Essentials of Regional Anesthesia

Second Edition

Alan David Kaye
Richard D. Urman
Nalini Vadivelu
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Electronic Supplementary Material can be downloaded from https://link.springer.com/book/10.1007/978-3-319-74838-2

ISBN 978-3-319-74837-5 ISBN 978-3-319-74838-2 (eBook)
https://doi.org/10.1007/978-3-319-74838-2

Library of Congress Control Number: 2018936917

Printed on acid-free paper

This Springer imprint is published by the registered company Springer International Publishing AG part of Springer Nature
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

For my wife Kim Kaye, M.D.,
for her patience, love, and wisdom
in raising our two wonderful children,
Aaron and Rachel.

Alan David Kaye

For my mentors, students, and trainees
for their inspiration; my parents,
Dennis and Tanya; my wife,
Zina Matlyuk-Urman, M.D.; and my children,
Abigail and Isabelle.

Richard D. Urman

For my parents;
my husband, Muthu; my sons, Gopal
and Vijay; and all my colleagues and friends
for their constant encouragement.

Nalini Vadivelu

Foreword

It is an enormous honor to pen this Foreword to *Essentials of Regional Anesthesia* in light of my following in the footsteps of the inimitable Professor P. Prithvi Raj, the author of the last edition's opening statement. In that regard alone, I am humbled to be next in line to comment upon the value of this extremely timely addition to our regional anesthesia armamentarium, which presents a fresh look at the evolving and expanding world of regional anesthesia.

I became immersed in the world of regional anesthesia when I sat in on Dr. Alon Winnie's address to the American Society of Anesthesiologist's Annual Meeting detailing his unique perspective on brachial plexus anesthesia in the early 1980s. Until that time there were only two regional anesthesia textbooks in common usage by clinicians, *Regional Block* by Dr. Daniel C. Moore from the Virginia Mason Medical Center in Seattle and *Neural Blockade, Pain Management* by Dr. Phil Bridenbaugh from Cincinnati along with Dr. Michael Cousins from Australia. From the moment I listened to Dr. Winnie and going forward I knew that there was no greater application of art to science in all of medicine than that presented by the challenge of providing discrete, localized, and well-circumscribed neural blockade to isolated areas of the body without engendering the trespass associated with use of general anesthesia, which up to that time continued to have a somewhat prohibitive incidence of grave consequences associated with its use. Coincidentally, while general anesthesia was about to become conspicuously safer with the advent and routine use of capnography and pulse oximetry, paradoxically instead of relegating regional anesthesia to the wayside, the popularity and utility of regional soared in the 1980s and beyond. This coincided with its own renaissance of sorts, including the practical identification that sound waves directed at a target or bundle of nerves while performing a block could enhance safety and efficacy of the procedures. In addition to safety and efficacy, ultrasound guidance empowered the meek and timid, who previously would have avoided regional block at all costs, and changed regional anesthesia from being a formidable and exclusionary practice limited to a mere few experts, to a practice that could be readily handed down from mentor to mentee with little fanfare and with extremely shallow learning curves. This is true particularly when contemporary training in regional is compared to the regional block training of the past, which relied heavily upon mastery of oftentimes imprecise percutaneous landmarks and the uncertainties of electrical neural stimulation.

Alon Winnie, like Prithvi Raj, was part of the renaissance of regional anesthesia, when in 1975 they partnered (along with Harold Carron, L. Donald Bridenbaugh, Jordan Katz) to resurrect the long-defunct American Society of Regional Anesthesia (ASRA; now known as the American Society of Regional Anesthesia and Pain Medicine), in anticipation of the resurgence of interest in regional anesthesia and pain management, recognizing that the two concepts are conjoined twins and always have been. It is no surprise and no coincidence that the great leaders of regional anesthesia have been and likely always will be experts at both pain management and in regional peripheral and neuraxial blockade. In that regard, Drs. Winnie and Raj; John Bonica and Daniel Moore; Gabor Racz and Steve Waldman; Michael Cousins and Phillip Bridenbaugh, among others, have each been considered to be world leaders not only in the domains of regional anesthesia but also in the management of acute and chronic pain conditions.

In 1999, while authoring a textbook chapter with Dr. Winnie for a novel text, *Pain Management and Regional Anesthesia in Trauma*, I was intrigued to note that Alon had included eight "postulates" concerning regional anesthesia and the expected benefits of peripheral nerve blocks, particularly when compared to use of general anesthesia or central neuraxial (spinal and epidural) types of blocks. Among his statements touting the major advantages of regional, he stated that it would...

- Provide Superior Postoperative Analgesia to Oral or IV Opioid Analgesics
- Improve Rehabilitation Efforts (due to analgesia)
- Lead to Decreased Perioperative Nausea and Vomiting (Less Opioid Use)
- Provide for Faster Emergence and Recovery
- Encourage Earlier Mobilization (Unilateral Block)
- Unilateral, Postganglionic Sympathetic Block with Less Associated Trespass
- Contribute to Faster Outpatient Discharge
- Have Extended Benefits via Continuous Catheters

In 1999, I was hesitant to include these bold statements into the body of a work which would be widely accessible to the masses and which would contribute to our enduring materials on regional anesthesia. Nevertheless, as usual he prevailed, and the result was that each and every one of these prescient proclamations of his did indeed come to fruition over time, affirming that this regional anesthesia business was "no humbug" indeed!

The use of regional anesthesia has become a centerpiece in our war against overuse of opioid analgesics. Providing profound analgesia to our patients reduces their need for consuming narcotics while also helping them bridge the time from surgical or procedural trespass to the safety of their homes. There is an abundance of clinical and experimental evidence available that unequivocally demonstrates these essential attributes of these techniques and which is a furtherance that opioid sparing, a mandate that all practicing clinicians must confront, can reasonably and reliably be accomplished by the use of regional block techniques using local anesthetics with and without adjunctive medications.

The present reference work is an affirmation of the concepts established long ago by the great leaders of regional anesthesia which has been succinctly presented in a manner most conducive to enhancing the educational process. Carefully organized into eight sections and expanded from the previous edition's 31 chapters to 39 chapters, this reference work should become a compulsory reading for anyone interested in expanding their horizons on all topics related to regional anesthesia, including basic and advanced constructs and incorporating the most up-to-date issues confronting those engaging in regional anesthesia practice. Among these are the considerations related to anticoagulation use; use of regional in acute settings such as the trauma patient; preemptive analgesia and the prevention of chronic postoperative pain; and the newer and more novel techniques of regional block popularized by the expansion of anatomical principles identified with the veritable explosion in ultrasound use.

Dr. Alan David Kaye has followed the pathways blazed by our great forefathers in combining his passions for pain management with those of regional anesthesia. After all, the lessons of the past have been transmitted to him through his unrivaled mentoring at the hands of Professors Racz and Raj, among others. These lessons permeate throughout this unique reference work, and whether the reader is a true regional anesthesia expert, is a first-time novice intent on becoming a master, or is merely a curious scopophiliac who wishes to expand their horizons, this book will provide a wonderful passageway to accomplishing these goals. With great pride, I too have made my humble contributions, and look upon this task as a most joyous triumph of scholastic achievement.

Chicago, IL, USA Kenneth D. Candido, MD

Preface

We are proud to present this updated 2nd edition of our popular practical guide to regional anesthesia techniques. The practice of regional anesthesia has undergone tremendous evolution in the past few decades. Until recently, older "blind" techniques were taught, and successful regional anesthetics were typically limited to a few extraordinary clinicians in each department of anesthesia worldwide. Ultrasound, electrical stimulation, fluoroscopy, and continuous catheters have contributed to a revolution in the fields of regional anesthesia and pain management. Advances in technology have changed these fields significantly, resulting in the development of formal regional anesthesia and pain fellowships. In addition, there is an increasing emphasis on regional anesthesia techniques for acute pain control in order to promote better patient outcomes.

The present field of regional anesthesia has challenged not only new residents and fellows but also older practicing anesthesiologists and other specialists to learn these new techniques and technologies in their clinical practices. Excellence and versatility in regional anesthesia can provide the means by which we better manage acute and chronic pain. Modern regional anesthesia provides hope and optimism for the comfort of future generations of patients afflicted with a wide array of medical conditions.

One of the strategies in creating this updated, evidence-based 2nd edition of *Essentials of Regional Anesthesia* was to make it practical for the clinician. To that end, we have recruited regional anesthesia experts, as well as fellowship directors and their fellows, as authors for most of the chapters in this book. We also requested that authors identify clinical pearls and help us create a databank of questions for trainees to facilitate learning of the subject material. The editors of the book agree that this has been a challenging but rewarding project. As with the first edition, we have endeavored to present the material with clarity and conciseness. Our goal has been a book of practical applicability for the anesthesia provider and others who would like to learn more about the field. Best of luck to each of you as you develop your clinical practices in regional anesthesia.

New Orleans, LA, USA — Alan David Kaye
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Part I

General Principles of Regional Anesthesia Practice

General Considerations for Regional Anesthesia Practice

1

Svetla Kurteva, Iyabo Olubunmi Muse, and Karina Gritsenko

Introduction

Setting up a regional anesthesia service requires a reliable and consistent product as well as a sound business plan. Technological advances in nerve stimulation, ultrasound guidance, and perineural catheters have led to rapid growth in the number and types of peripheral nerve block procedures available to regional anesthesia practitioners. Starting a new regional anesthesia program potentially adds monetary value to a facility's perioperative services by improving the quality of postoperative analgesia and recovery from surgery, thereby reducing perioperative costs and offering a competitive advantage over other surgical facilities. From the patient's perspective, a regional anesthesia program provides nonmonetary value by preventing pain and reducing the risk of nausea and vomiting after surgery. The goal of this chapter is to provide initial guidance to the anesthesiologist interested in starting a new regional anesthesiology and acute pain program.

While the argument in favor of developing a regional anesthesia program in terms of nonmonetary value is convincing in and of itself, determining monetary value is vital to initiating any new program. There are start-up costs to consider, and expected revenues are typically delayed. How does the individual anesthesiologist convince his or her own anesthesiology group or hospital administrators that a new service that provides peripheral nerve blocks and acute pain management is worth the investment? In this era of cost-effective healthcare delivery, all medical institutions, academic and private, are under similar financial pressures. There is a new plethora of literature beginning to show evidence for both regional anesthesia and acute pain services that provide benefit and cost efficiency even with the added investment [1–4]. For example, in a study investigating cost-effectiveness of ultrasound-guided interscalene nerve blocks in comparison with general anesthesia for arthroscopic shoulder surgery, Gonano et al. found that there were total cost savings as well as reduced anesthesia-related workflow (OR emergence and anesthesia control times, PACU time, etc.) [3]. For academic centers, there is an important primary educational mission that must be achieved in the setting of financial sta-

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_1

bility. That being said, regional anesthesia and acute pain medicine are established required components in the fund of knowledge required to become a board certified anesthesiologist and should be included in the training program of all anesthesiology residents, regardless of cost.

Reasons for Starting a Regional Anesthesia Program

As the definition of "outpatient" surgery continues to broaden, patients previously hospitalized for the same surgeries several years ago are now scheduled for discharge on the day of surgery and benefit from improved perioperative and postoperative analgesia. Lengthy hospitalizations for some major surgeries are gradually progressing to overnight admission. For example, total joint replacement postoperative protocols have been refined to the point that it is currently feasible to practice same-day discharge or short-stay admission for appropriate cases [5–7].

The causes of prolonged recovery after scheduled ambulatory surgery have been studied and are multifactorial. In addition to the type of surgery, these factors include postoperative nausea and vomiting, as well as pain following general anesthesia [8]. The proper application of regional anesthesia techniques in the ambulatory setting can minimize or avoid these common side effects and decrease the time required for patients to meet predetermined discharge criteria [9–11].

According to the results of a survey conducted by Dr. Macario and colleagues, nausea, vomiting, and pain are among the main side effects patients prefer to avoid after anesthesia [12]. It is increasingly important to consider patient preferences in the current healthcare system. Ensuring high patient satisfaction will likely lead a patient to return to a particular healthcare system for future surgical services if needed and potentially result in new referrals. Patients should be considered consumers with the right to the highest-quality service, and they have choices regarding their healthcare. Anesthesiology groups or hospitals who offer regional anesthesia services can employ marketing strategies to outcompete other anesthesiology groups and hospitals that do not offer similar services.

Regional anesthesia is not "one size fits all" anesthesia. The combination of specific peripheral nerve block techniques can produce anesthesia and postoperative analgesia that is nearly as selective as the surgical procedure itself. In a large case series, Klein and colleagues have demonstrated that peripheral nerve blocks in the ambulatory setting lead to reductions in perioperative intravenous opioid use and high patient satisfaction and can be used in conjunction with oral opioid analgesics [13]. For the non-orthopedic patient, other regional anesthesia techniques may offer similar advantages [14–16].

In order to extend the duration of site-specific pain relief beyond the immediate perioperative period, continuous peripheral nerve blocks (CPNB) and perineural local anesthetic infusions are currently used for a wide variety of surgeries in the ambulatory environment [17–24]. Randomized, placebo-controlled studies have conclusively demonstrated significant reductions in patient-reported pain after shoulder, foot, and distal upper extremity surgery as a result of CPNB [22–25]. By providing superior analgesia at home, a CPNB effectively reduces the need to hospitalize patients for pain control. Adjuvants to local anesthetics have also shown to produce reliable prolongation of analgesic duration and are being used more often in ambulatory surgery. A recent meta-analysis of randomized trials has demonstrated that the addition of dexamethasone to long-acting local anesthetics the sensory brachial plexus block by an average of 9.5 h [26]. This length can be increased even further in a dose-dependent fashion [27]. In addition, liposomal bupivacaine, which produces an extended release local anesthetic, has become increasingly popular in recent years, specifically for local infiltration, although there is interest in the use of Exparel with peripheral nerve blocks. Broader application of these advanced regional anesthesia techniques has contributed to the growing interest in ambulatory total joint replacement [6, 7, 28, 29].

Bringing Your "Product" to Market

Developing a new regional anesthesia program is like inventing a new product. In addition to ensuring the consistency and reliability of the product, the prospective clientele and demand for the product should be considered. Applying this analogy, it is essential to identify potential customers and their needs. The *patient* and *patient's family* are the most important customers, and improvements in the overall quality of postoperative recovery resulting from regional anesthesia offer meaningful benefits to them. S*urgeons* are clearly important customers to any anesthesiology practice, hospital, or surgical center. Surgeons' concerns regarding failed blocks, complications, and case delays must be addressed [30], and surgeons may rally behind a regional anesthesia program that improves operating room efficiency [31]. When a regional anesthesia program gains surgeon support, the dividends multiply. Since surgeons establish rapport with patients several days or weeks before the surgery, their recommendation in favor of regional anesthesia is likely to lead to higher utilization of these services.

Despite the common belief that surgeons represent the major obstacle to developing a regional anesthesia service, it is often the *anesthesiology practice* itself that requires the most convincing. Since the initial investment in money, training, and personnel is incurred by the practice, there must be tangible benefits from implementing a new service. The ability to recoup this cost is dependent on the model of regional anesthesia practice implemented and the anticipated volume of nerve block procedures. For a busy orthopedic hospital, a new regional anesthesia service may generate enough new revenue to support the salary of one dedicated regional anesthesia provider per clinical day. However, this type of "block room" model is not appropriate to all practices, and every regional anesthesia service should be developed with consultation from the individual practice manager and billing service to ensure proper financial planning.

The hidden customer when developing a new regional anesthesia service is *hospital administration*. Often, hospital administration receives the requests to purchase the initial capital equipment (e.g., nerve stimulators, regional block carts, and ultrasound machines). Although these overhead expenses may be large, they tend to be *fixed* and nonrecurring [32]. Administrators must be assured that this investment will result in quality improvement and possibly financial return, either from cost containment (e.g., reductions in hospital stays or nursing labor), increased revenue (e.g., attracting new patients or insurance contracts), or both. Some additional recurrent labor-intensive aspects may include support staff to maintain equipment and supplies and pharmaceutical staff to supply medications for the perioperative period. It is important to note that cost savings generated by regional anesthesia and perioperative pain management tend to benefit the hospital financially and not the individual anesthesiologist or anesthesiology group. In light of this fact, it is reasonable and expected to request the financial support of administration when developing a comprehensive perioperative pain management service employing regional anesthesia techniques.

Determine if Regional Anesthesia Will Save Money

Strategies to decrease the total cost of providing perioperative services must focus on *variable* costs despite the fact that the overwhelming majority of costs associated with any surgical procedure are *fixed* [32].

For ambulatory surgery, the use of peripheral nerve block techniques leads to increases in PACU bypass and shorter time to discharge compared to general anesthesia [9–11, 33, 34]. Cost savings can result from decreased recovery time associated with PACU bypass by reducing nursing time and labor [1]. In a high-volume orthopedic surgery center specializing in regional anesthesia for anterior cruciate ligament reconstruction (ACLR), the odds of bypassing PACU are nearly four times higher when the patient receives regional anesthesia compared to general anesthesia [1]. In this setting, PACU bypass has

been shown to reduce the average per-patient cost by approximately $420 (USD) [1].

The development of step-down (Phase II) recovery units alters traditional PACU nurse staffing since these units are not considered critical care units, and patients classified as Phase II may be staffed in a ratio of four or five patients to one nurse. By increasing PACU bypass from 0 to 40% with regional anesthesia, PACU staffing may be reduced by one full-time nurse in a typical surgical center employing full-time staff paid hourly with frequent overtime [35]. In practical terms, this increase in PACU bypass can reduce overtime and therefore ease the financial burden on short-staffed hospitals and surgery centers.

Shortening hospital stays and minimizing unplanned hospital admissions for outpatients are potential sources of cost savings. Cost containment is essential when dealing with insurers that only reimburse a fixed amount per surgical procedure regardless of charges. For scheduled outpatients undergoing ACLR, unplanned admission to hospital adds $385 (USD) per patient [1]. When employing femoral CPNB for scheduled surgical procedures requiring inpatient care such as total knee arthroplasty (TKA), patients may meet criteria for discharge home sooner compared to conventional analgesic techniques [5].

A major advantage of CPNB is that it may be provided on an outpatient basis unlike intravenous opioid patient-controlled analgesia or epidural analgesia. Shortening hospital stays for TKA by transitioning to outpatient femoral CPNB leads to a 34% decrease in overall hospitalization cost (US$ 2682) due mostly to room and board savings [2]. According to data gathered from the National Hospital Discharge Survey, the rate of primary TKA in the United States nearly tripled between 1990 and 2002 to over 400,000 per year and is expected to increase [36]. Given this trend, the potential cost savings afforded by employing a regional anesthesia service for just TKA is impressive; extrapolating these data to all joint replacements and other major surgeries generates a staggering figure that should change surgical practice.

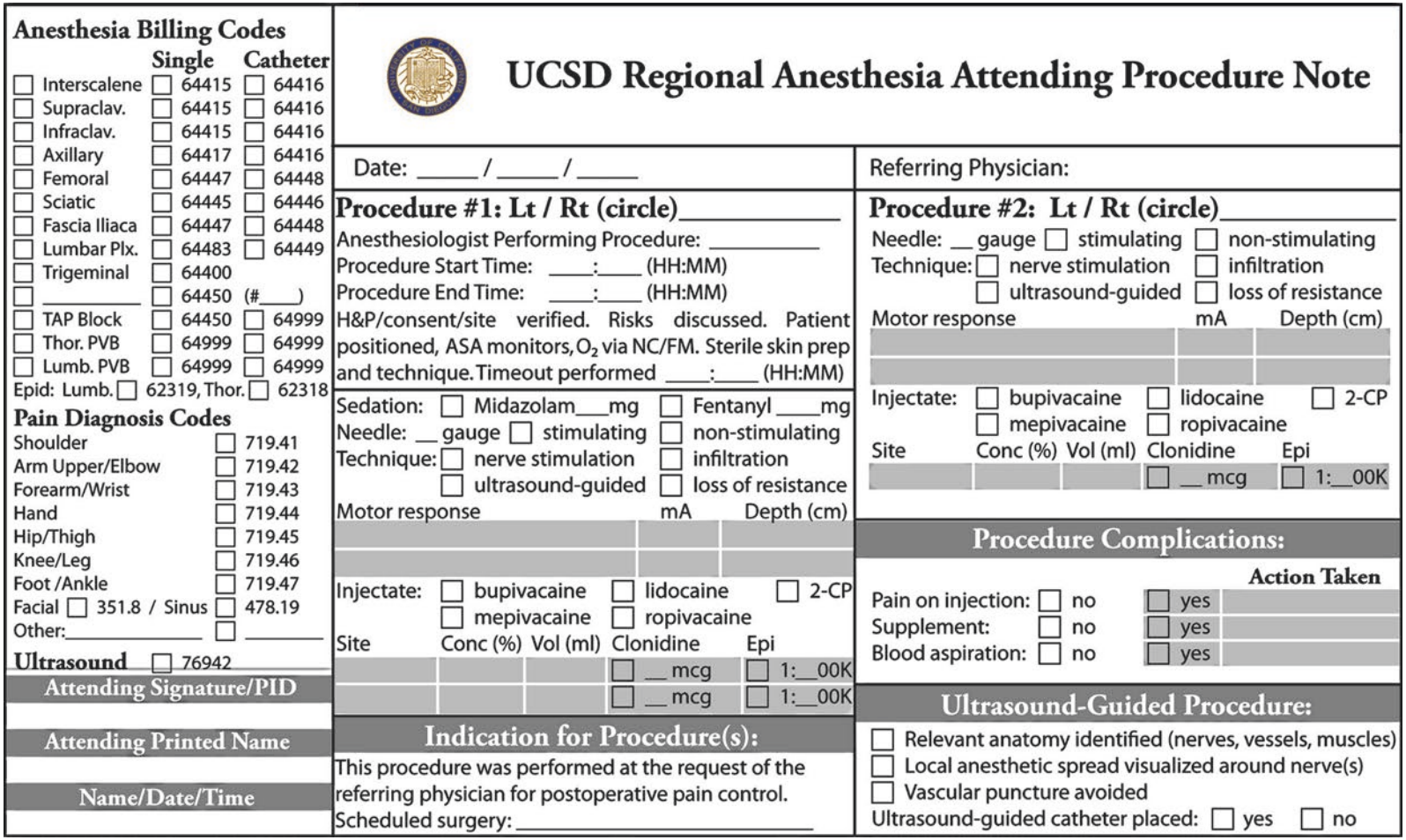
However, the decision to discharge a patient home early after TKA must be agreed upon by the entire healthcare team, and proper patient selection is essential. For patients to be discharged home with a femoral CPNB after joint replacement, he or she must have a caretaker 24 h/day, an established outpatient physical therapy program, and close follow-up by a healthcare provider [5].

Bill Effectively for Professional Fees

The *charge* is the sum listed on a bill for services rendered. Taking a hands-on approach to billing professional fees will maximize charges and lead to revenue generation for the individual anesthesiologist and anesthesiology group. Billing strategies should be constantly reevaluated as regulations and procedural codes change, and actual billing practices will vary based on the individual institution, geographic location, payor mix, and even negotiations with individual insurance providers. In addition, in the current political climate, insurance factors and finances may change further depending on any planned changes for the American health insurance system.

Use Appropriate Current Procedural Terminology (CPT) Codes and Modifiers

Anesthesia billing services should not be expected to interpret our handwritten procedure notes and deduce the appropriate codes for regional anesthesia. For example, simply writing "infraclavicular block" on an anesthesia record may not be correctly coded as "64415—brachial plexus block" unless the anesthesiologist educated the billing service staff in brachial plexus anatomy. To avoid confusion, consider doing your own coding on a standardized procedure note (Fig. 1.1). If using an electronic medical record, adding charges may become easier and faster. As an example, in our academic institution, every regional anesthesia provider completes their own coding electronically, directly upon completion of the nerve block and procedure note to minimize human error and missing documentation. In addi-



Anesthesia Billing Codes

	Single	Catheter
☐ Interscalene	☐ 64415	☐ 64416
☐ Supraclav.	☐ 64415	☐ 64416
☐ Infraclav.	☐ 64415	☐ 64416
☐ Axillary	☐ 64417	☐ 64416
☐ Femoral	☐ 64447	☐ 64448
☐ Sciatic	☐ 64445	☐ 64446
☐ Fascia Iliaca	☐ 64447	☐ 64448
☐ Lumbar Plx.	☐ 64483	☐ 64449
☐ Trigeminal	☐ 64400	
☐ _____	☐ 64450	(#____)
☐ TAP Block	☐ 64450	☐ 64999
☐ Thor. PVB	☐ 64999	☐ 64999
☐ Lumb. PVB	☐ 64999	☐ 64999

Epid: Lumb. ☐ 62319, Thor. ☐ 62318

Pain Diagnosis Codes

Shoulder	☐ 719.41
Arm Upper/Elbow	☐ 719.42
Forearm/Wrist	☐ 719.43
Hand	☐ 719.44
Hip/Thigh	☐ 719.45
Knee/Leg	☐ 719.46
Foot /Ankle	☐ 719.47
Facial ☐ 351.8 / Sinus	☐ 478.19
Other:_____	☐ _____

Ultrasound ☐ 76942

Attending Signature/PID

Attending Printed Name

Name/Date/Time

UCSD Regional Anesthesia Attending Procedure Note

Date: _____ / _____ / _____

Referring Physician:

Procedure #1: Lt / Rt (circle)_____

Anesthesiologist Performing Procedure: _____

Procedure Start Time: ____:____ (HH:MM)

Procedure End Time: ____:____ (HH:MM)

H&P/consent/site verified. Risks discussed. Patient positioned, ASA monitors, O_2 via NC/FM. Sterile skin prep and technique. Timeout performed ____:____ (HH:MM)

Sedation: ☐ Midazolam___mg ☐ Fentanyl ____mg

Needle: __ gauge ☐ stimulating ☐ non-stimulating

Technique: ☐ nerve stimulation ☐ infiltration ☐ ultrasound-guided ☐ loss of resistance

Motor response	mA	Depth (cm)

Injectate: ☐ bupivacaine ☐ lidocaine ☐ 2-CP ☐ mepivacaine ☐ ropivacaine

Site	Conc (%)	Vol (ml)	Clonidine	Epi
			☐ __ mcg	☐ 1:__00K
			☐ __ mcg	☐ 1:__00K

Indication for Procedure(s):

This procedure was performed at the request of the referring physician for postoperative pain control.

Scheduled surgery: _____

Procedure #2: Lt / Rt (circle)_____

Needle: __ gauge ☐ stimulating ☐ non-stimulating

Technique: ☐ nerve stimulation ☐ infiltration ☐ ultrasound-guided ☐ loss of resistance

Motor response	mA	Depth (cm)

Injectate: ☐ bupivacaine ☐ lidocaine ☐ 2-CP ☐ mepivacaine ☐ ropivacaine

Site	Conc (%)	Vol (ml)	Clonidine	Epi
			☐ __ mcg	☐ 1:__00K

Procedure Complications:

			Action Taken
Pain on injection:	☐ no	☐ yes	
Supplement:	☐ no	☐ yes	
Blood aspiration:	☐ no	☐ yes	

Ultrasound-Guided Procedure:

☐ Relevant anatomy identified (nerves, vessels, muscles)

☐ Local anesthetic spread visualized around nerve(s)

☐ Vascular puncture avoided

Ultrasound-guided catheter placed: ☐ yes ☐ no

Fig. 1.1 Example of a separate regional anesthesia procedure note

tion, our institution mandates education regarding billing and coding as a component of the regional and acute pain medicine rotation and as part of the system-based practice component in Accreditation Council for Graduate Medical Education (ACGME) core competencies. We hope this is a useful skill that is useful in their transition from residency to full-time employment.

Common CPT codes for regional anesthesia are listed in Tables 1.1 and 1.2. Be aware that CPT codes are revised periodically, so make it a habit to review the updated CPT codes every year. When billing for nerve block procedures performed for postoperative pain management, include the distinct procedure modifier −59 to distinguish the block from the intraoperative anesthetic technique (e.g., 64416–59 for a brachial plexus catheter placed for postoperative pain management) [37]. This is especially important when the same provider performs the nerve block and the intraoperative anesthesia. Some additional recent examples are listed below.

New ultrasound-guided transversus abdominis plane (TAP) block codes were introduced as of January 1, 2016. These new codes listed in Table 1.3 delineate between single and continuous infusion and unilateral versus bilateral procedures. Rectus sheath blocks and other TAP block variations can be billed using new codes for TAP blocks. It is important to note that these CPT codes include imaging as part of a bundle, therefore eliminating a separate code for ultrasound guidance. Ultrasound-guided ilioinguinal and iliohypogastric TAP blocks performed for inguinal hernia repair can be charged for using the separate 64425 CPT code; this code, however, does not include imaging or distinguish between unilateral and bilateral.

New ultrasound-guided paravertebral block (PVB) codes (Table 1.4) were also introduced in 2016. Once again these codes delineate between single injection and continuous infusion and

Table 1.1 Commonly used CPT codes (2017) and suggested unit value charges (ASA relative value guide) for single-injection nerve blocks

CPT code	Injection site	Units charged
64417	Axillary	8
64415	Brachial plexus	8
64447	Femoral	7
64445	Sciatic	7
64483	Lumbar plexus	9
64450	Peripheral nerve other	5
64999	Unlisted	N/A

Table 1.2 Commonly used CPT codes (2017) and suggested unit value charges (ASA relative value guide) for continuous peripheral nerve blocks

CPT code	Catheter site	Units charged
64416	Brachial plexus	13
64448	Femoral	12
64446	Sciatic	12
64449	Lumbar plexus	12
64999	Unlisted	N/A

Table 1.3 Ultrasound-guided transversus abdominis plane block CPT codes (2017)

CPT code	Description
64486	Transversus abdominis plane (TAP) BLOCK (abdominal plane block, rectus sheath block) *unilateral*; by injection(s)—includes imaging guidance, when performed
64487	Transversus abdominis plane (TAP) BLOCK (abdominal plane block, rectus sheath block) *unilateral*; by continuous infusion(s)—includes imaging guidance, when performed
64488	Transversus abdominis plane (TAP) BLOCK (abdominal plane block, rectus sheath block) *bilateral*; by injection(s)—includes imaging guidance, when performed
64489	Transversus abdominis plane (TAP) BLOCK (abdominal plane block, rectus sheath block) *bilateral*; by continuous infusion(s)—includes imaging guidance, when performed

Table 1.4 Ultrasound-guided paravertebral block CPT codes (2017)

CPT code	Description
64461	Paravertebral block (PVB) (paraspinous block), thoracic; single injection site—includes imaging guidance, when performed
64462	Paravertebral block (PVB) (paraspinous block), thoracic; second and any additional injection site(s)—includes imaging guidance, when performed
64463	Paravertebral block (PVB) (paraspinous block), thoracic; continuous infusion by catheter—includes imaging guidance, when performed

include imaging guidance. When more than one injection is given, the code 64462 should be used as an add-on code and billed together with 64461.

Create a Separate Procedure Note

When nerve blocks are performed for acute postoperative pain, separate from intraoperative anesthetic care, it is helpful to develop a distinct procedure note to document regional anesthesia techniques [38, 39]. The use of a different form physically separates the regional anesthesia procedure documentation from the documentation associated with the intraoperative anesthetic care and can even include common billing codes (Fig. 1.1). The procedure note must include indication for the procedure (acute postoperative pain) as well as the referring physician. When designing new forms, involve your managers to ensure compliance with hospital policies and mandates from regulatory agencies. When using a separate form to document and bill for nerve blocks placed for postoperative pain management, the anesthesia record should not indicate "nerve block" as being part of the intraoperative anesthetic management. However, nerve blocks performed as the intraoperative anesthetic technique should be billed as such and not billed separately for postoperative pain management.

Document Physician Referral for Pain Management Consultation

When performing regional anesthesia procedures for postoperative pain management, physician referral must be documented as well as the indication for the procedure. This also serves as the request for postoperative pain management consultation.

Billing for Ultrasound Guidance

When real-time ultrasound guidance is used for performing a nerve block, CPT code 76942 should be submitted as a separate charge. Proper utilization of this code requires documentation of the ultrasound image taken during the procedure as well as specific interpretation of findings. In some institutions, ultrasound images are able to be electronically transferred to become part of the computer record. Another way to fulfill this requirement is to print a copy of the ultrasound image and attach it to the procedure note, or, if the procedure note is electronic, to simply include the ultrasound image in the patient's paper chart. For completeness, it is important to annotate the ultrasound image with identification of relative anatomy (nerves, muscles, bones, arteries, veins, etc.) and document needle placement and spread of local anesthetic solution. CPT code 76942 takes into account both technical and professional components associated with real-time ultrasound use. The technical component includes equipment storage and maintenance costs related to owning an ultrasound machine, while the professional component takes into account the physician's application and interpretation of ultrasound only. If the ultrasound equipment is owned by the hospital and not by the anesthesiologist, the modifier −26 should be added to CPT code 76942 in order to limit the ultrasound charge to professional fee only. Without the modifier, the 76942 code includes a technical component charge for both equipment storage and maintenance.

Billing for Acute Pain Management and Daily Follow-Up of Continuous Peripheral Nerve Blocks

Multimodal postoperative analgesia as part of integrated clinical care pathways has led to the evolution of some regional anesthesia services into acute pain management services [40]. At times surgeons consult the regional anesthesiologist or designated acute pain medicine provider for acute pain management issues of challenging postoperative patients. Examples of consultation requests include evaluation of a hospitalized patient with severe obstructive sleep apnea with postsurgical pain who is sensitive to systemic narcotics or a patient on high doses of daily opioids for chronic pain who after surgery has difficulty transitioning from intravenous to oral pain medications. The anesthesiolo-

gist who evaluates these patients can use the CPT codes 99251–99254 to bill for the encounter, depending on the complexity of the case. In addition, patients with continuous peripheral nerve block catheters need to be evaluated daily for adequacy of pain control and potential catheter-related complications. Prior to January 2009, the CPT code for patients with such catheters included 10 days of routine follow-up management. Since then, the follow-up care has been unbundled, and the daily evaluation and management of such patients is now also a billable charge using 99231–99233 for established in-hospital consults.

Foster a Team Approach to Billing

Developing a good relationship with the people that send out claims and negotiate with insurance companies is essential. If regional anesthesia is new to an anesthesiology practice, meet with the billing service manager in person to clearly explain what regional anesthesia is, why it is performed, and the volume and variety of procedures that are expected. Since they are not directly involved in the provision of healthcare, it may be worthwhile to have the billing manager observe regional anesthesia procedures and witness patients' postsurgical recovery, as long as patient confidentiality can be preserved. The more the billing service manager understands the indications and benefits of regional anesthesia, the better he or she is equipped to negotiate charges and fair payment for regional and acute pain services. Similarly, when developing new aspects to any computer system, such as updating an electronic medical record, those who will build the technology and the supervisors of these systems are essential team members who must understand the flow and the needs of the regional and acute pain medicine services.

Organize an Efficient Regional Anesthesia Service

To make a regional anesthesia service successful, many pieces must fit together. All of the customers must be satisfied, including the patients, surgeons, and administrators. While patients may be satisfied by the superior pain control and improvement in the quality of post anesthesia recovery afforded by peripheral nerve blocks and CPNB, surgeons and administrators will also demand efficiency. In the busy outpatient surgery setting, the addition of a new regional anesthesia service does not have to detract from perioperative efficiency and may, in fact, contribute positively [31].

Staff and Training

Regardless of the regional anesthesia service model employed, specially trained personnel in regional anesthesia techniques are necessary. When developing a service that utilizes specific procedural skills and advanced technology (e.g., surface ultrasound and CPNB), hiring and training staff with these skills is the most important first step. This staffing model includes not only the anesthesiology physicians but can include NPs or nurses who specifically participate in the perioperative environment with the block team. The use of ultrasound guidance for regional anesthesia has become standard of care in many practices and may offer advantages in terms of procedural efficiency [41–44] but requires dedicated training [45, 46]. In the past, not all anesthesiology residency training programs have provided adequate training in regional anesthesia techniques, but this is becoming more commonplace [47–49] as the development of subspecialty regional anesthesia services at academic hospitals have led to increased volume and complexity of procedures performed by residents [50, 51]. For private practice anesthesiologists, skills can be learned from partners who have received specialized training or continuing education courses that fulfill certain educational parameters [45], such as the American Society of Anesthesiologist (ASA) and American Society of Regional Anesthesiology accredited meetings.

In addition, recognizing this need for further formalized training, the ACGME in October 2016 recognized an established set of guidelines

for a Regional Anesthesiology and Acute Pain Medicine Fellowship as the newest accredited subspecialty fellowship within anesthesiology. In 2016–2017, this will be the first year programs are able to apply for accreditation to the ACGME. Specific guidelines outlining fellowship training in regional anesthesiology and acute pain have been developed [52]. Graduates of these fellowship programs are expected to possess a higher knowledge and level of proficiency with regional techniques, be more efficient, be able to tackle challenging patient anatomies, and be comfortable with advanced technology with standard expectations as created through the ACGME and the ASA. These future fellowship trainees should be the experts at the multimodal approach to pain prevention and management. Their priority will be to aggressively treat severe acute pain when it occurs in the postsurgical period and prevent it from becoming chronic. Moreover, these highly trained regional anesthesiology graduates would be positioned to become physician leaders who may run acute pain medicine teams, educate residents and anesthesiology colleagues in regional techniques, and advocate the advantages and benefits of regional anesthesia among surgeons, hospital administrators, patients, and the general public. Finally, the development of subspecialty regional anesthesia fellowships and services at academic hospitals may lead to increased volume and complexity of procedures performed by residents [50, 51] and further advances in research and academia.

Consistency

By storing all necessary supplies in one central and convenient location, either a "block room" or regional anesthesia cart, performing peripheral nerve blocks and CPNB procedures can be as efficient as possible. All practitioners should conform to standardized supplies to simplify ordering, storage, and preparation of equipment associated with each procedure. This allows both for consistency in care and a more familiar environment to all participants in care.

All Team Members Working Together Toward Efficiency

In addition to having an efficient block room, it is imperative for all members of the team to be on time. All preoperative assessment must be in place, including the anesthesiologist assessment, the regional anesthesiology and acute pain medicine team assessment, and surgical paperwork to be complete. In addition, the surgeons must arrive at the hospital early to sign consent and mark the patient in order to avoid delay in first case starts. It is also helpful to improve operating room efficiency, if the surgeon marks the patient early between the cases thus to give the regional team ample time for block placement.

In many cases, patients also can attend an informational session on a day prior to surgery to improve their understanding of what will happen on the day of surgery and to optimize their own comfort level and understanding of the process.

Performing a Regional Nerve Block

When regional anesthesia procedures are performed in a regional anesthesia induction area or "block room" while the preceding case is still in the operating room, anesthesia-controlled time is reduced compared to both general anesthesia and nerve blocks performed in the operating room [31, 53]. This parallel processing model employing a block room may not work for every group practice or institution as it depends on the availability of resources and personnel [54]. For private anesthesia groups that function as a care team utilizing anesthesiologists and nurse anesthetists or academic anesthesiology departments with residents, a block room model is both feasible and recommended [39].

A "block room" does not have to be a dedicated enclosed space and may be created out of existing clinical space in a preoperative holding area or postanesthesia care unit. At a minimum, this space should contain standard ASA monitoring, an oxygen source, and resuscitation equipment in addition to regional anesthesia supplies

Fig. 1.2 Example of a regional anesthesia induction area or "block room" with standard ASA monitoring, oxygen source, resuscitation equipment, and regional anesthesia supplies

(Fig. 1.2). In addition, a portable regional anesthesia cart is advantageous because it can be transported from location to location when necessary. Since local anesthetic systemic toxicity remains one of the most devastating complications of regional anesthesia, it is important that lipid emulsion bags are readily available in the "block room" and clearly labeled with instructions for use in case of an emergency, as well as other standard emergency drugs.

Planning ahead will lead to effective time management on the day of surgery. Developing a reliable service is optimal because surgeons can discuss postoperative analgesia options including regional anesthesia with the patient in their clinics prior to scheduling surgery. A facility with a preanesthetic evaluation clinic can introduce the concept of regional anesthesia techniques for postoperative pain management. Written educational materials on regional anesthesia procedures for particular surgeries as well as answers to frequently asked questions may be printed or made available on Internet websites to help disseminate information prior to the day of surgery. Educating patients in advance saves time and minimizes patient anxiety on the day of surgery. Otherwise, each practitioner can call his or her own patients prior to surgery to discuss specific nerve block techniques. During this preoperative phone call, patients scheduled for surgery amenable to regional anesthesia techniques should be asked to check in at least 2 h prior to their scheduled surgery start time. It is also necessary to identify potential regional anesthesia patients to preoperative nursing and clerical staff so they may be triaged quickly through the admissions process and provide adequate time to perform procedures.

Diligent Follow-Up Is Key

Inserting the needle into the right place is the easiest part of regional anesthesia. The difficult part is developing an effective system of follow-up which is necessary for any program to succeed.

For example, in one survey of outpatients with perineural catheters, it was discussed that once-daily telephone calls from a healthcare provider is the optimal amount of contact, and 98% of patients are comfortable removing their own perineural catheters [55]. Only 4% would have liked a provider to remove his or her catheter, while 43% would have been satisfied with only written instructions and no person-to-person contact [55]. This may be a specific patient population but represents one collected opinion from one institution. Regardless, those patients who have received an ambulatory CPNB or a prolonged nerve block should be discharged with a caretaker (e.g., friend or family member) and a plan for postoperative care.

Patients who have received an ambulatory CPNB should receive specific written instructions for their portable infusion device as well

as provide a demonstration of the device function for the patient preoperatively. Written instructions should include expected CPNB issues (e.g., leakage and breakthrough pain) and contact information for a healthcare provider who will be available 24 h a day, 7 days a week. The patient should be followed on a daily basis until CPNB catheter removal, and each contact should be documented on a designated form [56].

Similarly, patients who had a single-injection peripheral nerve block with a long-acting local anesthetic in the ambulatory setting should only be discharged home after thorough education on the potential risks associated with a numb, insensate extremity. When patients lack pain sensation in a limb, they could easily physically injure it by inadvertently hitting or burning it. Placing a numb arm or leg in an awkward position can result in stretch or compression injury of a peripheral nerve or plexus. Patients who had blocks of the upper extremity should be discharged home with a protective sling. Patient who had femoral nerve blocks, for example, are of special concern since then can develop persistent quadriceps weakness leading to falls. These patients should receive written and verbal instructions not to bear weight on the affected extremity. When ready for discharge, they should be sent home with a knee immobilizer and crutches. Finally, all patients who undergo single-injection peripheral nerve blockade with long-acting local anesthetics in the ambulatory setting should receive a follow-up phone call the next day to assess for block resolution, adequacy of pain relief, and overall satisfaction with postoperative recovery.

For inpatients, a designated acute pain service provider or the anesthesiologist who performed the procedure should perform regular follow-up. Managing regional anesthesia patients, especially those with perineural catheters, is a team effort. A comprehensive clinical care team involving nurses, physical therapists, pharmacists, surgeons, and anesthesiologists can result in measurable patient benefits on a much broader scale [40].

A Guide to Team Building: Nursing Considerations in Regional Anesthesia

When it comes to developing positive physician–nurse interactions, an understanding of the basic tenets of modern nursing training is required. These are collaboration, approachability, autonomy, and education.

Collaboration fosters open communication with the staff [57] and prevents you, the physician, from feeling frustrated when managing nerve blocks and epidural catheters outside of the operating room. Collaboration with nursing management and education to appoint at least one nurse for each unit, covering all shifts, to be a regional-specific educator who can function as a resource to his or her fellow nurses can be a very useful aspect of care for the perioperative regional anesthesia patient. You can educate these appointed nurses initially with the information you feel is important (e.g., functional anatomy and physiology of nerve blockade, expected effects, and untoward side effects), and they can continue this education into their unit. This will build the bridge you need for open communication, avoidance of unnecessary pages, and mutual respect among team members. If a local resource can answer the questions for which you most commonly receive calls (e.g., catheter leakage and pain not covered by the block), it will be easier for everyone involved and lead to faster intervention for the patient when necessary.

Approachability. Make yourself approachable and allow nurses to put a face to your name. When performing inpatient rounds, talk to the nurses and develop a professional relationship. Make sure the nurses know that you *want* them to call you about your patients and why they should. Remember that nurses are at the patient's bedside 24 h a day, 7 days a week and are responsible for everything that happens to their patients. The two most common reasons that nurses call a physician are as follows: (1) they are familiar with the situation and suspect that something is wrong, or (2) they are faced with a new situation beyond their comfort level and are requesting guidance. Nurses expect that the physician is there to help

the patient and act as a guide when they are faced with unfamiliar clinical scenarios. Although there are times when contact may be inconvenient, it is important to respect the assessment of the nurse and discover the solution for the issue together. Later, you can address any underlying issues regarding education, so the nurses may become more confident in managing regional anesthesia patients in the future.

Autonomy defines modern-day nursing. Recent nursing graduates are not part of the generation that simply followed orders. Finding a way to approach new ideas with nursing that allows for empowerment and self-sufficiency will avoid pushback and encourage more forward progress. Let the nursing staff determine their own educational goals. For example, find out from the nurses how much they want to know about regional anesthesia procedures, patient management, and technology. Remember that simply demanding something *never* works. This is the best way to generate resistance from nursing.

Lastly, *education* is what ties the above concepts together. Education is the key to improving nurse–physician relationships, especially when implementing regional anesthesia into nursing practice [57]. Modern-day nursing thrives on autonomy, and in order to prevent resistance from the nursing staff, one must educate a nurse colleague to become most efficient and productive when providing care for the patient with nerve blocks and continuous catheters. Highlight the benefits of regional anesthesia in reducing nursing interventions [58]. Undoubtedly, the educational process will likely start with you, the physician, and then trickle down to nursing-specific education with dedicated nurse educators. Many hospitals throughout the nation already have pain resource nurses on individual units, and these nurses may be the ideal liaisons for you when disseminating regional anesthesia education.

Conclusion

Regional anesthesia techniques provide superior analgesia and can reduce the incidence of common side effects associated with postoperative recovery. However, an effective regional anesthesia product must offer benefits to all customers involved. For anesthesiologists starting a new regional anesthesia service, a hands-on approach is recommended to ensure the highest-quality patient care and strengthen relationships with surgeons and administrators. Emerging technology for ultrasound guidance in regional anesthesia and perineural catheter insertion has created a need for specialized training in these techniques. In addition to proper training, a successful regional anesthesia service requires a team effort and necessitates effective communication.

Acknowledgment The authors wish to thank Edward R. Mariano and Karley J. Mariano for their significant contribution to the prior edition.

Review Questions

1. Regarding the ways regional anesthesia provides "value" to an institution, all are true except:
   (a) Improving patient satisfaction with pain control and postoperative recovery
   (b) Creating a competitive edge versus other anesthesia groups and surgery centers
   (c) Reducing perioperative costs by decreasing the acuity of patients recovering from surgery and ensuring same-day discharge
   (d) Immediately generating new revenue
2. The cause of prolonged post anesthesia recovery is:
   (a) Postoperative nausea and vomiting
   (b) Acute postoperative pain
   (c) Multifactorial
   (d) Acute chronic pain
3. According to the study by Macario and colleagues, which side effect from anesthesia do patients most prefer to avoid?
   (a) Vomiting
   (b) Nausea
   (c) Pain
   (d) Gagging on the endotracheal tube

4. When considering regional anesthesia "customers," which of the following groups is most important?
   (a) Patients
   (b) Hospital administrators
   (c) Surgeons
   (d) Anesthesiology colleagues
5. Cost savings in the postoperative period attributable to regional anesthesia may result from all of the following except:
   (a) Reducing PACU length of stay
   (b) Avoiding unplanned hospitalization
   (c) Decreasing the patient to nurse ratio in PACU for bypass-eligible patients
   (d) Minimizing the need for pharmacologic interventions by PACU nurses
6. When designing the analgesic pathway for patients undergoing total knee arthroplasty, which of the following is false?
   (a) Intravenous patient-controlled opioid analgesia is typically administered on an inpatient basis.
   (b) Continuous nerve blocks may be managed effectively on an outpatient basis.
   (c) Epidural analgesia with local anesthetic solutions is most commonly maintained in the hospital setting.
   (d) None of the above.
7. Effectively billing for regional anesthesia procedures indicated for postoperative pain should involve which of the following?
   (a) Appropriate CPT coding.
   (b) Using a separate procedure note.
   (c) Including the distinct procedure modifier.
   (d) All of the above.
8. In a care team anesthesia delivery model, regional anesthesia may operate in the most efficient manner when:
   (a) Anesthesiologists are not familiar with regional anesthesia techniques
   (b) Equipment required for regional anesthesia is not centralized
   (c) Patients eligible for regional anesthesia are processed in "parallel"
   (d) Patients first learn about regional anesthesia on the day of surgery
9. When managing continuous nerve block catheters at home, which of the following is true?
   (a) Patients require a home nurse.
   (b) Patients must return to the hospital for catheter removal.
   (c) Patients should be called at home three times a day.
   (d) Patients should receive clear written and verbal instructions as well as contact information for a healthcare provider.
10. The clinical care team involved with managing patients with continuous regional anesthesia catheters should include:
    (a) Nursing
    (b) Pharmacists
    (c) Anesthesiologists
    (d) All of the above
11. Developing positive physician–nurse interactions when implementing a regional anesthesia program requires knowledge of all of the following except:
    (a) Collaboration
    (b) Assertiveness
    (c) Autonomy
    (d) Education
12. An nursing education program for regional anesthesia should include a discussion of:
    (a) Functional anatomy and physiology of nerve blockade
    (b) Expected effects of local anesthetics
    (c) Anticipated areas of pain not covered by blocks
    (d) All of the above
13. Nurses are most likely to call a physician about a regional anesthesia patient when:
    (a) They are familiar with the situation and suspect that something is wrong
    (b) They are bored
    (c) They are faced with a new situation beyond their comfort level and request guidance
    (d) (a) and (b)
14. Implementing a new regional anesthesia service requires all of the following except:
    (a) A master's degree in business administration (x)

(b) Specialized training in regional anesthesia
(c) Teamwork
(d) Effective communication

15. A "block room" requires all of the following except:
(a) Oxygen source
(b) Anesthesia machine (x)
(c) Standard ASA monitors
(d) Regional anesthesia supplies

16. Ambulatory patients discharged home after a single-injection peripheral nerve block with a long-acting local anesthetic:
(a) Have reliable resolution of sensory and motor blockade within 12 h of block placement
(b) Should receive a follow-up phone call the next week to assess for block resolution, adequacy of pain relief, and overall satisfaction with postoperative recovery
(c) Should be discharged with a protective sling if they had an upper extremity block
(d) Should be discharged home with a knee immobilizer if they had an adductor canal block

17. All of the following need to be completed prior to peripheral nerve block placement except:
(a) Patient attends informational session prior to day of surgery that includes an introduction to regional anesthesia techniques employed.
(b) Surgical consent and block consent signed by patient.
(c) Anesthesia assessment completed.
(d) Extremity to be blocked is marked by both surgeon and anesthesiologist.

18. New CPT codes for TAP blocks introduced in 2016:
(a) Delineate between single injection and continuous infusion and unilateral or bilateral procedures
(b) Include ultrasound imaging guidance
(c) Can be used to bill for rectus sheath blocks
(d) All of the above

19. Specialty training in regional anesthesiology techniques can be obtained:
(a) During residency
(b) By completing a Regional Anesthesiology and Acute Pain Fellowship
(c) By taking continuing medical education courses dedicated to regional anesthesia
(d) All of the above

20. Staffing a new regional anesthesia service includes training or hiring:
(a) Anesthesiology physicians trained in regional techniques
(b) Midlevel providers to assist with perioperative care and follow-ups
(c) A separate billing specialist
(d) (a) and (b)

Answers:

1. d
2. c
3. a
4. a
5. c
6. d
7. d
8. c
9. d
10. d
11. b
12. d
13. d
14. a
15. b
16. c
17. a
18. d
19. d
20. d

References

1. Williams BA, Kentor ML, Vogt MT, Vogt WB, Coley KC, Williams JP, et al. Economics of nerve block pain management after anterior cruciate ligament reconstruction: potential hospital cost savings via associ-

ated postanesthesia care unit bypass and same-day discharge. Anesthesiology. 2004;100:697–706.
2. Ilfeld BM, Mariano ER, Williams BA, Woodard JN, Macario A. Hospitalization costs of total knee arthroplasty with a continuous femoral nerve block provided only in the hospital versus on an ambulatory basis: a retrospective, case-control, cost-minimization analysis. Reg Anesth Pain Med. 2007;32:46–54.
3. Gonano C, Kettner SC, Ernstbrunner M, Schebesta K, Chiari A, Marhofer P. Comparison of economical aspects of interscalene brachial plexus blockade and general anaesthesia for arthroscopic shoulder surgery. Br J Anaesth. 2009;103(3):428–33.
4. Saporito A, Calciolari S, Ortiz LG, Anselmi L, Borgeat A, Aguirre J. A cost analysis of orthopedic foot surgery: can outpatient continuous regional analgesia provide the same standard of care for postoperative pain control at home without shifting costs? Eur J Health Econ. 2016;17(8):951–61.
5. Ilfeld BM, Gearen PF, Enneking FK, Berry LF, Spadoni EH, George SZ, et al. Total knee arthroplasty as an overnight-stay procedure using continuous femoral nerve blocks at home: a prospective feasibility study. Anesth Analg. 2006;102:87–90.
6. Ilfeld BM, Gearen PF, Enneking FK, Berry LF, Spadoni EH, George SZ, et al. Total hip arthroplasty as an overnight-stay procedure using an ambulatory continuous psoas compartment nerve block: a prospective feasibility study. Reg Anesth Pain Med. 2006;31:113–8.
7. Ilfeld BM, Vandenborne K, Duncan PW, Sessler DI, Enneking FK, Shuster JJ, et al. Ambulatory continuous interscalene nerve blocks decrease the time to discharge readiness after total shoulder arthroplasty: a randomized, triple-masked, placebo-controlled study. Anesthesiology. 2006;105:999–1007.
8. Chung F, Mezei G. Factors contributing to a prolonged stay after ambulatory surgery. Anesth Analg. 1999;89:1352–9.
9. Hadzic A, Williams BA, Karaca PE, Hobeika P, Unis G, Dermksian J, et al. For outpatient rotator cuff surgery, nerve block anesthesia provides superior same-day recovery over general anesthesia. Anesthesiology. 2005;102:1001–7.
10. Hadzic A, Karaca PE, Hobeika P, Unis G, Dermksian J, Yufa M, et al. Peripheral nerve blocks result in superior recovery profile compared with general anesthesia in outpatient knee arthroscopy. Anesth Analg. 2005;100:976–81.
11. Hadzic A, Arliss J, Kerimoglu B, Karaca PE, Yufa M, Claudio RE, et al. A comparison of infraclavicular nerve block versus general anesthesia for hand and wrist day-case surgeries. Anesthesiology. 2004;101:127–32.
12. Macario A, Weinger M, Carney S, Kim A. Which clinical anesthesia outcomes are important to avoid? The perspective of patients. Anesth Analg. 1999;89:652–8.
13. Klein SM, Nielsen KC, Greengrass RA, Warner DS, Martin A, Steele SM. Ambulatory discharge after long-acting peripheral nerve blockade: 2382 blocks with ropivacaine. Anesth Analg. 2002;94:65–70, table of contents.
14. Jamieson BD, Mariano ER. Thoracic and lumbar paravertebral blocks for outpatient lithotripsy. J Clin Anesth. 2007;19:149–51.
15. Mariano ER, Watson D, Loland VJ, Chu LF, Cheng GS, Mehta SH, et al. Bilateral infraorbital nerve blocks decrease postoperative pain but do not reduce time to discharge following outpatient nasal surgery. Can J Anaesth. 2009;56(8):584–9.
16. Klein SM, Bergh A, Steele SM, Georgiade GS, Greengrass RA. Thoracic paravertebral block for breast surgery. Anesth Analg. 2000;90:1402–5.
17. Grant SA, Nielsen KC, Greengrass RA, Steele SM, Klein SM. Continuous peripheral nerve block for ambulatory surgery. Reg Anesth Pain Med. 2001;26:209–14.
18. Nielsen KC, Greengrass RA, Pietrobon R, Klein SM, Steele SM. Continuous interscalene brachial plexus blockade provides good analgesia at home after major shoulder surgery-report of four cases. Can J Anaesth. 2003;50:57–61.
19. White PF, Issioui T, Skrivanek GD, Early JS, Wakefield C. The use of a continuous popliteal sciatic nerve block after surgery involving the foot and ankle: does it improve the quality of recovery? Anesth Analg. 2003;97:1303–9.
20. Bryan NA, Swenson JD, Greis PE, Burks RT. Indwelling interscalene catheter use in an outpatient setting for shoulder surgery: technique, efficacy, and complications. J Shoulder Elbow Surg. 2007;16:388–95.
21. Swenson JD, Bay N, Loose E, Bankhead B, Davis J, Beals TC, et al. Outpatient management of continuous peripheral nerve catheters placed using ultrasound guidance: an experience in 620 patients. Anesth Analg. 2006;103:1436–43.
22. Ilfeld BM, Morey TE, Wright TW, Chidgey LK, Enneking FK. Continuous interscalene brachial plexus block for postoperative pain control at home: a randomized, double-blinded, placebo-controlled study. Anesth Analg. 2003;96:1089–95, table of contents.
23. Ilfeld BM, Morey TE, Enneking FK. Continuous infraclavicular brachial plexus block for postoperative pain control at home: a randomized, double-blinded, placebo-controlled study. Anesthesiology. 2002;96:1297–304.
24. Ilfeld BM, Morey TE, Wang RD, Enneking FK. Continuous popliteal sciatic nerve block for postoperative pain control at home: a randomized, double-blinded, placebo-controlled study. Anesthesiology. 2002;97(4):959–65.
25. Mariano ER, Afra R, Loland VJ, Sandhu NS, Bellars RH, Bishop ML, et al. Continuous interscalene brachial plexus block via an ultrasound-guided posterior approach: a randomized, triple-masked, placebo-controlled study. Anesth Analg. 2009;108:1688–94.
26. Choi S, Rodseth R, McCartney JL. Effects of dexamethasone as a local anesthetic adjuvant for brachial

plexus block: a systematic review and meta-analysis of randomized trials. Br J Anaesth. 2014;112(3):427–39.
27. Woo JH, Kim YJ, Kim DY, Cho S. Dose-dependency of dexamethasone on the analgesic effect of interscalene block for arthroscopic shoulder surgery using ropivacaine 0.5%: a randomised controlled trial. Eur J Anaesthesiol. 2015;32(9):650–5.
28. Ilfeld BM, Wright TW, Enneking FK, Vandenborne K. Total elbow arthroplasty as an outpatient procedure using a continuous infraclavicular nerve block at home: a prospective case report. Reg Anesth Pain Med. 2006;31:172–6.
29. Ilfeld BM, Wright TW, Enneking FK, Mace JA, Shuster JJ, Spadoni EH, et al. Total shoulder arthroplasty as an outpatient procedure using ambulatory perineural local anesthetic infusion: a pilot feasibility study. Anesth Analg. 2005;101:1319–22.
30. Oldman M, McCartney CJ, Leung A, Rawson R, Perlas A, Gadsden J, et al. A survey of orthopedic surgeons' attitudes and knowledge regarding regional anesthesia. Anesth Analg. 2004;98:1486–90, table of contents.
31. Mariano ER, Chu LF, Peinado CR, Mazzei WJ. Anesthesia-controlled time and turnover time for ambulatory upper extremity surgery performed with regional versus general anesthesia. J Clin Anesth. 2009;21:253–7.
32. Macario A, Vitez TS, Dunn B, McDonald T. Where are the costs in perioperative care? Analysis of hospital costs and charges for inpatient surgical care. Anesthesiology. 1995;83:1138–44.
33. Ayalon O, Liu S, Flics S, Cahill J, Juliano K, Cornell CN. A multimodal clinical pathway can reduce length of stay after total knee arthroplasty. HSS J. 2011;7(1):9–15.
34. Corey JM, Bulka CM, Ehrenfeld JM. Is regional anesthesia associated with reduced PACU length of stay?: a retrospective analysis from a tertiary medical center. Clin Orthop Relat Res. 2014;472(5):1427–33.
35. Dexter F, Macario A, Manberg PJ, Lubarsky DA. Computer simulation to determine how rapid anesthetic recovery protocols to decrease the time for emergence or increase the phase I postanesthesia care unit bypass rate affect staffing of an ambulatory surgery center. Anesth Analg. 1999;88:1053–63.
36. Kurtz S, Mowat F, Ong K, Chan N, Lau E, Halpern M. Prevalence of primary and revision total hip and knee arthroplasty in the United States from 1990 through 2002. J Bone Joint Surg Am. 2005;87:1487–97.
37. Greger J, Williams BA. Billing for outpatient regional anesthesia services in the United States. Int Anesthesiol Clin. 2005;43:33–41.
38. Gerancher JC, Viscusi ER, Liguori GA, McCartney CJ, Williams BA, Ilfeld BM, et al. Development of a standardized peripheral nerve block procedure note form. Reg Anesth Pain Med. 2005;30:67–71.
39. Mariano ER. Making it work: setting up a regional anesthesia program that provides value. Anesthesiol Clin. 2008;26:681–92, vi.
40. Hebl JR, Kopp SL, Ali MH, Horlocker TT, Dilger JA, Lennon RL, et al. A comprehensive anesthesia protocol that emphasizes peripheral nerve blockade for total knee and total hip arthroplasty. J Bone Joint Surg Am. 2005;87(Suppl 2):63–70.
41. Mariano ER, Cheng GS, Choy LP, Loland VJ, Bellars RH, Sandhu NS, et al. Electrical stimulation versus ultrasound guidance for popliteal-sciatic perineural catheter insertion: a randomized controlled trial. Reg Anesth Pain Med. 2009;34:480–5.
42. Mariano ER, Loland VJ, Bellars RH, Sandhu NS, Bishop ML, Abrams RA, et al. Ultrasound guidance versus electrical stimulation for infraclavicular brachial plexus perineural catheter insertion. J Ultrasound Med. 2009;28:1211–8.
43. Mariano ER, Loland VJ, Sandhu NS, Bellars RH, Bishop ML, Afra R, et al. Ultrasound guidance versus electrical stimulation for femoral perineural catheter insertion. J Ultrasound Med. 2009;28:1453–60.
44. Mariano ER, Loland VJ, Sandhu NS, Bellars RH, Bishop ML, Meunier MJ, et al. A trainee-based randomized comparison of stimulating interscalene perineural catheters with a new technique using ultrasound guidance alone. J Ultrasound Med. 2010;29:329–36.
45. Sites BD, Chan VW, Neal JM, Weller R, Grau T, Koscielniak-Nielsen ZJ, et al. The American Society of Regional Anesthesia and Pain Medicine and the European Society of Regional Anaesthesia and Pain Therapy Joint Committee recommendations for education and training in ultrasound-guided regional anesthesia. Reg Anesth Pain Med. 2009;34:40–6.
46. Sites BD, Gallagher JD, Cravero J, Lundberg J, Blike G. The learning curve associated with a simulated ultrasound-guided interventional task by inexperienced anesthesia residents. Reg Anesth Pain Med. 2004;29:544–8.
47. Hadzic A, Vloka JD, Kuroda MM, Koorn R, Birnbach DJ. The practice of peripheral nerve blocks in the United States: a national survey [p2e comments]. Reg Anesth Pain Med. 1998;23:241–6.
48. Smith MP, Sprung J, Zura A, Mascha E, Tetzlaff JE. A survey of exposure to regional anesthesia techniques in American anesthesia residency training programs. Reg Anesth Pain Med. 1999;24:11–6.
49. Young A, Buvanendran A. Recent advances in multimodal analgesia. Anesthesiol Clin. 2012;30:91–100.
50. Richman JM, Stearns JD, Rowlingson AJ, Wu CL, McFarland EG. The introduction of a regional anesthesia rotation: effect on resident education and operating room efficiency. J Clin Anesth. 2006;18:240–1.
51. Martin G, Lineberger CK, MacLeod DB, El-Moalem HE, Breslin DS, Hardman D, et al. A new teaching model for resident training in regional anesthesia. Anesth Analg. 2002;95:1423–7, table of contents.
52. Regional Anesthesiology and Acute Pain Medicine Fellowship Directors Group. Guidelines for fellowship training in regional anesthesiology and acute pain medicine: third edition, 2014. Reg Anesth Pain Med. 2015;40:213–7.

53. Armstrong KP, Cherry RA. Brachial plexus anesthesia compared to general anesthesia when a block room is available. Can J Anaesth. 2004;51:41–4.
54. Drolet P, Girard M. Regional anesthesia, block room and efficiency: putting things in perspective. Can J Anaesth. 2004;51:1–5.
55. Ilfeld BM, Esener DE, Morey TE, Enneking FK. Ambulatory perineural infusion: the patients' perspective. Reg Anesth Pain Med. 2003;28:418–23.
56. Ilfeld BM, Enneking FK. Continuous peripheral nerve blocks at home: a review. Anesth Analg. 2005;100:1822–33.
57. Ninger LJ, Patterson P. Regional anesthesia has strong outcomes for care, efficiencies. OR Manager. 2004;20(1):9–12.
58. Williams BA, DeRiso BM, Engel LB, Figallo CM, Anders JW, Sproul KA, et al. Benchmarking the perioperative process: II. Introducing anesthesia clinical pathways to improve processes and outcomes and to reduce nursing labor intensity in ambulatory orthopedic surgery. J Clin Anesth. 1998;10:561–9.

2 Economic Impact, Cost, and Reimbursement Issues

Deepti Agarwal and Maunak V. Rana

Clinical Vignette

A 65-year-old male with a history of insulin-requiring diabetes mellitus and gastroesophageal reflux disease (GERD) presents for left total knee arthroplasty. The patient states that he had the same surgery on the right side 7 years prior, for which he received general anesthesia with an epidural for postoperative analgesia. The surgery time was 2.5 h, due to delayed emergence from general anesthesia. His postoperative hospital course lasted 3 days, which was complicated by postoperative nausea and vomiting (PONV). Upon discharge, the patient was discharged on oral oxycodone.

On this admission, you discuss the new protocol for Enhanced Recovery After Surgery (ERAS) that your hospital surgery center has instituted. Given the painful nature of total knee replacement surgery, you explain to the patient that the new protocol has been designed with a multimodal analgesic approach that will also help maximize early ambulation through physical therapy sessions and allow for discharge within 1–2 days. After reviewing the patient's medical history, you explain the anesthetic will consist of a spinal anesthetic with an adductor canal block.

Utilizing spinal anesthesia will help minimize the PONV the patient experienced last time, and an adductor canal block will help with pain control as well as spare motor function in the left leg postoperatively. The patient states that he had a relative who had surgery and asked about "going home with device that provides pain control." You explain that there is a possibility to insert a catheter adjacent to the nerve sheath. This approach will provide continuous local anesthesia and analgesia for up to 72 h via *an ON-Q pump that a patient can remove at home; however this service has not yet been set up at your surgical center.*

You proceed to explain the anesthetic to the patient and the details of the nerve block which will be performed by the acute pain team (dedicated nerve block anesthesiologist and an assistant). This block will be done in a dedicated block room that has an ultrasound and all the requisite supplies, in close proximity to the operating room.

Prior to going to surgery, the patient takes celecoxib and pregabalin, which is part of the multimodal analgesic regimen in the protocol. The patient proceeds to surgery. The surgery takes 1 h and 35 min to complete and does not have the increased time from the delayed emergence that occurred with the prior operation. The patient is able to bypass Phase I of PACU due to minimal sedation utilized during the surgery and the absence of nausea and vomiting. Postoperative

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_2

day one the patient is discharged to rehab after beginning his physical therapy and functioning with less pain than with his prior surgery.

Increasing Cost of Healthcare Economics

Healthcare costs have increased, with 17.8% of the gross domestic product (GDP) spent on healthcare costs and projected to be 19.3% by 2019, leading to heightened scrutiny, in an effort to balance safe and efficient care [1–3]. Therefore, interest in the economic assessment of healthcare processes and treatments with a precise understanding of which direct and indirect costs influence these processes will be important in the evaluation of healthcare economics.

As a result of this focused emphasis on cost containment, a shift has occurred in operative care of patients from what were once surgeries requiring inpatient admissions to now outpatient surgeries. This demand for increased efficiency and shorter hospital stays has magnified the role of anesthesiologists and their management of postoperative analgesia [4].

With a rapidly aging population and a higher level of morbidity exists, certain perioperative patients will require anesthetics that reduce the potential deleterious impact of agents on their cardiac and respiratory function. In addition, the increase in the number of obese patients has also posed challenges in the perioperative management of these patients. Without careful concern about the unique concerns of patients with comorbidities, healthcare costs may be increased in the postoperative phase of care. Diligent stratification and formalized protocols, however, serve to decrease the overall costs of readmission rates for pain, infection, or exacerbation of their comorbidities.

To that effect, the Enhanced Recovery After Surgery (ERAS) protocols have attracted importance as a way of providing value while decreasing complications, leading to a safer and cost-effective manner for patients undergoing surgery [5, 6]. ERAS has been utilized for patients undergoing colorectal, gynecologic surgery, and other surgical indications. As part of a collaborative care improvement model, ERAS programs have the potential to promote cost savings by leading shorter hospital stays as compared to non-pathway care. Investigation of patient factors leading to prolonged hospital stay after surgery has been evaluated [7]. In fact, the Triple Aim Framework from the Institute of Healthcare Improvement (IHI) highlights (a) improving population health, (b) improving patient experience, and (c) lowering per capita costs, as a goal that healthcare providers should embrace for the future of healthcare [8]. The prototype partners participating in the Triple Aim in the United States is available online and along with the associated measures/information [9].

These multidisciplinary protocols highlight anesthesiologists as key leaders in the perioperative arena. Anesthesiologists are uniquely poised to provide care preoperatively, intraoperatively, and when applicable postoperatively along the spectrum of a patient's surgical experience.

The Role of Anesthesiologist in Combating Costs

The cost for surgical care accounts for approximately 52% of hospital expenses in the United States [10]. Historically, surgeons served as the perioperative leader of the care team; however with increasing demands for intraoperative productivity, this role has shifted [11]. While hospitalists will help co-manage patients from a medical perspective, anesthesiologists have emerged as the preferred perioperative consultants because of their understanding of preoperative comorbidities, intraoperative management strategies, and postoperative pain management skills. The anesthesiologist therefore has a key role in standardization of care and demonstrating value with intervention choices in accordance with available financial capital. In evaluating healthcare economics, anesthesiologists play a central role in opportunity costs with the efficient use of limited resources in the future [12].

The anesthesiologist leads in evaluating cost minimization, cost-effectiveness, cost utility, and

cost benefit for technologies. Core processes in cost analysis must be considered by key decision-makers [12]. *Cost minimization* involves evaluating two alternative approaches in a process to reach an endpoint. This model strictly evaluates cost differential. *Cost-effectiveness* involves evaluating two alternatives, assessing their endpoints, and comparing the costs of achieving the differing endpoints. *Cost utility* allows for multiple outcomes (risks and benefits) combined into one measure. *Cost-benefit* evaluation analysis takes outcomes and translates them into financial outcomes, represented as dollar equivalents. As health entities design protocols for patient care, this will be met via the ERAS programs and also through the postsurgical home (PSH), distinct entities. Regardless of the platform, however, cost containment coupled with safe care is the common goal of protocol-driven patient management programs.

In evaluating the costs related to the anesthetic care of the patient, many factors are involved including the practice region, patient population, type of surgical procedure, and ability to have home-based acute care, along with fixed and variable costs [13]. The decision to offer a service should undergo a rigorous evaluation based on the resource availability, the resource cost, and the benefit potential and actually realized. Anesthesiologists play a key role in process evaluation for patient flow in the perioperative arena.

Despite the benefits from a patient satisfaction perspective and lower potential financial cost to regional anesthesia, there is perception among surgeons that the delivery of preoperative regional anesthesia slows down surgery times, leading to delays in the perioperative process [14]. In an evaluation done by Stahl et al., an "Operating Room of the Future" (ORF) was designed as a way to streamline and facilitate smooth transition between the preoperative, intraoperative, and postoperative phases [15]. They found that perioperative improvements other than trying to facilitate a faster operating time, actually, were the activities that led to greater cost containment. These adaptations are referring to activities that can be done in parallel with another simultaneous surgery (i.e., placing a peripheral nerve block in a patient in the preop area prior to surgery). On the other hand, Eappen et al. showed that despite surgeon perception, the anesthesia-controlled time (refer to paragraph below) was not improved with a separate regional anesthesia team, especially in the setting of longer operations with prolonged turnover times [16]. This study suggested exploring other areas as a source of potential delay as opposed to the preoperative block placement.

During the anesthesiologist's role of the patient in the perioperative setting, certain time concepts are important to consider, namely, anesthesia-controlled time (ACT) and turnover time. ACT refers to the time of operating room entry until the sterile prep and positioning of the patient. The time is then paused while the surgical intervention takes place. ACT then continues from the end of surgery until patient exit from the operating room. Turnover time (TOT) reflects the time from the patient leaving the operating room to the subsequent patient is brought into the operating room for the next procedure. Providing a regional anesthetic consecutively results in lower ACT time, lowest sum of ACT plus TOT times, and reduced unplanned hospital admissions most often related to pain and PONV [17].

Cost reduction and anesthetic choices are crucial factors demonstrating the strengths of regional anesthesia as a primary type of anesthetic when suitable. The choice of local anesthesia was evaluated for cost-benefit purposes. An observational case-control cost-minimization study was used to compare chloroprocaine with mepivacaine for outpatient popliteal block for foot surgery [18]. One hundred patients were given either 30 mL of 3% chloroprocaine or 30 mL 1.5% mepivacaine. The authors noted that onset time to block and duration of block (sensory and motor) was shorter with the use of chloroprocaine. These differences translated into a discharge time of 120 min earlier for the chloroprocaine group. Patient satisfaction and block efficacy were not impacted by the shorter-acting agent. Integrating this data into regional anesthesia protocols will be helpful to improve overall efficiency and economics.

Regional Anesthesia as a Way of Improving Economics

The infusion of regional anesthesia (RA) practice serves as a bona fide approach to combatting the rising costs of healthcare delivery in the perioperative milieu. Postoperative pain management has received considerable scrutiny from accreditation entities and pain management societies not only because of its' impact on patient satisfaction but also postoperative care [19]. Regional anesthesia can lead to fast-tracking of patients by optimizing postoperative analgesia by an opioid-sparing effect [20]. RA, compared to systemic analgesia, presents global improvements via cost savings as well as a reduction in opioid use and comorbidities [21]. These benefits are echoed in the orthopedic literature for anterior cruciate ligament surgery and total knee arthroplasty (TKA), allowing for decreased postoperative pain, morphine consumption, and adverse effects [22, 23].

Regional techniques have been advocated as a preferred approach due to the ability to decrease certain postoperative complications including, but not limited to, respiratory depression secondary to IV opioid use for pain control. A regional anesthesia approach in the perioperative setting has decreased the complications from general anesthesia, leading to improved pain scores, less PONV, earlier discharge, and less risk of hospital readmission. Richman et al. published a meta-analysis which confirmed the superiority of perineural analgesia via continuous catheter over opioid therapy for visual analog scores, nausea/vomiting, and pruritis [24].

In addition, the use of regional anesthesia allows for avoidance of tracheal intubation and positive pressure ventilation which may be less ideal in certain patients, reduces sympathetic activation and subsequent inflammation, and decreases venous stasis and the risk of developing pulmonary embolism. One of the significant sources of increased healthcare costs and decreased patient benefit after any surgical procedure is the persistence of debilitating pain along with the presence of postoperative nausea and vomiting (PONV). When effective analgesia as provided by regional anesthesia techniques occurs, concomitant antiemetic prophylaxis results in rapid recovery of patients, and PACU bypass, leading to a "fast-tracking" discharge of patients [25]. All of these advantages lead to long-term cost savings by reducing postoperative complications which may lead to longer hospital stays and increased medical expenses.

RA may also have a role in the fiscal health of an anesthesia group practice. A meta-analysis evaluated ten independent trials involving 330 patients undergoing general anesthesia and 348 patients undergoing neuraxial block and highlighted the salubrious nature of the neuraxial technique. From a potential cost savings, neuraxial block led to a quicker operating room time by 7 min and 275 mL/case less blood loss [26]. Williams et al. showed that using regional anesthesia for ambulatory orthopedic surgeries confirmed process improvement, efficiency, and benefit in recovery profiles [27].

While traditionally providing a powerful way of reducing complications and effects of general anesthesia in patients undergoing orthopedic surgery procedures, the use of regional techniques has also provided safe and efficient management of patients undergoing visceral surgical procedures. The transversus abdominis plane (TAP) block serves to anesthetize the lower portion of the abdomen from T10-L1. Blockade is effective for abdominal and inguinal surgery. Catheters have been placed to facilitate prolonged analgesia, allowing for outpatient discharge and recovery [28]. In a randomized triple-blind trial evaluating the efficacy of the TAP block after total laparoscopic hysterectomy, patients with the TAP block had a notable reduction in pain scores compared with a placebo group initially; however the benefit was not present, and no difference was present between treated and placebo patients at 24 and 48 h [29]. In a meta-analysis, however, TAP blocks were shown to provide improved postoperative pain, with an improvement in outcomes and a decrease in postoperative opioid consumption when administered in the setting of laparoscopic surgery [30]. TAP block has been used in the placement of peritoneal dialysis catheters under monitored anesthesia care in a case series [31]. Despite significant coexisting

medical conditions including renal dysfunction and cardiac and coagulation disorders, these seven patients in this study were managed with the TAP block along with supplemental analgesics. In this population, the use of RA to potentially bypass Phase I recovery and to avoid the prior mentioned concerns of GA serves as a boon to patients and providers [31].

The popularization of perioperative care pathways to achieve early recovery after surgical procedures has been to shown to improve outcomes. The development of ERAS and PROSPECT (evidence-based, procedure-specific postoperative pain management) has been designed initially for colorectal surgery patients but has since been expanded to many surgical specialties. Sammour and colleagues published a cost analysis of ERAS in colorectal surgery. Because of the reduction in total hospital stay, intravenous fluid use, complications, and duration of epidural use in the ERAS group, decreases in postoperative resource utilization resulted in greater cost savings over the long run in comparison with the cost of setting up and maintaining an ERAS program [32]. Regional anesthesia is bundled into the menu of options for patients as part of the ERAS enhanced recovery protocols (ERPs) and designed to standardize the anesthetic and analgesic regimen [33]. Data published has illustrated that regional anesthesia directly improved outcomes and the ability to achieve the goals of ERPs.

Regional anesthesia was noted to have a beneficial impact on measures of function and on economic outcomes, with noticeable improvements in patient outcomes [34]. Given these benefits, regional anesthesia has an important role in ERPs and can serve to achieve the goals of the Triple Aim, by improving pain and PONV and decreasing length of stay [33].

How Regional Anesthesia Fosters Fast-Track Elements/ERPs

Opioid-sparing effects/nausea and vomiting
- Early oral feeding
- Rapid patient mobility
- Quicker recovery from general anesthesia

Translating the seemingly salubrious benefit of regional anesthesia for surgical patients to financial benefits, that is, presenting a cost-benefit analysis, requires a more complex evaluation of all the factors leading to charges and costs during the perioperative process. An observational nonrandomized study done by Williams et al. combined hospital cost data with surgical outcome data [35]. It was found that the use of nerve blocks for ACL surgery reduced the PACU admission rate to 18% and led to a decrease in the unplanned hospital admission rate to 4%. This bypass lead to a mean reduction in hospital cost of $173 per patient. Furthermore, when the data were analyzed with multifactorial regression analysis, the effect of PACU bypass was to lower hospital costs by 12% ($420 per patient). On the converse side, patients who required hospital admission after surgery were responsible for an increased hospital cost of 11% (increase of $385 per patient). The additional effects on this improvement in patient flow would be a reduction of nursing staff in the PACU, which would theoretically lead to less nursing cost.

The advent of technology in regional anesthesia has led to cost benefits. Another analysis, presented as a letter to the editor, reported a cost analysis performed for equipment and supplies for patients undergoing infraclavicular block for upper extremity surgery [36]. Conventional nerve stimulator was evaluated against ultrasound technique for differences in procedure costs and time for procedure. The authors concluded that for infraclavicular catheter placement, ultrasound guidance led to faster blocks and shorter onset time, yielding savings of $13.90 per case.

A prospective cohort study evaluated 120 consecutive patients to two groups receiving popliteal block with an elastomeric pump with ropivacaine delivery as inpatients or outpatients after foot surgery [37]. The day discharge group had decreased total management costs as compared to the inpatient group. This change was due to a greater likelihood of PACU bypass. This study also followed patients and evaluated a cost analysis up to 6 months in terms of work lost and found benefit with those patients able to be in the day discharge group. The authors opine that

the main cost-saving factors which attributed to regional anesthesia include shorter ready-for-surgery time, reduction in length of stay, and recovery room bypass.

Choices by the perioperative physician are also of key importance in patients undergoing outpatient surgery. While patients may typically undergo general anesthesia for outpatient procedures, the use of regional anesthesia may provide a superior recovery as compared to general anesthesia. Hadzic et al. performed a randomized study comparing patients receiving general anesthesia and intra-articular injection of local anesthetic to patients receiving lumbar plexus/sciatic block in terms of operating room time, PACU bypass, and time to actual discharge home [38]. Patients receiving the blockade technique had a greater likelihood of PACU bypass and a shorter time to discharge than those patients undergoing general anesthesia, differences that would translate to a lesser cost of care for patients.

The comparative costs between general and regional anesthesia were compared in patients undergoing arthroscopic shoulder surgery as well, which showed an improvement in monetary costs for the regional group of 8 euros, with lower PACU costs and time [39]. It is no surprise that anesthesia workflow also improved with a regional approach. Time is saved on emergence, leading to less operating costs.

In addition to the cost benefits that regional anesthesia has for patients undergoing outpatient surgery, operations traditionally requiring inpatient admission have been shown to cost less with regional anesthesia. In a study evaluating the feasibility and the cost incurred for ambulatory vs. admitted patients after total knee arthroplasty, the ambulatory status of patients was facilitated with benefit from continuous femoral nerve blocks [40]. The ambulatory patients accrued 14% less charges than those patients who were admitted; the regional anesthetic was a significant factor in the cost savings in these patients.

Comorbid factors are associated with the development of surgical site infections that can be directly modulated by regional techniques [41]. Additionally, a retrospective propensity-matched cohort study evaluating data from the American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP) database for patients undergoing total hip arthroplasty from 2007 to 2011 was used to evaluate the benefits of regional techniques [42]. Logistic regression analysis was used for correlating anesthetic approach and risk of length of stay in hospitals, deep surgical site infections, mortality rates at 30 days, and cardiopulmonary complications. 5103 surgical procedures out of 12,929 were performed with a regional technique. Odds ratio analysis demonstrated that regional group had lower adjusted odds for deep infections, 5% decrease in hospital stay for patients with regional anesthesia, 27% decrease in odds of having a prolonged hospitalization, and a decreased incidence of cardiopulmonary complications.

Setting Up a Regional Service

As is evidenced by the power of the studies presented above, regional anesthesia leads to favorable costs during the healthcare delivery of surgical patients. Aside from cost benefits, enhanced patient satisfaction with analgesic quality impacts favorably on patient care and the overall hospital experience. As medical centers have improved patient satisfaction scores and better health outcomes, direct and indirect economic impact results.

The perioperative physician practicing regional anesthesia needs to take a careful and precise evaluation of the tools and equipment of the trade in order to effectively perform the tasks at hand. Intraoperative anesthesia costs make up approximately 6% of total costs in the perioperative period. Half of these costs are based directly on the choices of the anesthesiologist's clinical decisions [43]. Many factors may influence how the anesthesiologist decides which anesthetic technique would be best suited for their patient. Factors influencing decision-making include patient age, medical/surgical history, prior anesthetic history, scheduled surgery, and current medications including pain medications and/or anticoagulant therapies.

Considerations for Anesthetic Technique

- Age of patient
- Comorbidities (cardio/pulmonary status, anticoagulation status)
- Medications (chronic pain medications, blood-thinning medications)
- Surgical history (repeat/revision on an extremity)
- Any current issues that may influence the practitioner's decision (localized infection, high white blood cell count)
- Anesthetic complications in the past, side effects from anesthesia, family history of anesthetic problems (history of malignant hyperthermia may influence decision to proceed with regional)
- Patient consent/anesthetic concerns
- Inpatient or outpatient surgery, expected duration of hospitalization

The location of the perioperative setting (hospital vs. outpatient) and type of practice (private vs. academic) play an important role in determining whether the patient population will benefit from such a service and whether the services will result in overall cost containment while generating increased revenue.

A review by Swenson et al. highlights the important considerations for the regional physician to entertain when presenting and formulating a regional practice to provide care in a safe, efficient, and cost-conscious manner [44]. For example, the use of ultrasound (US) guidance as opposed to the most historic use of nerve stimulation (NS) is highlighted and presented as allowing for more successful blocks. While new adopters of US therapy may not be as facile as experienced sonographic anesthesiologists, the combined use of US and NS may allow the provider to perform ultrasound for real-time visualization of anatomy and medication deposition but allow for the familiar crutch of NS.

The combination of US and NS has led to needle redirects and slower block performance times than just utilizing US. Additionally, the use of echogenic needles is not necessarily advocated as a cost-saving tool despite this modification allowing for better needle visualization during encroachment into tissue. The use of specific nerve-stimulating catheters as compared to conventional catheters also does not appear to lead to a cost-savings benefit. Stimulating catheters have been described to lead to longer procedure times, procedural discomfort, increased failure rates, and iatrogenic injury. Newer technology may also add improved image fidelity: Swenson and colleagues found that magnetic-guided ultrasound improves positional needle accuracy when compared to conventional ultrasound in both novice and experienced practitioners [45]. This enhanced imaging could play a role in efficiency and overall quality of nerve blocks.

The infusion pump, typically delivering local anesthetic and/or clonidine after surgery, is the next technological tool that extends the operative analgesia into the perioperative state in the hospital or as an outpatient. The choice of type of infusion pump is an important consideration for anesthesiologists to balance finances with function. Disposable infusion pumps and non-disposable pumps are utilized in clinical practice. These pumps are further classified and have cost differences based on capability of infusions. For example, pumps may provide for fixed basal units to variable capable rate settings and also bolus capable units. It will be important to factor these decisions in deciding what tools will be necessary to create a successful regional program. Overall, the choice of supplies by regional anesthesia providers should emphasize a favorable cost-benefit ratio.

In order to develop a successful regional program, the customers of the anesthesiologist (patients, surgeons, and administrators) must be satisfied with the service and the results. While first and foremost, regional anesthesia must be safe and effective for the patient, perioperative readiness must also be taken into consideration as quickly as possible to enhance the flow of the operating room environment.

In order to balance these seemingly opposing demands, trained personnel proficient in regional anesthesia and the use of US nerve blocks must

lead the perioperative team for this service. The use of a "block room," where regional anesthesia procedures can be performed prior to the intended surgery, may serve to reduce ACT and allow for expedient patient surgical preparation from an anesthesia perspective [46]. The parallel workforce may inherently operate in a smoother fashion in academic settings where there are residents or in private settings where there are teams of anesthesiologists, nurse anesthetists, and anesthesia assistants working within their respective scope of practice, with the anesthesiologist as the leader of the team [47]. Additionally, the block room enables a controlled environment where equipment and dedicated supplies are readily available.

Preparing premade kits can help with efficiency, along with creating a cart in which all the equipment necessary for procedures are located. The mobility of this unit may allow for an easy transport to the bedside in different service locations. In order to provide a safe environment, resuscitative medication or a crash cart should also be available per medical center/surgery center policy.

Streamlining this perioperative regional anesthesia/analgesic process originates preoperatively when the patient initially decides to have surgery. Surgeons would ideally introduce the concept of a regional anesthesia for postoperative analgesia. This initial communication sets the table for the process from an established provider who the patient has rapport with. Surgeon buy-in to regional anesthesia allows for reducing anxiety about a new procedure discussed with the patient, at times, for the first time, on the day of surgery, shortly before the patient presents to the operating room. The preoperative discussion taking place much in advance of surgery allows for the patient to understand the benefits of regional anesthesia and to be mentally prepared for this process. It is ideal for the regionalist to meet with the patient to explain the procedure, risks, and benefits, along with alternatives for the patient. The key tenet to this discussion is clear communication between the patient and the perioperative physician in a concise and comfortable manner, allowing to empower the patient to be an important part of the perioperative process and not a passive participant in their choices for medical care.

Billing and Coding for Regional Anesthesia

The approach of the provider is to prepare a business model keeping in mind the customers, investors, and suppliers [48]. With regard to customer, both the patient and surgeon will need to be on board with the idea of nerve blocks in order for a successful regional program to operate.

It is important to take a hands-on approach to billing in order to maximize the amount of revenue generated for the hospital and the anesthesiology group. There is significant variability in reimbursement of regional anesthesia procedures for postoperative pain management and even between carriers within a certain region and across different regions [49].

From a billing perspective, a bona fide separate procedure note must be generated. Key points must be present in order to facilitate reimbursement success for claims. Detailed information includes requesting surgeon, indication of procedure, site of procedure, and technique. A key point to highlight is the distinction of block from the technique of anesthesia for the surgery, that is, to differentiate between postoperative analgesia from intraoperative techniques. For example, for a shoulder arthroscopic procedure, the interscalene nerve block must be clearly documented as for postoperative analgesia. The general anesthetic for the procedure would be the primary anesthetic in the intraoperative phase of the patient's care. From the anesthesiologist's perspective, unbundling of the regional technique from the intraoperative anesthetic is key for accurate and productive billing and may improve denial rate [50]. An additional way to highlight the distinction for postoperative analgesia from intraoperative care

is to have a separate team providing the block. If a separate practitioner is not possible, then ensure that the block is performed in a separate location from the anesthetizing location [17].

It is of crucial importance to document the number of locations that the regional anesthesiologist is performing. More than four sites categorize the physician as ***supervising*** as opposed to ***medically directing***, a redesignation that would lead to decreased reimbursement for anesthesia services. When evaluating billing for regional techniques, it is important to document the type of anesthetic technique for surgery. If the regional technique will be the sole anesthetic, then the block will be reimbursed as part of the global anesthetic fee; if another form of anesthesia is the primary type (general or neuraxial), then the nerve block should be billed as charge modifiers to the anesthetic fee. That is, these procedures are not billed by time units but by units assigned to blocks.

It is important to have a basic understanding of the costs, charges, and payments as they pertain to regional anesthesia in the implementation and execution of a regional anesthesia team [49]. Certain key concepts, while in colloquial use, are important to define, as financial viability is explored. ***Cost*** is the capital to buy resources to perform PNB, including salary of those performing the block. ***Charges*** reflect the hospital billing of the regional technique to the patient, which is generally more than the actual procedure costs. ***Payment*** is the amount of money received by the payer, which is usually a percentage of the charge and is determined by the payment schedule and payer mix. The payment or reimbursement should exceed the cost to maintain a fiscally efficacious practice model and to demonstrate viability to key stakeholders.

In order to maximize payments, familiarity with CPT codes and their relative value is important. CPT codes are utilized for submitting billing based on target nerve and specific block performed. Use modifiers like -59 to show that a peripheral nerve block is a distinct service, independent of other anesthetic services performed. If a procedure is performed bilaterally, the -50 modifier is used. The -51 modifying code is utilized for multiple blocks. Ultrasound guidance is billed with a separate and CPT code 76942. To appropriately use this code, the provider must document needle placement and image interpretation with a copy retained of the ultrasound image that highlights relevant sonographic anatomy and spread of local anesthetic. Patients may be continued with their peripheral nerve analgesic regimen with an infusion postoperatively, continuous peripheral nerve blockade (CPNB). This catheter system requires daily management of the patient to ensure analgesia and to evaluate for complications. E/M management codes are utilized for the daily management. Commonly used CPT codes and ICD-10 pain diagnosis codes are listed in Tables 2.1 and 2.2 [51].

Table 2.1 Anesthesia billing codes (CPT)

Type of block	Single shot	Catheter
Interscalene	64415	64416
Supraclavicular	64415	64416
Infraclavicular	64415	64416
Axillary	64417	64416
Femoral	64447	64448
Sciatic	64445	64446
Fascia iliaca	64447	64448
Lumbar plexus	64483	64449
TAP	64486/64488(bilateral)	64487/64489(bilateral)
Ultrasound guidance		76942

Table 2.2 ICD-10 pain diagnosis codes

Shoulder	M25.511	Pain in the right shoulder
	M25.512	Pain in the left shoulder
	M25.519	Pain in an unspecified shoulder
Upper arm/elbow	M25.521	Pain in the right elbow
	M25.522	Pain in the left elbow
	M25.529	Pain in an unspecified elbow
Forearm/wrist	M25.531	Pain in the right wrist
	M25.532	Pain in the left wrist
	M25.539	Pain in an unspecified wrist
Hand	M79.643	Pain in an unspecified hand
	M79.646	Pain in an unspecified finger(s)
Hip/thigh	M25.551	Pain in the right hip
	M25.552	Pain in the left hip
	M25.559	Pain in an unspecified hip
Knee/leg	M25.561	Pain in the right knee
	M25.562	Pain in the left knee
	M25.569	Pain in an unspecified knee
Foot/ankle	M25.571	Pain in the right ankle and joints of the right foot
	M25.572	Pain in the left ankle and joints of the left foot
	M25.579	Pain in an unspecified ankle and joints of an unspecified foot

Future Directions

Regional anesthesia techniques arm the anesthesiologists with tools to improve patient care and to advocate for fiscally sound choices for healthcare administration. The value will need to be demonstrated to key stakeholders in the healthcare paradigm. The perioperative home along with ERAS and ERPs is the key future direction for care of surgical patients. A way to directly document the powerful role that regional anesthesia has in the promulgation of these pathway is to compare ERPs that contain regional techniques with those that do not have it. The documented cost savings for pain medications, comparative incidence of PONV, and shorter time to discharge would lead credence to incorporating regional anesthesia as beneficial from value and cost-benefit purposes. These benefits make anesthesiologists poised to be the leaders of present and future patient care settings to provide the best possible value-driven care for patients.

Review Questions

1. Economic evaluations allow for comparisons of both costs and effects of an intervention. Which of the following is not part of this economic evaluation?
   (a) Cost minimization
   (b) Cost-effectiveness
   (c) Cost value
   (d) Cost benefit
   (e) Cost utility

   Answer: c) Cost value

   Cost minimization involves evaluating two alternative approaches in a process to reach an endpoint. This model strictly evaluates cost differential. Cost-effectiveness involves evaluating two alternatives, assessing their endpoints, and comparing the costs of achieving the differing endpoints. Cost utility allows for multiple outcomes (risks and benefits) combined into one measure. Cost-benefit evaluation analysis takes outcomes and translates them into financial outcomes, represented as dollar equivalents. Cost value is not a term used in economic evaluation.
2. A 57-year-old obese patient with a history of OSA on CPAP is receiving an ultrasound guided b/l TAP block for an abdominal hysterectomy in order to minimize postoperative pain and the need for IV narcotics given her obesity and OSA history. Which of the following modifiers would reflect that this procedure is being performed bilaterally with the use of ultrasound?
   (a) -59, 76942
   (b) -50, 76942
   (c) -49, 76940
   (d) -59, 76942
   (e) -50, 76940

   Answer: b) -50, 76942

   In order to maximize payments, familiarity with CPT codes and their relative value is important. CPT codes are utilized for submitting billing based on target nerve and specific

block performed. Use modifiers like -59 to show that a peripheral nerve block is a distinct service, independent of other anesthetic services performed. If a procedure is performed bilaterally the -50 modifier is used. The -51 modifying code is utilized for multiple blocks. Ultrasound guidance is billed with a separate and CPT code 76942.

3. Which of the following has *not* been shown to be a cost-effective benefit of performing a regional anesthetic?
   (a) Opioid-sparing effect
   (b) Decreased PONV
   (c) Earlier discharge to home
   (d) Reduced parasympathetic activation
   (e) Avoidance of general anesthesia in patients with respiratory pathology

   Answer: d) Reduced parasympathetic activation

   One of the significant sources of increased healthcare costs and decreased patient benefit after any surgical procedure is the persistence of debilitating pain along with the presence of postoperative nausea and vomiting (PONV). A regional anesthesia approach in the perioperative setting has decreased the complications from general anesthesia, leading to improved VAS scores, less PONV, decreased IV opioid use leading to an ***opioid-sparing effect***, earlier discharge, and less risk of hospital readmission.

   The stress response is in reference to the hormonal and metabolic changes which follow surgery. Regional anesthesia will prevent the endocrine and metabolic responses to surgery **decreasing sympathetic activation**. Both afferent input from the operative site to the central nervous system and the hypothalamic-pituitary axis and efferent autonomic neuronal pathways to the liver and adrenal medulla are blocked with epidural analgesia.

4. Which of the following has been shown to reduce ACT time as it relates to performing a regional anesthetic?
   (a) Performing the time-out in the preop area
   (b) Having an efficient janitorial staff in between cases
   (c) The use of a designated block room
   (d) Decreasing surgical operating times
   (e) Using a faster-acting local anesthetic

   Answer: c) The use of a designated block room

   Efficient management of operating rooms requires an understanding and analysis of the times needed for all components of surgical care. Two terms used when looking at anesthesia time in the operating room include anesthesia-controlled time (ACT) and turnover time (TOT).

   TOT = time (min) from previous patient out of the room to the next patient in the room

   ACT = time (min) from surgical closure to out of the room with previous patient + time (min) from the next patient in the room to anesthesia ready

   The use of a "block room" where regional anesthesia procedures can be performed prior to the intended surgery may serve to reduce ACT and allow for expedient patient surgical preparation from an anesthesia perspective. The parallel workforce may inherently operate in a smoother fashion in academic settings where there are residents or in private settings where there are teams of anesthesiologists, nurse anesthetists, and anesthesia assistants working within their respective scope of practice, with the anesthesiologist as the leader of the team. Additionally, the block room enables a controlled environment where equipment and dedicated supplies are readily available.

   Preparing premade kits can help with efficiency, along with creating a cart in which all the equipment necessary for procedures are located. The mobility of this unit may allow for an easy transport to the bedside in different service locations. In order to provide a safe environment, resuscitative medication or a crash cart should also be available per medical center/surgery center policy.

References

1. Center for Medicaid and Medicare Services. NHE fact sheet [Internet]. Baltimore: Center for Medicaid and Medicare Services; 2016 [cited 2016 Jan 30]. https://www.cms.gov/research-statistics-data-and-systems/

statistics-trends-and-reports/nationalhealthexpenddata/nhe-fact-sheet.html.
2. Fuchs VR. The gross domestic product and health care spending. N Engl J Med. 2013;369(2):107–9.
3. Sisko AM, Truffer CJ, Keehan SP, Poisal JA, Clemens MK, Madison AJ. National health spending projections: the estimated impact of reform through 2019. Health Aff (Millwood). 2010;29:1933–41.
4. Shuber A, Guirguis M. The role of peripheral nerve block analgesia in advancing therapeutic effectiveness spanning the episode of care. Tech Reg Anesth Pain Manag. 2014;18(4):118–22.
5. Alzahrani T, Eldawlatly A. The enigma of regional anesthesia in enhanced recovery after anesthesia protocols. Basic Res J Med Clin Sci. 2016;5(7):127–33.
6. Eldawatly A. Is enhanced recovery after anesthesia a synonym to enhanced recovery after surgery? Saudi J Anaesth. 2016;10:119–20.
7. Sibia US, MacDonald JH, King PJ. Predictors of hospital length of stay in an enhanced recovery after surgery program for primary total hip arthroplasty. J Arthroplast. 2016;31(10):2119–23.
8. Berwick DM, Nolan TW, Whittington J. The triple aim: care, health, and cost. Health Aff. 2008;27(3):759–69.
9. Institute for Healthcare Improvement. IHI triple aim initiative [Internet]. Cambridge, MA; 2010 [cited 2016 Jan 31]. http://www.ihi.org/Engage/Initiatives/TripleAim/Pages/Materials.aspx.
10. Healthcare Cost Institute. Health care cost and utilization report: 2011. Washington, DC: Health Care Cost Institute; 2012 [cited 2016 Feb 1]. http://www.healthcostinstitute.org/report/2012-health-care-cost-utilization-report/.
11. Sheldon GF, Ricketts TC, Charles A, King J, Fraher EP, Meyer A. The global health workforce shortage: role of surgeons and other providers. Adv Surg. 2008;42:63–85.
12. Martin J, Cheng D. Role of the anesthesiologist in the wider governance of healthcare and health economics. Can J Anaesth. 2013;60:918–28.
13. Macario A. What does one minute of operating room time cost? J Clin Anesth. 2010;22(4):233–6.
14. Oldman M, McCartney CJ, Leung A, Rawson R, Perlas A, Gadsen J, et al. A survey of orthopedic surgeons' attitudes and knowledge regarding regional anesthesia. Anesth Analg. 2004;98:1486–90.
15. Stahl JE, Sandberg WS, Daily B, et al. Reorganizing patient care and workflow in the operating room: a cost-effectiveness study. Surgery. 2006;139:717–28.
16. Eappen S, Flanagan H, Lithman R, Bhattacharya N. The addition of a regional block team to the orthopedic operating rooms does not improve anesthesia-controlled times and turnover time in the setting of long turnover times. J Clin Anesth. 2007;19(2):85–91.
17. Williams BA, Kentor ML, Williams JP, Figallo CM, Sigl JC, Anders JW, et al. Process analysis in outpatient knee surgery: effects of regional and general anesthesia on anesthesia-controlled time. Anesthesiology. 2000;93:529–38.
18. Saporito A, Anselmi L, Borgeat A, Aguirre JA. Can the choice of the local anesthetic have an impact on ambulatory surgery perioperative costs? Chloroprocaine for popliteal block in outpatient foot surgery. J Clin Anesth. 2016;32:119–26.
19. Chou R, Gordon DB, de Leon-Casasola OA, Rosenberg JM, et al. Management of postoperative pain: a clinical practice guideline from the American Pain Society, the American Society of Regional Anesthesia and Pain Medicine, and the American Society of Anesthesiologists' Committee on Regional Anesthesia, Executive Committee, and Administrative Council. J Pain. 2016;17(2):131–57.
20. Carli F, Kehlet H, Baldini G, Steel A, McRae K, Slinger P, et al. Evidence basis for regional anesthesia in multidisciplinary fast-track surgical care pathways. Reg Anesth Pain Med. 2011;36(1):63–72.
21. Hudson ME, Chelly JE, Williams BA. Economics: projecting costs and revenue for an interventional pain service in the ambulatory setting. Int Anesthesiol Clin. 2011;49(3):68–83.
22. Foster BD, Terrell R, Montgomery SC, Wang JC, Petrigliano FA, McAllister DR. Hospital charges and practice patterns for general and regional anesthesia in arthroscopic anterior cruciate ligament repair. Orthop J Sports Med. 2013;1(5):1–5.
23. MacFarlane AJR, Govindarajulu AP, Chan VWS, Brull R. Does regional anesthesia improve outcome after total knee arthroplasty? Clin Orthop Relat Res. 2009;467:2379–402.
24. Richman JM, Liu SS, Courpas G, Wong R, Rowlingson AJ, McGready J, et al. Does continuous peripheral nerve block provide superior pain control to opioids? A meta-analysis. Anesth Analg. 2006;102:248–57.
25. Williams BA, Kentor ML. Fast-track ambulatory anesthesia: impact on nursing workload when analgesia and antiemetic prophylaxis are near optimal. Can J Anaesth. 2007;54:243–4.
26. Mauermann WJ, Shilling AM, Zuo Z. A comparison of neuraxial block versus general anesthesia for elective total hip replacement: a meta-analysis. Anesth Analg. 2006;3(4):1018–25.
27. Williams BA, DeRiso BM, Figallo CM, Anders JW, Engel LB, Sproul KA. Benchmarking the perioperative process: III. Effects of regional anesthesia clinical pathway techniques on process efficiency and recovery profiles in ambulatory orthopedic surgery. J Clin Anesth. 1998;10:570–8.
28. Heil JW, Ilfeld BM, Loland VJ, et al. Ultrasound-guided transversus abdominis plane catheters and ambulatory perineural infusions for outpatient inguinal hernia repair. Reg Anesth Pain Med. 2010;35(6):556–8.
29. Calle GA, Lopez CC, Sanchez E, et al. Transversus abdominis plane block after ambulatory total laparoscopic hysterectomy: randomized controlled trial. Acta Obstet Gynecol Scand. 2014;93(4):345–50.
30. De Oliveira GS Jr, Castro-Alves LJ, Nader A, Kendall MC, McCarthy RJ. Transversus abdominis

plane block to ameliorate postoperative pain outcomes after laparoscopic surgery: a meta-analysis of randomized controlled trials. Anesth Analg. 2014;118:454–63.
31. Yamamoto H, Shido A, Sakura S, Saito Y. Monitored anesthesia care based on ultrasound-guided subcostal transversus abdominis plane block for continuous ambulatory peritoneal dialysis catheter surgery: case series. J Anesth. 2016;30(1):156–60.
32. Sammour T, Zargar-Shoshtari K, Bhat A, et al. A programme of Enhanced Recovery After Surgery (ERAS) is a cost-effective intervention in elective colonic surgery. N Z Med J. 2010;123:61–70.
33. McIsaac DI, Cole ET, McCartney CJL. Impact of including regional anaesthesia in enhanced recovery protocols: a scoping review. Br J Anaesth. 2015;115(52):ii46–56.
34. Dexter FD, Wachtel RE. Strategies for net cost reductions with the expanded role and expertise of anesthesiologists in the perioperative surgical home. Anesth Analg. 2014;118:1062–71.
35. Williams BA, Kentor ML, Vogt MT, Vogt WB, Coley KC, Williams JP, et al. The economics of nerve block pain management after anterior cruciate ligament reconstruction: significant hospital cost savings via associated PACU bypass and same-day discharge. Anesthesiology. 2004;100:697–706.
36. Sandu NS, Sidhu DS, Caplan LM. The cost comparison of infraclavicular brachial plexus block by nerve stimulation and ultrasound guidance. Anesth Analg. 2004;98:267–8.
37. Saporito A, Sturni E, Borgeat A, Aguirre J. Continuous popliteal sciatic nerve block for outpatient foot surgery: incidence of unplanned visits and readmissions compared to inpatient management—a randomized, controlled trial. Anaesthesia. 2014;69:1197–205.
38. Hadzic A, Karaca PE, Hobeika P, Unis G, Dermksian J, Yufa M, et al. Peripheral nerve blocks result in superior recovery profile compared with general anesthesia in outpatient knee arthroscopy. Anesth Analg. 2005;100:976–81.
39. Gonano C, Kettner SC, Ernstbrunner M, Schebesta K, Chiari A, Marhofer P. Comparison of economical aspects of interscalene brachial plexus blockade and general anaesthesia for arthroscopic shoulder surgery. Br J Anaesth. 2009;103(3):428–33.
40. Ilfeld BM, Mariano ER, Williams BA, Woodward JN, Macario A. Hospitalization costs of total knee arthroplasty with a continuous femoral nerve block provided only in the hospital versus on an ambulatory basis: a retrospective, case control, cost-minimization analysis. Reg Anesth Pain Med. 2007;32:46–54.
41. Urquhart DM, Hanna FS, Brennan SL, Wluka AE, Leder K, Cameron PA, et al. Incidence and risk factors for deep surgical site infection after primary total hip arthroplasty: a systematic review. J Arthroplast. 2010;25(8):1216–22.
42. Helwani MA, Avidan MS, Abdallah AB, Kaiser DJ, Clohisy JC, Hall BL, et al. Effects of regional versus general anesthesia on outcomes after total hip arthroplasty. J Bone Joint Surg Am. 2015;97:186–93.
43. Rinehardt EK, Sivarajan M. Costs and wastes in anesthesia care. Curr Opin Anaesthesiol. 2012;25:221–5.
44. Swenson JD, Davis JJ. Getting the best value for consumable supplies in regional anesthesia. Int Anesthesiol Clin. 2011;49:94–103.
45. Swenson JD, Klingler KR, Pace NL, Davis JJ, Loose EC. Evaluation of a new needle guidance system for ultrasound: results of a prospective, randomized, blinded study. Reg Anesth Pain Med. 2016;41(3):356–61.
46. Armstrong KP, Cherry RA. Brachial plexus anesthesia compared to general anesthesia when a block room is available. Can J Anaesth. 2004;51(1):41–4.
47. Drolet P, Girard M. Regional anesthesia, block room and efficiency: putting things in perspective. Can J Anaesth. 2004;51:1–5.
48. Kim TW, Mariano ER. Updated guide to billing for regional anesthesia (United States). Int Anesthesiol Clin. 2011;49(3):84–93.
49. Mariano ER. Making it work: setting up a regional anesthesia program that provides value. Anesthesiol Clin. 2008;26:681–92.
50. Gerancher JC, Viscusi ER, Liguori GA, McCartney CJ, Williams BA, Ilfeld BM, et al. Development of a standardized peripheral nerve block procedure note form. Reg Anesth Pain Med. 2005;30:67–71.
51. American Medical Association. CPT 2017 Professional Edition. American Medical Association; 2017.

3 Training and Education of a Physician for Regional Anesthesia

Chris M. Farlinger and Jonathan C. Beathe

Abbreviations

ACGME	Accreditation Council for Graduate Medical Education
ASRA	American Society of Regional Anesthesia
CA	Clinical anesthesia
ESRA	European Society of Regional Anaesthesia and Pain Therapy
PNB	Peripheral nerve block
QI	Quality improvement
RCC	Anesthesiology Residency Review Committee
USGA	Ultrasound for regional anesthesia

History

As Carl Koller demonstrated the first ophthalmologic surgical procedure using a local anesthetic in 1884, a new realm of possibilities in anesthesia emerged [1]. It was not long before upper extremity anesthesia was described utilizing both axillary and supraclavicular approaches to the brachial plexus in 1911. It was during this time that the earliest form of regional anesthesia education took place in the form of apprenticeships. Surgeons, such as Harvey Cushing, blocked nerves under direct vision during inhalational anesthesia, and such practices influenced subsequent pioneers in the field of regional anesthesia [2]. For instance, prior to publishing his classic text, *Regional Anesthesia*, Gaston Labat worked extensively in Paris with his mentor, surgeon Victor Pauchet [3]. While in Paris, his demonstration of a technique to provide full abdominal relaxation—without the complications of deep ether anesthesia—impressed observers such as Dr. Charles Mayo. Understanding the potential contribution to surgical practice, Dr. Mayo persuaded Labat to leave Paris for the Mayo Clinic. After his tenure at the Mayo Clinic, Labat would go on to become the first president of the original American Society of Regional Anesthesia (ASRA) in 1923. The teachings of Dr. Labat had significant influence on the next generation of anesthesiologists, including the creator of the modern specialty of anesthesiology, Emery Rovenstine [4]. Thus, it is impossible to separate the birth of anesthesiology as a specialty from the early work of the first regional anesthesia educators.

Evolution of Practice

Despite the initial enthusiasm for regional anesthesia techniques, interest and practice have fluctuated over the past century. This becomes

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_3

evident in review of anesthesiology resident education, which did not formally recognize a minimum number of regional anesthetic blocks as a requirement of training until 1996 [5]. It was at this time that the Anesthesiology Residency Review Committee (RRC) of the Accreditation Council for Graduate Medical Education (ACGME) addressed discrepancies in the regional anesthesia experience of graduating residents [6].

Multiple surveys have demonstrated how regional anesthesia training experience has evolved. For instance, training programs in 1980 reported the use of a regional anesthetic in 21.3% of cases. However, the discrepancies between training programs (2.8–55.7% of total delivered anesthetics) led to concern that some residents would not gain adequate experience to meet the growing demand to provide regional anesthetics [7]. When the popularity of epidural anesthesia and resident exposure to pain consultations expanded in the 1980s, survey data reflected the shift. In 1990, the reported use of regional anesthetics increased to 29.8% [8]. Unfortunately, wide variation in the use of regional anesthesia between training programs remained unchanged. The disparities in the number of regional anesthetics performed between residency training programs improved over the course of the next 10 years; however, the overall use of regional anesthesia by residents in the year 2000 appeared to plateau at 30.2% [9]. Since then, it is unlikely that the overall utilization of regional anesthesia by resident training programs has increased markedly. Consider 2006 data from a prominent tertiary teaching hospital: even when selecting for surgical procedures amenable to a regional anesthetic, only 36.5% of such patients are provided regional anesthesia [10]. Since that data was published in 2009, no new data has been published to indicate that the rate of regional anesthesia has grown outside of the obstetrical population.

As of July 1, 2016, the latest ACGME Program Requirements for Graduate Medical Education in Anesthesiology stated that residents should be exposed to 1 month of a regional analgesia experience rotation [5]. In addition, the stated minimum regional anesthesia clinical experience that should be obtained by each resident includes 40 epidurals, 40 spinals, and 40 peripheral nerve blocks (PNB). In addition to these regional experience, resident education must include a minimum of 1 month in an acute perioperative pain management rotation as well as a 1-month rotation for the assessment and treatment of inpatients and outpatients with chronic pain [5].

If you look at the Canadian Resident Curriculum set forth by the Royal College of Physicians and Surgeons of Canada, there is not a specific time frame or number of blocks to determine if someone is proficient in regional anesthesia. Within the 4 years of anesthesiology rotations, 18 months must be of adult anesthesiology which must include general and regional anesthetic experiences [11]. Furthermore, 1 month must be spent in a chronic pain management setting. Outside of rotation requirements, the National Curriculum details that the "Anesthesiologist shall demonstrate knowledge of the anatomy and physiology of, and an approach to, regional anesthesia" [11]. Although this is quite vague, it goes into further detail about specific requirements of pharmacology, physiology, technology (ultrasound, nerve stimulation, etc.), and clinical applications of neuraxial and peripheral nerve blocks [11].

In the United States, a 1999 survey discovered that 50% of graduating clinical anesthesia (CA) year three residents, although confident in performing neuraxial anesthesia, lacked adequate experience with many commonly performed peripheral nerve blocks [12]. A follow-up survey of graduating residents in 2011 showed that the number of PNBs performed during residency had significantly increased over the prior decade. However, although the study demonstrated that 91% of respondents met the ACGME criteria for PNBs, deficiencies in confidence still exist [13]. It is hypothesized that as the ACGME does not provide guidelines as to the specific types of blocks, it is possible that a resident may meet the requirement with one or two types of blocks and feel quite confident in those but lack training in other PNBs [13].

The question arises as to what is the best method of improving resident proficiency of

PNBs upon graduation. It has traditionally been felt that as PNBs have a steep learning curve, expanding resident exposure would be the most critical factor in improving resident education and confidence. This has resulted in many programs developing specific regional anesthesia rotations. However, debate still exists as consideration of the number of cases alone does not necessarily reflect resident mastery of the regional anesthetic technique.

The original concept of volume-based training model for surgical training dates back to 1889 and for more than 100 years did not change as it was expected that learners train under the direct supervision of a senior attending until they acquired enough "cases" to be competent [14]. Studies that address this topic also vary greatly in methodology and lack standardization. Existing studies do, however, provide insight into what level of experience may be necessary to achieve a specific endpoint of success. In one of the earliest investigations of trainee "learning curves" in regional anesthesia, Kopacz and colleagues demonstrated that significant improvement in success rate occurs after 20 spinal and 25 epidural anesthetics [15]. They also reported that a 90% success rate was not achieved until after performing 45 spinals and 60 epidural anesthetics. In a subsequent analysis of resident training at a Swiss institution using a standardized self-evaluation questionnaire, different results were demonstrated. Although a rapid improvement of success was also observed during the first 20 attempts, 71 attempts at spinal anesthesia were required to reach a success rate of 90% [16]. The study described epidural anesthesia as the most difficult task, with a success rate of only 80% after 90 attempts (Fig. 3.1). With the axillary approach to brachial plexus blockade, only a 70% success rate was achieved after 20 cases (Fig. 3.2). The first assessment of the number of attempts necessary for a resident to achieve proficiency in interscalene anesthesia demonstrated that 87.5% report "autonomous success" after 15 cases [17]. Of note, only 50% of residents were able to perform interscalene anesthesia autonomously after seven to nine previous block attempts. These studies, despite their limitations, suggest that the current RRC requirement that mandates experience with 40 *unspecified* peripheral nerve blocks is likely inadequate to ensure proficiency with various *specific* peripheral nerve blocks.

With the continued addition of emerging technology, the application of ultrasound for regional anesthesia (UGRA) is emerging as a standard of care, and its use in experienced hands is known to reduce complications and improve the overall quality of the procedure [14]. Gaining expertise in UGRA requires acquisition of new knowledge and technical skills, including, but not limited to, sonoanatomy, hand-eye coordination, and skills in ultrasound scanning and needle insertion [14]. Without the appropriate development of these skills, it has been shown that USGA of the bra-

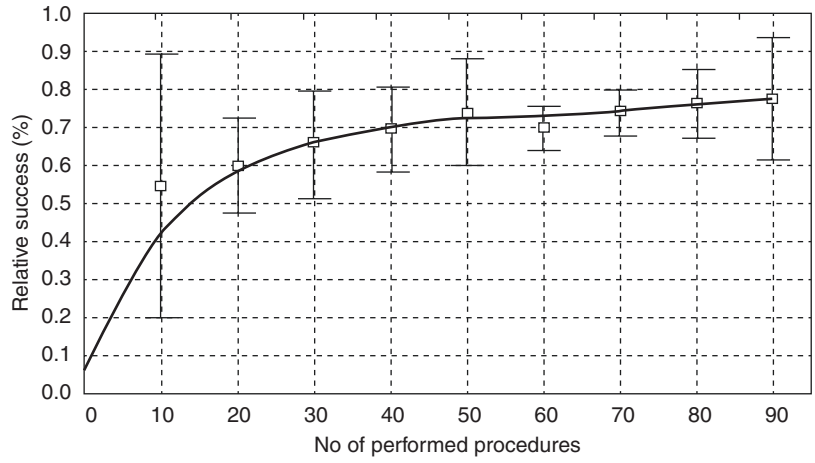

Fig. 3.1 Learning curve—epidural anesthesia

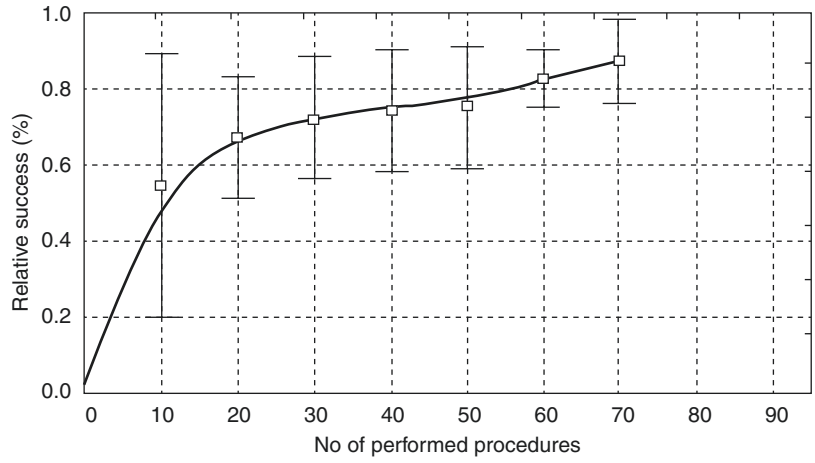

Fig. 3.2 Learning curve—brachial plexus anesthesia

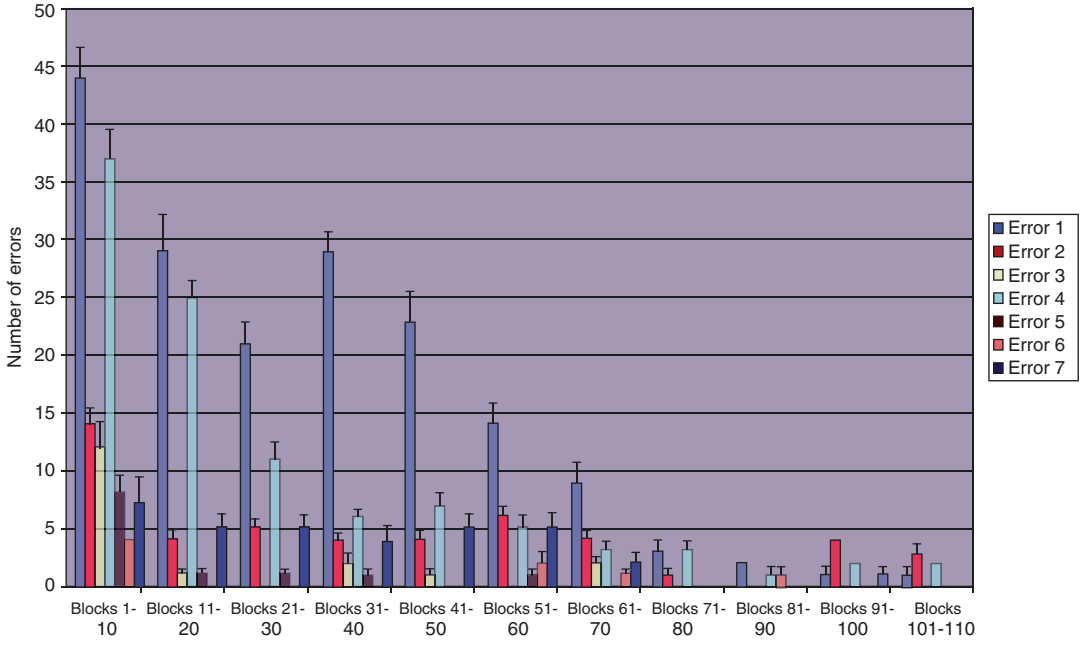

Fig. 3.3 Distribution of errors for all residents combined

chial plexus is not directly applicable to the safe performance of interscalene blockade utilizing the paresthesia technique in untrained hands. Sites and colleagues, in a study characterizing novice behavior associated with learning UGRA, explore the entirely new set of skills involved in UGRA [18]. By using video analyses of 520 nerve blocks performed by anesthesia residents, a multitude of errors such as "needle not visualized during advancement" were assessed. They observed that by the 60th block, the trainee was still committing an average of 2.8 errors per procedure (Fig. 3.3). It is a reasonable hypothesis that the necessary clinical exposure associated with proficiency is related to not only the specific peripheral nerve block but also to the method used to accomplish the technique. Few studies investigate the influence of ultrasound and related imaging technology upon learning curves for peripheral nerve blocks. Early data suggest,

however, that residents inexperienced in UGRA can rapidly master basic ultrasound skills in a simulated interventional procedure [19]. Thus, basic skill development common for all blocks, including ultrasound transducers positioning, ultrasound transducer orientation, as well as needle insertion, needle tracking, and needle tip visualization, could be beneficial prior to clinical exposure. This could be developed in junior years of training via simulation and target models.

Overcoming Obstacles and Expanding Practice

When attempting to improve the regional anesthesia exposure of residents, it is helpful to understand the potential obstacles that exist to expanding practice. As observed by Hanna and colleagues in a prospective observational study in 2009, a multitude of factors shape resident experience in regional anesthesia [10]. In over 2000 cases amenable to a regional anesthetic, the frequency and reasons for not performing such a technique were investigated. Surprisingly, they found that anesthesiology-related factors—not the surgeon, patient, or medical reasons—were the primary factors for not performing a regional anesthetic. They also observed, predictably, that designated regional anesthesia faculty performed regional anesthesia more often (68% of cases). In over 98% of the cases in which regional anesthesia was not performed, despite being an appropriate anesthetic selection, staff members not designated as "regional anesthesia faculty" were involved (Fig. 3.4). These findings support the assertion that the process of increasing resident exposure to regional anesthesia is facilitated by the presence of dedicated, trained faculty. Even with the addition of regional anesthesia experts to faculty rosters, the specialty faces additional challenges to improving the resident experience. The apprenticeship model of education remains the predominant teaching style in residency training programs, despite having significant limitations. Problems with inconsistency in the quality of learning experiences need to be addressed. Additional challenges include the development of improved methods of trainee evaluation and expanding curriculum development to achieve consistency in both technical and clinical achievement. To improve the environment for resident exposure and mastery of regional anesthesia, curriculum development has moved toward a competency model utilizing developing resources to foster knowledge and skill acquisition prior to performing in the clinical environment.

Curriculum Development

As most physicians lack formal training in education, the apprenticeship model of education remains the predominant teaching style in regional anesthesia. Unfortunately, progress in education methodology has not kept pace with the significant advances in medical technology and standards of care. Recently, educators in regional anesthesia have identified ways to improve upon existing models of teaching. Initial efforts to improve education have focused on increasing the numbers of peripheral nerve blocks performed by trainees. Martin and colleagues, outlining the use of a CA-3 resident in the preoperative area to perform regional anesthesia techniques, have described such an educational model [20]. This relatively simple modality significantly increased resident exposure to regional anesthesia (Fig. 3.5). In support of these findings, it has been demonstrated that residency programs that include a specific peripheral nerve block rotation expose their trainees to a greater number of peripheral nerve block techniques [21]. More recently, the focus of curricula has expanded to complement traditional patient care experiences with novel educational activities. In their sentinel article, Smith and colleagues at the Mayo Clinic have described in detail their institution's approach to regional anesthesia education [22]. As stated, the primary educational objectives of their curriculum include (1) standardizing educational content, (2) quality care and patient safety, and (3) resident evaluation and improvement. Some highlights of this "learner-centered" approach include a comprehensive "preclinical" educational program for UGRA, a list of criteria

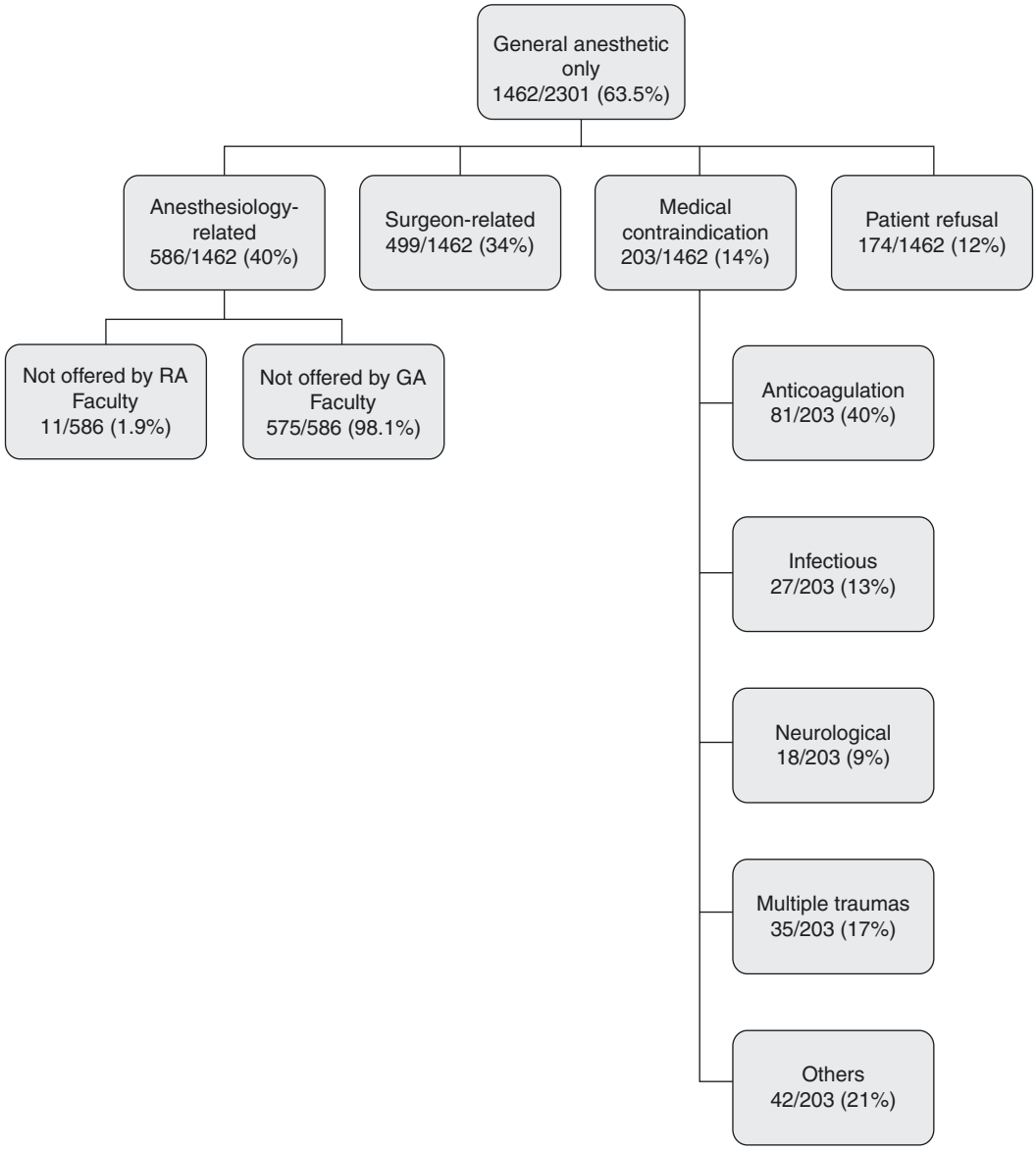

Fig. 3.4 When selecting for surgical procedures amenable to a regional anesthetic, many patients still receive general anesthesia (63.5%). This is primarily attributable (40%) to anesthesiology-related factors, such as a lack of regional anesthesia experience. *GA* general anesthesiology, *RA* regional anesthesiology

expected of residents during patient care, and CA-2 or CA-3 residents functioning in the role of a teacher of CA-1 residents during the 8-week rotation in regional anesthesia. Placing a senior resident in the role of educator is supported by evidence of potential benefit to academic performance and perceptions of clinical competency [23]. Clinical expectations during the regional anesthesia rotation and the performance checklist for UGRA proficiency are reproduced in Tables 3.1 and 3.2. Their work is a significant contribution to advancing the quality of regional anesthesia training and can be adapted to accommodate the needs of various institutions. However, work is needed to demonstrate the validity of this approach to improving resident education.

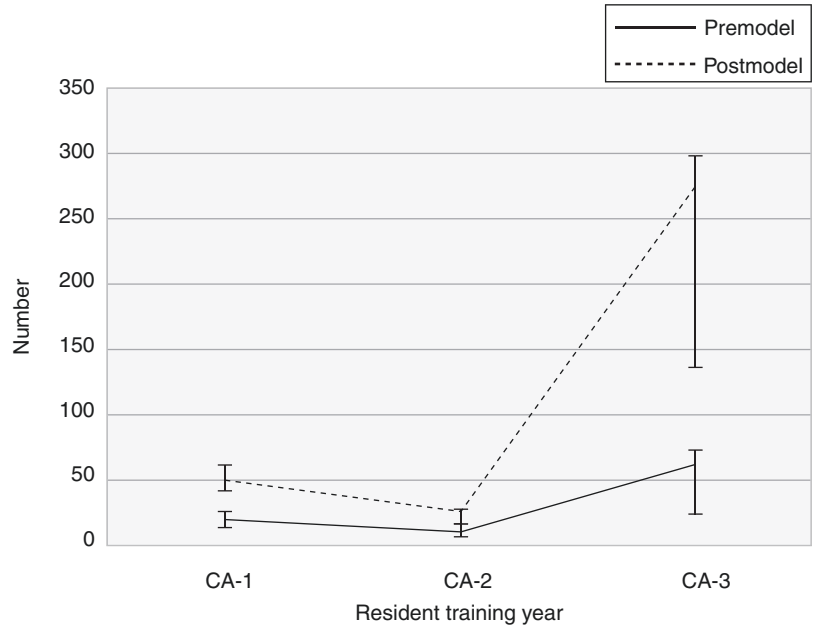

Fig. 3.5 An organized regional anesthesia rotation increases resident exposure to nerve blocks

In a follow-up to this study, faculty from five academic institutions jointly developed a pilot project to demonstrate how anesthesiology program could collaborate in the creation and sharing of education resources in regional anesthesia [24]. The goal of this was to develop an "Anesthesia Toolbox" to improve preclinical learning of regional procedures. Sample Toolbox Curriculum Map for teaching regional anesthesia across the CA years is available in their paper.

Additional challenges in curriculum development were created with the introduction and rapid evolution of UGRA. Questions surrounding the standards of care, assessment of competency, and definition of expert performance naturally follow the emergence of new technology. Efforts to define the scope of practice of UGRA have been summarized by the ASRA and the European Society of Regional Anaesthesia and Pain Therapy (ESRA) Joint Committee Recommendations for Education and Training in UGRA [25]. This collaborative effort is an example of the profession accepting responsibility for self-regulation and practice improvement while recommending that UGRA privileges be based at the individual institution level. The stated goals of the document are to (1) define and structure the common tasks used when performing an ultrasound-guided nerve block, (2) articulate the core competencies and skill sets associated with UGRA, (3) suggest a training process for both established practitioners and residents, and (4) recommend the establishment of a quality improvement (QI) process for UGRA. The skill sets that the ASRA-ESRA Joint Committee has associated with proficiency are reproduced in Table 3.3. The document provides an additional framework to enhance existing UGRA curricula, both for residency training and postgraduate pathways. It also further establishes the representative tasks that define the practice of UGRA, which aids the development of novel educational tools such as simulator-based instruction.

Comparisons between the training of laparoscopic surgery and USGA have led to further advancements in regional anesthesia training which highlights the need for a specifically designed curriculum where each skill has defined stages and outcomes and where each stage must be successfully completed in succession [26]. Based on theories and models, a number of components have been incorporated into separate phases of training. Niazi et al. discuss the breakdown of learned skills using interscalene plexus block as an example [14]. This method of training breaks

Table 3.1 Clinical expectations during the regional anesthesia rotation

Preoperative care
Review patient history and relevant laboratory data
Complete a physical examination
• Appropriate preoperative neurologic evaluation
Develop an anesthetic plan with the patient
• Understand impact of comorbid conditions
• Select appropriate regional technique
• Discuss risks, benefits, and alternatives of anesthetic options
Order preoperative analgesic medications
Discuss the patient with the attending anesthesiologist
Perform the appropriate regional procedure(s) under direct supervision
Intraoperative care
Evaluate the block before surgical incision
Develop an approach to managing field blocks
• Block supplementation (if indicated or appropriate)
• Conversion to general anesthesia
• Appropriate use of supplemental opioids and sedation
Postoperative care
Manage patient in the postanesthesia care unit
• Appropriately assess and manage pain
• Perform postoperative regional techniques (if applicable)
• Manage acute postoperative issues (e.g., nausea and vomiting, perineural catheters)
Participate in acute pain service rounds
• Evaluate the efficacy of the regional technique used
• Adjust postoperative analgesics as needed
• Monitor patients for potential complications

Data from [22]

down the learning of a procedure into knowledge, skill, and clinical procedure. The knowledge component, which includes specific anatomy, indications, and complications, can be done outside the operating room through didactic lectures or e-modules. The skill component can also be completed outside the OR through simulators, models, or other high-fidelity models. After completion of these steps, the trainee can then move to clinical exposure. It has been shown through a number of studies that preclinical simulation training allows trainees to perform blocks faster with fewer mistakes and improved performance scores. To further facilitate the development of a competency-based

Table 3.2 Performance checklist for ultrasound-guided regional anesthesia proficiency

Ultrasound-guided regional anesthesia element	Resident performance
Ultrasound equipment	• Navigate to patient demographic screen
	• Select appropriate probe and frequency for application
	• Adjust depth, gain, and contrast to optimize image
	• Capture appropriate image for medical record documentation
	• Navigate to "End Exam" screen
	• Store image for medical record documentation
Scanning techniques and sonoanatomy	• Hold and orient probe correctly
	• Perform basic probe movements
	• Use appropriate gel and probe pressure while scanning
	• Recognize basic image artifacts
	• Distinguish the sonographic appearance of the artery, vein, bone, muscle, and nerve
	• Identify the relevant neuroanatomy for interscalene, supraclavicular, infraclavicular, and axillary brachial plexus blockade
	• Identify and trace upper extremity peripheral nerves
	• Identify the sciatic nerve within the popliteal fossa
	• Identify the tibial nerve above the medial malleolus
Sonographic needle guidance	• Demonstrate appropriate hand positions for the probe and the needle
	• Demonstrate in-plane and out-of-plane needle-to-probe orientation
	• Identify simulated phantom target and needle insertion location
	• Adjust needle depth and trajectory to approximate target
	• Demonstrate basic techniques for optimizing needle visualization
	• Maintain needle and target imaging >80% of the time during simulation
	• Advance needle only when visualized

Data from [22]

Table 3.3 Skill sets associated with proficiency

Understanding ultrasound image generation and device operations	Image optimization (non-device related)	Image interpretation	Needle insertion and injection
Understanding basic technical principles of image generation	Learn the importance of transducer pressure	Identify nerves	Learn the in-plane technique, maximizing needle visualization
Selection of the appropriate transducer	Learn the importance of transducer alignment	Identify muscles and fascia	Learn the out-of-plane technique
Selection of the appropriate depth and focus settings	Learn the importance of transducer rotation	Identify blood vessels, distinguish artery from vein	Learn the benefits and limitations of both techniques
Understanding and appropriate use of both time gain compensation and overall gain	Learn the importance of transducer tilting	Identify bone and pleura	Learn to recognize intramuscular needle location
Understanding and application of color Doppler		Identify common acoustic artifacts	Learn to recognize correct and incorrect local anesthetic spread
Archiving images		Identify common anatomic artifacts (pitfall errors)	Conduct proper ergonomics
Follow ASRA-ESRA standardization for screen orientation to the patient		Identify vascularity associated with needle trajectory	Minimize unintentional transducer movement Identify intraneuronal needle location

Data from [25]

learning environment, Woodworth et al. developed a 47-item multiple choice-style online test of ultrasound interpretation which can be used to assess competency of milestones in the achievement of regional anesthesia training [27].

Fellowship Training

Regional anesthesia practice is not unlike most professions in that considerable time is required to establish expert performance. Evidence also exists that superior medical treatments are linked to more extensive training and specialization in associated medical fields [28]. As such, fellowship training in regional anesthesia should be considered a means to excel beyond basic competencies and become an expert in the field. Formal regional anesthesia fellowships emerged in the early 1980s, and subspecialty training offered by regional anesthesia fellowships has grown substantially to include 71 institutions to date (Table 3.4). This is an increase in 35 programs since the first edition of this chapter alone. However, with the absence of uniform standards, early programs did vary in duration, organization, and objectives. It was not until October 2003, after collaboration with the directors of several regional anesthesia fellowship programs, that the first Guidelines for Regional Anesthesia Fellowship Training were developed [29]. The guidelines recommend the necessary components of subspecialty fellowship training, emphasizing the clinical foundation of regional anesthesia, educational curricula, and opportunity for academic achievement. This document was reviewed in 2006, again in early 2009, and the most recent publication is from 2014 (3rd edition) [30]. Although they extend beyond clinical considerations, an important goal of the published guidelines is to provide a framework to progress beyond basic proficiency and achieve focused clinical expertise. With rapidly emerging technologies and new procedures, it becomes more difficult for physicians to safely integrate the latest regional anesthesia techniques into their practice. As mentioned, evidence exists that residency training alone is not adequate to

Table 3.4 Regional anesthesia fellowship programs from the ASRA website

Program	Location
Cedars-Sinai Medical Center	Los Angeles, CA
Harbor-UCLA	Torrance, CA
Keck School of Medicine, University of Southern California	Los Angeles, CA
Stanford University Medical Center	Stanford, CA
UCLA Department of Anesthesia and Perioperative Medicine	Los Angeles, CA
University of California at San Diego	San Diego, CA
University of California, Irvine	Orange, CA
University of California, San Francisco	San Francisco, CA
University of Colorado Denver School of Medicine	Aurora, CO
Integrated Anesthesia Associates (IAA)/Hartford Hospital	Hartford, CT
St. Francis Hospital and Medical Center	Hartford, CT
Yale University School of Medicine	New Haven, CT
Jackson Memorial Medical Center	Miami, FL
Mayo Clinic – Mayo School of Graduate Medical Education	Jacksonville, FL
The Andrews Institute for Orthopedics & Sports Medicine	Gulf Breeze, FL
University of Florida College of Medicine	Gainesville, FL
Emory University Hospital	Atlanta, GA
McGaw Medical Center of Northwestern University	Chicago, IL
University of Illinois at Chicago	Chicago, IL
University of Iowa	Iowa City, IA
The University of Kansa Hospital	Kansas City, KS
Ochsner Medical Center	New Orleans, LA
Johns Hopkins University School of Medicine	Baltimore, MD
University of Maryland	Baltimore, MD
Boston Children's Hospital	Boston, MA
Brigham and Women's Hospital	Boston, MA
Massachusetts General Hospital	Boston, MA
The University of Michigan	Ann Arbor, MI
Mayo Clinic – Mayo School of Graduate Medical Education	Rochester, MN
University of Minnesota	Minneapolis, MN
Washington University	St. Louis, MO
Geisel School of Medicine at Dartmouth	Lebanon, NH
University of New Mexico	Albuquerque, NM
Columbia University/NYPH Medical Center Columbia University	New York, NY
Hospital for Special Surgery	New York, NY
Montefiore Medical Center/Albert Einstein College of Medicine	Bronx, NY
Mount Sinai Medical Center, Mount Sinai School of Medicine	New York, NY
Mount Sinai St. Luke's Roosevelt Hospital	New York, NY
NYU/Hospital for Joint Disease	New York, NY
Weill Cornell Medical College/NYPH	New York, NY
Westchester Medical Center/New York Medical College	Valhalla, NY
Duke University Medical Center	Durham, NC
University of North Carolina at Chapel Hill	Chapel Hill, NC
Wake Forest School of Medicine	Winston-Salem, NC
Cleveland Clinic	Cleveland, OH
Ohio State University Medical Center	Columbus, OH

(continued)

Table 3.4 (continued)

Program	Location
Oregon Health & Science University	Portland, OR
Drexel University College of Medicine	Philadelphia, PA
Thomas Jefferson University	Philadelphia, PA
University of Pittsburgh	Pittsburgh, PA
Medical University of South Carolina	Charleston, SC
Vanderbilt University Medical Center	Nashville, TN
University of Texas Health Sciences Center at Houston	Houston, TX
University of Texas Southwestern Medical Center	Dallas, TX
University of Utah	Salt Lake City, UT
University of Virginia	Charlottesville, VA
University of Washington's Department of Anesthesiology and Pain Medicine	Seattle, WA
Virginia Mason Medical Center	Seattle, WA
Medical College of Wisconsin	Milwaukee, WI
University of Wisconsin Hospital and Clinics	Madison, WI
United States Military Walter Reed National Military Medical Center	Bethesda, MD
Centre Hospitalier de l'Universite de Montreal	Montreal, QC
Dalhousie University	Halifax, NS
McGill University	Montreal, QC
Toronto Western Hospital	Toronto, ON
University of Toronto – St. Michael's Hospital	Toronto, ON
Sunnybrook Health Sciences Center	Toronto, ON
University of Ottawa	Ottawa, ON
University of Alberta	Edmonton, AB
University of British Columbia	Vancouver, BC

achieve mastery in many regional anesthesia techniques, particularly peripheral nerve blockade. This deficiency likely translates to fewer acute pain management options for the patients of our graduating residents. In this regard, additional fellowship training can broaden the pain management strategies used by practitioners by providing the necessary expertise. An additional argument for fellowship training is to provide an expansion upon the limited experience that residency "block room" regional anesthesia training provides. The intraoperative portion of a regional anesthetic is not to be underestimated, as myriad challenges, complications, and emergencies occur during this time. Fellowship training provides further experience to anticipate, recognize, and appropriately treat the patient in a timely fashion when such scenarios present themselves. Over the course of a fellowship, trainees also have the opportunity to achieve advanced exposure to regional techniques on patients with significant comorbidities such as morbid obesity, severe scoliosis, ankylosing spondylitis, or significant cardiopulmonary disease. Following regional anesthesia fellowship, trainees are empowered to apply regional anesthesia techniques to clinical situations that are not traditionally endorsed. For instance, with appropriate execution and patient selection, neuraxial blockade can be a reasonable anesthetic option for hip surgery in the setting of aortic stenosis [31]. In order to ensure a safe outcome, advanced techniques such as hypotensive epidural anesthesia [32] also require a level of expertise that fellowship training provides. Although clinical considerations are arguably the foundation of fellowship training, didactic components are not of lesser value as well as the development of future competency-based models including simulation. Particularly, as regional practice expands in an era of regulatory oversight and evolving rules and measures, it will be increasingly important to inject our specialty with research initiatives that further validate and justify our

practice. Fellowship training is a platform that can be increasingly utilized to meet this end by providing important exposure to academic pursuits. With increasing interest and expansion of regional anesthesia technique and practice, fellowship training is an indispensable means to provide valued experts in the field.

The Practicing Anesthesiologist

Although fellowship training provides the definitive opportunity to achieve expertise in regional anesthesia, it is not always practical for physicians in the midst of their careers to dedicate a full year to formal training. Advances in science and technology have and will continue to challenge physicians in every discipline to stay current with modern practice and procedures. Parallels can be drawn between the practicing anesthesiologist seeking to advance his/her regional anesthesia skills and the experienced surgeon who would like to introduce a modern yet unfamiliar laparoscopic technique into his/her practice [33]. Similar questions are raised regarding the safety and efficacy of the new modality and if specific minimal educational requirements should be met [34]. Just as surgeons are faced with the daunting responsibility of ensuring the safe introduction of new procedures into practice, expert regional anesthesiologists must also adequately provide the educational opportunities required of lifelong learning and the development of new technical skills. This may take the form of the fellowship graduate arriving to a practice unfamiliar with UGRA, providing new skills and information to otherwise more experienced physicians. As fellowship graduates alone cannot meet this educational demand, a multitude of opportunities have developed to provide regional anesthesia exposure to the practicing anesthesiologist (Table 3.5). Although such opportunities provide a valuable service, extensive work remains to adequately define and assess competency in regional anesthesia. As we already know, limited training is associated with higher complication rates. Consider laparoscopic surgical data, in which the

Table 3.5 Regional anesthesia continuing education programs

Annual ASA Meeting
Annual Spring ASRA Meeting (focused on regional anesthesia)
Annual Fall ASRA Meeting (focused on pain medicine)
ASRA Excellence in Regional Anesthesia Workshops Regional Workshops Northwestern University's Feinberg Pavilion – Chicago, Illinois
Duke University – Durham, North Carolina
ASRA Ultrasound for Pain Medicine Workshops – Regional Locations: Rush University Medical Center, The Cleveland Clinic
Annual International Anesthesia Research Society Meeting
Annual HSS Regional Anesthesia Symposium – "Controversies and Fundamentals in Regional Anesthesia"
Annual NYSORA Meetings in Asia, Europe, and America
Annual International Anesthesia Research Society Meeting
Ultrasound for Regional Anesthesia (ISURA) 2010, Toronto, Canada
Ultrasound-Guided Regional Anesthesia Preceptorship, Duke University Medical Center, Durham, NC
Ultrasound-Guided Regional Anesthesia and Vascular Access, Northwest Anesthesia Seminars, various locations
Carolina Refresher Lectures: Care of the Surgical Patient 2010, Kiawah, SC
Dannemiller Anesthesiology Review Course 2010, Chicago, IL
"In Celebration of Patient Safety" Florida Society of Anesthesiologists (FSA) 2010 Annual Meeting, Palm Beach, FL
Ninth Biannual Hands on Ultrasound-Guided Regional Anesthesia Workshop, Houston, TX
Hawaii Anesthesiology Update 2010, Maui, HI
Texas Society of Anesthesiologists 2010 Annual Meeting, San Antonio, TX
First International Congress of Regional Anesthesia and Pain Interventions
Fourth Annual Regional Anesthesia in Children Conference, Seattle, WA
Anaesthesia in the Office-Based Setting, Boston, MA
Anesthesia Camp Laguna Beach, Laguna Beach, CA
Ontario Anesthesia Meeting, Toronto, Canada
Regional Anesthesia Study Center of Iowa (RASCI) Workshop, Iowa City, IA

(continued)

Table 3.5 (continued)

Introductory Ultrasound Workshop, Toronto, Canada
Advances in Physiology and Pharmacology in Anesthesia and Critical Care, White Sulphur Springs, WV
Illinois Society of Anesthesiologists Midwest Anesthesiology Conference (MAC), Chicago, IL
21st Annual University of California – Davis Anesthesiology Update, Monterey, CA
Pediatric Anesthesia Conference: New and Challenging Cases, Austin, TX
Survey of Current Issues in Surgical Anesthesia, Naples, FL
Advanced Ultrasound Workshop, Toronto, Canada
New York State Society of Anesthesiologists' 64th Postgraduate Assembly in Anesthesiology (PGA), New York, NY

rate of complications associated with the clinical learning curve can be decreased by additional education [35]. Future efforts to accomplish the difficult task of competency assessment will allow us to further optimize patient safety in regional anesthesia.

Review Questions

1. This physician helped teach the founder of the modern specialty of anesthesiology and became the first president of xthe original American Society of Regional Anesthesia in 1923:
   (a) Emery Rovenstine
   (b) Gaston Labat
   (c) Victor Pauchet
   (d) Carl Koller
2. Between the year 1980 and 2000, the reported use of regional anesthetics by training programs increased by approximately what percent?
   (a) 5%
   (b) 10%
   (c) 15%
   (d) 20%
3. Considering the use of regional anesthesia (RA), wide discrepancies existed between training programs in the 1980s. Approximately what range of RA case percentages were observed between low RA volume and high RA volume programs during this time?
   (a) <5–55%
   (b) 10–60%
   (c) 15–45%
   (d) 20–45%
4. By the year 2000, the overall use of regional anesthesia techniques by residents in training increased to approximately what percent of total case volume?
   (a) 25%
   (b) 30%
   (c) 35%
   (d) 40%
5. As of July 1, 2016, the ACGME Program Requirements for Graduate Medical Education in Anesthesiology state the following minimum number of epidural, spinal, and peripheral nerve blocks to be performed by each resident:
   (a) 40
   (b) 50
   (c) 60
   (d) 80
6. In the year 2011, approximately what percent of graduating anesthesia residents met the ACGME criteria for peripheral nerve blocks?
   (a) 75%
   (b) 80%
   (c) 85%
   (d) 90%
7. In early investigations of trainee "learning curves" in regional anesthesia, approximately what range of experience level was required to achieve a 90% success rate with spinal anesthesia?
   (a) 45–70 cases
   (b) 40–55 cases
   (c) 30–45 cases
   (d) 50–60 cases
8. After 60 ultrasound-guided nerve blocks performed by trainees, what is the approximate average number of errors committed per procedure?
   (a) 1
   (b) 3
   (c) 5
   (d) 7

9. Designated regional anesthesia faculty are observed to select a regional anesthetic technique for approximately what percentage of cases?
   (a) 25%
   (b) 50%
   (c) 65%
   (d) 30%
10. What has been observed to be the primary reason for not performing a regional anesthetic in clinical settings amenable to such a technique?
   (a) Surgeon preference
   (b) Patient refusal
   (c) Anesthesiology-related factors
   (d) Medical contraindications
11. Significant improvement in success rates of *spinal* anesthesia are observed after approximately what level of experience is achieved?
   (a) 10 cases
   (b) 15 cases
   (c) 20 cases
   (d) 25 cases
12. Significant improvement in success rates of *epidural* anesthesia are observed after approximately what level of experience is achieved?
   (a) 10 cases
   (b) 15 cases
   (c) 20 cases
   (d) 25 cases
13. After the experience of 90 cases is achieved, what is the approximate observed success rate of epidural anesthesia?
   (a) 50%
   (b) 70%
   (c) 80%
   (d) 94%
14. Comparisons between training in what surgery has led to further advancements in regional anesthesia training?
   (a) Shoulder arthroscopy
   (b) Laparoscopic surgery
   (c) Cystoscopy
   (d) Video-assisted thoracic surgery
15. The Accreditation Council for Graduate Medical Education (ACGME) did not formally recognize a minimum number of regional blocks as a requirement of training until:
   (a) 1980
   (b) 1996
   (c) 1976
   (d) 1970

Answers:

1. b
2. b
3. a
4. b
5. a
6. d
7. a
8. b
9. c
10. c
11. c
12. d
13. c
14. b
15. b

References

1. Koller C. On the use of cocaine for producing anaesthesia on the eye. Lancet. 1884;2:990–2.
2. Deschner B, Robards C, Somasundaram L, Harrops-Griffiths W. The history of local anesthesia. In: Hadzic A, editor. Textbook of regional anesthesia and pain management. New York: McGraw-Hill; 2007. p. 15.
3. Vachon CA, Bacon DR, Rose SH. Gaston Labat's regional anesthesia: the missing years. Anesth Analg. 2008;107(4):1371–5.
4. Bacon DR. Gaston Labat, John Lundy, Emery Rovenstine, and the Mayo Clinic: the spread of regional anesthesia in America between the World Wars. J Clin Anesth. 2002;14(4):315–20.
5. ACGME Program Requirements for Graduate Medical Education in Anesthesiology 2016. https://www.acgme.org/Portals/0/PFAssets/ProgramRequirements/040_anesthesiology_2016.pdf.
6. McDonald S, Neal J. Teaching regional anesthesia. In: Hadzic A, editor. Textbook of regional anesthesia and acute pain management. New York: McGraw-Hill; 2007. p. 1174.

7. Bridenbaugh L. Are anesthesia resident programs failing regional anesthesia? Reg Anesth. 1982;7:26–8.
8. Kopacz DJ, Bridenbaugh LD. Are anesthesia residency programs failing regional anesthesia? The past, present, and future. Reg Anesth. 1993;18(2):84–7.
9. Kopacz DJ, Neal JM. Regional anesthesia and pain medicine: residency training—the year 2000. Reg Anesth Pain Med. 2002;27(1):9–14.
10. Hanna MN, Jeffries MA, Hamzehzadeh S, Richman JM, Veloso PM, Cox L, et al. Survey of the utilization of regional and general anesthesia in a tertiary teaching hospital. Reg Anesth Pain Med. 2009;34(3):224–8.
11. National Curriculum for Canadian Anesthesiology Residency: Royal College of Physicians and Surgeons of Canada; 2015 [updated Nov 2015]. 2nd ed. http://www.royalcollege.ca/cs/groups/public/documents/document/ltaw/mti2/~edisp/rcp-00126601.pdf.
12. Smith MP, Sprung J, Zura A, Mascha E, Tetzlaff JE. A survey of exposure to regional anesthesia techniques in American anesthesia residency training programs. Reg Anesth Pain Med. 1999;24(1):11–6.
13. Moon TS, Lim E, Kinjo S. A survey of education and confidence level among graduating anesthesia residents with regard to selected peripheral nerve blocks. BMC Anesthesiol. 2013;13(1):16.
14. Niazi AU, Peng PW, Ho M, Tiwari A, Chan VW. The future of regional anesthesia education: lessons learned from the surgical specialty. Can J Anaesth. 2016;63(8):966–72.
15. Kopacz DJ, Neal JM, Pollock JE. The regional anesthesia "learning curve". What is the minimum number of epidural and spinal blocks to reach consistency? Reg Anesth. 1996;21(3):182–90.
16. Konrad C, Schupfer G, Wietlisbach M, Gerber H. Learning manual skills in anesthesiology: is there a recommended number of cases for anesthetic procedures? Anesth Analg. 1998;86(3):635–9.
17. Rosenblatt MA, Fishkind D. Proficiency in interscalene anesthesia-how many blocks are necessary? J Clin Anesth. 2003;15(4):285–8.
18. Sites BD, Spence BC, Gallagher JD, Wiley CW, Bertrand ML, Blike GT. Characterizing novice behavior associated with learning ultrasound-guided peripheral regional anesthesia. Reg Anesth Pain Med. 2007;32(2):107–15.
19. Sites BD, Gallagher JD, Cravero J, Lundberg J, Blike G. The learning curve associated with a simulated ultrasound-guided interventional task by inexperienced anesthesia residents. Reg Anesth Pain Med. 2004;29(6):544–8.
20. Martin G, Lineberger CK, MacLeod DB, El-Moalem HE, Breslin DS, Hardman D, et al. A new teaching model for resident training in regional anesthesia. Anesth Analg. 2002;95(5):1423–7, table of contents.
21. Chelly JE, Greger J, Gebhard R, Hagberg CA, Al-Samsam T, Khan A. Training of residents in peripheral nerve blocks during anesthesiology residency. J Clin Anesth. 2002;14(8):584–8.
22. Smith HM, Kopp SL, Jacob AK, Torsher LC, Hebl JR. Designing and implementing a comprehensive learner-centered regional anesthesia curriculum. Reg Anesth Pain Med. 2009;34(2):88–94.
23. Busari JO, Scherpbier AJ. Why residents should teach: a literature review. J Postgrad Med. 2004;50(3):205–10.
24. Woodworth G, Juve AM, Swide CE, Maniker R. An innovative approach to avoid reinventing the wheel: the anesthesia education toolbox. J Grad Med Educ. 2015;7(2):270–1.
25. Sites BD, Chan VW, Neal JM, Weller R, Grau T, Koscielniak-Nielsen ZJ, et al. The American Society of Regional Anesthesia and Pain Medicine and the European Society of Regional Anaesthesia and Pain Therapy joint committee recommendations for education and training in ultrasound-guided regional anesthesia. Reg Anesth Pain Med. 2010;35(2 Suppl):S74–80.
26. Grantcharov TP, Reznick RK. Teaching procedural skills. BMJ. 2008;336(7653):1129–31.
27. Woodworth GE, Carney PA, Cohen JM, Kopp SL, Vokach-Brodsky LE, Horn JL, et al. Development and validation of an assessment of regional anesthesia ultrasound interpretation skills. Reg Anesth Pain Med. 2015;40(4):306–14.
28. Ericsson KA. Deliberate practice and the acquisition and maintenance of expert performance in medicine and related domains. Acad Med. 2004;79(10 Suppl):S70–81.
29. Hargett MJ, Beckman JD, Liguori GA, Neal JM, Education Committee in the Department of Anesthesiology at Hospital for Special Surgery. Guidelines for regional anesthesia fellowship training. Reg Anesth Pain Med. 2005;30(3):218–25.
30. Merchant R, Chartrand D, Dain S, Dobson G, Kurrek MM, Lagace A, et al. Guidelines to the practice of anesthesia—revised edition 2015. Can J Anaesth. 2015;62(1):54–67.
31. Ho MC, Beathe JC, Sharrock NE. Hypotensive epidural anesthesia in patients with aortic stenosis undergoing total hip replacement. Reg Anesth Pain Med. 2008;33(2):129–33.
32. Sharrock NE, Salvati EA. Hypotensive epidural anesthesia for total hip arthroplasty: a review. Acta Orthop Scand. 1996;67(1):91–107.
33. Sachdeva AK, Russell TR. Safe introduction of new procedures and emerging technologies in surgery: education, credentialing, and privileging. Surg Clin North Am. 2007;87(4):853–66, vi–vii.
34. Bass BL, Polk HC, Jones RS, Townsend CM, Whittemore AD, Pellegrini CA, et al. Surgical privileging and credentialing: a report of a discussion and study group of the American Surgical Association. J Am Coll Surg. 2009;209(3):396–404.
35. See WA, Cooper CS, Fisher RJ. Predictors of laparoscopic complications after formal training in laparoscopic surgery. JAMA. 1993;270(22):2689–92.

Part II

Basics and Clinical Practice in Regional Anesthesia

4 The Anatomy of Pain and Its Implications for Regional Anesthesiology Practice

Harry J. Gould III and Alan David Kaye

Introduction

In years past, the prevailing approach to providing pain control was focused on identifying underlying etiologies or pathologic syndromes, e.g., low back pain, trigeminal neuralgia, and cancer pain, that produce the pain. While treating the presumed source of the pain, attempts to improve the accompanying discomfort relied largely on the use of non-opioid medications and the limited use of opioid and adjuvant analgesics. Over the past 25 years, however, there has been a dramatic increase in our understanding of the nervous system and how stimuli associated with actual or potential tissue injury are transduced, transmitted, modulated, perceived, and interpreted to form the basis for initiating appropriate evasive or protective behavior, thereby avoiding or limiting injury. Our current bank of knowledge has led to the recognition that (1) pain in the chronic state is in itself a disease deserving consideration, assessment, and management; (2) pain is not a single entity but a complex, multifaceted experience that warrants detailed and comprehensive evaluation to elucidate symptoms that may reflect specific associated mechanisms amenable to targeted treatment [1, 2]; and (3) treatment modalities and management approaches not heretofore considered can be effective and can improve the quality of life for those suffering with pain. This chapter will provide a brief overview of the anatomy of pain that forms the basis for current practice.

Considerations of General Organization

The somatosensory system provides the means through which living organisms explore and monitor the body's external and internal environment in order to recognize changes that may be beneficial and embraced or detrimental to survival and avoided.

The peripheral elements of the nervous system are organized in a segmental fashion that is determined during the somatic stage of development when the embryo more closely resembles phylogenetically earlier stages of evolution (Figs. 4.1 and 4.2). Neural crest cells that are destined to become sensory neurons establish connections with local tissues of the developing somatic and lateral plate mesoderm and project centrally to connect with elements of the central nervous system close to the entry zone. At that stage of

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_4

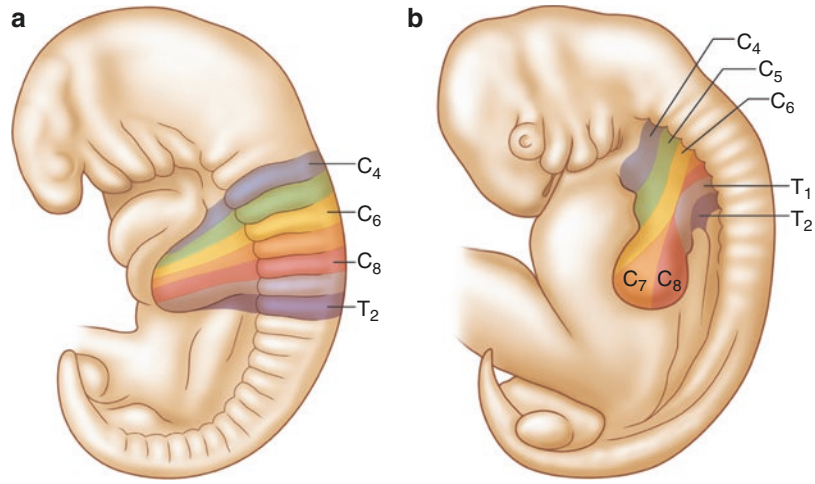


Fig. 4.1 Diagrams of the dermatomal pattern during early development of the pectoral limb bud at 5 weeks (**a**) and 6 weeks (**b**) of gestation

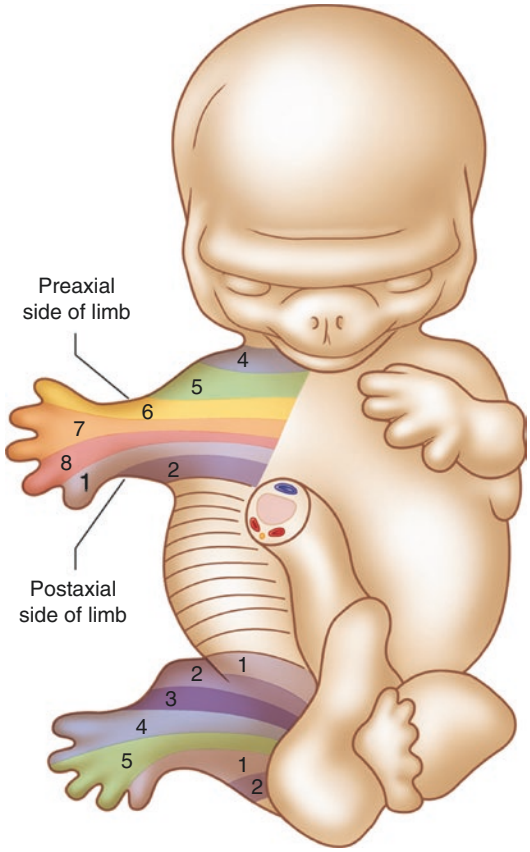


Fig. 4.2 Diagram of the ordered segmental distribution of peripheral afferents during an early stage of development

development, the pattern is clear. Sensory neurons from three levels are responsible for monitoring each region of the body to ensure redundancy of coverage and the integrity of the sensory monitoring system in the case of injury. Although the segmental relationship between the peripheral and central elements of the somatosensory nervous system remains and provides the basis for an ordered radicular or dermatomal pattern of innervation, the simple overlapping pattern is modified during later stages of development, resulting in a predictable increase in complexity of the basic dermatomal pattern (Fig. 4.3). The apparent change in distribution occurs during the process of differential growth and limb rotation through which the simple adult dermatomal pattern that is evident in the trunk is altered, leaving the inverted distribution of the segments of the trigeminal nerve in the head (Fig. 4.4), the autonomous regions of single root innervation in the limbs (Figs. 4.5 and 4.6), and the spiraling dermatomal pattern in the lower extremities (Fig. 4.6).

Axons that travel in close proximity to each other are packaged into nerve bundles that provide the conduits for neuronal traffic. Neurons innervating somatic derivatives of several dermatomal levels are packaged together and course through branches of spinal nerves that are distributed to the body wall and appendages (Figs. 4.7 and 4.8). The paths taken by neurons that innervate derivatives of the lateral plate mesoderm are less well defined in that they are variable and can course along blood vessels through elements of branches of the somatic nerves and through

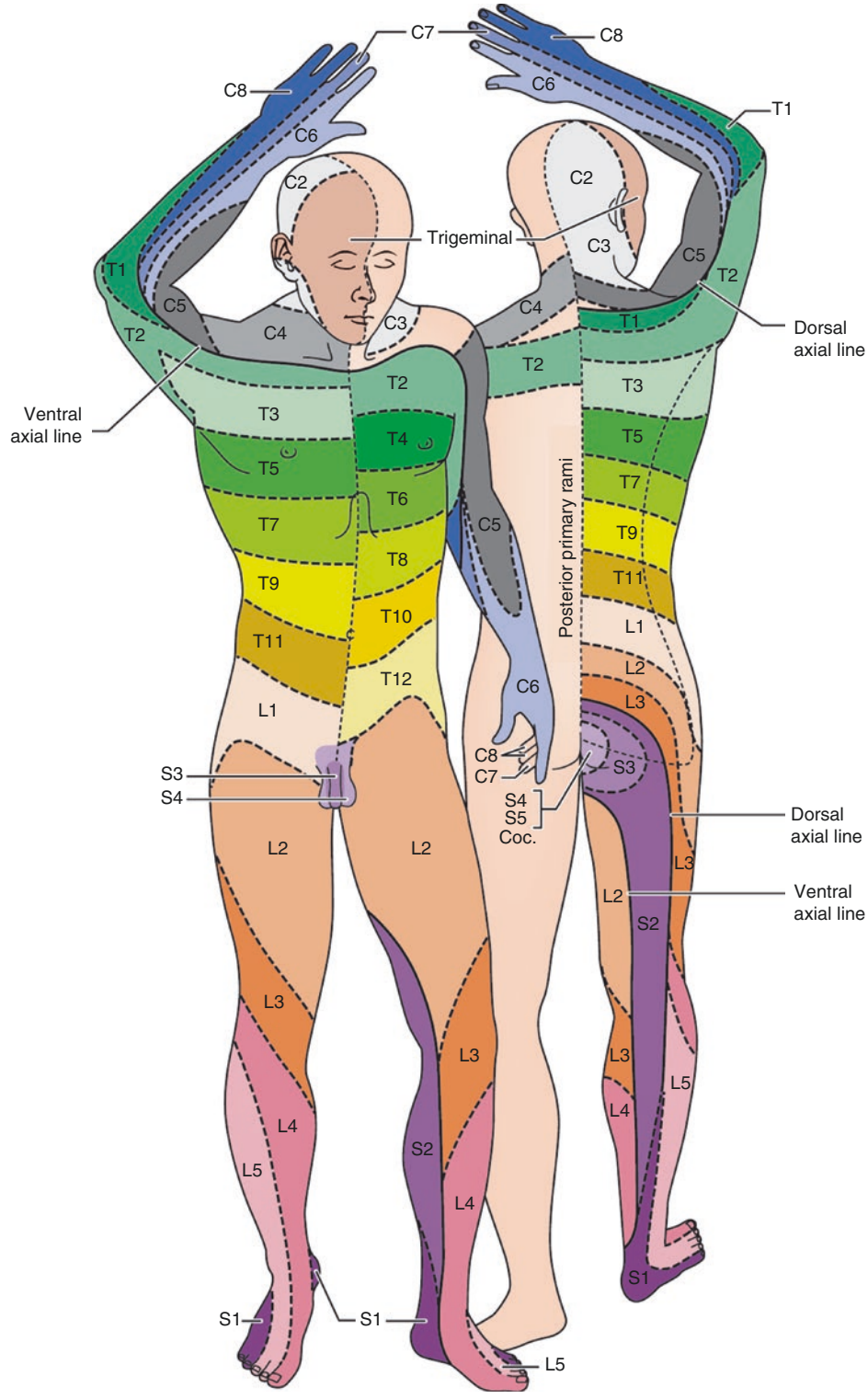


Fig. 4.3 Dermatomal distribution in the adult. Overlapping distribution of segments is indicated for the trunk (adapted from Lockhart RD, Hamilton GF, Fyfe FW. Anatomy of the Human Body. Philadelphia: J.B. Lippincott Company; 1972)

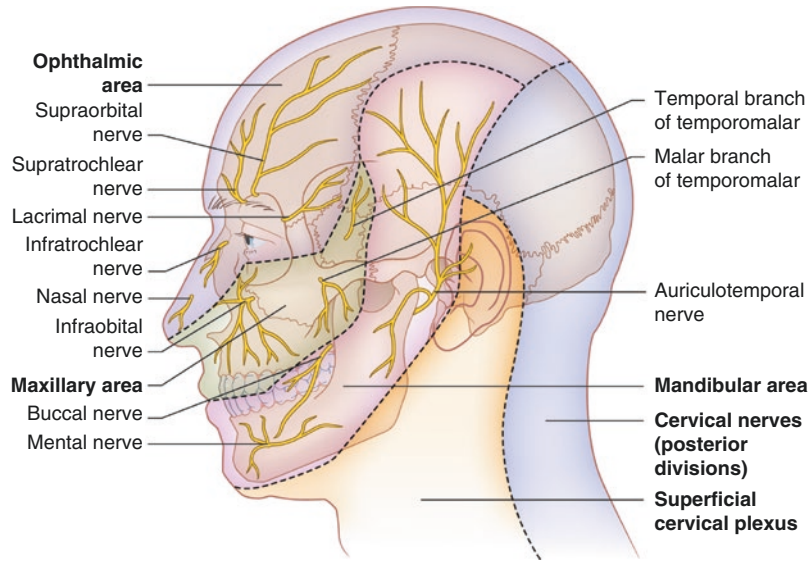


Fig. 4.4 Distribution of sensory nerves to the head (adapted from Gray's Anatomy, 1966)

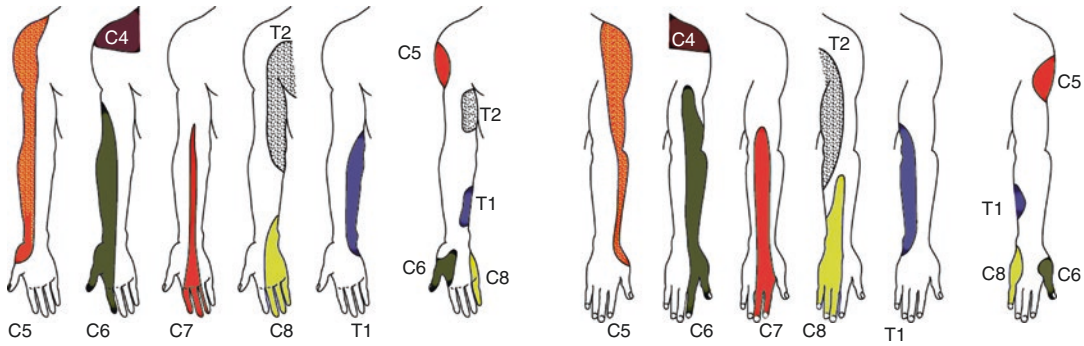


Fig. 4.5 Dermatomal distribution of the anterior (left) and posterior (right) upper extremity, showing areas supplied by only one segmental level (illustrations 6 and 12 from left) (adapted from Lockhart RD, Hamilton GF, Fyfe FW. Anatomy of the Human Body. Philadelphia: J.B. Lippincott Company; 1972)

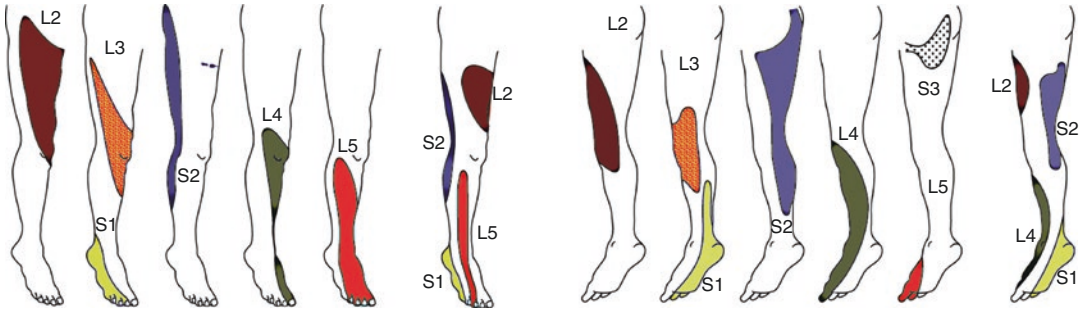


Fig. 4.6 Dermatomal distribution of the anterolateral (left) and posteromedial (right) lower extremity, showing areas supplied by only one segmental level (illustrations 6 and 12 from left) (adapted from Lockhart RD, Hamilton GF, Fyfe FW. Anatomy of the Human Body. Philadelphia: J.B. Lippincott Company; 1972)

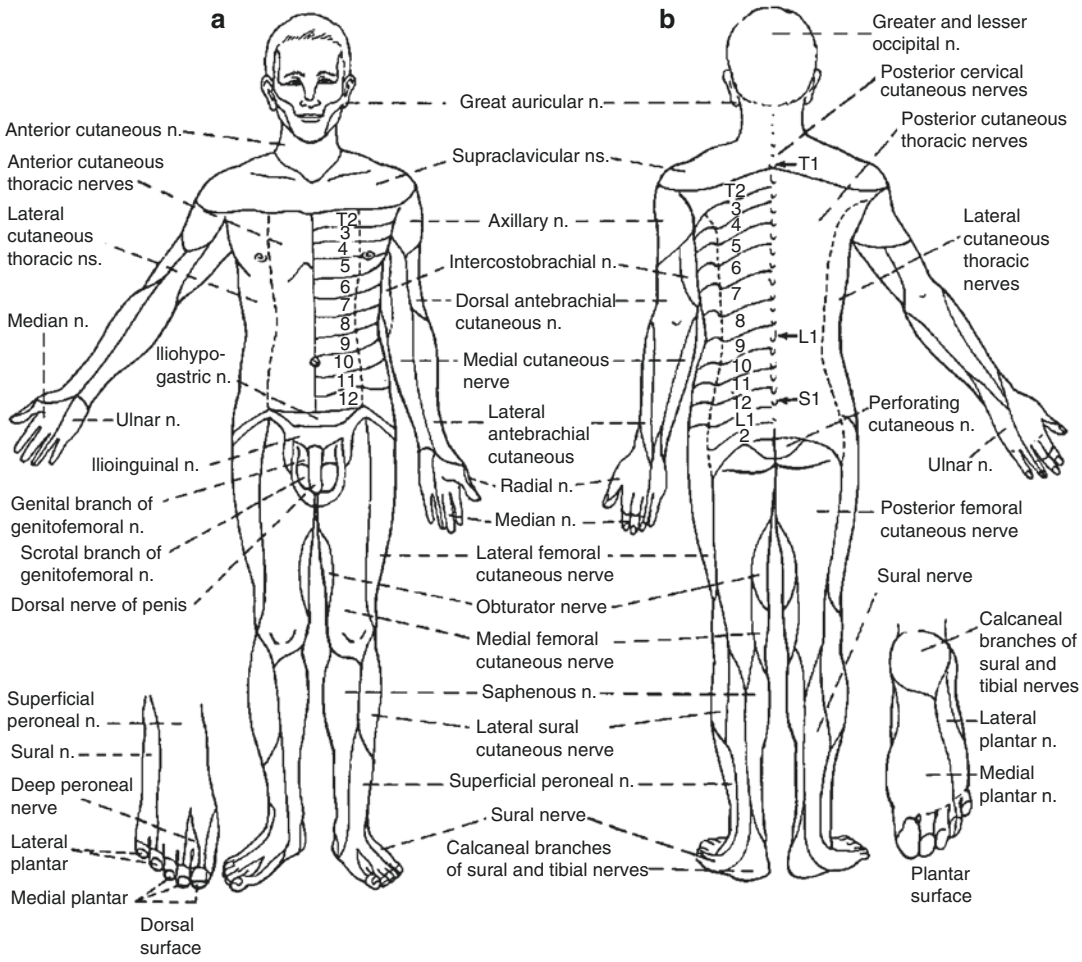


Fig. 4.7 The cutaneous fields of peripheral nerves (n.). (**a**) Anterior view. (**b**) Posterior view. In both figures, the numbers on the trunk refer to the intercostal nerves (modified from Haymaker W, Woodhall B. Peripheral nerve injuries: principles of diagnosis. Philadelphia: WB Saunders; 1945) (adapted from LeResche L, Bonica's Management of Pain, 3rd ed; 2001)

splanchnic components of the sympathetic nervous system (Fig. 4.9). These conduits ensure coverage of visceral tissues, smooth muscles, and glands located both in the body wall and in the core regions of the body. As long as the peripheral nerves are intact, damage to an individual nerve root will not result in complete loss of sensation in the area supplied by the damaged root. By contrast, damage to a peripheral nerve will result in a complete loss of sensation in the area served. An understanding of the differences between the patterns of dermatomal and peripheral nerve, thus, is important in assessing localization of site of injury and for determining the effect of diagnostic and therapeutic interventions.

Differential growth also results in an important disparity between boney vertebral levels, the location of the dorsal root ganglia, the location of the caudal end of the spinal cord, and the dorsal root entry zone of the spinal cord observed at different stages of development and in the adult. Figure 4.10 depicts the changes in the relative relationship between neural and boney elements from early stages in development (30, 67, and 111 mm) to shortly after birth (221 mm). In the

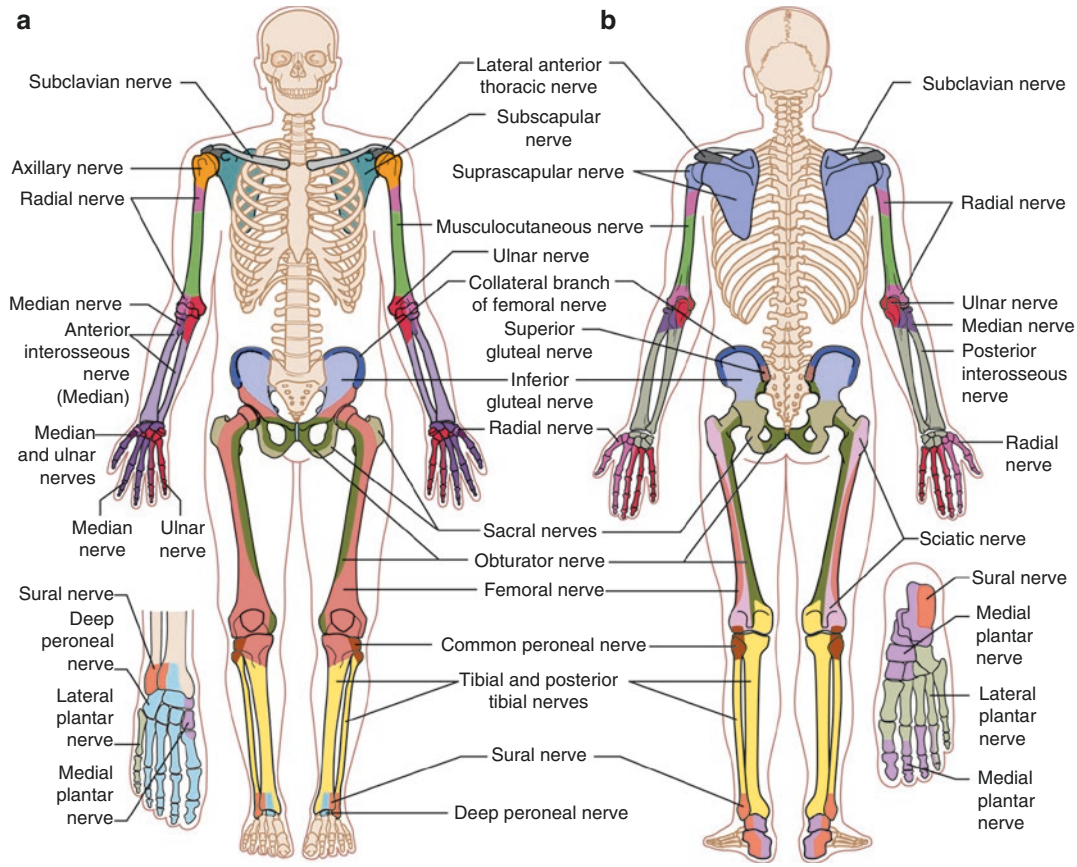


Fig. 4.8 The peripheral nerve supply of the skeleton. (**a**) Anterior view. (**b**) Posterior view. The various peripheral nerve fields are indicated by different patterns (modified from Dejérine J. Sémiologie du système nerveux. Paris: Masson; 1914) (adapted from LeResche L, Bonica's Management of Pain, 3rd ed; 2001)

adult, the relative disparity between level of the spinal nerve and its respective entry into the spinal cord generally follows the following formula: vertebral level (vertebral spinous process) + n = spinal cord level, where $n = 0$ for the upper cervical region, 1 between the lower cervical and upper thoracic region (vertebral prominence), 2 between T3 and T9, and 3 between T9 and T11 (Fig. 4.11). The conus medullaris is located between the spinous processes of the T12–L2 vertebrae. Figures 4.12, 4.13, and 4.14 depict boney landmarks and lines of reference to aid in identifying vertebral levels. An understanding of the disparity and knowledge of superficial landmarks is important for guiding and determining the best approaches for performing interventions on individual nerve roots and spinal cord levels. For example, the knowledge that the adult spinal cord extends inferiorly only to the L2–L3 vertebrae offers a degree of safety when inserting needles for obtaining spinal fluid from the lumbar cistern when the approach is made below the L3 vertebral level.

Stimulus Transduction and Transmission

Two fiber systems are responsible for the transmission of nociceptive signals from the body wall and viscera to the central nervous system, the Aδ and the C fiber systems (Fig. 4.15). A third system, the Aβ system, is primarily responsible for processing non-noxious mechanical

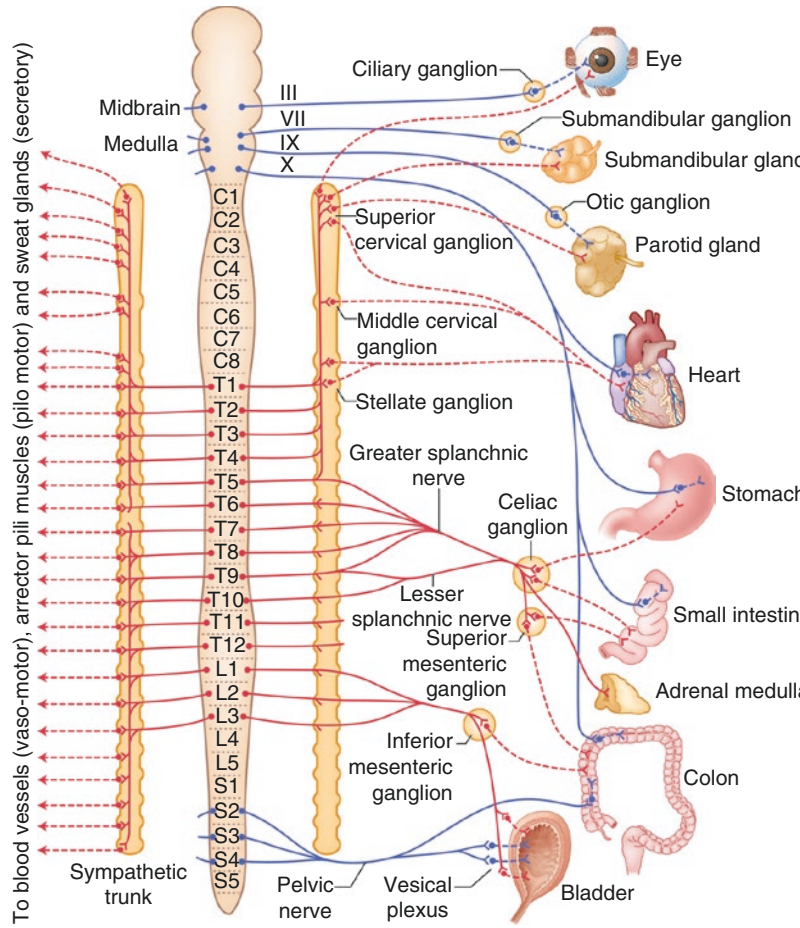


Fig. 4.9 Schematic diagram showing general arrangement of the autonomic system. The sympathetic components are shown in red, while the parasympathetic components are in blue. Solid lines represent preganglionic fibers; broken lines indicate postganglionic fibers. The sympathetic fibers to the blood vessels, hair, and sweat glands are not shown (adapted from Carpenter MB, Sutin J. Human Neuroanatomy. 8th ed. Baltimore: Williams & Wilkins; 1983)

stimuli and serves as a tactile discriminator, but it also plays a role in modulating nociceptive signals that enter the dorsal horn of the spinal cord (Fig. 4.15). These fiber systems are supported by pseudounipolar cell bodies that, along with supportive satellite cells, are located in the spinal dorsal root ganglia (DRG) and cranial nerves V, VII, IX, and X. The ganglia are located in or adjacent to intervertebral foramina of the spinal column or in or near boney canals and foramina of the skull, respectively. The intervertebral foramina and boney canals allow passage of elements of the peripheral nervous system into and out of the spinal cord and brain stem. The conducting elements of these fiber systems are composed of peripheral axons with free nerve endings or specialized receptor organs that are distributed in peripheral tissues and are contiguous with central elements that terminate either in the dorsal horn of the spinal cord or in nuclei of the brain stem. They are connected to their respective pseudounipolar perikarya by a T-segment of axonal membrane (Fig. 4.16). No synapses occur between primary afferents in the peripheral ganglia, but the proximity of the neuronal perikarya affords the possibility for electrochemical cross excitation between neurons to occur.

Free nerve endings comprise the distal terminals of nociceptive neurons. They are distributed within the epidermis of the skin, deep tissues, elements of the musculoskeletal system, and internal visceral organs. The nociceptors are optimally positioned to monitor changes in the thermal, mechanical, and chemical environment of every region of the body. Potentially injurious stimuli, when present in the peripheral tissues, trigger the release of a myriad of chemical mediators that set into motion a constellation of events

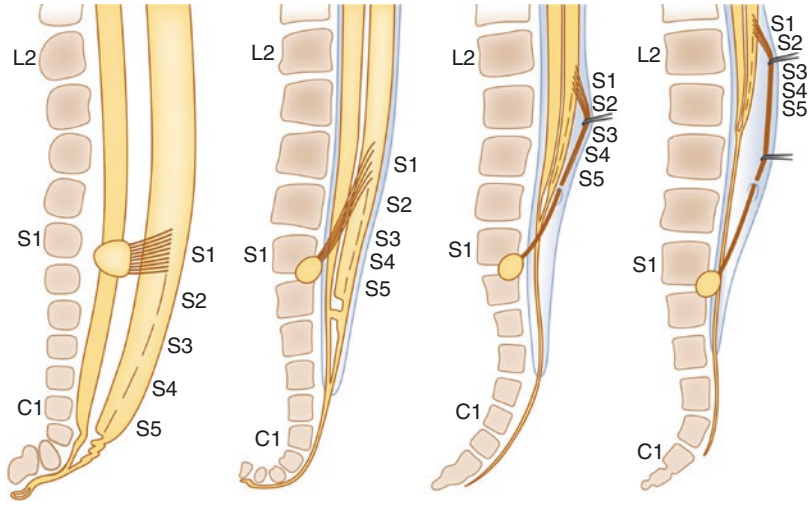


Fig. 4.10 Four successive stages in development of the caudal end of the human spinal cord (after Streeter). They show the formation of the filum terminale and the progressive obliquity of the first sacral nerve which is caused by the differential growth of the spinal cord and vertebral column. From left to right in the figure, the sizes of the embryos from which the reconstructions were made are as follows: 30, 67, and 221 mm (adapted from Hamilton WJ, Boyd JD, Mossman HW. Human embryology: Prenatal Development of Form and Function. 4th ed. London: Williams & Wilkins Company:Macmillian Press Ltd; 1978)

that alters the membrane permeability of afferent nerve terminals to charged ions. Among the inciting nociceptive events are the release of potassium ions, protons, and bradykinin and the initiation of the arachidonic acid cascade which leads to the production of prostaglandins and leukotrienes. Bradykinin, through activation of phospholipase C, stimulates the production of inositol 1, 4, 5-trisphosphate (IP3) and diacylglycerol (DAG) from membrane phospholipids. IP3 stimulates the release of calcium ions, while DAG, through protein kinase C (PKC)-mediated pathways, enhances the release of sodium ions and the production of arachidonic acid. The phospholipase A2-mediated metabolism of arachidonic acid increases tissue levels of adenylyl cyclase, cyclic AMP, and prostaglandins PGE_2 and PGI_2 [3–6]. These events, coupled with complimentary increases in the levels of mediators such as histamine, serotonin, adenosine, tumor necrosis factor-α (TNF-α), nerve growth factor (NGF), substance P (sP), glutamate, norepinephrine (NE), and cytokines (IL-1, IL-6), lead to a shift in the electrochemical gradient, the development of a generator current, the depolarization of the membrane, and the initiation of an action potential that is transmitted through the system of peripheral nerves to the central nervous system [5, 7] (Figs. 4.17 and 4.18).

Axons of the Aδ system range in diameter from 1 to 6 μm and are ensheathed by a thin layer of myelin [4]. The myelin provides a supportive and trophic effect for axons, and in addition to insulating axons within a nerve bundle from each other for the maintenance of temporal and spatial integrity of the signal, it serves to enhance conduction velocity. The Aδ axons are supported by cell bodies that measure 25–30 μm in diameter and serve small receptive fields. They respond to relatively low levels of noxious stimulation and conduct impulses at velocities between 5 and 30 m/s. Although they respond preferentially to mechanical stimulation, they also respond to noxious heat. As the axons approach the spinal cord, they diverge from the main nerve trunk and enter the dorsal root where they course by their cell bodies in the DRG and enter the spinal cord to terminate on neurons in Rexed laminae I, II, III, V, and X [8] (Fig. 4.19). The axons of the C fiber system are unmyelinated [5]. They are supported by DRG neurons measuring 10–15 μm in diameter, serve larger receptive fields than those served by Aδ

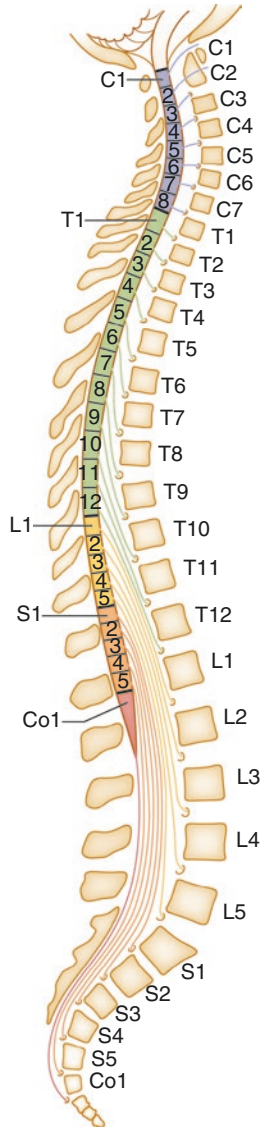


Fig. 4.11 Diagram of the position of the spinal cord levels in relation to the vertebral bodies and spinous processes of the vertebral column

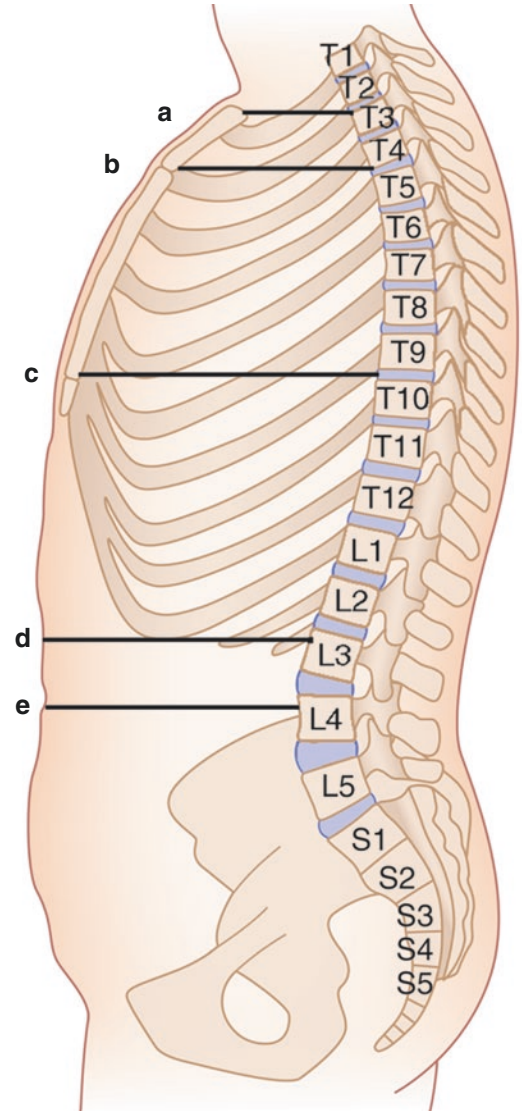


Fig. 4.12 A lateral view of the body, showing the vertebral levels of certain landmarks on the anterior thoracic and abdominal walls: (**a**) suprasternal notch, (**b**) sternal angle, (**c**) xiphisternal joint, (**d**) subcostal line, and (**e**) umbilicus (adapted from Crafts RC. A Textbook of Human Anatomy. 2nd ed. New York: Wiley Medical Publication; 1979)

fibers, require a higher stimulus intensity to initiate an action potential, and convey information at velocities between 0.5 and 2 m/s. C fibers respond to polymodal stimuli and can be classified into distinct populations [9]. Peptidergic C fibers respond to heat, but not to mechanical or cold stimuli. They also respond to sP, calcitonin gene-related peptide (CGRP), and capsaicin and express transient receptor potential cation channel subfamily V member 1 (TRP V1), tropomyosin receptor kinase (Trk) A, and μ-opioid receptors. Their activity is modulated by nerve growth factor (NGF). Peptidergic C fibers course medially in the dorsal root and terminate preferentially in Rexed lamina I, the outer portion of lamina II, and lamina V [8] (Fig. 4.20). A second population of C fibers

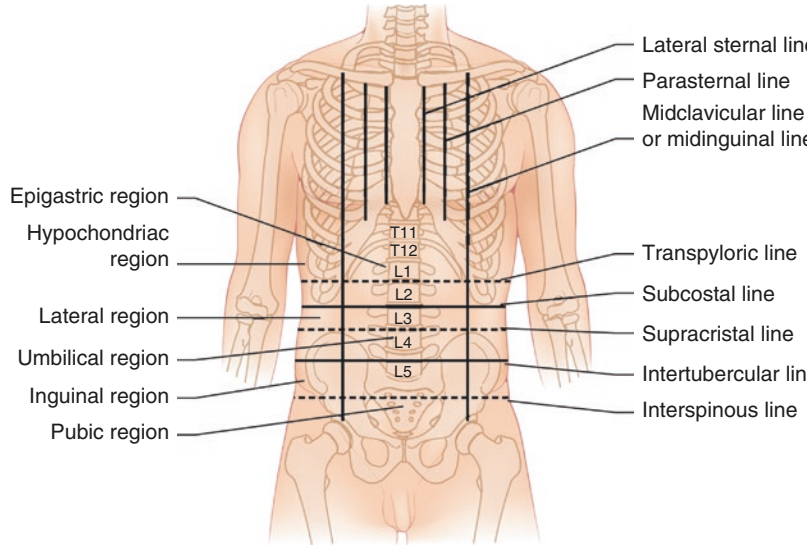


Fig. 4.13 Lines of reference on anterior thoracic and abdominal walls (adapted from Crafts RC. A Textbook of Human Anatomy. 2nd ed. New York: Wiley; 1979)

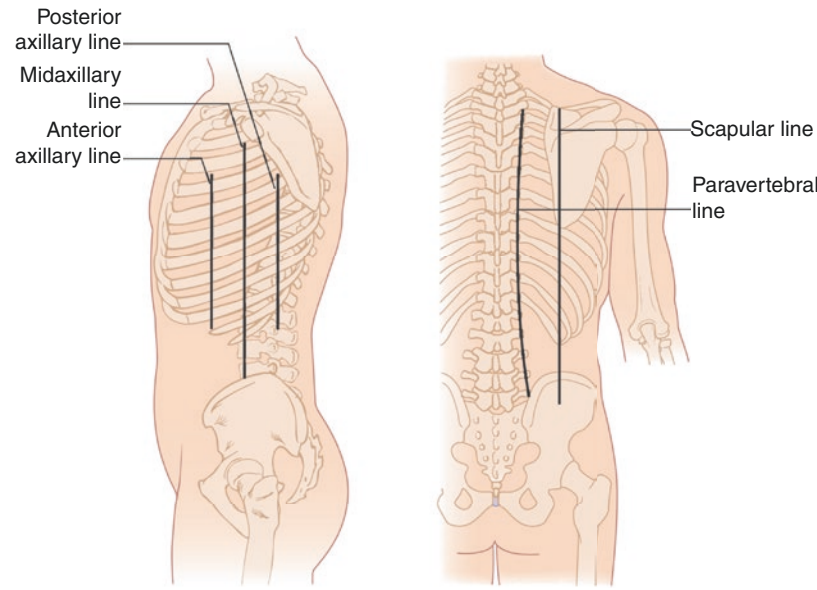


Fig. 4.14 Lines of reference on lateral and posterior chest wall (adapted from Crafts RC. A Textbook of Human Anatomy. 2nd ed. New York: Wiley; 1979)

responds to cold rather than to heat or mechanical stimulation and is not peptidergic. The cells respond to ATP and express P2X purinoceptor-3-isolectin B4, c-Ret neurotropin, and δ-opioid receptors. Unlike the peptidergic afferents, these cells terminate in the inner portion of lamina II and are modulated by glial-derived neurotrophic factor (GDNF). Some C fibers, "silent nociceptors," are typically unresponsive to normal noxious stimulation but become active during periods of inflammation or tissue injury, and others respond to peripherally released pruritogens. These specialized neurons release B-type natriuretic peptide (BNP) that activates natriuretic peptide receptor-A (Npra)-expressing neurons that subsequently release gastrin-releasing peptide (GRP) onto relay neurons in Rexed lamina I and II and are responsible for itch [10]. Upon entering the spinal cord, the axons of the primary nociceptors ascend and descend in the zone of Lissauer. The majority of these fibers ascend approximately two spinal levels before terminating in the dorsal horn.

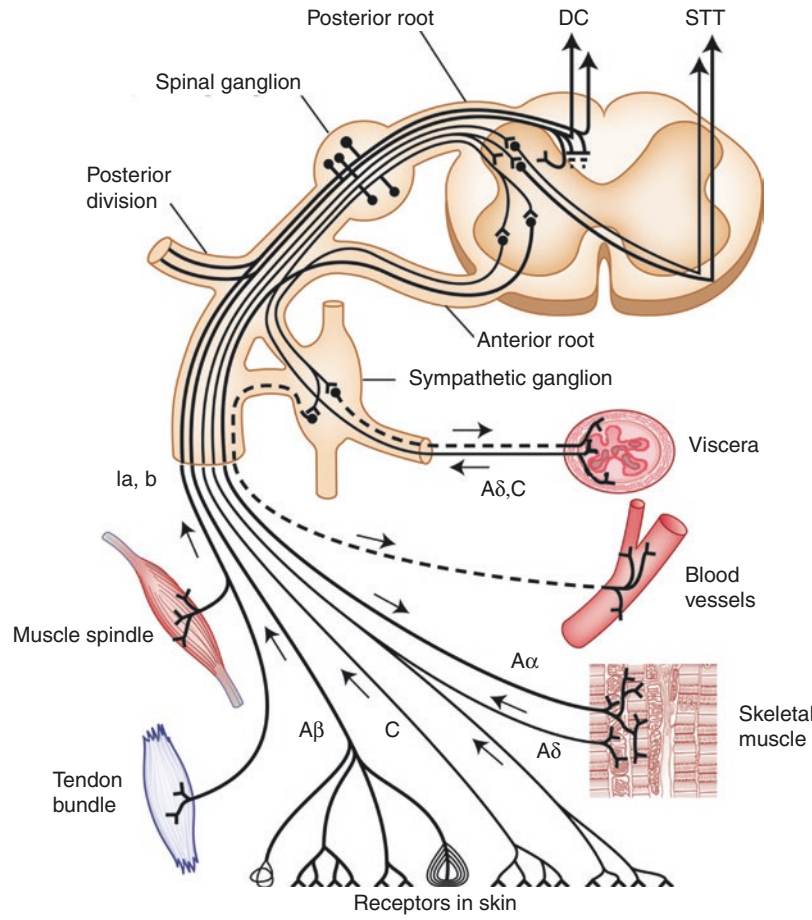


Fig. 4.15 A simplified schema of a spinal nerve and the different types of fibers contained therein (*DC* dorsal columns; *STT* spinothalamic tract) (adapted from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 26–72)

In the resting state, the free nerve endings of nociceptive afferents maintain a polarized membrane with a higher concentration of sodium ions outside the cell. Noxious heat (>45–55 °C), cold (8–25 °C), mechanical (pressure or distention; 60 g/mm^2), or chemically mediated stimuli increase the permeability of the membrane to charged ions, thereby setting up a generator current (Fig. 4.18) that leads to a subsequent shift in the electrochemical gradient and voltage across the membrane [7, 11]. The change in voltage alters the configuration of voltage-gated channels, allowing entry of predominantly sodium ions into the cell in exchange for potassium ions, and the initiation of an action potential, which is propagated along the axon to the central nervous system. In myelinated axons like those of the Aβ and Aδ system, the excitable membrane that supports the propagation of action potentials is found only in the intervals between adjacent segments of myelin, called nodes of Ranvier, where there is a high density of sodium channels. Since membrane depolarization occurs at the nodes of Ranvier, impulses "jump" from one node to the next in a saltatory fashion, resulting in rapid conduction of the action potential (Fig. 4.21). The fiber diameter and the internodal distance are primary determinants of the conduction velocity of the axon. In unmyelinated axons like those of the C fiber system, sodium channels are distributed along the entire length of the axon (Fig. 4.21). Depolarization is propagated contiguously between adjacent membrane segments, resulting in the slowest impulse conduction of any system. After the passage of the action potential, the electrochemical gradient is reestablished through energy-dependent sodium/potassium pumps (Na^+/K^+, ATPase) that transport sodium ions out of the cell in exchange for potassium ions.

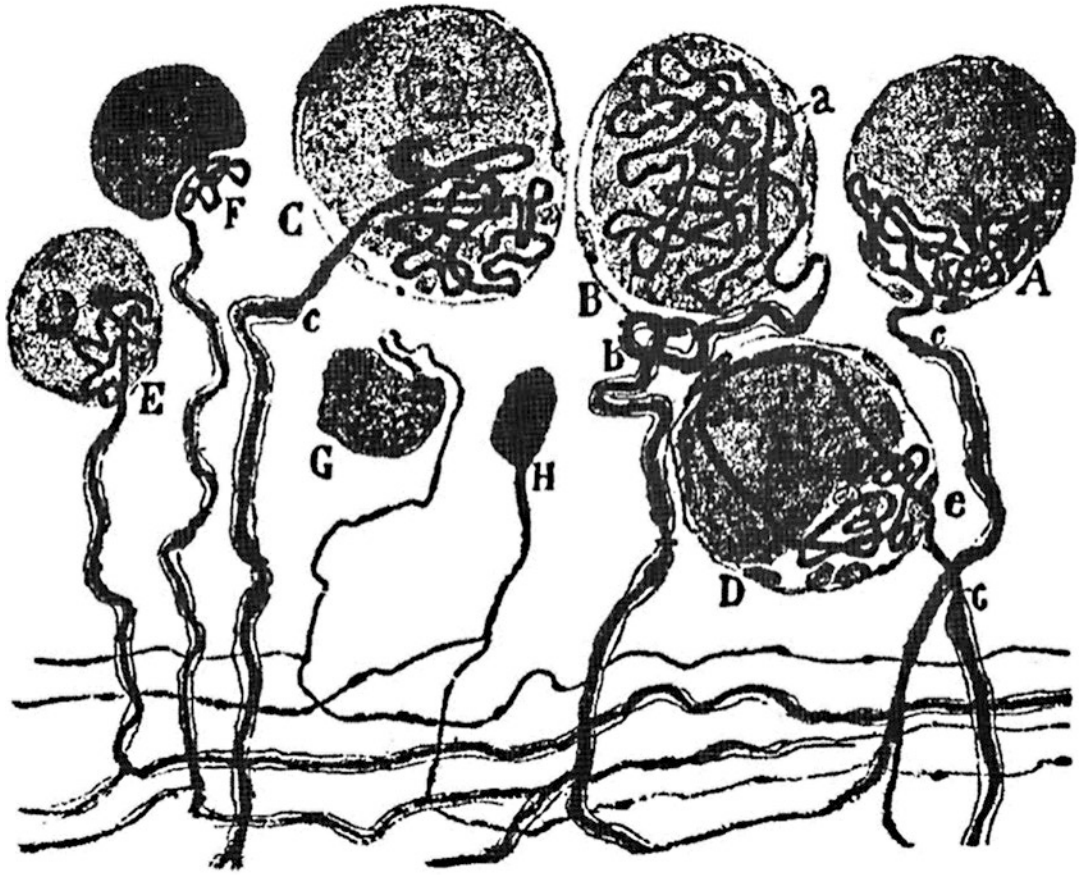


Fig. 4.16 Large and small DRG cell somata, the T-stem axon with its glomerulus, and the T-junction (adapted from Cajal, 1911, p. 428)

The differences in the receptive field sizes, the conduction velocity, and the thresholds for initiating action potentials between the Aδ and C fiber systems form the basis for the first and second pain responses. The first response occurs immediately upon stimulation, is often sharp in character, and is precisely localized. It results in a rapid, aversive withdrawal from the offending stimulus and a complimentary, supportive crossed extensor response. This basic mechanism is essential for survival and reduces the amount of tissue injury. Shortly after stimulation, a less well-localized feeling of discomfort is perceived, that is, often aching or throbbing in quality, and persists well after the stimulus has been removed. This second pain response raises the level of awareness of the injured body part during the healing process. The lowered threshold to activation of a nociceptive signal reduces the likelihood of additional injury due to subsequent activity and enhances vigilance until sufficient healing has occurred.

By contrast, when non-noxious mechanical stimuli are presented to specialized afferent end organs, e.g., Pacinian and Meissner corpuscles, Ruffini endings, and Merkel cells (Fig. 4.22), the membrane permeability of large (>25 μm in diameter), low-threshold neurons of the Aβ system is similarly altered, thus initiating action potentials conveying information of a non-noxious tactile nature [5]. These action potentials are conducted along large, 6–12-μm diameter myelinated axons at velocities between 30 and 70 m/s. Upon arriving at the spinal cord, the axons enter the cuneate

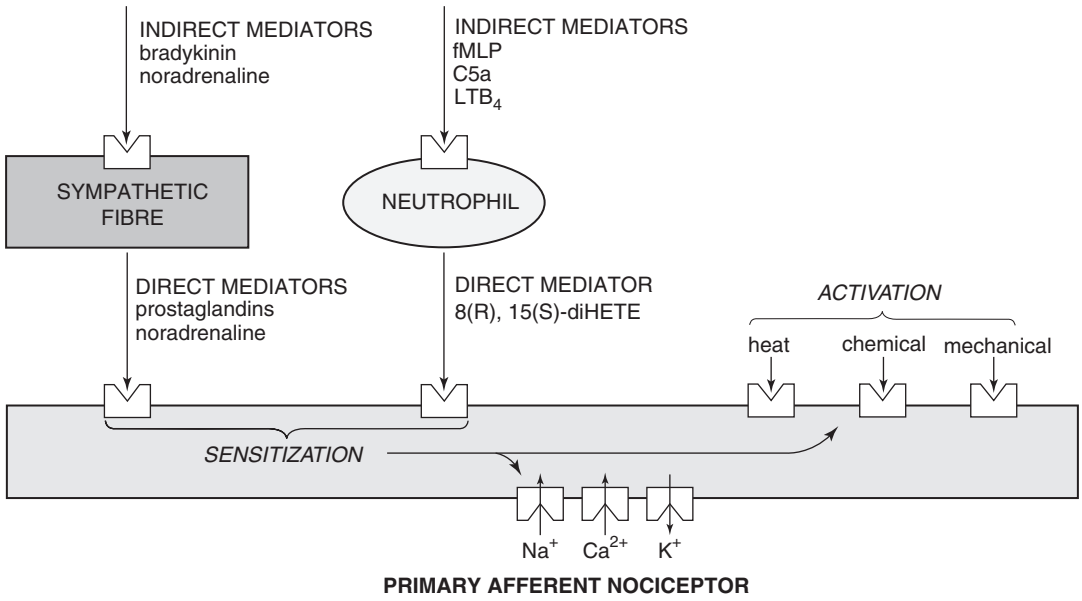


Fig. 4.17 Direct and indirect mechanisms by which inflammatory mediators sensitize primary afferent nociceptors (adapted from Levine JD, Reichling DB. Peripheral mechanisms of inflammatory pain. In: Wall PD, Melzack R, editors. Textbook of Pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 59–84)

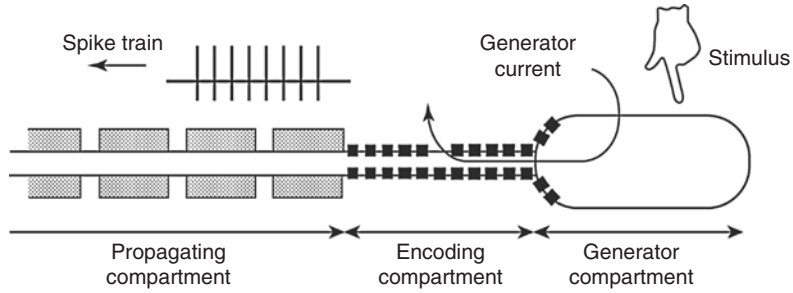


Fig. 4.18 Sketch of sensory ending showing generator, encoding, and propagating compartments (adapted from Devor M, Seltzer Z. The pathophysiology of damaged peripheral nerves. In: Wall PD, Melzack R, editors. Textbook of Pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 129–64)

and gracile fasciculi and ascend ipsilaterally in the spinal cord to terminate in the cuneate and gracile nuclei of the caudal medulla. Axons arising from neurons located in the cuneate and gracile nuclei then cross the midline of the neuraxis and ascend in the medial lemniscus to terminate in the lateral portion of the ventral posterior nucleus (VPN) of the thalamus. Collaterals from the Aβ afferents also project into the dorsal horn where they terminate in Rexed laminae III, IV, and V and, through stimulation of inhibitory interneurons, can reduce the intensity of nociceptive signals allowed through the dorsal horn (Fig. 4.23). Similar low-threshold tactile afferents arise from the head course in branches of the trigeminal nerve and enter the central nervous system at the level of the pons. These afferents terminate in the principal sensory nucleus. Axons arising from the principal sensory nucleus cross the midline, join the medial lemniscus, and ascend through the rostral brain stem to terminate in the medial portion of the VPN.

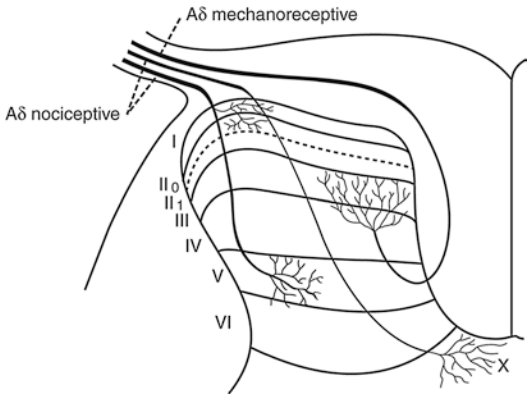


Fig. 4.19 Schematic diagrams of the course and termination of collaterals of the Aδ cutaneous fibers in the dorsal horn of the spinal cord (adapted from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 26–72)

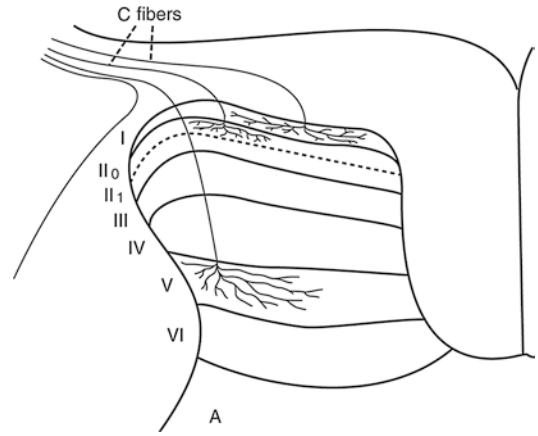


Fig. 4.20 Schematic diagrams of the course and termination of collaterals of unmyelinated C fibers in the dorsal horn of the spinal cord (adapted from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 26–72)

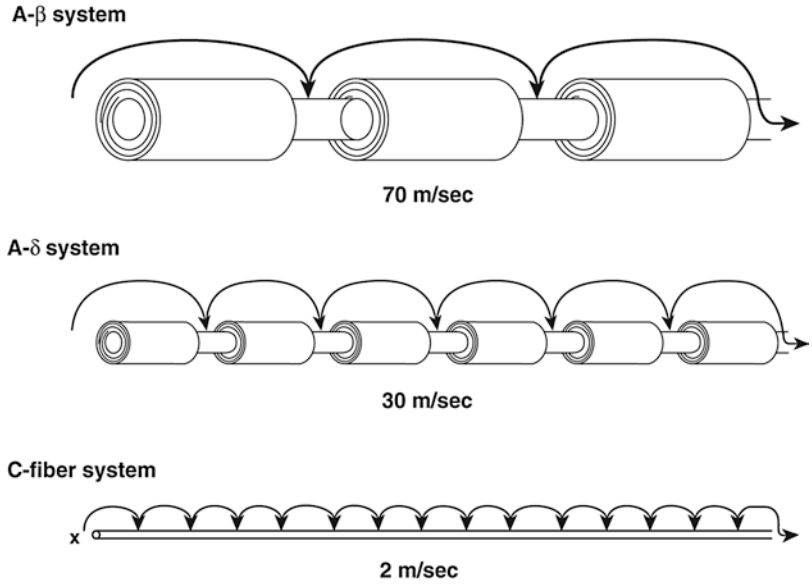


Fig. 4.21 Diagram depicts the relationship between fiber diameter, myelination, and conduction velocity in the peripheral nervous system (adapted from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

Stimulus Modulation

Upon entry of the central gray matter, primary afferents release stored excitatory neurotransmitters, thereby relaying the initial nociceptive signal to either wide dynamic range or nociceptive-specific neurons of the dorsal horn (Fig. 4.24). Through this connection, the modality and the temporal and spatial aspects of the nociceptive signal are integrated. The sum of that integration is then transmitted to higher levels of the nervous system for further processing. The wide dynamic range neurons are found primarily in lamina V and are responsible for much of the information that is transmitted to the brain stem and thalamus. These neurons receive not only

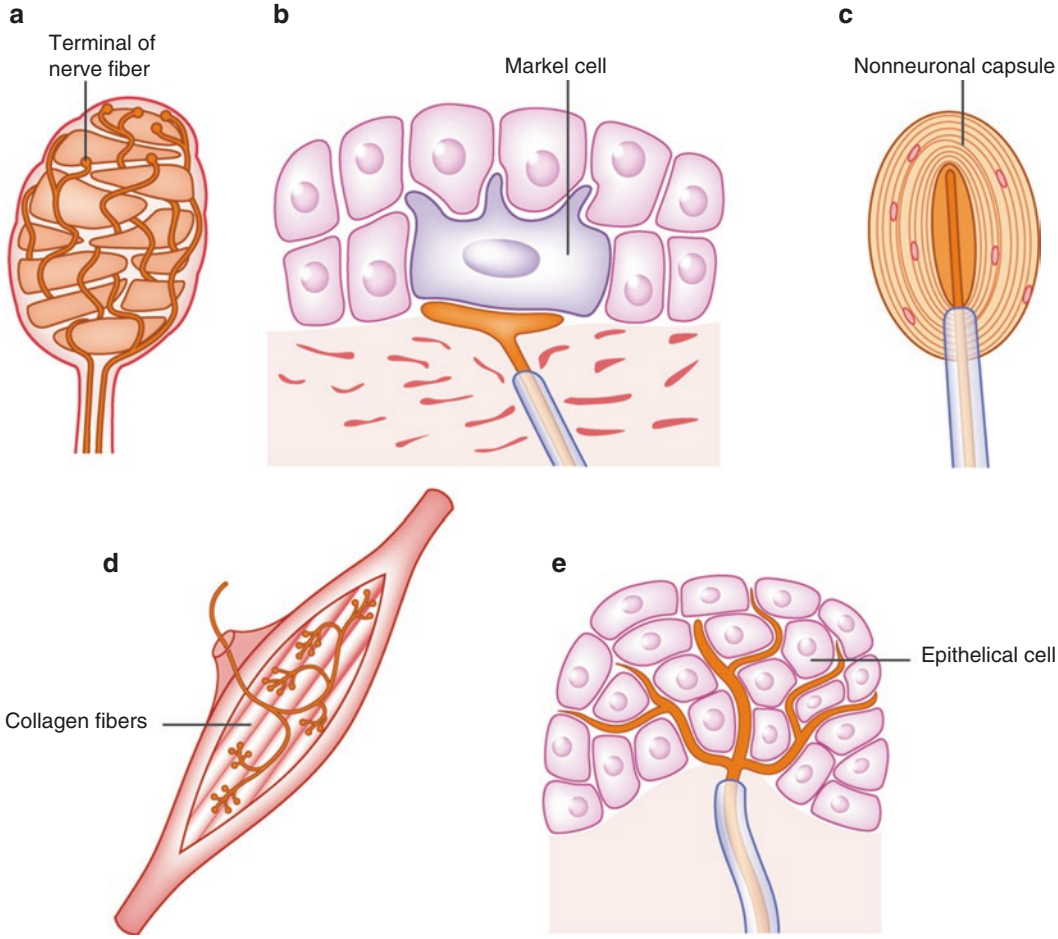


Fig. 4.22 Morphological features of somatosensory receptors, including the variation in non-neural components. (**a**) Meissner corpuscles are composed of axonal loops, separated by non-neuronal, supporting cells; (**b**) Merkel disks are characterized by the close association between afferent axons and Merkel cells; (**c**) Pacinian corpuscles include a central sensory axon, surrounded by a fluid-filled capsule that filters out all sustained stimuli; (**d**) Ruffini endings are driven by skin stretch because of the termination of primary afferents among collagen fibrils of the skin; and (**e**) free nerve endings, characteristic of nociceptors, are left unprotected from chemicals that are secreted or applied to the skin (adapted from Hendry SHC, Hsiao SS, Bushnell MC. Somatic sensation. In: Zigmond MJ, Bloom FE, Landis SC, Roberts JL, Squire LR, editors. Fundamental Neuroscience. San Diego: Academic Press; 1999)

polymodal inputs from high-threshold mechanical and heat-sensitive Aδ and C fiber nociceptors but also inputs from collaterals of non-nociceptive, low-threshold mechanical Aβ afferents and local internuncial neurons of the dorsal horn. They have a moderate threshold for initiating an impulse and are responsible for signals related to itch and flutter. Inputs to the wide dynamic range neurons provide the essential segmental framework for the "gate control theory" proposed by Melzack and Wall [12] whereby impulses transmitted by low-threshold mechanoreceptors can reduce the nociceptive signal that is relayed to higher integrative levels for conscious perception (Fig. 4.25). By comparison, nociceptive-specific neurons are located in laminae I and V and receive inputs only from high-threshold mechanical and heat-sensitive Aδ and C fiber nociceptors. Nociceptive-specific neurons receive inputs that may be either polymodal or

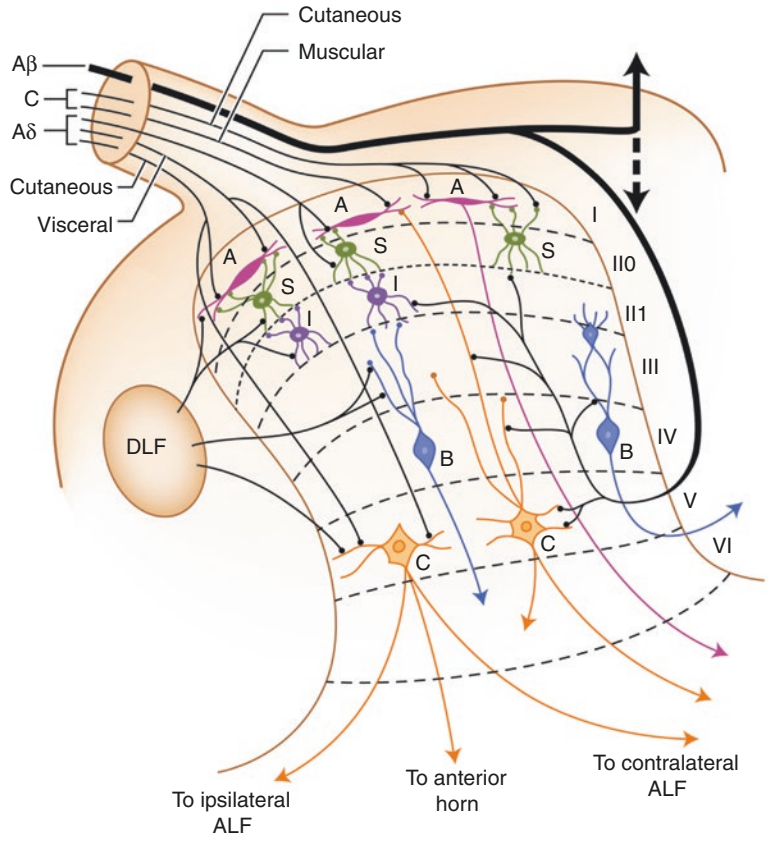


Fig. 4.23 Simplified schematic cross-sectional diagram of input and output of the dorsal horn of the spinal cord as well as interneurons and axonal terminals of descending control systems (adapted from Terman GW, Bonica JJ. Spinal mechanisms and their modulation. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 73–152)

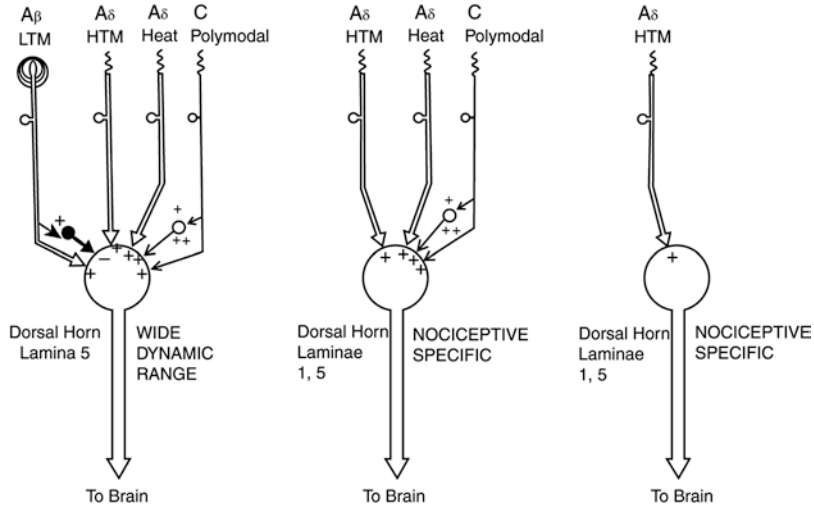


Fig. 4.24 Three types of nociceptive cells in the dorsal horn, their inputs from primary afferents, their location in the spinal cord, and their output to ascending systems. Wide dynamic range neurons receive inputs from low-threshold mechanoreceptive (LTM) primary afferents, high-threshold mechanoreceptive (HTM) primary afferents, and C-polymodal afferents. Nociceptive-specific neurons receive inputs exclusively from nociceptive afferents (adapted from Terman GW, Bonica JJ. Spinal mechanisms and their modulation. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001; p. 73–152)

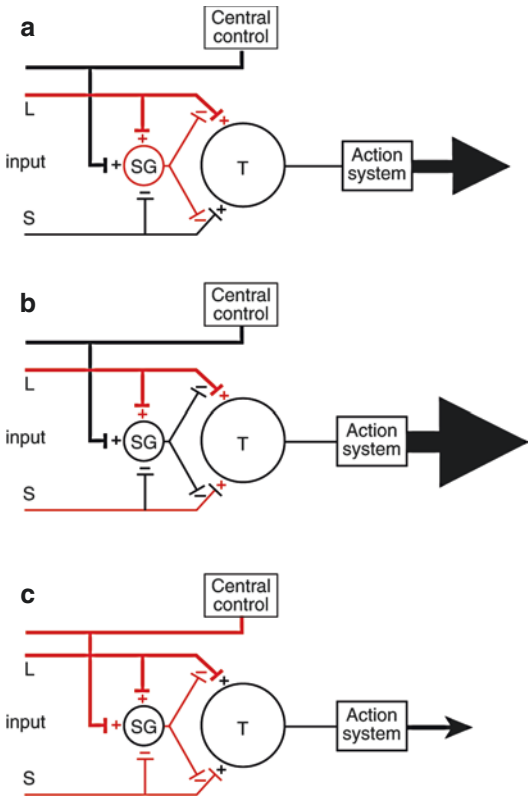


Fig. 4.25 Modified schematic diagram of the "gate control theory" of Melzack and Wall, 1965. SG represents an interneuron in the substantia gelatinosa of the dorsal horn. T represents a cell that transmits the nociceptive signal for higher central processing

modality specific and possess the capability of supporting after discharges, whereas the silent nociceptors are a special group of nociceptive-specific neurons that become active only during periods of inflammation and tissue injury and provide a means for amplifying the nociceptive signal.

When an action potential that is initiated by a nociceptive stimulus reaches the central afferent terminal, calcium enters the synaptic bouton through voltage-gated calcium channels (Fig. 4.26). In the presence of calcium, vesicles containing excitatory neurotransmitters, such as glutamate, aspartate, CGRP, sP, neurokinin, vasoactive intestinal peptide (VIP), neuropeptide Y (NP-Y), galanin, or somatostatin, fuse with the terminal cell membrane and release their contents into the synaptic cleft [4, 5]. The neurotransmitters cross the synaptic cleft, recognize receptors on the postsynaptic relay cell, and, through specific stoichiometric interaction, alter the membrane properties of ligand-gated receptors on the receiving neuron. The ligand-receptor interaction initiates a cascade of intracellular events that enables the triggering of the next impulse in the chain. The stability of the synapse is reestablished either by removal of the neurotransmitter from the synaptic cleft through

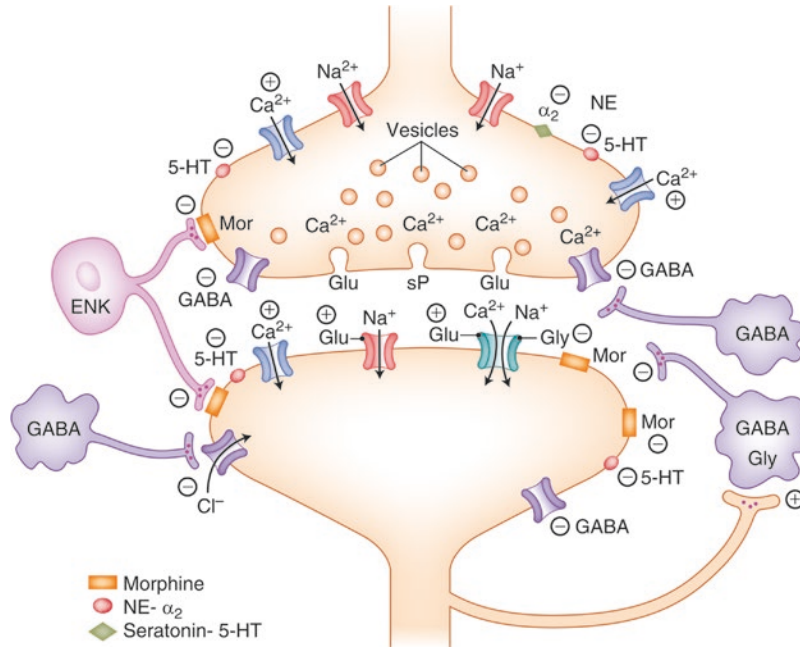


Fig. 4.26 Schematic depiction of a primary afferent synapse on a relay neuron in the dorsal horn of the spinal cord. *ENK* enkephalinergic neuron, *NE* norepinephrine, *5-HT* serotonin, *Glu* glutamate, *Gly* glycine, *MOR* μ-opioid receptor, and *GABA* gamma (γ)-aminobutyric acid (adapted from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. American New York: Academy of Neurology Press, Demos; 2007)

enzymatic degradation; through reuptake into the presynaptic terminal or transport via glutamate transporter 1 (GLT1) and glutamate-aspartate transporter (GLAST), into astrocytes that support the synapse [13]; or through the activation of processes that inhibit synaptic transmission. One such process is the collateral activation of inhibitory interneurons within the dorsal horn that release inhibitory neurotransmitters such as glycine and gamma (γ)-aminobutyric acid (GABA). These transmitters inhibit further release of excitatory neurotransmitters from the presynaptic terminal and stabilize the postsynaptic cell. It is the critical balance between the excitatory components of the afferent pathway whose role is to ensure transmission of the signal warning of impending injury. It is the inhibitory components that through amplification can reduce or through suppression can enhance the amount of nociceptive signal that is allowed to pass to higher levels of the nervous system and be perceived at any given time (Fig. 4.27).

Stimulus Perception and Interpretation

Axons en route to the thalamus from the spinal cord course through the ventral white commissure of the spinal cord, cross the midline, and enter the contralateral lateral spinothalamic tract where they project rostrally through the central nervous system to terminate in the VPN. Similar projections that subserve the territory of the trigeminal nerve receive inputs from axons that, upon entering the pons, descend in the spinal trigeminal tract and terminate on neurons in the spinal trigeminal nucleus. The projections that arise from the relay neurons in the spinal trigeminal nucleus cross the midline and join the spinothalamic tract en route to the VPN (Fig. 4.28). There are two components of the lateral spinothalamic pathway. The first component, the neospinothalamic tract, provides for discriminative functions and is related to the Aδ system. It projects directly to the VPN and is rapidly conducting, precisely

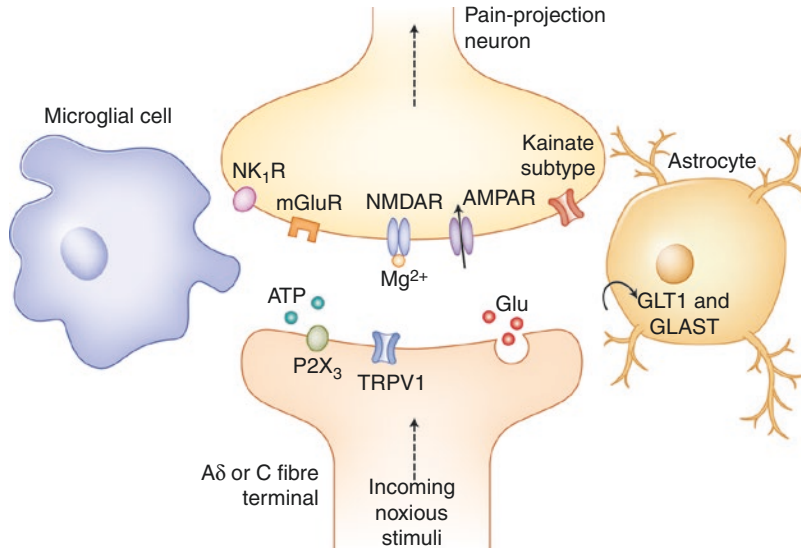


Fig. 4.27 Schematic depiction of the glial contribution to the processing of the primary afferent signal. Under healthy circumstances, low-frequency activation of Aδ and C fiber nociceptors by mild noxious stimuli leads to glutamate (Glu) release from the central presynaptic afferent nerve terminals in the spinal cord dorsal horn. Short-term activation of AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid) and kainite subtypes of ionotropic glutamate receptors ensues. Although also present, the NMDA (*N*-methyl-D-aspartate) ionotropic glutamate receptor subtype (NMDAR) remains silent because it is plugged by Mg^{2+}. This signaling to dorsal horn pain-projection neurons provides information about the time of onset, duration, and intensity of noxious stimuli from the periphery. Both astrocytes and microglia remain unchanged by these synaptic events (adapted from Milligan ED, Watkins LR. Pathological and protective roles of glia in chronic pain. Nat Rev. 2009;10:23–36)

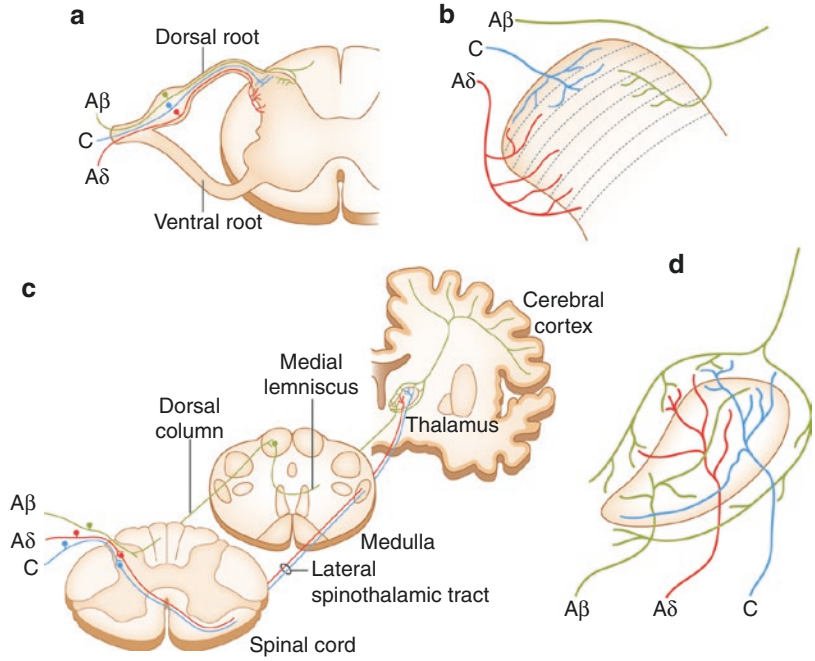


Fig. 4.28 Distribution of pathways involved in transmitting nociceptive information from peripheral nerves to higher levels of the brain for processing. (**a**) Depicts sensory nerves passing through dorsal roots en route to points of termination in the dorsal horn of the spinal cord and medulla. Specific fiber types terminate in different portions of the dorsal horn, as illustrated in (**b**). Signals effectively relayed in the spinal cord and brain stem course through the medical lemniscus and lateral spinothalamic tracts and terminate in a topographic fashion in the thalamus [(**c**) and enlarged in (**d**)] where the stimulus is consciously perceived. Projections from the thalamus connect with areas of the cerebral cortex (**c**) where further analysis and association with past experience are made (adapted from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

somatotopically organized, and modality specific. The second component, the paleospinothalamic tract, provides the basis for the affective and modulatory components of pain and is associated with the C fiber system. Its projections are more diffusely organized. In addition to projecting to the VPN, the paleospinothalamic tract provides collateral connections to the nuclei of the rostral ventromedial medulla (RVM), the lateral tegmental nucleus (LTN), the periaqueductal gray (PAG), the posterior and intralaminar nuclei of the thalamus, the basal telencephalic regions, limbic and paralimbic forebrain, amygdala, fornix, habenula, septal nuclei, and the hypothalamus. Upon termination in the thalamus, the nociceptive signal is consciously perceived [14].

Neurons in the VPN relay the nociceptive signal to the primary and secondary somatosensory cortices for the processing of location, intensity, and stimulus characterization and to the inferotemporal and frontal cortices for cognitive and contextual content and for cognitive, affective, and executive responses, respectively (Fig. 4.28). In the cortex, nociceptive signals are integrated and compared with past experience, emotions, mood, and current status for interpretation and implementation of a behavioral response. It is in this integrative process that the initial nociceptive signal is transformed into the complex, uncomfortable sensory and emotional experience that we call pain. It is the dynamic relationship between the thalamic neurons and the cortical modulating cells that determines the intensity of the unique painful experience perceived by each individual at any moment in time. Following the integration of the discriminative and affective components of the pain pathway, corticofugal projections return to VPN and surrounding

thalamic association nuclei, to the hypothalamus, and to brain stem nuclei. These projections can either augment or diminish the level of pain that is perceived for facilitation of a fight-or-flight response, depending on the state of the individual.

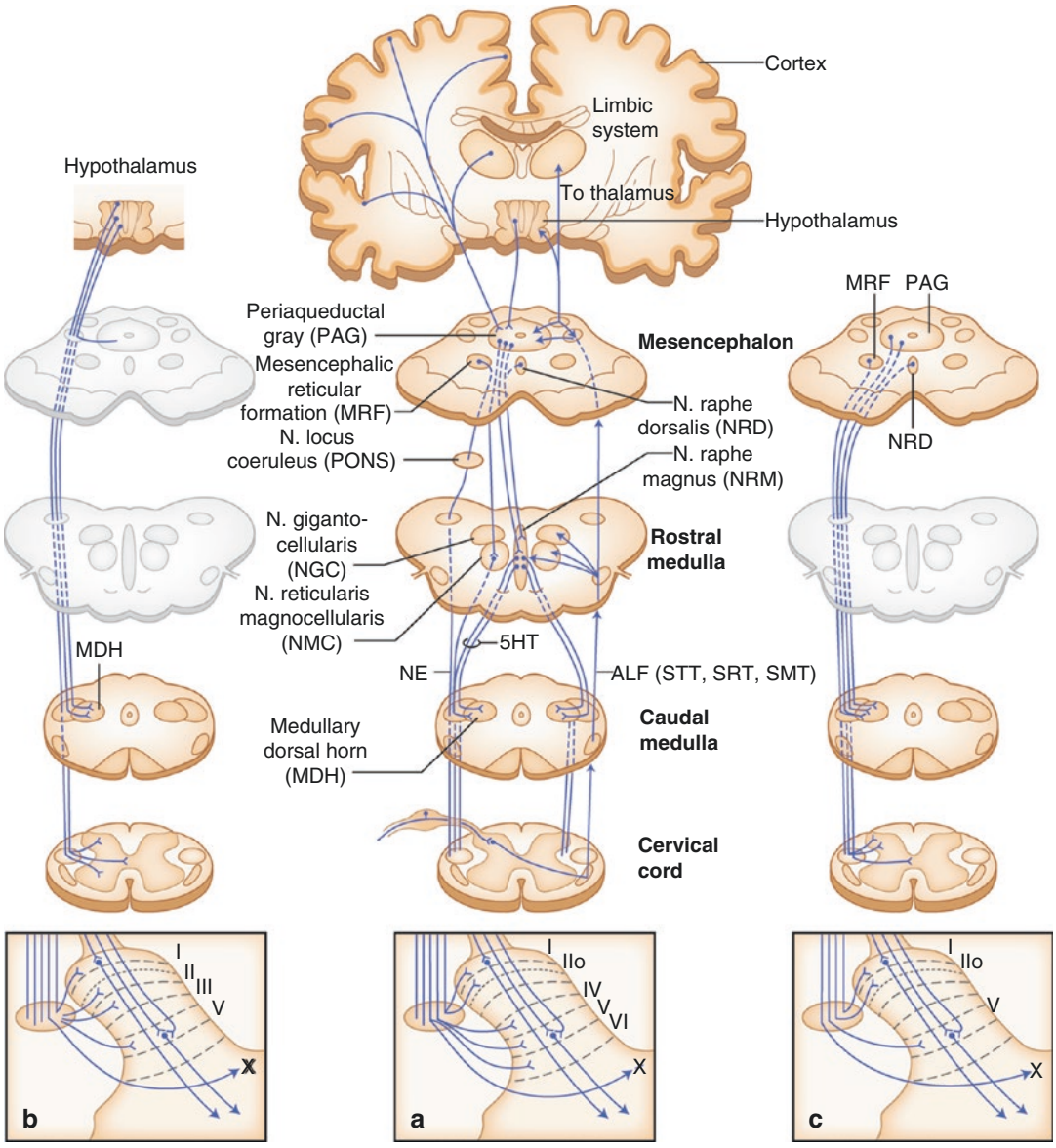
Stimulus Modulation and Behavioral Response

The hypothalamus monitors basal body functions, such as thirst, hunger, satiety, sexual function, blood pressure, temperature, and emotion, and influences behavior based on conscious and subconscious information sent from the cortex and from various body organs to maintain normal body function. Hypothalamic modulation of the behavioral response can be affected through the release of several hormones, including vasopressin, corticotropin-releasing factor (CRF), and pituitary adrenocorticotropic hormone (ACTH), that act centrally or peripherally to produce direct or indirect activity on pain-transmitting neurons. The process of modulation occurs through direct projections that affect the activity of enkephalinergic neurons of the PAG, the norepinephrine-containing neurons of the LTN, the serotonergic neurons of the RVM, and the neurons in the entry zones that receive primary afferent input [8, 15]. Projections from the RVM and the LTN descend through the brain stem and the dorsolateral funiculus of the spinal cord and synapse on the terminals of the primary afferent neurons and on inhibitory enkephalinergic and GABAergic interneurons of the dorsal horn, thereby indirectly affecting the transmission of nociceptive signals through the dorsal horn (Fig. 4.29). These projections can block the release of neurotransmitter from the primary afferent terminals, stimulate local inhibitory interneurons, or stabilize the membrane of the relay neurons and thus suppress the amount of nociceptive signal that is allowed to pass through the dorsal horn en route to higher integrative centers. Depending on the state of the individual, modulation of these descending systems can produce the opposite effect through reduction of the level of direct inhibitory input or through the disinhibition of local inhibitory circuits, thus amplifying noci-

Fig. 4.29 Descending endogenous pain inhibitory systems. (**a**) The most extensively studied and probably the most important descending system, composed of four-tiered parts. The ascending anterolateral fasciculus (ALF), composed of the spinothalamic, spinoreticular, and spinomesencephalic tracts, has important inputs into the nucleus raphe magnus (NRM), nucleus magnocellularis (NMC), nucleus reticularis gigantocellularis (NGC), and the periaqueductal gray (PAG) via the nucleus cuneiformis. The ALF also has input to the medullary/pontine reticular formation, the nucleus raphe dorsalis (NRD), and the mesencephalic reticular formation (MRF). The PAG receives input from such rostral structures as the frontal and insular cortices and other parts of the cerebrum involved in cognition and from the limbic system, thalamus, and hypothalamus, which sends β-endorphin axons to the PAG. The locus coeruleus in the pons is a major source of noradrenergic input to the PAG and dorsal horn (tract-labeled NE). These mesencephalic structures (PAG, NRD, MRF) contain enkephalin (ENK), dynorphin (DYN), serotonin (5-HT), and neurotensin (NT) neurons, but only the latter two send axons that project to NRM and NGC. Here, they synapse with neurons that are primarily serotonergic, whose axons project to the medullary dorsal horn and descend in the dorsolateral funiculus to send terminals to all laminae of the spinal gray (the densest populations are found in laminae I, II, and V of the dorsal horn and the motor neuron pools of lamina IX). The projection from NRM is bilateral, whereas the projection from NGC is ipsilateral. Noradrenergic fibers descend and project to the medullary dorsal horn and then descend in the dorsolateral funiculus of the spinal cord to send terminals to laminae I, II, IV, through VI, and X. (**b**) A simplistic schema to show the direct hypothalamospinal descending control system, which originates in the medial and paraventricular hypothalamic nuclei. This descending system consists of vasopressin and oxytocin neurons (and perhaps some enkephalinergic neurons), which not only send terminals predominantly to laminae I and X but also provide sparse input into laminae II and III and the lateral part of lamina V, as well as the homologous area in the medullary dorsal horn. (**c**) Direct PAG-spinal projection system, which bypasses the medullary nuclei and projects directly to the medullary dorsal horn and then descends in the dorsolateral funiculus to send terminals to laminae I, II_o, V, and X. Most of the axons are serotonergic and noradrenergic (adapted from Terman GW, Bonica JJ. Spinal mechanisms and their modulation. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 73–152)

ceptive signals and augmenting the likelihood that additional signals of a painful nature will be transmitted to the thalamus for perception [8].

For optimum survival, it is important to prepare the organism for an appropriate behavioral response and return the monitoring system to optimum levels of functioning in anticipation of additional warnings. This function is built into the nervous system. Since pain may well signal a threat to the survival of at least a part of an individual, painful stimuli automatically prepare the individual for rapid assessment of the afferent stimulus and the initiation of defensive "fight-or-flight" behavior through activation of the sympathetic nervous system (Fig. 4.30). The sympathetic nervous system controls blood pressure, heart and breathing rate, and the volume of blood that flows to specific tissues—more to voluntary muscles, heart, and lungs and less to the intestinal system and skin. The neurotransmitter that is released to produce these responses is norepinephrine. When released in the vicinity of peripheral afferent nerve



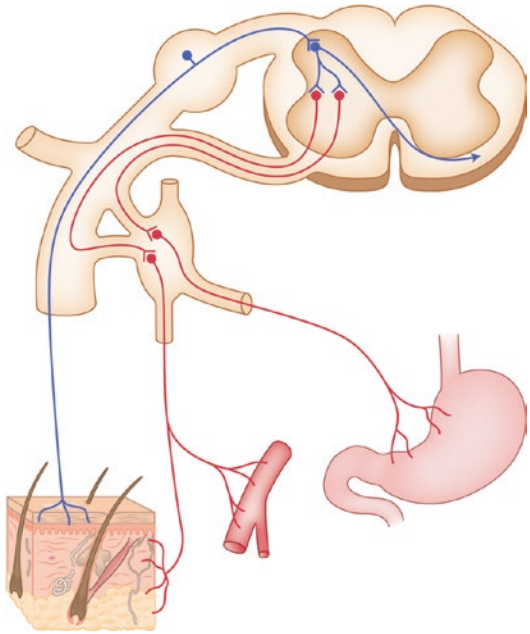


Fig. 4.30 Schematic depiction of sympathetic efferent projections in red that contribute to the "fight-or-flight" response to a nociceptive stimulus (from Gould 2007, modified from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's Management of Pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 26–72)

terminals, impulse generation is made easier. The sympathetic tone is modulated through descending cortical and hypothalamic projections that determine the firing frequency of preganglionic sympathetic neurons located in the intermediolateral cell column of the spinal gray matter from C8 (T1) to L1–L2 levels of the spinal cord.

After a nociceptive signal has been effectively relayed to the thalamus for further processing, the mechanisms responsible for receiving the nociceptive signals must be reset in the event that additional noxious stimuli requiring assessment arrive at the dorsal horn. To accomplish this, active relay neurons send axon collaterals to local inhibitory neurons in the dorsal horn that project back to the primary afferent terminal and to the initiating relay neuron to inhibit further activity and thus reduce the likelihood that multiple impulses will be sent to higher levels of analysis. The primary transmitters utilized by these inhibitory neurons are GABA and glycine.

Pathway Alterations Following Injury

After an injury, it is important to be aware of the area that has been injured so as not to subject it to further trauma that could exacerbate the injury. Mechanisms to enhance sensitivity in an injured region are also present in the normal nervous system, thereby aiding in recovery by increasing vigilance of the wound during the healing process. A significant portion of stimulus enhancement occurs during the process of peripheral sensitization. Peripheral sensitization is a by-product of the inflammation that is part of the mechanism of repair. It is present following injury and continues through the time that the wound has healed [16]. The process is initiated when tissue is injured by a thermal, mechanical, or chemical stimulus. Chemical mediators of inflammation are released from tissues in and around the site of injury. These chemicals increase blood flow to the injured area, carrying cells that engulf and destroy nonviable tissues and infectious agents, and increase levels of oxygen and nutrients necessary for repair. The inflammatory cells sequester particulate by-products of the cleanup and remove the by-products of metabolism. This process directly sensitizes the local nociceptors at the site of injury and, through the release of neurochemicals from collateral free nerve endings, indirectly sensitizes free nerve endings in adjacent tissues (Fig. 4.31). Consequently, the threshold for peripheral nociceptors is lowered, which increases the likelihood that a warning signal will be generated in a primary afferent nerve cell.

Abnormal sites for generation of a nociceptive signal that lead to repetitive firing and spontaneously generated pain can also develop when nerves are injured [7]. The mechanisms of injury vary, resulting in unique alterations in the normal function and integrity of the nerve. The alterations can include the destruction or damage to the neuronal cell bodies, the axons with their central and/or peripheral processes, the specialized endings in the peripheral tissues, and the supportive glial and Schwann cell elements as a result of toxic, metabolic, infectious, traumatic, and con-

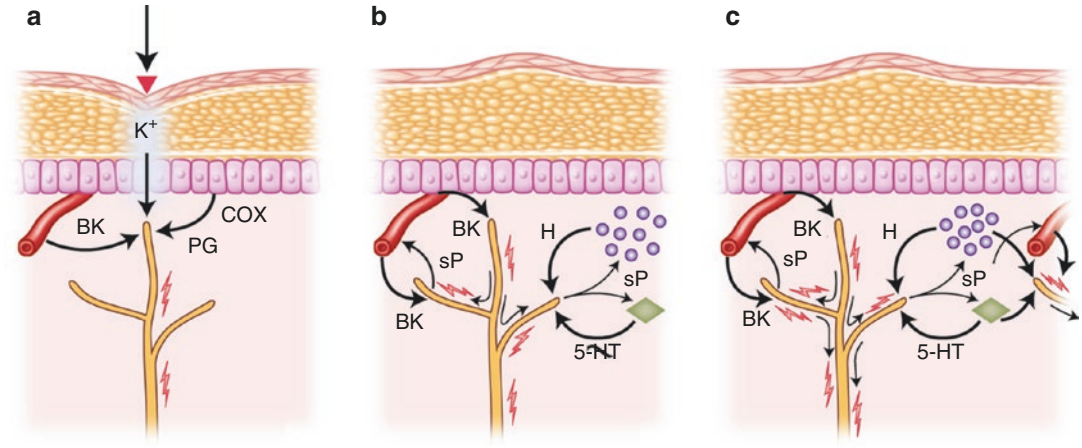


Fig. 4.31 Inflammatory enhancement of the pain signal. (**a**) Shows that noxious stimulation (arrow) results in the local release of protons (K^+) and inflammatory chemicals, bradykinin (BK) and prostaglandins (PG). A nociceptive signal is initiated and transmitted to the spinal cord. (**b**) Illustrates the nerve impulse as it extends into the peripheral terminal branches of free nerve endings causing the release of neurochemicals, e.g., substance P (sP), which stimulate the release of additional BK and other chemicals, histamine (H), and serotonin (5-HT). BK, H, and 5-HT make local and adjacent terminals (**c**) more sensitive to stimulation and thus more likely to generate a nociceptive signal. Modified from Byers and Bonica, 2001 (from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

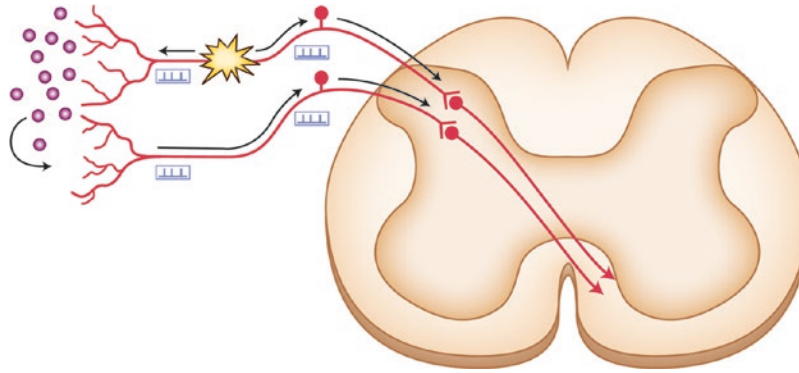


Fig. 4.32 Ectopic firing of injured nerve cells and peripheral sensitization. When peripheral nerves are injured, nerve impulses can be generated spontaneously at abnormal sites along the axon. An impulse is transmitted to the spinal cord and brain in the normal fashion but, in addition, is transmitted peripherally to the afferent terminals. Chemical mediators such as substance P are released and sensitize adjacent free nerve endings, enabling the initiation of a nociceptive signal in response to a non-noxious stimulus. Modified from Woolf and Mannion 1999 (from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

genital processes involving either the central or the peripheral nervous system. Such changes potentially lead to alterations in the numbers and relative densities of ion channels responsible for cellular excitability. In a significant portion of the population, neuronal injury results in such changes that make it possible for impulses to be generated at abnormal sites along the course of an axon rather than just at the generator zone of nerve terminals and at synapses (Fig. 4.32) [17]. Because of altered numbers, types, and distribution of ion channels, spontaneous channel openings allow the entry of sufficient sodium ions into the axon to depolarize the membrane [7, 18]. In

the periphery, the wave of depolarization proceeds away from the active site both toward the spinal cord and toward the body surface [19]. When a nerve impulse reaches the free endings of the afferent nerve terminal, neurochemical mediators are released from the terminals as described earlier, resulting in peripheral sensitization of the adjacent nerve terminals and the generation of nerve signals as a result of either noxious or nonnoxious stimuli. The signals then project centrally; reach the spinal cord, thalamus, and cortex; and are perceived as pain in the region of the body served by the aberrantly firing nerve. The resulting perception of pain can thus occur in the absence of a noxious stimulus being delivered to the body at the time of perception.

Enhanced peripheral activity associated with tissue injury, especially in individuals susceptible to developing neuropathic pain related to nerve injury, potentially lays the foundation for the development of persistent or permanent pain states. The regular and frequent signals are passed to the central nervous system, and through a process of central sensitization called "windup," the repetitive firing of peripheral C fibers produces a gradual increase in the perception of a stimulus irrespective of an increase in stimulus intensity [20]. This phenomenon effectively increases the likelihood that a stimulus will be relayed to levels of cognitive perception through a sensitization of relay neurons in the dorsal horn of the spinal cord. If the process of central sensitization is allowed to persist, high levels of sP and glutamate remain in the synaptic cleft (Fig. 4.33). When concentrations of sP and glutamate remain high in the synaptic cleft due to repetitive firing of primary afferent neurons, NK-1, kainite, ionotropic (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA), and metabotropic (mGluR) receptors, and through continued depolarization of the postsynaptic membrane in the presence of increased levels of glycine, released from local inhibitory interneurons, *N*-methyl-D-aspartate (NMDA) glutamate receptors are activated allowing entry of calcium as well as sodium into the postsynaptic cell. Other voltage-gated calcium channels present in the relay cell membrane also are activated and allow the entry of additional calcium into the relay neurons. Excess levels of intracellular calcium lead to activation of calcium-calmodulin protein kinase II α (CaMKIIα), cyclic adenosine

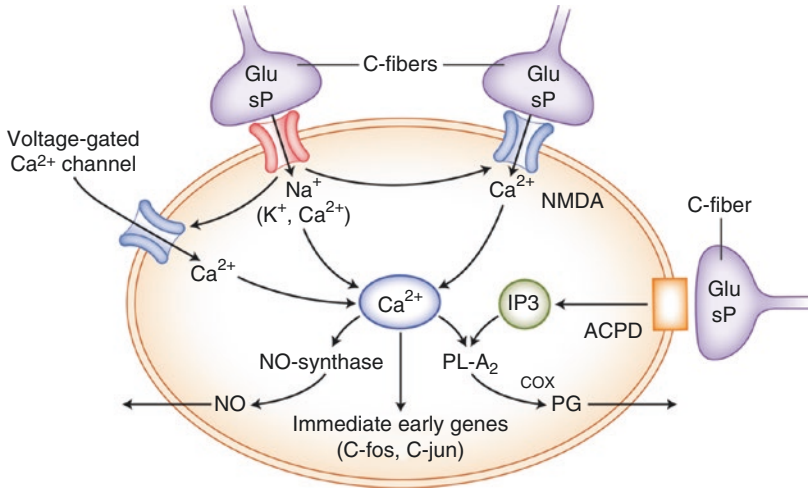


Fig. 4.33 Repetitive stimulation results in the activation of NMDA glutaminergic receptors and voltage-gated calcium channels. The entry of excess calcium stimulates the synthesis of nitric oxide and prostaglandins that are released from the neuron, resulting in sensitization of neighboring relay neurons and the possible initiation of the genetic process for the synthesis of cellular proteins. Modified from Ollat H, Cesaro P. Pharmacology of neuropathic pain. Clin Neuropharmacol. 1995;18:391–404 (from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

monophosphate (cAMP), brain-derived neurotrophic factor (BDNF), protein kinase A and C (PKA and PKC), and a further increase in AMPA receptor activation [21]. Through activation of the IP3 and DAG pathways, there is enhanced production of nitric oxide and prostaglandins that are released into the local neuropil. These mediators decrease the firing threshold of adjacent relay neurons, thus strengthening signal transmission and making it possible for adjacent neurons to reach firing threshold upon receiving an input generated by any level of stimulation (Fig. 4.34). In addition, the activation of a prostaglandin-dependent PKC pathway is thought to be a crucial component in "hyperalgesic priming," a process by which an injurious event produces changes in peripheral afferents that results in an exaggerated and prolonged hyperalgesic response to a subsequent minimally noxious or non-noxious stimulus that sets the stage for the development of chronic pain [22]. The neuromodulating chemicals that are also released from hyperactive relay neurons can affect additional transmitter release from the primary afferent neuron and affect the release of cytokines, neurotransmitters, and trophic agents from local microglia and astrocytes (Fig. 4.35) [12]. The resulting cascade of events enhances the likelihood that both noxious and non-noxious stimuli will be sufficient to initiate transmission of a nociceptive signal to higher levels of the nervous system. Finally, continued high levels of intracellular calcium may initiate the synthesis of proteins such as extracellular signal-regulated kinase (ERK), p38, and c-Jun N-terminal kinase (JNK) for further sensitization of pathways that enhance nociceptive transmission and the synthesis of immediate early genes that provide a basis for generating new and permanent neuronal connections and establish the basic framework for permanent hypersensitivity or centrally generated pain [23, 24].

Clearly, the processing of painful signals within the nervous system is complex and involves many components that function sequentially and simultaneously to enhance survival of the individual. The system provides many fail-safe assurances to ensure the integrity of the

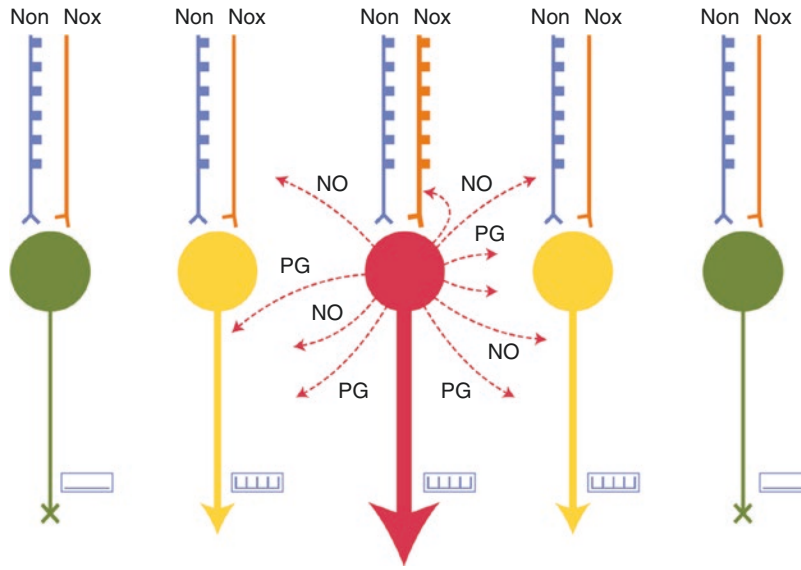
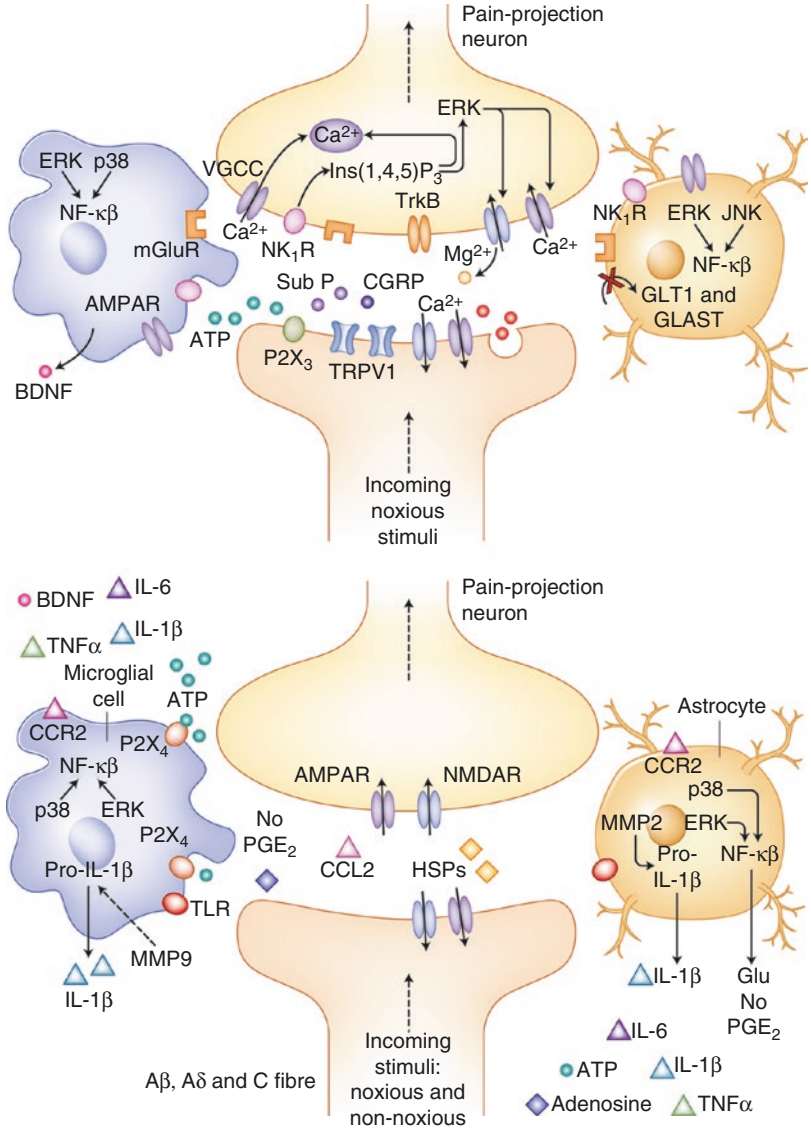


Fig. 4.34 Central sensitization enhances the transmission of a nociceptive signal. When nociceptive signals (Nox) repeatedly cause relay neurons to fire (arrowhead), prostaglandins (PG) and nitric oxide (NO) are released from the relay neuron (red), as illustrated in Fig. 4.32. PG and NO sensitize nearby nociceptive relay neurons (yellow) and enable them to respond to non-noxious stimuli (Non). Non-sensitized neurons (green) do not respond to non-noxious stimuli (from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

warning system to protect against serious injury, yet these assurances provide problems and frustration in achieving complete or even adequate pain relief. To achieve the best possible treatment of pain, all components must be considered as possible sources for pain generation and possible avenues for pain control. Knowledge of the anatomical and physiological basis for nociceptive processing and an understanding of the most likely sites where damage and intervention can occur are essential for providing optimum care for your patients.

Acknowledgments The authors wish to thank Dr. Dennis Paul for his helpful comments and suggestions in the preparation of this manuscript.

Fig. 4.35 Schematic depiction of the role of glia in processing repetitive nociceptive input and pain processing during inflammation. After repetitive synaptic communication, which can occur after a short barrage of nociceptive afferent input, there is an increase in the responsiveness of dorsal horn pain-projection neurons to subsequent stimuli (known as central sensitization). A co-release of glutamate and neurotransmitters such as substance P (sP) and calcitonin gene-related peptide (CGRP) mediates NMDAR activation, leading to voltage-gated Ca^{2+} currents (VGCCs). In addition, inositol-1,4,5-triphosphate ($Ins(1,4,5,)P_3$) signaling and mitogen-activated protein kinases, such as extracellular signal-regulated kinase (ERK), p38, and c-Jun N-terminal kinase (JNK), are activated. In neurons, ERK can further sensitize excited AMPA receptors (AMPARs) and NMDARs. Activation of purinoreceptors ($P2X_3$) by ATP, activation of sP receptors (the neurokinin 1 receptor (NK_1R)), activation of metabotropic glutamate receptors (mGluR), and release of brain-derived neurotrophic factor (BDNF) all contribute to enhanced nociceptive transmission. Astrocytes and microglia express various neurotransmitter receptors and are activated by glutamate, ATP, and sP. At synapses, the glutamate transporters, glutamate transporter 1 (GLT1), and glutamate-aspartate transporter (GLAST), which are crucial for clearing synaptic glutamate, become dysregulated after prolonged exposure to high levels of p38 and JNK activation in microglia and astrocytes. Each of these kinases can activate the transcription factor nuclear factor κB (NF-κB), which induces the synthesis of inflammatory factors. Upregulation of the V1 transient receptor potential channel (TRPV1) after inflammation further contributes to the sensitization to noxious signals. During this time, normally non-nociceptive Aβ fibers can also activate pain-projection neurons. If noxious input persists, such as during chronic inflammation or nerve damage, sustained central sensitization leads to transcriptional changes in dorsal horn neurons that alter these neurons' function for prolonged periods. Astrocytes respond to this ongoing synaptic activity by mobilizing internal Ca^{2+}, leading to the release of glutamate (Glu), ATP that binds to $P2X_4$, tumor necrosis factor-α (TNF-α), interleukin 1β (IL-1β), IL-6, nitric oxide (NO), and prostaglandin E_2 (PGE_2). Activated microglia are also a source of all of these proinflammatory factors. Matrix metalloproteinase 9 (MMP9) induces pro-IL-1β cleavage and microglial activation, whereas MMP2 induces pro-IL-1β cleavage and maintains astrocyte activation. The activation of p38 mitogen-activated protein kinase (p38 MAPK) is induced in both microglia and astrocytes on IL-1β signaling. Astrocytes and microglia express the chemokine receptors CX3CR1 (not shown) and CCR2 and become activated when the respective chemokines bind. After nerve damage, heat shock proteins (HSPs) are released and can bind to Toll-like receptors (TLRs) expressed on both astrocytes and microglia, leading to the further activation of these cell types (adapted from Milligan ED, Watkins LR. Pathological and protective roles of glia in chronic pain. Nat Rev. 2009;10:23–36)


Pain-projection neuron
ERK
Ca2+
VGCC
Ins(1,4,5)P3
TrkB
ERK p38
NF-κβ
mGluR
Ca2+
NK1R
Mg2+
Ca2+
NK1R ERK JNK
NF-κβ
GLT1 and GLAST
Sub P
CGRP
Ca2+
AMPAR
ATP
P2X3
TRPV1
BDNF
Incoming noxious stimuli
BDNF
IL-6
TNFα
IL-1β
Microglial cell
ATP
CCR2
P2X4
NF-κβ
p38
ERK
P2X4
Pro-IL-1β
TLR
MMP9
IL-1β
Pain-projection neuron
AMPAR
NMDAR
No PGE2
CCL2
HSPs
Astrocyte
CCR2
p38
MMP2
ERK
Pro-IL-1β
NF-κβ
IL-1β
Glu
No PGE2
IL-6
ATP
IL-1β
Adenosine
TNFα
Aβ, Aδ and C fibre
Incoming stimuli: noxious and non-noxious

Review Questions

1. Inhibitory interneurons within the dorsal horn release inhibitory neurotransmitters such as:
   (a) Glycine and gamma (γ)-aminobutyric acid (GABA)
   (b) Glutamate and aspartate
   (c) Calcitonin gene-related peptide (CGRP), galanin, and substance P (sP)
   (d) Neurokinin, vasoactive intestinal peptide (VIP), and neuropeptide Y (NP-Y)
2. There are two components of the lateral spinothalamic pathway:
   (a) Neospinothalamic tract and paleospinothalamic tract
   (b) Subthalamic tract and cerebellar vermis tract
   (c) Anterior and posterior longitudinal tract
   (d) Neocerebellar and tuberculum tract
3. When glutamate concentration remains high due to repetitive firing of primary afferent neurons, the depolarized postsynaptic membrane in the presence of increased levels of glycine, released from local inhibitory interneurons, stimulates the opening of:
   (a) Serotonin receptors
   (b) Bradykinin receptors
   (c) Muscarinic receptors
   (d) *N*-methyl-D-aspartate (NMDA) glutamate receptors
4. Inputs to the wide dynamic range neurons provide the essential segmental framework for the "gate control theory" proposed by:
   (a) Melzack and Wall (1965)
   (b) Racz and Raj (1971)
   (c) Bonica (1958)
   (d) Lema (1986)
5. The gate control theory:
   (a) Is completely false
   (b) States that impulses transmitted by low-threshold mechanoreceptors can reduce the nociceptive signal that is relayed to higher integrative levels for conscious perception
   (c) Explains the mechanism of the gamma reflex loop
   (d) Is the basis of our understanding of saltatory conduction
6. The regular and frequent signals which can be passed to the central nervous system and through a process of central sensitization are called:
   (a) "Windup"
   (b) Diffusion
   (c) Archicerebellum redundancy
   (d) Schmidt-Lanterman syndrome
7. The consequences of "windup" include:
   (a) Quicker reflexes
   (b) Increased micturition and defecation
   (c) The repetitive firing of peripheral C fibers which produces a gradual increase in the perception of a stimulus irrespective of an increase in stimulus intensity
   (d) The sequential discharge of β fibers which produces γ-mediated pain
8. Unique structures, which are depolarized by stimuli in response to tissue damage:
   (a) Touch receptors
   (b) Nociceptors
   (c) Temperature receptors
   (d) Chloride channels
9. As the axons approach the spinal cord, they diverge from the main nerve trunk and enter the dorsal root where they course by their cell bodies in the DRG and enter the spinal cord to terminate on neurons in:
   (a) Rexed laminae I and II
   (b) Rexed laminae III and V
   (c) Rexed lamina X
   (d) All of the above
10. The axons of the C fiber system:
    (a) Are unmyelinated
    (b) Are myelinated
    (c) Are never found in the peripheral nerves of the somatic sensory system
    (d) Have fast conduction velocity of over 20 m/s
11. Neurons in the ventral posterior nucleus (VPN) of the thalamus relay the nociceptive signal to:
    (a) The primary somatosensory cortex
    (b) The secondary somatosensory cortex
    (c) The inferotemporal and frontal cortices
    (d) All of the above
12. After an injury, a significant portion of stimulus enhancement can occur during the pro-

cess of peripheral sensitization and is limited to injury by:
(a) Thermal stimulus
(b) Mechanical stimulus
(c) Chemical stimulus
(d) All of the above

13. Which is false regarding wide dynamic range neurons?
(a) They are found primarily in lamina V.
(b) They are responsible for much of the information that is transmitted to the brain stem and thalamus.
(c) These neurons receive polymodal inputs.
(d) One limitation is that they do not receive inputs from collaterals of non-nociceptive, low-threshold mechanical Aβ afferents and local internuncial neurons of the dorsal horn.

14. C fibers:
(a) Respond to polymodal stimuli but preferentially respond to noxious heat.
(b) Their central elements course medially in the dorsal root and terminate on neurons in Rexed lamina I, the outer portion of lamina II, and lamina V.
(c) Upon entering the spinal cord, the axons of the primary nociceptors ascend and descend in the zone of Lissauer.
(d) The majority of these fibers ascend approximately two spinal levels before terminating in the dorsal horn.

15. In myelinated axons, the excitable membrane that supports the propagation of action potentials found only in the intervals between adjacent segments of myelin is called:
(a) Nodes of Ranvier
(b) Basilar sulci
(c) Nervus intermedius
(d) Riopelle lipofuscin

Answers:

1. a
2. a
3. d
4. a
5. b
6. a
7. c
8. b
9. d
10. a
11. d
12. d
13. d
14. d
15. a

References

1. Woolf CJ, Decosterd I. Implications of recent advances in the understanding of pain pathophysiology for the assessment of pain in patients. Pain. 1999;6(Suppl):S141–7.
2. Woolf CJ, Max MB. Mechanism-based pain diagnosis. Issues for analgesic drug development. Anesthesiology. 2001;95:241–9.
3. Bevan S. Nociceptive peripheral neurons: cellular properties. In: Wall PD, Melzack R, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 85–104.
4. Raja SN, Meyer RA, Ringkamp M, Campbell JN. Peripheral neural mechanisms of nociception. In: Wall PD, Melzack R, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 11–57.
5. Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's management of pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 26–72.
6. Rang HP, Bevan S, Dray A. Nociceptive peripheral neurons: cellular properties. In: Wall PD, Melzack R, editors. Textbook of pain. 3rd ed. Edinburgh: Churchill Livingstone; 1994. p. 57–78.
7. Devor M, Seltzer Z. The pathophysiology of damaged peripheral nerves. In: Wall PD, Melzack R, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 129–64.
8. Terman GW, Bonica JJ. Spinal mechanisms and their modulation. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's management of pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 73–152.
9. Basbaum AI, Bautista DM, Scherrer G, Julius D. Cellular and molecular mechanisms of pain. Cell. 2009;139:267–84.
10. Snyder LM, Ross SE. Itch and its inhibition by counter stimuli. Handb Exp Pharmacol. 2015;226:191–206.
11. Meyer RA, Campbell JN, Raja SN. Peripheral neural mechanisms of nociception. In: Wall PD, Melzack R, editors. Textbook of pain. 3rd ed. Edinburgh: Churchill Livingstone; 1994. p. 13–44.

12. Melzack R, Wall PD. Pain mechanisms: a new theory. Science. 1965;150:971–9.
13. Milligan ED, Watkins LR. Pathological and protective roles of glia in chronic pain. Nat Rev. 2009;10:23–36.
14. Adams RD, Victor M. Principles of neurology. 4th ed. New York: McGraw Hill; 1989.
15. Basbaum AI, Fields HL. Endogenous pain control systems: brain stem spinal pathways and endorphin circuitry. Annu Rev Neurosci. 1984;7:309–38.
16. Levine JD, Reichling DB. Peripheral mechanisms of inflammatory pain. In: Wall PD, Melzack R, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 59–84.
17. Paul D, Gould HJ III. Sodium channel modulation in the perception and management of pain. Spec Top Pain Manag Sci Am Pain Manag. 2018. In press.
18. Amir R, Devor M. Spike-evoked suppression and burst patterning in dorsal root ganglion neurons. J Physiol (Lond). 1997;501:183–96.
19. Woolf CJ, Mannion RJ. Neuropathic pain: aetiology, symptoms, mechanisms, and management. Lancet. 1999;353:1959–64.
20. Wall PD, Woolf CJ. The brief and the prolonged facilitatory effects of unmyelinated afferent input on the rat spinal cord are independently influenced by peripheral nerve section. Neuroscience. 1986;17:1199–205.
21. Voigt AW, Gould HJ III. Chronic daily headache—mechanisms and principles of management. Curr Pain Headache Rep. 2016;20:1–7.
22. Reichling DB, Levine JD. Critical role of nociceptor plasticity in chronic pain. Trends Neurosci. 2009;32:611–8.
23. Woolf CJ, Shortland P, Coggeshall RE. Peripheral nerve injury triggers central sprouting of myelinated afferents. Nature. 1992;355:75–8.
24. Kaye AD, Novitch MB, Gould HJ III. Pain, a comprehensive review and update. Neurosci Biobehav Psychology: Elsevier; 2017. ISBN 9780128093245.

Local Anesthetics and Adjuvants

5

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Introduction

Local anesthetics produce reversible and complete blockade of neuronal transmission when applied near the axons. They block primarily voltage-gated sodium channels. Their application results in interruption of nerve impulse conduction, allowing not only abolition of sensation from the area innervated by the corresponding nerves but also motor blockade. A number of compounds with local anesthetic activity occur in nature such as cocaine, eugenol derived from plants, tetrodotoxin derived from fish species in the family Tetraodontiformes, and saxitoxin derived from algae (*dinoflagellates*). The first reported medicinal use of a drug as a local anesthetic occurred in 1884 when Carl Koller used cocaine to anesthetize the eye by topical application.

It is also important to mention that recent publications have demonstrated additional properties of the local anesthetics other than being sodium channel blockers. They interact with various receptors and pathways and have an effect in chronic pain and demonstrate anti-inflammatory and potential antimetastatic properties [1–3].

This chapter describes the basic chemical structure of local anesthetics, the basic receptor pharmacology, and gives an overview over pharmacologic properties of the different drugs. Clinical use, advantages, and side effects are compared. The mechanism of action and effects of adjuvant drugs used in regional anesthesia is also explored. Finally, some clinical pearls are highlighted, and local anesthetic toxicity is described.

Local Anesthetics

Chemical Structure

Local anesthetic molecules are comprised of three basic building blocks: a hydrophobic aromatic ring, a hydrophilic tertiary amine, and an intermediate chain connecting the two. Hydrocarbon chain length varies between 6 and 9 Å. The chemical connection between the intermediate chain and the aromatic ring divides local anesthetics in "esters" and "amides" depending on whether the hydrocarbon chain is joined to the benzene-

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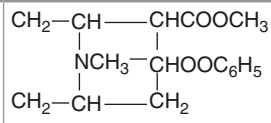
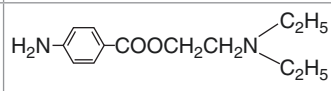
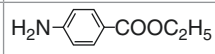
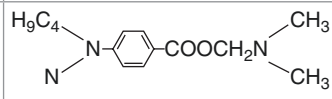
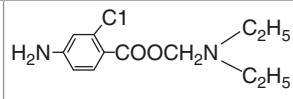
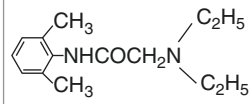
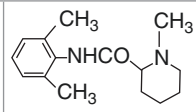
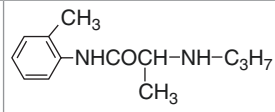
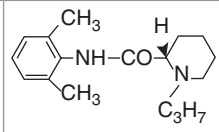
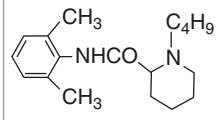
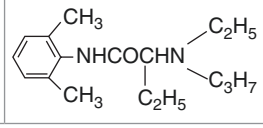
A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_5

derived moiety by an ester or an amide linkage. The type of linkage is important as it determines how local anesthetics are metabolized. Moreover, this chemical differentiation is clinically relevant because the amides are more stable and have less risk of allergic reaction than the esters (Table 5.1).

Table 5.1 Physiochemical properties of local anesthetics

Drug (brand name)	Type (year introduced)	Chemical structure	Relative in vitro potency			
			Rat sciatic nerve	pK_a	Partition coefficient[a]	Plasma protein binding
Cocaine	Ester		–	8.6	–	92
Procaine (Novocaine)	Ester (1905)		1	8.9	1.7	5.8
Benzocaine	Ester (1900)		–	3.5	81	–
Tetracaine (Pontocaine)	Ester (1930)		8	8.5	221	75.6
2-Chloroprocaine (Nesacaine)	Ester (1952)		1	8.7	9.0	NA
Lidocaine (Xylocaine)	Amide (1944)		2	7.72	2.4	64.3
Mepivacaine (Carbocaine, Polocaine)	Amide (1957)		2	7.6	21	77.5
Prilocaine (Citanest)	Amide (1960)		2	7.7	25	55
Ropivacaine (Naropin) Amide (1995)	Amide (1995)		4	8.1	115	95
Bupivacaine (Marcaine, Amide (1963) Sensorcaine) Levobupivacaine (Chirocaine)	Amide (1963)		8	8.1	346	95.6
Etidocaine (Duranest)	Amide (1972)		8	7.74	800	94

From: Mulroy MF. A Practical Approach to Regional Anesthesia. 4th ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2009:pg 3. Reprinted with permission from Wolters Kluwer Health

[a]Otanol: buffer pH 7.4

Site of Action and Nerve Conduction

Sodium Channel Structure

The human sodium channel is a transmembrane protein composed of three subunits forming a voltage-sensitive and sodium-selective channel [4]. Different isoforms are expressed in different tissues (muscle, heart, central nervous system, peripheral nervous system, etc.) [5]. Mutations with different sensitivity to local anesthetics are possible and have been shown in the experimental but not (yet) in clinical setting [6].

Conduction

With electrical excitation of the neuron, a depolarizing stimulus is conducted down an axon. A stimulus of significant magnitude changes the negative resting potential from −70 mV toward −55 mV, the threshold required for complete depolarization: sodium channels in the cell membrane are activated and open, permitting Na^+ ions to move down their electrochemical gradient intracellularly and locally "depolarize" the axonal membrane. This influx of cations rapidly changes the membrane potential to +35 mV. The resultant propagation of voltage change down the axon is defined as the action potential. Local anesthetic molecules traverse the cell membrane and then block the sodium channel from within the cell blocking propagation of the action along the nerve.

Repolarization

The sodium channel is inactivated after a few milliseconds by a time-dependent change in conformation closing an inactivation gate (Fig. 5.1). The inactivated state cannot conduct Na^+ and is not reopened if further stimulated (refractory period). Thereafter, the Na^+ channel changes further to the closed (resting) state. In this state, it cannot conduct Na^+ ions but, with a sufficiently strong stimulus, will convert the channel to the open state.

Binding of Local Anesthetics

Local anesthetics do not bind to a classical "receptor"; it is more a "binding" site which is located within the sodium channel near its intracellular opening [6]. It is, on the one hand, a hydrophobic region to which the hydrophobic part of the local anesthetic molecule "binds" and, on the other hand, a hydrophilic region with which the quaternary amine interacts. Any change in amino acid sequence can prevent local anesthetics from being effective.

Action potentials are blocked due to an inhibition of Na^+ movement through the Na^+ channel by direct blocking or influencing of the Na^+ channel conformation.

Pharmacodynamics and Physiochemical Properties of Local Anesthetics

Potency

The minimal local anesthetic concentration required to produce neural blockade is defined as potency. Lipophilicity correlates in in vitro settings well with local anesthetic potency. In vivo, this correlation exists but is less stable.

Phasic Block

The faster a nerve is stimulated, the lower the concentration of local anesthetic is needed to produce a blockade (in vitro). This observation is

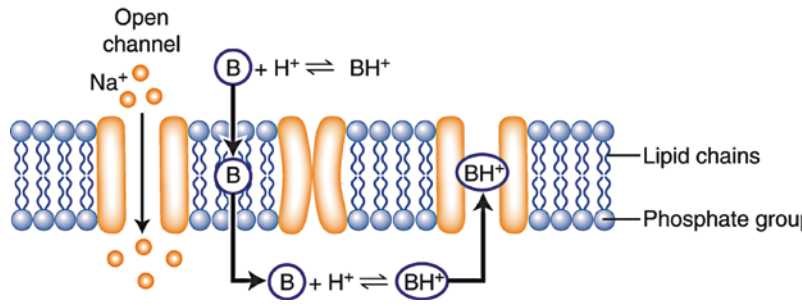


Fig. 5.1 Mechanism of action of local anesthetics. From: Rathmell JP. Regional Anesthesia: The requisites in anaesthesiology. 1st ed. Philadelphia:Elsevier Mosby; 2004:pg 17

called phasic block or rate-dependent block. Typically, phasic block occurs with more hydrophobic (potent) local anesthetics. They show a greater difference in their binding affinity in dependence of the different channel states compared to the less potent local anesthetics. There is no clear data about phasic block in the in vivo model, but phasic block seems to explain why hydrophobic local anesthetics are more cardiotoxic than hydrophilic local anesthetics.

Anesthetic Block in Dependency of Nerve/Axon Exposed

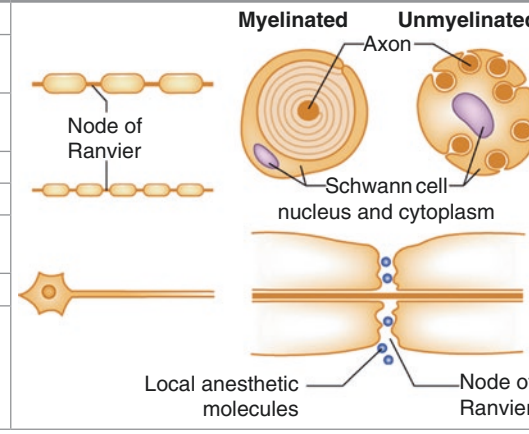
Axons are classified with respect to their structure (myelinated, unmyelinated), diameter, conduction velocity, and function. The characteristics of local anesthetic blockade vary among different axon types, but the exact role of size, myelination, or function in axonal blockade is, to date, not entirely clear (Table 5.2).

- Unmyelinated axons: the concentration of local anesthetic required to block conduction of unmyelinated axons decreases with increasing length of nerve exposed to the local anesthetic.
- Myelinated axons: myelin consists of Schwann cell plasma membranes wrapped around axons. There are gaps, called nodes of Ranvier, at fixed intervals between the myelinated areas. Myelination results in much faster conduction velocities because the axonal membrane needs to be only depolarized at the node. This process is called saltatory conduction.
- Unmyelinated axons (C fibers) are in vitro the most resistant to local anesthetic blockade, followed by large (Aα, Aβ fibers) and small (B fibers) myelinated axons [7]. Intermediate-size myelinated axons (Aδ, Aγ fibers) are the easiest axons to block in vitro.

Local anesthetics can gain access to axonal membrane of myelinated axons only at the nodes of Ranvier. In vitro, the Na^+ channels in approximately three consecutive nodes (0.4–4 mm) need to be blocked for axonal conduction to fail.

Table 5.2 Axon classification

Fiber type	Size (μm)	Function	Local anesthetic sensitivity (in vitro)	Illustrations
A				
α	12–20	Somatic motor, proprioception	++	
β	5–12	Touch, pressure motor to muscle spindles	++	
γ	3–6	Motor to muscle spindles	+++	
δ	2–5	Pain, temperature, touch	+++	
B	<3	Autonomic (preganglionic)	++	
C	0.3–1.4	Pain, reflex responses	+	
		Autonomic (postganglionic)		



From: Mulroy MF. A Practical Approach to Regional Anesthesia. 4th ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2009:pg 9. Reprinted with permission from Wolters Kluwer Health

Human axons are classified by size, presence or absence of myelin, and function; in vitro, small unmyelinated axons are most resistant to local anesthetic blockade, whereas large myelinated axons are the most sensitive. In vivo, however, the sensitivity to local anesthetic block is different for reasons that are not fully understood (see chapter on Clinical Pharmacology of Local Anesthetics). "+" indicates the relative sensitivity to local anesthetic block

Acid-Base and pK_a

Local anesthetics (except benzocaine) are weak bases (pK_a = 7.6–9.0) that are commercially prepared as an acidic solution, typically at pH 4–5. The pK_a defines the pH, where half of the drug is ionized (positively charged form, conjugate acid) and half is nonionized (base). The ionized and nonionized forms have different, but important, clinical effects. The nonionized form penetrates the nerve membrane, while the ionized form binds to proteins on the intracellular side of the sodium channel (Fig. 5.2). The percentage of each form present in a solution or in the tissue depends on the pH of the solution or tissue and can be calculated from the Henderson-Hasselbalch equation:

$$pK_a = pH - \log(\text{base})/(\text{acid}),$$

where pH is the pH in the solution/tissue and pK_a is the pH at which half the local anesthetic molecules are in the base form and half in the acid form.

The pK_a of each local anesthetic is unique and measures the tendency of the molecule to accept a proton in the base form or to donate a proton in the acid form. Most local anesthetics have a pK_a between 7.5 and 9.0.

Sodium bicarbonate can be added to local anesthetic solutions to raise the pH of the solution, thereby increasing the nonionized form. Other factors being similar, local anesthetics with more basic pK_a have a slower onset of blockade effect due to the lesser amount of nonionized local anesthetic molecules at physiologic pH. This relative lack of the nonionized form impairs local anesthetic movement across the cell membrane and thus delays block onset (Fig. 5.2).

Hydrophobicity

The charged form of all local anesthetics is more hydrophilic than the uncharged form. Hydrophobicity correlates with potency and, to a certain extent, to duration of action: the more hydrophobic the drug, the more potent it is. Hydrophobicity facilitates penetration of the neuronal cell membrane, which accelerates local anesthetic binding to the intracellular portion of the sodium channel.

Adding local anesthetic to a recipient containing two immiscible liquids like an aqueous buffer and a hydrophobic lipid is needed to determine hydrophobicity. The resultant ratio of the concentrations is called the "distribution coefficient" (partition coefficient).

Protein Binding

One of the most important clinical characteristics of local anesthetics is its duration of action, which correlates with the degree of local anesthetic protein binding (typically to albumin and α-1-acid-glycoprotein). Binding to plasma protein varies between 5 and 95%. In general, more hydrophobic drugs have higher protein binding.

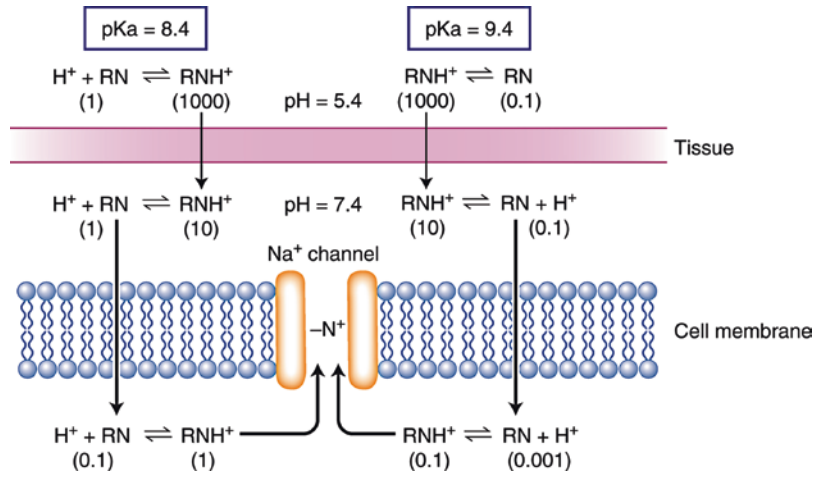


Fig. 5.2 Effect of ionization on activity. From: Rathmell JP. Regional Anesthesia: The requisites in anaesthesiology. 1st ed. Philadelphia:Elsevier Mosby; 2004:pg 18

However, plasma protein binding does not correlate necessarily with tissue protein binding.

Normally, short-acting local anesthetics have a fast onset of action, while long-duration local anesthetics have a slower onset of clinical effects. Serum protein binding also protects against drug toxicity because only the free (protein unbound) local anesthetic fraction can induce toxicity. However, once serum proteins are saturated, any additional administration or absorption of local anesthetics rapidly causes toxicity. Therefore, patients show a rapid progression from no signs of local anesthetic toxicity to manifestations of severe toxicity (CNS, cardiac) when highly protein-bound local anesthetics are used inadequately.

Binding to plasma proteins is mainly pH dependent: binding decreases during acidosis due to the decrease of available binding sites in an acidic environment.

Metabolism

Ester local anesthetics are primarily metabolized by ubiquitous plasma cholinesterases (pseudocholinesterase). These enzymes are synthesized by the liver and are found throughout the vascular system and in the cerebrospinal fluid (CSF). They are responsible for the metabolism of numerous drugs of relevance to the anesthesiologist, including ester local anesthetics, succinylcholine, and mivacurium. Because of the widespread distribution of these enzymes, plasma degradation of ester local anesthetics is typically rapid. In contrast, amide local anesthetics undergo degradations by hepatic enzymes and typically have a longer serum half-life.

Summary

The comprehension of the principles described in this chapter is essential to understand local anesthetic clinical pharmacology. However, one should keep in mind that the clinical setting is much more complicated as there are multiple influencing factors that cannot be reproduced in in vitro studies. It is also important to mention that recent publications have demonstrated additional properties of the local anesthetics other than being sodium channel blockers. They interact with various receptors and pathways and have an effect in chronic pain and demonstrate anti-inflammatory and potential antimetastatic properties [1].

Clinical Pharmacology of Local Anesthetics

Factors Determining Block Quality

Block Onset

The proximity of the injected local anesthetic to the nerve is the most important factor determining block onset; the nearer to the nerve, the shorter the time required to diffuse into the nerve.

The total local anesthetic dose and not the volume or concentration determines the onset time, the duration, and the intensity of the nerve block [8].

The choice of the local anesthetic is a crucial issue since hydrophobic agents are more prone to bind to hydrophobic sites on connective tissue compared to hydrophilic drugs. This explains the slower onset of hydrophobic local anesthetics despite their greater potency.

Block Duration

The main factor influencing block duration is the clearance rate of the local anesthetics.

The choice of local anesthetic greatly influences block duration; hydrophobic local anesthetics have a slower clearance compared to hydrophilic local anesthetics. Moreover, hydrophobic compounds have a higher potency. These two factors are responsible for a longer-lasting block. Furthermore, local anesthetics show variable vascular effects on local blood vessels. Vasoconstriction will reduce clearance, impairing its transport from the injection site. High concentrations of local anesthetics lead to a vasodilation increasing local blood flow and consequently their own clearance. But with decreasing concentration, vasoconstriction is present

reducing clearance and increasing the duration of the block. Differences among local anesthetics are listed below.

The dose influences duration: larger doses of local anesthetics produce a long-lasting block compared to lower doses. This is explained by the longer time required to clear the higher amount of drug.

Block Potency

Lipophilicity correlates with potency: the more lipid soluble the local anesthetic, the more potent it is. Lipophilicity facilitates penetration through the cell membrane, thereby accelerating the binding of the local anesthetic to the intracellular binding site of the Na^+ channel. Lipophilicity is influenced by the lateral chains of the benzene ring.

Individual Local Anesthetics

Common local anesthetics used in clinical practice and their applications are shown in Table 5.3.

Ester Local Anesthetics

Cocaine

Topical mucous membrane applications of cocaine (4% solution) result in very rapid anesthesia and vasoconstriction. At excessive doses, vasoconstrictive properties lead to hypertension, coronary ischemia, and arrhythmias. Mixtures of lidocaine with phenylephrine or oxymetazoline are safer alternatives to cocaine for anesthetizing and vasoconstricting mucous membranes. Attention must be paid not to mix cocaine with other vasoconstrictors (phenylephrine) because of the increased risk of acute myocardial infarction [9].

Cocaine is metabolized in the liver to active metabolites. The half-life is approximately 45 min. If taken together with alcohol, the metabolic pathway is altered, and the highly toxic cocaethylene is produced.

The maximum recommended dose of cocaine is 200 mg. Attention must be paid to the use of cocaine for awake fiber-optic nasal intubation: as local anesthetic toxicity is additive, the use of cocaine 4% and lidocaine 4–10% or benzocaine can lead to systemic toxic reaction.

Procaine

Procaine was the first synthetic local anesthetic used clinically. Unfortunately, procaine combines a short duration and limited tissue penetration. Procaine is still occasionally used for skin infiltration (0.25–1.0%) and short duration (30–45 min) spinal anesthesia (50–100 mg), although discharge readiness may be slightly longer than that seen with equipotent doses of spinal lidocaine. The block after spinal anesthesia is shorter compared to the block induced by lidocaine but has a higher failure rate (inadequate sensory block). On the other hand, less transient neurologic symptoms (TNS) have been reported [10]. Procaine is ineffective when used topically and is not reliable for epidural anesthesia. It is not recommended for peripheral block since it has a very slow onset time paired with a short-acting time. Procaine is metabolized in the plasma by the cholinesterase; its elimination half-life is approximately 8 min.

The 10% solution should be diluted to 5% with dextrose or saline. Procaine is metabolized to *para*-aminobenzoic acid (PABA), which can be associated with allergic reactions.

2-Chloroprocaine

Compared to procaine, it has a more rapid onset and slightly longer duration of action. The principal uses of chloroprocaine are in obstetrics and ambulatory anesthesia. It has rapid onset when used for epidural anesthesia and is therefore frequently chosen for urgent forceps or Cesarean deliveries. In the 2–3% concentrations, it is also used for spinal anesthesia and peripheral blocks. Like other ester local anesthetics, chloroprocaine is rapidly metabolized by plasma cholinesterase, and with a duration of action between 30 and 60 min, it is a good drug for outpatient procedures. Since serum half-life is approximately 40 s, fetal accumulation and systemic toxicity, in general, are extremely unlikely.

The preservative-free solution should be used for central neuraxial blocks because of the concern regarding potential neurotoxicity.

Table 5.3 Local anesthetic drug clinical doses

Drug (brand name)	Epidural[f]						Maximum recommended doses			
							Plain		With epinephrine	
	Topical[f] (%)	Spinal[f] (%)	Surgical[f] (%)	Obstetric[f] (%)	Peripheral nerve block (%)	Intravenous regional (%)	Total	mg/kg	Total	mg/kg
Cocaine	4	NA	NA	–	NA	NA	200	1.5	–	–
Benzocaine	5–20	NA	NA	–	NA	NA	–	–	–	–
Short duration										
Procaine (Novocaine)	NA	10	NI	NI	1	NI	500	–	–	–
2-Chloroprocaine (Nesacaine)	NI	NA	2–3	2–3	1–2	NI	800	11	1000	14
Intermediate duration										
Lidocaine (Xylocaine)	4	5	1.5	1.5	0.5	0.5	300	4.5	500	7
			2	2[a]	1					
Mepivacaine (Carbocaine, Polocaine)	NA	NA	1	NI	1	NA	400	–	550	[e]
			1.5							
			2							
Prilocaine (Citanest)	NA	NA	2–3	NI	1	0.5	–	–	500	–
Long duration										
Ropivacaine (Naropin)	NA	0.5[c]	0.75, 1[d]	0.2	0.5	NA	250	–	250	3
Bupivacaine (Marcaine, Sensorcaine)	NA	0.5	0.5	0.125[e]	0.25	0.25[c]	175	–	225	3
		0.5	0.5	0.125[e]	0.25	0.5				
					0.5[a]					
Levobupivacaine (Chirocaine)										
Etidocaine (Duranest)	NA	NA	1	NI	1	NI	300	4	400	6
			1.5							
Tetracaine (Pontocaine)	1–2	1	NA	NA	NA	NA				

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Drugs are grouped in general duration of action. Concentrations listed are those recommended for particular application

NA not available, *NI* not indicated, *PDR physicians' desk reference*

[a]Preservative-free solutions only

[b]Produces motor blockade suitable for cesarean delivery

[c]Not approved for this use

[d]For single injection only; lower concentrations should be used for follow-up injections of catheters

[e]Specific dose for epinephrine-containing solution not identified; this is the largest described dose

[f]Not prepared commercially; must be diluted at time of use

Tetracaine

Tetracaine is the longest-acting ester local anesthetic. It is used in spinal and ophthalmic anesthesia and is occasionally used for topical airway anesthesia. The latter application has declined with the recognition that tetracaine has a narrow margin between therapeutic and toxic doses that may lead to serious systemic toxicity after mucosal application. Metabolism is slower compared to procaine; therefore, the risk of systemic toxicity is greater.

Tetracaine is less chemically stable compared to lidocaine and bupivacaine. This instability may result in an occasional failed spinal anesthetic due to degradation of the local anesthetic during storage.

Benzocaine

Benzocaine was the first developed but not the first clinically used synthetic local anesthetic. Because of its low pK_a (3.5), it only exists in the uncharged form at physiologic pH, and it is hardly soluble in aqueous solutions.

Therefore, it is exclusively used as a topical spray or troche for mucous membranes or for topical application (cream and gel) for dermal hypesthesia.

Methemoglobinemia seems to be observed more frequently when benzocaine is used. This high risk and the difficulty of proper dosage (cream and spray) increase benzocaine potential risk for toxicity.

Amide Local Anesthetics

Lidocaine

Lidocaine is the most widely used local anesthetic. It combines significant potency, fast onset, intermediate duration, good tissue penetration, and minimal cardiac toxicity. Lidocaine is widely used for infiltration (1–2%), intravenous regional anesthesia (0.5%), peripheral nerve blocks (1 and 1.5%), topical airway (4%), spinal anesthesia (0.2–5%), and epidural anesthesia (2%). It produces moderate vasodilation. The allergic potency is very low.

Lidocaine 5% has been implicated in the occurrence of cauda equina syndrome with the use of small-diameter microcatheters for continuous spinal anesthesia. Spinal microcatheters have since then been withdrawn from the US market. Single-shot spinal anesthesia can be associated with TNS, the etiology of which is uncertain [11, 12].

Mepivacaine

Mepivacaine has similar pharmacokinetic profile to lidocaine, with slightly longer duration and better tissue penetration. Chemically, it is a cyclic tertiary amine-like bupivacaine and ropivacaine. It is used primarily for intermediate-duration infiltration and peripheral, epidural, and spinal nerve blocks in Europe. It has a mild vasoconstricting effect which may be responsible for its longer duration compared to lidocaine. Mepivacaine is not used anymore in obstetric epidural anesthesia since this drug is poorly metabolized in the fetus and neonate and may be responsible for lower neurobehavioral score in the first days of life [13].

Prilocaine

Prilocaine is similar to lidocaine in its clinical profile and is widely used for intravenous regional anesthesia outside the USA. It is the most rapidly metabolized amide local anesthetic. Within the USA, prilocaine was withdrawn from use following several cases of methemoglobinemia. Prilocaine is metabolized to nitro- and orthotoluidine, which can oxidize hemoglobin to methemoglobin. Prilocaine is mainly used commercially in topical eutectic mixture of local anesthetics (EMLA) cream, as well as in proprietary mixtures of local anesthetics specifically marketed for airway anesthesia. Significant methemoglobinemia has been reported in both of these applications.

Articaine

A structural local anesthetic that has a five-membered-thiophene ring instead of a benzene ring as its hydrophobic tail, articaine 4% is used only as dental local anesthetic and is the second most used local anesthetic for dentistry in the USA since its introduction in 2000. It is popular due to its rapid onset and long duration with a

low risk of allergy risk despite its ester side chain attached to the thiophene ring.

Bupivacaine

Bupivacaine was the first long-acting amide local anesthetic. Chemical structure makes bupivacaine significantly more hydrophobic than mepivacaine and lidocaine, slower in onset but of longer duration. Bupivacaine is highly protein bound, which is consistent with long duration and potential for cardiotoxicity. Indeed, the cardiotoxicity of bupivacaine prompted the development of ropivacaine and L-bupivacaine. Bupivacaine is popular for use in a wide array of applications, including infiltration (0.25%), peripheral nerve blocks (0.25–0.5%), and spinal (0.5 and 0.75%) and epidural (0.125 and 0.5%) anesthesia. Because of systemic toxicity, it is not used for IV regional anesthesia.

Bupivacaine has a lower therapeutic index, concerning cardiovascular toxicity compared to lidocaine. Bupivacaine is more slowly absorbed into plasma than lidocaine and produces plasma peak concentrations that are approximately 40% lower.

Clinically used concentrations of bupivacaine vary from 0.05% (epidural continuous infusions for labor analgesia and acute pain management) to 0.5% (spinal anesthesia and peripheral nerve blocks). Peripheral nerve blocks provide sensory block for 4–12 h, sometimes up to 24 h.

The 0.75% concentration is specifically contraindicated for obstetric epidural anesthesia due to concerns about cardiotoxicity. Contemporary epidural anesthesia incorporates the use of multihole catheters, test dosing regimens, incremental dosing, and low concentrations of local anesthetic via continuous infusion.

Levobupivacaine

Levobupivacaine is the levorotatory enantiomer of bupivacaine. Commercial bupivacaine is a racemic mixture of both enantiomers (R and S). Levobupivacaine is approximately equivalent to its racemic mixture for its use in regional anesthesia. Cardiac toxicity and CNS studies in animals and healthy volunteers indicated that levobupivacaine is approximately 35% less cardiotoxic compared to racemic bupivacaine [14, 15]. Levobupivacaine is used in the same concentrations, doses, and applications as racemic bupivacaine.

Ropivacaine

Ropivacaine is derived from mepivacaine. Ropivacaine is a long-acting amide local anesthetic which is supplied commercially like levobupivacaine as a single enantiomer. It is available as 0.2, 0.5, 0.75, and 1% solution.

This drug was specifically designed and formulated to minimize cardiotoxicity [16, 17]. At higher concentration (anesthetic), its potency is equivalent to that of bupivacaine [18]. At lower concentration (analgesic), ropivacaine was shown to be 40% less potent than bupivacaine [19]. The clinical experience for peripheral blocks shows that at equivalent doses, ropivacaine and bupivacaine produce similar onset and quality of block, but it can be stated that bupivacaine has a significantly longer duration. Ropivacaine is primarily used in epidural anesthesia/analgesia and peripheral nerve block applications. Ropivacaine appears to be approximately 40% less cardiotoxic as compared to racemic bupivacaine in animal models [16]. Ropivacaine produces vasoconstriction at clinically used concentrations for peripheral nerve blocks explaining the little advantage of adding epinephrine to additionally prolong peripheral nerve block or epidural analgesia [20].

Adjuvants

In the last 20 years, a number of randomized controlled trials and meta-analyses have examined the pros and cons of the use of various individual adjuvants thought to potentially enhance local anesthetic peripheral nerve or neuraxial blockade [21]. Moreover, recent animal safety and clinical observational work have introduced the concept of "multimodal perineural analgesia," whereby multiple agents with differing mechanisms of action are used with the goal of providing perineural analgesia while avoiding exposure to high and potentially toxic levels of individual agents [22].

Sodium Bicarbonate

Theoretically, sodium bicarbonate could fasten the onset time. However, results were not convincing, and actually, the practice of mixing sodium bicarbonate with local anesthetics is rarely used.

Hyaluronidase

It is used as adjuvant to local anesthetics to breakdown connective tissue in the extracellular matrix and thereby increase drug dispersion through tissue. Except for peribulbar block (sub-Tenon's block), it has been abandoned. Allergic reactions have also been described in this setting.

Vasoconstrictors

Adding epinephrine leads to vasoconstriction and thereby local blood flow and drug clearance are decreased. This prolongs block duration and decreases local anesthetic plasma concentration following spinal, epidural, and peripheral nerve blocks [23]. Lower peak plasma concentration decreases the risk for toxicity. However, epinephrine does not provide protection if accidental intravascular local anesthetic injection occurs [24].

Alpha-2-Adrenergic Agonists

Alpha-2-adrenergic agonists like clonidine and dexmedetomidine are analgesic drugs in their own right and have been shown to inhibit both C fibers and A fibers and to modestly inhibit local anesthetic clearance [25, 26]. When added to local anesthetics, clonidine prolongs sensory block during peripheral, central neuraxial, and intravenous regional anesthesia to a degree comparable to that produced by epinephrine. However, unlike epinephrine, clonidine does not prolong motor block when administered orally, as well as when added to the intrathecal local anesthetic [27]. Significant side effects have been described with the use of clonidine in the adult population, including arterial hypotension, orthostatic hypotension, bradycardia, and sedation [28].

Dexmedetomidine has been used since 2004 as an adjunct to peripheral or neuraxial analgesia. Multiple randomized controlled trials and meta-analyses have been conducted to examine its effectiveness as a peripheral nerve block additive. Abdallah et al. recently published a meta-analysis that examined four studies of dexmedetomidine as an additive for brachial plexus blocks [29]. This analysis found that dexmedetomidine significantly prolonged mean motor block by 268 min and time to first analgesic by 345 min. However, the mean sensory block prolongation of 284 min was not statistically significant. Two more recent studies have shown that the addition of dexmedetomidine to bupivacaine supraclavicular blocks and ropivacaine interscalene blocks prolonged the duration of the blocks by approximately 8 h [30] and 4 h, respectively [31].

The bulk of published data supports the efficacy of dexmedetomidine for peripheral nerve block prolongation of approximately 200 min at doses around 1 μg/kg, and it appears to be a viable option as an additive to ropivacaine or bupivacaine. Attention needs to be paid to the potential for bradycardia and hypotension with this medication.

A recent meta-analysis has shown that, compared with the control treatment, epidural dexmedetomidine administration prolonged the duration of analgesia, reduced the time to sensory block, decreased the requirement for rescue analgesia, and achieved a significantly higher sedation score [32].

Opioids

When added to short-duration local anesthetics used for spinal anesthesia, short-acting opioids (fentanyl and sufentanil) prolong and intensify sensory block without prolonging motor block or time to void, which is particularly advantageous for ambulatory spinal anesthesia [33]. However, postanesthesia nausea and vomiting and itching

can be a problem [34]. When added to local anesthetics or peripheral nerve block, fentanyl has also been shown to prolong sensory block, but at the expense for significantly slowing onset in some studies [35].

When added to intrathecal local anesthetics, the peak plasma concentrations for sufentanil occur between 20 and 30 min and are greater than what is necessary for postoperative analgesia [17]. This explains the many reports of "early" respiratory depression in mothers [18] and fetal heart rate abnormalities in infants when sufentanil is added to intrathecal local anesthetics for labor analgesia or Cesarean section [36].

Perineural buprenorphine has consistently shown the ability to prolong peripheral nerve blocks with no reported increase in side effects or clinical toxicity and may be considered a useful adjuvant for block prolongation. It should be noted, however, that in studies of isolated rat primary sensory neurons, high-concentration buprenorphine exposure for 24 h results in significant cell death [37]. Further laboratory analysis of neuronal exposure to clinically relevant concentrations of buprenorphine in isolation and in combination with local anesthetics and other perineural analgesic adjuvants is warranted.

Dexamethasone

Recent studies have shown safety and efficacy of dexamethasone as an adjuvant for peripheral nerve blockade. Dexamethasone prolongs both sensory blockade and motor blockade, with the latter somewhat limiting its clinical applications in the outpatient setting or when early rehabilitation programs are implemented.

Dexamethasone prolongs brachial plexus block with both intermediate (168–343 min)- and long-acting local anesthetics (730–1306 min). There is conflicting information regarding dosing, given certain randomized controlled trials describe equivalence when utilizing high doses of systemic and perineural administration of dexamethasone; however, low doses of perineural dexamethasone (1–2 mg) appear to prolong nerve block duration compared to equivalent or higher doses of IV dexamethasone (4 mg). Further studies need to look at the efficacy of low perineural doses of dexamethasone to determine if less may be preferable to minimize toxicity and systemic effects. Supra-clinical doses of dexamethasone have demonstrated neurotoxicity in in vitro animal models; however, recent in vivo animal safety models show no adverse event levels and potential neuroprotection and antihyperalgesic effects with clinically relevant dosing [38].

Depot Local Anesthetic Preparations

Depot preparations of local anesthetics are being investigated because they might allow prolong the action of local anesthetics to the point of decreasing the need for nerve catheters and pumps.

Gels, polymer microspheres, liposomes, and oil-water emulsions have been studied in animal models to produce long-acting anesthetic blocks [39]. The most studied formulation is liposome-encapsulated bupivacaine. To date, clinical evidence shows promising results for total knee arthroplasty, bunionectomy, and hemorrhoidectomy.

In a recent meta-analysis, liposomal bupivacaine infiltration has been shown to provide similar postoperative pain relief to femoral nerve block following total knee arthroplasty. In addition, liposomal bupivacaine infiltration could significantly reduce the consumption of morphine equivalents compared to femoral nerve block without an increased risk of adverse events [40].

Complications of Regional Anesthesia

Introduction

Overall incidence of neuropathy after peripheral nerve block varies from 0 to >5%. Studies which used closed claims databases ranked neuropathy at the second place, with 16% of all claims [41].

In a prospective French study, incidence of major neurologic adverse reactions was estimated at 3.5/10,000 [42]. Peripheral nerve damages following either spinal anesthesia or peripheral nerve blockades represented >50% of severe adverse reactions in this investigation.

Permanent injuries after regional anesthesia are rare [43–45]. Most surveys with large cohorts are retrospective [46, 47] or related to closed claims analysis [48, 49]. Few studies are prospective but focus on specific adverse reactions inducing limitation in their interpretation [42, 50, 51].

The largest recent clinical study was a voluntary reporting model used in France [42]. Data of 158,083 different blocks from 487 anesthesiologists were collected and analyzed. The incidence of serious complications such as central or peripheral nerve injury, seizure, death, etc. was described as 3.5/10,000 blocks. The risk of deaths was shown to be 1/400,000 regional blocks. All but one occurred during spinal anesthesia.

It can be concluded that the incidence of severe complications of regional anesthesia is similar to the one observed after general anesthesia.

Systemic Toxicity

Systemic toxicity is a significant and potentially dangerous problem [52]. Beside a local toxicity, an increase of the local anesthetic plasma concentration may lead to systemic toxicity, mainly neurologic and cardiovascular ones. Such an increase in local anesthetic plasmatic concentration may be related to inadvertent intravascular injection with a consecutive sudden plasmatic peak of concentration. The most frequent cause of systemic toxicity is related to a high and rapid resorption of local anesthetics through perinervous vessels. Toxicity occurs first in the CNS and then in the cardiovascular system (Fig. 5.3).

Toxicity

The incidence of seizures varies between 0.2 and 1/1000 cases and according to the anesthetic regional procedure [53, 54]. The clinical manifestation largely depends on the velocity of plasma concentration increment: a slow increase shows clear and reproducible series of typical CNS signs and symptoms. A rapid increase leads to generalized seizures as first clinical manifestation.

Sedatives and hypnotics such as propofol, benzodiazepines, and barbiturates raise seizure threshold and help protecting the CNS [55, 56].

The therapeutic to CNS toxicity ratio is for all local anesthetics, the same indicating that none of them are more or less propense to cause seizures.

The prevention and the treatment of CNS toxicity should be done according to published recommendations [57, 58].

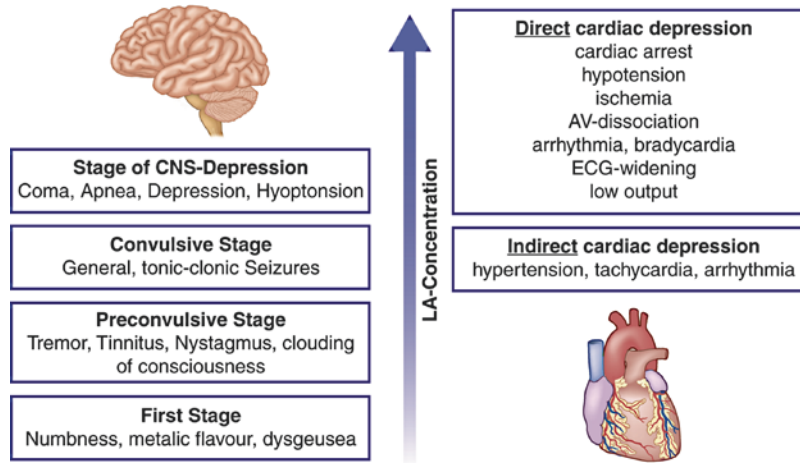

Fig. 5.3 Signs and symptoms of local anesthetic toxicity

Cardiac Toxicity

Estimated incidence of cardiac arrest related to local anesthetics varies between 1.8 and 3.1/10,000 cases [53, 59].

High plasma concentration of local anesthetics is needed to cause significant cardiovascular toxicity. This may occur, when the local anesthetic is injected intravenously, but a quick resuscitation is also possible. The therapeutic/cardiotoxic ratio is lower for hydrophobic local anesthetics (bupivacaine) compared to hydrophilic local anesthetics. Hydrophilic local anesthetics dissociate only after a greater amount of time from their binding sites; therefore, Na^+ channels are blocked when the next depolarization arrives. Cardiac toxicity can manifest as either malignant dysrhythmias (ventricular fibrillation), pulseless electrical activity, or asystole [24, 55, 60].

Cardiac toxicity should be prevented [58], but in case of patients experiencing signs or symptoms of local anesthetic systemic toxicity (LAST), treatment should be done according to the ASRA guidelines 2010 [57, 61].

Often, the doses of epinephrine in this setting are higher [24, 55, 60, 62]. Intralipid seems to be effective mainly in case of bupivacaine toxicity. A review about models and mechanisms of local anesthetic cardiac toxicity and a review of clinical presentations of local anesthetic systemic toxicity over the last 30 years have recently been published [63, 64].

Prevention of Toxicity

Toxicity depends on total dose of local anesthetic injected, type of local anesthetic, speed and site of injection, combination with adjuncts, patient's medical history, and concomitant use of other drugs leading to dangerous interactions, particularly with drugs presenting a hepatic metabolism action (hepatic blood flow modification, cytochrome P450 action, etc.). Interactions have been described among local anesthetics and β-blockers, amiodarone, cimetidine, and volatile agents [65–69]. Calculation of the optimal dose taking into account patient's age, pharmacokinetic and pharmacodynamic interactions with concomitant disease, and other drugs could be probably useful [70]. Development of nerve localization by ultrasonographic technique is thought to help reaching such objectives by limiting the volume of local anesthetic needed to block nerves [71]. However, clinical practice has shown that such a technique cannot always prevent intravascular injection or quick reabsorption [72].

A summary of strategies of prevention of local anesthetic systemic toxicity (LAST) has been recently published [58].

Local Tissue Toxicity

Nerve Injury/Transient Neurologic Syndrome

Direct nerve injury (Table 5.4) from local anesthetic is receiving increased scrutiny, particularly with regard to spinal anesthesia [73, 74]. Toxicity can result from either local anesthetics themselves or from additives, preservatives, antiseptics, or the pH of the formulations. The mechanism of local anesthetic-induced neurotoxicity is multifactorial [75, 76]. Direct nerve injury is evident when isolated nerves are exposed to high concentration of local anesthetics, particularly lidocaine and tetracaine. Local anesthetics also change the biologic milieu surrounding neurons, including localized changes in prostaglan-

Table 5.4 Classification of nerve injuries

Seddon	
Neurapraxia (*Sunderland 1*)	Myelin damage, conduction block
Axonotmesis (*Sunderland 2*)	Loss of axonal continuity, endoneurium intact, no conduction
Neurotmesis (*Sunderland 3*)	Loss of axonal and endoneurial continuity, perineurium intact, no conduction
(*Sunderland 4*)	Loss of axonal, endoneurial and perineurial continuity, epineurium intact, no conduction
(*Sunderland 5*)	Entire nerve trunk separated; no conduction

Based on data from Seddon H, Three types of nerve injury. Brain 1943;66:236–88; Sunderland S: A classification of peripheral nerve injuries producing loss of function. Brain 1951;74:491–516; and Lundborg G. Nerve injury and repair. Churchill Livingstone; 1988

din production, ionic permeability, and neural blood flow.

Compared with bupivacaine, lidocaine has a significantly greater potential for direct neurotoxicity, particularly when isolated nerves are exposed to high concentrations of lidocaine over long periods of time. Hyperbaric 5% lidocaine and tetracaine have been associated with cauda equina syndrome after continuous spinal anesthesia. In these cases, spinal microcatheters were used to administer supernormal doses (up to 300 mg) of hyperbaric 5% lidocaine. Because spinal microcatheters (25–32 gauge) greatly limit the speed of drug administration, high doses of local anesthetics presumably pooled near the catheter tip. As a result of the lordotic lumbar spine curvature, higher concentration of lidocaine remained in the lumbosacral cistern [75, 76].

Single-shot spinal anesthesia can cause transient pain, known as transient neurologic syndrome (TNS), which manifests as back and posterior leg discomfort with radicular symptoms lasting 1–3 days after spinal anesthesia. The etiology of TNS is unclear, but some have speculated that this syndrome represents a form of neurotoxicity. Transient neurologic symptoms occur more frequently with lidocaine than bupivacaine, which may relate to lidocaine's greater neurotoxicity in isolated nerve preparations [49, 77–80]. Additionally, several risk factors (lidocaine, lithotomy position, outpatient surgery, arthroscopic knee surgery, and obesity) for developing TNS have been identified [77, 78].

Needle Trauma

Recent ultrasonographic data have shown that injections between epineurium and perineurium did not produce significant neural injury [81]. If injection pressure is low (less than 12 psi), intraneural injection does not necessarily result in permanent injury but can lead to severe injury if pressures are high [82].

Studies over the last years have demonstrated that the correlation between needle-nerve proximity and the current necessary to elicit a motor response is poor and not always reliable, despite the high success rate of neurostimulation and its low complication rate [83, 84]. Moreover, also eliciting paresthesia has surprisingly poor correlation with nerve proximity [85, 86]. Case reports of intraneural, intravasal, and other complications despite the use of ultrasound have shown that also this promising technique does not guarantee a complete visualization of the targeted nerve to avoid further complications [87]. The best way to avoid needle-induced nerve trauma is to avoid long-bevel needle and perpendicular needle approaches to the nerve.

Clinical symptomatology of perimedullar complication following central nervous block is variable. Spinal cord injury can occur even when a patient did not complain of any paresthesia during puncture [88, 89]. Different risk factors have been identified to explain the occurrence of this complication [73]. Epidural hematoma can cause paraplegia following neuraxial anesthesia in patients concomitantly anticoagulated with low-molecular-weight heparin. Other causes of neural injury include positioning injuries, surgical trauma, and injuries related to the use of a limb tourniquet.

Guidelines on management of such complications following both central and peripheral nerve blocks have been published in 2010 by the American Society of Regional Anesthesia [73]. Decision-making algorithms have been proposed to help the clinician in case of neuropathy occurrence [76, 90] (Fig. 5.4).

Myotoxicity

Skeletal muscle toxicity is a rare and uncommon side effect of local anesthetic drugs. Intramuscular injections of these agents regularly result in reversible myonecrosis [91]. The extent of muscle damage is dose dependent and worsens with serial or continuous administration. This problem is probably underestimated as incidence of symptomatic clinical forms is unknown. Experimental studies have concluded that all LA cause muscular damages with concentration use in daily practice. The extent of such damage depends on pharmacologic properties of each local anesthetic, dose injected, and site of injection [92].

Animal studies in pigs showed lower mean damage score in muscles exposed to ropivacaine

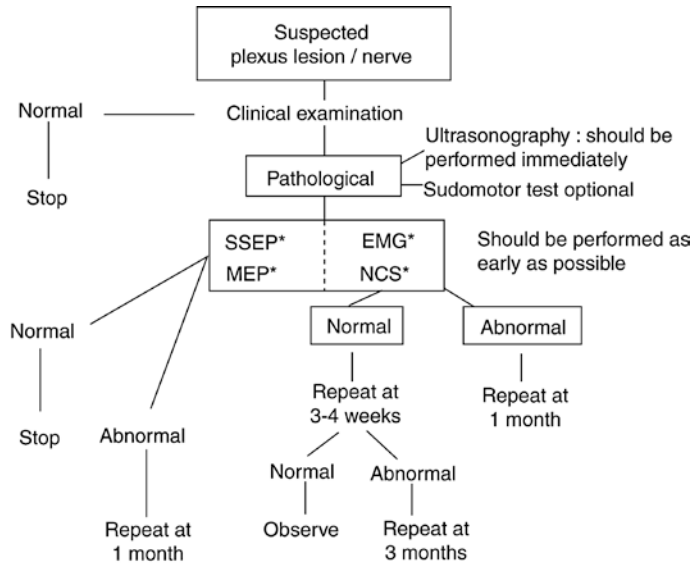

Fig. 5.4 Algorithm recommended to be performed in case of suspected plexus/nerve lesion

compared to exposure to bupivacaine [93, 94]. Stereospecificity of the drug seems also to play an important role in Ca^{2+} metabolism, which has been shown to be important in myotoxicity [95]. First reports of muscular dysfunction were related to retrobulbar injection of local anesthetics.

Bupivacaine seems to be the most toxic local anesthetic. Phenomena of apoptosis have been described only with bupivacaine but not with other LA [94, 96]. Interactions with the Ca^{2+} metabolism seem to be a key pathway and explain most damage [95, 97]. Also, changes in the mitochondrial metabolism induced by local anesthetics have been reported [96, 98, 99]. These effects are less pronounced with ropivacaine, a less lipophilic local anesthetic, compared with bupivacaine on heart cell preparation [100], but this was not shown in rat psoas muscle [101]. A recent study has concluded that mitochondrial bioenergetic alterations with bupivacaine were more severe in young rats compared to adults [102].

Chondrotoxicity

Complications from the use of pain pumps in orthopedic surgery have recently received considerable interest. Human and animal studies have reported on the chondrotoxicity of intra-articular application of bupivacaine [103–105]. Postarthroscopic glenohumeral chondrolysis is a noninfectious entity associated with factors including the use of radiofrequency tumoral instruments and intra-articular pain pumps that administer bupivacaine [106]. Also, the viability of bovine articular chondrocytes after exposure to corticosteroids alone or with lidocaine in a simulated inflammatory environment was assessed. The results showed a dose-dependent and time-dependent decrease in chondrocyte viability after exposure to methylprednisolone. The combination with lidocaine was toxic, with virtually no cells surviving the treatment [107]. Continuous 0.5% bupivacaine exposure was shown to have a clear detrimental effect on chondrocytes in an in vitro model [108]. There is a growing amount of evidence that intra-articular administration of bupivacaine is chondrotoxic, especially at a higher concentration and with a prolonged exposure.

Bupivacaine (0.5%), ropivacaine (0.75%), and mepivacaine (2%) have been shown to be chondrotoxic in vitro in a time-dependent, concentration-dependent, and drug-dependent manner [109]. However, exposure to concentrations up to 0.25% of bupivacaine, 0.5% of ropi-

vacaine, and 0.5% of mepivacaine did not reveal significant chondrotoxicity in flow cytometry. In the same study, cellular death rates were higher in osteoarthritic compared with intact cartilage after local anesthetic treatment. More studies are needed to clarify this issue.

Allergy

Allergic reactions may occur from preservatives added to some local anesthetics (sulfites and methylparaben). Actual allergic reactions to local anesthetics are quite rare, but are more common with ester local anesthetics compared to amides [110]. This is likely due to the breakdown products of ester local anesthetics, such as PABA. There are only a few convincing reports of allergic reactions to preservative-free amide local anesthetics.

If there is a history suggestive of true allergy, it may be worthwhile to perform allergy testing to preservative-free local anesthetics. Measurement of plasma esterase, which is increased in the event of "true" allergy, is useful. Skin testing is often performed to prospectively identify patients with local anesthetic allergy [111].

Bleeding Complications

This issue deals mainly with neuraxial blocks. Epidural (1:150,000 cases) or intrathecal (1:200,000 cases) hematomas can cause devastating neurologic injury. The increased use of antithrombotic prophylaxis has increased this risk after epidural/spinal anesthesia to 1:1000–1:10,000. In 2010 the ASRA has reviewed the risks attendant to performance of regional blocks in the anticoagulated patient and refreshed its guidelines [112, 113] which are also to be found in their website (www.asra.com). Patients may develop sensory changes, progressive weakness, and/or back pain. Confirmatory diagnosis with neuraxial imaging (CT and MRI) must be obtained in conjunction with immediate neurosurgical consultation. If more than 8 h pass between symptom onset and decompression, the likelihood of a full or partial recovery decreases dramatically.

Iatrogenic Coagulopathy

In fully anticoagulated patients (heparin and coumadin), epidural and spinal anesthesia should be avoided unless clear benefit outweighs the added risks.

As mentioned above, the ASRA has published guidelines for regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy [112].

Infection

Infection is a rare complication in regional anesthesia. Risk factors are indwelling catheters left in place for more than 5 days, immunocompromised patients, catheters in trauma patients, and lack of perioperative antibiotics [43].

Peripheral Nerve Blocks

Single-shot peripheral nerve blocks have a low risk of infection. The risk of colonization and infection increases when indwelling catheters are used. Despite the high colonization rate (70%, primarily *Staphylococcus epidermidis*), clinical evidence of infection is uncommon: less than 3%.

Central Neuraxial Blocks

Single-shot spinal and epidural anesthesia have a low risk of infection, but this risk seems to be higher than for peripheral nerve blocks. The incidence of meningitis after spinal anesthesia is estimated at less than 1:40,000; the risk of abscess after epidural anesthesia is less than 1:10,000 (Lit 2). Risk factors are the use of indwelling catheters and bacteremia [114].

Clinical Pearls

- Nerve-blocking potency of local anesthetics increases with increasing molecular weight and lipid solubility [115].
- The effectiveness of local anesthetics is influenced by the dose, site of administration, additives, temperature, and pregnancy [115].

- The plasma concentrations of local anesthetics are depending on the injection technique, place of injection, and addition of adjuvants to local anesthetics [115].
- In laboratory experiments, most local anesthetics will only produce cardiovascular toxicity after the blood concentration has exceeded three times that necessary to produce seizures [63].
- True allergic reactions to preservative-free amide-type local anesthetics are rare [110].
- True anaphylaxis is more common with ester local anesthetics that are metabolized directly to PABA than to amide local anesthetics [110].
- Some patients may react to preservatives, such as methylparaben, used in local anesthetics.
- In contrast to other shorter-acting amide local anesthetics, bupivacaine, levobupivacaine, and ropivacaine have a motor-sparing effect; they produce less motor block for a comparable degree of sensory analgesia.
- It is well accepted that lipid solubility usually goes hand in hand with local anesthetic potency. All things being equal, greater lipid solubility is related to increasing length of the aliphatic chain on the amino ring.
- Intraepidurally administered opioids reduce intraoperative requirements for volatile anesthetics significantly more compared to their intravenous administration. This proves site-specific action in the epidural space.
- Exceeding a total dose of 0.25 mg of epinephrine may be associated with cardiac arrhythmias.
- Adding epinephrine to spinal anesthetics will prolong motor blockade and delay the return of bladder function, thus preventing patients from achieving discharge criteria.
- When clonidine is used in combination with opiates, the analgesic effects are additive, but not synergistic. Patients require a smaller total dose of narcotics and have a decreased incidence of oxygen desaturation with equivalent analgesia.
- Generally, the bigger the size of the nerve fibers, the greater the amount of local anesthetic solution required to block conduction. Thus, fibers of small size are blocked sooner than those of larger diameter. The B fibers of the autonomic system constitute an exception of this rule: even though they are myelinated fibers, a minimum concentration of local anesthetic solution is required to produce an effective blockade. This explains why the sympathetic blockade is observed before the onset of sensory or motor blockade.
- The onset time of local anesthetic is influenced by the molecules pK_a (the higher the pK_a, the slower the onset time of the nerve block in a physiologic environment) and diffusibility [115].
- The ability to cross cell membrane depends on the molecular weight and the liposolubility of the molecule.
- The nonionized form of the molecule is more lipid soluble than the ionized one; therefore, it can cross more readily the cell membrane but diffuses less easily.
- The duration of the action of local anesthetic solutions depends on the protein binding as well as the clearance from the injection site.
- The closer the pK_a of local anesthetic is to physiologic pH, the shorter the onset time of the nerve block [115].
- Increasing the lipophilicity of local anesthetic increases its potency and toxicity, whereas protein binding is proportional to the duration of action of the local anesthetic.
- Sensory-motor differentiation is based on the different sizes and myelinization of the nerve fibers involved in pain conduction (Aδ and C) as compared to those involved in motor function (Aα).
- Postoperative maintenance is best performed with low concentration of a long-acting agent, like 0.2% ropivacaine and 0.125–0.2% levobupivacaine.
- Local toxicity with neurotoxicity primarily occurs in cases of intraneural injection rather than normal applications of clinically relevant concentrations of local anesthetics [116].
- To decrease the risk of nerve injury, utmost care should be taken during nerve localization; excessively high concentrations of local anesthetic and high injection pressures should be avoided [116].

- The larger the fascicle, the greater is the risk of accidental intraneural injection because large fascicles are easily speared by the needle.
- Injections into epineurium or perineural tissue do not result in significant injection resistance.
- When injection is difficult (injection pressures >20 psi), the injection should be stopped because of the risk of intraneural needle position [116].
- It is suggested that nerve stimulation with current intensity of 0.2–0.5 mA (0.1 ms) indicates close needle-nerve placement [117].
- Stimulation with current intensity of ≤0.2 mA may be associated with intraneural needle placement.
- Motor response to nerve stimulation may be absent even when the needle is inserted intraneurally [81].

Review Questions

1. Which of the following statements is correct regarding the perioperative use of intravenous lidocaine infusions?
   (a) They decrease pain beyond the duration of the infusion.
   (b) They have no effect on the duration of hospitalization.
   (c) Delay gastrointestinal motility.
   (d) None of the above.
2. Which of the following is true regarding local anesthetics?
   (a) Block voltage-gated sodium channels, potassium channels, and calcium channels.
   (b) Have anti-inflammatory properties.
   (c) There is in vitro evidence that they may have antimetastatic properties.
   (d) All of the above.
3. Adjuvants commonly used to enhance local anesthetic peripheral or neuraxial blockade include all of the following except:
   (a) Alpha-2-adrenergic agonists
   (b) Opioids
   (c) Dexamethasone
   (d) Neostigmine
4. Amide local anesthetics include all of the following except:
   (a) Lidocaine
   (b) Procaine
   (c) Ropivacaine
   (d) Prilocaine

Answers

1. a
2. d
3. d
4. b

References

1. van der Wal SE, van den Heuvel SA, Radema SA, van Berkum BF, et al. The in vitro mechanisms and in vivo efficacy of intravenous lidocaine on the neuroinflammatory response in acute and chronic pain. Eur J Pain. 2016;20(5):655–74.
2. Hollmann MW, Durieux ME. Local anesthetics and the inflammatory response: a new therapeutic indication? Anesthesiology. 2000;93(3):858–75.
3. Piegeler T, Votta-Velis EG, Liu G, Place AT, Schwartz DE, Beck-Schimmer B, Minshall RD, Borgeat A. Antimetastatic potential of amide-linked local anesthetics: inhibition of lung adenocarcinoma cell migration and inflammatory Src signaling independent of sodium channel blockade. Anesthesiology. 2012;117(3):548–59.
4. Nguyen HM, Goldin AL. Sodium channel carboxyl-terminal residue regulates fast inactivation. J Biol Chem. 2010;285:9077–89.
5. Goldberg YP, MacFarlane J, MacDonald ML, Thompson J, Dube MP, Mattice M, et al. Loss-of-function mutations in the Nav1.7 gene underlie congenital indifference to pain in multiple human populations. Clin Genet. 2007;71:311–9.
6. Yarov-Yarovoy V, Brown J, Sharp EM, Clare JJ, Scheuer T, Catterall WA. Molecular determinants of voltage-dependent gating and binding of pore-blocking drugs in transmembrane segment IIIS6 of the Na(+) channel alpha subunit. J Biol Chem. 2001;276:20–7.
7. Huang JH, Thalhammer JG, Raymond SA, Strichartz GR. Susceptibility to lidocaine of impulses in different somatosensory afferent fibers of rat sciatic nerve. J Pharmacol Exp Ther. 1997;282:802–11.
8. Ilfeld BM, Moeller LK, Mariano ER, Loland VJ, Stevens-Lapsley JE, Fleisher AS, et al. Continuous peripheral nerve blocks: is local anesthetic dose the only factor, or do concentration and volume influence infusion effects as well? Anesthesiology. 2010;112:347–54.

9. Ashchi M, Wiedemann HP, James KB. Cardiac complication from use of cocaine and phenylephrine in nasal septoplasty. Arch Otolaryngol Head Neck Surg. 1995;121:681–4.
10. Le Truong HH, Girard M, Drolet P, Grenier Y, Boucher C, Bergeron L. Spinal anesthesia: a comparison of procaine and lidocaine. Can J Anaesth. 2001;48:470–3.
11. Hodgson PS, Neal JM, Pollock JE, Liu SS. The neurotoxicity of drugs given intrathecally (spinal). Anesth Analg. 1999;88:797–809.
12. Duque S, Fernandez L. Delayed-type hypersensitivity to amide local anesthetics. Allergol Immunopathol (Madr). 2004;32:233–4.
13. Meffin P, Long GJ, Thomas J. Clearance and metabolism of mepivacaine in the human neonate. Clin Pharmacol Ther. 1973;14:218–25.
14. Gristwood RW. Cardiac and CNS toxicity of levobupivacaine: strengths of evidence for advantage over bupivacaine. Drug Saf. 2002;25:153–63.
15. Mather LE, Chang DH. Cardiotoxicity with modern local anaesthetics: is there a safer choice? Drugs. 2001;61:333–42.
16. Casati A, Putzu M. Bupivacaine, levobupivacaine and ropivacaine: are they clinically different? Best Pract Res Clin Anaesthesiol. 2005;19:247–68.
17. Zink W, Graf BM. Benefit-risk assessment of ropivacaine in the management of postoperative pain. Drug Saf. 2004;27:1093–114.
18. Fanelli G, Casati A, Beccaria P, Aldegheri G, Berti M, Tarantino F, et al. A double-blind comparison of ropivacaine, bupivacaine, and mepivacaine during sciatic and femoral nerve blockade. Anesth Analg. 1998;87:597–600.
19. Camorcia M, Capogna G, Columb MO. Minimum local analgesic doses of ropivacaine, levobupivacaine, and bupivacaine for intrathecal labor analgesia. Anesthesiology. 2005;102:646–50.
20. Weber A, Fournier R, Van Gessel E, Riand N, Gamulin Z. Epinephrine does not prolong the analgesia of 20 mL ropivacaine 0.5% or 0.2% in a femoral three-in-one block. Anesth Analg. 2001;93:1327–31.
21. Kirksey MA, Haskins SC, Cheng J, Liu SS. Local anesthetic peripheral nerve block adjuvants for prolongation of analgesia: a systematic qualitative review. PLoS One. 2015;10(9):e0137312.
22. Williams BA, Butt MT, Zeller JR, Coffee S, Pippi MA. Multimodal perineural analgesia with combined bupivacaine-clonidine-buprenorphine-dexamethasone: safe in vivo and chemically compatible in solution. Pain Med. 2015;16(1):186–98.
23. Buckenmaier CC III, Bleckner LL. Anaesthetic agents for advanced regional anaesthesia: a North American perspective. Drugs. 2005;65:745–59.
24. Bernards CM, Carpenter RL, Kenter ME, Brown DL, Rupp SM, Thompson GE. Effect of epinephrine on central nervous system and cardiovascular system toxicity of bupivacaine in pigs. Anesthesiology. 1989;71:711–7.
25. Butterworth JFT, Strichartz GR. The alpha 2-adrenergic agonists clonidine and guanfacine produce tonic and phasic block of conduction in rat sciatic nerve fibers. Anesth Analg. 1993;76:295–301.
26. Kopacz DJ, Bernards CM. Effect of clonidine on lidocaine clearance in vivo: a microdialysis study in humans. Anesthesiology. 2001;95(6):1371.
27. Ota K, Namiki A, Iwasaki H, Takahashi I. Dose-related prolongation of tetracaine spinal anesthesia by oral clonidine in humans. Anesth Analg. 1994;79:1121–5.
28. Pöpping DM, Elia N, Marret E, Wenk M, Tramèr MR. Clonidine as an adjuvant to local anesthetics for peripheral nerve and plexus blocks: a meta-analysis of randomized trials. Anesthesiology. 2009;111(2):406–15.
29. Abdallah FW, Brull R. Facilitatory effects of perineural dexmedetomidine on neuraxial and peripheral nerve block: a systematic review and meta-analysis. Br J Anaesth. 2013;110(6):915–25.
30. Agarwal S, Aggarwal R, Gupta P. Dexmedetomidine prolongs the effect of bupivacaine in supraclavicular brachial plexus block. J Anaesthesiol Clin Pharmacol. 2014;30(1):36–40.
31. Fritsch G, Danninger T, Allerberger K, Tsodikov A, Felder TK, Kapeller M, Gerner P, Brummett CM. Dexmedetomidine added to ropivacaine extends the duration of interscalene brachial plexus blocks for elective shoulder surgery when compared with ropivacaine alone: a single-center, prospective, triple-blind, randomized controlled trial. Reg Anesth Pain Med. 2014;39(1):37–47.
32. Zhang X, Wang D, Shi M, Luo Y. Efficacy and safety of dexmedetomidine as an adjuvant in epidural analgesia and anesthesia: a systematic review and meta-analysis of randomized controlled trials. Clin Drug Investig. 2017;37(4):343–54.
33. Liu S, Chiu AA, Carpenter RL, Mulroy MF, Allen HW, Neal JM, et al. Fentanyl prolongs lidocaine spinal anesthesia without prolonging recovery. Anesth Analg. 1995;80:730–4.
34. Mulroy MF, Larkin KL, Siddiqui A. Intrathecal fentanyl-induced pruritus is more severe in combination with procaine than with lidocaine or bupivacaine. Reg Anesth Pain Med. 2001;26:252–6.
35. Nishikawa K, Kanaya N, Nakayama M, Igarashi M, Tsunoda K, Namiki A. Fentanyl improves analgesia but prolongs the onset of axillary brachial plexus block by peripheral mechanism. Anesth Analg. 2000;91:384–7.
36. Van de Velde M, Teunkens A, Hanssens M, Vandermeersch E, Verhaeghe J. Intrathecal sufentanil and fetal heart rate abnormalities: a double-blind, double placebo-controlled trial comparing two forms of combined spinal epidural analgesia with epidural analgesia in labor. Anesth Analg. 2004;98:1153–9.
37. Williams BA, Hough KA, Tsui BY, Ibinson JW, Gold MS, Gebhart GF. Neurotoxicity of adjuvants used in perineural anesthesia and analgesia in comparison with ropivacaine. Reg Anesth Pain Med. 2011;36(3):225–30.

38. An K, Elkassabany NM, Liu J. Dexamethasone as adjuvant to bupivacaine prolongs the duration of thermal antinociception and prevents bupivacaine-induced rebound hyperalgesia via regional mechanism in a mouse sciatic nerve block model. PLoS One. 2015;10(4):e0123459.
39. Grant SA. The Holy Grail: long-acting local anaesthetics and liposomes. Best Pract Res Clin Anaesthesiol. 2002;16:345–52.
40. Ma J, Zhang W, Yao S. Liposomal bupivacaine infiltration versus femoral nerve block for pain control in total knee arthroplasty: a systematic review and meta-analysis. Int J Surg. 2016;36(Pt A):44–55.
41. Cheney FW, Domino KB, Caplan RA, Posner KL. Nerve injury associated with anesthesia: a closed claims analysis. Anesthesiology. 1999;90:1062–9.
42. Auroy Y, Benhamou D, Bargues L, Ecoffey C, Falissard B, Mercier FJ, et al. Major complications of regional anesthesia in France: the SOS Regional Anesthesia Hotline Service. Anesthesiology. 2002;97:1274–80.
43. Capdevila X, Bringuier S, Borgeat A. Infectious risk of continuous peripheral nerve blocks. Anesthesiology. 2009;110:182–8.
44. Popping DM, Zahn PK, Van Aken HK, Dasch B, Boche R, Pogatzki-Zahn EM. Effectiveness and safety of postoperative pain management: a survey of 18 925 consecutive patients between 1998 and 2006 (2nd revision): a database analysis of prospectively raised data. Br J Anaesth. 2008;101:832–40.
45. Neuburger M, Buttner J, Blumenthal S, Breitbarth J, Borgeat A. Inflammation and infection complications of 2285 perineural catheters: a prospective study. Acta Anaesthesiol Scand. 2007;51:108–14.
46. Horlocker TT. Complications of spinal and epidural anesthesia. Anesthesiol Clin North America. 2000;18:461–85.
47. Horlocker TT, McGregor DG, Matsushige DK, Schroeder DR, Besse JA. A retrospective review of 4767 consecutive spinal anesthetics: central nervous system complications. Perioperative Outcomes Group. Anesth Analg. 1997;84:578–84.
48. Lee LA, Posner KL, Domino KB, Caplan RA, Cheney FW. Injuries associated with regional anesthesia in the 1980s and 1990s: a closed claims analysis. Anesthesiology. 2004;101:143–52.
49. Ben-David B. Complications of regional anesthesia: an overview. Anesthesiol Clin North America. 2002;20:665–7. (ix).
50. Capdevila X, Pirat P, Bringuier S, Gaertner E, Singelyn F, Bernard N, et al. Continuous peripheral nerve blocks in hospital wards after orthopedic surgery: a multicenter prospective analysis of the quality of postoperative analgesia and complications in 1,416 patients. Anesthesiology. 2005;103:1035–45.
51. Phillips OC, Ebner H, Nelson AT, Black MH. Neurologic complications following spinal anesthesia with lidocaine: a prospective review of 10,440 cases. Anesthesiology. 1969;30:284–9.
52. Drasner K. Local anesthetic systemic toxicity: a historical perspective. Reg Anesth Pain Med. 2010;35:162–6.
53. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Samii K. Serious complications related to regional anesthesia: results of a prospective survey in France. Anesthesiology. 1997;87:479–86.
54. Brown DL, Ransom DM, Hall JA, Leicht CH, Schroeder DR, Offord KP. Regional anesthesia and local anesthetic-induced systemic toxicity: seizure frequency and accompanying cardiovascular changes. Anesth Analg. 1995;81:321–8.
55. Bernards CM, Carpenter RL, Rupp SM, Brown DL, Morse BV, Morell RC, et al. Effect of midazolam and diazepam premedication on central nervous system and cardiovascular toxicity of bupivacaine in pigs. Anesthesiology. 1989;70:318–23.
56. Mather LE, Copeland SE, Ladd LA. Acute toxicity of local anesthetics: underlying pharmacokinetic and pharmacodynamic concepts. Reg Anesth Pain Med. 2005;30:553–66.
57. Weinberg GL. Treatment of local anesthetic systemic toxicity (LAST). Reg Anesth Pain Med. 2010;35:188–93.
58. Mulroy MF, Hejtmanek MR. Prevention of local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35:177–80.
59. Kopp SL, Horlocker TT, Warner ME, Hebl JR, Vachon CA, Schroeder DR, et al. Cardiac arrest during neuraxial anesthesia: frequency and predisposing factors associated with survival. Anesth Analg. 2005;100:855–65.
60. Kasten GW, Martin ST. Comparison of resuscitation of sheep and dogs after bupivacaine-induced cardiovascular collapse. Anesth Analg. 1986;65:1029–32.
61. Neal JM, Bernards CM, JFt B, Di Gregorio G, Drasner K, Hejtmanek MR, et al. ASRA practice advisory on local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35:152–61.
62. Chadwick HS. Toxicity and resuscitation in lidocaine- or bupivacaine-infused cats. Anesthesiology. 1985;63:385–90.
63. Butterworth JF IV. Models and mechanisms of local anesthetic cardiac toxicity: a review. Reg Anesth Pain Med. 2010;35:167–76.
64. Di Gregorio G, Neal JM, Rosenquist RW, Weinberg GL. Clinical presentation of local anesthetic systemic toxicity: a review of published cases, 1979 to 2009. Reg Anesth Pain Med. 2010;35:181–7.
65. Freysz M, Beal JL, Timour Q, Bertrix L, Faucon G. Systemic toxicity of local anesthetics. Pharmacokinetic and pharmacodynamic factors. Ann Fr Anesth Reanim. 1988;7:181–8.
66. Siegmund JB, Wilson JH, Imhoff TE. Amiodarone interaction with lidocaine. J Cardiovasc Pharmacol. 1993;21:513–5.
67. Kuhnert BR, Zuspan KJ, Kuhnert PM, Syracuse CD, Brashear WT, Brown DE. Lack of influence of cimetidine on bupivacaine levels during parturition. Anesth Analg. 1987;66:986–90.

68. Mather LE, Runciman WB, Carapetis RJ, Ilsley AH, Upton RN. Hepatic and renal clearances of lidocaine in conscious and anesthetized sheep. Anesth Analg. 1986;65:943–9.
69. Bax ND, Tucker GT, Lennard MS, Woods HF. The impairment of lignocaine clearance by propranolol – major contribution from enzyme inhibition. Br J Clin Pharmacol. 1985;19:597–603.
70. Rosenberg PH, Veering BT, Urmey WF. Maximum recommended doses of local anesthetics: a multifactorial concept. Reg Anesth Pain Med. 2004;29:564–75. (discussion 24).
71. O'Donnell BD, Iohom G. An estimation of the minimum effective anesthetic volume of 2% lidocaine in ultrasound-guided axillary brachial plexus block. Anesthesiology. 2009;111:25–9.
72. Baciarello M, Danelli G, Fanelli G. Real-time ultrasound visualization of intravascular injection of local anesthetic during a peripheral nerve block. Reg Anesth Pain Med. 2009;34:278–9.
73. Neal JM, Bernards CM, Hadzic A, Hebl JR, Hogan QH, Horlocker TT, et al. ASRA practice advisory on neurologic complications in Regional Anesthesia and Pain Medicine. Reg Anesth Pain Med. 2008;33:404–15.
74. Sorenson EJ. Neurological injuries associated with regional anesthesia. Reg Anesth Pain Med. 2008;33:442–8.
75. Welch MB, Brummett CM, Welch TD, Tremper KK, Shanks AM, Guglani P, et al. Perioperative peripheral nerve injuries: a retrospective study of 380,680 cases during a 10-year period at a single institution. Anesthesiology. 2009;111:490–7.
76. Faccenda KA, Finucane BT. Complications of regional anaesthesia incidence and prevention. Drug Saf. 2001;24:413–42.
77. Freedman JM, Li DK, Drasner K, Jaskela MC, Larsen B, Wi S. Transient neurologic symptoms after spinal anesthesia: an epidemiologic study of 1,863 patients. Anesthesiology. 1998;89:633–41.
78. Pollock JE. Transient neurologic symptoms: etiology, risk factors, and management. Reg Anesth Pain Med. 2002;27(6):581.
79. Liu SS, McDonald SB. Current issues in spinal anesthesia. Anesthesiology. 2001;94:888–906.
80. Zaric D, Pace NL. Transient neurologic symptoms (TNS) following spinal anaesthesia with lidocaine versus other local anaesthetics. Cochrane Database Syst Rev. 2009;(2):CD003006.
81. Bigeleisen PE. Nerve puncture and apparent intraneural injection during ultrasound-guided axillary block does not invariably result in neurologic injury. Anesthesiology. 2006;105:779–83.
82. Kapur E, Vuckovic I, Dilberovic F, Zaciragic A, Cosovic E, Divanovic KA, et al. Neurologic and histologic outcome after intraneural injections of lidocaine in canine sciatic nerves. Acta Anaesthesiol Scand. 2007;51:101–7.
83. Choyce A, Chan VW, Middleton WJ, Knight PR, Peng P, McCartney CJ. What is the relationship between paresthesia and nerve stimulation for axillary brachial plexus block? Reg Anesth Pain Med. 2001;26:100–4.
84. Perlas A, Niazi A, McCartney C, Chan V, Xu D, Abbas S. The sensitivity of motor response to nerve stimulation and paresthesia for nerve localization as evaluated by ultrasound. Reg Anesth Pain Med. 2006;31:445–50.
85. Urmey WF, Stanton J. Inability to consistently elicit a motor response following sensory paresthesia during interscalene block administration. Anesthesiology. 2002;96:552–4.
86. Bollini CA, Urmey WF, Vascello L, Cacheiro F. Relationship between evoked motor response and sensory paresthesia in interscalene brachial plexus block. Reg Anesth Pain Med. 2003;28:384–8.
87. Liu SS, Ngeow JE, Yadeau JT. Ultrasound-guided regional anesthesia and analgesia: a qualitative systematic review. Reg Anesth Pain Med. 2009;34:47–59.
88. Tripathi M, Nath SS, Gupta RK. Paraplegia after intracord injection during attempted epidural steroid injection in an awake-patient. Anesth Analg. 2005;101:1209–11.
89. Tsui BC, Armstrong K. Can direct spinal cord injury occur without paresthesia? A report of delayed spinal cord injury after epidural placement in an awake patient. Anesth Analg. 2005;101(4):1212.
90. Borgeat A, Aguirre J, Curt A. Case scenario: neurologic complication after continuous interscalene block. Anesthesiology. 2010;112:742–5.
91. Hogan Q, Dotson R, Erickson S, Kettler R, Hogan K. Local anesthetic myotoxicity: a case and review. Anesthesiology. 1994;80:942–7.
92. Zink W, Graf BM. Local anesthetic myotoxicity. Reg Anesth Pain Med. 2004;29:333–40.
93. Zink W, Bohl JR, Hacke N, Sinner B, Martin E, Graf BM. The long term myotoxic effects of bupivacaine and ropivacaine after continuous peripheral nerve blocks. Anesth Analg. 2005;101:548–54.
94. Zink W, Seif C, Bohl JR, Hacke N, Braun PM, Sinner B, et al. The acute myotoxic effects of bupivacaine and ropivacaine after continuous peripheral nerve blockades. Anesth Analg. 2003;97:1173–9.
95. Zink W, Missler G, Sinner B, Martin E, Fink RH, Graf BM. Differential effects of bupivacaine and ropivacaine enantiomers on intracellular Ca2+ regulation in murine skeletal muscle fibers. Anesthesiology. 2005;102:793–8.
96. Irwin W, Fontaine E, Agnolucci L, Penzo D, Betto R, Bortolotto S, et al. Bupivacaine myotoxicity is mediated by mitochondria. J Biol Chem. 2002;277:12221–7.
97. Zink W, Graf BM, Sinner B, Martin E, Fink RH, Kunst G. Differential effects of bupivacaine on intracellular Ca2+ regulation: potential mechanisms of its myotoxicity. Anesthesiology. 2002;97:710–6.
98. Wakata N, Sugimoto H, Iguchi H, Nomoto N, Kinoshita M. Bupivacaine hydrochloride induces muscle fiber necrosis and hydroxyl radical

formation-dimethyl sulphoxide reduces hydroxyl radical formation. Neurochem Res. 2001;26:841–4.
99. Nouette-Gaulain K, Bellance N, Prevost B, Passerieux E, Pertuiset C, Galbes O, et al. Erythropoietin protects against local anesthetic myotoxicity during continuous regional analgesia. Anesthesiology. 2009;110:648–59.
100. Sztark F, Malgat M, Dabadie P, Mazat JP. Comparison of the effects of bupivacaine and ropivacaine on heart cell mitochondrial bioenergetics. Anesthesiology. 1998;88:1340–9.
101. Nouette-Gaulain K, Sirvent P, Canal-Raffin M, Morau D, Malgat M, Molimard M, et al. Effects of intermittent femoral nerve injections of bupivacaine, levobupivacaine, and ropivacaine on mitochondrial energy metabolism and intracellular calcium homeostasis in rat psoas muscle. Anesthesiology. 2007;106:1026–34.
102. Nouette-Gaulain K, Dadure C, Morau D, Pertuiset C, Galbes O, Hayot M, et al. Age-dependent bupivacaine-induced muscle toxicity during continuous peripheral nerve block in rats. Anesthesiology. 2009;111:1120–7.
103. Bailie DS, Ellenbecker TS. Severe chondrolysis after shoulder arthroscopy: a case series. J Shoulder Elbow Surg. 2009;18:742–7.
104. Chu CR, Izzo NJ, Papas NE, Fu FH. In vitro exposure to 0.5% bupivacaine is cytotoxic to bovine articular chondrocytes. Arthroscopy. 2006;22:693–9.
105. Rapley JH, Beavis RC, Barber FA. Glenohumeral chondrolysis after shoulder arthroscopy associated with continuous bupivacaine infusion. Arthroscopy. 2009;25:1367–73.
106. Busfield BT, Romero DM. Pain pump use after shoulder arthroscopy as a cause of glenohumeral chondrolysis. Arthroscopy. 2009;25:647–52.
107. Seshadri V, Coyle CH, Chu CR. Lidocaine potentiates the chondrotoxicity of methylprednisolone. Arthroscopy. 2009;25:337–47.
108. Anz A, Smith MJ, Stoker A, Linville C, Markway H, Branson K, et al. The effect of bupivacaine and morphine in a coculture model of diarthrodial joints. Arthroscopy. 2009;25:225–31.
109. Breu A, Rosenmeier K, Kujat R, Angele P, Zink W. The cytotoxicity of bupivacaine, ropivacaine, and mepivacaine on human chondrocytes and cartilage. Anesth Analg. 2013;117(2):514–22.
110. Amsler E, Flahault A, Mathelier-Fusade P, Aractingi S. Evaluation of re-challenge in patients with suspected lidocaine allergy. Dermatology. 2004;208:109–11.
111. Hein UR, Chantraine-Hess S, Worm M, Zuberbier T, Henz BM. Evaluation of systemic provocation tests in patients with suspected allergic and pseudoallergic drug reactions. Acta Derm Venereol. 1999;79:139–42.
112. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, Kopp SL, Benzon HT, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). Reg Anesth Pain Med. 2010;35:64–101.
113. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK. Executive summary: regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). Reg Anesth Pain Med. 2010;35:102–5.
114. Carp H, Bailey S. The association between meningitis and dural puncture in bacteremic rats. Anesthesiology. 1992;76:739–42.
115. Heavner JE. Local anesthetics. Curr Opin Anaesthesiol. 2007;20:336–42.
116. Hadzic A, Dilberovic F, Shah S, Kulenovic A, Kapur E, Zaciragic A, et al. Combination of intraneural injection and high injection pressure leads to fascicular injury and neurologic deficits in dogs. Reg Anesth Pain Med. 2004;29:417–23.
117. Jankovic D, Wells C. Brachial plexus. In: Jankovic D, Wells C, editors. Regional nerve blocks. Berlin: Blackwell Science; 2001. p. 58–86.

Monitoring and Sedation in Regional Anesthesia

6

James Kim and Jeff Gadsden

Introduction

General anesthesia has become increasingly safe over the last two decades, largely due to improvements in monitoring such as pulse oximetry and capnography [1]. These technologies, which allow for early detection of potentially catastrophic adverse events such as esophageal intubation, have aided in dramatically reducing anesthetic morbidity and mortality since the early 1980s [2, 3].

Regional anesthesia carries its own set of potential complications, principally nerve injury, systemic local anesthetic toxicity, and needle misadventure (e.g., pneumothorax or arterial puncture). In general, the morbidity and mortality related to these adverse events are both less common and less severe than those associated with airway disasters, but catastrophic outcomes still occur [4]. There are a variety of monitors that are utilized during the performance of peripheral nerve block in order to avoid such complications, although their routine use varies greatly. In general, the adoption of consistent, objective monitoring to prevent injury during regional anesthesia has lagged behind monitoring efforts during general anesthesia. This chapter will focus on the basic setup that should be employed during each and every regional anesthetic and the evidence base that supports the use of existing monitors. Since effective and judicious use of sedation is one of the keys to performing safe regional anesthetic techniques, a practical approach to sedation will also be covered.

Basic Setup

One of the principal means of avoiding adverse outcomes is to maintain consistency in safe practice: by using the *same* monitors routinely for *every* regional anesthetic, the likelihood of a physiologic derangement going undetected because a monitor was forgotten is minimized. Regional anesthesia is frequently performed in locations outside the operating room (i.e., the preoperative holding area, the labor room, or the postanesthesia care unit), but the same standards and monitors should be applied.

The use of standard monitors such as pulse oximetry, electrocardiography, and arterial blood pressure measurement are routinely recommended for any type of anesthetic (regional or general). Except in the obstetric population, neuraxial and peripheral nerve blocks are usually performed under some degree of sedation, both

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_6

Fig. 6.1 Basic monitors and equipment required for regional anesthesia procedures. Note monitor displaying electrocardiography, pulse oximetry, and noninvasive blood pressure, as well as immediate availability of wall suction, bag-valve-mask, and emergency drugs

for patient comfort and to raise the threshold for local anesthetic-induced seizures. As such, monitoring of oxygenation and ventilation is critical in order to detect hypoventilation, airway obstruction, and/or hypoxemia from excessive sedation. Pulse oximetry and frequent verbal contact with the patient are often sufficient to ensure adequate gas exchange; however, many centers employ capnography during peripheral nerve blockade in order to have a graphical representation of respiratory rate and guard against apnea.

Supplemental oxygen should also be administered, either by face mask or nasal cannulae. Hypoxia has been shown to potentiate the negative chronotropic and inotropic effects of both lidocaine and bupivacaine, worsening the hemodynamic status during cardiotoxicity [5]. Similarly, hypercapnia and acidosis from hypoventilation serve to increase the free fraction of bupivacaine in the plasma, as well as increase cerebral blood flow, two factors that may contribute synergistically to the development of systemic toxicity and seizures [6].

Electrocardiography (EKG) and blood pressure monitoring are essential in monitoring for early signs of cardiovascular systemic local anesthetic toxicity. Cardiac toxicity from local anesthetics typically begins with myocardial depression followed by an increase in heart rate, blood pressure, and contractility that coincides with the onset of central nervous system excitement. As drug concentration increases, QRS intervals widen, and, particularly in the case of bupivacaine, ventricular arrhythmias such as ventricular tachycardia or fibrillation occur. It is important to note that cardiac manifestations of systemic toxicity can precede the neurologic signs and symptoms, especially in the sedated patient, so vigilance for early changes in heart rate, blood pressure, and EKG morphology is vital [4]. An example of a patient with the basic monitors applied for regional anesthesia is illustrated in Fig. 6.1.

A variety of resuscitation equipment and medication should be immediately available during the performance of all regional blocks in order to facilitate rapid control of the airway, termination of seizures, stabilization of vital signs, and treatment of the cardiotoxic effects of local anesthetic-induced systemic toxicity. This list should include the following:

1. Self-inflating bag-valve-mask (i.e., Ambu® bag)
2. Suction
3. An oxygen source with face mask

4. Endotracheal tube(s), oral, and/or nasal airways
5. Laryngoscope (tested and functioning)
6. Emergency drugs:
   - A "sleep" dose of induction agent (e.g., 20 mL of propofol)
   - Succinylcholine
   - Atropine
   - A vasopressor such as ephedrine or phenylephrine
   - A 500-mL bag of intralipid for treatment of local anesthetic systemic toxicity (this does not necessarily have to be bedside but should be immediately available should the need arise to use it)

Specific Monitors

Besides the standard monitoring devices that are used for every anesthetic, regional anesthesia demands specific techniques and equipment that aid in preventing the three principal complications: nerve injury, local anesthetic systemic toxicity, and needle misadventure. The following section outlines each of the commonly used techniques and monitors.

Aspiration, Fractionation, and Speed of Injection

Aspiration immediately before and periodically during injection of local anesthetics appears intuitively to be a good practice, although there is scant evidence showing a safety advantage. In fact, there are multiple case reports of negative initial aspiration through a nerve block needle, followed by intravascular injection that was detected by a lack of spread of injectate on ultrasound [7, 8]. Similar cases have been reported for epidural catheters, especially the single-hole variety [9]. However, there is little to be lost by aspirating frequently, and it remains a recommended practice.

Slow, fractionated injection serves to reduce the maximum arterial concentration (C_{max}) of local anesthetic as shown by Mather et al. [10]. In a sheep model, prolonging the intravenous (IV) infusion time of 37.5 mg of levobupivacaine from 1 to 3 min reduced the C_{max} by approximately 40%. Constructing a simulation model based on these data, the investigators theorized that dividing a similar dose into six portions, each administered over 30 s, 1 min apart, would result in a reduction in C_{max} of approximately 30%. While a 6-min injection of local anesthetic for a peripheral nerve block may be excessive in most patients, the principle of a slow, fractionated injection is sound and should be considered a standard practice.

A slow injection speed may protect against nerve injury as well. An association with injection pressure and fascicular rupture has been shown in large animals [11], and recent evidence has demonstrated that the speed of injection is directly related to the risk of generating high pressure during femoral nerve blockade [12]. In this study, an injection speed of 10 mL/min carried a small incidence (6%) of pressure >1000 mmHg, a threshold that has been associated with injury in rabbits and dogs. In contrast, speeds of 20 and 30 mL/min were associated with dangerous pressures 35 and 44% of the time, respectively.

Intravenous Markers

Early detection of rising levels of local anesthetic in the plasma is critical to avoiding systemic toxicity. Toxic symptoms can occur acutely (in the first seconds to minutes after injection), as is the case with an accidental intravascular needle/catheter insertion, or subacutely (minutes to hours later), which is due to gradual vascular absorption. Several studies have investigated the utility of premonitory symptoms as a means of early detection. Moore and colleagues found that the sensitivity of a 1 mg/kg IV dose of lidocaine in unpremedicated volunteers was 100% in detecting early neurologic signs (e.g., tinnitus, perioral numbness, metallic taste) [13]. However, when given small doses of midazolam (1.5 mg) and fentanyl (75 μg) prior to injection, the sensitivity dropped to 60%. Similar results have been found

with other local anesthetics including bupivacaine, levobupivacaine, and 2-chloroprocaine [14, 15]. McCartney et al. found that a dose of 60 mg of ropivacaine did act as a reliable IV marker, even when volunteers were premedicated with 0.03 mg/kg of midazolam [16]. However, caution should be exercised when applying these results to clinical practice, as the use of such doses in elderly or frail patients may precipitate toxicity.

Epinephrine is the IV marker of choice for most regional anesthetic injectates. Besides reliably truncating the peak plasma concentration of local anesthetic [17], it also provides reliable and objective criteria by which to assess IV uptake of injectate. Guinard et al. demonstrated a 100% sensitivity and specificity for detecting an increase in heart rate 20 beats per minute or greater or an increase in systolic blood pressure 15 mmHg or greater, following the IV administration of 10–15 μg of epinephrine [18]. In the presence of acute beta-adrenergic blockade, the specificity of the blood pressure criterion dropped to 88%, while the sensitivity remained 100% (the heart rate remained 100% sensitive and specific). However, the sensitivity of the heart rate criterion appears reduced when patients are premedicated with fentanyl and midazolam (but not midazolam alone) [19]. These physiologic criteria may not be valid in the elderly, who are resistant to the effects of catecholamines, those under high neuraxial anesthesia/general anesthesia who may not mount the appropriate response, and laboring parturients, in whom increases in heart rate and blood pressure from labor may be misinterpreted. In this latter group, other strategies are available. A test dose of fentanyl 100 μg has been advocated, with patient sedation constituting a positive intravenous test [20]. Another option is the injection of 3 mL of air via the epidural catheter while listening via the fetal heart monitor over the mother's precordium—if the catheter is placed intravenously, a millwheel murmur will be heard [21].

A third physiologic criterion that is not affected by sedative medications is the T-wave amplitude. In the presence of 10–15 μg of epinephrine, the amplitude of the T-wave will reliably decrease by 25% or more [19]. While theoretically useful, this monitor may be somewhat impractical, as attempting to discern a 1–2-mm T-wave flattening on a single lead while maintaining a needle in a precise location by a nerve may require too much attention of a single practitioner.

Concern has been raised about the use of epinephrine and vasoconstriction of small nutritive blood vessels in the nerve, which could potentially cause ischemia [22]. Epinephrine is known to produce a dose-dependent prolongation of neural blockade, the mechanism of which is partly thought to be due to nerve ischemia [23]. Combination with inherently vasodilating local anesthetics does not reverse this effect; Myers and Heckman demonstrated that the combination of lidocaine 2% and epinephrine 5 μg/mL reduced rat sciatic endoneurial blood flow by 78% [24]. However, more dilute concentrations of epinephrine may be beneficial: Partridge showed that epinephrine 2.5 μg/mL applied to the rat sciatic nerve transiently increased neural blood flow by 20% for several minutes, before returning to baseline, suggesting that at reduced concentrations, the beta-adrenergic effects may predominate [25]. An ideal test dose might therefore be 6 mL of a local anesthetic solution containing 2.5 μg/mL (15 μg) of epinephrine. Our institutional practice is to use this dose (2.5 μg/mL or 1:400,000) in almost every block (an exception might be made for those blocks where miniscule volumes of local anesthetic—3–5 mL—are being used).

Neurostimulation

Electrical nerve stimulation has been used as a means of nerve localization for almost four decades. While there is a lack of evidence showing improved block success or patient safety compared with the paresthesia technique, it remains a commonly used method [26]. However, its sensitivity as a means of neurolocalization has been questioned. Several experiments have found that needle-nerve contact and, in some cases, intraneural needle tip placement still require

current intensities in excess of 0.5–1.0 mA in order to obtain a motor response [27–29]. This contradicts the common belief that "the closer the needle to the nerve, the stronger the twitch." On the other hand, extremely low current thresholds may be associated with intraneural needle tip positioning. Tsai et al. demonstrated in a pig model that, while extraneural current thresholds varied with distance to the nerve, motor responses obtained using currents less than 0.2 mA were always associated with intraneural needle tip positioning [30]. In another pig study, Voelckel et al. performed percutaneous sciatic nerve blocks using two different current intensities and examined the relationship to histologic changes [31]. Those nerves for which currents of 0.3–0.5 mA were accepted showed no signs of injury; however, when the blocks were performed using currents less than 0.2 mA, 50% of nerves showed signs of inflammatory changes. More recently, Bigeleisen et al. provided clinical evidence that a motor response of 0.2 mA or less indicated intraneural needle placement in an ultrasound-guided supraclavicular block model [32]. While neurostimulation may not be a sensitive method of placing needles next to nerves (i.e., high current intensities may still be required if within the nerve), it appears to carry a high specificity for ruling out intraneural placement, using 0.2 mA as a cutoff for safe practice.

Ultrasonography

The use of ultrasound-guided nerve blockade has many apparent benefits, the most obvious being the ability to guide the needle under real time toward the target. Its use may also confer several safety advantages, chief among them being the ability to decrease the amount of local anesthetic used to achieve a successful block. Among the first to demonstrate this were Casati et al., who showed in an up-and-down design that ultrasound guidance was able to reduce the minimum effective anesthetic volume required for a femoral block by 42% [33]. In an era where systemic toxicity from even "clinical doses" of local anesthetics is regularly published [34], this is not an insignificant finding. In addition, not all of the volume reduction benefits are of a systemic nature. Riazi et al. compared the effect of performing ultrasound-guided interscalene blocks using 5 vs. 20 mL of 0.5% ropivacaine on phrenic nerve palsy and found the incidence of diaphragmatic paralysis to be 45 and 100%, respectively, while pain scores and analgesic consumption in the first 24 h were identical [35]. Vandepitte et al. conducted an up-down study on the minimal effective volume required to establish an effective block through an interscalene catheter and found the ED95 to be 7 mL [36]. The shift to using lower volumes of local anesthetic is probably responsible in part for the significant reduction in local anesthetic systemic toxicity (LAST) that occurs with ultrasound-guided peripheral nerve blocks compared with non-ultrasound-guided techniques. In a review of a database of over 25,000 peripheral nerve blocks, Barrington and Kluger reported that ultrasound guidance resulted in a 77% relative risk reduction (OR 0.23, 95% CI 0.088–0.59, $P = 0.002$) in the incidence of LAST compared to techniques not using ultrasound [37]. This is the best evidence that we currently have demonstrating a safety advantage and will be a key factor in establishing ultrasound guidance as a standard of care during regional anesthesia.

Ultrasound may be a useful tool in demonstrating intraneural injection. In some studies, even volumes less than 1 mL can result in obvious nerve swelling on the ultrasound image [38, 39]. This is not a consistent finding however, and other investigations have shown that the ability of anesthesiologists to correctly identify intraneural injection of 0.5 mL of injectate into the sciatic nerve is only 76% sensitive; in other words, anesthesiologists appear to miss one quarter of intraneural injections with volumes of 0.5 mL [40]. This is important, as it has been well established that as little as 0.05 mL injected within a fascicle can result in irreparable axonal destruction [22]. Despite this, there are case series and retrospective studies showing that intraneural injection does not necessarily lead to unavoidable neurologic injury [41–43]. The explanation put forward for this discrepancy is that in most instances

of inadvertent (or deliberate) nerve puncture and/or intraneural injection, the needle passes by or in between fascicles, passing through the loose, epineurial matrix without fascicular trauma. Unfortunately, ultrasound currently lacks the resolution to precisely determine extrafascicular versus intrafascicular needle tip location. Moreover, there are both blood vessels and fine interfascicular connections that exist in the epineurium that are at risk during an intraneural, extrafascicular needle position [44]. This reinforces the decades-old wisdom that maintaining an extraneural needle location is important for patient safety.

Another potentially beneficial aspect of ultrasonography that seems intuitive is the avoidance of vascular or pleural puncture. Just as the rate of carotid puncture during internal jugular cannulation appears to be reduced significantly when using ultrasound guidance [45], one large prospective audit of more than 7000 peripheral nerve blocks showed a significant reduction in the incidence of inadvertent vascular puncture [46]. In a systematic review of four studies, Abrahams et al. found a significant reduction (OR 0.16, 95% CI 0.05–0.47) in the incidence of vascular puncture compared to nerve stimulation-guided blocks [47]. However, another large registry database demonstrated no difference in vascular puncture when using ultrasound [37]. What we do know is that there are numerous reports of vascular and neural impalement despite the use of ultrasound [48, 49], as well as reports of pneumothoraces following ultrasound-guided brachial plexus block [50, 51]. These data suggest that, for the time being, either the technology or the manner in which it is being used is not foolproof. On the other hand, ultrasound has been advocated as a routine procedure to rule out pneumothorax prior to discharging patients home after supraclavicular blockade for ambulatory surgery [52].

Injection Pressure Monitoring

A complementary monitor that may aid in avoiding neural injury is injection pressure monitoring. Hadzic et al. showed that high pressures (>25 psi) at the commencement of intraneural injections in dogs were associated with neurologic deficit and destruction of neural architecture [11]. This is likely due to the rupture of the fascicle after the expansion of these low-compliance spaces. Monitoring of injection pressure has been shown to predict needle-nerve contact and the inability to inject local anesthetic in both interscalene and femoral blocks [53, 54]. In both studies, a blinded observer halted the syringe pump when the opening injection pressure reached 15 psi. The sensitivity of injection pressure monitoring to indicate a needle tip position abutting the nerves was 97% and 90%, respectively, for these two blocks. In contrast, only 75% of subjects had a motor response when the needle tip was indenting the C5 or C6 nerve roots with a current intensity of 0.5 mA, suggesting that injection pressure monitoring may be a superior warning system than nerve stimulation.

Hand feel is often cited as an acquired skill that permits an experienced anesthesiologist to determine a dangerous injection pressure. However, blinded in vitro and meat model studies of what anesthesiologists felt to be appropriate injection pressures do not support this [55, 56]. Therefore, the objective monitoring of injection pressure may be a useful modality. This can be achieved through one of several ways: either through commercially available in-line devices (Fig. 6.2) or by the use of a "compressed air injection technique" (Fig. 6.3) [57]. By filling a 30-mL syringe with liquid and leaving 10 mL of air, a compression of the air component to no more than half its original volume (i.e., 5 mL)

Fig. 6.2 A commercial in-line pressure transducer with graduated markings on the side of a piston, indicating pressure in psi (BSmart™, BBraun Ltd, Bethlehem, PA)

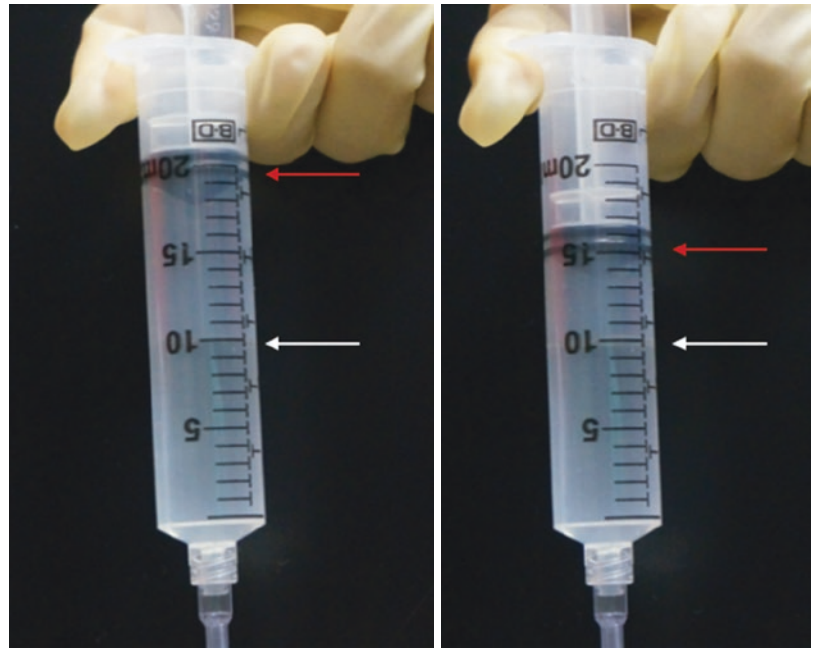

Fig. 6.3 The compressed air technique. Prior to injection (left), the syringe contains 10 mL of local anesthetic and 10 mL of air. After applying pressure to the plunger, the air bubble decreases in volume. A decrease in half of its original size (right), or 5 mL, indicates an increase in pressure within the system of 1 atmosphere or 14.7 psi

will ensure that injection pressure is kept at 20 psi or less. Since this technology is relatively new, evidence has not accumulated showing a safety advantage. However, since it is inexpensive and easy to use, and since the animal data are compelling, the reasons not to use it are few.

Pressure monitoring has other applications as well—a recent study showed that bilateral spread during lumbar plexus block occurred in 60% of patients exposed to injection pressures >20 psi compared with 0% in those with pressures <15 psi [58]. One patient had an epidural block with a T4 sensory level, a situation that could prove dangerous in a vulnerable patient.

Monitors of Consciousness and Cerebral Perfusion

When regional anesthesia is used alone (i.e., not in combination with general anesthesia), mild to moderate sedation is usually desirable for reasons of patient comfort, anxiolysis, and amnesia. While monitoring the level of sedation can be achieved clinically, some have employed processed electroencephalography, such as the bispectral index (BIS), to titrate sedation to a specific depth. This may carry advantages for certain populations at risk for adverse outcomes as a result of oversedation. For example, one study of elderly patients undergoing repair of fractured hips under spinal anesthesia randomized subjects to receive propofol sedation titrated to a BIS of approximately 50 vs. a BIS of 80 or greater [59]. The incidence of postoperative delirium was significantly reduced in the group with the higher BIS target, 19 vs. 40%, suggesting that the use of clear BIS targets to minimize oversedation may aid in avoiding this all-too-common outcome.

Several observations have to be made with respect to monitors of brain activity and their use in regional anesthesia. First, neuraxial anesthesia itself is known to contribute to sedation and decrease the requirements for further anesthetic drugs [60]. This is effected by a combination of decreased afferent neural input to the reticular activating system and a direct effect of local anesthetics in the cerebrospinal fluid [61, 62]. The spinal level of the block may be important: Nishikawa and colleagues demonstrated that a spinal resulting in a sensory block to T4 in unpremedicated patients resulted in a significant decrease from baseline BIS scores, whereas an L3 level did not [63]. Secondly, while propofol

and midazolam both appear to predictably reduce BIS scores, nitrous oxide may not, even though the clinical effect is apparent. For instance, in patients who received epidural anesthesia for lower extremity surgery, increasing the concentration of nitrous oxide in oxygen (33, 50, and 67%) that was administered resulted in steadily decreasing sedation scores as judged by a blinded observer [64]. However, BIS scores remained unchanged throughout, suggesting that BIS may not be an effective monitor of level of consciousness in the presence of nitrous oxide.

Another group of patients that may benefit significantly from central nervous system monitoring during regional anesthesia are those positioned in the beach chair position. Used primarily for procedures on or about the shoulder, this position has been associated with several cases of perioperative ischemic cerebral events [65, 66]. These may be caused by underperfusion to the brain when brachial artery pressure, which is generally used to guide hemodynamic management during these cases, overestimates the cerebral perfusion pressure, as the pressure differential may be as much as 30 mmHg. A contributing factor is the frequent request by the surgeon to lower arterial blood pressure to reduce bleeding into the joint and improve visibility during arthroscopic shoulder surgery. Cerebral oximetry using near-infrared spectroscopy is a method by which cerebral tissue oxygenation can be continuously assessed noninvasively. A study comparing cerebral oxygenation using the beach chair vs. the lateral decubitus position for shoulder surgery showed that 80% of patients in the beach chair position had what were defined as "critical desaturation events," compared with 0% in the lateral decubitus position [67]. This group also experienced a sevenfold increase in the incidence of nausea and vomiting in the recovery room, although all other recovery variables were similar. Interestingly, the BIS scores between groups did not differ at any time, suggesting that oximetry is a more sensitive predictor of nausea and vomiting. The question that arises is whether there might be more subtle neurologic changes in patients at risk (e.g., the elderly or hypertensive patients) that are occurring regularly without obvious signs in the immediate postoperative period. It is not known whether cerebral oximetry can be used to guide treatment in this population; patients were treated promptly in this study when the events were diagnosed and there was no control arm. Also, these patients all received general anesthesia; further research efforts should focus on conducting similar studies using regional anesthesia alone.

Sedation for Regional Anesthesia

Sedation is a continuum ranging from mild anxiolysis to unconsciousness and unresponsiveness (i.e., general anesthesia). There are multiple reasons why sedation during regional anesthesia procedures is advantageous:

1. Sedation alleviates the stress associated with fear of needles, procedural pain, and recall of the nerve block [68, 69].
2. Sedation increases patient satisfaction during regional anesthesia and increases global tolerance of regional blocks [70, 71].
3. Sedation decreases the requirement for opioid analgesics, potentially reducing the risk of opioid-related adverse events such as nausea, vomiting, or respiratory depression [72, 73].
4. Sedation with benzodiazepines or propofol increases the seizure threshold, thereby potentially reducing the risk of central nervous systemic toxicity [74].

A retrospective study of over 42,000 patients who received regional anesthetic procedures with or without sedation demonstrated that sedation significantly reduced the risk of premature termination of the block as well as the risk of primary failure [75]. This may be related to improved operating conditions associated with a calm, immobile patient. Clearly, sedation is not without risk, particularly respiratory and hemodynamic depression, highlighting the need for appropriate monitoring and access to resuscitation drugs and equipment. There is also a risk of sedating a patient to a level of consciousness where

Table 6.1 Commonly used sedative drugs during regional anesthesia

Drug	Commonly used IV dose range for sedation	Onset (min)	Notes
Midazolam	1–4 mg	1–2	Rapid anxiolysis. Associated with anterograde amnesia, especially in higher doses. Synergistic depression of respiratory function with opioids. Minimal residual effect
Fentanyl	25–100 μg	3–5	Some sedation when given alone. Excellent analgesic. Associated with facial pruritis
Alfentanil	250–500 μg	1–2	Short-acting, little residual analgesia after block. May match block procedure duration the best of all opioids
Remifentanil	20–80 μg	0.5–1	Higher cost, very short duration of action
Clonidine	50–100 μg	5–10	Prolongs sensory block, good sedative. Associated with hypotension, prolonged sedation
Dexmedetomidine	50 μg	5–10	Prolongs sensory block, good sedative. Faster offset than clonidine
Ketamine	5–20 mg		Weak sedation, excellent analgesia. Positive effect on hemodynamic stability. Minimal if any dysphoria/ hallucinations in subhypnotic doses
Propofol	10–50 mg	<1	Potent respiratory depressant, must be prepared for apnea. Quick, clean offset with no "hangover." May have to re-dose depending on the length of procedure (i.e., multiple catheters)

disinhibition and unexpected movement could occur during the block, resulting in injury [76].

There are a variety of drugs available for use when sedating patients for regional blocks. In a retrospective review of the German Network for Regional Anesthesia database, 44% of patients received opioid alone, 21% received benzodiazepine alone, and 26% received a combination of the two [75]. The remainder (<10%) received medications such as propofol, alpha-2 agonists, and ketamine. Table 6.1 outlines commonly used drugs for sedation in regional anesthesia and their properties.

Case Study

A 21-year-old college athlete presents for elective arthroscopic rotator cuff repair. He is interested in regional anesthesia in order to avoid a general anesthetic but is concerned about the risk of nerve injury associated with a brachial plexus block, especially since he is an elite athlete. During your preoperative discussion, you explain that the risk of nerve injury is very low but that you will take all of the relevant steps to minimize and prevent complications. The patient is agreeable to a block, but appears quite anxious.

Your Plan Is to Perform a Single-Injection Interscalene Brachial Plexus Block. How Will You Sedate This Patient?

Your primary goal here is anxiolysis, while at the same time providing ideal operating conditions for the block procedure. As such, a short-acting benzodiazepine such as midazolam 1–2 mg is appropriate. Propofol in small doses (e.g., 10–30 mg) is also a good choice; however, care must be taken to avoid provoking apnea. Excessive sedation may also disinhibit the patient, causing him to move during the procedure and putting him at risk for needle trauma. A single-injection interscalene brachial plexus block is associated with minimal discomfort, so the use of opioids is optional. In fact, an "opioid-free" technique may be desired in order to avoid opioid-related adverse events (e.g., nausea or vomiting) in an outpatient procedure on a high-functioning patient.

You Plan to Use Ultrasound Guidance for the Block Placement. Is There Benefit to Using Nerve Stimulation as Well?

Yes. Ultrasound has now become the primary means of locating targets, guiding needle advancement and confirming appropriate spread of injectate. However, as the needle tip is not always visible, even in experienced hands, it is prudent to use other monitors to safeguard against needle-nerve contact. Electrical nerve stimulation is insensitive (i.e., your tip may be *inside* the nerve with no motor response at a high current) but is very specific: if you observe a motor response to a current intensity below 0.2 mA, all the available data points to a needle tip location abutting or within the nerve.

Are There Any Other Monitors That Can Be Used to Prevent Nerve Injury?

Injection pressure monitoring is complementary to nerve stimulation in this regard. Certainly it is a non-specific monitor—high resistance to injection may result from a clotted needle, tip position against fascia, or any other number of reasons; however, if injectate *can* occur using pressures less than 15 psi, then there appears to be a 90–97% likelihood that that needle tip is in a safe position (i.e., not impinging on the epineurium or within a fascicle).

What Special Equipment Is Required for Injection Pressure Monitoring?

While commercial products do exist to make the task of monitoring injection pressure easy, a clever application of Boyle's law ($P_1V_1 = P_2V_2$) is all one needs to ensure opening injection pressures remain in the safe (<15 psi) range in a syringe. The local anesthetic syringe is made to contain a known volume of air and is held upright (plunger toward the ceiling). Once injection begins, as long as the air bubble does not reach half of its original volume, the pressure in the system will not double (i.e., reach 14.7 psi), a conveniently safe threshold for preventing injury.

Review Questions

1. Ultrasonography during peripheral nerve blocks has been shown to:
   (a) Reduce the incidence of nerve injury compared to nerve stimulation techniques
   (b) Reduce the likelihood of pneumothoraces compared to nerve stimulation techniques
   (c) Reduce the need for monitoring during nerve block performance
   (d) Reduce the incidence of accidental vascular puncture
2. The use of injection pressure monitoring:
   (a) May reduce the incidence of nerve injury
   (b) Is complicated and requires special equipment
   (c) Is unnecessary if the practitioner is experienced and can gauge "hand feel" for himself/herself
   (d) Should be used only for high-risk blocks such as interscalene brachial plexus blocks
3. Which of the following is *not* critical to observe during performance of all ultrasound-guided peripheral nerve blocks?
   (a) Expansion of the tissue at the time of local anesthetic injection at the target site
   (b) Vasculature that is adjacent to the target site
   (c) The tip of the nerve block needle
   (d) The target nerve or plexus
4. Which of the following is true regarding the use of nerve stimulation during peripheral nerve blockade?
   (a) There is a predictable relationship between needle tip-nerve distance and the current required to cause a motor response.
   (b) Nerve stimulation has little value in the era of ultrasonography.
   (c) The generation of a motor response at extremely low currents (i.e., <0.2 mA) has been associated with intraneural needle tip placement.

(d) If a motor response is NOT generated at currents of 0.5 mA or greater, intraneural needle tip placement is not possible.

5. Essential equipment for the performance of a femoral nerve block includes:
   (a) Electrocardiography
   (b) Nerve stimulation
   (c) Ultrasound
   (d) A laryngeal mask airway
6. Epinephrine is often added to local anesthetic solutions for all of the following reasons EXCEPT:
   (a) To truncate the peak plasma level of local anesthetic
   (b) To hasten the onset of the analgesic block
   (c) To prolong the duration of nerve blockade
   (d) To serve as an intravenous marker in case of intravascular absorption
7. When used for peripheral nerve blockade, the use of ultrasonography significantly reduces the incidence of:
   (a) Pneumothorax
   (b) Nerve injury
   (c) Unwanted motor blockade
   (d) Local anesthetic systemic toxicity

Answers

1. d
2. a
3. c
4. c
5. a
6. b
7. d

References

1. Buhre W, Rossaint R. Perioperative management and monitoring in anaesthesia. Lancet. 2003;362:1839–46.
2. Auroy Y, Benhamou D, Péquignot F, Bovet M, Jougla E, Lienhart A. Mortality related to anaesthesia in France: analysis of deaths related to airway complications. Anaesthesia. 2009;64:366–70.
3. Li G, Warner M, Lang BH, Huang L, Sun LS. Epidemiology of anesthesia-related mortality in the United States, 1999-2005. Anesthesiology. 2009;110:759–65.
4. Di Gregorio G, Neal JM, Rosenquist RW, Weinberg GL. Clinical presentation of local anesthetic systemic toxicity: a review of published cases, 1979 to 2009. Reg Anesth Pain Med. 2010;35:181–7.
5. Heavner JE, Dryden CF, Sanghani V, Huemer G, Bessire A, Badgwell JM. Severe hypoxia enhances central nervous system and cardiovascular toxicity of bupivacaine in lightly anesthetized pigs. Anesthesiology. 1992;77:142–7.
6. Rosen MA, Thigpen JW, Shnider SM, Foutz SE, Levinson G, Koike M. Bupivacaine-induced cardiotoxicity in hypoxic and acidotic sheep. Anesth Analg. 1985;64:1089–96.
7. Martínez Navas A, DE LA Tabla González RO. Ultrasound-guided technique allowed early detection of intravascular injection during an infraclavicular brachial plexus block. Acta Anaesthesiol Scand. 2009;53:968–70.
8. Robards C, Clendenen S, Greengrass R. Intravascular injection during ultrasound-guided axillary block: negative aspiration can be misleading. Anesth Analg. 2008;107(5):1754.
9. Mulroy MF, Norris MC, Liu SS. Safety steps for epidural injection of local anesthetics: review of the literature and recommendations. Anesth Analg. 1997;85:1346–56.
10. Mather LE, Copeland SE, Ladd LA. Acute toxicity of local anesthetics: underlying pharmacokinetic and pharmacodynamic concepts. Reg Anesth Pain Med. 2005;30:553–66.
11. Hadzic A, Dilberovic F, Shah S, Kulenovic A, Kapur E, Zaciragic A, et al. Combination of intraneural injection and high injection pressure leads to fascicular injury and neurologic deficits in dogs. Reg Anesth Pain Med. 2004;29:417–23.
12. Gebhard RE, Ghelber O, Warters RD, Pivalizza EG, Szmuk P. Local anesthetic injection-pressure monitoring during nerve blocks with the Compuflo Injection Pump; The Anesthesiology annual meeting 2006.
13. Moore JM, Liu SS, Neal JM. Premedication with fentanyl and midazolam decreases the reliability of intravenous lidocaine test dose. Anesth Analg. 1998;86:1015–7.
14. Owen MD, Gautier P, Hood DD. Can ropivacaine and levobupivacaine be used as test doses during regional anesthesia? Anesthesiology. 2004;100:922–5.
15. Mulroy MF, Neal JM, Mackey DC, Harrington BE. 2-Chloroprocaine and bupivacaine are unreliable indicators of intravascular injection in the premedicated patient. Reg Anesth Pain Med. 1998;23:9–13.
16. McCartney CJL, Murphy DB, Iagounova A, Chan VWS. Intravenous ropivacaine bolus is a reliable marker of intravascular injection in premedicated healthy volunteers. Can J Anaesth. 2003;50:795–800.
17. Wildsmith JA, Tucker GT, Cooper S, Scott DB, Covino BG. Plasma concentrations of local anaesthetics after interscalene brachial plexus block. Br J Anaesth. 1977;49:461–6.

18. Guinard JP, Mulroy MF, Carpenter RL, Knopes KD. Test doses: optimal epinephrine content with and without acute beta-adrenergic blockade. Anesthesiology. 1990;73:386–92.
19. Tanaka M, Sato M, Kimura T, Nishikawa T. The efficacy of simulated intravascular test dose in sedated patients. Anesth Analg. 2001;93:1612–7.
20. Guay J. The epidural test dose: a review. Anesth Analg. 2006;102:921–9.
21. Leighton BL, Norris MC, DeSimone CA, Rosko T, Gross JB. The air test as a clinically useful indicator of intravenously placed epidural catheters. Anesthesiology. 1990;73:610–3.
22. Selander D, Brattsand R, Lundborg G, Nordborg C, Olsson Y. Local anesthetics: importance of mode of application, concentration and adrenaline for the appearance of nerve lesions. An experimental study of axonal degeneration and barrier damage after intrafascicular injection or topical application of bupivacaine (Marcain). Acta Anaesthesiol Scand. 1979;23:127–36.
23. Fink BR, Aasheim GM, Levy BA. Neural pharmacokinetics of epinephrine. Anesthesiology. 1978;48:263–6.
24. Myers RR, Heckman HM. Effects of local anesthesia on nerve blood flow: studies using lidocaine with and without epinephrine. Anesthesiology. 1989;71:757–62.
25. Partridge BL. The effects of local anesthetics and epinephrine on rat sciatic nerve blood flow. Anesthesiology. 1991;75:243–50.
26. Liguori GA, Zayas VM, YaDeau JT, Kahn RL, Paroli L, Buschiazzo V, et al. Nerve localization techniques for interscalene brachial plexus blockade: a prospective, randomized comparison of mechanical paresthesia versus electrical stimulation. Anesth Analg. 2006;103:761–7.
27. Urmey WF, Stanton J. Inability to consistently elicit a motor response following sensory paresthesia during interscalene block administration. Anesthesiology. 2002;96:552–4.
28. Chan VWS, Brull R, McCartney CJL, Xu D, Abbas S, Shannon P. An ultrasonographic and histological study of intraneural injection and electrical stimulation in pigs. Anesth Analg. 2007;104:1281–4.
29. Perlas A, Niazi A, McCartney C, Chan V, Xu D, Abbas S. The sensitivity of motor response to nerve stimulation and paresthesia for nerve localization as evaluated by ultrasound. Reg Anesth Pain Med. 2006;31:445–50.
30. Tsai TP, Vuckovic I, Dilberovic F, Obhodzas M, Kapur E, Divanovic K-A, et al. Intensity of the stimulating current may not be a reliable indicator of intraneural needle placement. Reg Anesth Pain Med. 2008;33:207–10.
31. Voelckel WG, Klima G, Krismer AC, Haslinger C, Stadlbauer KH, Wenzel V, et al. Signs of inflammation after sciatic nerve block in pigs. Anesth Analg. 2005;101:1844–6.
32. Bigeleisen PE, Moayeri N, Groen GJ. Extraneural versus intraneural stimulation thresholds during ultrasound-guided supraclavicular block. Anesthesiology. 2009;110:1235–43.
33. Casati A, Baciarello M, Di Cianni S, Danelli G, De Marco G, Leone S, et al. Effects of ultrasound guidance on the minimum effective anaesthetic volume required to block the femoral nerve. Br J Anaesth. 2007;98:823–7.
34. Dhir S, Ganapathy S, Lindsay P, Athwal GS. Case report: ropivacaine neurotoxicity at clinical doses in interscalene brachial plexus block. Can J Anaesth. 2007;54:912–6.
35. Riazi S, Carmichael N, Awad I, Holtby RM, McCartney CJL. Effect of local anaesthetic volume (20 vs 5 ml) on the efficacy and respiratory consequences of ultrasound-guided interscalene brachial plexus block. Br J Anaesth. 2008;101:549–56.
36. Vandepitte C, Gautier P, Xu D, Salviz EA, Hadzic A. Effective volume of ropivacaine 0.75% through a catheter required for interscalene brachial plexus blockade. Anesthesiology. 2013;118:863–7.
37. Barrington MJ, Kluger R. Ultrasound guidance reduces the risk of local anesthetic systemic toxicity following peripheral nerve blockade. Reg Anesth Pain Med. 2013;38:289–97.
38. Altermatt FR, Cummings TJ, Auten KM, Baldwin MF, Belknap SW, Reynolds JD. Ultrasonographic appearance of intraneural injections in the porcine model. Reg Anesth Pain Med. 2010;35:203–6.
39. Lupu CM, Kiehl T-R, Chan VWS, El-Beheiry H, Madden M, Brull R. Nerve expansion seen on ultrasound predicts histologic but not functional nerve injury after intraneural injection in pigs. Reg Anesth Pain Med. 2010;35:132–9.
40. Krediet AC, Moayeri N, Bleys RL, Groen GJ. Intraneural or extraneural: diagnostic accuracy of ultrasound assessment for localizing low-volume injection. Reg Anesth Pain Med. 2014;39:409–13.
41. Bigeleisen PE. Nerve puncture and apparent intraneural injection during ultrasound-guided axillary block does not invariably result in neurologic injury. Anesthesiology. 2006;105:779–83.
42. Robards C, Hadzic A, Somasundaram L, Iwata T, Gadsden J, Xu D, et al. Intraneural injection with low-current stimulation during popliteal sciatic nerve block. Anesth Analg. 2009;109:673–7.
43. Hara K, Sakura S, Yokokawa N, Tadenuma S. Incidence and effects of unintentional intraneural injection during ultrasound-guided subgluteal sciatic nerve block. Reg Anesth Pain Med. 2012;37:289–93.
44. Sunderland S. The anatomy and physiology of nerve injury. Muscle Nerve. 1990;13:771–84.
45. Hind D, Calvert N, McWilliams R, Davidson A, Paisley S, Beverley C, et al. Ultrasonic locating devices for central venous cannulation: meta-analysis. BMJ. 2003;327:361.
46. Barrington MJ, Watts SA, Gledhill SR, Thomas RD, Said SA, Snyder GL, et al. Preliminary results of the

Australasian Regional Anaesthesia Collaboration: a prospective audit of more than 7000 peripheral nerve and plexus blocks for neurologic and other complications. Reg Anesth Pain Med. 2009;34:534–41.
47. Abrahams MS, Aziz MF, Fu RF, Horn J-L. Ultrasound guidance compared with electrical neurostimulation for peripheral nerve block: a systematic review and meta-analysis of randomized controlled trials. Br J Anaesth. 2009;102:408–17. https://doi.org/10.1093/bja/aen384.
48. Loubert C, Williams SR, Hélie F, Arcand G. Complication during ultrasound-guided regional block: accidental intravascular injection of local anesthetic. Anesthesiology. 2008;108:759–60.
49. Schafhalter-Zoppoth I, Zeitz ID, Gray AT. Inadvertent femoral nerve impalement and intraneural injection visualized by ultrasound. Anesth Analg. 2004;99:627–8.
50. Koscielniak-Nielsen ZJ, Rasmussen H, Hesselbjerg L. Pneumothorax after an ultrasound-guided lateral sagittal infraclavicular block. Acta Anaesthesiol Scand. 2008;52:1176–7.
51. Bryan NA, Swenson JD, Greis PE, Burks RT. Indwelling interscalene catheter use in an outpatient setting for shoulder surgery: technique, efficacy, and complications. J Shoulder Elbow Surg. 2007;16:388–95.
52. Luyet C, Wipfli M, Eichenberger U, Farkas ZS. Performing ultrasound-guided supraclavicular blocks in the outpatient setting-an additional security measure. Reg Anesth Pain Med. 2010;35:224.
53. Gadsden JC, Choi JJ, Lin E, Robinson A. Opening injection pressure consistently detects needle–nerve contact during ultrasound-guided interscalene brachial plexus block. Anesthesiology. 2014;120:1246–53.
54. Gadsden J, Latmore M, Levine DM, Robinson A. High opening injection pressure is associated with needle-nerve and needle-fascia contact during femoral nerve block. Reg Anesth Pain Med. 2016;41:50–5.
55. Claudio R, Hadzic A, Shih H, Vloka JD, Castro J, Koscielniak-Nielsen Z, et al. Injection pressures by anesthesiologists during simulated peripheral nerve block. Reg Anesth Pain Med. 2004;29:201–5.
56. Theron PS, Mackay Z, Gonzalez JG, Donaldson N, Blanco R. An animal model of "syringe feel" during peripheral nerve block. Reg Anesth Pain Med. 2009;34:330–2.
57. Tsui BCH, Knezevich MP, Pillay JJ. Reduced injection pressures using a compressed air injection technique (CAIT): an in vitro study. Reg Anesth Pain Med. 2008;33:168–73.
58. Gadsden JC, Lindenmuth DM, Hadzic A, Xu D, Somasundarum L, Flisinski KA. Lumbar plexus block using high-pressure injection leads to contralateral and epidural spread. Anesthesiology. 2008;109:683–8.
59. Sieber FE, Zakriya KJ, Gottschalk A, Blute M-R, Lee HB, Rosenberg PB, et al. Sedation depth during spinal anesthesia and the development of postoperative delirium in elderly patients undergoing hip fracture repair. Mayo Clin Proc. 2010;85:18–26.
60. Ben-David B, Vaida S, Gaitini L. The influence of high spinal anesthesia on sensitivity to midazolam sedation. Anesth Analg. 1995;81:525–8.
61. Shono A, Sakura S, Saito Y, Doi K, Nakatani T. Comparison of 1% and 2% lidocaine epidural anaesthesia combined with sevoflurane general anaesthesia utilizing a constant bispectral index. Br J Anaesth. 2003;91:825–9.
62. Doufas AG, Wadhwa A, Shah YM, Lin C-M, Haugh GS, Sessler DI. Block-dependent sedation during epidural anaesthesia is associated with delayed brainstem conduction. Br J Anaesth. 2004;93:228–34.
63. Nishikawa K, Hagiwara R, Nakamura K, Ishizeki J, Kubo K, Saito S, et al. The effects of the extent of spinal block on the BIS score and regional cerebral oxygen saturation in elderly patients: a prospective, randomized, and double-blinded study. J Clin Monit Comput. 2007;21:109–14.
64. Park KS, Hur EJ, Han KW, Kil HY, Han TH. Bispectral index does not correlate with observer assessment of alertness and sedation scores during 0.5% bupivacaine epidural anesthesia with nitrous oxide sedation. Anesth Analg. 2006;103:385–9.
65. Friedman DJ, Parnes NZ, Zimmer Z, Higgins LD, Warner JJP. Prevalence of cerebrovascular events during shoulder surgery and association with patient position. Orthopedics. 2009;32(4). pii: orthosupersite.com/view.asp?rID=38058.
66. Pohl A, Cullen DJ. Cerebral ischemia during shoulder surgery in the upright position: a case series. J Clin Anesth. 2005;17:463–9.
67. Murphy GS, Szokol JW, Marymont JH, Greenberg SB, Avram MJ, Vender JS, et al. Cerebral oxygen desaturation events assessed by near-infrared spectroscopy during shoulder arthroscopy in the beach chair and lateral decubitus positions. Anesth Analg. 2010;111:496–505.
68. Macario A, Weinger M, Carney S, Kim A. Which clinical anesthesia outcomes are important to avoid? The perspective of patients. Anesth Analg. 1999;89:652–8.
69. Gajraj NM, Sharma SK, Souter AJ, Pole Y, Sidawi JE. A survey of obstetric patients who refuse regional anaesthesia. Anaesthesia. 1995;50:740–1.
70. Ironfield CM, Barrington MJ, Kluger R, Sites B. Are patients satisfied after peripheral nerve blockade? Results from an International Registry of Regional Anesthesia. Reg Anesth Pain Med. 2014;39:48–55.
71. Jlala HA, Bedforth NM, Hardman JG. Anesthesiologists' perception of patients' anxiety under regional anesthesia. Local Reg Anesth. 2010;3:65–71.
72. Avramov MN, White PF. Use of alfentanil and propofol for outpatient monitored anesthesia care: determining the optimal dosing regimen. Anesth Analg. 1997;85:566–72.
73. Pavlin DJ, Coda B, Shen DD, Tschanz J, Nguyen Q, Schaffer R, et al. Effects of combining propofol and

alfentanil on ventilation, analgesia, sedation, and emesis in human volunteers. Anesthesiology. 1996;84:23–37.
74. Horikawa H, Tada T, Sakai M, Karube T, Ichiyanagi K. Effects of midazolam on the threshold of lidocaine-induced seizures in the dog–comparison with diazepam. J Anesth. 1990;4:265–9.
75. Kubulus C, Schmitt K, Albert N, Raddatz A, Gräber S, Kessler P, et al. Awake, sedated or anaesthetised for regional anaesthesia block placements? A retrospective registry analysis of acute complications and patient satisfaction in adults. Eur J Anaesthesiol. 2016;33:715–24.
76. Martínez-Tellería A, Cano ME, Carlos R. [Paradoxical reaction to midazolam after its use as a sedative in regional anesthesia]. Rev Esp Anestesiol Reanim. 1992;39:379–80.

7 Anticoagulation and Regional Anesthesia Concerns

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Introduction

Interventional pain management is an emerging specialty that focuses on the use of procedures to diagnose and to treat chronic pain. Most of these procedures are performed percutaneously and carry a potential risk of bleeding [1]. Regional anesthesia similarly uses percutaneous injection techniques for surgery, perioperatively, and postoperative analgesia. Patients undergoing these treatments may be receiving exogenous anticoagulants or have impaired hemostasis.

Interventional pain management and regional anesthesia procedures carry a risk of bleeding with potentially hazardous consequences. The American Society of Regional Anesthesia has published guidelines addressing the risk of bleeding and hematomas following regional and neuraxial techniques in the setting of pharmacological anticoagulation, with the most recent updated guidelines from 2010 [2]. It is clear and obvious that the clinician performing regional anesthesia must be aware of the potential significant morbidity and mortality of attempting to complete such procedures in an anticoagulated patient receiving antithrombotics or thrombolytic therapy.

Interventional pain management consists of a larger variety of procedures [3], delivered in the outpatient setting. The ASRA guidelines [2], although useful, were not created for these practitioners. A bleeding risk stratification and summary of the literature have been developed for interventional pain physicians [1]. Bleeding risks must be weighed against procedural benefits. The practitioner must decide between performing and canceling the procedure, after assessing the risk for bleeding. Appraising bleeding risk involves understanding coagulation physiology and pathophysiology, pharmacology of anticoagulants, and the technical risks associated with a particular procedure.

Coagulation Physiology

Under normal circumstances, a tight equilibrium between clotting and bleeding is maintained. A complex interplay between activators, cofactors,

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_7

inhibitors, and feedback loops exists. Hemostasis involves three simultaneous processes: (1) primary hemostasis, (2) secondary hemostasis, and (3) fibrinolysis.

Platelets play a critical role in primary hemostasis [4]. Vascular endothelial injury occurring after needle trauma creates a friable platelet plug by the deposition of platelets at the site of injury in order to stop bleeding. This process of platelet deposition is referred to as primary hemostasis. When vascular endothelial injury occurs that exposes the underlying extracellular matrix, platelets undergo a series of changes in three phases: adhesion, activation, and aggregation.

A platelet is a cell fragment which is derived from bone marrow megakaryocytes, which are surrounded by a coat of glycoproteins; these glycoproteins adhere to injured endothelium. Von Willebrand factor (vWF), a protein found in subendothelial tissues, facilitates platelet adhesion. Platelets express a glycoprotein Ib receptor that binds to vWF. Upon adhesion, the platelet activates. Platelets change shape, initiate a granule release reaction, and express glycoprotein IIb/IIIa receptors on their surface. Phosphatidylserine is translocated to the outer side of the platelet. Plasma clotting factors interact with the activated platelet surface. Fibrinogen adheres to the GpIIb/GpIIIa receptors. The process is highly regulated, and endothelial cells prevent platelet aggregation outside the area of vascular injury.

The coagulation cascade is critical to secondary hemostasis [4]. Clotting factors circulate in an inactive form and are activated by exposure to tissue factor or foreign surfaces. All of the procoagulants are synthesized in the liver except von Willebrand factor, which is produced in megakaryocytes and endothelial cells, and factor VIII which is also produced by endothelial cells. These procoagulants are cleaved into an activated enzyme. In succession, this enzyme cleaves the next procoagulant and so on. This cascade terminates by converting the water-soluble fibrinogen into the insoluble fibrin. Fibrin acts as "glue" that stabilizes the platelet plug. Fibrin cross-linking strengthens the clot. There are two clotting pathways, intrinsic and extrinsic, which lead to a final common pathway. The final common pathway begins with the binding of factor Xa to cofactor Va and platelet phospholipids (PF-3). This complex converts prothrombin (II) to thrombin (IIa). PF-3-bound thrombin complex then cleaves fibrinogen (I) to fibrin (Ia). The extrinsic pathway can be rapid and must occur in the presence of tissue trauma. Tissue factor modulates factor VIIa activity, and this interaction is the primary physiologic event in initiating clotting [5]. The intrinsic pathway can be induced in the absence of extrinsic tissue components and only requires factors "intrinsic" to the blood. Both the intrinsic and extrinsic pathways work in tandem and lead to the final common pathway.

Coagulation must be restricted to the area of injury; control occurs via three types of inhibitory pathways: (1) antithrombin III, (2) thrombomodulin, and (3) tissue factor inhibitor. Antithrombin III inhibits several factors, particularly Xa. Thrombomodulin binds to thrombin and activates proteins C and S, which causes proteolysis of factors Va and VIIIa. Tissue factor pathway inhibitor blocks factor Xa via a negative feedback loop. The enzymatic degradation of fibrin is governed by the fibrinolytic system. Plasmin is a proteolytic enzyme that digests fibrin, fibrinogen, factor V, factor VIII, prothrombin, and factor XII. Plasmin is derived from plasminogen. Tissue-type plasminogen activator is a serine protease that binds to a fibrin clot and activates plasminogen. Proteolysis is confined to the clot itself.

Coagulation Pathophysiology

Coagulation pathophysiology clinically manifests as a hemorrhagic or thrombotic disorder. Hemorrhagic disorders may be hereditary or acquired disorders of hemostasis. The three most common hereditary coagulation disorders are von Willebrand's disease, hemophilia A, and hemophilia B [6, 7].

Von Willebrand's disease is the most common hereditary bleeding disorder, afflicting 1–3% of the general population. There may be a quantitative or qualitative impairment in von Willebrand factor [7]. As previously mentioned, vWF func-

tions in primary hemostasis by binding to both platelets and endothelial components, forming an adhesive bridge between platelets and subendothelial components and between adjacent platelets. VWF also serves as a carrier protein for factor VIII which increases its half-life. In von Willebrand's disease, platelets lose their adhesive properties, and factor VIII levels are reduced. The condition is inherited as an autosomal dominant disorder. Patients develop bruising and mucosal bleeding. Prolonged bleeding from mucosal surfaces may occur, such as epistaxis or menorrhagia. Surgical bleeding is localized to the area of injury, and distant site bleeding is uncommon. Platelet function analyses, PFA-100, can diagnose the problem. Specialized assays, such as one that directly measures the von Willebrand factor antigen, can confirm the diagnosis [7]. Treatment involves desmopressin and factor replacement.

Hemophilia A is a bleeding disorder resulting from a defect in factor VIII:C, a cofactor involved in the activation of factor X [8]. The disease primarily afflicts men since the disease is X-linked. All patients with hemophilia A have normal plasma concentrations of vWF. Life-threatening hemorrhage can occur. Spontaneous bleeding into joints, neural compartments, and intracranial structures can occur. Central nervous system hemorrhaging is associated with a 30% mortality rate. The activated partial thromboplastin time (aPTT) is abnormal. The prothrombin or bleeding times are normal. Disease severity is proportional to plasma concentrations of factor VIII. Factor replacement is essential in planning surgical procedures. Factor VIII [8] replacement can be plasma derived or recombinant. Recombinant VIIa, a "universal" hemostatic agent, can be used to control surgical or trauma-associated bleeding in patients with hemophilia A. Hemophilia B is a bleeding disorder resulting from a defect in factor IX. Inheritance and clinical patterns are indistinguishable from hemophilia A. Treatment requires plasma-derived or recombinant factor IX.

Vitamin K deficiency develops with malnutrition, fat malabsorption, antibiotic usage, and liver disease [4]. Microsomal carboxylase, a liver enzyme dependent on vitamin K, is necessary to convert factors II, VII, IX, and X into their functionally active forms. Vitamin K deficiency leads to a reduction in these factors and, consequently, a bleeding diathesis. Patients develop melena, hematuria, ecchymosis, and hematomas [4]. Supplemental vitamin K can be given 4–8 h prior to a procedure in the at-risk patient.

Liver disease can lead to thrombocytopenia, platelet dysfunction, reduced production of clotting factors, increased factor consumption, and increased fibrinolysis. A continuum of bleeding disorders may result, and all stages of liver disease increase the risk of bleeding [9]. Preprocedural screening of liver disease patients may include hemoglobin, PT, aPTT, platelet count, platelet function analysis, fibrinogen level, and bilirubin levels. Management strategies include vitamin K supplementation, fresh frozen plasma, platelets, and cryoprecipitate. Vitamin K supplementation may be sufficient in patients with biliary tract disorders [9].

Renal disease causes hemostatic defects due to multiple reasons [10]. Defects in platelets, subendothelial metabolism, and platelet vessel interactions occur. Renal disease augments the effects of antiplatelet drugs and low-molecular-weight heparins. A comprehensive coagulation profile must be performed in renal failure patients. Bleeding time may be useful to assess bleeding risk. Elevations in the PT or aPTT denote the effect of other clotting problems. Coagulopathy [10] may be treated with dialysis, anemia correction, desmopressin, cryoprecipitate, estrogens, and avoidance of antiplatelet drugs.

Drugs That Impair Hemostasis

Cyclooxygenase [COX] inhibitors disrupt the formation of thromboxane A2, which disturbs vasoconstriction and secondary platelet aggregation. A primary platelet plug may still form and be adequate for small injuries; however, this will not be sufficient for stopping surgically induced bleeding. Aspirin irreversibly inhibits cyclooxygenase for the life of platelet, whereas nonsteroidals are reversible in their inhibition of

COX. Procedural postponement may not be necessary, except in situations whereby bleeding times are excessively prolonged. Another option is to have patients withhold aspirin for 7–10 days and NSAIDs for 3–5 days [11, 12].

Thienopyridine inhibitors, such as clopidogrel, ticagrelor, ticlopidine, and prasugrel, interfere with primary and secondary platelet aggregation [13]. These agents interfere with ADP binding and subsequent activation of the GpIIb/GpIIIb receptor complex. Clopidogrel interferes with platelets to platelet, platelet to fibrinogen, and platelet to endothelium interactions. Clopidogrel reaches a steady state in 7 days, but is reversible. Clopidogrel prolongs bleeding time more than aspirin. Purpura and epistaxis can occur in up to 5% of patients. Serious bleeding occurs at a rate of 1–2% [13, 14]. Recent studies suggest bleeding is more common with prasugrel. A recent study comparing prasugrel vs. clopidogrel showed a significantly increased number of bleeding complications for patients receiving prasugrel [15]. Extra caution with this medication is suggested as its clinical use is increasing.

Glycoprotein receptor antagonists interfere with the final common pathway of platelet aggregation and cross-linking of adjacent platelets via interaction with fibrinogen [16]. GpIIb/GpIIIa receptor antagonists, which include abciximab, eptifibatide, and tirofiban, are often used in the management of acute coronary syndromes. Platelet function normalizes 8–24 h after stopping the infusion. During the infusion period, significant bleeding occurs 2% of the time, and the majority of patients develop some degree of bleeding. These agents are not encountered in the outpatient setting, but are important in the perioperative setting. Elective surgery should be delayed for 24–48 h following abciximab and 4–8 h after tirofiban. Eptifibatide is another product in the same class that would be expected to have similar bleeding risks.

Warfarin [17] is an oral anticoagulant that interferes with the carboxylation of vitamin K-dependent coagulation factors. Vitamin K-dependent factors II, VII, IX, and X and the anticoagulant proteins C and S become depleted. The intensity of warfarin therapy depends on the proportion of inactive factors and factor half-lives. Factor VII has the shortest half-life (6–8 h) and is most likely to affect the prothrombin time/international normalized ratio. When factor VII is at 40%, the INR approaches 1.5 [17]. Hemostasis is presumed to be normal at an INR $\leq$ 1.5. Age, diet, race, drug interactions, gender, body weight, and comorbidities influence the response to warfarin. A warfarin overdose manifests as ecchymoses and mucosal bleeding. Most surgical procedures can be carried out at an INR of 1.5. However, many practitioners will hold warfarin for 4–5 days prior to a surgical procedure. Balancing the risks of withdrawing anticoagulation in patients with a history of stroke or venous thromboembolic disease prior to performing surgery is a challenge and must be reviewed with the risks versus benefits in mind.

Heparin [18] is a glycosaminoglycan that is widely used for anticoagulation during surgical procedures. Unfractionated heparin has a heterogeneous range of molecular weights with correspondingly heterogeneous anticoagulant properties. Heparin interacts with antithrombin III, which inactivates factors IIa, Xa, IXa, XIa, and XIIa. By inactivating thrombin, heparin prevents fibrin formation and inhibits thrombin-induced activation of platelets and factors V, VIII, and XI. The anticoagulant effect is nonlinear. Low doses have a half-life of 60 min, and high doses have a half-life of up to 150 min. Significant bleeding has been reported in association with different types of heparin therapy. Subcutaneous hemorrhages and deep tissue hematomas may occur. Spontaneous bleeding may occur, even with an aPTT 1.5–2 times the normal.

Low-molecular-weight heparin [18] has a longer half-life, compared to unfractionated heparin. They are derived from unfractionated heparin by depolymerization and are approximately one third the molecular weight of heparin. It binds to antithrombin and inactivates factor Xa. Due to their size, 50–75% of the chains are too small to simultaneously bind antithrombin and thrombin. Therefore, the inhibition of thrombin is minimal. These products include enoxaparin, dalteparin, ardeparin, danaparoid, tinzaparin, and nadropa-

rin. No blood test can adequately monitor the anticoagulant effect. LMWH primarily is associated with anti-Xa activity. Two to four hours following subcutaneous administration, therapeutic levels are reached, and up to 50% of this effect can be maintained at 12 h. Abnormal renal function, advanced age, and concomitant NSAID use enhance the effects of LMWH. LMWH is commonly used to reduce the risk of venous thromboembolism (VTE).

Herbal medications [19] such as garlic, ginkgo, and ginseng may cause bleeding. Garlic irreversibly inhibits platelet aggregation and has fibrinolytic activity. Ginkgo inhibits platelet-activating factor. Ginseng interferes with platelet aggregation. Several other herbal medications have been implicated as antihemostatic agents, including feverfew, green tea, horse chestnut, cat's claw, ginger, fenugreek, and chamomile. Fish oil (omega-3 fatty acid) supplements have been reported to increase the risk for significant bleeding. While well-controlled studies have not provided solid evidence for increased risk of clinically significant bleeding with fish oil, clinicians should be aware of the possibility of increased risk when used in conjunction with anticoagulants.

SSRIs and Other Antidepressants

Selective serotonin reuptake inhibitors (SSRIs) include fluoxetine (Prozac), fluvoxamine (Luvox), paroxetine (Paxil), sertraline (Zoloft), citalopram (Celexa), clomipramine (Anafranil), and trazodone (Desyrel). Nonselective serotonin reuptake inhibitors include amitriptyline (Elavil), imipramine (Tofranil), and doxepin (Sinequan). These medications are commonly used for depression, anxiety, and other psychological conditions. Observational studies have shown that SSRIs can increase the risk of bleeding when combined with NSAIDs, aspirin, or anticoagulants. Bleeding complications that have been observed include upper gastrointestinal bleeding, stroke, postpartum hemorrhage, and intraoperative bleeding as well as minor episodes of mucosal bleeding, petechiae, purpura, and easy bruising; upper gastrointestinal bleeding is consistently observed across multiple studies [20]. Depletion of serotonin (5-HT) levels in platelets inhibits induced aggregation and has the potential to prevent formation of clots. Among the different SSRIs, there is not strong evidence that any specific antidepressant is more strongly associated with bleeding. Clinicians should be aware of the potential of increased bleeding when these medications are combined with other anticoagulants.

Other Antiplatelet Drugs

Dipyridamole (Persantine) is an older drug with a mechanism of action that is thought to involve the inhibition of cyclic AMP phosphodiesterase. It is often combined with aspirin and used in the prevention of stroke and transient ischemic attack. Due to the inhibition of platelet functioning, discontinuation of dipyridamole 3–5 days before a surgical procedure would be prudent.

Thrombin Inhibitors

Direct thrombin inhibitors (DTIs) are a class of anticoagulants that bind directly to and block thrombin, acting as allosteric inhibitors which prevent the binding of other substrates. These inhibitors of thrombin may carry a lower risk of bleeding as compared to high-dose intravenous heparin. Recombinant hirudin derivatives (desirudin, lepirudin, bivalirudin) inhibit free and clot-bound thrombin. They are an alternative to heparin in patients with unstable angina undergoing percutaneous coronary interventions although no DTI has been shown to be safe for long-term treatment of acute coronary syndromes. DTIs may also have a lower risk of major hemorrhage as compared to heparin. The half-life is approximately 1 h. Serum levels decrease to zero 2 h after discontinuation of these IV agents. Argatroban, an L-arginine derivative, also provides reversible thrombin inhibition. It is used in the treatment and prophylaxis of heparin-induced thrombocytopenia. Lepirudin is also a direct

thrombin inhibitor that is approved in the United States to treat HIT. Treatment with both of these agents decreases the rate of new thrombotic episodes in HIT patients [21].

Dabigatran is a new oral direct thrombin inhibitor that is used in patients for the prevention of venous thromboembolic disease and for the prevention of stroke in patients with atrial fibrillation. Like other anticoagulants, dabigatran increases the risk of bleeding in patients. Dabigatran has a black box warning for an increased risk of spinal or epidural hematoma for patients undergoing regional anesthesia or spinal taps. A new reversal agent for dabigatran called idarucizumab has recently come onto the market. This new drug can provide rapid reversal of spinal hematoma complications in regional anesthesia and pain procedures as well as reversals of intracranial and gastrointestinal hemorrhage. The reversal of the anticoagulant effect can occur within minutes of administration of idarucizumab [22].

Newer Anticoagulants

Direct factor Xa inhibitors are a new class of anticoagulants that inactivate factor Xa. These oral drugs have become very popular in the patients that require prophylactic prevention of embolic stroke from atrial fibrillation as well as prevention and treatment of deep vein thrombosis and pulmonary embolism. Direct factor Xa inhibitors are oral agents only with no parental form available. There are also no reversal agents available for direct factor Xa inhibitors, but there are currently reversal drugs in clinic trials. The most commonly prescribed factor X inhibitors in the United States are rivaroxaban and apixaban. Because both of these drugs do not require routine monitoring of coagulation markers and do not affect vitamin K metabolism, they are seen as more convenient options for patients who need anticoagulation. Like all anticoagulants, both of these agents increase the risk of bleeding events, and both have a black box warning for epidural and spinal hematomas after procedures such as spinal punctures and neuraxial anesthesia [3, 23].

Bleeding Complications in Association with Interventional Pain Practice and Regional Anesthesia

The incidence of spontaneous spinal hematoma is extremely rare with an estimate of 1 patient per 1,000,000 patients per year [3, 24]. The incidence of clinically significant spinal hematoma has been estimated with 95% confidence to be <1 of 150,000 epidural anesthetics and <1 of 220,000 spinal anesthetics for noncardiac surgical cases. Ruppen et al. estimated the risk of epidural hematoma following epidural analgesia in the obstetric population to be 1 in 168,000 [25]. For cardiac surgery with anticoagulation during cardiopulmonary bypass, the estimated risk of hematoma with 95% confidence is 1 in 3552 according to a meta-analysis done in 2015 [26].

The rate of spinal hematomas in all non-obstetric patients is 18.5 per 100,000 patients [27]. In general, the risk of complications in epidural analgesia is low for obstetric cases and increases substantially in vascular surgery [27]. There are also certain risk factors that increase the risk of hematoma as an adverse event after epidural placement. These risk factors include female gender, difficult spinal anatomy, traumatic epidural, current antithrombotic medication treatment, and inherited or acquired coagulopathy [24, 28].

Epidural catheterization and regional blocks have been successfully carried out in patients with hemophilia A, when factor VIII replacement was carried out [29, 30]. Another recombinant factor, VIIa, is sometimes referred to as the universal hemostatic agent because it can be used to control surgical and trauma-associated bleeding in patient with hemophilia A. Epidural catheterization has been successfully performed in patients with von Willebrand's disease when vWF:Ag and RiCof levels met certain thresholds [31, 32]. Advanced liver disease, with associated portal hypertension and hypersplenism, poses a unique risk in procedure-related bleeding. Epidural hematomas following catheterization have been reported in patients with mild liver disease [33]. In the setting of renal disease, a delayed

epidural hematoma has been seen despite normal coagulation studies and bleeding history in this patient [34].

Anticoagulation increases the risk of bleeding. Spontaneous bleeding occurs in 3–7% of patients receiving warfarin [35]. Bleeding occurs in less than 3% of patients receiving fractionated or unfractionated heparin [35]. Thrombolytics present the greatest risk of bleeding with 6–30% [35]. In the setting of these agents, procedure-induced bleeding risk increases with anticoagulation.

The relative risk of neuraxial procedures in the presence of anticoagulation has been estimated [36]. There is no increased risk in the presence of aspirin therapy. Traumatic insertion increases the relative risk to 11. Traumatic insertion in the presence of systemic heparinization increases the relative risk to 111. Aspirin and intravenous heparin therapy increase the risk to 26. The closer in time that heparin is restarted in a patient, the higher the risk of bleeding. If heparinization is started within 1 h of a neuraxial procedure, the risk is 25. If heparinization is delayed more than 1 h, then the risk drops to 2.

Aspirin and NSAIDs have not been proven to increase the risk of spinal epidural hematoma. These agents are safe to be used in patients receiving neuraxial anesthesia. On the contrary, aspirin and NSAIDs can increase the risk of bleeding in patients taking another medication that affects coagulation, including fish oil products. It has been advised for these patients to stop taking aspirin 7–10 days before an epidural or neuraxial procedure if the patient is on another antithrombotic agent. NSAIDS should be stopped 3 days prior to procedure if on another antithrombotic medication [12].

Recommendations differ for P2Y12 receptor blockers. Clopidogrel should be stopped 7 days before a patient undergoes neuraxial anesthesia, and ticlopidine should be held even longer, 14 days, before a procedure is performed. Both drugs may be restarted immediately after the neuraxial catheter has been discontinued [37]. Glycoprotein receptor inhibitors such as abciximab, eptifibatide, and tirofiban affect the way platelets function in the body, so patients on these medications must be cautious when undergoing a needle or catheter placement. It is recommended that eptifibatide and tirofiban be stopped at least 8 h before a procedure and abciximab be stopped 48 h prior to procedure or catheter placement. Patients must wait until 4 weeks after a surgery to restart these agents [2].

Oral anticoagulation is a contraindication to neuraxial anesthesia procedures [2]. Atraumatic epidural catheterization in an anticoagulated patient has led to paraplegia in rare cases [38]. The American Society of Regional Anesthesia suggests that patients undergoing neuraxial procedures stop warfarin 4–5 days prior to procedure to ensure safety [2]. The prothrombin time and international normalized ratio should be checked prior to the neuraxial block. There is no "safe" INR for performing neuraxial procedures [1], but ideally, an INR less than 1.3 should be sought. Neuraxial procedures should not be performed in any patient who is currently on thrombolytics [1, 2].

Vandermeulen et al. [28] reported 30 cases of epidural hematoma in patients receiving fractionated or unfractionated heparin therapy. For unfractionated heparin, the ASRA guidelines [2] suggest that neuraxial techniques should be avoided in patients with concomitant coagulopathies. ASRA also recommends that heparinization should be delayed for 1 h after needle placement, indwelling catheters should be removed 2–4 h after the last heparin dose, and reheparinization should be delayed for 1 h after catheter removal. Postoperative neurological monitoring should occur after all procedures even with minimal use of local anesthetics, and caution should be exercised if needle insertion is difficult or traumatic. In these scenarios, patients should not be on any other antithrombotic medications including aspirin. Similar guidelines should be considered for nonneuraxial procedures [2]. Neuraxial anesthesia can be performed after prophylactic heparin doses with minimal to no risks increased the risk of procedure [39].

The recommendations for the perioperative administration of low-molecular-weight heparin by the American Society of Regional Anesthesia advise avoidance of concomitant antiplatelet and oral anticoagulants, due to increased risk of

adverse outcomes. Needle placement should be delayed 12 h following the last dose of prophylactic LMWH, and it should be delayed 24 h following the last dose of therapeutic LMWH. LMWH should be held at least 24 h following surgery if an indwelling catheter is in place and also at least 24 h following a traumatic tap. The catheter should be removed prior to initiation of LMWH therapy, and this therapy should not be started for at least 4 h after catheter removal [2, 40].

Ho et al. [41] summarized the safety precautions to minimize the risk of spinal hematoma following epidural catheterization during cardiac surgery. Their recommendations included patient-specific factors (anticoagulation status) and technique-specific factors (epidural catheterization): (1) normalization of coagulation before needle or catheter insertion, (2) avoidance of repeated attempts, (3) postponement of surgery for 24 h after bloody tap, (4) needle or catheter insertion 1 h before systemic heparinization, (5) optimization of hemostasis after cardiopulmonary bypass, (6) removal of epidural catheter only after normal hemostasis has been restored postoperatively, (7) close neurologic surveillance, (8) use of a midline technique, (9) administration of saline solution through the needle to distend the epidural space before insertion of the catheter, and (10) neuraxial instrumentation postoperatively only after normalization of coagulation. Ho et al. [41] advised that significant breaching of such protocols would likely increase the risk of adverse events. Notably, their paper was published with no spinal hematomas following epidural catheterization during cardiac surgery.

Bleeding Risk Assessment

A bleeding risk score (Table 7.1) can be estimated based on various bleeding risks, such as specific anticoagulants and bleeding disorders. The goal of this scoring system is to quickly identify patients that are at increased bleeding risk. This framework helps physicians and nonphysicians to assess the bleeding risk in specific patients. The patient flow is rapid in a surgical center or pain procedure suite, and a bleeding risk score is a swift, convenient way to ensure that patients at risk are not missed during a busy workday. A bleeding risk instrument enables nonphysicians to quickly bring a potential bleeding problem to the attention of the physician.

Table 7.1 Components of bleeding risk associated with interventional pain procedure

Risk factors associated with technique	Score
Proximity to significant vascular structures	1
Proximity to significant neurological structures	1
Target in a confined space	1
Use of a sharp, rather than blunt, needle to reach target	1
Multiple passages	1
Contrast not used, if applicable	1
Fluoroscopy not used, if applicable	1
Aspiration not performed or the presence of blood at the needle hub	1
Needle size: larger than 20 gauge	1
Continuous, not single shot, procedure	1

Data from [40]

Bleeding risks differ based on the type of procedure and its anatomic location (Table 7.2) [1]. These risks depend on whether the target structure is near a major vascular or neurological structure or if it is in a confined space [1]. The gauge of the needle and number of attempts at the procedure also affect the bleeding risk. A bigger needle and multiple passes of the needle into a designated area increase the risk of bleeding [1]. The use of fluoroscopy and contrast and the use of aspiration are factors that decrease the risk of bleeding [1]. Finally, a procedure that is a "single shot" may have a lower risk of bleeding compared to a continuous infusion [1].

The bleeding risk score can be calculated by using different risk factors from the type of patient or the type of procedure involved. This score may support clinical decision-making about whether to cancel or carry out the procedure [1]. Regardless, patient flow is rapid in interventional pain management—prone to the possibility of failing to identify patients at an elevated bleeding risk. The bleeding risk score is

Table 7.2 Components of bleeding risk associated with impaired hemostasis and anticoagulant

Hemostasis	Modifying factors	Score
Normal	None	2
Normal	History of self-limited, transient bleeding disorder	4
Normal	Normal coagulation studies despite the intake of medications that theoretically may affect hemostasis	6 (nutraceuticals, serotonin reuptake inhibitors)
Normal	Normal coagulation studies after discontinuation of known anticoagulants (the score may be modified, depending on when the drug was stopped relative to the period of drug effect)	6 (e.g., warfarin was stopped 5 days earlier, aspirin was stopped 7–10 days earlier, heparin infusion held for >6 h) 8 (e.g., aspirin was stopped 3 days earlier) 10 (e.g., warfarin was stopped 2 days earlier, heparin infusion was stopped 4 h earlier) 6–10 (e.g., factor or blood product replacement therapy in specific acquired and congenital bleeding disorder)
Abnormal	Active consumption of anticoagulants that cannot be held (the score may be modified based on the specific anticoagulant and abnormal coagulation studies)	10 (low-dose aspirin, NSAIDS) 12 (subcutaneous heparin, low-dose Coumadin (INR < 1.4), medium- to high-dose aspirin, ticlopidine, clopidogrel) 14 (low-molecular-weight heparin, Coumadin (INR 1.5–2, GpIIb/GpIIIa inhibitors)) 16 (intravenous heparin bolus, Coumadin [INR 2–3]) 16–18 (thrombin inhibitors) 18 (high-dose intravenous heparinization and warfarin, INR >3)
Abnormal	Known history of medical bleeding disorder (the score may be modified if there is a history of easy bruisability, deep versus superficial bleeding episodes, or spontaneous versus traumatically induced bleeding episodes)	20 (thrombolytics) 10 (thrombocytopenia >80,000) 12 (thrombocytopenia <80,000, idiopathic thrombocytopenic purpura, renal failure-uremia) 12–14 (von Willebrand's disease, depending on severity) 14 (vitamin K deficiency) 14–18 (hemophilia A and B, depending on the severity of factor deficiency) 14–18 (liver disease, depending on severity)
Abnormal	Known history of significant bleeding with procedures but cause not identified	18
Abnormal	Major hemorrhage due to incompetent coagulation system	20 (disseminated intravascular coagulation)

Adapted from [41]

helpful for physicians, nonphysicians, and health-care teams to rapidly identify patients at risk of bleeding. This numerical bleeding score improves health-care safety with the intent of facilitating health-care communication.

In the absence of anticoagulation, neuraxial procedures are low-risk procedures, in regard to the development of a spinal hematoma. Neuraxial procedures can be safe even if the patient is taking aspirin or nonsteroidal anti-inflammatory drugs. This systematic review suggests that no definitive conclusion can be reached regarding minimizing spinal hematoma or major bleeding risk, in the setting of anticoagulant therapy or impaired hemostasis. In this scenario, guidelines and recommendations based on sensible practices are advised. An adequately powered study to understand the true risk of bleeding in patients on anticoagulation or with a coagulopathy following an interventional spine procedure would

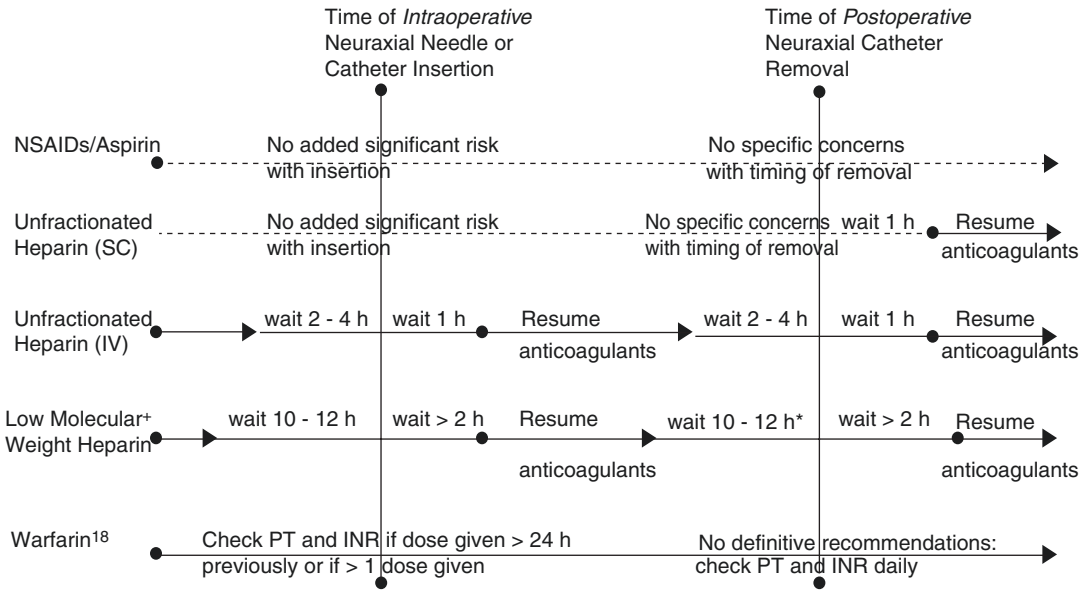

Fig. 7.1 Recommendations for anticoagulated patients undergoing image-guided spinal procedures (adapted from [43])

require a very large number of subjects. This would put many patients at risk and would be unethical. The FDA MedWatch system [42] alerted practitioners to the risks of spinal hematoma in patients on low-molecular-weight heparin. This changed the precautions that were taken in interventional pain and regional anesthesia procedures in America. Now, there are guidelines or mathematical estimations of risk [41], such as those proposed by the American Society of Regional Anesthesia (Fig. 7.1) [38]. Nonetheless, spinal hematomas can still arise with close adherence to guidelines. Furthermore, interventional spine procedures have greater variations with respect to anatomical localization, technique, practitioner expertise, and needle or catheter type. In the absence of sufficient information, a bleeding score/risk assessment may be incorporated into the clinical decision-making of interventional pain practitioners.

Conclusion

Interventional pain physicians perform procedures that carry a finite amount of bleeding risk. Many patients at increased risk either have congenital or acquired bleeding disorders or are taking anticoagulant agents. Bleeding risk assessments aid in patient safety so that risk factors are not overlooked. Understanding the possible bleeding complications of patients at increased risk and interventional procedures that are high risk due to anatomic location is imperative to improving patient safety. Pain physicians and anesthesiologists must understand the risks of many different bleeding assessments in order to make informed decisions about interventional procedures.

The patient should be counseled extensively on the risk-benefit analysis, for the purposes of informed consent and shared decision-making. Patients must understand that there is no way to reduce the risk of a spinal hematoma or major bleeding to zero. Physicians have many difficult decisions in patients with higher risk such as stopping an anticoagulant, correcting a hemostatic disorder, modifying an interventional technique, or pursuing less-invasive pain therapies.

The bleeding risk assessment is a summation of the patient- and technique-specific bleeding risks that are added together to create an overall risk of bleeding score [1]. Physicians and nonphysicians can both quickly and efficiently calculate this overall risk and use this vital piece of information to increase patient safety. This overall risk

score may facilitate clinical decision-making as well as improve safety [1].

Based on the authors' review of the literature, physician best practice guidelines should be followed to help decrease the risk of spinal epidural hematoma and other adverse reactions. A clinician should use these recommendations along with his or her own clinical judgment in order to decrease the risk of adverse events in a patient. Clinical guidelines and mathematical estimations of risk may partially fill this void. To help with decision-making, an overall risk of bleeding risk score has been proposed by a narrative review [2]. This score may support clinical decision-making about whether to cancel or carry out the procedure [2]. As for specific best practice recommendations, we support the Assessment of Bleeding Risk of Interventional Techniques: A Best Evidence Synthesis of Practice Patterns and Perioperative Management of Anticoagulant and Antithrombotic Therapy recommendations of continuing NSAIDs and low-dose aspirin and phosphodiesterase inhibitors, e.g., dipyridamole, cilostazol, and Aggrenox, during interventional techniques. Recommendations for discontinuation of antiplatelet therapy with platelet aggregation inhibitors, including clopidogrel, ticlopidine, and prasugrel, are variable with clinical judgment to continue or to discontinue based on factors such as patient's condition, the planned procedure, risk factors, and cardiologist's input. Low-molecular-weight heparin or unfractionated heparin may be discontinued 12 h prior to performing interventional techniques. Warfarin should be discontinued or international normalized ratio be normalized to 1.4 or less for high-risk procedures and 2 or less for low-risk procedures based on risk factors. It is also recommended to discontinue Pradaxa for 24 h for paravertebral interventional techniques in 2–4 days for epidural interventions in patients with normal renal function and for longer periods of time in patients with renal impairment, and to discontinue rivaroxaban for 24 h prior to performing interventional techniques [1]. Finally, consider holding agents that can cause additive or synergistic effects, such as fish oil or herbal products which can interfere with the coagulation cascade, etc.

Clinical Pearls

- Discontinue chronic warfarin therapy 4–5 days before spinal procedure—the INR should be within the normal range at the time of procedure.
- No contraindications with aspirin or NSAIDs.
- Thienopyridine derivatives: clopidogrel should be discontinued at least 7 days prior to procedure, and ticlopidine should be discontinued at least 14 days before a procedure.
- GpIIb/GpIIIa inhibitors should be discontinued to allow recovery of platelet function prior to procedure (8 h for tirofiban and eptifibatide, 48 h for abciximab).
- Thrombolytics/fibrinolytics: an extremely high risk of bleeding; neuraxial techniques should be avoided; no recommendations for timing of catheter removal in those patients who unexpectedly receive fibrinolytic or thrombolytic therapy. Follow fibrinogen level to assist in timing of catheter removal and observe for signs of neural compression.
- LMWH: delay procedure at least 10–12 h from the last dose of thromboprophylaxis LMWH. For "treatment" dosing of LMWH, at least 24 h should elapse prior to procedure. LMWH should not be administered until 6–8 h after neuraxial needle or catheter placement. Therapeutic LMWH should not be given until 4 h after catheter removal.
- Unfractionated SQ heparin: patients receiving twice daily dosing of 5000 U can have neuraxial anesthesia with little increased risk. There are no contraindications to neuraxial procedure if total daily dose is less than 10,000 U. For higher dosing regimens, manage according to intravenous heparin guidelines.
- Unfractionated IV heparin: delay spinal puncture 2–4 h after the last dose; document normal aPTT. Heparin may be restarted 1 h following procedure.
- There should be a low threshold for obtaining an MRI if a patient demonstrates progressive neurological impairment.
- The incidence of clinically significant spinal hematoma has been estimated to be <1 of 150,000 epidural anesthetics and <1 of 220,000 spinal anesthetics for noncardiac surgical cases.

- During preoperative assessment, have patients discontinue products that can potentially interfere with the coagulation cascade, such as herbal products, fish oil, etc.
- There is an ASRA app that can be downloaded which will provide guidance on how long to hold any medication that has a blood thinning effect. The app divides procedures from low, medium and high risk procedures.

Review Questions

1. A bleeding risk score is useful for estimating which of the following?
   (a) Physicians can estimate bleeding risk based on patient- and technique-specific factors.
   (b) Physicians can estimate the amount of blood a patient may lose during an intervention pain procedure.
   (c) This score may support clinical decision-making about whether to cancel or carry out a procedure.
   (d) (a) and (c).
2. Neuraxial procedures are usually safe, with respect to the development of a spinal hematoma, in the absence of which of the following?
   (a) Warfarin
   (b) Aspirin or nonsteroidal anti-inflammatory drugs
   (c) Low-molecular-weight heparin
   (d) (a) and (d)
3. Which of the following is a contraindication to neuraxial anesthesia?
   (a) Oral anticoagulation
   (b) Thrombolytics
   (c) Unfractionated heparin
   (d) All of the above
4. Which of the following is considered thienopyridine derivative?
   (a) Clopidogrel
   (b) Dipyridamole
   (c) Lepirudin
   (d) Fondaparinux
5. Neuraxial procedures should be avoided for 4 weeks following a glycoprotein receptor antagonist. Which of the following is not a glycoprotein receptor antagonist?
   (a) Abciximab
   (b) Tirofiban
   (c) Eptifibatide
   (d) Prasugrel
6. Which of the following can lead to platelet dysfunction and hemostatic defects?
   (a) Renal disease
   (b) Liver disease
   (c) Vitamin K deficiency
   (d) All of the above
7. Which of the following is considered the "universal" hemostatic agent and can be used to control surgical or trauma-associated bleeding in patients with hemophilia A?
   (a) Recombinant VIIa
   (b) Recombinant VIa
   (c) Recombinant IVa
   (d) Recombinant VIIIa
8. Which of the following is *not* a characteristic of von Willebrand's disease?
   (a) It is the most common hereditary bleeding disorder.
   (b) Afflicts 1–3% of the general population.
   (c) Factor VIII levels are reduced.
   (d) The condition is not an autosomal dominant disorder.
9. Which of the following is accurate concerning interventional pain management?
   (a) Pain procedures should be done only after other interventions such as PT or oral non-opiods have failed.
   (b) Uses procedures to diagnose and treat chronic pain.
   (c) Perform procedures percutaneously that often carry a risk of bleeding.
   (d) All of the above.
10. Which of the following usually occurs after needle trauma?
    (a) A platelet plug is created in order to stop bleeding.
    (b) Glycoproteins adhere to injured endothelium.

(c) Von Willebrand factor (vWF), a protein found in subendothelial tissues, facilitates platelet adhesion.
(d) All of the above.

11. Which of the following is true concerning cyclooxygenase inhibitors?
(a) They disrupt the formation of thromboxane A2.
(b) They stimulate the production of thromboxane A2.
(c) NSAIDs irreversibly inhibit cyclooxygenase for the life of platelet.
(d) Aspirin reversibly inhibits this enzyme.
12. Thienopyridine inhibitors interfere with primary and secondary platelet aggregation due to ADP binding and subsequent activation of the GpIIb/GpIIIb receptor complex. Which of the following is not a thienopyridine inhibitor?
(a) Heparin
(b) Prasugrel
(c) Ticlopidine
(d) All of the above are thienopyridine inhibitors
13. Clopidogrel should be held for how many days prior to a neuraxial procedure?
(a) 9 days
(b) 2 days
(c) 5 days
(d) 7 days
14. Ideally, what should be sought prior to the neuraxial block?
(a) An INR less than 1.3
(b) An INR less than 2.3
(c) An INR less than 2.5
(d) An INR less than 3
15. With regard to communicating the risk-benefit analysis with patients, which of the following is true?
(a) Patients must understand that there is no way to reduce the risk of a spinal hematoma or major bleeding episode to zero.
(b) Difficult decisions about stopping anticoagulants may need to be made.
(c) Pursuing less-invasive pain therapies may be a safer choice for a patient with hemostatic disorders.
(d) All of the above are true.

Answers:

1. d
2. d
3. d
4. a
5. d
6. d
7. a
8. d
9. d
10. d
11. a
12. a
13. d
14. a
15. d

References

1. Manchikanti L, Falco FJ, Benyamin RM, Caraway DL, Kaye AD, Helm S 2nd, Wargo BW, Hansen H, Parr AT, Singh V, Swicegood JR, Smith HS, Schultz DM, Malla Y, Hirsch JA. Assessment of bleeding risk of interventional techniques: a best evidence synthesis of practice patterns and perioperative management of anticoagulant and antithrombotic therapy. Pain Physician. 2013;16(2 Suppl):SE261–318. Review.
2. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). Reg Anesth Pain Med. 2017;35(1):64–101.
3. Manchikanti L, Abdi S, Atluri S, Benyamin RM, et al. An update of comprehensive evidence based guidelines for interventional techniques in chronic spinal pain. Part II: guidance and recommendations. Pain Physician. 2013;16(2 Suppl):S49–283.
4. Meisenberg G, Simmons WH. Principles of medical biochemistry. Philadelphia: Elsevier/Mosby; 2012.
5. Rapaport SI, Rao LV. The tissue factor pathway: how it has become a "prima ballerina". Thromb Haemost. 1995;74(1):7–17.
6. Ellison N. Diagnosis and management of bleeding disorders. Anesthesiology. 1977;47(2):171–80.
7. Mannucci PM, Federici AB. Management of inherited von Willebrand disease. Best Pract Res Clin Haematol. 2001;14(2):455–62.
8. Mannucci PM, Tuddenham EGD. The hemophilias—from royal genes to gene therapy. N Engl J Med. 2001;344(23):1773–9.
9. Sallah S, Bobzien W. Bleeding problems in patients with liver disease. Ways to manage the many

hepatic effects on coagulation. Postgrad Med. 1999;106(4):187–90, 193–5.
10. Weigert AL, Schafer AI. Uremic bleeding: pathogenesis and therapy. Am J Med Sci. 1998;316(2):94–104.
11. Horlocker TT, Wedel DJ, Schroeder DR, Rose SH, Elliott BA, McGregor DG, Wong GY. Preoperative antiplatelet therapy does not increase the risk of spinal hematoma associated with regional anesthesia. Anesth Analg. 1995;80(2):303–9.
12. Cronberg S, Wallmark E, Söderberg I. Effect on platelet aggregation of oral administration of 10 nonsteroidal analgesics to humans. Scand J Haematol. 1984;33(2):155–9.
13. CAPRIE Steering Committee. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. Lancet (London, England). 1996;348(9038):1329–39.
14. Tam NLK, Pac-Soo C, Pretorius PM. Epidural haematoma after a combined spinal–epidural anaesthetic in a patient treated with clopidogrel and dalteparin. Br J Anaesth. 2006;96:262–5.
15. Doktorova M, Motovska Z. Clinical review: bleeding—a notable complication of treatment in patients with acute coronary syndromes: incidence, predictors, classification, impact on prognosis, and management. Crit Care. 2013;17(5):239.
16. O'Neill WW, Serruys P, Knudtson M, van Es GA, et al. Long-term treatment with a platelet glycoprotein-receptor antagonist after percutaneous coronary revascularization. EXCITE Trial Investigators. Evaluation of Oral Xemilofiban in Controlling Thrombotic Events. N Engl J Med. 2000;342(18):1316–24.
17. Xi M, Béguin S, Hemker HC. The relative importance of the factors II, VII, IX and X for the prothrombinase activity in plasma of orally anticoagulated patients. Thromb Haemost. 1989;62(2):788–91.
18. Hirsh J, Raschke R, Warkentin TE, Dalen JE, Deykin D, Poller L. Heparin: mechanism of action, pharmacokinetics, dosing considerations, monitoring, efficacy, and safety. Chest. 1995;108(4 Suppl):258S–75S.
19. Kaye AD, Clarke RC, Sabar R, Vig S, Dhawan KP, Hofbauer R, Kaye AM. Herbal medicines: current trends in anesthesiology practice—a hospital survey. J Clin Anesth. 2000;12(6):468–71.
20. Opatrny L, Delaney JA, Suissa S. Gastro-intestinal haemorrhage risks of selective serotonin receptor antagonist therapy: a new look. Br J Clin Pharmacol. 2008;66(1):76–81.
21. Di Nisio M, Middeldorp S, Büller HR. Direct thrombin inhibitors. N Engl J Med. 2005;353(10):1028–40.
22. Pollack CV, Reilly PA, Eikelboom J, Glund S, et al. Idarucizumab for dabigatran reversal. N Engl J Med. 2015;373(6):511–20.
23. Martin K, Beyer-Westendorf J, Davidson BL, Huisman MV, Sandset PM, Moll S. Use of the direct oral anticoagulants in obese patients: guidance from the SSC of the ISTH. J Thromb Haemost. 2016;14(6):1308–13.
24. Moen V, Dahlgren N, Irestedt L. Severe neurological complications after central neuraxial blockades in Sweden 1990-1999. Anesthesiology. 2004;101(4):950–9.
25. Ruppen W, Derry S, McQuay H, Moore RA. Incidence of epidural hematoma, infection, and neurologic injury in obstetric patients with epidural analgesia/anesthesia. Anesthesiology. 2006;105(2):394–9.
26. Landoni G, Isella F, Greco M, Zangrillo A, Royse CF. Benefits and risks of epidural analgesia in cardiac surgery. Hardman JG, ed. Br J Anaesth. 2015;115(1):25–32.
27. Rosero EB, Joshi GP. Nationwide incidence of serious complications of epidural analgesia in the United States. Acta Anaesthesiol Scand. 2016;60(6):810–20.
28. Vandermeulen EP, Van Aken H, Vermylen J. Anticoagulants and spinal-epidural anesthesia. Anesth Analg. 1994;79(6):1165–77.
29. Christie-Taylor GA, McAuliffe GL. Epidural placement in a patient with undiagnosed acquired haemophilia from factor VIII inhibitor. Anaesthesia. 1999;54(4):367–71.
30. Kang SB, Rumball KM, Ettinger RS. Continuous axillary brachial plexus analgesia in a patient with severe hemophilia. J Clin Anesth. 2003;15(1):38–40.
31. Stedeford JC, Pittman JAL. Von Willebrand's disease and neuroaxial anaesthesia. Anaesthesia. 2000;55(12):1228–9.
32. Milaskiewicz RM, Holdcroft A, Letsky E. Epidural anaesthesia and Von Willebrand's disease. Anaesthesia. 1990;45(6):462–4.
33. Dunn D, Dhopesh V, Mobini J. Spinal subdural hematoma: a possible hazard of lumbar puncture in an alcoholic. JAMA. 1979;241(16):1712–3.
34. Grejda S, Ellis K, Arino P. Paraplegia following spinal anesthesia in a patient with chronic renal failure. Reg Anesth. 1989;14(3):155–7.
35. Levine MN, Raskob G, Beyth RJ, Kearon C, Schulman S. Hemorrhagic complications of anticoagulant treatment. Chest. 2004;126(3):287S–310S.
36. Stafford-Smith M. Impaired haemostasis and regional anaesthesia. Can J Anaesth. 1996;43(5 Pt 2):R129–41.
37. Benzon HT, McCarthy RJ, Benzon HA, Kendall MC, et al. Determination of residual antiplatelet activity of clopidogrel before neuraxial injections. Br J Anaesth. 2011;107(6):966–71.
38. Wille-Jørgensen P, Jørgensen LN, Rasmussen LS. Lumbar regional anaesthesia and prophylactic anticoagulant therapy. Is the combination safe? Anaesthesia. 1991;46(8):623–7.
39. Liu SS, Mulroy MF. Neuraxial anesthesia and analgesia in the presence of standard heparin. Reg Anesth Pain Med. 1998;23(6 Suppl 2):157–63.
40. Gogarten W, Vandermeulen E, Van Aken H, Kozek S, Llau JV, Samama CM, European Society of Anaesthesiology. Regional anaesthesia and antithrombotic agents: recommendations of the European Society of Anaesthesiology. Eur J Anaesthesiol. 2010;27(12):999–1015.
41. Ho AM, Chung DC, Joynt GM. Neuraxial blockade and hematoma in cardiac surgery: estimating the risk of a rare adverse event that has not (yet) occurred. Chest. 2000;117(2):551–5.
42. Lumpkin MM. FDA public health advisory. Anesthesiology. 1998;88(2):27A–8A.
43. Layton KF, Kallmes DF, Horlocker TT. Recommendations for anticoagulated patients undergoing image-guided spinal procedures. AJNR Am J Neuroradiol. 2006;27(3):468–70.

8 Regional Anesthesia in the Community Practice Setting

Joseph Marino and Brian E. Harrington

Introduction

The appropriate management of pain has many benefits. Evidence for improved patient outcomes, in particular, has given physicians a popular and professional mandate to better manage pain [1]. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) now requires the recording of pain as a "5th vital sign." Unfortunately, there is well-publicized evidence that pain continues to be inadequately managed [2, 3].

By virtue of their clinical training, scope of practice, and historical innovation, anesthesiologists are uniquely qualified and, indeed, expected to assume a leadership role in acute perioperative pain management. As the primary practitioners of regional techniques, anesthesiologists play a critical role in the delivery of state-of-the-art multimodal opioid-sparing techniques designed to maximize pain relief while minimizing side effects [4]. Within the specialty, this has led to a renaissance in the field of regional anesthesia. Yet, effectively responding to the many challenges presented by the expansion of anesthesia practice into the realm of pain management requires a conscious effort by practitioners, especially by those in community practice whose formal training may not have adequately prepared them for this eventuality. Therefore, it is not surprising that despite evidence-based data to support their benefits, these opioid-sparing regional techniques appear to remain underutilized, especially in the community practice setting.

In many respects, the pain associated with orthopedic surgical procedures is ideally suited to a multimodal approach. The significant degree of pain associated with many orthopedic procedures warrants the time and effort of regional anesthesia. Advanced pain management is further justified as it allows many orthopedic procedures to be performed on an ambulatory basis that would otherwise require hospitalization. Preservation of oral intake usually permits the utilization of a wide spectrum of pharmacologic agents. Regional techniques are often able to be targeted at extremity pain with minimal hemodynamic effects. Finally, certain orthopedic procedures (e.g., total hip and knee arthroplasty) are performed frequently enough to warrant the development of standardized multimodal analgesic pathways.

It is easy to appreciate that what actually constitutes "community practice" is an incredibly diverse reality. Practitioners may be solo or have any number of department members (which may include subspecialty-trained physicians, CRNAs,

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_8

or nurse practitioners), with practice settings varying from hospitals to ambulatory surgery centers to office-based care. It would be impossible to address the unique issues of orthopedic pain management in each community practice circumstance. The intent of this chapter is to identify the common hurdles that exist in a community practice environment and present broad concepts and directions to overcome these hurdles to achieve a common goal: creating a culture of consistent and efficient acute pain management that extends beyond the operating room.

Identifying the Challenges

Important differences exist between academic and community practice. The realities of the modern community practice setting often present obstacles to the effective delivery of regional anesthesia. For many anesthesiologists in community practice, the issue is not whether regional anesthesia can benefit patients, but whether these techniques are realistically transportable from the academic setting into the community practice arena. While practice environments vary greatly among facilities, some generalizations include the following:

Institutional Challenges

Physicians in community practice are often wedged in a culture of conformity. A general anesthetic utilizing postoperative opiate therapy is reliable and requires less technical skill and minimal organizational adaptations. Institutions lacking leadership in acute pain medicine are poorly positioned to fully utilize the many recent advances in this rapidly growing field. Furthermore, once a culture of medical practice is established, a transformation in this culture is difficult to accomplish. Implementing regional anesthesia-based acute pain protocols under these entrenched circumstances requires considerable effort and vision. If the institutional hierarchy fails to appreciate the many benefits of advanced pain management, this lack of support may make it difficult to obtain necessary staff, supplies, and equipment. This is especially true for expensive technology like ultrasound equipment.

Community practices also frequently lack accommodating facilities commonly encountered in academic environments, such as designated areas for the performance of regional blocks (block rooms) (Fig. 8.1). The optimal timing and location for regional anesthesia under these circumstances tends to be dictated by individual circumstances (nurse and anesthesia personnel staffing, room turnover times, patient flow within a facility, available equipment, etc.). In an effort to overcome these infrastructural hurdles, physicians often must either perform regional techniques in less than desirable locations or abort the prospect altogether.

Fig. 8.1 Photo of a block area. At our hospital, epidural and peripheral nerve blockade are frequently performed in the PACU. The block area is a dedicated patient location that includes full monitoring, the regional block ultrasound unit, stimulating catheters, and a fully stocked regional anesthesia cart. It is immediately adjacent to the operating room and allows rapid turnover with minimal distraction

Time Pressures

Anesthesiologists in community hospital practice often operate in a competitive, fast-paced, high volume, fee-for-service environment. The focus of this environment is clearly on the efficient performance of surgery including operating room turnover and not the optimal management of postoperative pain. One example of the accelerated pace of community practice is the striking difference that has been noted between the median duration of surgery for private practice (1.5 h) and academic centers (2.6 h) [5]. A consequence of the high volume and accelerated pace along with the need to satisfy surgeons is the desire to avoid delays at all costs. Compounding this situation, anesthesiologists in community practice are commonly unable to be freed from a case to perform a block on their next patient. These considerations can create significant time pressures that can easily compromise the management of pain. These issues are compounded as they are set against the background of capricious insurance reimbursement and a hostile medicolegal environment familiar to all practitioners.

Surgeon Resistance

Any discussion of anesthesia choices in private practice must address the influence of surgeons, who are often considered either proponents or opponents of regional anesthesia. Just as surgical support for regional techniques can greatly facilitate their acceptance, resistance from surgical colleagues can be a significant hurdle. A 2002 survey of orthopedic surgeons found that the two principal reasons for not favoring regional anesthesia were OR delays and unpredictable success. The principal reasons for favoring regional anesthesia were less postoperative pain, decreased nausea and vomiting, and safety. If we can convince our surgical colleagues regarding the benefits of regional anesthesia, they may instead act as advocates in our mission to educate the public. These issues may be resolved with physician education, improvements in training, and organization of the regional anesthesia facility [6]. There are a myriad of opportunities for anesthesiologists to adequately position themselves in a variety of hospital settings to champion the benefits of their craft; establishing a presence in the presurgical testing is an ideal platform to set expectations and develop a collaborative analgesic plan that allows patients both to feel empowered and to be drivers of their postoperative experience.

Deficiencies in Training

Few anesthesiologists in community practice have advanced clinical training in regional anesthesia or pain management. While the training of anesthesia residents is generally adequate for spinal and epidural techniques, exposure to peripheral nerve blocks may be inadequate. Kopacz and Neal reported in 2002 that as many as 40% of anesthesiology residents in the United States may not be receiving the minimal required level of exposure to peripheral nerve blocks [7]. Given the large number of different regional techniques, anesthesiologists may complete their residency training without sufficient experience in perineural techniques to feel confident as they enter community practice. Reflecting this narrowed comfort zone, German anesthesiologists who practice in small hospitals have been shown to rely heavily on basic regional techniques, in contrast to consultants at teaching institutions [8]. The explosive growth of perineural techniques has clearly outpaced the experience of many already in practice. Given these observations, it is not surprising that anesthesiologists in community practice have been noted to perform significantly fewer peripheral nerve blocks than those who practice in teaching institutions ($p = 0.05$) [9]. Finally, those who are trained in advanced pain therapies may be *challenged* to find that many anesthesia colleagues in community practice may be uncomfortable or disinterested in providing cross-coverage for unfamiliar pain management techniques.

Personnel Issues

Many anesthesia departments in community practice settings are small or minimally staffed; assistance with blocks may be unpredictably available and involve personnel having minimal experience with regional procedures. Galvanizing the nursing staff may be the most effective alternative for anesthesiologists in community practice to cultivate a reliable first assistant with peripheral nerve blockade. During regular hours, practitioners may be largely confined to the operating room, unable to be freed from a case to perform a block on their next patient, and having limited ability to attend to the needs of hospitalized patients. In many cases, pain management coverage during odd hours may well be covered from home.

Patient Resistance

The public's fears and distorted perceptions of pain from needle passage, paralysis, and a wakeful state can also hinder the assimilation of regional techniques into daily practice. There is a serious and underappreciated risk of serious injury from parenteral opiates. The public does not understand the risks and benefits of regional anesthesia in addition to having an underappreciation of the dangers of postoperative opiates [10]. More problematic is the concept that anesthesiologists do not understand the general public's fears of regional anesthesia. This is evidenced by the finding that anesthesiologists' perceptions differed from the actual fears of interviewed patients. The anesthesiology community has not been successful in keeping the public well-informed regarding regional anesthesia. Future anesthesia-related educational programs should address the concerns of the public about anesthesia matters, particularly regional anesthesia [11].

Overcoming the Challenges

The issues presented above represent significant hurdles to the management of pain in the community practice setting and mandate a disciplined and pragmatic approach to this aspect of patient care. Successfully overcoming these hurdles requires a thoughtful and comprehensive approach.

Create a Physical Environment Conducive to Regional Anesthesia

A block room can greatly facilitate the preoperative performance of regional techniques and in one study resulted in an operating room time savings of over 20 min per case [12]. However, the economic feasibility of a dedicated block room is questionable, and a designated preoperative "block area" can be a reasonable alternative. Pressures to maintain OR flow and limit delays make the postanesthesia care unit (PACU) an excellent substitute for a block room if one does not exist. Consider isolating a single patient bay in a corner of the PACU to perform regional techniques preoperatively (Fig. 8.1). While many regional procedures can be performed with minimal assistance, each should be preceded by a "time-out." In addition to participating in the time-out, preanesthetic site verification with a signature of the involved extremity by the procedurist may help to prevent wrong-sided block errors. PACU nurses are exceptionally trained in monitoring and can serve as excellent assistants if dedicated personnel are unavailable. Furthermore, patients can be expeditiously transferred because of the PACU's close proximity to the OR. Regardless of locality, several regional anesthesia texts should be readily available wherever blocks are performed.

The efficiency of regional anesthesia is enhanced by keeping supplies together in a standardized "block cart," which has the additional advantages of being mobile and able to hold resuscitative equipment (Fig. 8.2). A sufficient supply of intralipid should be stocked wherever local anesthetics are to be used. Lipid emulsion bolus followed by infusion represents a novel resuscitation method that has demonstrated efficacy in the treatment of local anesthetic toxicity [13]. Contents of a regional anesthesia cart should now include a 500-ml vial of 20% intralipid, 60-ml syringe, and a macrodrip infusion kit. A lipid rescue algorithm (Appendix 1) should be posted on this block cart (see Fig. 8.2) to aid the

Fig. 8.2 Photo of the contents of a typical regional anesthesia cart. The cart includes catheters, stimulators, local anesthetic solutions, gowns, gloves, and prep solutions. Of importance, the cart is also stocked with resuscitative medications and intralipid solutions for emergency treatment of local anesthetic-induced cardiotoxicity

practitioner and to provide immediate visual cues in the event of an unintended intravascular injection. An educational website has been created (http://www.lipidrescue.org) and serves as an excellent instructional resource for physicians to learn about lipid emulsion therapy.

The postanesthesia care unit (PACU) serves as an important environmental "hub" in the management of acute postoperative pain. It is here that a smooth transition from surgical anesthesia to postoperative analgesia must occur. Having standardized infusion solutions for peripheral nerve blocks available in the PACU facilitates this smooth transition by greatly enhancing the ability to promptly initiate analgesic regimens. The PACU also frequently serves as the pain management communication center, where patients are identified as requiring postoperative rounds by the acute pain service. A pain management logbook or index card file (Appendix 2) usually serves this purpose.

Establish a Multidisciplinary Pain Management Team

Implementation of evidenced-based guidelines for pain management alone is inadequate to achieve advances in patient outcomes. A consistent and comprehensive approach to the management of acute pain involves the patient and every member of their care team. Success of the service is predicated on collaboration among physicians, nurses, ancillary staff, and hospital administration. The cornerstone of this interdisciplinary effort is communication. Shortcomings in the effective management of acute pain can usually be overcome through efforts to improve communication, education, and coordination of care. Integrated collaborations between the medical, nursing, and ancillary staff are needed to achieve the full benefits of an improved analgesic regimen [2, 14]. A process for inpatient postoperative follow-up is a clinical imperative. It is useful to briefly consider how anesthesiologists may effectively interact with each component of this interdisciplinary effort.

Senior Leadership

Coordination of a successful pain management program requires strong institutional support. Plans for major initiatives should be disseminated to the senior leadership at both medical and hospital board levels delineating the benefits of the service. Institutional support for pain management efforts is essential if additional staffing will be required and also necessary to obtain necessary supplies and equipment. It is of no small import in this regard that ultrasound guidance for regional anesthesia can often be viewed as an institutional revenue generator [15]. Any efforts that will look to maximize patient safety, improve patient care, enhance operating room efficiency, and decrease length of stay will certainly be embraced and highlight the efforts of the department toward developing new standards of practice and "service excellence."

Anesthesia Department

Establish a Core Group Within Your Ranks

Surprisingly, the greatest resistance to the successful integration of regional techniques in community practice may come from within the department of anesthesiology itself. A lack of interest or inexperience and consequent medicolegal concerns may lead some colleagues to

oppose implementing techniques that are perceived to require greater technical skill. The collaborative effort for the success of the initiative needs to start within the department of anesthesiology and an important core group of partners is needed to support the formation of the regional anesthesia service. Establish a minimum level of proficiency within the department by creating opportunities to mentor partners with less experience with both didactic and practical instruction. Establishing a single primary location for block placement (such as the PACU) facilitates the education of other anesthesia team members, where members can gather together, learn each other's techniques, and share information. Creating a core group of partners promotes an infrastructure of technical support making these analgesic techniques available to all patients as well as allowing the burden of work to be shared. There is encouraging evidence that motivated practitioners can successfully utilize even the most complex regional techniques in the community practice setting, as was demonstrated for ambulatory continuous interscalene blocks [16].

Appoint a Leader/Physician Champion

The challenge to overcome obstacles to regional anesthesia will tend to fall on the shoulders of one individual within the anesthesia department. Ideally, one member of the anesthesia staff will assume the role of "physician champion" for the acute pain service. While this individual may or may not be uniquely qualified by virtue of training or experience, it is essential that they possess a genuine interest in acute pain medicine as well as good communication and problem-solving skills. Let there be no mistake; the passion and persistence of one individual to persevere through the initial resistance of surgical, nursing, and anesthesia ranks is critical to the initiative! This individual must shoulder the responsibility of staff education, standardization, documentation *and advocacy.* Recognition of this individual within the institution and the department of anesthesiology as the leader in acute pain management will assure program quality and continuity.

Physician Organization

Surgeons

Surgeon acceptance of the use of regional anesthesia is critical. The fact that advanced anesthesia-based pain control methods can result in superior pain control is generally insufficient in itself to justify the additional time and effort required to generate genuine surgical support. Successful implementation of a multimodal approach to pain management is grounded in a close collaboration with surgical colleagues. Surgeons must be involved in the development of pain management protocols for their patients and, ultimately, endorse the chosen plan. Assuming responsibility for postoperative analgesia orders by the anesthesia-based acute pain service avoids the duplication of efforts by both departments as well as mitigates the presumed "burden" of managing pain from the surgical specialty. This approach also strengthens our desired perception as involved participants in patient care.

Surgeons can be the greatest advocates for the routine use of regional anesthesia and are the drivers of patient acceptance; an effective platform for this advocacy can be the creation of a standing multidisciplinary grand rounds format with representation and attendance by both anesthesia and orthopedic colleagues [17]. As noted above, patients will tend to be more receptive to regional techniques if they are introduced to the possibility by their surgeons. Identifying which surgeons are supportive of the initiative before implementing the service to the entire department will ensure acceptance of the techniques and increase success. The survey mentioned above regarding resistance to regional anesthesia among orthopedic surgeons provides some valuable insight into the rationale involved [6]. Although surgeons reported predictable concerns with regional anesthesia regarding operating room delays and unpredictable success, when data were reanalyzed, investigators found that these perceptions of delays or success rate were surprisingly not predictive of their preferences for regional anesthesia [18]. Instead, they found that a surgeon's preference for peripheral nerve

blocks for his or her own surgery strongly predicted their preference for his or her patients. Importantly, a significant number of surgeons would want peripheral nerve blocks for some surgical procedures but not others, probably based on perceptions of how painful a surgery may be. These data serve to emphasize the value of discussing procedure-specific anesthesia choices with surgeons, focusing on what they would want for their anesthetic if they were the patient and why. Finally, distribution of educational pamphlets delineating analgesic options in addition to highlighting the multidisciplinary collaboration between the two disciplines is an effective way to demonstrate our ownership of success in this initiative.

Nonsurgeon Physicians

Primary care physicians are intimately involved in the care of many sicker patients postoperatively and also commonly deal with acutely painful but nonsurgical conditions. Education of these practitioners can, through a clearer understanding of the benefits and limitations of anesthesia-based pain management modalities, generate appropriate referrals and improve the quality of care. Presentation at medical grand rounds is an effective means of efficiently educating these providers.

An often overlooked area of pain management in hospitals is the emergency room. There is ample evidence that pain continues to be inadequately managed in the ER setting and could be improved upon [19]. The early performance of a fascia iliaca block for patients with hip fractures, for example, is a safe and simple intervention that can control pain and minimize opioid use in a frail, elderly population [20]. Anesthesiologist attendance at an emergency room departmental meeting can be one means of educating emergency physicians and help expand the service beyond the operating room. Creation of a "code hip" process where the admission of a hip fracture triggers a contemporaneous anesthesia consult for both expedient operative intervention in addition to evaluation of pain management modalities is an example of this multidisciplinary collaboration.

Nursing Staff

Optimal analgesia requires careful therapeutic fine-tuning to maximize the benefits and minimize the risks and side effects of therapy, necessitating an organized service beyond the operating room [21]. Nursing staff support is an implicit prerequisite to the viability of an anesthesia-based acute pain management service. While physician leadership is required to champion the goals of the service in a physician-directed nurse-delivered model, the nursing staff is empowered to assess, manage, and ultimately treat the patient. Regardless of the diversity that exists in the variety of anesthesia staffing models, this arrangement creates an infrastructure of support resulting in close patient surveillance preventing the occurrence of any analgesic gaps. Establishing this link allows advanced regional techniques to be safely utilized in any institutional setting.

Analgesic protocols and order forms serve as an extension of the physician (Appendices 3–5). The nursing staff utilizes these guidelines as an instrument for the ongoing care of the patient. Implementing a nursing assessment flow sheet has been a valuable tool to allow our nursing staff to both monitor as well as intervene along an algorithmic decision tree to facilitate care (Appendix 6). Although certain institutions have found optimal function with the addition of a clinical nurse specialists specially trained in pain management, our experience has demonstrated that floor nurses can accomplish our goals of continuous monitoring and adjustment of therapy without the need for additional personnel.

It is important that the degree of insight by nurses into acute pain management modalities extends deeper than the physician orders. While written orders should clearly delineate nursing responsibilities, nurses should also understand the rationale for pain management choices and appreciate the nuances of each. Direct involvement by the department of anesthesiology in nursing education is one means of effectively preparing hospital staff for full participation in the management of acute pain. The didactic instruction should include a comprehensive description of the normal side effects and complications from regional anesthesia techniques,

simulation of a collaborative team approach to the expedient treatment of local anesthetic systemic toxicity (LAST), care for/troubleshoot catheters and infusion pumps, and the delineation of discharge instructions to patients (Appendix 7). A system for follow-up with outpatients must also be established (with a phone call from nursing generally being sufficient). A formal process of continuing education where the nursing staff is credited with continuing education units (CEUs) maintains the integrity of the service and ensures optimal nursing assessment and management skills.

Given the large number of nurses required to fill all shifts and the inevitable turnover of staff, institutions should plan for continuous training in pain management protocols. A video presentation, even as simple as a recording of an inservice provided by anesthesia staff, can be an effective tool for ongoing nursing education. The hospital newsletter can also be an effective vehicle to communicate certain pain control issues to nursing as well as all hospital staffs.

Ancillary Staff

The department of physical therapy plays a crucial role in the transition from the acute postoperative period to eventual functional outcome. Better management of pain facilitates more aggressive physiotherapy regimens, which may improve outcomes and decrease hospital length of stays [22]. Physical therapists need to be educated regarding the potential for motor blockade with lower extremity regional techniques and how this may impact ambulation. Inpatient falls with consequent injury are viewed by our regulatory bodies as hospital-acquired conditions reflecting poor quality with the possible consequence of financial penalties for the institution. Advances and innovations in the field of regional anesthesia (i.e., adductor canal blocks vs. femoral nerve block, periarticular infiltration of local anesthetic) continue to address the association between quadriceps blockade and fall risk while driving equianalgesic outcomes launched on a platform of perineural blockade and multimodal analgesia [23].

While pharmacists are often viewed as being somewhat removed from direct patient care, their involvement is essential to a smoothly operating acute pain management system. Standardizing the volume and concentration of analgesic infusion solutions can help reduce the risk of medication error. Stocking supplies of premixed standardized infusion agents in a convenient location (e.g., the PACU) is more efficient than an on-demand system for pharmacy and also helps to ensure the timely availability of solutions. Using appropriate sterile procedures, pharmacists may also be able to fractionate certain agents into clinically useful amounts (e.g., 1 mg preservative-free clonidine into 100 μg single-dose volumes).

Due to the variability in staffing models that exist in a variety of community practice settings, assistance with blocks may be unpredictable. Ancillary personnel have become an integral part of preanesthetic site verification to prevent wrong-sided block errors. With specialty training in monitoring and respiratory function, the recruitment of recovery room personnel and respiratory therapists can effectively accomplish many goals; they can become critical components of the preprocedure "time-out," monitor patients during and after block placement, and provide effective support during emergency situations.

Multimodal anesthetic techniques can improve discharge predictability and accelerate discharge eligibility. If social services are not involved early in the patients' perioperative course, these advantages can go essentially unrecognized. Preoperative patient education sessions describing the perioperative course may help to overcome common social delays in discharge (nursing home placement, patient transportation, lack of home readiness by family members, patient concerns resulting in requests for extended hospital stay), facilitating early discharge planning. Engaging the social service department in a comprehensive patient care plan at the beginning of hospital admission allows for the timely discharge of patients [24].

The Public

Informed patients, through more accurate perceptions and realistic expectations, enable the successful management of their own acute postoperative pain. Due to the limited opportunity for anesthesiologists to establish rapport in the rapid operating room environment, early preoperative patient education is desirable. Patients who are first informed of pain management techniques by their surgeon (e.g., interscalene block for shoulder surgery, adductor canal/femoral block for knee surgery) are more likely to be readily accepting of anesthesiology-based pain management pathways.

Despite limited personal contact, there are a variety of approaches through which anesthesiologists may preoperatively educate the public: procedure-specific pain management literature can be made available in surgeons' offices, anesthesiologists can contribute to or attend "joint replacement classes," and patients may be directed to appropriate sources of information. Websites sponsored by the American Society of Anesthesiologists (http://www.asahq.org) and American Society of Regional Anesthesia and Pain Medicine (http://www.asra.com) have useful areas dedicated to patient education.

Finally, it is essential that anesthesiologists rapidly and clearly communicate acute pain management plans during the preoperative visit. The general public has many misconceptions regarding anesthesia and pain management that are often best discussed in a one-on-one manner [10].

Formulate and Implement an Acute Pain Management Plan

The community practice environment mandates a pragmatic, team approach to pain management. This will maximize the likelihood of satisfactory analgesia while minimizing risks to patients or compromise the smooth delivery of care. Ideally, a well-formulated plan will prove to be sufficient from the outset and not require further intervention. Important concepts in this regard include:

Multimodal Analgesia

Since the pathophysiology of pain is a complex of interrelated systems, one method of analgesia alone is usually not sufficient to provide optimal pain relief. Simultaneously utilizing several approaches for analgesia takes advantage of additive and synergistic effects of different pharmacologic drug classes and has the potential to provide superior pain control, avoid analgesic gaps, and minimize adverse effects (notably those associated with opioids). Available evidence, although limited, strongly supports this concept of multimodal analgesia. The American Society of Anesthesiologists Task Force on Postoperative Pain Management, which included members from a spectrum of practice environments, concluded in its practice guidelines for acute pain management in the perioperative setting:

> Whenever possible, anesthesiologists should employ multimodal pain management therapy. Unless contraindicated, all patients should receive an around-the-clock regimen of NSAIDs, COXIBs, or acetaminophen. In addition, regional blockade with local anesthetics should be considered. Dosing regimens should be administered to optimize efficacy while minimizing the risk of adverse events. The choice of medication, dose, route, and duration of therapy should be individualized [4].

These evidence-based recommendations serve to reinforce several points. First, overreliance on opioid analgesia in the postoperative period is to be avoided. Second, simple nonopioid measures like acetaminophen and NSAIDS/COXIBS should not be overlooked [25]. Third, whether employed for surgical anesthesia or not, regional blocks are an essential component in the optimal postoperative management of pain. Finally, any analgesic plan, including established clinical pathways, must be tailored to each individual patient.

Finally, while regional anesthesia is a high profile component of multimodal analgesia, anesthesiologists must not lose sight of the potential benefits of multimodal therapy even in the absence of regional techniques. Several important aspects of acute pain management, generally outside of the direct administration by

anesthesiologists, should be mentioned. These include infiltration of the wound with local anesthetic (as a one-time procedure or continuously administered [26], which may allow for patient-controlled boluses) and intra-articular agents (e.g., intra-articular morphine) [27]. Another consideration is the preoperative administration of analgesics (usually orally) whose duration would be anticipated to extend into the postoperative period, such as extended-release opiates (e.g., extended-release oxycodone) or anti-inflammatories (e.g., celecoxib). Other less well-established adjunctive modalities such as ketamine, gabapentin, and clonidine are being actively investigated and may assume greater importance in the future. As recently demonstrated for pregabalin, there may also be significant promise for these and other agents in the prevention of chronic postoperative pain [28].

Clinical Pathways

Surgical procedures that entail complex perioperative processes have long been identified as fertile ground for improving the quality and coordination of medical care. There is evidence that procedure-specific "clinical pathways," which delineate a standardized multimodal, multidisciplinary care process, can improve efficiency and quality while preserving patient satisfaction. Many orthopedic procedures, especially total joint arthroplasties (e.g., hip and knee), are extremely well-suited for such management.

Anesthesiologists in community practice are encouraged to standardize their contributions to care in a procedure-specific fashion where, for example, every knee replacement procedure receives a perineural approach to the femoral nerve (adductor canal/femoral block) and every shoulder replacement receives a perineural approach to the brachial plexus (interscalene block) utilizing identical equipment, supplies, and labeled syringes on each patient. Starting the discussion of perioperative routines in the surgeons' office and later confirming these options during the preanesthetic visit begins to establish a habitual course of action where the pathway is familiar to patients and caregivers. With variability minimized, standardization of the service instills familiarity and reliability in the process, which saves time and reduces the risk of iatrogenic errors.

Usually, multimodal pathways for orthopedic surgeries prominently feature regional anesthesia. Optimal management of pain, largely accomplished through the addition of regional techniques, can help minimize complications while facilitating aggressive physiotherapy, which can result in improved functional outcomes and decreased hospital length of stays [29]. Rather than assume a lead role in the design of standardized protocols, physicians in community practice are encouraged to investigate the current practice at academic centers. Many leaders in the development orthopedic care maps have published their experiences (e.g., the Mayo Clinic) [30]. Analyzing and adapting such protocols from academic centers, which have been used successfully on a large scale, is likely to prove safe and effective in the community hospital environment [31] (Appendix 8). Recent updated evidence-based recommendations are also available for several common orthopedic procedures on the PROSPECT website (http://www.postoppain.org) and published in recent review articles [13, 32].

Judicious Use of Regional Blocks

While it may be possible to perform a regional technique that may be useful for virtually any orthopedic procedure, anesthesiologists in community practice are encouraged to exercise appropriate judgment and restraint (particularly in settings where regional anesthesia is not routine). This means that practitioners must carefully pick their battles and often limit regional blocks to what would be considered to be "essential" and ideally require minimal time and effort.

Situations where basic blocks result in obvious patient benefits (the "low-hanging fruit") should be considered to be the foundation for regional acceptance within an institution. It is easy, for example, to generate a consensus of support for a perineural approach to the femoral nerve (adductor canal/femoral block) after total knee arthroplasty. Momentum generated through

a single routine can then be used to further promote regional techniques for other indications.

In the community practice environment, management should be streamlined whenever possible. While combinations of peripheral blocks may be necessary to provide complete pain relief following certain surgeries, single block approaches are generally more practical. The lack of functional improvement with the addition of sciatic block following total knee arthroplasty, for example, makes the perineural approach to the femoral nerve (adductor canal approaches/femoral block) alone an attractive choice in community practice [33]. The addition of periarticular infiltration of local anesthetic has been demonstrated to further reduce opioid requirements and reduce pain scores and may have a place as another ingredient in the multimodal recipe for joint replacement pain [34]. Likewise, although catheter techniques can provide superior long-term pain relief, single-shot blocks are generally preferred unless severe pain is expected to extend for several days.

Conceptually, the approach to regional blocks in a community practice setting is often starkly pragmatic when compared to an academic environment. Practitioners should thoughtfully consider specific regional blocks in light of the following three "ideal" attributes: a single-injection site, short needle (50 mm or less), and supine positioning. Blocks that have high success rates with single injections are clearly preferable to blocks that rely on delivery of local anesthetic to multiple locations. Supra- and infraclavicular blocks are thereby able to be performed more expeditiously than multiple-stimulation axillary block. Efficacy can also be improved through knowledge of optimal target responses for successful block with single-injection sites (i.e., posterior cord stimulation with infraclavicular block [35] and tibial nerve stimulation for popliteal block [36]). Blocks that can be done using short needles are able to be more quickly performed and tend to be associated with fewer needle passes, less patient discomfort, and possibly lower complication rates. The ability to maintain the supine position generally allows for patient care to proceed along a usual flow, despite sometimes necessitating the use of longer needles (e.g., lateral popliteal block [36] or anterior sciatic block [37]).

Keys to Success with Regional Anesthesia in Community Practice

Given the realities presented above, it is apparent that the successful performance of regional techniques is critical to an anesthesia-based acute pain service. Yet the modern community practice environment can often make these techniques seem impractical, if not impossible, to put into practice. Successfully performing and expanding the use of regional anesthesia under such circumstances requires a pragmatic approach, which can be summarized as follows:

Operate Within the "Comfort Zone"

Start slowly. Each institution has its own "comfort zone," which, while capable of being expanded, should not be violated. The overzealous forcing of change is rarely sustainable, as lasting change will only take hold through popular support. The evolution of acute pain management, with the integration of new modalities, usually necessitates an incremental culture change. This progression must be accompanied by appropriate communication and education.

In general, and especially with new approaches to acute pain, it is ideal that these modalities require minimal attention outside of the operating room and normal working hours. The availability of concomitant intravenous patient-controlled analgesia (IV PCA), in particular, is a major consolation when initiating more advanced nonopioid pain management modalities (i.e., single-injection or continuous nerve blocks). The patient-titrated nature of IV PCA has the advantage of minimizing nursing care while being capable of independently providing adequate postoperative analgesia. The extent of IV PCA use (or more accurately, the extent to which it was not used) also to some degree reflects the efficacy of nonopioid techniques being simultaneously utilized.

Once a "comfort zone" for the concomitant use of postoperative opioids is established, a transition to extended-release oral opiates as seen in published analgesic care maps can obviate the need for parenteral use and its consequent side effects (Appendix 8).

Operating within the comfort zone also means that practitioners should strive to gain sufficient experience with single-injection options before taking on continuous techniques and develop familiarity with pain management innovations in inpatients before extending their use to ambulatory patients.

Learn in a Logical Progression

Given the large number of different regional techniques, it is apparent that few anesthesiologists will have sufficient experience during residency with peripheral nerve blocks to feel broadly confident as they enter community practice. Considered in its proper perspective, regional anesthesia training must be viewed as an introduction to a lifelong commitment to further learning. Just as an anesthesiologist must acquire experience when a new inhalational agent is marketed, they should approach overcoming deficiencies in regional anesthesia training with the same intellectual curiosity. Effectively removing surgical pain from the equation along with the unpleasant side effects of opioids is where regional anesthesia has evolved. The explosive growth that orthopedic anesthesia has witnessed should not mandate specialty training for regional techniques to be implemented in community practice. Just as we have not created a subspecialty for the placement of arterial lines or administration of total intravenous anesthesia (TIVA), we do not need specialty training for perioperative blocks. Every anesthesia provider should be able to perform these techniques if they are willing to choose so.

It is easy to appreciate that some regional procedures (e.g., spinal anesthesia) are more readily mastered than others. All anesthesiologists possess some regional skills and should therefore strive to expand their regional anesthesia practice in a stepwise manner. They should take care not to violate institutional or their own personal comfort zones, but rather seek to reasonably expand these zones. With this concept in mind, regional procedures have been classified into basic, intermediate, and advanced categories [38]. An awareness of this stratification can help practitioners develop competence and confidence with regional techniques in a logical progression. Proficiency with manual skills is developed through practice, and skills learned with one block will generally build confidence with all regional procedures. Anesthesiologists should liberally utilize regional techniques in appropriate clinical situations, not just when it is crucial that they work.

In any practice setting, regional anesthesia is heavily dependent upon appropriate patient selection as well as a working knowledge of the relevant anatomy and block risks and benefits. A brief review of anatomy, block technique, side effects, and potential complications should precede every regional block as practitioners strive to solidify their knowledge base. Initially, a reasonable goal is to become proficient in three or four blocks, knowing that skills learned in one technique will have a crossover to others. Continuous techniques are always more advanced than single-shot blocks and should be reserved until comfort is attained with more basic procedures. Continuous femoral nerve block deserves special mention, as it is the most commonly performed continuous technique and is particularly appropriate for pain management following total knee arthroplasty. Novices should consider continuous femoral block as the ideal "training ground" to develop comfort and familiarity with all continuous perineural techniques.

Incorporate Ultrasound into Your Practice

Anatomical diversity in patients coupled with a challenging body habitus has led some practitioners with marginal regional experience to navigate through an attempt at regional blockade with trepidation in a "poke and hope" approach.

Unpredictable block success, patient discomfort, and technical delays will negatively reinforce future attempts at perineural techniques.

Advances in the science of regional anesthesia have seen the technique of nerve location progress from utilizing paresthesias to nerve stimulation to ultrasound guidance. Ultrasound guidance of regional anesthesia is currently an area of intense interest and has created the potential of simplifying peripheral nerve blockade. The prediction of Dr. Alon Winnie many years ago was: "Sooner or later someone will make a sufficiently close examination of the anatomy involved, so that exact techniques will be developed" [39]. While it is not yet viewed as the gold standard, the literature suggests that this technology may be capable of improving the efficiency and efficacy of regional blocks [40]. Compared to nerve stimulation techniques, ultrasound-guided blocks are performed more quickly, using less local anesthetic, with fewer needle passes as well as a reduced incidence of vascular puncture [41]. The increase in current thresholds caused by the injection of conducting solutions hampers the ability to instantly reinject local anesthetic after a failed block. By confirming local anesthetic spread around the target nerve or perivascular anatomy, ultrasound can overcome this phenomenon of electrical interference and offers practitioners a powerful tool for block rescue and the potential for increased block success. Furthermore, ultrasound guidance provides the practitioner with a renewed opportunity to perform interventions on patients difficult to stimulate with the peripheral nerve stimulator (i.e., diabetic patients).

Visualizing the relationships between nerves and other structures in "real time" is an appealing aspect of ultrasound-guided regional anesthesia as we can finally see the anatomy of our target nerves. This visual feedback gives the practitioner the ability to assess the anatomic variations in a particular patient's individual anatomy. This improved visual model has the potential to empower and energize practitioners to expand the use of regional techniques in community practice. Despite the fact that the vast majority of anesthesiologists in community practice are untrained in ultrasound use, proficiency may be quickly attained through one of many hands-on courses currently offered by recognized experts easily accessed through the ASA/ASRA websites.

Keep Regional Blocks in Proper Perspective

While studies published from academic centers often compare regional to general anesthesia, in reality there is no need to compare or contrast these complementary techniques. Intraoperatively, regional block is usually best viewed as a supplement to general anesthesia and an integral component of a balanced anesthetic. Even in situations where regional anesthesia could conceivably serve as a sole anesthetic, a planned light general will compensate for delays in onset and occasional block failure. This perspective eliminates the problem of blocks that are not necessarily failures but may be inadequate to stand alone as a sole analgesic.

In the community practice arena, regional anesthesia is usually best thought of as being primarily used for postoperative analgesia. This approach accelerates the start of surgery and reduces the need for postoperative opiates, facilitating a more rapid discharge. This is consistent with the recommendations of the ASA Task Force on Acute Pain Management, which advocate consideration of regional blockade "whenever possible." Once this advantage is recognized, the surgical staff welcomes the slightly longer start times used to implement regional techniques as their prolonged analgesic effects translate into reduced phone calls for analgesic intervention.

Dealing with Block Failures

Plans for regional anesthesia often suffer from a failure to consider reasonable alternatives in a timely manner. Visualizing success with regional anesthesia is in many ways similar to management of the airway. If plan A (laryngoscopy) does not meet with success, then plan B (LMA) and

even C (fiber-optic bronchoscopy, etc.) should be pursued. Likewise, if certain regional techniques are not proceeding smoothly, they can be appropriately followed by "plan B" blocks. Difficulties with infraclavicular or femoral blocks can be expeditiously addressed by performing axillary and fascia iliaca blocks, respectively. Wound infiltration with local anesthetic by the surgeon is usually a reasonable plan C option.

Practitioners must also have a realistic perspective on abandoning frustrating unsuccessful efforts at regional block in a timely manner. Although beneficial in many respects, regional techniques are rarely essential for patient care, and stubbornly persisting with attempts at regional anesthesia in difficult situations is seldom in the best interests of the patient. Acknowledging acceptance of an alternative plan is often a sign of sound clinical judgment and the mark of a mature practitioner.

In the event of a true block failure that becomes evident in the postanesthesia care unit (PACU), reattempting the same block is usually not considered prudent. However, incomplete pain relief in some anatomic regions may be adequately covered by similar techniques. Failure of interscalene and femoral blocks, for example, can be safely and effectively followed by suprascapular [42] and fascia iliaca blocks [20], respectively. More selective distal blocks are often ideal following the failure of a more proximal block (e.g., ulnar, median, or radial blocks at the elbow after failed brachial plexus blocks).

Be Cost-Conscious

Anesthesiologists must be knowledgeable regarding the hospital cost of supplies and consistently choose cost-efficient means of providing pain control. Incorporating considerations for cost awareness is a subcompetency of one of our core competencies: systems-based practice. While few supplies are essential, practitioners are faced with a number of important choices whenever regional techniques are contemplated. Opponents of ultrasound will claim that the initial investment in machinery is prohibitively expensive. Increased block success and a reduction in complications that accompanies visualization of the needle shaft and tip can more than overcompensate for the initial cost of the machine. The addition of local anesthetic adjuvants may obviate the need for continuous catheter techniques further reducing the cost of supplies. Costs may also be reduced through the use of a prep sponge and sterile towel pack instead of a commercially manufactured block tray, choosing bupivacaine over ropivacaine as circumstances permit, and utilizing reusable pumps as opposed to disposable infusion devices.

In this era of cost containment, the conscious and purposeful choice of supplies can help to justify the more frequent use of regional techniques. Furthermore, the economical use of equipment may also make practitioners less hesitant to appropriately abandon a difficult (i.e., time-consuming and possibly futile) block procedure.

Avoid Delays (Even the Perception of Delays)

The production pressures mentioned above require that practitioners ensure that regional techniques not be perceived as a cause of delays. On the contrary, a systematic multimodal approach to acute pain management, which includes regional analgesia, should be viewed as the ideal strategy to improve efficiency through "fast-tracking" (bypass of phase 1 recovery) and speeding discharge readiness [43].

Regional techniques must be performed expeditiously. When performing regional blocks, anesthesiologists should develop a reasonable degree of "clock consciousness" and may find it a useful exercise to occasionally time themselves. As a general rule, single-injection techniques should be able to be completed within 10 min and continuous techniques within 15 min. Practitioners who are unable to perform regional techniques within these parameters should strive to improve their skills when extra time can be easily afforded, such as before the first case of the day or postoperatively in the PACU. The first case of the day generally presents an ideal opportunity to perform

blocks in a preoperative area. Preoperative performance also allows for greater "soak time" and evaluation of block effects.

In an effort to avoid delays, anesthesiologists in community practice may elect to perform regional anesthesia in anesthetized or heavily sedated patients. Practice has been noted to vary widely in this regard. While performing regional anesthesia on insensate patients may ensure guaranteed cooperation and maximize flexibility in the timing of these procedures, it may also expose the patient and practitioner to unnecessary risk. Anesthesiologists should be aware of the recent practice advisory on this subject [44]. In this advisory, the authors acknowledge that the decision to perform regional anesthesia under these circumstances is "controversial, complicated, and must be made in the absence of traditional forms of evidence-based medicine." Notably, interscalene block is the only regional technique explicitly contraindicated in anesthetized or heavily sedated patients.

Documentation

In order to create an environment conducive to the optimal management of pain, anesthesiologists must effectively take ownership of the task. The department of anesthesia should generate any orders necessary for pain management and be intimately involved in any modification of hospital policies and nursing duties in this regard. The ultimate goal should be to raise the profile of anesthesiology such that any pain management issues within the institution are naturally directed to the department.

Proper documentation is an essential component of modern medical care. Documentation of pain management techniques primarily serves as a basic communication tool between anesthesiologists and all other members of the care team. However, the ramifications of accurate descriptions of interventions performed for the management of pain extend well beyond the clinical setting and are of obvious importance as legal records and to satisfy billing and regulatory requirements.

Most institutions require that patients provide written informed consent for anesthesia care, which is separate from surgical care. Practitioners may wish to obtain additional consent for pain management procedures, which can be considered apart from surgical anesthesia care. Procedures performed for postoperative pain are considered separate from the anesthesia care provided for surgery. As such, these procedures should be documented on a form separate from the anesthesia record. The key elements to a standardized peripheral nerve block procedure note form have been described and analyzed [45]. Dedicated procedure notes have been developed for both peripheral nerve blockade [45] and neuraxial techniques [46], which can be readily combined into a single form (Appendix 9). These forms can be transcribed into the electronic medical record if computerized physician order entry is used in a particular institution.

Finally, the importance of documentation in the context of reimbursement cannot be overstated. Several aspects of the procedure note are specifically included to address reimbursement issues. Namely, the form should specifically state that the procedure was performed for the purpose of postoperative analgesia (not surgical anesthesia), the indication for pain control (i.e., the location of pain being treated rather than the surgical procedure performed), and that anesthesia-based pain management has been requested by the attending surgeon (some have advocated obtaining the surgeon's signature on this form to more fully document this request). While the issue of reimbursement for pain management services involves a multitude of variables and is beyond the scope of this discussion, it is fair to state that proper reimbursement begins with proper documentation.

Following Through on an Acute Pain Management Course

Proper follow-through is a duty of ownership and critical to the long-term success of any patient care program. Efforts by anesthesiologists which clearly extend to the conclusion of care are

necessary to maximize benefits and minimize risks associated with acute pain management and will ensure the highest levels of satisfaction from both patients and surgeons.

Follow-Through for Outpatients

Adequate analgesia is an obvious prerequisite for ambulatory surgery, where inadequate pain control has been shown to be a common reason for prolonged postoperative stays and unanticipated admissions. Furthermore, it is essential to anticipate pain-related issues that may become evident following discharge in ambulatory patients as inadequate pain management has been shown to be a leading and preventable cause for readmissions [47]. In ambulatory surgery, regional techniques including single-injection and continuous perineural catheters provide improved analgesia, less opioid-related side effects, and the potential for earlier discharge [48].

Successfully caring for patients on an ambulatory basis requires that an individualized plan be devised for the ongoing multimodal management of pain. Outpatients should be provided with written instructions concerning further out-of-hospital management of their pain (e.g., oral analgesics), precautions regarding the care of an insensate limb (if they have had regional blocks), and a 24-h telephone contact number should they have any problems or concerns (Appendix 10). Patients discharged with continuous perineural infusions must have explicit instructions regarding the care of an indwelling catheter and should be capable of discontinuing the catheter at home without necessarily returning for personal medical attention.

Each institution must establish a system for follow-up with outpatients. As alluded to above, a brief telephone call 24–72 h postoperatively, usually by a nurse, is generally sufficient. General questions regarding patient satisfaction with intraoperative anesthesia and postoperative analgesia should be asked and any degree of patient dissatisfaction promptly passed on to the department of anesthesiology through established channels. The essence of these follow-up efforts should be documented and maintained by the department of quality management for a reasonable period of time (but does not necessarily need to be placed in the patient's permanent medical record) (Appendix 10). If efforts by telephone are unsuccessful, a card may be sent by mail to the patient explaining that reasonable attempts were made to establish routine postoperative follow-up by telephone and encouraging the patient to provide feedback regarding their perioperative experience either by telephone or in writing.

Follow-Up for Inpatients

Hospitalized patients, by virtue of their higher acuity of illness and injury, may stand to benefit the most from the effective management of pain through minimizing complications and possibly preventing chronic pain. Following up on inpatients is a primary function of an acute pain service. It has been repeatedly acknowledged that there is no consensus regarding the optimal structure or function of an acute pain service [49]. In the diverse reality of community practice, an acute pain service may take many forms but must at least consist of involved physician (e.g., anesthesia) and nursing personnel.

Nurses are at the core of inpatient follow-up and are empowered to assume the leading role in assessing and treating postoperative pain. Regular assessment of pain, commonly every 4 h utilizing a 0–10 pain rating scale, is noted on pain assessment flow sheets which serve to track the "5th vital sign" (i.e., pain) over time and record responses to treatment (see Appendix 6), although such documentation is now often computerized. Multimodal treatment of pain based on scores >4 is usually included in standing pain management orders. This approach has been used successfully in many practice settings and shown to result in improved pain control and patient satisfaction, but can also be associated with an increased incidence of opioid-induced oversedation [50]. This oversedation is usually preceded by a gradual decrease in the patient's level of consciousness, which underscores the critical importance of frequent clinical assessment by nursing.

Written/computerized entry orders are necessary to enable nurses to assume the leading hands-on role in the treatment of acute postoperative pain. Orders should be devised for each of the three basic anesthesia-based modalities: intravenous PCA, central neuraxial techniques (subarachnoid and epidural), and peripheral nerve/plexus blocking techniques (see Appendices 3–5, 7). Dedicated orders are recommended for each approach as this provides the clearest direction to nursing staff and serves to emphasize important difference between central and peripheral techniques, such as anticoagulation issues and the addition of other analgesics. Orders should allow for prudent adjustments of each of the primary modalities as well as provide direction for the addition of supplemental or adjunctive measures preventing any analgesic gaps. The coordination of postoperative pain management orders with the department of surgery avoids the duplication of services preventing overdosage and adverse drug interactions.

With the exception of patients receiving IV PCA, all patients enrolled in the acute pain service must be seen by anesthesia staff on a daily basis. This visit serves as a single-time assessment of pain management as well as an important opportunity to interact with nursing staff. Support of and collaboration with nursing staff can be the tipping point of success in a community-based regional anesthesia practice. A proactive effort to address any nursing-related concerns regarding pain management at this time can alleviate a number of night and cross-coverage issues. Anesthesiologists should also use postoperative visits as a means of extracting the greatest amount of experience from each pain management intervention (e.g., the efficacy and duration of single-injection blocks). Documentation of daily pain management follow-up should be placed in the patient's chart as well as submitted for billing purposes. One successful approach to the various documentation requirements has been the development of a carbon copy peel-and-stick form, where the procedure with billing codes is documented at the top, a self-adhesive daily "SOAP" format note can be placed in the progress notes, and the carbon copy submitted for billing purposes (Appendix 11). Adaptations of this note may be transposed into an electronic format to blend the needs of computerized order entry and patient follow-up. Alternatively, using an index card system, notes may be *recorded* directly in the patient's chart and, at the conclusion of pain service involvement, the updated index card submitted for billing of daily pain management.

Although the acute pain service in many community practice settings is not a formal, distinct entity, prompt 24-h coverage is essential. Instructions for appropriate contact of anesthesia personnel should be included in all pain management orders. An acute pain service beeper can help maintain continuity of communication within a system. If in-house anesthesia coverage is available, then an on-call physician manages overnight pain-related issues. If in-house overnight coverage is not available, then a mechanism that provides for off-hour patient evaluation needs to be devised. One solution is to specifically train selected night shift nursing personnel to evaluate and troubleshoot common issues concerning acute pain management (for continuous infusions, e.g., this would include occlusion alarms, catheter disconnections, and evaluation of skin entry sites).

Management of Complications

The ideal management of complications begins with the tacit acknowledgment that complications are inevitable. Having realistic preoperative discussions with patients regarding potential complications, obtaining meaningful written informed consent, and keeping accurate records comprise the foundations of appropriately dealing with adverse events. The traditional model of anesthesia care involved the placement of regional techniques with the "occasional" participation in postoperative pain management. The surgeons' office was frequently used as the "middle man" to manage block-related complications. Unhappy patients coupled with a lack of knowledge regarding block-related sequelae created an adversarial relationship between the two working disciplines. Adopting a "patient-centric"

approach where the anesthesiologist collaborates closely with the surgical staff on any postoperative block-related issues creates a cooperative approach to the management of complications. Furthermore, taking ownership of our interventions will certainly result in a more vigilant approach improving procedural efficacy.

One goal of any anesthesia-based acute pain service should be to promptly and directly deal with any adverse outcomes potentially related to pain management. Certain complications should be anticipated and managed proactively. Making contact with patients, either personally or by telephone, into a routine part of postoperative care will help to ensure the consistent and early discovery of any complications. If any potential complications of acute pain management are first encountered by nursing personnel, they should be reported without delay to designated anesthesia personnel (as well as to the surgeon's office).

Human beings make mistakes, distractions are ubiquitous, and memory fails during stressful situations. Medication errors, wrong-sided nerve blocks, and misconnected continuous infusions are examples of errors that can result in patient harm and threaten the viability of a regional anesthesia service. The above examples are all preventable errors which are problems in search of system solutions; therefore, an annual review of the system process by the physician leader is warranted in order to maintain the integrity of the service and promote a culture of safety.

A detailed discussion of the multitude of possible complications associated with acute pain management is beyond the scope of this chapter. Since appropriate management of complications will depend on individual circumstances, it is critical that each be personally evaluated. Fortunately, most potential adverse events are rare and/or self-limited. In the unlikely event of a serious complication, cultivating a professional relationship with a department of neurology can help to facilitate prompt consultations and referrals.

To a degree that would be considered appropriate, anesthesiologists are encouraged to stay involved in the care of any patients suffering adverse outcomes secondary to pain management efforts. It should be emphasized that taking an active interest in potential complications does not imply fault or negligence by anesthesiologists, but reinforces the commitment to quality health care and serves to legitimize the pain service in the eyes of other medical professionals. Continued personal communication with the patient helps to reinforce the desired message of genuine concern.

The complete management of complications secondary to pain management requires that all occurrences be compulsively included in quality improvement efforts.

Quality Improvement

A process for quality improvement (QI), also commonly referred to as quality management (QM), is a fundamental requirement of all healthcare organizations. Although QI for the department of anesthesiology largely concerns the operative period, in the case of an anesthesiology-based acute pain service, it must extend through the entire duration of management. Quality improvement efforts allow for clinically significant data concerning pain management to be collected and monitored with the goal of improving performance and enhancing patient safety. The American Society of Anesthesiologists website is an excellent resource regarding quality improvement (http://www.asahq.org). The Quality Management Template found at the ASA website, developed by ASA committees and provided without charge, serves as an indispensable guide to implementing a quality improvement program in any practice setting [51].

The ready availability of occurrence reporting forms is a key element in the consistent self-reporting of adverse events. For cases in the operating room, reporting forms are often attached to the anesthesia record. Similarly, anesthesia-specific incident reporting forms should be immediately at hand as nurses and anesthesiologists are engaged in following through on an acute pain management plan. While occurrence forms are usually completed manually, if large amounts of data will require analysis, it is advisable that these forms be capable of being scanned.

A number of computer-ready process improvement tracking tools are commercially available, with several examples provided in ASA's Quality Management Template. Although self-reporting of adverse outcomes has inherent weaknesses, it has been shown to be more reliable than medical chart review or incident reports and tends to be successful in environments where it is perceived that participation may result in improved patient care [52].

Finally, it is essential that one member of the department of anesthesiology assumes the leadership role regarding quality improvement. This individual is responsible for assuring the consistent reporting of sentinel events (a significant limitation of self-reporting), managing the appropriate analysis of data (usually consisting of at least some type of peer review), and overseeing the adoption of appropriate measures to improve performance and safety.

Conclusion

Anesthesiologists currently have the knowledge as well as the pharmacologic and technological tools necessary to successfully control postoperative orthopedic surgery pain; however, inadequate analgesia continues to be a prominent medical issue. Meeting the challenges of acute pain management in modern community practice requires a comprehensive appreciation of the entire process, physician leadership, and an organizational commitment. Incorporating regional techniques into community practice offers anesthesiologists an opportunity to extend themselves beyond the OR into all patient care areas. Primarily through the coordinated efforts of our surgical colleagues, anesthesiology and nursing staff, a culture of consistent and efficient pain management can be established in any practice setting in a physician-directed nurse-delivered model.

Clinical Pearls

- Appoint a physician leader.
- Establish a core group within the partnership.
- Identify which surgeons are supportive of the initiative.
- Empower the nursing staff.
- Create a mobile block cart and utilize the PACU as a block room.
- Think "complementary."
- Operate within your comfort zone.
- Learn in a logical progression.
- Develop "clock consciousness" and avoid delays.
- *Incorporate ultrasound into your practice*.
- Manage complications directly.

Ultrasound Pearls

- After attending a workshop, practice probe ergonomics and visualization of the anatomy on staff members on a daily basis in order to gain proficiency with ultrasound use.
- Reinforce knowledge of the anatomy by didactic review in a color atlas with ultrasound practice on live models to develop an understanding of the target structures.
- Start with simple blocks located near easily identifiable structures (i.e., femoral, interscalene).
- Learn your machine; master knobology, etc. Become familiar with the technical adjustments of the ultrasound machine. Know how to set the optimum balance of frequency, contrast, and depth.
- Using the in-plane approach where the needle shaft is visualized maximizes the chance of seeing the tip of the needle as you navigate toward the intended structure minimizing the risk of complication.

Review Questions

1. All of the following are examples of interventions used in a standard multimodal analgesic pathway except:
   (a) Acetaminophen
   (b) NSAIDs

(c) Periarticular local anesthetic infiltration of soft tissues
(d) Spinal anesthetic with continuous femoral block
(e) General anesthetic with rapid sequence induction

2. The two principal reasons for not favoring regional anesthesia when surveying orthopedic surgeons are:
   (a) Operating room delay and excessive motor block
   (b) Operating room delay and high injection pressures
   (c) Unpredictable success and medicolegal complications
   (d) Unpredictable success and operating room delay
   (e) Medicolegal complications and operating room delay
3. Success of a regional anesthesia service is predicated on:
   (a) Collaboration with ancillary staff
   (b) Implementation of evidenced-based guidelines for pain management
   (c) Minimizing wrong-sided blocks with the performance of a "time-out"
   (d) Avoiding operating room delays
   (e) All of the above
4. Contents of a standardized regional anesthesia block cart should include all of the following except:
   (a) Resuscitative medications
   (b) Endotracheal tubes
   (c) Intralipid
   (d) EMLA cream
   (e) Ester local anesthetics
5. Contents necessary for a successful resuscitation with lipid rescue include all of the following except:
   (a) 20% intralipid
   (b) Macrodrip infusion kit
   (c) 60 cc syringe
   (d) Propofol
6. Regional techniques for ambulatory surgery result in all of the following except:
   (a) Improved analgesia
   (b) Less opioid-related side effects
   (c) Potential to bypass the postanesthesia care unit
   (d) Increase use of antiemetics
   (e) Reduced incidence of readmission
7. Coordination of a successful pain management program requires strong institutional support. Didactic instruction by the department of anesthesiology in nursing education should consist of:
   (a) Care for/troubleshoot catheters and infusion pumps
   (b) Expecting quadriceps weakness as a normal component of a femoral block
   (c) How to administer intralipid for resuscitation of local anesthetic toxicity
   (d) Delineation of discharge instructions
   (e) All of the above
8. All of the following factors may explain why anesthesiologists in community practice perform fewer peripheral nerve blocks as compared to practitioners in academic institutions except:
   (a) Lack of an accommodating infrastructure
   (b) Deficient exposure during residency training
   (c) Time pressures
   (d) Patient request
   (e) Lack of assistance
9. Regional anesthetic techniques can improve discharge predictability and accelerate discharge eligibility. Social service involvement early in the patients' perioperative course can:
   (a) Overcome delays in nursing home placement
   (b) Arrange for patient transportation
   (c) Anticipate lack of home readiness by family members facilitating timely discharge

(d) Addressing patient concerns resulting in requests for extended hospital stay
(e) All of the above

10. Useful approaches when dealing with block-related complications include:
    (a) Having realistic preoperative discussions with patients regarding potential complications
    (b) Obtaining meaningful written informed consent
    (c) Keeping accurate records
    (d) All of the above

Answers:

1. e
2. d
3. e
4. d
5. d
6. d
7. e
8. d
9. e
10. d

Appendix 1: Lipid Rescue Algorithm (Fig. 8.3)

LipidRescue™

TREATMENT FOR LOCAL ANESTHETIC-INDUCED CARDIAC ARREST

PLEASE KEEP THIS PROTOCOL ATTACHED TO THE INTRALIPID BAG

In the event of local anesthetic-induced cardiac arrest that is unresponsive to standard therapy, in addition to standard cardio-pulmonary resuscitation, Intralipid 20% should be given i.v. in the following dose regime:

- Intralipid 20% 1.5 mL/kg over 1 minute
- Follow immediately with an infusion at a rate of 0.25 mL/kg/min,
- Continue chest compressions (lipid must circulate)
- Repeat bolus every 3-5 minutes up to 3 mL/kg total dose until circulation is restored
- Continue infusion until hemodynamic stability is restored. Increase the rate to 0.5 mL/kg/min if BP declines
- A maximum total dose of 8 mL/kg is recommended

In practice, in resuscitating an adult weighing 70kg:

- *Take a 500ml bag of Intralipid 20% and a 50ml syringe.*
- *Draw up 50ml and give stat i.v., X2*
- *Then attach the Intralipid bag to an iv administration set (macrodrip) and run it .i.v over the next 15 minutes*
- *Repeat the initial bolus up to twice more – if spontaneous circulation has not returned.*

If you use Intralipid to treat a case of local anaesthetic toxicity, please report it at www.lipidrescue.org. Remember to restock the lipid. Ver7/06

Appendix 2: Pain Management Log Book (Fig. 8.4)

Anesthesiology Postoperative Pain Management Procedure Record

Postoperative pain management specifically requested by_____

Medical indication (e.g. pain location) _____

"Time Out" immediately before starting procedure @ _____ correct patient ID using 2 identifiers ( )

Team members present:_____ Correct side and site ( )

Approach	Patient Condition	Skin Prep
☐ Midline ☐ Right ☐ Paramedian ☐ Left	☐ Awake ☐ Sedated ☐ Anesthetized	☐ Alcohol ☐ Chlorhexidine
	Patient Position ☐ RLD ☐ Supine ☐ Sitting	☐ Povidone-Iodine ☐ Iodophor/isopropyl
☐ Ultrasound-assisted	☐ LLD ☐ Prone	

Needle:_____ Gauge/Length_____mm ☐ Quincke ☐ Pencil-point

☐ Insulated ☐ Tuohy ☐ Short-bevel ☐ Other:_____

Single-Injection Techniques

Peripheral Nerve Blockade	Neuraxial Blockade
Block performed: _____	Technique: ☐ Subarachnoid ☐ Epidural
Technique: ☐ Infiltration ☐ Paresthesia	Approximate interspace: _____
☐ Nerve stimulation: _____mA	Epidural loss-of resistance: ☐ Air ☐ Saline
Comments:	Epidural depth: _____cm
	Comments:

Continuous Techniques

Peripheral Nerve Blockade	Neuraxial Blockade (Epidural)
Block Performed: _____	Approximate interspace: _____
Nerve stimulation: _____mA at _____depth (cm)	Epidural loss-of resistance: ☐ Air ☐ Saline
Catheter secured at skin:_____cm	Depths: Epidural _____cm Catheter_____ cm
Comments:	Comments:

Injectate			Narrative	
Local Anesthetic	[%]	Volume (ml)	☐ Blood aspirated	☐ Unanticipated CSF
			☐ Pain on injection	☐ Unanticipated paresthesia
Adjunct(s):			☐ (+) Test dose of IV / subarachnoid placement	
Epinephrine:			Comments/actions:	
☐ Incremental injection ☐ (--) Epinephrine test dose				

Performed by: _____ _____ _____ _____

Name Signature Date Time

Billings Clinic

Patient Identification

Appendix 3: Pain Management Order Sheet (Fig. 8.5)

Name:
DOB: Age: Sex:
Acct#: Religion:
MR#:
Attending MD:
Admitted on:

North Shore LIJ Huntington Hospital
North Shore-Long Island Jewish Health System

PAIN MANAGEMENT ORDER SHEET
INTRAVENOUS PCA

(Recommended for patients over 40 kg)

Allergies: _____ Height: _____ Weight: _____lb _____ kg ☐ Actual ☐ Estimated
Pregnant: ☐ Yes ☐ No Breast Feeding: ☐ Yes ☐ No

DATE: _____ TIME: _____

1. SELECT drug therapy (ONE DRUG ONLY): if questions, please contact prescriber

☐ MORPHINE 5 mg/mL	☐ HYDROMORPHONE 1 mg/mL	☐ FENTANYL 50mcg/mL
Loading dose (2-5mg) _____mg	Loading dose (0.3-0.5mg) _____mg	Loading dose (25-75mcg) _____mcg
☐ One dose only	☐ One dose only	☐ One dose only
☐ Repeat X ___, ____ minutes apart	☐ Repeat X ____ , ____ minutes apart	☐ Repeat X ____ , ____ minutes apart
PCA dose (1-2mg) _____mg	PCA dose (0.2-0.4mg) _____mg	PCA dose (10-25mcg) _____mcg
Lockout interval (5-15 min) _____minutes	Lockout interval (5-15 min) _____minutes	Lockout interval (5-15 min) _____minutes
Continuous rate (1-2mg/hr) _____mg/hr	Continuous rate (0.2-0.4mg/hr) ____mg/hr	Continuous rate (10-25mcg/hr) ____mcg/hr
Total dose _____mg in 4 hrs (50 mg maximum)	Total dose _____mg in 4 hrs (10 mg maximum)	Total dose _____mcg in 4 hrs (500 mcg maximum)

2. SUPPORTIVE therapy medication(s) while on PCA.
   For itching: Naloxone (Narcan®) 0.1mg SC q 2h PRN

   For nausea: Ondansetron (Zofran®) 4mg IVP q 6h PRN
   If ineffective after 20 minutes call anesthesiologist/prescriber

   ☐ Oxygen via nasal cannula at _____ L/min

3. While on PCA NO sedatives, opioids or other respiratory depressants are to be given, except by order of an anesthesiologist.

4. MONITOR vital signs (BP, HR, RR), sedation level, pain level and pump settings and document:
   a. q 1 hour X 2, then q 4 hours
   b. q 4 hours for duration of PCA.
   c. q 1 hour X 2 after any change, then q 4 hours

5. RESCUE: If respiratory rate falls below 6 per minute with changes in level of sedation:
   a. Stop PCA infusion pump
   b. Give naloxone (Narcan®) 0.2 mg IVP, may repeat X 1 in 5 minutes if RR remains below 6 per minute.
   c. Call prescriber immediately.

6. OTHER instructions: _____
   _____

Signature: _____ # _____ Beeper # _____

Orders verified by: _____ RN _____ RN

1PO

Rev. 4/07 #1-369

Appendix 4: Pain Management Order Sheet (Fig. 8.6)

North Shore LIJ Huntington Hospital
North Shore-Long Island Jewish Health System

PAIN MANAGEMENT ORDER SHEET
CONTINUOUS REGIONAL ANALGESIA

Name:
DOB: Age: Sex:
Acct#: Religion:
MR#:
Attending MD:
Admitted on:

Allergies: _____

Height: _____

Weight: _____lb _____ kg ☐ Actual ☐ Estimated

Pregnant: ☐ Yes ☐ No Breast Feeding: ☐ Yes ☐ No

Date: _____ Time: _____

1. Catheter site:
   - ☐ Axillary
   - ☐ Infraclavicular
   - ☐ Interscalene
   - ☐ Fascia iliac
   - ☐ Femoral
   - ☐ Popliteal
   - ☐ Psoas
   - ☐ Other (specify): _____

2. ENSURE that catheter site, infusion and tubing (no ports) are clearly labeled.
   Catheter positioned at _____ cm at skin.
   DO NOT MANIPULATE catheter.

3. DRUG:
   - ☐ Ropivacaine (Naropin®) 0.2% (2mg/mL)
   - ☐ Other: _____

4. DOSING:
   - ☐ Manual Loading (by anesthesiologist only): Dose _____ mL
   - ☐ Continuous Infusion via pump: Rate _____ mL/hr (Max. 25mL/hr).
   - ☐ Titrate: _____
   - ☐ Other: _____

5. MAINTAIN IV access during drug administration (Saline lock).

6. MONITOR and document data as per Pain Management Flowsheet q 4 hours.

7. Additional pain management:
   - ☐ PCA (see PCA order sheet).
   - Other: _____

8. CALL anesthesiologist if patient has:
   a. Inadequate pain relief.
   b. Signs of toxicity (e.g. ringing in the ears, perioral numbness or tingling, change in sedation level or mental changes).
   c. SBP above _____ or below _____; sustained heart rate above _____ bpm or below _____ bpm.
   d. Kinking or dislodgment of catheter.
   e. Catheter site problems (e.g. leaking, edema, erythema and/or signs of infection).
   f. Lower Extremity Motor Block; score of 2 or above on the 0-3 Bromage scale.

9. CONTACT anesthesiologist on call, for any problems (Ext. 2491 or 2353) if primary anesthesiologist is unavailable (after 8 pm, on weekends & holidays).

10. AMBULATE Patient may ambulate only under the following circumstances:
    a. Have a physician's order to ambulate.
    b. Registered nurse assesses the patient and verifies absence of residual weakness or motor block.
    c. Patient is able to stand without assistance.
    d. Patient must be assisted by RN, LPN or P.T. while ambulating.

Signature: _____ # _____ Telephone # _____ Beeper # _____

Orders verified by: _____ RN _____ RN

1PO

#1-370 Rev, 4/07

Appendix 5: Pain Management Order Sheet (Fig. 8.7)

North Shore LIJ Huntington Hospital
North Shore-Long Island Jewish Health System

Name:
DOB: 00 / 00 / 00 Age: Sex:
Acct#: 0000000 Religion:
MR#: 0000000
Attending MD:
Admitted on: 00 / 00 / 00

PAIN MANAGEMENT ORDER SHEET
EPIDURAL INFUSION

Allergies:_____

Height: _____ Weight: _____kg Pregnant: ☐Yes ☐No Breast Feeding: ☐Yes ☐No

The patient has an epidural catheter in place, which is to be handled by an anesthesiologist only. Patient has received:
Drug(s):_____ Time: _____ Date: _____

Do NOT administer dalteparin (Fragmin®) to any patient with an indwelling epidural catheter.
Do NOT administer dalteparin (Fragmin®) until 4h after epidural catheter is discontinued.
Please notify anesthesiologist BEFORE IV or SC heparin therapy is started.
Please notify anesthesiologist if warfarin (Coumadin®) is ordered.
Epidural catheter must be removed prior to 2nd dose of warfarin (Coumadin®).

CHECK appropriate box: ☐ Discontinue OR ☐ Continue
☐ Alprazolam ☐ Lorazepam ☐ Diazepam ☐ Zolpidem ☐ Morphine ☐ Hydromorphone ☐ Oxycodone
Other:_____

SELECT drug therapy (ONE preservative free drug ONLY) and initiate via Epidural Infusion Pump

Morphine 50 mcg/ML + bupivacaine 0.04%	**Hydromorphone 10 mcg/ML+** bupivacaine 0.04%	**Fentany 4 mcg/ML +** bupivacaine 0.04%
Continuous Rate: _____ mL/hr	Continuous Rate: _____ mL/hr	Continuous Rate: _____ mL/hr
Demand Dose (PCEA):	**Demand** Dose (PCEA):	**Demand** Dose (PCEA):
☐3mL every 10 minutes	☐3mL every 10 minutes	☐3mL every 10 minutes
☐5mL every 10 minutes	☐5mL every 10 minutes	☐5mL every 10 minutes
☐5mL every 15 minutes	☐5mL every 15 minutes	☐5mL every 15 minutes
☐_____ mL every _____ minutes	☐ _____ mL every _____ minutes	☐ _____ mL every _____ minutes

SUPPORTIVE THERAPY medication(s) while on epidural
For itching: Naloxone (Narcan®) 0.1 mg SC q 2h PRN
For nausea: Ondansetron (Zofrn®) 4 mg IVP q 6 h PRN. if ineffective after 20 minutes call anesthesiologist.
☐ Oxygen via nasal cannula at _____L/min

MAINTAIN IV saline lock for duration of epidural infusion.

RESCUE If Respiratory Rate (RR) falls below 8/min with changes in sedation level.
a. Stop infusion pump
b. Give naloxone (Narcan®) 0.2 mg IVP, may repeat X 1, in 5 minutes if RR remains below 8/min
c. Call anesthesiologist immediately

MONITOR BP, IIR, RR, sedation level, pain level and pump settings. Document on PMFS q15min x1h, then q2h for duration of infusion

CALL anesthesiologist if patient has:
a. Change in level of sedation, lethargy, increased somnolence.
b. Systolic BP less than 90
c. Evidence of airway obstruction, change in respiratory pattern, decrease in respiratory effort, respiratory rate less than 10/min.
d. Complains of weakness or numbness in lower extremities, pain, urinary retention, severe itching, severe nausea or vomiting.

CHECK and document ability to maintain motor function in lower extremities. May ambulate only under the following circumstances:
a. Have a surgical order to ambulate.
b. Registered nurse assesses the patient and verifies absence of residual weakness or motor block.
c. Patient is able to stand without assistance
d. Patient must be assisted by RN or LPN while ambulating

CONTACT anesthesiologist on call if primary anesthesiologist is unavailable (after 8 pm, on weekends & holidays).

FILL a NEW Pain Management Order Sheet **EPIDURAL INFUSION** for any change in order.

Date: _____ Time: _____

Signature: _____ # _____ Beeper #: _____

Orders verified by: _____ RN _____ RN

1PO

#1-371 Rev. 4/07; 10/07; 3/08
8/09

Appendix 6: Nursing Assessment Flow Sheet (Fig. 8.8)

Route of Infusion # 1: ______________________
Drug: ____________________________________
Concentration: ______________/mL
Initiated:

Date	Time	INIT	INIT

Discontinued:

Date	Time	Waste	INIT	INIT

Route of Infusion # 2: ______________________
Drug: ____________________________________
Concentration: ______________/mL
Initiated:

Date	Time	INIT	INIT

Discontinued:

Date	Time	Waste	INIT	INIT

ONLY PATIENTS ARE ALLOWED TO PUSH BUTTON

DATE	TIME	Loading or Bolus Dose			Continuous (basal) Infusion Dose			Demand Dose (PCA or PCEA)			INITIAL	2nd INITIAL (Witness)
		CRA	Epidural	IV	CRA	Epidural	IV	PCA or PCEA Dose	Lockout Interval	Total Delivered		

Appendix 7: Patient Instruction Sheet for Outpatients Receiving Regional Blocks (Fig. 8.9)

North Shore LIJ Huntington Hospital
North Shore-Long Island Jewish Health System

Pain Management Flow Sheet (PMFS)

Name:
DOB: Age: Sex:
Acct#: Religion:
MR#:
Attending MD:
Admitted on:

Signature/Title	Initial	Signature/Title	Initial

Allergies: _____

Patient comfort/goal level (0 to 10): _____

Date	Time	Sedation Scale	Pain Level / Scale Used	Pain Location	Characteristic	Clinical Signs	BP**	Heart Rate**	Resp. Rate	Bromage Scale	O_2 Sat***	Cardiac Monitor	Catheter Dressing	Intervention	Initial	Outcomes**** Pain Level / Scale Used	Outcomes**** Time	Outcomes**** Initial

Sedation Scale
1 Alert (Arousable by minimal stimuli)
2 Lethargic (Arousable by increased stimuli)
3 Stuporous (Arousable by vigorous stimuli)
4 Comatose (Unarousable)

Bromage Scale
0 Full flexion of knees and feet.
1 Able to flex knees full flexion of feet.
2 Unable to flex knees still flexion of feet.
3 Unable to move legs or feet.

Pain level Scale
0 – 10

Pain Rating Scale
N Numerical
W Wong/Baker faces
F FLACC

Location
A Abdominal
B Back
C Chest
E Extremity
H Head
I Incisional
P Perineal
***O** Other

Characterisitics
SP Sharp Pain
DP Dull Pain
TP Throbbing Pain
AP Aching Pain
B Burning
***O** Other

Interventions
D Drug (see MAR)
E Education/Support
I Ice Pack
H Heat Pack
M Massage
P Position Change
S Sitz Bath
***O** Other

Clinical Signs
A Anxiety
C Calm
D Diaphoresis
M Myoclonus
N Nausea
P Pruritus
R Restlessness
V Vomiting
WS Without Sign
***O** Other

Cardiac Monitor
N = No
Y = Yes

Catheter Dressing
(Epidural or CRA)
I Intact/occlusive
B Bloody
L Leaking
P Purulent
S Soiled
***O** Other

* Other document on IPN
** BP & HR q 2h for epidural
q 4h for PCA (after initial 1st 2 hours)
Once a shift for all other analgesics.
*** O_2 Saturation if applicable
**** Outcome Pain Level/Scale used: 1 hour after PO, IM, SQ, IV, change in IV PCA and all other non pharmacologic interventions

3PMF

#1-341 Rev 4/07

Appendix 8: Post-op Multimodal Pain Management Orders (Fig. 8.10)

Patient Instruction Sheet for Outpatients Receiving Regional Blocks

Your anesthesiologist is treating your postoperative pain, in part, with a regional block. Regional blocks use local anesthetics (like 'xylocaine' and 'novacaine') to make part of your body numb instead of painful. Depending on a number of factors, especially the particular local anesthetic agent used, you may experience numbness for many hours (not uncommonly up to 36 hours). In addition to numbness ("sensory block"), you may also experience significant weakness ("motor block") in the affected area.

It is important that you protect your numb limb. If your block involves the upper extremities (shoulders and arms), you should wear a sling if one has been provided and avoid sleeping on the affected side. If your block involves the lower extremities (legs), you should not try to bear weight, walk without assistance, or drive a car until all numbness has worn off.

It is normal after regional blocks to experience:

* Tenderness, mild swelling, or bruising at the site of injection
* A "pins and needles" sensation as the block wears off

And in the case of regional block performed for shoulder surgery:

* Temporary hoarseness, a droopy eyelid, and difficulty swallowing

It is usual to use other medications in combination with regional blocks to fully control postoperative pain. You should take all pain medications prescribed to you by your surgeon as directed. To avoid unnecessary discomfort, pain medications should be started before your block has fully worn off.

You should contact the on-call anesthesiologist 24 hours a day at the numbers shown below for any of the following:

* Enlarging redness or drainage at the site of injection
* Numbness lasting longer than 48 hours
* Shooting or burning pain that seems more related to the block than your surgery
* Any urgent concerns regarding your regional block

Contact numbers: Tell the hospital operator that you need to speak with the on-call anesthesiologist.

Local XXX-XXXX

Long Distance (Toll Free) 1-800-XXX-XXXX

Appendix 9: Anesthesiology Postoperative Pain Management Procedure Record (Fig. 8.11)

North Shore LIJ Huntington Hospital
North Shore-Long Island Jewish Health System

POST-OP
MULTIMODAL PAIN MANAGEMENT ORDERS

Name:
DOB: Age: Sex:
Acct#: Religion:
MR#:
Attending MD:
Admitted on:

Allergies:_____

Height:_____ Weight:_____ kg

Pregnant: - Yes No Breast Feeding:- Yes No

In conjunction with CRA (see CRA order form)

KNEE Arthroplasty
<75 YEARS OLD
Celecoxib (Celebrex®) 200 mg PO daily x 72h
Acetaminophen (Tylenol®) 650 mg PO q6h x 72h
Oxycodone SR (Oxycontin®) 20 mg PO q12h x 72h. Hold HR≤50, sedation scale ≥3
Breakthrough pain: Oxycodone 10 mg PO q6h prn mild pain (1 – 4) x 72h Oxycodone 20 mg PO q6h prn moderate pain (5 – 6) x 72h Hydromorphone (Dilaudid®) 1 mg SC q3h prn severe pain (7 – 10) x 72h
≥75 YEARS OLD
Celecoxib (Celebrex®) 200 mg PO daily x 72h
Acetaminophen (Tylenol®) 650 mg PO q6h x72h
Oxycodone SR (Oxycontin®) 10 mg PO q12h x 72h Hold HR≤50, sedation scale ≥3
Breakthrough pain: Oxycodone 5 mg PO q6h prn mild pain (1 – 4) x 72h Oxycodone 10 mg PO q6h prn moderate pain (5 – 6) x 72h Hydromorphone (Dilaudid®) 1 mg SC q3h prn severe pain (7 – 10) x 72h
HIP Arthroplasty
<75 YEARS OLD
Celecoxib (Celebrex®) 200 mg PO daily x 72h
Acetaminophen (Tylenol®) 650 mg PO q6h x 72h
Oxycodone SR (Oxycontin®) 10 mg PO q12h x 72h Hold HR≤50, sedation scale ≥3
Breakthrough pain: Oxycodone 10 mg PO q6h prn mild pain (1 – 4) x 72h Oxycodone 20 mg PO q6h prn moderate pain (5 – 6) x 72h Hydromorphone (Dilaudid®) 0.5 mg SC q3h prn sever pain (7 – 10) x 72h
≥75 YEARS OLD
Celecoxib (Celebrex®) 200 mg PO daily x 72h
Acetaminophen (Tylenol®) 650 mg PO q6h x 72h
Oxycodone SR (Oxycontin®) 10 mg PO x1 as soon as patient gets to the floor then q AM x 72h Hold HR≤50, sedation scale ≥3
Breakthrough pain: Oxycodone 5 mg PO q6h prn mild pain (1 – 4) x 72h Oxycodone 10 mg PO q6h prn mild pain (5 – 6) x 72h Hydromorphone (Dilaudid®) 0.5 mg SC q3h prn severe pain (7 – 10) x 72h
Additional Orders for Opioid Tolerant Paitents (as determined by Anesthesiologist)
Gabapentin 100 mg PO q8h
Clonidine (Catapres- TTS® -2) 0.2 mg/24h apply once weekly

Date: _____ Time: _____ Signature _____ LIP # _____

* 1 P O *

* 1 P O *

Appendix 10: Outpatient Postoperative Contact Form (Fig. 8.12)

Billings Clinic

Patient Identification

Anesthesiology Postoperative Pain Management Procedure Record

Postoperative pain management specifically requested by _____

Medical indication (e.g. pain location) _____

Proper side confirmed: ☐	Patient Condition ☐ Awake ☐ Sedated ☐ Anesthetized	Skin Prep ☐ Alcohol ☐ Chlorhexidine ☐ Povidone-iodine ☐ Iodophor/isopropyl
Approach ☐ Midline ☐ Right ☐ Paramedian ☐ Left ☐ Ultrasound-assisted	Patient Position ☐ RLD ☐ Supine ☐ Sitting ☐ LLD ☐ Prone	☐ Sterile ☐ Aseptic

Needle: _____ Gauge / Length _____ mm ☐ Quincke ☐ Pencil-point

☐ Insulated ☐ Tuohy ☐ Short-bevel ☐ Other: _____

Single-Injection Techniques

Peripheral Nerve Blockade	Neuraxial Blockade
Block performed: _____	**Technique**: ☐ Subarachnoid ☐ Epidural
Technique: ☐ Infiltration ☐ Paresthesia ☐ Nerve stimulation: _____ mA Comments:	Approximate interspace: _____ Epidural loss-of-resistance: ☐ Air ☐ Saline Epidural depth: _____ cm Comments:

Continuous Techniques

Peripheral Nerve Blockade	Neuraxial Blockade (**Epidural**)
Block Performed: _____	Approximate interspace: _____
Nerve stimulation: _____ mA at _____ depth (cm)	Epidural loss-of-resistance to: ☐ Air ☐ Saline
Catheter secured at skin: _____ cm	Depths: Epidural _____ cm Catheter _____ cm
Comments:	Comments:

Injectate		Narrative
Local Anesthetic	[%] Volume (ml)	☐ Blood aspirated ☐ Unanticipated CSF
		☐ Pain on injection ☐ Unanticipated paresthesia
		☐ (+) Test dose for IV/subarachnoid placement
Adjunct(s):		Comments/actions:
Epinephrine:		
☐ Incremental injection ☐ (-) Epinephrine test dose		

Performed by: _____ _____ _____ _____

Name Signature Date Time

Appendix 11: Peel-and-Stick Form (Figs. 8.13 and 8.14)

Billings Clinic.

Patient Identification

Outpatient Postoperative Contact Form

Patient Information (to be completed upon entry into Outpatient Surgery)	
Date: _____	Address: _____
Procedure: _____	Telephone: _____
Anethesiologist: _____	Parents: _____

Telephone Interview

Date/Time of Callback: _____

Did you have any problems after leaving the hospital?
(i.e. pain control, nausea/vomiting, incision site drainage/bleeding, fever, bowel/bladder etc.)

_____

_____

_____

Did you meet and talk with your anesthesiologist before surgery?

_____

Do you have any other questions, comments, or suggestions?

_____

_____

_____

_____

Actions

Actions taken by RN:

_____

_____

_____

_____

_____

☐ Unable to contact by telephone. Card sent to address above on _____ (date)

RN: Signature _____ Printed Name _____

HUNTINGTON HOSPITAL
ACUTE PAIN MANAGEMENT SERVICE

_____ PCA
_____ EPI
_____ CRA

Surgeon: _____ Diagnosis code:
Anesthesiologist: _____ _____
Operation: _____ ROOM NO.
Date of service: _____

HUNTINGTON HOSPITAL ACUTE PAIN MANAGEMENT SERVICE: THERAPY INITIATION

CHECK ONE:

☐ I. V. PCA	☐ EPIDURAL/NEURAXIAL	☐ PERIPHERAL NERVE BLOCK
CPT: 01997 **INITIAL SETTINGS:** ☐ Morphine ☐ Hydromorphone Continuous Rate: _____ mg./hr. Demand Dose _____ mg. Lockout Interval _____ Min. 4 Hr. Dose Limit _____	CPT ☐ Thoracic 62318 + 99231 ☐ Lumbar 62319 + 99231 ☐ Postop Pain Rx only (Daily Mgmt.) 01996 ☐ Postop Visit (Single Shot) 99231 ☐ Blood Patch 62273 **INITIAL SETTINGS:** ☐ Continuous Rate: ___ml./hr./Titrate ____ to ___ Bupivacaine: _____ % Ropivacaine _____% + Fentanyl ___ mcg./ml., Hydromorphone___mcg./ml. or Preserv. Free Morphine ____mcg./ml. ☐ PCEA Dose _____ ml. Delay _____ Min. Other: _____	Brachial Plexus ☐ Single shot 64415 ☐-59 ☐ Continuous 64416 ☐-22 Sciatic ☐ Single shot 64445 ☐ Continuous 64446 Femoral ☐ Single shot 64447 ☐ Continuous 64448 Psoas ☐ Continuous 64449 Bolus _____ ml. Continuous Rate: _____ ml./hr. Ropivacaine _____ % Other _____ During Placement: ☐ Yes ☐ No Home ☐ Yes ☐ No Paresthesia ☐ Yes ☐ No Pain on Injection ☐ Yes ☐ No Low Resistance to Inj. PCRA Dose _____ ml. Delay _____ Min.

☐ Procedure Explained to Patient including Risks/Benefits/Alternatives. Patient Consents to Procedure.

POSTOP DAY #___

HUNTINGTON HOSPITAL · ACUTE PAIN MANAGEMENT SERVICE

SUBJECTIVE: _____

Date: _____ **OBJECTIVE:** Pain Score: _____ /10

☐ PCA ☐ Epidural ☐ Peripheral nerve block ☐ Single shot neuraxial

Time: _____ ☐ Vital signs stable ☐ Alert & oriented ☐ No motor/sensory block ☐ Nausea ☐ Pruritus ☐ Headache

☐ Bromage Score _____

ASSESSMENT/PLAN: ☐ Continue current Rx ☐ Catheter removed, tip intact ☐ Further pain Rx plan _____

Provider Signature: _____ **COMMENTS:** _____

_____

_____

POSTOP DAY #___

HUNTINGTON HOSPITAL · ACUTE PAIN MANAGEMENT SERVICE

SUBJECTIVE: _____

Date: _____ **OBJECTIVE:** Pain Score: _____ /10

☐ PCA ☐ Epidural ☐ Peripheral nerve block

Time: _____ ☐ Vital signs stable ☐ Alert & oriented ☐ No motor/sensory block ☐ Nausea ☐ Pruritus ☐ Headache

☐ Bromage Score _____

ASSESSMENT/PLAN: ☐ Continue current Rx ☐ Catheter removed, tip intact ☐ Further pain Rx plan _____

Provider Signature: _____ **COMMENTS:** _____

_____

_____

POSTOP DAY #___

HUNTINGTON HOSPITAL · ACUTE PAIN MANAGEMENT SERVICE

SUBJECTIVE: _____

Date: _____ **OBJECTIVE:** Pain Score: _____ /10

☐ PCA ☐ Epidural ☐ Peripheral nerve block

Time: _____ ☐ Vital signs stable ☐ Alert & oriented ☐ No motor/sensory block ☐ Nausea ☐ Pruritus ☐ Headache

☐ Bromage Score _____

ASSESSMENT/PLAN: ☐ Continue current Rx ☐ Catheter removed, tip intact ☐ Further pain Rx plan _____

Provider Signature: _____ **COMMENTS:** _____

_____

_____

FORM 1-324 (REV. 11/05)

References

1. Hanna MN, Murphy JD, Kumar K, et al. Regional techniques and outcome: what is the evidence? Curr Opin Anaesthesiol. 2009;22:672–7.
2. Apfelbaum JL, Chen C, Mehta SS, et al. Postoperative pain experience: results from a national survey suggest postoperative pain continues to be undermanaged. Anesth Analg. 2003;97:534–40.
3. McGrath B, Elgendy H, Chung F, et al. Thirty percent of patients have moderate to severe pain 24 hr after ambulatory surgery: a survey of 5,703 patients. Can J Anesth. 2004;51:886–91.
4. American Society of Anesthesiologists Task Force on Acute Pain Management. Practice guidelines for acute pain management in the perioperative setting. An updated report by the American Society of Anesthesiologists Task Force on Acute Pain Management. Anesthesiology. 2004;100:1573–81.
5. Abouleish AE, Prough DS, Whitten CW, et al. Comparing clinical productivity of anesthesiology groups. Anesthesiology. 2002;97:608–15.
6. Oldman M, McCartney CJL, Leung A, et al. A survey of orthopedic surgeons' attitudes and knowledge regarding regional anesthesia. Anesth Analg. 2004;98:1486–90.
7. Kopacz DJ, Neal JM. Regional anesthesia and pain medicine: residency training – the year 2000. Reg Anesth Pain Med. 2002;27:9–14.
8. Heid F, Jage B, Jage J. Current practice in regional anaesthesia in Germany. Eur J Anaesthesiol. 2006; 23:346–50.
9. Gaba DM, Howard SK, Jump B. Production pressure in the work environment. California anesthesiologists' attitudes and experiences. Anesthesiology. 1994;81:488–500.
10. Weinger MB. Dangers of postoperative opioids. Anesthesia Patient Safety Foundation Newslett. 2007;21:61–8.
11. Matthey PW, Finegan BA, Finucane BT. The public's fears about and perceptions of regional anesthesia. Reg Anesth Pain Med. 2004;29:96–101.
12. Armstrong KPJ, Cherry RA. Brachial plexus anesthesia compared to general anesthesia when a block room is available. Can J Anesth. 2004;51:41–4.
13. Leskiw U, Weinberg GL. Lipid rescue for local anesthetic toxicity: is it really lifesaving? Curr Opin Anaesthesiol. 2009;22:667–71.
14. White PF, Kehlet H. Improving postoperative pain management. What are the unresolved issues? Anesthesiology. 2010;112:220–5.
15. Liu SS, John RS. Modeling cost of ultrasound versus nerve stimulator guidance for nerve blocks with sensitivity analysis. Reg Anesth Pain Med. 2010;35: 57–63.
16. Frederickson M, et al. Successful continuous interscalene analgesia for ambulatory shoulder surgery in a private practice setting. Reg Anesth Pain Med. 2008;33:122–8.
17. Kahn RL, Nelson DA. Regional anesthesia group practice in multihospital private practice settings and in orthopedic specialty hospitals. Int Anesthesiol Clin. 2005;43:15–24.
18. Masursky D, et al. Predicting orthopedic surgeons' preference for peripheral nerve blocks for their patients. Anesth Analg. 2008;106:561–7.
19. Rupp T, Delaney KA. Inadequate analgesia in emergency medicine. Ann Emerg Med. 2004;43:494–503.
20. Foss NB, Kristensen BB, Bundgaard M, et al. Fascia iliaca compartment blockade for acute pain control in hip fracture patients: a randomized, placebo-controlled trial. Anesthesiology. 2007;106:773–8.
21. Viscusi ER, Rehanna J, Warshawsky D. An acute pain management service with regional anesthesia: how to make it work. Tech Reg Anesth Pain Manage. 2002;6:40–9.
22. Sharma V, Morgan PM, Cheng EY. Factors influencing early rehabilitation after THA. A systematic review. Clin Orthop Relat Res. 2009;467:1400–11.
23. Kwofie MK, Shastri UD, Gadsden JC, Sinha SK, Abrams JH, Xu D, Salviz EA. The effects of ultrasound-guided adductor canal block *versus* femoral nerve block on quadriceps strength and fall risk: a blinded, randomized trial of volunteers. Reg Anesth Pain Med. 2013;38:321–5.
24. Hebl JR, Dilger JA, Byer DE, et al. A pre-emptive multimodal pathway featuring peripheral nerve block improves postoperative outcomes after major orthopedic surgery. Reg Anesth Pain Med. 2008;33:510–7.
25. Schug SA, Manopas A. Update on the role of non-opioids for postoperative pain treatment. Best Pract Res Clin Anaesthesiol. 2007;21:15–30.
26. Liu SS, Richman JM, Thirlby RC, et al. Efficacy of continuous wound catheters delivering local anesthetic for postoperative analgesia: a quantitative and qualitative systematic review of randomized controlled trials. J Am Coll Surg. 2006;203:914–32.
27. Gupta A, Bodin L, Holmstrom B, et al. A systematic review of the peripheral analgesic effects of intraarticular morphine. Anesth Analg. 2001;93:761–70.
28. Buvanendran A, Kroin JS, Della Valle CJ, et al. Perioperative oral pregabalin reduces chronic pain after total knee arthroplasty: a prospective, randomized, controlled trial. Anesth Analg. 2010;110:199–207.
29. Barbieri A, Vanhaect K, Van Herck P, et al. Effects of clinical pathways in the joint replacement: a meta-analysis. BMC Med. 2009;7:32.
30. Lennon RL, Horlocker TT. Mayo Clinic analgesic pathway: peripheral nerve blockade for major orthopedic surgery. Florence, KY: Taylor and Francis Group; 2006.
31. Walter FL, Bass N, Bock G, et al. Success of clinical pathways for total joint arthroplasty in a clinical hospital. Clin Orthop Relat Res. 2006;457:133–7.
32. Fisher HB, Simanski CJ, Sharp C, et al. A procedure-specific systematic review and consensus recommendations for postoperative analgesia following total knee arthroplasty. Anaesthesia. 2008;63:1105–23.

33. Pham Dang C, Gautheron E, Guilley J, et al. The value of adding sciatic block to continuous femoral block for analgesia after total knee replacement. Reg Anesth Pain Med. 2005;30:128–33.
34. Toftdahl K, Nikolajsen L, Haraldsted V, Madsen F, Tonnesen EK, Soballe K. Comparison of peri- and intraarticular analgesia with femoral nerve block after total knee arthroplasty: a randomized clinical trial. Acta Orthop. 2007;78:172–9.
35. Lecamwasam H, Mayfield J, Rosow L, et al. Stimulation of the posterior cord predicts successful infraclavicular block. Anesth Analg. 2006;102:1564–8.
36. Arcioni R, Palmisani S, Della Valle CJ, et al. Lateral popliteal sciatic nerve block: a single injection targeting the tibial branch of the sciatic nerve is as effective as a double-injection technique. Acta Anaesthesiol Scand. 2007;51:115–21.
37. Wiegel M, Reske A, Hennebach R, et al. Anterior sciatic nerve block – new landmarks and clinical experience. Acta Anaesthesiol Scand. 2005;49:522–7.
38. Hargett MJ, Beckman JD, Ligouri GA, et al. Guidelines for regional anesthesia fellowship training. Reg Anesth Pain Med. 2005;30:218–25.
39. Kapral S, Marhofer P, Grau T. Ultrasound in local anesthesia. Part 1: technical developments and background. Anaesthetist. 2002;51:931–7.
40. Griffin J, Nicholls B. Ultrasound in regional anesthesia. Anaesthesia. 2010;65(Suppl 1):1–12.
41. Neal JM, Brull R, VWS C, et al. The ASRA evidence-based medicine assessment of ultrasound-guided regional anesthesia and pain medicine. Executive summary. Reg Anesth Pain Med. 2010;35:S1–9.
42. Singelyn FJ, Lhotel L, Fabre B. Pain relief after arthroscopic shoulder surgery: a comparison of intraarticular analgesia, suprascapular nerve block, and interscalene brachial plexus block. Anesth Analg. 2004;99:589–92.
43. White PF, Kehlet H, Neal JM, et al. The role of the anesthesiologist in fast-track surgery: from multimodal analgesia to perioperative medical care. Anesth Analg. 2007;104:1380–96.
44. Bernards CM, Hadzic A, Suresh S, et al. Regional anesthesia in anesthetized of heavily sedated patients. Reg Anesth Pain Med. 2008;33:449–60.
45. Gerancher JC, Viscusi ER, Liguori GA, et al. Development of a standardized peripheral nerve block procedure note form. Reg Anesth Pain Med. 2005;30:67–71.
46. Viscusi ER, Gerancher JC, Weller R, et al. Not documented? Not done! A proposed procedure note for neuraxial blockade. American Society of Regional Anesthesia and Pain Medicine 30th Annual Spring Meeting and Workshops. Abstract 68, April 21–24, 2005.
47. Coley KC, Williams BA, DaPos SV, et al. Retrospective evaluation of unanticipated admissions and readmissions after same day surgery and associated costs. J Clin Anesth. 2002;14:349–53.
48. O'Donnell BD, Iohom G. Regional anesthesia techniques for ambulatory orthopedic surgery. Curr Opin Anaesthesiol. 2008;21:723–8.
49. Rawal N. Organization, function, and implementation of acute pain service. Anesthesiol Clin North Am. 2005;23:211–25.
50. Vila H, Smith RA, Augustyniak MJ, et al. The efficacy and safety of pain management before and after implementation of hospital-wide pain management standards. Anesth Analg. 2005;101:474–80.
51. Committee on Performance and Outcomes Measurement (CPOM), Committee on Quality Management and Departmental Administration (QMDA): Quality Management Template, http://www.asahq.org/quality/qmtemplate013105.pdf.2004.
52. Katz RI, Lagasse RS. Factors influencing the reporting of adverse perioperative outcomes to a quality management program. Anesth Analg. 2000;90:344–50.

Part III

Ultrasound-Assisted Nerve Blocks

9 Ultrasound-Guided Peripheral Nerve Blockade

Alan Bielsky and David M. Polaner

Introduction

The introduction of ultrasonographic guidance for peripheral nerve blockade has provided today's anesthesiologist with a powerful imaging tool for managing the precision and safety of needle and catheter placement and of local anesthetic injection. Due to the wide range of blocks performed, and the inability to blind observers when studying blocks, it is difficult to determine with certainty if ultrasound-guided blocks are better than other techniques, but numerous studies provide evidence that using ultrasound confers increased efficacy, lower local anesthetic requirements and improved safety compared with landmark or nerve stimulator techniques [1–11]. Randomized, controlled trials have assessed individual ultrasound-guided blocks and suggest a reduced risk of vascular puncture, improved block quality, faster onset time, and reduced time to perform the block [8–10]. Additionally, some investigations have suggested that ultrasound guidance may permit successful blockade with lower volumes of local anesthetics, which might have implications regarding reduced risk for toxicity [12, 13].

Basic principles of ultrasound-guided peripheral nerve blockade require an understanding of nomenclature, physics, and descriptions of probe manipulation and the orientation of the probe relative to the needle. An ultrasound probe uses a cyclic sound pressure beam which penetrates a medium and then measures the reflection signature, creating an image [14]. This permits the operator to visualize the inner structural details of many media, including soft tissue.

When describing an ultrasound image, one uses the terms hyperechoic, hypoechoic, and anechoic. Hyperechoic refers to a bright, white appearance of structures, while hypoechoic refers to a darker, duller appearance of structures. Anechoic refers to a completely dark appearance. Typically, tendons, nerves, and fascia appear as hyperechoic, while fat and muscle appear as heterogeneous, hypoechoic structures. Fluid, which fills the arteries and veins, appears as anechoic. Air produces a bright, hyperechoic image (Fig. 9.1).

In order to optimize images with the tools available on ultrasound machines, one must understand some basic principles. The ultrasound wave frequency can be chosen both by probe selection and by changing the settings on the machine itself. Higher-frequency beams improve axial resolution, which is the ability to distinguish between two objects at different depths in line

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_9

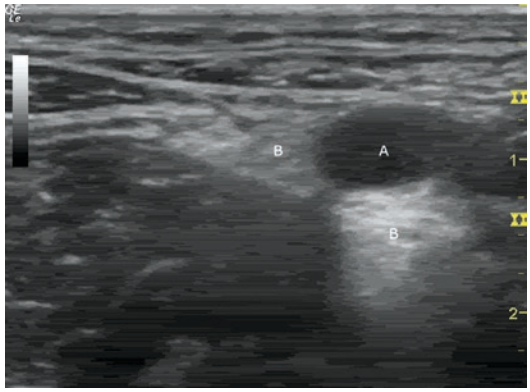

Fig. 9.1 Image of hyper- and hypoechoic. (*A*) The fluid-filled femoral artery appears as a *dark*, anechoic structure, and (*B*) the fascial structure appears *bright* and, as such, is hyperechoic

with the axis of the beam [15]. Thus, increasing the frequency of the ultrasound probe (10–13 MHz) will improve resolution of superficial structures at the expense of visualizing deeper structures. Conversely, a lower frequency will improve image quality of deeper structures at the expense of resolution of more superficial structures. The term "gain" refers to the degree to which the ultrasound machine amplifies returning ultrasound waves, making them appear brighter. Gain will increase the brightness of the entire image, but also increases artifact from background noise. "Time gain compensation" is a form of gain manipulation that allows the operator to adjust the gain at specific depths in the field. Time gain compensation is useful in filtering out background noise and focusing on the depth of the target, though it may make visualization of the needle more difficult. Altering depth penetration also can be used to enhance the image. Once a target is identified, if a greater-than-necessary depth is selected, the target will appear small due to the change in aspect ratio of the image. If the set depth is too shallow, the target may be obscured or fall deeper than the penetration of the ultrasound beam appears on the screen. The final manipulation is "focus," which allows the operator to place the focal zone of the beam at various points in the field to limit beam divergence, thereby improving lateral resolution, which is the ability to distinguish between two structures that sit side by side [16, 17].

In addition to optimizing the machine settings, an image can be enhanced by physical manipulations of the probe by the operator. The basic motions of probe manipulation are pressure, rotation, alignment, and tilt (Fig. 9.2). The needle direction in relation to the ultrasound beam can be described as in-plane or out-of-plane (Fig. 9.3). It is also useful, before needle placement, to establish the ultrasound probe's orientation in relation to the right and left sides of the screen and to center the target in the image on the screen.

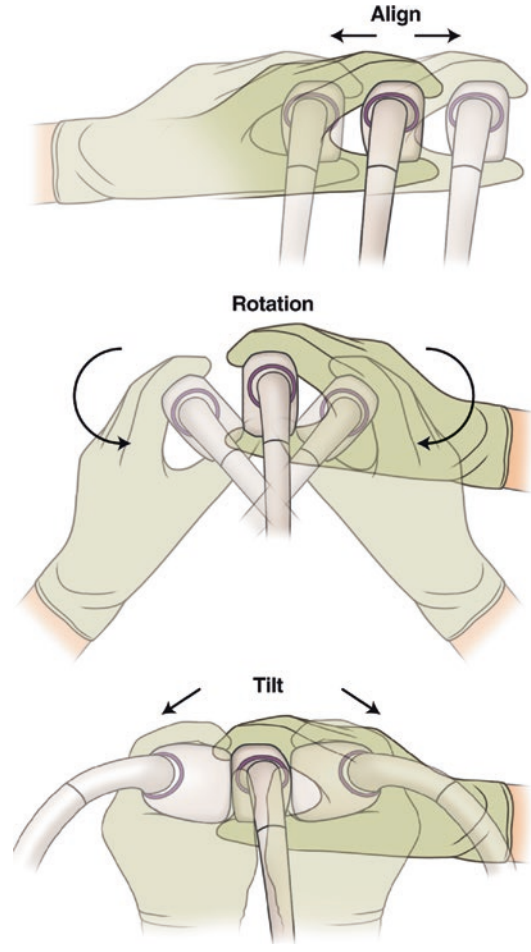

Fig. 9.2 The basic principles of ultrasound probe manipulation are pressure, rotation, alignment, and tilting

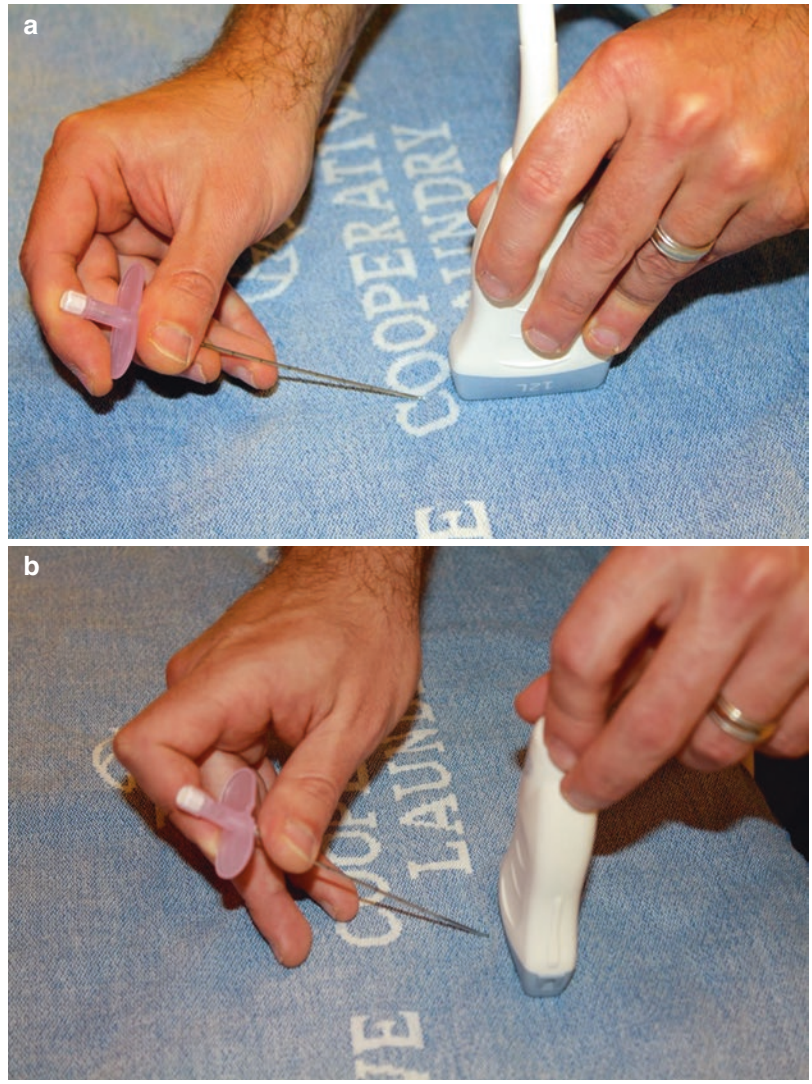

Fig. 9.3 (**a**) The needle is in-plane with the ultrasound probe. (**b**) The needle is out-of-plane with the ultrasound probe

Local Anesthetic Dosing

It is difficult to give absolute dose recommendations for specific blocks. In some cases it appears to be better to use as small a dose of drug as possible, in order to reduce the incidence of side effects (e.g., to reduce diaphragmatic paresis with interscalene block) [5]. For other blocks (e.g., transversus abdominal plane blocks), however, increasing volume while not exceeding the accepted safe limits for toxicity may improve block quality [18]. Many experts, on the basis of both empiric evidence and limited study data, recommend that ultrasound imaging can help guide the adequacy of administered volume by visualizing the spread of injected local anesthetic around the target nerves. General recommendations for average volumes of local anesthetic for specific blocks are found in Table 9.1. In all cases one must never exceed the recommended toxic limits of local anesthetic, which should be calculated in advance and must also take into account

Table 9.1 Dosing

Area of block	Suggested volume (mL/kg)	Postoperative analgesic dose	Anesthetic dose	Max dose (mL)
Above umbilicus	0.2–0.4	0.2% Ropivacaine or 0.25% bupivacaine	0.35% Ropivacaine or 0.375% bupivacaine	30
Below umbilicus	0.3–0.5	0.2% Ropivacaine or 0.25% bupivacaine	0.35% Ropivacaine or 0.375% bupivacaine	40
Compartment block (TAP, quadratus lumborum, paravertebral)	0.25	0.2% Ropivacaine or 0.25% bupivacaine	Surgical anesthetic dose cannot be obtained (not applicable)	40

Table 9.2 Local anesthetic toxic dose limits

Agent	Maximum dose (mg/kg)
Lidocaine	3
Lidocaine with epinephrine	6
Bupivacaine	2
Bupivacaine with epinephrine	2.5
Ropivacaine	3
Ropivacaine with epinephrine	4

any addition local anesthetic dose administered to the patient by the anesthesiologist or surgeon (Table 9.2). Local anesthetic systemic toxicity is a rare event during peripheral nerve blockade but must be assiduously avoided [19].

Interscalene Nerve Block

The use of ultrasound guidance in the placement of interscalene peripheral nerve blockade has been validated by studies addressing success rate, block quality, and time to perform the block [20, 21]. The interscalene nerve block aims to inject local anesthetic at the level of the trunks in the brachial plexus, thereby providing anesthesia to the upper arms and shoulder.

The trunks of the brachial plexus are most effectively accessed at the level of C6, at which location cadaver studies have shown a minimum distance of 23 mm from skin to vertebral foramen [22]. Here, the plexus passes through a compartment formed by the fascia-encased anterior and middle scalene muscles (Fig. 9.4).

To perform an interscalene nerve block with ultrasound guidance, the patient is placed supine with the head rotated between 30 and 45° away from the side of the block. After sterile preparation of the skin and the ultrasound probe, visual inspection reveals the sternocleidomastoid muscle and the thyroid prominence, which is slightly above the C6 level. A linear probe is placed on the skin overlying the sternocleidomastoid at this level in an axial oblique plane in order for the ultrasound beam to transect the plexus (Fig. 9.5). Initial ultrasonographic anatomic landmarks include the sternocleidomastoid, which can be identified by its tapering appearance as one examines more laterally. The carotid artery and jugular artery can be recognized as pulsatile, anechoic, round structures (Fig. 9.6). Moving laterally, the anterior and middle scalene muscles appear in cross section, identifiable by their round, striated nature. In between these two muscles, potentially between their hyperechoic-appearing investing fascia, lay the trunks of the brachial plexus. Adjustments should be made to the ultrasound image in order to maximize frequency and minimize field depth. The trunks appear as round, hypoechoic structures that may be separated by hyperechoic fascial septae (Fig. 9.7). The stacked linear orientation has been described as resembling a snowman.

A 2–4 cm needle is placed at the lateral border of the linear ultrasound probe and advanced medially toward the trunks under constant ultrasound visualization. The needle tip should be visualized passing lateral to the sternocleidomastoid and skirt along the medial border of the middle scalene fascia until it reaches the midpoint of the viewed trunks (Fig. 9.8). After aspiration, a small test dose of local anesthetic should be administered, with good spread being visualized around the trunks and not in muscle or vascular tissue.

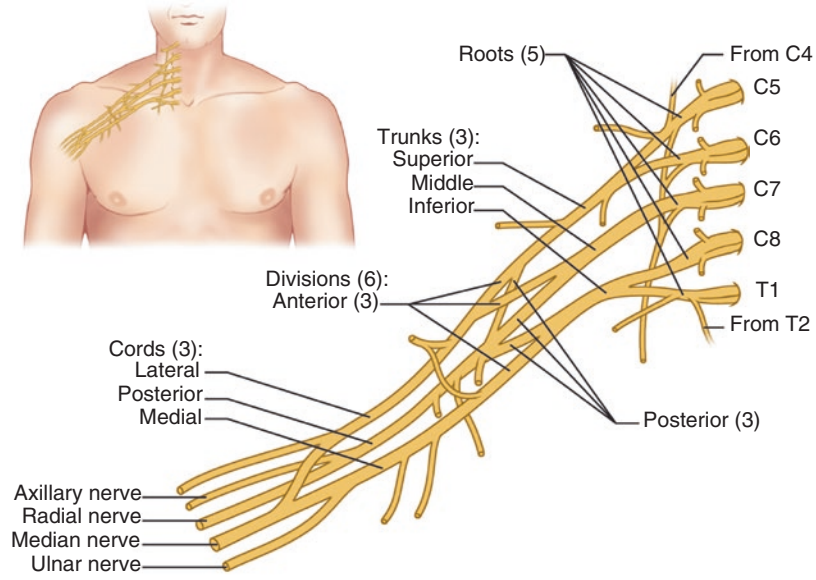

Fig. 9.4 The brachial plexus

Fig. 9.5 Performing an interscalene block

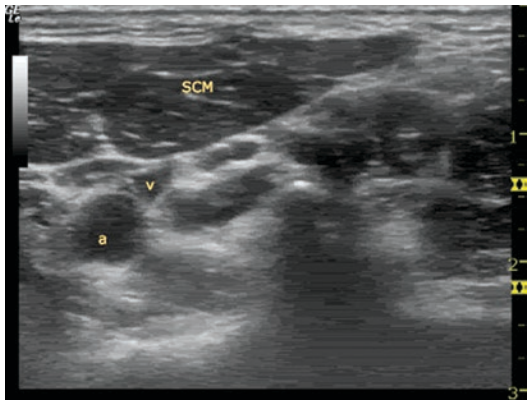

Fig. 9.6 Relationship between the carotid artery (*a*), internal jugular vein (*v*), and sternocleidomastoid (*scm*)

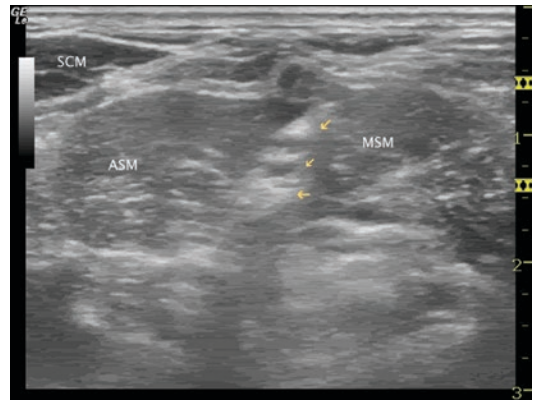

Fig. 9.7 Ultrasound image of the interscalene approach to the brachial plexus showing the trunks of the plexus. Displayed are the sternocleidomastoid (*scm*), the anterior scalene muscle (*asm*), the middle scalene muscle (*msm*), and the trunks of the brachial plexus (arrows)

Complications from interscalene nerve blockade can be dramatic due to the proximity to major vascular and neuraxial structures. Pneumothorax, spinal cord injection with resultant permanent paralysis, epidural injection, intrathecal injection, and intravascular injection are all concerns, and although rare, have been reported [23, 24]. Additionally, neck hematoma and sepsis have been described [25]. Persistent neuropathy after interscalene block has been assessed prospec-

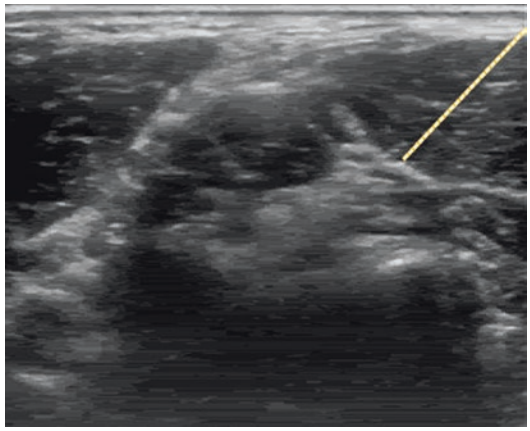

Fig. 9.8 Interscalene injection. Needle trajectory of the interscalene nerve block (dashed line)

tively and does occur with an incidence of between 4 and 16% within the first week after the block but only 0.1–0.2% permanently [6]. These numbers were not different for ultrasound-guided versus nerve stimulator-guided blocks. More common side effects may include a Horner's-type syndrome, transient vocal changes, and transient phrenic blockade. As such, patients should be advised of these phenomena preoperatively, as to alleviate postoperative concerns. Whereas phrenic nerve paresis is nearly universal when nerve stimulation is used to guide needle placement and may produce significant hypoxemia or respiratory insufficiency in susceptible subjects, reports suggest that ultrasound guidance may dramatically reduce its incidence due to the reduction in necessary volume [5].

Of particular note, interscalene nerve blockade has been considered a high-risk (or even contraindicated) procedure in the heavily sedated or anesthetized patient due to the inability of the patient to report paresthesias that could herald entry into the vertebral foramina. Recent results from an analysis of the prospective Pediatric Regional Anesthesia Network database found that the risk of performing this block using ultrasound guidance in anesthetized children bears no greater risk than that reported in awake adults [26]. Comparable data in adults are not yet available.

Supraclavicular Nerve Block

After fading away from the anesthesiologist's armamentarium due to an elevated risk of pneumothorax when performed with surface landmarks, the ultrasound-guided supraclavicular approach to the brachial plexus has gained widespread acceptance due to the ability to easily visualize and inject structures in this area. Subsequent analysis has shown an exceedingly low incidence of pneumothorax when ultrasound is employed [27]. The supraclavicular nerve block aims to anesthetize the divisions of the brachial plexus as they pass over the first rib, under the clavicle (Fig. 9.9). Here, the divisions are located posterolateral to the subclavian artery, medial to the middle scalene muscle, and superior to both the first rib and the pleura [27–29]. The supraclavicular nerve block provides fast-acting and dense anesthesia for procedures distal to the midhumerus.

To perform the block, the operator stands at either the head of the bed or facing the ipsilateral shoulder. The patient's head is turned between 30 and 45° away from the side to be blocked. After sterile preparation of the skin and probe, the supraclavicular fossa is visually identified, noting the sternocleidomastoid muscles, clavicle, and coracoid process. A high-frequency linear probe is placed in a coronal oblique plane, which can be approximated by orienting the probe roughly parallel to the clavicle. Some may find it easiest to "step off" the clavicle and let the probe seat into the supraclavicular fossa (Fig. 9.10).

Ultrasound examination begins by locating the subclavian artery in the short axis view, where, with appropriate rotation and tilting, the artery will appear as a round, pulsating, and anechoic structure. At this point, one will also see the hypoechoic first rib underneath the artery and, possibly, the pleura. Ultrasonographically, the pleura will have a mixed pattern of hyper- and hypoechoic signals due to the presence of air in the interstitium and will move with respiration, while the rib will not. Lateral to the subclavian artery, the operator will appreciate the middle scalene, which is scanned in short axis and is

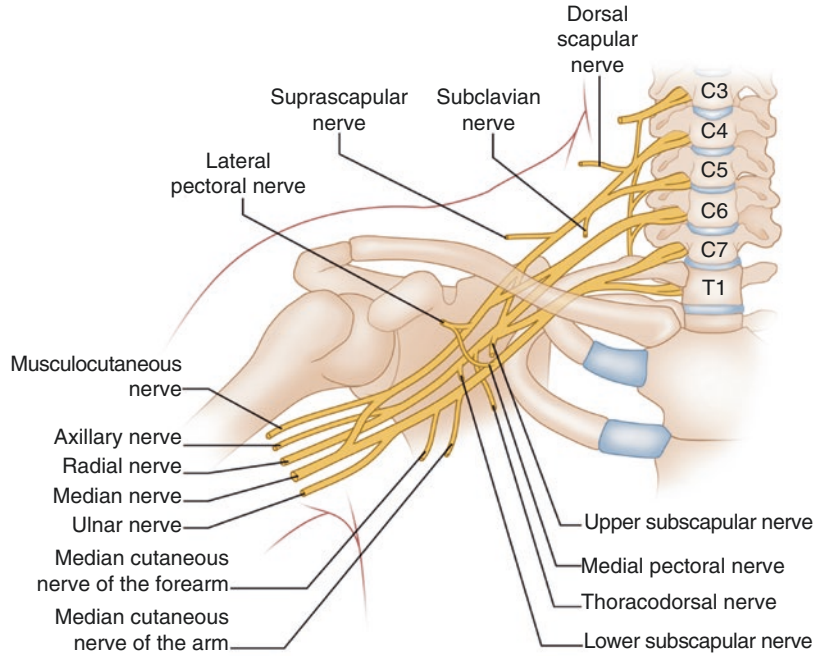

Fig. 9.9 Passage of the brachial plexus in the supraclavicular area

Fig. 9.10 Photo of performance of the supraclavicular block

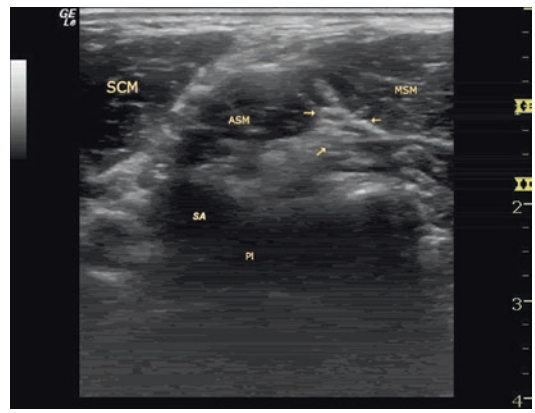

Fig. 9.11 View of the brachial plexus in the supraclavicular fossa. Note the sternocleidomastoid (*scm*), subclavian artery (*SA*), anterior scalene muscle (*asm*), middle scalene muscle (*msm*), first rib shadow and pleura (*pl*), and the brachial plexus divisions (arrows)

notable for its often-striated appearance. In between the subclavian and the middle scalene lie the divisions of the brachial plexus, which appear as a hypoechoic, grape-cluster-like structure (Fig. 9.11).

After identification of the brachial plexus in the supraclavicular fossa, the ultrasound image is optimized by increasing frequency and decreasing image depth to focus on the plexus and the first rib. The block needle is then advanced under constant visualization in an in-plane fashion along the medial border of the middle scalene, toward the lateral portion of the plexus. In order to obtain proper needle position, it is often necessary to pierce the middle scalene muscle (Fig. 9.12). Here, a test dose reveals spread of the anesthesia in the fascial layer surrounding the divisions of the brachial plexus. If indicated, subtle movements can be used to penetrate small fascial layers to provide adequate local anesthetic spread.

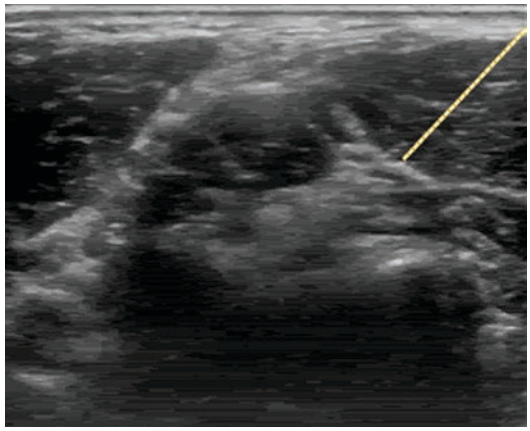

Fig. 9.12 The needle trajectory (yellow dashed line) for supraclavicular nerve block

Inherent to the supraclavicular nerve block is the risk of pneumothorax, the risk of which is reduced by cautious and deliberate needle advancement under ultrasound guidance. Other risks include intravascular injection with resultant local anesthetic toxicity, neck hematoma, and abscess. In the largest cohort reported to date, the incidence of accidental vascular puncture and transient sensory deficits were both 0.4%. Horner Syndrome and hemidiaphragmatic paresis occurred in 1% and there were no pneumothoraces [30].

Infraclavicular Nerve Block

The infraclavicular nerve block targets the brachial plexus as it emerges from underneath the clavicle. Here, the lateral, medial, and posterior cords surround the axillary artery and are easily identified by ultrasound examination. This block is utilized in surgeries distal to the midhumerus. Though quite similar to the supraclavicular nerve block, it offers benefits of diminished risk of phrenic nerve blockade and ease of catheter placement [31]. The ultrasound-guided infraclavicular nerve block has shown similar, if not improved, efficacy compared with the axillary approach to the plexus as well as greater patient comfort and willingness to undergo the same procedure when performed in the awake or unsedated subject [32, 33].

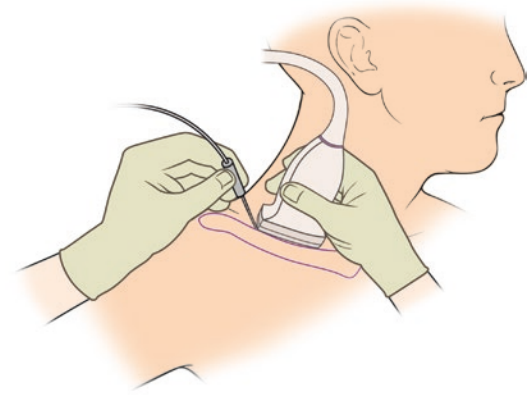

Fig. 9.13 Performing the infraclavicular block

To perform the infraclavicular nerve block, the operator preferably stands at the head of the bed. Visual inspection reveals the sternocleidomastoid, the clavicle, and the coracoid process. It may be helpful to mark these points on the patient with a soft-tip marker. Additionally, it may be useful to abduct the arm to 90°, externally rotate the shoulder, and flex the elbow (Fig. 9.13). This action may bring the plexus closer to the skin, allowing the ultrasound image to be optimized [34].

After sterile preparation of the skin, a linear ultrasound probe is placed in the coronal plane in the infraclavicular fossa. Of note, in the larger patient, a lower-frequency (8–10 MHz) ultrasound beam may be required, as it may need to penetrate deeper than for other blocks. The hypoechoic clavicle is identified, and then the axillary artery is visualized in cross section. Rotation and tilting may be used to enhance the artery's round, pulsating image. Once this is achieved, one can appreciate the two muscle layers immediately anterior to the artery, comprised of the pectoralis major and pectoralis minor. The image depth and frequency are then optimized. It is important to orient oneself to the location of the beam, specifically which side is caudal and which is rostral. Another landmark to note may be the heterogeneous lung pleura, which moves with inspiration.

In the infraclavicular fossa, the cords of the brachial plexus appear as hyperechoic star-like structures surrounding the hypoechoic axillary artery. Chan classically described the positions of

the nerve cords in relation to the axillary artery in terms of a clock face with the lateral cord located cranially at 09:00 h, the posterior cord between 06:00 and 07:00 h, and the medial cord lying at 04:00–05:00 h, often between the axillary artery and vein (Figs. 9.14 and 9.15) [29, 35].

Using an in-plane approach, a needle is guided from either the rostral or caudal end of the ultrasound probe, under constant visualization. It is often easier to insert the needle at the rostral end of the probe just underneath the clavicle, as a 45° angle is usually sufficient to initially reach the posterior cord, and, after injection, draw back to the lateral cord and inject further local anesthetic. When the needle reaches the posterior cord, injection may result in the "double bubble sign," which consists of the hypoechoic axillary artery anteriorly and the spreading local anesthetic posteriorly [34]. One may see additional spread of the local anesthetic in a u-shaped fashion along the posterior border of the axillary artery.

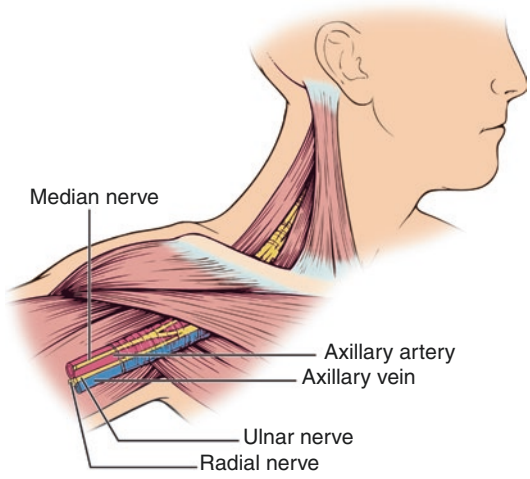

Fig. 9.14 Location of the cords around the axillary artery

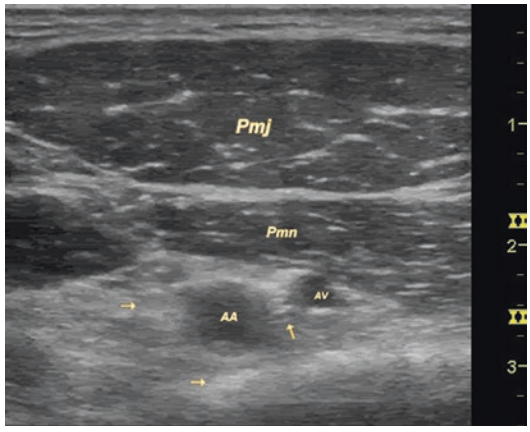

Fig. 9.15 Ultrasound appearance of the approach to the infraclavicular nerve block. The pectoralis major (*pmj*) and pectoralis minor (*pmn*) cover the brachial plexus noted by arrows, which surrounds the axillary artery (*AA*). The small axillary vein (*AV*) is located caudal to the artery

Complications associated with the infraclavicular nerve block include hematoma, infection, vascular puncture, and local anesthetic toxicity. Pneumothorax is avoided by maintaining the needle in the sagittal plane and avoiding medial movement. Minor dysesthesia has been noted in 2% of patients in large cohorts, though permanent nerve injury is only rarely described following this block [31, 33].

Axillary Nerve Block

A time-tested approach to the brachial plexus exists in the axillary nerve block, although with the advent of ultrasound guidance for the previously described brachial plexus techniques, this block has become less frequently performed. Here, the brachial plexus is blocked at the level of terminal nerves as they pass through the axilla. This technique lends itself to ultrasound guidance due to the superficial orientation of the plexus. Of note, this is an excellent "starter" block for newcomers to ultrasound-guided blocks, due to the easy visualization of structures, the ability to handle the needle with multiple passes under ultrasound guidance, and the lack of critical structures to avoid. Much like the infraclavicular block, the axillary nerve block provides anesthesia for extremities distal to the midhumerus.

To perform the axillary nerve block, the arm is abducted to 90° and externally rotated. After sterile preparation, a linear probe is placed in a parasagittal orientation in the axilla (Fig. 9.16). On initial exam, one notices the striated biceps and triceps muscles and the pulsating axillary artery. As the probe is centered over the axillary artery, the image is optimized by increasing

Fig. 9.16 Performing the axillary nerve block

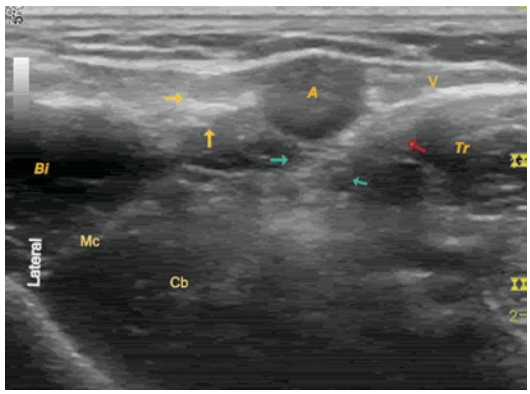

Fig. 9.17 The ultrasonography of the axillary nerve block. The axillary artery (*A*) is located centrally, bounded laterally by the biceps muscle (*Bi*) and medially by the triceps muscle (*Tr*) and compressible axillary vein (*V*). The terminal nerves surrounding the artery are the median nerve (yellow arrows), the radial nerve (blue arrows), and the ulnar nerve (red arrow). The musculocutaneous nerve (*Mc*) is separately blocked as it runs in between the biceps and the coracobrachialis (*Cb*)

frequency, adjusting gain, and reducing depth. The median, radial, and ulnar nerves are visualized as heterogeneous, typically honeycomb-shaped structures around the artery. While there does tend to be variation in location of the nerves, the median nerve is located most laterally, in close proximity to the biceps; the radial nerve tends to lie deep to the axillary artery; and the ulnar nerve lies medially, close to both the triceps muscle and the axillary vein (Fig. 9.17) [36].

An in-plane approach is used to guide a needle to the plexus. There is consensus among some experts that multiple injection passes are needed to provide adequate anesthesia to the distal upper extremity [37–39]. Typically, one may insert the needle from the lateral aspect of the brachial plexus and advance the needle to the radial nerve, which lies underneath the axillary artery, typically at a 06:00 h position. The needle is then withdrawn to the 09:00 h position, where the median nerve is injected. The current evidence suggests that selective ulnar nerve injection is not necessary for block success because diffusion of the solution within tissue planes will produce adequate blockade of that nerve [37]. Block success seems to be improved when it is easy to visualize the spread of hypoechoic local anesthetic around the nerve bundles [40].

A specific problem frequently encountered with the axillary plexus block is the early (proximal) exit of the musculocutaneous nerve, which innervates the surface of the medial arm. This frequently necessitates the direct blocking of the musculocutaneous nerve, which courses through the body of the coracobrachialis muscle. To block the nerve here, one simply places the probe on the coracobrachialis muscle in a cross-sectional fashion and finds the nerve by its hyperechoic signature within the muscle mass. This can be achieved with distal and superior scanning of the ultrasound probe on the arm. A small volume of local anesthetic then is placed at this site [41].

Complications of the axillary nerve block include hematoma, infection, vascular puncture, and local anesthetic toxicity. Nerve injury from this block is exceedingly rare [39].

Femoral Nerve Block

The femoral nerve block is utilized for procedures involving the anterior thigh and knee. This block is easy to perform given the size of the nerve sheath and its adjacent structures and finds many uses in orthopedic practice. The femoral nerve is blocked in the proximal thigh as it exits below the inguinal ligament and above both the psoas and iliacus muscles. It is easily identifiable as it courses lateral to the femoral artery, surrounded in a triangular fashion by the artery and iliopectineal ligament medially, fascia lata

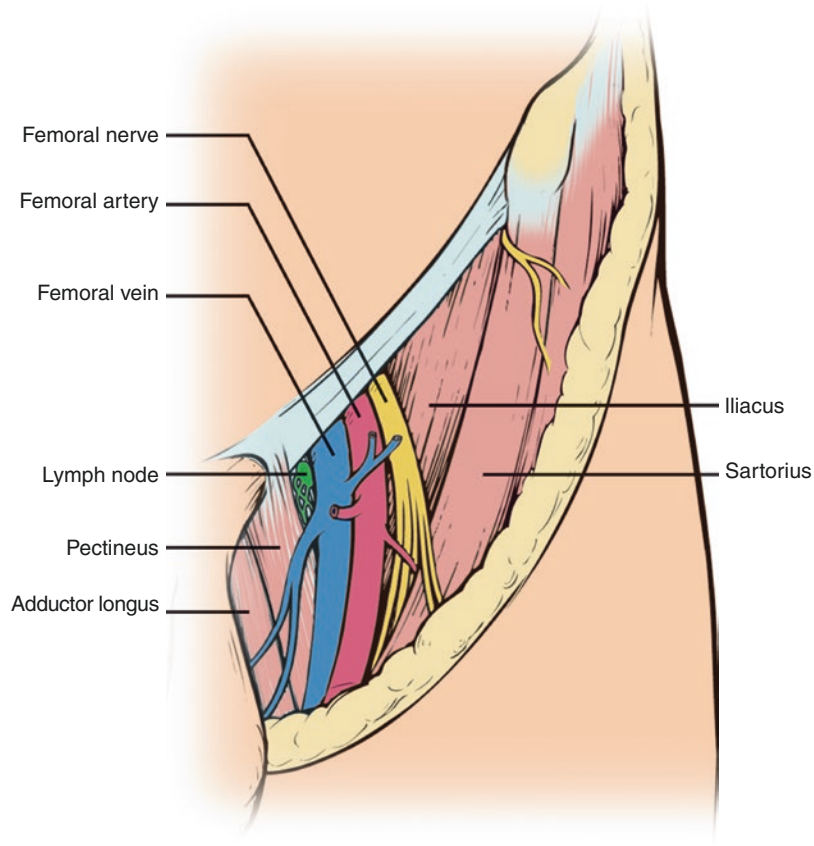

Fig. 9.18 The femoral nerve and its relation to the artery and vein

superiorly, and iliacus muscle and its investing fascia inferiorly (Fig. 9.18).

To perform the block, the patient is placed supine, with the legs in a neutral position. After sterile preparation, a linear probe is placed in a transverse orientation just distal to the inguinal ligament (Fig. 9.19). The first and most prominent landmark is the pulsating, anechoic femoral artery. The operator then moves the probe distally to observe the takeoff of the profunda femoris artery from the femoral artery. Blockade of the femoral nerve should be proximal to this landmark. The nerve is identified as a hyperechoic, heterogeneous structure located lateral to the artery. It often appears as a "comet trail," which is due to its surrounding fascia lata and iliacus muscle. If difficulty is encountered visualizing the nerve, the probe can be tilted in a caudal-rostral fashion until the image improves (Fig. 9.20) [42].

The image is optimized using frequencies between 8 and 10 MHz, by adjusting depth and gain. In an in-plane approach, the needle is passed to the inferolateral border of the "comet tail." Here, local anesthetic is deposited. Frequently, with injection the round, hyperechoic nerve becomes more visible as the local anesthetic surrounds the nerve. If there is inadequate spread of local anesthetic medially, one can

Fig. 9.19 Performing the femoral nerve block

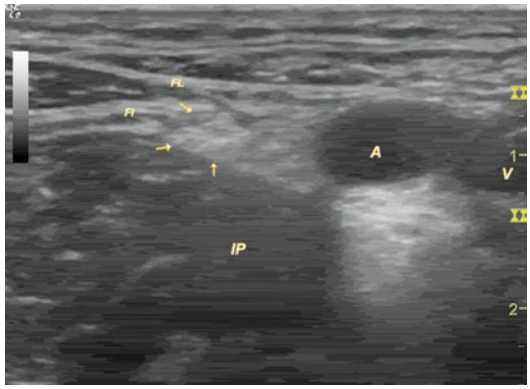

Fig. 9.20 Ultrasonographic appearance of the femoral nerve. The femoral nerve (arrows) is observed superior to the iliopsoas muscle (*IP*) and lateral to the femoral artery (*A*) and femoral vein (*V*). It is encased by the fascia iliaca (*FI*) and bounded superiorly by the fascia lata (*FL*)

reposition the needle on top of the nerve and continue injection, though it is wise to stay below the hyperechoic, linear-appearing fascia iliaca.

Complications of the femoral nerve block include hematoma, abscess, vascular puncture, and local anesthetic toxicity. Transient and permanent nerve injury after femoral nerve blockade is exceedingly rare [31].

Sciatic Nerve Block

While it is a large nerve, ultrasound imaging of the sciatic nerve is impeded by its depth and by the large amounts of tissues that surround it in its proximal region. For this reason, one must consider whether a proximal or distal sciatic nerve block is appropriate. If anesthesia of the posterior thigh is required, a proximal sciatic block should be performed. If anesthesia is only required at and below the knee, a popliteal fossa sciatic nerve block is sufficient.

The sciatic nerve is formed from the L4, L5, and S1–S3 nerve roots, leaving the pelvis via the greater sciatic foramen, deep to the gluteus maximus, and along the medial side of the femur [43]. The sciatic nerve splits in the popliteal fossa to form the tibial and peroneal nerves, which provides innervation to the lower extremity distal to the knee. As such, the sciatic nerve can be blocked proximally, near the gluteus maximus, or distally in the popliteal fossa.

Sciatic nerve blockade near the gluteus maximus poses challenges of depth to anesthesia guidance. To perform the block at this level, the patient is placed in a lateral position with the hips and knees flexed. To obtain an image, a lower-frequency (2–5 MHz) curved array probe is placed just inferior to the buttock in line with the ischial tuberosity and greater trochanter [44]. Here the sciatic nerve will be found deep to the gluteus maximus and medial to the ischial tuberosity. The depth of the nerve varies between 3 and 5 cm. The nerve appears as an elliptical hyperechoic structure that may be difficult to distinguish from the fascia surrounding the gluteus maximus (Fig. 9.21). For this reason, a stimulating needle may be of benefit in confirming its position [45]. Using an in-plane or out-of-plane technique, an insulated stimulating needle is passed to the nerve. It is wise to anticipate that a longer-length needle may be needed. Once the needle has reached the sciatic nerve, neurostimulation may reveal the need for readjustment.

At the midthigh, the sciatic nerve becomes more superficial, though it may still be difficult to see in patients with more muscle or adipose mass. In this setting, a lower-frequency (6–10 MHz) linear probe is used. The patient's leg is flexed at the knee and positioned so that the posterior

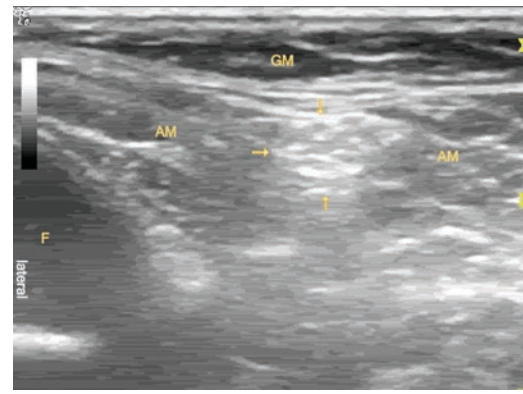

Fig. 9.21 The proximal sciatic nerve. The sciatic nerve (arrows) is bounded superficially by the gluteus maximus (*GM*) and deeper by the adductor magnus muscles (*AM*). The hypoechoic femur (*F*) serves as the lateral point of reference

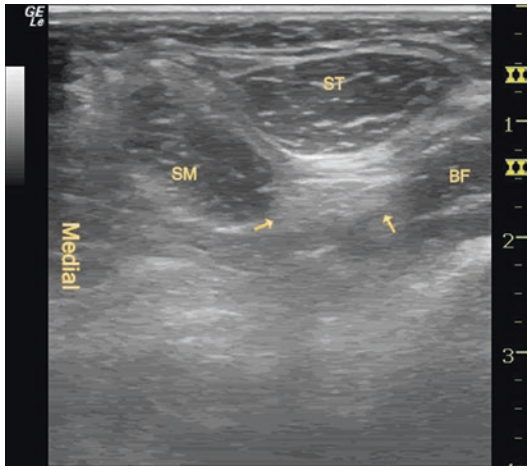

Fig. 9.22 The midthigh sciatic nerve. The sciatic nerve (arrows) lying deep to the semimembranosus (*SM*), semitendinosus (*ST*), and biceps femoris (*BF*)

aspect of the thigh is accessible to probe placement. The probe is then applied laterally to the posterior aspect of the thigh. Anatomic structures of note in the view are the biceps femoris, adductor magnus, semitendinosus muscle, and semitendinosus muscle (Fig. 9.22). The sciatic nerve appears as a honeycombed oval or elliptical structure deep to these muscles [45, 46]. If further confirmation is needed, one can use the "scan down-scan up" technique in which the operator scans, in the same plane, distally to observe the bifurcation of the sciatic nerve into the tibial and peroneal nerves. The "scan back" portion then tracks the nerve proximally and proceeds with needle placement. Needle insertion can be in-plane or out-of-plane. It is often useful to use an in-plane, trans-vastus lateralis approach. For this technique, the needle is inserted in-plane and passed in a lateral fashion from the side of the thigh (Fig. 9.23). An advantage of the trans-vastus approach is that the needle angle remains constant, allowing the operator to optimize the needle image and then advance in that optimized plane.

The popliteal fossa provides an easy location to perform sciatic nerve blockade, though it will not provide anesthesia to the posterior thigh proximally to the popliteal fossa. Here, the thick muscular tissues part, allowing easy visualization

Fig. 9.23 The trans-vastus approach for needle placement

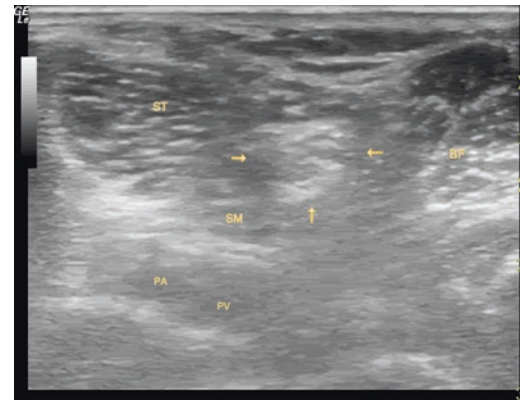

Fig. 9.24 The popliteal fossa. The sciatic nerve (arrows) is bounded laterally by the semitendinosus (*ST*) and semimembranosus (*SM*) muscles and medially by the biceps femoris muscles (*BF*). The popliteal artery (*PA*) and popliteal vein (*PV*) are also seen

of the sciatic nerve. The patient is placed in either a lateral position or a supine position with the hip flexed and the knee flexed. An 8- to 10-MHz probe is placed transversely at the posterior portion of the popliteal crease. The tendons of the semimembranosus and semitendinosus are identified medially, and the biceps femoris is located laterally. Additional landmarks noted by ultrasound exam include the anechoic, the pulsatile popliteal artery, and the compressible popliteal vein. The vessels are located medially to the sciatic nerve, which appears as a hyperechoic, round structure with hypoechoic honeycombing (Fig. 9.24) [28]. Needle choice is made, and the needle can be introduced in an in-plane or out-of-plane fashion. Again, as in the midthigh region,

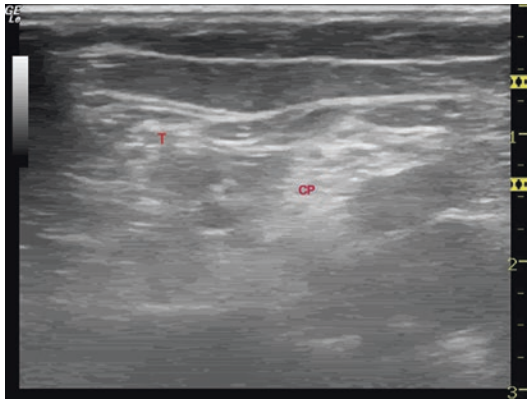

Fig. 9.25 The bifurcation of the sciatic nerve into the common peroneal (*CP*) and tibial nerves (*T*)

the in-plane needle insertion can be performed in a trans-vastus approach or in an angular approach from the side of the probe. The "scan back" technique is often useful for this approach as well, noting the separate tibial and peroneal nerves and then moving the transducer proximally until their point of bifurcation from the single sciatic nerve is identified (Fig. 9.25).

Complications

Complications of the sciatic nerve block include hematoma, abscess, vascular puncture, and local anesthetic toxicity. Transient and permanent nerve injury after sciatic nerve blockade is exceedingly rare [3, 31].

Saphenous Nerve Block/Adductor Canal Block

The saphenous nerve, a terminal sensory branch of the posterior division of the femoral nerve, provides innervation to the medial aspect of the upper thigh, lower leg, ankle, and foot. As the nerve exits the adductor canal, it courses along with the saphenous branch of the descending genicular artery [47, 48]. The nerve then gives off an infrapatellar branch innervating the knee, and a sartorial branch, which courses to the posterior portion of the knee.

The saphenous nerve is easily blocked in the midthigh, owing to its course along with the sartorius muscle, the femoral artery, and, more distally, the descending genicular artery. The patient is placed supine, and after sterile preparation, the probe is placed in a transverse fashion on the anterior thigh, midway between the inguinal crease and knee. Typically, owing to the deeper location of the nerve, a frequency between 6 and 1 MHz is selected. Initial ultrasound examination will identify, most superficially and medially, the sartorius muscle and, laterally and deeper, the vastus medialis. The nerve can be identified as it courses laterally and deep to the sartorius muscle, in an anterolateral relation to the femoral artery. Here, the nerve will appear as a hyperechoic, star-like structure that directly abuts the artery (Fig. 9.26). If difficulty is encountered, the probe can be moved more distally in order to attempt to image the nerve as it exits the adductor canal, though it may change its orientation to both the sartorius and the descending genicular artery in

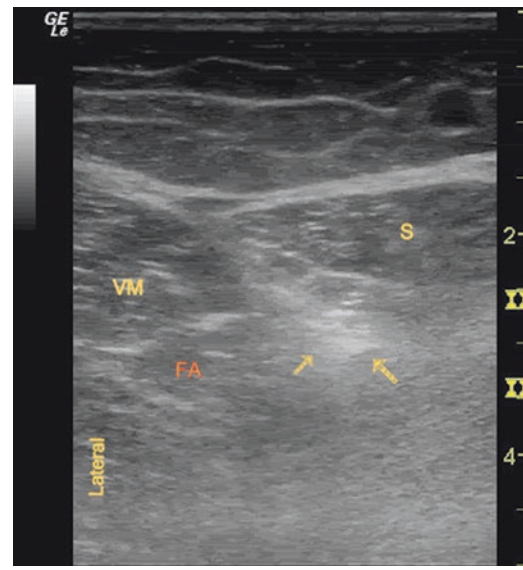

Fig. 9.26 The trans-sartorial saphenous block. The saphenous nerve (arrows) in relation to the vastus medialis (*VM*), the femoral artery (*FA*), and the sartorius (*S*)

this view. The nerve can also be identified by a "scan back technique" in which the operator locates the femoral nerve and artery in the inguinal crease and then traces these structures down to the midthigh, thereby identifying the saphenous nerve [49].

Needle selection is based on the depth of the nerve on ultrasound examination, and an in-plane approach is used from the lateral edge of the ultrasound probe. After the needle reaches the saphenous nerve, aspiration is performed to exclude vascular placement, and local anesthetic is injected incrementally. At times, the needle may be repositioned to ensure injection within the fascial sheath running in between the vastus medialis and the sartorius.

Use of the term "adductor canal block" has become more frequent in relation to surgeries of the lower extremity. This block is performed in the same fashion as the saphenous nerve block, but a higher volume of injectate is utilized [50, 51]. The higher volume of local anesthesia may provide additional proximal spread to cover the vastus medialis nerve, the middle femoral cutaneous nerve, articular branches of the obturator nerve, and the medial retinacular nerve, thereby providing sensory block to the medial and anterior aspects of the knee and the upper tibia [47, 48, 52].

Truncal Blocks

Ilioinguinal-Iliohypogastric Nerve Block

The ilioinguinal nerve innervates the upper medial part of the thigh and the upper part of the genitalia, while the iliohypogastric nerve provides sensation to the buttock and abdominal wall above the pubis. As such, the ilioinguinal-iliohypogastric nerve block (IIIHNB) can provide analgesia for procedures including inguinal hernia repair, orchiopexy, and hydrocele repair [53]. The ilioinguinal and iliohypogastric nerves are terminal portions of the L1 root that emerge from the lateral border of the psoas major muscle, cross the quadratus lumborum muscle obliquely, and perforate the transverse abdominis muscle, where they course together in the plane between the internal oblique and transverse abdominis (Fig. 9.27) [44].

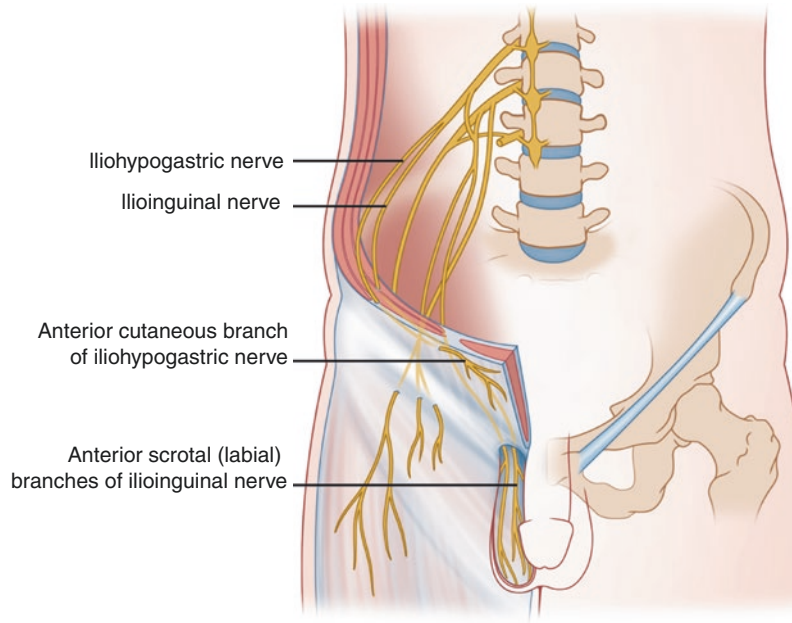

Fig. 9.27 The course of the ilioinguinal and iliohypogastric nerves

The nerve block is performed with the patient supine. After sterile preparation of the skin and probe, the probe is placed in a transverse orientation directly medial to the anterior superior iliac spine (ASIS). Here it is useful to tilt and rotate the probe so that it runs parallel to a line drawn between the ASIS and umbilicus. The operator then gently moves the probe medially, "rolling off" the ASIS. Ultrasound examination will reveal laterally the hypoechoic shadow of the ASIS and the three layers of abdominal musculature: the external oblique, internal oblique, and transverse abdominis. Below the transverse abdominis, the bowel can be seen. The ilioinguinal and iliohypogastric nerves can be visualized as elliptical honeycomb structures that run between the internal oblique and transverse abdominis (Fig. 9.28). They may often be mistaken for vascular structures due to their round, hypoechoic centers.

After frequency, depth, and gain are adjusted to optimize the image, a needle is passed from the lateral border of the probe toward the nerve (Fig. 9.29). After an aspiration is performed to rule out vascular placement of the needle, a small test dose is injected. Ultrasound examination of correct placement reveals a lemon-shaped appearance of local spread around the nerve bundle. Incorrect placement of the needle will often result in a round appearance of local anesthetic spread, typical of intramuscular injection.

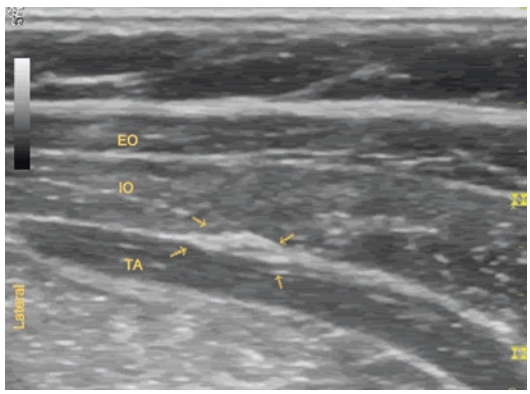

Fig. 9.28 Ultrasound of the ilioinguinal and iliohypogastric nerves. The ilioinguinal and iliohypogastric nerves (arrows) lie in between the internal oblique (*IO*) and transverse abdominis muscles (*TA*). Also pictured is the more superficial external oblique

Fig. 9.29 Performing the ilioinguinal-iliohypogastric nerve block

Specific complications associated with IIIHNB are bowel hematoma, bowel puncture, pelvic hematoma, femoral nerve block, and local anesthetic toxicity [53]. As such, it is prudent to constantly visualize the tip of the needle with specific attention to the depth of both the needle and the location of the intraperitoneal contents.

Transverse Abdominis Plane Block

The transverse abdominis plane (TAP) block can be used as an analgesic supplement in procedures involving the abdominal wall and anterior parietal peritoneum [53]. The transverse abdominis plane exists between the internal oblique and transverse abdominis muscles and consists of an interconnected plexus of nerves comprised of the somatic afferents of T8–L1 (Fig. 9.30) [53, 54]. It should be noted that this block will not provide analgesia of the deep intraperitoneal structures.

To perform the TAP block, a linear probe is selected. After sterile preparation, the probe is placed on the abdomen, approximately at the level of the planned incision, in a transverse fashion just lateral to midline. Initial ultrasound examination will identify the large, ellipse-like muscular structure of the rectus abdominis muscles (Fig. 9.31). Once this is identified, the probe is moved laterally, noting the edge of the rectus abdominis muscle. Directly adjacent to the lateral

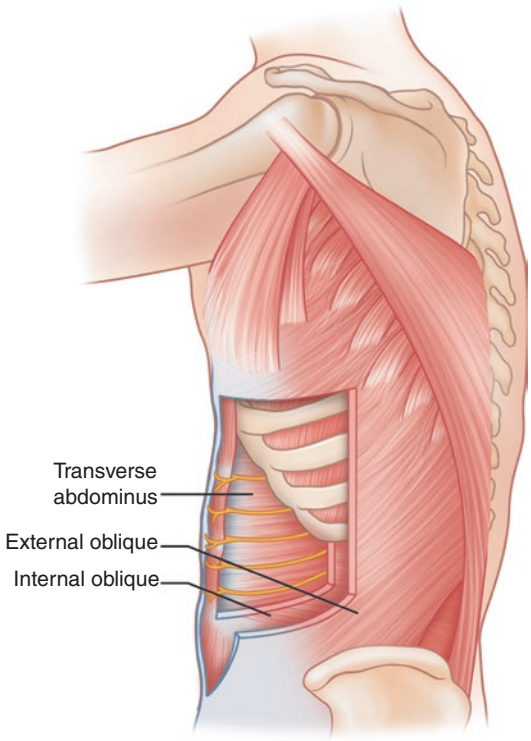

Fig. 9.30 The TAP plexus

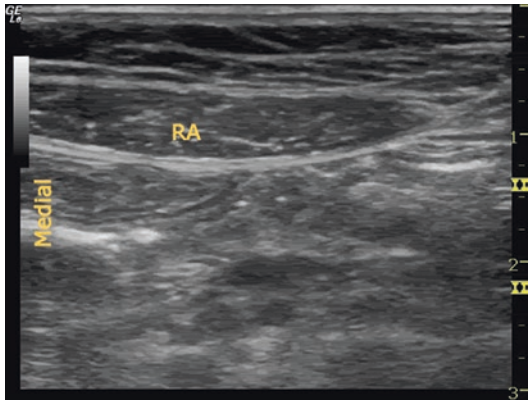

Fig. 9.31 The cross-sectional ultrasonographic appearance of the rectus abdominis

edge of the rectus abdominis muscle, the three linear layers of muscle can be visualized, consisting of the external oblique most superficially, then the internal oblique, and, deepest, the transverse abdominis (Fig. 9.32). Once identified, depth, focus, and frequency (typically 12–14 MHz) are adjusted to optimize the image.

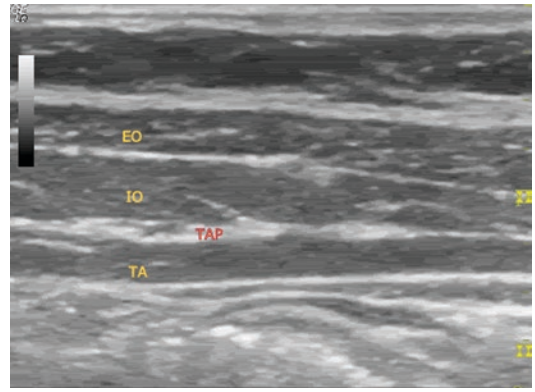

Fig. 9.32 TAP block. The external oblique muscle (*EO*), internal oblique muscle (*IO*), and transverse abdominis muscle (*TA*) are visualized. The transverse abdominis plane (*TAP*) lies between the internal oblique and transverse abdominis muscle

The needle is introduced in-plane from the medial edge of the ultrasound probe. Under constant visualization, it is passed into the hyperechoic fascial layer between the internal oblique muscle and the transverse abdominis muscle. After aspiration, a test dose injection is performed. Ideal visualization of local spread will show a "lemon"-shaped spread of local anesthetic between fascial planes, as opposed to the more circular-shaped appearance of an intramuscular injection [55].

Potential complications of the TAP block include intravascular injection, local anesthetic toxicity, peritoneal puncture with or without visceral injury, and infection at the injection site [56–58].

Rectus Sheath Block

The rectus sheath block can provide analgesia for procedures involving the anterior abdominal wall such as vertical midline laparotomy and laparoscopy. Its advantages include the large and recognizable size of the rectus muscle and lack of large vascular structures in that area [59].

The ventral roots of T6–L1 innervate the central portion of the abdominal wall and lie between the belly of the rectus abdominis muscle and, posteriorly, the fascia of the rectus sheath. Here,

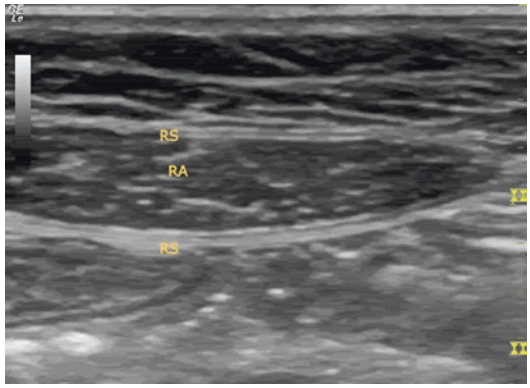

Fig. 9.33 The rectus sheath (*RM*) is encapsulated anteriorly and posteriorly by the rectus sheath (*RS*)

an injection of local anesthetic will spread in a caudocephalad manner, anesthetizing the terminal branches of the nerves [60].

To perform the nerve block, the patient is placed supine. A linear probe is selected, with typical frequency ranging from 10 to 12 MHz. After sterile preparation of the skin and probe, the probe is placed in a transverse fashion along the lateral edge of the umbilicus. Here, the rectus abdominis muscle is identified in cross section, and the image is optimized by decreasing depth and selecting the best focus depth and frequency. The probe is then directed laterally to identify the lateral, beak-like border of the rectus muscle. Directly posterior to the muscle lies the hyperechoic fascia of the rectus sheath (Fig. 9.33).

Needle selection is based on the depth of the border of the rectus muscle and rectus sheath. In an in-plane fashion, a needle is introduced from the lateral edge of the rectus muscle and is placed between the rectus muscle and the posterior rectus sheath fascia. After aspirating to rule out intravascular needle placement, a test dose injection will reveal an ellipse-like spread of local anesthetic in the fascial plane. Incorrect intramuscular placement of the needle will result in the local anesthetic injection forming a circular appearance. Once correct needle placement is confirmed, the remaining local anesthetic is injected with the goal of separating the rectus muscle and sheath [61].

Potential complications of the rectus sheath block include intravascular injection, local anesthetic toxicity, rectus sheath hematoma, peritoneal puncture with or without visceral injury, and infection at the injection site.

Quadratus Lumborum Block

The quadratus lumborum block aims to deposit local anesthetic in a similar fascial plane as the TAP block, only more dorsally in the abdominal wall (Fig. 9.34). The aim of the block is to anesthetize the ventral rami of the spinal nerve roots as they course anterior to the quadratus lumborum muscle [62]. The block may impart both a longer duration of sensory block, as well as a more complete spread of sensory block over the upper and lower abdomen [63, 64]. The block is thus ideal for surgeries that involve more extensive somatic pain over the abdominal wall, such as laparoscopic surgeries with multiple ports.

To perform the block, a linear probe is selected. After sterile prep, the probe is placed on the lateral abdomen, similar to the TAP block. The external oblique, internal oblique, and transverse abdominis are identified just as in the TAP block. The probe is then moved laterally, following the transverse abdominis to its lateral border, where it comes to a point. Below the tip of the

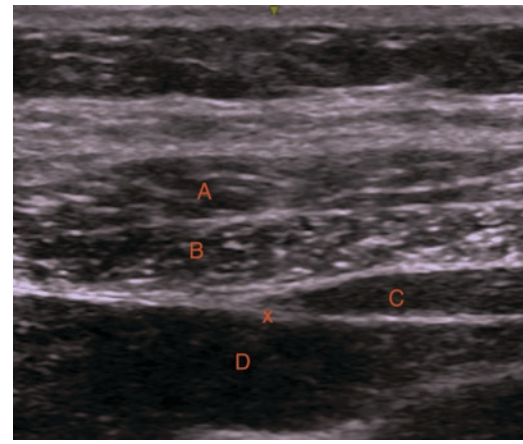

Fig. 9.34 The quadratus lumborum block: similar to the TAP block, the external oblique (*A*), internal oblique (*B*), and transversus abdominis muscles (*C*) are identified. Below the transversus abdominis lies the quadratus lumborum (*D*). Injection occurs at the conjunction of the transversus abdominis and the quadratus lumborum (*x*)

transverse abdominis lies the quadratus lumborum muscle, which is surrounded by a hyperechoic, thick thoracolumbar fascia [62]. It is important to ensure that the kidney is not mistaken for the quadratus lumborum, and as such, Doppler interrogation of the target is prudent. The needle is introduced medially and directed laterally, with the goal of placement directly under the thoracolumbar fascia encasing the quadratus lumborum muscle. Once test dose reveals subfascial injection, injection of a larger volume of more dilute local anesthetic will result in a successful block.

Potential complications of the quadratus lumborum block include intravascular injection, local anesthetic toxicity, peritoneal puncture with or without visceral injury, kidney injury, and infection at the injection site [65].

Thoracic Paravertebral Block

The thoracic paravertebral space provides an area to perform unilateral somatic and sympathetic nervous blockade to the chest and abdominal wall (Fig. 9.35). Continued experience with ultrasound guidance has popularized this technique. Its potential benefits over thoracic epidural blockade include hemodynamic stability with more dense blockade, ability to ambulate, no risk of urinary retention, thereby eliminating the need for Foley catheter placement, and the ability to perform neurologic examination of the lower extremities [66].

The thoracic paravertebral space is bounded medially by the vertebral bodies, though it maintains a connection to the epidural space [67]. Laterally, the paravertebral space tapers and becomes the intercostal space. The posterior border of the thoracic paravertebral space is the superior costotransverse ligament, while the anterior border is the parietal pleura. Interconnectivity exists at the anteromedial border of the paravertebral space, allowing spread of the injectate to multiple levels [68].

Two methods exist for ultrasound visualization of the thoracic paravertebral space: sagittal and transverse. In the sagittal view, a linear probe is placed on the midline of the selected level of blockade. The spinous processes are identified, specifically noted by their "mountain chain" appearance. The probe is the moved laterally, and utilizing tilt, the hypoechoic transverse processes surrounding the hypoechoic paravertebral space are visualized (Fig. 9.36). Some have likened this image to "Mickey Mouse ears." Of note, the paravertebral space is superiorly bordered by the costotransverse ligament in this view, which appears

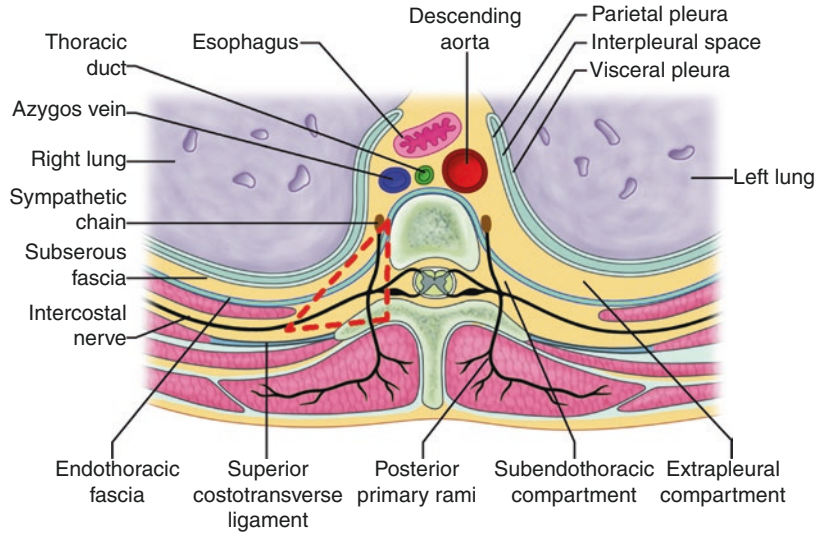

Fig. 9.35 The thoracic paravertebral space (red dashed line)

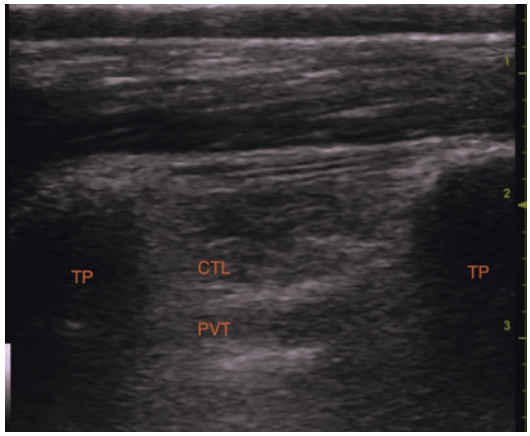

Fig. 9.36 The sagittal view of the thoracic paravertebral space. The transverse processes (TP) about the hypoechoic paravertebral space (PVT) superiorly bordered by the costotransverse ligament (CTL). Injection of the paravertebral space will lead to a round increasing size of the paravertebral space and a depression of the hyperechoic pleura below

as a more hyperechoic but heterogenous layer. The paravertebral space will be a hypoechoic linear space directly below this layer, connecting the "Mickey Mouse ears." Of note, the parietal pleura is also seen here as a hyperechoic linear and dynamic structure moving with respiration.

To inject the paravertebral space in the sagittal view, the patient is placed either lateral or prone. A linear probe is selected, with typical frequency ranging from 8 to 12 MHz. After sterile preparation of the skin and probe, the probe is placed as above. Once the paravertebral space is identified, the needle is passed in an in-plane fashion between the acoustic images of the transverse processes to the hypoechoic paravertebral space. Often, a distinct pop is felt when the needle passes through the costotransverse ligament. It is prudent to intermittently hydrodissect as the needle is advanced to avoid entry into the pleural cavity. Once the paravertebral space is entered with the needle, injection will increase the area of the hypoechoic paravertebral space, also depressing the hyperechoic parietal pleura. If difficulty is encountered guiding the needle to the paravertebral space, one can perform the "off-sides" maneuver of Abdallah and Brull, in which the paravertebral space is placed "off-sides" to the lateral side of the ultrasound screen allowing for a steeper angle of the needle to reach the paravertebral space. This cephalad or caudad translational movement of the linear transducer slows the needle to clear the transverse process and permits easier access to the paravertebral space [69].

The transverse view technique for thoracic paravertebral blockade utilizes the same anatomy, but only focuses on one transverse process at a time. To obtain an image of the thoracic paravertebral space, the probe is initially placed midline to view the spinous process, which appears similar to a mountain peak (Fig. 9.37). The probe is the slid laterally to view the transverse process (Fig. 9.38). Beneath the transverse

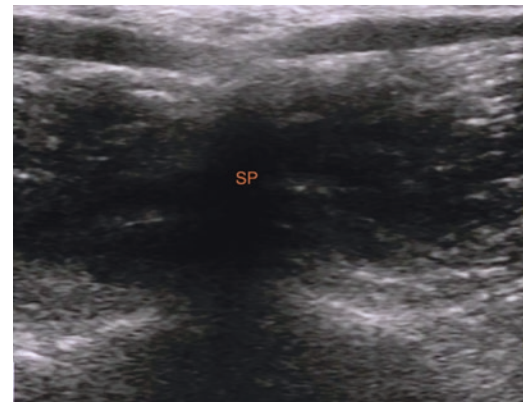

Fig. 9.37 The transverse view of the thoracic vertebrae. Note the hypoechoic "mountain peak" signature of the spinous process (SP)

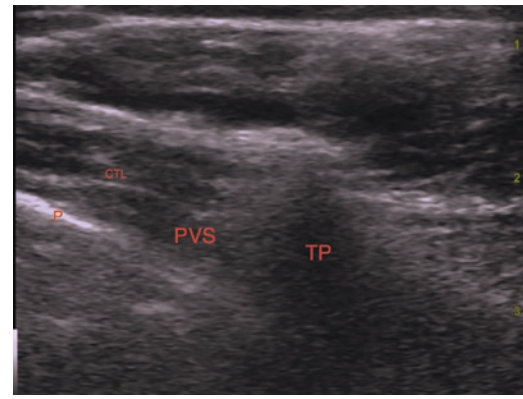

Fig. 9.38 The transverse approach to the thoracic paravertebral block. The hypoechoic transverse process (TP) is bordered laterally by the paravertebral space (PVS). Note the hyperechoic pleura (P) and superiorly bordering costotransverse ligament (CTL)

process lies the wedge shaped, hypoechoic paravertebral space overlying the hyperechoic and dynamic parietal pleura. Inserting a needle in-plane from the lateral side, the needle is advanced to the paravertebral space with hydrodissection to avoid intrapleural injection. As in the sagittal technique, injection into the paravertebral space will result in depression of the parietal pleura [68].

Peripheral Nerve Blockade in Children

Peripheral nerve blockade in the pediatric setting has undergone a slower evolution when compared with the adult world. Before the development of ultrasound-guided peripheral nerve blockade techniques, significant challenges in pediatric patients included the difficulty in targeting nerve structures that course dangerously closely to other critical structures, the constant risk of local anesthetic toxicity with smaller children and higher volume blocks, and the concern of nerve injury when performing a nerve block on a heavily sedated or completely anesthetized patient [70]. The development of ultrasound-guided techniques and subsequent large-scale evaluation of outcomes have led to both increased numbers of peripheral nerve blocks performed in children and clinical data proving efficacy and safety [1, 3]. Performing peripheral nerve blocks with ultrasound on anesthetized children is now generally well accepted and has led to an increase in practice [71].

The pediatric patient offers several advantages over the adult patient in relation to ultrasonography. Typically, the child has less adipose tissue and has smaller structures, which allows the operator to utilize higher frequencies and improve resolution and image quality of blocks that are often difficult in adults. Secondly peripheral nerve blocks in a heavily sedated or anesthetized child have been shown to be safe, which provides the operator with an immobile patient [26, 72]. A disadvantage encountered when performing ultrasound-guided regional nerve blocks on children is related mainly to size. In this setting, smaller, more specific equipment may be needed, including "hockey stick" probes with higher available frequencies and short block needles. Additionally, there is often limited application space for ultrasound probe placement on the skin in small children.

Performing nerve blocks provides an excellent analgesic alternative to systemic pain medication. Considering the fragile nature of infants and children, peripheral nerve blockade can provide a safe, long-lasting analgesic devoid of the risk of respiratory depression and hemodynamic instability. Current trends support this claim, as the gap between neuraxial blockade, once the most commonly practiced regional anesthetic option, and peripheral nerve blockade in pediatric patients narrows [1, 3].

Systemic local anesthetic toxicity has always been a concern when placing regional blocks in infants and children. Ultrasound guidance has been shown to reduce the volume of local anesthetic needed for block success, thereby significantly reducing this risk to an acceptable level [3, 73].

Clinical Pearls

Interscalene Block

- The trunks of the plexus lie lateral to the easily identifiable sternocleidomastoid (SCM). The tapered tip of the SCM lies directly above and medial to the anterior scalene muscle.
- It is easy to mistake the lateral border of the SCM for the brachial plexus. This can be avoided by identification of the anterior scalene muscle.
- The anterior scalene muscle can be small as the probe is moved rostrally. It may be beneficial to move the probe caudally in order to identify it.
- If the brachial plexus is not visualized in the interscalene position, gently roll the probe down to the supraclavicular position, and then trace the plexus back to the interscalene position.

Supraclavicular Nerve Block

- The plexus at this level appears as a "bunch of grapes" as opposed to the more linear, "snowman" appearance in the interscalene block.
- The probe should be tilted to transect the brachial plexus at this level.
- It is often easy to mistake the fascia enveloping the sternocleidomastoid for the brachial plexus. It is important, here, to visualize the SCM, the anterior scalene, and the middle scalene to correctly identify the brachial plexus lying between the anterior and middle scalene.

Infraclavicular Nerve Block

- This is a deeper block; therefore, a curvilinear probe with lower frequencies may be necessary to penetrate tissue.
- If there is difficulty in locating the brachial plexus in the infraclavicular fossa, it is often helpful to identify the brachial plexus in the supraclavicular fossa and then mark on the skin where the plexus is located there. This surface landmark can now serve as the medial border of the area to scan in the infraclavicular fossa.
- In children, it may be difficult to pass a needle at a correct angle in between the probe and the clavicle, particularly with larger probes. If this is the case, it is acceptable to use an in-plane needle placement from the inferior edge of the probe. A hockey stick probe is often beneficial.

Femoral Nerve Block

- The optimal block location is proximal to the femoral artery bifurcation.
- Pressure on the transducer is often useful in obtaining the image of the femoral artery.
- The point of injection must always be below the fascia lata.
- A small amount of rostral tilt may improve the image.

Sciatic Nerve Block

- Lower-frequency probes may be useful when the block is performed more proximally.
- When introducing the needle from the lateral thigh, first measure the depth of the nerve itself, and then use that distance from the probe as the insertion site on the lateral thigh.
- Probe tilting may improve the image as you are "transecting" the nerve at different points of the thigh.
- The bifurcation of the sciatic nerve into the tibial and peroneal nerves should be visualized first during the popliteal fossa block (Fig. 9.25). The nerve should be traced back and blocked proximal to this bifurcation, or the common peroneal and tibial nerves should be blocked with separate injections.

Ilioinguinal-Iliohypogastric Nerve Block

- It is often useful to "roll off" the iliac crest in order to situate the probe in its proper alignment.
- Useful landmarks may include the thick iliacus muscles which lie below the transverse abdominis muscles.
- The nerves may appear similar to vasculature. Doppler interrogation may be used to determine whether it is a vascular or nervous structure.

Transverse Abdominis Plane Block

- The TAP block provides analgesia for abdominal wall and peritoneal pain. Supplementation with NSAIDs and opiates should be added for visceral pain.
- Higher-frequency probes will improve the resolution of the image to the superficial nature.
- The TAP block should be performed as lateral as possible on the abdominal wall, specifically where the latissimus dorsi muscle begins to obscure the three muscle layers.

- The rectus abdominis muscle can serve as an excellent landmark medially because this is where the three muscle layers become evident.

Rectus Sheath Block

- The rectus sheath is easily visualized in the midline.
- A lateral approach may be easier as it allows the operator to avoid the thick rectus muscle.

Review Questions

1. Axial resolution refers to:
   (a) The ability to distinguish two points side by side
   (b) The ability to distinguish two points in the same line of axis
   (c) The ability to minimize background noise
   (d) The optimal depth in order to visualize a structure
2. The interscalene block aims to inject the brachial plexus between:
   (a) The sternocleidomastoid and the first rib
   (b) The omohyoid muscle and the sternocleidomastoid
   (c) The anterior and middle scalene muscles
   (d) The pectoralis minor and axillary artery
3. When placing an ultrasound-guided supraclavicular nerve block, the pulsating artery found at the inferior portion of the target area is the:
   (a) Subclavian artery
   (b) External carotid artery
   (c) Internal carotid artery
   (d) Vertebral artery
4. After placing an axillary nerve block, the patient has sensory sparing of the medial upper arm. The nerve responsible for this is:
   (a) The radial nerve
   (b) The ulnar nerve
   (c) The median nerve
   (d) The musculocutaneous nerve
5. The femoral nerve is bounded medially by the femoral artery, inferiorly by the iliacus muscle, and superiorly by the hyperechoic:
   (a) Iliopectineal ligament
   (b) Fascia lata
   (c) Femoral nerve
   (d) Psoas muscle
6. After performing a popliteal fossa sciatic nerve block for an ankle procedure, the patient is experiencing pain on the dorsum of the foot. A likely scenario of failure is:
   (a) The femoral nerve has been mistakenly blocked.
   (b) The saphenous nerve was not adequately anesthetized.
   (c) There is typically sparing of anesthesia in this location.
   (d) The block occurred distal to the bifurcation of the sciatic, only including the tibial nerve.
7. The "trackback" technique involves locating the saphenous nerve related to its branching from the:
   (a) Femoral nerve
   (b) Sural nerve
   (c) Sciatic nerve
   (d) Obturator nerve
8. The ilioinguinal and iliohypogastric nerves are blocked as they course between the:
   (a) Transverse abdominis muscle and the parietal peritoneum
   (b) External and internal oblique muscles
   (c) Internal oblique and transverse abdominis muscles
   (d) Iliacus and transverse abdominis muscles
9. When utilizing a TAP block for a laparoscopic appendectomy, the following is false:
   (a) Parenteral opiates or NSAIDs are useful for the treatment of uncovered visceral pain.
   (b) The anterior abdominal wall is anesthetized by the TAP block.
   (c) The anterior parietal peritoneum is blocked by the TAP block.
   (d) The TAP block should cover components of visceral pain.

10. When injecting a local anesthetic solution during ultrasound-guided peripheral nerve block placement, you notice large hyperechoic artifacts filling the target area. The likely problem is:
    (a) Vascular perforation
    (b) Air has been injected through the needle
    (c) Intraneural injection
    (d) Vasospasm
11. While performing a TAP block, injection reveals a round, circular spread of injectate in an area presumed to lie between the transverse abdominis and internal oblique muscles. The block fails. The likely problem is:
    (a) This was an intramuscular injection.
    (b) This was an intravascular injection.
    (c) The nerve was missed.
    (d) The volume of local anesthetic was insufficient.
12. While performing an interscalene nerve block, the image appears as bright, causing difficulty in identifying neural structures. An appropriate control to manipulate would be:
    (a) The depth
    (b) The frequency
    (c) The gain
    (d) The focus position
13. While placing a femoral nerve block, difficulty is encountered in identifying the needle placement. Injection of a small amount of local anesthetic results in a widening of the femoral nerve image. A likely scenario is:
    (a) Intraneural injection
    (b) Correct placement
    (c) Intravascular injection
    (d) Air injection
14. While placing a femoral nerve block, you notice an asymmetric, expanding hypoechoic element next to the nerve and artery. This is likely:
    (a) Extravasation of a small amount of local anesthetic from the needle
    (b) Separation of the fascial plains
    (c) Mild damage to and extravasation of the lymphatic system
    (d) Formation of a hematoma from the femoral artery
15. While performing an interscalene block, it is difficult to obtain an acceptable image of the axillary artery and brachial plexus due to the depth of the structures. The following would be appropriate manipulations of the ultrasound machines *EXCEPT*:
    (a) Decreasing frequency
    (b) Increasing frequency
    (c) Manipulating the TGC
    (d) Increasing depth

Answers:

1. b—Axial resolution refers to the ability to distinguish two points in the same line of axis. Higher-frequency beams improve axial resolution at the cost of poorer depth penetration and visualization.
2. d—The interscalene block aims to inject the brachial plexus at the level of the trunks, which usually exists at C6. Here, the trunks lie between the anterior and middle scalene, though the anterior scalene may appear small.
3. a—The brachial plexus follows a similar course to the subclavian artery, which becomes the axillary artery. As such, this is a useful landmark in locating the brachial plexus.
4. d—The musculocutaneous nerve is frequently missed in the axillary nerve block. It can be visualized with an ultrasound and injected separately or accessed by directly injecting into the body of the coracobrachialis muscle.
5. b—The fascia lata is the superior border of the femoral nerve sheath. It separates from the fascia iliaca at the femoral nerve and rejoins laterally.
6. d—Blocking the sciatic nerve in the popliteal fossa must be performed proximal to the bifurcation of the sciatic nerve into the tibial and peroneal nerves.
7. a—The saphenous nerve is a branch of the femoral nerve. As such, it is often responsible for medial foot pain when only a sciatic block is performed.

8. c—Much like the TAP block, the ilioinguinal and iliohypogastric nerves course between the internal oblique muscle and the transverse abdominis muscle.
9. d—While the TAP block anesthetized the terminal branches of T8–T11, it does not typically block visceral pain. As such, the anterior abdominal wall and the parietal peritoneum will likely be covered, but visceral pain must be addressed with either parenteral medicines or additional neuraxial anesthetics.
10. b—As air is hyperechoic, even small amounts can significantly worsen imaging when injected. Therefore, it is essential to always flush needles before ultrasound-guided peripheral nerve blockade.
11. a—The TAP block relies on anesthetic deposition between two fascial layers. As such, the injectate should appear as an elliptical spread of hypoechoic fluid. Round-appearing injectate is frequently indicative of intramuscular injection.
12. c—Gain refers to the amplification of received signals. As such, too much gain leads to an amplification of background noise and, in this case, should be lowered.
13. a—Widening of the nerve image is often indicative of intraneural injection, which has been associated with nerve injury.
14. d—Inadvertent vascular puncture is often seen as a hypoechoic expansion of the perivascular space.
15. b—By decreasing frequency and depth, deeper structures are better imaged. TGC may help optimize the gain on attenuated deeper structures.

References

1. Ecoffey C. Safety in pediatric regional anesthesia. Pediatric anesthesia. Blackwell, Oxford; 2012;22(1):25–30.
2. Neal JM, Brull R, VWS C, Grant SA, Horn J-L, Liu SS, et al. The ASRA evidence-based medicine assessment of ultrasound-guided regional anesthesia and pain medicine. Reg Anesth Pain Med. 2010;35(Suppl 1):S1–9.
3. Polaner DM, Taenzer AH, Walker BJ, Bosenberg A, Krane EJ, Suresh S, et al. Pediatric Regional Anesthesia Network (PRAN): a multi-institutional study of the use and incidence of complications of pediatric regional anesthesia. Anesth Analg. 2012;115(6):1353–64.
4. Neal JM. Ultrasound-guided regional anesthesia and patient safety: update of an evidence-based analysis. Reg Anesth Pain Med. 2016;41(2):195–204.
5. McNaught A, Shastri U, Carmichael N, Awad IT, Columb M, Cheung J, et al. Ultrasound reduces the minimum effective local anaesthetic volume compared with peripheral nerve stimulation for interscalene block. Br J Anaesth. 2011;106(1):124–30. Oxford University Press.
6. Liu SS, Zayas VM, Gordon MA, Beathe JC, Maalouf DB, Paroli L, et al. A prospective, randomized, controlled trial comparing ultrasound versus nerve stimulator guidance for interscalene block for ambulatory shoulder surgery for postoperative neurological symptoms. Anesth Analg. 2009;109(1):265–71.
7. Orebaugh SL, Williams BA, Vallejo M, Kentor ML. Adverse outcomes associated with stimulator-based peripheral nerve blocks with versus without ultrasound visualization. Reg Anesth Pain Med. 2009;34(3):251–5.
8. Abrahams MS, Aziz MF, Fu RF, Horn JL. Ultrasound guidance compared with electrical neurostimulation for peripheral nerve block: a systematic review and meta-analysis of randomized controlled trials. Br J Anaesth. 2009;102(3):408–17.
9. Casati A, Baciarello M, Cianni SD, Danelli G, De Marco G, Leone S, et al. Effects of ultrasound guidance on the minimum effective anaesthetic volume required to block the femoral nerve. Br J Anaesth. 2007;98(6):823–7. Oxford University Press.
10. Suresh S, Schaldenbrand K, Wallis B, De Oliveira GS. Regional anaesthesia to improve pain outcomes in paediatric surgical patients: a qualitative systematic review of randomized controlled trials. Br J Anaesth. 2014;113(3):375–90. Oxford University Press.
11. Guay J, Suresh S, Kopp S. The use of ultrasound guidance for perioperative neuraxial and peripheral nerve blocks in children. Cochrane Database Syst Rev. 2016;(2):CD011436.
12. Marhofer P. Ultrasonographic guidance reduces the amount of local anesthetic for 3-in-1 blocks*1. Reg Anesth Pain Med. 1998;23(6):584–8.
13. Willschke H, Marhofer P, Bösenberg A, Johnston S, Wanzel O, Cox SG, et al. Ultrasonography for ilioinguinal/iliohypogastric nerve blocks in children. Br J Anaesth. 2005;95(2):226–30. Oxford University Press.
14. Novelline RA, Squire LF. Squire's fundamentals of radiology. 6th ed. Cambridge: Harvard University Press; 2004.

15. Brull R, Macfarlane AJR, Tse CCH. Practical knobology for ultrasound-guided regional anesthesia. Reg Anesth Pain Med. 2010;35(2 Suppl):S68–73.
16. Sites BD, Brull R, Chan VWS, Spence BC, Gallagher J, Beach ML, et al. Artifacts and pitfall errors associated with ultrasound-guided regional anesthesia. Part II: a pictorial approach to understanding and avoidance. Reg Anesth Pain Med. 2007;32(5):419–33.
17. Sites B, Brull R, Chan V, Spence B, Gallagher J, Beach M, et al. Artifacts and pitfall errors associated with ultrasound-guided regional anesthesia. Part I: understanding the basic principles of ultrasound physics and machine operations. Reg Anesth Pain Med. 2007;32(5):412–8.
18. Suresh S, Taylor LJ, De Oliveira GS. Dose effect of local anesthetics on analgesic outcomes for the transversus abdominis plane (TAP) block in children: a randomized, double-blinded, clinical trial. Pediatr Anesth. 2015;25(5):506–10.
19. Sites BD, Taenzer AH, Herrick MD, Gilloon C, Antonakakis J, Richins J, et al. Incidence of local anesthetic systemic toxicity and postoperative neurologic symptoms associated with 12,668 ultrasound-guided nerve blocks. Reg Anesth Pain Med. 2012;37(5):478–82.
20. Fredrickson MJ, Ball CM, Dalgleish AJ. A prospective randomized comparison of ultrasound guidance versus neurostimulation for interscalene catheter placement. Reg Anesth Pain Med. 2009;34(6):590–4.
21. Kapral S, Greher M, Huber G, Willschke H, Kettner S, Kdolsky R, et al. Ultrasonographic guidance improves the success rate of interscalene brachial plexus blockade. Reg Anesth Pain Med. 2008;33(3):253–8.
22. Lombard TP, Couper JL. Bilateral spread of analgesia following interscalene brachial plexus block. Anesthesiology. 1983s;58(5):472.
23. Borgeat A. All roads do not lead to Rome. Anesthesiology. 2006;105(1):1–2.
24. Benumof JL. Permanent loss of cervical spinal cord function associated with interscalene block performed under general anesthesia. Anesthesiology. 2000;93(6):1541–4.
25. Clendenen SR, Robards CB, Wang RD, Greengrass RA. Continuous interscalene block associated with neck hematoma and postoperative sepsis. Anesth Analg. 2010;110(4):1236–8.
26. Taenzer AH, Walker BJ, Bosenberg AT, Martin L, Suresh S, Polaner DM, et al. Asleep versus awake: does it matter?: pediatric regional block complications by patient state: a report from the Pediatric Regional Anesthesia Network. Reg Anesth Pain Med. 2014;39(4):279–83.
27. Perlas A, Lobo G, Lo N, Brull R, Chan VWS, Karkhanis R. Ultrasound-guided supraclavicular block. Reg Anesth Pain Med. 2009;34(2):171–6.
28. Fingerman M, Benonis JG, Martin G. A practical guide to commonly performed ultrasound-guided peripheral-nerve blocks. Curr Opin Anaesthesiol. 2009;22(5):600–7.
29. Chan VWS, Perlas A, Rawson R, Odukoya O. Ultrasound-guided supraclavicular brachial plexus block. Anesth Analg. 2003;97:1514–7.
30. Perlas A, Lobo G, Lo N, Brull R, Chan VWS, Karkhanis R. Ultrasound-guided supraclavicular block: outcome of 510 Consecutive cases. Reg Anesth Pain Med. 2009;34(2):171–6.
31. Neal JM. Ultrasound-guided regional anesthesia and patient safety: an evidence-based analysis. Reg Anesth Pain Med. 2010;35(2):S59–67.
32. Tran DQH, Clemente A, Tran DQ, Finlayson RJ. A comparison between ultrasound-guided infraclavicular block using the "double bubble" sign and neurostimulation-guided axillary block. Anesth Analg. 2008;107(3):1075–8.
33. Tedore TR, YaDeau JT, Maalouf DB, Weiland AJ, Tong-Ngork S, Wukovits B, et al. Comparison of the transarterial axillary block and the ultrasound-guided infraclavicular block for upper extremity surgery. Reg Anesth Pain Med. 2009;34(4):361–5.
34. Bigeleisen P, Wilson M. A comparison of two techniques for ultrasound guided infraclavicular block. Br J Anaesth. 2006;96(4):502–7. Oxford University Press.
35. Sauter AR, Smith H-JR, Stubhaug A, Dodgson MS, Klaastad I. Use of magnetic resonance imaging to define the anatomical location closest to all three cords of the infraclavicular brachial plexus. Anesth Analg. 2006;103(6):1574–6.
36. Retzl G, Kapral S, Greher M, Mauritz W. Ultrasonographic findings of the axillary part of the brachial plexus. Anesth Analg. 2001;92:1271–5.
37. Koscielniak-Nielsen ZJ. Multiple injections in axillary block. Reg Anesth Pain Med. 2006;31(3):192–5.
38. Handoll HHG, Koscielniak-Nielsen ZJ. Single, double or multiple injection techniques for axillary brachial plexus block for hand, wrist or forearm surgery. In: Koscielniak-Nielsen ZJ, editor. Cochrane Database Syst Rev. Chichester: Wiley; 2006;(1):CD003842.
39. Neal JM, Gerancher JC, Hebl JR, Ilfeld BM, McCartney CJ, Franco CD, Hogan QH. Upper extremity regional anesthesia: essentials of our current understanding, 2008: Erratum in Reg Anesth Pain Med. 2010;35(4):407.
40. Chan V. Ultrasound imaging for regional anesthesia: a practical guide booklet , Etobicoke; Ontario, 2008.
41. Spence B, Sites B, Beach M. Ultrasound-guided musculocutaneous nerve block: a description of a novel technique. Reg Anesth Pain Med. 2005;30(2):198–201.
42. Soong J, Schafhalter-Zoppoth I, Gray AT. The importance of transducer angle to ultrasound visibility of the femoral nerve. Reg Anesth Pain Med. 2005;30(5):505.
43. Tsui BCH, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents. Anesthesiology. 2010;112(2):473–92.
44. Saranteas T, Chantzi C, Paraskeuopoulos T, Alevizou A, Zogojiannis J, Dimitriou V, et al. Imaging in anesthesia: the role of 4 MHz to 7 MHz sector array ultrasound probe in the identification of the sciatic nerve at different anatomic locations. Reg Anesth Pain Med. 2007;32(6):537–8.

45. Chan VWS, Nova H, Abbas S, McCartney CJL, Perlas A, Quan Xu D. Ultrasound examination and localization of the sciatic nerve. Anesthesiology. 2006;104(2):309–14.
46. Barrington M, Lai S, Briggs C, Ivanusic J, Gledhill S. Ultrasound-guided midthigh sciatic nerve block—a clinical and anatomical study. Reg Anesth Pain Med. 2008;33(4):369–76.
47. Horn J-L, Pitsch T, Salinas F, Benninger B. Anatomic basis to the ultrasound-guided approach for saphenous nerve blockade. Reg Anesth Pain Med. 2009;34(5):486–9.
48. Krombach J, Gray AT. Sonography for saphenous nerve block near the adductor canal. Reg Anesth Pain Med. 2007;32(4):369–70.
49. Kirkpatrick JD, Sites BD, Antonakakis JG. Preliminary experience with a new approach to performing an ultrasound-guided saphenous nerve block in the mid to proximal femur. Reg Anesth Pain Med. 2010;35(2):222–3.
50. Wang D, Yang Y, Li Q, Tang S-L, Zeng W-N, Xu J, et al. Adductor canal block versus femoral nerve block for total knee arthroplasty: a meta-analysis of randomized controlled trials. Sci Rep. 2017;7:40721. Nature Publishing Group.
51. Kim DH, Lin Y, Goytizolo EA, Kahn RL, Maalouf DB, Manohar A, et al. Adductor canal block versus femoral nerve block for total knee arthroplasty a prospective, randomized, controlled trial. Anesthesiology. 2014;120(3):540–50.
52. Manickam B, Perlas A, Duggan E, Brull R, Chan VWS, Ramlogan R. Feasibility and efficacy of ultrasound-guided block of the saphenous nerve in the adductor canal. Reg Anesth Pain Med. 2009;34(6):578–80.
53. Abrahams MS, Horn J-L, Noles LM, Aziz MF. Evidence-based medicine: ultrasound guidance for truncal blocks. Reg Anesth Pain Med. 2010;35(2):S36–42.
54. McDonnell J, ODonnell B, Farrell T, Gough N, Tuite D, Power C, et al. Transversus abdominis plane block: a cadaveric and radiological evaluation. Reg Anesth Pain Med. 2007;32(5):399–404.
55. Suresh S, Chan VWS. Ultrasound guided transversus abdominis plane block in infants, children and adolescents: a simple procedural guidance for their performance. Pediatr Anesth. 2009;19(4):296–9. Blackwell.
56. Farooq M, Carey M. A case of liver trauma with a blunt regional anesthesia needle while performing transversus abdominis plane block. Reg Anesth Pain Med. 2008;33(3):274.
57. O'Donnell BD, Mannion S. A case of liver trauma with a blunt regional anesthesia needle while performing transversus abdominis plane block. Reg Anesth Pain Med. 2009;34(1):75–6.
58. Jankovic Z, Ahmad N, Ravishankar N, Archer F. Transversus abdominis plane block: how safe is it? Anesth Analg. 2008;107(5):1758–9.
59. Azemati S, Khosravi MB. An assessment of the value of rectus sheath block for postlaparoscopic pain in gynecologic surgery. J Minim Invas Gynecol. 2005;12(1):12–5.
60. Dolan J, Lucie P, Geary T, Smith M, GNC K. The rectus sheath block: accuracy of local anesthetic placement by trainee anesthesiologists using loss of resistance or ultrasound guidance. Reg Anesth Pain Med. 2009;34(3):247–50.
61. Willschke H. Ultrasonography-guided rectus sheath block in paediatric anaesthesia–a new approach to an old technique. Br J Anaesth. 2006;97(2):244–9.
62. Carline L, McLeod GA, Lamb C. A cadaver study comparing spread of dye and nerve involvement after three different quadratus lumborum blocks. In: Colvin L, editor. Br J Anaesth. 2016;117(3):387–394. Oxford University Press.
63. Murouchi T, Iwasaki S, Yamakage M. Quadratus lumborum block: analgesic effects and chronological ropivacaine concentrations after laparoscopic surgery. Reg Anesth Pain Med. 2016;41(2):146–50.
64. Barrington MJ, Ivanusic JJ, Rozen WM, Hebbard P. Spread of injectate after ultrasound-guided subcostal transversus abdominis plane block: a cadaveric study. Anaesthesia. 2009;64(7):745–50. Blackwell.
65. Børglum J, Moriggl B, Jensen K, Lönnqvist PA, Christensen AF, Sauter A, et al. Ultrasound-guided transmuscular quadratus lumborum blockade. Br J Anaesth. 2013;111:(eLetters Supplement).
66. Davies RG, Myles PS, Graham JM. A comparison of the analgesic efficacy and side-effects of paravertebral vs epidural blockade for thoracotomy—a systematic review and meta-analysis of randomized trials. Br J Anaesth. 2006;96(4):418–26. Oxford University Press.
67. Karmakar MK. Thoracic paravertebral block. Anesthesiology. 2001;95(3):771–80.
68. Krediet AC, Moayeri N. Different approaches to ultrasound-guided thoracic paravertebral block. An illustrated review. Anesthesiology. 2015;123(2):459–74.
69. Abdallah FW, Brull R. Off side! A simple modification to the parasagittal in-plane approach for paravertebral block. Reg Anesth Pain Med. 2014;39(3):240–2.
70. Tsui BCH, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents. Anesthesiology. 2010;112(3):719–28.
71. Ivani G, Suresh S, Ecoffey C, Bosenberg A, Lonnqvist P-A, Krane E, et al. The European Society of Regional Anaesthesia and Pain Therapy and the American Society of Regional Anesthesia and Pain Medicine Joint Committee Practice Advisory on Controversial Topics in Pediatric Regional Anesthesia. Reg Anesth Pain Med. 2015;40(5):526–32.
72. Taenzer A, Walker BJ, Bosenberg AT, Krane EJ, Martin LD, Polaner DM, Wolf C, Suresh S. Interscalene brachial plexus blocks under general anesthesia in children: is this safe practice?: A report from the Pediatric Regional Anesthesia Network (PRAN). Reg Anesth Pain Med. 2014;39(6):502–5.
73. Willschke H, Marhofer P, Machata AM, Lönnqvist PA. Current trends in paediatric regional anaesthesia. Anaesthesia. 2010;65(s1):97–104. Blackwell.

Part IV

Continuous Regional Analgesia and Techniques for Regional Anesthesia

10 Equipment and Clinical Practice: Aids to Localization of Peripheral Nerves

Bryan Tischenkel, Beverly Pearce-Smith, Johnny K. Lee, and Karina Gritsenko

Introduction

Prior to the beginning of the discussion on localization of peripheral nerves, one must consider the location and circumstances under which one is performing these peripheral nerve blocks. It is imperative to ensure the presence of standard monitoring equipment (EKG, blood pressure, pulse oxygenation), wall oxygen, emergency medications (e.g., phenylephrine, ephedrine, epinephrine), resuscitation equipment (bag valve mask, laryngoscopes, and endotracheal tubes), and intralipid rescue prior to performance of a nerve block.

Identifying nerve location begins by identifying standard anatomical landmarks, which are used as a basis for subsequent invasive needle exploration. The successful endpoint used may be anatomical (transarterial axillary block), ultrasonographic (real-time imaging), or functional [a sensory paresthesia or a motor response to electrical nerve stimulation (NS)].

In the 1960s, electrical nerve stimulation techniques were developed. Even more recently, small, battery-operated, portable handheld devices have been introduced [1–3]. The theory behind the use of the nerve stimulator is that an identifiable specific muscle twitch can be observed by nerve stimulation during needle advancement to provide a reference for the appropriate distance of the needle to the nerve. The technique is further described below. After its invention, this technique enjoyed widespread use, with a proven clinical efficacy and safety record. Electrical stimulus of the nerve is based on factors such as conductive area of the electrode, resistance to electrical stimulation, distance between skin and nerve, current flow, and pulse duration (Fig. 10.1) [4].

Although ultrasound had previously been applied in other areas of clinical practice, after the advent of the peripheral nerve stimulator, the use of ultrasound to locate both peripheral nerves and their surrounding structures became progressively more widespread in the world of regional anesthesia. This allows the user to have concrete visual feedback to avoid damage to surrounding structures, including nerves and vasculature, while locating the needle tip at all times. It also allows visualization of the spread of local anesthetic [5].

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_10

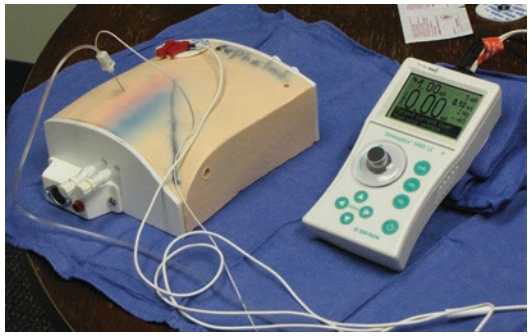

Fig. 10.1 Nerve stimulator, with stimulating needle/ground lead, attached to a simulated model

Peripheral Nerve Stimulation

A nerve stimulator works by applying a weak, direct current (DC) electrical current to a stimulating needle by an oscillating current generator. Assuming that a square pulse of current is used to stimulate the nerve, the total energy (charge) applied to the nerve is the product of the current intensity and the pulse duration. Ohm's law ($V = IR$) assists us in calculating the current generated by the stimulator [6]:

$$\text{Voltage output} / \text{impedance} = \text{current output (Ohm's law)}$$

or

$$V / R = I.$$

Technique

The nerve stimulator is first connected to a stimulating needle. A current is passed through the needle at an amplitude/intensity of 1–2 mA, a frequency (f) of 1–2 Hz, and a pulse duration of 0.1–0.2 ms. The needle is then inserted through the skin and slowly advanced toward the expected anatomical location of the targeted nerve. In each anatomical location, an expected twitch will be observed based on targeting specific nerves to be stimulated. When the desired motor response is elicited, the current intensity is gradually decreased until it is abolished. If the motor contraction is abolished at a relatively higher intensity (>0.6 mA), the needle should be advanced until a further motor response is elicited. This process should be repeated until the motor response disappears at approximately 0.4 mA. This indicates that the needle tip is in close enough proximity to the nerve (1–2 mm) to inject the desired local anesthetic. If a muscle twitch is generated at a current strength of less than 0.4 mA, the stimulating needle may have penetrated the epineurium, thus risking a subsequent intraneural injection. It is therefore important to ensure that the muscle twitch disappears at or higher than a current of 0.4–0.5 mA.

As a side note, it is possible for a current to be generated through a muscle leading to direct generalized muscle stimulation; this should not be confused with a neural stimulation as an intramuscular injection likely would not provide an accurate nerve block.

In practice, the PNS should not be used as a substitute for proper knowledge of anatomy, as no motor response will occur if the needle tip is greater than 1 cm from the targeted nerve. PNS should be used to refine the search endpoint, guiding the needle through the final 5 mm or so.

Another limitation of the PNS technique is that PNS is limited in application to mixed peripheral nerves because a motor response endpoint is desired. Although pure sensory nerves may be stimulated, ultimately obtaining a sensory paresthesia, this is not commonly performed clinically.

Electrophysiology

Energy

The amount of electrical energy required to propagate a nerve impulse is the product of the stimulus strength (mA) and current duration (ms). For any nerve type, there is a minimum current strength required in order to generate an impulse—this is referred to as the rheobase. Below this minimum level, an impulse will not be generated. The chronaxie is defined as the stimulus duration needed for impulse generation,

when employing a current strength of twice the rheobase.

Myelinated fibers are much more sensitive and require less electrical energy for stimulation than unmyelinated fibers. Therefore, if less electrical energy is required to propagate a motor nerve impulse, this means that either the stimulus strength required may be lower than that for a sensory nerve or the current duration may be lower than that required for a sensory nerve (lower rheobase or shorter chronaxie). The clinical relevance of this concept and application to peripheral nerve stimulator use is that the goal of nerve stimulation is to stimulate muscular contractions while avoiding painful sensory nerve stimulation. Since the chronaxie of A alpha fibers is 50–100 μs while A delta fibers require 170 μs and C fibers require 400 μs, it would be wise to use shorter impulse durations (<0.17 ms) in order to attempt to stimulate only the A alpha fibers. Alternatively, one may use a weaker stimulus strength with a longer pulse duration (e.g., >0.5 mA) to elicit the same motor response. If either current intensity or pulse duration becomes too high, uncomfortable paresthesia-like sensations often occur.

Polarity

A nerve impulse is propagated when a threshold potential is reached, causing depolarization of the nerve. Typically, the nerve has a resting potential of around −60 mV, with negative charges inside the cell and positive charges on the outer membrane. To cause a decrease in the potential difference between the inside and outside of the cell, a negative charge should be introduced outside the cell. Therefore, less electrical energy would be required if the negatively charged cathode is close to the nerve, inducing direct depolarization. The reverse is true with an anodal (positive) needle since the direction of flow would induce hyperpolarization of the target nerve. This, in turn, requires a higher current to stimulate the nerve. For these reasons, the needle polarity is designated negative by default. The site of placement of the positive (return) electrode, however, is probably irrelevant, as long as quality grounding electrodes are used and good electrical contact is made.

Distance

The relationship between the constant current stimulus intensity and the distance from the nerve is governed by Coulomb's law:

$$I = K\left(\frac{Q}{r^2}\right),$$

where I is the stimulus intensity, K is a constant, Q is the minimal current needed for stimulation, and r is the distance from the stimulus to the nerve.

Rearranging this formula, we get:

$$\frac{I}{K} = \frac{Q}{r^2}$$

and

$$\frac{I}{K}r^2 = Q.$$

Since $\frac{I}{K}$ is a constant:

$$r^2 \sim Q.$$

This means that as distance from the nerve increases, the charge required to stimulate the nerve increases by the square of the increased distance, requiring a very high current intensity as the needle moves further away from the nerve.

Stimulus Frequency

As the needle is advanced, a muscle twitch by the stimulating current indicates that the needle is approaching the target nerve. If the frequency of impulses is too low, the nerve may be inadvertently penetrated. If the frequency is too high, painful muscle twitches (tetany) may be induced. A frequency of 2 Hz (cycles/s) is a good compromise as well as a suggested nee-

dle advancement speed of approximately 1 mm/s [12].

Summary

A peripheral nerve stimulator should provide as a minimum:

1. A square wave impulse with a duration of 0.1 ms.
2. The negative lead connected to the stimulating needle.
3. 2 Hz frequency.
4. Initial current level of 1–2 mA, seeking the nerve.
5. A final current level of 0.4–0.6 mA, positioning the needle tip close to the nerve.
6. Current delivery down to 0.1–0.2 mA, to ensure no intraneural stimulation.

Additional safety features include:

1. Accurate current delivery in the range of 0–5.0 mA.
2. Constant current square wave pulse.
3. Display of current flowing into the patient as well as that delivered internally from the device.
4. Open circuit alarm.
5. Excessive impedance alarm.
6. Low battery alarm.
7. Internal malfunction alarm [6].

New Developments in Nerve Stimulation

Percutaneous Electrode Guidance

The percutaneous electrode guidance (PEG) technique is a modification of transcutaneous NS: a percutaneous nerve electrode coupled to a nerve stimulator can be used to locate an underlying nerve by passing the superficial electrode over standard anatomic landmarks. Cutaneous stimulation of the underlying nerve occurs at nerve stimulator settings between 2 and 10 mA, with a 0.1-ms pulse duration (alternatively, 0–5 mA, pulse duration 0.2–1.0 ms). Cutaneous stimulation benefits from a longer pulse duration (0.2–1.0 ms), which enables an electrical motor response at a lower current. Since much of the initial stimulation is done by the probe, which indents the skin toward the nerve, the stimulating needle tip (inserted from within the outer PEG cannula) travels only a short distance in order to finally contact the nerve. Skin indentation during the performance of the PEG technique allows for a decrease in impedance as well as a maximal increase in electrical conductance. Thus, PEG has the net effect of eliciting a motor response with minimal discomfort to the patient [6, 13, 14].

Sequential Electrical Nerve Stimulation

Presently, current amplitude (amperage) is continuously varied, deliberately maintaining a constant frequency and pulse duration (one degree of freedom). Therefore, only one constant fixed pulse duration has been used (e.g., 0.1 or 0.2 ms). Some newer nerve stimulators allow the pulse duration to be preset at different fixed pulse widths (e.g., 0.05, 0.1, 0.3, 0.5, or 1.0 ms). However, this pulse duration cannot be easily varied during the actual block performance. Urmey and Grossi [15] evaluated a novel technique for nerve localization utilizing an electrical nerve stimulator programmed to deliver sequenced electrical nerve stimuli (SENS). The nerve stimulator generated alternating sequential electrical pulses of differing pulse durations at an overall set frequency of 3 Hz (3 cycles/s). Repeating pulse duration sequences of 0.1, 0.3, and 1.0 ms (shortest to longest) were generated, with 1/3-s period intervals separating each pulse.

Selective attenuation of the applied current resulted in the three pulses having more equivalent charges. In each case, the needle was advanced at an initial current amplitude of 1 mA until appropriate motor responses (MR) occurred. If 1 MR/s or 2 MR/s were noted, the needle was continually advanced until all 3 MR/s were visible. Current was then decreased until MR/s

decreased to 1 or 2. At this point, the needle was again advanced slowly. When 3 MRs occurred at ≤0.5 mA, indicating that the 0.1-ms pulse was stimulating the nerve, final needle position was held constant. Prior to final injection, current was then slowly decreased with the needle held immobile.

Conventionally, increasing the current flow has been the only parameter used to increase stimulation range since it directly enables stimulation at a greater distance from the nerve. Additionally, with SENS, pulse durations of 0.3–1.0 ms were used almost simultaneously to increase the range, in distance, of successful stimulation at a given current amplitude. Therefore, higher pulse durations increase sensitivity for successful NS with the stimulator needle at a distance, whereas specificity is then enhanced by decreasing the pulse duration down to the standard 0.1 ms. By employing sequential long and short pulses, successful neurostimulation was able to occur at a much greater needle to nerve distance. Prior to SENS, these elicited motor responses did not occur with the standard 0.1-ms pulses. Thus, the near simultaneous variance of two separate parameters (applied current together with pulse width duration) enhanced successful PNS of the targeted motor nerve [6].

Ultrasound

As mentioned above, the use of ultrasound to locate peripheral nerves and their surrounding structures has become progressively more widespread in regional anesthetic practices. Ultrasound allows the operator to visualize their target nerve while seeing their needle tip in real time. It also allows the visualization of local anesthetic spread around the target nerve and allows us to avoid undesirable structures (i.e., intravascular or intraneural injections) [7]. Many of these benefits may not be provided by the anatomic and nerve stimulator-guided techniques of peripheral nerve block insertion. One study showed that in an appropriately imaged supraclavicular nerve block, a peripheral nerve stimulator adds no benefit [8]. This study demonstrated that ultrasound guidance may serve as a substitute for peripheral nerve stimulation (in patients with normal anatomy). Another study showed that for sciatic nerve block, ultrasound guidance resulted in higher success, a faster block onset, and faster progression of sensorimotor block while not increasing nerve block performance time or complications as compared to peripheral nerve stimulator-guided procedure [9]. According to the American Society of Regional Anesthesia and Pain Medicine, ultrasound guidance has improved the incidence of pneumothorax and local anesthetic systemic toxicity and the incidence and intensity of hemidiaphragmatic paralysis (unpredictable manner), but has no significant effect on the incidence of postoperative neurological symptoms [10]. While these benefits have been affirmed, one must always take into account the technical capabilities of both the ultrasound machine and the operator [11]. As ultrasonography is discussed elsewhere in this textbook, for a more in-depth discussion on ultrasound's involvement with regional anesthesia, please see Chap. 9.

Positioning

As with any aspect of clinical care, the positioning of the patient can decide the success or failure of the procedure at hand. Optimization of anatomy is essential to the success of any nerve block no matter what adjuvant techniques are available. This becomes even more important when taking into consideration patients of a different body habitus than average.

In some cases, pillows, towels, or a second practitioner may be used to assist in positioning a patient (supine, lateral, prone, sitting, etc.). In addition, specific positioning devices that assist practitioners when performing blocks are also available. One example includes a device which can maintain an elevated extremity; for example, a supine patient with a planned popliteal nerve block can be maintained with less need for counterpressure along the leg for visualization of the nerve. A table which can allow for adjustable height also can help the practitioner raise the leg instead of using stacks of blankets or pillows.

These examples allow the patient to remain supine while properly propping the lower extremity in position for the peripheral nerve block. This also allows access to the airway in the setting of sedation, allows for efficient nerve blocks, and requires no additional space, as it is placed directly on the patient's bed.

Regional Anesthesia Equipment Tray

When performing a regional anesthetic procedure, pre-made kits with needed supplies can be useful to perform the block. Not only does this provide organization and efficiency while performing the block but can also be added as a separate billable charge bundle, as the equipment used is not part of the typical general anesthetic protocol, leading to possible revenue [16]. Depending on the institution, regional anesthetic equipment kits can include, but are not limited to, nasal cannulae, EKG electrodes, pulse oximeter, needles, syringes, stopcock, sterile gloves, midazolam and fentanyl for sedation, and insulated nerve block needles. When placing a continuous nerve block catheter, the kit may be expanded to include a nerve block catheter, sterile drapes, sterile dressing, and a local anesthetic infusion device. Kits can also be custom-made to address the needs of the particular clinical setting and expected procedures to be performed.

Skin Preparation

Infection is a rare complication associated with regional anesthetic procedures but is a concern nonetheless. The concern has grown greater with the growing popularity of perineural catheters, as these can be indwelling devices. Two common antiseptics used to prepare the skin for the procedure are povidone-iodine and chlorhexidine. While both may kill organisms on the skin and some meta-analyses show similar results, there is a wealth of literature supporting the use of chlorhexidine over povidone-iodine. Chlorhexidine was demonstrated to have more rapid of onset and more efficacy at antisepsis with longer duration when being used for epidural or even central venous catheter or arterial line placement [17–21]. While this may be true and despite the increased effectiveness of chlorhexidine (chlorhexidine is bactericidal in nature) over povidone-iodine, chlorhexidine is not FDA approved for use during perineural administration due to concerns for neurotoxicity. There have been several case reports of chronic adhesive arachnoiditis associated with both epidurally and spinally injected chlorhexidine [22]. This concern may be secondary to the chlorhexidine itself or to the primary ingredient in the chlorhexidine skin preparation (alcohol) [23]. Alcohol has long been known to be used for neurolytic blocks and thus may be the cause of neurotoxicity, though it is unclear.

Ultrasound Transducer Covers

For single-shot and continuous peripheral nerve blocks in practice of the authors, the ultrasound transducer is typically covered in a clear, plastic, sterile sheath. This allows the practitioner to visualize ultrasound gel properly covering the transducer, permitting the ultrasound machine to have proper coupling between sound waves and tissue. In some practices, the ultrasound transducer is covered with a transparent film dressing only. The advantage to using the longer probe sheath is that the procedure is performed with higher sterility. The disadvantages, as identified by Tsui et al., consist of increased cost and the possibility of air tracking between the transducer and the inside of the sheath, producing a poorer image quality [24].

Injection Pressure Monitoring [25]

Another method that has been developed in order to improve safety and help avoid intraneural injection has been to monitor the injection pressure during perineural local anesthetic injection. One study performed in canines indicated that high injection pressures may indicate intraneural needle placement, leading to neurologic injury

and deficits. It also found that injecting intrafascicularly with a pressure monitor required pressures >25 psi due to the low compliance of injecting into perineurium [26]. There are multiple techniques to monitor injection pressures. The compressed-air technique is when one draws up 10 mL of air with 10 mL of saline. If one holds the syringe upright and only compresses the air to half of its original volume when injecting, the practitioner cannot exceed 15 psi of pressure [27]. Another option that has been used is a disposable manometer. Placed between the syringe and tubing, the manometer can measure pressure directly from the syringe into the spring-loaded manometer of pressures <15, 15–20, or >20 psi.

All of the above being true, the cost of extra devices can be high, the compressed-air technique can be time-consuming to set up, and the risk of nerve injury is quite low; therefore, it is not commonplace to monitor injection pressures in all practices. In addition, it is a common belief that intraneural injection can be avoided by the sensation of resistance during injection and that smaller volume syringes enable the clinician to better feel this resistance, but in a recent animal study, the conclusion was that syringe feel was no better than chance at detecting intraneural injection when using a 20 mL syringe [28].

Needles

There are several different needles employed during regional anesthetic procedures. Each needle provides different advantages.

Stimulating Needles

Stimulating needles, also known as insulated needles, have a protective nonconducting sheath over the shaft of the needle, with the exception of the tip. By applying an electrical current to the needle using a nerve stimulator, it is possible to stimulate a nerve as the tip of the needle comes in proximity of the nerve.

When comparing nerve blocks using a nerve stimulator with stimulating needles versus ultrasound alone, studies have demonstrated quicker onset of sensory and motor blockade and longer duration with US guidance compared to peripheral nerve stimulation alone [29, 30].

Echogenic Block Needles

As there is an increasing prevalence in the use of ultrasound to guide the placement of peripheral nerve blocks and catheters, the development of an echogenic block needle was a logical next step. The primary purpose of these needles is to better visualize the needle tip during particularly difficult nerve block procedures, such as those requiring steeper insertion angles, though they may be used to improve visualization in novice anesthesiologists for blocks with shallow angles as well. Many of these needles exist in various forms and with different names (textured, reflector, cornerstone/corner cube reflector [CCR]). Textured reflecting surfaces are typically placed at the tip of the needles, which allow ultrasonic waves to reflect back to the ultrasound probe at any insertion angle. This technology is similar to bicycle reflectors [31]. Kamada et al. used gel phantoms to compare CCR needles with standard block needles and showed that a lower optical density (better echogenicity) and a better luminance occurred with the CCR needles than with standard block needles at an insertion angle of 30° [32]. Kilicaslan et al. used beef phantom models to demonstrate that inexperienced users of block needles were able to complete a block procedure more quickly with better visibility of the needle tip at insertion angles between 42 and 64° [33]. Finally, Brookes et al. demonstrated in vivo that patients receiving proximal sciatic nerve blocks for total knee arthroplasties had shorter procedure times, fewer needle redirections, and decreased patient discomfort with an echogenic needle versus a plain stimulating needle [34].

Needle Gauge

In general, a 21–22 G needle is used to place single-shot nerve blocks. Needles with smaller

gauge improve patient comfort while placing the block; however, they are more difficult to inject through. These needles are usually used for local infiltration at the skin.

Larger bore needles can be useful when placing catheters. A 17–18 G Tuohy needle is commonly used to place perineural catheters as a larger needle is needed in order for a catheter to be able to pass through a port site.

Needle Bevel

There have been several studies concerning mechanical trauma in regard to the needle bevel. Short-beveled and <45° needles may have less incidence of nerve trauma: however, when nerve injury does occur, it may be more severe compared to long-beveled needles, >45° [35–38].

Catheters

Recently, perineural catheters have become popular to provide anesthesia and analgesia for surgical cases, especially orthopedic and vascular procedures. Brachial plexus catheters have been shown to have favorable outcomes [39]. Interscalene catheters have been shown to decrease pain and opioid usage after shoulder surgery [40]. Sciatic nerve catheters in both the popliteal fossa and the subgluteal crease improve analgesia in the postoperative period following foot surgery [41]. Finally, adductor canal catheters have also been used successfully with dilute local anesthetics to improve pain control following total knee replacement while minimizing the motor blockade associated with continuous femoral nerve blockade [42].

Infusion catheters have been useful for continuous infusion of perineural local anesthetic. Catheters usually come in a 19–22 G size. Smaller gauge catheters may increase resistance to infusing medication through. They also may be more difficult to thread and more likely to kink.

The catheters themselves have been constructed with several different properties and varieties. They can be single port or multiorifice. Single-port catheters have their opening at the distal tip of the catheter. With single-port catheters, test injectate is all expelled from the single orifice, allowing for a more reliable test dose to the same area, should the orifice of the catheter be intravascular or intrathecal. In contrast, multiorifice catheters may be less likely to plug given the multiple openings and may be more reliable for detection of intrathecal or intravascular placement via aspiration.

Newer catheters may be made of different materials, leading to differences in stiffness and flexibility. Metal reinforced catheters may be difficult to kink; however, they are not MRI compatible and should be noted when placed. In addition, those catheters known to be more stiff may have a greater likelihood to puncture a structure, such as a vessel.

In addition, stimulating catheters that are able to conduct an electrical current have been used. By applying an electrical current along the catheter and finding a subsequent muscle twitch along the expected nerve distribution, the catheter is theoretically in closer proximity to that nerve. Data comparing stimulating catheters to nonstimulating catheters has been conflicting. While some studies have demonstrated that stimulating catheters may lead to better analgesic quality [43] and a higher success rate following nerve block in combination with ultrasound guidance [44], other studies have demonstrated a similar quality of analgesia and increased time to proper catheter positioning [45].

Given the prevalent use of ultrasound in today's regional anesthetic practices, and the continued use of perineural catheters to prolong patient analgesia, echogenic nerve catheters are now an additional tool of our anesthetic practice. Data comparing stimulating catheter and needle versus echogenic needles showed a decrease in procedure time and patient discomfort with echogenic needle and catheter versus stimulating needle and catheter, with no difference in visibility during sciatic nerve block with a low-frequency ultrasound transducer [34]. In addition, ultrasound has been used in unique ways to identify the location of a perineural

catheter. Using a catheter with a guidewire, the removal and reinsertion of the guidewire (pumping maneuver) produced a color Doppler effect along the track of the catheter and created an M-mode tracing to help identify its proper placement [46].

Conclusion

As we look back upon the history of regional anesthesia, it is clear that while peripheral nerve blockade was previously performed without adjuncts for nerve localization, the complication rates were higher, and the success rates were lower. With further technological advances (i.e., ultrasound, peripheral nerve stimulation, injection pressure monitors, positioning devices, echogenic needles and catheters), we are now able to perform peripheral nerve block more safely and successfully.

Review Questions

1. Identifying nerve location can be:
   (a) Anatomic
   (b) Ultrasonographic
   (c) Functional
   (d) All of the above
2. Electrical nerve stimulation is based on:
   (a) Conductive area
   (b) Resistance
   (c) Distance
   (d) All of the above
3. A nerve stimulator:
   (a) Applies a constant current impulse
   (b) Charge = (I) × (pulse duration)
   (c) Utilizes an oscillating current
   (d) (b) and (c)
4. Nerve stimulator current:
   (a) Is pulsed at 1–2 Hz (cycles/s)
   (b) Starts at 10 mA
   (c) Pulse duration of 0.1–0.2 ms
   (d) (a) and (c)
5. Motor contractions:
   (a) Occur at low current (0.2–0.5 mA)
   (b) Do not occur greater than 1 cm from needle tip to nerve
   (c) Start at a current of 1–2 mA
   (d) All of the above
6. Pure sensory nerves:
   (a) May be stimulated
   (b) Endpoint is a sensory paresthesia
   (c) Are not commonly stimulated clinically
   (d) All of the above
7. When using a high-intensity current (>1 mA):
   (a) Motor and sensory stimulation occurs
   (b) Painful C fiber activity occurs
   (c) More current is required with longer duration (>0.5 mA)
   (d) (a) and (b)
8. True statements regarding needle polarity and grounding are:
   (a) Needle is negative by default
   (b) The grounding electrode should be applied within 6 in. of the target nerve
   (c) A positive needle requires a higher stimulating current
   (d) (a) and (c)
9. Ultrasound-guided regional anesthesia provides all of the following benefits except:
   (a) Decreased incidence of pneumothorax
   (b) Decreased incidence of local anesthetic systemic toxicity
   (c) Decreased incidence of postoperative neurological symptoms
   (d) Decreased incidence of hemidiaphragmatic paralysis
10. Chlorhexidine is more advantageous over povidone-iodine in regard to antisepsis because it:
    (a) Is more rapid in onset
    (b) More efficacious
    (c) Has longer duration antimicrobial activity
    (d) All of the above
11. Short-beveled needles compared to long-beveled needles:
    (a) Have less incidence of nerve trauma
    (b) Have less severe nerve trauma when trauma occurs
    (c) Are >45°
    (d) Are more comfortable for the patient
12. Tuohy needles:
    (a) Have an orifice at the tip in line with the shaft of the needle
    (b) Are nonstimulating needles

(c) Traverse through tissue in a straight line
(d) Are larger bore needles that allow advancement of a catheter through its shaft

13. Single-port catheters compared to multiorifice catheters:
(a) Are more reliable when tested with test injectate
(b) Are more reliable when aspirated for return of fluid
(c) Are more likely to become obstructed by a plug
(d) (a) and (c)
14. Stimulating catheters compared to nonstimulating catheters:
(a) Are equivalent in analgesic quality
(b) Improve block success when used in conjunction with ultrasound when placing infraclavicular blocks
(c) Are only single orifice
(d) (a) and (c)

Answers:

1. d
2. d
3. d
4. d
5. d
6. d
7. d
8. d
9. c
10. d
11. a
12. d
13. a
14. b

References

1. Pither C, et al. The use of peripheral nerve stimulators for regional anaesthesia. A review of experimental characteristics, techniques and clinical applications. Reg Anesth. 1985;10:49–58.
2. Hadzic A, et al. Nerve stimulators used for peripheral nerve blocks vary in their electrical characteristics. Anesthesiology. 2003;98:969–74.
3. Bathram CN. Nerve stimulators for nerve localization: are they all the same? Anaesthesia. 1997;52:761–4.
4. Urmey W. Using the nerve stimulator for peripheral or plexus nerve blocks. Minerva Anestesiol. 2006;72(6):467–71.
5. Marhofer P, Greher M, Kapral S. Ultrasound guidance in regional anesthesia. Br J Anaesth. 2005;94(1):7–17.
6. Gebhard R, Hadzic A, Urmey W. Dual guidance: a multimodal approach to nerve location. Bethlehem: B. Braun Medical Inc.; 2008.
7. Sites BD, Brull R. Ultrasound guidance in peripheral regional anesthesia: philosophy, evidence-based medicine, and techniques. Curr Opin Anaesthesiol. 2006;19(6):630–9.
8. Beach ML, Sites BD, Gallagher JD. Use of a nerve stimulator does not improve the efficacy of ultrasound-guided supraclavicular nerve blocks. J Clin Anesth. 2006;18(8):580–4.
9. Perlas A, Brull R, Chan VW. Ultrasound guidance improves the success of sciatic nerve block at the popliteal fossa. Reg Anesth Pain Med. 2008;33(3):259–65.
10. Neal JM. Ultrasound-guided regional anesthesia and patient safety: update of evidence-based analysis. Reg Anesth Pain Med. 2016;41(2):195–204.
11. Neal JM, Brull R, Horn JL, et al. The Second American Society of Regional Anesthesia and Pain Medicine Evidence-Based Medicine Assessment of Ultrasound-Guided Regional Anesthesia: executive summary. Reg Anesth Pain Med. 2016;41(2):181–94.
12. Tew D, et al. B. Braun satellite symposium XXII. ESRA Congress Malta, 12 Sept 2003.
13. Urmey W, et al. Percutaneous electrode guidance (PEG): a noninvasive technique for pre-location of peripheral nerves to facilitate nerve block. Reg Anesth Pain Med. 2002;27:261–7.
14. Urmey W, et al. Percutaneous electrode guidance (PEG) and subcutaneous stimulating electrode guidance (SSEG): modifications of the original technique. Reg Anesth Pain Med. 2003;28:253–5.
15. Urmey W, et al. Use of sequential electrical nerve stimuli (SENS) for location of the sciatic nerve and lumbar plexus. Reg Anesth Pain Med. 2006;31:463–9.
16. Mariano E. Making it work: setting up a regional anesthesia program that provides value. Anesthesiol Clin. 2008;26(4):681–92.
17. Kinirons B, Mimoz O, Lafendi L, Naas T, Meunier J, Nordmann P. Chlorhexidine versus povidone iodine in preventing colonization of continuous epidural catheters in children: a randomized, controlled trial. Anesthesiology. 2001;94:239–44.
18. Haley CE, Marling-Cason M, Smith JW, Luby JP, Mackowiak PA. Bactericidal activity of antiseptics against methicillin-resistant *Staphylococcus aureus*. J Clin Microbiol. 1985;21:991–2.
19. Maki DG, Ringer M, Alvarado CJ. Prospective randomised trial of povidone-iodine, alcohol, and chlorhexidine for prevention of infection associated with central venous and arterial catheters. Lancet. 1991;338:339–43.
20. Mimoz O, Karim A, Mercat A, Cosseron M, Falissard B, Parker F, et al. Chlorhexidine compared with povidone–iodine as skin preparation before blood cul-

ture: a randomized, controlled trial. Ann Intern Med. 1999;131:834–7.
21. Birnbach DJ, Meadows W, Stein DJ, Murray O, Thys DM, Sordillo EM. Comparison of povidone iodine and DuraPrep, an iodophor-in-isopropyl alcohol solution, for skin disinfection prior to epidural catheter insertion in parturients. Anesthesiology. 2003;98:164–9.
22. Killeen T, Kamat A, Walsh D, Parker A, Aliashkevich A. Severe adhesive arachnoiditis resulting in progressive paraplegia following obstetric spinal anaesthesia: a case report and review. Anaesthesia. 2012;67:1386–94.
23. Patle V. Arachnoiditis: alcohol or chlorhexidine? Anaesthesia. 2013;68:425.
24. Tsui BCH, Twomey C, Finucane BT. Visualization of the brachial plexus in the supraclavicular region using a curved ultrasound probe with a sterile transparent dressing. Reg Anesth Pain Med. 2006;31(2):182–4.
25. Gadsden J, Hadzic A. Chapter 5: Monitoring and documentation. In: Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. NewYork: Mcgraw-Hill; 2012. p. 71–9.
26. Hadzic A, Dilberovic F, Shah S, et al. Combination of intraneural injection and high injection pressure leads to fascicular injury and neurologic deficits in dogs. Reg Anesth Pain Med. 2004;29(5):417–23.
27. Lin JA, Lu HT. A convenient alternative for monitoring opening pressure during multiple needle redirection. Br J Anaesth. 2014;112(4):771–2.
28. Theron PS, Mackay Z, Gonzalez JG, Donaldson N, Blanco R. An animal model of "syringe feel" during peripheral nerve block. Reg Anesth Pain Med. 2009;34(4):330–2.
29. Casati A, Danelli G, Baciarello M, et al. A prospective, randomized comparison between ultrasound and nerve stimulation guidance for multiple injection axillary brachial plexus block. Anesthesiology. 2007;106:992–6.
30. Marhofer P, Sitzwohl C, Greher M, et al. Ultrasound guidance for infraclavicular brachial plexus anaesthesia in children. Anaesthesia. 2004;59:642–64.
31. Hebard S, Hocking G. Echogenic technology can improve needle visibility during ultrasound-guided regional anesthesia. Reg Anesth Pain Med. 2011;36(2):185–9.
32. Kamada T, Yasumura R, Takao R, et al. A quantitative comparative study of a new echogenic needle for nerve blocks. Anesthesiology. 2008;109:A344.
33. Kilicaslan A, Topal A, Tavlan A, et al. Differences in tip visibility and nerve block parameters between two echogenic needles during a simulation study with inexperienced anesthesia trainees. J Anesth. 2014;29(3):460–2.
34. Brookes J, Sondekoppam R, Armstrong K, et al. Comparative evaluation of the visibility and block characteristics of a stimulating needle and catheter vs an echogenic needle and catheter for sciatic nerve block with a low-frequency ultrasound probe. Br J Anaesth. 2015;115(6):912–9.
35. Selander D, Dhuner KG, Lundborg G. Peripheral nerve injury due to injection needles used for regional anesthesia. Acta Anaesthesiol Scand. 1977;21:182–8.
36. Hirasawa Y, Katsumi Y, Kusswetter W, Sprotte G. Peripheral nerve injury due to injection needles: an experimental study. Reg Anaesth. 1990;13:11–5.
37. Maruyama M. Long-tapered double needle used to reduce needle stick injury. Reg Anesth. 1997;22:157–60.
38. Rice AS, McMahon SB. Peripheral nerve injury caused by injection needles used in regional anesthesia: influence of beveled configuration, studied in a rat model. Br J Anaesth. 1992;69:433–8.
39. Klein SM, Grant SA, Greengrass RA, Nielsen KC, Speer KP, White W, et al. Interscalene brachial plexus block with a continuous catheter insertion system and a disposable infusion pump. Anesth Analg. 2000;91:1473–8.
40. Ilfeld BM, Vandenborne K, Duncan PW, Sessler DI, Enneking FK, Shuster JJ, et al. Ambulatory continuous interscalene nerve blocks decrease the time to discharge readiness after total shoulder arthroplasty: a randomized, triple-masked, placebo-controlled study. Anesthesiology. 2006;105:999–1007.
41. di Benedetto P, Casati A, Bertini L, et al. Postoperative analgesia with continuous sciatic nerve block after foot surgery: a prospective, randomized comparison between the popliteal and subgluteal approaches. Anesth Analg. 2002;94(4):996–1000.
42. Zhang W, Hu Y, Tao Y, et al. Ultrasound-guided continuous adductor canal block for analgesia after total knee replacement. Chin Med J (Engl). 2014;127(23):4077–81.
43. Morin AM, Kranke P, Wulf H, et al. The effect of stimulating versus nonstimulating catheter techniques for continuous regional anesthesia: a semi-quantitative systematic review. Reg Anesth Pain Med. 2010;35:194–9.
44. Dhir S, Ganapathy S. Comparative evaluation of ultrasound-guided continuous infraclavicular brachial plexus block with stimulating catheter and traditional technique: a prospective-randomized trial. Acta Anaesthesiol Scand. 2008;52:1158–66.
45. Gandhi K, Lindenmuth DM, Hadzic A, et al. The effect of stimulating versus conventional perineural catheters on postoperative analgesia following ultrasound-guided femoral nerve localization. J Clin Anesth. 2011;23:626–31.
46. Eisharkawy H, Salmasi V, Abd-Elsayed A, et al. Identification of location of nerve catheters using pumping maneuver and M-mode- a novel technique. J Clin Anesth. 2015;27(4):325–30.

Neuraxial Blockade: Subarachnoid Anesthesia

11

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Introduction

The administration of a local anesthetic in the subarachnoid space acts on the spinal roots causing reversible blockage of these nerves. It is a classic technique that has been refined over time and expanded in its practical applications. The development of new drugs and special techniques has been crucial and has greatly influenced the use of spinal anesthesia and its indications.

Anatomy of the Spine

The spine consists of 33 vertebrae: seven cervical, 12 thoracic, five lumbar, five that are fused to form the sacrum, and four fused to form the coccyx. The spine has four curves. The thoracic and sacral curves have concave forward curvature and are primary curvatures formed at birth. The cervical and lumbar curves have forward convex curvatures and are secondary curvatures developed after birth. When the patient is supine the highest point is in L3 and the lowest point is in T5 (Fig. 11.1).

The vertebrae consist of two essential parts: an anterior solid segment or body and a posterior segment or arch. The arch is divided on each side into a pedicle attached to the body and a lamina at the back. The spinous process extends backward from the junction of the two laminae. The junction of the pedicles and laminae forms the transverse process, which extends outward from each side of the arch. The pedicles of each vertebral arch are notched forming an incomplete ring, the intervertebral foramen. The spinal nerves enter and exit through these holes from each side of the vertebral canal (Fig. 11.2).

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The Intervertebral Discs

The intervertebral discs are a pad of fibrous cartilage between adjacent surfaces of the vertebral bodies. They provide mobility and shock absorption to the spine.

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_11

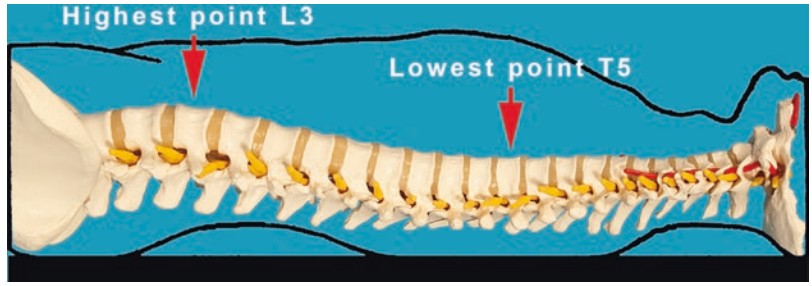

Fig. 11.1 Highest and lowest points on the spine in the supine position

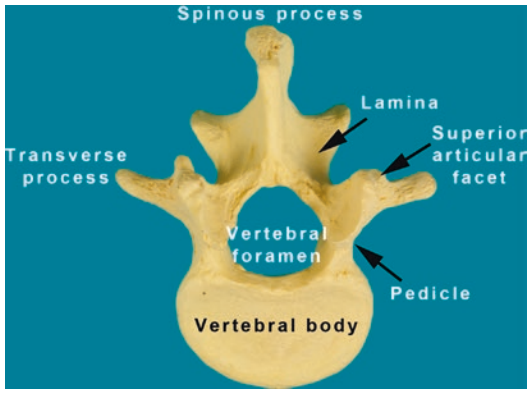

Fig. 11.2 The vertebrae

Five Ligaments

Five ligaments connect the vertebral processes: The supraspinous ligament connects the tips of the spinous processes, and the interspinous ligament connects the spinous processes.

The ligamentum flavum connects the laminae of adjacent vertebrae, forms the posterior border of epidural space at each interlinear space, and consists of elastic fibers. It becomes progressively thicker from front to back, and it is easily recognized by the increased resistance to the passage of the needle. The other two ligaments are the posterior longitudinal ligament and the anterior longitudinal ligament.

The Epidural Space

The epidural space extends from the foramen magnum to the sacral hiatus. It is bounded by the posterior longitudinal ligament at the sides by the pedicles and the intervertebral foramen and posteriorly by the ligamentum flavum. It contains nerve roots, venous plexuses, arteries, and fat.

The Spinal Cord

The spinal cord originates in the brainstem and continues through the occipital foramen magnum ending in the conus medullaris. This distal end ranges from L3 in infants up to the bottom of L1 in adults due to differences in growth between the bony spinal canal and central nervous system. It ends at the conus medullaris from where the lumbar nerve, sacral and coccygeal roots emerge to form the cauda equina (horse tail). It is in this area (below L2) that spinal needles are inserted.

Meninges and Spaces

Meninges and spaces include the pia mater, which is the inner most layer closely attached to the spinal cord and brain. It ends as terminal filum and is highly vascularized. The arachnoid is an avascular membrane tightly attached to the outermost layer, the dura mater. It seems that the arachnoid acts as a major barrier to the flow of drugs from the cerebrospinal fluid (CSF); thus, it would be responsible for 90% of the drug resistance to migration [1, 2].

The dura mater is the third and outermost membrane of the spinal canal. It is the continuation of the cranial dura mater, extending from foramen magnum to S2 (Fig. 11.3).

The subarachnoid space lies between the pia mater and the arachnoid. In it is found the CSF, the spinal nerves, a network of trabeculae between the two membranes, and blood vessels

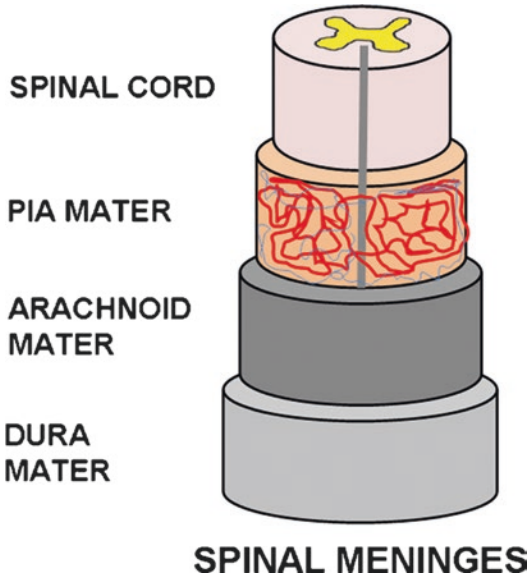

Fig. 11.3 Spinal meninges

supplying the spinal cord. It extends from S2 to the cerebral ventricles. The subdural space is a virtual space between the dura and the arachnoid that contains small amounts of serous fluid that allows the membranes to move past each other.

The Cerebrospinal Fluid (CSF)

The cerebrospinal fluid (CSF) is formed continuously at a rate of 450 mL/day by way of the secretion or plasma ultrafiltration from the choroidal arterial plexus located in the lateral ventricle and the third and fourth ventricle. The CSF is reabsorbed into the bloodstream through the arachnoid villi and granulations, which protrude from the dura mater to be in contact with the endothelium of the cerebral venous sinuses. The CSF serves to protect the brain and spinal cord. The CSF is a determinant of the effects of intrathecally administered substances because all the drugs injected into the subarachnoid space are diluted in the CSF before reaching their target in the spinal cord. It has been noted that the volume of CSF is one of the most important factors affecting the level of sensory block and duration of spinal anesthesia [3, 4]. The volume of CSF varies from one individual to another and, with the exception of weight, is not related to the anthropometric values clinically available.

Physical Properties of CSF

- Clear, liquid, colorless
- Specific gravity: 1003–1009 at 37 °C
- Total volume: 120–150 mL
- Spinal CSF volume: 25–30 mL
- Ventricular CSF volume: 60–75 mL
- Average pressure: 100–150 cm of water
- pH: 7.6

Thirty-One Pairs of Spinal Nerves

Thirty-one pairs of spinal nerves emerge from the spinal cord by the anterior and posterior roots. Each spinal nerve innervates a specific area of skin or dermatome and skeletal muscles.

The Anterior Spinal Root

The anterior spinal root is efferent and contains:

1. Motor fibers (voluntary muscles).
2. Preganglionic sympathetic fibers (T1–L2) join spinal nerves to form the sympathetic chain. The sympathetic chain extends along the entire column (anterolateral side of the vertebral bodies). It gives rise to the stellate ganglion, splanchnic nerves, and celiac plexus. Sympathetic cardio accelerator fibers arise from T1 to T4.
3. The fibers of the parasympathetic nervous system travel in the anterior roots of S2–S4 and supply organs of the pelvis.

The Posterior Spinal Root

The posterior spinal root is afferent. All afferent impulses from the body, including viscera, pass through the posterior roots. Each has a dorsal root ganglion.

Physiology

Neural Blockade

The small-diameter unmyelinated (sympathetic) fibers are blocked more easily than larger myelinated fibers (sensory and motor). As a result, the level of autonomic blockade extends two or three segments above the sensory block, and in the same way, the sensory block extends one to four segments above the motor block. Weaker concentrations of local anesthetic can produce sensory block without causing motor paralysis. The sequence of the blockade of nerve fibers is generally in the following order: (1) vasomotor block, (2) pain, (3) touch, (4) motor, (5) pressure, (6) proprioception.

Cardiovascular Physiology

The nerve block produces sympathectomy two to four dermatomes above the sensory level. This causes arterial and venous vasodilatation, the venodilator effect being predominant because smooth muscle in the arterial side retains a considerable degree of autonomous tone. This causes a decrease in systemic vascular resistance, venous return, and cardiac output, all of which contribute to lower blood pressure. Vasodilation in the lower extremities can be compensated by vasoconstriction in the upper extremities. However, with high thoracic anesthetic levels, vasoconstriction in upper extremities and in the splanchnic bed may be highly reduced, and this can lead to significant hemodynamic instability. Similarly, heart rate in a high neuraxial block decreases by the blockade of sympathetic cardio accelerator fibers (T1–T5), giving rise to a predominant vagal parasympathetic tone. Furthermore, the reduction in venous return induced by spinal anesthesia paradoxically increases vagal tone, and this leads to a marked bradycardia and possible asystole [5].

Respiratory Physiology

The anesthesia in low spinal sites has no effect on ventilation. If the blockade reaches to thoracic areas, there is a paralysis of the intercostal muscles. This has little effect on ventilation since diaphragmatic breathing is regulated by the phrenic nerve. However, the patient may complain of difficulty breathing, and in patients with inadequate respiratory reserve, ventilation might be insufficient. The paralysis of both intercostal and abdominal muscles decreases the patient's ability to cough and clear secretions.

Digestive Physiology and Function

Gastrointestinal hyperperistalsis occurs as a consequence of unopposed parasympathetic activity (vagal). It can cause nausea and vomiting but responds well to atropine.

Genitourinary Physiology and Function

Sacral blockade produces an atonic bladder able to retain large volumes of urine. Efferent sympathetic blockade (T5–L1) causes an increased sphincter tone producing retention. The risk of urinary retention seems lower under spinal anesthesia with a short half-life [6–9]. The most prudent approach is to avoid excessive use of crystalloid intravenous solutions [8, 9].

Thermoregulation Issues

Hypothermia due to spinal anesthesia is caused by the redistribution of heat as a direct result of vasodilation accompanying sympathetic block [10, 11]. This is the most important cause of core hypothermia during the first hour [11]. Hypothermia can remain in patients who undergo major operations under spinal anesthesia if sympathetic blockade persists. Hypothermia risk factors are as follows: age less than 1 month, low temperature in the operating room, second- and third-degree burns, combined general and spinal anesthesia, age over 70, low temperature of the patient before induction, low body weight, and large blood loss, in that order [12]. To reduce the

risk of intraoperative hypothermia, several strategies are recommended: (a) Monitor core temperature. (b) Perform active heating with air blankets (as treatment or, in certain cases, as a prophylactic treatment. (c) Heat fluids to approximately 37 °C. (d) Maintain the temperature of the operating room to over 25 °C. (e) Cover the skin to reduce cutaneous heat loss [10]. (f) Avoid high spinal blocks where possible [13].

Techniques

Preparation is important before the injection of spinal anesthesia. Preoperative evaluation is important. A medical history and physical examination of the patient, including lower back, should be undertaken. The site where the procedure is performed must be equipped with an oxygen source, as well as immediate access to emergency drugs and equipment for resuscitation and intubation. In addition, the patient should be monitored and sedated so he is both comfortable and cooperative.

Spinal needles (Fig. 11.4) are classified into two major categories: those that have conical points and separate the dural fibers (Fig. 11.5) and the ones that cut the dura (Fig. 11.6). Among the former are the Whitacre and Sprotte needles (Fig. 11.5), and the latter will encompass the Quincke-Babcock needles (Fig. 11.6). With the needles that have conical-shaped tips and side openings, the incidence of post-dural puncture headaches (PDPH) decreases [14]. The incidence of PDPH decreases with fine-gauge needles, although it also may increase if numerous attempts to puncture are made since thin needles produce worse tactile sensation during needle placement.

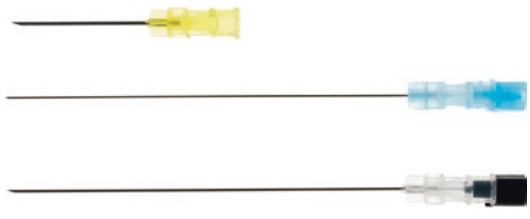

Fig. 11.4 Spinal anesthesia needle types

Fig. 11.5 Quincke-Babcock needles (sharp point)

Fig. 11.6 Whitacre and Sprotte needles (conical point)

Position of the Patient

The choice of position for spinal anesthesia is influenced by a combination of several factors: the preference of the anesthesiologist, patient characteristics, and the baricity of local anesthetic solutions in conjunction with the surgical site. Optimal patient positioning is critical to the success of neuraxial procedures.

Lateral Decubitus Position

The patient is placed with the affected side down if a hyperbaric solution is used or with the affected side up if the anesthetic solution is hypobaric. The spine should be horizontal and parallel to the edge of the table or bed. Maximum deflection of the column must be obtained with the

knees bending to the chest, and the chin should be flexed down into the chest (Fig. 11.7).

Seated Position

The head and shoulders are bent down over the trunk, feet resting on a stool, and the patient's back should be near the edge of the table or bed. One must have an assistant to stabilize the patient, who should not be over-sedated (Fig. 11.8). Many anesthesiologists prefer this position because it helps to identify the midline, especially in obese patients. The line connecting the upper edges of the iliac crests crosses the vertebral body of L4 or the L3–L4 interspaces (Fig. 11.9). If the patient is left in this position for several minutes (with hyperbaric anesthetic solution), a block to the sacral dermatomes (saddle block) is obtained. To achieve a higher spinal block, the patient is placed in supine position immediately after the intrathecal injection. One must be aware of the arterial pressure while the patient is sitting since this position favors the decrease in venous return due to sympathetic effects of spinal block.

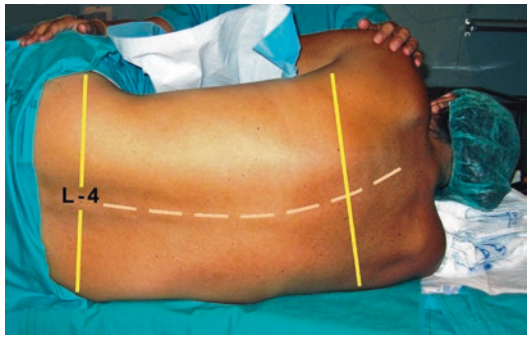

Fig. 11.7 Lateral decubitus position

Fig. 11.8 Seated position

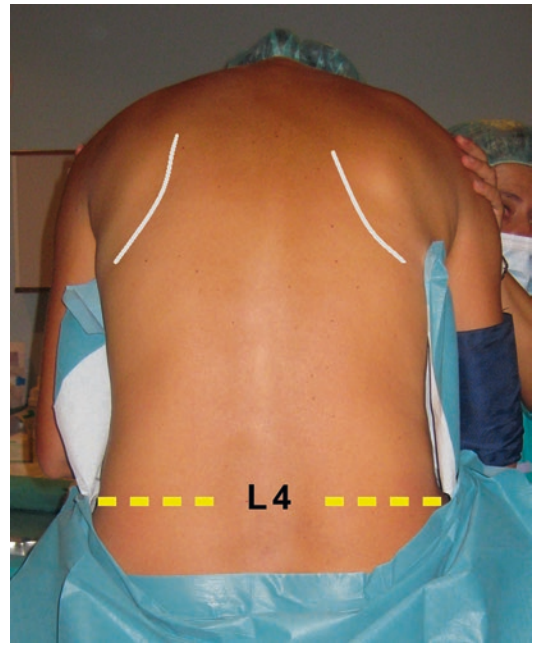

Fig. 11.9 Reference lines and vertebral body of L4

Prone or Jackknife Position

It is used in conjunction with hypobaric anesthesia for procedures in the rectum, perineum, and anus. It is possible to use this position both for surgery and anesthesia (Fig. 11.10).

Anatomic Approach

In general, for spinal anesthesia, spaces L3–L4 and L4–L5 are used. To avoid traumatic puncture of the conus medullaris, the puncture should be below L2. A large area of skin should be prepared,

Fig. 11.10 Prone or jackknife position

avoiding contamination with any antiseptic solution that could be potentially neurotoxic. This should be followed with 1% lidocaine at the site of the spinal needle puncture.

Midline Approach

Using the midline pathway decreases the lumbar lordosis by inserting the spinal needle between adjacent spinous processes. By palpation, the spinous and interspinous spaces are located. A subcutaneous wheal with local anesthetic is then used. The needle used for infiltration with local anesthetic is also used to verify the alignment between the spinous processes. It is necessary to use a spinal needle introducer when the needles are small (25–27 gauges with a pencil tip). If the introducer is properly placed, it should be firmly inserted between the fibers of the interspinous ligament. A spinal needle is inserted through the introducer along its cephalic angulation. The insertion should be slow to heighten the sense of passing through tissue planes to notice the characteristic change when the needle passes through the ligamentum flavum and the dura, where a pop indicating loss of resistance is noted. The stylet must be placed to prevent obstruction of the aperture with tissue. After the stylet is removed, CSF must appear. If CSF does not appear, rotate the needle and, if necessary, adjust the needle tip (e.g., advance, redirect, or withdraw the needle) and repeat the procedure until the CSF is noted. Flow of CSF confirms the position of the needle into the subarachnoid space, and that the tip of the needle has come into contact with part of the cauda equina. If the patient describes a paresthesia at any time, needle advancement should be stopped. Normally, paresthesia serves as an indication that the subarachnoid space has been reached and is usuallly transient and mild. The stylet is then removed, and appearance of CSF should be observed. If the paresthesia has been resolved, a local anesthetic injection should follow. If paresthesia recurs on injecting the local anesthetic, under no circumstances should local anesthetic be injected. It will be necessary to remove the needle and insert it again. The most common cause of not obtaining CSF is that the needle was inserted away from the midline. Another common mistake is insertion of the needle at an excessive cephalad angulation. During the injection, a gentle aspiration of 0.1 or 0.2 mL of CSF to confirm the position in the subarachnoid space could be performed before the local anesthetic injection. The depth of the dura from the skin in patients of normal body habitus is 5.1 cm with a tolerance of 1 cm [15]. The midline approach is suitable for most patients, is easy to learn, and also provides a relatively avascular approach (Fig. 11.11a).

Paramedian Approach

This route is useful in patients who cannot bend properly or when the interspinous ligament is ossified. Local anesthetic is injected 1 cm lateral and 1 cm caudal to the superior spinous process. The needle with the introducer is directed medially and in a slightly cephalad direction and is passed laterally to the supraspinous ligament. The most common error, as occurs in the medial approach, is to go in an excessively cephalad

Fig. 11.11 Midline (**a**) and paramedian (**b**) approach

direction at the insertion. Nevertheless, if a contact with the vertebral lamina is made, the needle should be redirected and introduced in a medial and cephalad direction. As in the case of the midline pathway, the characteristic feeling of the passage through the ligament and the dura can be perceived, although the needle requires a greater depth of insertion. Once CSF is obtained, the injection is performed in the same manner as described in the midline approach (Fig. 11.11b).

Lumbosacral Approach (Taylor)

The lumbosacral vertebral foramen is the largest of the spine. In patients in which the approaches described above do not allow entry into the spinal canal due to calcification, or fusion of the intervertebral spaces, the lateral oblique pathway L5–S1 space (or Taylor approximation) can be the most appropriate to reach the subarachnoid space. It can be used in a seated, lateral, or prone position. The posterior superior iliac spine is identified, and skin is marked 1 cm medial and 1 cm caudal. Also identified and marked is the intervertebral space L5–S1. A spinal needle 120–125 mm long is inserted because the oblique angle creates a great distance to reach the subarachnoid space. A cutaneous wheal is created. Then, the needle is inserted and is directed 45° medial and 45° caudal to the L5–S1 space. Changes of resistance to the passage of the needle through the ligamentum flavum and dura mater are the same as in the medial pathway (Fig. 11.12).

Pre-procedure Ultrasonography

Ultrasound can be used to identify the lumbar interspace for the best needle placement and it is really helpful in determining the depth of the subarachnoid space in obese patients or those with difficult anatomy. The spine can be scanned in transverse and sagittal view (Figs. 11.13 and 11.14). To perform the scan it is important to have experience and training. Also, since it takes additional time and equipment, its benefits are

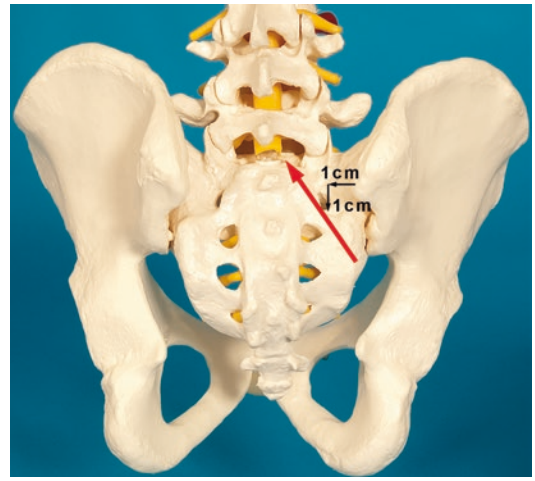

Fig. 11.12 Lumbosacral approach (Taylor)

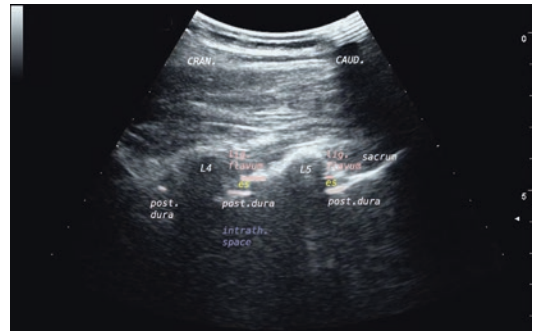

Fig. 11.13 Paramedial oblique sagittal scan of the lumbar spine. Note the hyperechoic bony lines of sacrum caudally and L4 and L5 cranially, *es* epidural space

Fig. 11.14 Transverse scan of the lumbar spine, interspinous L4–L5 level

arguable. Therefore, ultrasonography for spinal anesthesia is not yet routinely used [16].

Pharmacology

General Considerations

Local anesthetics are substances capable of producing a reversible block of the conduction in nerve fibers. Its chemical structure is an aromatic radical attached to an amine structure with a link that can be ester or amide. Amino ester anesthetics are procaine and tetracaine; the amino amides are lidocaine, prilocaine, mepivacaine, bupivacaine, and ropivacaine. The most important clinical properties of local anesthetics are potency, onset, and duration of action. They are differentiated by lipid solubility, pKa, and protein binding. They may be short duration of action or long duration of action. The anesthetics of short duration (≤90 min) are procaine, lidocaine, and mepivacaine. Those of long duration are tetracaine, bupivacaine, ropivacaine, and levobupivacaine.

Local Anesthetics for Spinal Anesthesia

Procaine

It has been used as a local anesthetic for 100 years. Lidocaine replaced it by providing a faster onset and longer duration of action. The recent association of lidocaine with the appearance of transient neurological symptoms (TNS) has renewed interest in the use of procaine in spinal anesthesia because it seems to cause fewer TNS, but it produces more frequent failure of the blockade and also nausea. Additionally, the recuperation period is greater than with lidocaine [17].

Chloroprocaine

It is also a short-acting local anesthetic. In clinical studies with volunteers, it has been shown to have a profile similar to lidocaine, with a lower incidence of TNS. Not approved by the FDA for intrathecal use. Neurotoxicity attributed to preservative sodium bisulfite [18].

Lidocaine

It is considered to be a local anesthetic of intermediate/short-term action. It has historically been the most widely used as a local anesthetic for spinal anesthesia. Its use has greatly diminished because of the incidence of TNS, which varies between 15 and 33% depending on the type of surgery [3, 19].

Mepivacaine

Mepivacaine is associated with a high incidence of transient neurological symptoms, as is the case with lidocaine [19].

Bupivacaine

It is the anesthetic most widely used for long-acting effect. The extent and duration of the blockade is dose dependent. However, it has a great variability due to its high lipid solubility. It is cardio toxic, but the doses used in spinal anesthesia (maximum 20 mg) are too small to produce toxicity. In ambulatory surgery, it has been used in low doses as an alternative to lidocaine, albeit with wide variability and high failure rate [20]. Although a low dose of bupivacaine (e.g., 4–6 mg) has been described for outpatient anesthesia, the prolonged recovery time makes bupivacaine less desirable for ambulatory surgery.

Ropivacaine and Levobupivacaine

These are two long-acting anesthetics that have been developed as an alternative to bupivacaine for epidural anesthesia and nerve blocks due to

Table 11.1 Doses and duration of local anesthetic in spinal anesthesia

Anesthetic	Dose (mg)	To T10 (mg)	To T4 (mg)	Duration (min)	With epinephrine (min)
Procaine	50–200	125	200	45	60
Chloroprocaine	30–100	15–40	40–100	80	–
Lidocaine	25–100	50–75	75–100	60–75	75–100
Mepivacaine	30–60	60	80	70–90	120–180
Tetracaine	5–20	8–14	14–20	70–90	100–150
Bupivacaine	5–20	8–12	12–20	90–110	100–150
Ropivacaine	8–25	12–18	18–25	80–120	–
Levobupivacaine	5–20	8–10	12–20	90–120	100–150

their reduced cardiotoxicity. For spinal anesthesia, cardiotoxicity is not a relevant clinical issue because of the low dose used. Ropivacaine has lower potency than bupivacaine but also allows (40%) early motor recovery. Levobupivacaine is the L-enantiomer of bupivacaine. It is no longer marketed in the USA.

Tetracaine

It is the prototype of the ester type of local anesthetic of long duration. Compared with bupivacaine, it is more potent and has longer life, but it seems to produce more hypotension [21] (see Table 11.1) and has therefore been replaced with bupivacaine.

Local Anesthetic Additives (Adjuvants)

There are two main reasons for using additives with local anesthetics: to improve the quality and duration of the spinal block and to decrease the dose of local anesthetic injected, thereby reducing the cardiovascular effects and improving the clinical profile of the spinal block [1]. Alpha-adrenergic agents and opioids are substances that are associated most frequently with local anesthetics.

Alpha-Adrenergic Agents

These act by reducing the elimination of local anesthetics due to the vasoconstriction they produce, thus reducing their absorption into the systemic circulation and increasing the duration of action thereby contributing to postoperative analgesia.

Epinephrine

Its vasoconstrictor action is due to the direct alpha-adrenergic effect [22]. Clinically, the effectiveness of intrathecal epinephrine depends on the local anesthetic with which it is associated [22–24]. The recommended dose is 0.2–0.3 mg epinephrine. Large doses of epinephrine may decrease blood flow to the spinal cord, whereas doses of 0.2 mg appear to not affect the blood supply to the spinal cord [21, 23, 24]. However, they can contribute to the development of transient neurological symptoms (TNS) [21]. On the other hand, the intrathecal epinephrine significantly delays the return of the sacral autonomic function and the capacity for spontaneous urination [25].

Phenylephrine

The mechanism of action and effects are similar to those of epinephrine. It is used clinically in doses of 5 mg. Its use has declined since it seems that TNS increases when associated with tetracaine [26].

Clonidine

It is not used in the USA. It prolongs anesthesia and analgesia. Doses are 15–150 mcg. It is associated with more episodes of intraoperative hypotension [27].

Magnesium

It is an antagonist of the NMDA receptors and may modify nociceptive modulation. It appears that additional study would be required [28].

Opioids

These interact synergistically because they block afferent stimuli in action sites different from

those of local anesthetics. They improve intraoperative and postoperative analgesia. However, they also produce different side effects such as itching, nausea, vomiting, and respiratory depression, all in a dose-dependent manner.

Morphine

It has a slow onset of action (30–60 min) and a prolonged duration of action, providing extensive postoperative analgesia. Doses of 0.1–0.2 mg provided an extensive spinal analgesia for up to 12–36 h for different surgical procedures such as cesarean delivery, radical prostatectomy, hysterectomy, and total hip arthroplasty. With these low doses, the risk of respiratory depression is quite rare. However, the minimum dose needed in total knee arthroplasty is 0.3–0.5 mg. With these doses, the side effects such as nausea, vomiting, urinary retention, and pruritus increased significantly compared with lower doses. The risk of delayed respiratory depression is dose dependent (6–18 h after intraoperative injection).

Fentanyl and Sufentanyl

Fentanyl is the most commonly used intrathecal opioid. Its lipophilic profile allows a quick onset of action (5–10 min) and intermediate duration of action (60–120 min). Doses of fentanyl of 15–25 mcg, in association with lidocaine or bupivacaine, prolong the duration of anesthesia without increasing complete sensory, motor, and bladder recovery time. Fentanyl and sufentanyl provide minimal postoperative analgesia and are not associated with delayed respiratory depression [29].

Determinants of Intrathecal Local Anesthetic Distribution

Baricity

Baricity is a ratio that compares the density of one solution in another. In the case of spinal anesthesia, it is defined as the ratio between the density of the local anesthetic solution compared with the density of the patient's CSF at 37 °C. Local anesthetic solutions that have the same density as the CSF are called isobaric. Local anesthetic solutions with a higher density than the CSF fluid are called hyperbaric, while solutions with a lower density than the CSF fluid are called hypobaric. Hyperbaric solutions will flow in the direction of gravity and settle in the most dependent areas of the intrathecal space, while hypobaric mixtures will rise in relation to gravitational pull. The effects of gravity are determined by the choice of the patient's position and in the supine position on the curvatures of the spine. There is wide variability in the density of CSF among the various population subgroups. Therefore, a local anesthetic solution can be isobaric in one individual while hypobaric in another.

Hyperbaric Spinal Anesthesia

Hyperbaric solutions are prepared by mixing the local anesthetic solution with glucose. With the patient in supine position, hyperbaric solutions tend to be distributed by gravity to the most sloped points of the thoracic (T6–T7) and sacral (S2) curves (Fig. 11.1). We use hyperbaric solution when we desire sensory level higher than T10. Hyperbaric solutions have faster onset, shorter duration, and a greater extent of sensory block. If an anesthetic hyperbaric solution is injected into a patient in a seated position, its distribution will be restricted during 5–10 min to the lumbosacral dermatomes, thereby producing a "saddle block." Similarly, if hyperbaric solutions are injected in the lateral supine position with the surgical side sloped down, and remains in this position for 10–15 min, it is possible to get a unilateral spinal anesthesia [30].

Hypobaric Spinal Anesthesia

Commercial solutions are prepared by diluting isobaric solutions with sterile distilled water. Although it is less commonly used for perineal and perirectal surgical procedures performed with the patient in the prone position or "jackknife" position, it provides significant advantages in hip surgery in lateral position.

Isobaric Spinal Anesthesia

These solutions are not completely isobaric but tend to be slightly hypobaric. Consequently, depending on the patient's position during the

injection and during surgery, the solution can behave unpredictably rather than as a true isobaric solution. Isobaric solutions are not distributed far from the initial point of injection and are particularly useful when sensory block in the high thoracic dermatomes is not desirable.

Dose, Volume, and Concentration

The importance of concentration, dose, and volume of different local anesthetic solutions on the extent of spinal block has been considered. A change in one of the variables causes changes in the others. However, it appears that the dose (as weight in mg) is the most important of the three. The volume and concentration are not as important. The greater the dose of local anesthetic injected, the greater the extent and the duration of the blockade [27, 31].

Intervertebral Space for the Injection

Different researchers have studied the importance of injection site in the extension of the spinal anesthesia. It seems that the effect of the injection site is superseded by the baricity of the anesthetic solution and the position during the injection.

Position of the Side Holes of the Spinal Needle

This is of importance when using pencil-point needles with a side opening (Whitacre and Sprotte). If the hole is directed either caudal or cranial, it appears that it may affect the extent and duration of the spinal anesthesia differently [32].

Age

With increasing age, there appears to be a tendency to increase the extent of spinal anesthesia. This seems to be related to decreased CSF and to the demyelination that occurs in elderly patients [22, 33, 34].

Height

There is no significant correlation between height and extension of the anesthesia [22].

Body Mass Index

It seems that there is a tendency toward an increase in the extension of the blockade in obese patients, but a significant statistical correlation has not yet been observed. The most likely mechanism is the compression in the subarachnoid space due to an increase in abdominal mass that produces a decrease in the CSF causing the extent of the spinal anesthesia to be higher. This also occurs in pregnant patients [35, 36].

Indications and Contraindications

Indications

The anesthesiologist must determine the correct segmental level for surgery to be performed and assess that the physiological effects of the required anesthetic level are not harmful to the patient. Visceral sensitivity and viscerosomatic reflexes have spinal segmental levels that are much higher than what could be predicted from skin dermatomes. Table 11.2 shows the levels needed for common surgical interventions.

Contraindications

The most important contraindications for spinal anesthesia are patient refusal and increased intracranial pressure. Other contraindications may be infection of the puncture site, severe hypovolemia, or coagulation disorder. For patients with preexisting neurological diseases (peripheral neuropathies, demyelinating diseases), it is controversial because there is no clinical study showing that spinal anesthesia worsens these diseases. Rather, it appears that the contraindication is largely based on legal considerations.

Table 11.2 Dermatomal levels of spinal anesthesia for common surgical procedures

Procedure	Dermatomal
Hysterectomy, cesarean delivery, inguinal herniorrhaphy, appendectomy	T4
TURP(transurethral resection of the prostate), cistoscopy, hysteroscopy, total hip replacement, femoral popliteal bypass, varicose vein stripping	T10
Lower extremity surgery with tourniquet use. Knee replacement and arthroscopy. Below knee amputation	T8–T10
Foot and ankle surgery	L1
Perirectal and perineal	S2–S5

Complications

Cardiovascular Side Effects

The most frequent and severe side effects are hypotension and bradycardia. There is an incidence of hypotension around 33% in non-obstetric populations [3]. Risk factors for hypotension in the non-obstetric population include block height above T5, age over 40, systolic blood pressure less than 120 mmHg, and chronic hypertension. The severity of the decline in blood pressure correlates with the height of the blockade and with the patient's intravascular volume [37]. Prophylactic measures to prevent hypotension include prehydration with crystalloids or colloids or administration of vasoactive drugs [1, 3]. The crystalloid solutions are quickly distributed from intravascular to extravascular space. It has been observed that the administration of Ringer's lactate during the induction of spinal anesthesia is more effective than when it is administered 20 min before [38]. Administration of large volumes of crystalloids (more than 1 L) does not seem to offer great additional benefits over small volumes (250 mL) and may be harmful in patients with limited cardiopulmonary reserve [3]. Prophylactic treatment with vasoactive agents may be more effective than prehydration for the prevention of hypotension [39]. Ephedrine, in intravenous increments of 5–10 mg, produces increased cardiac output in addition to vasoconstriction. Phenylephrine (bolus of 40–160 mcg IV or infusion of 20–200 mcg/min IV) increased peripheral vascular resistance but can decrease the frequency and cardiac output. It would be first choice in obstetrics, especially if tachycardia is present [40].

The incidence of bradycardia is about 13% in non-obstetric populations. The heart rate decreases with a high block height by blockade of cardioaccelerator sympathetic fibers and a decrease in venous return, leading to a predominant vagal tone. Risk factors favoring bradycardia include block height above T5, decreased age (American Society of Anesthesiologists physical status) ASA I, average heart rate < 60 BPM, prolonged PR interval, and treatment with beta blockers. Cardiovascular collapse associated with spinal anesthesia is not uncommon and often is preceded by bradycardia and hypotension. This rare but severe complication seems mainly related to decreased venous return to the heart which activates vagal tone. The incidence of cardiac arrest is difficult to determine and depends on the interpretation of data and definitions. Large observational studies indicate an incidence of (0.04–10)/10,000 [40]. The excessive sedation and a delay in treatment with vasoactive drugs may exacerbate the effects of hypotension and bradycardia. Therefore, treatment should be immediate and aggressive. In addition to treatment with crystalloids and/or colloids, treatment should be continued in steps with atropine (0.5–1 mg), ephedrine (25–30 mg), and epinephrine (0.2–0.3 mg) [41]. If severe bradycardia is present, apply ACLS protocols.

Total Spinal Anesthesia

Total spinal anesthesia occurs when the local anesthetic spreads so high that there is a sensory block of all the spinal cord beyond the cervical region. Subsequent to a complete sympathetic block, severe hypotension and bradycardia occur, followed by respiratory arrest and loss of consciousness. Fortunately, when the local anesthetic spreads so cephalad, the total amount is low, and the motor paralysis is limited and of short duration. Recognition and prompt treatment are essential to prevent cardiac arrest and hypoxic brain injury. Supportive treatment for the duration of the blockade includes vasopressors, atropine, and fluids, in addition to oxygen and controlled ventilation. Morbidity and mortality should not occur if ventilation and circulation are maintained until the blockade is resolved [42].

Subdural Anesthesia

The subdural space is a potential space between the dura and arachnoid, which contains only small amounts of serous fluid that allows the two membranes to move over each other. On rare

occasions, during the course of spinal or epidural anesthesia, local anesthetics may be injected into this space. If the amount of local anesthetic is small (spinal anesthesia), the result is an extensive but minimal anesthesia and may explain many cases of failed spinal anesthesia. If the dose was injected for epidural anesthesia, a wide spreading of the local anesthetic into the subdural compartment can occur with an unexpected spread of the sensory and motor blockade with symptoms resembling total spinal anesthesia.

Spinal Hematoma

The formation of a hematoma within the spinal canal can produce spinal cord compression and ischemic damage. The hematoma can occur in patients with normal coagulation because of damage from the needle or catheter in the epidural venous plexus, but the risk is increased in patients with impaired hemostasis. It is estimated that the incidence of hematoma is less than 1/150,000 in the case of epidural puncture and 1/220,000 in the case of subarachnoid puncture. Suspect a potential hematoma problem when a spinal block is unusually long. Early detection is critical because a delay of more than 8 h in decompressing the spinal cord worsens the prognosis [43, 44].

Infectious Complications

These may occur as localized infection of the skin, spinal abscess, or meningitis. Spinal abscess manifests itself as back pain accompanied by radicular pain, motor deficits, and fever. The diagnosis is made with an MRI. Treatment includes intravenous antibiotics and drainage/surgical decompression.

Neurological Complications

The incidence of neurological injury in large series of spinal and epidural anesthesia ranges from 0.03 to 0.1% [19, 45]. Blunt trauma from the needle causes paresthesia. Intraneural injection causes a more severe paresthesia and worsens nerve injury. Therefore, when a paresthesia occurs while performing a spinal puncture, the advancement of the needle should be stopped, and the local anesthetic injection should be discontinued by removing the needle and waiting for the disappearance of paresthesia. Laboratory studies suggest that all local anesthetics are potentially neurotoxic, but clinical experience suggests that nerve injury induced by these agents is rare. The syndrome of the cauda equina is associated with the use of microcatheters during continuous spinal anesthesia, as well as with 5% lidocaine [46]. Transient neurological symptoms or transient radicular irritation is a syndrome that manifests itself with back pain that radiates to the thighs and lower extremities after spinal anesthesia. The pain on a scale 1–10 has an average value of 6.2. Most patients report an onset of symptoms from 12 to 24 h after surgery for a period of between 6 h and 4 days. The neurological examination is standard [19]. Included among the risk factors are ambulatory surgery, surgical position (in lithotomy, knee arthroscopy), and obesity. One of the most important risk factors is the use of lidocaine. Although all local anesthetics can produce TNS, lidocaine is the one with a higher incidence. Although this is a transitory situation, its symptoms can be very disabling. Treatment is symptomatic with anti-inflammatory analgesics and opiates [19, 45, 47].

Hearing Loss

Incidence is 0.4–0.5%. This complication has been described with increasing frequency. It has been demonstrated by audiometry [46]. It is believed to be due to loss of CSF after lumbar puncture and lower CSF pressure that is transmitted to the perilymph [48].

Nausea

The most frequent causes are hypotension, the predominance of vagal tone that produces sympathetic blockade leading to gastrointestinal hyperactivity and the use of intrathecal opioids.

Post-dural Puncture Headache

This is a common complication of spinal anesthesia. The mechanism appears to be related to the leak of CSF through the puncture site. The leak causes a decrease in CSF pressure and traction of the intracranial structures when the patient changes position from supine to seated [1]. It is an intense occipital headache radiating to the posterior cervical region and may be accompanied by nausea, vomiting, and photophobia. The main characteristic is the positional nature. Other neurological symptoms, such as diplopia and hearing loss, may indicate a severe case of dural puncture headache. It occurs between 12 and 48 h after puncture and usually lasts no more than 2–7 days. It is more common in the young and females. Maternity cases present the greatest risk, and this relates to the type of needle employed or to the increase in abdominal pressure.

The differential diagnosis with regard to other types of headaches, such as meningitis, subarachnoid hemorrhage, and those associated with eclampsia and preeclampsia, should be made. During recent years, the incidence of post-dural puncture headache has decreased, thanks to the use of smaller needles and the introduction of pencil-point needles [49].

Social factors influence treatment because it is a headache that is relieved by supine position and worsened with movement. Thus, it does not affect a woman who has to care for her newborn child in the same way as it would a recently operated patient who cannot move (e.g., trauma patients). Therefore, the treatment of dural puncture headache is, initially, rest, hydration, and treatment with NSAIDs. The treatment of choice is the epidural blood patch [50]. If the headache is severe and lasts more than 24 h, a treatment should be initiated consisting of a spinal epidural injection of 10–15 mL of the patient's sterilized blood. The effectiveness is between 70 and 98% but may require a second blood patch. After the completion of the blood patch, the patient should be lying down for 1–2 h.

Transnasal sphenopalatine ganglion block has been used in the treatment of headaches and facial pain. Studies based on a small number of cases showing use of this block for the treatment of post-dural puncture headaches have been published [51].

Clinical Pearls

Anatomy of the Spine

- The ligamentum flavum is easily recognized by the increased resistance to the passage of the needle.
- The spinal needles are inserted below L2.
- The arachnoid acts as a major barrier to the flow of drugs from the CSF.
- The subdural space is a virtual space between the dura and the arachnoid.
- The volume of CSF is one of the most important factors affecting the level of sensory block and duration of spinal anesthesia.
- Each spinal nerve innervates a specific area of skin or dermatome and skeletal muscles.

Physiology

- The heart rate and the blood pressure in a high neuraxial block decrease by the reduction in venous return that also increases the vagal tone, and this leads to a marked hypotension, bradycardia, and possible asystole.
- If the blockade reaches to thoracic areas, ventilation might be insufficient.
- Hypothermia is caused by the redistribution of heat as a direct result of vasodilation accompanying sympathetic block.

Techniques

- Appropriate sedation may be used although it is important to communicate with the patient.
- Patient positioning and subsequent repositioning must be carefully considered in real time.
- The technique should be done in the shortest time possible without excessive haste. Refrain from making multiple punctures.

- Ultrasound can be used to identify the lumbar interspace and the depth of the subarachnoid space in obese patients or those with difficult anatomy.
- The site should have prompt access to equipment for resuscitation and intubation.

Pharmacology

- Spinal blockade anesthetics should produce (a) a rapid onset to facilitate the start of surgery, (b) a duration commensurate with the length of the surgical procedure, and (c) a recovery that facilitates the expected recovery time and patient discharge.
- Lidocaine use has decreased due to the incidence of TNS, which varies between 15 and 33% depending on the type of surgery.
- Additives to the anesthetic improve the quality and duration of the spinal block and decrease the dose of the local anesthetic injected, thereby reducing the cardiovascular effects and improving the clinical profile of the spinal block.
- The risk of respiratory depression with intrathecal morphine is dose dependent.
- The three most important factors in determining distribution of local anesthetics are baricity, position of the patient during and just after injection, and dose.
- The anesthesiologist must determine the correct segmental level for surgery and must also assess that the physiological effects are not harmful to the patient.
- The contraindications for spinal anesthesia are patient refusal, increased intracranial pressure, infection of the puncture site, severe hypovolemia, or coagulation disorder.

Complications

- The severity of the decline in blood pressure correlates with the height of the blockade and with the patient's intravascular volume.
- A large volume of crystalloids does not offer great benefits over small volumes and may be harmful in patients with limited cardiopulmonary reserve.
- Cardiovascular collapse associated with spinal anesthesia is not uncommon and often is preceded by bradycardia and hypotension.
- A potential hematoma should be suspected when a spinal block is unusually long.
- When a paresthesia occurs while performing a spinal puncture, the advancement of the needle and the local anesthetic injection should be stopped, and disappearance of paresthesia should be awaited.
- If neurological damage is suspected, immediate diagnosis is essential.
- TNS is a syndrome with back pain that radiates to the thighs and lower extremities after spinal anesthesia. The risk factors are the use of lidocaine and surgical position.
- The main characteristic of post-dural puncture headache (PDPH) is the positional nature. The treatment of choice is the epidural blood patch.

Case Study

Loss of Hearing after Spinal Anesthesia [48, 52, 53]

Male, age 25, with no personal history of interest (weight 80 kg, height 190 cm) is programmed for knee arthroscopy. Spinal anesthesia is performed with 24G spinal pencil tip needle. 12 mg of 0.5% hyperbaric bupivacaine is injected. During the intervention 250 mL of Ringer's lactate solution is infused. The surgery lasts 45 min without incident. The patient is maintained with hemodynamic parameters within normal limits during the intervention.

The next day the patient reports hearing loss that is confirmed by audiometry.

Hearing loss following spinal anesthesia is believed to be due to loss of cephalorrachidian fluid through the puncture pore. The dynamics of the cerebrospinal fluid is fundamental for the functioning of the inner ear. Due to the decrease in the pressure of the cerebrospinal fluid related to the opening of the dura mater after spinal

puncture, large volumes of perilymph enter the cerebrospinal fluid and this is reflected in the decrease in the pressure of the perilymph in the labyrinth. The imbalance between endolymph and perilymph is likely to be the cause of hearing loss following spinal anesthesia. This temporary hearing loss observed after spinal anesthesia seems to be due to different factors. The volume of cerebrospinal fluid leakage is associated with hearing loss and also with post-puncture-dural headache. This is related to the size and type of needle being used since studies show that the incidence of hearing loss is more frequent with larger needles (more frequent for size 22G than for 24G and very rare when using 27G).

Circulating blood volume and administration of intravenous solutions also appear to be related to the incidence of hearing loss. Proper fluid therapy can prevent hearing loss and improves internal ear perfusion.

A higher incidence of hearing loss in young people has been observed and this is due to the fact that there is a greater leakage of cerebrospinal fluid after dural puncture that correlates to a higher incidence of post-dural-puncture headache in these patients. Its treatment is therefore superimposable on that of post-dural-puncture headaches.

The hearing loss usually does not last more than a week.

Review Questions

1. A female, 60 years old, is perineorrhaphy operated on in the lithotomy position under spinal anesthesia. The next day, she complains of severe low back pain radiating down her legs. What is the most common cause of this pain?
   (a) TNS (transient neurological symptoms)
   (b) Epidural spinal hematoma
   (c) Epidural abscess
   (d) Decubitus
2. What is the path you should follow?
   (a) Consult with a neurologist
   (b) MRI
   (c) CT at 24 h
   (d) Expect spontaneous resolution
3. Which of the following is true of post-dural puncture headache?
   (a) Commences within 12–48 h of dural puncture.
   (b) Traction of the intracranial structures appears when the patient changes position from supine to seated.
   (c) The technique of blood patch is between 70 and 98% effective. Some cases require a second patch.
   (d) All of the above.
4. What is the minimum level to be achieved for spinal anesthesia in cesarean delivery?
   (a) T1
   (b) T4
   (c) T10
   (d) T8–T10
5. In relation to spinal anesthesia and "transient neurological symptoms" (TNS):
   (a) One of the most important risk factors is the use of lidocaine.
   (b) Onset of symptoms appears between 6 h and 4 days after surgery.
   (c) Although this is a transitory situation, its symptoms can be very disabling.
   (d) All of the above.
6. In a neonate, the spinal cord terminates at the lower border of:
   (a) T12
   (b) L1
   (c) L2
   (d) L3
7. In the case of a patient sitting upright with his arms by his side, a line drawn between the tips of the scapulae will correspond to the vertebral body of:
   (a) T6
   (b) T7
   (c) T8
   (d) T9
8. Indicate which of the statements is false:
   (a) The subarachnoid space lies between the pia mater and arachnoids.
   (b) The subdural space is a virtual space between the dura and the arachnoids.
   (c) Spinal CSF volume is approximately 25 mL.
   (d) Ventricular CSF volume is approximately 150 mL.

9. Risk factors for hypotension in the non-obstetric population include:
   (a) Block height greater than T5.
   (b) Systolic blood pressure less than 120 mmHg.
   (c) Chronic hypertension.
   (d) All of the above.
10. Which of the following is true of the baricity of anesthetic solutions?
   (a) Local anesthetic solutions that have the same density as the CSF are called hyperbaric.
   (b) Local anesthetic solutions with a higher density than the CSF fluid are called isobaric.
   (c) Hyperbaric solutions are distributed to areas most nondependent on intrathecal space while the hypobaric are distributed to dependent intrathecal areas.
   (d) The effects of gravity are determined by the choice of the patient's position and in the supine position on the curvatures of the spine.
11. The line connecting the upper edges of the iliac crest crosses the vertebral body of:
   (a) Interspaces L2–L3
   (b) L3
   (c) L4
   (d) Interspaces L5–S1

Answers

1. a
2. a
3. d
4. b
5. d
6. d
7. b
8. d
9. d
10. d
11. c

References

1. Casati A, Vinciguerra F. Intrathecal anesthesia. Curr Opin Anaesthesiol. 2002;15:543–51.
2. Reina MA, De Leon Casasola O, Lopez A, et al. The origin of the spinal subdural space: ultrastructure findings. Anesth Analg. 2002;94:991–5.
3. Liu SS, McDonald SB. Current issues in spinal anesthesia. Anesthesiology. 2001;94:888–906.
4. Carpenter RL, Hogan Q, Liu SS, et al. Lumbar cerebrospinal fluid volume is the primary determinant of sensory block extent and duration during spinal anesthesia. Anesthesiology. 1998;89:24–9.
5. Greene NM. Physiology of spinal anaesthesia. 3rd ed. Baltimore: Williams & Wilkins; 1981.
6. Mulroy MF, Larkin KL, Hodgson PH, et al. A comparison of spinal, epidural and general anaesthesia for outpatient knee arthroscopy. Anesth Analg. 2000;91(4):860.
7. Mulroy MF, Salinas FV, Larkin KL, Polissar NL. Ambulatory surgery patients may be discharged before voiding after short-acting spinal and epidural anesthesia. Anesthesiology. 2002;97:315–9.
8. Salinas FV, Sueda LA, Liu SS. Physiology of spinal anaesthesia and practical suggestions for successful spinal anaesthesia. Best Pract Res Clin Anaesthesiol. 2003;17(3):289–303.
9. Kamphius ET, Ionescu TI, Kuipers PWG, et al. Recovery of storage and emptying functions of the urinary bladder after spinal anesthesia with lidocaine and bupivacaine in men. Anesthesiology. 1998;88:310–6.
10. Sessler DI. Mild perioperative hypothermia. N Engl J Med. 1997;336:1730–7.
11. Sessler DI. Perioperative heat balance. Anesthesiology. 2000;92:578–96.
12. Macario A, Dexter F. What are the most important risk factors for a patient's developing intraoperative hypothermia? Anesth Analg. 2002;94:215–20.
13. Frank SM, EI-Rahmany HK, Cattaneo CG, Barnes RA. Predictors of hypothermia during spinal anesthesia. Anesthesiology. 2000;92:1330–4.
14. Ready LB, Cuplin S, Haschke RH, et al. Spinal needle determinants of rate of transdural fluid leak. Anesth Analg. 1989;69:457.
15. Gnaho A, Nguyen V, Villevielle T, et al. Assesing the depth of the subarachnoid space by ultrasound. Rev Bras Anestesiol. 2012;62:520.
16. Chin KJ, Perlas A, Chan V, et al. Ultrasound imaging facilitates spinal anesthesia in adults with difficult surface anatomic landmarks. Anesthesiology. 2011;115:94.
17. Hodgson PS, Liu SS, Batra MS, et al. Procaine compared to lidocaine for incidence of transient neurologic symptoms. Reg Anesth Pain Med. 2000;25:218.

18. Hejtmanek MR, Pollock JE. Chloroprocaine for spinal anesthesia for spinal anesthesia: a retrospective analysis. Acta Anaesthesiol Scand. 2011;55:267.
19. Pollock JE. Neurotoxicity of intrathecal local anaesthetics and transient neurological symptoms. Best Pract Res Clin Anaesthesiol. 2003;17(3):471–83.
20. Liu SS, Ware PD, Allen HW, et al. Dose-response characteristics of spinal bupivacaine in volunteers. Clinical implications for ambulatory anaesthesia. Anesthesiology. 1996;85:729–36.
21. Kozody R, Palahnuik RJ, Cummings MO. Spinal cord blood flow following subarachnoid tetracaine. Can Anaesth Soc J. 1985;32:23–9.
22. Pitkanen M, Rosenberg PH. Local anaesthetics and additives for spinal anaesthesia—characteristics and factors influencing the spread and duration of the block. Best Pract Res Clin Anaesthesiol. 2003;17(3):305–22.
23. Kozody R, Ong B, Palahnuik RJ, et al. Subarachnoid bupivacaine decrease spinal cord blood flow in dogs. Can Anaesth Soc J. 1985;32:216–22.
24. Kozody R, Palahnuik RJ, Biehl DR. Spinal cord blood flow following subarachnoid lidocaine. Can Anaesth Soc J. 1985;32:472–8.
25. Moore JM, Liu SS, Pollock JE, et al. The effect on epinefrine on small-dose hyperbaric bupivacaine spinal anesthesia: clinical implications for ambulatory surgery. Anesth Analg. 1998;86:973–7.
26. Sakura S, Sumi M, Sakaguchi Y, et al. The addition of phenylephrine contributes to the development of transient neurologic symptoms after spinal anesthesia with 0.5% tetracaine. Anesthesiology. 1997;87:771–8.
27. Sheskey MC, Rocco AG, Bizarri-Scgmid M, et al. A doses response study of bupivacaine for spinal anaesthesia. Anesth Analg. 2003;97:589–94.
28. Elia N, Culebras X, Mazza C, et al. Clonidineas an adjuvant to intrathecal local anesthetics for surgery: systematic review of randomized trials. Reg Anesth Pain Med. 2008;33:159.
29. Morrison AP, Hunter JM, Halpern SH, Banerjee A. Effect of intrathecal magnesium in the presence of absence of local anesthetic with and without lipophilic opioids: a systematic review and metaanalysis. Br J Anaesth. 2013;110:702.
30. Liu S, Chiu AA, Carpenter RL, et al. Fentanyl prolongs lidocaine spinal anesthesia without prolonging recovery. Anesth Analg. 1995;80:730.
31. Van Zundert AA, Grouls RJ, Korsten HH, et al. Spinal anesthesia. Volume or concentration: what matters? Reg Anesth. 1996;21:112.
32. Urmey WF, Stanton J, Bassin P, et al. The direction of the Whitacre needle aperture affects the extent and duration of isobaric spinal anaesthesia. Anesth Analg. 1997;84:337–41.
33. Pitkanen M, Haapaniemi L, Tuominem M, Rosenberg PH. Influence of age on spinal anaesthesia with isobaric o, 5% bupivacaine. Br J Anaesth. 1984;56:279–384.
34. Cameron AE, Arnold RW, Ghorisa MW, Jamieson V. Spinal analgesia using bupivacaine 0.5%. Variation in the extent of the block with patient age. Anesthesia. 1981;36:318–22.
35. Pitkanen M. Body mass spread of spinal anesthesia with bupivacaine. Anesth Analg. 1987;66:127–31.
36. McCulloch WJ, Littlewood DG. Influence of obesity on spinal analgesia with isobaric 0, 5% bupivacaine. Br J Anaesth. 1986;58:610–4.
37. Arndt JO, Bomer W, Marquardt KJ. Incidence and time course of cardiovascular effects during spinal anesthesia after prophylactic administrations of fluids and vasoconstrictors. Anesth Analg. 1998;87:347–54.
38. Mojica JL, Melendez HJ, Bautista LE. The timing of intravenous crystalloid administration and incidence of cardiovascular side effects during spinal anaesthesia; the results from a randomized controlled trial. Anesth Analg. 2002;94:432–7.
39. Chan WS, Irwin MG, Tong WN, Lam YH. Prevention of hypotension during spinal anesthesia for caesarean section: ephedrine infusion versus fluid preload. Anesthesia. 1997;52:908–13.
40. Ngan Kee WD, Lee A, Khaw KS, et al. A randomized double-blinded comparison of phenylephrine and ephedrine infusion combinations to maintain blood pressure during spinal anesthesia for cesarean delivery: the effects on fetal acid-base status and hemodynamic control. Anesth Analg. 2008;107:1295.
41. Pollard JB. Cardiac arrest during spinal anaesthesia: common mechanisms and strategies for prevention. Anesth Analg. 2001;92:252–6.
42. Furst SR, Reisner LS. Risk of high spinal anesthesia following failed epidural block for caesarean delivery. J Clin Anesth. 1995;7(1):71–4.
43. Horlocker TT, Wedel DJ, Benzon HT, et al. Regional anesthesia in the anticoagulated patient: defining the risk (the second ASRA consensus conference on neuroaxial anesthesia and anticoagulation). Reg Anesth Pain Med. 2003;28:172–97.
44. Horlocker TT, Wedel DJ, Rowlingson JC, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy. American Society of Regional Anesthesia and Pain Medicine Evidenced-Based Guidelines (Third Edition). Reg Anesth Pain Med. 2010;35(1):64–101.
45. Kane RE. Neurologic deficits following epidural or spinal anesthesia. Anesth Analg. 1981;50:150–61.
46. Rigler ML, Drasner K, Krejcie TC, et al. Cauda equina syndrome after continuous spinal anesthesia. Anesth Analg. 1991;72:275–81.

47. Zaric D, Pace NL. Transient neurologic symptoms (TNS) following spinal anesthesia with lidocaine versus other local anesthetics. Cochrane Database Syst Rev. 2009;2:CD003006.
48. Cosar A, Yetiser S, Sizlan A, et al. Hearing impairment associated with spinal anaesthesia. Acta Otolaryngol. 2004;124:1159–64.
49. Spencer HC. Postdural puncture headache: what matters is technique. Reg Anesth Pain Med. 1998;23:374–9.
50. Harrington BE. Postural puncture headache and the development of the epidural blood patch. Reg Anesth Pain Med. 2004;29:136–63.
51. Cohen S, Sakr A, Katyal S, Chopra D. Sphenopalatine ganglion block for postdural puncture headache. Anesthesia. 2009;64:574.
52. Janecka-Placek A, et al. Cochlear function monitoring after spinal anesthesia. Med Sci Monit. 2015;21:2767–73.
53. Vilhena D, et al. Sudden sensorineural hearing loss after orthopedic surgery under combined spinal and epidural anesthesia. Case Rep Otolaryngol. 2016;2016:4295601.

Neuraxial Blockade: Epidural Anesthesia

12

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Introduction

The first epidural injection was performed in 1901 by Jean-Athanase Sicard and Ferdinand Catheline through the caudal route. The Touhy needle was developed for continuous spinal catheter technique and later adapted for epidural anesthesia by Manual Martinez Curbelo. Its popularity increased due to the potential serious neurological sequelae of spinal injections and the availability of long-acting local anesthetic agents such as bupivacaine

The versatility of epidurals, in their use as a sole anesthetic, supplement for general anesthesia or for analgesia and added benefits in obstetric conditions, makes it a popular regional technique in the USA and UK.

The epidural space can be approached at all levels to provide segmental analgesia and this allows it to have a role in a wide variety of subspecialties including; chronic pain, pediatrics, obstetrics; vascular and even emergency laparotomy patients. Other benefits include attenuating the stress response to surgery, reducing postoperative complications and intraoperative blood loss (and therefore the need for blood transfusion). Reduction in postoperative cardiovascular, respiratory, and metabolic complications, improved wound healing and reduced incidence of venous thrombosis are further advantages [1].

The technique of an epidural can be more challenging than a spinal injection and take longer to perform, the onset of analgesia/anesthesia is longer and the motor blockade is less dense compared to spinal techniques. The incidence of post-dural-puncture headache (PDPH) is significantly higher compared to spinal injections.

Anatomy of the Epidural Space

The epidural space is also known as the extradural or peridural space and extends from the base of the skull to the tip of the sacrum. It encircles the dura from the dural reflections at the foramen magnum cranially down to the sacrococcygeal

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_12

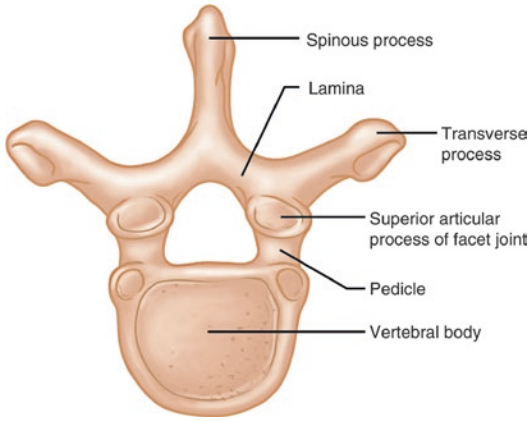

Fig. 12.1 Posterolateral structures

ligament caudally. It is thinnest in the cervical region (2 mm) and thickest in the lumbar region (6 mm).

The vertebral column is made up of 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 3–5 coccygeal vertebrae. The latter two are fused together to form the sacrum and the coccyx, respectively. Though the morphology of the five types of vertebrae differs considerably with regard to size and shape, the basic components for the vertebra remain same. These are the anterior body, lateral pedicles and posterior spinous process. The lamina and the pedicle form the posterolateral structures (Fig. 12.1). The size and shape of the vertebrae vary as we move down along the vertebral column from the cervical to sacral region (see Fig. 12.3), which has implications on the technique of needle insertion into the epidural space. Of notable importance is the variation in angle of the spinous process at the various levels. In the cervical and lumbar regions, the spinous processes are almost horizontal, which permits a midline approach to the space. In the thoracic region, these processes are more acutely angled making a paramedian approach technically easier.

The anatomical borders of the epidural space are superiorly; the foramen magnum, inferiorly; the sacrococcygeal ligament which covers the sacral hiatus and fuses with the coccyx, anteriorly; the vertebral bodies and intervertebral disks and posteriorly the ligamentum flavum.

Ligaments

There are three important ligaments that provide posterior support for the vertebral column and are important when accessing the epidural space.

The *supraspinous* ligament is a continuation of the ligamentum nuchae, which is a thin structure running along the vertebral column joining the tips of adjacent spinous processes. Its thickness increases gradually inferiorly and is maximum in the lumbar region. The *interspinous ligaments* lie beneath the supraspinous ligament and connect adjacent spinous processes. They are thin and inconsequential. The *ligamentum flavum* is a midline structure, which is paired and usually fused in the midline. It is much thicker and offers resistance to a needle passing through it (when the two halves are separate, then it can lead to difficulties in identifying the epidural space by the midline approach with increased risk of dural tap). The *ligamentum flavum* is thinnest in the cervical region and thickens in the thoracic and lumbar region. This is the only ligament that is encountered in the paramedian approach to the epidural space unlike the midline approach where the needle will pass through all three.

Contents of the Epidural Space

The epidural space contains *fat*, *epidural veins*, *spinal nerve roots*, and *connective tissue* (Fig. 12.2). The *epidural fat* lies between the dura and vertebral canal surrounding the spinal cord. It might have a protective role in reducing accidental dural taps during epidural needle insertion. To some extent, this fat can potentially act to modify the effect of drugs injected into the epidural space depending on their lipid solubility. However, the exact role played by this is not very clear. The *epidural venous plexus* is a network of valveless veins known as Batson plexus. They

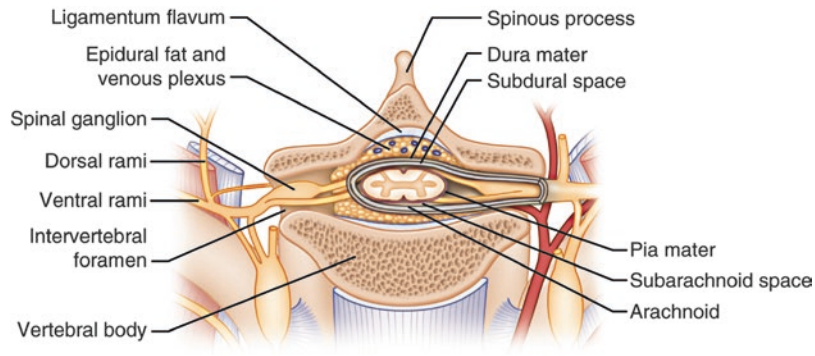

Fig. 12.2 Contents of the epidural space

form a reticular network in the epidural space and transmit pressure fluctuations in the thorax and abdomen as happens during coughing, straining, or during pregnancy. In pregnancy, especially during active labor, these epidural plexuses become highly engorged reducing the volume of the epidural space. The spinal nerve roots lie in the epidural space and, as they exit the spinal cord, carry a short length of the dura, which forms a cuff around these roots. Finally, *the connective tissue* loosely arranged in the epidural space may have some bands and poorly defined septae which can rarely interfere with passing of a catheter or spread of local anesthetic solutions.

Surface Anatomy

Surface landmarks (Table 12.1) and palpation are most commonly used to identify intervertebral level, although both lack accuracy. The vertebra prominens is the most prominent structure noticeable descending down the vertebral column. The other useful surface landmarks are demonstrated in Fig. 12.3. The line joining the superior aspect of the iliac crests is known as Tuffier's line and taken to mark the L4 level.

Table 12.1 Anatomical landmarks for epidural siting

Surface marking	Vertebral level
Vertebra prominence	C7
Root of spine of scapula	T3
Inferior angle of scapula	T7
Rib margin 10 cm from midline	L1
Superior aspect of iliac crest	L4
Posterior superior iliac spine	S2

Special Anatomical Considerations for Caudal Epidural

Although the termination of the spinal cord (conus medullaris) is generally at the level of L2, the cauda equina extends for a variable distance below this (Fig. 12.4) and remains encased within the dural sac, which extends down into the sacral canal. The epidural space can be accessed here, via the sacral hiatus, in the form of caudal epidural anesthesia (Fig. 12.5). The sacral hiatus is the area of S5 (significant individual variability) where the spinous process is absent and can be identified cephalad to the coccyx and in-between the two sacral cornua.

There is significant anatomical variation and in some patients the sacral hiatus lies in close proximity to the anus, which may increase the infection risk of caudal anesthesia.

Physiological Effects of Epidural Blockade

The physiological effects of epidural anesthesia are similar to that of subarachnoid (spinal) anesthesia [2]. The key differences are the onset time

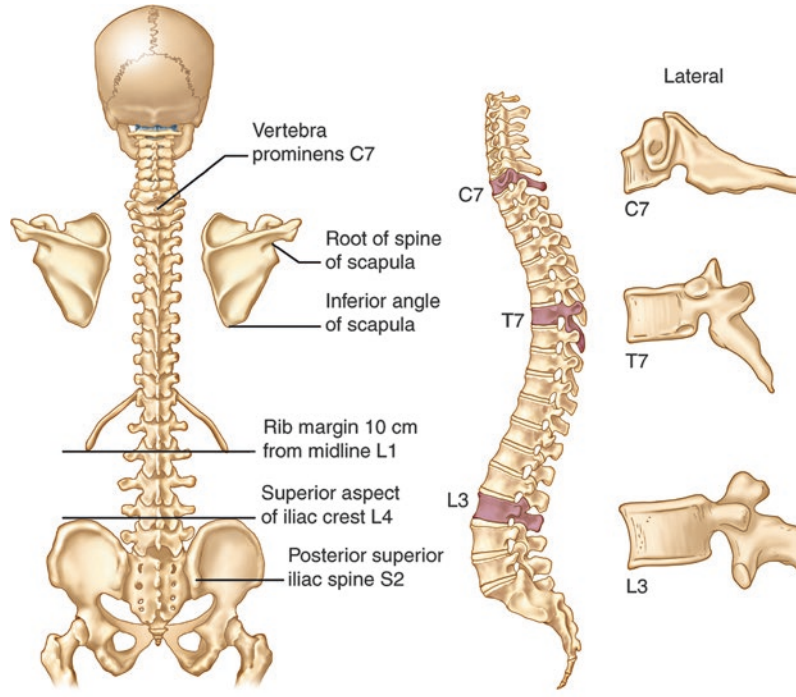

Fig. 12.3 Surface landmarks

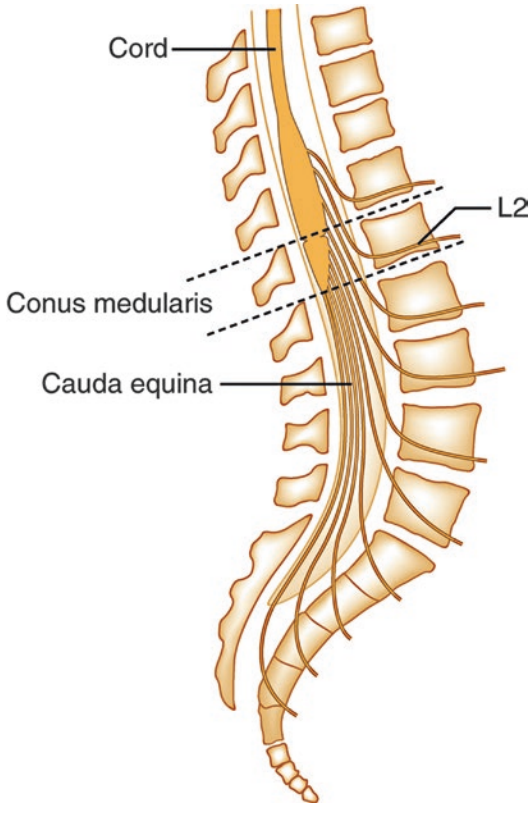

Fig. 12.4 Cauda equine

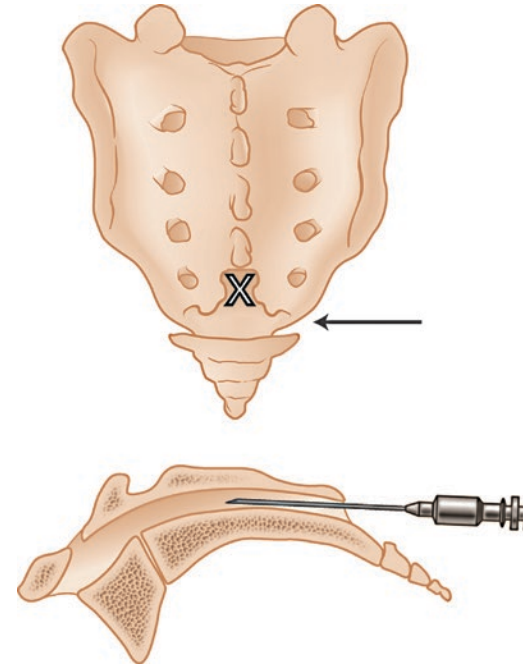

Fig. 12.5 Needle insertion

and the segmental nature of the block produced, due to the restricted epidural spread of the drugs injected. This is an advantage in certain clinical situations (e.g., patients with cardiovascular or respiratory illnesses) where a slower more controlled establishment of blockade is required.

Nervous System

Epidural anesthesia is based on the principle that local anesthetic drugs injected into the epidural space can block spinal nerves at their roots when they leave the spinal cord (Fig. 12.2). Epidural blockade affects both the autonomic and peripheral nervous systems.

Autonomic Nervous System

The sympathetic nerves exit the spinal cord between T1 and L2 and form a sympathetic chain (bilaterally). Blockade of these nerves frequently results in vasodilatation and subsequent hypotension. Higher blocks affecting T1–T5 (cardioaccelerator branches) may result in a reduction in myocardial oxygen demand by reducing inotropy and chronotropy.

Peripheral Nervous System

Epidurals provide a segmental blockade of the peripheral nervous system with caudal and cephalad spread from the point of insertion, the dermatomal distribution of the sensory nerves is shown in Fig. 12.6. This is in contrast to spinal anesthesia, which generally provides complete neural blockade below and, to a variable level, above the level of injection.

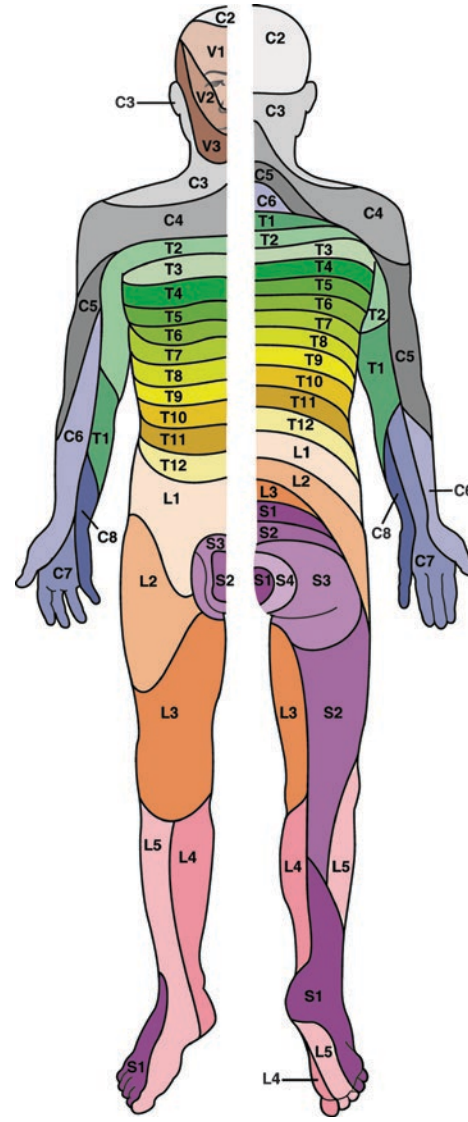

Fig. 12.6 The dermatomal distribution of the sensory nerves

Cardiovascular System

The cardiovascular effects of epidural anesthesia are a result of sympathetic blockade and depend on the level of block and dosage of agents used. Extensive blockade will cause vasodilation, reduced venous return and hypotension as well as reduced adrenal medullary secretions. Compensatory vasoconstriction in the upper body can lead to bradycardia due to the baroreceptor-mediated reflex [3]. In selective lumbar and thoracic epidurals this feature of compensatory vasoconstriction in unblocked segments provides the benefit of hemodynamic stability.

High thoracic blockade affecting T1–T5 will inhibit the cardioaccelerator sympathetic fibers causing bradycardia. This provides a theoretical benefit of reduced oxygen demand, improved coronary perfusion and oxygenation as long as the blood pressure is maintained (spontaneously or pharmacologically).

Respiratory System

There is minimal effect on the respiratory function with epidurals unless the level of block produced is too high. A high thoracic block can affect the function of the accessory muscles of respiration causing distress to patients in the form of difficulty in breathing. Rarely the block can extend above C5 and the phrenic nerve (C3–C5) will be affected causing diaphragmatic paralysis and requiring mechanical ventilation until the block wears off.

The use of epidural analgesia perioperatively improves respiratory function as it provides effective pain relief with minimal motor block, allowing patients to maintain good respiratory function and reduce the risk of respiratory complications such as infection and collapse in the postoperative period.

Gastrointestinal (GI) System

Effects on the GI system are due to blockade of the autonomic nervous system; splanchnic nerve blockade (T5–L1) leads to unopposed parasympathetic activity culminating in increased GI secretions, hypermotility and relaxation of sphincters. These effects can be beneficial in that a small contracted bowel improves access during bowel surgery. An increase in visceral perfusion and early return of postoperative GI motility are preferable following bowel operations. Nausea and vomiting associated with epidural anesthesia are secondary to increased vagal tone and reduction in blood pressure.

Genitourinary System

Epidural anesthesia has no direct effect on renal function; however, a sacral blockade (S2–S4) can lead to urinary retention, requiring catheterization of the bladder.

Effect on Thermoregulation

Shivering is a common feature of epidural injections and the exact mechanism is still not fully known. Suggested mechanisms include vasodilatation and reduction in core body temperature and disruption of the normal thermoregulatory mechanism. The latter is a result of differential nerve blockade allowing selective conduction of cold sensation to the thermoregulatory center or blocking descending inhibitory pathways to the spinal cord.

Indications

These can be broadly divided into three major categories: obstetric anesthesia/analgesia, surgical anesthesia/analgesia and chronic pain interventions.

Obstetric Analgesia

Epidural analgesia has been used for the treatment of labour pains for over 40 years and is considered the gold standard for labor analgesia, despite a number of controversies on its effect on labor.

Epidurals are not only used for analgesia but can also be used to "top up" a block to provide anesthesia for instrumental deliveries, caesarean sections and other operative procedures. They can be sited de novo for elective and emergency procedures where the option to extend and prolong a block will be beneficial especially if extensive postoperative analgesia is anticipated.

The obstetric anesthetists' association (OAA) has produced an information card to be used when obtaining informed consent for labour epidurals outlining common (failure, hypotension, shivering, post-dural-puncture headache (PDPH) and urinary incontinence), rare (nerve damage), and very rare but serious (infection, hematoma, and paralysis) side effects. These should be discussed at the earliest most appropriate opportunity although the practicality of doing this when patients are in severe distress is questionable.

Adjunct to General Anesthesia

Epidural injections or catheters may be sited preoperatively at various levels in the vertebral column to provide both intraoperative and postoperative analgesia.

Cervical Epidurals

Cervical epidural analgesia (CEA) is a useful technique for surgeries of the upper body however is not commonly used other than the treatment of radicular pain in the upper limbs. The epidural space in the cervical region is narrow with a greater depth of space from the skin compared to lumbar and thoracic regions and has a greater potential for complications. CEA blocks the cardioaccelerator fibers, can partially or completely block the phrenic nerve and needs to be considered in patients with cardiorespiratory disease.

A recent systematic review failed to produce specific recommendations on the use of CEA due to limited evidence and given the significant potential for serious harm the technique should only be performed by experienced providers where there is a strong rationale for its use.

Thoracic Epidurals

These are increasingly being utilized for major abdominal, vascular and cardiothoracic surgery. Thoracic epidural analgesia (TEA) not only provides effective analgesia for the targeted dermatomal site and a selective bilateral sympathetic block, with relative sparing of the lower dermatomes. This facilitates early postoperative ambulation, allows deep breathing and improves postoperative recovery.

The sympathetic block is associated with reduced myocardial oxygen demand and subsequently reduces the risk of postoperative myocardial ischemia. In addition, it reduces the incidence of postoperative ileus and has respiratory function benefits also.

The benefits must be balanced with the risks of performing TEA, particularly in the following patient groups:

1. Shocked patients: in this situation, the loss of sympathetic tone may result in unacceptably severe hypotension with fatal consequences.
2. Patients with potential for major blood loss - the derangement in clotting may make the indwelling epidural catheter a potential risk for epidural hematoma.

Lumbar Epidurals

In addition to use on labor ward as mentioned previously, these can be utilized for abdominal and lower limb surgery. The ensuing sympathetic block can be beneficial in improving tissue perfusion to the lower limbs following vascular or plastic surgery.

The use of a combined spinal–epidural (CSE) technique may be desirable in a procedure where rapid onset, complete anesthesia is required but where the procedure may last longer than the duration of the spinal component alone. In this situation the epidural component may be utilized to prolong the anesthesia without the need to convert to general anesthesia. With CSE higher lumbar level approaches should be avoided to reduce accidental damage to the spinal cord by the spinal needle.

Epidurals can be used to identify landmarks in technically challenging patients due to poor anatomical landmarks or difficult positioning. Here identifying the epidural space with an epidural needle first, followed by the spinal needle through the epidural needle, can rescue the spinal block. This technique avoids multiple attempts and use of lower gauge (thicker) spinal needle can potentially reduce the incidence of PDPH.

Management of Chronic Pain

Steroid injections into the epidural space are frequently used in the treatment of radicular and low back pain. Previously radicular pain was thought to be due to nerve compression; however, more recent work suggests it may be secondary to release of inflammatory markers from damaged intervertebral disks; corticosteroid injection into the epidural space is thought to inhibit this inflammatory process [4]. Delivering

the steroids directly to the injured area reduces the systemic effects of steroids and increases the concentration of the drug at the target site. These injections are commonly used to treat nonspecific radiculitis, spinal canal stenosis, and vertebral compression fracture resulting in radicular pain. Its use has also been documented in post-laminectomy syndrome, postherpetic and post-traumatic neuralgia, diabetic neuropathy and myofascial pain.

Spinal Cord Stimulation (SCS) [4]

SCS is supported by randomized controlled trials in the management of failed back surgery syndrome, complex regional pain syndrome, neuropathic pain and ischemic pain. Stimulator electrodes are placed in the epidural space, which can be accessed via a needle or open laminectomy to allow subsequent catheter placement. Spinal cord stimulation has also been used successfully to treat conditions such as urine and fecal incontinence.

Contraindications

Absolute
- Patient refusal
- Local anesthetic allergy
- Infection at the site of injection

Relative
- Shocked patients (hypovolemia, sepsis)
- Aortic stenosis
- Uncooperative patient
- Spinal deformity/previous surgery
- Coagulopathy (sepsis, pharmacological, low platelets, bleeding disorders)
- Risk of major blood loss

There are many clinical scenarios when the risks of the procedure will outweigh the potential benefits and each case needs to be assessed on an individual basis.

Compromised Hemodynamic States

In patients who are shocked (e.g., hypovolemia, trauma, sepsis) and those with a fixed cardiac output (e.g., aortic stenosis and other significant valvular lesions, restrictive cardiac disease) administration of neuraxial blockade can pose serious risks as they are unable to compensate for the fall in systemic vascular resistance (SVR) that ensues once sympathetic blockade has been established following epidural anesthesia. Coronary perfusion will fall and can result in cardiac arrest - resuscitation of such patients is particularly difficult.

Coagulopathy and Bleeding

Patients with uncorrected clotting disorders and major blood loss are at risk of epidural hematoma formation, which is a surgical emergency. The clot must be evacuated without delay to prevent permanent spinal cord damage as a result of increased pressure in the epidural space. The AAGBI has recently published clear guidelines on the timing of performing neuraxial blocks, stopping and restarting anticoagulation and the removal of epidural catheters in patients with coagulation abnormalities.

Infection and Allergy

Local infection or inflammation around the site of desired catheter insertion risks the spread of the infection into the epidural space and in septic patients the risk of spreading infection with an epidural injection is significantly high. History of allergy to the drugs used is another contraindication; however, this can be overcome by using alternate drugs.

Epidural: The Procedures

As with all anesthetic techniques inserting an epidural can be described in terms of pre-procedure, procedure and post-procedure considerations.

Technique of Inserting an Epidural

Pre-procedure

- Pre-assessment
  - History and examination
  - Investigations (coagulation, spinal imaging)
- Optimization
  - Fluid status (correction of abnormalities e.g., dehydration, cardiac failure)
  - Reviewing and stopping anticoagulants
- Consent

Procedure

- Preparation
  - Equipment
  - Drugs: local anesthetics, adjuvants, vasopressors
  - Large bore IV access and connect fluids
  - Aseptic precautions (gown, gloves, hat & mask)
- Prepare the patient:
  - Positioning: sitting, lateral
  - Skin disinfection (0.5% chlorhexidine in alcohol left to dry)
- Monitoring
  - AAGBI standards
  - Skilled assistance
- Sedation/GA
  - This should be established prior to epidural insertion in certain patients
- Anatomical landmarks
  - Identify site of insertion
  - Cover the patient with a sterile fenestrated drape
  - Infiltrate skin with local anesthetic
- Technique:
  - Open the epidural pack onto a sterile trolley, ensuring there is no exposure to disinfectant
  - Flush the epidural catheter, connector and filter with saline and disassemble
  - Insert Tuohy needle through infiltrated area until ligamentum flavum identified
  - Remove inner trocar
  - Use LOR syringe to identify the epidural space
  - Attach the stabilizer and insert the epidural catheter
  - Carefully remove the Tuohy needle keeping catheter at appropriate depth
  - Secure at correct depth
- Testing
  - Give a test dose of local anesthetic
  - Check level of motor and sensory (light touch and cold)

Post-procedure

- Continue monitoring for whole duration of epidural analgesia (AAGBI standards, block density and height)
- Give anesthetic/analgesic by intermittent boluses or continuous infusion

Pre-procedure

The technique must be performed by trained practitioners with skilled assistance only and nurses with specific training and skills in the management of epidural analgesia should be present on wards where epidurals are to be managed.

Pre-assessment

The patient should undergo formal pre-assessment as for a general anesthetic; history, examination, and review of relevant investigations paying particular attention to cardiorespiratory status, anticoagulants and clotting abnormalities and assessment of back and veins. The current recommendations of when anticoagulants should be stopped prior to neuraxial blockade are shown in Table 12.2. Blood results, specifically full blood count and coagulation screen should be reviewed as well as markers of infection. In certain patients, such as those with previous spinal surgery or scoliosis, reviews of spine X-rays or CT scans may be necessary.

Table 12.2 Epidural analgesia and drugs affecting hemostasis: current recommendations

Drug	When to stop
Aspirin	Continue
NSAID	Continue
Heparin	Stop before 4 h
LMWH	Stop before 12 h (prophylactic dose) Stop before 24 h (treatment dose)
Warfarin	Stop before 5 days (INR to be below 1.5)
Clopidogrel	Stop before 1 week (range 5–10 days)
Teicoplidine	Stop before 2 weeks
GIIa/III	Stop before 4 weeks

Optimization

Patients' fluid status should be assessed and preloading or co-loading of crystalloid should take place as well reviewing any anticoagulant medication that needs to stopped, continued, or converted to bridging therapy (in high risk patients) [35].

Consent

Informed consent should be obtained and should include indications, contraindications (relevant to the patient), a description of the procedure, management during the block, risks and benefits and post-procedure management. Patients should be given the opportunity to ask questions, refuse and alternatives should be discussed.

Procedure

In theory epidurals may be inserted at any spinal level but in practice the most common sites are thoracic, lumbar and caudal. The technique is broadly similar for each and here we describe a lumbar epidural and the specific considerations/variations for the other two main types.

Once all of the pre-procedure steps are completed (above) the patient should be moved to an area with appropriate resuscitation and airway monitoring facilities readily available and all equipment and drugs should be checked and prepared. The procedure should be performed using an aseptic technique, skin decontaminated with 0.5% chlorhexidine and left to dry while preparing the equipment taking care that the disinfectant does not come into contact with the equipment or gloves of the practitioner. Large bore IV access (typically 16 gauge venflon) should be sited and connected to fluids prior to starting the procedure. Patients who are likely to be cardiovascularly unstable should have vasopressors and/or inotropes prepared and ready to use.

Equipment

Many centers provide pre-sterilized epidural packs and separate individual kit when the technique needs to modified for a CSE procedure or patient with high BMI. Each individual equipment is described in detail below.

The latest guidelines recommend the use of 0.5% chlorhexidine in 80% alcohol for skin preparation prior to siting an epidural based on evidence of effectiveness and association to neurotoxicity.

Epidural Needles

Although there are a range of available epidural needles (Touhy, Husted, and Crawford), the Touhy needle is the most widely used. The main difference between these needles is the angle of the blunt tip, which varies from 15 to 30° (Fig. 12.7).

A standard Touhy needle consists of an 8 cm metal shaft with markings at 1 cm intervals (to help measure depth of needle tip from the skin) attached to a hub (making the total length 10 cm). A wing or flange is attached to the hub to help in stabilizing the needle during insertion into the epidural space. In some needles the flange is fused with hub and in others it is attached to the needle just prior to the hub.

The Touhy needle has a rounded tip which should be pointed upward and this is called a Huber point. The deflected bevel top makes the cutting surface perpendicular to the needle shaft and is designed to reduce the coring of tissue and septa. The bevel at the tip of the needle reduces the risk of dural puncture and has the effect of directing the epidural catheter cranially during insertion.

The hollow of the needle is occupied by a removable trocar, which does not protrude through the end of the needle and is only removed once the needle is introduced through the skin and immediate soft tissues. The function of the

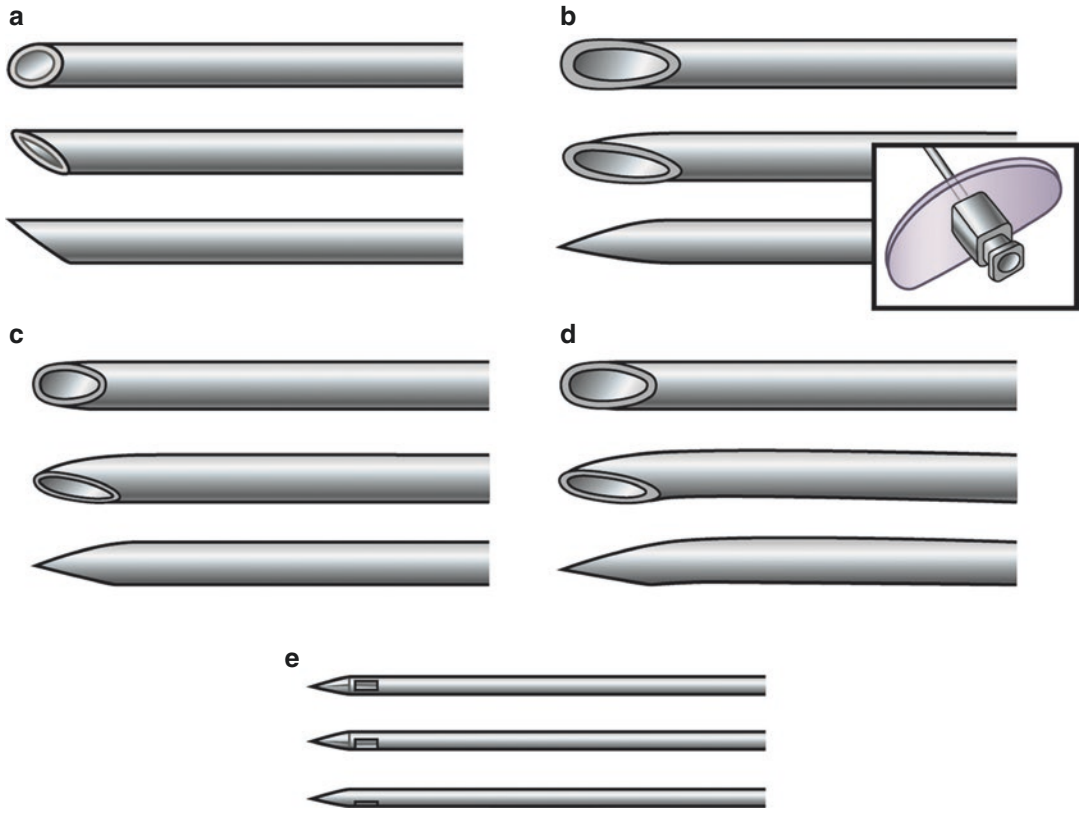

Fig. 12.7 Types of needles and their tips. Inspired by Regional Anesthesia and Analgesia, WB Saunders, Philadelphia

trocar is to prevent a plug of skin or tissue from blocking the needle and many anesthetists will feel for the "grip" of the ligamentum flavum before removing the trocar and connecting the loss of resistance syringe.

Another commonly seen epidural needle is one specifically designed for a needle through needle CSE technique. As well as spinal needle which will protrude through the tip of the Touhy needle, the Touhy needle has a slightly altered tip which will allow the smooth passage of the spinal needle through the end without the slight resistance which may occur if the technique is performed through a standard Touhy needle.

Epidural needles are available in various gauges and lengths; 16, 17, and 18G are the most frequently used and a 10 cm needle is suitable for most adults (a 15 cm needle is available when extra length is required) and separate pediatric sizes are available.

Epidural needles need a greater caliber to allow the easy flow of saline, which is required to detect a loss of resistance. However, it is possible to use smaller caliber spinal needles to access the epidural space in specific situations such as X-ray guided procedures in management of chronic pain conditions including caudal anesthesia in adults.

Epidural Catheters

These are made of either nylon or Teflon, are biologically inert, transparent, and 90 cm long. The distal tip is colored (to help identify it during removal) and may be closed or open; closed tip catheters are claimed to reduce the risk of intravascular injection and have side ports on the distal end. The first 15 cm of the catheter has marking every 5 cm and then there are 1 cm markings from 5 to 15 cm from of the distal end. The proximal open end is connected to the catheter connector, which in turn connects to the filter.

Connectors

Connectors for the epidural catheters come in various designs; some are screw-in and others snap-up. The epidural kits also contain stabilizers that help to facilitate easy passage of catheters through the needle.

Epidural Filters

The pore size of these filters is 0.22 μm in size and helps to remove viruses and bacteria as well as foreign bodies such as glass particles (from ampoules).

Loss of Resistance (LOR) Syringes

Traditionally LOR syringes have been Luer lock syringes, with low plunger resistance allowing easy identification of the sudden loss of resistance as the needle enters the epidural space. However, a recent NPSA alert has meant many centers have or are shifting towards non-Luer devices to reduce the risk of inadvertent intravenous administration of drugs intended for intrathecal/epidural or regional route and vice versa. The syringes are of 10 mL capacity and made of PVC or PP and should be filled with saline prior to attaching the epidural needle.

Aids to Identify Epidural Space

There are a variety of devices used to detect the entry of the tip of the needle into the epidural space, such as pre-filled balloons, spring-loaded syringes, radiological imaging, or ultrasonography but these are not routinely used in clinical practice and are no substitute for technical acquired with clinical practice. In technically challenging cases (e.g., extreme obesity or anatomical abnormality), adjuvants such as X-ray or ultrasound guidance could prove valuable.

Prepare the Patient

The patient should be positioned either sitting or in the left lateral position with the spine fully flexed. Epidurals can also be performed in prone position although this is not a common occurrence. There are advantages and disadvantages to each position and the choice will depend on patient factors, technical factors and operator preference.

In the lateral decubitus position, the patient will be more stable without the need for an assistant to support them and this position allows a greater degree of sedation to be employed. The patient should lie with their back parallel to the edge of the bed/trolley, a pillow should be placed under the head to keep the spine level. The knees should be drawn up to the abdomen with thighs flexed, with the upper arm across the chest and the lower arm projecting 90° from the body. Ideally the patient should adopt a fetal position with the spine maximally flexed to open the spaces between the vertebrae. If necessary, the patient can be asked to increase the flexion by grasping the back of the head/neck and attempting to draw elbows and knees together. In obese patients the lateral position can make identification of the midline more difficult as the tissue midline is distorted by the subcutaneous adipose tissue being displaced by gravity.

In the sitting position, patients should sit up at the edge of the bed, with their feet on a stool or other support. They should start with a straight back, with their chin on their chest, arms hugging a pillow, or on a Mayo table or stand in front of them. It is important they do not lean forward and should arch their back (terminology such as “angry cat” or “slouch” are commonly used). Lateral rotation and flexion of the spine should be avoided and an assistant may help to prevent this and to encourage the patients to keep their shoulders level [5].

The level at which the epidural is inserted should be identified; landmarks that can be used to identify spinous processes and hence vertebral level will be dealt with in the following section (Table 12.3). Evidence has shown that the accuracy of anatomical landmarks is known to be poor and an ultrasound scanner could be helpful in difficult situations.

The bed should be positioned at a height convenient for the operator to work at the insertion level. The midline should be identified and can be marked with an indelible skin marker. The operator should then adopt an aseptic technique, including surgical scrub, face mask, hat, and sterile gloves. There is controversy regarding the use of a face mask [6], however, wearing one has

Table 12.3 Suggested catheter levels for specific surgical procedures

Surgery/procedure	Level of catheter insertion
Procedure in the neck	C6–T1
Mastectomy	T6–T7
Thoracotomy	T4–T6
Upper abdominal	T6–T8
Lower abdominal	T10–T12
Lower limb	L2–L4
Labor/delivery	T10–S4
First stage	T10–L1
Second stage	S2–S4

Adapted from Regional Anaesthesia—The Requisites in Anesthesiology

been shown to reduce the incidence of infection rates during central venous catheterization and the author would continue to advocate their use. Eye protection should also be worn in case of inadvertent aerosolization of local anesthetic during the procedure.

The skin should be prepared with 0.5% chlorhexidine gluconate in alcohol and a sterile fenestrated drape used to isolate the area where the epidural will be inserted. The applied disinfectant should remain in contact with the skin for the recommended duration (e.g., alcohol-based disinfectants should be left to dry on its own).

Monitoring

Monitoring according to AAGBI standards should be implemented prior to and continued after establishment of epidural analgesia. Without guarantee of these basic standards the procedure should not even be attempted.

AAGBI Monitoring Standards

- HR
- Blood pressure
- Respiratory rate
- Sedation score
- Temperature
- Pain score
- Degree of motor and sensory block

Sedation/GA

Sedation is used depending on the situation and when employed the patient should be able to communicate and cooperate with the operator. This optimal sedation can improve patient comfort without losing cooperation. Epidurals are not commonly done under general anesthesia to minimize the risk of complications going undetected (an awake patient can communicate pain or paresthesia on insertion allowing adjustment or resiting).

Anatomical Landmarks

The level of epidural insertion depends on the indication for the epidural as well as patient factors.

The type and site of surgery as well as the purpose of the epidural (Table 12.3) will determine where best to perform the procedure to produce optimum analgesia. The epidural drugs should be injected into the spine at the level corresponding to the dermatome corresponding with the midpoint of surgical incision. When using a catheter, the tip of the catheter should correspond to the midpoint of the surgical incision (this is not very accurate without radiological screening).

Patient factors influence the site of epidural insertion; ease of palpation of spinous processes (obese patients), size of interspinous space (narrow spaces common in elderly patients), presence of localized infection, anatomical and other abnormalities of the spine (e.g., scoliosis, metal rods), previous spinal surgery and level of cooperation. The experience and familiarity of the operator with the technique and availability of adjuvants will also play a role.

Technique

The operator should adopt a good position that is convenient for them; some will work from a standing position, while others work from a sitting position. Building up dexterity and adaptability from early years of training will prove valuable in later years of career.

Preparing the procedure tray in advance is a useful and rewarding habit to learn; the epidural catheter should be connected to the filter and connector and be flushed with saline to ensure

patency of the orifices and then each component should be disassembled. The LOR syringe should be tested to ensure free movement and we advocate the use of saline with the syringe being filled with 5–10 mL of saline.

A skin weal should be raised at the target site with local anesthetic (1% or 2% lignocaine) and local infiltration performed to the supraspinous and interspinous ligaments. This needle can be used as a "seeker" to identify the depth to the ligamentum flavum if the patient's body habitus is favorable and also to determine cephalad angulation required to pass between the spinous processes. It can also be used to identify the bony landmarks.

The epidural needle should be connected to the flange and inserted with the stylet in situ and bevel facing cephalad or caudad, perpendicular to the skin in vertical and horizontal planes. Common techniques used for holding and advancing the needle observed by the authors include holding the flanges between thumb and index finger of both hands and bracing the remaining fingers against the back to prevent too rapid advancement, and holding the needle with the thenar eminence at the hub, index and middle finger supporting the needle with thumb held parallel to the axis of the needle. The needle is then slowly advanced until increased resistance is met, representing the ligamentum flavum. The stylet should then be removed and the loss of resistance (LOR) syringe attached and the needle re-angled slightly cephalad.

For loss of resistance to saline (LORS), the non-dominant hand is used to brace against the back (see Fig. 12.8), to stabilize the needle and prevent sudden rapid forward motion. The syringe is held in the dominant hand, and constant pressure is applied to the plunger of the syringe with the thumb as the needle is advanced. While the bevel of the needle is within the ligaments, there will be considerable resistance to pressure, but as the bevel exits the ligamentum flavum and enters the epidural space, there will be a sudden loss of

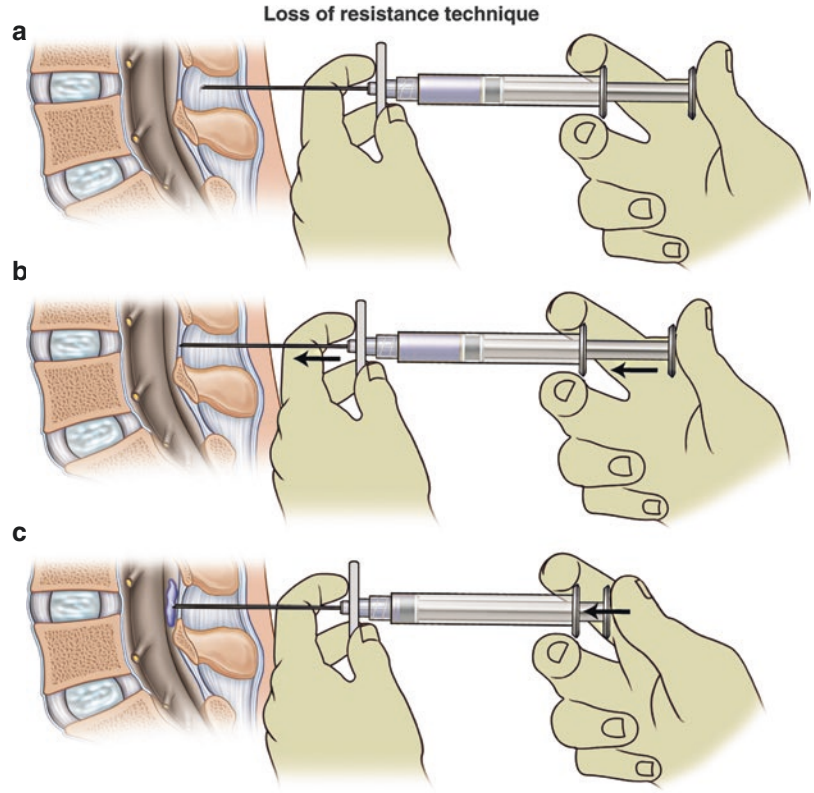

Fig. 12.8 Loss of resistance technique. Inspired by Visser L. Epidural Anesthesia

that resistance, the contents of the syringe will be discharged into the space, and the needle will cease in its forward motion. It is important to warn the patients that they will feel pressure in the back, but they should not feel pain. If they feel pain or discomfort, the operator should ask the patients whether they feel it to left or right, or in the midline, as it may be necessary to reevaluate the direction of insertion of the needle.

Alternatively a technique of intermittent advancement may be used, where the needle is advanced 1–2 mm at a time using the same hold as described for the insertion of the needle, and the plunger of the needle is depressed intermittently to assess for loss of resistance. A small air bubble purposefully introduced into the LOR syringe can help to monitor the pressure inside the syringe. As long as the needle tip is inside the ligamentum flavum, the bubble can be observed to be compressible within the syringe by exerting pressure over the plunger.

Once LOR has occurred, the needle should be advanced 1 mm further to ensure the opening is fully within the epidural space and the syringe removed from the hub, ensuring the needle does not move. A small amount of fluid may now drain from the needle, but should stop after only a few drops. If not, then this may be CSF and dural puncture may have occurred. If fluid runs freely, then definite dural puncture has occurred, and there are several options available to the operator, which will be discussed below. If blood appears in the needle, then it should be withdrawn and either redirected or re-sited depending on assessment of the landmarks. In the absence of blood or CSF, the depth to which the needle has been inserted should be noted. The patient should be told to remain absolutely still to prevent displacement. If a single shot technique is to be used, injection should take place now.

If a catheter is to be inserted, the stabilizer should be placed in the needle hub and the catheter advanced slowly into the epidural space so that 15–18 cm of catheter is within the needle and space, while the needle is stabilized. The patients should be warned that they may feel paresthesia, burning, tingling, or electric shock, but that this should only be transient.

Catheter advancement may be made easier if a 5–10 mL of saline is injected into the space once LOR has occurred, which may separate the tissues slightly to allow passage of the catheter. If the sensation persists after halting advancement, then it may be necessary to remove the catheter and re-site the needle. Withdrawing the catheter through the needle could lead to shearing of the catheter on the bevel and this is not recommended and should be avoided.

The needle is then withdrawn over the catheter, ensuring non-displacement of the catheter. It is recommended that 4–5 cm of catheter be left in the epidural space [7] and the catheter should be withdrawn now to a depth of 4–5 cm plus the depth of the space. For example, if the skin is at the 6 cm mark on the needle, the catheter should be withdrawn to the 10 or 11 cm. The filter and connector should be attached at the proximal end of the catheter using a sterile technique and then aspirated to check for CSF or blood. If blood is freely aspirated, then placement within a blood vessel must be assumed and the catheter should be removed and a new attempt at insertion made.

If CSF is aspirated at any point during the procedure (either via the needle or the catheter), then the option of placing/leaving an intrathecal catheter may be considered. This may then be used to provide spinal anesthesia, with the relevant cautions applied but this technique is beyond the scope of this chapter. Alternatively the catheter can be withdrawn and re-sited. The patient should be warned about the possibility of PDPH and a note of dural puncture should be made in the medical/anesthetic records.

Once the catheter is at the required depth, it should be secured either with a proprietary securing device or a clear transparent dressing. If the latter is used, one or two loose loops of the catheter should be made on the skin so that tension on the catheter will unravel the loops rather than displacing it. A further alternative includes tunnelling the catheter under the skin for a short distance before making the loops. The catheter is then brought over the patient's shoulder and secured in situ with a cloth tape such as Mepore® or Hypafix®.

Testing

A test dose of 3 mL of local anesthetic (with or without 1:200,000 epinephrine equivalent to 15 μm) may now be given and the patient asked to return to the supine position. Monitoring for signs of intravascular injection or intrathecal injection should be done (20% increase in heart rate, fall in blood pressure, spinal anesthesia). It should be borne in mind that sedation may reduce the reliability of the lidocaine only test in an awake patient [8]. If after 5–10 min no signs of either have been detected, then incremental injection of the desired analgesic/anesthetic drugs may occur. Continued observation for signs of systemic toxicity and catheter displacement into the dural space should be continued. Epidural block can take up to 20 min to become fully established.

Block height and density should be assessed at regular intervals. This can be done by using the Bromage scale and by assessing for loss of sensation to cold, touch, and pinprick. The dermatome level at which sensation is lost should be recorded (Table 12.4) [9, 10].

Special Techniques

Loss of resistance to air is another technique for identifying the epidural space. As above, the LOR syringe with 5–10 mL of air inside is attached to the needle hub when the needle is in the interspinous ligament or ligamentum flavum. The wings of the needle are gripped between the thumb and forefinger of both hands with the dorsa of the hand resting against the patient's back and the needle advanced 2 mm at a time. The plunger is gently pressed, and if there is resistance (colloquially termed "bounce"), the needle is very carefully advanced another 2 mm. As the needle enters the epidural space, a sudden "give way" or "click" may be felt. At this point air can be freely injected into the epidural space. The syringe is removed and the catheter threaded as above. Provided great care is taken in advancing the needle it should not pierce the dura. As this technique requires intermittent removal of one hand and testing for LOR, it is relatively slower but probably safer as there is less chance for the needle to overshoot and produce an accidental dural puncture.

A further possible technique is the "hanging drop," where a drop of saline is placed at the end of the needle once the stylet has been withdrawn. As the needle is advanced into the epidural space, the negative pressure that exists within the space withdraws the drop into the needle (due to the denting of the dural by the needle). This technique used to be popular for thoracic epidural injections.

Table 12.4 The Bromage scale (1965)

Degree of block	Bromage criteria	Score (%)
No block	Full flexion of knees and feet	0
Partial block	Just able to flex knees and full flexion of feet	33
Almost complete	Unable to flex knees, some foot flexion	66
Complete	Unable to move legs or feet	100

Thoracic Epidural

The thoracic vertebral spinous processes are much more steeply angled and project further. The dura is more closely aligned to the ligamentum flavum and the spinal cord may lie closer to the dura. The positioning of the patient and the technique of advancement remain substantially the same as for lumbar epidural, but with the proviso that the epidural needle should only be advanced 1 mm at most after LOR.

The paramedian approach is an alternative and may be preferable in the thoracic level and when ligaments are calcified. The needle is inserted 1–2 cm lateral to the spinous process of the more cephalad vertebra. The needle is then advanced perpendicular to the skin until contact is made with the lamina or the pedicle of the vertebra. The needle should then be redirected cephalad 15–30° and medially 15–30° and the needle "walked off" the bone. A loss of resistance technique is then used to detect the epidural space.

Caudal Approach

Here the patient is positioned either in the lateral decubitus or prone position. Sacral hiatus is then

identified by palpation. The surface marking of sacral hiatus is that it lies at the apex of an equilateral triangle base of which is formed by the line joining the two posterior superior iliac spines. The hiatus lies in its apex with the sacral cornua on either side. A 22 (or 23)-G needle is then inserted at 45° angle with its bevel facing the operator into the ligament and once the ligament is perforated (felt as a "pop") the needle is advanced at a more acute angle further (2 cm in adults and about 1 cm in children) into the caudal canal (Fig. 12.5). Care should be taken not to advance the needle too much as this will increase the risk of dural puncture. A cannula over the needle technique can also be used where the cannula is left in epidural space after withdrawing the needle. The anesthetic solution is injected after careful aspiration for CSF or blood.

Post-procedure

Epidurals should only be managed in environments with adequately trained staff with access to emergency equipment and drugs required to manage known complications. Epidural solutions can continue to be administered by intermittent bolus injections or continuous infusions.

The patient should continue to be monitored after the anesthetic has been given for signs of local anesthetic toxicity. These include lightheadedness, tinnitus, circumoral and tongue numbness, paresthesiae, visual disturbances, muscular twitching, convulsions, unconsciousness, coma, respiratory arrest and cardiovascular collapse.

Troubleshooting

Difficult Anatomy

In pregnant or obese patients, it may be difficult to identify the midline, especially in the lateral decubitus position and where possible these patients should be asked to adopt the sitting position. It may be helpful, though not necessarily reliable, to ask the patients whether they feel that the operator's hand/needle is in the midline. Alternatively, it may be possible to use ultrasound to identify the midline and interspinous space.

Repeated Contact with Bone

Position is the most common reason for repeated bone contact; the patient should be asked to flex more, or position should be changed from lateral decubitus to sitting or vice versa. Other techniques are to reinsert the needle slightly away from the midline, withdrawing the needle to the subcutaneous tissue level and repositioning the needle at a steeper angle or inserting the needle closer to lower border of the upper spine. In the lateral decubitus position, there is a tendency for the soft tissues to sag under gravity leading to the midline of the back to move away from the spinous processes and this can mislead the operator (happening more frequently in obese individuals).

Difficulty Threading the Catheter

The stabilizer that comes in most commercial packs should be used to aid in inserting the catheter, as it will prevent kinking in the relatively large hub of the needle. Slight rotation of the needle about the longitudinal axis may facilitate insertion. Sometimes, even after obtaining LOR to saline or air, the tip of the needle will be only halfway through the ligamentum flavum making it difficult for the catheter tip to pass into the epidural space; this could be solved by advancing the needle slightly further in.

Fluid or Blood Returns Via the Needle or Catheter

Fluid in the needle or catheter can either be saline, which should stop after a few seconds, or CSF where flow doesn't stop. If flow does stop, then incremental doses of anesthetic should be given while observing the patient for signs of intrathecal block; this applies whether or not a catheter is used. Testing the fluid for glucose using an indicator strip of glucometer can distinguish between CSF and the saline.

Blood in the catheter indicates likely intravascular placement; if blood flow stops on withdrawing the catheter and blood can no longer be aspirated, then the catheter may be used

cautiously. This is under the proviso that all doses should be preceded by aspiration to check for blood and should be given incrementally while monitoring the patient very closely for signs of toxicity. The catheter should be flushed with saline before a test dose is given.

Pain on Insertion

A brief sensation of electrical shock or paresthesia on insertion of a catheter is common but if it persists the catheter is likely up against a nerve root and should be withdrawn a few millimeters until the sensation stops. If sufficient catheter remains within the space, then it may still be used otherwise it should be re-sited.

Unilateral Block

The precise cause for this can be difficult to determine; it could be that the tip of the catheter has moved out of the epidural space through the intervertebral foramen (more common when more than 4 cm of catheter is left in the epidural space) or that there are connective tissue septa which theoretically prevent uniform distribution of the local anesthetic solution. This problem is managed by pulling out the catheter so that about 3–4 cm of it remains inside the epidural space and then giving another top-up or by turning the patient on the unblocked side before the top-up and keeping in this position for about 15 min - if this fails then the epidural will have to be re-sited.

Pharmacology of Epidural Blockade

Site of Action of Epidurally Administered Drugs

The exact mechanism of how epidurally administered drugs exert their effects is not fully understood. Hogan [11, 12] demonstrated that the spread of solutions injected into the epidural space results to form a coat around the cylindrical dural sac while some of it passes through the foramina.

There are four potential possibilities for these drugs to exert their observed effects: (1) once injected the drug passes along the intervertebral foramina into the paravertebral space and acts directly on the nerve roots and plexuses, (2) the drug diffuses through the dura into the subarachnoid space, (3) the drug penetrates the dural cuffs of the spinal nerves and interferes with nerve conduction, and (4) the other possible pathway is by axonal transmission. The large network of epidural veins, known as Batson's plexus, also contributes to the systemic absorption of the drugs administered.

Drugs and Doses

At one time or another almost all local anesthetic agents were used for providing epidural anesthesia or analgesia, either alone or in combination with a variety of other drugs ranging from epinephrine to ketamine to opioids. All preparations of any drug used for neuraxial blockade should be preservative free to minimize the risk of neurotoxicity.

Local Anesthetics (LA)

The choice of LA depends on the indication for which the epidural has been sited and the pharmacokinetic effects of each agent. For example, prilocaine and lignocaine will be more beneficial for short procedures whereas bupivacaine and ropivacaine would be preferable for longer procedures and analgesia during labor (Table 12.5).

The duration of LA action in epidural analgesia can be described in terms of "two-dermatome regression" or "complete resolution." The former is the time taken for the block to recede by two dermatomes from its maximum extent, while the latter is the time taken for the sensory block to wear off completely. This is influenced by dural surface area, volume of fat in the epidural space, and velocity of blood flow in the epidural space [13]. The recommended dose and duration of action of commonly used local anesthetics [14]:

Table 12.5 Recommended dose and duration of action of commonly used local anesthetics

Drug	Presentation	Onset of action (min)	Usual dose	Duration of action
Chloroprocaine	As a solution in 2% and 3% concentration	6–12	15–25 mL	40–50 min
Lidocaine	As solution of lidocaine (usually hydrochloride) with or without epinephrine, in concentrations of 1, 1.5 and 2%	10–20	10–20 mL (1%)	Without epinephrine from 1 to 2 h
			10–15 mL (1.5%)	
			10–20 mL (2%)	With epinephrine may be considerably longer
			Depending on site and desired level of block	
Mepivacaine	As a solution in 1, 1.5 or 2% concentration	3–20	15–30 mL (1%)	2–2.5 h
			10–25 mL (1.5%)	
			10–20 mL (2%)	
Ropivacaine	As a solution in 0.2, 0.5, and 1% concentration	5–13	10–20 mL (0.2%) at 30–60 min intervals or infused at 4–14 mL/h	3–5 h
			15–30 mL (0.5%) for surgery	
			15–20 mL (0.1%)	
Bupivacaine (and Levobupivacaine, which is equipotent but with fewer cardiotoxic effects)	As a solution in 0.25, 0.5 or 0.75% in 5–10 mL vials or ampoules	5–20	10–20 mL of either concentration at 1–2 h intervals, or infusions of 5–15 mL/h of 0.1% solutions	Up to 2–2.5 h
	As a solution of 0.1% in infusion bags or syringes of 50–500 mL, often with fentanyl 2 μg/mL or 4 μg/mL for epidural infusion			

Chloroprocaine

- Rapid onset (6–12 min)
- Duration of approximately 40–50 min
- Can be used as an infusion
- Available as 2% and 3% concentrations
- Epidural dose 15–25 mL [15]
- Higher doses associated with backache
- Reduced efficacy of adjuvants such as morphine and clonidine

Lidocaine

- Most widely used LA
- Rapid onset (10–20 min)
- Available in range of concentrations including 1, 1.5, and 2%. It has a
- Dose is 10–20 mL of 1%, 10–15 mL of 1.5% or 10–20 mL of 2% depending on site and desired block level
- Duration of action from 1 to 2 h (without epinephrine) [16]
- Tachyphylaxis limits long-term use
- Popular topping-up agent for cesarean sections (alone or with epinephrine and sodium bicarbonate; the latter ensures rapid onset by altering the pH so that more of the unionized drug is available to penetrate the neural tissues)

Mepivacaine
- Available as 1, 1.5, or 2% concentrations
- Onset of action ranges from 3 to 20 min
- Duration of action of up to 2.5 h
- Dose ranges are 15–30 mL 1%, 10–25 mL 1.5%, and 10–20 mL 2% [17–19]

Ropivacaine
- Single isomer
- Moderately rapid onset of action (within 5–13 min)
- Available as 0.2, 0.5, and 1% concentrations (0.2% solution used for analgesia; 10–20 mL at 30–60 min intervals or at infusion rates of 6–14 mL/h)
- For surgery, 15–30 mL of 0.5% or 15–20 mL of 0.10% ropivacaine may be used
- Duration of action 3–5 h [20]
- It does not have any significant motor sparing action as claimed

Bupivacaine
- Available as 0.25 or 0.5 or 0.75% solutions in 5–10 mL vials or ampoules
- Widely available as a 0.1% solution in large volume bags (100–250 mL or more) often with fentanyl 2 or 4 μg/mL for epidural infusion
- Bolus doses of 10–20 mL with repeat dosing at 2-h intervals, depending on desired block
- Infusion rates from 5 to 15 mL/h for the 0.1% solutions [21, 22]
- Levobupivacaine, the levo form, has fewer cardiotoxic side effects without loss of the anesthetic potency [23]

Adjuvant Drugs

There are many drugs which are used to augment or supplement the effects of LA but the following are the commonly used in clinical practice:

Opioids

Opioids are the most commonly used adjuvant drugs; morphine, fentanyl, sufentanil, hydromorphone, and diamorphine have all been used as epidural adjuvants [24–26]. They prolong the duration of analgesia without any effect on the motor system. This effect is mediated by opioid receptors in spinal cord and the recommended infusion regimens are shown in Tables 12.6 and 12.7.

Epinephrine

Epinephrine can be used to increase the depth and duration of block through its effects of local vasoconstriction. This decreases the clearance of the LA from the tissues, allowing a reduced concentration and dose of drug to be administered. The usual concentration of epinephrine is 1:200,000 (5 μm/mL) [27] and it prolongs duration of both sensory and motor blockade. This is a significant feature in the case of LA with short and intermediate duration of action but in contrast this effect is not seen in the case of longer acting LA.

Epinephrine also exerts effects on the α2 adrenergic receptors present in the spinal cord, reducing transmission of nociceptive impulses. In addition to the neuraxial effects, epinephrine produces reduction in systemic vascular resistance as a result of its systemic absorption from the epidural space. This is the result of β2 stimulation of arterial adrenergic receptors which reduces mean arterial pressure leading to a reflex tachycardia.

Clonidine

Clonidine is a selective α-2 adrenergic agonist, which has been used extensively in epidural and spinal anesthesia for many years. Side effects include hypotension, bradycardia, and sedation which should be considered when choosing to use it in certain patients groups (e.g., pediatrics & elderly). It has been used as a sole agent in a dose of 300–600 μg and in conjunction with LA both intra- and postoperatively at doses of 75–150 μg.

Table 12.6 Epidural opioids recommended dose as continuous infusion

Drug	Solution (mg/mL)/(%)	Bolus dose (mg)	Basal infusion (per h)	Breakthrough doses (mg)	Increments in breakthrough (mg)
Morphine	0.1/0.01	4–6	0.5–0.8 mg	0.2–0.3 every 10–15 min	0.1
Hydromorphone	0.05/0.005	0.8–1.5	0.15–0.3 mg	0.15–0.3 every 10–15 min	0.05
Fentanyl	0.010/0.001	0.0005–0.0015	0.0005–0.001 mg/kg	0.010–015 every 10–15 min	0.010
Sufentanyl	0.001/0.0001	0.0003–0.0007	0.0001–0.0002 mg/kg	0.005–0.007 every 10–15 min	0.005
Alfentanyl	0.25/0.125	0.01–015	0.10–0.018 mg/kg	0.25 every 10 min	0.25

Table 12.7 Epidural Opoid-Bupivaine combination administered as infusion

Drug combinations	Solution (%)	Basal infusion (mL/h)	Breakthrough doses mL (interval, min)	Increments in breakthrough (mL of the solution)
Morphine	0.01	6–8	1–2 (10–15)	1
Bupivacaine	0.05–0.1			
Hydromorphone	0.0025–0.005	6–8	1–3 (10–15)	1
Bupivacaine	0.05–0.1			
Fentanyl	0.001	0.1–0.15/kg	1–1.5 (10–15)	1
Bupivacaine	0.05–0.1			
Sufentanyl	0.0001	0.1–0.2/kg	1–1.5 (10–15)	1
Bupivacaine	0.05–0.1			

Adapted from de Leon-Casasola OA, Lema MJ. Postoperative epidural opioid analgesia: What are the choices? Anesth Analg. 1996;83:867–875

Clonidine has been shown to reduce opioid use by 50% and prolong the analgesic effects of local anesthetics by 100% [28].

Ketamine

Ketamine has been used in conjunction with LA and opioids for intra- and postoperative analgesia in doses ranging from 0.5 to 1 mg/h (with morphine) up to 0.25 mg/kg/h (with sufentanil) and single boluses of up to 1 mg/kg have also been used. Potential side effects include dizziness, diplopia, dysphoria, dreams, hallucinations and disorientation, strange sensations, lightheadedness, sleep difficulties and confusion, although these are less common when ketamine is used epidurally (Table 12.8) [29].

Factors Affecting Spread of Drugs in the Epidural Space [30]

Patient Factors

These have minor effects on the epidural spread of local anesthetics:

Age

- In elderly patients intervertebral foraminal narrowing leads to higher spread of anesthetic
- In younger individuals, part of the injected dose moves out through the foraminae

Weight

- Obesity increases the spread of injected drugs (possibly due to raised intra abdominal pressure, as seen in pregnancy, which decreases

Table 12.8 Epidural Adjuvants

Drug	Dose	Effect
Epinephrine	1:200,000 (5 μg/mL)	Increases local vasoconstriction thereby decreasing the clearance of local anesthetic from the tissues. This effect means that often the concentration and dose of drug used can be reduced
Clonidine	300–600 μg as a sole agent	Selective alpha-2 adrenergic agonist
	75–150 μg in combination with local anesthetic (intra- or postoperatively)	Side effects include hypotension, bradycardia, and sedation, and operators should bear this in mind when choosing to use it
		It has been shown to reduce opioid use by 50% and prolong the analgesic effects of local anesthetics by 100%
Ketamine	0.5–1 mg/h (with morphine) up to 0.25 mg/kg/h (with sufentanil). Single boluses up to 1 mg/kg have also been used	Potential side effects include dizziness, diplopia, dysphoria, dreams, hallucinations disorientation, strange sensations, light headedness, sleep difficulties, and confusion, though these are less common when ketamine is used epidurally
	In combination with local anesthesia for caudals 0.5 mg/kg has been used	

the volume of epidural space because of venous engorgement
- These patients require smaller doses, relative to their weight, for a given level of blockade, although there is significant individual variability

Height

- Taller patients require higher although there is significant individual variability

Drug Factors

Total Dose

- This is a *major* factor determining the spread of epidural injections
- Higher doses produce higher level of blockade
- It is impossible to accurately predict the level of blockade for a given dose

Volume

- Greater volumes produce greater spread of anesthetic
- This can lead to reduction in the concentration of the drug if the total dose is kept constant
- This can lead to reduced intensity of epidural block
- When the concentration is maintained, greater volumes increase the dermatomal spread (in a nonlinear fashion)

Technical Factors

Position of Patient

- The position of the patient during injection has a minimal effect.

Site of Injection

- Site of injection is a *major* determinant of spread of epidural drugs
- Thoracic injections require lower dose as the epidural space is narrow in this location
- Caudal injections require higher volumes as there is a greater volume
- The effect is immediate and maximum at the site of injection and spreads cephalad and caudad over time (Fig. 12.9)

Type of Pharmacological Agent

Local anesthetics produce sensory, motor, and sympathetic blockade, while opiates produce analgesia without any of the above effects.

Complications of Epidural Blockade

Complications following epidural can be immediate or delayed as shown below in Table 12.9.

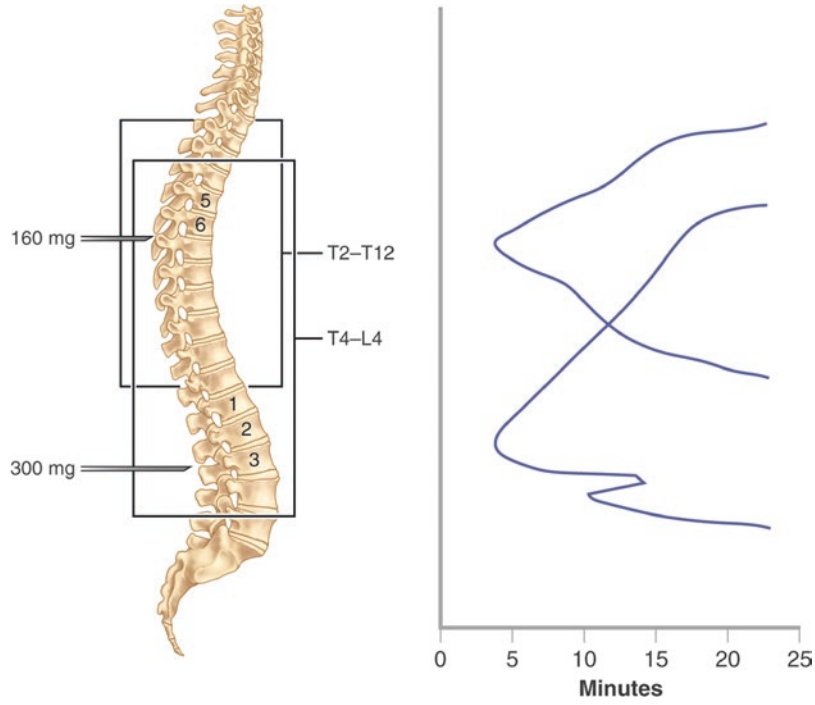

Fig. 12.9 Site of injection and spread of epidural drugs. Inspired by Mulroy MF. Regional anesthesia—an illustrated procedural guide. 3rd ed. Philadelphia, PA: Lippincott.

Table 12.9 Complications of epidural anesthesia

Immediate	Delayed
Nausea and vomiting	Residual neurological damage
Shivering	Post-dural-puncture headache
Itching	Back pain
Trauma	Epidural hematoma
Dural puncture	Epidural abbess
Secondary effects of sympathetic blockade	Arachnoiditis and cauda equine syndrome
Inadvertent intravascular injection of drugs	Retained catheter
Total spinal	
Subdural injection	
Incomplete block	

Immediate Complications

Immediate minor side effects are nausea, vomiting (treat hypotension, antiemetics), shivering (may respond to small doses of pethidine or ketamine) and itching (commonly due to opioids treated with nalbuphine, naloxone, or antihistamines).

Trauma

Poor technique, multiple attempts, poor positioning, and lack of patient cooperation can all contribute to injury of ligaments and soft tissues. Trauma to the vertebrae and intervertebral disks, though rare, is possible. This can be minimized with experience, careful planning, proper positioning, a gentle technique and good communication with the patient. Identification of the epidural space can be facilitated by using aids such as ultrasound.

Neurological Damage

Neurological damage, though rare, can occur during the procedure or as later sequelae. It may be traumatic or the result of inadvertent injection of neurotoxic drugs. Though the damage is produced during the procedure, clinical presentation with persistence of sensory loss or abnormal sensation or as neuropathic pain might be delayed until the effect of epidural injection wears off.

Performing an epidural with the patient awake can minimize this complication and confirmation of diagnosis is by detailed neurological examination

and radiological imaging. In many cases of neurological damage, though stressful both for patient and physician, is followed by complete or near complete recovery with only a few cases where the damage is permanent.

Bleeding

Bleeding can potentially occur from any tissue layer all along the path of the needle. If bleeding occurs through the needle, then it may have to be withdrawn and the procedure should be attempted at a different level. Bleeding from catheter can be managed by withdrawing the catheter by a few millimeters and flushing it with saline and repeating process until the catheter becomes clear (beware of catheter being completely pulled out of epidural space, or too little of it being inside; the latter runs the risk of delayed dislodgement) or by resiting at a different level. Initial injections of local anesthetic solutions then need to be administered through the catheter slowly and with frequent aspirations as a precaution.

Dural Tap

This usually presents as a dramatic flow of CSF through the Touhy needle or less frequently after the passage of epidural catheter. Sometimes a dual tap can remain concealed and present later as an inadvertent high spinal or a PDPH; there is a high incidence of PDPH (about 75%) following a dural tap. Management options include performing epidural in another level or converting it into a continuous spinal by inserting an intrathecal catheter (needs to be very careful when administering local anesthetic through this catheter as there is risk of accidental overdosing of local anesthetic).

Sympathetic Blockade

This is truly an excessive physiological effect of the extended epidural blockade.

Hypotension

Hypotension occurs secondary to vasodilatation and subsequent reduction in venous return. It usually has a slow onset in comparison with spinal and is easily treated with fluid loading and/or vasopressors (ephedrine, metaraminol - or phenylephrine). This is exaggerated in the presence of intravascular volume depletion secondary to dehydration or hemorrhage. Epidural epinephrine administered as an additive with local anesthetic can exaggerate this effect by its beta effect.

Bradycardia

Bradycardia is secondary to inhibition of sympathetic cardioaccelerator fibers (T1–T5) and reduced venous return. The treatment, in the presence of hemodynamic compromise, is with vagolytic agents (atropine or glycopyrrolate). Ephedrine by virtue of its effect both on heart rate and on blood pressure is yet another option.

Bronchospasm/Hypoxemia

Bronchospasm, though rare, is a possibility secondary to loss of bronchodilatory effect of the sympathetic system and the unopposed parasympathetic activity.

Hypotension can lead to an increase in pulmonary shunt and lead to *hypoxemia*, which is treated with supplementation of oxygen and by measures to increase blood pressure.

LA Toxicity

Accidental intravascular injection of local anesthetic drugs can lead to a range of side effects from mild symptoms like dizziness and circumoral tingling sensation to life-threatening circulatory collapse and convulsion. Management of this is in the line of airway, breathing, circulation (ABC) and organ support. Intralipid infusion [31] is claimed to be effective in reversing the effects of LA toxicity.

Total Spinal Anesthesia

This is the result of a large volume of LA injected accidentally into the subarachnoid space. Clinical manifestations include profound hypotension, respiratory insufficiency and loss of consciousness. Management is supportive following an ABC approach and a general anesthesia and artificial ventilation may need to be induced until recovery of the block.

Prevention is the best strategy to avoid a total spinal and this is done by giving a test dose before the full dose followed by testing motor functions

of lower limbs. As this complication can potentially occur at any point until the epidural is stopped and catheter is removed, it is prudent to administer LA after careful aspiration of the catheter for CSF every time a top-up is given. Often LA solutions in the epidural space that are returning through the catheter can be confused with CSF and testing for the presence of glucose in the CSF is helpful in this situation.

Subdural Injection

The subdural space lies between the dural and arachnoid layers and is a potential space that, unlike epidural space, extends into the cranial cavity. Relatively small volumes of local anesthetic solutions entering the subdural space following injection through a misplaced catheter can produce high levels of blockade. The incidence of these complications is very low [32] (less than 1/1000 epidurals) and associated with slow onset of high block with a predominant sensory blockade and motor sparing. This may be associated with Horner's syndrome. Management is supportive and allows the block to wear off spontaneously.

Inadequate Block

This can present in a variety of ways such as a unilateral or patchy block due to uneven distribution of the local anesthetic solution or pain sensation. Potential causes include epidural catheter tip positioned outside the space, mechanical obstruction to spread of anesthetic solution (for sacral sparing), bands or septae separating the individual nerve roots or the presence of an air bubble into the epidural space (higher in LOR techniques using air not saline). This can be managed by pulling the catheter back, increasing the concentration of local anesthetic solution topping up on the side with reduced block, adding adjuvants such as a narcotic or if all these fail—resiting the epidural.

Delayed Complications

Delayed complications can present at a varying time frame from epidural insertion and most centers routinely institute a policy whereby an anesthetist will follow up patients 24 h post epidural removal to pick up potentially serious complications.

Post-dural-Puncture Headache

This results from leakage of CSF and subsequent reduction in the CSF pressure and is more common following a dural tap. The onset is usually 24–48 h following the dural puncture and can last up to 10 days. It presents as frontal or occipital headache with nuchal extension, characteristically worse on standing and associated symptoms include nausea, tinnitus, hearing loss, photophobia and diplopia.

The initial approach is conservative management with simple analgesia, rehydration (oral or i.v.), abdominal binding and bed rest. Other medications that are found to be of benefit but without significant evidence include caffeine, sumatriptan and ACTH. If this fails, then an epidural blood patch is performed using the patient's own blood. The procedure requires two doctors; one to draw the patient's blood in an aseptic manner, while the other performs the epidural and injects the blood (20 mL or until the patient complains of pressure). The dural rent is sealed off by the subsequent formation of a blood clot and often this produces a dramatic relief of PDPH; however, this may have to be repeated if the headache returns.

Back Pain

Back pain is secondary to local trauma and is common and usually resolves with simple analgesics. Those due to soft tissue hematoma especially in the ligaments take a longer time (6–8 weeks) to resolve. Chronic back pain per se due to tissue (bone, disk, or ligament) injury during epidural injection is possible but very rare.

Epidural Hematoma

This is a rare yet serious delayed complication of epidural neuraxial blockade with an incidence of 1/150,000 (preexisting coagulopathy is a risk factor). Pain may be the first presenting symptom and followed by loss of neurological functions, which is a result of compression of the cord by

the expanding hematoma inside the rigid spinal canal. Undue prolongation of residual motor block should raise the suspicion of this condition. Radiological imaging should be undertaken immediately to confirm or rule out hematoma. Surgical decompression should be instituted early (within 6 h) as a delay can cause permanent neurological damage.

Epidural Abscess

Epidural abscess is another rare but serious complication of epidural injection. Risk factors include systemic sepsis and history of intravenous drug abuse. Initially it presents with non-specific symptoms such as fever and backache several days following epidural injection. There may be local tenderness over the spine and later progressing on to sensory loss and paraplegia. Laboratory findings may include elevated white cell count and raised ESR and CRP. Diagnosis is confirmed by MRI and treatment is again urgent surgical decompression and intravenous antibiotics.

Adhesive Arachnoiditis and Cauda Equina Syndrome

This complication has been reported [33, 34] following epidural injections and is the result of neurotoxicity of injectates (e.g., lignocaine, glass particles from ampoules). Clinical features are bowel and bladder disturbances, pain, paresthesia and patchy sensory abnormalities affecting the perineal regions and lower limb. Chemical arachnoiditis due to glass is prevented by not using medications from glass ampoule or using a glass filter for drawing up the injectate. Treatment is conservative and recovery may be incomplete.

Retained Catheter

Excessive lengths of epidural catheter introduced into the epidural space can lead to knotting and removal can be difficult or impossible. Leaving excessive length of catheter inside the epidural space is probably not a good practice. The terminal portion of catheter can break off and get lodged in the epidural space following an attempt to withdraw it through the needle. These catheters can be left in the epidural space without undue fear of tissue reaction as they are implantation tested. However, the patient should be warned of its presence in order to avoid future confusion with regard to the presence of a foreign body.

Clinical Pearls

Successful administration of epidural block begins from the pre-assessment phase right through to cessation of epidural analgesia. Detailed attention to patient selection and thoughtful planning and execution, as well as follow-up of patients, are necessary for a successful procedure with minimal complications.

A summary of the key points relating to epidural anesthesia:

Pre-assessment

Specifically Explore

History

- Symptoms of autonomic neuropathy
- Hypovolemia
- Features of local infection or sepsis
- Coagulopathy (INR < 1.5 is acceptable)
- Medications affecting haemostasis (Table 12.2)

Examination

- Airway assessment (potential for failure or complications)
- Spine (ease of palpation of spinous processes)
- Back (assess any abnormality and evidence infections/tattoos)
- Venous access

Investigations

- FBC
- Coagulation screen

Procedure

Surface Anatomy

- The depth of the epidural space is most shallow in the lumbar region deepest in the cervical region
- Vertebral flexion in the cervical and lumbar spines means making the patient maximally flex is beneficial
- Thoracic vertebrae only permit rotation so flexion makes little difference.

Preparation

- Check and prepare emergency drugs and GA equipment
- Maintain verbal contact with patient at all times (can give early warning of immediate/imminent complications and anility to speak is a good sign of adequate cerebral perfusion)
- Proper positioning can make the procedure far easier
- Palpating the thoracic spines downward helps identify landmarks in obese individuals.
- LA needle can be used as a seeker needle in technically challenging cases
- Test all epidural catheters prior to insertion
- Full aseptic technique should be followed at all times including even when topping up

Procedure

- Avoid rotating the Touhy needle inside the epidural space to help in threading the epidural as this can produce dural tear with subsequent passage of catheter into the subdural space

Complication

Bloody Tap

- Avoid administering heparin for 2 h and LMWH for 24 h

Subdural Injection

- Can manifest as patchy epidural block, high or total spinal
- Rapid injections can force the LA into the subdural space, while slow injections lead to epidural spread

Neurological

- Performing the epidural awake is the safest option as the patient can guide the operator in avoiding nerve damage

Catheter Migration

- Aspirating the catheter for blood and CSF is mandatory before each top-up (failure to do so can end in a catastrophe)

Review Questions

1. Regarding epidural analgesia the following statement is correct:
   (a) Reduces postoperative mortality
   (b) There is a reduction in venous thrombosis
   (c) It increases catecholamine release
   (d) Can lead to metabolic dysfunction
2. Epidural space extends from:
   (a) Cranial cavity to sacral foramina
   (b) Foramen magnum to lower border of L2
   (c) Foramen magnum to sacrococcygeal ligament
   (d) Foramen magnum filum terminale
3. Epidural space is thinnest in:
   (a) Cervical region
   (b) Thoracic region
   (c) Lumbar region
   (d) Caudal region
4. Regarding surface markings, the following statement is NOT true:
   (a) Vertebra prominence corresponds to C8 vertebra
   (b) Inferior angle of the scapula corresponds to T7 vertebra

(c) Superior aspect of iliac crest corresponds to L4 vertebra
(d) Posterior superior iliac spine corresponds to S2 vertebra

5. When performing thoracic epidural injection:
   (a) It is always performed by a paramedian approach
   (b) Midline approach is impossible because of the acute angulation of the thoracic spinous processes
   (c) It is possible to perform it by inserting the needle in the midline at an acute angle
   (d) Motor blockade produced at this level can paralyze the diaphragm
6. When performing a caudal epidural injection, the structures through which the needle passes through are:
   (a) Skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, and ligamentum flavum
   (b) Skin, subcutaneous tissue, sacrococcygeal membrane and ligamentum flavum
   (c) Skin, subcutaneous tissue, interspinous ligament, and sacrococcygeal membrane
   (d) Skin subcutaneous tissue and sacrococcygeal membrane
7. A 75-year-old man with a history of ischemic heart disease (IHD) is undergoing hip arthroplasty under epidural. His base line heart rate and mean arterial pressure (MAP) prior to anesthesia were 75/min and 100 mm of Hg, respectively. After administration of epidural you notice that his heart rate has dropped to 55/min and MAP to 50 mm of Hg. Choose the most logical intervention:
   (a) There is no need for any intervention as the reduction in heart rate has reduced the myocardial oxygen demand
   (b) Atropine is indicated for bradycardia
   (c) Hypotension need not be treated as it helps to reduce blood loss
   (d) Treat hypotension as the reduction in myocardial oxygen demand will be offset by reduced oxygen supply secondary to hypotension
8. All of the following are true regarding epidural blockade except:
   (a) A reduction in heart rate can be due to blockade of cardioaccelerator fibers
   (b) A reduction in heart rate can be due to baroreceptor-mediated reflex bradycardia
   (c) Reduction in blood pressure due to vasodilatation reduced venous return and reduced adrenal cortical secretions
   (d) Selective lumbar or thoracic blockade has the benefit of providing hemodynamic stability as a result of compensatory vasoconstriction in unblocked segments
9. Which of the following effect is NOT caused by epidural blockade?
   (a) Parasympathetic blockade leading to reduction in blood pressure and nausea
   (b) Small, contracted bowel
   (c) Increased upper GI motility
   (d) Increased GI secretions
10. Electrodes for spinal cord stimulation are place in:
   (a) Subarachnoid space
   (b) Subdural space
   (c) Intrathecal space
   (d) Epidural space
11. The following is an absolute contraindication for epidural blockade:
   (a) Coagulopathy
   (b) Patient refusal
   (c) A bleeding patient
   (d) A septic patient
12. You are performing an epidural catheter insertion for labor analgesia. The epidural space is identified at a depth of 6 cm. A catheter is then passed and on withdrawing the Touhy needle the marking at the skin corresponds to 15 cm. The length of catheter remaining inside the epidural space probably is:
   (a) 15 cm
   (b) 10 cm
   (c) 9 cm
   (d) 11 cm

13. The structures negotiated by a Touhy needle during the paramedian approach are:
    (a) Skin, subcutaneous tissue, paraspinous muscles, and ligamentum flavum
    (b) Skin, subcutaneous tissue, interspinous ligament, and ligamentum flavum
    (c) Skin, subcutaneous tissue, paraspinous muscles, and interspinous ligament
    (d) Skin, subcutaneous tissue, supraspinous ligament, and ligamentum flavum
14. When epinephrine is used as an additive in epidural anesthesia:
    (a) The dose of local anesthetic should be reduced to avoid drug toxicity
    (b) It increases the depth of neural blockade
    (c) There is a rapid systemic absorption of the drugs injected which is indicated by an increase in heart rate
    (d) A sudden increase in heart rate indicates successful epidural deposition of the drug
15. The following statement is NOT true regarding PDPH:
    (a) CSF pressure is low
    (b) The treatment of choice is immediate blood patch
    (c) Can present with cranial nerve symptoms
    (d) Its relation to posture is characteristic

Answers

1. b—Epidural analgesia reduces the stress response to surgery by providing effective analgesia, which is beneficial for cardiovascular, respiratory, and metabolic functions. There is a reduction in postoperative hypercoagulable state, which in turn reduces the incidence of deep vein thrombosis. Though there is a reduction in postoperative morbidity, there is no evidence for reduction in mortality after surgery.
2. c
3. a—It is 2 mm in thickness at the cervical region and 5–6 mm in the lumbar region.
4. a—There is no C8 vertebra though there is C8 nerve root.
5. c—Though paramedian approach is relatively easy to perform, a midline approach can also be used to administer thoracic epidural injection. Motor blockade at thoracic level does not affect diaphragmatic function as the innervation of diaphragm is by the phrenic nerve, which originates at cervical level (C3–C5).
6. d—There is no ligamentum flavum over the sacral hiatus as it fuses with the caudal lamina.
7. d—Though reduction in heart rate reduces myocardial oxygen demand, oxygen supply will be maintained only if MAP is maintained. In the presence of IHD, it is safe not to allow the MAP to drop below 20% of the base line value.
8. c—Reduction in blood pressure is due to a reduction in adrenal *medullary* secretions.
9. a—Sympathetic blockade leads to unopposed vagal dominance.
10. d
11. b—All the others are relative contraindications.
12. c
13. a—Paramedian approach avoids both supraspinous and interspinous ligaments, and hence, it is easier by this approach in the elderly who may have calcified ligaments.
14. b—Epidural epinephrine produces local vasoconstriction and decreases systemic absorption of local anesthetic, and hence, a higher dose of local anesthetic can be used. A sudden increase in heart rate indicates the intravascular injection of the drug (into epidural veins).
15. b—First line of treatment for PDPH is bed rest, simple analgesics, and hydration. If this fails then epidural blood patch is indicated.

References

1. Moraca RJ, Sheldon DG, et al. The role of epidural anesthesia and analgesia in surgical practice. Ann Surg. 2003;238(5):663–73.
2. Neal JM. Epidural anesthesia. In: Rathmell JP, Neal JM, Viscomi CM, editors. Regional anesthesia—the requisites in anaesthesiology. Philadelphia, PA: Elselvier/Mosby; 2004. p. 99–113.
3. Baron J, Decaux-Jacolot A, Edouard A, et al. Influence of venous return on baro-reflex control of heart rate during lumbar epidural anesthesia in humans. Anesthesiology. 1986;64:188.
4. Kunnumpurath S, et al. Spinal cord stimulation: principles of past, present and future practice: a review. J Clin Monit Comput. 2009;23(5):333–9.
5. Stone PA, Kilpatrick AW, Thorburn J. Posture and epidural catheter insertion. The relationship between skill, experience and maternal posture on the outcome of epidural catheter insertion. Anaesthesia. 1990;45(11):920–3.
6. AnaesthesiaDotCalm.Org Epidural Abscess. 2007. http://anaesthesiadotcalm.org/Epidural%20 Abscess.pdf. Accessed 13 May 2010.
7. Hamilton CL, Riley ET, Cohen SE. Changes in the position of epidural catheters associated with patient movement. Anesthesiology. 1997;86(4):778–84.
8. Moore JM, Liu SS, Neal JM. Premedication with fentanyl and midazolam decreases the reliability of intravenous lidocaine test dose. Anesth Analg. 1998;86:1015–7.
9. OAA Epidural Information card. 2008. Online: http://www.oaa-anaes.ac.uk/ui/content/content.aspx?id=161. Accessed 24 Nov 2016.
10. Shanthanna H, Mendis N, Goel A. Cervical epidural analgesia in current anaesthesia practice: systematic review of its clinical utility and rationale and technical considerations. Br J Anaesth. 2016;116(2):192–207.
11. Camorcia M, Capogna G. Sensory assessment of epidural block for Caesarean section: a systematic comparison of pinprick, cold and touch sensation [Abstract]. Eur J Anaesthesiol. 2006;23(7):611–7.
12. Hogan Q. Epidural catheter tip position and distribution of injectate evaluated by computed tomography. Anesthesiology. 1999;90:964–70.
13. Hogan Q. Distribution of solution in the epidural space: examination by cryomicrotome section. Reg Anesth Pain Med. 2002;27:150–6.
14. Higuchi H, et al. Factors affecting the spread and duration of epidural anesthesia with ropivacaine. Anesthesiology. 2004;101(2):451–60.
15. Mulroy MF, Bernards CM, McDonald SB, Salinas FV. A practical approach to regional anesthesia. 4th ed. Phildelphia: Lippincott; 2009.
16. Nesacaine Product Monograph (Revised 29 Jun 2006). http://www.astrazeneca.ca/documents/ProductPortfolio/NESACAINE_PM_en.pdf. Accessed 14 May 2010.
17. Xylocaine Parenteral Injections Product Monograph (Revised 17 Apr 2008). http://www.astrazeneca.ca/documents/ProductPortfolio/XYLOCAINE%20Parenterals_PM_en.pdf. Accessed 14 May 2010.
18. Mepivacaine Product Monograph. http://www.medscape.com/druginfo/monograph?cid=med&drugid=150529&drugname=Mepivacaine+(PF)+Inj&monotype=monograph. Accessed 14 May 2010.
19. Carbocaine Product Monograph (Revised Apr 2010). http://www.drugs.com/pro/carbocaine.html. Accessed 14 May 2010.
20. The New York School of Regional Anesthesia. Local anesthetics (16 Mar 2009). http://www.nysora.com/regional_anesthesia/equipment/3116-local_anesthetics.html. Accessed 14 May 2010.
21. Naropin Product Monograph (Prepared Feb 2000, Rev Mar 2008). http://www.astrazeneca.ca/documents/ProductPortfolio/NAROPIN_PM_en.pdf. Accessed 14 May 2010.
22. Marcaine Product Monograph. 2009. http://www.medscape.com/druginfo/monograph?cid=med&drugid=76904&drugname=Marcaine+(PF)+Inj&monotype=monograph&secid=4. Accessed 14 May 2010.
23. Anaesthetics (Parenteral-Local). http://www.drugs.com/mmx/chirocaine.html. Accessed 14 May 2010.
24. Morrison SG, Dominguez JJ, Frascarolo P, Reiz S. A comparison of the electrocardiographic cardiotoxic effects of racemic bupivacaine, levobupivacaine, and ropivacaine in anesthetized swine. Anesth Analg. 2000;90:1308–14.
25. Block BM, Liu SS, Rowlingson AJ, Cowan AR, Cowan JA Jr, Wu CL. Efficacy of postoperative epidural analgesia: a meta-analysis. JAMA. 2003;290(18):2455–63.
26. Lowson SM, Alexander JI, Black AM, Bambridge AD. Epidural diamorphine infusions with and without 0.167% bupivacaine for post-operative analgesia. Eur J Anaesthesiol. 1994;11(5):345–52.
27. de Leon-Casasola OA, Lema MJ. Postoperative epidural opioid analgesia: what are the choices. Anesth Analg. 1996;83:867–75.
28. Mulroy MF, Norris MC, Liu SS. Safety steps for epidural injection of local anesthetics: review of the literature and recommendations. Anesth Analg. 1997;85:1346–56.
29. Eisenach J, De Kock M, Klimscha W. Alpha-2 adrenergic agonists for regional anesthesia: a clinical review of clonidine (1984–1995). Anesthesiology. 1996;85(3):655–74.
30. Subramaniam K, Subramaniam B, Steinbrook RA. Ketamine as adjuvant analgesic to opioids: a quantitative and qualitative systematic review. Anesth Analg. 2004;99(2):482–95.

31. Mulroy MF. Regional anesthesia—an illustrated procedural guide. 3rd ed. Philadelphia, PA: Lippincott; 2002. p. 93–118.
32. Weinberg G, et al. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. Reg Anesth Pain Med. 2003;28:198–202.
33. Lubenow T, Keh-Wong E, Kristof K, et al. Inadvertent subdural injection: a complication of epidural block. Anesth Analg. 1988;67:175–9.
34. Drasner K, Rigler ML, Sessler ID, et al. Cauda equina syndrome after incidental total spinal anesthesia with 2% lidocaine. Anesthesiology. 1992;77:582–5.
35. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines. Reg Anesth Pain Med. 2010;35(1):64–101.

Suggested Reading

Moore DC. Regional block: a handbook for use in the clinical practice of medicine and surgery. 4th ed (10th printing). Charles C Thomas: Springfield, IL; 1981.
Mulroy FM, Bernards CM, McDonald SB, Salinas FV. A practical approach to regional anesthesia. 4th ed. Philadelphia, PA: Lippincott; 2009.
Pinnock CA, Lin T, Smith T. Fundamentals of anaesthesia. 2nd ed. London: Greenwich Medical Media Ltd; 2002.
Wong CA. Spinal and epidural anesthesia. 1st ed. McGraw-Hill Professional: Columbus, OH; 2007.
Yentis SM, Hirsch NP, Smith GB. Anaesthesia and intensive care A-Z an encyclopaedia of principles and practice. 3rd ed. London: Elsevier (Butterworth Heineman); 2004.

13 Peripheral Nerve Blocks for the Lower Extremity

Sylvia H. Wilson and Anna Uskova

Clinical Case

A 54-year-old male presents to the preoperative clinic prior to total knee arthroplasty. He had a previously arthroplasty and experienced great pain in the postoperative period. He further comments that opiate medicines make him hallucinate and he would like to minimize these medications as well. He is interested in regional anesthetic techniques that may minimize his postoperative pain.

Introduction

Certain steps must be taken before offering a peripheral nerve block to a patient:

- Surgeon and primary anesthesia care team aware and in agreement.
- Patient meets all criteria for surgery and anesthesia: NPO, cardiopulmonary status, etc.
- Patient evaluation is complete: laboratory values, EKG, and CXR.
- Paperwork is completed: patient identification, surgical consent, and laterality.
- Contraindications to block are not present: coagulopathy, refusal (see later section).
- Patient consents to block: risks, benefits, and options explained.

Preparation

- Monitors: blood pressure cuff and pulse oximeter.
- Supplemental oxygen: nasal cannula or face mask.
- Laterality verification (when applicable) by two independent parties.
- Pillows or blankets for positioning.
- Skin marker for laterality and landmarks.
- Drugs: emergency drugs (local anesthetic toxicity, hypotension) and sedative (optional).
- Sterile supplies: antiseptic soap, gloves, needle (see each section), and gauze.
- Local anesthetic: lidocaine to anesthetize skin, local anesthetic or saline to be injected through block needle.
- Special equipment: nerve stimulator (set to 1.5 mA, 2 Hz, and 0.1 ms; may need to increase frequency to 0.3 ms in patients with neuropathy) and ultrasound (US).

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_13

- Catheters sterile supplies (when applicable): towels or drapes, dressing materials to secure the catheter, and sleeve for US probe.
- Nurse or trained assistant to help with documentation, monitoring, and emergencies.

Complications

1. Local anesthetic toxicity from intravascular injection: aspirate prior to the initial injection of local anesthetic and again after every 5 mL injected.
2. Nerve injury: this is much more likely due to a number of other phenomena than direct needle trauma. Differential diagnosis includes direct surgical trauma, ischemic injury from prolonged tourniquet application, and compressive injury from improper patient positioning. Nerve injury during regional anesthesia can be permanent but usually resolves over weeks. It may result from compression by local hematoma formation, injury by intraneural injection, and needle-associated direct nerve trauma. Minimize this complication by avoiding paresthesias, pain with injection, injection with muscle stimulation at <0.2 mA, and injection with increased resistance.
3. Mask the onset of lower extremity compartment syndrome: lower extremity nerve blocks may be relatively contraindicated in patients with fractures of the tibia and fibula or elective orthopedic procedures of the tibia and fibula.
4. If placing a catheter, confirm with the patient's surgical team that a tourniquet will not be placed over the catheter during the operation. While much evidence is not available, concern exists that tourniquet placement over a peripheral nerve catheter may cause or be associated with an increased risk of nerve damage.

Contraindications

- Patient refusal (number one reason not to do a nerve block).
- Allergy to local anesthetics.
- Infection at the site of injection, sepsis, and generalized systemic infections (elevated temperature and white blood cell count).
- Coagulopathy (more concerning at noncompressible/deep block sites, e.g., lumbar plexus).
- History or diagnostic evaluation that would cause cancellation of surgical procedure.

Sciatic Nerve Block

Anatomy

The largest nerve of the human body, the sciatic nerve, provides motor and sensory innervation to the posterior thigh and lower leg. Sciatic nerve blockade is indicated for pain management associated with lower extremity surgery. Since the saphenous nerve, a branch of the femoral nerve, supplies the medial aspect of the lower leg, very few surgical procedures can be performed with the sciatic block alone, and it is usually combined with a lumbar plexus, femoral, or saphenous nerve block.

The sciatic nerve can be blocked from several different locations depending on the desired area of analgesia. We will discuss each of these in groups according to how the patient is positioned for the nerve block.

Indication

The sciatic nerve arises from the lumbosacral plexus (ventral rami of L4–5 and S1–3) and is actually two nerves in close apposition (tibial—medial—and common peroneal—lateral—nerves). In the pelvis, it is part of the sacral plexus with superior gluteal nerve, inferior gluteal nerve, and posterior femoral cutaneous nerve. The sciatic nerve leaves the pelvis though the greater sciatic foramen below the piriformis muscle, runs between muscle layers in the gluteal region (superficial to superior and inferior gemellus, quadratus femoris, and obturator internus muscles; deep to the gluteus

maximus muscle), and continues distally toward the posterior thigh between the greater trochanter and ischial tuberosity.

Response to Nerve Stimulation

Successful stimulation of the sciatic nerve is identified by plantar flexion/inversion (tibial nerve) or dorsiflexion/eversion (common peroneal nerve). However, several studies have shown tibial stimulation to be associated with a more frequent success rate compared with peroneal stimulation with various approaches [1, 2].

Procedure

Needle: 10 cm insulated needle (15 cm—obese patients; 5 cm—prone popliteal approach).

Posterior Approaches

Place the patient in the lateral position with the nonoperative down, operative side up. Straighten the patient's dependent, nonoperative leg and flex the operative extremity as much as they are able at the hip and to a lesser extent at the knee. Flexing the knee at the hip flattens the gluteal muscles and brings the sciatic nerve into a more superficial position.

1. Labat or "Classic" Approach. First described in 1924, it has the advantage of blocking the posterior femoral cutaneous nerve [3]. (The parasacral approach does this as well).

   Landmarks
   - Posterior superior iliac spine (PSIS).
   - Greater trochanter.
   - Sacral hiatus.

   Draw a line between the greater trochanter and PSIS (line 1). Draw a second line from the sacral hiatus to the greater trochanter (line 2). Draw a perpendicular, caudad line from the midpoint of line 1 that intersects line 2 (this is line 3). The needle insertion points in where lines 2 and 3 intersect (Fig. 13.1).

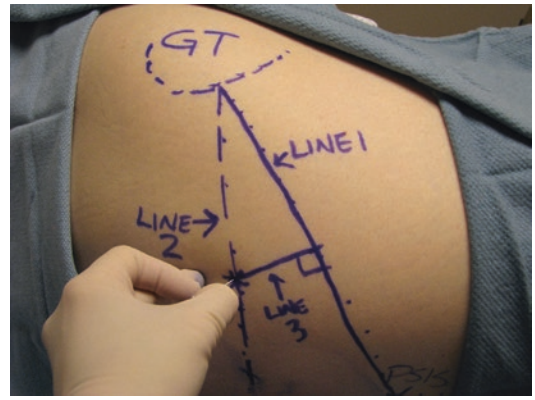

Fig. 13.1 Labat approach. The greater trochanter (GT), posterior superior iliac spine (PSIS), and sacral hiatus (SH) are identified. *Line 1* connects GT to PSIS. *Line 2* connects GT to SH. *Line 3* bisects and is perpendicular to *line 1* and is drawn caudad to intersect *line 2*. The needle is inserted where *line 3* intersects *line 2*

   Insert the needle perpendicular to all planes. Stimulation of the gluteus maximus muscle is often encountered just before the sciatic nerve stimulation.

   Alternative

   Instead of drawing line 2, you may draw a perpendicular and caudad line (equivalent to line 3) from the midpoint of line 1 that is 5 cm long. The needle is inserted at the end of this line perpendicular to all planes.

   Tips
   - Hamstring muscle stimulation: redirect the needle tip laterally.
   - Piriformis muscle stimulation is very painful to the patient and is deep to the nerve.

2. *Parasacral Approach (Sacral Plexus Block)*. The most proximal approach to the sciatic nerve at the level of the sacral plexus, the nerves are targeted in the greater sciatic foramen. Stimulating the superior gluteal nerve (gluteus medius/minimus or tensor fascia lata), inferior gluteal (gluteus maximus) or sciatic (peroneal, tibial) nerve is acceptable at this level. The posterior femoral cutaneous nerve is purely sensory. Superior gluteal nerve stimulation is preferred for patients undergoing hip surgery (innervates hip capsule).

Unlike other sciatic nerve blocks, the parasacral approach also will block the obturator nerve [4].

Landmarks
- PSIS.
- Ischial tuberosity.

  Draw a line connecting the PSIS to the ischial tuberosity. Insert the needle 7–8 cm caudal to the PSIS along this line, perpendicular to all planes (Fig. 13.2). The nerve is usually 6–8 cm below the skin.

Ultrasound

Scan the region caudal to the posterior superior iliac spine with a low-frequency probe and identify the ischial bone and the lateral border of the sacrum. (These define the greater sciatic foramen). The sacral plexus appears as a hyperechoic bundle (Fig. 13.3). Visualization may be improved by aiming at the greater trochanter.

Tips
- If os is contacted, "walk off" caudally and laterally until the needle advances through the sciatic foramen. If you continue to touch os, reexamine landmarks.
- The needle insertion point should be approximately at the level of the intergluteal cleft (see Fig. 13.2).

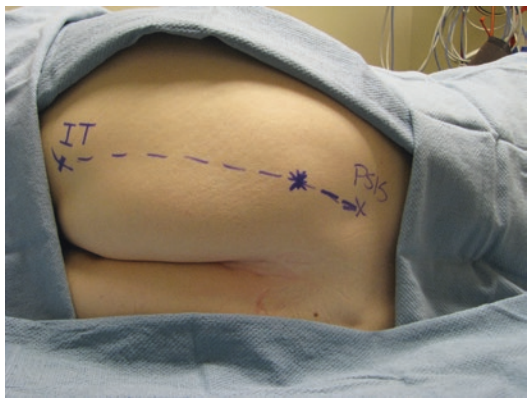

Fig. 13.2 Parasacral approach. Posterior superior iliac spine (PSIS) and ischial tuberosity (IT) are identified. A line is drawn between PSIS and IT. The needle insertion point is 7–8 cm caudal to the PSIS along this line

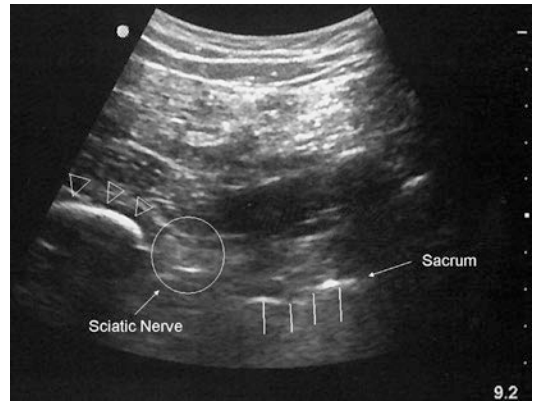

Fig. 13.3 Ultrasound-guided parasacral approach. *Arrow heads* identify the ischial bone (IB) and *straight lines* identify the sacrum. The sacral plexus is marked by a *circle*

3. *Mid-gluteal (Carlo Franco) Approach.*

Landmarks
- Gluteal Crease.
- Identify the midpoint of the gluteal crease. Draw a line perpendicular to the crease 10 cm in length. The insertion point is at the 10 cm mark, perpendicular to all planes.

4. *Gluteal and Subgluteal Approach.*

Landmarks
- Greater trochanter.
- Ischial tuberosity.
- *Gluteal*: Draw a line connecting these landmarks and identify the midpoint of this line. Insert the needle perpendicular to all planes (Fig. 13.4).

Ultrasound

If using an ultrasound, identify the greater trochanter and ischial tuberosity under ultrasound. The nerve will be located between these and may have a "flat" appearance (Fig. 13.5). Insert the needle from the lateral aspect of the probe (near the greater trochanter).

Subgluteal: Draw a line connecting these landmarks and, at the midpoint, draw a 3- to 5-cm perpendicular line in the caudad

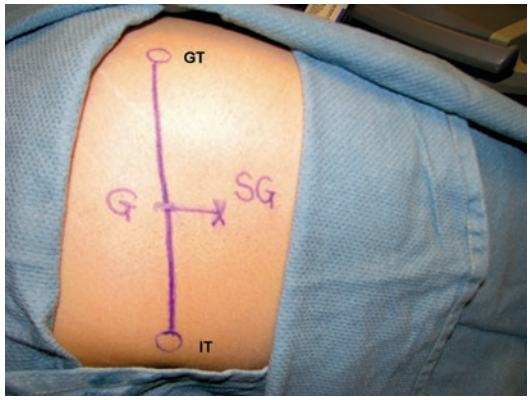

Fig. 13.4 Gluteal and subgluteal approach. The patient is positioned laterally (superior = lateral; inferior = medial). The *lateral circle* marks the greater trochanter (GT), and the *medial circle marks* the ischial tuberosity (IT). A line connects GT and IT. The midpoint of this line marks the insertion point for a gluteal (G) approach. Caudad 3–5 cm to the midpoint of the line is the insertion point for a subgluteal (SG) approach

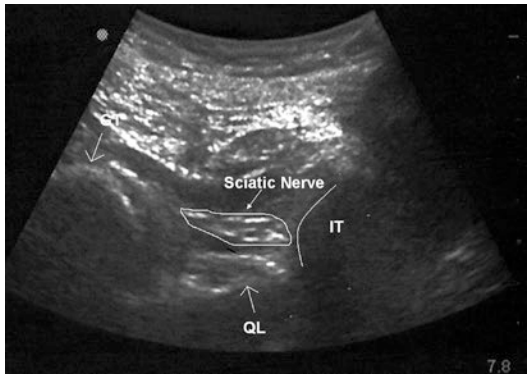

Fig. 13.5 Ultrasound-guided gluteal approach. The *arrow* on the left identifies the greater trochanter. The *arrow* at the bottom identifies the quadratus lumborum. The *curve line* on the right identifies the ischial tuberosity. The sciatic nerve is outlined by a *white line* and lies in the middle of the other structures

direction (see Fig. 13.4). Insert the needle perpendicular to all planes.

Tips

- Look or palpate for a longitudinal groove that runs along the posterior thigh starting at the bottom of the buttocks. This groove represents the origin of the semitendinosus from the ischial tuberosity and the biceps femoris long head. It is the path of the sciatic nerve.
- The subgluteal approach is beneficial due to the superficial position of the sciatic nerve. This approach will also not cause direct muscle stimulation of the gluteus maximus which may be painful for some patients.
- In the gluteal approach, stimulation of the gluteus maximus gives you an approximate depth of needle insertion. The sciatic nerve is 1–2 cm deeper than the gluteus maximus.

Supine Approaches

Use these approaches when it is not possible to move patients out of the supine position. Position the patient completely supine. A pillow may be placed behind the head if needed.

1. *Raj Approach*. Known as the lithotomy approach, the patient remains supine with the hip and knee flexed at 90° with the foot resting on a table or held by an assistant.

   Landmarks
   - Greater trochanter.
   - Ischial tuberosity.

   Draw a line connecting these landmarks and identify the midpoint of this line. Insert the needle perpendicular to all planes (Fig. 13.6).

2. *Anterior Approach*. In this approach, the sciatic nerve is blocked just beyond the hip joint (posterior cutaneous nerve of the thigh will likely not be blocked). The depth of insertion is also commonly greater than the depth for a posterior approach, and a longer needle may be required. Externally rotate the patient's leg if possible [5, 6].

   Landmarks
   - Pubic tubercle (PT).
   - Anterior superior iliac spine (ASIS).

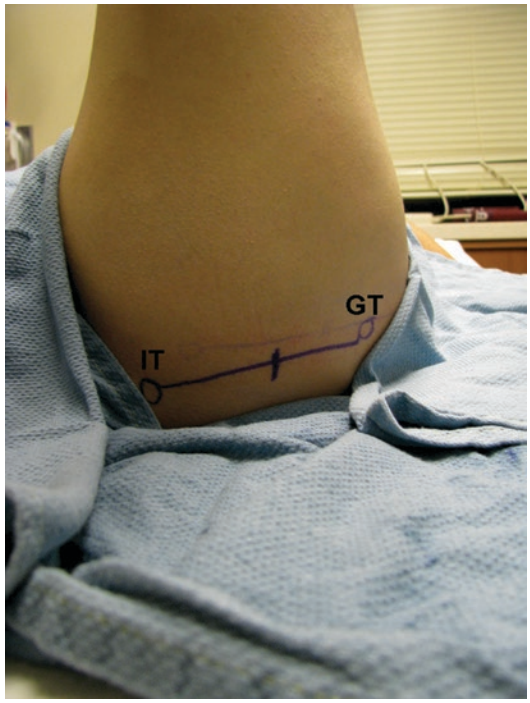

Fig. 13.6 Raj approach. The *lateral circle* marks the greater trochanter, and the *medial circle* marks the ischial tuberosity. The middle of the line connecting these points marks the needle insertion point

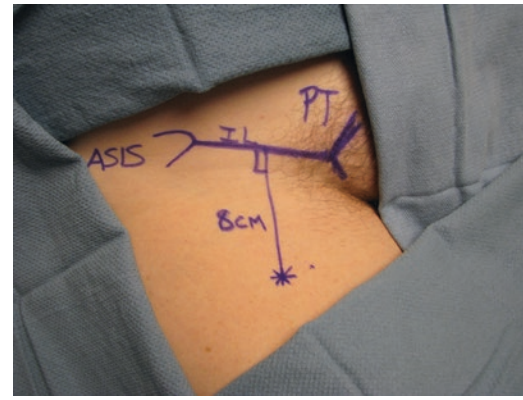

Fig. 13.7 Anterior approach. The pubic tubercle (PT) and anterior superior iliac spine (ASIS) are connected by a *line* representing the inguinal ligament. At the midpoint of this line, a perpendicular 8 cm line is drawn caudally. The insertion point is at the end of this line

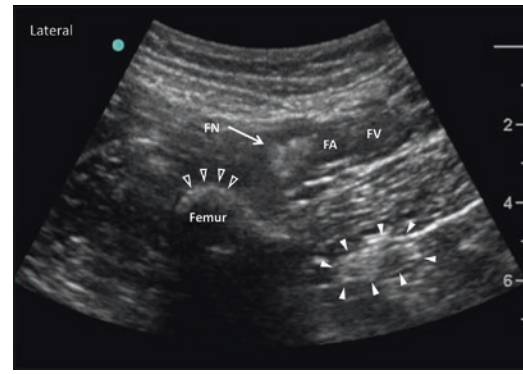

Fig. 13.8 Ultrasound-guided anterior-medial approach. The femur is marked by four open *arrow heads*, while the femoral nerve and vessels are *noted superficially*: nerve (FN), artery (FA), and vein (FV). The sciatic nerve is identified (closed arrow heads) medial and deep to the femur and lateral to the adductor magnus (*left* is lateral and *right* is medial)

Identify the ASIS and PT and connect with a line, which represents the inguinal ligament. Bisect the initial line and extend a perpendicular line 8 cm caudad (Fig. 13.7). Insert the needle perpendicular to the skin in a slight lateral direction. The needle will likely strike close to the medial edge of the femur at approximately the level of the greater trochanter. Remove the needle slightly and redirect the tip more medially (needle change to a more vertical orientation) and advance the needle. Repeat this until the needle "walks off" the medial edge of the femur. This approach has a high risk of vascular puncture, and aspiration for intravascular placement is particularly important.

Ultrasound

Ultrasound may facilitate this block greatly since there is no need to palpate bony structures and allows visualization of femoral vessels. The nerve will be lateral to the femur and 2 cm dorsal. The needle may be inserted medial or lateral to the probe, but a medial insertion with help avoid puncture of the femoral vessels. The nerve will be located between the femur and the adductor magnus muscle (Fig. 13.8).

Tips

- Once bone is identified, note this depth. The final depth of the sciatic nerve should be an additional 5 cm or less past this depth.
- Not successful: insert needle 1–2 cm medial to the original insertion site. (This

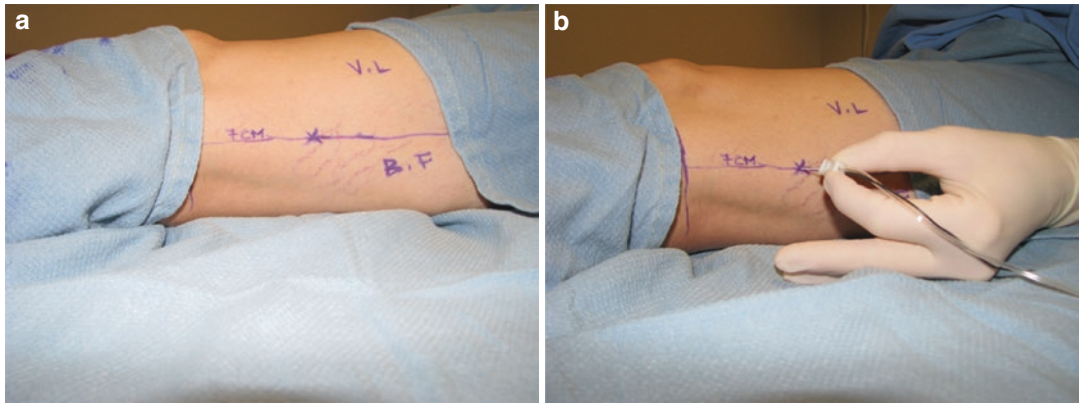

Fig. 13.9 Lateral popliteal approach. (**a**) The vastus lateralis (VL) and biceps femoris (BF) muscles are identified. (**b**) The popliteal crease is identified, and the needle insertion point is marked 7 cm cephalad

allows the needle to pass the femur's medial edge at a greater angle and directs the tip posterior to the femur).

3. *Popliteal Lateral Approach*. Ideal for surgeries of the lower leg, foot, and ankle, this approach preserves the function of the hamstrings. To help avoid an incomplete block, a larger volume of local anesthetic (30–40 mL) is often used, but volume may be decreased and success rate increased with US guidance or a two injection technique [7].

Landmarks

- Vastus lateralis muscle.
- Biceps femoris muscle.
- Head of fibula.
- Superior border of patella.

Identify the vastus lateralis and biceps femoris muscles (Fig. 13.9a). Draw a line demarcating the space between the two muscles. Identify the popliteal crease. The insertion point is 7–10 cm cephalad to the popliteal crease along the original line (Fig. 13.9b). A cephalad insertion point increases the likelihood that the peroneal and tibial components are in close proximity and is more likely to result in a successful block [6, 8, 9].

Alternative Approach

Identify the head of the fibula. Draw a line cephalad, parallel to the floor/bed. Identify the superior border of the patella and draw a line perpendicular to the floor. The intersection of these lines marks the insertion point. Insert the needle perpendicular to the skin and parallel to the floor. First, the more lateral peroneal nerve will be stimulated. Note this depth of needle insertion and advance the needle until a tibial response is illicited after depositing two thirds of the anesthetic around the tibial nerve, withdraw the needle to the peroneal depth noted earlier, and deposit the remaining local anesthetic [10].

Ultrasound

Scan over the popliteal fossa and identify the popliteal artery (deep) and tibia nerve (superficial). Follow the nerve cephalad and slightly lateral until a second nerve (peroneal nerve) is observed to join the first. The nerve block should be performed where the two nerves are in close proximity (Fig. 13.10) or appear to combine. The needle is inserted in the lateral thigh approximately 2 cm deep to the skin so that the needle is parallel to the probe.

Tips

- If the femoral shaft is contacted, withdraw the needle to the skin and redirect 30° posteriorly/dorsal to the initial insertion plane. The nerve should be 1–1.5 cm deeper than the distance to the femur.

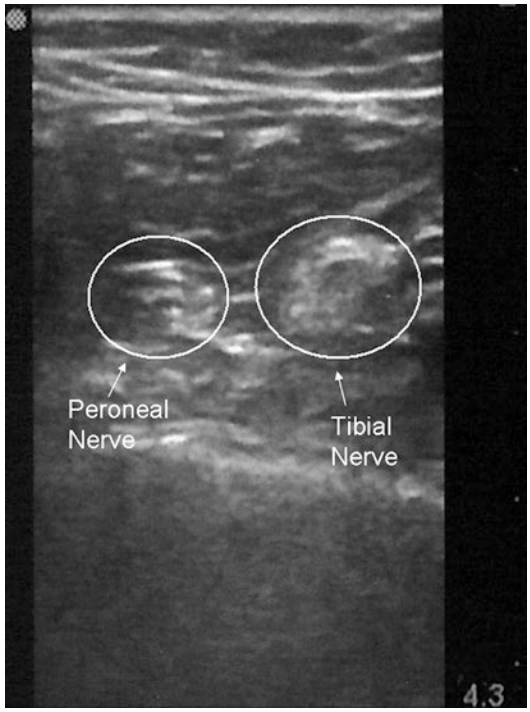

Fig. 13.10 Ultrasound-guided popliteal approach. The common peroneal nerve (lateral) and tibial nerve (medial) are identified in close approximation (*left* is lateral and *right* is medial)

Prone Approaches

When not contraindicated by fractures, the prone position allows full access to the target area. Place the patient prone with a folded blanket or pillow below the lower leg of the operative lower extremity. The operative lower extremity should be slightly bent at the knee, the lower leg supported, and the foot should rests freely above the bed.

1. Popliteal Prone Approach

   Landmarks
   - Semimembranosus muscle (medial).
   - Biceps femoris muscle (lateral).
   - Popliteal crease (inferior).

   Identify and mark the medial, lateral, and inferior borders of the popliteal triangle. Needle insertion should be 7–10 cm cephalad and 1 cm lateral to the midline of the popliteal crease (Fig. 13.11). Insert the needle in a cephalad direction at a 45–60° angle to the skin.

   Ultrasound

   Start by scanning the patient at the popliteal crease and identify the popliteal artery and tibial nerve (superficial to the artery). Trace the tibial nerve proximally until the peroneal structures are visualized in close approximation (see Fig. 13.10). Insert the needle medial or lateral to the probe.

   Tips
   - Unable to visualize landmarks: have the patient bend the knee against resistance.
   - No nerve stimulation with needle insertion: redirect the needle tip 1 cm lateral to the initial insertion site.
   - Stimulation of biceps femoris muscle: redirect the needle tip slightly medial. (Note that more medial insertion increases the risk of inadvertent vascular puncture).

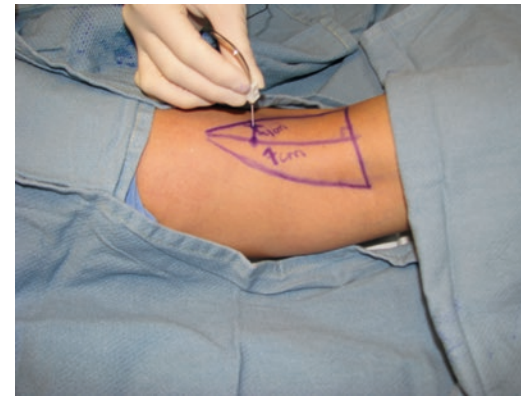

Fig. 13.11 Prone popliteal approach. Semimembranosus muscle (medial), biceps femoris muscle (lateral), and popliteal crease (inferior) are marked. The insertion point is 7 cm cephalad and 1 cm lateral

Lumbar Plexus

Introduction and Anatomy

The lumbar plexus originates from the ventral rami of L1–4 (variable contributions from T12 and L5), forms within the body of the psoas muscle, and supplies the lower abdomen and upper

leg. It consists of six peripheral nerves: femoral, obturator, lateral femoral cutaneous, ilioinguinal, genitofemoral, and iliohypogastric nerves. Consequently, a lumbar plexus block consistently blocks the three nerves supplying the lower extremity (femoral, obturator, and lateral femoral cutaneous nerves). The lumbar plexus block remains controversial because of the deep location of the plexus within the psoas muscle and the potential for bleeding into the retroperitoneum, a noncompressible area. Contacting and identifying the L4 transverse process before entering the plexus is very important in a lumbar plexus bock. This landmark serves as a needle depth safety point that should prevent advancing the needle too deep into the retroperitoneum [11–14].

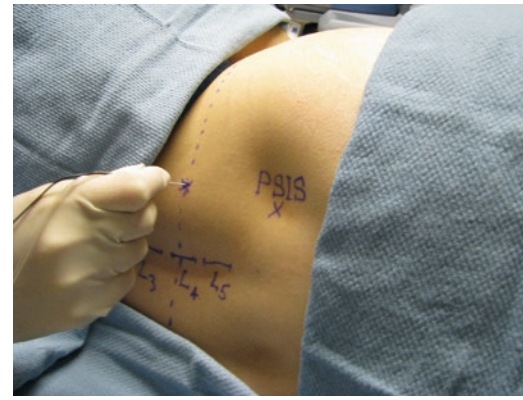

Fig. 13.12 Lumbar plexus nerve block. The intercristal line (*dashed line*) is identified using the posterior iliac crest. The needle insertion site is 4–5 cm from midline and is approximately as lateral as the patient's posterior superior iliac spine (PSIS)

Procedure

Needle: 10 cm insulated needle (15 cm needle may be needed for obese patients).

Position: Place the patient to a lateral position (Sim's position) with the nonoperative down, operative side up.

Landmarks

- Posterior iliac crest.
- Spinous processes (midline).

Draw a line from the top of the posterior iliac crest to midline. This line (intercristal line) is positioned over the L4 transverse process. Mark 4–5 cm lateral to midline on the operative side (approximately as lateral as the PSIS from the midline) on the intercristal line (Fig. 13.12).

Insert the block needle perpendicular to all planes with the bevel pointed superior or lateral and monitor for a quadriceps muscle twitch (patellar snap). This twitch indicates stimulation of the femoral nerve and proximity to the lumbar plexus. If the L4 transverse process is not contacted 5–6 cm deep to the skin and stimulation of the quadriceps has not been noted, make small adjustments cephalad or caudad in attempt to contact the transverse process. The needle may need to be advanced further in more obese patients. Once os has been contacted and the L4 transverse process identified, withdraw the needle 1–2 cm and redirect the tip 15° cephalad/caudal with the goal of placing the tip just superior/inferior to the L4 transverse process. The plexus should be stimulated 2 cm or less beyond the transverse process. Make sure to check for CSF as well as blood when aspirating before injection [11–14].

Alternative: Loss of Resistance Technique

Contact the transverse process as above. Flush the needle with 1 mL (remove possible tissue plug) and attach loss of resistance (LOR) syringe. Advance needle checking for LOR. (This is more subtle than the typical LOR found with epidurals). This is a fascial plane block (inserts local anesthetic between the quadratus lumborum and psoas muscles) and is volume dependent. Consequently, catheters may need increased rates.

Tips

- Unable to find transverse process: remove needle and reinsert 4 cm lateral to midline but perpendicular to all planes. (Do not deviate

the needle tip medially as this will increase the risk of epidural or subarachnoid block).
- No stimulation with repeated attempts: change to loss or resistance technique.
- Stimulation of hamstrings indicates stimulation of the sacral plexus (do not accept this twitch). Needle is too caudal and/or medial. Redirect lateral and/or cephalad. (Injection with stimulation of the sciatic nerve may increase the risk of retrograde spread of the local anesthetic into the epidural space [12]).
- Stimulation of adductors indicates obturator nerve stimulation. The needle is likely too medial and should be directed laterally.
- Os is repeatedly encountered: needle is likely hitting the posterior pelvis and has been inserted too caudad at L5 level or lower. Remove needle, recheck landmarks (likely reinsert needle at more cephalad location).
- Always insert with needle tip lateral or cephalad (never medial). This will decrease the chance of diffusion into the epidural space and decrease the incidence of epidural catheter placement with continuous techniques.
- Place the dressing as far lateral as possible when placing a lumbar plexus catheter. This will leave the midline uncovered and allow for a subarachnoid block if so desired for surgical anesthesia.

Complications

Complications specific to lumbar plexus blocks include epidural injection or diffusion (most common complication), intrathecal injections, intravascular injection, and retroperitoneal bleeding (especially in patients on therapeutic anticoagulants).

Femoral Nerve Block

Introduction and Anatomy

Derived from L2–4, the femoral nerve is the largest branch of the lumbar plexus. After traveling through the psoas muscle, the nerve passes anterior to the iliopsoas, under the inguinal ligament, and becomes superficial in the anterior thigh deep to the fascia lata and fascia iliacus. Here, it is separated from the femoral artery and vein by the fascia iliacus.

Femoral nerve blockade provides analgesia to the anterior thigh, anterior knee and medial leg, ankle, and foot. A femoral nerve block has been referred to as the "3-in-1" block inferring that the femoral, lateral femoral cutaneous, and obturator nerves could all be blocked by one injection [15, 16]. However, studies have demonstrated that the obturator nerve is often missed with this technique [17–20].

Procedure

Needle: 5 cm insulated needle.

Position: Position the patient supine. A pillow may be placed under the head, if needed. Retract the patient's pannus with tape as needed (do not cover the insertion site).

Landmarks

- Inguinal ligament.
- Pubic tubercle.
- Anterior superior iliac spine.
- Femoral artery.

Identify the inguinal ligament by drawing a line from the pubic tubercle to the anterior superior iliac spine. Palpate the femoral artery just distal to the ligament (Fig. 13.13). Insert the needle 1–1.5 cm lateral to the femoral artery. (Remember that the nerve is lateral to the artery with the mnemonic NAVELS: Nerve, Artery, Vein, Empty space, Lymphatics, and Symphysis). Insert the block needle in a cephalad direction at a 45–60° angle to the skin and monitor for an evoked response of the rectus femoris (patellar snap).

Ultrasound

Identify the femoral vessels (medial) and femoral nerve (lateral; Fig. 13.14). Clinicians may insert

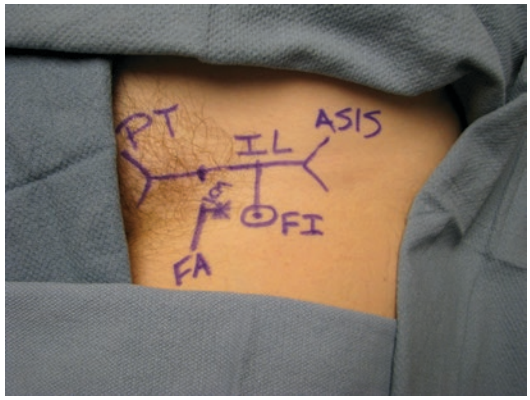

Fig. 13.13 Femoral nerve block. A *line* connects the pubic tubercle (PT) to the anterior superior iliac spine (ASIS) and identifies the inguinal ligament (IL). The femoral artery (FA) is palpated and marked. The needle insertion site is 1 cm lateral FA

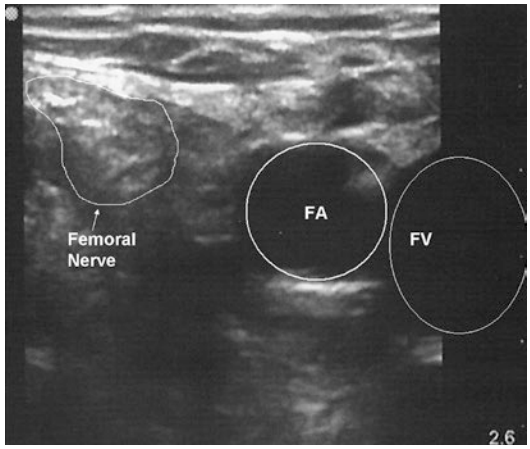

Fig. 13.14 Ultrasound-guided femoral nerve block. Lateral to medial, the femoral nerve (FN), artery (FA), and vein (FV) are identified (*left* is lateral and *right* is medial)

the needle for in-plane (preferred by author) or out-of-plane approach.

Tips

- Stimulation of the medial thigh occurs: redirect tip laterally. [Do not accept this evoked motor response (likely a superficial branch or direct vastus medialis or sartorius muscle stimulation)].
- Do not direct your tip medially; you will increase the risk of vascular puncture. If you think you are too lateral, reinsert the tip 0.5 cm medially but maintain the cephalad direction with insertion.
- You may note two "pops" with needle insertion. This is due to penetration of fascia lata (superficial) and fascia iliaca.

Alternative: Fascia Iliaca Block

This technique is useful for patients on anticoagulation, patients with a recent vascular study or cardiac catheterization in the groin of the operative side, or patients with intense pain negating nerve stimulation. Needle placement is farther from the vessels and nerves are not stimulated.

Identify the above landmarks except the femoral artery. Divide line on inguinal ligament into thirds. At the lateral mark (between lateral and middle thirds), draw a 1- to 2-cm line perpendicular to the original line (Fig. 13.15). Insert the needle at the bottom of this second line perpendicular to all planes. Feel for two "pops": fascia lata and fascia iliacus. The neural structures are present in this plan. Insert local anesthetic.

Contraindications

A prosthetic femoral artery graft is a relative contraindication to femoral nerve block since it is

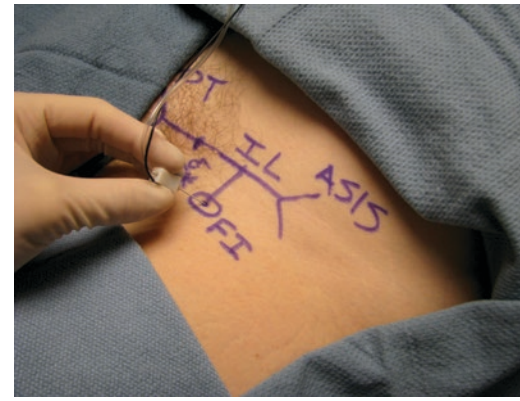

Fig. 13.15 Fascia iliaca block. The inguinal ligament is identified by drawing a line from the pubic tubercle to the anterior superior iliac spine. This line is divided into thirds. At the lateral mark (between lateral and middle thirds), a 1–2-cm line is drawn perpendicular to the original line. This marks the needle insertion site

difficult to know the orientation of the femoral nerve and native vessel to the palpable vascular graft. An ultrasound should be utilized in these patients in order to fully evaluate the altered vascular anatomy.

Adductor Canal Block

Introduction and Anatomy

In an effort to minimize the quadriceps weakness associated with the femoral nerve block, the adductor canal block has gained popularity as a novel approach for primarily sensory analgesia to the anterior knee [21–23]. The motor sparing of the quadriceps muscles allow for improved physical therapy participation and maintenance of balance with equivalent postoperative analgesia. Although this technique primarily blocks the saphenous nerve, it also anesthetizes the nerve to the vastus medialis and may still have minor associated motor blockade.

Procedure

Needle: 5–10 cm insulated needle.

Position: Position the patient supine with the leg to be blocked bent and externally rotated. A pillow may be placed under the knee of the blocked leg for support, if needed.

Landmarks

- Middle third of the thigh.

Ultrasound

Place the probe over the ventral surface of the mid-thigh (halfway between the patella and anterior superior iliac crest). Identify the femur (deep) and vastus medialis muscle (superficial). Scan medially and observe the sartorius muscle with branches of the femoral vessels just deep to the sartorius muscle. The adductor canal is formed by the vastus medialis muscle (lateral), sartorius muscle (anterior), and adductor muscles (adductor magnus and brevis; posterior-medial; Fig. 13.16).

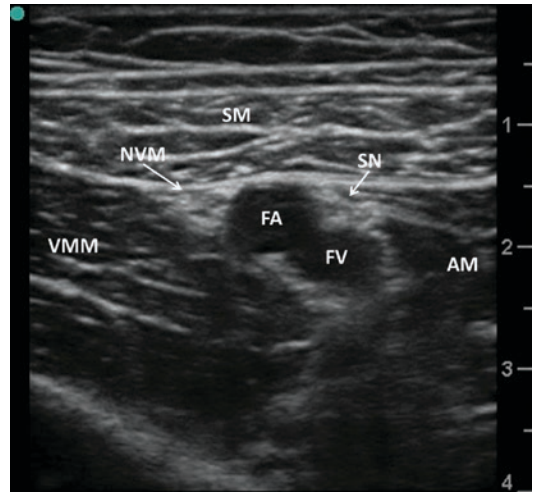

Fig. 13.16 Ultrasound-guided adductor canal block. The adductor canal is formed by the vastus medialis muscle (VMM; lateral), sartorius muscle (SM; anterior), and adductor muscles (AM; posterior-medial). The saphenous nerve (SN), nerve to vastus medialis (NVM) and branches of the femoral artery and vein (FA, FV) are visualized

Clinicians may insert the needle for in-plane (preferred by author) or out-of-plane approach.

Tips

- Although the saphenous nerve and nerve to vastus medialis are often visible near the branches of the femoral artery and vein, simply injecting local anesthetic in the adductor canal below the sartotius muscle will provide an adequate block.
- If you have difficulty identifying the vessels below the sartorius muscle, use color doppler.
- If planning to place a catheter for a knee procedure, discuss with the surgeon. It may be necessary to place the catheter more proximally to avoid placement in the surgical field.

Quadratus Lumborum Block

Introduction and Anatomy

First described, as ultrasound-guided abdominal wall block with injection of local anesthetic close

to anterior-lateral border of quadratus lumborum muscle, a quadratus lumborum block (QLB) provides a sensory block in T7-L1 distribution. It is believed that spread of local anesthetic to the thoracic paravertebral space is responsible for blockade of anterior and lateral branches of T7-L2, providing somatic and visceral analgesia [24].

Procedure

Needle: 8–10 cm 22G Tuohy (18G Tuohy with 20G catheter)

Position: supine, supine with a roll under the blocking flank or lateral

Landmarks:

- Mid-axillary line between the rib cage and iliac crest

Ultrasound

Place the curved array transducer (2–6 MHz) in transverse plane between the rib and iliac crest, halfway between the umbilicus and mid-axillary line to identify all three abdominal wall muscles (external oblique, internal oblique, and transverse abdominal). Scan laterally and observe transverse abdominal muscle (TAM) tapering into the aponeurosis with lateral extension into quadratus lumborum muscle (QLM). Visualize the peritoneal cavity with moving loops of bowel separated from QLM by a "pocket" of retroperitoneal fat.

The needle is inserted in-plane to the transducer (medial to lateral) and the tip is advanced through all three layers of abdominal wall muscles pointing towards transverse abdominis aponeurosis and lateral portion of the QLM. Penetration of aponeurosis feels like "a pop" and gives a clear pattern of LA spread under the aponeurosis in posterior direction on both sides of QL muscle (Fig. 13.17).

Two types of QLB have been described depending on injection end point. In a Type I (anterior) approach, the needle penetrates the aponeurosis of TAM and spread on both sides of QLM. In a Type II (lateral) approach, the needle is aimed more lateral (Fig. 13.18).

Tips

- Medial to lateral needle direction with bevel pointed laterally is preferable to avoid intraperitoneal organs injury.
- Although depth to the target usually is less than 4 cm, a curved array transducer provides

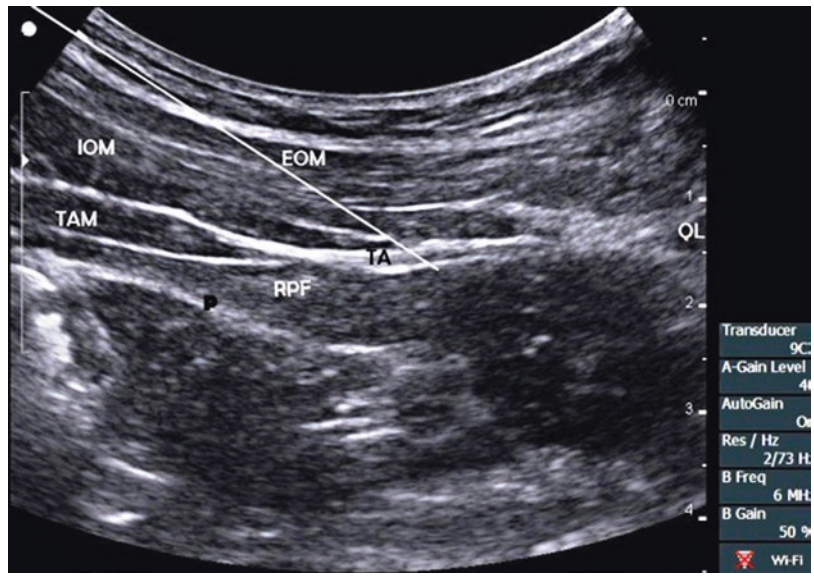

Fig. 13.17 Ultrasound-guided Quadratus Lumborum Block (dot is medial). The External Oblique Muscle (EOM), Internal Oblique Muscle (IOM), Transverse Abdominis Muscle (TAM), Aponeurosis of TAM (TA), Quadratus Lumborum Muscle (QL), and retroperitoneal fat (RPF) are visualized. The correct needle position is demonstrated by the white line with a medial to lateral insertion

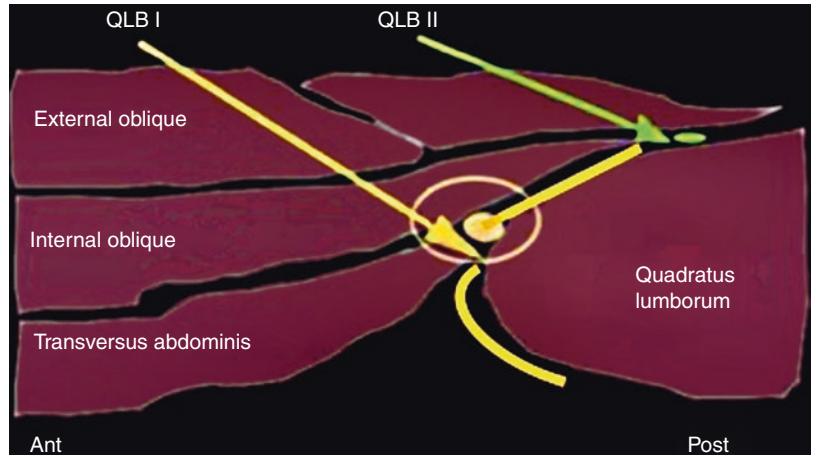

Fig. 13.18 Anatomy and Injection Sites for Quadratus Lumborum Block Type I and II (QLM I and QLM II). Yellow lines demonstrate pattern of local anesthetic spread around the quadratus lumborum muscle

better in-plane needle tip visualization, especially through aponeurosis and TAM.

- This is a fascial plane block and high volume of local anesthetic is required to achieve sufficient spread (20–30 mL; 0.2–0.5% Ropivacaine). In our experience, additives such as dexmedetomidine, dexamethasone, and epinephrine improve quality of block and prolong analgesia up to 48 h.
- QLB provides excellent analgesia for hip surgeries (THA, hemiarthroplasty, ORIF) with no detectable motor block in hip flexors (iliopsoas muscle) and quadriceps femoris, making this block almost ideal for early rehabilitation.

Review Questions

1. While performing a sciatic nerve block with nerve stimulation, you observe foot dorsiflexion. What part of the sciatic nerve are you stimulating?
2. Which sciatic nerve block(s) will also block the posterior femoral cutaneous nerve of the thigh?
3. Which sciatic nerve block may also block the obturator nerve?
4. While performing a popliteal nerve block from the lateral approach, you contact the femur. How should you redirect your needle?
5. What muscle contraction should be observed with stimulation of the lumbar plexus?
6. You are having difficulty placing a lumbar plexus block and change to a loss or resistance technique for a psoas compartment block. After you have found the transverse process, what should you do before attaching the loss of resistance syringe?
7. What nerve roots does the femoral nerve originate from?
8. Where is the femoral nerve anatomically located in relation to the femoral artery?
9. In a fascia iliaca block, what two fascial layers cause the two "pops"?
10. What is the number one contraindication to performing a nerve block?

Answers

1. Common peroneal
2. Labat and parasacral
3. Parasacral
4. 30° Posterior/dorsal
5. Quadriceps (patellar "snap" due to femoral nerve stimulation)
6. Flush the needle with 1 mL of solution to clear any plug at the tip
7. Lumbar roots 2–4
8. Nerve is lateral to artery (NAVELS)
9. Fascia lata (superficial) and fascia iliaca (deep)
10. Patient refusal

References

1. Hagon BS, Itani O, Bidgoli JH, et al. Parasacral sciatic nerve block: does the elicited motor response predict the success rate? Anesth Analg. 2007;150:163–266.
2. Taboada M, Atanassoff PG, Rodríguez J, et al. Plantar flexion seems more reliable than dorsiflexion with Labat's sciatic nerve block: a prospective, randomized comparison. Anesth Analg. 2005;100(1):250–4.
3. Labat G. Its technique and unique applications. In: Regional anaesthesia. 2nd ed. Philadelphia: WB Saunders; 1924. p. 45–55.
4. Morris GF, Lang SA, Dust WN, Van der Wahl M. The parasacral sciatic nerve block. Reg Anesth. 1997;22:223–8.
5. Chelly JE, Delaunay L. A new anterior approach to the sciatic nerve block. Anesthesiology. 1999;91:1655–60.
6. Vloka JD, Hadzic A, April E, Thys DM. The division of the sciatic nerve in the popliteal fossa: anatomical implication for popliteal nevre blockade. Anesth Analg. 2001;92:215–7.
7. Perlass A, Brull R, Chan VWS, et al. Ultrasound guidance improves the success of sciatic nerve block in the popliteal fossa. Reg Anesth Pain Med. 2008;33:259–65.
8. Pacqueron X, Bouaziz H, Macalou D, et al. The lateral approach to the sciatic nerve at the popliteal fossa: one or two injection? Anesth Analg. 1999;89:1221–5.
9. Vloka JD, Hadzic A, Singson R, et al. The popliteal nerve block revisited: results of an MRI study. Anesth Analg. 1997;84:S344.
10. O'Neill T. Lateral popliteal sciatic-nerve block made easy. Reg Anesth Pain Med. 2007;32:93–4.
11. Parkinson SK, Mueller JB, Little WL, Bailey SL. Extent of blockade with various approaches to the lumbar plexus. Anesth Analg. 1989;68:243–8.
12. Turker G, Uckunkaya N, Yavascaglu B, et al. Comparision of catheter technique psoas compartment block and the epidural block for analgesia in partial hip replacement surgery. Acta Anesthesiol Scand. 2003;47:30–6.
13. Capdevila X, Coimbra C, Choquet O. Approaches to the lumbar plexus: success, risks, and outcome. Reg Anesth Pain Med. 2005;30:150–62.
14. Macaire P, Dadure C, Choquet O, et al. Continuous psoas compartment block for postoperative analgesia after total hip arthroplasty: new landmarks, technical guidelines, and clinical evaluation. Anesth Analg. 2002;94:1606–13.
15. Khoo ST, Brown TCK. Femoral nerve block—the anatomical basis for a single injection technique. Anaesth Intensive Care. 1983;11:40–2.
16. Winnie AP, Ramamurthy S, Durrani Z. The inguinal paravascular technique of lumbar plexus anesthesia: the "3-in-1 block". Anesth Analg. 1973;52(6):989–96.
17. Lang SA, Yip RW, Chang PC, Gerard MA. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5(4):292–6.
18. Seeberger MD, Urwyler A. Paravascular lumbar plexus block: block extension after femoral nerve stimulation and injection of 20 vs. 40 ml mepivacaine 10 mg/ml. Acta Anaesthesiol Scand. 1995;39(6):769–73.
19. Marhofer P, Nasel C, Sitzwohl C, Kapral S. Magnetic resonance imaging of the distribution of local anesthetic during the three-in-one block. Anesth Analg. 2000;90:119–24.
20. Atanassoff PG, Weiss BM, Brull SJ, et al. Electromyographic comparison of obturator nerve block to three-in-one block. Anesth Analg. 1995;81:529–33.
21. Jæger P, Zaric D, Fomsgaard JS, Hilsted KL, Bjerregaard J, Gyrn J, Mathiesen O, Larsen TK, Dahl JB. Adductor canal block versus femoral nerve block for analgesia after total knee arthroplasty: a randomized, double-blind study. Reg Anesth Pain Med. 2013;38(6):526–32.
22. Kim DH, Lin Y, Goytizolo EA, Kahn RL, Maalouf DB, Manohar A, Patt ML, Goon AK, Lee YY, Ma Y, YaDeau JT. Adductor canal block versus femoral nerve block for total knee arthroplasty a prospective, randomized, controlled trial. Anesthesiology. 2014;120(3):540–50.
23. Kwofie MK, Shastri UD, Gadsden JC, Sinha SK, Abrams JH, Xu D, Salviz EA. The effects of ultrasound-guided adductor canal block versus femoral nerve block on quadriceps strength and fall risk: a blinded, randomized trial of volunteers. Reg Anesth Pain Med. 2013;38(4):321–5.
24. Dam M, Moriggl B, Hansen CK, Hoermann R, Bendtsen TF, Børglum J. The pathway of injectate spread with the transmuscular quadratus lumborum block: a cadaver study. Anesth Analg. 2017;125(1):303–312.

Regional and Topical Anesthesia for Endotracheal Intubation

14

Ryan Philip Ellender, Shilpadevi S. Patil, Lien Tran, Scott M. Kleinpeter, Elyse M. Cornett, and Alan David Kaye

Introduction

Management of difficult airways is a crucial patient safety issue. The incidence of difficult intubation in patients without pathologic airway anatomy was 5.8% in a meta-analysis study. The incidence is higher in patients with abnormal anatomic features of head, neck, or airway [1]. Difficult tracheal intubation, if not managed appropriately, can cause severe patient morbidity and mortality in the form of brain damage and death as documented in several closed claim analysis, national audits, and patient complaints. Each unsuccessful attempt at intubation not only compromises the patient but also decreases the chances of successful intubation with fiber-optic scope. Therefore, it is imperative to have the knowledge and skills necessary to manage a difficult airway in a simple, safe, and reliable way [2, 3].

Despite recent advancements in video and digital laryngoscopes, awake intubation continues to be an important modality for anesthesiologists to secure the airway. Awake intubation (AI) can be performed for a variety of conditions that result in a difficult airway. Congenital anomalies involving the head and neck, tumors of the airway, morbid obesity, cervical spine pathology, patients with a history of difficult intubation, and trauma associated with facial and cervical instability all make direct laryngoscopy challenging and can be indications for awake intubation. During the airway exam, limited mouth opening, limited thyromental distance, reduced neck mobility, inability to prognath, and higher mallampatti scores are some of the predictors for difficult airway that in turn may necessitate awake intubation [1].

On the other hand, contraindications to regional and topical anesthesia of the airway include patient refusal, coagulopathy, infection at the blockade site, allergy to local anesthetics, and

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_14

a patient in whom the loss of protective airway reflexes would be unsafe.

There are many benefits to performing a fiber-optic intubation in an awake as opposed to an anesthetized or apneic patient. The awake patient can manipulate the tongue and swallow secretions, thereby clearing the airway. An awake patient can also breathe spontaneously, allowing the anesthesiologist more time to secure the airway. Furthermore, the patient can phonate, which can assist in visualization of the glottic opening and vocal cords. The largest hurdle to awake intubation is often patient cooperation. Although drugs such as midazolam, fentanyl, remifentanil, dexmedetomidine, ketamine, and propofol can be used as useful adjuncts to provide sedation while performing awake intubation, excessive salivation, intact reflexes, and patient discomfort can lead to failure of AI. This can be overcome by adequate anesthesia of airway structures with topical and regional anesthetic techniques, often in addition to sedation.

Common Indications for Awake Fiber-optic Intubation [1]

Known difficult intubation

Suspected difficult intubation by direct laryngoscopy (e.g., history of difficult intubation, limited mouth opening, decreased neck mobility, decreased thyromental distance)

TMJ ankylosis

Oral submucous fibrosis

Unstable cervical spine or cervical spine pathology (e.g., rheumatoid arthritis)

Abnormal anatomy of the airway

- Congenital airway deformities (e.g., Pierre Robin syndrome)
- Head and neck cancers (e.g., supraglottic tumors)
- Goiter

Trauma
- Face/neck
- Airway trauma

Tumors
- Head and neck
- Airway tumors

Difficult mask ventilation

High risk of aspiration

Head and neck Scarring and contractures
- Radiation
- Burns

Infection
- Submandibular abscess
- Parapharyngeal access

Relevant Airway Anatomy

The anatomy of the upper airway and, in particular, its pattern of sensory innervation of the component subdivisions of the upper airway must be considered when performing an awake oral or nasal intubation. It is important that the physician can locate the nerves based on anatomical landmarks to deliver adequate anesthesia to the patient.

The upper airway includes the nasal cavity, oral cavity, and the three subdivisions of the pharynx: the nasopharynx, the oral pharynx, and the laryngopharynx (hypopharynx). These subdivisions of the upper airway are innervated primarily by branches of the trigeminal (CN V), glossopharyngeal (CN IX), and vagus (CN X) cranial nerves, respectively (Fig. 14.1).

The trigeminal nerve (CN V) has three divisions, the ophthalmic division (CN V1), the maxillary (CN V2), and mandibular division (CN V3), each of which is involved in supplying sensory innervation to different parts of the upper airway.

The anterior aspect of the nasal cavity is supplied by the anterior ethmoidal nerve, a branch of the nasociliary nerve from the ophthalmic division of the trigeminal nerve (CN V1). It subdivides into lateral internal nasal branches to the anterior aspect of the lateral wall of the nasal cavity and medial internal nasal branches to anterior superior parts of the nasal septum. After giving off these branches, this nerve continues as the

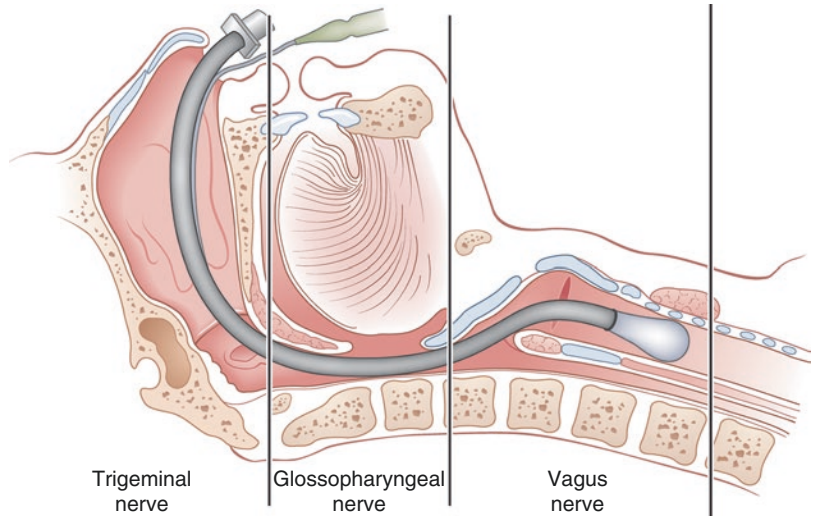

Fig. 14.1 Simplified innervation of airway structures. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

external nasal nerve which exits the nasal cavity between the nasal bone and nasal cartilages to provide sensory innervation to the skin on the external surface to the tip of the nose inferior to the nasal bones and superior part of the nares.

The infraorbital nerve, the terminal branch of the maxillary division of the trigeminal nerve (CN V2), gives rise to internal nasal branches which supply sensory innervation to the inferior part of the nares, the skin on the side of the nose, the nasal vestibule, and the anterior movable part of the nasal septum at which point it interdigitates with terminal branches of the anterior ethmoidal nerve. Branches of the anterior superior alveolar nerves from the infraorbital nerve supply the inferior part of the nasal septum, the floor of the nasal cavity near the anterior nasal spine, and the anterior one-fourth of the lateral nasal wall up to the level of the opening of the maxillary sinus. The posterior three-fourths of the lateral nasal wall, roof, and floor receive sensory innervation from the lateral posterior superior nasal nerves (to the mucous membrane covering the superior and middle concha) and posterior inferior nasal nerves which are branches coursing anteriorly from the greater palatine nerve as it courses inferiorly from the pterygopalatine ganglion. The pterygopalatine ganglion is located within the pterygopalatine fossa in line with the middle turbinate. The posterior and inferior parts of the nasal septum are supplied by the medial posterior superior nasal nerves, which branch from the pterygopalatine ganglion and enter the nasal cavity through the sphenopalatine foramen. One of the large medial posterior superior nasal nerves is called the nasopalatine nerve which courses from posterior superior to anterior inferior between the periosteum and the mucosa of the nasal septum. The terminal part of the nasopalatine nerve courses through the incisive canal in the anterior midline of the hard palate to provide sensory innervation to the most anterior part of the hard palate within the oral cavity. The nerve of the pterygoid canal (Vidian nerve) supplies the most posterior and superior part of the nasal septum (Fig. 14.2).

The nasal cavity communicates posteriorly with the nasopharynx by means of the posterior nasal choanae. Sensory innervation to the nasopharynx posterior to the opening of the pharyngotympanic tube is mediated primarily by the pharyngeal nerve, a branch from the pterygopalatine ganglion that courses anteriorly through the palatovaginal or pharyngeal canal to enter the posterior aspect of the nasopharynx.

The oral cavity receives its sensory innervations from branches of the trigeminal, facial, and glossopharyngeal nerves.

Sensory innervation of the tongue is provided by the lingual nerve, a branch of the mandibular division of the trigeminal nerve (CN V3) to the anterior two-thirds of the tongue and lingual

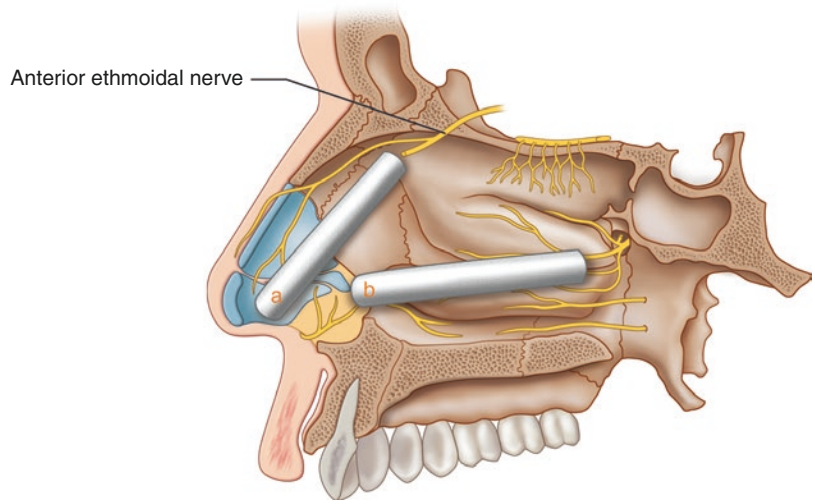

Fig. 14.2 Innervation of the right nasal cavity. (**a**) And (**b**) cotton pledgets can provide topical anesthesia to the nasal cavity. Adapted from Drake RL et al. *Gray's Anatomy for Students*, 2nd Ed, 2010

branches of the glossopharyngeal nerve (CN IX) to its posterior one-third. The glossopharyngeal nerve provides general sensory innervation of the posterior one-third of the tongue and also provides the sensation of taste from the same area.

The hard palate is innervated by the greater palatine nerve and nasopalatine nerve, both of which are branches of the maxillary division of the trigeminal nerve (CN V2). Its terminal fibers overlap the distribution of fibers from the nasopalatine nerve, which enters the oral cavity through the incisive canal and innervates the mucosa covering the most anterior part of the hard palate.

The soft palate receives its sensory innervation from the lesser palatine nerves. The lesser palatine nerves also descend through the greater palatine canal and course through the lesser palatine foramina on the posterior lateral aspect of the hard palate. They course posteriorly into the soft palate to innervate the mucosa of the soft palate, palatine tonsil, and uvula.

The oropharynx is continuous anteriorly with the oral cavity, is bounded superiorly by the soft palate, and extends inferiorly to the level of the upper border of the epiglottis. The oral cavity communicates with the oropharynx through the oropharyngeal isthmus, which is demarcated by the palatoglossal arch. The palatine tonsil and the palatopharyngeal arch (posterior tonsillar pillar) comprise its lateral boundary, while the pharyngeal aspect of the tongue is located anteriorly.

Cranial nerve VII (the facial nerve) and cranial nerve IX (the glossopharyngeal nerve) supply the sensory innervation to this part of the upper airway. The mucous membranes of the palate and nasopharynx, i.e., caudal parts of the oral cavity, have sensory afferent fibers that are in the facial nerve. The palatine tonsil receives not only sensory nerve branches from the lesser palatine branch of the maxillary division of the trigeminal nerve (CN V2) but also has sensory innervation from the glossopharyngeal nerve (CN IX), which is located just lateral to the palatine tonsil. The posterior tonsillar pillar or palatopharyngeal arch is innervated by a dense plexus formed by the pharyngeal branches of the cranial nerves IX and X. The pharyngeal mucosa from the inferior opening of the pharyngotympanic tube (Eustachian tube) to approximately the middle level of the aryepiglottic fold is supplied by glossopharyngeal sensory fibers. Sensory nerve fibers from the caudal pole of the palatine tonsil and root of the tongue have been reported to be very dense [4].

The glossopharyngeal nerve exits the jugular foramen anterior to the vagus and spinal accessory nerves and courses anteriorly and inferiorly between the internal carotid artery and the internal jugular vein posteromedial to the styloid process and its associated muscles. The nerve courses anteriorly between the internal carotid artery and external carotid artery crosses the stylopharyngeus

muscle and enters the pharynx between the superior and middle constrictor muscles. After entering the pharynx, it branches to supply the posterior one-third of the tongue and the mucous membrane of the palatine tonsil and pharynx. Pharyngeal branches of the glossopharyngeal nerve unite at approximately the middle constrictor muscle to contribute the sensory component to the pharyngeal plexus. Tonsillar branches supply sensory branches to the mucous membrane over the tonsil and soft palate. In the posterior aspect of the oral cavity, the glossopharyngeal nerve is located just lateral to the palatine tonsil and posterior tonsillar pillar and medial to the lateral pharyngeal space. It also has an intimate anatomical relationship to the significant anastomosing vascular supply to the palatine tonsil.

The laryngopharynx (hypopharynx) extends from the superior border of the epiglottis and glossoepiglottic folds to the inferior border of the cricoid cartilage. The piriform fossa is a small paired depression that lies medial to the laminae of the thyroid cartilage and lateral to either side of the laryngeal aditus, separated from the cavity of the larynx by the aryepiglottic folds.

The sensory innervation of the larynx and the laryngopharynx is mediated by branches of the vagus nerve (CN X). The vagus nerve exits the jugular foramen and descends within the carotid sheath between the internal jugular vein and the internal carotid artery. After giving off branches, which form the motor component of the pharyngeal plexus, the vagus nerve, at approximately the level of the C1–C2 vertebra, gives rise to the superior laryngeal nerve. The superior laryngeal nerve arises from the inferior ganglion of the vagus and descends along the lateral aspect of the pharynx, initially posterior and subsequently medial to the internal carotid artery. The superior laryngeal nerve subsequently divides into a small external laryngeal nerve and a larger internal laryngeal nerve.

The external branch of the superior laryngeal nerve is motor to the cricothyroid and the cricopharyngeus muscles and does not participate in the sensory innervation of the larynx or hypopharynx. The external laryngeal nerve lies between the pretracheal fascia and the inferior pharyngeal constrictor muscle. The external and internal branches of the superior laryngeal nerve are the only nerves coursing from lateral to medial at this level.

The internal branch of the superior laryngeal nerve carries afferent sensory innervation from the mucosa of the larynx, extending from the laryngeal surface of the epiglottis and back of the tongue inferiorly to the level of the vocal folds. It is also responsible for sensory innervation of the piriform fossa. The sensory innervation to the hypopharyngeal wall is dense [4]. The internal branch courses anteriorly from its origin crosses the greater cornu of the hyoid bone and pierces the thyrohyoid membrane superior to the superior laryngeal branch of the superior thyroid artery. After perforating the thyrohyoid membrane, the internal laryngeal nerve arborizes to innervate the laryngeal mucosa from the upper surface of the vocal folds to the base of the tongue.

The trunk of the internal laryngeal nerve enters the larynx through the thyrohyoid membrane and subdivides into three major branches. The anterior (superior) "epiglottic branch" supplies the mucosa on the lingual surface of the epiglottis and a small part of the anterior wall of the epiglottic vallecula. The middle "ventricular branch" supplies the aryepiglottic fold and courses caudally to innervate the mucosa of the ventricular fold. The posterior "post cricoid branch" courses directly to the piriform sinus and also sends a few small branches to the inter arytenoid muscles.

The density and laterality of the sensory innervation of the airway vary. The posterior tonsillar pillars, the laryngeal surface of the epiglottis, and the post cricoid and arytenoid regions have a high density of sensory innervation. Other areas such as the posterior part of the hypopharynx were reported to have little or no identifiable sensory nerve supply.

The sensory innervation of the mucosa of the larynx below the level of the vocal folds is from the right and left recurrent laryngeal nerves which also supply motor innervation to all of the intrinsic muscles of the larynx except the cricothyroid muscle. As a result of the embryologic

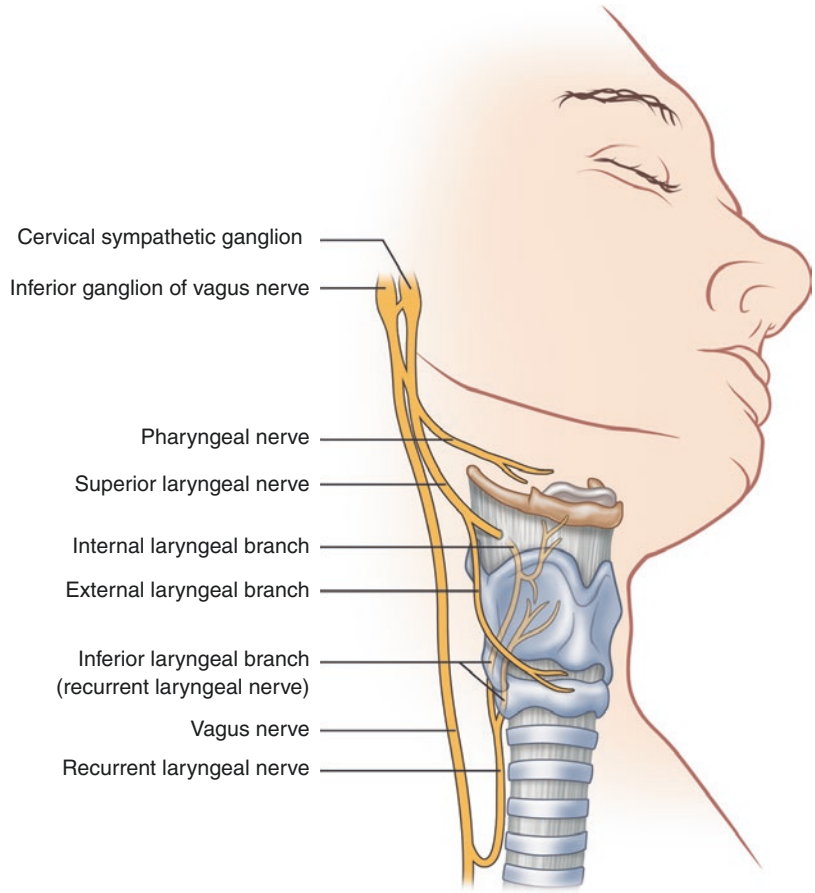

Fig. 14.3 Innervation of the larynx. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

development of the aortic arches, the right and left recurrent laryngeal nerves arise at different levels. The right recurrent laryngeal nerve arises anterior to the first part of the subclavian artery as the nerve winds around the vessel. The left recurrent laryngeal nerve arises on the left side of the arch of the aorta where it winds around this vessel posterior to the attachment of the ligamentum arteriosum.

The recurrent laryngeal nerve can be identified as it enters the larynx just posterior to the inferior cornu of the thyroid cartilage. In the lower portion of its course, the nerve can be palpated on the surface of the trachea. It courses superiorly in the groove between the trachea and the esophagus. Before entering the larynx, the nerve supplies branches for the cricopharyngeus muscle. Below the border of the inferior constrictor, the recurrent laryngeal nerve divides into an anterior branch, which is mainly motor (sometimes referred to as the inferior laryngeal nerve) and a posterior branch, which is mainly sensory. The sensory branch supplies the mucosa of the larynx inferior to the vocal cords and communicates with branches of the internal laryngeal nerve (Fig. 14.3).

Preparation

Preparation of the patient is the key for safe and successful awake intubation. After reviewing the clinical issues and prior anesthetic records the first step in preparation for airway manipulation in a conscious patient is education. The anesthesiologist needs to explain to the patient the reasons behind the decision to perform an awake intubation, as well as the steps involved.

The patient needs to understand that his or her cooperation is necessary to facilitate the process. A calm patient can be intubated much easier than a restless one.

The individual performing the fiber-optic intubation is the airway manager. As communication is an important part of the preparation, he or she should also inform the other individuals of the operating room team involved with the intubation plan. This includes notifying a surgeon, who would be available to perform an emergent cricothyroidotomy, if necessary.

Next, an important step is checking the proper functioning of necessary equipment such as fiber-optic bronchoscope, video laryngoscope, necessary laryngoscope blades and handles, intubating airways or supraglottic devices, increasingly sized nasopharyngeal airways, laryngeal mask airway(LMA), and suction system.

Lastly, appropriate monitors should be applied to the patient. A nasal canula can be used to supplement oxygenation during oral fiber-optic intubation. Likewise, a specialized face mask that can deliver oxygen and permit mask ventilation during fiber-optic intubation can be used or a conventional face mask placed inferiorly to cover the mouth and chin can be used to supplement oxygenation during nasal fiber-optic intubation [5]. Either method should allow end-tidal CO_2 monitoring. Oxygen saturation and blood pressure should also be monitored. Electrocardiogram leads should be attached, and intravenous access should be obtained [6].

Appropriate premedication and medications necessary for the intubation as described below have to be kept ready for use.

Premedication

Antisialagogue should be administered early before airway instrumentation in an awake patient to minimize secretions. By doing so, antisialagogues improve not only the fiber-optic view but also the ability of local anesthetics to reach the airway mucosa. Intravenously administered glycopyrrolate is the preferred agent. Atropine i.v. or i.m. is an alternative. Intramuscular administration of atropine is favored over intravenous injection to avoid tachycardia and, less commonly, psychosis [6]. Scopolamine can also be given; however, it may cause delirium, especially in older patients [5].

Topical vasoconstrictors should also be administered before performing a nasotracheal intubation. This will help to decrease bleeding and open the airways, as topical anesthesia tends to reduce the caliber of a normal airway [7]. The nasal passages can be accessed for patency by asking the patient to note the air movement from each nostril after the contralateral side is compressed. Each nostril can also be examined fiber-optically to identify pathology as well as access the degree of patency [8]. After choosing the nasal passage, deposit at least two sprays of 1% phenylephrine or 0.05% oxymetazoline into each nostril [5].

Although no sedation approach can be used, most patients experience some degree of anxiety associated with an awake intubation; therefore, administration of sedatives and hypnotics may be appropriate [9]. However, it is important to remember that sedation cannot compensate for inadequate topical anesthesia and can be dangerous in a patient with a critical airway [7]. Short-acting and intravenous medications are ideal for this process. Midazolam and fentanyl are commonly used, as they are easy to titrate and reverse. Dexmedetomidine can be used and is suitable for this setting because it does not cause significant respiratory depression [10]. Propofol and ketamine can also be used as a last resort when comfortable sedation cannot be achieved (Table 14.1).

Approach to Anesthesia of the Airway

Once the decision has been made to proceed with an awake tracheal intubation, the next step is to decide the route of intubation whether oral or nasal. This depends on the mouth opening and the site of surgery. In patients with severe trismus, a large tongue or if the surgical site is the mouth then the nasal approach is preferred. Nasal route is anatomically favorable as the laryngeal opening is

Table 14.1 Commonly used medications for awake intubation

Medication	Dosage and route	Class	Side effects
Glycopyrrolate	3–6 μg/kg IV,IM	Antisialogogue	Slight tachycardia
Atropine	7–10 μg/kg IV,IM	Antisialogogue	Tachycardia, psycosis
Scopolamine	0.3–0.6 mg IV,IM,SC	Antisialogogue	Sedation, delirium
Midazolam	20–40 μg/kg IV, IM	Benzodiazepine	Delayed awakening
Fentanyl	1–2 μg/kg IV	Opioid	Respiratory depression
Dexmedetomidine	Loading dose: 1 μg/kg over 10 min	α_2-Agonist	Bradycardia, hypotension
	Infusion: 0.2–0.7 μg/kg/h		
Propofol	0.25 mg/kg IV	Hypnotic	Cardiovascular depression
Ketamine	0.2–0.5 mg/kg	Hypnotic	Increased secretions
Remifentanil	Loading dose:0.75 μg/kg	Opioid	Respiratory depression
	Infusion: 0.075–0.15 μg/kg/min		

easily seen by fibreoptic scope as it courses past the nasopharynx with less obstruction by the tongue. Communication with the surgical team is extremely important to proceed with the preferred route. In the case of using the nasal route, the patients are asked to compare their nasal airflow while alternatively breathing through the right and the left nostril. The nostril with the best airflow is the preferred choice for naso tracheal intubation.

The next step is to decide between needle based local anesthetic blocks or go with entirely topical anesthesia. Each one has their advantages. Some may prefer nerve blocks due to rapid onset and longer duration and also because of the belief that chances of local anesthetic toxicity may be lesser due to smaller dose requirement of local anesthetics. However, complications of airway blocks are not insignificant and include intra-arterial injection, hematoma formation, and tracheal injury. Needle blocks are relatively contraindicated in patients with coagulopathies or those who are on anticoagulants.

The next step is to decide the method of choice for tracheal intubation. One can either choose fiber-optic intubation, video laryngoscopy, or even a regular direct laryngoscopy [9]. However, the flexible fiber-optic bronchoscope (FOB) is regarded as the gold standard airway tool for management of difficult airway. Awake fiber-optic intubation can be performed with the patient in seated or supine position [1, 11].

As the patient is prepared and premedicated for an awake fiber-optic intubation, the process of anesthetizing the airway with topically applied local anesthetics may be started. This is a dynamic process that can also involve performing regional anesthesia on specific nerves. Because both non-invasive and invasive means of airway anesthesia are available, it is up to the anesthesiologist to decide which combination will work best for the individual patient. Various local anesthetics may be utilized; however, lidocaine has a better safety profile than other agents used for airway anesthesia [12]. Also, excessive doses of local anesthetics can cause toxicity, and the total amount used in both topical and regional techniques must be considered. The route of administration of lidocaine determines the time to the peak concentration of the local anesthetic. Therefore, the route of endotracheal intubation and the specific technique of localization will determine which anatomic structures will need to be anesthetized first.

Ultimately, familiarity, experience, and skill, as well as availability of equipment, will help the anesthesia provider to consider and choose one approach over the other [1].

Topicalization of the Airway

Topicalization of the Nasal Cavity and Nasopharynx

Awake nasotracheal intubation can be facilitated by first anesthetizing the nasal cavity. A topical anesthetic can be used to block the nerves that lie just beneath the nasal mucosa. Cotton pledgets soaked in 2–4% lidocaine can be used to topicalize the

anterior ethmoidal nerve and pterygopalatine ganglion. To start, vasoconstrictors, such as oxymetazoline, should be applied to the nasal mucosa. Then pledgets should be positioned as illustrated in Fig. 14.2 (a) anteriorly and superiorly along the middle turbinate to anesthetize the anterior ethmoidal nerve and several minutes after (b) inferiorly and posteriorly along the inferior turbinate to anesthetize the pterygopalatine ganglion. An oral approach with an injection of local anesthetic may also be utilized to block the pterygopalatine ganglion [13]. A nasal trumpet covered in 2% lidocaine jelly or 4% viscous lidocaine is another means of anesthetizing the nasal cavity. The plastic sheath of a 20-guage angiocatheter or laryngotracheal mucosal atomizer placed through the nasal trumpet allows the injection of 4 mL of 1% lidocaine directly into the nasopharynx. The spread of local anesthetic to more distal airways is then facilitated by a cough that this procedure causes.

Topicalization of the Oral Cavity and Oral Pharynx

A local anesthetic can be sprayed onto the mucosa of the oral cavity and oral pharynx to allow for AI. Cetacaine is a preparation composed of benzocaine, tetracaine, and aminobenzoate delivered in an oily foam that is commonly used for this purpose. Care must be taken when using any benzocaine-based product, though, as it can result in methemoglobinemia within 1–2 s of use. Also, the use of any aerosolized sprays containing preservatives can cause a sore throat following the procedure [13].

Topicalization of the Airway with Aerosolized Local Anesthetic

An aerosolized local anesthetic is one of the most simple and comfortable means of providing an anesthetic to the airway down to the level of the trachea. The main shortfall of this method is that some degree of patient cooperation is required, which can present a problem in some patient populations. 5 mL of 4% lidocaine is nebulized with oxygen at 6 L/min via a face mask to provide the patient with airway topicalization. The level of anesthesia achieved through this technique is highly variable and is the main disadvantage of this approach [6]. Also, the lack of dense airway anesthesia in combination with a working gag reflex can make it challenging.

Spray-As-You-Go Technique

The spray-as-you-go (SAYGO) technique is a method of noninvasively and intermittently anesthetizing the airway. It requires more patience on the part of the anesthesiologist and more patient cooperation but is useful in patients who are at risk for gastric aspiration because it allows the patient to maintain his or her reflexes as long as possible. The patient is first premedicated and has the appropriate monitors applied before the nose or mouth is anesthetized. The fiber-optic bronchoscope is advanced into the pharynx, and 4% lidocaine is injected via the suction port by one of two methods. One method involves attaching a 5 mL syringe with a local anesthetic to the suction port. The least amount needed to anesthetize an area, 0.2 mL increments, are then injected into the port. The physician then waits approximately 1 min before advancing the bronchoscope and repeating the maneuver. The other method involves an epidural catheter being placed through the suction port of the bronchoscope to deliver the local anesthetic.

Regional Anesthesia of the Airway

Glossopharyngeal Nerve Block

In most patients, topicalization of the airway is sufficient to allow awake intubation. However, a pronounced gag reflex found in some patients leads to discomfort and subsequent failure of AI despite adequate topicalization. This occurs because the pressure receptors at the root of the tongue, which initiate the gag reflex, lie in the submucosa and are not consistently blocked by topical anesthesia [14]. A bilateral glossopharyngeal nerve block can be

performed using either an intraoral or peristyloid approach to suppress the gag reflex. Aspiration before injection must be performed during this block, as both approaches involve deposition of local anesthetic near the carotid artery.

The intraoral approach to the glossopharyngeal nerve block is performed in a patient who can open his or her mouth widely. As depicted in Fig. 14.3, after the patient's oral cavity is anesthetized and the mouth is opened, a number 3 Macintosh laryngoscope blade is used to assist with visualization of the palatine tonsil and the posterior tonsillar pillar. A 22-gauge or smaller spinal needle is then inserted 0.5 cm into the submucosa at the base of the posterior tonsillar pillar. Following a negative aspiration test, 2 mL of 1% lidocaine is then injected. The block is then repeated on the contralateral side. A variation of the intraoral approach is often performed by otolaryngologists and involves blocking the nerve from a posterior direction (Fig. 14.4) [13].

An alternative to the intraoral approach is the peristyloid approach to the glossopharyngeal nerve block. This technique is more appropriate for a patient who is unable to open his or her mouth wide enough for intraoral blockade. The patient is placed in a supine position, with the head in a neutral position. A line is then drawn to connect the mastoid process and the angle of the mandible, as shown in Fig. 14.5. The styloid process is then palpated using deep pressure near the middle of this line. A short 22-gauge needle is then inserted and advanced until the styloid process is contacted. The needle is then withdrawn slightly and directed posteriorly off of the styloid process. Five milliliters of 1% lidocaine are injected after a negative aspiration test. The block is then repeated on the contralateral side.

A modified peristyloid approach can also be performed using an echogenic needle under ultrasound guidance. The ultrasound is used to identify the carotid artery and can decrease the risk of vascular injury and intravascular injection. This modification may improve the accuracy and safety of the glossopharyngeal nerve block [15].

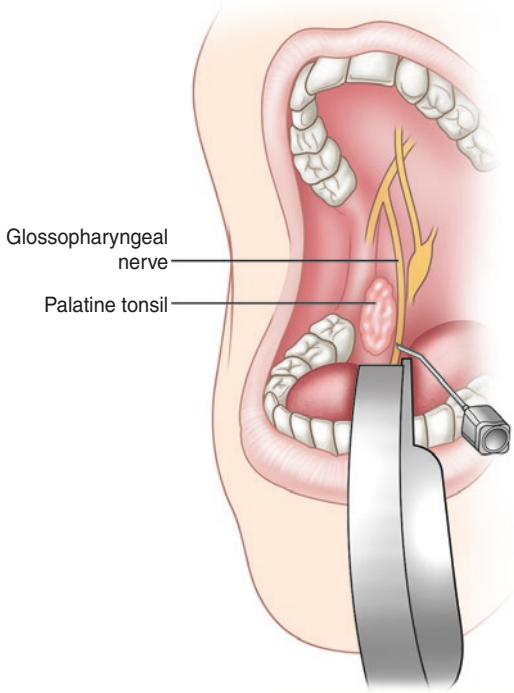

Fig. 14.4 Glossopharyngeal nerve block—intraoral approach. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

Superior Laryngeal Nerve Block

During an AI, the superior laryngeal nerve is blocked bilaterally to facilitate passage of the endotracheal tube through the larynx. To block this nerve, the patient is placed in a supine or sitting position with the neck maximally extended. The neck region is prepped with aseptic technique. The hyoid bone is displaced ipsilaterally. A 25-gauge needle is inserted and walked of the greater cornu inferiorly. The needle is then advanced 2–3 mm, thereby piercing the thyrohyoid membrane. Aspirate, and if no air or blood encountered, 2 mL of 1% lidocaine is then injected. If air is aspirated, the needle is probably in the airway; redirect and repeat the procedure. If blood is aspirated, the needle is intravascular; redirect and repeat the procedure (Fig. 14.6).

Recurrent Laryngeal Nerve Block

The transtracheal or trans laryngeal block is performed to block the recurrent laryngeal nerve,

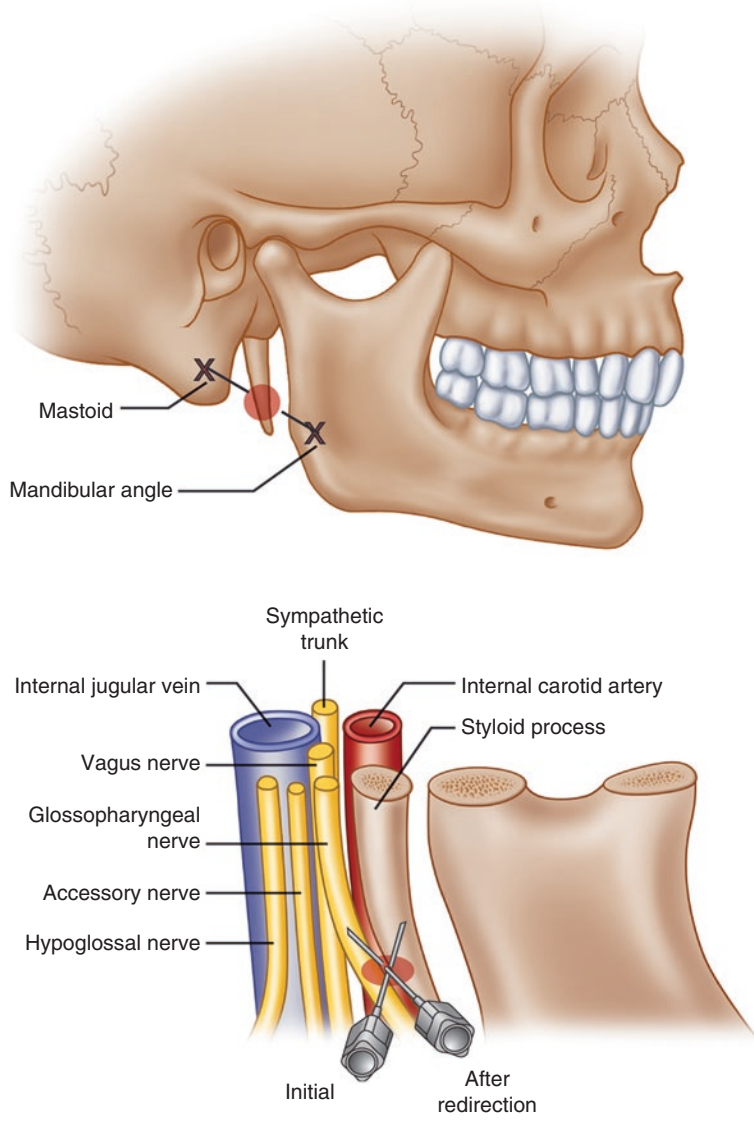

Fig. 14.5 Glossopharyngeal nerve block—peristyloid approach. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

thus properly anesthetizing the area below the vocal cords. This block prevents the patient from coughing as the endotracheal tube passes through the vocal cords, and it is also useful during an awake tracheostomy. The block is typically not performed on patients at risk for elevated intracranial pressure or intraocular pressure. To block this nerve, the patient cartilage is placed in a supine or sitting position. Aseptic technique is used to prep the neck. The thyroid cartilage and cricoid cartilage is palpated. The space between the two is the cricothyroid membrane. A 20-gauge angiocatheter is advanced midline in the neck through the cricothyroid membrane while aspirating for air. Once air is aspirated, the angiocatheter is advanced into the airway. The needle is removed, and then the angiocatheter is again aspirated for air to confirm correct placement. At this point, 4 mL of 4% lidocaine is injected into the airway. The patient should cough, confirming placement of the local anesthetic in the airway. Only the sensory aspect of the recurrent laryngeal nerve is blocked (Figs. 14.7 and 14.8).

Fig. 14.6 Superior laryngeal nerve block

Authors' Approach

Education of the patient is paramount for a successful awake intubation. A cooperative patient is much easier to intubate than a combative one; therefore, the anesthesiologist must explain the steps of the procedure to the patient. After placement of the appropriate monitors, an antisialagogue is given to help minimize secretions. Light sedation is given to establish a level at which the patient is comfortable while at the same time responsive and breathing spontaneously. Topical anesthetic spray, aerosolized local anesthetics, or ointment can be used to help anesthetize the airway. Appropriate nerve blocks are then performed, as described above.

In a patient lying in the supine position, the tongue falls posteriorly into the back wall of the pharynx causing obstruction of the view with the fiber-optic bronchoscope; therefore, the head of the bed is raised. This forces the scope deeper into the back of the pharynx. Furthermore, in an upright patient, saliva pools low in the pharynx out of the field of view. The length of the scope is lubricated, and an appropriate size endotracheal

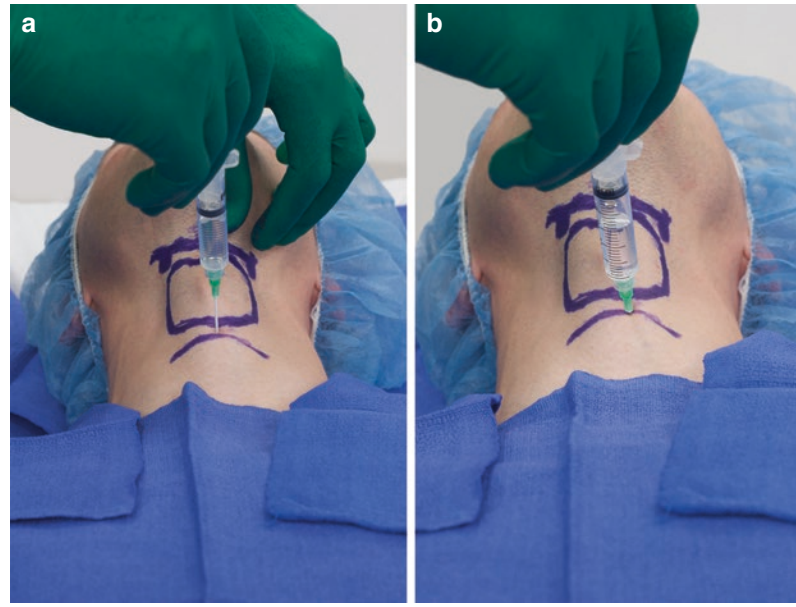

Fig. 14.7 (**a**, **b**) Translaryngeal nerve block

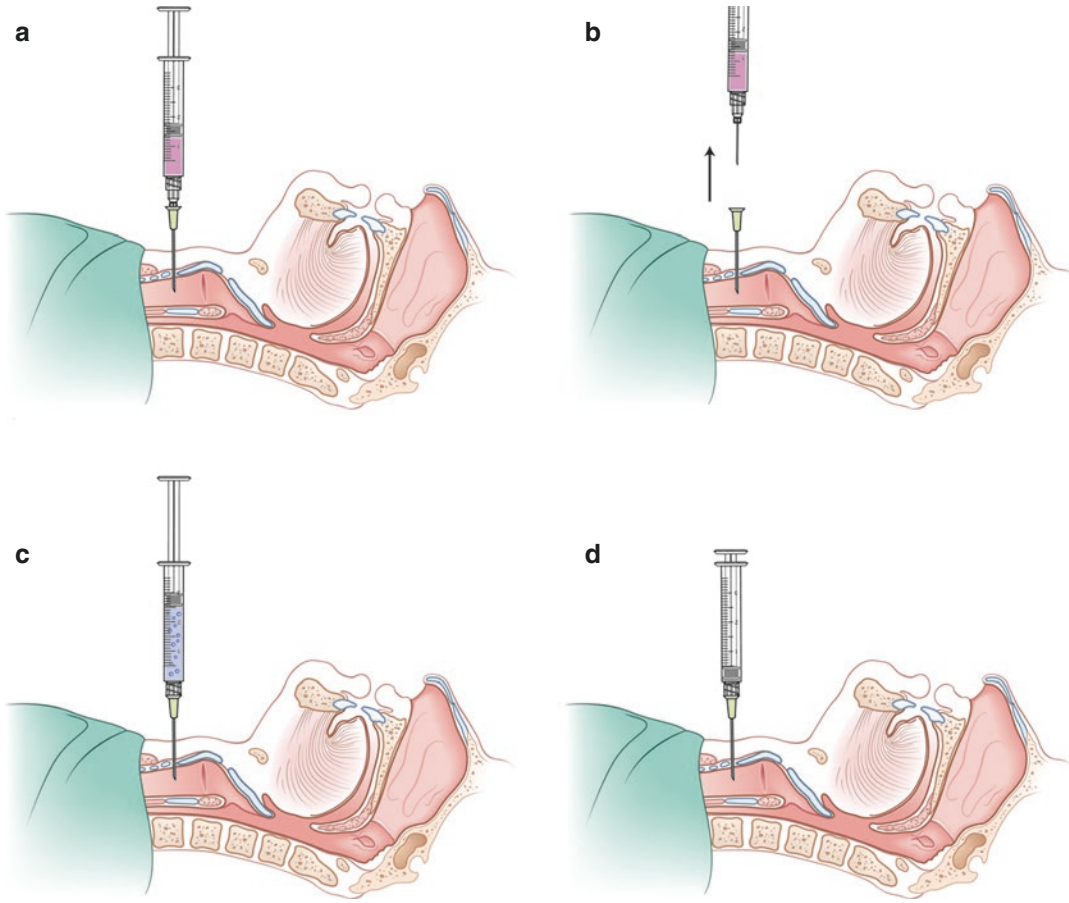

Fig. 14.8 Translaryngeal nerve block—midsagittal view of angiocatheter technique. (**a**) Insertion of angiocatheter. (**b**) Removal of the needle. (**c**) Confirmation of aspiration test. (**d**) Injection of local anesthetic. Adapted from Hagberg CA. *Benumof's Airway Management* 2nd Ed, 2007

tube is placed over it. A monitor is recommended as it allows everyone in the room to visualize what the airway manager is doing.

Face the patient and have an assistant pull the tongue forward with gauze or provide a jaw thrust [5]. An intubating airway is used to prevent the patient from biting on the scope. The scope is placed in the patient's mouth. The epiglottis and glottic opening should be visualized midline. Move the scope toward the glottic opening and pass it between the vocal cords. Once tracheal rings are visualized, advance the scope until the carina is observed and pass the endotracheal tube over the scope. Confirm visualization of the carina and then remove the scope. Inflate the cuff of the ETT and attach it to the breathing circuit. Check for end-tidal CO_2 and bilateral chest rise. Induction of the patient is appropriate at this time.

Acknowledgments The authors would like to acknowledge Valeriy V. Kozmenko, M.D. for the photography that appears in the chapter.

Review Questions

1. Which of the following nerves could be blocked to prevent a gag reflex?
   (a) Branches of the trigeminal nerve
   (b) Glossopharyngeal nerve

(c) Superior laryngeal nerve
(d) Recurrent laryngeal nerve

2. Care must be taken when performing a glossopharyngeal nerve block due to the potential for infiltration of the:
   (a) Carotid artery
   (b) Jugular vein
   (c) Thyroid cartilage
   (d) Vagus nerve
3. Bilateral blockade of the recurrent laryngeal nerve can result in:
   (a) Airway dilation
   (b) Airway obstruction
   (c) Pneumothorax
   (d) Hoarseness
4. Which of the following nerves must be blocked during an awake tracheostomy?
   (a) Trigeminal
   (b) Glossopharyngeal
   (c) Superior laryngeal
   (d) Recurrent laryngeal
5. The anterior ethmoidal nerve is a branch of:
   (a) Cranial nerve I (Olfactory)
   (b) Cranial nerve III (Orbital)
   (c) Cranial Nerve V (Trigeminal)
   (d) Cranial Nerve VII (Facial)
6. Which of the following muscles is innervated by the superior laryngeal nerve?
   (a) Cricoarytenoid
   (b) Cricothyroid
   (c) Thyroarytenoid
   (d) Vocalis
7. Which of the following local anesthetic used in topical anesthesia of the airway is associated with methemoglobinemia?
   (a) Benzocaine
   (b) Cocaine
   (c) Lidocaine
   (d) Tetracaine
8. Which of the following structures lies near the internal branch of the superior laryngeal nerve as it pierces the thyrohyoid membrane?
   (a) Inferior thyroid artery
   (b) Lingual artery
   (c) Superior thyroid artery
   (d) Subclavian artery
9. Displacement of the hyoid bone in which of the following directions aids in the blockade of the superior laryngeal nerve:
   (a) Contralateral
   (b) Inferior
   (c) Ipsilateral
   (d) Superior
10. The left recurrent laryngeal nerve winds around which of the following structures:
   (a) Arch of the aorta
   (b) Left subclavian artery
   (c) Left subclavian vein
   (d) Left vertebral artery

Answers

1. b
2. a
3. b
4. d
5. c
6. b
7. a
8. c
9. c
10. a

References

1. Collins SR, Blank RS. Fiberoptic intubation: an overview and update. Respir Care. 2014;59(6):865–80.
2. Rosenstock CV, Thøgersen B, Afshari A, Christensen AL, Eriksen C, Gätke MR. Awake fiberoptic or awake video laryngoscopic tracheal intubation in patients with anticipated difficult airway management. Anesthesiology. 2012;116(6):1210–6.
3. Ajay S, Singhania A, Akkara AG, Shah A, Adalja M. A study of flexible fiberoptic bronchoscopy aided tracheal intubation for patients undergoing elective surgery under general anesthesia. Indian J Otolaryngol Head Neck Surg. 2013;65(2):116–9.
4. Mu L, Sanders I. Sensory nerve supply of the human oro- and laryngopharynx: a preliminary study. Anat Rec. 2000;258(4):406–20.
5. Finucane BT, Tsui BCH, Santora AH. Principles of airway management. Springer: New York, NY; 2011.
6. Pani N, Kumar RS. Regional & topical anaesthesia of upper airways. Indian J Anaesth. 2009; 53(6):641–8.

7. Henderson J. Airway management in the adult. In: Miller R, editor. Miller's anesthesia. Philadelphia: Elsevier; 2010. p. 1573–610.
8. Smith JE, Reid AP. Identifying the more patent nostril before nasotracheal intubation. Anaesthesia. 2001;56(3):258–62.
9. Doyle DJ. Airway anesthesia. Anesthesiol Clin. 2015;33(2):291–304.
10. Avitsian R, Lin J, Lotto M, Ebrahim Z. Dexmedetomidine and awake fiberoptic intubation for possible cervical spine myelopathy: a clinical series. J Neurosurg Anesthesiol. 2005;17(2):97–9.
11. Alvis BD, King AB, Hester D, Hughes CG, Higgins MS. Randomized controlled pilot trial of the rigid and flexing laryngoscope versus the fiberoptic bronchoscope for intubation of potentially difficult airway. Minerva Anestesiol. 2015;81(9):946–50.
12. Baptista R, Silva S, Dias P, Monteiro P, et al. In-hospital prescription of QT-prolonging drugs in a cohort of more than 100,000 patients. Int J Cardiol. 2011;147(1):165–6.
13. Ramkumar V. Preparation of the patient and the airway for awake intubation. Indian J Anaesth. 2011;55(5):442–7.
14. Quinn FB, Ryan MW, Stoner Quinn M. Regional Anesthesia in Head and Neck Surgery SOURCE: Grand Rounds Presentation. 2006.
15. Bedder M. Glossopharyngeal nerve. In: Hahn M, McQuillan P, Sheplock G, editors. Regional anesthesia: an atlas of anatomy and techniques. Philadelphia: Elsevier; 1995. p. 75–8.

Regional Anesthetic Techniques for the Pediatric Patient

15

Vaughn E. Nossaman and Bobby D. Nossaman

Introduction

Since the original debate in the 1980s regarding the pros and cons of pediatric regional anesthesia [1, 2], safe and effective treatment of acute pain in children remains a high priority as clinical studies have shown pediatric patients experience pain from medical illnesses, during and following therapeutic and diagnostic procedures, and following trauma and surgery [3–11]. Although the safety profile of opiate administration in children has been established [12–17], elimination half-lives in newborns are longer with decreased metabolic clearances when compared to older children and adults [18, 19]. The optimal plasma concentrations for effective opiate analgesia are variable with careful titrations required to obtain effective analgesia while minimizing side effects [19–23].

Regional anesthesia has been shown to be beneficial when compared to general anesthesia. These benefits include reductions in morbidity and mortality [24–35], superior postoperative analgesia [36–43], and cost-effectiveness [39, 44–48]. There have been progressive developments in regional anesthetic techniques for the pediatric patient, since the original publications of the 1950s [49–52], but these techniques are still slow to be implemented due to concerns about neurologic complications, operator inexperience, and availability of proper equipment [53–58]. Many of these concerns were addressed in a sentinel article published in 1996, in a prospective study of greater than 24,000 pediatric blocks, in which 89% were performed under sedation or general anesthesia, with an incidence of 0.9/1000 complications and with no deaths nor long-term sequelae [33]. These findings were confirmed with subsequent studies [34, 35, 43, 59–63]. When properly performed, regional anesthesia is a safe, clinical practice with risk profiles similar to general anesthesia [34, 35, 43, 59–67].

Ultrasonography

All clinical techniques have an incidence of failure. Neurovascular anatomy is variable with subcutaneous electrical current stimulation techniques providing nerve localization with little to no information in proper placement of local anesthetics. Therefore, percutaneous techniques utilizing surface anatomy and projection, even in the best of hands, are fraught with failure [61, 68–70]. With the development of high-resolution portable ultrasound (US) analysis of anatomic

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_15

relationships and observed real time spread of administered local anesthetics have made this modality feasible in the conduct of pediatric regional anesthesia [71–75]. To develop skill sets in the use of US for regional anesthesia, one should attend an US-guided regional anesthesia course, begin with simple blocks, then progress to more complicated procedures as experience develops [68, 70, 76–79].

Local Anesthetic Blocks

The technical expertise required in delivering regional anesthesia is tempered with concerns about producing neurologic complications, availability of proper equipment, costs and time limitations as to why regional anesthetic techniques are not utilized in the pediatric population [56–58, 68, 70, 76–80]. In children, most regional techniques require general anesthesia to provide a safe procedural environment [34, 76, 81]. With regard to selection of local anesthetics, the delivery site and the metabolic maturity of the child are also important considerations [82–85]. The introductions of the newer local anesthetics, levobupivacaine and ropivacaine, have similar pharmacokinetic profiles when compared to racemic bupivacaine, and are reported to be less cardiac toxic [84, 86–88], and are shown to be beneficial in children [86, 89–91]. Although local anesthetic toxicity is rare in children, reports of seizures, transient neuropathic symptoms, dysrhythmias, and cardiovascular collapse have been reported [85, 86, 90–93].

Topical Analgesia

As with adults, topical anesthesia is used to anesthetize the skin by local infiltration before intravenous catheter insertion or other minor procedures [94–98]. Likewise, local anesthetic infiltration is also employed to provide postoperative analgesia for incisional pain. Dosing guidelines are comparable to those guidelines for adults [99–102].

Early studies from the 1950s employed mixtures of tetracaine, adrenalin (epinephrine), and cocaine (TAC) in pediatric patients for repair of minor skin lacerations in emergency departments [103–107]. In a large-scale pediatric series, this form of anesthesia resulted in quicker surgical repair times, markedly improved patient acceptance, with wound complication rates not significantly different when compared to lidocaine subcutaneous infiltration. Subsequent studies confirmed these findings [104, 105, 107].

A eutectic mixture of local anesthetics (EMLA) cream was developed in the 1980s that contains 2.5% lidocaine and 2.5% prilocaine [108]. This mixture results in an oil-water emulsification with a total local anesthetic concentration of 5% and has the ability to anesthetize intact skin to a depth of 5 mm [109, 110]. Recommended application is 45 min to 1 h before invasive procedures, with an occlusive dressing applied over the proposed procedural site. Because of EMLA's potential for systemic toxicity, the cream should not be in prolonged contact with mucous membranes or with traumatized skin [111–113]. Common uses include anesthesia for venipuncture, neonatal circumcision, lumbar punctures, vaccinations, biopsies, and laser ablation of port wine stains [85, 114–125].

Another local anesthetic cream with a shorter onset of action (~30 min), ELA-Max is also available and is composed of 4% liposomal lidocaine [126]. One study by Eichenfield and colleagues observed comparable efficacy between ELA-Max at 30 min and EMLA cream applied 60 min before the procedure [127]. ELA-Max may also decrease the incidence of methemoglobinemia as it does not contain prilocaine [85]. ELA-Max has been beneficial for intravenous cannulations and in office meatotomies [127–131].

Applications of local anesthetics to mucous membranes have been reported to decrease discomfort during nasotracheal intubation, nasogastric tube insertions, and bronchoscopy [132–136]. This application may be accomplished by a number of methods including direct spray, nebulization, or ointment or jelly application [137–140].

Regional Anesthetic Blocks

Head and Neck Blocks

Blockade of the great auricular nerve acts as an opioid sparing technique for tympanomastoidectomy and otoplasty, and in the treatments of moyamoya disease and postherpetic neuralgia [141–144]. The nerve arises from the superior cervical plexus (C_2, C_3) and provides sensory innervation to the lateral occipital region and medial auricle. The nerve ascends superficial to the posterior belly of the clavicular head of the sternocleidomastoid muscle (Fig. 15.1). Local anesthetic is injected along this subcutaneous region at the level of the cricoid cartilage. Complications include intravascular injection of the carotid artery or internal jugular vein and phrenic nerve block resulting in Horner's syndrome [54, 145].

Effective pain relief for cleft lip repair as well as for sinus surgery, rhinoplasty, and nasal septal reconstruction can be provided by an infraorbital nerve block [146, 147]. The sensory nerve is derived from the second maxillary division of the trigeminal nerve and exits the skull through the foramen rotundum before passing through the infraorbital foramen. It then divides into four branches—internal and external nasal, superior labial, and inferior palpebral branches. These branches innervate the skin of the upper lip, lower eyelid and cheek and lateral nose. Two field blocks, extraoral and intraoral can block the nerve (Figs. 15.2 and 15.3). The external approach involves locating the infraorbital foramen approximately 0.5 cm inferior to the lower orbital margin. A 27-gauge needle is then inserted until bone is contacted. The needle is then withdrawn slightly and following negative aspiration a small amount of local anesthetic (0.25–0.5 mL) is injected. The intraoral approach starts with the same landmark by palpating the infraorbital foramen with the non-dominant hand to maintain position. The upper lip is then lifted and a 25–27-gauge needle is used to inject 0.5–1.5 mL of local anesthetic following negative aspiration along the inner surface of the lip along the maxillary premolar toward the infraorbital foramen. Other than swelling around the eyelid, which can be reduced by pressure over the injection site for several minutes, complications from this block are rare.

Indications for supraorbital and supratrochlear nerve blocks include procedures on the scalp and forehead such as frontal craniotomies, ventriculoperitoneal shunt revisions, excision of skin lesions,

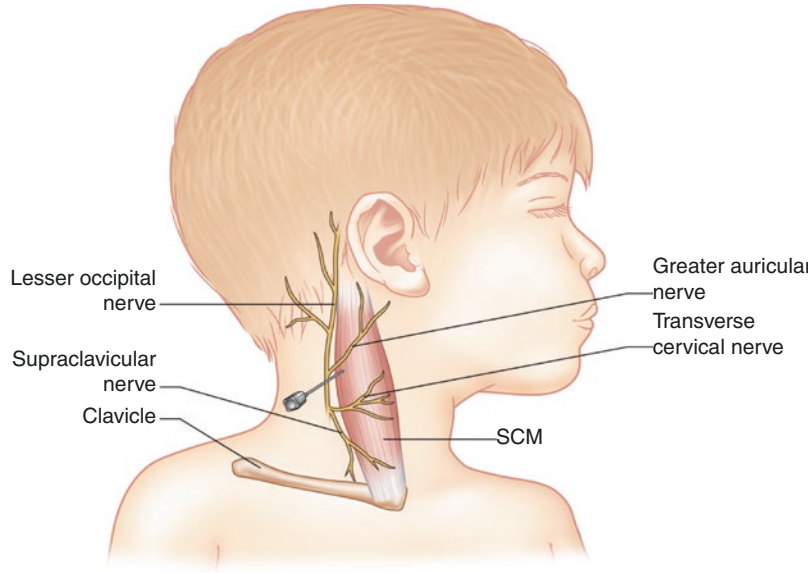


Fig. 15.1 Great auricular nerve block

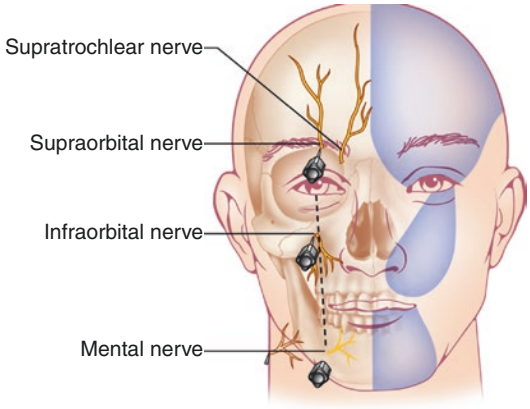


Fig. 15.2 Extraoral nerve block

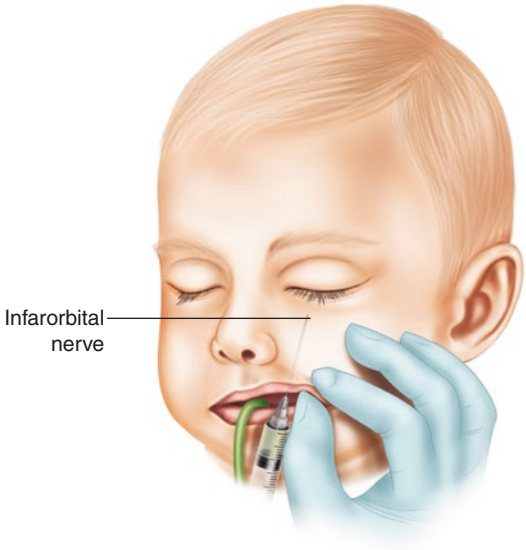


Fig. 15.3 Intraoral nerve block

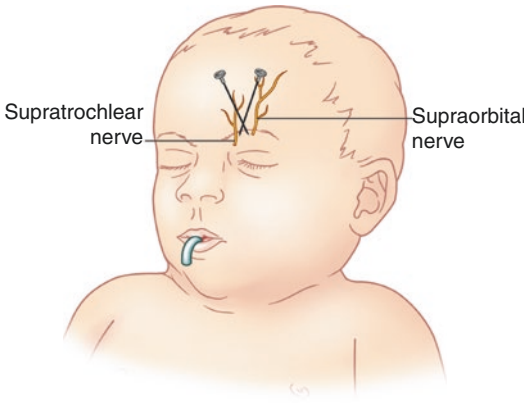


Fig. 15.4 Supraorbital and supratrochlear nerve block

and laser therapy for hemangiomas (Fig. 15.4) [143, 148, 149]. The nerves are branches of the ophthalmic division of the trigeminal nerve and supply the skin of the forehead and conjunctiva of the upper eyelid. The supraorbital nerve is found in the upper margin of the orbit at the supraorbital notch and the supratrochlear nerve is in close proximity and just medially. After palpating the supraorbital notch, a 27-gauge needle is inserted superior to the notch until bone is contacted. Local anesthetic (1 mL) is injected after slight withdrawal and negative aspiration for blood. The needle is withdrawn and directed slightly medially before injecting another 1 mL of local anesthetic following negative aspiration. Hematomas and periorbital edema are common complications [150, 151], but can be minimized by applying pressure for approximately 5 min.

Brachial Plexus Block

Although there are several approaches to the brachial plexus in children, the axillary approach is commonly used for brachial plexus blockade [152, 153]. Recently, the use of US allows infraclavicular and supraclavicular approaches to the brachial plexus [81, 154–157]. The brachial plexus arises from the cervical nerve roots (C_5-T_1). Brachial plexus blocks are easy to perform in children, due to less adipose tissue when compared to adults, and the axillary artery is easier to palpate and isolate [158, 159]. The arm is abducted to a 90° angle in relation to the chest wall. The artery is palpated and fixed in the axilla, and the 22-guage, short-bevel, 2-in. needle allows accurate placement around and when necessary through the axillary artery (Fig. 15.5). With 'through and through' axillary artery puncture technique continuous aspiration is required as the needle is advanced until no blood is aspirated, then one-half of the local anesthetic is injected into the distal portion of the sheath. As the needle is withdrawn, again the needle is continuously aspirated until no blood can be withdrawn, and the remaining half of the local anesthetic can be injected into the proximal portion of the sheath. The recommended dose of local anesthetic is 1 mL/kg of either 0.25% bupivacaine or 0.2%

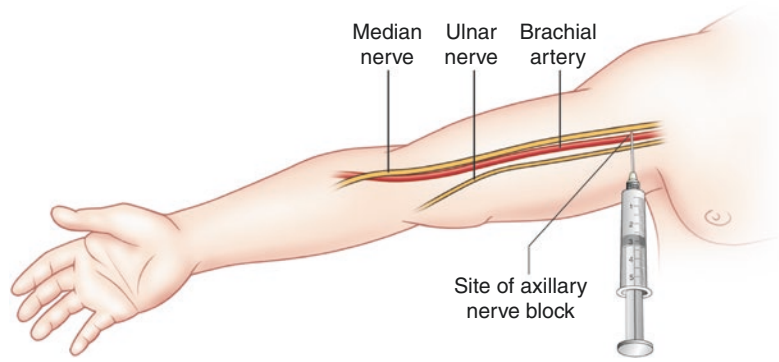


Fig. 15.5 Axillary block

ropivacaine [102]. Vigilant aspiration should be performed to minimize intravascular injection. An additional circumferential subcutaneous cuff block for the intercostobrachial nerve to minimize tourniquet pain is also recommended.

The use of a nerve stimulator can assist the operator in advancing the 22-guage, short-bevel 2-in. needle into the sheath of the brachial plexus superior to the axillary artery. Once a twitch is elicited, local anesthetic solution can be injected into the sheath. Again a ring of local anesthetic can be subcutaneously injected in a ring around the upper arm to block the intercostobrachial nerve to provide tourniquet-related pain relief.

Ultrasound is also effective in visualizing the interscalene approach to the brachial plexus [81, 160–164]. A recent review of the Pediatric Regional Anesthesia Network reported placement of interscalene blocks in children under general anesthesia identified no serious adverse events [81].

Paravertebral Block

With the ability to target specific dermatomes, single-sided paravertebral blockade is indicated for patients undergoing renal surgery, thoracotomy, unilateral abdominal procedures such as cholecystectomy and even inguinal surgery [165, 166]. The bilateral approach expands its use in chronic management of pancreatitis or to procedures that cross or involve the midline, such as Nuss repair of pectus excavatum or following laparoscopic cholecystectomy [167–169]. Lönnqvist and others demonstrated continuous paravertebral blockade to be superior to continuous epidural blockade in reducing morphine requirements in children undergoing renal surgery [165, 166]. Berta and others demonstrated benefits observed in single case reports [167–169] and in patients undergoing major renal surgery [170]. Loftus and colleagues reported beneficial use of paravertebral continuous infusion pain catheters following pectus excavatum repair surgery resulting in shorter hospital length of stays [171].

A wedge-shaped area, the paravertebral space is bound anteriorly by the parietal pleural, posteriorly by the superior costotransverse ligament, laterally by the posterior intercostal membrane, and medially by the vertebra (Fig. 15.6). The space contains spinal roots emerging from the intervertebral foramina from the dorsal and ventral rami and the sympathetic chain. Blockade may involve several dermatomes and can produce sensory, sympathetic, and motor blockade. In the pediatric population, the block is usually performed under general anesthesia with the patient in the lateral position. After establishing the midline, the point of lateral approach is estimated by measuring the distance between spinous processes. The needle is inserted perpendicular to skin until contact with the transverse process. The needle is then slightly retraced and directed caudal to walk off the process. In adults, the needle is then advanced 1 cm deeper than the transverse process, while in children the space is usually more superficial. Further confirmation may be obtained by a loss of resistance technique similar to epidural placement. A "pop" may be felt as the needle penetrates the paravertebral space. At this point, a drop of sterile fluid

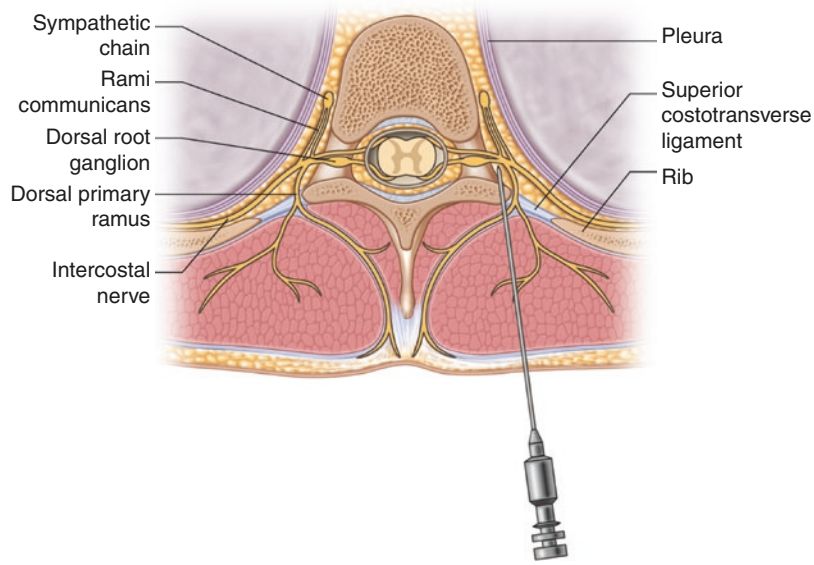


Fig. 15.6 Paravertebral block

is placed at the needle hub and the patient is given a deep breath to rule out intrapleural placement. A 22-gauge blunt needle is then used to inject 0.5 mL/kg of local anesthetic for unilateral blockade. Ropivacaine 0.2% or bupivacaine 0.25% is typically used. A Touhy needle can be used to thread a catheter for continuous techniques. Typical infusion rates are 0.25 mL/kg/h in children and 0.2 mL/kg/h in infants of 0.1–0.125% local anesthetic.

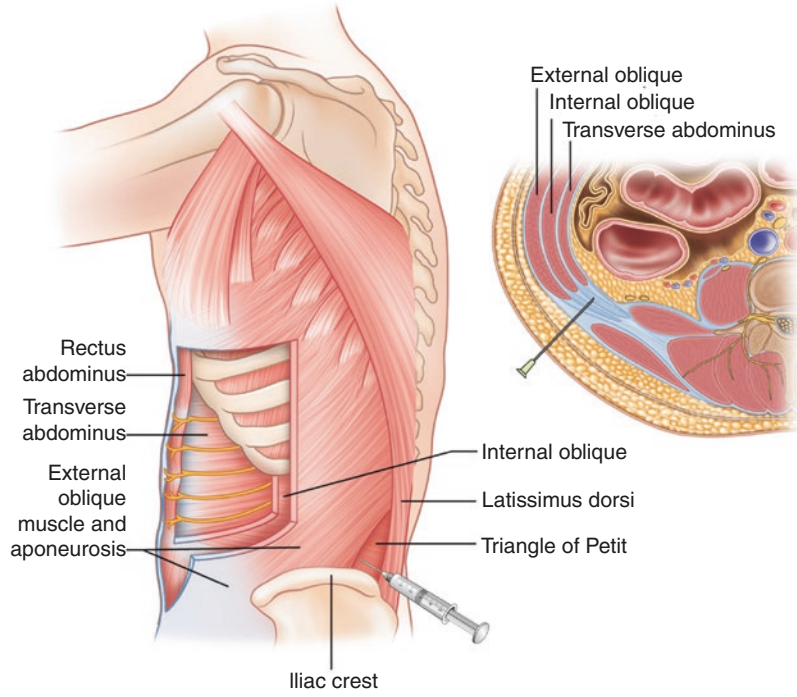
The proximity of this block to the epidural space leads to the possibility of inadvertent epidural or spinal blockade resulting in hypotension or rarely a "high spinal" [172, 173]. Other complications include vascular or pleural puncture and pneumothorax [174, 175]. A 10.7% failure rate in adults and 6.2% in children was demonstrated in one series of 367 patients by Lönnqvist and others [176]. However the use of bilateral paravertebral technique doubled the likelihood of accidental vascular puncture (9% vs. 5%) and with an eightfold increase in pleural puncture and pneumothorax complications (3% vs. 0.4%) when compared with unilateral blocks [177].

Transversus Abdominis Plane Block

As a landmark-based technique, the transversus abdominis plane block (TAP) has provided excellent analgesia in adults undergoing lower abdominal surgery including hernia repair, appendectomy, abdominal hysterectomy, and caesareans [178–182]. Application to the pediatric population, in which landmarks are difficult or impossible to palpate, has been eased by the use of US [9, 183–189]. The TAP block is especially useful in cases where neuraxial blockade is contraindicated [184]. A TAP block may substitute for the ilioinguinal/iliohypogastric block and can also provide analgesia for more superior abdominal incisions from laparotomy or laparoscopy. Incisional pain can be well controlled but the block is less effective for visceral pain [9, 183–190].

The anterolateral abdominal wall is innervated by the anterior rami of T_7-L_1 and include the ilioinguinal, iliohypogastric, intercostal, and subcostal nerves (Fig. 15.7) [191]. These nerves travel in the intercostal space before entering the abdominal wall between the internal oblique and transversus abdominis muscles. This plane serves as the target for the TAP block. The landmark technique involves locating the lumbar triangle of Petit. The base of the triangle lies on the highest point of the iliac crest and the apex is at the costal margin. Anterior and posterior borders include the external oblique muscle and latissimus dorsi muscle, respectively. A blunt 22-gauge 2-in. needle is inserted in this location and passes through the external oblique muscular fascia,

Fig. 15.7 TAP block



then the internal oblique muscular fascia (Fig. 15.7). After these two fascial "pops" are appreciated, local anesthetic is injected following negative aspiration with obvious care not to exceed toxic levels. A bilateral TAP block may be used for midline incisions or procedures involving both sides [9, 182, 183, 185–189, 192, 193].

Aside from real-time visualization, US offers a distinct advantage for this block in the pediatric population as the triangle of Petit is difficult to ascertain in children and loss of resistance through less developed internal and external oblique muscles can be difficult to appreciate [191, 194]. Placement of the US probe in the transverse plane above the iliac crest usually provides excellent visualization of the external and internal obliques fascial planes, transversus abdominis fascial plane, and peritoneal plane although the US probe may need to be directed more medially in some patients. Local anesthetic is deposited following negative aspiration as the needle tip is visualized deep to the internal oblique fascial plane. Spread within the internal oblique and transversus abdominis fascial plane confirms accurate placement. An US-guided Tuohy needle can be used to place a continuous catheter 2–3 cm beyond the needle tip if prolonged analgesia is required [9, 186, 187].

Complications are similar to those reported with ilioinguinal blockade including peritoneal perforation and femoral nerve palsy [182, 192, 195]. The catheter-based technique has a theoretical risk of infection. There are no reported complications with the US-guided technique [9, 182, 185–189, 192].

Ilioinguinal/Iliohypogastric Block

Analgesia for inguinal hernia repair, hydrocelectomy, and orchiopexy is provided by an ilioinguinal/iliohypogastric block [41, 79, 196, 197]. Originating from the lumbar plexus, the ilioinguinal and iliohypogastric nerves pass superficial to the transversus abdominus near the anterior superior iliac spine (Fig. 15.8). These nerves can be blocked at this site before separating. The iliohypogastric nerve supplies skin over the lower anterior abdominal wall, while the ilioinguinal supplies skin over the scrotum or labium majoris.

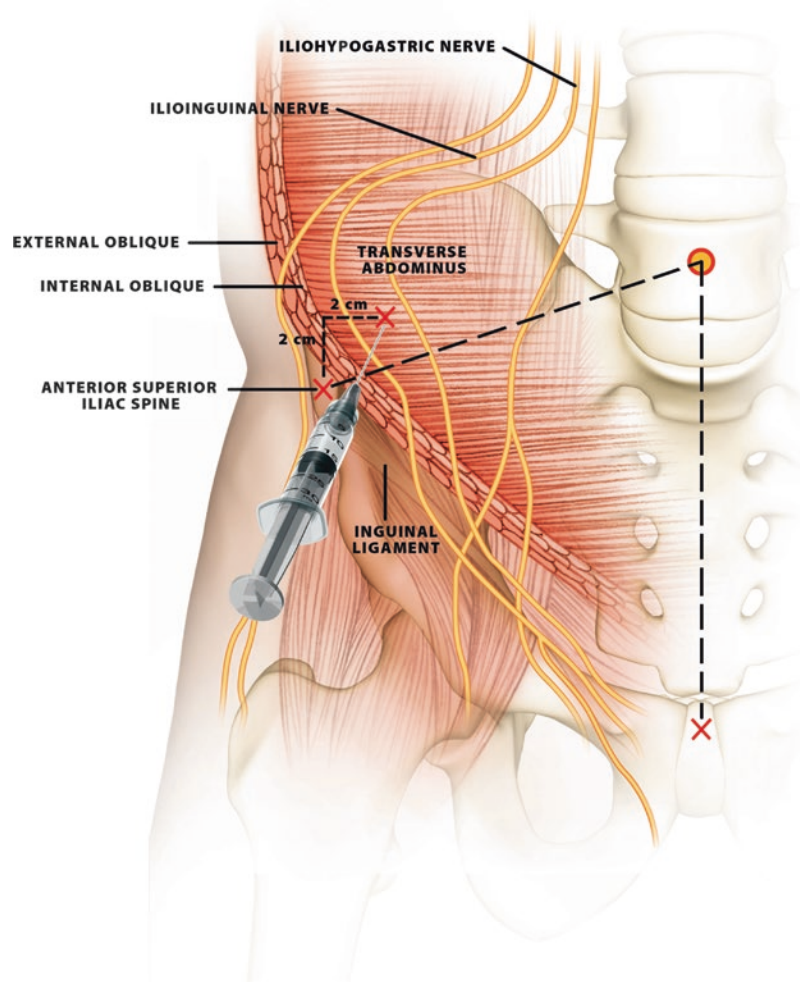


Fig. 15.8 Ilioinguinal/iliohypogastric block

A blunt 22–25 gauge needle is inserted 1 cm superior and 1 cm medial to the anterior superior iliac spine (ASIS) (Fig. 15.8). A field block is then performed directing the needle parallel to the muscle wall in the direction of the ASIS. The needle is withdrawn while injecting anesthetic and redirected toward the inguinal ligament with care not to puncture the ligament. Penetration of the oblique muscles results in a characteristic "pop" after while local anesthetic is again injected. The block can also be performed post surgically by the surgeon under direct vision. Bupivacaine 0.25% or ropivacaine 0.2% or 0.5% are typically used.

Ultrasound guidance involves direct visualization of the nerve or nerves by placement of the probe just medial to the superior aspect of the ASIS. An out-of-plane technique is typically employed as the nerves' proximity to the ASIS can make the in-plane technique challenging [198]. At this location, the nerves are typically less than 1 cm deep and run between the internal oblique and transversus abdominus muscle.

Serious complications are rare and include small bowel or colonic perforation [199]. Transient femoral blockade resulting in motor weakness of the quadriceps can occur in up to 5% of patients if the local anesthetic tracks inferior to the inguinal ligament [200].

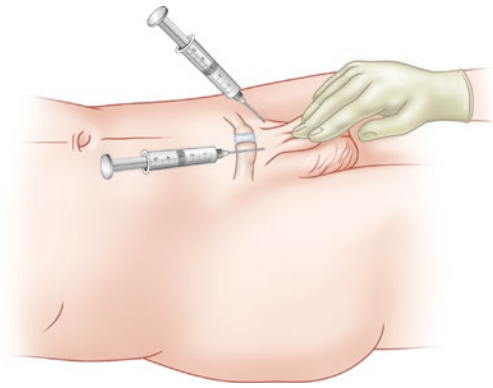


Fig. 15.9 Penile nerve block

Penile Nerve Block

Arising from the sacral plexus, innervation of the distal two-thirds of the penis is supplied by branches of the pudendal nerve known as dorsal nerves. The nerves are surrounded by Buck's fascia and are near dorsal vessels (Fig. 15.9). Various techniques exist for anesthetizing these nerves for intraoperative and postoperative pain secondary to circumcision and uncomplicated hypospadias repair. They include application of topical cream, subcutaneous ring block, dorsal nerve block, and suprapubic nerve block [124, 201–203]. Studies have shown the subcutaneous ring block to be more effective than the other techniques [201, 203].

Application of topical cream is the simplest method and has been employed because of its ability to penetrate intact foreskin [203, 204]. As absorption may be increased through mucous membranes, care must be taken to use the minimum amount necessary. Subcutaneous ring block involves placing a skin wheal of local anesthetic circumferentially around the base of the penis [205]. Injection of local anesthetic to the penis bilaterally at the symphysis pubis is known as the dorsal penile block. With downward traction of the penis, a 25-gauge needle is directed medially and caudally until Buck's fascia is penetrated at 10:30 and at 1:30 until a characteristic "pop" is felt. Frequent aspiration is necessary due to the close proximity of the dorsal vessels at this location [206–209].

Most sources recommend the avoidance of epinephrine with these blocks as vasoconstriction can theoretically result in necrosis [210, 211]. A volume of 0.1 mL/kg of bupivacaine 0.25–0.5% or ropivacaine 0.2% is typically used and provides approximately 4–6 h of analgesia. Complications include hematoma formation resulting in necrosis, intravascular injection, and tissue edema affecting surgical conditions [101, 205, 212]. Recent studies examined the role of US and found improved efficacy with the block [213–217].

Caudal Block

Although regional block needles are used in the performance of the pediatric caudal block, a number of studies advocate the use of styletted, short-beveled 22-guage needles [218–220], as the styletted needle may reduce the risk of introduction of a dermal plug into the caudal space [219]. The approach to the caudal canal is dependent upon proper angle of the needle as parallel insertion to the sacrum is required through the sacrococcygeal membrane (Fig. 15.10). Final needle placement is dependent upon a "pop" as the blunt needle pierces the sacrococcygeal membrane. Aspiration should be performed prior to injection of the local anesthetic solution. A test-dose including epinephrine (0.5 mcg/kg) helps identify that the needle is not in the intravascular or intraosseous space (Fig. 15.10 bottom cartoon). During injection, the lack of subcutaneous swelling is a helpful sign of proper needle placement. Relaxation of the anal sphincter also predicts successful blockade [221].

Extension of the Caudal Catheter into the Lumbar or Thoracic Regions

Caudal catheters were used in the past in adults, but lost their popularity with the development of lumbar and thoracic approaches to the epidural space [222]. However, there has been a recent resurgence in caudal catheter epi-

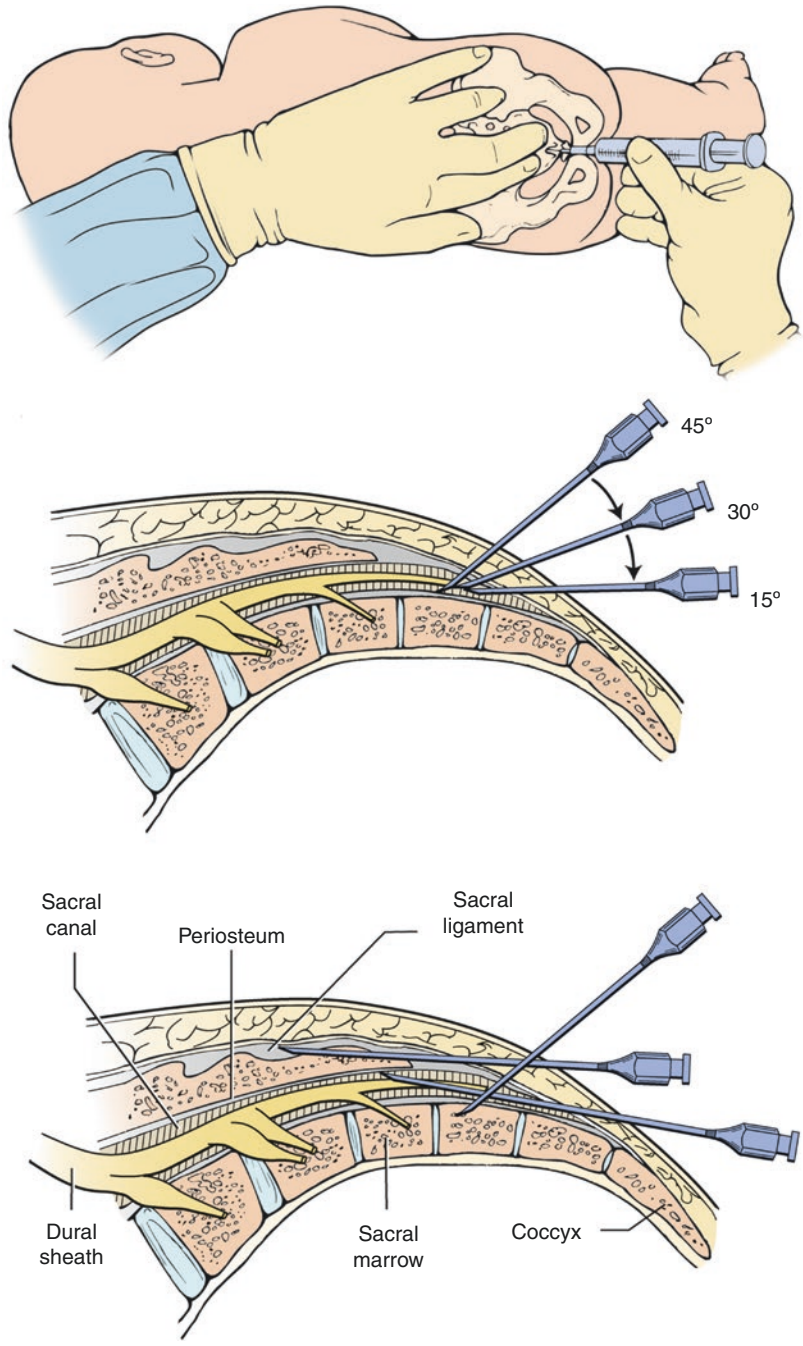


Fig. 15.10 Caudal block

dural placement in neonates and in infants as they can be used to facilitate the surgical anesthetic and be a component of a postoperative analgesia regimen. A recent large review of the Pediatric Regional Anesthesia Network in over 18,000 caudal blocks reported a 1.9% complication rate due to block failure, blood aspiration, and intravascular injection. There were no permanent sequelae reported [65]. The caudal canal in neonates can allow easy access to the

lumbar and thoracic segments with minimal resistance in passage of the catheter [222–226]. However, in older patients, the addition of fibrous and fatty tissue and development of septal membranes in the epidural space, can impede caudal catheter advancement [227, 228].

Summary

The benefits of regional analgesia in the management of postoperative pain are clearly recognized. Despite many reported advantages, the use of peripheral nerve blocks in perioperative care for children continues to be underutilized. Although these regional techniques are safe, they are not without risk [85, 99, 229]. The application of ultrasonography should decrease some of these risks [34, 61, 70, 99, 230]. Regional anesthesia can be an important component to multi-modal analgesia [217, 231, 232]. Certainly the role of the parents regarding postoperative instructions is important in the transition of analgesic regimens as the regional block wanes [233]. However, these postoperative analgesia instructions should not be significantly different than what is currently employed for pediatric patients following general anesthesia. In any perioperative plan of care, the risks and benefits of any technique lie with the skill and experience of the caregiver. Nevertheless, regional anesthesia is an effective method of providing postoperative analgesia in the pediatric patient.

Review Questions

1. Opiate metabolic clearance rates in newborns are:
   (a) Increased when compared to older children
   (b) Unchanged when compared to older children or adults
   (c) Decreased when compared to adults
   (d) Decreased when compared to older children
2. Complications of great auricular nerve blocks in children are:
   (a) Intravascular injection of the carotid artery
   (b) Intravascular injection of the internal jugular vein
   (c) Horner's syndrome
   (d) All the above
3. The use of interscalene blocks under general anesthesia are contraindicated in children
   (a) True
   (b) False
4. Paravertebral blockade is indicated in children undergoing:
   (a) Renal surgery
   (b) Thoracic surgery
   (c) Cholecystectomy
   (d) Inguinal surgery
   (e) a, b, c
   (f) All the above
5. Penile nerve blocks in children can be most effective with:
   (a) Application of topical ELMA
   (b) Subcutaneous ring block
   (c) Doral nerve block
   (d) Suprapubic nerve block
6. Caudal nerve blocks in children:
   (a) Regional nerve block needles with echogenic features improve placement
   (b) Risk intraosseous injection of local anesthetic solution
   (c) Styletted needles may reduce risk of dermal plug into the caudal space
   (d) a and b
   (e) a and c
   (f) b and c
   (g) a, b, and c
7. Extending caudal catheters into the lumbar or thoracic regions in newborns risk permanent neurologic sequelae.
   (a) True
   (b) False
8. Complications of ilioinguinal/iliohypogastric nerve blocks include:
   (a) Small bowel perforation
   (b) Colon perforation
   (c) Quadriceps motor weakness
   (d) All the above

Answers

1. a
2. d
3. b
4. f
5. b
6. f
7. b
8. d

References

1. Armitage EN. Is there a place for regional anesthesia in pediatrics?—Yes! Acta Anaesthesiol Belg. 1988;39(3):191–5.
2. Dick W. Is there a place for regional anesthesia in pediatrics?—No! Acta Anaesthesiol Belg. 1988;39(3):185–9.
3. Bhatt-Mehta V, Rosen DA. Management of acute pain in children. Clin Pharm. 1991;10(9):667–85.
4. Anand KJ, Hickey PR. Pain and its effects in the human neonate and fetus. N Engl J Med. 1987;317(21):1321–9.
5. Fitzgerald M, Beggs S. The neurobiology of pain: developmental aspects. Neuroscientist. 2001;7(3):246–57.
6. Amory C, et al. Is ilioinguinal/iliohypogastric nerve block always totally safe in children? Paediatr Anaesth. 2003;13(2):164–6.
7. Ivani G, Tonetti F. Postoperative analgesia in infants and children: new developments. Minerva Anestesiol. 2004;70(5):399–403.
8. Wolf AR. Effects of regional analgesia on stress responses to pediatric surgery. Paediatr Anaesth. 2012;22(1):19–24.
9. Abu Elyazed MM, et al. The effect of ultrasound-guided transversus abdominis plane (TAP) block on postoperative analgesia and neuroendocrine stress response in pediatric patients undergoing elective open inguinal hernia repair. Paediatr Anaesth. 2016;26(12):1165–71.
10. Rafique MB, et al. Anesthesia for children with mitochondrial disorders: a national survey and review. J Anesth. 2013;27(2):186–91.
11. Disma N, Hansen TG. Pediatric anesthesia and neurotoxicity: can findings be translated from animals to humans? Minerva Anestesiol. 2016;82(7):791–6.
12. El Sayed MF, et al. Safety profile of morphine following surgery in neonates. J Perinatol. 2007;27(7):444–7.
13. Anderson BJ, Palmer GM. Recent pharmacological advances in paediatric analgesics. Biomed Pharmacother. 2006;60(7):303–9.
14. Suominen PK, et al. Intrathecal morphine provides effective and safe analgesia in children after cardiac surgery. Acta Anaesthesiol Scand. 2004;48(7):875–82.
15. Dial S, et al. Pediatric sedation for procedures titrated to a desired degree of immobility results in unpredictable depth of sedation. Pediatr Emerg Care. 2001;17(6):414–20.
16. Wood CM, et al. Randomised double blind trial of morphine versus diamorphine for sedation of preterm neonates. Arch Dis Child Fetal Neonatal Ed. 1998;79(1):F34–9.
17. Barker DP, et al. Randomised, double blind trial of two loading dose regimens of diamorphine in ventilated newborn infants. Arch Dis Child Fetal Neonatal Ed. 1995;73(1):F22–6.
18. Inturrisi CE. Clinical pharmacology of opioids for pain. Clin J Pain. 2002;18(4 Suppl):S3–13.
19. Simons SH, Anand KJ. Pain control: opioid dosing, population kinetics and side-effects. Semin Fetal Neonatal Med. 2006;11(4):260–7.
20. Lynn AM, et al. Respiratory effects of intravenous morphine infusions in neonates, infants, and children after cardiac surgery. Anesth Analg. 1993;77(4):695-701.
21. Farrington EA, et al. Continuous intravenous morphine infusion in postoperative newborn infants. Am J Perinatol. 1993;10(1):84–7.
22. Hunt A, et al. Transdermal fentanyl for pain relief in a paediatric palliative care population. Palliat Med. 2001;15(5):405–12.
23. Hunt A, et al. Population pharmacokinetics of oral morphine and its glucuronides in children receiving morphine as immediate-release liquid or sustained-release tablets for cancer pain. J Pediatr. 1999;135(1):47–55.
24. Rodgers A, et al. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomised trials. BMJ. 2000;321(7275):1493.
25. Bouwmeester NJ, et al. Developmental pharmacokinetics of morphine and its metabolites in neonates, infants and young children. Br J Anaesth. 2004;92(2):208–17.
26. Urwin SC, Parker MJ, Griffiths R. General versus regional anaesthesia for hip fracture surgery: a meta-analysis of randomized trials. Br J Anaesth. 2000;84(4):450–5.
27. Liu SS, Wu CL. Effect of postoperative analgesia on major postoperative complications: a systematic update of the evidence. Anesth Analg. 2007;104(3):689–702.
28. Liu SS, Wu CL. The effect of analgesic technique on postoperative patient-reported outcomes including analgesia: a systematic review. Anesth Analg. 2007;105(3):789–808.
29. Wu CL, et al. Effect of postoperative epidural analgesia on morbidity and mortality after total hip replace-

ment surgery in medicare patients. Reg Anesth Pain Med. 2003;28(4):271–8.
30. Ballantyne JC, et al. The comparative effects of postoperative analgesic therapies on pulmonary outcome: cumulative meta-analyses of randomized, controlled trials. Anesth Analg. 1998;86(3):598–612.
31. Ganapathy S, et al. Best evidence in anesthetic practice: prevention: epidural anesthesia and analgesia does not reduce 30-day all-cause mortality and major morbidity after abdominal surgery. Can J Anaesth. 2003;50(2):143–6.
32. Beattie WS, Badner NH, Choi PT. Meta-analysis demonstrates statistically significant reduction in postoperative myocardial infarction with the use of thoracic epidural analgesia. Anesth Analg. 2003;97(3):919–20.
33. Weiss M, et al. Safe anesthesia for every tot—the SAFETOTS initiative. Curr Opin Anaesthesiol. 2015;28(3):302–7.
34. Ecoffey C. Safety in pediatric regional anesthesia. Paediatr Anaesth. 2012;22(1):25–30.
35. Polaner DM, Drescher J. Pediatric regional anesthesia: what is the current safety record? Paediatr Anaesth. 2011;21(7):737–42.
36. Buist RJ. A survey of the practice of regional anaesthesia. J R Soc Med. 1990;83(11):709–12.
37. Chelly JE, et al. Anesthesia and postoperative analgesia: outcomes following orthopedic surgery. Orthopedics. 2003;26(8 Suppl):s865–71.
38. Hadzic A, et al. Peripheral nerve blocks result in superior recovery profile compared with general anesthesia in outpatient knee arthroscopy. Anesth Analg. 2005;100(4):976–81.
39. Bolte RG, et al. Mini-dose Bier block intravenous regional anesthesia in the emergency department treatment of pediatric upper-extremity injuries. J Pediatr Orthop. 1994;14(4):534–7.
40. Walker BJ, et al. Peripheral nerve catheters in children: an analysis of safety and practice patterns from the pediatric regional anesthesia network (PRAN). Br J Anaesth. 2015;115(3):457–62.
41. Visoiu M. Paediatric regional anaesthesia: a current perspective. Curr Opin Anaesthesiol. 2015;28(5):577–82.
42. Vicchio N, Mossetti V, Ivani G. Evaluation of 18279 blocks in a pediatric hospital. Anesth Pain Med. 2015;5(2):e22897.
43. Muhly WT, Gurnaney HG, Ganesh A. Regional anesthesia for pediatric knee surgery: a review of the indications, procedures, outcomes, safety, and challenges. Local Reg Anesth. 2015;8:85–91.
44. Siegel AL, Snyder HM, Duckett JW. Outpatient pediatric urological surgery: techniques for a successful and cost-effective practice. J Urol. 1986;136(4):879–81.
45. Everett LL. Newer drugs in pediatric anesthesia. Semin Pediatr Surg. 1999;8(1):6–12.
46. Pershad J, Todd K, Waters T. Cost-effectiveness analysis of sedation and analgesia regimens during fracture manipulation in the pediatric emergency department. Pediatr Emerg Care. 2006;22(10):729–36.
47. Hall-Burton DM, et al. Regional anesthesia is cost-effective in preventing unanticipated hospital admission in pediatric patients having anterior cruciate ligament reconstruction. Reg Anesth Pain Med. 2016;41(4):527–31.
48. Piacevoli Q, et al. Costs and quality in loco-regional anesthesia. Minerva Anestesiol. 2005;71(9):543–7.
49. Kaloud H. The autonomic block in pediatrics. Wien Med Wochenschr. 1957;107(32–33):664–6.
50. Anastasov K. Conduction anesthesia in children. Stomatologiia (Sofiia). 1954;6:360–4.
51. Haupt H, Nagel W. Application of autonomic blockade with phenothiazine derivatives. Monatsschr Kinderheilkd. 1954;102(1):4–9.
52. Anastasov K. Novocaine anesthesia and block in children. Stomatologiia (Sofiia). 1953;6:354–60.
53. Mossetti V, Ivani G. Controversial issues in pediatric regional anesthesia. Paediatr Anaesth. 2012;22(1):109–14.
54. Brull R, et al. Practice patterns related to block selection, nerve localization and risk disclosure: a survey of the American Society of Regional Anesthesia and Pain Medicine. Reg Anesth Pain Med. 2008;33(5):395–403.
55. Constantine E, et al. The use of local anesthetic techniques for closed forearm fracture reduction in children: a survey of academic pediatric emergency departments. Pediatr Emerg Care. 2007;23(4):209–11.
56. Ford S, et al. Defining the reliability of sonoanatomy identification by novices in ultrasound-guided pediatric ilioinguinal and iliohypogastric nerve blockade. Anesth Analg. 2009;109(6):1793–8.
57. Schuepfer G, et al. Generating a learning curve for pediatric caudal epidural blocks: an empirical evaluation of technical skills in novice and experienced anesthetists. Reg Anesth Pain Med. 2000;25(4):385–8.
58. Rubin K, Sullivan D, Sadhasivam S. Are peripheral and neuraxial blocks with ultrasound guidance more effective and safe in children? Paediatr Anaesth. 2009;19(2):92–6.
59. Gurnaney H, et al. Safety of pediatric continuous interscalene block catheters placed under general anesthesia: a single center's experience. Acta Anaesthesiol Scand. 2015;59(3):377–83.
60. Gupta A, Saha U. Spinal anesthesia in children: a review. J Anaesthesiol Clin Pharmacol. 2014;30(1):10–8.
61. Boretsky KR. Regional anesthesia in pediatrics: marching forward. Curr Opin Anaesthesiol. 2014;27(5):556–60.
62. Russell P, von Ungern-Sternberg BS, Schug SA. Perioperative analgesia in pediatric surgery. Curr Opin Anaesthesiol. 2013;26(4):420–7.

63. Llewellyn N, Moriarty A. The national pediatric epidural audit. Paediatr Anaesth. 2007;17(6):520–33.
64. Deer JD, Sawardekar A, Suresh S. Day surgery regional anesthesia in children: safety and improving outcomes, do they make a difference? Curr Opin Anaesthesiol. 2016;29(6):691–5.
65. Suresh S, et al. Are caudal blocks for pain control safe in children? An analysis of 18,650 caudal blocks from the Pediatric Regional Anesthesia Network (PRAN) database. Anesth Analg. 2015;120(1):151–6.
66. Giaufre E, Dalens B, Gombert A. Epidemiology and morbidity of regional anesthesia in children: a one-year prospective survey of the French-Language Society of Pediatric Anesthesiologists. Anesth Analg. 1996;83(5):904–12.
67. Bernards CM, et al. Regional anesthesia in anesthetized or heavily sedated patients. Reg Anesth Pain Med. 2008;33(5):449–60.
68. Liu Y, et al. Comparison of the development of performance skills in ultrasound-guided regional anesthesia simulations with different phantom models. Simul Healthc. 2013;8(6):368–75.
69. Amiri HR, Espandar R. Upper extremity surgery in younger children under ultrasound-guided supraclavicular brachial plexus block: a case series. J Child Orthop. 2011;5(1):5–9.
70. Roberts S. Ultrasonographic guidance in pediatric regional anesthesia. Part 2: techniques. Paediatr Anaesth. 2006;16(11):1112–24.
71. Tsui BC, Pillay JJ. Evidence-based medicine: assessment of ultrasound imaging for regional anesthesia in infants, children, and adolescents. Reg Anesth Pain Med. 2010;35(2 Suppl):S47–54.
72. Tsui BC, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents: a review of current literature and its application in the practice of neuraxial blocks. Anesthesiology. 2010;112(3):719–28.
73. Tsui B, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents: a review of current literature and its application in the practice of extremity and trunk blocks. Anesthesiology. 2010;112(2):473–92.
74. Mariano ER, et al. Feasibility of ultrasound-guided peripheral nerve block catheters for pain control on pediatric medical missions in developing countries. Paediatr Anaesth. 2008;18(7):598–601.
75. Baldi C, et al. Ultrasound guidance for locoregional anesthesia: a review. Minerva Anestesiol. 2007;73(11):587–93.
76. Johr M. Practical pediatric regional anesthesia. Curr Opin Anaesthesiol. 2013;26(3):327–32.
77. Moore DL, Ding L, Sadhasivam S. Novel real-time feedback and integrated simulation model for teaching and evaluating ultrasound-guided regional anesthesia skills in pediatric anesthesia trainees. Paediatr Anaesth. 2012;22(9):847–53.
78. Liu Y, Glass NL, Power RW. Technical communication: new teaching model for practicing ultrasound-guided regional anesthesia techniques: no perishable food products! Anesth Analg. 2010;110(4):1233–5.
79. Ivani G, Mosseti V. Pediatric regional anesthesia. Minerva Anestesiol. 2009;75(10):577–83.
80. Arul GS, Spicer RD. Where should paediatric surgery be performed? Arch Dis Child. 1998;79(1):65–70; discussion 70–2
81. Taenzer A, et al. Interscalene brachial plexus blocks under general anesthesia in children: is this safe practice?: a report from the Pediatric Regional Anesthesia Network (PRAN). Reg Anesth Pain Med. 2014;39(6):502–5.
82. Tobias JD, Flannagan J. Regional anesthesia in the preterm neonate. Clin Pediatr (Phila). 1992;31(11):668–71.
83. Mazoit JX. Pharmacokinetic/pharmacodynamic modeling of anesthetics in children: therapeutic implications. Paediatr Drugs. 2006;8(3):139–50.
84. Casati A, Putzu M. Bupivacaine, levobupivacaine and ropivacaine: are they clinically different? Best Pract Res Clin Anaesthesiol. 2005;19(2):247–68.
85. Gunter JB. Benefit and risks of local anesthetics in infants and children. Paediatr Drugs. 2002;4(10):649–72.
86. Mather LE. The acute toxicity of local anesthetics. Expert Opin Drug Metab Toxicol. 2010;6(11):1313–32.
87. Santos AC, DeArmas PI. Systemic toxicity of levobupivacaine, bupivacaine, and ropivacaine during continuous intravenous infusion to nonpregnant and pregnant ewes. Anesthesiology. 2001;95(5):1256–64.
88. Groban L, et al. Cardiac resuscitation after incremental overdosage with lidocaine, bupivacaine, levobupivacaine, and ropivacaine in anesthetized dogs. Anesth Analg. 2001;92(1):37–43.
89. De Negri P, et al. New local anesthetics for pediatric anesthesia. Curr Opin Anaesthesiol. 2005;18(3):289–92.
90. Fuzier R, et al. Adverse drug reactions to local anaesthetics: a review of the French pharmacovigilance database. Drug Saf. 2009;32(4):345–56.
91. Copeland SE, et al. The effects of general anesthesia on the central nervous and cardiovascular system toxicity of local anesthetics. Anesth Analg. 2008;106(5):1429–39.
92. Corman SL, Skledar SJ. Use of lipid emulsion to reverse local anesthetic-induced toxicity. Ann Pharmacother. 2007;41(11):1873–7.
93. Mather LE, Copeland SE, Ladd LA. Acute toxicity of local anesthetics: underlying pharmacokinetic and pharmacodynamic concepts. Reg Anesth Pain Med. 2005;30(6):553–66.
94. Fein JA, Gorelick MH. The decision to use topical anesthetic for intravenous insertion in the pediatric emergency department. Acad Emerg Med. 2006;13(3):264–8.

95. Houck CS, Sethna NF. Transdermal analgesia with local anesthetics in children: review, update and future directions. Expert Rev Neurother. 2005;5(5):625–34.
96. Sohmer B, et al. EMLA cream is an effective topical anesthetic for bronchoscopy. Can Respir J. 2004;11(8):587–8.
97. Kundu S, Achar S. Principles of office anesthesia: part II. Topical anesthesia. Am Fam Physician. 2002;66(1):99–102.
98. Weise KL, Nahata MC. EMLA for painful procedures in infants. J Pediatr Health Care. 2005;19(1):42–7; quiz 48–9
99. Neal JM, et al. The second American Society of Regional Anesthesia and pain medicine evidence-based medicine assessment of ultrasound-guided regional anesthesia: executive summary. Reg Anesth Pain Med. 2016;41(2):181–94.
100. Neal JM, et al. The ASRA evidence-based medicine assessment of ultrasound-guided regional anesthesia and pain medicine: executive summary. Reg Anesth Pain Med. 2010;35(2 Suppl):S1–9.
101. Markakis DA. Regional anesthesia in pediatrics. Anesthesiol Clin North Am. 2000;18(2):355–81. vii
102. Wilder RT. Local anesthetics for the pediatric patient. Pediatr Clin N Am. 2000;47(3):545–58.
103. Pierluisi GJ, Terndrup TE. Influence of topical anesthesia on the sedation of pediatric emergency department patients with lacerations. Pediatr Emerg Care. 1989;5(4):211–5.
104. Bonadio WA, Wagner V. Efficacy of TAC topical anesthetic for repair of pediatric lacerations. Am J Dis Child. 1988;142(2):203–5.
105. White WB, Iserson KV, Criss E. Topical anesthesia for laceration repair: tetracaine versus TAC (tetracaine, adrenaline, and cocaine). Am J Emerg Med. 1986;4(4):319–22.
106. Schaffer DJ. Clinical comparison of TAC anesthetic solutions with and without cocaine. Ann Emerg Med. 1985;14(11):1077–80.
107. Pryor GJ, Kilpatrick WR, Opp DR. Local anesthesia in minor lacerations: topical TAC vs lidocaine infiltration. Ann Emerg Med. 1980;9(11):568–71.
108. Ehrenstrom Reiz GM, Reiz SL. EMLA—a eutectic mixture of local anaesthetics for topical anaesthesia. Acta Anaesthesiol Scand. 1982;26(6):596–8.
109. Wahlgren CF, Quiding H. Depth of cutaneous analgesia after application of a eutectic mixture of the local anesthetics lidocaine and prilocaine (EMLA cream). J Am Acad Dermatol. 2000;42(4):584–8.
110. Bjerring P, Arendt-Nielsen L. Depth and duration of skin analgesia to needle insertion after topical application of EMLA cream. Br J Anaesth. 1990;64(2):173–7.
111. Shachor-Meyouhas Y, Galbraith R, Shavit I. Application of topical analgesia in triage: a potential for harm. J Emerg Med. 2008;35(1):39–41.
112. Kopecky EA, et al. Safety and pharmacokinetics of EMLA in the treatment of postburn pruritus in pediatric patients: a pilot study. J Burn Care Rehabil. 2001;22(3):235–42.
113. Brisman M, et al. Methaemoglobin formation after the use of EMLA cream in term neonates. Acta Paediatr. 1998;87(11):1191–4.
114. Choi EK, et al. The use of EMLA cream reduces the pain of skin puncture associated with caudal block in children. Korean J Anesthesiol. 2016;69(2):149–54.
115. Rogers TL, Ostrow CL. The use of EMLA cream to decrease venipuncture pain in children. J Pediatr Nurs. 2004;19(1):33–9.
116. Jacobson RM, et al. Making vaccines more acceptable—methods to prevent and minimize pain and other common adverse events associated with vaccines. Vaccine. 2001;19(17–19):2418–27.
117. Datta S. Use of a eutectic mixture of local anesthesia in newborns. Indian Pediatr. 1999;36(7):726–7.
118. Butler-O'Hara M, LeMoine C, Guillet R. Analgesia for neonatal circumcision: a randomized controlled trial of EMLA cream versus dorsal penile nerve block. Pediatrics. 1998;101(4):E5.
119. Zempsky WT, Karasic RB. EMLA versus TAC for topical anesthesia of extremity wounds in children. Ann Emerg Med. 1997;30(2):163–6.
120. Vetter TR. A comparison of EMLA cream versus nitrous oxide for pediatric venous cannulation. J Clin Anesth. 1995;7(6):486–90.
121. Steward DJ. Eutectic mixture of local anesthetics (EMLA): what is it? What does it do? J Pediatr. 1993;122(5 Pt 2):S21–3.
122. Robieux IC, et al. The feasibility of using EMLA (eutectic mixture of local anaesthetics) cream in pediatric outpatient clinics. Can J Hosp Pharm. 1990;43(5):235–6, xxxii.
123. Dutta S. Use of eutectic mixture of local anesthetics in children. Indian J Pediatr. 1999;66(5):707–15.
124. Brady-Fryer B, Wiebe N, Lander JA. Pain relief for neonatal circumcision. Cochrane Database Syst Rev. 2004;4:CD004217.
125. Shavit I, et al. Lidocaine-based topical anesthetic with disinfectant (LidoDin) versus EMLA for venipuncture: a randomized controlled trial. Clin J Pain. 2009;25(8):711–4.
126. Altman DA, Gildenberg SR. High-energy pulsed light source hair removal device used to evaluate the onset of action of a new topical anesthetic. Dermatol Surg. 1999;25(10):816–8.
127. Eichenfield LF, et al. A clinical study to evaluate the efficacy of ELA-Max (4% liposomal lidocaine) as compared with eutectic mixture of local anesthetics cream for pain reduction of venipuncture in children. Pediatrics. 2002;109(6):1093–9.
128. Spanos S, et al. Jet injection of 1% buffered lidocaine versus topical ELA-Max for anesthesia before peripheral intravenous catheterization in children:

a randomized controlled trial. Pediatr Emerg Care. 2008;24(8):511–5.
129. Smith DP, Gjellum M. The efficacy of LMX versus EMLA for pain relief in boys undergoing office meatotomy. J Urol. 2004;172(4 Pt 2):1760–1.
130. Luhmann J, et al. A comparison of buffered lidocaine versus ELA-Max before peripheral intravenous catheter insertions in children. Pediatrics. 2004;113(3 Pt 1):e217–20.
131. Kleiber C, et al. Topical anesthetics for intravenous insertion in children: a randomized equivalency study. Pediatrics. 2002;110(4):758–61.
132. Zhijun C, et al. Therapeutic experience from 1428 patients with pediatric tracheobronchial foreign body. J Pediatr Surg. 2008;43(4):718–21.
133. Randell T, et al. Topical anaesthesia of the nasal mucosa for fibreoptic airway endoscopy. Br J Anaesth. 1992;68(2):164–7.
134. Wolfe TR, Fosnocht DE, Linscott MS. Atomized lidocaine as topical anesthesia for nasogastric tube placement: a randomized, double-blind, placebo-controlled trial. Ann Emerg Med. 2000;35(5):421–5.
135. West HH. Topical anesthesia for nasogastric tube placement. Ann Emerg Med. 1982;11(11):645.
136. Wangemann BU, Jantzen JP. Fiberoptic intubation of neurosurgical patients. Neurochirurgia (Stuttg). 1993;36(4):117–22.
137. Gjonaj ST, Lowenthal DB, Dozor AJ. Nebulized lidocaine administered to infants and children undergoing flexible bronchoscopy. Chest. 1997;112(6):1665–9.
138. Ohzeki K, et al. Local anesthetic cream prepared from lidocaine-tetracaine eutectic mixture. Yakugaku Zasshi. 2008;128(4):611–6.
139. Hakim OM, El-Hag YG, Haikal MA. Strabismus surgery under augmented topical anesthesia. J AAPOS. 2005;9(3):279–84.
140. Haasio J, et al. Topical anaesthesia of gingival mucosa by 5% eutectic mixture of lignocaine and prilocaine or by 10% lignocaine spray. Br J Oral Maxillofac Surg. 1990;28(2):99–101.
141. Suresh S, Wheeler M. Practical pediatric regional anesthesia. Anesthesiol Clin North Am. 2002;20(1):83–113.
142. Suresh S, et al. Postoperative pain relief in children undergoing tympanomastoid surgery: is a regional block better than opioids? Anesth Analg. 2002;94(4):859–62.
143. Ahn HJ, et al. Effect of preoperative skull block on pediatric moyamoya disease. J Neurosurg Pediatr. 2008;2(1):37–41.
144. Wang SM. An integrative approach for treating postherpetic neuralgia—a case report. Pain Pract. 2007;7(3):274–8.
145. Aldoori MI, Baird RN. Local neurological complication during carotid endarterectomy. J Cardiovasc Surg. 1988;29(4):432–6.
146. Suresh S, et al. Regional anaesthesia to improve pain outcomes in paediatric surgical patients: a qualitative systematic review of randomized controlled trials. Br J Anaesth. 2014;113(3):375–90.
147. Salloum ML, et al. Combined use of infraorbital and external nasal nerve blocks for effective perioperative pain control during and after cleft lip repair. Cleft Palate Craniofac J. 2009;46(6):629–35.
148. Szperka CL, Gelfand AA, Hershey AD. Patterns of use of peripheral nerve blocks and trigger point injections for pediatric headache: results of a survey of the American Headache Society Pediatric and Adolescent Section. Headache. 2016;56(10):1597–607.
149. Wiewrodt D, Wagner W. Long-term significance of injury to the supraorbital or supratrochlear nerves during frontoorbital advancement in infancy. Childs Nerv Syst. 2009;25(12):1589–91.
150. Barnett P. Alternatives to sedation for painful procedures. Pediatr Emerg Care. 2009;25(6):415–9; quiz 420–2.
151. Ismail AR, et al. Regional nerve block of the upper eyelid in oculoplastic surgery. Eur J Ophthalmol. 2006;16(4):509–13.
152. Kriwanek KL, et al. Axillary block for analgesia during manipulation of forearm fractures in the pediatric emergency department a prospective randomized comparative trial. J Pediatr Orthop. 2006;26(6):737–40.
153. Desbordes J, et al. Brachial plexus anesthesia via an axillary route for emergency surgery: comparison of three approach methods. Ann Fr Anesth Reanim. 1998;17(7):674–80.
154. Ponde VC. Continuous infraclavicular brachial plexus block: a modified technique to better secure catheter position in infants and children. Anesth Analg. 2008;106(1):94–6.
155. Loland VJ, et al. Ultrasound-guided perineural catheter and local anesthetic infusion in the perioperative management of pediatric limb salvage: a case report. Paediatr Anaesth. 2009;19(9):905–7.
156. Amiri HR, Espandar R. Upper extremity surgery in younger children under ultrasound-guided supraclavicular brachial plexus block: a case series. J Child Orthop. 2010;4(4):315–9.
157. de Jose Maria B, Tielens LK. Vertical infraclavicular brachial plexus block in children: a preliminary study. Paediatr Anaesth. 2004;14(11):931–5.
158. Wall JJ. Axillary nerve blocks. Am Fam Physician. 1975;11(5):135–42.
159. Ross DM, Williams DO. Combined axillary plexus block and basal sedation for cardiac catheterization in young children. Br Heart J. 1970;32(2):195–7.
160. Torrillo TM, Rosenblatt MA. Ultrasound-guided interscalene catheters performed under general anesthesia in a patient with Huntington's disease. Minerva Anestesiol. 2010;76(8):645–8.
161. Koscielniak-Nielsen ZJ, Rasmussen H, Hesselbjerg L. Long-axis ultrasound imaging of the nerves and

advancement of perineural catheters under direct vision: a preliminary report of four cases. Reg Anesth Pain Med. 2008;33(5):477–82.
162. Fredrickson MJ. Ultrasound-assisted interscalene catheter placement in a child. Anaesth Intensive Care. 2007;35(5):807–8.
163. Jan van Geffen G, Tielens L, Gielen M. Ultrasound-guided interscalene brachial plexus block in a child with femur fibula ulna syndrome. Paediatr Anaesth. 2006;16(3):330–2.
164. Lee JH, et al. Ultrasound-guided interscalene brachial plexus block in a pediatric patient with acute hepatitis—a case report. Korean J Anesthesiol. 2012;62(6):568–70.
165. Lonnqvist PA, Hildingsson U. The caudal boundary of the thoracic paravertebral space. A study in human cadavers. Anaesthesia. 1992;47(12):1051–2.
166. Lonnqvist PA, Olsson GL. Paravertebral vs epidural block in children. Effects on postoperative morphine requirement after renal surgery. Acta Anaesthesiol Scand. 1994;38(4):346–9.
167. Hutchins J, et al. Postoperative pain control with paravertebral catheters after pediatric total pancreatectomy and islet autotransplantation: a retrospective cohort study. Paediatr Anaesth. 2016;26(3):315–20.
168. Visoiu M, Cassara A, Yang CI. Bilateral paravertebral blockade (T7-10) versus incisional local anesthetic administration for pediatric laparoscopic cholecystectomy: a prospective, randomized clinical study. Anesth Analg. 2015;120(5):1106–13.
169. Hall Burton DM, Boretsky KR. A comparison of paravertebral nerve block catheters and thoracic epidural catheters for postoperative analgesia following the Nuss procedure for pectus excavatum repair. Paediatr Anaesth. 2014;24(5):516–20.
170. Berta E, et al. Single injection paravertebral block for renal surgery in children. Paediatr Anaesth. 2008;18(7):593–7.
171. Loftus PD, et al. Paravertebral regional blocks decrease length of stay following surgery for pectus excavatum in children. J Pediatr Surg. 2016;51(1):149–53.
172. Karmakar MK, et al. Continuous thoracic paravertebral infusion of bupivacaine for pain management in patients with multiple fractured ribs. Chest. 2003;123(2):424–31.
173. Chung YT, et al. Inadvertent epidural spread after subpleural-paravertebral block with 0.5% bupivacaine. Ma Zui Xue Za Zhi. 1989;27(4):381–4.
174. Lall NG, Sharma SR. "Clicking" pneumothorax following thoracic paravertebral block. Case report. Br J Anaesth. 1971;43(4):415–7.
175. Tenicela R, Pollan SB. Paravertebral-peridural block technique: a unilateral thoracic block. Clin J Pain. 1990;6(3):227–34.
176. Lonnqvist PA, et al. Paravertebral blockade. Failure rate and complications. Anaesthesia. 1995;50(9):813–5.
177. Naja Z, Lonnqvist PA. Somatic paravertebral nerve blockade. Incidence of failed block and complications. Anaesthesia. 2001;56(12):1184–8.
178. Conaghan P, et al. Efficacy of transversus abdominis plane blocks in laparoscopic colorectal resections. Surg Endosc. 2010;24(10):2480–4.
179. Araco A, et al. Transversus abdominis plane block reduces the analgesic requirements after abdominoplasty with flank liposuction. Ann Plast Surg. 2010;65(4):385–8.
180. Niraj G, et al. Analgesic efficacy of ultrasound-guided transversus abdominis plane block in patients undergoing open appendicectomy. Br J Anaesth. 2009;103(4):601–5.
181. Niraj G, Kelkar A, Fox AJ. Oblique sub-costal transversus abdominis plane (TAP) catheters: an alternative to epidural analgesia after upper abdominal surgery. Anaesthesia. 2009;64(10):1137–40.
182. Belavy D, et al. Ultrasound-guided transversus abdominis plane block for analgesia after Caesarean delivery. Br J Anaesth. 2009;103(5):726–30.
183. Suresh S, Chan VW. Ultrasound guided transversus abdominis plane block in infants, children and adolescents: a simple procedural guidance for their performance. Paediatr Anaesth. 2009;19(4):296–9.
184. Taylor LJ, et al. Children with spinal dysraphism: transversus abdominis plane (TAP) catheters to the rescue! Paediatr Anaesth. 2010;20(10):951–4.
185. Hernandez MA, Vecchione T, Boretsky K. Dermatomal spread following posterior transversus abdominis plane block in pediatric patients: our initial experience. Paediatr Anaesth. 2017;27(3):300–4.
186. Hamill JK, et al. Rectus sheath and transversus abdominis plane blocks in children: a systematic review and meta-analysis of randomized trials. Paediatr Anaesth. 2016;26(4):363–71.
187. Faasse MA, et al. Perioperative effects of caudal and transversus abdominis plane (TAP) blocks for children undergoing urologic robot-assisted laparoscopic surgery. J Pediatr Urol. 2015;11(3):121 e1–7.
188. Long JB, et al. Transversus abdominis plane block in children: a multicenter safety analysis of 1994 cases from the PRAN (Pediatric Regional Anesthesia Network) database. Anesth Analg. 2014;119(2):395–9.
189. Mai CL, Young MJ, Quraishi SA. Clinical implications of the transversus abdominis plane block in pediatric anesthesia. Paediatr Anaesth. 2012;22(9):831–40.
190. Carney J, et al. Ipsilateral transversus abdominis plane block provides effective analgesia after appendectomy in children: a randomized controlled trial. Anesth Analg. 2010;111(4):998–1003.
191. Jankovic ZB, du Feu FM, McConnell P. An anatomical study of the transversus abdominis plane block: location of the lumbar triangle of Petit and adjacent nerves. Anesth Analg. 2009;109(3):981–5.

192. Ra YS, et al. The analgesic effect of the ultrasound-guided transverse abdominis plane block after laparoscopic cholecystectomy. Korean J Anesthesiol. 2010;58(4):362–8.
193. McDonnell JG, et al. The analgesic efficacy of transversus abdominis plane block after cesarean delivery: a randomized controlled trial. Anesth Analg. 2008;106(1):186–91.
194. Petersen PL, et al. The transversus abdominis plane block: a valuable option for postoperative analgesia? A topical review. Acta Anaesthesiol Scand. 2010;54(5):529–35.
195. Carney J, et al. The transversus abdominis plane block provides effective postoperative analgesia in patients undergoing total abdominal hysterectomy. Anesth Analg. 2008;107(6):2056–60.
196. Broadman LM. Blocks and other techniques pediatric surgeons can employ to reduce postoperative pain in pediatric patients. Semin Pediatr Surg. 1999;8(1):30–3.
197. Jagannathan N, et al. Unilateral groin surgery in children: will the addition of an ultrasound-guided ilioinguinal nerve block enhance the duration of analgesia of a single-shot caudal block? Paediatr Anaesth. 2009;19(9):892–8.
198. Willschke H, et al. Ultrasonography for ilioinguinal/iliohypogastric nerve blocks in children. Br J Anaesth. 2005;95(2):226–30.
199. Frigon C, et al. Bowel hematoma following an iliohypogastric-ilioinguinal nerve block. Paediatr Anaesth. 2006;16(9):993–6.
200. Derrick JL, Aun CS. Transient femoral nerve palsy after ilioinguinal block. Anaesth Intensive Care. 1996;24(1):115.
201. Lander J, et al. Comparison of ring block, dorsal penile nerve block, and topical anesthesia for neonatal circumcision: a randomized controlled trial. JAMA. 1997;278(24):2157–62.
202. Howard CR, et al. A randomized, controlled trial of a eutectic mixture of local anesthetic cream (lidocaine and prilocaine) versus penile nerve block for pain relief during circumcision. Am J Obstet Gynecol. 1999;181(6):1506–11.
203. Serour F, et al. Efficacy of EMLA cream prior to dorsal penile nerve block for circumcision in children. Acta Anaesthesiol Scand. 1998;42(2):260–3.
204. Lehr VT, et al. Lidocaine 4% cream compared with lidocaine 2.5% and prilocaine 2.5% or dorsal penile block for circumcision. Am J Perinatol. 2005;22(5):231–7.
205. Serour F, Mori J, Barr J. Optimal regional anesthesia for circumcision. Anesth Analg. 1994;79(1):129–31.
206. Fontaine P, Dittberner D, Scheltema KE. The safety of dorsal penile nerve block for neonatal circumcision. J Fam Pract. 1994;39(3):243–8.
207. Lee JJ, Forrester P. EMLA for postoperative analgesia for day case circumcision in children. A comparison with dorsal nerve of penis block. Anaesthesia. 1992;47(12):1081–3.
208. Carlsson P, Svensson J. The duration of pain relief after penile block to boys undergoing circumcision. Acta Anaesthesiol Scand. 1984;28(4):432–4.
209. Soliman MG, Tremblay NA. Nerve block of the penis for postoperative pain relief in children. Anesth Analg. 1978;57(4):495–8.
210. Dalens B. Some current controversies in paediatric regional anaesthesia. Curr Opin Anaesthesiol. 2006;19(3):301–8.
211. Gul M, et al. Epinephrine injection associated scrotal skin necrosis. Case Rep Urol. 2015;2015:187831.
212. Johr M, Berger TM. Recent developments in paediatric regional anaesthesia. Curr Opin Anaesthesiol. 2004;17(3):211–5.
213. Faraoni D, et al. Does ultrasound guidance improve the efficacy of dorsal penile nerve block in children? Paediatr Anaesth. 2010;20(10):931–6.
214. Boybeyi O, et al. Investigation of the effect of dorsal penile block to penile tissue. J Pediatr Urol. 2015;11(5):268 e1–5.
215. O'Sullivan MJ, Mislovic B, Alexander E. Dorsal penile nerve block for male pediatric circumcision—randomized comparison of ultrasound-guided vs anatomical landmark technique. Paediatr Anaesth. 2011;21(12):1214–8.
216. Beyaz SG, Tokgoz O, Tufek A. Regional anaesthesia in paediatric surgery: results of 2200 children. J Pak Med Assoc. 2011;61(8):782–6.
217. Stein AL, et al. Updates in pediatric regional anesthesia and its role in the treatment of acute pain in the ambulatory setting. Curr Pain Headache Rep. 2017;21(2):11.
218. Newman PJ, Bushnell TG, Radford P. The effect of needle size and type in paediatric caudal analgesia. Paediatr Anaesth. 1996;6(6):459–61.
219. Goldschneider KR, Brandom BW. The incidence of tissue coring during the performance of caudal injection in children. Reg Anesth Pain Med. 1999;24(6):553–6.
220. Tsui BC, Berde CB. Caudal analgesia and anesthesia techniques in children. Curr Opin Anaesthesiol. 2005;18(3):283–8.
221. Verghese ST, et al. Caudal anesthesia in children: effect of volume versus concentration of bupivacaine on blocking spermatic cord traction response during orchidopexy. Anesth Analg. 2002;95(5):1219–23.
222. Seefelder C. The caudal catheter in neonates: where are the restrictions? Curr Opin Anaesthesiol. 2002;15(3):343–8.
223. Gunter JB, Eng C. Thoracic epidural anesthesia via the caudal approach in children. Anesthesiology. 1992;76(6):935–8.
224. van Niekerk J, et al. Epidurography in premature infants. Anaesthesia. 1990;45(9):722–5.
225. Rasch DK, et al. Lumbar and thoracic epidural analgesia via the caudal approach for postoperative pain relief in infants and children. Can J Anaesth. 1990;37(3):359–62.

226. Bosenberg AT, et al. Thoracic epidural anesthesia via caudal route in infants. Anesthesiology. 1988;69(2):265–9.
227. Savolaine ER, et al. Anatomy of the human lumbar epidural space: new insights using CT-epidurography. Anesthesiology. 1988;68(2):217–20.
228. Blanco D, et al. Thoracic epidural anesthesia by the caudal route in pediatric anesthesia: age is a limiting factor. Rev Esp Anestesiol Reanim. 1994;41(4):214–6.
229. Taenzer AH, et al. Asleep versus awake: does it matter?: pediatric regional block complications by patient state: a report from the Pediatric Regional Anesthesia Network. Reg Anesth Pain Med. 2014;39(4):279–83.
230. Lonnqvist PA. Is ultrasound guidance mandatory when performing paediatric regional anaesthesia? Curr Opin Anaesthesiol. 2010;23(3):337–41.
231. Schultz-Machata AM, Weiss M, Becke K. What's new in pediatric acute pain therapy? Curr Opin Anaesthesiol. 2014;27(3):316–22.
232. Verghese ST, Hannallah RS. Acute pain management in children. J Pain Res. 2010;3:105–23.
233. Lonnqvist PA, Morton NS. Paediatric day-case anaesthesia and pain control. Curr Opin Anaesthesiol. 2006;19(6):617–21.

Head and Neck Regional Anesthesia

16

Desiree Persaud and Sébastien Garneau

Introduction

Even though head and neck blocks are among the easiest to perform due to constant and reliable landmarks, they are still infrequently used by anesthesiologists in the operating room. This is in part because general anesthesia offers a safe and easy alternative for most surgeries involving these anatomical areas. Nonetheless, neural blockade has become the mainstay of anesthetic techniques for (a) most ophthalmologic cases, (b) neurosurgical procedures or carotid endarterectomies where intraoperative neurological assessment is required, and (c) a safe alternative for patients with low functional reserve that would have poor tolerance to general anesthesia. These blocks can also prove to be useful for the anesthesiologists themselves, in techniques for airway management in awake patients.

In this chapter, we review blocks that are relevant to the anesthesiologist working in the operating room, focusing on acute and surgical pain management. Many other resources will provide you with information about blocks of the head and neck for chronic pain patients.

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The Scalp Block/Block of the Head

An 84-year-old male is scheduled for an emergent evacuation of a large left frontotemporal subdural hematoma. He presented to the emergency department for a complaint of increasing ataxia and memory disturbance. There is no midline shift and he is calm and cooperative. His past medical history includes severe chronic obstructive pulmonary disease and mild ischemic cardiomyopathy.

The surgeon plans to evacuate the hematoma through two burr holes and leave a drain in situ. The patient's head will rest on a horseshoe headrest.

Due to the patient's comorbidities, general endotracheal tube anesthesia could compromise the patient's respiratory and cardiovascular systems. Scalp blocks provide reliable surgical anesthesia and would be a viable option in this scenario.

The scalp block is not a block in itself but a combination of multiple nerve blocks, namely, the supraorbital, supratrochlear, greater auricular, auriculotemporal, and greater and lesser occipital nerve blocks.

Anatomy

The innervation of the scalp can be divided into anterior and posterior distributions: anteriorly,

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_16

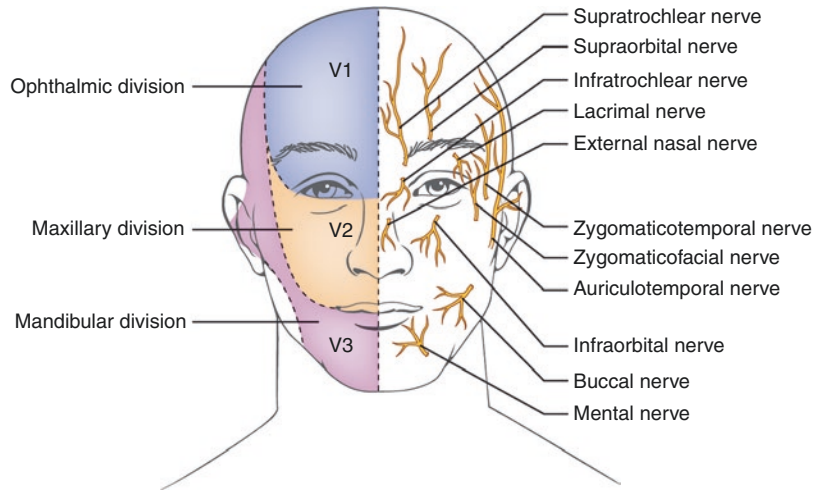


Fig. 16.1 Innervation territories of the scalp

from branches of the trigeminal nerve (fifth cranial nerve), and, posteriorly, from branches of the cervical plexus and posterior spinal rami (Fig. 16.1).

Trigeminal Nerve Branches

All originating from the gasserian ganglion, the branches of the trigeminal nerve are the supraorbital and supratrochlear nerves (branches of V1—traveling within the conus of the eye) innervating the forehead, and the zygomaticotemporal nerve (V2) and the auriculotemporal nerve (V3) innervating the temporal area, and, for the latter nerve, the anterior part of the ear.

Occipital Nerves

The greater auricular nerve and lesser occipital (from the superficial cervical plexus) and the greater occipital nerve (originating from the dorsal primary ramus of C2) are responsible for the sensory innervation of the posterior aspect of the head (Fig. 16.2). The greater occipital nerve innervation area covers the skin located between the nuchal line and the vertex of the skull, whereas the lesser occipital and greater auricular innervate the skin behind/below the ear and in front (over the parotid) of the ear, respectively.

Indications

1. Awake craniotomies [1]
2. Stereotactic biopsies
3. Attenuation/prevention of the sympathetic stimulation and stress response associated with Mayfield pins insertion [2, 3]
4. Postoperative analgesia for craniotomies [4–6]
5. Plastic procedures of the scalp [7]

Local Anesthetics

Most local anesthetics are suitable for facial nerve blocks, and less concentrated local anesthetics will work well on these sensory nerves; 0.5% ropivacaine, 0.25% bupivacaine, or 1% lidocaine would be good choices. Epinephrine is a good adjunct to identify an inadvertent intravascular injection and to lower the possibility of local bleeding at the puncture site.

Techniques

As with all neural blockade techniques, the patient should be monitored, and appropriate resuscitative drugs and equipment should be readily available. A strict sterile technique is not always possible (for blocks performed in hair-covered areas), but the technique should be as aseptic as possible.

Supraorbital and Supratrochlear

1. After palpation of the supraorbital foramen, slightly medial to the plane of the centered

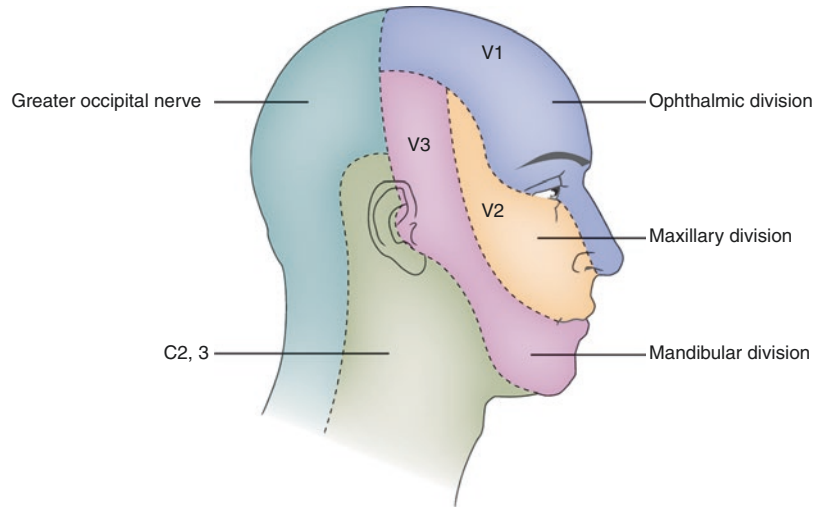
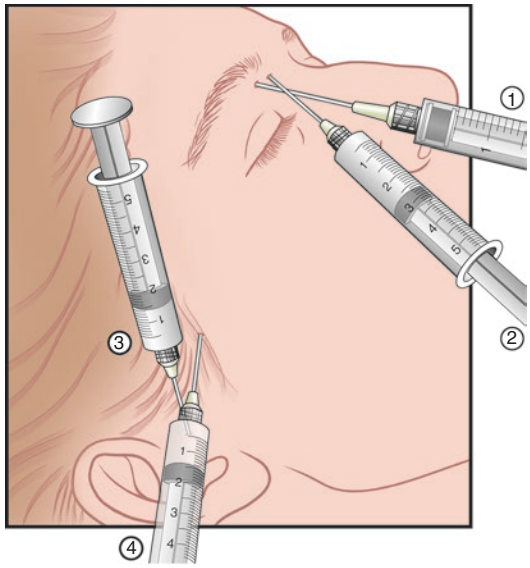


Fig. 16.2 Specific areas of innervation of the scalp



1. Supraorbital
2. Supratrochlear
3. Auricotemporal
4. Zygomaticotemporal

Fig. 16.3 Anterior scalp blocks

pupil on the superior orbital rim (approximately 2–2.5 cm lateral to midline), a 25-G 30-mm needle is inserted above or below the eyebrow toward the foramen (but not entering it) (Fig. 16.3).

2. A subcutaneous infiltration of 2–3 mL of local anesthetic solution suffices, followed by local pressure to prevent hematoma formation.
3. The supratrochlear nerve can be specifically blocked at the intersection of the superior orbital rim and the nasal bridge, with the needle aiming at this intersection, pulled back slightly after bony contact, and then injecting 1 mL of solution (Fig. 16.3). An alternative is to infiltrate local anesthetics subcutaneously from the supratrochlear notch to the nasal bridge.

Auriculotemporal

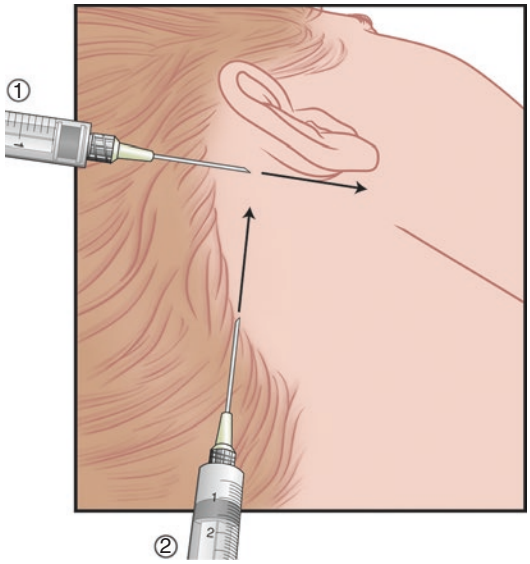
A 25-G needle is inserted just between the tragus and the superficial temporal artery, just on the posterior aspect of the zygomatic bone. Three to five milliliter of anesthetic solution is injected (Fig. 16.3).

Zygomaticotemporal

A 25-G needle is inserted 2.5 cm anterior to the tragus, perpendicular to all planes, and injection of 3–5 mL of anesthetic solution is performed deep to the fascia and superficially as the needle is withdrawn (Fig. 16.3).

Greater Auricular

A 25-G needle is used to create a subcutaneous wheal of local anesthetics following the posterior aspect of the ear, over the mastoid process, with an approximate volume of 5 mL (Fig. 16.4).



1. Greater auricular
2. Occipital

Fig. 16.4 Posterior scalp blocks

Greater and Lesser Occipital Nerve

1. After palpation of the occipital artery, approximately midway between the occipital protuberance and the mastoid process, a bent 25-G needle is inserted lateral to the occipital artery toward the mastoid process.
2. A subcutaneous infiltration is made lateral to this point, following the nuchal line, using 3–5 mL of solution, after negative aspiration for blood, as the needle is withdrawn (Fig. 16.4).

Risks and Complications

All these blocks yield risks related to local anesthetic hypersensitivity, hematoma formation, and infection. The supratrochlear and supraorbital nerve blocks have an additional risk of intraneural injection. The clinician should recognize the risk of intravascular injection with the auriculotemporal and greater occipital nerve blocks.

Ophthalmologic Blocks

In many countries, the blocks of or around the eye are performed by the ophthalmologist. This section focuses on the two more relevant techniques for the anesthesiologist, the retrobulbar and peribulbar blocks. Those blocks are not suitable for open globe surgery, and the reader should note that less invasive surgical techniques used nowadays also permit topical anesthesia to be sufficient for some lens and anterior chamber procedures.

Anatomy

Like most structures of the head, branches from the trigeminal nerve give sensory innervation to the eye and adnexa. However, ophthalmologic surgery requires immobility as much as insensibility.

1. Motor innervation is supplied by the oculomotor nerve (CN III), trochlear nerve (CN IV), and abducens nerve (CN VI), as well as by the temporal division of the facial nerve (CN VII) to the orbicularis oculi muscle.
2. Sensory innervation is provided by branches emerging from the ophthalmic (V1) branch of the trigeminal nerve through the ciliary ganglion.
3. The branches of the ophthalmic division of the trigeminal and most of these previously mentioned nerves travel in the orbit behind the globe, which provides a space for local anesthetic deposition (Fig. 16.5). All the nerves can thus be blocked here except for the temporal division of the facial nerve. The main difference between the two discussed techniques is the site of injection of the local anesthetic solution, intraconal (inside the cone formed by the rectus muscles) for the retrobulbar or extraconal for peribulbar. The reader should note that the cone is anatomically incomplete and there is no anatomic structure linking the extrinsic ocular muscles together [8]. Clinically, the peribulbar block is considered safer [9] than the retrobulbar, since the structures at risk are intraconal. It also has the advantage of providing complete akinesia of the eye adnexa, owing to direct diffusion to the eyelids. However, the success rate of the peribulbar block may be lower (this is controversial, many studies show similar success rates [9, 10]) and necessitates a larger volume

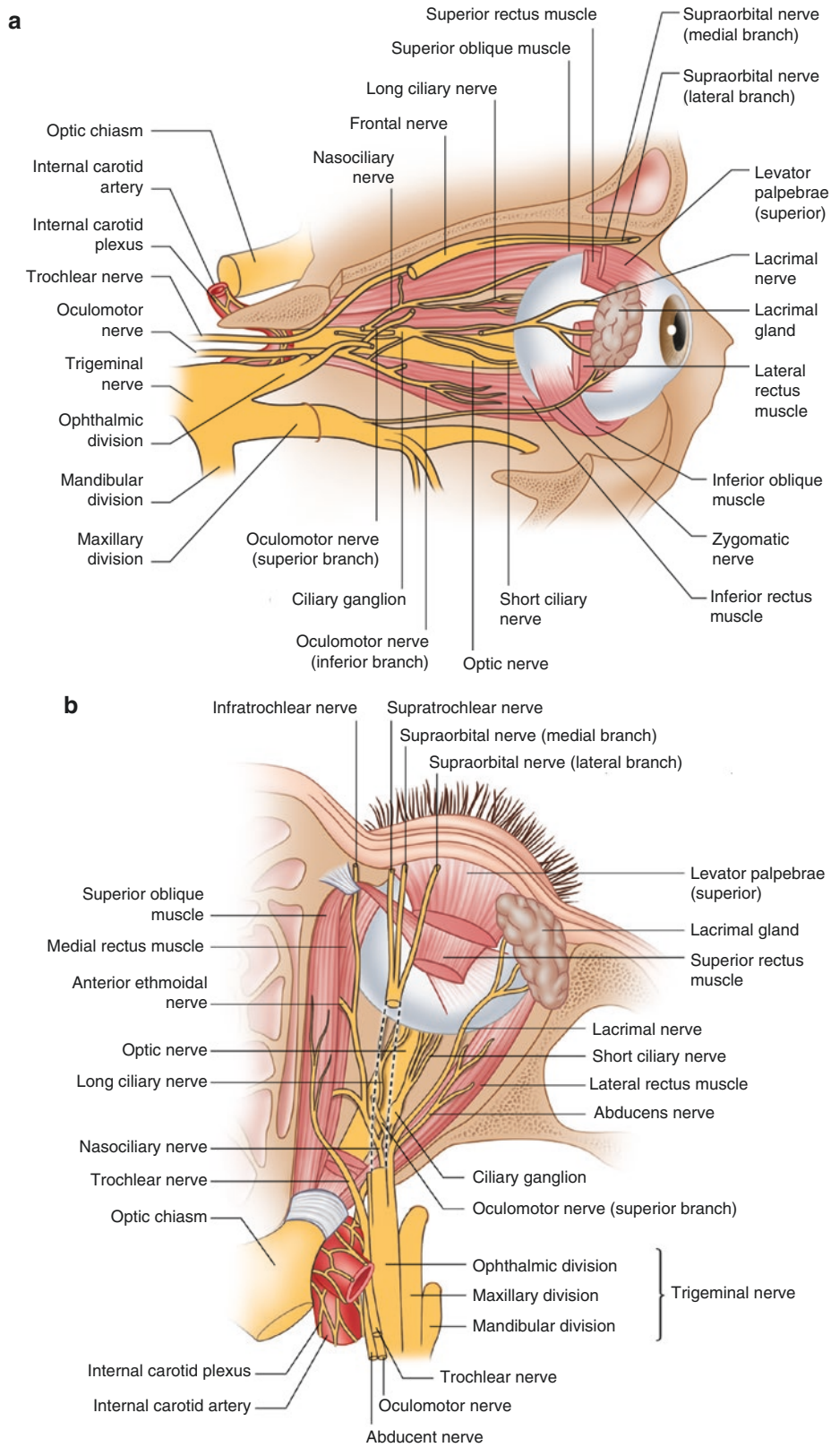


Fig. 16.5 (**a**, **b**) Innervation of the eye and adnexa

of solution compared to the retrobulbar block. The onset of the peribulbar block is also slower than that of the retrobulbar block due to the time needed for diffusion of the local anesthetic [11].

Local Anesthetics

1. For shorter cases, 2% lidocaine is used, and 0.5% bupivacaine or 0.5% ropivacaine can be used for longer procedures.
2. Use of adjuncts such as hyaluronidase, bicarbonates, and epinephrine is controversial and could be considered for selected cases.

Techniques

As with all blocks, patients should be monitored and resuscitation drugs and equipment should be ready for use. A strict aseptic technique should be used.

Retrobulbar

1. With the patient's gaze neutral or oriented inferonasally, thus pulling the optic nerve and meningeal sheath away from the injection site, a 25-G 30-mm short-bevel needle is inserted at the inferolateral side, transpalpebral, tangentially to the globe, until the equatorial line of the globe is reached (approximately 1.5–2 cm) (Fig. 16.6).
2. The needle is then redirected superiorly, towards the point situated just below the apex of the orbit, and advanced for a maximum depth of 25–30 mm. The eye will rotate slightly inferiorly and will suddenly return to neutral position as the conus is entered. Keeping the bevel facing the sclera reduces the risk of globe perforation.
3. After a negative aspiration test, 3–4 mL of local anesthetic solution should be injected slowly and painlessly. There should be minimal resistance to injection.
4. After injection, intermittent mechanical pressure should be applied on the globe for 5–10 min to promote even distribution of local anesthetic.
5. For complete akinesia, this block needs supplementation of the temporal branch of the facial nerve to avoid orbicularis oculi movement during the surgical procedure. A 25-G needle is inserted 1 cm lateral to the lateral canthus. Then a subcutaneous infiltration is made along the inferior and the superior orbital border, with 2–3 mL of solution on each side (Fig. 16.7).

Peribulbar

1. A 25-G 30-mm short-bevel needle is inserted at the junction of the middle and the lateral thirds of the lower lid and directed to the equator of the globe (Fig. 16.8).

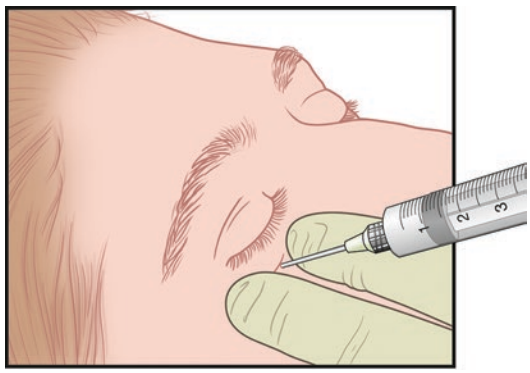


Fig. 16.6 Retrobulbar anesthesia

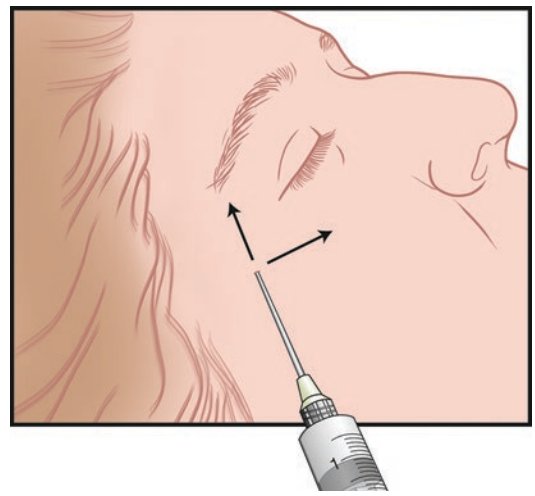


Fig. 16.7 Block of the temporal branch of the facial nerve (CN VII)

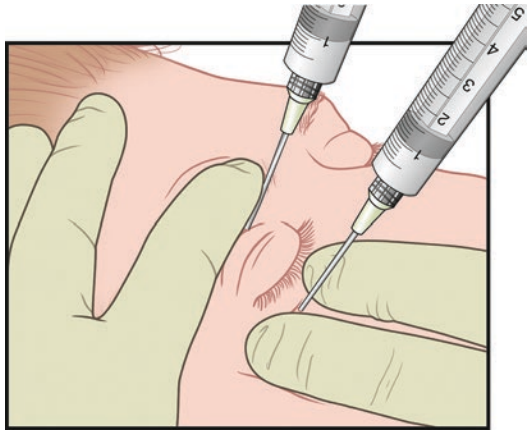


Fig. 16.8 Peribulbar anesthesia, inferolateral and superonasal approach

2. After a negative aspiration test, 5 mL is injected.
3. In most patients, this inferolateral injection alone produces satisfactory anesthesia and akinesia [12]. If not, a superonasal approach can be added. This is essentially the same approach, but this time the insertion site is at the intersection of the medial and middle thirds of the upper lid (Fig. 16.8). Note that the onset time for surgical block can be as long as 20 min [11].

Risks and Complications

1. Retrobulbar [10, 13]:
   (a) Trauma to adjacent structures
   (b) Globe perforation
   (c) Retrobulbar hemorrhage
   (d) Optic nerve injury
   (e) Oculocardiac reflex
   (f) Misplaced injections
   (g) Intra-arterial injection, with associated seizures
   (h) Subarachnoid injection
2. Peribulbar [9, 13]:
   (a) Trauma to adjacent structures
   (b) Globe perforation
   (c) Peribulbar hemorrhage
   (d) Central retinal artery occlusion
   (e) Oculocardiac reflex
   (f) Toxic injury to rectus muscles, with persistent paresis [14]

Cervical Blocks

Cervical blocks are useful for anterior neck surgery, such as carotid endarterectomy, thyroidectomy, and lymph node biopsies. Superficial cervical plexus block is the most frequently performed technique because of the ease of performance and low risk. Deep cervical plexus block has been less popular because some evidence has shown not only no added benefits to superficial plexus block for carotid and thyroid surgery, but also a definitive increase in risk [14, 15]. Recently, some authors have advocated the use of a high interscalene block to obtain the same endpoints as with the formal deep cervical plexus block with one injection site and more complete block [16].

Anatomy

1. The cervical plexus originates from the anterior rami of C1–C4 spinal nerves, which emerge from the intervertebral foramen behind the vertebral artery. Superficial branches come out on the posterior aspect of the sternocleidomastoid to give sensory innervation to the skin via the lesser occipital, the great auricular, the transverse cervical, and the supraclavicular nerves.
2. The deep branches innervate deeper structures of the neck and participate in the constitution of the phrenic nerve (Fig. 16.9).

Local Anesthetics

Good choices for the superficial cervical plexus block are 1% lidocaine or 0.25% bupivacaine, whereas 2% lidocaine, 0.5% ropivacaine, or 0.5% bupivacaine is more suitable for a deep cervical plexus block or for the high interscalene block. The solution used for the two latter blocks should contain at least 2.5 μg/mL of epinephrine in order

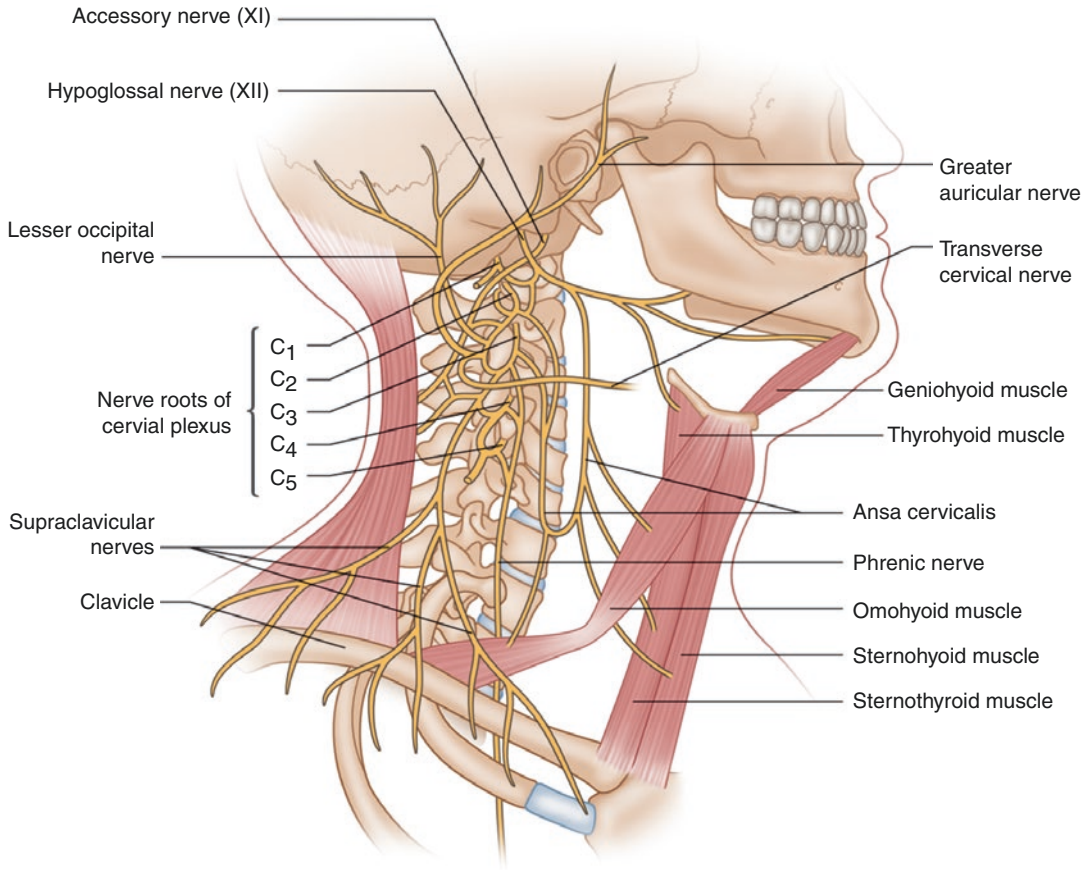


Fig. 16.9 Anatomy of the cervical plexus

to identify inadvertent intravenous injection (intra-arterial injection will be obvious with or without epinephrine).

Techniques

Superficial Cervical Plexus Block

The needle is inserted at the intersection of a line drawn horizontally from the cricoid cartilage and a vertical line drawn along the posterior border of the clavicular head of the sternocleidomastoid muscle. 5 mLs of local anesthetic is infiltrated along the middle third (Fig. 16.10). It is possible to use ultrasound guidance to perform this block, the local anesthetic solution being deposited just under the sternocleidomastoid muscle border using an in-plane approach (Fig. 16.11).

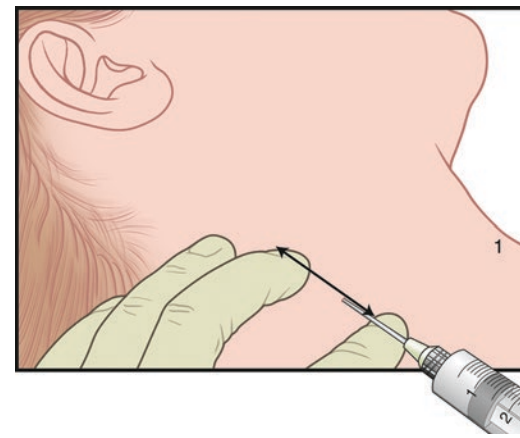


Fig. 16.10 Superficial cervical plexus block. A subcutaneous infiltration is made along the posterior border of the sternocleidomastoid muscle

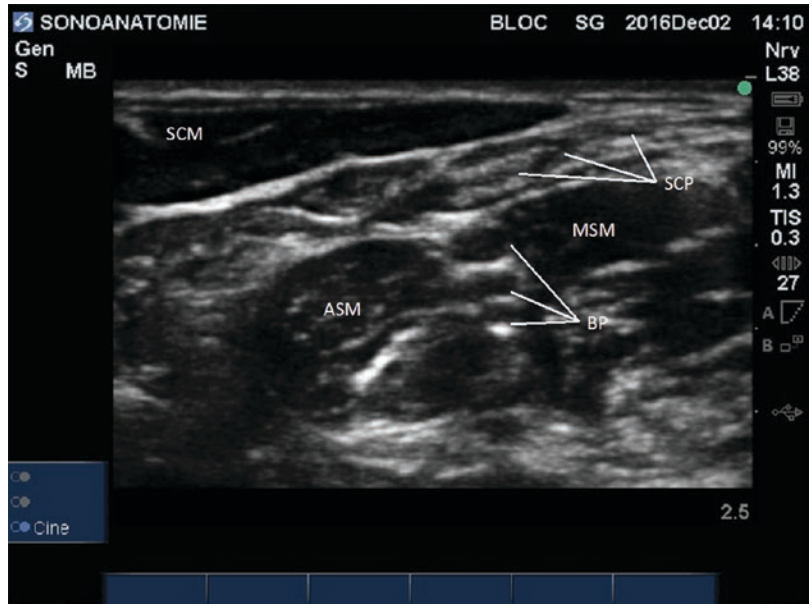


Fig. 16.11 Sonoanatomy of the superficial cervical plexus. *SCM* sternocleidomastoid muscle, *ASM* anterior scalene muscle, *MSM* middle scalene muscle, *BP* brachial plexus (superior, middle, and inferior trunks), *SCP* superficial cervical plexus branches

Deep Cervical Plexus Block

This block is a cervical paravertebral block. Three injections have to be done on the transverse processes of C2, C3, and C4.

1. The insertion points are located on a vertical line drawn between the mastoid process and the C6 transverse process (Chassaignac's tubercle), which is in the same craniocaudal plane as the cricoid cartilage.
2. The lower border of the mandible is the approximate level of C4, which is marked on the line.
3. The space between the mastoid process and this point can now be divided into thirds, showing the insertion points for C2 and C3.
4. The 22-G needle is inserted in the posterior-medial-caudal direction until contact is made with the transverse process.
5. An aspiration test should be made to ensure that the vertebral artery or the subarachnoid space has not been punctured, and only then 3–4 mL of local anesthetic solution should be injected per level (Fig. 16.12).

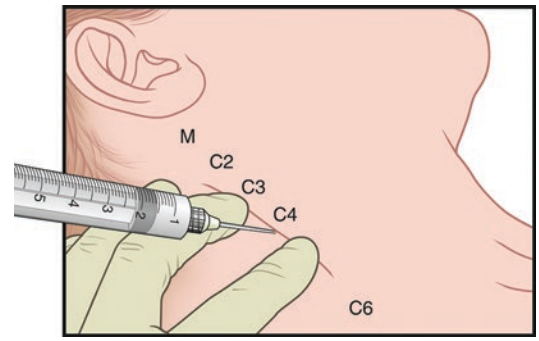


Fig. 16.12 Deep cervical plexus block

High Interscalene

This approach provides the opportunity of using a single-injection technique to block the cervical plexus (deep and superficial components).

1. The interscalene groove is palpated and marked, and the needle is inserted at the highest point of the interscalene groove usually coinciding with a horizontal line from the lower angle of the jaw (C4), with a posteromedial and slightly caudad angle (Fig. 16.13).

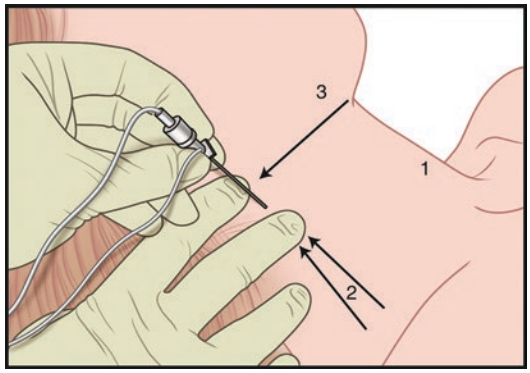


Fig. 16.13 High interscalene block

2. Levator scapulae movement as demonstrated by elevation and internal rotation of the scapula should be sought with the nerve stimulator.
3. A 30 mL volume should be used, and digital pressure should be applied caudally to the needle to promote cephalad diffusion. This block can also be ultrasound guided [17].

Risks and Complications

1. Superficial cervical plexus block is a low-risk procedure, the main risk being inadvertent intravascular injection.
2. Deep cervical plexus block and high interscalene:
   (a) Intravertebral injection, seizures
   (b) Intrathecal injection, total spinal anesthesia/brainstem anesthesia
   (c) Phrenic and recurrent laryngeal nerve block
   (d) Brachial plexus blockade
   (e) Hematoma

Airway Blocks

These blocks are particularly useful for the anesthesiologist performing an awake fiber-optic intubation and for tracheotomy and bronchoscopy. Blocks involve the laryngeal branches of the vagus nerve, namely, the superior laryngeal nerve and the recurrent laryngeal nerve.

Anatomy

Superior Laryngeal Nerve

The laryngeal surface of the epiglottis and the laryngeal inlet, down to the vocal folds, are innervated by the superior laryngeal nerve, which leaves the vagus trunk, crosses the greater cornu of the hyoid, and then penetrates the thyrohyoid membrane. This nerve also has a motor component (external branch) innervating the cricothyroid muscle.

The Recurrent Laryngeal Nerve

The recurrent laryngeal nerve, as its name suggests, is a branch of the vagus nerve traveling lower down the thorax and then ascending in the neck between the trachea and the esophagus. This nerve provides sensory innervation of the larynx below the vocal cords and the trachea, and motor innervation to all intrinsic muscles of the larynx, except the cricothyroid muscle (Fig. 16.14).

Glossopharyngeal Nerve

The other structures of the oropharynx and the ventral portion of the epiglottis are innervated by the glossopharyngeal nerve, which can be blocked easily by a number of noninvasive techniques including mouthwash/gargles with local anesthetics mixture or spraying of local anesthetics.

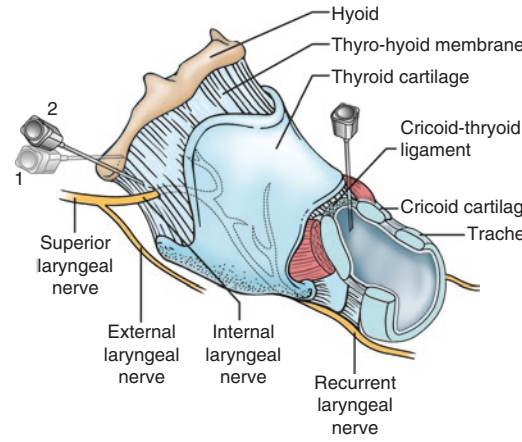


Fig. 16.14 Innervation of the larynx

Local Anesthetics

The short duration and onset time of 2% lidocaine make it perfect for most airway blocks, except for the gargles which are usually made with a 4% viscous lidocaine preparation.

Techniques

Superior Laryngeal Nerve

1. A 25-mm 25-G needle is inserted over the greater cornu of the hyoid bone and is then walked off the caudad aspect of it until the thyrohyoid membrane is pierced. The needle should not go deeper than the hyoid itself.
2. After aspiration, 3 mL of 1% lidocaine is injected.
3. To get precise landmarks, the hyoid should be slightly pushed toward the site to be blocked while the cricoid cartilage is held medial (Fig. 16.15).
4. This block could also be done under ultrasound guidance [18].

Recurrent Laryngeal Nerve

Although this nerve can be blocked directly, it is easier to use a translaryngeal technique to approach.

1. A 20-G needle is introduced in the cricothyroid membrane until air is aspirated.
2. Then the local anesthetic solution (usually 3–5 mL of 2% or 4% lidocaine) is quickly injected and the needle immediately withdrawn (to avoid trauma as the patient coughs).

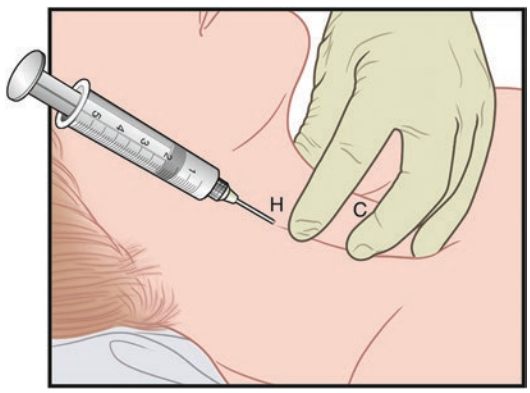


Fig. 16.15 Superior laryngeal nerve block. *H* hyoid cornu, *C* cricoid cartilage

Risks and Complications

1. Systemic toxicity
2. Aspiration of gastric content, since airway reflexes will be abolished

Clinical Pearls

The Scalp Block/Block of the Head

- Scalp block is an effective means of preventing the elevation of blood pressure and associated elevation of intracranial pressure when Mayfield pins are inserted for head positioning [2, 3].
- Scalp blocks are painful, and appropriate sedation should be given to patients before proceeding.

Ophthalmologic Block

- Oculocardiac reflex is most frequent in the pediatric and geriatric populations. Patients with preexisting high vagal tone, stressed, or on beta-blockers, are also considered to be higher risk patients.
- Highly myopic patient is at increased risk for globe perforation, so a discussion with the ophthalmologist before blockade can help define the relative risk for a given patient.

Cervical Blocks

- The superficial cervical plexus block alone is often enough for most cervical surgeries, provided that the surgeon infiltrates the deeper structures as he or she dissects through them [14, 15].

- The caudad orientation of the needle is the key to avoid penetration of the subarachnoid space and vertebral artery during deep cervical plexus and high interscalene block.
- For carotid endarterectomy surgery, the surgeon must additionally infiltrate directly around the carotid artery to block autonomic responses associated with manipulation of the carotid. At the present time, however, direct infiltration into the carotid sinus is not recommended for prevention of postoperative hemodynamic lability [19].
- If akinesia of the trapezius is indicated, the accessory nerve can be blocked with the same insertion point as for the superficial plexus block, with the needle penetrating just below the fascia, perpendicular to all planes.

Airway Block

- A mouthwash with local anesthetics is as effective as the glossopharyngeal block and better tolerated to produce anesthesia of the pharyngeal structures [20].

Review Questions

1. All of the following nerves must be blocked in order to provide anesthesia/analgesia to the scalp EXCEPT:
   (a) Cranial nerve V
   (b) Greater occipital nerve
   (c) Supraorbital nerve
   (d) Lesser auricular nerve
2. Compared to retrobulbar block the peribulbar block has the following property:
   (a) More complete akinesia of the eye
   (b) Uses less volume
   (c) More likely to have globe perforation.
   (d) Faster onset time
3. The superficial branches of the cervical plexus include the following nerves EXCEPT:
   (a) Lesser occipital nerve
   (b) Greater auricular nerve
   (c) Suprascapular nerve
   (d) Transverse cervical nerve
4. For high interscalene block with a nerve stimulator, the most reliable motor endpoint is:
   (a) Shoulder abduction
   (b) Scapular elevation and internal rotation
   (c) Lateral rotation of the neck
   (d) Arm flexion and pronation
5. This nerve provides sensory innervation of the larynx above the vocal cords:
   (a) Superior laryngeal nerve
   (b) Glossopharyngeal nerve
   (c) Recurrent laryngeal nerve
   (d) External branch of the superior laryngeal nerve
6. Which complication is specific to the retrobulbar block?
   (a) Globe perforation
   (b) Oculocardiac reflex
   (c) Subarachnoid injection
   (d) Trauma to adjacent structures
7. While performing a scalp block, which of these specific branches does not need aspiration prior to injection?
   (a) Supratrochlear
   (b) Supraorbital
   (c) Auriculotemporal
   (d) Greater occipital
8. During a retrobulbar block, the patient's gaze should be ideally oriented:
   (a) Superonasally
   (b) Inferonasally
   (c) Superolaterally
   (d) Inferolaterally

Answers

1. d
2. a
3. c
4. b
5. a
6. c
7. a
8. b

References

1. Sinha PK, Koshy T, Gayatri P, Smitha V, Abraham M, Rathod RC. Anesthesia for awake craniotomy: a retrospective study. Neurol India. 2007;55(4):376–81.
2. Geze S, Yilmaz AA, Tuzuner F. The effect of scalp block and local infiltration on the haemodynamic and stress response to skull-pin placement for craniotomy. Eur J Anaesthesiol. 2009;26(4):298–303.
3. Pinosky ML, Fishman RL, Reeves ST, Harvey SC, Patel S, Palesch Y, et al. The effect of bupivacaine skull block on the hemodynamic response to craniotomy. Anesth Analg. 1996;83(6):1256–61.
4. Bala I, Gupta B, Bhardwaj N, Ghai B, Khosla VK. Effect of scalp block on postoperative pain relief in craniotomy patients. Anaesth Intensive Care. 2006;34(2):224–7.
5. Nguyen A, Girard F, Boudreault D, Fugère F, Ruel M, Moumdjian R, et al. Scalp nerve blocks decrease the severity of pain after craniotomy. Anesth Analg. 2001;93(5):1272–6.
6. Guilfoyle MR, Helmy A, Duane D, Hutchinson PJA. Regional scalp block for Postcraniotomy analgesia: a systematic review and meta-analysis. Anesth Analg. 2013;116(5):1093–102.
7. Finco G, Atzeni M, Musu M, Maxia S, Ribuffo D. Greater occipital nerve block for surgical resection of major infiltrating lesions of the posterior scalp. Plast Reconstr Surg. 2010;125(2):52e–3.
8. Ripart J, Lefrant JY, de La Coussaye JE, Prat-Pradal D, Vivien B, Eledjam JJ. Peribulbar versus retrobulbar anesthesia for ophthalmic surgery: an anatomical comparison of extraconal and intraconal injections. Anesthesiology. 2001;94(1):56–62.
9. Davis DB 2nd, Mandel MR. Efficacy and complication rate of 16,224 consecutive peribulbar blocks. A prospective multicenter study. J Cataract Refract Surg. 1994;20(3):327–37.
10. Alhassan MB, Kyari F, Ejere HO. Peribulbar versus retrobulbar anaesthesia for cataract surgery. Cochrane Database Syst Rev. 2008;16(3):CD004083.
11. Perron Y, Ripart J. Blocs en ophtalmologie. In: Gaertner E, Choquet O, Macaire P, Zetlaoui PJ, editors. Anesthésie régionale—Anesthésie tronculaire et plexique de l'adulte. Arnette: Rueil-Malmaison; 2001. p. 41–57.
12. Clausel H, Touffet L, Havaux M, Lamard M, Savean J, Cochener B, et al. Anesthésie péribulbaire: efficacité d'une seule injection et d'un volume d'anesthésiques locaux limité. J Fr Ophtalmol. 2008;31(8):781–5.
13. Eke T, Thompson JR. Serious complications of local anaesthesia for cataract surgery: a 1 year national survey in the United Kingdom. Br J Ophthalmol. 2007;91(4):470–5.
14. Suh YJ, Kim YS, In JH, Joo JD, Jeon YS, Kim HK. Comparison of analgesic efficacy between bilateral superficial and combined (superficial and deep) cervical plexus block administered before thyroid surgery. Eur J Anaesthesiol. 2009;26(12):1043–7.
15. Guay J. Regional anesthesia for carotid surgery. Curr Opin Anaesthesiol. 2008;21(5):638–44. Review.
16. Merle JC, Mazoit JX, Desgranges P, Abhay K, Rezaiguia S, Dhonneur G, et al. A comparison of two techniques for cervical plexus blockade: evaluation of efficacy and systemic toxicity. Anesth Analg. 1999;89(6):1366–70.
17. Roessel T, Wiessner D, Heller AR, Zimmermann T, Koch T, Litz RJ. High-resolution ultrasound-guided high interscalene plexus block for carotid endarterectomy. Reg Anesth Pain Med. 2007;32(3):247–53.
18. Kaur B, Tang R, Sawka A, Krebs C, Vaghadia H. A method for ultrasonographic visualization and injection for the superior laryngeal nerve: volunteer study and cadaver simulation. Anesth Analg. 2012;115(5):1242–5.
19. Tang TY, Walsh SR, Gillard JH, Varty K, Boyle JR, Gaunt ME. Carotid sinus nerve blockade to reduce blood pressure instability following carotid endarterectomy: a systematic review and meta-analysis. Eur J Vasc Endovasc Surg. 2007;34(3):304–11.
20. Sitzman BT, Rich GF, Rockwell JJ, Leisure GS, Durieux ME, DiFazio CA. Local anesthetic administration for awake direct laryngoscopy. Are glossopharyngeal nerve blocks superior? Anesthesiology. 1997;86(1):34–40.

Upper Extremity Nerve Blocks

17

De Q.H. Tran, Maria Francisca Elgueta, and Juan Francisco Asenjo

Introduction

For more than a century, brachial plexus blockade has been an indispensable tool in the regional anesthesiologist's armamentarium. By providing surgical anesthesia and postoperative analgesia to the entire upper limb, it has been intimately linked to advances in orthopedic and ambulatory anesthesia. Furthermore, with the advent of ultrasonography, upper extremity blocks are being rediscovered under a new light. Every month, anesthesia journals report novel methods to anesthetize different parts of the brachial plexus. Navigating this plethora of studies can be a daunting task. This chapter aims to present a pragmatic and logical discussion of approaches and techniques for brachial plexus blockade.

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Clinical Anatomy of the Brachial Plexus

The brachial plexus (Fig. 17.1) is derived from the anterior primary rami of the fifth, sixth, seventh, and eighth cervical nerves as well as the first thoracic nerve in about 75% of the individuals, with variable contributions from the fourth cervical nerve in 15–62% of cases ("prefixed" brachial plexus) and the second thoracic nerve in 16–73% of cases ("postfixed" brachial plexus).

The length of the roots, from foramina to trunk, varies between 30 mm (C8 and T1), 40 mm (C5), 50 mm (C6), and 60 mm (C7). The duramater and the epidural connective tissues in the vertebral canal follow the roots to form the perineurium and epineurium, respectively. The roots leave the intervertebral foramina and course between the anterior and middle scalene muscles in the posterior triangle of the neck. Before forming the three trunks (superior, inferior, and middle), the roots give rise to the following nerves:

(a) The long thoracic nerve (C5, C6, and C7), which innervates the anterior serratus muscle, either traverses the middle scalene muscle or exits between the posterior and the middle scalene muscles.
(b) The dorsal scapular nerve (C5), which innervates the rhomboid and levator scapulae muscles, exits behind the middle scalene muscle.

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_17

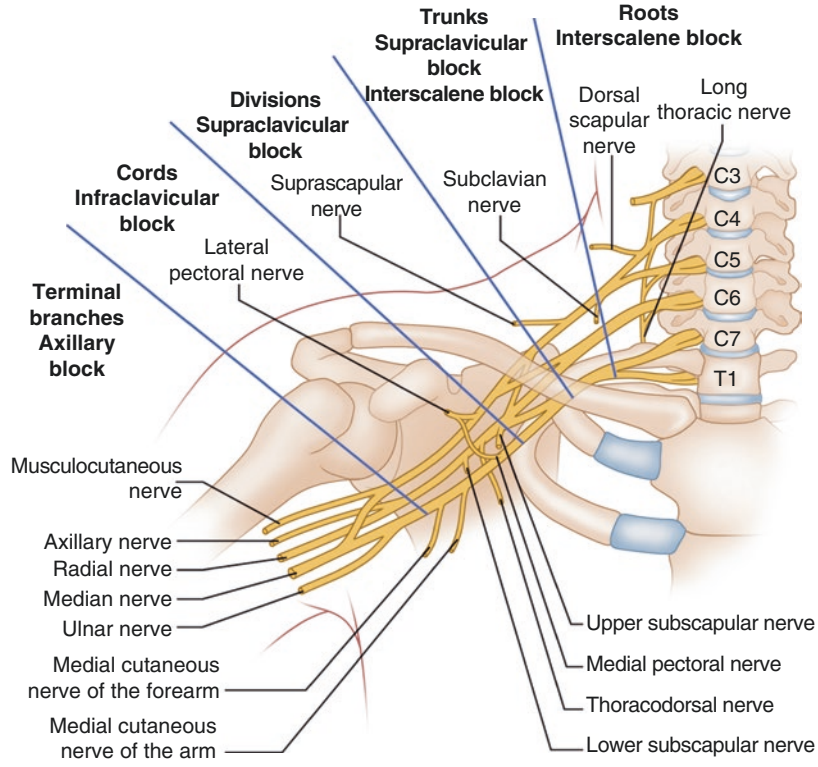

Fig. 17.1 Anatomy of the brachial plexus

(c) Although the phrenic nerve stems from the C3, C4, and C5 nerves, in 20% of cases, it originates entirely from the roots of the brachial plexus.
(d) The C5, C6, C7, and C8 roots also provide innervation to the scalene and longus colli muscles.

Of the three trunks, the only one giving rise to peripheral branches is the superior trunk. The suprascapular nerve (C5 and C6), which supplies the supra- and infraspinatus muscles, and the nerve to the subclavius muscle (C5 and C6) both originate from the latter.

At the lateral edge of the first rib, each trunk separates into anterior and posterior divisions.

Subsequently, the divisions join to form three cords. The cords are termed lateral (formed from the anterior divisions of the superior and middle trunks, therefore C5 + C6 + C7), posterior (formed from all posterior divisions, C5 + C6 + C7 + C8 + T1), and medial (formed from the anterior divisions of the lower trunk, C8 + T1) based on their relationship with the axillary artery. The cords give rise to multiple side branches:

(a) The lateral pectoral nerve originates from the lateral cord.
(b) The medial pectoral nerve, the medial cutaneous nerve of the arm, and the medial cutaneous nerve of the forearm originate from the medial cord.
(c) The upper subscapular nerve, the lower subscapular nerve, and the thoracodorsal nerve originate from the posterior cord.

At the lateral border of the pectoralis minor muscle, the cords divide into terminal branches: the musculocutaneous nerve (lateral cord), axillary nerve (posterior cord), radial nerve (posterior cord), median nerve (lateral and medial cords), and ulnar nerve (medial cord).

Choosing the Right Approach

Surgery of the Shoulder, Clavicle, and Proximal Humerus

The clavicle and the (posterior) proximal humerus are innervated by the subclavian and suprascapular nerve, respectively (Fig. 17.2). Because they target the latter prior to their take-off from the superior trunk, the cervical paravertebral, interscalene, and supraclavicular approaches can be used.

Although some authors claim that the cervical paravertebral approach differs from its interscalene counterpart because the posterior, and not anterior, cervical roots are anesthetized [1], this remains ambiguous. In a large randomized trial (n = 80), no differences were found between the two blocks in terms of success rate, extent of the block, as well as onset and offset times [2]. One study has compared interscalene and supraclavicular blocks. Although block duration, patient satisfaction, postoperative pain scores, and analgesic requirements were similar, the supraclavicular approach resulted in fewer side effects (Horner's syndrome, recurrent laryngeal nerve palsy, and symptomatic diaphragmatic paralysis) [3].

Surgery of the Distal Humerus, Forearm, and Hand

The supraclavicular, infraclavicular, and axillary approaches can be used for surgical procedures involving the distal humerus, forearm, and hand. Humeral canal blocks should be reserved for surgery distal to the elbow.

When optimal techniques are utilized for each approach, the literature seems to suggest that supraclavicular, infraclavicular, axillary, and humeral canal blocks result in comparable success rates. Expectedly, approaches requiring a multiple-injection technique (axillary and humeral canal) can be associated with a longer performance time, more needle passes, or higher block-related pain scores [4–7].

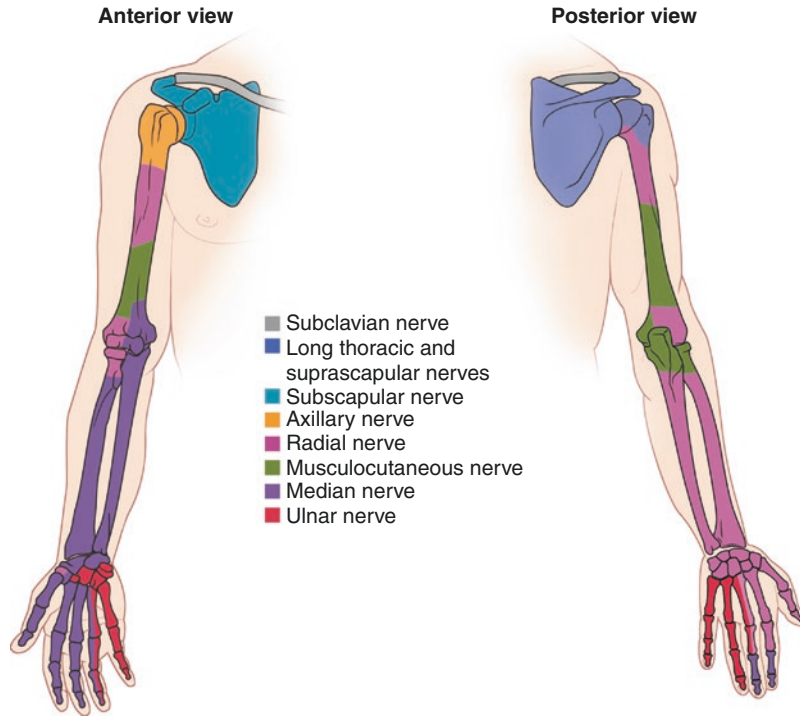

Fig. 17.2 Bony innervation of the upper extremity

Interscalene Brachial Plexus Block

Background

The interscalene approach anesthetizes the brachial plexus at the level of the roots and trunks. Identification of the plexus in the interscalene groove can be achieved with elicitation of paresthesia, nerve stimulation, or ultrasonography. Three trials have compared elicitation of paresthesia and neurostimulation with mixed results. In two studies, no differences were found [8, 9]. In contrast, the third trial recorded higher failure rates (10% vs. 0%) and postoperative pain scores in the paresthesia group [10].

Comparison between neurostimulation and ultrasonography has also yielded contradictory results. In one study, echoguidance improved the rate of surgical anesthesia (98.8% vs. 91.3%) as well as the onset and offset times [11]. In contrast, another trial observed no differences in performance time, surgical anesthesia, and postoperative neural deficits. However, patients in the ultrasound group required fewer passes [12].

The Techniques

Nerve Stimulation

The patient is supine with the head turned toward the contralateral side. At the level of the cricoid cartilage, posterior to the sternocleidomastoid muscle, the neck is palpated to identify the groove between the anterior and middle scalene muscles (Fig. 17.3).

The skin is infiltrated with local anesthesia. Because the plexus is very superficial, a small volume (<0.3 mL) should be used; otherwise, the evoked motor response may be abolished. A 2.5-cm block needle, connected to a nerve stimulator set at a current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is inserted in the interscalene groove. The needle is oriented in a slight caudad direction to avoid penetration of the intervertebral foramen. Typically, contraction of the deltoid, biceps, triceps, or pectoral muscles is seen. All four constitute acceptable evoked motor responses. If diaphragmatic contraction is encountered, the needle tip is close to the phrenic nerve (situated on the anterior scalene muscle) and thus should be redirected posteriorly. Conversely, if the needle is too posterior, stimulation of the dorsal scapular nerve and shoulder elevation (contraction of the rhomboid and levator scapulae muscles) will occur. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 20–30 mL of local anesthetic is injected.

Fig. 17.3 Landmarks for interscalene brachial plexus block (*IS* interscalene, *SCM* sternocleidomastoid muscle, *X* puncture site)

Ultrasound Guidance

The patient is placed in a supine or semisitting position with the head turned toward the contralateral side. At the level of the cricoid cartilage, the neck is scanned with a high-frequency probe (Fig. 17.4). The brachial plexus appears as a column of hypoechoic nodules. The exact nature of the latter (roots vs. trunks) remains controversial (Fig. 17.5). Using an in-plane technique and a lateral to medial direction, the skin and subcutaneous tissues are infiltrated with local anesthesia. A 5-cm block needle is then inserted. Care must be taken to visualize the entire length of the needle during the advancement process. The classic target for this block is situated between the first and second hypoechoic nodules. However, an injection in the middle scalene muscle, next to the interscalene groove but without penetration of the latter, provides a similar efficacy coupled with a slightly shorter block duration [13]. A volume of 10–20 mL of local anesthetic is commonly used.

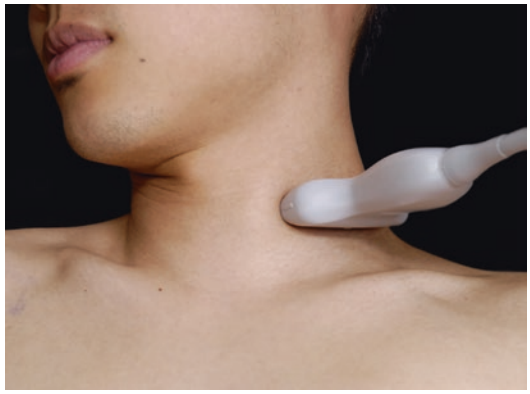

Fig. 17.4 Position of the ultrasound probe for interscalene brachial plexus block

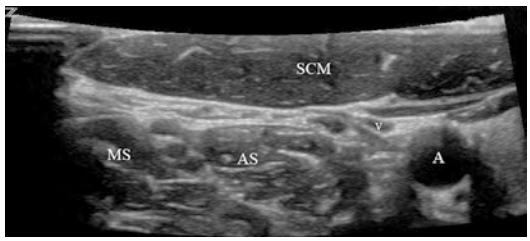

Fig. 17.5 Ultrasonographic appearance of the interscalene and cervical paravertebral brachial plexus (*A* carotid artery, *AS* anterior scalene muscle, *MS* middle scalene muscle, *SCM* sternocleidomastoid muscle, *V* internal jugular vein)

Complications

Due to the proximity of the cervical sympathetic chain and the recurrent laryngeal nerve, Horner's syndrome and hoarseness can occur. With appropriate technique and equipment, some complications can be prevented: a slight caudad orientation of the needle will decrease the risk of dural cuff puncture and vertebral artery or neuraxial injection. Similarly, limiting the length of needle insertion can prevent the occurrence of a pneumothorax. The most vexing side effect remains the 100% incidence of ipsilateral hemidiaphragmatic paralysis caused by migration of local anesthetics to the C3–5 roots or the phrenic nerve itself [14]. Usually well tolerated by healthy subjects, it becomes a prohibitive risk in patients with pulmonary compromise. To date, no preventive measure has been found. For instance, a reduction in local anesthetic volume (from 45 to 20 mL) and digital pressure above the injection site did not reduce phrenic paralysis [15–18]. Although 10 mL of bupivacaine 0.25% was not associated with changes in pulmonary function in healthy volunteers [19], a recent study reported a 27% incidence of diaphragmatic paralysis despite limiting the dose of local anesthetic to 5 mL of ropivacaine 0.75% [20].

Cervical Paravertebral Brachial Plexus Block

Background

The cervical paravertebral approach anesthetizes the brachial plexus at the level of the roots and proximal trunks. Identification of the plexus can be carried out with loss of resistance, nerve stimulation, or ultrasonography. No study has compared these three modalities. Most clinicians seem to prefer the latter two.

The Techniques

Nerve Stimulation

The patient is placed in a sitting or lateral decubitus position with the surgical side uppermost. The neck is flexed to facilitate palpation of the C6 and C7 spinous processes. Three to four centimeters lateral to the latter, a paravertebral line is traced in a cephalocaudal axis. This often corresponds to the groove between the levator scapulae and trapezius muscles. The puncture site is located on the midpoint of this paravertebral line (Fig. 17.6).

The skin and subcutaneous tissues are infiltrated with local anesthesia. A 10-cm block needle, connected to a nerve stimulator set at a current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is inserted perpendicularly to the skin until contact with the pars intervertebralis or transverse process. It is then walked laterally off the bone and advanced until contraction of the deltoid, biceps, triceps, or pectoral muscles is seen. After ensuring that the evoked motor

Fig. 17.6 Landmarks for cervical paravertebral brachial plexus block (*X* puncture site)

Fig. 17.7 Position of the ultrasound probe for cervical paravertebral brachial plexus block

response is still present at a current of 0.2–0.5 mA, 20–30 mL of local anesthetic is injected.

Ultrasonography

The patient is placed in a lateral decubitus position with the surgical side uppermost. At the level of the cricoid cartilage, the neck is scanned with a high-frequency probe to identify the carotid artery (Fig. 17.7). The probe is moved laterally until the interscalene groove can be identified. The brachial plexus appears as a column of hypoechoic nodules (Fig. 17.5). The puncture site for this block is situated in the groove between the levator scapulae and trapezius muscles. The skin and subcutaneous tissues are infiltrated with local anesthesia. Using an in-plane needle, a 10-cm block needle is directed toward the brachial plexus. Care must be taken to visualize the entire length of the needle during the advancement process. The target for this block can be the plexus itself (between the hypoechoic nodules) or the middle scalene muscle, next to the interscalene groove but without penetration of the latter [13]. A volume of 10–20 mL of local anesthetic is commonly used.

Complications

Adverse events related to the cervical paravertebral approach are similar to those associated with interscalene blocks (Horner's syndrome, hoarseness, vascular breach, and hemidiaphragmatic paralysis). Two potential complications deserve special mention. Because the needle traverses the extensor muscles of the neck, muscular pain can be problematic; inserting the needle in the groove between the levator scapulae and trapezius muscles may decrease the incidence of neck pain. Neuraxial spread of local anesthetic agents can occur in up to 4% of cervical paravertebral blocks [21]. To minimize this risk, some authors recommend avoiding sharp needles, which can pierce the dural cuffs. The vertebral artery is situated anterior to the pars intervertebralis or articular column of the vertebrae. Therefore vascular breach is unlikely if the needle is introduced to contact bone and walked laterally off the latter.

Supraclavicular Brachial Plexus Block

Background

The supraclavicular approach anesthetizes the brachial plexus at the level of the trunks and divisions. This block can be performed by elicitation of paresthesia, neurostimulation, or ultrasonography. The last two modalities are usually preferred. Although various surface landmarks have been described, the plumb bob technique is most commonly used [22]. For nerve stimulation, currents of 0.9 and 0.5 mA yield similar success rates, onsets, and durations of anesthesia [23]. For ultrasound guidance, the "targeted

intracluster injection" technique, whereby local anesthetic is injected inside all clusters formed by the trunks and divisions of the brachial plexus, seems to provide a reliable method to block the brachial plexus [24]. Compared to neurostimulation, ultrasonography results in a similar success rate coupled with a lower incidence of phrenic nerve blockade [25].

The Techniques

Nerve Stimulation

For the "plumb bob" technique, the patient is supine with the head turned toward the contralateral side. The head is raised to identify the insertion of the lateral border of the sternocleidomastoid on the clavicle. A 5-cm block needle is inserted at this point perpendicularly to the floor (Fig. 17.8). Failure to elicit an evoked motor response should be followed by redirection of the needle in a cephalad or caudad direction (in a parasagittal plane). Care is taken not to exceed an arc of 30°. After ensuring that the evoked motor response is still present at a current of 0.9 mA or lower, 30–40 mL of local anesthetic is commonly used.

Ultrasound Guidance

The patient is supine or semisitting with the head turned toward the contralateral side. Using a high-frequency probe, the supraclavicular area is scanned to identify a short-axis view of the subclavian artery (Fig. 17.9). The first rib can be identified under the vessel. Lateral and superolateral to the artery, a collection of neural clusters (trunks and divisions of the brachial plexus) can be seen. The skin and subcutaneous tissues are infiltrated with local anesthesia. Using an in-plane technique and a lateral to medial direction, a 5-cm block needle is directed toward the main (largest) neural cluster (Fig. 17.10). Care must be taken to visualize the entire length of the needle during the advancement process. Half the volume of local anesthetic is injected in this location. Subsequently, the remaining half is divided into equal aliquots and deposited inside the satellite (smaller) clusters. A volume of 30–35 mL of local anesthetic is commonly used [26].

Complications

Vascular puncture, recurrent laryngeal nerve paralysis, and Horner's syndrome can occur after supraclavicular blocks. The risk of pneumothorax can be as high as 6% if traditional techniques, which direct the needle in a cephalocaudal direction toward the lung, are used. Because phrenic nerve blockade can occur in 67% of cases, like its interscalene counterpart, this block is contraindicated in patients with pulmonary compromise [27].

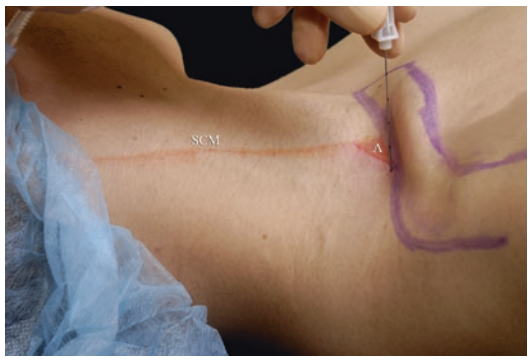

Fig. 17.8 Landmarks for supraclavicular brachial plexus block (*A* subclavian artery, *SCM* sternocleidomastoid muscle)

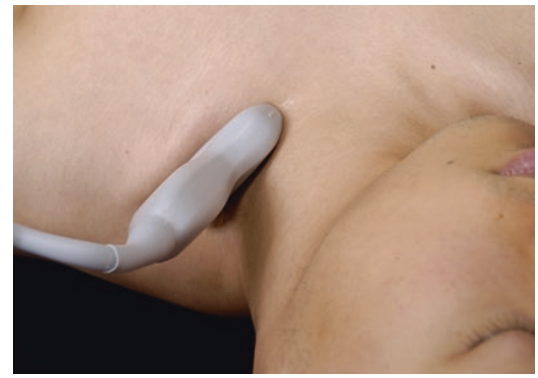

Fig. 17.9 Position of the ultrasound probe for supraclavicular brachial plexus block

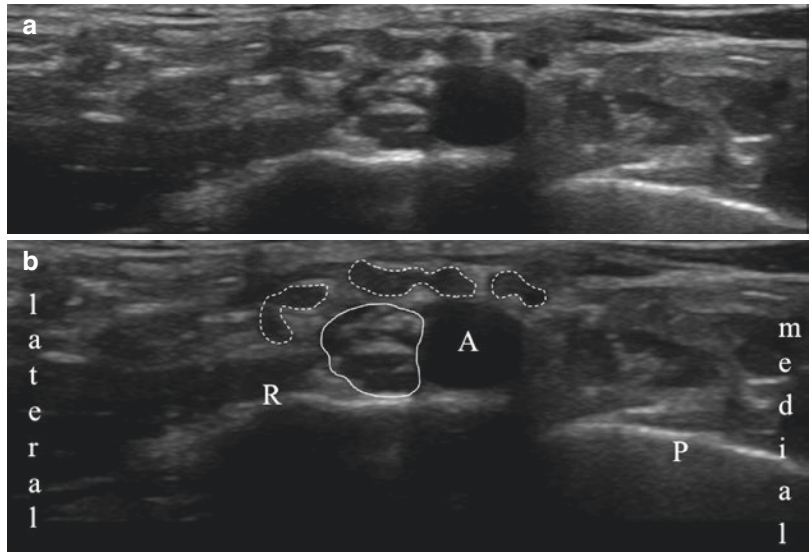

Fig. 17.10 (**a**) Ultrasonographic appearance of the supraclavicular brachial plexus. (**b**) Line drawing (*A* subclavian artery, *P* pleura, *R* first rib). Continuous line indicates main neural cluster. Dotted lines indicate satellite neural clusters

Infraclavicular Brachial Plexus Block

Background

The infraclavicular approach anesthetizes the brachial plexus at the level of its cords This block can be performed with neurostimulation or ultrasonography. For neurostimulation-guided infraclavicular blocks, the available literature favors a double-injection technique (avoiding the musculocutaneous/median combination) or a single-injection technique aiming for radial-type stimulation [28]. For ultrasound-guided infraclavicular blocks, the optimal technique consists of a single-injection dorsal to the axillary artery [29, 30].

Comparison of nerve stimulation and ultrasonography has yielded mixed results. Two trials comparing single-stimulation infraclavicular block with single- or multiple-injection ultrasound-guided block found similar rates of surgical anesthesia and onset times [31, 32]. However, in another study, ultrasonography was associated with a higher rate of surgical anesthesia, a shorter performance time, and fewer paresthesia [33]. Although some practitioners routinely combine neurostimulation and ultrasonography, this practice provides minimal benefits. Compared to ultrasound guidance alone, the combination of both modalities unnecessarily increased the performance time [34, 35] and led to a lower success rate [34].

The Techniques

Neurostimulation

Since the first description by Raj et al. [36], several sets of landmarks have been described for infraclavicular blocks. In North America, the most popular method is the pericoracoid technique [37]. With the patient supine, the arm to be blocked is adducted. A point 2 cm medial and 2 cm caudad to the tip of the coracoid process is identified [37] (Fig. 17.11). The skin and subcutaneous tissue are infiltrated with local anesthesia. A 5- to 10-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is inserted perpendicularly to the skin. Usually, elbow flexion (lateral cord stimulation) is encountered first. Using a parasagittal plane, the needle tip is redirected in a caudad direction in search of a radial-type response (extension of the forearm, wrist, or fingers). After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 30–40 mL of local anesthetic is deposited.

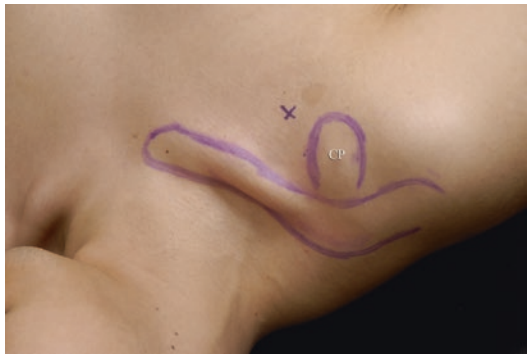

Fig. 17.11 Landmarks for infraclavicular brachial plexus block (*CP* coracoid process, *X* puncture site)

Fig. 17.12 Position of the ultrasound probe for infraclavicular brachial plexus block

Ultrasound Guidance

The patient is positioned supine. The arm is flexed so that the forearm and hand can rest comfortably on the torso. A high-frequency ultrasound probe is placed in the infraclavicular fossa, medial to the coracoid process, to obtain a short-axis view of the axillary vessels (Fig. 17.12). The axillary artery and vein can be found under the pectoralis major and minor muscles. The pleura can sometimes be seen under the vessels (Fig. 17.13). Local anesthetic is used to infiltrate the skin, subcutaneous tissues, and pectoralis muscles. Using an in-plane technique and a cephalad to caudad direction, a 10-cm block needle is advanced until the tip lies just dorsal to the artery. Care must be taken to visualize the needle during the advancement process. Usually, a pop can be felt just before the needle assumes the correct position. A volume of 30–35 mL of local anesthetic is commonly used [38].

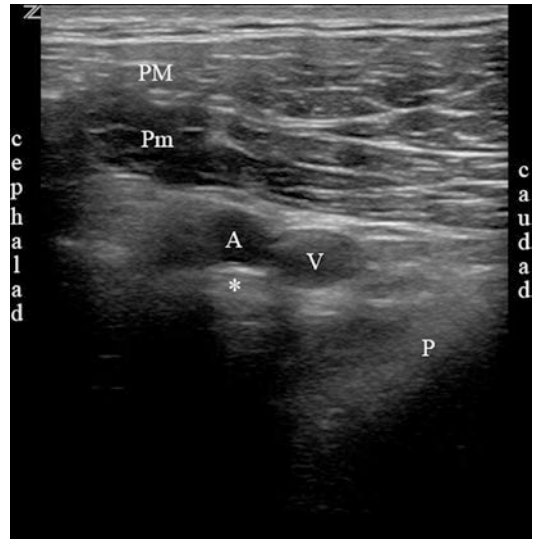

Fig. 17.13 Ultrasonographic appearance of the infraclavicular brachial plexus (*A* axillary artery, *P* pleura, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *V* axillary vein, *asterisk* indicates target)

Complications

Vascular puncture can occur. Because of the depth of the vessels, external compression can be difficult to achieve. Thus, caution should be exercised in coagulopathic patients, and perhaps, a different approach, considered. There have also been reports of Horner's syndrome, phrenic paralysis [39], and pneumothorax [40] associated with infraclavicular blocks.

Axillary Brachial Plexus Block

Background

The axillary approach anesthetizes the brachial plexus at the level of its four main terminal branches (musculocutaneous, median, radial, and ulnar nerves). Performing this block by fascial clicks, elicitation of paresthesia, transarterial injection, and single-nerve stimulation yields a similar success rate between 70 and 80% [41].

Thus, most practitioners prefer multiple-nerve stimulation and ultrasound guidance.

With neurostimulation, evidence suggests that a triple-injection technique (in which the ulnar nerve is not located) provides a similar efficacy to the four-injection technique [42, 43]. Furthermore, for the radial nerve, a distal motor response (wrist or finger extension) should be preferred to a proximal response (forearm extension) [44].

Compared to a multiple-stimulation technique, a higher success rate and shorter onset time have been reported with ultrasonography [45]. Two techniques exist for ultrasound-guided axillary block [46]. With the perineural method, each of the four branches is identified and individually anesthetized. In contrast, the perivascular method only requires the identification of the musculocutaneous nerve; subsequently local anesthetic is injected next to the axillary artery and anesthesia of the median, radial, and ulnar nerves is achieved through local anesthetic diffusion inside the neurovascular sheath. Both techniques result in similar efficacy. However the perivascular method offers greater simplicity (shorter performance time, fewer needle passes) [46].

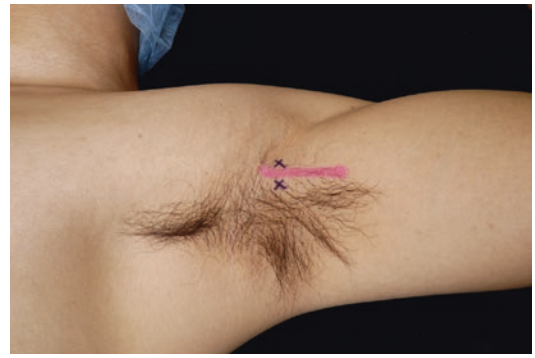

Fig. 17.14 Landmarks for axillary brachial plexus block (*X* puncture sites)

Fig. 17.15 Position of the ultrasound probe for axillary brachial plexus block

The Techniques

Nerve Stimulation

The patient is positioned with the shoulder abducted and the elbow flexed. The axillary area is palpated to identify the axillary artery. In the axilla, the musculocutaneous and median nerves are most often situated above the artery, whereas the radial and ulnar nerves can be found below the latter. However, a great deal of anatomical variability can occur. For this block, two distinct puncture sites (above and below the artery) are required (Fig. 17.14). The skin is infiltrated with local anesthesia. Because the median nerve is very superficial, a small volume (<0.3 mL) should be used above the artery; otherwise, the evoked motor response may be abolished for the median nerve. A 5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is first inserted above the artery to locate the musculocutaneous nerve (elbow flexion). After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 mL of local anesthetic is deposited. Subsequently, the needle is repositioned to locate the median nerve (above the artery) and radial nerve (below the artery). Wrist/finger flexion is sought for the former, whereas wrist/finger extension is sought for the latter. For each of these two nerves, a local anesthetic volume of 10–14 mL can be used.

Ultrasound Guidance

The patient is positioned with the shoulder abducted and the elbow flexed. The axilla is scanned with a high-frequency linear ultrasound probe to identify a short-axis view of the axillary artery (Fig. 17.15). The musculocutaneous

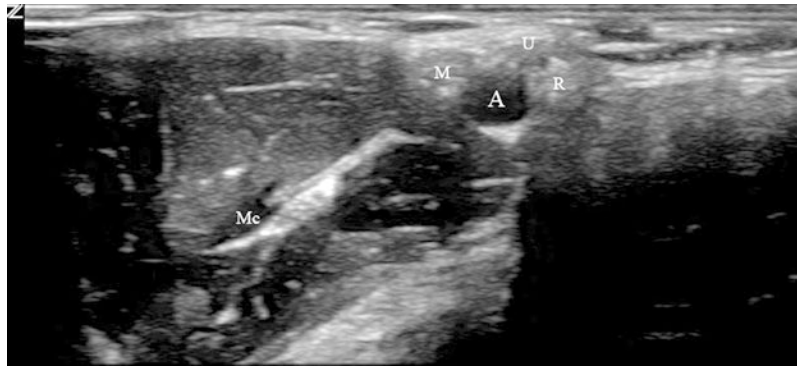

Fig. 17.16 Ultrasonographic appearance of the axillary brachial plexus (*A* axillary artery, *M* median nerve, *Mc* musculocutaneous nerve, *R* radial nerve, *U* ulnar nerve)

nerve, a hyperechoic structure, can be found anterior and lateral to the artery, in the belly of the coracobrachialis muscles, or in a plane between the coracobrachialis and biceps muscles (Fig. 17.16).

Using an in-plane technique, the skin and subcutaneous tissues are infiltrated with local anesthesia. A 5-cm block needle is then inserted. Care must be taken to visualize the entire length of the needle during the advancement process. The needle is first directed toward the musculocutaneous nerve. Six milliliters of local anesthetic is injected. Subsequently, the needle is redirected toward the dorsal aspect (6 o'clock position) of the axillary artery. Twenty-four milliliters of local anesthetic is deposited in this location [47]. To ensure proximity between needle tip and axillary artery, injection of the first few milliliters of local anesthetic must result in a "silhouette sign." The latter is defined as the blurring of the dorsal aspect of the arterial wall and results from the superposition of anechoic blood and anechoic local anesthetic [48].

Complications

Transient numbness, vascular puncture, intravascular injection, bruising, and soreness at the injection site have been reported, but the overall safety margin for the block is very high.

Humeral Canal Block

Background

Similar to the axillary approach, the humeral canal block anesthetizes the brachial plexus at the level of its terminal branches.

The Techniques

Nerve Stimulation

The patient is positioned with the shoulder abducted and the elbow flexed. Midway between the shoulder and elbow, the arm is palpated to identify the axillary artery. The musculocutaneous and median nerves are most often situated above the artery, whereas the radial and ulnar nerves can be found below the latter. However, the radial nerve can be difficult to find because it courses posterior to the humerus. For this block, two distinct puncture sites (above and below the artery) are required (Fig. 17.17). Because the median and ulnar nerves are very superficial, a small volume (<0.3 mL) is used; otherwise, the evoked motor responses could be abolished. A 5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is first inserted above the artery to locate the musculocutaneous (elbow flexion) and median (wrist/finger flexion) nerves. Subsequently, the needle is

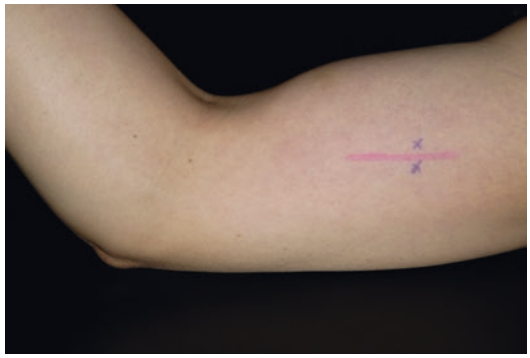

Fig. 17.17 Landmarks for humeral canal block (*X* puncture sites)

Fig. 17.18 Position of the ultrasound probe for humeral canal block

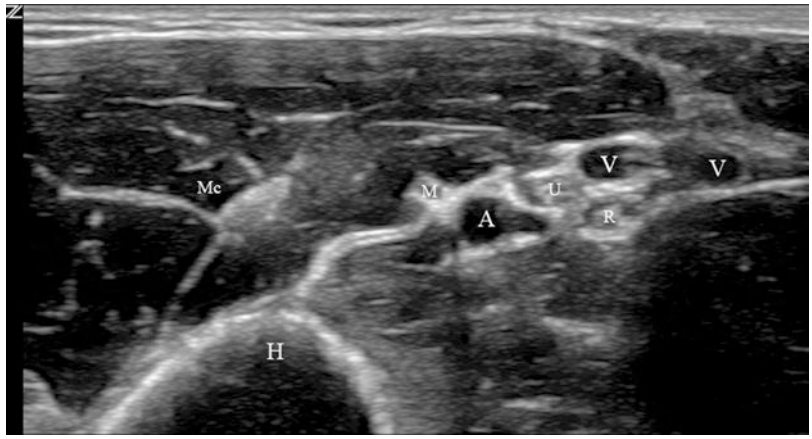

Fig. 17.19 Ultrasonographic appearance of nerves in the humeral canal (*A* brachial artery, *H* humerus, *M* median nerve, *Mc* musculocutaneous nerve, *R* radial nerve, *U* ulnar nerve, *V* brachial vein)

repositioned under the artery to locate the radial (wrist/finger extension) and ulnar (extension of the fourth/fifth fingers and ulnar deviation of the wrist) nerves. For the median and radial nerve, currents of 0.8 mA or lower and 0.6 mA or lower should be used, respectively. For the ulnar and musculocutaneous, a threshold of 0.7 mA or lower is recommended [49]. A volume of 5–7 mL of local anesthetic is deposited for each nerve.

Ultrasound Guidance

The patient is positioned with the shoulder abducted and the elbow flexed. The arm is scanned with a high-frequency, linear ultrasound probe to identify a short-axis view of the axillary artery (Fig. 17.18). The musculocutaneous and median nerves are situated above the artery, whereas the radial and ulnar nerves can be located below the latter (Fig. 17.19). Using an in-plane technique and puncture sites above or below the artery, a 5-cm block needle is directed toward each of the four neural structures. Care must be taken to visualize the entire length of the needle during the advancement process. Local anesthetic is injected until circumferential spread is achieved for each nerve. Five to seven milliliters are commonly used per nerve.

Complications

Although vascular puncture, bruising, and soreness at the injection site can occur, the overall safety margin for the block is very high.

Supplemental Blocks

In the event of an incomplete brachial plexus block, missing nerves can be anesthetized in a more distal location.

Suprascapular Nerve Block

Background

The suprascapular nerve can be blocked with neurostimulation or ultrasonography. No randomized control trial has compared these two modalities. Cadaveric dissection suggests that ultrasonography targets the suprascapular nerve in the suprascapular fossa, whereas nerve stimulation contacts the nerve in the notch [50].

The Techniques

Neurostimulation

The patient is positioned sitting and leaning forward slightly. The spine of the scapula is identified and is traced. A vertical line passing through the tip of the scapula is also drawn. These two lines divide the scapula into four quadrants. A bisector is drawn for the superolateral quadrant. The puncture site is located 2–3 cm along this bisector (Fig. 17.20). A 5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is introduced at this point perpendicular to the skin. If the scapula is contacted, the needle is redirected superior and medially to enter the suprascapular notch. Abduction or external rotation of the arm is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 10 mL of local anesthetic is deposited.

Ultrasound Guidance

The patient is positioned in the lateral decubitus position so that the side to be blocked is nondependent (Fig. 17.21). Using a high-frequency, linear ultrasound probe, the area cephalad to the scapular spine is scanned to identify the suprascapular fossa (Fig. 17.22). The skin and subcutaneous tissues are infiltrated with local anesthesia. Using an out-of-plane technique, a 10-cm block needle is advanced toward the suprascapular fossa. The needle tip can be followed through tissue distortion. A volume of 10 mL of local anesthetics is deposited in the fossa.

Upper Extremity Distal Nerve Blocks (Radial, Median, and Ulnar Nerves)

Background

The radial, median, and ulnar nerves can be blocked at the elbow or wrist. Blocks performed at the elbow offer more versatility because they

Fig. 17.20 Landmarks for suprascapular nerve block (*X* puncture site)

Fig. 17.21 Position of the ultrasound probe for suprascapular nerve block

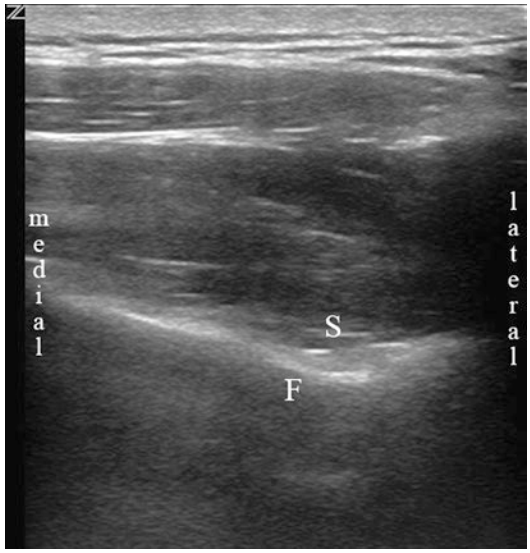

Fig. 17.22 Ultrasonographic appearance of the supra-scapular fossa (*F* fossa, *S* supraspinatus muscular fascia)

can be used for forearm, wrist, and hand surgery. In contrast, blocks performed at the wrist can only be used for procedures involving the hand.

The Techniques

At the Elbow

Neurostimulation

(a) Radial Nerve
The patient is supine with the upper extremity supinated and abducted. The radial nerve is located lateral to the bicipital tendon between the brachialis and brachioradialis muscles (Fig. 17.23). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is inserted to a depth of 1–2 cm. Wrist or finger extension is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 mL of local anesthetic is deposited.

(b) Median Nerve
The patient is supine with the upper extremity supinated and abducted. The median nerve is located just medial to the brachial artery (Fig. 17.23). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is inserted medial to the brachial artery at a depth of 1–2 cm. Wrist or finger flexion is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 mL of local anesthetic is deposited.

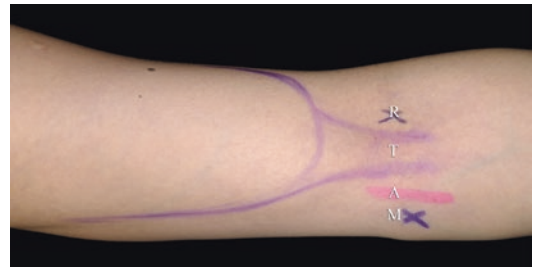

Fig. 17.23 Landmarks for supplemental median and radial nerve blocks at the elbow (*A* brachial artery, *M* median nerve, *R* radial nerve, *T* bicipital tendon)

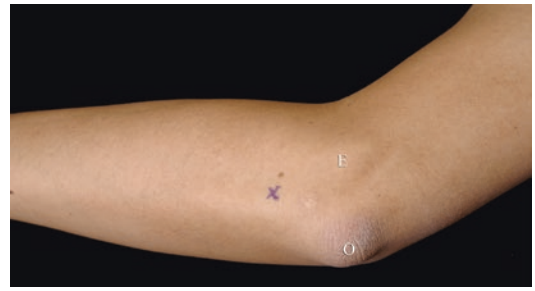

Fig. 17.24 Landmarks for supplemental ulnar nerve block at the elbow (*E* medial epicondyle, *O* olecranon, *X* puncture site)

(c) Ulnar Nerve
The patient is supine with the forearm flexed on the arm to locate the ulnar groove. The nerve is located in the groove between the medial epicondyle and the olecranon process (Fig. 17.24). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is inserted 1–3 cm proximal to a line joining the bony landmarks and directed along the longitudinal axis of the humerus. Ulnar deviation of the wrist and

flexion of the little finger are sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 mL of the local anesthetic agent is deposited.

Ultrasound Guidance

(a) Radial Nerve
The patient is positioned supine with the upper extremity abducted. At the level of the elbow crease, a high-frequency, linear ultrasound probe is used (Fig. 17.25). The radial nerve appears as a hyperechoic crescent (Fig. 17.26). Using an in-plane technique, a 5-cm block needle is advanced toward the nerve. A volume of 5–7 mL of local anesthetic is deposited.

(b) Median Nerve
The patient is supine with the upper extremity abducted. At the level of the elbow crease, a high-frequency, linear ultrasound probe is used (Fig. 17.25). The median nerve is located medial to the brachial artery (Fig. 17.27). Using an in-plane technique, a 5-cm block needle is advanced toward the nerve. A volume of 5–7 mL of local anesthetic is deposited.

(c) Ulnar Nerve
The patient is positioned supine. The elbow is flexed and the forearm internally rotated so that its radial aspect rests comfortably on the torso. A high-frequency, linear ultrasound probe is used to scan the proximal forearm (Fig. 17.28). The ulnar nerve appears as a hyperechoic structure (Fig. 17.29). Using an in-plane technique, a 5-cm block needle is advanced toward the nerve. A volume of 5–7 mL of local anesthetic is deposited.

At the Wrist

(a) Radial Nerve
The radial nerve can be blocked at the wrist without the use of neurostimulation or ultrasonography. A field block is performed by

Fig. 17.25 Position of the ultrasound probe for supplemental median and radial nerve blocks at the elbow

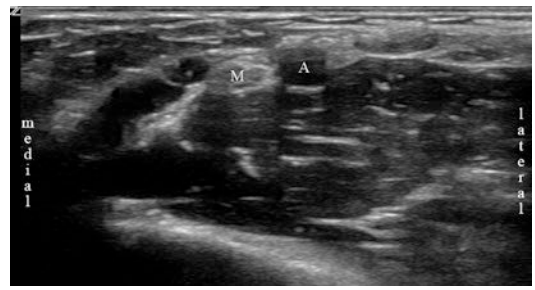

Fig. 17.27 Ultrasonographic appearance of the median nerve at the elbow (*A* brachial artery, *M* median nerve)

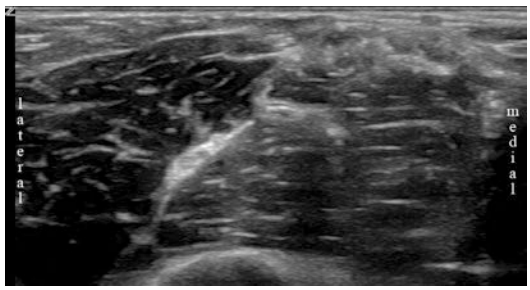

Fig. 17.26 Ultrasonographic appearance of the radial nerve at the elbow

Fig. 17.28 Position of the ultrasound probe for supplemental ulnar nerve block at the elbow

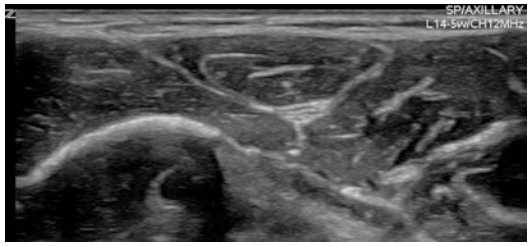

Fig. 17.29 Ultrasonographic appearance of the ulnar nerve at the elbow

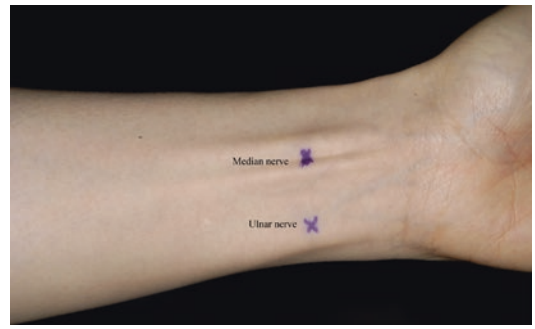

Fig. 17.31 Landmarks for supplemental median and ulnar nerve block at the wrist

Fig. 17.30 Landmarks for supplemental radial nerve block at the wrist (*X* site of infiltration)

Fig. 17.32 Position of the ultrasound probe for supplemental median and ulnar nerve block at the elbow

injecting 5–7 mL of local anesthetic subcutaneously in and around the anatomical "snuff box" (Fig. 17.30).

(b) Median and Ulnar Nerves

Neurostimulation

(a) Median
The median nerve is located between the tendons of the flexor palmaris longus and the flexor carpi radialis (Fig. 17.31). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is introduced approximately 3 cm above the wrist crease. Thumb flexion is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 3–5 mL of local anesthetic is deposited.

(b) Ulnar
The ulnar nerve is located medial to the ulnar artery, below the tendon of the flexor carpi ulnaris muscle. A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is introduced medial to the artery, 3 cm proximal to the wrist crease (Fig. 17.31). Flexion of the fifth finger is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 3–5 mL of local anesthetic is deposited.

Ultrasound Guidance

The patient is positioned supine with the upper extremity abducted. The distal forearm is scanned with a high-frequency, linear ultrasound probe (Fig. 17.32). The median nerve appears in the middle of the screen (Fig. 17.33). The ulnar nerve is medial to the ulnar artery (Fig. 17.34). Using an in-plane technique, a 2.5–5-cm block needle is advanced toward each nerve. A volume of 5–7 mL of local anesthetic is deposited for each nerve.

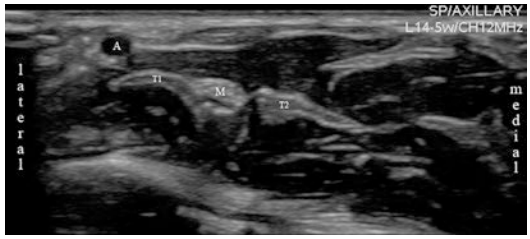

Fig. 17.33 Ultrasonographic appearance of the median nerve at the wrist (*A* radial artery, *M* median nerve, *T1* tendon of the flexor carpi radialis muscle, *T2* tendon of the palmaris longus muscle)

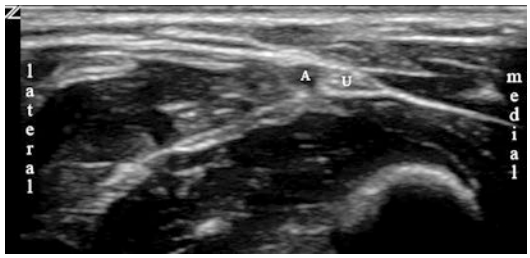

Fig. 17.34 Ultrasonographic appearance of the ulnar nerve at the wrist (*A* ulnar artery, *U* ulnar nerve)

Digital Nerve Block

This block is performed with the hand in the prone position. A 2.5-cm block needle is introduced into the web space of the finger to be anesthetized: this corresponds to the proximal phalanx. A volume of 1–2 mL of local anesthetic is deposited on either side of the finger.

Complications

Most supplemental blocks possess a high safety profile. Vascular puncture (brachial, ulnar, or suprascapular arteries) can occur. For suprascapular nerve blockade, care must be taken not to advance the needle too far past the suprascapular notch: this can lead to a pneumothorax.

Continuous Brachial Plexus Block

Background

Continuous brachial plexus blockade can be achieved using a blind catheter, a technique whereby the block needle locates the plexus with neurostimulation, and the catheter is simply advanced 1–5 cm past the needle tip. Alternatively, a stimulating catheter can be used: after the obtention of a satisfactory evoked motor response with the needle, the nerve stimulator is connected to the catheter to ensure that, during the latter's advancement, muscular contractions are preserved. Lastly, ultrasound guidance can also be used to confirm the proximity of needle and catheter to the brachial plexus.

To date, two trials have compared these different techniques. In the setting of shoulder surgery, blind and ultrasound-guided interscalene catheters resulted in similar pain scores and local anesthetic/opioid consumption postoperatively. However, ultrasonography yielded a slightly quicker performance time and lower block-related pain score [51]. The second study compared blind, stimulating, and ultrasound-guided infraclavicular catheters. Unfortunately, the results are difficult to interpret because of the differences in evoked motor responses between groups [52]. Although four trials have demonstrated that, compared to neurostimulation, ultrasound guidance results in a quicker performance time for interscalene and infraclavicular catheters, these studies did not rigorously assess pain control during the postoperative period [53–56].

The Techniques

Blind and Stimulating Catheters

With landmarks similar to single-shot blocks, the block needle is used to locate the brachial plexus. Because neural structures are very superficial in the interscalene and axillary areas, the needle should be inserted with a flat angle to the skin to facilitate subsequent catheter advancement (Figs. 17.35 and 17.36). Although some authors dilate the perineural sheath with a small bolus (5–10 mL) of D5W prior to threading the catheter, this seems to provide minimal benefits [57]. For blind catheters, normal saline or local anesthetic can be used; for stimulating catheters, D5W will preserve the evoked motor response for catheter

Fig. 17.35 Needle angulation for continuous neurostimulation-guided interscalene brachial plexus block

Fig. 17.36 Needle angulation for continuous neurostimulation-guided axillary brachial plexus block

advancement. After an evoked motor response is obtained with the needle at 0.5 mA, the blind catheter is simply advanced 3–4 cm past the needle tip. A distance greater than 4 cm should be avoided to prevent catheter coiling [58]. If a stimulating catheter is used, the nerve stimulator is disconnected from the needle and connected to the catheter. During the advancement process (3–4 cm), care must be taken to ensure that the evoked motor response and stimulatory threshold do not change. The operator may need to withdraw the catheter into the needle and change the latter's bevel orientation or angulation to accomplish this. After the blind or stimulating catheter has been successfully inserted, the needle is carefully withdrawn over the catheter and the latter secured to the skin with adhesive dressings.

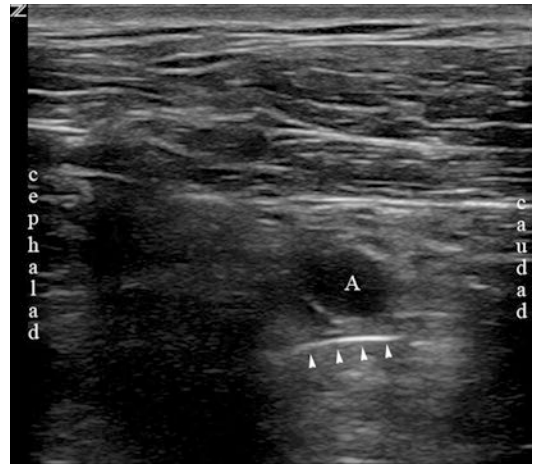

Fig. 17.37 Ultrasonographic appearance of a continuous infraclavicular catheter inserted with ultrasonography (*A* axillary artery, *arrows* indicate catheter)

Ultrasound Guidance

After the bolus of local anesthetic has been injected through the needle, the catheter is advanced 3–4 cm past the needle tip. Care must be taken to visualize in real time its passage into the perineural space (Fig. 17.37). This may require the help of an assistant, as a third hand is needed to hold the probe while the operator inserts the catheter through the needle. If the catheter cannot be seen with certainty, its tip can be identified with the injection of a few milliliters of local anesthetics or saline. Alternately, 1 mL of air can be used. This will produce an unmistakable hyperechoic shadow. Air should be used sparingly in order to preserve the quality of the image. After successful placement of the ultrasound-guided catheter, the needle is carefully withdrawn over the catheter and the latter secured to the skin with adhesive dressings.

Clinical Pearls

Clinical Anatomy of the Brachial Plexus

- Although most textbooks recommend selecting nerve blocks based on the cutaneous innervation of the surgical site (Fig. 17.38),

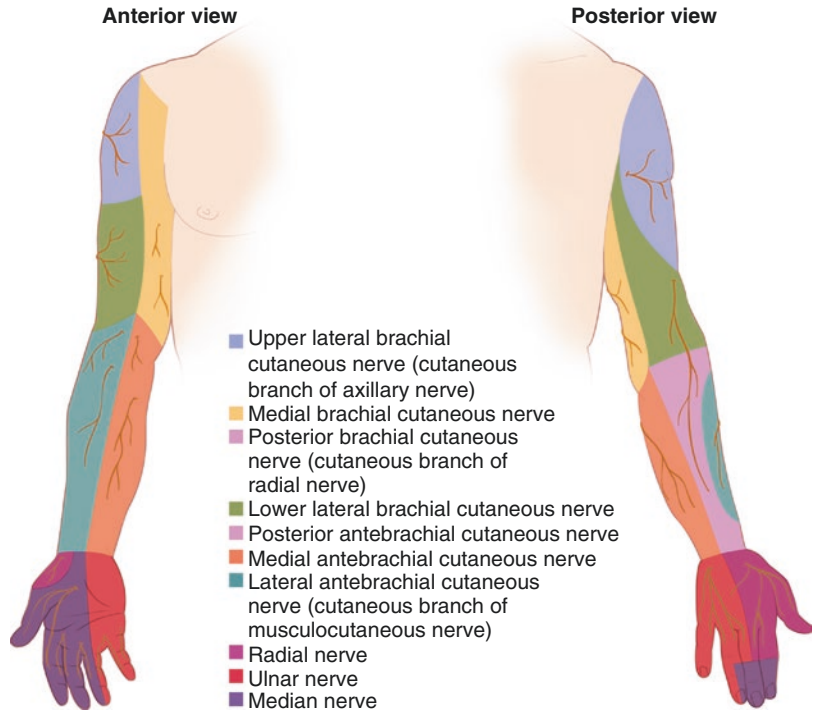

Fig. 17.38 Cutaneous innervation of the upper extremity

knowledge of the osseous innervation (Fig. 17.2) is far more important as postoperative pain rarely stems from trauma to skin.

- The medial and lateral pectoral nerves (which innervate the pectoral muscles) arise from the medial and lateral cords, respectively. Thus, pectoral contraction is an acceptable evoked motor response when performing neurostimulation-guided interscalene or cervical paravertebral brachial plexus block.
- Pectoral contraction should not be accepted for neurostimulation-guided infraclavicular brachial plexus block since it may result from direct stimulation of the pectoral muscles.

Choosing the Right Approach

- For single-shot blocks, the cervical paravertebral, interscalene, and supraclavicular approaches can be used to anesthetize the for shoulder and proximal humeral surgery. For continuous perineural catheters, interscalene and cervical paravertebral blocks offer an advantage over supraclavicular blocks because of the latter's proximity to the surgical site. Cervical paravertebral catheters provide an elegant option because they can be tunneled around the hairline and secured on the nonoperative shoulder.
- In light of the comparable efficacy, the selection between supraclavicular, infraclavicular, axillary, and humeral canal approaches should be dictated by potential adverse events and patient characteristics. For instance, supraclavicular blocks, and their inherent risk of phrenic paralysis, should be avoided in patients with pulmonary compromise. Infraclavicular blocks may be technically difficult in subjects with ample pectoral muscles or breast tissue. Axillary and humeral canal blocks should be avoided in patients (with fractures) who cannot comfortably abduct the upper limb.

Interscalene Brachial Plexus Block

- To ensure that the interscalene groove has been properly identified, palpation of the

latter above the clavicle should reveal the presence of an arterial pulsation (subclavian artery).

- If interscalene blocks are performed postoperatively with nerve stimulation, shoulder elevation (dorsal scapular nerve stimulation) can be mistaken for abduction (brachial plexus stimulation) because of the presence of slings and surgical dressings. Before injecting the local anesthetic, the operator should palpate the deltoid muscle and confirm the presence of abduction.
- If the brachial plexus cannot be identified with ultrasonography at the interscalene level, the supraclavicular area can be scanned to locate the subclavian artery. Typically, the brachial plexus (cluster of trunks and divisions) is situated superolateral to the latter. Next, the plexus is slowly traced back toward the cricoid cartilage until it becomes a column of hypoechoic nodules (roots or trunks).
- For patients in whom phrenic nerve block constitutes a prohibitive risk, combined infraclavicular brachial plexus block and suprascapular nerve block can be used to anesthetize the anterior and posterior shoulder, respectively [59]. Although some authors propose targeted suprascapular and axillary nerve blocks as an alternative to interscalene brachial plexus blocks [60], this option fails to cover the upper humerus, which receives innervation from the radial nerve (Fig. 17.2). Furthermore one cannot insert a perineural catheter next the axillary nerve as it would interfere with the surgical field.

Supraclavicular Brachial Plexus Block

- For neurostimulation, a distal evoked motor response (wrist or hand) seems to provide a better block.
- The risk of pneumothorax is decreased when this block is performed with ultrasound guidance because the entire length of the needle can be visualized.
- Because of its multiple targets, the targeted intracluster injection technique may prove difficult for beginners. The double-injection technique, whereby half the local anesthetic volume is injected inside the largest neural cluster and half, at the "corner pocket" (intersection of the first rib and subclavian artery), provides a simpler alternative. However onset time will be slower [24].

Infraclavicular Brachial Plexus Block

- Magnetic resonance imaging reveals a great deal of anatomic variability in the location of the three cords around the axillary artery. For instance, despite its name, the medial cord is usually posterior (dorsal) to the artery [61].
- With the pericoracoid technique, in order to minimize the risk of pneumothorax, the needle should never be directed medially when searching for the appropriate evoked motor response.
- If the needle tip is correctly located with ultrasonography, injection of the first few milliliters of local anesthetic will give a picture resembling a "double bubble" [62]. The "superior bubble" represents the axillary artery in a short axis, and the "inferior bubble," the pool of local anesthetic (Fig. 17.39). As more local anesthetic is deposited, the inferior bubble will turn into a U shape,

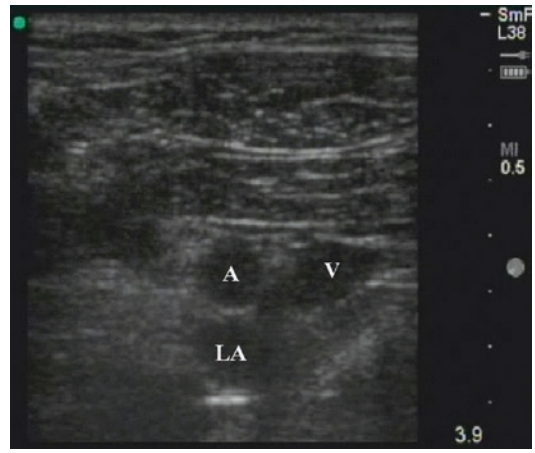

Fig. 17.39 The "double bubble" sign (*A* axillary artery, *LA* local anesthetic agent, *V* axillary vein)

wrapping itself around the artery, and the latter will be gently pushed ventrally. If a "double bubble" fails to form or the artery does not rise with injection, the needle tip may be too dorsal in relation to artery; thus, it should be repositioned to lie immediately adjacent to the vessel.
- Occasionally, two axillary veins (cephalad and caudad to the artery) can be present. In this situation, another approach should be considered to avoid vascular puncture.

Axillary Brachial Plexus Block

- In some patients, the musculocutaneous nerve travels with the axillary artery instead of inside the coracobrachialis/biceps muscles. If the musculocutaneous nerve cannot be seen (ultrasound guidance) or elicited (neurostimulation) inside the latter, the entire volume of local anesthetic is injected at the 6 o'clock position of the axillary artery (ultrasound guidance) or upon elicitation of the median, radial, and ulnar nerves (neurostimulation).
- Care must be taken not to apply too much pressure with the ultrasound probe. This may lead to compression of superficial veins and unrecognized intravascular injection [63].

Humeral Canal Block

- For ultrasound-guided humeral canal blocks, if the radial nerve cannot be identified dorsal to the humerus, the probe can be moved proximally toward the axilla: the nerve will be located more superficially around the brachial artery.
- If the musculocutaneous, median, and ulnar nerves cannot be identified, they can be traced from the axilla downward. Alternatively, the median and ulnar nerves can be traced back from the elbow.
- In their practice, the authors seldom use humeral canal blocks, as they provide no clear benefits compared to their axillary counterparts.

Supplemental Blocks

- At the elbow, for ulnar nerve block, local anesthetic should not be injected directly into the groove: high pressure in this tight fascial compartment can damage the nerve.
- Although many textbooks recommend supplementing brachial plexus blocks with an intercostobrachial nerve block (subcutaneous infiltration of the medial arm with 5–7 mL of local anesthetic) for tourniquet tolerance, this step is seldom necessary. Tourniquet-related pain stems from muscular compression and should be covered by the brachial plexus block. In contrast, the intercostobrachial nerve only provides sensory innervation to the skin.
- For digital nerve block, epinephrine must be avoided in the local anesthetic solution as this may produce ischemia of the fingertips.

Continuous Brachial Plexus Block

- The insertion of stimulating perineural catheters may require multiple attempts. A systematic approach is required to find the optimal combination of needle angulation and bevel orientation. The needle is first rotated 90° at a time to attempt catheter advancement. After the four quadrants have been unsuccessfully explored, the needle angulation is changed and the four quadrants tried again. These two steps (change of angulation and exploration of four quadrants) are repeated until the catheter can be inserted 3–4 cm past the needle tip with preservation of the evoked motor response.
- The optimal stimulatory threshold for perineural catheter placement has not been established. In their practice, the authors tolerate a threshold as high as 1.0 mA (pulse width = 0.1 ms).
- If the perineural catheter is required for a longer postoperative period (e.g., to facilitate complex rehabilitation), the authors prefer neurostimulation over ultrasound guidance in order to minimize the risk of dislodgement.

Case Study

A 75-year-old patient requires surgical repair of a left upper humeral fracture sustained during a fall from his own height. The patient has a long-standing history of osteoarthritis and stable hypertension. Ten years ago, he had undergone a right pneumonectomy for the (successful) treatment of lung cancer. Besides the antihypertensive agents, the patient is taking no other medications. His exercise tolerance is difficult to assess in light of decreased mobility due to osteoarthritis.

What Are the Options for Postoperative Analgesia?

Although general anesthesia can be provided for intraoperative care, effective (and safe) postoperative analgesia remains a challenge. Surgical procedures of the shoulder are notoriously painful for patients. Intravenous opioids coupled with multimodal analgesia (acetaminophen, selective COX 2 inhibitors, pregabalin) may not provide sufficient pain relief. Regional anesthesia constitutes a more effective strategy. The anterior and posterior aspects of the proximal humerus receive innervation from the axillary/radial and suprascapular/radial nerves, respectively (Fig. 17.2). Of these three branches, the suprascapular nerve leaves the brachial plexus the earliest (upper trunk) (Fig. 17.1). Thus the operator would need to anesthetize the brachial plexus at the level of the roots or trunks. The interscalene, cervical paravertebral, and supraclavicular approaches would theoretically fit the bill. Unfortunately, in light of the previous right pneumonectomy, left phrenic nerve block (and left hemidiaphragmatic paralysis) constitutes a prohibitive obstacle. Despite decreases in local anesthetic volume, interscalene, cervical paravertebral, and supraclavicular blocks still carry a significant risk of phrenic nerve block.

Therefore the optimal analgesic strategy for this patient requires anesthetizing the axillary, radial, and suprascapular nerves. The first two originate from the posterior cord and could be efficiently targeted with an infraclavicular block (Fig. 17.13). The suprascapular nerve could be selectively blocked in the suprascapular fossa (Fig. 17.22). Moreover, since both infraclavicular and suprascapular fossae are located far from the surgical site (upper humerus), perineural catheters could be left in place to maximize postoperative analgesia.

Acknowledgments The authors thank Dr. Shubada Dugani for her invaluable help in acquiring the figures displayed in this chapter.

Review Questions

1. All the following nerves originate from the brachial plexus EXCEPT:
   (a) Thoracodorsal nerve
   (b) Intercostobrachial nerve
   (c) Lateral pectoral nerve
   (d) Long thoracic nerve
2. All the following nerves originate from the C5 nerve root EXCEPT:
   (a) Suprascapular nerve
   (b) Dorsal scapular nerve
   (c) Phrenic nerve
   (d) Ulnar nerve
3. An injury to the posterior cord will lead to all the following deficits EXCEPT:
   (a) Decreased shoulder abduction
   (b) Decreased elbow extension
   (c) Decreased sensation of the lateral aspect of the shoulder
   (d) Decreased sensation over the medial aspect of the forearm
4. For clavicular surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Cervical paravertebral block
   (b) Supraclavicular block
   (c) Superficial cervical plexus block
   (d) None of the above
5. For shoulder surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Infraclavicular combined with suprascapular nerve blocks
   (b) Infraclavicular combined with superficial cervical plexus blocks

(c) Cervical paravertebral block
(d) None of the above

6. For elbow surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Infraclavicular block
   (b) Axillary block
   (c) Supraclavicular block
   (d) None of the above
7. For hand surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Interscalene block
   (b) Humeral canal block
   (c) Supraclavicular block
   (d) None of the above
8. All the following are potential side effects of interscalene blocks EXCEPT:
   (a) Hoarseness
   (b) Exophthalmos
   (c) Myosis
   (d) Dyspnea
9. All the following are potential side effects of infraclavicular blocks EXCEPT:
   (a) Horner's syndrome
   (b) Dyspnea
   (c) Winged scapula
   (d) Perioral numbness
10. All the following evoked motor responses are acceptable for neurostimulation-guided interscalene blocks EXCEPT:
    (a) Shoulder elevation
    (b) Pectoral contraction
    (c) Finger flexion
    (d) Wrist extension
11. All the following evoked motor responses are acceptable for neurostimulation-guided infraclavicular blocks EXCEPT:
    (a) Pectoral contraction
    (b) Elbow extension
    (c) Wrist extension
    (d) Finger flexion
12. Which of the following evoked motor responses is considered suboptimal in the performance of neurostimulation-guided axillary blocks?
    (a) Elbow flexion
    (b) Elbow extension
    (c) Thumb opposition
    (d) Wrist extension
13. With ultrasonography, all the following structures are hyperechoic EXCEPT:
    (a) Musculocutaneous nerve
    (b) Lateral cord
    (c) Superior trunk
    (d) Median nerve in the forearm
14. With ultrasonography, all the following structures are hypoechoic EXCEPT:
    (a) Inferior trunk
    (b) Phrenic nerve
    (c) C7 root
    (d) Medial cord
15. With ultrasonography, all the following nerves can be anesthetized using a perivascular injection EXCEPT:
    (a) Interscalene brachial plexus
    (b) Axillary brachial plexus
    (c) Median nerve at the elbow
    (d) Ulnar nerve at the wrist

Answers

1. b
2. d
3. d
4. c
5. b
6. d
7. a
8. b
9. c
10. a
11. a
12. b
13. c
14. d
15. a

References

1. Boezaart AP. That which we call a rose by any other name would smell as sweet—and its thorns would hurt as much. Reg Anesth Pain Med. 2009;34:3–7.
2. Rettig HC, Gielen MJM, Jack NTM, Boersma E, Klein J. A comparison of the lateral and posterior approach for brachial plexus block. Reg Anesth Pain Med. 2006;31:119–26.

3. Dewees JL, Schultz CT, Wilkerson FK, Kelly JA, Biegner AR, Pellegrini JE. Comparison of two approaches to brachial plexus anesthesia for proximal upper extremity surgery: interscalene and intersternocleidomastoid. AANA J. 2006;74:201–6.
4. Deleuze A, Gentili M, Marret E, Lamonerie L, Bonnet F. A comparison of a single-stimulation lateral infraclavicular plexus block with a triple-stimulation axillary block. Reg Anesth Pain Med. 2003;28:89–94.
5. Koscielniak-Nielsen ZJ, Rasmussen H, Hesselbjerg L, Nielsen TP, Gurkan Y. Infraclavicular block causes less discomfort than axillary block in ambulatory patients. Acta Anaesthesiol Scand. 2005;49:1030–4.
6. Minville V, Fourcade O, Idabouk L, et al. Infraclavicular brachial plexus block versus humeral block in trauma patients: a comparison of patient comfort. Anesth Analg. 2006;102:912–6.
7. Tran DQH, Russo GL, Munoz L, Zaouter C, Finlayson RJ. A prospective, randomized comparison between ultrasound-guided supraclavicular, infraclavicular and axillary brachial plexus blocks. Reg Anesth Pain Med. 2009;34:366–71.
8. Liguori GA, Zayas VM, YaDeau JT, et al. Nerve localization techniques for interscalene brachial plexus blockade: a prospective, randomized comparison of mechanical paresthesia versus electrical stimulation. Anesth Analg. 2006;103:761–7.
9. Smith BL. Efficacy of a nerve stimulator in regional anesthesia; experience in resident training programme. Anaesthesia. 1976;31:778–82.
10. Boezaart A, de Beer J, du Toit C, van Rooyen K. A new technique of continuous interscalene nerve block. Can J Anesth. 1999;46:275–81.
11. Kapral S, Greher M, Huber G, et al. Ultrasonographic guidance improves the success rate of interscalene brachial plexus blockade. Reg Anesth Pain Med. 2008;33:253–8.
12. Liu SS, Zayas VM, Gordon MA, et al. A prospective, randomized, controlled trial comparing ultrasound versus nerve stimulator guidance for interscalene block for ambulatory shoulder surgery for postoperative neurological symptoms. Anesth Analg. 2009;109:265–71.
13. Spence BC, Beach ML, Gallagher JD, Sites BD. Ultrasound-guided interscalene blocks: understanding where to inject the local anesthetic. Anaesthesia. 2011;66:509–14.
14. Urmey WF, Talts KH, Sharrock NE. One hundred percent incidence of hemidiaphragmatic paresis associated with interscalene brachial plexus anesthesia as diagnosed by ultrasonography. Anesth Analg. 1991;72:498–503.
15. Urmey WF, Gloeggler PJ. Pulmonary function changes during interscalene brachial plexus block: effects of decreasing local anesthetic injection volume. Reg Anesth. 1993;18:244–9.
16. Urmey WF, Grossi P, Sharrock N, Stanton J, Gloeggler PJ. Digital pressure during interscalene block is clinically ineffective in preventing anesthetic spread to the cervical plexus. Anesth Analg. 1996;83:366–70.
17. Bennani SE, Vandenabele-Teneur F, Nyarwaya JB, Delecroix M, Krivosic-Horber R. An attempt to prevent spread of local anesthetic to the phrenic nerve by compression above the injection site during interscalene brachial plexus block. Eur J Anaesthesiol. 1998;15:453–6.
18. Sala-Blanch X, Lazaro JR, Correa J, Gomez-Fernandez M. Phrenic nerve block caused by interscalene brachial plexus block: effects of digital pressure and a low volume of local anesthetic. Reg Anesth Pain Med. 1999;24:231–5.
19. Al-Kaisy AA, Chan VWS, Perlas A. Respiratory effects of low-dose bupivacaine interscalene block. Br J Anaesth. 1999;82:217–20.
20. Stundner O, Meissnitzer M, Brummett CM, et al. Comparison of tissue distribution, phrenic nerve involvement, and epidural spread in standard- vs low-volume ultrasound-guided interscalene plexus block using contrast magnetic resonance imaging: a randomized, controlled trial. Br J Anaesth. 2016;116:405–12.
21. Boezaart AP, de Beer JF, Nell ML. Early experience with continuous cervical paravertebral block using a stimulating catheter. Reg Anesth Pain Med. 2003;28:406–13.
22. Brown DL, Cahill DR, Bridenbaugh D. Supraclavicular nerve block: anatomic analysis of a method to prevent pneumothorax. Anesth Analg. 1993;76:530–4.
23. Franco CD, Domashevich V, Voronov G, Rafizad AB, Jelev TJ. The supraclavicular block with a nerve stimulator: to decrease or not to decrease, that is the question. Anesth Analg. 2004;98:1167–71.
24. Techasuk W, González AP, Bernucci F, Cupido T, Finlayson RJ, Tran DQH. A randomized comparison between double-injection and targeted intracluster-injection for ultrasound-guided supraclavicular brachial plexus block. Anesth Analg. 2014;118:1363–9.
25. Renes SH, Spoormans HH, Gielen MH, Rettig HC, van Geffen JG. Hemidiaphragmatic paresis can be avoided in ultrasound-guided supraclavicular brachial plexus block. Reg Anesth Pain Med. 2009;34:595–9.
26. Tran DQH, Dugani S, Correa JA, Dyachenko A, Alsenosy N, Finlayson RJ. Minimum effective volume for ultrasound-guided supraclavicular block. Reg Anesth Pain Med. 2009;34:215–8.
27. Knoblanche GE. The incidence and aetiology of phrenic nerve blockade associated with supraclavicular brachial plexus block. Anaesth Intensive Care. 1979;7:346–9.
28. Rodriguez J, Taboada M, Oliveira J, et al. Single-stimulation of the posterior cord is superior to dual nerve stimulation in a coracoid block. Acta Anaesthesiol Scand. 2010;54:241–5.
29. Tran DQH, Bertini P, Zaouter C, Munoz L, Finlayson RJ. A prospective, randomized comparison between single- and double-injection ultrasound-guided infraclavicular brachial plexus block. Reg Anesth Pain Med. 2010;35:16–21.
30. Desgagnes MC, Levesque S, Dion S, et al. A comparison of a single or triple injection technique for

ultrasound-guided infraclavicular block: a prospective randomized controlled study. Anesth Analg. 2009;109:668–72.
31. Sauter AR, Dodgson MS, Stubhaug A, Halstensen AM, Klaastad O. Electrical nerve stimulation or ultrasound guidance for lateral sagittal infraclavicular blocks: a randomized, controlled, observer-blinded, comparative study. Anesth Analg. 2008;106:1910–5.
32. Taboada M, Rodriguez J, Amor M, et al. Is ultrasound guidance superior to conventional nerve stimulation for coracoid infraclavicular brachial plexus block? Reg Anesth Pain Med. 2009;34:357–60.
33. Brull R, Lupu M, Perlas A, Chan VWS, McCartney CJL. Compared with nerve stimulation, ultrasound guidance shortens the time for infraclavicular block performance. Can J Anesth. 2009;56:812–8.
34. Dingemans E, Williams SR, Arcand G, et al. Neurostimulation in ultrasound-guided infraclavicular block: a prospective randomized trial. Anesth Analg. 2007;104:1275–80.
35. Gurkan Y, Tekin M, Acar S, Solak M, Toker K. Is nerve stimulation needed during an ultrasound-guided lateral sagittal infraclavicular block? Acta Anaesthesiol Scand. 2010;54:403–7.
36. Raj PP, Montgomery SJ, Nettles D, Jenkins MT. Infraclavicular brachial plexus block—a new approach. Anesth Analg. 1973;52:897–904.
37. Wilson JL, Brown DL, Wong GY, et al. Infraclavicular brachial plexus block: parasagittal anatomy important to the coracoid technique. Anesth Analg. 1998;87:870–3.
38. Tran DQH, Dugani S, Dyachenko A, Correa JA, Finlayson RJ. Minimum effective volume of lidocaine for ultrasound-guided infraclavicular brachial plexus block. Reg Anesth Pain Med. 2011;36:190–4.
39. Petrar SD, Seltenrich ME, Head SJ, Schwarz SKW. Hemidiaphragmatic paralysis following ultrasound-guided supraclavicular versus infraclavicular brachial plexus blockade. Reg Anesth Pain Med. 2015;40:133–8.
40. Crews JC, Gerancher JC, Wellers RS. Pneumothorax after coracoid infraclavicular brachial plexus block. Anesth Analg. 2007;105:275–7.
41. Tran DQH, Clemente A, Doan J, Finlayson RJ. Brachial plexus blocks: a review of approaches and techniques. Can J Anesth. 2007;54:662–74.
42. Sia S, Bartoli M. Selective ulnar nerve localization is not necessary for axillary brachial plexus block using a multiple nerve stimulation technique. Reg Anesth Pain Med. 2001;26:12–6.
43. Sia S. A comparison of injection at the ulnar and the radial nerve in axillary block using triple stimulation. Reg Anesth Pain Med. 2006;31:514–8.
44. Sia S, Lepri A, Magherini M, Doni L, Di Marco P, Gritti G. A comparison of proximal and distal radial nerve motor responses in axillary block using triple stimulation. Reg Anesth Pain Med. 2005;30:458–63.
45. Chan VWS, Perlas A, McCartney CJL, Brull R, Xu D, Abbas S. Ultrasound guidance improves success rate of axillary brachial plexus block. Can J Anesth. 2007;54:176–82.
46. Bernucci F, González F, Finlayson RJ, Tran DQH. A prospective randomized comparison between perivascular and perineural ultrasound-guided axillary brachial plexus block. Reg Anesth Pain Med. 2012;37:473–7.
47. González AP, Bernucci F, Pham K, Coorea JA, Finlayson RJ, Tran DQH. Minimum effective volume of lidocaine for double-injection ultrasound-guided axillary block. Reg Anesth Pain Med. 2013;38:16–20.
48. Tran DQH, Pham K, Dugani S, Finlayson RJ. A prospective, randomized comparison between double-, triple-, and quadruple-injection ultrasound-guided axillary brachial plexus block. Reg Anesth Pain Med. 2012;37:248–53.
49. Carles M, Pulcini A, Macchi P, Duflos P, Raucoules-Aime M, Grimaud D. An evaluation of the brachial plexus block at the humeral canal using a neurostimulator (1417 patients): the efficacy, safety, and predictive criteria of failure. Anesth Analg. 2001;92:194–8.
50. Peng PWH, Wiley MJ, Liang J, Bellingham GA. Ultrasound-guided suprascapular nerve block: a correlation with fluoroscopic and cadaveric findings. Can J Anesth. 2010;57:143–8.
51. Fredrickson MJ, Ball CM, Dagleish AJ, Stewart AW, Short TG. A prospective randomized comparison of ultrasound and neurostimulation as needle end points for interscalene catheter placement. Anesth Analg. 2009;108:1695–700.
52. Dhir S, Ganapathy S. Comparative evaluation of ultrasound-guided continuous infraclavicular brachial plexus block with stimulating catheter and traditional technique: a prospective-randomized trial. Acta Anaesthesiol Scand. 2008;52:1158–66.
53. Mariano ER, Loland VJ, Sandhu NS, et al. A trainee-based randomized comparison of stimulating interscalene perineural catheter with a new technique using ultrasound guidance alone. J Ultrasound Med. 2010;29:329–36.
54. Mariano ER, Loland VJ, Bellars RH, et al. Ultrasound guidance versus electrical stimulation for infraclavicular brachial plexus perineural catheter insertion. J Ultrasound Med. 2009;28:1211–8.
55. Danelli G, Bonarelli S, Tognú A, et al. Prospective randomized comparison of ultrasound-guided and neurostimulation techniques for continuous interscalene brachial plexus block in patients undergoing coracoacromial ligament repair. Br J Anaesth. 2012;108:1006–10.
56. Dhir S, Armstrong K, Armstrong P, et al. A randomized comparison between ultrasound and nerve stimulation for infraclavicular catheter placement. Aanesthesia. 2016;71:198–204.
57. Ficarotta MR, Morey TE, Boezaart AP. Does "Opening the Perineural Space" before stimulating catheter placement for continuous nerve block add value in clinical practice? Reg Anesth Pain Med. 2010;35:245–8.

58. Tran DQH, De La Cuadra Fontaine JC, Chan SY, Kovarik G, Asenjo JF, Finlayson RJ. Coiling of stimulating perineural catheters. Anesthesiology. 2007;106:189–90.
59. Martinez J, Sala-Blanch X, Ramos I, Gomar C. Combined infraclavicular plexus block with suprascapular nerve block for humeral head surgery in a patient with respiratory failure: an alternative approach. Anesthesiology. 2003;98:784–5.
60. Price Price DJ. The shoulder block: a new alternative to interscalene brachial plexus blockade for the control of postoperative shoulder pain. Anaesth Intensive Care. 2007;35:575–81.
61. Sauter AR, Smith HJ, Stubhaug A, et al. Use of magnetic resonance imaging to define the anatomical location closest to all three cords of the infraclavicular brachial plexus. Anesth Analg. 2006;103(6):1574.
62. Tran DQH, Charghi R, Finlayson RJ. The "Double Bubble" sign for successful infraclavicular brachial plexus blockade. Anesth Analg. 2006;103:1048–9.
63. Loubert C, Williams SR, Helie F, Arcand G. Complication during ultrasound-guided regional block: accidental intravascular injection of local anesthetic. Anesthesiology. 2008;108:759–60.

Suggested Reading

Tran DQH, Clemente A, Doan J, Finlayson RJ. Brachial plexus blocks: a review of approaches and techniques. Can J Anesth. 2007;54:662–74.
Neal JM, Gerancher JC, Hebl JR, Ilfeld BM, McCartney CJL, Franco CD, et al. Upper extremity regional anesthesia- essentials of our current understandings 2008. Reg Anesth Pain Med. 2009;34:134–70.

18 Local Anesthesia of the Masticatory Region

Henry A. Gremillion, Christopher J. Spencer, Alex D. Ehrlich, and Janice A. Townsend

Introduction

Local anesthetics are used in a wide range of clinical situations in dentistry. Some indications are to alleviate sensory input, thus minimizing discomfort during treatment procedures, to treat inflammatory and chronic pain, and for diagnostic and prognostic purposes. Knowledge of the scope of anesthesia effects and specific anatomical factors associated with proper technique is essential for their safe and efficacious use in order to achieve intended goals.

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The trigeminal nerve provides the vast majority of sensory innervation to the masticatory region. The cell bodies for the sensory fibers comprise the semilunar (Gasserian) ganglion, which lies in Meckel's cave in the inferior-medial aspect of the middle cranial fossae. Originating in the ganglion are the three major branches of nerve: the ophthalmic, maxillary, and mandibular divisions. Knowledge of the neural pathways of the maxillary and mandibular divisions is essential for effective deposition of local anesthetic agents.

Maxillary Nerve Anesthesia

The maxillary nerve is purely sensory. It passes through the foramen rotundum to enter the pterygopalatine fossa. Branches of the maxillary nerve supply sensory innervation to the hard (teeth and bone) and soft (mucosa and gingival) tissue of the upper jaw. The following nerve blocks are those primarily utilized in daily practice.

Local anesthesia is typically administered via infiltration (superperiosteal injections) or nerve block injections. Infiltration is when local anesthetic is delivered near the apices of the teeth and it diffuses through cortical bone. It is most effective for pulpal anesthesia of the maxillary teeth and surrounding periodontium (bone and gingiva). Typically local infiltration has a quick onset and is only adequate for one to two teeth. If local anesthesia is required for multiple teeth block injections are preferred.

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_18

The Posterior Superior Alveolar Block

The area anesthetized by this block includes much of the posterior and lateral aspects of the tuberosity of the maxilla with its associated mucosa. Pulpal anesthesia for the maxillary second and third molars and the distal and palatal roots of the maxillary first molar is also achieved with this block. The placement of the needle for administration of the anesthetic is posterior and superior to the maxillary second molar. The needle is directed toward the distal aspect of the maxillary tuberosity where branches of the posterior superior nerve enter into the maxilla via their respective alveolar foramina (Fig. 18.1).

The most common complication for anesthetic injections into the region involves needle trauma to the superior alveolar venous plexus or posterior superior artery, which results in hemorrhage, swelling, and hematoma formation. The hematoma may become visible externally on the face. Harn et al. describe a triangle of safety just superior to the maxillary second molar that is 99% free of significant vascularization [1], which entails reduced risk of vascular compromise. Harn reports decrease in hematoma formation with precautions. Precautions include placement of the needle with the bevel toward the periosteum and utilization of an aspirating syringe in order to avoid intravascular injection of anesthetic. It is important that needles be replaced if bone is bumped during an injection or when several injections are to be performed. The needle tip can become barbed or jagged which will increase the risk to neuronal or vascular tissues in the region. Another rare complication is transient diplopia or strabismus, which can be very disconcerting to the individuals involved [2]. No absolute explanation has been made for this type of complication. The distal placement of the anesthetic needle does also present a very small risk for needle breakage; however, the risk is much less than that of the inferior alveolar nerve block [3].

The Middle Superior Alveolar Block

The area anesthetized with this block includes lateral aspect of the maxillary alveolar process with its associated mucosa. The middle superior alveolar nerve provides innervation for the mesiobuccal root of the maxillary first molar, the primary molars, the second premolar, and likely the first premolar. The posterior superior alveolar nerve and middle superior alveolar nerves may form an anastomosis, which results in the superior dental plexus, which can then provide joint innervations to the maxillary posterior teeth. The middle superior alveolar nerve is contained in a thin "rib" of bone in the lateral aspect of the maxillary sinus. When performing this block, the needle is placed superior to the first maxillary

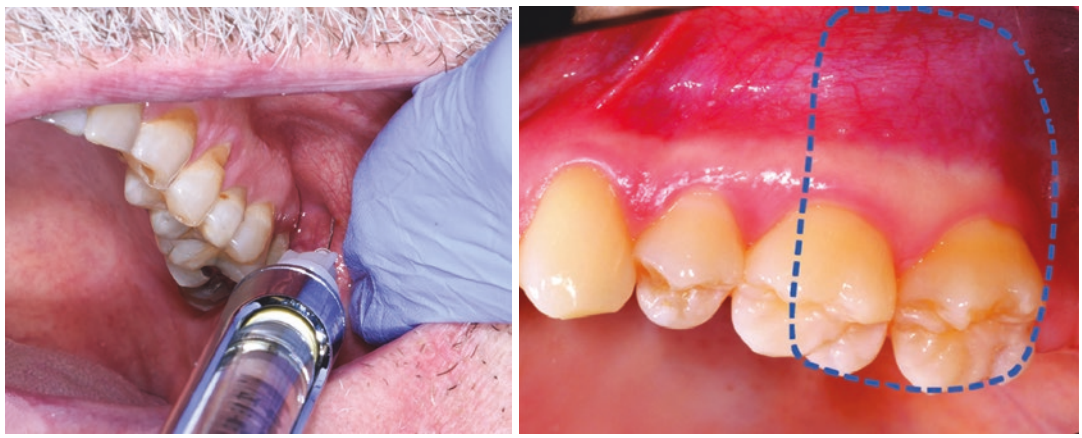

Fig. 18.1 Area anesthetized utilizing the posterior superior alveolar nerve block

molar anterior to the zygomatic process of the maxilla high in the buccal vestibule under the mucosa where approximately 1 mL of anesthetic is deposited. There are few reported adverse reactions associated with the middle superior alveolar nerve block. However, it must be remembered that all local anesthetics are both neurotoxic and myotoxic with small risks associated even with dental injections (Fig. 18.2).

The Anterior Superior Alveolar Block

The region anesthetized by this block includes the anterior aspect of the maxillary alveolar process with its associated mucosa and the innervation for the maxillary cuspid, lateral incisor, and central incisor. The anterior superior alveolar nerve runs in a thin "rib" of bone in the anterior wall of the maxillary sinus approximating the nasal labial fold externally. When performing this block, the needle is directed to the labial vestibule superior to the maxillary canine and lateral incisor. The injection is made submucosally with a deposit of approximately 1 mL of anesthetic. There have been few reported adverse reactions with the anterior superior alveolar block (Fig. 18.3).

Infraorbital Block

The region anesthetized by the infraorbital block is widespread, providing dermal, alveolar process, and dental anesthesia. The dermal region of

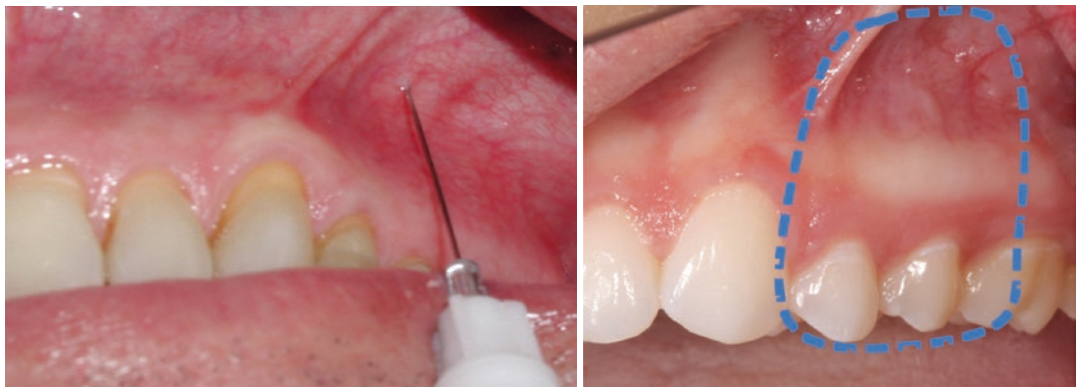

Fig. 18.2 Area anesthetized utilizing the middle superior alveolar nerve block

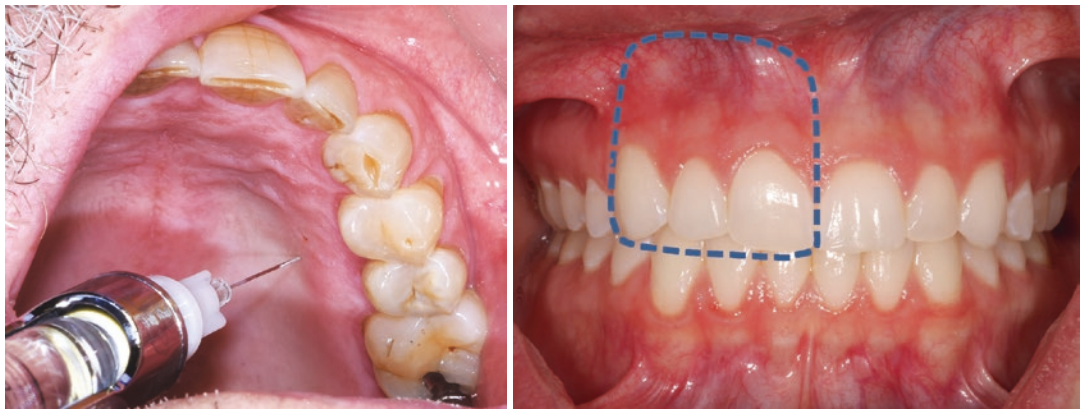

Fig. 18.3 Area anesthetized utilizing the anterior superior alveolar nerve block

anesthesia involves the upper lip, lateral aspect of the nose, and the region below the eye to the lateral canthus of the eye [4]. Intraorally, the anesthesia is provided to the alveolar process and the central incisor, lateral incisor, canine, and first premolar, second premolar, and usually the buccal root of the first maxillary molar (likely through the anterior superior alveolar nerve and middle superior alveolar nerve) along with the adjacent alveolar process and mucosa. There have been reports that the infraorbital blocks provide profound pulpal anesthesia for second premolars, first premolars, and canines (but not central incisors and lateral incisors) for endodontic procedures [5].

The target zone for this injection is the infraorbital foramen, which can be externally palpated below the orbital rim (a depression). This orientation of the orbital rim makes the injection safe as a bony barrier between the infraorbital nerve and the contents of the orbit. The foramen is readily accessible via intraoral approach. The alveolar process is flattened in this region and provides reasonable access to the infraorbital foramen. A finger can be placed extraorally palpating the infraorbital notch, which lies immediately superior to the foramen. The needle is directed to the buccal vestibule superior to the maxillary first premolar. Once the needle approximates the foramen, approximately 1 mL of anesthetic is deposited. While an extraoral approach is possible, the intraoral approach is more kind to the patient since the mucosa is readily anesthetized with 20% benzocaine and is much easier to penetrate. The intraoral approach also minimizes the chance of infraorbital bruising. No difference in efficacy for intraoral and extraoral approaches has been found [6]. There have been few reports of adverse events associated with this anesthetic block (Fig. 18.4).

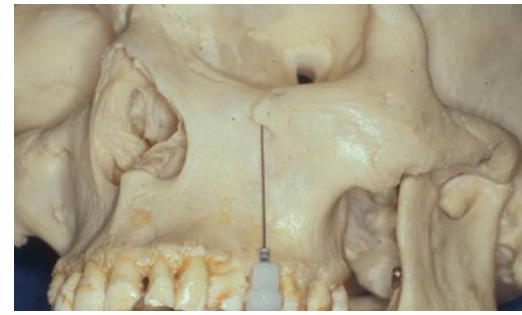

Fig. 18.4 Needle approximating the infraorbital foramen

Greater Palatine Block

The region anesthetized by the greater palatine block is the hard palate including the palatal process of the maxilla and the soft tissues overlying the bony plate beginning anteriorly at the level of the first premolar extending to the posterior aspect of the hard palate from the midline to the lingual marginal gingival surrounding the teeth. The target area for this block is the greater palatine foramen located distal, medial, and superior to the maxillary second molar. If the patient is in a full primary dentition the injection should be administered approximately 10 mm posterior to the distal surface of the second primary molar [7]. This injection site is located clinically with digital palpation of the depression in the soft tissue formed by the foramen. The needle will likely need to be bent in a 45° angle approximately 1 in. from the tip [8] so that the tip of the needle can probe for the opening of the foramen which is 2–3 mm in diameter. The palatal tissue is very fibrous and tightly bound to the hard palate. Thus expression of anesthetic in this area can result in significant discomfort to the patient. Preinjection application of topical benzocaine 20% may reduce discomfort associated with insertion of the needle; however, the topical anesthetic effect does not eliminate pain associated with injection of the initial bolus of local anesthetic [9]. Utilization of pressure and/or cold [10] with pressure can alleviate much of this discomfort. The handle of a dental mirror can be pressed against the tissue firmly while the initiation of the injection is made. Once a few drops of local anesthetic are deposited, the mirror handle may be removed. Slow, gradual deposition of local anesthetic is recommended. One study concluded that the injection pressure below 306 mm of mercury [11] was effective in pain reduction. Use of Computer Controlled

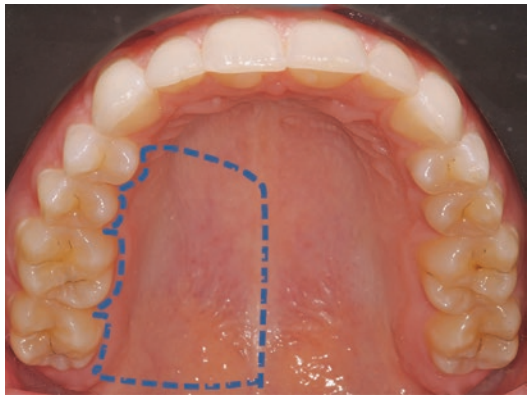

Fig. 18.5 Area anesthetized utilizing the greater palatine nerve block

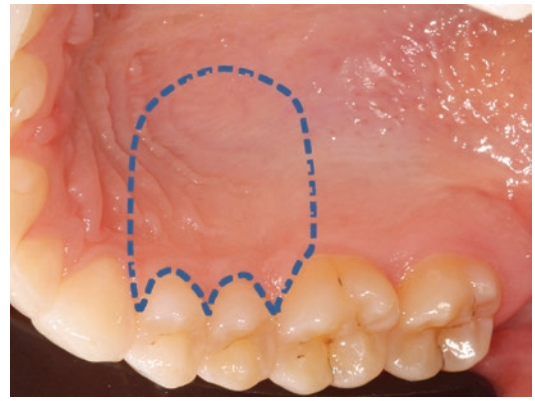

Fig. 18.6 Area anesthetized utilizing the anterior middle superior nerve block

Local Anesthetic Delivery (**CCLAD**) has been shown to significantly reduce the pain perception of palatal anesthesia in children [12]. There are few reports of adverse effects from this injection (Fig. 18.5).

Anterior Middle Superior Alveolar Block

The region anesthetized includes the palate and associated soft tissue adjacent to the first and second premolars. The target zone is the palatal tissue approximating half of the distance between the midpalatal suture and the marginal gingiva of the first and second premolars. The needle is placed, and 0.5 mL of anesthetic is deposited into the very dense fibrous palatal tissue [13]. Often the tissue blanches indicating the extent of the anesthesia to the tissue. As with any palatal injections, pressure or cold anesthesia can help to alleviate some of the discomfort associated with this block. There are few reports of adverse effects from this anesthetic block (Fig. 18.6).

Maxillary Nerve Block (V_2)

The maxillary nerve (V_2) enters the pterygopalatine fossa through foramen rotundum. This region may be accessed by several approaches.

Greater Palatine Foramen Approach

Using the same technique for the greater palatine nerve block, the greater palatine foramen can be located. This provides access to the palatine canal through which the second division of the trigeminal nerve which lies within the pterygopalatine fossa may be anesthetized [14]. The maxillary nerve block effectively anesthetizes all branches of the maxillary nerve including the greater and lesser palatine, posterior superior alveolar, middle superior alveolar, anterior superior alveolar, nasal palatine, and infraorbital nerves. This approach also provides anesthesia to the parasympathetic nerve fibers, which synapse in the sphenopalatine ganglion and even some sympathetic that travel through the region.

Once the greater palatine foramen is located, the needle (27 gauge long) is advanced within the palatine canal with a gentle probing with redirection-as-needed technique. The needle may need to be withdrawn slightly and redirected if progress up the canal is impeded. Once the needle has been advanced half to two-third of its length, 0.5–1 mL of anesthetic is deposited slowly. Forceful advancement of the needle or injection of anesthetic against resistance is contraindicated due to the potential for unnecessary neural or vascular damage. The greater palatine artery travels adjacent to the greater palatine nerve in the greater palatine canal. Therefore, the

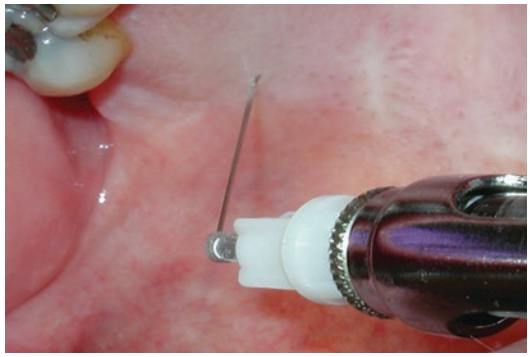

Fig. 18.7 Greater palatine foramen approach to maxillary nerve block

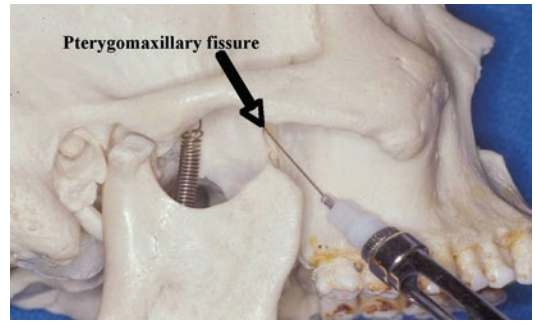

Fig. 18.8 Pterygomaxillary fissure approach to the sphenopalatine fossa for maxillary nerve block

risk of hematoma formation is present though very small. Any hematoma formation would be limited by the tightly restricted space of the greater palatine canal. One study reports that the risk of this event or other complications was very small including only one incidence of positive aspiration of blood during the injection [15]. Interestingly, otolaryngologists may utilize anesthesia in the greater palatine canal for the treatment of epistaxis with efficacy for reduction in bleeding with no associated serious complications [16]. The extraoral approach as described below can be utilized; however, the risk for complications is much greater for hematoma formation (maxillary artery) among other much more serious complications such as brainstem anesthesia and respiratory arrest (Fig. 18.7) [17].

Extraoral Approach Through the Pterygomaxillary Fissure

This approach utilizes the external lateral face in the region just below the zygomatic process of the maxilla in the region of the superficial masseter muscle as landmarks for initial needle placement. The patient then opens the mouth wide, and the needle is inserted superior or just anterior to the coronoid process into the infratemporal fossa. The pterygomaxillary fissure lies deep, and at least a 27-gauge-long needle would need to be utilized. The posterior border of the maxilla would be tracked distally until the needle inserts just anterior to the lateral pterygoid plate of the sphenoid bone. The needle would be advanced almost to its full length. Stojcev reports that an angle of 60° to the sagittal plane and 10° to the horizontal plane help provide access to the pterygomaxillary fissure. The study also reports a success rate of 75% utilizing this approach [18]. Approximately 1.8 mL of anesthetic would be deposited in the region following careful aspiration. The risks of intravascular injection of anesthetic or trauma to vascular structures are much greater that attend this injection as compared to the greater palatine approach. The vascularity of the posterior superior alveolar region has been discussed previously. Inside the pterygomaxillary fissure is the very large maxillary artery, which is a terminal branch of the external carotid and provides a large exposure as it moves in a convoluted loop through this region. The risks of hematoma and other vascular adverse events need to be assessed for this approach (Fig. 18.8).

Sphenopalatine Foramen Approach

There are different approaches to placement of local anesthetic in the pterygopalatine fossa. The specific approach may be influenced by the desired outcome: anesthesia required for dental treatment, differential diagnosis of orofacial pain conditions, or management of orofacial pain conditions. Importantly, the pterygopalatine ganglion is associated with the maxillary nerve and the

transnasal approach attempts to anesthetize the ganglion in order to affect the parasympathetic afferents which synapse in the ganglion. The target is the sphenopalatine foramen in the nasal cavity posterior to the middle turbinate and the pterygopalatine ganglion, which lies lateral to the foramen. The nares are prepared first with 2% viscous lidocaine which provides both lubrication and topical anesthetic. Cotton-tipped applicators with appropriate anesthetic (lidocaine, tetracaine, cocaine 10%) are inserted through the nose to the desired location and left for 20–30 min. The patient may experience lacrimation, light-headedness along with a bitter taste, and numbness in the back of the throat [19]. If cocaine is utilized, then cardiac monitoring and pulse oximetry are utilized for possible cardiac adverse effects.

Anesthetic Block Through Nares

An alternative to the application of cotton-tipped applicators is the utilization of lidocaine transnasal spray. While this approach may not achieve effective blockade to the entire maxillary division, it may serve to provide invaluable diagnostic and/or therapeutic effects. Concentrations of 4–8% lidocaine are sprayed unilaterally or bilaterally (two sprays per side) utilizing a metered spray bottle into the nares [20]. The advantage to this technique is the ease of application and the rapidity of efficacy of the anesthetic (in certain cases within minutes). This technique has been utilized for V_2 pain associated with trigeminal neuralgia [21, 22] and neurovascular pain associated with different headache entities. The benefit of transnasal lidocaine for the management of migraine headaches has been suggested. One study shows the likely decrease of parasympathetic outflow through the pterygopalatine ganglion with application of lidocaine [23]. Interestingly, there was significant pain relief but no effect on the peripheral allodynia due to central nervous system involvement. A case report demonstrated the ability of intranasal lidocaine 4% for the prevention of migraine following aura [24]. The benefits of the lidocaine occur primarily in the nasal cavity and pterygopalatine fossa with little systemic uptake as documented by Kanai (only 0.6 μg per mL^{-1} maximum plasma concentration). Studies report no significant adverse effects with only a small amount of burning associated with the lidocaine and the bitter taste from postnasal drip.

A combination of 3% tetracaine and 0.05% oxymetazoline has been FDA approved (Kovanaze, St. Reatus) for anesthesia of the anterior superior and middle superior alveolar nerves through an intranasal spray. The success rate of maxillary pulpal anesthesia of the incisors, canines, and premolars is 88% with reduced success on the second premolars [25]. This may be due to an absent superior alveolar nerve and subsequent innervation of the second premolars by the posterior superior alveolar nerve branch.

Nasopalatine Block: Incisive Canal

The nasopalatine block effectively provides anesthesia to the anterior portion of the hard palate with its associated mucosa from the first premolar to the incisive papilla bilaterally. The target zone for placement of anesthetic solution is the incisive papilla and the incisive canal above it in which the nasal palatine nerve enters into the palate. The needle is placed into the incisive papilla and directed superiorly several millimeters. Approximately 0.5 mL of anesthetic is deposited into the canal. The injection is painful and tactics such as providing pressure anesthesia as previously mentioned can be helpful. McArdle presents a technique for the deposition of a small amount of anesthetic in the facial gingival, which is less painful as compared to the palatal tissue. Then sequential small increments of anesthesia can be directed through the interdental papilla into the palatal tissues. Once the surface is anesthetized, then an injection into the incisive papilla is much less uncomfortable [26]. There are few reported adverse effects to this block, but patients will remember for a long time if it is very painful. Occasionally, the incisive papilla can be sore and slightly swollen as sequelae, which can be a problem if the patient's lower incisors occlude on the papilla (Fig. 18.9).

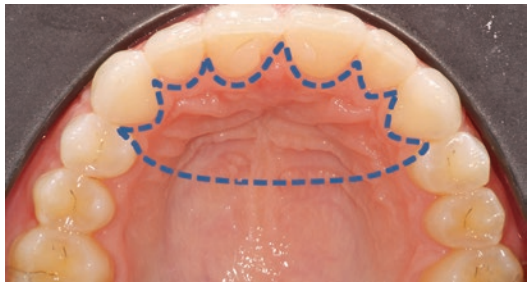

Fig. 18.9 Area anesthetized utilizing the nasopalatine nerve block

Mandibular Anesthetic Blockade

The mandibular nerve (third division of the trigeminal nerve) is a mixed nerve with two roots: a large sensory and a smaller motor root. Once branching off of the trigeminal ganglion, it reaches the infratemporal fossa via the foramen ovale. Branches of the mandibular nerve supply sensory innervation to the hard (teeth and bone) and soft (mucosa, gingiva, and tongue) tissues of the lower jaw and floor of the mouth. Motor branches to the muscles of mastication, the tensor tympani, and the tensor veli palatini muscles leave the trunk in the infratemporal fossa. The nerve gives off numerous sensory branches.

Supraperiosteal anesthesia or infiltration is less effective for anesthesia of permanent mandibular teeth due to the dense cortical bone. Mandibular infiltration is more effective in the pediatric population but is inadequate for pulp therapy and extractions [27]. Mandibular infiltration for adults can provide anesthesia of varying degrees and some argue that the use of articaine can enhance efficacy. There is a benefit to use of supplemental infiltration of articaine, after successful mandibular block anesthesia, in achieving profound pulpal anesthesia [28].

Inferior Alveolar Block

The region anesthetized includes all mandibular teeth to the midline, body of the mandible, inferior portion of the ramus, buccal mucoperiosteum and mucus membranes anterior to the first molar, anterior two-third of the tongue and floor of the mouth, and lingual mucus membranes with the associated periosteum to the midline. The target of this anesthetic block is the inferior alveolar nerve before it enters into the mandibular foramen and travels inside the body of the mandible inside the mandibular canal. The access to the inferior alveolar nerve is limited by the sphenomandibular ligament that attaches to the lingual and the medial aspect of the ramus of the mandible. To correctly navigate this small region, there are several external landmarks that will guide the needle into the correct location. The three-dimensional direction of the needle is determined by the plane of occlusion, the depth of the coronoid notch, and the pterygomandibular raphe. In adults the height of the injection is typically 6–10 mm above the occlusal plane. Typically penetration in 20–25 mm in adults and a long 27-gauge needle should be used. Bone must be contacted at approximately two-thirds to three-fourths of the needle length. The anesthetic should be slowly deposited following careful aspiration. At least 1–1.8 mL of anesthetic is utilized. In children aged seven and younger the height of the injection is at the occlusal plane. The depth of penetration is decreased so a 27-gauge short needle may be used as long as it does not reach the hub. If utilizing mandibular bilateral blocks it is best to prepare the patent that a very broad area of the mandible would be anesthetized. Some patients report perceived or actual difficulty in swallowing or an increase in iatrogenic trauma such as lip biting (Fig. 18.10).

The literature provides more case reports of adverse effects for the inferior alveolar block than any other oral anesthetic block. The most common complication is paresthesia which is almost universally associated with inferior alveolar block. In one study [29], out of 182 reports of paresthesia, all but 2 were associated with the inferior alveolar block. Of those, the lingual nerve was implicated at least twice as often as the inferior alveolar nerve. Following are complications for the inferior alveolar block.

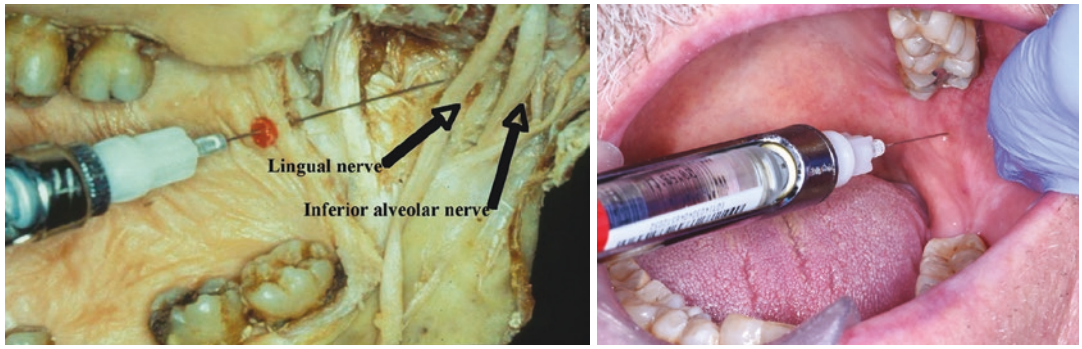

Fig. 18.10 Needle placement for inferior alveolar and lingual nerve blocks

Electric Shock

Electric shocks are most often experienced by direct contact with the lingual nerve during the injection because it is so close to the surface of the mucosa. The inferior alveolar nerve will respond in the same way, but it is much better protected. It has been shown that there is no correlation with the patient experiencing an electrical shock and neurologic damage to either nerve [30].

Injection Injuries to the Lingual and Inferior Alveolar Nerve

One study [22] reports that 42 of 54 injuries occurred to the lingual nerve and 12 of 54 to the inferior alveolar nerve. The lingual nerve injuries are much more incapacitating than the inferior alveolar nerves resulting in sensory loss to both feeling and taste (damage to the chorda tympani nerve as well). There is no clarity on whether these injuries will spontaneously improve. Krafft and Hickel report a high spontaneous resolution of 17 of 18 in 6 months. Hillerup reports the opposite. The etiology of the reason behind the injury is unclear. There are several rationales proposed:

Direct trauma: This was the earliest proposed rationale. There is no strong evidence to support this. There is no evidence for the electric shock—direct trauma with correlation of paresthesias.

Neurotoxicity: There is mounting evidence that this may be the most likely causative factor for nerve injury in respect to local anesthesia. Most of the injuries are associated with the local anesthetics with 4% concentration. Hillerup reports that 54% of nerve injuries were associated with articaine 4% following its utilization in Denmark [22]. In a 21-year retrospective study, Haas [31] reports on Canadian reports of paresthesias. In 1993, there were 14 cases of paresthesia of which articaine 4% was apparently implicated in 10 of 14, and prilocaine 4% was implicated in the other 4 out of 14 cases.

Neural ischemia associated with neural toxicity: Perineural and endoneurial fibrotic changes are likely associated with high concentration of local anesthetic. This can likely result in endoneurial edema and injury to the nerve [32].

Intravascular injections: The medial aspect of the ramus in the region is heavily vascularized with the inferior alveolar artery and other sources. An intra-arterial injection of local anesthetic with epinephrine concentrates the anesthetic in the peripheral tissues. This may be demonstrated by blanching of facial skin, intraoral regions, and even eye symptoms. These sequelae normally fade away within 60 min or less [33].

Diplopia: This transient response is also seen with inferior alveolar blocks as well as posterior superior alveolar blocks [34].

Broken needles: Although very rare, broken needles occur in the anesthetic block for the inferior alveolar nerve more than any other. In one study, 16 of 17 reports of broken needles occurred

in this region [3]. Needle breakage is associated with the use of 30-gauge short needles and in children who are reported to have moved violently [3]. To prevent this complication needles should be of adequate length to avoid penetration to the hub, 25- or 27-gauge needles should be used, needles should not be bent, and adequate head stabilization is required for young children or phobic patients who may move during local anesthetic administration [35].

Postoperative Iatrogenic Trauma

This typically involves lip and cheek biting although tongue biting and scratching can occur as well. The complication primarily affects young children or patients with special health care needs. All patients and/or guardians should be warned about the potential for postoperative trauma. A warning sticker reminder is helpful for parents and some dentists use a cotton roll with a floss ligature as a reminder.

Of all the anesthetic blocks, the inferior alveolar block has the most reports of inefficacy. Conservatively, inferior alveolar blocks fail at least 15% of the time [36]. Another study reports that 87% of subjects with an inferior alveolar block reported numb lips after 5 min [37]. Endodontic literature reports that pulpal anesthesia with teeth with irreversible pulpitis resulting from inferior alveolar blockade demonstrates much less than expected efficacy (36%) [38].

Why do inferior alveolar blocks demonstrate more unpredictability than other blocks? The reason likely involves the complexity of the anatomy and the difficulty in access. There are several common errors that can contribute to less desirable predictability.

Injection Error: An Approach That Is Too Straight Rather Than from the Contralateral Premolar Region

If the approach is from the region of the central incisors, the needle often contacts the ascending ramus of the mandible, thereby stopping the injection before there is any access to the region of the lingula and the mandibular foramen. If the injection is more medial, the needle will be on the medial aspect of the sphenomandibular ligament which prevents infiltration of the anesthetic from reaching the inferior alveolar nerve. This may result in the deposition of anesthetic in the tail of the parotid gland that wraps around the distal aspect of the ramus often with the resultant taste to the patient and even some anesthesia of the facial nerve. The correction involves correction of the approach by directing the needle from the opposite premolar region so that the needle can reach the desired location.

Injection Error: An Approach That Is Too Inferior

The needle needs to be directed ideally at a bisection of the depth of the coronoid notch and at the same inclination as the occlusal plane. If the needle is below this plane, the anesthetic will be deposited in a region that is below the mandibular foramen and below the sphenomandibular ligament without access to the inferior alveolar nerve. The correction involves raising of the needle to the correct plane.

Accessory Innervations to the Mandibular Molar Region

Other accessory innervations to the mandibular molar region have been documented including branches from the cervical plexus from C_2 and C_3, branches from the motor nerve to the mylohyoid muscle [39], and even additional branches from the inferior alveolar nerve entering the mandible [40]. The solution to possible accessory innervations to mandibular posterior teeth or even anterior teeth would be additional anesthesia likely with a buccal approach or even a lingual approach. Infiltrations in the proper depth and location will anesthetize addition innervations.

Buccal Block

The buccal block anesthetizes the region of the mucous membranes including gingiva and periosteum to the buccal of the mandibular molars. This injection is used adjunctively with the

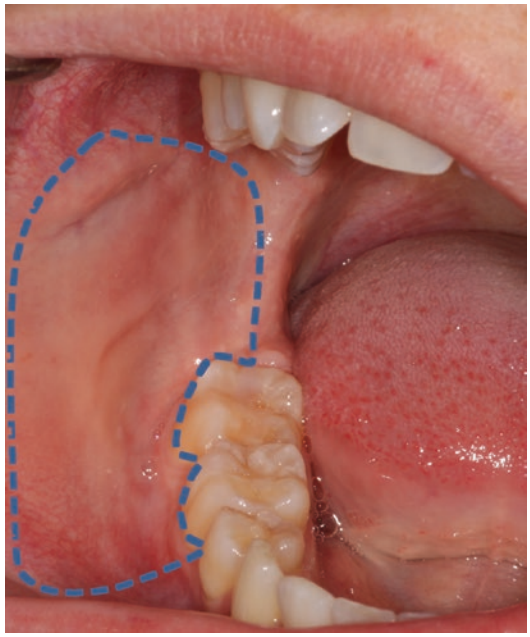

Fig. 18.11 Area anesthetized utilizing a long buccal nerve block

inferior alveolar block, which will not anesthetize the buccal tissues. The target is the buccal mucosa lateral to the maxillary second molar and lateral to the mandibular coronoid process. Approximately 0.5 mL of anesthetic is deposited under the mucosa. There are few reported adverse effects for this injection (Fig. 18.11).

Mental Block

The mental block anesthetizes the buccal mucous membranes anterior to the mental foramen to the midline. This usually involves the gingiva, the buccal mucosa adjacent to the mandibular first molar to the lower lip, but may also include either the first or the second premolar if sufficient anesthetic enters the mental foramen. The target is the mental nerve as it leaves its respective foramen. The mental foramen is most likely present on the buccal osseous surface of the mandible apical to the second premolar or between the apices of the first and second premolars. It may vary in position from the canine all the way to the mandibular first molar [41]. The needle is inserted in a

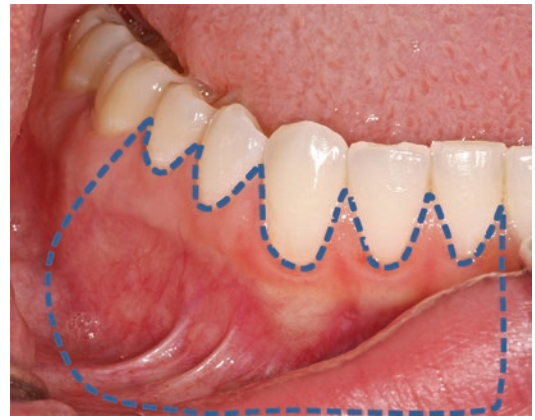

Fig. 18.12 Area anesthetized utilizing a mental nerve block

posterior and inferior direction at the base of the mandibular vestibule and moved into position to the region of the periosteum over the foramen. Approximately 0.5 mL of anesthetic is deposited. Very few adverse effects are reported in the literature. There are suggestions for utilizing mental blocks as an alternative to the inferior alveolar block if the area of concern is anterior to the mental foramen since this block is much more readily accessible (Fig. 18.12) [42].

Incisive Block

The incisive block anesthetizes the mucous membranes including the gingiva and buccal mucosa of the vestibule and lip. The pulps of the premolars, canines, and incisors may also become anesthetized. The target is the buccal mucosa in the bottom of the vestibule in the apical region of the teeth including the mental foramen adjacent to the mandibular premolars or soft tissue that requires anesthetic. The needle is inserted and approximately 0.5 mL of anesthetic is deposited. There are few recorded adverse reactions to this injection. It has been reported that articaine 4% with epinephrine anesthetic provides superior anesthesia as compared to lidocaine 2% with epinephrine both in regard to duration and depth of anesthesia for the mental and incisive blocks (Fig. 18.13) [42].

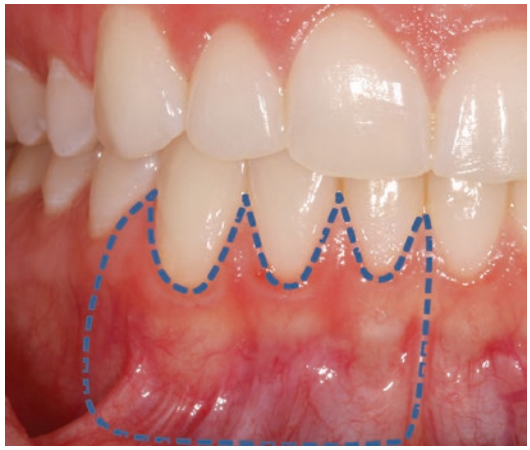

Fig. 18.13 Area anesthetized utilizing an incisive nerve block

Gow-Gates Mandibular Block

The area anesthetized by the Gow-Gates block is the entire region of the mandibular branch of the trigeminal nerve. It is essentially a V_3 division block, which includes the body and inferior portion of the mandible, teeth, buccal mucosa, anterior two-third lingual mucosa and tongue, and preauricular region of the face associated with the auriculotemporal nerve. This block is utilized following failure of an inferior alveolar nerve block but has many applications of its own for both diagnostic anesthesia and dental anesthesia. The target for the needle is in a plane that is much higher in a superior direction to the occlusal plane and that of the inferior alveolar block. The region of injection is the anteromedial aspect of the condyle. The mouth must be opened fully with the needle inserted but below the second molar with the approach from the opposite mandibular premolar region. The needle is inserted 25–28 mm [43], with check for aspiration, and 1.8 mL of anesthetic deposited.

There are a number of benefits for utilizing the Gow-Gates block as compared to the inferior alveolar block. The risk of positive aspiration or intravascular injection is greatly diminished. This is related to the decrease in vascularity of the region as compared to the medial aspect of the mandible. It is reported that once mastered, the Gow-Gates block has a risk of approximately 1.6% for aspiration of blood as compared to a range of 3.6–22% for the inferior alveolar block [43]. Gow-Gates (the author) reports a more predictable anesthetic technique as well with a success rate of 98–100% as compared to 83.9–85.4% for the inferior alveolar block. Other reports vary and suggest that the efficacy of a Gow-Gates block is comparable to an inferior alveolar block for extractions and pulp testing [44]. So it seems likely that adverse effects in relationship to vascular compromise are lessened and possibly risks of paresthesia as well. There are few, if any, reports of paresthesia related to Gow-Gates blocks. The few reports of adverse effects are related to temporary vision defects [45].

Auriculotemporal Block

The utility for use of an auriculotemporal nerve block is diagnostic and procedure related. The block can serve as an aid in determining the degree of involvement of the temporomandibular joint (TMJ) in cases of orofacial pain. Since there is a significant convergence from the masseter muscle region and the TMJ, it not surprising that it is difficult to readily determine where the pain is coming from. The auriculotemporal block anesthetizes the auriculotemporal nerve, a branch of the mandibular nerve, which provides sensory innervation to the vast majority of the TMJ, the preauricular region, and the portion of the scalp superior to the helix of the ear. The target area for placement of the 27-gauge-short needle is a small concavity anterior to the base of the tragus as the mandible opens. The needle is angled at a 20° anterior angle matching the external ear canal to prevent inadvertent deposition of the anesthetic in that area. The needle is advanced until in some cases it gently contacts the posterior aspect of the mandibular ramus/condylar neck [46]. If this occurs, the patient is asked to open the mouth, thus allowing the needle to pass just behind the ramus to the medial side. Then, approximately 0.5–0.8 mL of anesthetic is deposited following aspiration (Fig. 18.14).

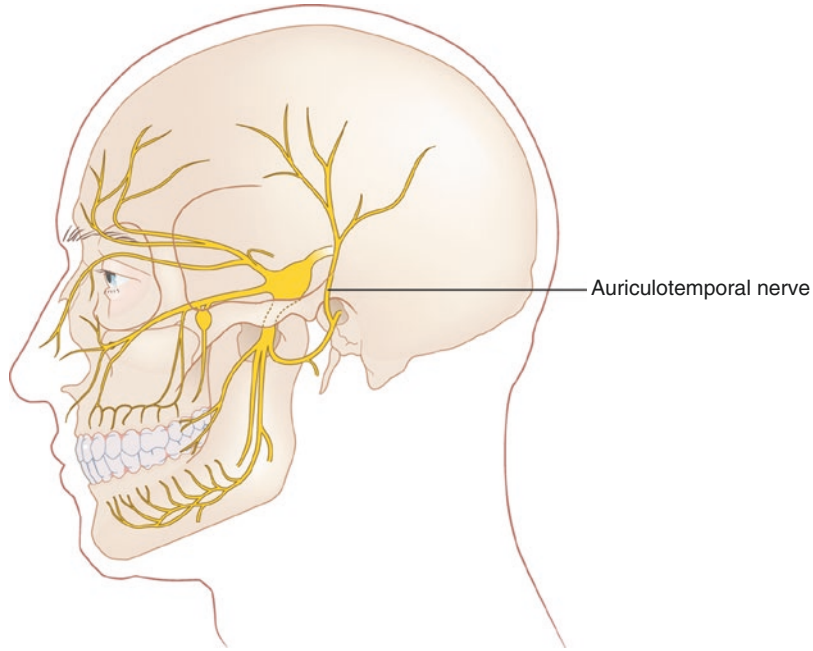

Fig. 18.14 Auriculotemporal nerve, adapted from *Atlas of Human Anatomy* 3rd edition by Frank H. Netter, Icon Learning Systems Teterboro, New Jersey 2003

There are few reported risks associated with the auriculotemporal block. However, patient education prior to the block is important since the normal short-lived-associated factors induce unnecessary anxiety. Since the facial nerve is in close proximity to the injection target zone, it will be likely affected for the duration of the anesthetic resulting in temporary paresis of the muscles of facial expression subserved by the temporal, zygomatic, and buccal branches. Should the patient be unable to close their eyelid, this can be accomplished manually. A moistened 2 × 2 gauze can hold the lid closed.

Trigger-Point Injections

Local anesthetic is utilized in the diagnosis and treatment of myofascial pain in the head and neck region. The focal point of this injection is the "trigger point" identified within the painful muscle. This region of taut band is identified through musculoskeletal examination. The trigger point presents as a hard knot-like area in a muscle that when palpated duplicates the patients' chief pain concern and is associated with pain not only in the local region but also at a distant site known as the "zone of reference." Typically, a 27-gauge-short needle is utilized. The muscle must be stabilized so that the trigger point does not move out of position as the needle is inserted through the skin or intraoral mucosa. As the trigger point is reached, the patient often will describe pain that duplicates their chief concern and the muscle itself may exhibit a "twitch" response. Approximately 0.3–0.5 mL of local anesthetic without vasoconstrictor is deposited. The needle is then redirected in the region of anesthetized tissue, and adjacent muscle fibers are also anesthetized. Following the injection, pressure is applied for 5 min in order to control bleeding. Cross-frictional massage followed by gentle stretching with ice massage is recommended following injection so as to soften the taut band in the muscle. The patient is given postoperative instructions to limit vigorous utilization of the injected muscle(s). It must be strongly emphasized that trigger-point injections are part of an overall comprehensive approach to management of

myofascial pain. For the masticatory system, this multidisciplinary approach always involves patient education, patient involvement with stretching and self-behavior modification techniques, regimens that incorporate improvements related to sleep hygiene, and often utilization of nutritional supplementation. Other aspects may include physical therapy, clinical psychology (habit modification/relaxation training), and pharmacotherapy.

Case Study #1

A 37-year-old Caucasian male who was a military veteran presented to a university orofacial pain center with a chief concern of bilateral pain in the preauricular, cervical, and temporal regions. He also reported a history of migraine headaches. The patient noted that the most significant pain was located in the right preauricular region. His medical history was significant for anxiety, depression, hypercholesterolemia, and insomnia. The patient's medical conditions were managed by the Veterans Administration physicians. The patient was currently taking the following medications:

Simvastatin 20 mg, tizanidine 20 mg divided in three doses per day, divalproex 500 mg per day, venlafaxine 100 mg per day, zolmitriptan 5 mg prn pain, indomethacin 50 mg prn pain.

The patient described the right jaw pain as throbbing pain "like a muscle" with radiation to the ear and temporal region. The pain once it began could last for weeks at a time and was exacerbated by gritting his teeth, opening wide, and chewing hard foods. He reported only one modality, a temporary oral appliance, that had provided some pain relief. The migraine headaches were associated with nausea, photophobia, phonophobia, and osmophobia. They were exacerbated by his jaw pain resulting in an increase in frequency.

The clinical examination revealed soreness in the right temporomandibular joint associated with clicking, and postural compromise with head forward position. He was aware of masticatory parafunction (clenching his teeth).

Palpation of the masticatory and cervical muscles that radiated pain to distant sights duplicating his chief concern was:

- Right superficial masseter that radiated pain to the eyes, ears, and temporal region
- Right posterior temporalis muscle that radiated pain to the frontal region
- Right sternocleidomastoid insertion and midbody that radiated pain to the apex of the head
- Bilateral trapezius muscle: insertion at the superior nuchal line, paraspinal and anterior trapezius that radiated pain in a superior direction and duplicated his headache pain

Panoramic and lateral cephalometric images were obtained which demonstrated cervical degenerative joint disease, grossly normal temporomandibular joints, and maxillary sinuses (Fig. 18.15).

Fig. 18.15 Periapical image of tooth #7 with an endodontic procedure. Note that no periapical or coronal disease processes are evident

Diagnoses determined included myofascial pain with referral, migraine headaches, temporomandibular joint articular disc displacement with reduction, insomnia associated with sleep bruxism, postural compromise, and psychological factors that affected the medical diagnosis.

The management of the patient's condition began with education about myofascial pain, self-physical therapy such as stretching, and moist heat. Iontophoresis that utilized bupivacaine 0.5% and dexamethasone 4 mg/mL was implemented for the right masseter muscle. The patient was reappointed for delivery of an oral appliance. When the patient returned he reported a new problem. Tooth #7, the right maxillary incisor, had become painful and had been recently treated with an endodontic procedure. However the pain was unchanged. Upon examination of tooth #7, no pathology was identified. It was determined that the pain was not of odontogenic origin. Further assessment via muscle palpation provoked a referral pattern not seen in the initial examination resulting in a duplication of the pain in tooth #7 which he rated as 6/10.

A trigger-point injection utilizing 1 cm^3 of 3% mepivacaine, no vasoconstrictor, was directed at the right superficial masseter (Fig. 18.16). Following the injection, the muscle could now be palpated without triggering the pain. The patient noted that the pain in tooth #7 was no longer present confirming a diagnosis of myofascial pain with referral. As the management of the patient's myofascial pain proceeded, the patient's overall pain levels were reduced significantly and tooth #7 never became symptomatic again. However it must be kept in mind that myofascial pain is known to be cyclical in nature and may be associated with numerous contributing factors which must be addressed in the overall care for the patient. Therefore, trigger-point injections may serve as a component of an overall management plan.

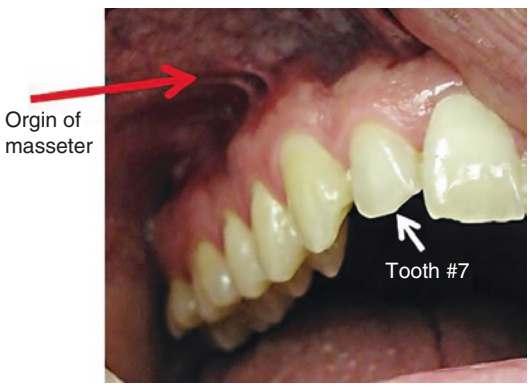

Fig. 18.16 Origin of the right superficial masseter with the arrow pointing to the location of the trigger-point injection

Review Questions/Discussion

1. How did the trigger-point injection help identify a referral pattern from another structure to tooth #7?
2. Why was it important to document the visual analogue scale both before and after diagnostic injections?
3. How did trigger-point injections act as a part of the overall pain management strategy?
4. At what stage of treatment were the trigger-point injections implemented?

Case Study #2

A 53-year-old Caucasian female presented to a university orofacial pain center with a significant history of right temporomandibular pain and dysfunction. She had been diagnosed with right-sided temporomandibular joint articular disc displacement with reduction and pain. Nonsurgical management of her condition was not effective. Surgical intervention was accomplished via an open temporomandibular joint procedure involving articular disc repositioning and placation. Following surgery the patient experienced improved function and elimination of pain.

Four years later this patient was injured in a motor vehicle accident in which she was rear-ended. Following the accident, she experienced temporomandibular joint sounds and severe pain in the same right preauricular region. She was examined by an oral surgeon and it was determined that a second temporomandibular joint

surgery was indicated. She decided to have a second opinion at a university orofacial pain center.

Upon presentation, she reported a constant soreness in the right preauricular region that varied in intensity throughout the day. She noted that the pain was present upon awakening and was exacerbated with masticatory function. The patient reported that the pain during the day ranged from 4/10 to 7/10. Her medical history was noncontributory with exception of the temporomandibular surgery. She did report that the utilization of ibuprofen provided modest benefit.

The examination revealed a normal range of motion with moderate crepitus in the right temporomandibular joint. The joint was also mildly tender to palpation. The examination revealed severe pain in the insertion (attachment) of the right anterior temporalis muscle as it enveloped the coronoid process which duplicated the patient's chief concern. The superficial masseter and medial pterygoid muscle were also palpated at moderate to severe level. The trapezius muscle and sternocleidomastoid muscle were also painful.

In order to help to determine the source of the pain diagnostic anesthesia was utilized. She reported her pain level to be 7/10 on the visual analogue scale. The skin on her face was prepped and an auriculotemporal block was administered with 1 cm^3 of 3% mepivacaine without vasoconstrictor. After waiting 15 min her pain level dropped to 6/10 with little reported pain relief. It was determined that since the attachment of the right anterior temporalis muscle duplicated the pain, a diagnostic trigger-point injection would be utilized in this region. One cubic centimeter of mepivacaine 3% was administered in the region high on the coronoid process: lateral, medial, and anterior. The pain level dropped from 6/10 after 15 min to 0/10. All pain in the region of the chief concern was gone. It was determined that the pain of the chief concern was associated with the insertion of the anterior temporalis and not the temporomandibular joint.

The patient's diagnosis responsible for her pain was myofascial pain associated with the insertion of the anterior temporalis muscle. She also was diagnosed with myofascial pain with spreading which affected other muscles of the masticatory and cervical regions. The temporomandibular joint did demonstrate slight soreness likely associated with mild inflammation but demonstrated no structural damage due to trauma.

At the same appointment 1 cm^3 of 4 mg/mL of dexamethasone was injected into the now-determined source of the pain since the attachment is very tendonous and the likely etiology was of inflammatory origin. The patient was already wearing an oral appliance at night and was actively engaged in a gentle stretching protocol for her masticatory muscles. The patient was referred to physical therapy for her cervical muscle concerns. The result was control of her pain over the next 2 years which was 0/10 on most days. She required no surgery since the pain was not a result of traumatic reinjury to the temporomandibular joint.

Review Questions/Discussion

1. How was diagnostic anesthesia utilized in the examination process to either confirm or rule out a temporomandibular joint diagnosis as the cause of the patient's pain?
2. The diagnostic anesthetic injections proved beneficial in the examination of the temporomandibular joint that was a possible source of the pain associated with the motor vehicle accident. How did it help both the doctor and the patient to understand which anatomical structures were likely the source of the patient's chief concern?
3. What was the most significant benefit of the auriculotemporal diagnostic block used during the examination? What did it likely prevent?
4. Why was the utilization of a second diagnostic injection, which was the trigger-point injection into the insertion of the right anterior temporalis, important?

Conclusion

The use of local anesthetics is ubiquitous in health care. There are adverse reactions, but they are "extraordinarily negligible" [47].

Fig. 18.17 Masseter muscle trigger-point injection. Note that the operator has isolated the taut band in order to stabilize the target zone within the muscle

However, efficacy and comfort are predicated on a detailed knowledge of anatomy, physiology, neural pathways, and pharmacodynamics and pharmacokinetics of the anesthetic solutions utilized (Fig. 18.17).

References

1. Harn SD, Durham TM, Callahan BP, Kent DK. The triangle of safety: a modified posterior superior alveolar injection technique based on the anatomy of the PSA artery. Gen Dent. 2002;50(6):554–7.
2. Goldenberg AS. Transient diplopia as a result of block injections. Mandibular and posterior superior alveolar. NY state. Dent. 1997;63(5):29–31.
3. Pogrel MA. Broken local anesthetic needles: a case series of 16 patients with recommendations. J Am Dent Assoc. 2009;140(12):1517–22.
4. Hwang K, Suh MS, Chung IH. Technical strategies, cutaneous distribution of the infraorbital nerve. J Craniofac Surg. 2004;15(1):1–5.
5. Berberich G, Reader A, Drum M, Nusstein J, Beck M. A prospective, randomized, double blind comparison of the anesthetic efficacy of two percent lidocaine with 1:100,000 and 1:50,000 epinephrine and three percent mepivicaine in the intraoral, infraorbital nerve block. J Endod. 2009;35(11):1498–504.
6. Karkut B, Reader A, Drum M, Nusstein J, Beck M. A comparison of the local anesthetic efficacy of the extraoral versus the intraoral infraorbital block. J Am Dent Assoc. 2010;141(2):185–92.
7. Jones JE, Dean JA. Local anesthesia and pain control for the child and adolescent. In: Dean JA, editor. McDonald and Avery's dentistry for the child and adolescent. 10th ed. St. Louis, MI: Elsevier; 2016. p. 274–85.
8. Douglas R, Wormald PJ. Pterygopalatine fossa infiltration through the greater palatine foramen: where to bend the needle. Laryngoscrope. 2006;116(7):1255–7.
9. Bhalla J, Meechan JG, Lawrence HP, Grad HA, Haas DA. Effect of time on clinical efficacy of topical anesthesia. Anesth Prog. 2009;56(2):36–41.
10. Harbert H. Topical ice: a precursor to palatal injections. J Endod. 1989;15(1):27–8.
11. Kudo M. Initial injection pressure for dental local anesthesia: affects on pain and anxiety. Anesthe Prog. 2005;52(3):95–101.
12. Mittal M, Kumar A, Srivastava D, Sharma P, Sharma S. Pain perception: computerized versus traditional local anesthesia in pediatric patients. J Clin Pediatr Dent. 2015;39(5):470–4.
13. Nusstein J, Lee S, Reader A, Beck M, Weaver J. Injection pain and postinjection pain of the anterior middle superior alveolar injection administered with the Wand or conventional syringe. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2004;98(1):124–31.
14. Nish IA, Pynn BR, Holmes HI, Young ER. Maxillary nerve block: a case report and review of the intraoral technique. J Can Dent Assoc. 1995;61(4):305–10.
15. Schwartz-Arad D, Dolev E, Williams W. Maxillary nerve block—a new approach using computer-controlled anesthetic delivery system for maxillary sinus elevation procedure. A prospective study. Quintessence Int. 2004;35(6):477–80.
16. Bharadwaj VK, Novotny GM. Greater palatine canal injection: an alternative to the posterior nasal packing and arterial ligation in epistaxis. J Otolaryngol. 1986;15(2):94–100.
17. Nique TA, Bennett CR. Inadvertent brainstem anesthesia following extraoral trigeminal V2–V3 blocks. Oral Surg Oral Med Oral Pathol. 1981;51(5):468–70.
18. Stojcev Stajcic L, Gacic B, Popovic N, Stajcic Z. Anatomical study of the pterygopalatine fossa pertinent to the maxillary nerve block at the foramen rotundum. Int J Oral Maxillofac Surg. 2010;39(5):493–6.
19. Windsor RE, Jahnke S. Sphenopalatine ganglion blockade: a review and proposed modification of the transnasal technique. Pain Physician. 2004;7(2):283–6.
20. Kanai A, Suzuki A, Kobayashi M, Hoka S. Intranasal lidocaine 8% spray for second-division trigeminal neuralgia. Br J Anaesth. 2006;97(4):559–63.
21. Obah C, Fine PG. Intranasal sphenopalatine ganglion block: minimally invasive pharmacotherapy for refractory facial and headache pain. J Pain Palliat Care Pharmacother. 2006;20(3):57–9.
22. Hillerup S, Jensen R. Nerve injury caused by mandibular block analgesia. Int J Oral Maxillofac Surg. 2006;35(5):437–43.
23. Yarnitsky K, Goor-Aryeh I, Bajwa ZH, Ransil BI, Cutrer FM, Sottile A, et al. 2003 Wolff Award: possible parasympathetic contributions to peripheral and central sensitization during migraine. Headache. 2004;44(5):447.

24. Maizels M. Intranasal lidocaine to prevent headache following migraine aura. Headache. 1999;39(6):439–42.
25. Hersh EV, Pinto A, Saraghi M, Saleh N, Pulaski L, Gordon SM, et al. Double-masked, randomized, placebo-controlled study to evaluate the efficacy and tolerability of intranasal K305 (3% tetracaine plus 0.05% oxymetazoline) in anesthetizing maxillary teeth. J Am Dent Assoc. 2016;147(4):278–87.
26. McArdle BF. Painless palatal anesthesia. J Am Dent Assoc. 1997;128(5):647.
27. Oulis CJ, Vadiakas GP, Vasilopoulou A. The effectiveness of mandibular infiltration compared to mandibular block anesthesia in treating primary molars in children. Pediatr Dent. 1996;18(4):301–5.
28. King J, McDonagh M, Sedgley CM. Does articaine provide an advantage over lidocaine in patients with symptomatic irreversible pulpitis? A systematic review and meta-analysis. J Endod. 2015;41(11):1784–94.
29. Gaffen AS, Haas DA. Retrospective review of voluntary reports of nonsurgical paresthesia in dentistry. J Can Dent Assoc. 2009;75(8):579.
30. Krafft TC, Hickel R. Clinical investigation into the incidence of direct damage to the lingual nerve caused by local anesthesia. J Craniomaxillofac Surg. 1994;22(5):294–6.
31. Haas DA, Lennon D. A 21 year retrospective study of reports of paresthesia following local administration. J Can Dent Assoc. 1995;61(4):319–30.
32. Selander D. Peripheral nerve injury caused by injection needles. Br J Anaesth. 1992;69(5):433–8.
33. Webber B, Orlansky H, Lipton C, Stevens M. Complication of an intra-arterial injection from an inferior alveolar nerve block. J Am Dent Assoc. 2001;132(12):1702–4.
34. Scott JK, Moxham BJ, Downie IP. Upper lip blanching and diplopia associated with local anaesthesia of the inferior alveolar nerve. Br Dent J. 2007;202(1):32–3.
35. Malamed SF, Reed K, Poorsattar S. Needle breakage: incidence and prevention. Dent Clin N Am. 2010;54(4):745–56.
36. Gaum LI, Moon AC. The Art mandibular nerve block: a new approach to accomplishing regional anesthesia. J Can Dent Assoc. 1997;63(6):454–9.
37. Donkor P, Wong J, Punnia-Moorthy A. An evaluation of the closed mouth mandibular block technique. Int J Oral Maxillofac Surg. 1990;19(4):216–9.
38. Aggarwal V, Singla M, Kabi D. Comparative evaluation of anesthetic efficacy of Gow-Gates mandibular conduction anesthesia, Vaziriani-Akinosi technique, buccal-plus-lingual infiltrations, and conventional inferior alveolar nerve anesthesia in patients with irreversible pulpitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2010;109(2):303–8.
39. Bennett S, Townsend G. Distribution of the mylohyoid nerve: anatomical variability and clinical implications. Aust Endod J. 2001;27(3):109–11.
40. Gover PS, Lorton L. Bifid mandibular nerve as a possible cause of inadequate anesthesia in the mandible. J Oral Maxillofac Surg. 1983;41:177.
41. Greenstein G, Tarnow D. The mental foramen and nerve: clinical and anatomical factors related to the dental implant placement: a literature review. J Periodontol. 2006;77(12):1933–43.
42. Batista da Silva C, Berto LA, Volpato MC, Ramacciato JC, Motta RH, Ranali J, et al. Anesthetic efficacy of articaine and lidocaine for incisive/mental nerve block. J Endod. 2010;36(3):438–41.
43. Gow-Gates G, Watson JE. Gow-Gates mandibular block—applied anatomy and histology. Anesth Prog. 1989;36:192–200.
44. Hung PC, Chang HH, Yang PJ, Kuo YS, Lan WH, Lin CP. Comparison of the Gow-Gates mandibular block and inferior alveolar nerve block using a standardized protocol. J Formos Med Assoc. 2006;105(2):139–46.
45. Fish LR, McIntire DN, Johnson L. Temporary paralysis of cranial nerves III, IV, and VI after a Gow-Gates injection. J Am Dent Assoc. 1989;119(1):127–8.
46. Schmidt BL, Progrel MA, Necoechea M, Kaerns G. The distribution of the auriculotemporal nerve around the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;86(2):165–8.
47. Mehra P, Caiazzo A, Maloney P. Lidocaine toxicity. Anesth Prog. 1998;45(1):38–41.

Regional Anesthetic Techniques for Foot Surgery

19

Rick C. Chen and Peter A. Blume

Introduction

Over the last decade, outpatient surgery has consistently gained in popularity by providing a significant reduction in the cost of hospitalization and the patient's length of stay. Foot and ankle surgery procedures are commonly performed in an outpatient setting [1]. Key issues in foot and ankle surgery include rising demand for outpatient procedures, managing postoperative pain and decreasing the use of opiates, and avoiding the side effects of general anesthesia in certain patient populations [2].

Foot and ankle surgeries produce moderate-to-severe postoperative pain that is sometimes difficult to control with oral pain medications alone [3]. Research has shown that regional anesthesia has been used successfully in foot and ankle surgeries to reduce postoperative pain [1], with one study reporting that regional anesthesia reduces perioperative opioid requirements [4]. Another study indicated that monitored intravenous sedation can be safely and effectively carried out together with regional anesthesia in foot surgeries. The article reported high patient satisfaction and reduction in postoperative pain using this combination [5]. Using monitored intravenous sedation instead of general anesthesia significantly reduces side effects, including nausea, vomiting, and throat discomfort. Intravenous sedation also reduces recovery time and avoids unwanted admission to the hospital [6]. The combination of regional anesthesia with monitored intravenous sedation can also be used in American Society of Anesthesiologists (ASA) 3 and 4 patients undergoing lower limb-preservation procedures without increasing their pulmonary or cardiac complications. This finding is significant because historically it has been assumed that ASA 3 and 4 patients needed to be under general anesthesia regardless of the surgical procedure due to the higher rate of complications associated with this patient population [7]. There are also other specific patient populations in which regional anesthesia may be a superior anesthetic technique. Patients with asthma, for example, benefit greatly from regional anesthesia because it avoids airway manipulation [1].

Despite the numerous benefits of regional anesthesia reported in recent studies, there is some anecdotal evidence that performing regional anesthesia increases operating room time and delays turnovers [2]. However, with judicious preoperative timing and planning as well as skillful regional anesthesia administration, delayed turnover can be minimized. This chapter discusses several common regional anesthesia techniques in foot surgeries and offers clinical pearls.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_19

Anesthetic Agents

Lidocaine (Xylocaine), 1 and 2%, and bupivacaine (Marcaine), 0.25 and 0.5%, are commonly used agents in foot and ankle surgery. Lidocaine has a faster onset of action than bupivacaine, but bupivacaine has a longer duration of action than lidocaine [8]. Many surgeons would also use 1:1 ratio mixtures of lidocaine and bupivacaine to take advantage of the faster onset of lidocaine and longer duration of bupivacaine. The maximum safe dosage of lidocaine without epinephrine in a normal, healthy adult is 4.5 mg/kg, which is approximately 300 mg of lidocaine in a 70-kg adult. With the addition of epinephrine, the maximum dosage increases to approximately 500 mg. The maximum safe dosage of bupivacaine without epinephrine is 2.5 mg/kg or approximately 175 mg in a 70-kg adult. With the addition of epinephrine, the maximum dose becomes 300 mg [5]. Epinephrine (1:100,000) is frequently added to lidocaine and Marcaine to utilize its vasoconstriction properties and enhance the effects of the anesthesia. However, it is generally not recommended to use local anesthetics with epinephrine when injecting circumferentially around the ankle joint or toes due to the risk of causing tissue ischemia.

There have been efforts in recent years to develop an anesthetic again which will provide prolonged analgesia without the use of indwelling catheter. Exparel® (Fig. 19.1) was developed by Pacira Pharmaceutical as a postsurgical analgesic agent. It is an extended-release liposome bupivacaine. Exparel uses an innovative delivery system called Depo Foam®, which incases the anesthetic agent in an aqueous chamber without altering its chemical structures. The medication is released over time due to erosion and/or reorganization of the lipid membrane. The result is a reliable release of a low-dose bupivacaine over a period of 2–3 days. This eliminates the need of dose titration and external device to achieve prolonged analgesia at the surgical site. The formulation comes in either 266 mg/20 mL or 133 mg/10 mL single-use vials. It can be injected directly around the surgical site and can be injected undiluted. It can also expand to a total of 300 mL with normal saline (0.9%) or lactate ringers to accommodate large surgical sites. A recent randomized, multicenter study looked at the efficacy of Exparel in patients undergoing total knee arthroplasty. The study showed significant lower use of opioid medication in patients receiving Exparel injection postoperatively compared to the placebo [9]. Exparel has been gaining popularity in foot and ankle surgery with promising anecdotal results. In a prospective, comparative study, Exparel achieved good analgesia and reduction of concurrent narcotic use in patients undergoing forefoot surgery [10]. Further research is under way to examine the efficacy of Exparel in specific foot and ankle procedures.

Fig. 19.1 Vial of Exparel®

Ankle Block

Introduction

This block involves the blocking of five nerves that innervate the entire foot: posterior tibial, sural, superficial peroneal, saphenous, and deep peroneal nerves. These nerves are the terminal branches of principal nerve trunks that innervate the proximal leg. Each nerve needs to be blocked individually in order to achieve complete anesthesia.

Anatomy

Posterior Tibial Nerve

This nerve is one of the two branches of the sciatic nerve. It courses down the posterior leg along with the posterior tibial artery and vein. At the ankle joint, it runs just posterior to the medial malleolus and gives off the medial calcaneal branch before it dives plantar medially toward the sole of the foot and divides into the medial and plantar nerves (Fig. 19.2). This nerve pro-

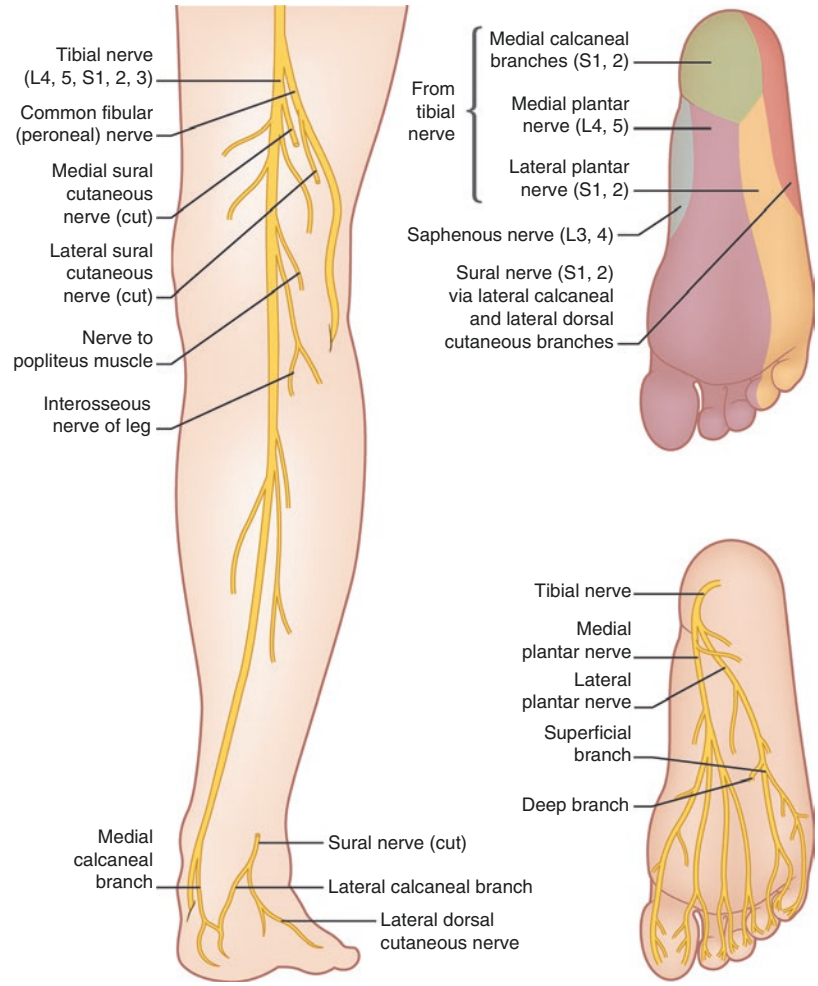

Fig. 19.2 Posterior and plantar sensory nerve distributions and innervations of the sole

vides sensory innervations to the medial heel as well as the entire sole of the foot (see Fig. 19.5).

Sural Nerve

This nerve arises from both the posterior tibial and common peroneal nerves. It courses down the posterior lower leg with the small saphenous vein and gives off the lateral calcaneal branch before it curves posterior and inferior to the lateral malleolus; it then runs along the lateral side of the foot before it becomes one of the digital nerves of the fifth toe (Fig. 19.2, Fig. 19.4). This nerve provides sensory innervations to the area of the lateral malleolus and lateral heel, as well as to the lateral side of the foot (Fig. 19.3).

Superficial Peroneal Nerve

This nerve arises from the common peroneal nerve as it wraps around the fibular head. It courses down the lateral compartment of the lower leg and pierces the deep fascia at the level of the ankle and divides into the medial and intermediate dorsal cutaneous nerves. This nerve provides sensory innervation to the dorsum of the foot (Fig. 19.4).

Saphenous Nerve

This nerve arises from the femoral nerve. It exits the adductor canal and courses down the medial side of the leg along with the great saphenous vein. At the level of the ankle, it runs anterior to the medial mal-

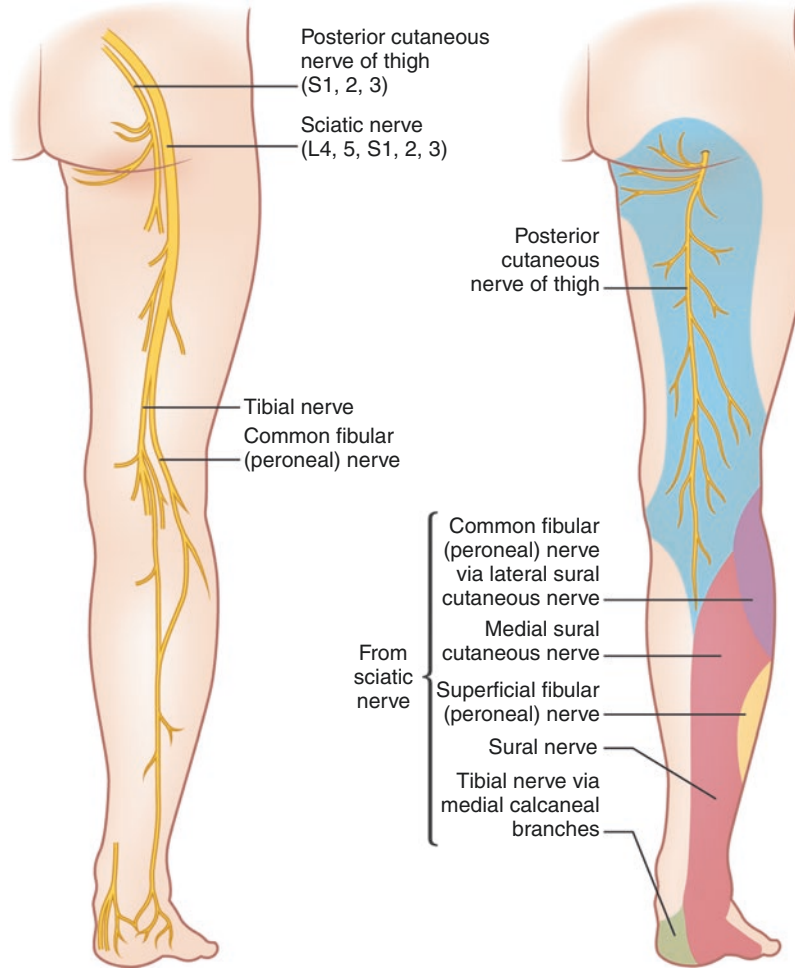

Fig. 19.3 Posterior leg sensory nerve distribution and innervations of heel

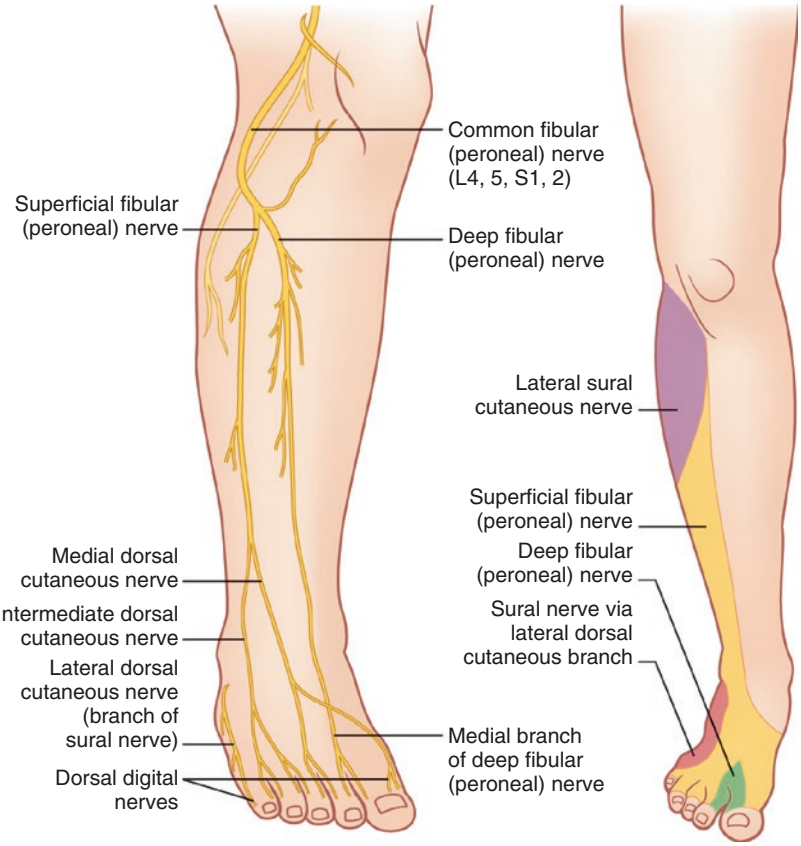

Fig. 19.4 Anterior leg sensory nerve distribution and innervations

leolus and ends at the medial side of the midfoot (Fig. 19.2). This nerve provides the sensory innervation of the medial malleolus (Fig. 19.5).

Deep Peroneal Nerve

This nerve arises from the common peroneal nerve after it wraps around the fibular head. It courses deep down the anterior compartment of the lower leg and dorsal foot. This nerve provides mostly motor innervations of the anterior compartment muscles of the lower leg. The only sensory innervation the deep peroneal nerve provides is at the first interdigital space (Fig. 19.5).

Indications

- All surgical procedures of the foot
- Providing postoperative pain relief in both adults and children
- Supplemental anesthesia to incomplete proximal regional nerve block, including popliteal or sciatic block

Procedure

Posterior Tibial Nerve Block

Positioning: Prone with pillow under the ankle, or supine with the leg externally rotated.

Landmarks:

- Medial malleolus
- Achilles tendon
- Posterior tibial artery

Injection Technique: The point of needle insertion is approximately two fingerbreadths posterior to the medial malleolus and 1–1.5 cm anterior to the Achilles tendon. The pulse of the posterior tibial artery is palpated, and a 25-gauge, 1.5-in.-long nee-

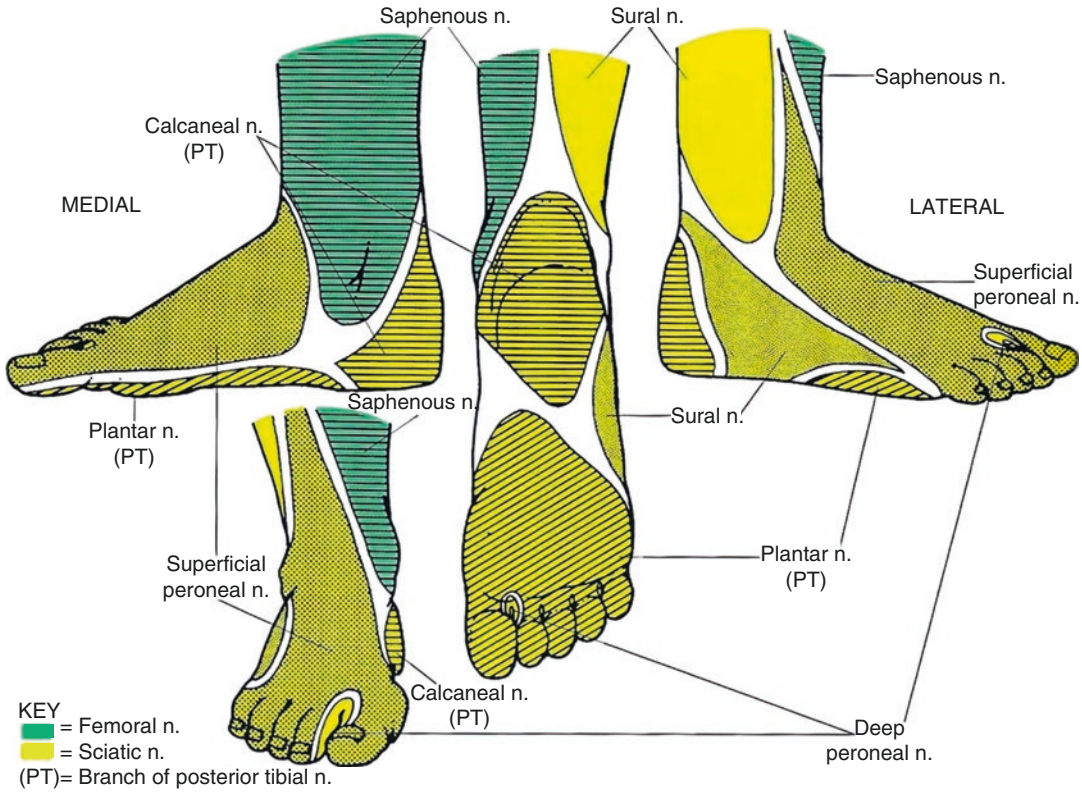

Fig. 19.5 Sensory nerve innervations of the foot and ankle. From Shah S, Tsai T, et al. Outpatient regional anesthesia for foot and ankle surgery. Int Anesthesiol Clin. 2005;43(3):143–51. Reprinted with permission from Wolters Kluwer Health, Inc.

dle is inserted just posterior to the palpated pulse angle toward the medial malleolus. Advance the needle until the posterior side of the medial malleolus is encountered. Withdraw the needle about 2–3 mm and aspirate to ensure that the needle tip is not within the Tibial artery. Inject 5–8 mL of local anesthetic at this spot (Figs. 19.6 and 19.7).

Sural Nerve Block

Positioning: Supine with leg internally rotated

Landmarks:

- Lateral malleolus
- Achilles tendon

Injection Technique: The point of needle insertion is just anterior to the Achilles tendon and 2–3 cm above the lateral malleolus. A 25-gauge, 1.5-in.-long needle is inserted, and a small wheal is raised after a negative aspiration test. Advance the needle toward the fibula and approximately 5 mL of local anesthetic is injected subcutaneously (Fig. 19.6).

Superficial Peroneal and Saphenous Nerve Blocks

Positioning: Supine with leg in neutral position

Landmarks:

- Medial and lateral malleoli

Injection Technique: Insert a 25-gauge, 1.5-in.-long needle at the anterior border of the medial malleolus and 2–3 cm above the ankle joint. After a small wheal is raised and a negative aspiration test, advance the needle across the anterior ankle until the anterior border of the lateral malleolus is reached. Inject 5–10 mL of local anesthetic subcutaneously while advancing the needle. The needle might need to be withdrawn and reinserted in order to reach across the entire area. Perform an aspiration test each time the needle is reinserted to ensure that the local anesthetic is not being injected into a blood vessel (Fig. 19.8).

Fig. 19.6 Posterior view of the foot and ankle. *Arrow 1* shows the direction of needle insertion for anesthetizing the posterior tibial nerve. *Arrow 2* shows the direction of needle insertion for anesthetizing the sural nerve

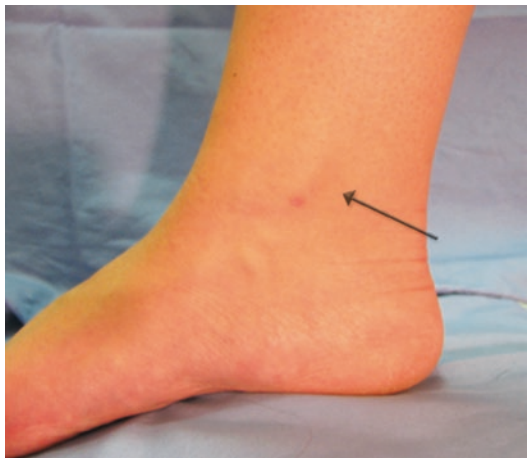

Fig. 19.7 The *arrow* shows the direction of the needle insertion in posterior tibial nerve block from the lateral view

Deep Peroneal Nerve Block

Positioning: Supine with the leg in neutral position

Landmarks:

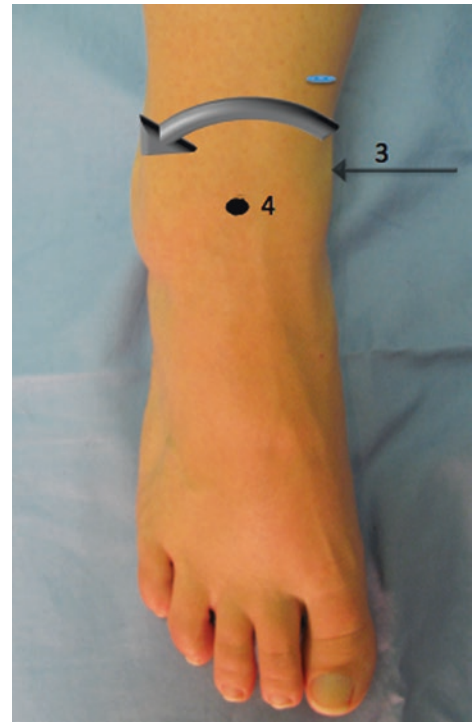

Fig. 19.8 Anterior view of the foot and ankle. *Arrow 3* shows the place of needle insertion in superficial peroneal nerve block. The *gray arrow* indicates the direction of needle advancement. *Arrow 4* shows the direction of needle insertion in deep peroneal nerve block. Note that the needle is perpendicular to the coronal plane

- Dorsalis pedis artery
- Extensor hallucis longus tendon
- Medial and lateral malleoli

Injection Technique: At the level of the malleoli, identify the extensor hallucis longus tendon by dorsiflexing and plantarflexing the hallux. Palpate the pulse of the dorsalis pedis artery just lateral to the tendon. Insert a 25-gauge, 1.5-in.-long needle just lateral to the palpated artery pulse and raise a small wheal after a negative aspiration test. Advance the needle deep through the deep fascia and inject 2–3 mL of local anesthetic (Fig. 19.8).

Mayo Block

Introduction

This is essentially a field block for surgeries involving the first metatarsal phalangeal joint as

well as any hallux-related surgeries. This technique allows the surgeon to use less local anesthetic while still achieving the desired level of anesthesia to perform the surgery. This field block can also be used in the fifth toe and ray surgeries and is termed "reverse Mayo block."

Anatomy

The hallux is innervated by four nerves: one on each side of the hallux dorsally as well as plantarly. The dorsal medial nerve is one of the terminal branches from the medial dorsal cutaneous nerve. The plantar nerves arise from the medial plantar nerve. The lateral dorsal nerve is the sensory branch of the deep peroneal nerve as it innervates the first interdigital space. The medial dorsal nerve arises from the medial dorsal cutaneous nerve (Figs. 19.2 and 19.4).

Indications

- All surgeries involving the first metatarsal phalangeal joint, and hallux, including bunionectomy, amputation, and joint arthrodesis.

Procedure

Landmarks:

- First metatarsal phalangeal joint
- First intermetatarsal space

Positioning: Supine

Injection Technique: Insert the needle medially proximal to the first metatarsal phalangeal joint and approximately two-thirds down the metatarsal shaft. Direct the needle laterally and inject local anesthetic as it crosses the first metatarsal dorsally. The needle is then withdrawn and redirected plantarly. First, advance the needle laterally and inject local anesthetic subcutaneously just below the skin. Second, withdraw the needle and then direct it deep just below the first metatarsal and inject local anesthetic as the needle advances laterally. Additional local anesthetic is then deposited distally at the first interdigital space (Figs. 19.9 and 19.10).

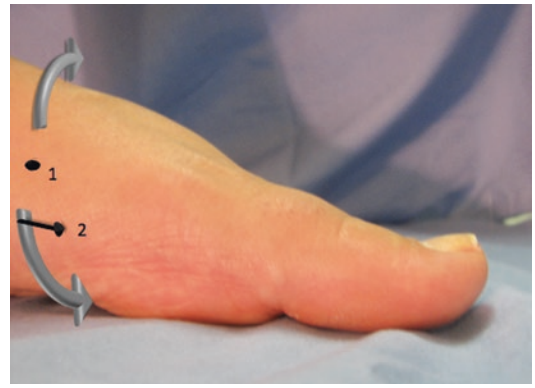

Fig. 19.9 Lateral view of the forefoot. *Arrow 1* shows the direction the needle insertion for anesthetizing the dorsal and plantar nerves around the first ray. Note that the needle is perpendicular to the sagittal plane. The *gray arrows* show the direction of needle advancements. *Arrow 2* shows the needle insertion for anesthetizing the deep branch of the plantar nerve

Fig. 19.10 AP view of the forefoot. *Arrow 3* shows the placement of needle for anesthetizing the sensory branch of the deep peroneal nerve

Digital Block

Introduction

This is a field block technique utilized when performing toe surgeries. This technique can be repeated to anesthetize multiple toes. This block can also be performed more proximally to include the metatarsals.

Anatomy

Each toe is innervated by four nerves: two dorsal proper digital nerves and two plantar proper digital nerves. Each pair of nerves courses along both sides of the toe (Figs. 19.2 and 19.4).

Indications

- All surgeries involving the toe, including amputation, arthroplasty, and arthrodesis
- Nail procedures

Procedure

Landmarks:

- Metatarsal phalangeal joint
- Metatarsal head and neck
- Proximal phalanx

Positioning: Supine

Injection Technique: Insert the needle at one side of the toe at the level just proximal to the metatarsal neck. Raise a wheal with 0.25–0.5 mL of local anesthetics. Advance the needle plantarly until just beneath the plantar skin and deposit 0.25–0.5 mL of local anesthetics. Repeat the technique on the other side of the digit to complete the block. For distal toe surgeries such as nail avulsion procedures, this block can be performed more distally at the level of the proximal phalanx. Using the same technique, local anesthetic is deposited on each side of the proximal phalanges at the midshaft (Fig. 19.11).

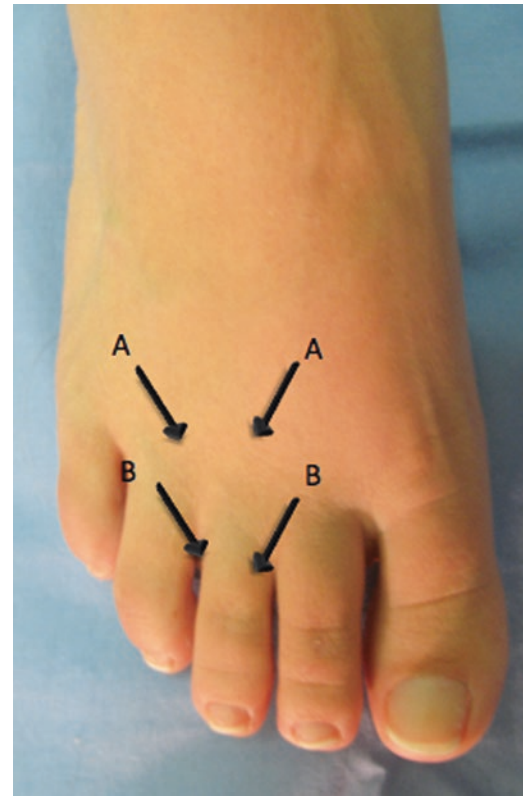

Fig. 19.11 AP view of the forefoot. *Arrows A* show the needle placement for anesthetizing the third ray. *Arrows B* show a more distal needle placement for anesthetizing only the third toe distal to the metatarsal phalangeal joint

Complications

Complications are rare in local foot and ankle anesthesia. Most allergic reactions are associated with methylparaben, a preservative in local anesthetics [8], although true allergy to local anesthetics can occur. Local toxicity occurs when large amounts of local anesthetics are injected directly into nerves or skeletal muscles; this could cause irreversible conduction loss and muscle necrosis, respectively. Systemic toxicity is also possible if sufficient amount of local anesthetic is injected intravenously or intra-arterially. Symptoms of systemic toxicity include tinnitus, convulsions, and cardiac dysrhythmias [11]. Necrosis or gangrene of the toes is another possible complication when excessive volume of local anesthetics is injected circumferentially around the toe. The risk of gangrene increases when epinephrine is added due to its vasoconstriction properties.

Case Studies

A 50-year-old female is scheduled for a chevron bunionectomy to correct a painful bunion of the left foot. She has a history of hypertension and asthma and her medication consists of a beta-blocker and albuterol inhaler as needed. She has no allergies and is otherwise healthy. This is an excellent setting to utilize regional anesthesia to achieve good intraoperative analgesia to avoid risks associated with general anesthesia. A Mayo field block (Figs. 19.9 and 19.10) would be deployed to anesthetize the four nerves innervating the first metatarsal-phalangeal joint. These nerves include the terminal branch from the medial dorsal cutaneous nerve, the terminal branches of the medial plantar nerve, and the deep peroneal nerve (Figs. 19.2 and 19.4). This method gives complete analgesia of the entire great toe distally to the mid-shaft of the first metatarsal bone.

A 20-year-old college student is seen in the office with complaint of a painful ingrown toenail of the right second toe to both nail borders. He has no medical history, takes no medications, and has no allergies. A digital local field block is great in this case to allow the procedure to be done in the office without any other adjunctive anesthesia. Complete analgesia of the second toe can be achieved by anesthetizing the two dorsal and two plantar cutaneous nerves on each side of the toe (Fig. 19.11). The injection can be performed at the base of the proximal phalanx (B sites) since only the distal port of the toe needs to be anesthetized.

Review Questions

1. Which regional anesthesia is preferred for a patient undergoing ORIF of metatarsal fractures 1–4 with dislocation of the tarsal-metatarsal joints?
   (a) Mayo block
   (b) Digital block
   (c) Posterior tibial block
   (d) Ankle block
2. Anesthetizing which pedal nerve would achieve complete analgesia of both medial and lateral plantar nerve?
   (a) Sural nerve
   (b) Posterior tibial nerve
   (c) Deep peroneal nerve
   (d) Saphenous nerve
3. All the following nerves are anesthetized in a Mayo block except:
   (a) Deep peroneal nerve
   (b) Dorsal medial cutaneous nerve
   (c) Sural nerve
   (d) Terminal branch of medial plantar nerve

Answers

1. d
2. b
3. c

References

1. Shah S, Tsai T, et al. Outpatient regional anesthesia for foot and ankle surgery. Int Anesthesiol Clin. 2005;43(3):143–51.
2. Pearce C, Hamilton P. Current concepts review: regional anesthesia for foot and ankle surgery. Foot Ankle Int. 2010;31(8):732–9.
3. Samuel R, Sloan A, et al. The efficacy of combined popliteal and ankle blocks in forefoot surgery. J Bone Joint Surg Am. 2008;90:1443–6.
4. Singelyn F. Single-injection applications for foot and ankle surgery. Best Prac Res Clin Anaesthesiol. 2002;16(2):247–54.
5. Ptaszek A, Morris S, et al. Midfoot field block anesthesia with monitored intravenous sedation in forefoot surgery. Foot Ankle Int. 1999;20(9):583–6.
6. Migues A, Slullitel G, et al. Peripheral foot blockade versus popliteal fossa nerve block: a prospective randomized trial in 51 patients. J Foot Ankle Surg. 2005;44(5):354–7.
7. Vadivelu N, Gesquire M, et al. Safety of local anesthesia combined with monitored intravenous sedation for American Society of Anesthesiologists 3 and 4 patients undergoing lower limb-preservation procedures. J Foot Ankle Surg. 2010;49:152–4.
8. Cavaliere R, Pappas E. Anesthesia. In: Banks A, Downey M, Martin D, Miller S, editors. McGlamry's comprehensive textbook of foot and ankle surgery, vol. 1. 3rd ed. Pennsylvania: Lippincott Williams & Wilkins; 2001.

9. Hadzic A, Minkowitz H, et al. Liposome bupivacaine femoral nerve block for postsurgical analgesia after total knee arthroplasty. Anesthesiology. 2016;124:1372–83.
10. Robbins J, Green C, et al. Liposome bupivacaine in forefoot surgery. Foot Ankle Int. 2015;36(5):503–7.
11. Veering B. Complications and local anaesthetic toxicity in regional anaesthesia. Curr Opin Anaesthesiol. 2003;16(5):455–9.

Selective Regional Anesthesia Options in Surgical Subspecialties

20

Hong Yan, Alan David Kaye, and Henry Liu

Iliohypogastric and Ilioinguinal Nerve Block

Introduction

These two nerves usually lie very close to each other and are frequently blocked together with the same needle insertion. Dr. Harvey Cushing reported in the Annals of Surgery in 1900 that "almost all cases of hernia, with the possible exception of those in young children, could undoubtedly be subjected to the radical operation under local anesthesia" [1]. Block of these two nerves is not commonly used in modern days; some authors claim that the block is truly underutilized for herniorrhaphy [2]. Yilmazlar and colleagues compared the ilioinguinal-iliohypogastric nerve blocks to spinal anesthesia for inguinal herniorrhaphy. They found that patients receiving the ilioinguinal-iliohypogastric nerve blocks had shorter time-to-home readiness, quicker oral intake postoperatively, and no need for recovery room care [3]. Recently, Stav et al. conducted a prospective, randomized, controlled, and observer-blinded clinical trial. They studied 166 adult male patients who were randomly assigned to one of the three groups: a preoperative transversus abdominis plane (TAP) block group, a preoperative ilioinguinal-iliohypogastric block group, and a control group. An intraoperative block of the genital branch of the genitofemoral nerve was performed in all three groups in all patients. Postoperative patient-controlled intravenous analgesia with morphine was available to all patients. The pain intensity and morphine utilization immediately after surgery and first 24 h after surgery were significantly lower in both block groups than the control group. However, during the first 24 h after surgery, morphine consumption in the ilioinguinal-iliohypogastric block group was lower compared with the TAP group. Thus they concluded that ultrasound-guided ilioinguinal-iliohypogastric blocks provided better pain control than ultrasound-guided posterior TAP following the Lichtenstein patch tension-free method of open inguinal hernia repair in men during 24 h after surgery [4].

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_20

Indications

Ilioinguinal-iliohypogastric block is indicated as a treatment for both acute and chronic pain involving the groin area, lower abdominal wall, and inguinal region. If the block is used for herniorrhaphy, the hernia sac needs additional local infiltration because it contains peritoneum and visceral nerves. There is no specific contraindication for this block.

Anatomy

The iliohypogastric nerve may have a small contribution from T12, but it primarily originates from L1. The nerve travels around the body, starting posteriorly, and then heading laterally before emerging anteriorly. At the anterior superior iliac spine (ASIS) area, the iliohypogastric nerve pierces through the posterior portion of the transverse abdominal muscle and then divides into lateral and anterior branches. The lateral branches penetrate both the internal and external oblique muscles and provide sensation to the skin of the posterior lateral gluteal region. The anterior branch penetrates through the internal oblique muscle approximately 2 cm medial to the anterior superior iliac spine and perforates the external oblique muscle, distributing sensory fibers to the skin of the abdomen above the pubis [5] (Fig. 20.1).

The relatively smaller ilioinguinal nerve originates from L1. It emanates from the upper part of the lateral border of the psoas major muscle and runs caudad to the iliohypogastric nerve. The nerve crosses obliquely and anteriorly to the quadratus lumborum and iliacus muscles and then perforates the transverse abdominis muscle near the anterior part of the iliac crest. In the anterior abdominal trunk, the nerve travels between the transverse abdominis and the internal oblique muscles (Fig. 20.1). It occasionally anastomoses with branches of the iliohypogastric nerve at the ASIS level. It pierces the internal oblique muscle and accompanies the spermatic cord through the inguinal ring into the inguinal canal. It provides skin sensation over the root of the penis, to the superior inner aspect of the thigh, to the upper part of the scrotum in males, and to the skin covering the mons pubis and lateral part of labia in females [6].

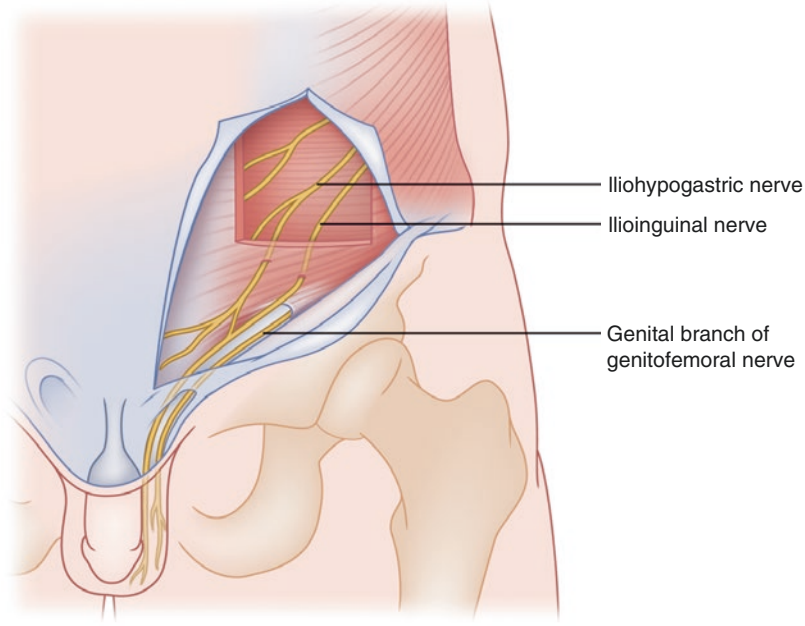

Fig. 20.1 Three nerves (iliohypogastric nerve, ilioinguinal nerve, and genitofemoral nerve) starting from spinal cord to exiting inguinal canal

Technique

Ultrasound-Guided Technique

The patient is usually in a supine position. After locating and labeling the ASIS and the umbilical button, a line is drawn between the ASIS and the umbilical button. A linear high- frequency (7–13 MHZ) ultrasound probe is usually used for this block. By adjusting the ultrasound setting (depth is usually better at 1–3 cm), the differential muscle layers can be visualized, and the nerves can sometimes be imaged. Unfortunately, the nerves are often very difficult to consistently identify. The ilioinguinal nerve is usually located in the plane between the transverse abdominal muscle and the internal oblique muscle above the ASIS, while the iliohypogastric nerve usually is located between the internal oblique muscle and the external oblique muscle. We typically use a 22-gauge or 23-gauge needle and inject locally throughout the needle's path with 5–8 mL of local anesthetic agent directly deposited to each nerve if possible. The total local anesthetic volume is 20–30 mL.

Ideally, one should identify and target the individual nerve; however, the nerves cannot always be identified. In this case, the reliable end points for the ilioinguinal-iliohypogastric nerve blocks are the transverse abdominal/internal oblique muscle plane where the ilioinguinal nerve is reported to be found in 100% of patients [7], and the plane between the internal oblique muscle and the external oblique muscle, which contains the iliohypogastric nerve (Fig. 20.2).

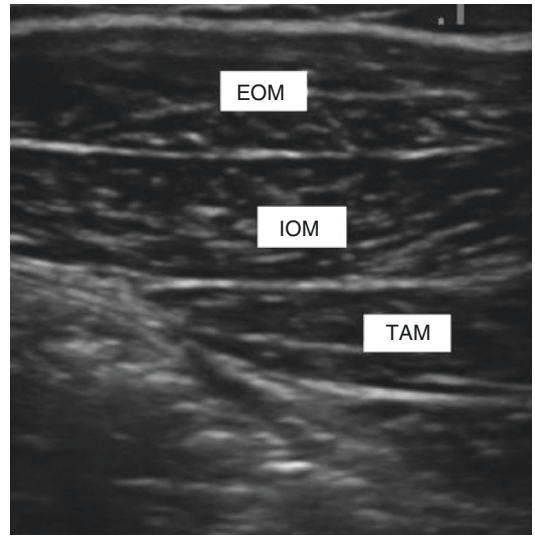

Fig. 20.2 Ultrasound image showing the layers of abdominal muscles. *EOM* external oblique muscle, *IOM* internal oblique muscle, *TAM* transverse abdominal muscle [5]

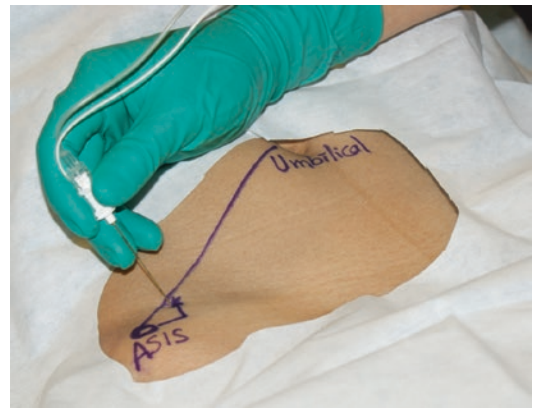

Fig. 20.3 Illustrating the ilioinguinal/iliohypogastric nerve block needle entry point: 3 cm medial and 3 cm above ASIS [5]

Using Anatomical Landmarks

The ultrasound-guided technique has gained so much popularity; this block is rarely done by just using anatomical landmarks in modern practice. The patient is usually placed in a supine position. After marking the ASIS and drawing a line between the ASIS and the umbilical button, the patient's lower quadrant should be sterilely prepared. The injection site is located 3 cm medial to the ASIS and 3 cm above the ASIS (Fig. 20.3). As aforementioned, the key to adequate blockade is injecting sufficient local anesthetic agent into the two planes: the plane between the transverse abdominal muscle and the internal oblique muscle (the ilioinguinal nerve) and the plane between the internal oblique muscle and the external oblique muscle (the iliohypogastric nerve). Using a 22-gauge or 23-gauge needle, advance the needle at a right angle to the skin in all planes. A "click" is usually felt as the needle passes through the external oblique muscle. Before advancing further, inject 8–10 mL of local anesthetic agent into this plane. Then, advance the needle until a

second "click" is felt. This indicates that the needle has advanced through the internal oblique muscle. At this point, another 8–10 mL of local anesthetic agent is injected. One should inject 8–10 mL into each plane and along the needle path. We usually limit our total dose to less than 40 mL in volume and under toxic drug dose.

Some anesthesiologists use two separate needle entry points for ilioinguinal and iliohypogastric blockade. To access and block the iliohypogastric nerve, a needle is directed 3 cm medial and 3 cm superior to the ASIS. Blockade of the ilioinguinal nerve can be accomplished by placing a needle 2 cm medial to the ASIS and 2 cm inferior to the entry point for the iliohypogastric nerve. Next, the needle is directed toward the pubic symphysis in a fanlike manner, piercing through the fascia of the external oblique muscle and depositing local anesthetic along its path. Because the ilioinguinal and iliohypogastric nerves are located at different fascial planes among the three muscles (IO, EO, and TA), these blind techniques have a low success rate.

Anesthetic Agents

Our groups typically use 0.5% bupivacaine or 1% ropivacaine for surgical anesthesia and 0.25% bupivacaine or 0.5% ropivacaine for postoperative analgesia or chronic pain analgesia. Beaussier et al. reported that adding clonidine (75 μg) to local anesthetic (ropivacaine) can reduce motion pain but may increase the chance of orthostatic hypotension [8]. Popping et al. analyzed multiple studies and they concluded that adding clonidine to intermediate- or long-acting local anesthetics for single-shot peripheral nerve or plexus blocks prolongs the duration of analgesia and motor blockade by about 2 h [9]. The increased incidence of hypotension, fainting, and sedation may limit its use and there is a considerable additional cost for this adjuvant medication. After comparing three concentrations (0.125, 0.25, and 0.375%) of levobupivacaine, Disma et al. reported that 0.25% levobupivacaine provided satisfactory postoperative analgesia with the fewest side effects [10].

Continuous ilioinguinal-iliohypogastric nerve block with ultrasound-guided placement of bilateral catheters has been reported [1]. This block provides intraoperative and postoperative analgesia for procedures using a Pfannenstiel incision. Also, this block provides a good option for patients when epidural analgesia is contraindicated. The technique involves inserting an 18-gauge Tuohy epidural needle at the same entry point as single-shot block (3 cm medial and 3 cm above the ASIS). With ultrasound guidance, after penetrating the external oblique muscle and internal oblique muscle, a multi-orifice catheter is threaded through the Tuohy needle into the plane between the internal oblique muscle and the transverse abdominal muscle. The catheter should be directed medially about 3 in. This block should be performed bilaterally. Once placed, each catheter is connected to a continuous infusion of 0.5% ropivacaine or 0.2–0.25% bupivacaine set at a flow rate of 2 mL/h. This technique is very similar to TAP but differs from TAP block in two ways: (1) the needle entry for the ilioinguinal-iliohypogastric continuous block is more medial than the technique for the TAP block and (2) this technique aims for blockade of L1 and T12, while the TAP technique blocks sensory fibers from T10 to L1, or even higher. Gucev et al. placed continuous catheters into the plane between the internal oblique muscle and the transverse abdominal muscle. Ilioinguinal-iliohypogastric block with 0.2% ropivacaine plus oral ibuprofen for postoperative analgesia after cesarean delivery resulted in low pain scores postoperatively, minimal use of opioid supplement, and no report of nausea and vomiting [11]. This suggests that continuous ilioinguinal-iliohypogastric nerve blockade deserves further clinical studies to validate this technique to be an important component of multimodal analgesia after cesarean delivery.

Complications

1. Hemodynamic changes are usually minimal because this block does not cause sympathetic blockade. Patient may develop hypotension if clonidine is added to the local anesthetic solution.

2. Local anesthetic toxicity is always a concern, especially when it is done bilaterally. But the possibility in this block is very small, even though this block involves multiple-point injections. The volume is small, and the blood circulation at the injection sites is less luxurious than the epidural or intercostal spaces. The total dose is significantly lower than the toxic dosage.
3. There are reports of small and large bowel perforations, so a blunt needle is recommended for this block. When inserting the needle, try to avoid being too deep or inserting without assurance of needle location. In most patients, the needle is inserted no more than 1.5 cm after passing through the external oblique muscle layer.
4. Subcutaneous hematoma can occur after this block.
5. Pelvic hematomas have been reported, so have bowel hematomas in pediatric patient [12].
6. Transient femoral anesthesia was reported to occur in about 3.5–7% of the patients who received ilioinguinal/iliohypogastric nerve block and occurs more frequently when the injection site is located lower than the ASIS and the needle tip is deep [13].

Genitofemoral Nerve Block

Introduction

The genitofemoral nerve block is utilized as a treatment for chronic pain of the pelvis, the perineal area, and the upper thigh and can be combined with ilioinguinal/iliohypogastric nerve blocks for surgical procedures involving the groin area.

Indications

1. Performed with ilioinguinal and iliohypogastric nerve blocks for inguinal hernia repair, orchiopexy, and hydrocelectomy.
2. As a nerve block supplementing femoral nerve block for long saphenous vein stripping.
3. For the diagnosis of genitofemoral neuralgia.
4. For chronic pain syndromes in the pelvic or groin areas, such as border nerve syndrome [14].
5. The genitofemoral block can also be used for hemiscrotal anesthesia and pain treatment [15].

There are no specific contraindications for this block.

Anatomy

The genitofemoral nerve originates from the L1 and L2 ventral rami and is formed within the psoas major. The nerve, primarily sensory in function, contains a small motor component and descends obliquely, advancing through the psoas muscle to emerge at its abdominal surface near the medial border. There, the genitofemoral nerve divides into femoral and genital branches at various distances from the inguinal ligament. The femoral branch joins the femoral artery and travels underneath the inguinal ligament, penetrating the fascia lata. It supplies sensation to a small area of skin immediately below the inguinal ligament. The genital branch enters the inguinal canal through the deep ring and travels with the spermatic cord to supply the cremaster and dartos muscles and sends small terminal sensory fibers to the skin of the scrotum in males. It runs inside the inguinal canal with terminal fibers to the round ligament of the uterus and the skin of labium majus in females [5].

The Technique

Ultrasound-Guided Technique

For the block of the genital branch, we will use the technique described by Peng [14]. The probe is placed perpendicular to the inguinal ligament. By adjusting the probe position we can identify the femoral artery. It serves as a reference structure. Next, we identify the internal ring and spermatic cord, which is oval or circular in shape and contains one or two arteries (the testicular artery and the artery to the vas deferens). The vas deferens is often seen as a thick tubular structure within the spermatic cord. The probe is then moved medially and caudally to a

final location approximately 1 in. lateral to the pubic tubercle. We usually use out-of-plane technique with the needle approaching the skin from the lateral aspect of the probe. Local anesthetic without epinephrine is used to avoid the possible vasoconstriction effect on the testicular artery. Because of the anatomical anomalies found with the location of the genital branch in the genitofemoral nerve, we suggest injecting 5 mL of local anesthetic agent inside and another 5 mL outside the spermatic cord to provide adequate blockade [14, 16].

The femoral branch can usually be visualized with the ultrasound probe immediately lateral to the femoral artery. It sometimes appears attached to the femoral artery lateral wall. Block of the femoral nerve can also block this nerve.

Anatomical Landmark Technique

The femoral branch is blocked by locating the femoral artery pulse. After inserting a 25-gauge needle just lateral to the femoral artery pulse, inject 5 mL of local anesthetic solution. Next, inject 5 mL of local anesthetic in a fanlike pattern along a 5–7 cm path inferior to femoral pulse. The genital branch is blocked by identifying the pubic tubercle and inserting a 25-gauge needle 1 in. lateral to the pubic tubercle and below the inguinal ligament. A total local anesthetic volume of 10 mL, without epinephrine, is needed to achieve this block (Fig. 20.4).

Complications

1. Local pain.
2. Local anesthetic toxicity is always a concern, but with this block risk is low because the total local anesthetic dose is significantly below the toxic dose. Also, blood flow is not as rich as in the epidural or intercostal spaces.
3. Subcutaneous hematoma can occur after this block.

Penile Block

For many years, the penile block has been widely used for circumcisions and other penile surgeries. However, the anatomy of the penile block still confuses many anesthesiologists and contributes to variations of technique.

Indications

1. Circumcision
2. Phimosis and paraphimosis reduction
3. Dorsal penile skin surgery
4. Distal hypospadia repair
5. Postoperative analgesia in penile surgery

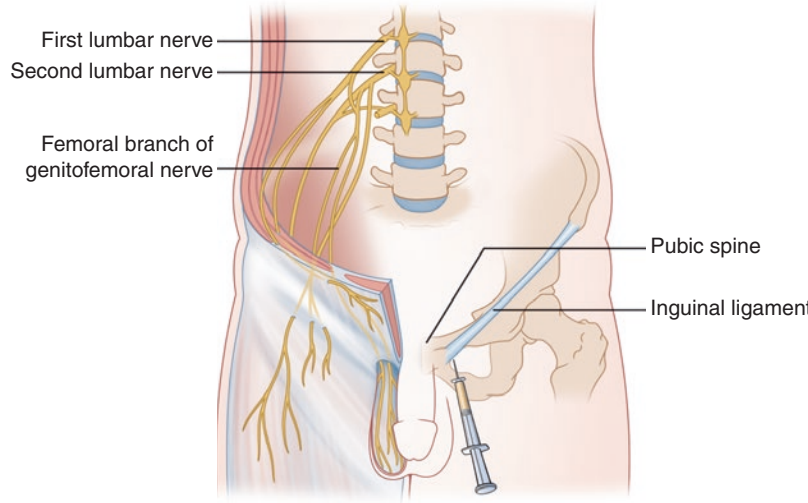

Fig. 20.4 Anatomic landmark of genital branch of genitofemoral nerve

Contraindications

1. Suspected testicular torsion
2. Infection of the skin at the proposed injection site

Anatomy

The penile nerve is derived from the pudendal nerve (S2–4). The penile nerve usually divides into the right and left dorsal nerves of the penis and courses under the pubis symphysis. Then it travels under Buck's fascia to supply sensory innervations to the penis. Both left and right penile nerves travel lateral to the penile arteries (Fig. 20.5).

Technique

Although many variations in blockade technique exist, the most common approach targets the two dorsal penile nerves for local anesthetic injection as well as subcutaneous local administration circumferentially. Recent studies indicate that to achieve adequate foreskin analgesia, supplemental dorsal nerve blocks with ventral subcutaneous infiltration just proximal to the incision line will improve surgical anesthesia and avoid inconsistency [17]. Metzelder found that the penile block for hypospadias repair in children works better than caudal anesthesia (significantly less impaired micturition) [18].

Ultrasound-Guided Penile Dorsal Nerve Block

Sandeman described this ultrasound-guided penile block in children under general anesthesia [19]. They used real-time scanning to guide bilateral injections of local anesthetics into the subpubic space, deep to Scarpa's fascia either side of the midline fundiform ligament. Scanning can confirm that the local anesthetic has spread to contact the deep fascia on each side. A subcutaneous wheel of local anesthetic along the penoscrotal junction completes the block. Gurkan et al. described ultrasound-guided penile block in adult patient [20].

Complications

1. Inadequate block is common.
2. Hematoma occurs.
3. Penile ischemia is very rare.

Clinical Pearls

Iliohypogastric and Ilioinguinal Nerve Block

- Sedate the patient before proceeding with the block.
- The reliable end point for the inexperienced practitioner using ultrasound guidance for the

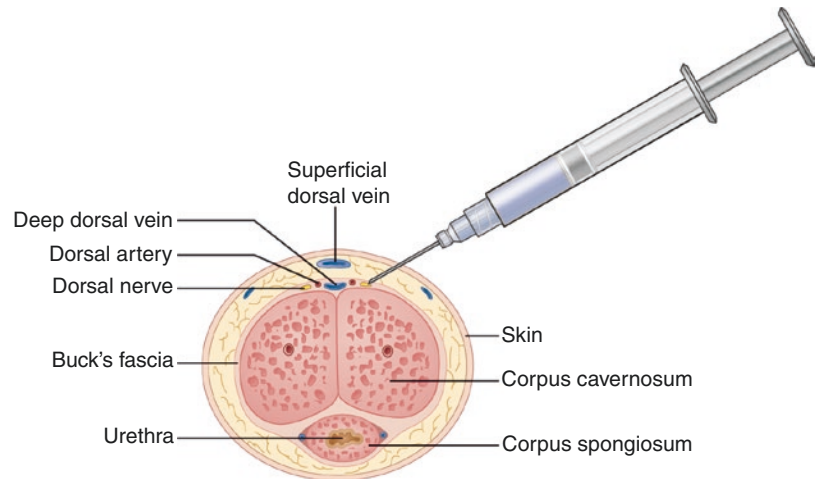

Fig. 20.5 Penile block

block of the ilioinguinal-iliohypogastric nerves is the plane between the transverse abdominal and the internal oblique muscles. The nerves are located in this plane in almost 100% of patients [7].
- One should feel resistance while moving through muscle tissue and a loss of this resistance when exiting the muscle. A blunt needle will usually make the loss of resistance more appreciable.
- Regardless of the technique, if the nerves are not easily identifiable, target the anatomic plane where the nerves lie to inject the local anesthetic solution.
- If you can locate the target nerves, try to keep them in the middle of the ultrasound image.
- Pay attention to the needle insertion depth. Do not insert the needle too deep and avoid getting into peritoneal cavity. This will reduce the incidence of bowel perforation and hematoma.
- Adding a genitofemoral nerve block to the ilioinguinal-iliohypogastric nerve block may not offer any extra benefit to pediatric patients undergoing hernia repair [18].

Genitofemoral Nerve Block

- Successful injection of the genitofemoral branches requires appropriate volume, typically requiring 10 mL or more of local anesthetic solution.
- A multi-direction infiltration will help the adequacy of the block.
- Just use plain local anesthetics; do not mix with epinephrine.
- Sterile preparation is important because the area is a breeding ground for pathogens.

Penile Block

- If possible, try to feel the pulse of the penile artery. The needle insertion site is less important because of the skin mobility; inject lateral to the pulse. The superficial dorsal vein can serve as a landmark for midline. Deposit local anesthetics under Buck's fascia where the penile nerves travel.
- Because the superficial and deep dorsal veins are both located at the dorsal midline, try to avoid a straight-down midline approach. This will significantly minimize the occurrence of hematoma.
- Penile ischemia can be prevented by avoiding puncture of the penile arteries, avoiding a larger than necessary volume of local anesthetic, and avoiding hematoma formation.

Summary

In summary, these blocks are valuable for a wide range of indications. Appreciation of anatomy and proper technique with ultrasound can improve efficacy of these selective nerve blocks and reduce potential side effects. In this regard, many groups have moved away from bupivacaine because of its potential toxicity with intravascular injection; however, other groups still utilize bupivacaine, making good technique that is much more significant for ensuring best outcomes and patient safety.

Review Questions

1. The primary nerve root supplying the ilioinguinal and iliohypogastric nerves is:
   (a) L3
   (b) L2
   (c) L1
   (d) T12
2. All the following are advantages of ilioinguinal and iliohypogastric blocks compared to spinal except:
   (a) Quicker postoperative discharge
   (b) Faster postoperative oral intake
   (c) Less need for recovery room
   (d) Less postoperative surgical complications
3. The ilioinguinal nerve supplies sensation to all the following areas except:
   (a) Skin covering the base of the penis
   (b) Skin covering the upper scrotum

(c) Skin covering the mons pubis
(d) Skin covering the posterior aspect of the upper thigh

4. The ilioinguinal and iliohypogastric nerves are commonly located between:
   (a) The transverse abdominal muscle and the internal oblique muscle
   (b) The internal oblique muscle and the external oblique muscle
   (c) The transverse abdominal muscle and the rectus sheath
   (d) The rectus sheath and aponeurosis of the external oblique muscle
5. Continuous ilioinguinal and iliohypogastric blocks for Pfannenstiel incisions:
   (a) Place catheter unilaterally
   (b) Place bilateral catheters between the transverse abdominal and the internal oblique muscles
   (c) Place bilateral catheters between the external and the internal oblique muscles
   (d) Place bilateral catheters between the internal and the external oblique muscles aiming medially
6. Complications to the ilioinguinal and iliohypogastric block include all except:
   (a) Hemodynamic instability
   (b) Bowel perforation
   (c) Subcutaneous hematoma
   (d) Pelvic hematoma
7. Indications for genitofemoral nerve block include all except:
   (a) Supplemental block for hernia surgery
   (b) Aid in diagnosis of genitofemoral neuralgia
   (c) Treatment of some chronic pelvic pain syndromes
   (d) Primary block for orchiopexy surgery
8. The genitofemoral nerve originates from:
   (a) Dorsal rami of T12 and L1
   (b) Dorsal rami of L1 and L2
   (c) Ventral rami of L1 and L2
   (d) Ventral rami of T12 and L1
9. The genital branch of the genitofemoral nerve:
   (a) Enters the inguinal area through the deep ring
   (b) Travels with the spermatic cord
   (c) Supplies the cremaster and dartos muscles
   (d) All of the above
10. Anatomical landmarks used for ultrasound block of the genital branch include all except:
   (a) ASIS
   (b) Umbilical button
   (c) Inguinal ligament
   (d) Quadriceps muscle
11. Clinical pearls for genitofemoral block include all except:
   (a) Use epinephrine mixed with local anesthetic
   (b) Use multidirectional infiltration
   (c) Sterile preparation extremely important
   (d) Use local anesthetic without epinephrine
12. Indications for penile block include:
   (a) Cystoscopy
   (b) Retrograde urethrogram
   (c) Circumcision and distal hypospadias repair
   (d) Testicular torsion
13. Contraindications for penile block:
   (a) Dorsal penile skin surgery
   (b) Postoperative analgesia for penile surgery
   (c) Penile skin infection
   (d) Phimosis surgery
14. All of the following concerning the penile nerve are true except:
   (a) Derived from S2–4
   (b) Courses under the pubic symphysis
   (c) Travels under Buck's fascia
   (d) Blocked with local anesthetic containing epinephrine
15. Complications from penile block:
   (a) Inadequate block
   (b) Penile ischemia is common
   (c) Hematomas are rare
   (d) Local anesthetic toxicity from large-volume doses

Answers

1. c
2. d
3. d

4. a
5. b
6. a
7. d
8. c
9. d
10. d
11. a
12. c
13. c
14. d
15. b

References

1. NYSORA-II/IH continuous Block: http://www.nysora.com/peripheral_nerve_blocks/classic_block_tecniques/3066-ilioinguinal_and_iliohypogastric_blocks.html.
2. Wang H. Is ilioinguinal-iliohypogastric nerve block an underused anesthetic technique for inguinal herniorrhaphy? South Med J. 2006;99(1):15.
3. Yilmazlar A, Bilgel H, Donmez C, Guney A, Yilmazlar T, Tokat O. Comparison of ilioinguinal-iliohypogastric nerve block versus spinal anesthesia for inguinal herniorrhaphy. South Med J. 2006;99(1):48–51.
4. Stav A, Reytman L, Stav MY, Troitsa A, Kirshon M, Alfici R, Dudkiewicz M, Sternberg A. Transversus abdominis plane versus ilioinguinal and iliohypogastric nerve blocks for analgesia following open inguinal herniorrhaphy. Rambam Maimonides Med J. 2016;7(3):e0021. https://doi.org/10.5041/RMMJ.10248. PMID: 27487311.
5. Liu H, Fox CJ, Yarborough MJ, Kaye AD. Selective regional anesthesia options in surgical subspecialties. In: Kaye D, Urman RD, Vadivelu N, editors. Essentials of regional anesthesia. New York: Springer; 2012. p. 525–39. https://doi.org/10.1007/978-1-4614-1013-3. ISBN 978-1-4614-1012-6. Chapter 20.
6. Katz G. Ilioinguinal and iliohypogastric nerves. In: Atlas of regional anesthesia. Connecticut: Appleton-Century-Crofts/Norwalk; 1985. p. 114–5.
7. Ford S, Dosani M, Robinson AJ, Campbell GC, Ansermino JM, Lim J, et al. Defining the reliability of sonoanatomy identification by novices in ultrasound-guided pediatric ilioinguinal and iliohypogastric nerve blockade. Anesth Analg. 2009;109(6):1793–8.
8. Beaussier M, Weickmans H, Abdelhalim Z, Lienhart A. Inguinal herniorrhaphy under monitored anesthesia care with ilioinguinal-iliohypogastric block: the impact of adding clonidine to ropivacaine. Anesth Analg. 2005;101(6):1659–62.
9. Pöpping DM, Elia N, Marret E, Wenk M, Tramèr MR. Clonidine as an adjuvant to local anesthetics for peripheral nerve and plexus blocks: a meta-analysis of randomized trials. Anesthesiology. 2009;111(2):406–15.
10. Disma N, Tuo P, Pellegrino S, Astuto M. Three concentrations of levobupivacaine for ilioinguinal/iliohypogastric nerve block in ambulatory pediatric surgery. J Clin Anesth. 2009;21(6):389–93.
11. Gucev G, Yasui GM, Chang TY, Lee J. Bilateral ultrasound-guided continuous ilioinguinal-iliohypogastric block for pain relief after cesarean delivery. Anesth Analg. 2008;106(4):1220–2.
12. Frigon C, Mai R, Valois-Gomez T, Desparmet J. Bowel hematoma following an iliohypogastric-ilioinguinal nerve block. Paediatr Anaesth. 2006;16(9):993–6. https://doi.org/10.1111/j.1460-9592.2006.01909.x. PMID: 16918666.
13. Tani M, Sato M, Son I, Tanabe H, Hirota K, Shirakami G, Fukuda K. Transient femoral nerve palsy after ilioinguinal-iliohypogastric nerve block: comparison of ultrasound-guided and landmark-based techniques in adult inguinal hernia surgery. Masui. 2015;64(6):603–9. PMID: 26437548.
14. Peng PW, Tumber PS. Ultrasound-guided interventional procedures for patients with chronic pelvic pain—a description of techniques and review of literature. Pain Physician. 2008;11(2):215–24. PMID: 18354713.
15. Pankhania M, Ali S. The genitofemoral nerve block: a method for hemiscrotal anaesthesia at the bedside. Ann R Coll Surg Engl. 2012;94(4):272. https://doi.org/10.1308/003588412X13171221591259a. PMID: 22613308.
16. Long RM, McCartan D, Cullen I, Harmon D, Flood HD. A preliminary study of the sensory distribution of the penile dorsal and ventral nerves: implications for effective penile block for circumcision. BJU Int. 2010;105(11):1576–8.
17. Metzelder ML, Kuebler JF, Glueer S, Suempelmann R, Ure BM. World J Urol. 2010;28(1):87–91.
18. Sasaoka N, Kawaguchi M, Yoshitani K, Kato H, Suzuki A, Furuya H. Evaluation of genito- femoral nerve block, in addition to ilioinguinal and iliohypogastric nerve block, during inguinal hernia repair in children. Br J Anaesth. 2005;94(2):243–6.
19. Sandeman DJ, Dilley AV. Ultrasound guided dorsal penile nerve block in children. Anaesth Intensive Care. 2007;35(2):266–9. PMID: 17444318.
20. Gürkan Y, Kus A, Aksu C, Çiftçi S, Çulha M, Pandin P. Ultrasonography-guided penile block for adult penile surgery. Can J Anesth. 2016;63(6):780–1.

Obstetric Anesthesiology

21

Debbie Chandler, Ray Paschall, Leslie Robichaux, Burton Beakley, Elyse M. Cornett, and Alan David Kaye

Introduction

Obstetric anesthesiology gained noble notoriety when Prince Albert requested Dr. John Snow to provide analgesia for Queen Victoria during the birth of Prince Leopold. This unique beginning is reflected by the continuing uncommon nature of the practice [1].

No hard guidelines can be established to govern the conduct of obstetric anesthesia because each case is truly distinct and is best served by tailored response to the individual circumstances presented. Answers are often unclear or drift and put the capability of the anesthesia provider in focus because the outcomes for two patients are at risk. Providers of obstetric anesthesiology commonly use regional anesthesia techniques to provide care for their patients. This represents a growing trend based on the belief that there are an increased safety profile and maternal satisfaction associated with using regional anesthesia for the delivery process [2]. The classic technical skills required are mastered through repetition and training although the use of ultrasound in the labor suite can be anticipated to become more prevalent to facilitate catheter placement [3]. Historically, the suggested learning curve for proficiency is 60 regional anesthetic placements [4, 5].

The remarkable and persistent myth of painless childbirth has been the natural history still propagated despite the cumulative evidence to the contrary. Consequently, patient anxiety about accepting regional anesthesia remains challenging because of complex familial, personal, or cultural factors frequently inconsistent with values or perception of the provider. How to lower these anxieties lacks a simple answer. Impressions made by the provider during first communications can relieve apprehensions and create a mutual understanding that is a functional anxiolytic without pharmacologic administration. The decision to use or decline regional anesthesia will always be an individual patient preference involving more complex variables than that can be succinctly summarized. The usual elements of informed consent must always

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_21

be respected, but the patient in overwhelming labor pain is frequently the patient who has reconsidered an earlier decision to decline regional anesthesia.

It seems inconceivable that any laboring patient would wish to suffer from pain approximating that of distal amputation of a finger as depicted by the pain rating index in Fig. 21.1 [6]. Indeed the American College of Obstetricians and Gynecologists has issued position statements reflecting support for the provision of pain relief during labor that requires only a patient request [7, 8]. Pain has personal meaning to each patient, and once their tolerance has been met, or their threshold exceeded, then the option for regional anesthesia should be quickly offered. To not do so has ethical implications, and current obstetric anesthesia practices have evolved to make available regional anesthesia earlier and later than has been the historical custom. Active management of labor by the obstetrical team makes the need for waiting until prescribed cervical dilation has been met a moot point. If the anesthesia provider is comfortable and capable, then late intervention also has no limits relative to dilation or station.

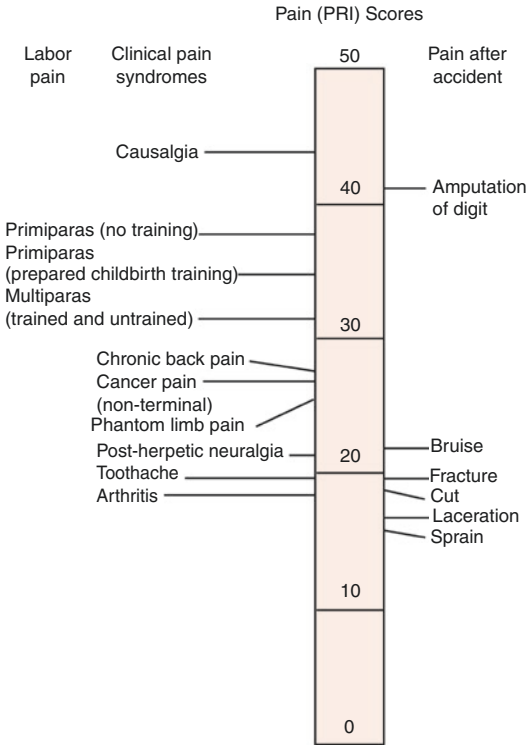

Fig. 21.1 Pain rating index

Communication is essential to providing safe care for mother and fetus. Changes in the obstetric population and obstetric practice have increased the number of high-risk patients likely to be encountered. If the risk of childbirth is increased, then the need to continually examine practice habits becomes important to assure a good outcome for mother and fetus. The ASA guidelines for obstetric anesthesia should always guide practice conduct [9]. Verbatim, the guidelines state "The choice of analgesic technique depends on the medical status of the patient, progress of labor, and resources at the facility. When sufficient resources (e.g., anesthesia and nursing staff) are available, neuraxial catheter techniques should be one of the analgesic options offered."

Physiologic Changes of Pregnancy

Increased maternal metabolic rate, cardiac modifications, and especially circulatory volume changes which mediate the hemodynamic response to neuraxial anesthesia are the focus of most obstetric anesthesia providers because the likelihood of these changes directly impacting the mother or fetus is common. Also, during pregnancy, many normal alterations occur in the pulmonary system. Those physiologic changes of pregnancy predispose difficulty with mask ventilation, intubation, rapid desaturation, and high airway pressure during mechanical ventilation. Obviously, all organ systems are impacted by the pregnant state. Total body water change (6.5–8.5 L gain at term) is impressive and is a significant hypervolemic adaptation of pregnancy. Proper respect paid to the changes known to occur in each system with appropriate risk determinations has made the danger of unintended morbidity a rare event. Multiple consensus summaries of important physiologic and anatomic changes are available for review [10, 11].

Cardiovascular Changes During Pregnancy

- Systolic and diastolic blood pressure decreases until midpregnancy with a return to prepregnancy values by term. Lowest blood pressures occur at 16–20 weeks.
- Decreased systemic vascular resistance 20%: All vascular beds are affected, especially uterine, which increases from 50 mL/min at 10 weeks to 500 mL/min at term. Increased progesterone is implicated as a smooth muscle relaxant.
- Increased intravascular volume by 25–40%: ECHO reveals 10% increased end diastolic volume.
- Increased heart rate by 15–20% and cardiac output by 30–50% which is due to stroke volume and is position dependent: In fact, CO begins to increase by 5 weeks of gestation and is approximately 40% above baseline at the end of the first trimester.
- Increased total blood volume by 25% and plasma volume by 40–45% which is reflected in increased SV.
- Aortocaval compression with supine position: CO decreases 14% when the patient is supine rather than tilted. The sitting position may also lead to aortocaval compression with a 10% of the CO.
- Autotransfusion with uterine contraction can also add a 300–500 mL bolus to the circulating volume which could add another 10% to cardiac output at the time of measurement.
- First heart sound (S1) is accentuated and there is a typical systolic ejection murmur. S3 and S4 may also be heard but there is no clinical significance.
- Because of the elevation of the diaphragm by the gravid uterus, there is a leftward displacement of point of maximal cardiac impulse.
- On EKG, especially during the last trimester, PR and QT interval may be shortened. This might have some clinical implications for women with prolonged QT syndrome. It is not uncommon to notice depressed ST segments and low-voltage T-waves in the last trimester.

Pulmonary Changes During Pregnancy

- Elevated diaphragm.
- Increased upper airway edema and friability.
- Decreased functional residual capacity 20%.
- Increased minute ventilation 40–50%.
- Partially compensated respiratory alkalosis, pCO2 27–32 mmHg, pH 7.40–7.45.
- Depleted bicarbonate, 17–22 mEq implying limited buffering capacity.
- Oxygen consumption is increased 20% which creates a tendency for rapid maternal and fetal hypoxia and maternal rapid desaturation during supine position and during intubation.
- Total lung capacity is slightly reduced whereas tidal volume is increased by 45%.
- No change in vital capacity.

Hematologic/Laboratory Changes During Pregnancy

Physiology

- Plasma volume begins to expand as early as 6 weeks of gestation to reach its maximum by 34–36 weeks of gestations.
- Physiologic anemia, hemoglobin 10–12 mg/dL: Indeed red cell mass is increased, but plasma volume is increased much more dramatically which is commonly called dilutional anemia. That decrease in blood viscosity or "dilutional anemia" is an essential component of maintaining the uteroplacental vascular bed patent.
- Leukocytosis, WBC count 12,000–25,000.
- Slight decrease or unchanged platelet count.
- Fibrinogen doubled at term.
- Pregnancy is a hypercoagulable state with increases in most procoagulant factors and a decrease in some of the natural inhibitors. Factors I, VII, VIII, and XII increase. (Thromboembolic disease is the #1 cause of maternal death in the USA making obstetricians very much quick to utilize anticlotting drugs to manage at-risk patients.). Factors XI and XIII are decreased and factors II and V are unchanged.

- BUN and creatinine decrease as a result of increased glomerular filtration rate.

Gastrointestinal/Endocrine Changes During Pregnancy

- GERD is very common among pregnant women, and almost 40–50% of women will experience it during pregnancy. Most of them will complain of regurgitation rather than heartburn. While GERD prevalence is low during the first trimester (10%), it reaches the maximum in the third trimester (55%).
- Decreased gastric motility and emptying representing an increased risk of aspiration due to progesterone effects: Gastrin secretion increases and motilin secretion decreases.
- Bowel displaced cephalad in the third trimester.
- A normal pregnant woman will remain euthyroid. However, the thyroid gland can be enlarged by 50–70% during pregnancy due to follicular hyperplasia and increased vascularity.
- Pregnancy conveys resistance to insulin due to human placental lactogen.
- Neuraxial analgesia effectively mitigates many of the physiologic changes that can be detrimental to labor. If pain or stress causes maternal hyperventilation, then hypocarbia results with a decrease in uterine blood flow. Hyperventilation also causes a left shift in the maternal oxyhemoglobin dissociation curve decreasing the transfer of oxygen across the placenta [12]. Neuraxial analgesia results in a decrease in maternal oxygen consumption [13].
- Cardiac output increases less in labor with neuraxial analgesia. The decrease in systemic vascular resistance is usually beneficial to preeclamptic parturients and fetuses [14, 15]. Neuraxial analgesia blunts the stress response during labor. The normal increase in maternal circulating norepinephrine and epinephrine levels decreases after neuraxial analgesia [16, 17].
- Neuraxial analgesia may have a beneficial effect on fetal heart rate patterns and be advantageous for the fetus with marginal uteroplacental circulation [18]. Neuraxial analgesia is associated with lower maternal, fetal, and neonatal lactic acid levels [19].

Neuraxial Analgesia and the Progress of Labor

Neuraxial labor analgesia remains a controversial subject regarding the potential to slow the progress of labor and resultant delivery. Investigations have concluded that neuraxial analgesia improves dysfunctional labor [19]. Recently, there has been a focal concern that neuraxial labor analgesia may prolong labor and increase the rate of operative delivery. Some observational studies have loosely associated neuraxial analgesia with prolonged labor and higher rates of instrument and cesarean delivery. There is no clear causal link to any of these findings. Controlling for the variable of early painful labor suggests that independent of neuraxial analgesia, parturients with early pain have a higher incidence of dystocia that would require instrumental deliveries [20]. Cesarean delivery rates differ markedly. Higher cesarean rates are only partially explained by patient characteristics but are greatly influenced by nonmedical factors such as provider density, private insurance, capacity of the local health-care system, and malpractice pressure. Areas with higher usage rates perform the intervention in medically less appropriate populations—that is, relatively healthier births—and do not see improvements in maternal or neonatal mortality [21].

The best available evidence has to be interpreted that neuraxial analgesia given when needed has no significant impact on cesarean section or instrument delivery. A meta-analysis concluded that neuraxial analgesia does not increase the risk of cesarean delivery [22]. As shown by Wong et al. the provision of early analgesia via CSE will decrease total labor time compared to narcotic analgesia [23]. The beta-2 effect of epinephrine is to act as a known myometrial relaxant that could prolong labor and negatively impact the fetus due to increased oxygen consumption if maternal stress or pain responsible for raising epinephrine levels is not attenuated.

Long term, the likelihood of developing postpartum depression is reduced when adequate analgesia is provided intrapartum [24]. Within the 2009 health-care reform package is a concentration on the complications of postpartum depression that will be emphasized as a forward focus for prevention. Cognitive function is also apparently impaired when a woman suffers through labor with inadequate analgesia [25].

Numerous articles have reported on the psychological, cultural, and emotional components of pain. Juhan described the individual's attitude toward pain as the main determinant in the regulation of stress hormones [26]. The idea that sensory experience is shaped by one's attitude and beliefs has become widely accepted [27]. The attitudes of those communicating with the individual who is experiencing pain contribute to the individual's regulation of that pain. Social support, a sense of control and empowerment, and planning are all vital determinants of an individual's experience of pain especially evident when a woman is in labor [28, 29].

Labor Pain Pathways

Labor pain is transmitted from low thoracic, high lumbar, and low sacral nerve roots which are segmentally involved as labor stages change (Fig. 21.2).

First-stage labor pains are related to the physical process of cervical dilation and stretching associated with uterine contractions. By definition, this occurs with the onset of labor and culminates when the cervix is completely dilated. This pain is a nociceptive visceral pain mediated through nerve roots T10–L1. By convention, the classic Friedman curve analysis, which depicts the progress of labor, is composed of a graph that is divided into the latent and active phases of labor. The active phase is subdivided into acceleration, maximum slope, and deceleration. Dysfunctional labor patterns or falling off the curve represent a patient more likely to require frequent anesthetic intervention. Uterine pain fibers combine in the inferior hypogastric plexus and Frankenhauser's ganglia and can be blocked by a paracervical regional technique if the patient is in first-stage labor. The lumbar paravertebral sympathetic chain is also part of this process and conducts sympathetic efferent nociception. The T10–L1 dorsal nerve roots and their connection to the spinothalamic tract are the spinal cord destination for these first-stage nociceptive impulses which are also potentially an analgesic target for paravertebral block.

The second stage of labor is dominantly a somatic pain pathway conducted via the pudendal nerve with contributions from the S2–S4 roots. Second-stage pain most likely also continues with visceral pain associated with continued

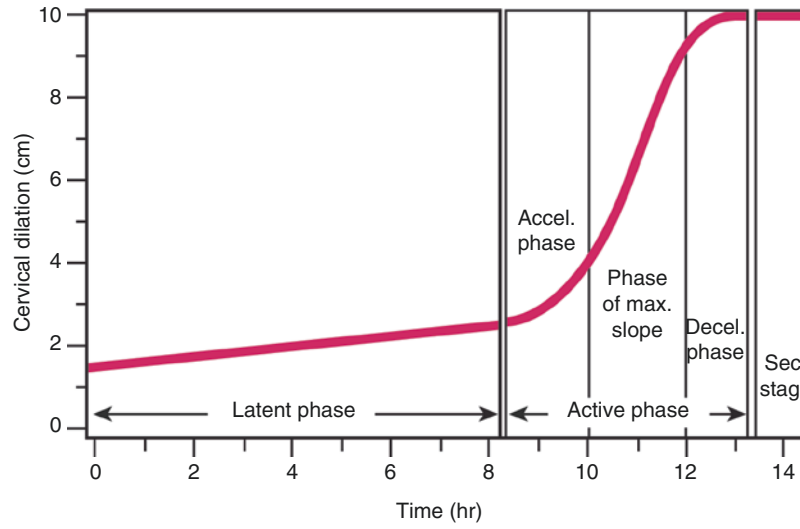

Fig. 21.2 Labor phases

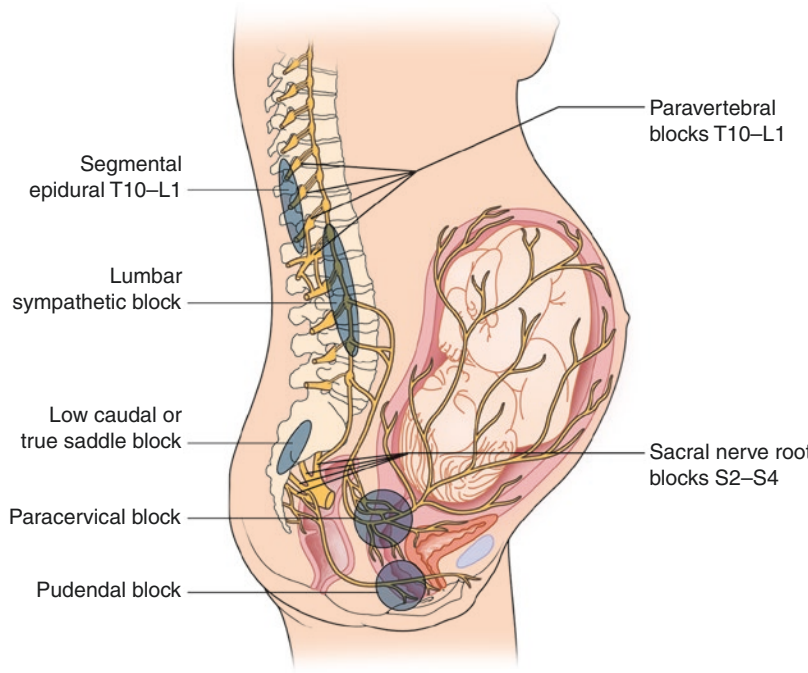

Fig. 21.3 Obstetric block anatomy

uterine contractions and is defined as commencing with complete dilation and terminating with delivery. An alternative block to relieve second-stage labor pain is a pudendal block (Fig. 21.3).

Physiology of Labor Pain

By convention, there are four major steps involved in the production of a pain reaction: (1) transduction, (2) transmission, (3) perception, and (4) modulation. Transduction refers to the peripheral afferent conversion of a stimulus into an action potential. The action potential is then transmitted via C fibers and A-delta fibers along the length of the neurons to cells in the spinal cord, specifically the dorsal horn tract of the spinal cord in Rexed's laminae.

Regional anesthesia functionally targets transduction and transmission when utilizing local anesthetic agents and the addition of narcotic or adjuvants like alpha or serotonin receptor agents can additionally target modulation in the descending pathways where amplification or dampening of the pain signal is thought to occur. Perception is an intricate, multifaceted conscious awareness that is based on very complex interactions of expectations, individual responses to pain or its relief, and communication of these experiences.

Drug Response in Pregnancy

It is commonly recognized that pregnancy impacts dose requirements to provide equivalent anesthesia and analgesia compared to the nonpregnant state due to anatomic and physiologic changes. Increased levels of progesterone and anatomic compression of the epidural space by venous engorgement are reasons long known for less local anesthetic being needed to provide equianalgesic coverage of targeted dermatomes compared to the nonpregnant state. Progesterone has been characterized as a brain anesthetic acting more like a sedative than a gestational hormone and has the potential to be additive with many anesthetic processes [30]. Altered volumes of distribution, changes in protein binding, and increased hepatic and renal blood flow certainly change drug pharmacokinetic behavior in the pregnant state and require fine-tuning the doses administered to achieve the expected result.

Drug diffusion into the CSF from the epidural space may be exaggerated by the increased pres-

sure due to the fetal growth occupying the abdominal cavity. Cephalad spread can be accentuated due to increased lumbar lordosis. Venous engorgement increases the risk for intravascular injection or more rapid uptake to the CNS, heart, or liver.

Highly protein-bound drugs function differently due to the oncotic pressure decrease that is a usual physiologic change of pregnancy resulting in an increased free fraction of circulating drug.

Maternal endorphin and enkephalin levels are also higher in pregnancy and provide natural pain relief and decrease the drug levels needed to provide analgesia in the nonpregnant state.

Altered drug response is more crucial to the fetus because the uteroplacental perfusion is subject to hypotensive perturbation that can have profound fetal effects. Maternal, fetal, and placental issues along with drug pharmacology influence the passage of drugs from maternal circulation to the fetus. Uterine blood flow, molecular size of the drug, ionization, lipophilicity of the drug, pKa of the drug, and maternal/fetal pH are the controllable factors influencing how much drug the baby encounters. Ion trapping as predicted by an acidotic fetal environment is the principal contingency to be aware of when choosing drugs to administer in large quantities such as for an impending cesarean section [31].

Lipophilicity determines the behavior and duration of narcotics administered in the epidural space, and there are functionally no changes when pregnant other than repeating that there is a decreased dose requirement to provide obstetric analgesia equivalent to the nonpregnant state [32]. Amide local anesthetics are metabolized by the liver and excreted by the kidney and have little pharmacologic change in pregnancy. Ester local anesthetics are degraded by pseudocholinesterases, which are decreased during pregnancy but not to a sufficient extent to influence the choice of local anesthetic.

Anesthesia for Labor

Epidurals or combined spinal-epidurals (CSE) offer the best solution for a labor expected to end with vaginal delivery. A probable duration of 12 h in labor is reasonable although highly variable. Currently utilized local anesthetic and narcotic solutions permit neuraxial techniques to achieve a controllable segmental block of sympathetic and sensory nerves with relative motor neural-function sparing. Solutions injected into the epidural space travel cephalad and caudad in the path of least resistance to provide analgesia to the dermatomes of interest [33]. The risk of incompletely or not blocking a painful area is very real and could persist despite all rescue maneuvers and should be discussed prominently in the informed consent process.

The lateral path of local anesthetic spreads through intervertebral foramina to the dural cuff. The local anesthetic can spread further through the dural cuff via arachnoid villa to the CSF. The foramen magnum and sacral foramina represent the cephalad and caudal limits of epidural local anesthetic spread. Epidural block occurs at mixed spinal nerves, dorsal root ganglia, and spinal cord [34].

Epidural or CSE is usually placed at the L2–L3, L3–L4, or L4–L5 interspace which represents a midpoint between the targeted dermatomes for first-stage and second-stage labor relief. These interspaces are usually identified using the intercristal or Tuffier's line to identify the L4 vertebral body and palpating above or below for the most favorable interspace placement. Successful identification is presumptive but remains the best available technique unless ultrasound is available. Unfortunately, when misidentification occurs, the trend is to miss at a higher space than appreciated which could lead to serious morbidity [35–38].

A rule of thumb is that 1–2 mL of epidural local anesthetic per segment to be blocked will be needed to establish the desired level of conduction analgesia. Modifiers such as body habitus or medical status will change the dosing for each individual. Morbid obesity has been shown to cause a dramatic minimum local anesthetic concentration change requiring approximately 40% less infusate to achieve desired anesthesia. Obesity usually results in a 2-dermatomal higher spread from an equal injection volume in a nonobese patient [39]. Divided incremental dosing should always be practiced but is especially emphasized in the obese patient. Obesity is a risk escalator for all morbidities in the pregnant patient.

Absolute and relative contraindications to epidural placement include patient refusal, infection at the site, uncorrected hypovolemia or coagulopathy, significant thrombocytopenia (<75,000), severe aortic or mitral stenosis, local anesthetic allergy, increased ICP, and neurologic disease such as those which are demyelinating and likely to be adversely effected by local anesthetics.

Low-dose local anesthetic in combination with low-dose narcotic solutions provides the most comfort and satisfaction to the patient especially when a PCEA mode is utilized. Bupivacaine and ropivacaine have been the local anesthetics of choice because they have intermediate duration and are principally sensory in their clinical effect. In recent years, many providers have moved away from utilizing epidural bupivacaine for concern of potential systemic local anesthetic toxicity (not seen with ropivacaine). In particular, it is well understood that physiological changes of pregnancy can lead to engorgement of epidural veins and higher risk of potential intravascular injection. With regard to opioids, fentanyl or sufentanil is the analgesic additive with the most clinical use. Minimum local anesthetic concentrations necessary to maintain analgesia in labor have been established as 0.11% for ropivacaine and 0.067% for bupivacaine [40].

CSE can be argued to be the single best choice for labor analgesia since Wong has shown that shorter labor, better analgesia, and no change in operative delivery exist even with early neuraxial analgesia [23]. Earlier work had also suggested that combined spinal-epidural was associated with a 1 cm/h more rapid cervical dilation in nulliparous women [41]. Camian covered the validity of the published studies in detail and provided a careful explanation to account for confounding findings in his review of regional anesthesia and analgesia for labor and delivery [42]. This information continues to be dynamic and as such will not have universal acceptance or interpretation. The clear consensus is that neuraxial anesthesia is not detrimental to labor but is more likely beneficial to safe outcomes and satisfaction.

The optimal recipe for labor analgesia has not been agreed upon and multiple combinations of local anesthetic and narcotic have enjoyed success in the labor suite. Cost and perceived difference in the incidence of side effects are likely to drive the choice of solutions used by any particular practice.

Low-range dosing with either ropivacaine or bupivacaine at 0.625% combined with fentanyl 2–3 mcg/mL or sufentanil 0.3–0.4 mcg/mL has had widespread favorable results as a continuous or PCEA infusion. I utilize a 0.1% ropivacaine plus 2 mcg/mL hydromorphone for anticipated prolonged deliveries and have received very positive patient feedback on its analgesic effect with no evidence of fetal depression.

A systematic review by Halpern and Carvalho concluded that background infusions and larger bolus settings for PCEA resulted in better analgesia. High-volume, dilute local anesthetic solutions of ropivacaine or bupivacaine were the most successful strategy and met more clinical goals related to maternal satisfaction, lack of motor block, and need for clinician rescue intervention [43].

Saddle block for impending delivery is a technique to be considered when delivery is imminent no more than 90 min from placement. Low-dose hyperbaric bupivacaine can provide second-stage labor analgesia utilizing a small spinal needle rather than risking a dural puncture with a larger epidural needle in a patient likely to be a moving target while attempting regional anesthesia. L4–L5 or L5–S1 via a Taylor's approach is an interspace target to consider. Saddle block can also be used to provide immediate analgesia for a parturient unable to cooperate with positioning requests due to her labor pain but is unlikely to deliver before the spinal anesthetic wears off. After the spinal has achieved its desired analgesic effect, then the patient may be positioned more comfortably for epidural placement intended to last the duration of labor. Saddle-block dosing is also employed for cerclage placement for the patient with an incompetent cervix. Dosing for labor analgesia is 2.5–5 mg of bupivacaine with fentanyl 5–10 mcg or sufentanil 2.5–5 mcg added. Successful analgesic dosing requirement is likely to be on the lower end for either local anesthetic or narcotic.

Motor blocking is not desired but is a minimal detriment to pushing in labor since successful pushing is done more by diaphragmatic force than abdominal muscle contraction force [44]. In fact, parturients with spinal cord injury who are unable to sense contractions or are unable to push voluntarily deliver vaginally without difficulty. It does seem that despite its long history in the labor suite coached maternal expulsive efforts do little to speed delivery. Nonetheless, a motor block should be avoided if possible because it can increase the length of the second stage of labor.

Anesthesia for Cesarean Section

Cesarean delivery accounts for at least 31% of the births in the USA and is likely to become a higher percentage in the future due to the litigious risk perceived by obstetricians [45]. Cesarean section is one of the most common major surgical procedures and is estimated at 1 million/year, and accounts for more than 30% of all procedures. An emerging trend is that of the primary elective cesarean delivery for maternal preference independent of maternal or fetal medical need. Is this because the perceived risk of obstetric anesthesia and operative delivery by the patient is so low? ACOG has issued a qualified acceptance of this preference: "Acknowledgment of the importance of patient autonomy and increased patient access to information has prompted more patient-generated requests for surgical interventions not necessarily recommended by their physicians. Decision making in obstetrics and gynecology should be guided by the ethical principles of respect for patient autonomy, beneficence, nonmaleficence, justice, and veracity. Each physician should exercise judgment when determining whether the information presented to the patient is adequate. When working with a patient to make decisions about surgery, it is important for obstetricians and gynecologists to take a broad view of the consequences of surgical treatment and to acknowledge the lack of firm evidence for the benefit of one approach over another when evidence is limited" [46].

The choice of an anesthetic technique for cesarean section depends on maternal, fetal, or obstetrical factors. In general, neuraxial anesthesia is the most common maternal preference due to maternal desires to witness the birth. The trend of whether to offer spinal or epidural for elective cesarean delivery is continuing to favor the use of spinal anesthesia. In 2002, the preference for regional anesthesia for elective cesarean section was 94.9% with spinal anesthesia accounting for 86.6% [47]. Many anesthesia providers think that spinal is a more simple technique which allows for both a rapid administration and onset of surgical block, reduces systemic toxicity, and has an increased density of block which provides better comfort to the patient [48]. A survey of anesthesia providers for cesarean delivery in the USA revealed an 85% preference to administer a spinal, 11% choosing combined spinal-epidural, and 4% choosing epidural [49]. Confidence that there will be no complications and that your colleague obstetrician can complete the operation within the time offered by one-shot spinal is obviously key to choosing this pathway. Indications and contraindications to either spinal, epidural, or combined spinal epidural (CSE) are relative and based on individual clinical presentation.

One meta-analysis suggests that fetal blood gas results favor epidural and general over spinal anesthesia [50]. When hypotension is controlled and phenylephrine is utilized as the vasopressor of choice, then the fetal blood gas is more favorable when utilizing a spinal anesthetic technique [51]. Phenylephrine appears to be the vasopressor of choice to treat hypotension for surgical anesthesia since it has the most demonstrable benefit to fetal oxygenation. From our lab in the dual-perfused, single-isolated cotyledon, human placental model, exposure of the maternal circulation to ephedrine and phenylephrine caused an increase in fetal arterial perfusion pressure, whereas exposure to norepinephrine, epinephrine, and methoxamine did not. The pharmacodynamic mechanisms underlying these differences have yet to be explained. Thus, the clinical implications of the findings are as yet unclear [52]. The best management strategy is clearly to maintain maternal normotension utilizing whatever vasopressor is most effective [53].

Top-up dosing of in situ epidurals for failed trials of labor and general anesthetics for emergency cesarean delivery when medical contraindications to neuraxial anesthesia are present will cover the great majority of techniques needed in the operating room for nonscheduled cesarean operative cases.

Case urgency may be the driving force for choosing a technique, but in even the most extreme emergencies the fallback should always be to that technique which the provider is most comfortable performing. Taking care of the mother and optimizing her physiology is what is best for the fetus and constitutes fetal resuscitation that may buy time in the operating room for more orderly provision of surgical anesthesia. The advocated 30-min declaration to delivery is a soft guideline that is often not achievable, but is a widely accepted community goal despite the lack of clinical evidence showing better fetal outcomes [54]. On a case-by-case basis, all the currently available data should be interpreted with caution, and each declared urgent or emergent delivery accomplished as expeditiously as can be safely done [55]. Approximately 1% or less of cesarean deliveries will be crash sections.

Abnormal fetal heart rate tracings will be the number one cause for rapid transport to the operating room. Placental abruption, cord prolapse, preeclampsia, placenta previa, and failed instrument delivery are probable causes for the urgent declaration [56].

There are many recognized combinations of local anesthetic/opioid for spinal anesthesia to cover cesarean delivery. Unless the provider mistakenly uses an exceptionally large dose of local anesthetic, total spinal is a rare complication (1:10,000), which is modifiable by utilizing hyperbaric preparations to reduce this risk when performing in an emergent setting.

Factors known to impact or suggested to influence the height of the block achieved after injection include the volume of cerebrospinal fluid in the lumbosacral region, vertebral column length, baricity of the solution, volume of the solution, and speed of injection. Patient height and weight have never been strongly linked to influencing the spread of the local anesthetic to the heights desired [57].

Even if a T6 sensory tested level is documented, patients still have a significant risk of visceral pain, which interestingly represents the greatest obstetric patient fear in a survey of preferences to control to optimize their surgical experience [58]. Pruritus, PONV, and other variables ranked much lower than fear of pain. Patients reported a willingness to accept a pain VAS score of 5 in the study design. Surgical anesthesia should not be compromised due to concerns about hypotension and its impact on fetal perfusion. Hypotension is easily treatable and maternal pain that needs rescue may then represent a greater threat to fetal compromise than would have the hypotension immediately before delivery.

The spinal drug of choice is usually hyperbaric bupivacaine in doses between 7.5 and 15 mg. However, in mothers who receive less than 10 mg, there is a 71% risk of intraoperative pain [59]. Bupivacaine administered at doses less than 12.5 mg has not been found to abolish visceral pain [60]. The ED95 providing an effective spinal block of women undergoing elective cesarean section has been calculated at 0.06 mg hyperbaric bupivacaine/cm height [61]. Given that the usual approach will be to combine a local anesthetic with narcotics for spinal cesarean delivery, the ED50 of 7.6 mg hyperbaric bupivacaine and the ED95 of 11.2 mg hyperbaric bupivacaine should be kept in mind, and the ED95 dosing plus narcotic chosen to more reliably provide comfort when the peritoneum is incised, the bladder flap is made, or the uterus is exteriorized. An important clinical investigation advising against the use of doses of intrathecal bupivacaine less than the ED95 has proven to be a good practice strategy to implement. An exception to this guideline would be if the spinal dosing is part of a CSE technique that would allow for extending the block [61].

Continuous spinal anesthesia is a method to provide dense titratable anesthesia for cesarean delivery. Currently, the only method for providing continuous spinal anesthesia is limited to using an epidural needle to identify the intrathecal space and then threading a catheter through that needle to provide spinal anesthesia. This carries an

enhanced risk of a spinal headache from using a large-gauge needle to place the catheter. An ongoing government clinical trial (NCT00990574) with an over-the-needle Wiley catheter is being investigated at Stanford. The stated principal outcome investigation is to assess hypotension occurrence and vasopressor need to treat comparing the continuous Wiley spinal catheter to single-shot spinals at equal dosing. Headache incidence will also be evaluated. From a practical clinical perspective, a continuous spinal technique would seem to have many advantages over either single-shot spinal or epidural anesthesia. Continuous spinal would advantageously allow the development of a denser block compared to an epidural, and also be incrementally dosable which represents an advantage over single-shot spinal dosing. Potentially, it would offer the opportunity to provide multiple doses for postoperative analgesia and utilize lower doses to minimize side effects after each dosing.

Epidural or CSE techniques possibly offer the advantage of less hypotension, which could make them a better choice for urgent operative delivery if maternal-fetal compromise allows time for their placement. Avoiding maternal hypotension greater than 20% of presenting baseline is a reasonable goal. Choosing opioids to add to the intrathecal dose can reduce local anesthetic hypotension but add their own side effect complications. Narcotic-induced side effects include pruritus, nausea, vomiting, and respiratory depression. Even with low dosing of narcotics, it is likely that the more minor side effects associated with intrathecal narcotics will be present. Respiratory depression in clinically relevant dosing is extremely unlikely. The severity of side effects may be dose dependent, but their occurrence seems to be a patient-dependent phenomenon [62]. Clinically relevant intrathecal dosing for morphine is 50–200 mcg, fentanyl range is 10–25 mcg, and sufentanil is 2.5–5 mcg. Increasing narcotic dosing does not improve analgesia nor significantly extend its duration. In this regard, it is more likely to potentiate side effects.

Top-up dosing of an indwelling epidural catheter for a cesarean section can be accomplished rapidly with reported time requirement in the range of 3–14 min independent of prior sensory levels established for the trial of labor. Proven epidurals can be rapidly dosed incrementally with little fear of toxicity or respiratory compromise. Lidocaine and chloroprocaine are the favored agents for fast onset, but bupivacaine, ropivacaine, and combinations of agents have all been reported to have successfully topped up an existing epidural catheter to surgical levels quickly [63–66]. Currently, the expected failure to achieve adequate surgical anesthesia with a labor epidural catheter is less than 5%, but that would mean approximately 1:20 parturients going for urgent cesarean delivery from labor could be expected to need conversion to general anesthesia or deep MAC supplementation [67]. This emphasizes the need to continually prove the analgesic efficacy of a labor catheter and to have multidisciplinary communication if there is perceived escalating risk of fetal compromise that would require urgent operative delivery. Delay in instituting anesthetic care and failure to communicate were standout reasons for obstetric anesthesiologist closed-claim liabilities [68].

Epidural catheter alone for an elective cesarean section is an excellent option in the morbidly obese or massively morbidly obese patient because the sitting can be made higher in the lumbar spine where bony landmarks are more easily palpated and can be dosed slowly to mitigate the risks associated with neuraxial anesthesia in obese patients.

Combined spinal-epidural is an excellent choice for nonurgent and selected urgent presentations for cesarean delivery. Small spinal needles passing through a larger epidural needle that functions as a long introducer make this a popular choice for identifying the target rapidly in an obese patient. The rapid benefits of spinal anesthesia with the security of an epidural catheter and low risk for postoperative spinal headache represent the rationale most often given for choosing this anesthetic method. However, the currently accepted expectation for failure to obtain cerebrospinal fluid when attempting a CSE approximates 10%. Known causes for this phenomenon include inadequate needle length to

Table 21.1 Tabular guide for which technique to consider utilizing

Epidural	CSE	Spinal
Routine labor with anticipated imminent delivery	Urgent delivery vaginal delivery	Advanced dilation or saddle-block station
Morbidly obese pt. for C/S	Scoliosis	Routine C/S
Multiple prior C/S, therefore anticipated long case	Harrington rods or other spinal surgery	Continuous catheter for morbid obesity
Special considerations: L2–L3 catheter sitting for early or disproportionate pain which suggests possible C/S for dystocia	Prior failed epidural analgesia for trial of labor	Impending instrument delivery; supplemental for existing block
Site L3–L4 for more optimal development of comfort for first- and second-stage labor		

penetrate the dura and lateral epidural space identification making it hard to position the port of the spinal needle in the subarachnoid space. There will also be a time lag to be able to prove that the epidural catheter can functionally extend the block until the initial spinal dosing has worn off (Table 21.1).

Adjuvant Drugs Used for Neuraxial Anesthesia

Adjuvant drugs are used for different purposes, either to prolong postoperative anesthesia/analgesia, to improve the quality of intraoperative anesthesia, or to reduce the dose of local anesthetics which therefore decreases their side effects. Multiple adjuvants such as dextrose, opioids, and epinephrine have been successfully used. However, newer adjuvants have been added to the list such as neostigmine and clonidine. Those medications are undergoing clinical investigations. Spinal and epidural clonidine, 60–200 μg, can decrease shivering and any additional opioid requirement for post-cesarean section pain control. However, because of opioid association with sedation and hypotension, it is not yet FDA approved for intraoperative analgesia.

While neostigmine (spinal and epidural) can improve analgesia after cesarean section, it is associated with high risk of nausea following spinal administration as well as intraoperative shivering and sedation following an epidural injection. Therefore neostigmine is not FDA approved as an adjuvant for neuraxial anesthesia.

Adjuvant and Alternative OB Blocks

Paracervical block is essentially reserved for obstetricians and is unlikely to be performed due to the high incidence of fetal compromise reported even when the block is performed well. Paracervical block is an excellent intervention reserved for procedures like a dilation and curettage when there is a fetal demise. A field infiltration with local anesthetic via a spinal needle at the 4–5 o'clock and 7–8 o'clock positions where the cervix joins the vagina is all that is required. Appreciation of the correct tissue plane is difficult even when there is no presenting fetal head. Multiple reports of bad outcomes due to local anesthetic toxicity in a delivering fetus make this a very rare choice for analgesic intervention. This is not a block likely to be encountered and can only provide limited analgesia for the first stage of labor [69].

A pudendal block is also more likely to be performed by an obstetrician since the most successful block technique requires working within the vaginal cavity. This block would provide analgesia for the second stage of labor, but the usual clinical conditions of a patient unable to cooperate to facilitate the procedure make its use very rare. Palpation of the ischial spines and passing a spinal needle posterior to the bony process while requesting the writhing patient to be still make this a truly tough field block to accomplish. Vascular injury or injections with resultant local anesthetic toxicity are the most common complications noted in performing

this block [70]. Transperineal approach to the space is possible but adjacent vascular structures are again a high risk to injure or inject, and the success is low such that the risk-benefit ratio is unacceptable to offer to the patient for comfort. Saddle-block anesthesia is much easier to perform and far more likely to be successful.

Paravertebral blocks do not have much utility since they are painful to perform and can only treat first-stage labor pain. The potential to place a catheter in the paravertebral space could increase their potential analgesic potential and reduce the number of needle sticks necessary to cover the T10–L1 dermatomes. Identification of the paravertebral space is done by walking a needle off the transverse process of the selected vertebral segment (usually L2), injecting local anesthetic just anterior to the medial attachment of the psoas muscle bathing the exiting spinal nerve and the nearby sympathetic chain. Catheters can be placed in this anatomic space and a typical epidural solution infused to provide labor analgesia. The probability and predictability of success with this technique are less than those of an epidural and probably it has little use as an alternative to epidural placement for anatomic abnormalities that might make an epidural placement difficult. Known common complications with this technique include painful placement and hypotension. Rare complications such as high spinal and retroperitoneal hematoma are known to have occurred [71].

Utilization of regional anesthetic techniques other than a central neuraxial approach to provide comfort to parturients is gaining interest. Since the 2001 description of a transverse abdominis plane or TAP block technique by Rafi and succeeding descriptions of "how to" approaches for doing a TAP block [72, 73], multiple reports analyzing the utility of doing said block have populated the literature [74–76]. As might be expected, there exists disagreement as to how successful and therefore applicable the technique can be, but it seems to be gaining popularity because of how simple the block is to perform. TAP block functions like a distal intercostal block (ICB). The history of ICB in providing postoperative analgesia for the abdomen is long [76, 77]. For each potential case, the question of how to perform the block and what to utilize when performing the block will be an individual decision. The greatest potential complication is the risk of local anesthetic toxicity whether it is systemic overdosing or unintended intravascular injection [78].

As the body of evidence mounts, it has become clear that TAP blocks can indeed be an excellent adjuvant for postoperative analgesic provision. Spinal and epidural anesthesia will remain the gold standards for providing obstetric anesthesia and analgesia, but having command of the ability to provide further analgesia or an alternative to traditional methods is useful when the need arises.

TAP blocks are the most intriguing alternative regional anesthetic technique being utilized for post-cesarean analgesia or operative bring backs for wound complications in the lower abdomen which is the usual obstetric approach to the hysterotomy required for cesarean delivery.

The interest in utilizing truncal blocks to provide prolonged analgesia post-cesarean delivery has become a focal topic of interest among obstetric anesthesia providers. The potential to be a long-acting analgesic adjuvant absent side effects like pruritus, nausea, hypotension, or fetal drug accumulation makes the block attractive. The list for known side effects related to using neuraxial anesthesia to provide postoperative analgesia is extensive and especially common when long-acting duramorph is utilized in the intraspinal space.

The TAP block can be performed more predictably with ultrasound, which would obviously be the safest and most predictable way to administer the block. Utilizing TAP blocks to target upper thoracoabdominal surgeries for post-analgesia is reported [79]. The TAP block can be performed without ultrasound and has a proven, repeatable success rate once the learning curve and adequate numbers have been performed using the double fascial click palpation technique.

The block will have limited application since the anticipated dermatomal distribution for the block seems to be T10 to L1 when utilizing

perceived safe volumes of local anesthetic of choice [80, 81]. Further, it has to be emphasized that a TAP block will be limited to somatic relief only and not address visceral pain typical in postoperative obstetric cases. A specific investigation into post-cesarean analgesia with the TAP block did yield promising results [74]. The TAP block has proven success in gynecologic, urologic, and bowel surgery, which involves an incision in the anterior abdominal wall in the dermatomes suggested to be covered by the block.

The triangle of Petit or the lumbar triangle is the anatomic identifier that marks the entry point for the needle to access the fascial plane between the internal oblique muscle and the transversus abdominis muscle. The superior aspect of the iliac crest serves as the origin for the latissimus dorsi muscle that marks the posterior-lateral border of the triangle while the external oblique aponeurosis inserts on the anterior half of the iliac crest marking the anterior-medial border of the triangle. The clinical appreciation of the true cephalad extent of the iliac crest is important and often more difficult to appreciate than expected when a large pannus is present that hides the correct insertion point without ultrasound guidance. The anterior superior iliac spine is often more easily palpable and could be a confounder for trying to identify the appropriate needle entry point. If the morphology of the patient is a challenge, it may be easier to identify the latissimus dorsi muscle and walk the palpating hand down to the iliac crest by taking advantage of the ability to identify and isolate the latissimus muscle. Take advantage of the insertion of the latissimus on the humerus and have the patient extend or medially rotate the humerus to isolate and definitively identify the latissimus dorsi connection to the iliac crest.

The needle of choice will be a blunt bevel whether using ultrasound-guided assistance or the double-click technique. Standard epidural 17-gauge or 18-gauge Tuohy needles with centimeter markings or a B bevel regional needle (typically a 22-gauge needle) is acceptable. Flexing the OR table while the patient is supine will make the iliac crest more prominent. Airplaning the table away from the intended block side will also make the target zone more appreciable. When using the double-pop technique, the needle is advanced perpendicular to the coronal plane encountering a more subtle external oblique pop and then a more resistant internal oblique pop which is the stopping point for needle advancement and local anesthetic injection after aspiration. The anticipated resistance to injection should be similar to that when injecting an epidural catheter; if there is more resistance to injection than anticipated the probability is that the needle has advanced into the transversus abdominis muscle. Withdraw slightly, aspirate, and repeat the injection trial. If the needle is truly in the intended plane, then the resistance will be very minimal to injection.

Utilizing ultrasound, the needle entry point will not be as important because the three muscles of interest are so clear sonographically. Orienting the probe to obtain in plane real-time images documenting needle advancement can be easily accomplished by placing the probe transversely in the triangle of Petit. Appreciation of the fascial pops as the needle advances serves as a double reassurance when performing the block.

My preference for local anesthetic is 20 mL of ropivacaine 0.5% with 2 mg dexamethasone in each hemiabdomen. If an epinephrine-containing local anesthetic solution is desired for nonvascular confirmation, then an easy approach is to dilute equal volumes of 2% lidocaine with 1:200,000 epinephrine commonly available to most obstetric anesthesia providers with the ropivacaine. This creates a 1:400,000 epinephrine-combined ropivacaine-lidocaine solution. Adding fresh epinephrine is the best solution, but the addition of 2% lidocaine with epinephrine to ropivacaine or bupivacaine is a quick and easy pharmacologic solution available to almost all practitioners.

The biggest concern for performing a TAP block especially a bilateral TAP block will be safe dosing of the local anesthetic injectate. The potential for complication in a pregnant patient is heightened due to systemic vascular engorgement, which could result in accidental intravenous injection and the typical local anesthetic toxicity manifestations. The concept of test dosing as is done in

epidural placement with an epinephrine-containing solution is a good practice habit that could detect an intravascular sitting before a full-dose single-shot injection is performed.

Cumulative maximum safe dosing for single-shot injections is an easy calculation and can be done by classic methods that then allow the practitioner to choose agent, concentration, and volume. It can be expected that a 15–20 mL injection will be needed for each injection to achieve the dermatomal coverage targeted. The costal margin, iliac crest, and lateral border of the rectus will be the anatomical sites limiting spread. An excellent anatomic review by Rozen and colleagues found that only T9–L1 nerves were found in the midaxillary line below the costal margin to the inguinal ligament. Circumferential and cephalad spread are approximately equal and cannot be overcome with volume since the plane is permeable and leaky into spaces adjacent, therefore missing the targeted nerves to be blocked.

Indeed, because ventral rami and segmental nerves branch extensively and communicate widely with adjacent nerves a single-shot TAP block is effectively a plexus block that is easily accomplished.

Complications

The mother and her fetus typically represent 120 life years when complications are litigated. Familiarity with the potential for harm and how to mitigate those consequences is the next best thing to preventing them.

Approximately 2.4 million epidurals per year are performed to provide labor analgesia in the USA. Currently, in the USA, approximately 60% of laboring women receive epidurals for intrapartum pain relief [82]. Accurate risk estimates for hematoma can be stated as 1 in 168,000; deep epidural infection, 1 in 145,000; persistent neurologic injury, 1 in 240,000; and transient neurologic injury, 1 in 6700. Meta-analysis shows that these risk incidences have improved compared to earlier reports because the data available has improved and can be more accurately quoted to the patient [83].

High Block

A high block is an iatrogenic event that requires early recognition and rapid management to protect the patient. A clinical setting in which this is likely to occur is the patient with a functioning epidural who has failed a trial of labor and has gone to the operating room for cesarean delivery. Despite large-volume dosing of local anesthetic, the epidural block will not rise to a sufficient level for surgical anesthesia. The determination to expedite the case and perform a spinal to provide a denser and higher block is made. What determines the dermatomal height achieved after spinal injection is inconclusive, but there is good evidence to suggest that the volume of lumbosacral CSF is a determinant of how high an intrathecal injection will rise, and in this case the volume is diminished due to epidural fluid mass effect. By virtue of this compression, the likelihood of high spinal is increased even when using reduced dosing or usual spinal dosing. Mass effect plus the fact that sodium channels are occupied from epidural infusion that has crossed into the CSF make it very easy to overdose and get a high spinal in this setting [84]. The ED50 and ED95 of intrathecal isobaric bupivacaine are 7.25 and 13.0 mg [85], which can serve as guides for dosing or if the provider follows the recommendations of Norris [86] to not modify the dose of bupivacaine and to use a full 15 mg dose then the risk for high spinal is probably increased. It is an unsolved dilemma as to how much dosing is required in a patient with an inadequate but demonstrable level whether it was from epidural or intrathecal cause.

Patient awareness of ascending block precedes dramatic changes in blood pressure, heart rate, EKG rhythm, or pulse oximetry which is often expressed by the patient as a sense of impending doom or fear. This may represent medullary hypoperfusion. Aggressively treat hypotension and hypoxemia and assess block height continuously while being ready to secure the airway by intubation. For hyperbaric solutions, the anatomic hindrance to continued cephalad spread is the T5 or T6 point of maximum kyphosis. If this barrier has been breached then

the cephalad spread is rapid and the consequences are overwhelming. Airway compromise and hypotension should be the focal points to guide treatment.

Sedation to provide anxiolysis is likely to compromise further an already at-risk airway, so it is advisable to be very judicious in choosing this pathway if a patient has not yet lost their airway. Checking handgrip and changes in phonation is the method to assess the height of the spinal block. Be cautious when administering benzodiazepines to a weak patient who has not yet crossed the threshold for intubation. Do not obtund the patient unless inducing the patient for intubation. High spinals have been shown to decrease the sensitivity to midazolam meaning much less goes much further toward creating respiratory embarrassment [87].

Pulmonary Aspiration

The risk of pulmonary aspiration is a constant for any pregnant patient especially those who present emergently for stat cesarean delivery requiring general anesthesia. After 16 weeks of amenorrhea, all obstetric patients are at increased risk for pulmonary aspiration [88]. The risk of aspiration during high spinal is also very real and less so for laboring patients or patients presenting for nonurgent cesarean delivery. Even if pharmacologic prophylaxis may not have sufficient time to be optimized in such a setting, it should be insisted upon being given. Protecting the mother protects the fetus at risk and any step needed to optimize maternal safety cannot be compromised. Administration of nonparticulate antacid, metoclopramide, and an H2 blocker would be the preferred regimen for a patient known to be at high risk for aspiration. An assistant skilled at providing cricoid pressure should be a part of the team preparedness for such an emergency. Difficult intubations also increase the risk for aspiration. Studies show that the incidence of difficult intubations is increased in pregnant patients. Difficult intubation is 1 in 30 pregnant patients. The impossible intubation is 1 per 280 in obstetrics which is 8 times greater than the general population [88]. Extubation represents another risk point for aspiration that can be mitigated by using an OG or NG tube to suction the stomach.

Hypotension

Hypotension is rarely a significant medical risk to the parturient unless she has a medical comorbidity that cannot be compromised by hypotension such as valvular heart disease. The sympathectomy that characterizes successful regional anesthetic placement can be anticipated and appropriate interventions can be taken to reduce the degree of hypotension resultant. These measures can include preloading or more appropriately coloading with fluids as the block is established. Recent studies have shown that a preloading mixture of 500 mL 6% hydroxyethyl starch (HES) 130/0.4 + 500 mL Ringer's lactate (RL) significantly reduces hypotension associated with sympathetic blockade [89]. Aggressive administration of vasoactive drugs to lessen the hypotensive effect is also an effective measure. Phenylephrine is now accepted for routine obstetric intervention and should be the vasopressor of choice for blocks below the T10 level [90]. Ephedrine is still useful and is probably a clinically better drug to treat hypotension as the block progresses cephalad above T10. Maternal heart rate may influence the choice between phenylephrine and ephedrine. Drops in maternal MAP represent a greater risk to the fetus because the driving force for uteroplacental perfusion is maternal MAP. A good clinical strategy is to identify the fetus at potential risk and intervene aggressively if decelerations are noted after neuraxial placement that represents a change from FHR baseline. If hypotension is absent, but fetal decelerations are significant, then the suspicion should be that a hypertonic uterus is responsible. This is common after a CSE technique when rapid analgesia has resulted from intrathecal narcotic injection, but the maternal baseline pressure has not changed. Tocolytic treatment with terbutaline or nitroglycerin rather than urgent transport to the operating room should effectively remedy the deceleration problem.

Local Anesthetic Toxicity

A consensus statement has been issued by the ASRA practice advisory on local anesthetic systemic toxicity, which summarizes the diagnosis and treatment of this complication [91]. The incidence of local anesthetic systemic toxicity is low and occurs in approximately 1 in 12,688 patients undergoing regional anesthesia [92]. Classic signs and symptoms of local anesthetic toxicity include but are not limited to auditory changes, circumoral numbness, metallic taste, and agitation, which can lead to seizures. Local anesthetic CNS toxicity occurs before cardiac toxicity, which manifests as arrhythmia and is a function of potency. No single intervention has been identified which eliminates the risk of local anesthetic toxicity, but early detection of intravascular dosing remains the focus. Minimizing the amount of local anesthetic needed to achieve the desired clinical effect is emphasized. Atypical presentations of local anesthetic toxicity occur in approximately 40% of reported cases. Provider vigilance to variability is crucial. Incremental dosing is always the correct dosing and adding an intravascular marker is another step to reduce the risk of complication that should be routinely used.

Treatment of local anesthetic toxicity should focus on preventing airway compromise and hypoxemia which potentiate the complication. Compared to lidocaine, bupivacaine is more dysrhythmic and more resistant to resuscitation. For small-dose inadvertent intravascular injection, the fact that pregnancy increases hepatic blood flow and increases amide clearance is beneficial. Benzodiazepines are the drugs of choice for halting seizures; if a benzodiazepine is not available, then propofol can be used. Early use of 20% lipid emulsion accelerates the removal of bupivacaine from the circulation [93]. Recommended bolus dosing is 1.5 mL/kg over 1 min repeated up to three times at 3-min intervals, and then titrate to effect with an expected dosing of 0.25 mL/kg/min. The below figure shows the guidelines for managing local anesthetic systemic toxicity with lipid emulsion injection published by AAGBI in 2010.

If these measures fail, then CPR should be begun and having cardiopulmonary bypass should be made available. Given enough time, the metabolism of the local anesthetic will occur, but aggressive life support will be needed to buy that time (Table 21.2).

Chloroprocaine is not included above because of the safety profile associated with esters. Esters have low toxic potential but are much more likely to generate an allergic reaction because of their linkage and the release of a PABA-like molecule after hydrolysis. Esters are not of themselves immunogenic, but the metabolites of esters are much more potentially allergenic than are the metabolites of amide-linked local anesthetic. The metabolites of many drugs are truly the culprits causing adverse reactions or unwanted side effects that all anesthesia providers fear, and this is indeed the cause for reported ester local anesthetic allergic potential.

Spinal Headache

Postdural puncture headache is a risk that has a long and continued history in regional anesthesia. Usual risk quotations for epidural and spinal

Table 21.2 Local anesthetic drug information

Drug	Lidocaine	Prilocaine	Bupivacaine	Levobupivacaine	Ropivacaine
Description	Amide	Amide	Amide	Amide	Amide
Relative potency	2	2	8	8	6
Onset (min)	5–10	5–10	10–15	10–15	10–15
Duration without epinephrine (h)	1–2	1–2	3–12	3–12	3–12
Duration with epinephrine (h)	2–4	2–4	4–12	4–12	4–12
Max dose without epinephrine (mg/kg)	3	6	2	2.5[a]	3[a]
Max dose with epinephrine (mg/kg)	7	9	2.5	3[a]	4[a]

[a]Indicates probable safe maximum dose (insufficient data)

approaches resulting in a headache are 1–2%. It has been established that the dura is not a watertight meningeal layer and is therefore not truly the violated layer responsible for CSF leak causing a headache. More appropriately, it can be referred to as a meningeal puncture headache because the arachnoid layer must be breached to cause the headache [94]. Unfortunately, iatrogenic headaches from accidental dural puncture represent the most frequent temporary claim for litigated injury, which is why it should rank high on the list for risks in the informed consent process.

An accidental dural puncture during epidural anesthesia may cause severe postdural puncture headache. A systematic meta-analysis was performed aiming to identify anesthetic techniques that may reduce the incidence of accidental dural puncture while administering epidural blocks. The study was unable to give recommendations on the best technique to use while performing epidurals. No significant difference was found in the incidence of accidental dural puncture for combined spinal-epidural analgesia, maternal position, type of the catheter, needle size, bevel direction, operator experience, or use of ultrasound. No concrete recommendation was made on a specific technique to reduce ADP. They concluded that clinicians should focus on measures to prevent or treat PDPH once ADP has occurred [95].

Why CSF loss generates a headache is controversial and probably multifactorial. Loss of intracranial support creating brain sag and compensatory vasodilatation by cerebral vessels are thought to be the causes for cephalgia. The clinical picture is that the headache is postural, generally occurring 12–48 h after an epidural or a spinal, and is a bilateral headache that can be frontal, occipital, or both. A headache that is exacerbated with 15 min of assuming an upright position and improved within 15 min of lying down is a meningeal puncture headache. Of the meningeal puncture headaches, 67% are severe, 23% are moderate, and 11% are mild [96]. Recent studies have shown that BMI may be inversely related to the incidence of postdural puncture headache, meaning there is a higher incidence of PDPH in obstetric patients of normal BMI and lower incidence in patients with an increased BMI [95].

If doubt exists about the cause for a postdelivery headache after regional anesthesia, then more extensive workup including radiologic evaluation or neurological consultation to rule out potential serious etiologies like dural venous sinus thrombosis, subdural hematoma, subarachnoid hemorrhage, or preeclampsia must be done before further treatment.

Treatment options include conservative management and therapies focused on supportive comfort including utilizing pharmacology approaches that yield more success when using models for treating vascular or migraine headaches. The definitive treatment for meningeal puncture headache is still a blood patch and should be offered as soon as conservative management fails to provide patient relief. Epidural blood patch is thought to be an effective treatment for PDPH, but there is insufficient evidence to support its use as a prophylactic procedure [97]. When a wet tap happens, the option of placing an intrathecal catheter and leaving it in situ for 24 h has been shown to decrease the incidence of PDPH. This adds an increased risk of infection and is not a universally accepted clinical practice. Further studies are needed to help determine the best treatment of patients experiencing postdural puncture headache after an accidental dural puncture during epidural administration [98]. Minidose duramorph through the indwelling catheter before removal has been used which may only delay the onset of a headache but has an analgesic effect. Dosing with preservative-free saline has also been tried with an upper limit of 20 mL or stopping if the patient complains of paresthesia, tinnitus, or uncomfortable pressure in her head.

Neurologic Complication

Neurologic complications are usually short and transient but can be more sinister and difficult to diagnose when persistent. Circumspect technique and vigilant adherence to meticulous preparation

will reduce the chance of complication, but will not abolish it. Serious neurologic complications from regional anesthesia techniques have a lower incidence in the obstetric population than in the general population. Major complications are rare and can be divided into damage caused by the needle, damage caused by the catheter, or complication caused by technique.

The estimated frequency for direct nerve damage is 1–10,000 to 1–30,000 and can be caused by needle or catheter placement. There is nothing to offer as treatment and the duration of time needed to appreciate improvement is 1–6 months. Direct trauma to nervous tissue may occur at the level of the spinal cord, nerve root, or peripheral nerve. Paresthesia or pain during sitting or injection should be respected, and the process stopped immediately. Two-thirds of anesthesia-related neurological complications are associated with either paresthesia or pain during injection.

The complication of hemorrhagic complication is estimated to be a frequency greater than 1:150,000 for epidural placement and 1:220,000 or greater for spinal placement. Neurologic dysfunction that results can be catastrophic requiring immediate recognition and treatment to avoid permanent paraplegia. The cause can be needle or catheter placement and has been suggested to happen more often with epidural catheter removal than with placement. Anticoagulation therapy increases the risk of hematoma as does the progressive thrombocytopenia typical in preeclampsia, eclampsia, or HELLP patients. The symptoms of epidural hematoma are bilateral leg weakness, urinary incontinence, and loss of rectal sphincter tone. These severe neurologic deficits may be preceded by sharp pain in the back or legs. Prolonged motor paralysis without regression warrants workup. Stat MRI is the radiologic exam of choice to identify potential epidural hematoma with neurosurgical notification of the potential problem as soon as possible. Symptomatic epidural hematoma must be decompressed surgically as rapidly as possible to facilitate recovery. ASRA issued its Third Consensus Statement that serves as a model to guide practice standards if regional anesthesia is perceived to be a risk due to antithrombotic or thrombolytic therapy. Understanding that this is a collection of observations and experiences of many experts, and then if any particular drug or class of drugs is encountered in a parturient, the recommended waiting times for placement and safest time to remove a catheter should be sought in this reference [99]. Below is a table showing commonly used anticoagulants and the recommended waiting time after their administration before performing a neuraxial procedure.

Thrombocytopenia can be a risk factor for hematoma formation during epidural and spinal anesthesia. Obstetric patients can come in with thrombocytopenia-associated HELLP syndrome or gestational thrombocytopenia. In these instances, thromboelastography can be used to assess the ability of the patient to form clots adequately. The use of thromboelastography (TEG) in patients at risk of bleeding has been recommended, if available, as an option to traditional laboratory testing [9]. The thromboelastograph (TEG) measures viscoelastic properties of clot formation and dissolution (platelet function, coagulation, fibrinogen-platelet interaction, and fibrinolysis). TEG has shown that many obstetric patients who are thrombocytopenic are still hypercoagulable due to pregnancy changes. As a result, the TEG has shown that neuraxial block in patients with low platelet counts can be performed without complication [100].

Epidural abscess is very rare, approximately 1 in 500,000 in the obstetric population. It is usually due to hematogenous seeding of the epidural space and *Staphylococcus aureus* is the most common organism causing epidural abscesses [100]. Obstetric anesthesia providers should use hat, mask, and gown to decrease the risk of inoculating the patient. Oropharyngeal secretions are known to be potential sources for particularly bad infections. Presentation is usually within days of placement but commonly occur about 1 week out because that amount of time must pass to allow for the pressure of the developing abscess to be sensed. Symptoms include fever, malaise, headache, and back pain. Palpation of the site or adjacent paraspinal areas will reveal focal pain. White blood cell count will be elevated. Pyrexia is usually

present. Neurologic deficits will progress if the spinal cord is compressed and may manifest as lower extremity pain, weakness, bowel and bladder dysfunction, and paraplegia. Urgent CT scan or MRI with gadolinium should be done to confirm the diagnosis of epidural abscess [100]. Surgical intervention and a long course of appropriate antibiotics represent the best treatment [101, 102].

The spinal cord, nerve roots, or peripheral nerves are vulnerable to needle or catheter injury. Most anesthetic-related neurological complications have the harbinger of paresthesia or pain upon placement or injection [103]. Resultant deficits are usually detectable within 24–48 h and spinal anesthesia is three times more likely to result in neurologic injury or radiculopathic injury compared to epidural anesthesia. Spinal administration has an added risk of resultant neurologic injury without paresthetic warning being noted [104]. Single nerve root neuropathy is rare with approximating 1:10,000 incidence and more likely to resolve completely and more quickly than spinal cord, plexus, or polyneuropathy injuries [105, 106].

Epidural catheters rarely may break or shear but should be left indwelling unless increasing neurologic symptoms are expressed or a compromise in daily activities occurs. The epidural space should accommodate the presence of a benign catheter with less risk for complication than would be suffered by the patient if surgical retrieval were made.

Parturients who do not receive regional anesthesia frequently experience compression nerve injury. The reported incidence of permanent neurological deficits is as high as 1:2100 deliveries [107]. Factors that increase risk are prolonged labor, maternal positioning during delivery, fetal presentation, fetal size, and instrument delivery. The lumbosacral plexus is most likely to be involved especially the lateral femoral cutaneous, femoral, and obturator nerves. The peroneal nerve is vulnerable to injury if prolonged or poorly positioned legs in stirrups occur during delivery. These nerve injuries are probably ischemic in origin and will usually be transient but may persist for as long as 6 weeks postdelivery [108].

Postpartum Back Pain

Backache postdelivery is common and is independent of whether an epidural was chosen for analgesia. One-third to one-half of women will experience back pain more pronounced after delivery than before becoming pregnant. Epidural analgesia has been clearly shown not to be a contributing risk. Yet, recent studies show that patients undergoing cesarean delivery with epidural anesthesia are at increased risk of developing lower back pain compared with patients undergoing vaginal delivery. These studies also suggest that age and urinary tract infections are factors causing postpartum back pain [109]. Suggested causes for long-term backache postpartum include a change in pelvic tilt, ligamentous relaxation causing spinal anatomy changes due to the release of the hormone relaxin at delivery, musculoskeletal injury or stretch not appreciable due to analgesia or excitement of delivery, and more. It is now clear that the use of epidural analgesia is not a direct cause of postpartum backache nor does it modify the risk of developing backache [110, 111]. Can an obstetric epidural cause adhesive arachnoiditis has been asked and functionally answered in the negative. Adhesive arachnoiditis is particularly painful and debilitating, and an extremely rare potential complication of obstetrical epidurals and the infusions commonly run in them [112].

High-Risk Anesthetic Patients

The three leading causes of maternal mortality are hemorrhage, thromboembolic disease, and preeclampsia. Maternal mortality has recently increased across the globe although anesthetic deaths are declining in prevalence having fallen to the seventh leading cause of maternal deaths [113, 114]. Maternal mortality from anesthesia is approximately 1 out of 1 million parturients but is increased to 6 out of 1 million parturients undergoing caesarean delivery [115]. General anesthesia and associated airway loss complications represent the greatest threat to life for anesthetic-caused maternal death. Being able to predict, prepare, and handle the difficult airway in obstetrics cannot be overemphasized [116].

Worldwide, the quotation is that one parturient per minute dies which is true by actuarial analysis of reported data. Life-threatening maternal etiologies can be expected to be encountered in any obstetric anesthesia practice whether in a small community or large tertiary care hospital. Hypertensive diseases of pregnancy are increasing in frequency, and because of high-risk medical management those afflicted are successfully carrying a pregnancy to further gestations compatible with viable delivery feasible at approximately 24 weeks of gestation. Obesity, advanced maternal age, black race, cesarean delivery, and multiple pregnancies are all factors that increase the risk of maternal morbidity and mortality [115].

Hypertensive Disorders of Pregnancy

The overall incidence of hypertensive disorders in pregnancy approximates 6%. Eclampsia is reported at 1–2 per 1000 deliveries. Women with preeclampsia and eclampsia have a 3- to 25-fold increased risk of severe complications, such as abruptio placentae, thrombocytopenia, disseminated intravascular coagulation, pulmonary edema, and aspiration pneumonia. More than half of the women with preeclampsia and eclampsia require a cesarean delivery. African-American women not only have a higher incidence of hypertensive disorders in pregnancy but also tended to have a greater risk for most severe complications. Preeclamptic and eclamptic women younger than 20 years or older than 35 years have substantially higher morbidity [117].

Pregnancy is a thrombogenic condition, and pulmonary embolism tends to be considered a later pregnancy problem. However, symptoms suggestive of pulmonary embolism need to be taken seriously and treated and investigated at any stage of pregnancy. The potential for thromboembolic complications has introduced the relatively common usage of low-molecular-weight heparins for thromboprophylaxis, which carries the potential to be a strong contraindication to using neuraxial anesthesia if the last dose administered falls within the recognized time frame for unacceptable intraspinal bleeding.

Hypertensive disorders of pregnancy have defied modeling that can be agreed upon as representing disease course. Terminal end points being the most severe disease manifestation are usually recognized as acute fatty liver syndrome and eclampsia, but whether these end points are on a continuum including preeclampsia as a common pathway is debatable.

Preeclampsia morbidity and mortality are related to systemic endothelial dysfunction, vasospasm, and small-vessel thrombosis leading to tissue and organ ischemia. Possible organ involvement includes CNS events such as seizures, strokes, or hemorrhage; renal tubular necrosis; hepatic coagulopathies; and placental abruption in the mother. Each of these complications can precipitate a request for urgent delivery.

Probable Predictors for OB Requested Urgent Delivery

Preeclampsia

Blood pressure: 140 mmHg or higher systolic or 90 mmHg or higher diastolic after 20 weeks of gestation in a woman with previously normal blood pressure

Proteinuria: 0.3 g or more of protein in a 24-h urine collection (usually corresponds with 1+ or greater on a urine dipstick test)

Severe preeclampsia

Blood pressure: 160 mmHg or higher systolic or 110 mmHg or higher diastolic on two occasions at least 6 h apart in a woman on bed rest

Proteinuria: 5 g or more of protein in a 24-h urine collection or 3+ or greater on urine dipstick testing of two random urine samples collected at least 4 h apart

Other features: Oliguria (less than 500 mL of urine in 24 h), cerebral or visual disturbances, pulmonary edema or cyanosis, epigastric or right upper quadrant pain, impaired liver function, thrombocytopenia, intrauterine growth restriction

Eclampsia is defined as a seizure in a patient with preeclampsia and carries added risk for respiratory compromise and all the known complications of an intracranial bleed. After airway control has been established, then pharmacologic treatment of the cause can be instituted. Magnesium is the treatment of choice for preeclamptics or eclamptics. The special pharmacology of magnesium as it relates to anesthetic drug choices must always be kept in mind along with its impact on renal physiology. The results of the MAGPIE study demonstrated that magnesium is the drug of choice to treat hypertensive disorders of pregnancy, and obstetric anesthesia providers should expect it to be on board when consulted for anesthetic management of a preeclamptic [118].

Preeclampsia in the USA is treated with prophylactic magnesium sulfate to prevent the escalation to eclampsia. Magnesium can potentiate maternal hypotension after the initiation of neuraxial anesthesia that is difficult to treat because magnesium attenuates the response to vasopressors. Dose reduction for depolarizing and nondepolarizing neuromuscular is wise in a patient on magnesium therapy because there will be a delayed recovery of muscular strength to acceptable levels for extubation if a general anesthetic is needed. Calcium chloride is the drug of choice for magnesium toxicity.

A good strategy for the anesthetic management of preeclampsia is to intervene early. The disease progression can rapidly devolve into an escalating high-risk anesthetic encounter. The patient usually has intravascular hypovolemia despite obvious third-space overload expressed as independent edema in all tissues. A progressive coagulopathy must be ruled out before approaching the patient to discuss risks and benefits of neuraxial anesthesia. A comprehensive review of the history of the present illness and its systemic manifestations is crucial to being able to provide safe anesthesia care.

If lab values are acceptable and appropriate intravascular resuscitation can be made then neuraxial anesthesia is the technique of choice whether the obstetric management plan is to attempt vaginal delivery or the plan is for cesarean delivery.

The number of platelets needed to be reassured about placing a neuraxial catheter in a preeclamptic/eclamptic patient is debatable. If the disease is mild, our absolute acceptable number is 75,000 platelets, but at least 10% of all preeclamptics will fall below 100,000 platelets which seems to have a more formal acceptance threshold. Anesthetic judgment about offering neuraxial anesthesia to a parturient with less than 75,000 platelets is justifiable when the clinical picture supports that assessment. How functional those platelets are is as much of an issue as the absolute number. Thromboelastographic analysis or platelet function analysis may be needed if the clinical history suggests a bleeding trend despite the absolute platelet numeric count being acceptable. A platelet count of 50,000 seems to be the absolute lowest limit acceptable to consider providing neuraxial anesthesia.

HELLP syndrome describes a constellation of hemolysis (anemia with blood film evidence of hemolysis), elevated liver enzymes showing hepatic dysfunction (transaminitis greater than twice the normal range), and low platelets (platelet count of less than 150,000/dL). HELLP occurs in around 5% of preeclampsia cases or 10–20% of severe preeclampsia, and is associated with 1.1% maternal mortality and severe morbidity including disseminated intravascular coagulopathy, liver hematoma, liver failure, and renal failure [119]. If there is a clinical picture suggestive of HELLP, then my practice is to place an epidural catheter several hours before the patient requires analgesia. Any patient with HELLP will be delivered expeditiously, and to place a catheter before the platelet count is unacceptably low allows for the option of neuraxial analgesia for a trial of labor. In parturients with low platelet counts, a platelet count should be determined before removing the epidural catheter.

Neuraxial anesthesia is sometimes requested by obstetricians for blood pressure control, which should not be the primary reason to place an epidural, but rather to reap the secondary gain associated with good pain relief. Neuraxial analgesia does blunt the exaggerated hypertensive response to labor in the preeclamptic population, which results in better maternal blood pressures. In the

interest of the fetus, epidural analgesia may improve intervillous blood flow and decrease the likelihood of urgent cesarean delivery for monitored non-reassuring fetal tracing. If an urgent cesarean delivery is needed, it is better to have a functioning in situ epidural to provide the anesthesia for operative delivery than to go through the increased risks of general anesthesia and a possible difficult intubation in a stress setting.

I consider spinal anesthesia to be the preferred method for operative anesthesia whether elective, urgent, or emergent in the preeclamptic patient. As noted in the section on hypotension for complications and under cesarean anesthetic conduct, it has been found that patients with preeclampsia have less severe spinal anesthesia-induced hypotension compared to general anesthesia. It had always been felt that sudden sympathectomy could compromise either the parturient or the fetus, but multiple studies cited earlier have dispelled this belief and shown that using low-dose vasopressors creates a hemodynamically smooth course for cesarean delivery after spinal anesthesia. The increased risk associated with an indwelling catheter in a potentially coagulopathic patient might need to be endured if a long cesarean section is anticipated that requires a combined spinal-epidural technique to allow for extending the block duration. Fetal outcomes are favorable for spinal anesthesia delivery in the preeclamptic.

Maternal Hemorrhage

Maternal hemorrhage is the most preventable cause of maternal mortality. Rapid anesthetic response is important to outcomes for the parturient and the fetus. Clinical scenarios with potential extreme urgency include placenta previa, placental abruption, cesarean hysterectomy from abnormal placentation, and postpartum hemorrhage. Maternal and fetal status require rapid assessment for frank bleeding or when bleeding is suspected but not obvious. If a patient is suspected of having a high risk for hemorrhage early enough to convene a joint care conference to possibly include nursing, obstetrics, blood bank, interventional radiology, and anesthesiology, that should be undertaken to explore the options for management and to understand the decision process guiding patient care among each specialty.

Placenta Previa

Placenta previa has an incidence of 0.5%, often presenting with painless vaginal bleeding. Before 37 weeks gestation, it is managed with bed rest and observation, and after 37 weeks gestation management is delivery via cesarean. For nonurgent antenatal bleeding associated with placenta previa, most anesthesia providers will consider neuraxial anesthesia if the risk for cesarean hysterectomy is not obviously increased. However general anesthesia is the preferred route when there is active bleeding or an unstable patient requiring a cesarean hysterectomy. For a patient with multiple prior cesarean sections or a history of prior placenta previa, the presumption that a placenta accreta will be encountered should always be a part of the anesthetic plan and appropriate contingencies to deal with the complication made. At least two large-bore IV access and blood bank readiness to administer products are mandatory before commencing operative delivery.

Cesarean section for placenta previa diagnosed preoperatively with appropriate resuscitation before entering the operating room represents a good opportunity to provide neuraxial anesthesia absent any other risk factors. Large volumes of blood can be hidden in a partial abruption. The potential for maternal hypotension that compromises the uteroplacental flow will still exist but is treatable and in my opinion is not a contraindication to the use of neuraxial anesthesia.

A cesarean hysterectomy can be anticipated to lose between 2500 and 4500 mL of blood, and the difficulty for surgeons trying to secure vascular pedicles is increased due to uterine size. A spontaneously breathing patient makes anatomical structure identification more difficult for the surgeon especially where the uterine artery dives under the ureter. A strong case can

be made that it is best to secure the airway before the surgery begins because airway edema markedly worsens during large-volume resuscitation. Trying to secure an airway is also more difficult with an open abdomen and potential significant hypotension. Tenable arguments against the use of neuraxial anesthesia include the risk of severe maternal hypotension, patient discomfort associated with intraperitoneal manipulation and traction, and patient discomfort associated with a prolonged surgical procedure. In the circumstance of placenta previa and acute hemorrhage, general anesthesia is the best choice [120].

Having interventional radiologists place uterine artery catheters before cesarean delivery can mitigate blood loss. If blood loss continues postoperatively, then uterine artery catheters can be used to embolize the uterus and hopefully prevent postpartum hemorrhage and hysterectomy [121]. The uterus has collateral artery blood supplies that are branches of the ovarian and rectal artery system, which means that all bleeding is not controlled by securing the uterine artery supply.

Placental Abruption

The anesthetic considerations in patients with placental abruption are similar to those with placenta previa. The incidence of placental abruption ranges from 1 to 2% of deliveries but is mostly encountered as mild or moderate without extreme risk. Risk factors the anesthesia provider should be aware of for placental abruption include a history of trauma, hypertension, alcohol/cocaine abuse, multiparity, and prolonged PROM. These patients will often present with painful vaginal bleeding. The incidence of abruption that impacts anesthetic management is approximately 1 in 150 deliveries. Of abruptions, 90% will have no fetal distress evident. However, large volumes of blood can be hidden in a partial abruption. An additional consideration is the possible presence of disseminated intravascular coagulation (DIC) triggered by the abruption. Neuraxial anesthesia is contraindicated in the presence of DIC. Patients who are hemodynamically stable without ongoing hemorrhage and a coagulopathy are candidates for neuraxial analgesia/anesthesia. Vaginal delivery is possible for most cases of abruption. General anesthesia is indicated for acute hemorrhage or in the presence of DIC. Cesarean delivery for placental abruption can result in massive blood loss; patients should proceed to the operating room with appropriate IV access for massive transfusion of blood products as well as coagulation factors and platelets.

Postpartum Hemorrhage

The anesthetic considerations in postpartum hemorrhage are similarly focused on achieving hemodynamic stability and assessing maternal blood volume issues. Postpartum hemorrhage is often underestimated by simple underaccounting or may also be anatomically hidden in the retroperitoneal gutters or in the broad ligament from delivery trauma. If an epidural is still present from delivery, then an extension of epidural analgesia or initiation of neuraxial anesthesia is appropriate. However, in the face of hypovolemia and hemodynamic instability, general anesthesia is the anesthetic of choice.

Retained placenta and uterine atony are other commonly encountered causes for postpartum hemorrhage. Pharmacologic treatment using oxytocin, methylergonovine, prostaglandin F2 alpha, and misoprostol is used to try and target the myometrium to contract sufficiently to stop hypotonic bleeds while retained placenta requires manual extraction. Nitroglycerin or extension of epidural block facilitates cervical dilation and extraction. If operative removal is needed, then an extension of neuraxial anesthesia of provision of deep MAC anesthesia is preferred. Inhalation agents are profound uterine relaxants and can increase bleeding after extraction and should be avoided. A hypotonic or atonic uterus has a rich blood supply and the amount of blood lost can accumulate rapidly. The uterine perfusion at delivery is 500–700 mL/min and the risk for uterine atony complication is 2–5% of all deliveries [122].

VBAC

Uterine rupture is a potential risk for all parturients electing a VBAC trial who decline a repeat cesarean delivery. The risk for hemorrhage depends on the manifestation of the "rupture." However, the anesthesia provider should be aware of the common presentations of uterine rupture including pain, bleeding, loss of uterine tone, fetal distress, or hypotension. True rupture requires emergent intervention on behalf of the mother and fetus while dehiscence is an urgent event with less blood than might be anticipated since the scar dehiscence should be in the relatively avascular lower uterine segment. It is strongly advised that the parturient trying to achieve VBAC should have an epidural for the labor. Epidurals will not mask the pain of uterine rupture, changing in character to a continuous pain often with associated hypotension, and can be used to facilitate cesarean delivery urgently as needed avoiding the risk of general anesthesia [123]. The anesthesia provider should be prepared for administering volume replacement and potentially converting to a general anesthetic if applicable.

Massive Transfusion

Obstetric hemorrhage can be profound with rapidly developing shock despite the physiologic adaptations of pregnancy that provide the parturient with more red blood cells and circulating volume. These adaptations may delay detection of impending collapse. We have massive transfusion guidelines that we institute when faced with extreme obstetrical hemorrhage. The following is a short synopsis of our guiding principles as developed by the Vanderbilt surgical trauma service [124, 125]:

1. The initial dose will consist of:
   (a) 6 RBCs: If trauma units are used; Rh pos for males and females with expected age >50; Rh neg for females with expected age <50.
   (b) 4 FFP: If trauma units are used, select AB FFP.
   (c) 1 Platelet dose.

   Products are sent together as *complete* doses as described above:
   (d) Only the number and type of products as outlined in this protocol can be issued.
   (e) Requests for additional numbers or type of product must be preapproved by the Blood Bank resident or BB attending.
   (f) RBCs, FFP, and platelets must be issued *together* for each cycle. RBC and platelets will not be sent without the FFP.
   (g) Exception: If FFPs are not thawed and ready at the initiation of the massive transfusion protocol; RBCs if requested, cryoprecipitate can be issued.
   (h) Given the high ratio of plasma infused for each cycle, cryoprecipitate is not necessary. If MTP is started late in the resuscitation and the clinical team feels that fibrinogen may have been low from the beginning, then cryo may be considered.
2. An emergency release form is issued with any uncross-matched units.
3. RBCs and FFP are packed in a cooler with ice:
   (a) Platelets are placed in a plastic ziplock bag, labeled with "Do Not Place Platelets in Cooler" sticker.
4. When the cycle is ready, the patient location is called to notify the staff that the cooler is ready for pickup, and asked if the MTP is to continue.
   (a) If an OR room telephone line is busy, it is permissible to call the OR Board to tell them that the cooler is ready, but the *OR room must be called to ask if the MTP is to continue*.
5. If the protocol is to continue, additional coolers will be supplied as soon as all products in the cycle are ready. This is approximately every 30 min.
6. The second and subsequent doses will consist of:
   (a) 6 RBCs
   (b) 4 FFP
   (c) 1 Platelet dose
7. When each dose is ready, the patient location is called to notify them that the cooler is ready. At this time, Blood Bank asks if the protocol is to continue.

8. This process is continued until the attending surgeon or anesthesiologist tells the Blood Bank to discontinue.

Additional Considerations

Many of the following comments and pearls were first described by Dr. Ray Paschall at Vanderbilt Department of Anesthesiology in the first edition of this chapter. Obstetric anesthesia is a subspecialty full of unpredictability and challenge. Nothing is normal no matter what it may look like on the surface, and the ability to always remain vigilant is Sisyphean. Fortunately, patient outcomes are usually happy as the process of childbirth is a much-anticipated event that is more appreciated when the parturient is made comfortable. Interactions and feedback with obstetricians and nursing are vital to be kept in the loop and contribute to patient safety. Because the ambient environment of obstetrics obeys the second law of thermodynamics, there is always an entropic outbreak about to happen. Remaining calm and rational in the flight to the OR for emergent delivery is sometimes viewed as the anesthesiologist just not having the right appreciation or perspective for the events that triggered the chaos. The subspecialty is indeed the most gratifying when the patient recognizes us for making her comfortable, keeping her and the baby safe, and being able to control all the variables making her anxious about the whole process. How to conduct a case is always a matter of confidence in your ability and is as much an art as a science as can exist in anesthesia. The pressure to do something for medicolegal issues should be resisted if it conflicts with what you think is the right management plan.

Remifentanil can be liberally used for procedure placement like arterial lines, epidurals, and spinals. From our experiences with fetal surgery and our in utero monitoring, babies are far more averse to having their umbilical cord compromised to a far greater extent than anesthetic drugs such as opioids or benzodiazepines. Babies wake up about the same time as their mother after our general anesthetic/epidural/narcotic administration for in utero repair. Inform the neonatal team of drugs administered, and that team will know how to handle the consequences to the baby. It is all about multi-collaborative teamwork and sharing information.

The possibility of developing chronic pain after a routine cesarean delivery is real, so multimodal paths are used to achieve anesthesia and analgesia. Routine use of clonidine in spinals at a dose of 0.2 mcg/kg is routine. Ketamine and propofol are used as an adjunct to cover patchy epidural blocks or to sedate the patient uncomfortable with being awake for cesarean surgery. A usual induction drug sequence is to give 100 mg lidocaine IVP, then remifentanil 2 mcg/kg IVP, and then 1 mg/kg propofol which has ketamine mixed at 2.5 mg/mL and succinylcholine 1 mg/kg. For inductions or preceding spinal placement, it is common to add 400 mcg phenylephrine/L to crystalloid and infuse it rapidly to coload and to mitigate hypotension. Match the hatch is a fishing phrase that translates well to any individual obstetric anesthesia practice because the obstetric anesthesia provider has the onus of doing his or her job to fit the obstetrician's ability and expectations. Fortunately, there are a wide armamentarium of drugs to achieve this goal.

The only constant in the field is change, and it behooves the anesthesia provider to know more about the practice of obstetrics than does an obstetrician feel compelled to understand our specialty. Much is expected of obstetric anesthesia providers, and we have met the challenge for the most part and have advanced the safety of mother and baby mostly through regional anesthetic use. Ultrasound will be common in the near future especially if the sonography improves to allow for real-time imaging when performing neuraxial techniques.

Clinical Pearls

- Early pain may be a predictor for likely C/S; therefore consider placing epidural catheter higher rather than lower to facilitate conversion to surgical anesthesia.

Anesthesia for Labor

- When continuous fetal heart rate tracing is being performed the L&D nurses usually place the bands holding the toco and fetal heart monitors over the iliac crests which are usually visible along the patient back roughly equivalent to Tuffier's line.
- Data from trials to assess the risk for cesarean delivery cannot control for the factor thought to be most responsible for operative rates which are obstetrician preference.
- Maintain analgesia because tachyphylaxis or tolerance is possible when more rescue dosing is needed due to inadequate analgesia especially with bupivacaine.
- Hyperbaric bupivacaine 2.5–5 mg can be utilized to cover sacral nerves when utilizing CSE, spinal, or continuous IT catheter. Sacral sparing is often an unwanted epidural fact.
- Opioids help relieve persistent perineal pain and hot spots of missed segments; sufentanil has the least dermatomal spread and hydromorphone the most dermatomal spread for narcotics that are lipophilic.

Anesthesia for Cesarean Section

- Phenylephrine is my drug of choice for hypotension caused by sympathectomy below T10; from T10 to T5 ephedrine will have more effective pressor action.
- The only epidural catheter worth keeping is one that can be predicted to be dosed rapidly to section anesthesia. Redosing more than three times in an hour is an indicator of a catheter that should be replaced.

Adjuvant and Alternative OB Blocks

- Although no longer frequently used, both the paracervical and pudendal blocks served as tools to differentially map the course of labor.

Hypotension

- Time to a clinically appreciable pressor action of phenylephrine is approximately 1 min, while ephedrine clinical onset is approximately 2 min.
- The Institute for Safe Medication Practices has placed oxytocin on the High Alert list due to the potential of oxytocin to cause profound hypotension.

Neurologic Complication

- The course of labor and childbirth delivery is more likely to cause nerve injury than is anesthesia conduct.

Review Questions

1. A G4P3003 presents for third repeat C/S. BMI is 45, and pt. has comorbidities of gestational DM not requiring insulin. VS are BP 145/90, P 98, and SpO2 is 96% on RA. Hct is 32 and blood glu is 145. Last C/S took 2 h to complete due to adhesions encountered. Best choice for anesthesia?
   (a) Spinal
   (b) Combined spinal-epidural
   (c) Epidural
   (d) General anesthesia
2. Which of the following is an inappropriate rescue drug for high spinal?
   (a) Vasopressin 2 U IVP
   (b) Phenylephrine 200 mcg IVP
   (c) Midazolam 2 mg
   (d) Ephedrine 10 mg
3. A parturient presents for VBAC at 37 weeks, now G3P2002 with both prior deliveries by C/S due to CPD. U/S reveals anterior placenta. VS are normal and pt. has no significant medical history except for gestational nausea and backache. Pt. wishes to have natural labor. Which of the following is not true?

(a) Pt. is at low risk for abnormal placental implantation.
(b) Early neuraxial anesthesia placement is preferred.
(c) Pt. needs a type and screen in the blood bank.
(d) Pt. is at high risk for abnormal placental implantation.

4. Preeclampsia:
(a) Occurs before 20-week gestation.
(b) Can occur after delivery.
(c) Is a contraindication to neuraxial anesthesia.
(d) Has a mild, moderate, and severe form.

5. Spinal block height:
(a) Is strongly predicted by pt. height.
(b) Is reduced in obese patients.
(c) Is best predicted by volume of lumbosacral CSF.
(d) Is adequate if a T8 level is achieved for C/S.

6. Pregnancy:
(a) Is a hypocoagulable state.
(b) Is a hypercoagulable state.
(c) Has higher baseline maternal BP to feed the fetus.
(d) Offers no challenges to the anesthesia provider.

7. Neuraxial anesthesia:
(a) Slows labor.
(b) Increases the risk for C/S.
(c) Causes maternal backache postpartum.
(d) Decreases maternal stress catecholamine levels.

8. Urgent C/S will be performed most frequently:
(a) For a VBAC patient
(b) Fetal heart tracing abnormalities
(c) Placental abruption
(d) Eclampsia

9. Spinal headache after wet tap:
(a) Occurs immediately.
(b) Is rarely severe.
(c) Is exacerbated by lying down.
(d) The definitive treatment is blood patch.

10. Magnesium sulfate therapy:
(a) Is a relative contraindication to neuraxial anesthesia.
(b) Potentiates neuromuscular blockade.
(c) Is algolic.
(d) Requires higher dosing of local anesthesia to achieve labor analgesia.

11. Paracervical blocks:
(a) Are easier and safer to perform than neuraxial anesthesia.
(b) Are safer for the fetus.
(c) Are limited to first stage of labor relief.
(d) Are limited to second stage of labor relief.

12. Combined spinal-epidural anesthesia:
(a) Has been associated with shorter labors.
(b) Doubles the risk for postpartum headache.
(c) Should only be performed when a parturient wants a walking epidural.
(d) Causes profound hypotension.

13. A patient is seen the day after a prolonged vaginal delivery with epidural analgesia. She complains of numbness only in the lateral femoral cutaneous nerve distribution. Which of the following is true?
(a) The epidural could not have caused this because it is only a sensory nerve.
(b) The effect is probably permanent if it does not resolve within 2 weeks.
(c) The obstetric team probably caused it by compressing the nerve under her inguinal ligament during delivery.
(d) No follow-up is necessary.

14. Pt. with epidural catheter now s/p SVD with partial abruption but continues to ooze despite 30 U of oxytocin. History of gestational PIH but not preeclamptic.
(a) Administer methergine
(b) Draw DIC labs
(c) Order trauma blood from the blood bank
(d) Administer 10 more units of oxytocin

15. The most likely complication for neuraxial anesthesia is:
(a) Wet tap
(b) Transient neural injury
(c) Inadequate analgesia or failed block
(d) Infection

Answers

1. The answer is c because the expected duration could exceed single-shot spinal dosing and an

epidural is not proven to work in the CSE technique since it cannot be tested after spinal dosing. General anesthesia is least desirable

2. c
3. The answer is a. Pt. has 24–40% chance for accreta and therefore large-volume blood loss
4. The answer is b. As noted, it is in the differential diagnosis for postpartum hypertensive headache
5. c
6. b
7. d
8. b
9. d
10. b
11. c
12. a
13. c
14. The answer is b because that is the probable cause for continued bleeding. Trauma blood is inappropriate without Blood Bank current cross match due to the possibility of changed profile associated with fetal-maternal blood mixing with abruption. Methergine is contraindicated with hypertensive disease of pregnancy
15. c

References

1. Wong CA. Advances in labor analgesia. Int J Women's Health. 2010;1:139–54.
2. Traynor AJ, Aragon M, Ghosh D, Choi RS, Dingmann D, Vu Tran Z, Bucklin BA. Obstetric anesthesia workforce survey: a 30-year update. Anesth Analg. 2016;122(6):1939–46.
3. Carvalho JC. Ultrasound-facilitated epidurals and spinals in obstetrics. Anesthesiol Clin. 2008;26(1):145–58, vii–viii.
4. Kopacz DJ, Neal JM, Pollock JE. The regional anesthesia "learning curve". What is the minimum number of epidural and spinal blocks to reach consistency? Reg Anesth. 1996;21(3):182–90.
5. de Oliveira Filho GR. The construction of learning curves for basic skills in anesthetic procedures: an application for the cumulative sum method. Anesth Analg. 2002;95(2):411–6, table of contents.
6. Melzack R. The myth of painless childbirth (the John J. Bonica lecture). Pain. 1984;19(4):321–37.
7. American College of Obstetrics and Gynecology. ACOG practice bulletin. Obstetric analgesia and anesthesia. Number 36, July 2002. American College of Obstetrics and Gynecology. Int J Gynaecol Obstet. 2002;78(3):321–35.
8. ACOG Committee opinion #295: pain relief during labor. Obstet Gynecol. 2004;104(1):213.
9. American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Practice guidelines for obstetric anesthesia. Anesthesiology. 2007;106(4):843–63.
10. Soma-Pillay P, Nelson-Piercy C, Tolppanen H, Mebazaa A. Physiological changes in pregnancy. Cardiovasc J Afr. 2016;27(2):89–94.
11. Sibai BM, Frangieh A. Maternal adaptation to pregnancy. Curr Opin Obstet Gynecol. 1995;7(6):420–6.
12. Aarnoudse JG, Oeseburg B, Kwant G, Zwart A, Zijlstra WG, Huisjes HJ. Influence of variations in pH and PCO2 on scalp tissue oxygen tension and carotid arterial oxygen tension in the fetal lamb. Biol Neonate. 1981;40(5–6):252–63.
13. Sangoul F, Fox GS, Houle GL. Effect of regional analgesia on maternal oxygen consumption during the first stage of labor. Am J Obstet Gynecol. 1975;121(8):1080–3.
14. Jouppila P, Jouppila R, Hollmén A, Koivula A. Lumbar epidural analgesia to improve intervillous blood flow during labor in severe preeclampsia. Obstet Gynecol. 1982;59(2):158–61.
15. Ramos-Santos E, Devoe LD, Wakefield ML, Sherline DM, Metheny WP. The effects of epidural anesthesia on the Doppler velocimetry of umbilical and uterine arteries in normal and hypertensive patients during active term labor. Obstet Gynecol. 1991;77(1):20–6.
16. Cascio M, Pygon B, Bernett C, Ramanathan S. Labour analgesia with intrathecal fentanyl decreases maternal stress. Can J Anaesth. 1997;44(6):605–9.
17. Shnider SM, Abboud TK, Artal R, Henriksen EH, Stefani SJ, Levinson G. Maternal catecholamines decrease during labor after lumbar epidural anesthesia. Am J Obstet Gynecol. 1983;147(1):13–5.
18. Lederman RP, Lederman E, Work B Jr, McCann DS. Anxiety and epinephrine in multiparous women in labor: relationship to duration of labor and fetal heart rate pattern. Am J Obstet Gynecol. 1985;153(8):870–7.
19. Thalme B, Raabe N, Belfrage P. Lumbar epidural analgesia in labour. II. Effects on glucose, lactate, sodium, chloride, total protein, haematocrit and haemoglobin in maternal, fetal and neonatal blood. Acta Obstet Gynecol Scand. 1974;53(2):113–9.
20. Yancey MK, Pierce B, Schweitzer D, Daniels D. Observations on labor epidural analgesia and operative delivery rates. Am J Obstet Gynecol. 1999;180(2 Pt 1):353–9.
21. Kozhimannil KB, Law MR, Virnig BA. Cesarean delivery rates vary tenfold among US hospitals; reducing variation may address quality and cost issues. Health Aff (Millwood). 2013; 32(3):527–35.

22. Halpern S, Leighton B. Epidural analgesia and the progress of labor. In: Halpern SH, Douglas MJ, editors. Evidence-based obstetric anesthesia. Oxford: Blackwell; 2005. p. 10–22.
23. Wong CA, Scavone BM, Peaceman AM, McCarthy RJ, et al. The risk of cesarean delivery with neuraxial analgesia given early versus late in labor. N Engl J Med. 2005;352(7):655–65.
24. Hiltunen P, Raudaskoski T, Ebeling H, Moilanen I. Does pain relief during delivery decrease the risk of postnatal depression? Acta Obstet Gynecol Scand. 2004;83(3):257–61.
25. Eidelman AI, Hoffmann NW, Kaitz M. Cognitive deficits in women after childbirth. Obstet Gynecol. 1993;81(5 (Pt 1)):764–7.
26. Juhan D. Job's body: a handbook for bodywork. New York: Station Hill Press; 2003.
27. Wager TD, Rilling JK, Smith EE, Sokolik A, et al. Placebo-induced changes in FMRI in the anticipation and experience of pain. Science. 2004;303(5661):1162–7.
28. Wright ME, McCrea H, Stringer M, Murphy-Black T. Personal control in pain relief during labour. J Adv Nurs. 2000;32(5):1168–77.
29. Lowe NK. The nature of labor pain. Am J Obstet Gynecol. 2002;186(5 Suppl Nature):S16–24.
30. Datta S, Lambert DH, Gregus J, Gissen AJ, Covino BG. Differential sensitivities of mammalian nerve fibers during pregnancy. Anesth Analg. 1983;62(12):1070–2.
31. Johnson RF, Herman NL, Johnson HV, Arney TL, Paschall RL, Downing JW. Effects of fetal pH on local anesthetic transfer across the human placenta. Anesthesiology. 1996;85(3):608–15.
32. Bernards CM. Understanding the physiology and pharmacology of epidural and intrathecal opioids. Best Pract Res Clin Anaesthesiol. 2002;16(4):489–505.
33. Hogan Q. Epidural catheter tip position and distribution of injectate evaluated by computed tomography. Anesthesiology. 1999;90(4):964–70.
34. Cousins C, Veering B. Epidural neural blockade. In: Neural blockade in clinical anesthesia and management of pain, M. Cousins and P. Bridenbaugh, Eds. Philadelphia: Lippincott Williams & Wilkins, 1998, pp. 242–321.
35. Broadbent CR, Maxwell WB, Ferrie R, Wilson DJ, Gawne-Cain M, Russell R. Ability of anaesthetists to identify a marked lumbar interspace. Anaesthesia. 2000;55(11):1122–6.
36. Chakraverty R, Pynsent P, Isaacs K. Which spinal levels are identified by palpation of the iliac crests and the posterior superior iliac spines? J Anat. 2007;210(2):232–6.
37. Windisch G, Ulz H, Feigl G. Reliability of Tuffier's line evaluated on cadaver specimens. Surg Radiol Anat. 2009;31(8):627–3.
38. Reynolds F. Damage to the conus medullaris following spinal anaesthesia. Anaesthesia. 2001;56(3):238–47.
39. Panni MK, Columb MO. Obese parturients have lower epidural local anaesthetic requirements for analgesia in labour. Br J Anaesth. 2005;96(1):106–10.
40. Polley LS, Columb MO, Naughton NN, Wagner DS, van de Ven CJ. Relative analgesic potencies of ropivacaine and bupivacaine for epidural analgesia in labor: implications for therapeutic indexes. Anesthesiology. 1999;90(4):944–50.
41. Tsen LC, Thue B, Datta S, Segal S. Is combined spinal-epidural analgesia associated with more rapid cervical dilation in nulliparous patients when compared with conventional epidural analgesia? Anesthesiology. 1999;91(4):920–5.
42. Eltzschig HK, Lieberman ES, Camann WR. Regional anesthesia and analgesia for labor and delivery. N Engl J Med. 2003;348(4):319–32.
43. Halpern SH, Carvalho B. Patient-controlled epidural analgesia for labor. Anesth Analg. 2009;108(3):921–8.
44. Liao JB, Buhimschi CS, Norwitz ER. Normal labor: mechanism and duration. Obstet Gynecol Clin N Am. 2005;32(2):145–64, vii.
45. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Kirmeyer S, Munson ML. Centers for Disease Control and Prevention National Center for Health Statistics National Vital Statistics System. Births: final data for 2005. Natl Vital Stat Rep. 2007;56(6):1–103.
46. American College of Obstetricians and Gynecologists. ACOG Committee opinion no. 395: surgery and patient choice. Obstet Gynecol. 2008;111(1):243–7.
47. Davoudi M, Tarbiat M, Ebadian MR, Hajian P. Effect of position during spinal anesthesia on postdural puncture headache after cesarean section: a prospective, single-blind randomized clinical trial. Anesth Pain Med. 2016;6(4):e35486.
48. Ng K, Parsons J, Cyna AM, Middleton P. Spinal versus epidural anaesthesia for caesarean section. Cochrane Database Syst Rev. 2004;2:CD003765.
49. Aiono-Le Tagaloa L, Butwick AJ, Carvalho B. A survey of perioperative and postoperative anesthetic practices for cesarean delivery. Anesthesiol Res Pract. 2009;2009:510642.
50. Reynolds F, Seed PT. Anaesthesia for caesarean section and neonatal acid-base status: a meta-analysis. Anaesthesia. 2005;60(7):636–53.
51. Ngan Kee WD, Khaw KS, Tan PE, Ng FF, Karmakar MK. Placental transfer and fetal metabolic effects of phenylephrine and ephedrine during spinal anesthesia for cesarean delivery. Anesthesiology. 2009;111(3):506–12.
52. Minzter BH, Johnson RF, Paschall RL, Ramasubramanian R, Ayers GD, Downing JW. The diverse effects of vasopressors on the fetoplacental circulation of the dual perfused human placenta. Anesth Analg. 2010;110(3):857–62.
53. Riley ET, Editorial I. Spinal anaesthesia for caesarean delivery: keep the pressure up and don't spare the vasoconstrictors. Br J Anaesth. 2004;92(4):459–61.

54. Bloom SL, Leveno KJ, Spong CY, Gilbert S, et al. Decision-to-incision times and maternal and infant outcomes. Obstet Gynecol. 2006;108(1):6–11.
55. Tuffnell DJ, Wilkinson K, Beresford N. Interval between decision and delivery by caesarean section-are current standards achievable? Observational case series. BMJ. 2001;322(7298):1330–3.
56. Hillemanns P, Strauss A, Hasbargen U, Schulze A, Genzel-Boroviczeny O, Weninger E, Hepp H. Crash emergency cesarean section: decision-to-delivery interval under 30 min and its effect on Apgar and umbilical artery pH. Arch Gynecol Obstet. 2005;273(3):161–5.
57. Pargger H, Hampl KF, Aeschbach A, Paganoni R, Schneider MC. Combined effect of patient variables on sensory level after spinal 0.5% plain bupivacaine. Acta Anaesthesiol Scand. 1998;42(4):430–4.
58. Carvalho B, Cohen SE, Lipman SS, Fuller A, Mathusamy AD, Macario A. Patient preferences for anesthesia outcomes associated with cesarean delivery. Anesth Analg. 2005;101(4):1182–7, table of contents.
59. Ben-David B, Miller G, Gavriel R, Gurevitch A. Low-dose bupivacaine-fentanyl spinal anesthesia for cesarean delivery. Reg Anesth Pain Med. 2000;25(3):235–9.
60. Oğün CO, Kirgiz EN, Duman A, Okesli S, Akyürek C. Comparison of intrathecal isobaric bupivacaine-morphine and ropivacaine-morphine for caesarean delivery. Br J Anaesth. 2003;90(5):659–64.
61. Ginosar Y, Mirikatani E, Drover DR, Cohen SE, Riley ET. ED50 and ED95 of intrathecal hyperbaric bupivacaine coadministered with opioids for cesarean delivery. Anesthesiology. 2004;100(3):676–82.
62. Raffaeli W, Marconi G, Fanelli G, Taddei S, Borghi GB, Casati A. Opioid-related side-effects after intrathecal morphine: a prospective, randomized, double-blind dose-response study. Eur J Anaesthesiol. 2006;23(7):605–10.
63. Price ML, Reynolds F, Morgan BM. Extending epidural blockade for emergency caesarean section. Evaluation of 2% lignocaine with adrenaline. Int J Obstet Anesth. 1991;1(1):13–8.
64. Regan KJ, O'Sullivan G. The extension of epidural blockade for emergency caesarean section: a survey of current UK practice*. Anaesthesia. 2008;63(2):136–42.
65. Abboud TK, Kim KC, Noueihed R, Kuhnert BR, DerMardirossian N, Moumdjian J, Sarkis F, Nagappala S. Epidural bupivacaine, chloroprocaine, or lidocaine for cesarean section—maternal and neonatal effects. Anesth Analg. 1983;62(10):914–9.
66. Lucas DN, Ciccone GK, Yentis SM. Extending low-dose epidural analgesia for emergency caesarean section. A comparison of three solutions. Anaesthesia. 1999;54(12):1173–7.
67. Halpern SH, Soliman A, Yee J, Angle P, Ioscovich A. Conversion of epidural labour analgesia to anaesthesia for caesarean section: a prospective study of the incidence and determinants of failure. Br J Anaesth. 2009;102(2):240–3.
68. Davies JM, Posner KL, Lee LA, Cheney FW, Domino KB. Liability associated with obstetric anesthesia: a closed claims analysis. Anesthesiology. 2009;110(1):131–9.
69. Rosen MA. Paracervical block for labor analgesia: a brief historic review. Am J Obstet Gynecol. 2002;186(5 Suppl Nature):S127–30.
70. King JC, Sherline DM. Paracervical and pudendal block. Clin Obstet Gynecol. 1981;24(2):587–95.
71. Yantis S, Hirsch N, Smith G. Anesthesia and intensive care. 3rd ed. London: Elsevier; 2004.
72. Rafi AN. Abdominal field block: a new approach via the lumbar triangle. Anaesthesia. 2008;56(10):1024–6.
73. Bonnet F, Berger J, Aveline C. Transversus abdominis plane block: what is its role in postoperative analgesia? Br J Anaesth. 2009;103(4):468–70.
74. McDonnell JG, Curley G, Carney J, Benton A, Costello J, Maharaj CH, Laffey JG. The analgesic efficacy of transversus abdominis plane block after cesarean delivery: a randomized controlled trial. Anesth Analg. 2008;106(1):186–91, table of contents.
75. Hebbard P. Audit of rescue analgesia using TAP block. Anaesth Intensive Care. 2007;35(4):617–8.
76. Moore DC. Intercostal nerve block for postoperative somatic pain following surgery of thorax and upper abdomen. Br J Anaesth. 1975;47(suppl):284–6.
77. Bunting P, McGeachie JF. Intercostal nerve blockade producing analgesia after appendicectomy. Br J Anaesth. 1988;61(2):169–72.
78. Salinas F, Liu S, Scholz A. Ion channel ligands/sodium channel blockers/local anesthetics. In: Evers A, Maze M, editors. Anesthetic pharmacology. Physiologic principles and clinical practice. Philadelphia: Churchill Livingstone; 2004. p. 507–38.
79. Mukhtar K. Transversus abdominis plane (TAP) block. J NYSORA. 2009;12:28–33.
80. Tran TMN, Ivanusic JJ, Hebbard P, Barrington MJ. Determination of spread of injectate after ultrasound-guided transversus abdominis plane block: a cadaveric study. Br J Anaesth. 2009;102(1):123–7.
81. Rozen WM, Tran TM, Ashton MW, Barrington MJ, Ivanusic JJ, Taylor GI. Refining the course of the thoracolumbar nerves: a new understanding of the innervation of the anterior abdominal wall. Clin Anat. 2008;21(4):325–33.
82. Rosero EB, Joshi GP. Nationwide incidence of serious complications of epidural analgesia in the United States. Acta Anaesthesiol Scand. 2016;60(6):810–20.
83. Ruppen W, Derry S, McQuay H, Moore RA. Incidence of epidural hematoma, infection, and neurologic injury in obstetric patients with epidural analgesia/anesthesia. Anesthesiology. 2006;105(2):394–9.
84. Carpenter RL, Hogan QH, Liu SS, Crane B, Moore J. Lumbosacral cerebrospinal fluid volume is the

primary determinant of sensory block extent and duration during spinal anesthesia. Anesthesiology. 1998;89(1):24–9.
85. Carvalho B, Durbin M, Drover DR, Cohen SE, Ginosar Y, Riley ET. The ED50 and ED95 of intrathecal isobaric bupivacaine with opioids for cesarean delivery. Anesthesiology. 2005;103(3):606–12.
86. Norris M. Patient variables and the subarachnoid spread of hyperbaric bupivacaine in the term parturient. Anesthesiology. 1990;72(3):478–82.
87. Ben-David B, Vaida S, Gaitini L. The influence of high spinal anesthesia on sensitivity to midazolam sedation. Anesth Analg. 1995;81(3):525–8.
88. Boutonnet M, Faitot V, Keïta H. Airway management in obstetrics. Annales francaises d'anesthesie et de. Reanimation. 2011;30(9):651–64.
89. Ciechanowicz S, Patil V. Lipid emulsion for local anesthetic systemic toxicity. Anesthesiol Res Pract. 2012;2012:131784.
90. Tanaka M, Balki M, Parkes RK, Carvalho JCA. ED95 of phenylephrine to prevent spinal-induced hypotension and/or nausea at elective cesarean delivery. Int J Obstet Anesth. 2009;18(2):125–30.
91. Neal JM, Bernards CM, Butterworth JF 4th, Di Gregorio G, et al. ASRA practice advisory on local anesthetic systemic toxicity. ASRA practice advisory on local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35(2):152–61.
92. Sites BD, Taenzer AH, Herrick MD, Gilloon C, Antonakakis J, Richins J, Beach ML. Incidence of local anesthetic systemic toxicity and postoperative neurologic symptoms associated with 12,668 ultrasound-guided nerve blocks: an analysis from a prospective clinical registry. Reg Anesth Pain Med. 2012;37(5):478–82.
93. Weinberg GL. Treatment of local anesthetic systemic toxicity (LAST). Reg Anesth Pain Med. 2010;35(2):188–93.
94. Bernards CM. Sophistry in medicine: lessons from the epidural space. Reg Anesth Pain Med. 2005;30(1):56–66.
95. Heesen M, Klöhr S, Rossaint R, Van De Velde M, Straube S. Can the incidence of accidental dural puncture in laboring women be reduced? A systematic review and meta-analysis. Minerva Anestesiol. 2013;79(10):1187–97.
96. Lybecker H, Djernes M, Schmidt JF. Postdural puncture headache (PDPH): onset, duration, severity, and associated symptoms. An analysis of 75 consecutive patients with PDPH. Acta Anaesthesiol Scand. 1995;39(5):605–12.
97. Agerson AN, Scavone BM. Prophylactic epidural blood patch after unintentional dural puncture for the prevention of postdural puncture headache in parturients. Anesth Analg. 2012;115(1):133–6.
98. Suescun H, Austin P, Gabaldon D. Nonpharmacologic neuraxial interventions for prophylaxis of postdural puncture headache in the obstetric patient. AANA J. 2016;84(1):15–22.
99. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). Reg Anesth Pain Med. 2010;35(1):64–101.
100. Kowe O, Waters JH. Neurologic complications in the patient receiving obstetric anesthesia. Neurol Clin. 2012;30(3):823–33.
101. Kindler CH, Seeberger MD, Staender SE. Epidural abscess complicating epidural anesthesia and analgesia. An analysis of the literature. Acta Anaesthesiol Scand. 1998;42(6):614–20.
102. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev. 2000;23(4):175–204; discussion 205.
103. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Samii K. Serious complications related to regional anesthesia: results of a prospective survey in France. Anesthesiology. 1997;87(3):479–86.
104. Cook TM, Counsell D, Wildsmith JA. Royal College of Anaesthetists Third National Audit Project. Major complications of central neuraxial block: report on the Third National Audit Project of the Royal College of Anaesthetists. Br J Anaesth. 2009;102(2):179–90.
105. Scott DB, Hibbard BM. Serious non-fatal complications associated with extradural block in obstetric practice. Br J Anaesth. 1990;64(5):537–41.
106. Scott DB, Tunstall ME. Serious complications associated with epidural/spinal blockade in obstetrics: a two-year prospective study. Int J Obstet Anesth. 1995;4(3):133–9.
107. Holdcroft A, Gibberd FB, Hargrove RL, Hawkins DF, Dellaportas CI. Neurological complications associated with pregnancy. Br J Anaesth. 1995;75(5):522–6.
108. Graham JG. Neurological complications of pregnancy and anaesthesia. Clin Obstet Gynaecol. 1982;9(2):333–50.
109. Chia YY, Lo Y, Chen YB, Liu CP, Huang WC, Wen CH. Risk of chronic low back pain among parturients who undergo cesarean delivery with neuraxial anesthesia: a nationwide population-based retrospective cohort study. Medicine (Baltimore). 2016;95(16):e3468.
110. Macarthur AJ, Macarthur C, Weeks SK. Is epidural anesthesia in labor associated with chronic low back pain? A prospective cohort study. Anesth Analg. 1997;85(5):1066–70.
111. Howell CJ, Dean T, Lucking L, Dziedzic K, Jones PW, Johanson RB. Randomised study of long term outcome after epidural versus non-epidural analgesia during labour. BMJ. 2002;325(7360):357.
112. Rice I, Wee MY, Thomson K. Obstetric epidurals and chronic adhesive arachnoiditis. Br J Anaesth. 2004;92(1):109–20.
113. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP. Anesthesia-related deaths during obstetric delivery

in the United States, 1979–1990. Anesthesiology. 1997;86(2):277–84.
114. Ngan Kee W. Confidential enquiries into maternal deaths: 50 years of closing the loop. Br J Anaesth. 2005;94(4):413–6.
115. Ranasinghe JS, Birnbach D. Current status of obstetric anaesthesia: improving satisfaction and safety. Indian J Anaesth. 2009;53(5):608–17.
116. McDonnell NJ, Paech MJ, Clavisi OM, Scott KL, ANZCA Trials Group. Difficult and failed intubation in obstetric anaesthesia: an observational study of airway management and complications associated with general anaesthesia for caesarean section. Int J Obstet Anesth. 2008;17(4):292–7.
117. Zhang J, Meikle S, Trumble A. Severe maternal morbidity associated with hypertensive disorders in pregnancy in the United States. Hypertens Pregnancy. 2003;22(2):203–12.
118. Altman D, Carroli G, Duley L, Farrell B, et al. Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: a randomised placebo-controlled trial. Lancet. 2002;359(9321):1877–90.
119. Townsend R, O'Brien P, Khalil A. Current best practice in the management of hypertensive disorders in pregnancy. Integr Blood Press Control. 2016;9:79–94.
120. Parekh N, Husaini SW, Russell IF. Caesarean section for placenta praevia: a retrospective study of anaesthetic management. Br J Anaesth. 2000;84(6):725–30.
121. Sundaram R, Brown AG, Koteeswaran SK, Urquhart G. Anaesthetic implications of uterine artery embolisation in management of massive obstetric haemorrhage. Anaesthesia. 2006;61(3):248–52.
122. Herbert WN, Cefalo RC. Management of postpartum hemorrhage. Clin Obstet Gynecol. 1984;27(1):139–47.
123. Bucklin BA. Vaginal birth after cesarean delivery. J Am Soc Anesthesiol. 2003;99(6):1444–8.
124. Gunter OL Jr, Au BK, Isbell JM, Mowery NT, Young PP, Cotton BA. Optimizing outcomes in damage control resuscitation: identifying blood product ratios associated with improved survival. J Trauma. 2008;65(3):527–34.
125. Cotton BA, Au BK, Nunez TC, Gunter OL, Robertson AM, Young PP. Predefined massive transfusion protocols are associated with a reduction in organ failure and postinjury complications. J Trauma. 2009;66(1):41–8; discussion 48–9

Acute Situations: Trauma in Surgical Specialties

22

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Overview

Trauma is a major cause of mortality and morbidity worldwide, and pain is the most common symptom reported by patients entering the Emergency Department [1]. Each year, more than 100,000 deaths in the USA and about 8% of all deaths worldwide are caused by traumatic injuries [2]. Trauma is also a leading cause of death in persons younger than 30 years [3]. An estimated 5.3 million people in the United States have long-term disabilities resulting from traumatic brain injuries and another 200,000 from spinal cord injuries [3].

Among all the treatment modalities for trauma patients, pain management has become the core intervention because improved pain management has not only led to increased comfort in trauma patients, but has also been shown to reduce morbidity and improve long-term outcomes [4, 5]. Conversely, inadequate pain control leads to drastic clinical consequences, such as thromboembolic and pulmonary complications, lengthy hospital stay, and development of posttraumatic stress disorder [6–8]. Since trauma patients usually experience significantly more stress than patients undergoing elective surgery, trauma patients tend to have increased morbidity as a result of stress-induced higher myocardial oxygen consumption if pain is not adequately controlled [9]. It has also been shown that the persistence of severe, uncontrolled pain can lead to series of anatomic and physiologic changes in the nervous system [10]. These neuroplastic changes underlie the development of chronic, disabling neuropathic pain. For example, one study [11] reported that inadequate pain control resulted in chronic pain syndromes in 69% of patients with spinal cord injuries.

Unfortunately, multiple studies have reported that trauma-related pain is still inadequately controlled [12]. A recent study by Whipple et al. [13] assessed adequacy of pain treatment in 17 patients with multiple trauma injuries. While 95% of staff and 81% of nurses reported adequate analgesia, 74% of patients rated their pain as moderate to severe. Lack of recognition of pain and its related symptoms, limited acknowledgment of various pain management approaches, excessive concern about narcotics-induced hemodynamic instability, respiratory depression, and addiction all contribute to the inadequacy of pain management in trauma patients.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_22

Therefore, pain management in trauma patients still remains a challenge to clinical practitioners. Plus, the need to preserve the hemodynamic stability, the respiratory function, and patients' level of consciousness in this patient population further complicates the challenge.

Traumatic injuries can be associated with severe blood loss and coagulation derangements. Hemorrhage might not be evident at first, and especially in young patients, onset of hypotension is subtle and might not appear until >30% of blood volume is lost. Early signs of significant blood loss are low pulse pressure (less than 25% of the systolic value) and tachycardia with heart rate above 120 [14]. Long bone and pelvic fractures are associated with significant bleeding: a fractured femur, for example, can lead to a 2 L blood loss into the thigh. Blunt abdominal trauma can also lead to hemorrhagic shock, even if not evident at first (e.g., delayed splenic rupture).

Besides, up to one-third of trauma patients develop an endogenous coagulopathy very early in the clinical course [15]. This is a multifactorial condition that results from a combination of bleeding-induced shock, tissue injury with thrombin-thrombomodulin-complex generation, and the activation of anticoagulant and fibrinolytic pathways.

When choosing the optimal pain management protocol for the trauma patient, and even more so when regional anesthesia techniques are being considered, one must take into account that, although not apparent upon the patient's arrival, hemodynamic instability and coagulopathy might develop.

Multimodal analgesia has been increasingly used to manage pain in trauma patients [16]. This wide range of measures includes regional anesthesia procedures, opioids, NSAIDs, NMDA receptor blockers, anticonvulsants, antidepressants, and α2-agonists. Although each modality has its own strength and weakness, regional anesthesia, e.g., peripheral nerve blocks, stands out as an important technique especially in the perioperative setting because many traumatic injuries eventually require surgical interventions [3]. Regional anesthesia can become the first choice of analgesia in patients with isolated orthopedic injuries and burning injuries because this technique avoids many adverse side effects associated with systemic opioids, such as nausea/vomiting, pruritus, urinary retention, hypotension, and respiratory depression.

Even though evidence that shows improvement of outcomes by regional anesthesia in trauma patients is still lacking, it is generally agreeable that adequate analgesia via regional anesthesia reduces incidence of intubation and postoperative morbidity related to traumatic injuries, resulting in positive outcomes [17].

Any regional anesthesia techniques applicable in the elective surgery patient are potentially useful in the trauma patient. Nevertheless, the challenges to manage both pain and other trauma-related complications simultaneously require clinicians to take into account all possible risks and benefits of this technique in order for an optimum patient care to be achieved.

Peripheral Nerve Blocks for Pain Management in Trauma to the Extremities

Peripheral nerve blocks (PNBs) provide rapid and effective analgesia with less opioid-related side effects, such as nausea/vomiting, pruritis, urinary retention, constipation, sedation, and respiratory depression [18].

Depending on the site of injury and the planned operative procedure, peripheral nerve blocks should only be in a designated clinical context.

Before placing the block, it is very important to perform a neurological exam of the patient, documenting sensory and/or motor impairments [3]. A preexisting neurological injury does not represent per se an absolute contraindication to peripheral nerve blocks, but it is important to document it both for medicolegal reasons and for considerations in developing a clinical plan of treatment. Continuous peripheral nerve blocks have been shown to decrease pain scores, increase joint range of motion, and decrease hospital stay and rehabilitation times compared to intravenous patient-controlled analgesia [19]. They also

produce fewer side effects compared with epidural analgesia [20].

Continuous infusion of local anesthetics through implanted catheters has often been found necessary in order to manage trauma-induced pain because single injections do not usually provide long enough pain coverage [3]. Ideally, regional techniques should initially be used to diminish the inflammatory response caused by tissue injury and then continue as long as the painful insults persist [21]. This "preventive" strategy, as opposed to preemptive analgesia which, by definition, only covers the earliest phase of the inflammatory insult, has been postulated to be more beneficial in terms of preventing chronic pain syndromes, although clear evidence is still lacking. In this perspective, multiple sequential catheters are sometimes indicated in order to provide optimal long-lasting analgesia [22]. This strategy has been successfully applied in military medical care, especially for soldiers wounded in combat [14].

Since trauma can occur at multiple sites, nerve blocks at multiple sites are often necessary in order to effectively reduce the amount of IV opioids required. It has been demonstrated that trauma patients may safely benefit from multiple simultaneous continuous peripheral nerve catheter infusions to treat multiple injuries [18].

Both peripheral nerve stimulation and ultrasound may be used to guide needle placement for peripheral nerve localization: neither technique has been proven to be superior to the other in terms of block success, although ultrasound may potentially decrease the time and number of attempts to complete a block [23]. Moreover, eliciting an evoked motor response across a fractured site may cause increased pain, and ultrasound brings a certain advantage in this setting [3]. In cases of traumatic nerve injuries, ultrasound has obvious indications and benefits [24].

Upper Extremity Trauma

Patients frequently presenting to the Emergency Department (ED) with upper extremity injuries such as fractures, dislocations, lacerations, and burns often require immediate pain relief provided by peripheral nerve block. Brachial plexus block usually provides adequate analgesia for upper extremity injuries. Depending on the injury sites, different approaches can be used, for example, interscalene, supraclavicular, or axillary blocks should provide effective pain relief for injuries at mid-distal arm, elbow, forearm, and/or hand [25]. Alternatively, various blocks at forearm should deliver adequate analgesia for hand or wrist injuries.

These nerve block approaches, however, are sometimes associated with risks of having various complications. For example, interscalene nerve block, which is often indicated for anesthesia and/or analgesia in patients with shoulder injuries, can cause Horner's syndrome that obscures the neurological assessment of the patients' consciousness level [3]. Accidental phrenic nerve block can result in an impairment of the ipsilateral diaphragmatic function [26]. The interscalene approach can also present an increased risk of infection should tracheostomy be performed or internal jugular vein catheter be implanted [3]. It is also known that both supraclavicular and infraclavicular nerve blocks are associated with pneumothorax [27]. Among these different approaches, axillary nerve block is probably the least desirable because it requires the largest scale of movement of the injured upper extremity and catheter positioning, and maintenance becomes difficult under the arm [3].

Use of ultrasound in upper extremity nerve blocks has improved the accuracy of needle insertion and catheter placement. The advantage of ultrasound-guided nerve block becomes obvious when it is hard to locate skin landmarks due to either excessive adipose tissues or anatomic distortions caused by neck injuries. It is noteworthy that the presence of C-collar is not a contraindication to performing upper extremity nerve blocks. Once cervical traumatic injuries are ruled out by proper imaging tests, C-collar can be removed and an ultrasound-guided nerve block can be performed.

Sympathectomy that follows regional anesthesia of the upper extremity is often beneficial for revascularization, reimplantation, or in any

Table 22.1 Principal upper extremity blocks in trauma

Upper extremity blocks			
	Indications	Pro	Cons
Interscalenic	Shoulder, proximal humerus,	Effective analgesia with low volume of anesthetic Possibility of continuous block	Ipsilateral diaphragmatic paralysis Risk of vascular puncture
Supraclavicular	Distal humerus, elbow, forearm, wrist	Effective analgesia with low volume of anesthetic Possibility of continuous block	Risk of pneumothorax and vascular puncture
Infraclavicular	Distal humerus, elbow, forearm, wrist	Effective analgesia with low volume anesthetic Lower risk of pneumothorax compared to supraclavicular Possibility of continuous block	Risk of pneumothorax and vascular puncture in non-compressible site
Axillary	Distal humerus, elbow, forearm, wrist	Safest: no risk of pneumothorax, vascular puncture in compressible site	Need for appropriate patient positioning More painful (multiple needle directions) Only single shot block possible

other cases where blood flow is compromised [28]. Before performing the block, the risks and benefits should be discussed with the surgeon. Every effort should be made to avoid radial compartment syndrome. If necessary, a short-acting local anesthetic may be preferred and can be used reliably for surgical anesthesia.

See Table 22.1 for an overview of indications, pro, and cons of upper extremity blocks in trauma.

Case Study #1

A 76-year-old lady is brought to the ED after a fall while riding her bicycle. She is diagnosed with a displaced comminuted left humeral head fracture, and scheduled for surgery the following day. She has moderate Chronic Obstructive Pulmonary Disease (COPD) and her medications include Bronchodilators and low dose Aspirin. She is in considerable pain at rest (NRS 6), which becomes unbearable upon mobilization (NRS 10). Discuss the options for pain management in this patient.

Discussion

Continuous interscalene block is a good option for this patient. An eco-guided procedure should be preferred, in order to avoid the intense pain associated with nerve stimulation in a fractured limb. This block provides good analgesia to the shoulder, upper arm, and elbow. Since the patient is scheduled for surgery, placing a perineural catheter allows to prolong analgesia into the intra- and postoperative period. Interscalene block is associated with phrenic nerve block and consequent ipsilateral hemi-diaphragm paralysis: for this reason it is generally contraindicated in patients with severe pulmonary disease, but should not be a major concern in a patient with moderate COPD. Cautious evaluation should be undertaken in trauma patients with ipsi and contralateral pleural effusions, pulmonary contusions, and pneumonia.

Case Study #2

A 20-year-old boy suffers a displaced fracture of his right forearm after a skateboard accident. He is in considerable pain and is very concerned about the planned reduction maneuver. A conservative treatment is planned, and he will be discharged home with a cast. Discuss the options for pain management in this patient.

Discussion

If a regional anesthesia technique is chosen, single shot supraclavicular, infraclavicular, and axillary block are all good options. The latter avoids the risk of pneumothorax, but the required position

with the abducted arm might be difficult to sustain for the patient. Again, we recommend echo-guided technique and avoidance of neural stimulation. Before discharge, the patient should be informed on the planned duration of the block, and instructed to seek medical advice if numbness/paresthesia persist beyond a reasonable time. An oral analgesic prescription (Acetaminophen or NSAID) should also be provided for the following days.

Lower Extremity Trauma

Regional anesthesia at lower extremity usually includes lumbar plexus block and sacral plexus block at different sites. These nerve block procedures have been proved superior over morphine PCA in providing analgesia in patients with lower extremity trauma [29, 30]. They are also considered safer with less complications comparing with epidural block [3].

Lumbar plexus can be blocked using either anterior or posterior approaches. Anterior approaches at the level of inguinal ligament (3-in-1 or fascia iliaca blocks) have the obvious advantage of not having to put the patients in the lateral position, and have been demonstrated to be safe and at least equivalent to intravenous analgesia in reducing pain in both adult and pediatric patients with femur fractures [31, 32]. The downside is that bocks at this level do not effectively cover all the branches of lumbar plexus, and larger volumes of local anesthetics are often needed to provide adequate analgesia [33].

Posterior approaches (psoas compartment block) usually provide excellent analgesia [34] with relatively small dose of local anesthetics because adequate coverage of all lumbar plexus branches can be achieved at the level of psoas muscle compartment.

Various approaches to sacral plexus block have also been used to provide analgesia to where sciatic nerve is distributed.

Depending on the injury sites, either lumbar plexus or sacral plexus or both have to be used to warrant adequate analgesia. For example, acetabular or femoral neck injury may only require a lumbar plexus block, whereas knee/patellar injuries and ankle injuries require both femoral and sciatic nerves to be blocked [3].

Complications from continuous lower extremity nerve blocks are rare, although minor events like local inflammation and vascular puncture may be common [35]. The incidence of infection associated to nerve blocks is poorly defined in literature, and even if the rate of catheter tip contamination results between 23 and 57%, the incidence of clinical local infections is only 0–3% [3].

Among these complications, it is worth noting that psoas compartment block could lead to epidural and/or intrathecal injection of local anesthetics either due to catheter displacement or local anesthetic spread resulting in bilateral block and hypotension [34]. The risk of neuraxial block may be lowered by avoiding medial direction of the needle when psoas compartment block is performed.

See Table 22.2 for an overview of indications, pro, and cons of lower extremity blocks in trauma.

Table 22.2 Principal lower extremities blocks

Lower extremity blocks			
Psoas Compt. Lumbar plexus block	Hip, femur, knee fractures	Effective analgesia with low volume of anesthetic, possibility for continuous block	Need for lateral decubitus Possible peridural spread of anesthetic Possible vascular puncture in non compressible site
Fascia iliaca, 3 in 1 block, Femoral block	Hip, femur, knee, ankle fracture	Patient in the supine position, superficial blocks	Not all branches effectively blocked, may require larger volume of anesthetic or intravenous top-up
Sciatic block	Hip, leg, ankle fractures	Can be performed at different sites, effective analgesia with low volume of anesthetic, possibility of continuous block	As all peripheral nerve blocks, may mask compartment syndrome or neurological injuries

Case Study #3

A 50-year-old woman reports a right spiral femur shaft fracture after a ski accident. Her medical history includes hypertension and moderate chronic renal failure due to nephroangiosclerosis. She reports moderate pain at rest (NRS 5), and severe pain upon mobilization (NRS 10). She is scheduled for surgery on the following day. Discuss the options for pain management in this patient.

Discussion

Continuous lumbar plexus block at the psoas compartment is likely to be a very effective option for perioperative pain management in this patient, since it would provide effective analgesia for the pre-, intra-, and postoperative period. However, the patient has unbearable pain at mobilization, and muscle twitches associated with nerve stimulation is likely to cause great discomfort. Eco-guided Fascia Iliaca block might represent a valid alternative, since it can be performed in the supine position. The sensory block provided by fascia iliaca block is not as intense as with lumbar plexus block, and additional spinal or general anesthesia will be required for surgery.

Case Study #4

A 22-year-old male basketball player has to undergo reduction of a trimalleolar ankle fracture, and surgical repair is scheduled for the following days. Discuss the possible analgesic plan for this patient.

Discussion

Pain from trimalleolar ankle fracture is usually very intense, and peripheral nerve block is indicated. Femoral and Sciatic Bi-block will be necessary to provide complete analgesia and intraoperative anesthesia of the ankle. Femoral nerve block provides anesthesia of the medial aspect of the ankle, and can be maintained on a bolus-based fashion if total local anesthetic dose is a concern. Continuous sciatic block will cover the remaining of the surgical site.

Acute Compartment Syndrome

In lower extremity musculoskeletal trauma, acute compartment syndrome is a potentially devastating complication, whose incidence has been previously described as 7.3 per 100,000 in men and 0.7 per 100,000 in women [36]. The most common cause of acute compartment syndrome is usually fracture (69%) with tibial fracture being the most common injury; soft tissue injury without fractures is the most common cause (23%) with 10% of these occurring in patients taking anticoagulants or with a bleeding disorder [37]. Pain out of proportion to the injury, aggravated by passive stretching of muscle groups in the corresponding compartment, is one of the earliest and most sensitive clinical signs of compartment syndrome, even though it can be diminished or absent in an established compartment syndrome [38]. Anesthetic techniques have been reported to contribute to delay of the diagnosis [39]. Patients receiving epidural analgesia with local anesthetics and opioids have been reported to have a fourfold increased risk of neurologic complications than patients receiving systemic narcotics [40]: epidural analgesia with local anesthetics and opioids is therefore not recommended in at-risk patients. However, in the trauma patient, the absence of pain in a compartment syndrome is often caused by superimposed central or peripheral neural deficit, and pressure or firmness in the compartment remains the earliest and sometimes the only objective finding of early compartment syndrome [37]. Various methods of measuring tissue pressure have been described [41], and their application is recommended any time the clinical picture may be borderline or the patient examination can be ambiguous. In this scenario, a peripheral nerve block is not absolutely contraindicated, and each specific case should be addressed and discussed with the trauma team.

Case Study #5

A 25-year-old male soccer player is brought to the emergency department after a sport accident. He is diagnosed with tibial shaft fracture, with no

associated injuries. Neurological examination, although impaired by intense pain, reveals mild hypoesthesia of the toes and weakness on toe extension. Calcaneal skeletal traction is placed and the patient is scheduled for surgery on the following day. Intramedullary nailing is performed under general anesthesia, and is uneventful. On postoperative day 1, unbearable leg pain, paresthesia of the toes and frank motor deficit upon toe extension develop, the skin of the anterior leg compartment looks very tight and tissue pressure measure raise a high suspicion for acute compartment syndrome. Emergency fasciotomy is performed and the patient slowly recovers during the following days. At 3 months, he shows no signs of motor or sensory neurological deficit. Discuss the role of regional anesthesia for this patient.

Discussion

This patient presents several risk factors for acute compartment syndrome development (young athlete, sport injury, tibial shaft fracture). Furthermore, some degree of neurological impairment is already present upon admission. Regional anesthesia is not absolutely contraindicated in this setting, but the possibility that peripheral nerve block may mask or delay the diagnosis of ACS should be always kept in mind. If a perineural sciatic catheter block is inserted for continuous analgesia, it may be advisable to use a short-acting local anesthetic at low concentration (e.g., mepivacaine 0.5%) for continuous infusion, or a protocol based on discrete boluses, which can be administered by care-givers after physical and neurological examination.

Peripheral Nerve Blocks for the Management of Chest Trauma

Rib fractures are the most common thoracic injuries with an incidence ranging from 10% to almost 30% in patients after trauma. Mortality rate of patients with rib fractures range from 5.8% (single rib fracture) to 34.4% (multiple rib fractures) with an overall rate of 10%. Pain associated with rib fractures usually impairs pulmonary function and increases pulmonary morbidity. Therefore, appropriate pain management in a timely manner should be a core intervention in managing these patients. Various techniques have been used to manage pain in patients with rib fractures. These include systemic opioids, intercostal nerve blocks, epidural analgesia, and intrapleural and paravertebral nerve blocks. Clear superiority of one technique over the others in terms of efficacy and safety has not been demonstrated in the literature.

Intercostal Nerve Block

Both multiple single-shot injections with local anesthetics above and below the fracture site and continuous intercostal infusions have been shown to be successful in relieving pain caused by rib fractures. However, the exact mechanism underlying the intercostal analgesia is still unknown. Anesthetics are supposed to spread to the paravertebral space, epidural space, or a combination of both. An early case report showed paravertebral spread of local anesthetic after 20 mL of 0.5% bupivacaine was injected into the intercostal space. The same mechanism was confirmed by Mowbray et al. [42], who followed the spread of intercostal injection of 20 mL of bupivacaine and methylene blue through a catheter at thoracotomy in the paravertebral space. Indeed, a recent study verified a paravertebral catheter placement through the intercostal space. Therefore, it is possible that the major component of segmental block during intercostal catheterization may be secondary to paravertebral spread.

Intrapleural Nerve Block

There are many reports of the successful use of unilateral and bilateral interpleural blockade in patients with multiple rib fractures. This technique produces multi-segmental intercostal nerve blockade by gravity-dependent retrograde diffusion of the local anesthetics to reach the intercos-

tal nerve. A few studies have compared the interpleural nerve block with epidural block, paravertebral nerve block, and conventional opioids for analgesic efficacy in chest wall trauma with contrasting results. Some reasons for the conflicting results include catheter position, presence of hemothorax, location of fractured ribs, characteristics of local anesthetics, loss of local anesthetic through chest tubes, and dilution in pleural effusion. Among these reasons, it is interesting to notice that the location of rib fractures may affect the analgesic efficacy by interpleural nerve block. It appears that interpleural nerve block is most useful in clinical settings such as lateral or posterior rib fractures in the healthy chest cavity.

Epidural Nerve Block

Many studies have shown that thoracic epidural nerve block with local anesthetics, opioids, or a combination of both produces dramatic analgesia in patients with multiple rib fractures. Pulmonary function such as functional residual capacity, forced vital capacity, airway resistance, maximal inspiratory force, and maximal tidal volume is also reported improved by epidural analgesia. Although evidence that epidural nerve block improves subjective pain score and a variety of pulmonary functions in rib fracture patients is abundant and compelling, there is limited evidence that epidural nerve block improves outcomes. In a meta-analysis by Carrier et al. [43], evaluating seven randomized controlled studies (232 patients), epidural analgesia did not demonstrate significant benefits related to mortality, ICU length of stay, hospital length of stay, or duration of mechanical ventilation compared to other analgesia modalities, including opioid PCA or IV/IM opioid boluses and interpleural nerve blocks. Moreover, hypotension proved to be more frequent in patients receiving epidural analgesia. Thus, the evidence does not support the strength of the recent clinical practice guidelines on pain management in blunt thoracic trauma laid out by the East Association for the Surgery of Trauma (EAST), which stated that epidural analgesia may improve clinically significant outcomes in this population (Grade B recommendation) and that it should be considered the preferred analgesic modality (Grade A recommendation). In addition, in patients with mechanical ventilation and sedation, epidural analgesia is usually relatively contraindicated because of the patients' altered level of consciousness. Therefore, considering the potential for rare but major adverse events of epidural nerve block, clinically significant benefits other than better pain control need to be demonstrated to endorse the use of epidural nerve block as a standard of care in adult patients with traumatic rib fractures.

Paravertebral Nerve Block

Paravertebral nerve block is a regional anesthetic technique in which a single injection of anesthetic or a continuous infusion is delivered to the thoracic paravertebral space, producing a unilateral, multilevel, somatic, and sympathetic block. Since it is simple to perform, is associated with a low incidence of complications, requires no additional nursing surveillance, and has few absolute contraindications, paravertebral nerve block has recently been used to control pain in a variety of conditions involving the chest and abdomen. Evidence is also accumulating in support of this modality in patients with trauma, such as rib fractures: single injection of 0.5% bupivacaine into the thoracic paravertebral space led to significant improvement of pain scores and vital capacity in patients suffering from blunt or penetrating thoracic trauma; continuous paravertebral anesthetic (0.5% bupivacaine at 0.1–0.2 mL/kg/h for 4 days) in 15 patients with isolated unilateral rib fractures also provided significant improvements in analogue pain scores, vital capacity, peak expiratory flow rate, oxygen saturation (SaO_2), and O_2 index (PaO_2/FiO_2 ratio). Compared with epidural nerve block, paravertebral nerve blocks have been shown to produce comparable pain relief and similar improvements in respiratory function in patients with unilateral fractured ribs, although epidural was complicated by a higher incidence of hypotension. A downside of paravertebral

nerve blocks is that fewer practitioners are familiar with the technique, and large clinical trials are still lacking in the trauma population. Nevertheless, an increasing availability of data in the literature supports their efficacy in other clinical scenarios such as thoracotomies, which share a common pain mechanism with rib fractures, that is, intercostal nerve damage. Preoperative paravertebral nerve blocks have been demonstrated to significantly lower postoperative pain scores and better preserve postoperative lung function, measured by forced vital capacity, when compared to epidural analgesia. Moreover, a few review papers reported that paravertebral nerve block provided at least equally effective analgesia to epidural with fewer side effects, such as urinary retention, nausea/vomiting, and hypotension. Moreover, while epidural technique is contraindicated in the setting of coagulopathy due to the risk of hematoma and subsequent cord compression, the margin of safety is much higher with a paravertebral block and the more distensible paravertebral space.

See Table 22.3 for an overview of indications, pro, and cons of locoregional techniques in thoracic trauma.

Case Study #6

A 67-year-old male pedestrian is brought to the emergency department after being run over by a car. Medical history reveals hypertension and chronic atrial fibrillation treated with Warfarin. He reports a ruptured spleen and multiple (6th to 10th) left rib fractures with pneumothorax. After anti Vitamin K reversal with Prothrombin Complex Concentrate and chest tube positioning, he undergoes emergency splenectomy. During surgery he develops mild hypotension that is treated with crystalloids and transfusion of 3 Units of Red Blood Cells. On POD 1, despite endovenous analgesia with acetaminophen, NSAIDs, and morphine, he reports severe thoracic pain at inspiration, which limits chest expansion to shallow breathing, and becomes unbearable with deeper inspirations. Discuss alternative pain management options for this patient.

Discussion

This patient would certainly benefit from thoracic peridural catheter positioning. However, there are some concerns regarding this choice in this particular setting. The patient has suffered considerable blood loss and might still be at risk for developing hypotension with epidural analgesia. Careful titration with a short-acting local anesthesia may be warranted. Moreover, this patient will have to resume oral or subcutaneous (Low molecular Weight Heparin, LMWH) anticoagulant therapy for chronic atrial fibrillation. This is not an absolute contraindication for epidural catheter placement, with strict adhesion to correct timing between LMWH administration and catheter positioning/removal. Continuous paravertebral block may be a suitable alternative in this case, where hypotension and coagulopathy are major concerns. The same recommendations regarding catheter handling and anticoagulant timing should be followed, but paravertebral block may carry a lower risk of spinal hematoma in the case of accidental catheter removal. Other suitable alternatives are intrapleural block and intercostal nerve block, the latter being less desirable since it would require several injections to cover pain from multiple fractures.

Table 22.3 Principal blocks in thoracic trauma

Thoracic blocks			
Epidural	Rib fractures Laparotomy	Effective analgesia for multiple and bilateral injuries, improvement of pulmonary function	Risk of hypotension, spinal hematoma, Motor block, nausea
Paravertebral block	Rib fractures	Effective analgesia, lower risks compared to epidural	Multiple injections required if bilateral injuries
Intercostal, Intrapleural	Rib fractures	Lower risks compared to epidural	Multiple injections required if bilateral injuries

Clinical Pearls

- Improved pain management has been shown to reduce morbidity and improve long-term outcomes.
- Multimodal analgesia has been increasingly used to manage pain in trauma patients; this wide range of measures includes regional anesthesia procedures.
- Peripheral nerve blocks (PNBs) provide rapid and effective analgesia with less opioid-related side effects, such as nausea/vomiting, pruritis, urinary retention, constipation, sedation, and respiratory depression.
- Brachial plexus block usually provides adequate analgesia for upper extremity injuries.
- Use of ultrasound in upper extremity nerve blocks has improved the accuracy of needle insertion and catheter placement. Its advantages become especially obvious when it is hard to locate skin landmarks.
- Regional anesthesia at lower extremity usually includes lumbar plexus block, fascia iliaca block, and sacral plexus block at different sites.
- The posterior approaches to the lumbar plexus usually provide excellent analgesia with relatively small dose of local anesthetics, while fascia iliaca block has the advantage of keeping the patient in the supine position. Various approaches to sacral plexus block have also been used to provide analgesia to where sciatic nerve is distributed.
- Pain associated with rib fractures usually impairs pulmonary function and increases pulmonary morbidity: appropriate pain management in a timely manner should be a core intervention in managing these patients.
- Pulmonary function such as functional residual capacity, forced vital capacity, airway resistance, maximal inspiratory force, and maximal tidal volume is reported improved by epidural analgesia in patients with chest trauma.
- Paravertebral nerve blocks have been shown to produce comparable pain relief and similar improvements in respiratory function than epidural analgesia in patients with unilateral fractured ribs, although epidural was complicated by a higher incidence of hypotension.
- Paravertebral nerve blocks provide at least equally effective analgesia to epidural with fewer side effects, such as urinary retention, nausea/vomiting, and hypotension in patients with chest trauma.

Review Questions

1. How many deaths in the USA are caused by traumatic injuries every year?
   (a) 100,000
   (b) 500,000
   (c) 1,000,000
   (d) 250,000
   (e) 25,000
2. Which percentage of deaths is caused by traumatic injuries worldwide?
   (a) 0.1%
   (b) 8%
   (c) 25%
   (d) 0.25%
   (e) 12%
3. In which percentage of patients' inadequate pain control resulted in chronic pain syndromes after spinal cord injuries?
   (a) 20%
   (b) 30%
   (c) 40%
   (d) 50%
   (e) 70%
4. In the recent study by Whipple et al. [13] about adequacy of pain treatment in patients with multiple trauma injuries, which percentage of patients rated pain as moderate to severe?
   (a) 24%
   (b) 10%
   (c) 0.2%
   (d) 74%
   (e) 99%
5. Which upper extremity block can have Horner's syndrome as a complication?
   (a) Axillary block
   (b) Infraclavicular block
   (c) Supraclavicular block
   (d) Interscalene block
   (e) Ulnar block at the elbow

6. In lower extremity musculoskeletal trauma, acute compartment syndrome is a potentially devastating complication, whose incidence has been previously described as:
   (a) 7.3 per 100,000 in men and 0.7 per 100,000 in women
   (b) 0.7 per 100,000 in women and 7.3 per 100,000 in men
   (c) 30 per 100,000 in men and women
   (d) 0.5 per 100,000 in men and 0.01 per 100,000 in women
   (e) 70 per 100,000 in men and women
7. The most common cause of acute compartment syndrome is usually:
   (a) Burn injury
   (b) Soft tissue injury
   (c) Fracture
   (d) Crush injury
   (e) Tissue edema
8. The most common fracture that can be complicated by compartment syndrome is:
   (a) Humerus fracture
   (b) Scaphoid fracture
   (c) Tibial fracture
   (d) Femur fracture
   (e) Scapular fracture
9. A predisposing factor for compartment syndrome in soft tissue injuries is:
   (a) Regional anesthesia
   (b) Hypertension
   (c) Anticoagulants or bleeding disorders
   (d) Hypotension
   (e) Vascular diseases
10. One of the earliest and most sensitive clinical signs of compartment syndrome is:
    (a) Pain out of proportion
    (b) Motor and sensory block
    (c) Paresthesia
    (d) Absence of pain
    (e) Pallor
11. Mortality rate of patients with single rib fractures is around:
    (a) 1%
    (b) 10%
    (c) 6%
    (d) 50%
    (e) 0.1%
12. Mortality rate of patients with multiple rib fractures is around:
    (a) 90%
    (b) 80%
    (c) 70%
    (d) 35%
    (e) 25%
13. The East Association for the Surgery of Trauma (EAST) stated that one of the following may improve clinically significant outcomes in this population (Grade B recommendation) and that it should be considered the preferred analgesic modality (Grade A recommendation):
    (a) Intrapleural block
    (b) Epidural block
    (c) Intercostal block
    (d) Paravertebral block
    (e) Morphine PCA
14. When compared to epidural, paravertebral nerve blocks have been demonstrated to cause less:
    (a) Hypotension and urinary retention
    (b) Failed block
    (c) Compartment syndrome
    (d) Foot drop
    (e) Infections
15. Which of the following have been demonstrated to provide comparable analgesia?
    (a) Intercostal and intrapleural blocks
    (b) Intercostal and paravertebral blocks
    (c) Epidural and intrapleural blocks
    (d) Paravertebral and epidural blocks
    (e) Paravertebral and intrapleural blocks

Answers

1. a
2. b
3. e
4. d
5. d
6. a
7. c
8. c
9. c
10. a
11. c
12. d

13. b
14. a
15. d

References

1. Cordell WH, Keene KK, Giles BK, et al. The high prevalence of pain in emergency medical care. Am J Emerg Med. 2002;20(3):165–9.
2. Rosenberg A, Grande C, Bernstein R. Pain management and regional anesthesia in trauma. Philadelphia, PA: WB Saunders; 2000.
3. Clark L, Varbanova M. Regional anesthesia in trauma. Adv Anesth. 2009;27:191–222.
4. Davidson EM, Ginosar Y, Avidan A. Pain management and regional anaesthesia in the trauma patient. Curr Opin Anaesthesiol. 2005;18:169–74.
5. Cohen SP, Christo PJ, Moroz L. Pain management in trauma patients. Am J Phys Med Rehabil. 2004;83:142–61.
6. Tuman KJ, McCarthy RJ, March RJ, et al. Effects of epidural anesthesia and analgesia on coagulation and outcome after major vascular surgery. Anesth Analg. 1991;73:696–704.
7. Sorenson RM, Pace NL. Anesthetic techniques during surgical repair of femoral neck fractures: a meta-analysis. Anesthesiology. 1992;77:1095–104.
8. Liu SS, Carpenter RL, Macket DC, et al. Effects of perioperaitve analgesic technique on rate of recovery after colon surgery. Anesthesiology. 1995;83:757–65.
9. Yeager MP, Glass DD, Neff RK, et al. Epidural anesthesia and analgesia in high-risk surgical patients. Anesthesiology. 1987;66:729–36.
10. Woolf CJ, Salter MW. Neuronal plasticity: increasing the gain in pain. Science. 2000;288:1765–9.
11. Turner JA, Cardenas DD, Warms CA, et al. Chronic pain associated with spinal cord injuries: a community survey. Arch Phys Med Rehabil. 2001;82(4):501–9.
12. Rotondi AJ, Chelluri L, SIrio C, et al. Patients' recollections of stressful experiences while receiving prolonged mechanical ventilation in an intensive care unit. Crit Care Med. 2002;30:746–52.
13. Whipple JK, Lewis KS, Quebbeman EJ, et al. Analysis of pain management in critically ill patients. Pharmacotherapy. 1995;15:592–9.
14. American College of Surgeons (ACS) Committee on Trauma. Advanced trauma life support for doctors: ATLS student course manual. Chicago: ACS; 2008.
15. Rossaint R, Bouillon B, Cerny V, et al. The European guideline on management of major bleeding and coagulopathy following trauma: fourth edition. Crit Care. 2016;20:100.
16. Malchow RJ, Black IH. The evolution of pain management in the critically ill trauma patient: emerging concepts from the global war on terrorism. Crit Care Med. 2008;36(7 suppl):S346–57.
17. Gregoretti C, Decaroli D, Miletto A, Mistretta A, et al. Regional anesthesia in trauma patients. Anesthesiol Clin. 2007;25:99–116.
18. Richman JM, Liu SS, Courpas G, et al. Does continuous peripheral nerve blocks provide superior pain control to opioids? A meta-analysis. Anesth Analg. 2006;102:248–57.
19. Le-Wendling L, Enneking FK. Continuous peripheral nerve blocks for postoperative analgesia. Curr Opin Anaesthesiol. 2008;21:602–9.
20. Plunkett AR, Buckenmaier CC. Safety of multiple, simultaneous continuous peripheral nerve block catheters in patients receiving therapeutic low-molecular weight heparin. Pain Med. 2008;9:624–7.
21. Pogatzki-Zahn EM, Zahn PK. From preemptive to preventive analgesia. Curr Opin Anaesthesiol. 2006;19(5):551.
22. Moiniche S, Kehlet H, Dahl JB. A qualitative and quantitative systematic review of preemptive analgesia for postoperative pain relief: the role of timing of analgesia. Anesthesiology. 2002;96:725–41.
23. de Tran QH, Munoz L, Russo G, et al. Ultrasonography and stimulating perineural catheters for nerve blockade: a review of evidence. Can J Anaesth. 2008;55:447–57.
24. Plunkett AR, Brown DS, Rogers JM, et al. Supraclavicular continuous peripheral nerve block in a wounded soldier: when ultrasound is the only option. Br J Anaesth. 2006;97:715–7.
25. Delaunay L, Chelly JE. Indications for upper extremity blocks. In: Chelly JE, editor. Peripheral nerve blocks: a color atlas. Philadelphia, PA: Lippincott Williams and Wilkins; 1999. p. 17–27.
26. Sala-Blanch X, Lazaro JR, Correa J, et al. Phrenic nerve block caused by interscalene brachial plexus block: effects of digital pressure and a low volume of local anesthetic. Reg Anesth Pain Med. 1999;24:231–5.
27. Jandard C, Gentili ME, Girar DF, et al. Infraclavicular block with lateral approach and nerve stimulation: extent of anesthesia and adverse effects. Reg Anesth Pain Med. 2002;27:37–42.
28. Taras JS, Behrman MJ. Continuous peripheral nerve block in reimplantation and revascularization. J Reconstr Microsurg. 1998;14:17–21.
29. Fletcher AK, Rigby AS, Boughrouph J, et al. Three-in-one femoral nerve block as analgesia for fractured neck of femur in the emergency department: a randomized, controlled trial. Ann Emerg Med. 2003;41:227–33.
30. Sia S, Pelusio F, Marbagli R, et al. Analgesia before performing a spinal block in the sitting position in patients with femoral shaft fracture: a comparison between femoral nerve block and intravenous fentanyl. Anesth Analg. 2004;98:1785–8.
31. Chesters A, Atkinson P. Fascia iliaca block for pain relief from proximal femoral fracture in the emergency department: a review of the literature. Emerg Med J. 2014;31:84–7.

32. Wathen JE, Gao D, Merritt G, Georgopoulos G, Battan FK. A randomized controlled trial comparing a fascia iliaca compartment nerve block to a traditional systemic analgesic for femur fractures in a pediatric emergency department. Ann Emerg Med. 2007;50(2):162–71.
33. Capdevila X, Biboulet P, Bouregba M, et al. Comparison of the three-in-one and fascia iliaca compartment blocks in adult: clinical and radiographic analysis. Anesth Analg. 1998;86:1039–44.
34. Chelly JE, Casati A, Al-Samsam T, et al. Continuous lumbar plexus block for acute postoperative pain management after open reduction internal fixation of acetabular fractures. J Orthop Trauma. 2003;17(5):362–7.
35. Neuburger M, Buttner J, Blumenthal S, et al. Inflammation and infectious complications of 2295 perineural catheters: a prospective study. Acta Anaesthesiol Scand. 2007;51:108–14.
36. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome: who is at risk? J Bone Joint Surg Br. 2000;82:200–3.
37. Olson SA, Glasgow RR. Acute compartment syndrome in lower extremity musculoskeletal trauma. J Am Acad Orthop Surg. 2005;13:436–44.
38. McQueen MM, Christie J, Court-Brown CM. Acute compartment syndrome in tibial diaphyseal fractures. J Bone Joint Surg Br. 1996;78:95–8.
39. Hyder N, Kessler S, Jennings AG, et al. Compartment syndrome in tibial shaft fracture missed because of a local nerve block. J Bone Joint Surg Br. 1996;78:449–500.
40. Strecker WB, Wood MB, Bieber EJ. Compartment syndrome masked by epidural anesthesia for postoperative pain. J Bone Joint Surg Am. 1986;68:1447–8.
41. Moed BR, Thorderson PK. Measurement of intracompartmental pressure: a comparison of the slit catheter, side-ported needle and simple needle. J Bone Joint Surg Am. 1993;75:231–5.
42. Mowbray A, Wong KK, Murray JM. Intercostal catheterisation. An alternative approach to the paravertebral space. Anaesthesia. 1987;42(9):958–61.
43. Carrier FM, Turgeon AF, Nicole PC, Trépanier CA, Fergusson DA, Thauvette D, Lessard MR. Effect of epidural analgesia in patients with traumatic rib fractures: a systematic review and meta-analysis of randomized controlled trials. Can J Anaesth. 2009;56(3):230–42.

23 Preemptive Analgesia, Regional Anesthesia, and Prevention of Chronic Postoperative Pain

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Historical Background and Foundations in Preemptive Analgesia

In 1914, Crile and Lower stated that shock was any form of vital energy expenditure in the human body such as traumatic, hemorrhagic, or emotional, and they proposed that these states of shock "cause physical alterations in the cells of the brain." They also theorized that these "cells, which reach a certain degree of alteration [could] not be restored" once undergoing a certain level of injury [1]. Furthermore, they sought to find a solution to this permanent change that can occur in the nervous system in response to pain which led to the formulation of the concept of preemptive analgesia [1]. Since then, Crile, Lower, and future generations of clinicians have worked to develop best strategies or techniques of preventing post-procedural pain through combinations of anesthetics and analgesics. Preemptive analgesia is focused, therefore, on prevention of postoperative pain as opposed to just treatment [2]. Although Crile and Lower laid these foundations over 100 years ago, research still continues to achieve a better understanding of pain, its prevention, and its most effective treatments.

Physiology of Central and Peripheral Analgesia

Sensitization is an increase in the intensity and duration of painful sensation caused by continuous application of a noxious stimulus [3].

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_23

Sensitization has been identified in both the peripheral and central nervous systems and is a testament to the malleability of the nervous system. Peripheral sensitization starts at cutaneous nerve endings; specifically, nociceptors along with the accumulation of neuropeptides and inflammatory mediators. For instance, an incision site results in the release of inflammatory mediators such as bradykinin, substance P, prostaglandins including thromboxanes, platelet-activating factor, leukotrienes, and nitric oxide, along with simultaneous activation of nociceptors by neurotransmitters [4]. The fast, myelinated Aδ fibers transmit an initial pain sensation when activated that is then followed by the more dull sense of pain associated with the slow, unmyelinated C nerve fiber activation [5]. These signals are transmitted to the dorsal horn of the spinal cord where they are carried to the brain via excitatory signals and interneurons that modulate signals to second order neurons [6]. These inflammatory mediators and processes work in tandem in the periphery and can lead to states of hyperalgesia or allodynia with sustained exposure to noxious stimuli. This "sensitization soup" of inflammatory mediators and neuropeptides in the periphery reduce the threshold at which the primary afferent terminals are activated [2]. This lowering of the threshold can result in acute and chronic hypersensitivities to noxious stimuli.

Similarly, exposure to noxious stimuli for prolonged periods or of high frequency in the periphery can lead to changes in the central nervous system pain processing centers. These changes typically lead to a reduced threshold for initiating pain and increased perception of pain. Central sensitization is an activity-dependent process that takes place under the influence of peripheral afferent nociceptor signaling [3]. Woolf describes the theory of central sensitization using the wind-up phenomenon [7]. Wind-up is defined as an increase in the number of action potential discharges released with repeated stimulation by a signal of unchanging strength and frequency [7]. Increasing the amplitude of each produced signal is another phenomenon associated with sensitization that is called cumulative depolarization. This depolarization activates *N*-methyl-D-aspartic acid (NMDA) and tachykinin receptors in the spinal neurons. This activation alters second messenger levels and subsequently protein kinase activity. This leads to expressed changes in the spinal neurons' response to primary afferent signals [2]. The wind-up and cumulative depolarization are not necessarily related, and varying neurons in the dorsal root can achieve both, one, or none of these methods for sensitization; however, both of these mechanisms lead to hyperalgesia [3].

Sensitization is an important aspect to consider when determining an appropriate analgesic plan for patients. A myriad of factors are at play related to postoperative pain and an individual patient. The duration of sensitization is categorized as either short-term or long-term sensitization; the former lasting seconds to minutes and the latter persisting for hours to months. Long-term sensitization appears to be very similar to the type of pain associated with injury, e.g., induced chronic pain [3]. The idea of blocking these pain signals or decreasing the perception of pain in the periphery which would lead to decreased acute or chronic pain has led to the development of the concept behind preemptive anesthesia.

Preemptive Analgesia

The idea of preemptive anesthesia originated as a means to decrease postoperative pain in patients. The increased pain experienced at the site of surgery or the uninjured surrounding area is related to sensitization, both centrally and peripherally. Once sensitization is established, pain-controlling treatments become less effective and must be used for longer periods of time postoperatively [2]. This realization led to research in prevention of postoperative pain with preemptive analgesia. Crile and Lower were the first to theorize that local anesthesia along with general anesthesia may lead to more successful surgical outcomes long-term by reducing postoperative pain.

Preoperative treatment has proven to be effective in the reduction of postoperative or post-injurious pain in some animal models; however, clinical trials are still under way to determine the

success rates of preemptive analgesia as well as which medications or techniques which are the most efficacious in preventing chronic postoperative pain. Gadek et al. completed a clinical trial in patients receiving hallux valgus surgery, which showed a statistically significant decrease in postoperative pain and rescue analgesia in patients who received local anesthetic preoperatively versus patients who received placebo injection [8]. However, a meta-analysis on the efficacy of pretreatment with oral NSAIDs in the prevention of postoperative pain after molar extraction showed no significant benefit compared to control groups [9].

Many agents can be used as preemptive analgesic treatments. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used to decrease the peripheral prostaglandin release at the incision site leading to decreased sensitization [2]. Local anesthetics inhibit action potential activation in peripheral afferent nerve fibers which leads to a decrease in sensation. Opioids act centrally by decreasing the presynaptic release of neurotransmitters and postsynaptic hyperpolarization of the dorsal horn neuron membranes. The use of NMDA and tachykinin receptor antagonists could potentially prevent the changes in protein expression that typically occur during central sensitization [2]. Other treatment ideas exist that target different areas of this system. All of these treatments aim to either decrease pain perception and signaling thereby preventing the development of sensitization or inhibit the changes that occur on a cellular level during sensitization.

Another facet of preemptive analgesia is the duration of pre-, peri-, and postoperative treatment with the chosen analgesic. How long must a patient receive preemptive analgesia to successfully thwart the effects of peripheral and central sensitization? Two phases of nociceptor activation exist with tissue damage. The initial input results directly from the injury to the tissue such as caused by a surgical incision. The second phase of sensory input results from the release of inflammatory chemicals within the tissue which contribute to activation and sensitization of the peripheral nociceptors [2]. Therefore, the establishment of sensitization can continue postoperatively as opposed to just perioperatively related to this variable state of inflammation. O'Connor and Abram demonstrated that the administration of intrathecal morphine before formalin injection in rats but then reversed with naloxone before the onset of the second phase of sensory input still enabled suppression of sensitization. These results show that adequate suppression of the first sensory input phase could prevent the second phase of input and thereby sensitization overall; however, the study group that showed the highest level of sensitization suppression received halothane inhalation along with intrathecal morphine. This highlights the synergistic or additive effects of general anesthesia along with specific preemptive analgesics [10].

Clinical Evidence

The literature surrounding preemptive analgesia and its conclusions are variable depending on several factors, although the most current literature is inconclusive. A meta-analysis reviewed 66 randomized controlled trials and data from 3261 patients and specifically compared preoperative analgesic interventions with postoperative analgesic interventions via the same route for five different analgesic modalities and showed mixed results. Results showed that while there were improvements in patient pain intensity scores for preemptive epidural analgesia, intervention via preemptive local anesthetic wound infiltration and NSAID administration did not improve postoperative pain scores [11]. Similarly, a prospective double-blind study was conducted in patients undergoing total abdominal hysterectomies. Patients were allocated into four groups with four different interventions, either pre- and postoperative 0.9% saline, preoperative saline and postoperative local anesthetic mixture (10 mL 2% lidocaine added to 10 mL 0.5% bupivacaine), preoperative local anesthetic mixture and postoperative saline, or preoperative and postoperative local anesthetic mixture. The results showed no significant difference between the amount of morphine used by any of the groups postoperatively, and likewise there was no difference in the intensity of

pain between any groups. Local anesthetic infiltration was thus concluded to not reduce the intensity of postoperative pain in this patient population [12]. Of note, several studies have produced opposite conclusions regarding local anesthetic infiltration, which are named and discussed further in this chapter.

There does appear to be variability with medications chosen for preemptive analgesia and with the type of technique chosen for preemptive analgesia. Patients recieving pre-incisional intravenous dexketoprofen trometamol, or acetaminophen were compared to a control group for elective septorhinoplasty. Sixty patients were followed with result parameters of patient satisfaction, tramadol usage postoperatively, and overall postoperative pain. There was no difference in tramadol consumption between the control group and the acetaminophen group. However, both of these groups consumed more tramadol than the group which received IV dexketoprofen. First analgesic requirement time and side effect results were no different between groups. The study concluded that both preemptive IV dexketoprofen and acetaminophen were effective for the reduction of postoperative pain, however also that preemptive dexketoprofen trometamol was more effective than acetaminophen for postoperative analgesia, highlighting the importance of medication choice for preemptive analgesia [13].

There is also a relationship between the type of procedure chosen and medications. A study following lumbar laminectomy patients suggested that preemptive infiltration of the wound site with levobupivacaine alone provided effective pain control with reduced opiate use after operative measures. Further, the data indicated that preemptive injection of levobupivacaine or levobupivacaine-methylprednisolone into the muscle near the operative site provided more effective analgesia than infiltration of these drugs and wound closure [14]. In this regard, a separate study came to the conclusion that the administration of preemptive bupivacaine did not appear to have any advantage over postoperative administration in patients undergoing ambulatory breast biopsy [15]. The literature involving preemptive analgesia seems to vary with medication choice, technique, and operative measure.

Local Wound Infiltration

Local wound infiltration is the continuous administration of an anesthetic or pharmacologic agent into a wound to decrease pain. Most often it is used in conjunction with other techniques such as preoperative or intraoperative nerve blocks and is quickly becoming a well-known combination technique for more efficient postoperative analgesia. For example, a widely used technique for patients undergoing thoracotomies is intraoperative intercostal block combined with catheter wound infiltration that provides prolonged analgesia [16]. Local wound infiltration for small incisions performed under monitored anesthesia is also widely used for surface injuries, such as ring blocks for digits and circumcision [17]. Larger operative measures require larger amounts of medication for local wound infiltration, and success has been reported in hysterectomies, cesarean sections, abdominal surgeries, laparoscopic procedures, abdominal surgeries, and many others [18–26]. As discussed earlier, the efficacy of preemptive local anesthetic wound infiltration may depend on the medication used, the amount of medication, operative or procedural measure, and technique used.

In addition to analgesia, local anesthetic wound infiltration has other notable benefits. Local anesthetics exhibit bacteriostatic and bactericidal actions. In one particular study, human breast cancer cells and mammary epithelial cell lines were exposed to lignocaine and bupivacaine. There was inhibition of cell viability, with apoptosis of the breast tumor cells. These findings point to the option of local anesthetics for skin infiltration during oncological surgeries of the breast [27, 28].

There are considerable benefits of local wound infiltration. However, there are questions of the efficacy of local anesthetic agents when administered alone, without conjunction of other techniques. Mixed results have been reported, and most studies with local anesthetics as a group

alone also administer NSAIDs and vasoconstrictors for additional pain relief [29]. For example, a meta-analysis suggested preemptive local anesthetic wound infiltration improved analgesic consumption and time to first rescue analgesic request, but not overall postoperative pain scores [11, 30]. An additional concern is the effect of local anesthetics on inflammatory responses to injury. In an RCT of 38 patients undergoing a cesarean section, local wound anesthetic resulted in significant reductions in levels of IL-10 and increases of substance P in the wounds of the bupivacaine-treated group compared to a saline-treated group. The result has been replicated in rats, with impairment of wound healing overall. The study above showed there is increased risk with continuous infusions of local anesthetic, and the same can be attenuated with lower concentrations of local anesthetics. There are, however, no definitive statistically convincing evidence against the use of local anesthetics for preemptive analgesia when their analgesic benefits are taken into account [31, 32].

Regional Anesthesia

Utilization of regional anesthesia has grown tremendously over the past several years as the use of ultrasound has become standard practice. Regional anesthesia encompasses peripheral nerve, intravenous, and neuraxial blockade. Studies by Liu et al. demonstrated that peripheral nerve blocks were associated with reduced pain scores and a decreased need for postoperative analgesics [33]. Also, patient satisfaction was greater with peripheral nerve blockade and a multimodal analgesic approach compared to conventional general anesthesia [34].

Peripheral nerve blocks are used for a wide variety of procedures to provide intraoperative anesthesia and postoperative analgesia and for the diagnosis and treatment of chronic pain syndromes. They have become exponentially more popular in the outpatient setting as ultrasound usage and competence continues to increase among providers. Ultrasound offers the potential for a safer, faster, and overall more precise and efficacious analgesic [35]. Blockade for surgical procedures can be accomplished via single-shot techniques or insertion of continuous perineural catheters. Perineural catheter insertion has several benefits in the ambulatory setting. Continuous infusion of local anesthetics can improve pain control at home after patient discharge and minimize unwanted effects of parenteral opioid usage [33].

Multiple studies have shown that regional anesthesia can prevent surges in both stress hormone and catecholamine levels in response to surgical stimulation [36, 37]. While this observation correlates closely with the concept of preemptive analgesia, clinical studies thus far have shown varying degrees of success. Adequacy of pain control is dependent on multiple factors including concentration and duration of local anesthetic used, proficiency of the clinician, and type of surgery. Whether the block is performed pre-incision or post-incision does not appear to have a major influence on postoperative pain scores. The use of preemptive intercostal block was compared to postoperative intercostal block for thoracotomy pain [38]. It was found that preemptive blockade only moderately decreased pain in patients taking a vital capacity breath during the first 48 h compared to the postoperative group. There was no significant difference in other measures studied including VAS scores, analgesic consumption, and extent or duration of blockade [38]. The use of perineural catheters with continuous administration of local anesthetic is associated with the greatest amount of pain control and patient satisfaction [36]. However, more research needs to be done to study these modalities as they relate specifically to preemptive analgesia.

Epidural and Caudal Analgesia

Neuraxial blockade includes intrathecal, epidural, and combined spinal-epidural techniques. Various combinations of local anesthetics, opiates, and other medications can be utilized in each of these modalities to maximize analgesic efficacy. Epidural techniques with catheter placement allow for the administration of medication

anytime in the perioperative period and are thus the most amendable in regard to studying preemptive analgesia. A meta-analysis of 66 studies found that epidural techniques had the most profound effects as it pertains to preemptive analgesia. However, other meta-analyses have failed to show any correlation between epidural anesthesia and preemptive analgesia [39]. Theoretically, neuraxial blockade should be an ideal method for preventing central sensitization and resulting in preemptive analgesia. However, definitive evidence of preemptive benefit with neuraxial blockade alone has been difficult to prove, and more research needs to be done.

NSAIDs

NSAIDs exert an analgesic effect at the site of injury by targeting COX enzymes to inhibit prostaglandin production, preventing peripheral sensitization, and reducing pain hypersensitivity [2]. Induction of cyclooxygenase (COX) enzymes occurs after tissue injury leading to increased conversion of arachidonic acid to prostaglandins and causing pain hypersensitivity in the postoperative period. Prostaglandins released at the surgical site produce peripheral sensitization by increasing the excitability and decreasing the pain threshold of nociceptive nerve terminals [40], generating hyperalgesia at the site of injury (primary hyperalgesia). The increase in peripheral nociceptor afferent activity causes central sensitization of neurons in the spinal cord, resulting in hyperalgesia in the uninjured tissue surrounding the surgical site (secondary hyperalgesia) [41].

Tissue injury also increases COX-2 expression within the central nervous system (CNS) producing elevated prostaglandin levels in the CNS and contributing to the production of postoperative pain hypersensitivity [42]. Surgical incision in rats has been demonstrated to produce an increase in spinal cord levels of COX-2 [43], and increased prostaglandin levels in the cerebrospinal fluid (CSF) have been shown to have a positive correlation with postoperative pain [44]. When administered directly to the CNS in mice, prostaglandins increase the response to painful stimuli (hyperalgesia) as well as causing pain with normally painless stimuli (allodynia) [45]. NSAIDs also exert an analgesic effect by acting within the CNS to inhibit the increased excitability of spinal cord neurons, preventing central sensitization [2] by inhibiting COX-2 expression within the spinal cord. Intrathecal administration of a COX-2 inhibitor decreases spinal prostaglandin levels and reduces hyperalgesia in rats [46]. In patients undergoing total hip arthroplasty, preoperative administration of a COX-2 inhibitor decreases prostaglandin levels in both the CSF and the surgical site along with decreased postoperative pain [44].

A reduction in postoperative pain and opioid consumption has long been evident with the postoperative administration of NSAIDs [47, 48]. More recently, studies have examined the effects of preoperative NSAID administration with inconsistent results. Many studies have found a beneficial effect, with preemptive NSAIDs reducing postoperative pain and opioid consumption compared to placebo [49–51], while others have found no benefit [52]. Preemptive NSAID use may be more beneficial than postoperative NSAID administration by acting before the start of surgical tissue injury to inhibit the release of prostaglandins, preventing peripheral and central sensitization from occurring and thus decreasing postoperative hyperalgesia [53]. Recent studies have supported this theory, demonstrating greater analgesic efficacy with preoperative NSAID administration than administration of the same medication postoperatively [53, 54].

Reviews of the literature have also shown different conclusions on the preemptive use of NSAIDs. A review by Moiniche et al. [39] of 20 randomized controlled trials of preemptive NSAID use in various surgical procedures found only four trials demonstrating an analgesic benefit with preoperative versus postoperative NSAID use, suggesting no preemptive benefit. Conversely, Ong et al. [11] analyzed 17 trials and found that although preemptive NSAID use did not significantly decrease postoperative pain, it did significantly decrease postoperative analgesic consumption and increased the time to first analgesic. A recent review by Nir et al. [55] of 24 randomized controlled trials found a significant

analgesic benefit with preemptive NSAID use. Twelve of the analyzed trials used preemptive COX-2 inhibitors, which produced a significant decrease in analgesic consumption. The other NSAID classes did not demonstrate a significant preemptive analgesic effect, suggesting that COX-2 inhibitors are more effective when administered preoperatively in reducing hyperalgesia after surgery than other NSAIDs.

While NSAIDs have demonstrated a preemptive analgesic benefit alone, they may also have a significant benefit as part of a multimodal preemptive analgesic regimen composed of agents acting on different components of the pain pathway [56, 57].

Opioids and Other Pharmacological Agents

There has been an increase in the use of postoperative analgesics in an attempt to decrease postoperative pain and opioid consumption [55]. Opioids are extremely useful analgesics in the treatment of pain during the postoperative period. Preemptive opioids can decrease postoperative hyperalgesia and allodynia by preventing central sensitization [58]. Results from trials on the preemptive use of opioids have been contradictory. While some studies have demonstrated a reduction in postoperative pain and opioid consumption with preemptive opioids compared to placebo [59, 60], others have found no preemptive analgesic benefit [61]. The reviews by Moiniche et al. [39], Ong et al. [11], and Nir et al. [55] however, have all reached the same conclusion that preemptive opioids have no analgesic benefit compared to postoperative opioid administration.

The NMDA receptor antagonist, ketamine, is used intraoperatively as an anesthetic, while the use of NMDA receptor antagonists as analgesics remains unclear [62]. Activation of NMDA receptors in the CNS occurs with the increase in afferent activity of peripheral nociceptors after surgical tissue injury, leading to central sensitization [63]. Preemptive NMDA receptor antagonist administration may decrease the central sensitization and hyperalgesia that occurs after surgery [63]. While some studies have failed to find a preemptive analgesic benefit with either ketamine or dextromethorphan [62, 64], others demonstrated a decrease in postoperative pain and postoperative opioid consumption [65, 66]. A randomized controlled trial in patients undergoing abdominal surgery demonstrated preemptive dextromethorphan to be more effective in decreasing postoperative pain and opioid consumption compared to both placebo and postoperative dextromethorphan administration [62]. Despite some promising studies, reviews by Moiniche et al. [39] and Ong et al. [11] both concluded that there was no preemptive analgesic benefit with NMDA receptor antagonists.

The gabapentinoids are structural analogs of gamma-aminobutyric acid which bind voltage-dependent calcium channels within the CNS, modifying the release of excitatory neurotransmitters and potentially preventing central sensitization [67]. Studies have demonstrated a preemptive analgesic benefit with both gabapentin [68] and pregabalin [69, 70], while some studies have failed to find a beneficial effect [67, 71]. Two recent meta-analyses concluded that preemptive gabapentin use produced a significant decrease in postoperative pain and opioid consumption [72, 73]. The preemptive analgesic benefit of pregabalin remains less clear. While a recent meta-analysis by Mao et al. [74] concluded that there is a benefit with either gabapentin or pregabalin use preemptively in patients undergoing total hip arthroplasty, the review by Nir et al. [55] only found a preemptive analgesic benefit with gabapentin use and not with pregabalin.

Preemptive analgesia may be more effective in reducing postoperative pain than the traditional postoperative approach, and a review of the current literature suggests that COX-2 inhibitors and gabapentin are the most promising preemptive analgesic agents [55].

Chronic Pain Syndromes Following Surgery

Of the chronic pain syndromes, the four syndromes that the healthcare provider is likely to encounter include complex regional pain syndrome (CRPS), phantom limb pain (PLP),

chronic donor site pain, and post-thoracotomy pain syndrome and will be discussed in detail below.

Complex Regional Pain Syndrome

CRPS is a neuropathic disorder with a constellation of sensory, motor, and autonomic manifestations. It is accompanied by typical neuropathic symptoms such as burning, hyperalgesia, and allodynia. Autonomic features can manifest through altered sweating, changes in skin color, and changes in temperature. Trophic changes of the skin and nails may also be present, but these are rare. The range of symptoms of CRPS often varies between individuals and can even change over time.

There are approximately 50,000 new cases of CRPS each year within the USA. The risk for developing CRPS is higher in women compared to men (3–4:1) and increases with age with the highest incidence in fifth to seventh decade [75, 76]. Trauma is the most common cause for the development of CRPS with 40% of these cases developing after a fracture resulting in an overall incidence of 3.8–7.0%. Symptoms usually begin 4–8 weeks after the injury [77]. CRPS has also been known to arise spontaneously in 10–16% of cases [76]. Other insults such as surgery, contusions, sprains, and crush injuries can also result in CRPS. The upper extremities are twice as likely to be affected compared to the lower extremities [78]. It is also more likely for symptoms to develop distally as opposed to proximally as rarely does CRPS develop in sites such as the shoulder or knee. Any psychological factor or personality trait that predisposes to the development of CRPS is yet to be identified [79]. Mild cases can heal spontaneously after several weeks, and 85% of patients will have an improvement in their condition within the first 12 months. If there is no improvement of symptoms within the first year, it is less likely that the symptoms will resolve [76].

The pathophysiological mechanisms behind CRPS are complex and considered to be multifactorial. Some of the proposed pathophysiologic mechanisms include classic and/or neurogenic inflammation, maladaptive neuroplasticity, autonomic nervous system dysfunction, and central sensitization [79]. Patients that have had CRPS for longer periods of time are more likely to report symptoms of central sensitization. This may partially explain why peripherally targeted treatments are less effective in these patients [80].

Diagnosis of CRPS is clinical and based solely on the history and physical exam. There is no gold standard test for diagnosing CRPS. However, X-rays and bone scintigraphy can assist with diagnosis in some cases. The clinical diagnostic criteria are outlined in the Budapest criteria below. CRPS is separated into several subtypes, which are classified by the absence (CRPS-I) or presence (CRPS-II) of a known peripheral nerve injury identifiable with either EMG or nerve conduction studies [79]. A third subtype is known as CRPS-NOS (not otherwise specified), composed of patients that have been diagnosed with CRPS but do not fulfill the current Budapest clinical diagnostic criteria. CRPS can also present with different phenotypes. This is based upon symptomatology and skin temperature (warm or cold) at onset. The warm type accounts for approximately 70% of cases and is characterized with a warm, red, and swollen affected limb. The cold type represents around 30% of cases and presents with a limb that is cold and either dark or pale. Prognosis is often more favorable for those with the warm phenotype [77].

Once diagnosed, patients should be treated early and aggressively in an attempt to avoid the development of chronic symptoms and involve a multidisciplinary approach [81]. Unfortunately, there is little evidence to support the effectiveness of many current CRPS therapies. Some guidelines suggest physical therapy modalities as a first-line treatment. However, a Cochrane review could not find convincing evidence for the effectiveness of physical therapy interventions. The authors did find some low-quality evidence that graded motor imagery, and mirror therapy may provide benefit in reducing pain and disability in CRPS-I [82]. Pharmacological treatments include calcitonin, bisphosphonates, baclofen, NSAIDs, vasodilating drugs, gabapentin, antidepressants, opioids, and the free radical scavengers dimethyl sulfoxide (DMSO), vitamin C, and *N*-acetyl-cysteine [78]. Many of these

therapies are lacking sufficient evidence for their effectiveness, but are still widely used in clinical practice. There is evidence supporting the use of bisphosphonates, which may be most beneficial in patients with disease duration of fewer than 12 months. Calcitonin appears to offer the most benefit in more advanced stages of CRPS when utilized as a short-term medication [83]. If there is no response to conventional therapy after 12–16 weeks, then interventional techniques should utilized [79]. Other possible treatment modalities include physical therapy, occupational therapy, and spinal cord stimulators. Local anesthetic sympathetic blockade (LASB) has also been used for the treatment of CRPS. A Cochrane review did not conclude on the efficacy of LASB as an effective treatment. The authors did comment that "the existing evidence is not encouraging" [84]. Spinal cord stimulators (SCS) and intrathecal drug delivery systems are often used as a result of ineffective medical treatments [75]. SCS can reduce pain scores and improve quality of life and patient satisfaction [85]. Newer techniques such as intravenous immunoglobulin (IVIG) therapy and plasma exchange have shown promise in providing relief in a small subset of patients with chronic disease [86].

Budapest criteria for clinical diagnosis of complex regional pain syndrome
1. Continual pain that is disproportionate to the inciting event
2. Patient must report at least one symptom in three of the four following categories
3. Patient has at least one sign in two or more of the following categories
Sensory: Hyperalgesia (to pinprick) and/or allodynia (to deep somatic pressure and/or light touch and/or temperature sensation) and/or kinesthetic allodynia (pain on joint movement)
Vasomotor: Temperature asymmetry greater than 1 deg. Celsius and/or skin color asymmetry and/or skin color changes (red, blue or blotchy)
Sudomotor/edema: Sweating changes and/or sweating asymmetry and/or edema
Motor/trophic: Decreased range of motion and/or motor dysfunction (weakness, dystonia, tremor) and/or trophic changes (skin, nail, hair)
4. Signs and symptoms cannot be better explained by another diagnosis
Adapted from [79, 87]

Phantom Limb Pain

Phantom limb pain (PLP) is pain experienced in the area of a missing limb following traumatic or surgical amputation. This is different from phantom limb sensation (PLS) and stump pain. PLS is any sensation felt in the area of the absent limb that is not painful. Sensations of temperature change, tingling, itching, or movement are often described. Stump pain is pain that is localized to the site of amputation/stump. PLP is not just associated with a limb but has been seen with digits, the nose, teeth, tongue, eyes, breast, and even menstrual cramps after hysterectomy [88]. PLP can also present in patients with congenital inexistence of limbs. The prevalence of PLP ranges from 50 to 85%. Most cases (75%) begin within 24 h to 1 week of amputation [88, 89]. Cases may also present years later, but the prevalence has been shown to decrease with time. Risk factors that have been associated with the development of PLP include female gender, preamputation pain, post-amputation pain, loss of an upper extremity, and time since amputation [88]. In one study population, patients with an amputation of the upper extremity developed PLP at a higher rate and experienced more frequent and severe pain [90].

The underlying mechanisms for PLP are still unclear, although originally it was believed that PLP stemmed from psychogenic causes and pathological changes in the area of the stump [91]. However, it is now considered to be a neuropathic type pain due to the involvement of both the central and peripheral nervous systems [92]. Peripheral mechanisms involve neuronal injury and deafferentation leading to the formation of neuromas. These neuromas have increased expression of sodium channels, which leads to spontaneous firing and hyperexcitability resulting in the sensation of pain. Central mechanisms involve in PLP are cortical reorganization, windup, and central sensitization [88].

Currently there are no guidelines for the management of PLP. Most treatment recommendations are extrapolated from the recommendations based on management of neuropathic pain [88, 91]. Treatment of PLP is based on a multidisciplinary approach that includes regional techniques,

desensitization, pharmacotherapy, adjuvant therapies, and psychotherapy. Results for preemptive approaches to reduce the risk and severity have been mixed, and it may not be possible to prevent the development of PLP [93]. Some evidence suggests nerve blocks and epidurals within the first 3 days after amputation do not prevent the occurrence of PLP [94]. There are several studies that have shown regional anesthesia to be effective in prevention and treatment of PLP. In one study perineural infusions of 0.5% ropivacaine for periods ranging from 4 to 83 days were successful at both treating and preventing PLP [89]. Another study used optimized epidural analgesia and PCA beginning 48 h preoperatively and continued 48 hours postoperatively which decreased PLP at 6 months [95]. However, a meta-analysis from 2015 concluded that the use of peripheral nerve catheters did not affect the occurrence of phantom limb pain [96]. Due to either design flaws or small sample sizes, more randomized controlled trials are needed to help determine the effectiveness of regional anesthesia in the prevention of PLP [97]. In patients with established PLP, the use of spinal or epidural anesthesia has been associated with exacerbation and/or recurrence of symptoms. Due to these reports, some clinicians will avoid the use of neuraxial anesthesia in these patients.

Many different pharmacological agents have been employed for the treatment of PLP. Morphine, gabapentin, and ketamine have demonstrated short-term effectiveness in the treatment of PLP, but these results are also based on studies with small sample sizes. Memantine may provide some benefit immediately following amputation but failed to provide analgesia in chronic stages [98]. In regard to long-term effectiveness and safety of pharmacological therapy, more large, randomized controlled trials are needed [99]. Based upon current literature, combinations of drugs are not superior to the use of individual pharmacological agents [92]. A Cochrane review concluded that there was not sufficient evidence to support any particular agent for the treatment of PLP [99]. Mirror therapy has some evidence as an effective treatment. Most of the studies, however, have small sample size, therefore, strong evidence is lacking for its efficacy as a first-line treatment [100]. There is some evidence that spinal cord stimulation is effective in reducing symptoms in PLP [101], as well as massage, acupuncture, and transcutaneous electrical nerve stimulation (TENS).

Chronic Donor Site Pain

When a bone graft is taken, it may lead to the development of chronic pain at the site of harvest; this is known as chronic donor site pain. The exact underlying mechanism for the development of chronic donor site pain is unknown and likely multifactorial. It may be periosteal or muscular in origin, and there may also be a neuropathic component. Nerves that may be injured or involved include the ilioinguinal, lateral femoral cutaneous and superior cuneal nerves.

An autogenous iliac crest bone graft (ICBG) is considered the "gold standard" of bone graft material and will be the primary discussion of the following text. Chronic donor site pain has been reported to cause significant morbidity and quality of life issues for patients. The pain is typically over the donor site and commonly described as sharp, shooting, aching, burning, or similar to that of a toothache. It can be severe and even supersede the pain of the surgical site [102]. Rates of chronic donor site pain vary within the literature, often depending on the study design or population and are often higher for spinal surgeries. The higher prevalence of donor site pain in patients undergoing spinal surgery has been theorized to stem from an inability to discern it from chronic back pain.

Some patients see an improvement in their symptoms within the first 6 months, but many patients can still experience pain at the donor site years after surgery. Around 33% of patients have been reported to experience pain at 1 year, and 2 years between 26.1 and 31% of patients reported some pain [102–104]. Even after 3 years, up to 20% of patients can still experience significant pain that often impacts their quality of life [105]. The more recent literature reported that 87% of patients did not acquire chronic donor site pain at the iliac crest and also cited female gender as a

significant risk factor [106]. However, another study found no difference in the incidence of donor site pain in regard to gender or obesity [107]. Skeppholm et al. found that donor site pain was significant within the first 3 months postoperatively but did not seem to have a major impact on quality of life after 4 weeks [108]. In a 10-year follow-up of patients to assess pain at the donor site, there was no significant difference in pain scores at 1 month and 1 year, with a declining trend in pain scores beginning at 3 years postoperatively [109].

Management of chronic donor site pain that is directed at preventing its development can be attempted through altering surgical techniques and/or perioperative local anesthetic infusions at the graft site. Established chronic donor site pain can be treated with medications such as steroids, NSAIDs, or opioids. Interventional techniques including peripheral nerve blocks and neurolysis have also been used for treatment [110, 111]. Singh et al. showed that a 96 mL infusion of 0.5% bupivacaine resulted in 0 out of 9 patients developing chronic pain after 4 years compared to 7 out of 10 in the control group [112]. Another study showed that intraoperative injection of ropivacaine to the harvest site resulted in no complaints of chronic pain at 6 months postoperatively [113]. On the other hand, administration of 5 mg morphine into the iliac crest donor site did not reduce the incidence of chronic pain [114]. Many modalities can be used to alleviate acute pain at the donor site, but chronic donor site pain is often difficult to treat, and spontaneous resolution is not common.

Post-Thoracotomy Pain Syndrome

The International Association for the Study of Pain (IASP) define post-thoracotomy pain syndrome (PTPS) as "pain that recurs or persists along with a thoracotomy incision for at least two months following the surgical procedure." The pain is often described as aching or burning and can affect around 50% of patients following thoracic surgery [115–117]. Severe acute postoperative pain has been shown to be a significant risk factor for the development of PTPS, and females are also at an increased risk [115, 116]. It has been postulated that adequate management of postoperative pain may decrease the likelihood of developing PTP [115].

There are several proposed mechanisms for PTPS and is believed to have a neuropathic component. The first proposed mechanism is damage to the intercostal nerves. These nerves may be damaged through cutting, compression, or entrapment. Compression is most likely caused by retraction, while catching the nerve with a suture when closing the chest is usually the cause of entrapment. Other proposed mechanisms of PTPS include inflammation of the chest muscles, neuroma formation, and fractured or compressed ribs [117]. The type of surgical incision has not yet proven to influence the evolution of PTPS [118].

Several regional techniques have shown to be effective for controlling acute pain after a thoracotomy. The use of epidural anesthesia and paravertebral blocks may reduce the risk of developing chronic pain in around one in every four patients [119]. Thoracic epidurals are considered the gold standard for acute pain management. They have shown to be most effective with local anesthetic and combined opioid techniques but have also shown benefit when either is administered as a solo agent. The combined opioid and local anesthetic techniques have a synergistic analgesic effect while reducing the risk of side effects. Thoracic epidurals are usually administered in the preoperative phase and continued for 48–72 h after surgery. Also, paravertebral blocks can provide similar analgesia when compared to thoracic epidurals and may reduce the risk of developing minor complications [120, 121]. As with epidurals, a bolus dose is often given which is followed by a continuous infusion for 48–72 h. In regard to minor complications, paravertebral blocks have been associated with less urinary retention, pruritus, hypotension, and even nausea and vomiting [121–123]. There is currently not enough data to determine if either thoracic epidurals or paravertebral blocks are superior to the other in preventing chronic pain [122].

Ketamine is an effective treatment for acute pain. However, some studies have shown it to be

unsuccessful at preventing the development of chronic pain after a thoracotomy [124, 125]. One study has demonstrated that an infusion of dexmedetomidine at 0.5 μg/kg/h from initiation of anesthesia until extubation resulted in a decrease in PTPS from 52 to 22% [117].

We conducted a systematic literature search which yielded 15 randomized control trials evaluating the efficacy of ketamine in the treatment of acute post-thoracotomy pain, with fewer studies assessing its effect on attenuating chronic post-thoracotomy pain. The majority of reviewed studies demonstrated that ketamine has efficacy in reduction of acute pain, but the evidence is limited on long-term benefit of ketamine to prevent post-thoracotomy pain syndrome, regardless of the route of administration. A nested analytical study found there is a statistically significant reduction in acute post-thoracotomy pain with IV or epidural ketamine. However currently, the evidence for a role of ketamine as a preventative agent for chronic post-thoracotomy pain is insufficient related to the heterogeneity of the studies reviewed in this review with regard to the route of administration, dosage, and outcome measures. Therefore, the majority of randomized controlled trials reviewed show no role for ketamine in attenuating or preventing post-thoracotomy pain syndrome at variable follow-up lengths, and additional research is warranted with consideration of risk factors and long-term follow-up for chronic post-thoracotomy pain though the evidence for benefit appears clear for acute post-thoracotomy pain.

When a thoracic epidural or paravertebral block cannot be performed, other methods can be attempted to reduce pain. However, it should be noted that the perioperative use of pregabalin has not shown to be effective in reducing PTPS [126]. A single intrathecal dose of opioids has been shown to provide more effective analgesia for up to 24 h when compared to patient-controlled analgesia [120]. The intrapleural administration of local anesthetics is currently not recommended due to its potential for toxicity from high absorption rates and failure to show efficacy when compared to other techniques [120]. Cryoanalgesia and intercostal nerve blocks have both been shown to be ineffective at reducing PTPS, and there is evidence that cryoanalgesia may lead to an increase in chronic pain. There are currently no studies in regard to NSAIDs and acetaminophen and their impact on the development of PTPS [127].

The pharmacological treatment of PTPS is similar to that of other neuropathic pains and can include gabapentinoids, tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, and tramadol. If conventional treatments are inadequate then interventional techniques such as intercostal nerve blocks and pulsed radiofrequency of the dorsal root ganglion can be considered, however, there is currently no convincing evidence for their effectiveness [127].

Multimodal Preemptive Analgesia

As previously stated, the goal of multimodal preemptive analgesia is to achieve optimal pain relief, while reducing opioid requirements and undesirable side effects [128]. Common analgesic modalities mentioned in this chapter include opioids, nonsteroidal anti-inflammatory drugs (NSAIDS), acetaminophen, analgesic adjuncts (steroids, ketamine, anticonvulsants, antidepressants, α-2 agonists, neuroleptics, and antihistamines), and local anesthetic techniques (peripheral nerve blocks, neuraxial blocks, and local infiltration) [128]. While there is controversial evidence that any one of these treatments is the most effective preemptive analgesic agent, it remains clear that the best approach is multimodal, includes various or multiple pharmacological agents, and is tailored to the patient's needs.

Some authors suggest that preemptive analgesia should provide antinociceptive protection that extends sufficiently into the postoperative period to cover the inflammatory phase of central sensitization to be effective [129]. Furthermore, anesthesiologists and perioperative pain medicine practitioners are highly interested in both the short-term and long-term control of postoperative pain. In the short term, pain control likely

leads to better patient satisfaction and more efficient postanesthesia care unit throughput. Long-term effects of postoperative pain may cause debilitating sequelae such as chronic pain and mental illness [130, 131]. Some argue that a combination of preemptive analgesia and multimodal pain management is an ideal approach to decrease postoperative pain [132]. Specifically, multimodal analgesia, an important component of preventive analgesia, is theorized to lead to a decrease in long-term pain sensitivity at the central and peripheral levels by lengthening the duration of action of analgesic drugs [132]. Overall, multimodal preemptive analgesia is an attractive area of study with much ongoing research and promise for clinicians as well as their patients.

Summary

Despite the compelling experimental evidence, the effectiveness of preemptive analgesia is challenging to prove in a clinical setting. This might be related to several factors including the by-product of various methodologies implemented by different researchers. Furthermore, it is difficult to manipulate and study complex physiologic events such as postoperative pain. However, given these obstacles, a growing body of research suggests that techniques which involve continuous infusions of a local anesthetic like epidurals are most promising. In particular, continuous perineural catheters, which are used to prolong the effect of a peripheral nerve block, achieve good clinical results as patients benefit from being discharged to the comfort of their home while, at the same time, potentially reducing the inflammatory sensitization that would otherwise occur. In the future, we may see the role of these catheters grow as long-term pain control and reduction in healthcare costs become more important to healthcare providers, administrators, and patients.

The value of comprehensive pain control for surgical patients should not be underestimated. Some clinicians focus on providing preventive analgesia rather than the more narrowly defined preemptive analgesia. Preventive analgesia emphasizes the fact that central neuroplasticity is induced by preoperative, intraoperative, and postoperative nociceptive inputs. Thus, the goal of analgesia is to reduce the sensitization that arises from noxious inputs arising through the entire postoperative period and not just from those occurring during the surgical incision. Multimodal analgesic interventions aimed at both short- and long-term pain management may serve to insulate the susceptible neural pathways from a relatively lengthy period of continuous nociceptive inputs. Effective preventive analgesic techniques may be not only useful in reducing acute pain but also chronic postsurgical pain and disability.

As our understanding of pain prevention improves, anesthesiologists and perioperative pain management practitioners may employ preemptive analgesia more widely. While further research on preemptive analgesia is needed, postoperative pain control leads to an increase in patient comfort and a reduction in pain-related sequelae, thereby leading to improved postanesthesia care unit efficiency and shorter hospital stays.

Review Questions

1. What is gabapentin's mechanism of action?
   (a) Interacts with an auxiliary subunit of voltage-sensitive Ca2+ channels in brain membranes
   (b) Acts on GABAA receptors
   (c) Acts on GABAB receptors
   (d) Acts on both GABAA and GABAB receptors
2. What is the first-line treatment for CRPS Type II (formerly causalgia)?
   (a) Spinal cord stimulator
   (b) NSAIDS
   (c) Opioids
   (d) Physical therapy
3. What is the best type of anesthesia for a morbidly obese 54-year-old man with hypertension and insulin-dependent diabetes mellitus undergoing a total knee arthroplasty?

(a) General anesthesia with a post-op femoral and sciatic nerve block.
(b) Epidural anesthesia with a catheter in place.
(c) Spinal anesthesia with a post-op femoral and sciatic nerve block.
(d) No recommendation can be given at this time. Different anesthetics have pros and cons that must be weighed on a case-by-case basis.

4. What is the first sign of a high spinal after caudal anesthesia in a 7-month-old girl?
(a) Respiratory failure
(b) Hypotension and bradycardia
(c) Hypotension and tachycardia
(d) Pupillary constriction

Answers

1. a
2. d
3. d
4. a

References

1. Crile G, Lower W. Anoci-association. Ann Surg. 1915;61(1):123.
2. Woolf CJ, Chong MS. Preemptive analgesia—treating postoperative pain by preventing the establishment of central sensitization. Anesth Analg. 1993;77(2):362–79.
3. Baranauskas G, Nistri A. Sensitization of pain pathways in the spinal cord: cellular mechanisms. Prog Neurobiol. 1998;54(3):349–65.
4. Petho G, Reeh PW. Sensory and signaling mechanisms of bradykinin, eicosanoids, platelet-activating factor, and nitric oxide in peripheral nociceptors. Physiol Rev. 2012;92(4):1699–775.
5. Millan MJ. The induction of pain: an integrative review. Prog Neurobiol. 1999;57(1):1–164.
6. Renn CL, Dorsey SG. The physiology and processing of pain: a review. AACN Clin Issues. 16(3):277–90.
7. Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. J Pain. 2009;10(9):895–926.
8. dek AG, Liszka H, Wordliczek J. Postoperative pain and preemptive local anesthetic infiltration in hallux valgus surgery. Foot Ankle Int. 2015;36(3):277–81.
9. Costa FWG, Esses DFS, de Barros Silva PG, Carvalho FSR, Sá CDL, Albuquerque AFM, Bezerra TP, Ribeiro TR, Fonteles CSR, Soares ECS. Does the preemptive use of oral nonsteroidal anti-inflammatory drugs reduce postoperative pain in surgical removal of third molars? A meta-analysis of randomized clinical trials. Anesth Prog. 2015;62(2):57–63.
10. O'Connor TC, Abram SE. Halothane enhances suppression of spinal sensitization by intrathecal morphine in the rat formalin test. Anesthesiology. 1994;81(5):1277–83.
11. Ong CK-S, Lirk P, Seymour RA, Jenkins BJ. The efficacy of preemptive analgesia for acute postoperative pain management: a meta-analysis. Anesth Analg. 2005;100(3):757–73., table of contents.
12. Hariharan S, Moseley H, Kumar A, Raju S. The effect of preemptive analgesia in postoperative pain relief—a prospective double-blind randomized study. Pain Med. 2009;10(1):49–53.
13. Keskioğlu İ, Aktay İnal M, Özlü O. The effectiveness of preemptive analgesic techniques on postoperative analgesia in patients undergoing open septorhinoplasty. Agri. 2016;28(3):135–42.
14. Gurbet A, Bekar A, Bilgin H, Korfali G, Yilmazlar S, Tercan M. Pre-emptive infiltration of levobupivacaine is superior to at-closure administration in lumbar laminectomy patients. Eur Spine J. 2008;17(9):1237–41.
15. O'Hanlon DM, Colbert ST, Keane PW, Given FH. Preemptive bupivacaine offers no advantages to postoperative wound infiltration in analgesia for outpatient breast biopsy. Am J Surg. 2000;180(1):29–32.
16. Kristek J, Kvolik S, Sakić K, Has B, Prlić L. Intercostal catheter analgesia is more efficient vs. intercostal nerve blockade for post-thoracotomy pain relief. Coll Antropol. 2007;31(2):561–6.
17. Pryor GJ, Kilpatrick WR, Opp DR. Local anesthesia in minor lacerations: topical TAC vs lidocaine infiltration. Ann Emerg Med. 1980;9(11):568–71.
18. Petersen PL, Mathiesen O, Stjernholm P, Kristiansen VB, Torup H, Hansen EG, Mitchell AU, Moeller A, Rosenberg J, Dahl JB. The effect of transversus abdominis plane block or local anaesthetic infiltration in inguinal hernia repair. Eur J Anaesthesiol. 2013;30(7):415–21.
19. Saeed M, Andrabi WI, Rabbani S, Zahur S, Mahmood K, Andrabi SI, Butt HA, Chaudhry AM. The impact of preemptive ropivacaine in inguinal hernioplasty—a randomized controlled trial. Int J Surg. 2015;13:76–9.
20. Bamigboye AA, Justus HG. Ropivacaine abdominal wound infiltration and peritoneal spraying at cesarean delivery for preemptive analgesia. Int J Gynecol Obstet. 2008;102(2):160–4.
21. Mecklem DW, Humphrey MD, Hicks RW. Efficacy of bupivacaine delivered by wound catheter for post-caesarean section analgesia. Aust N Z J Obstet Gynaecol. 1995;35(4):416–21.
22. Givens VA, Lipscomb GH, Meyer NL. A randomized trial of postoperative wound irrigation with

local anesthetic for pain after cesarean delivery. Am J Obstet Gynecol. 2002;186(6):1188–91.
23. Ventham NT, Hughes M, O'Neill S, Johns N, Brady RR, Wigmore SJ. Systematic review and meta-analysis of continuous local anaesthetic wound infiltration versus epidural analgesia for postoperative pain following abdominal surgery. Br J Surg. 2013;100(10):1280–9.
24. Ventham NT, O'Neill S, Johns N, Brady RR, Fearon KCH. Evaluation of novel local anesthetic wound infiltration techniques for postoperative pain following colorectal resection surgery. Dis Colon Rectum. 2014;57(2):237–50.
25. Kong T-W, Park H, Cheong J-Y, Min S-K, Ryu H-S. Efficacy of continuous wound infiltration of local anesthetic for pain relief after gynecologic laparoscopy. Int J Gynecol Obstet. 2014;124(3):212–5.
26. Scott NB. Wound infiltration for surgery. Anaesthesia. 2010;65:67–75.
27. Sakuragi T, Ishino H, Dan K. Bactericidal activity of clinically used local anesthetics on Staphylococcus Aureus. Reg Anesth. 1996;21(3):239–42.
28. Chang Y-C, Liu C-L, Chen M-J, Hsu Y-W, Chen S-N, Lin C-H, Chen C-M, Yang F-M, Hu M-C. Local anesthetics induce apoptosis in human breast tumor cells. Anesth Analg. 2014;118(1):116–24.
29. Dahl JB, Møiniche S. Relief of postoperative pain by local anaesthetic infiltration: efficacy for major abdominal and orthopedic surgery. Pain. 2009;143(1):7–11.
30. Bhaskar SB. Case for local infiltration analgesia: is all the evidence in black and white? Indian J Anaesth. 2015;59(1):1–4.
31. Abrão J, Fernandes CR, White PF, Shimano AC, Okubo R, Lima GB, Bachur JA, Garcia SB. Effect of local anaesthetic infiltration with bupivacaine and ropivacaine on wound healing: a placebo-controlled study. Int Wound J. 2014;11(4):379–85.
32. Brower M, Johnson ME. Adverse effects of local anesthetic infiltration on wound healing. Reg Anesth Pain Med. 2003;28(3):233–40.
33. Liu SS, Strodtbeck WM, Richman JM, Wu CL. A comparison of regional versus general anesthesia for ambulatory anesthesia: a meta-analysis of randomized controlled trials. Anesth Analg. 2005;101(6):1634–42.
34. Thavaneswaran P, Rudkin GE, Cooter RD, Moyes DG, Perera CL, Maddern GJ. Paravertebral block for anesthesia. Anesth Analg. 2010;110(6):1740–4.
35. Merritt CK, Mariano ER, Kaye AD, Lissauer J, Mancuso K, Prabhakar A, Urman RD. Peripheral nerve catheters and local anesthetic infiltration in perioperative analgesia. Best Pract Res Clin Anaesthesiol. 2014;28(1):41–57.
36. Prabhakar A, Mancuso KF, Owen CP, Lissauer J, Merritt CK, Urman RD, Kaye AD. Perioperative analgesia outcomes and strategies. Best Pract Res Clin Anaesthesiol. 2014;28(2):105–15.
37. Barton J, Grant SA. Outcome studies and infection control in regional anesthesia. In: Essentials of regional anesthesia. New York, NY: Springer; 2012. p. 741–77.
38. Doyle E, Bowler GMR. Pre-emptive effect of multimodal analgesia in thoracic surgery. Br J Anaesth. 1998;80(80):147–51.
39. Møiniche S, Kehlet H, Dahl JB. A qualitative and quantitative systematic review of preemptive analgesia for postoperative pain relief: the role of timing of analgesia. Anesthesiology. 2002;96(3):725–41.
40. Samad T, Abdi S. Cyclooxygenase-2 and antagonists in pain management. Curr Opin Anaesthesiol. 2001;14(5):527–32.
41. Reuben SS, Bhopatkar S, Maciolek H, Joshi W, Sklar J. The preemptive analgesic effect of rofecoxib after ambulatory arthroscopic knee surgery. Anesth Analg. 2002;94(1):55–9., table of contents.
42. Vardeh D, Wang D, Costigan M, Lazarus M, Saper CB, Woolf CJ, FitzGerald GA, Samad TA. COX2 in CNS neural cells mediates mechanical inflammatory pain hypersensitivity in mice. J Clin Invest. 2009;119(2):287–94.
43. Kroin JS, Ling ZD, Buvanendran A, Tuman KJ. Upregulation of spinal cyclooxygenase-2 in rats after surgical incision. Anesthesiology. 2004;100(2):364–9.
44. Buvanendran A, Kroin JS, Berger RA, Hallab NJ, Saha C, Negrescu C, Moric M, Caicedo MS, Tuman KJ. Upregulation of prostaglandin E2 and interleukins in the central nervous system and peripheral tissue during and after surgery in humans. Anesthesiology. 2006;104(3):403–10.
45. Minami T, Nishihara I, Uda R, Ito S, Hyodo M, Hayaishi O. Characterization of EP-receptor subtypes involved in allodynia and hyperalgesia induced by intrathecal administration of prostaglandin E2 to mice. Br J Pharmacol. 1994;112(3):735–40.
46. Yaksh TL, Dirig DM, Conway CM, Svensson C, Luo ZD, Isakson PC. The acute antihyperalgesic action of nonsteroidal, anti-inflammatory drugs and release of spinal prostaglandin E2 is mediated by the inhibition of constitutive spinal cyclooxygenase-2 (COX-2) but not COX-1. J Neurosci. 2001;21(16):5847–53.
47. Hodsman NB, Burns J, Blyth A, Kenny GN, McArdle CS, Rotman H. The morphine sparing effects of diclofenac sodium following abdominal surgery. Anaesthesia. 1987;42(9):1005–8.
48. Gillies GW, Kenny GN, Bullingham RE, McArdle CS. The morphine sparing effect of ketorolac tromethamine. A study of a new, parenteral non-steroidal anti-inflammatory agent after abdominal surgery. Anaesthesia. 1987;42(7):727–31.
49. Aynehchi BB, Cerrati EW, Rosenberg DB. The efficacy of oral Celecoxib for acute postoperative pain in face-lift surgery. JAMA Facial Plast Surg. 2014;16(5):306.
50. Molnár C, Simon É, Kazup Á, Gál J, Molnár L, Novák L, Bereczki D, Sessler DI, Fülesdi B. A single

preoperative dose of diclofenac reduces the intensity of acute postcraniotomy headache and decreases analgesic requirements over five postoperative days in adults: a single center, randomized, blinded trial. J Neurol Sci. 2015;353(1–2):70–3.
51. Węgorowski P, Stanisławek A, Domżał-Drzewicka R, Sysiak J, Rząca M, Milanowska J, Janiszewska M, Dziubińska A. The effect of pre-emptive analgesia on the level of postoperative pain in women undergoing surgery for breast neoplasm. Contemp Oncol (Poznan, Poland). 2016;20(2):158–64.
52. Schuster R, Stewart D, Schuster L, Greaney G, Waxman K. Preoperative oral rofecoxib and postoperative pain in patients after laparoscopic cholecystectomy: a prospective, randomized, double-blinded, placebo-controlled trial. Am Surg. 2005;71(10):827–9.
53. Munteanu AM, Cionac Florescu S, Anastase DM, Stoica CI. Is there any analgesic benefit from preoperative vs. postoperative administration of etoricoxib in total knee arthroplasty under spinal anaesthesia? Eur J Anaesthesiol. 2016;33(11):840–5.
54. Boccara G, Chaumeron A, Pouzeratte Y, Mann C. The preoperative administration of ketoprofen improves analgesia after laparoscopic cholecystectomy in comparison with propacetamol or postoperative ketoprofen. Br J Anaesth. 2005;94(3):347–51.
55. Nir R-R, Nahman-Averbuch H, Moont R, Sprecher E, Yarnitsky D. Preoperative preemptive drug administration for acute postoperative pain: a systematic review and meta-analysis. Eur J Pain. 2016;20(7):1025–43.
56. Kang H, Ha Y-C, Kim J-Y, Woo Y-C, Lee J-S, Jang E-C. Effectiveness of multimodal pain management after bipolar hemiarthroplasty for hip fracture. J Bone Joint Surg Am. 2013;95(4):291–6.
57. Trabulsi EJ, Patel J, Viscusi ER, Gomella LG, Lallas CD. Preemptive multimodal pain regimen reduces opioid analgesia for patients undergoing robotic-assisted laparoscopic radical prostatectomy. Urology. 2010;76(5):1122–4.
58. Sandkühler J, Ruscheweyh R. Opioids and central sensitisation: I. Pre-emptive analgesia. Eur J Pain. 2005;9(2):145–8.
59. Rahimi M, Farsani D, Naghibi K, Alikiaii B. Preemptive morphine suppository for postoperative pain relief after laparoscopic cholecystectomy. Adv Biomed Res. 2016;5(1):57.
60. Wang N, Wang Y, Pang L, Wang J. Effect of preemptive analgesia with intravenous oxycodone in the patients undergoing laparoscopic resection of ovarian tumor. Pakistan J Med Sci. 2015;31(2):300–3.
61. Rizos EN, Siafakas N, Katsantoni E, Lazou V, Sakellaropoulos K, Kastania A, Kossida S, Chatzigeorgiou K-S, Arsenis G, Zerva L, Katsafouros K, Lykouras L. Association of the dopamine D3 receptor Ser9Gly and of the serotonin 2C receptor gene polymorphisms with tardive dyskinesia in Greeks with chronic schizophrenic disorder. Psychiatr Genet. 2009;19(2):106–7.
62. Abdolahi M, Soltani HA, Montazeri K, Soleymani B. Preemptive low-dose of ketamine does not effective on anesthetic consumption, perioperative analgesic requirement and postoperative pain, nausea and vomiting in painful ophthalmic surgery. J Res Med Sci. 2013;18(7):583–7.
63. Helmy SA, Bali A. The effect of the preemptive use of the NMDA receptor antagonist dextromethorphan on postoperative analgesic requirements. Anesth Analg. 2001;92(3):739–44.
64. Movafegh A, Beigi N, Mahmoodzadeh H. Preoperative oral dextromethorphan does not reduce pain or morphine consumption after open cholecystectomy. Saudi J Anaesth. 2009;3(2):13.
65. Entezary SR, Farshadpour S, Alebouyeh MR, Imani F, Emami Meybodi MK, Yaribeygi H. Effects of preoperative use of oral dextromethorphan on postoperative need for analgesics in patients with knee arthroscopy. Anesthesiol Pain Med. 2014;4(1):e11187.
66. Siddiqui K, Khan F. Effect of preinduction low-dose ketamine bolus on intra operative and immediate postoperative analgesia requirement in day care surgery: a randomized controlled trial. Saudi J Anaesth. 2015;9(4):422.
67. Paech MJ, Goy R, Chua S, Scott K, Christmas T, Doherty DA. A randomized, placebo-controlled trial of preoperative oral Pregabalin for postoperative pain relief after minor gynecological surgery. Anesth Analg. 2007;105(5):1449–53.
68. Valadan M, Banifatemi S, Yousefshahi F. Preoperative gabapentin to prevent postoperative shoulder pain after laparoscopic ovarian cystectomy: a randomized clinical trial. Anesthesiol. Pain Med. 2015;5(6):e31524.
69. Ahn S, Byun SH, Park K, Ha JL, Kwon B, Kim JC. Analgesic efficacy of preemptive pregabalin administration in arthroscopic shoulder surgery: a randomized controlled trial. Can J Anesth. 2016;63(3):283–9.
70. Aydoğan H, Kucuk A, Yuce HH, Karahan MA, Ciftci H, Gulum M, Aksoy N, Yalcin S. Adição de 75mg de pregabalina ao regime analgésico reduz escores de dor e consumo de opiáceos em adultos após nefrolitotomia percutânea. Brazilian J Anesthesiol. 2014;64(5):335–42.
71. Sadatsune EJ, Leal P d C, Cossetti RJD, Sakata RK. Effect of preoperative gabapentin on pain intensity and development of chronic pain after carpal tunnel syndrome surgical treatment in women: randomized, double-blind, placebo-controlled study. Sao Paulo Med J. 2016;134(4):285–91.
72. Alayed N, Alghanaim N, Tan X, Tulandi T. Preemptive use of gabapentin in abdominal hysterectomy. Obstet Gynecol. 2014;123(6):1221–9.
73. Arumugam S, Lau CS, Chamberlain RS. Use of preoperative gabapentin significantly reduces postoperative opioid consumption: a meta-analysis. J Pain Res. 2016;9:631–40.
74. Mao Y, Wu L, Ding W. The efficacy of preoperative administration of gabapentin/pregabalin in improv-

ing pain after total hip arthroplasty: a meta-analysis. BMC Musculoskelet Disord. 2016;17(1):373.
75. Bruehl S. An update on the pathophysiology of complex regional pain syndrome. Anesthesiology. 2010;113(3):713–25.
76. de Mos M, de Bruijn AGJ, Huygen FJPM, Dieleman JP, Stricker BHC, Sturkenboom MCJM. The incidence of complex regional pain syndrome: a population-based study. Pain. 2007;129(1–2):12–20.
77. Bruehl S. Complex regional pain syndrome. BMJ. 2015;351:h2730.
78. Maihöfner C, Seifert F, Markovic K. Complex regional pain syndromes: new pathophysiological concepts and therapies. Eur J Neurol. 2010;17(5):649–60.
79. Bussa M, Guttilla D, Lucia M, Mascaro A, Rinaldi S. Complex regional pain syndrome type I: a comprehensive review. Acta Anaesthesiol Scand. 2015;59(6):685–97.
80. Golmirzaie G, Holland LS, Moser SE, Rastogi M, Hassett AL, Brummett CM. Time since inciting event is associated with higher centralized pain symptoms in patients diagnosed with complex regional pain syndrome. Reg Anesth Pain Med. 2016;41(6):731–6.
81. van Eijs F, Stanton-Hicks M, Van Zundert J, Faber CG, Lubenow TR, Mekhail N, van Kleef M, Huygen F. Evidence-based interventional pain medicine according to clinical diagnoses. 16. Complex regional pain syndrome. Pain Pract. 2011;11(1):70–87.
82. Smart KM, Wand BM, O'Connell NE. Physiotherapy for pain and disability in adults with complex regional pain syndrome (CRPS) types I and II. In: Smart KM, editor. Cochrane database of systematic reviews. Wiley: Chichester, UK; 2013.
83. Wertli MM, Kessels AGH, Perez RSGM, Bachmann LM, Brunner F. Rational pain management in complex regional pain syndrome 1 (CRPS 1)—a network meta-analysis. Pain Med. 2014;15(9):1575–89.
84. O'Connell NE, Wand BM, Gibson W, Carr DB, Birklein F, Stanton TR. Local anaesthetic sympathetic blockade for complex regional pain syndrome. In: Stanton TR, editor. Cochrane database of systematic reviews. Wiley: Chichester, UK; 2016.
85. Visnjevac O, Costandi S, Patel BA, Azer G, Agarwal P, Bolash R, Mekhail NA. A comprehensive outcome-specific review of the use of spinal cord stimulation for complex regional pain syndrome. Pain Pract. 2017;17(4):533–45.
86. Aradillas E, Schwartzman RJ, Grothusen JR, Goebel A, Alexander GM. Plasma exchange therapy in patients with complex regional pain syndrome. Pain Physician. 18(4):383–94.
87. Harden RN, Oaklander AL, Burton AW, Perez RSGM, Richardson K, Swan M, Barthel J, Costa B, Graciosa JR, Bruehl S. Reflex sympathetic dystrophy syndrome association, "complex regional pain syndrome: practical diagnostic and treatment guidelines", 4th edition. Pain Med. 2013;14(2):180–229.
88. Subedi B, Grossberg GT. Phantom limb pain: mechanisms and treatment approaches. Pain Res Treat. 2011;2011:864605.
89. Borghi B, D'Addabbo M, White PF, Gallerani P, Toccaceli L, Raffaeli W, Tognù A, Fabbri N, Mercuri M. The use of prolonged peripheral neural blockade after lower extremity amputation: the effect on symptoms associated with phantom limb syndrome. Anesth Analg. 2010;111(5):1308–15.
90. Davidson JH, Khor KE, Jones LE. A cross-sectional study of post-amputation pain in upper and lower limb amputees, experience of a tertiary referral amputee clinic. Disabil Rehabil. 2010;32(22):1855–62.
91. Flor H, Nikolajsen L, Staehelin Jensen T. Phantom limb pain: a case of maladaptive CNS plasticity? Nat Rev Neurosci. 2006;7(11):873–81.
92. Fang J, Lian Y, Xie K, Cai S. Pharmacological interventions for phantom limb pain. Chin Med J. 2013;126(3):542–9.
93. Nikolajsen L, Jensen TS. Phantom limb pain. Br J Anaesth. 2001;87(1):107–16.
94. Borghi B, D'Addabbo M, Borghi R. Can neural blocks prevent phantom limb pain? Pain Manag. 2014;4(4):261–6.
95. Karanikolas M, Aretha D, Tsolakis I, Monantera G, Kiekkas P, Papadoulas S, Swarm RA, Filos KS. Optimized perioperative analgesia reduces chronic phantom limb pain intensity, prevalence, and frequency: a prospective, randomized, clinical trial. Anesthesiology. 2011;114(5):1144–54.
96. Bosanquet DC, Glasbey JCD, Stimpson A, Williams IM, Twine CP. Systematic review and meta-analysis of the efficacy of perineural local anaesthetic catheters after major lower limb amputation. Eur J Vasc Endovasc Surg. 2015;50(2):241–9.
97. Luo Y, Anderson TA. Phantom limb pain. Int Anesthesiol Clin. 2016;54(2):121–39.
98. Loy BM, Britt RB, Brown JN. Memantine for the treatment of phantom limb pain: a systematic review. J Pain Palliat Care Pharmacother. 2016:1–7.
99. Alviar MJM, Hale T, Dungca M. Pharmacologic interventions for treating phantom limb pain. Cochrane Database Syst Rev. 2011;(12):CD006380.
100. Barbin J, Seetha V, Casillas JM, Paysant J, Pérennou D. The effects of mirror therapy on pain and motor control of phantom limb in amputees: a systematic review. Ann Phys Rehabil Med. 2016;59(4):270–5.
101. Aiyer R, Barkin RL, Bhatia A, Gungor S. A systematic review on the treatment of phantom limb pain with spinal cord stimulation. Pain Manag. 2017;7(1):59–69.
102. Silber JS, Anderson DG, Daffner SD, Brislin BT, Leland JM, Hilibrand AS, Vaccaro AR, Albert TJ. Donor site morbidity after anterior iliac crest bone harvest for single-level anterior cervical discectomy and fusion. Spine. 2003;28(2):134–9.
103. Sasso RC, LeHuec JC, Shaffrey C, Spine Interbody Research Group. Iliac crest bone graft donor site pain after anterior lumbar interbody fusion: a prospective patient satisfaction outcome assessment. J Spinal Disord Tech. 2005;18(Suppl):S77–81.

104. Kim DH, Rhim R, Li L, Martha J, Swaim BH, Banco RJ, Jenis LG, Tromanhauser SG. Prospective study of iliac crest bone graft harvest site pain and morbidity. Spine J. 2009;9(11):886–92.
105. Schwartz CE, Martha JF, Kowalski P, Wang DA, Bode R, Li L, Kim DH. Prospective evaluation of chronic pain associated with posterior autologous iliac crest bone graft harvest and its effect on postoperative outcome. Health Qual Life Outcomes. 2009;7(1):49.
106. Kim CH, Chung CK, Choi Y, Hwang ST, Kim S-M, Park SB. The patient-reported outcome of chronic pain after the harvest of anterior iliac bone for anterior cervical arthrodesis. J Clin Neurosci. 2016;36:102–7.
107. Heary RF, Schlenk RP, Sacchieri TA, Barone D, Brotea C. Persistent iliac crest donor site pain: independent outcome assessment. Neurosurgery. 2002;50(3):510–6.
108. Skeppholm M, Olerud C. Pain from donor site after anterior cervical fusion with bone graft: a prospective randomized study with 12 months of follow-up. Eur Spine J. 2013;22(1):142–7.
109. Qi L, Liu Y, Li H, Zhang Y. Results of 10-year follow-up of the iliac donor site of graft patients. J Int Med Res. 2014;42(6):1348–52.
110. Yanow JH, Di Lorenzo L, Worosilo SC, Pappagallo M. Successful treatment of chronic donor site pain. Anesthesiol. Pain Med. 2015;5(5):e18777.
111. Mahli A, Coskun D, Altun NS, Simsek A, Ocal E, Kostekci M. Alcohol neurolysis for persistent pain caused by superior cluneal nerves injury after iliac crest bone graft harvesting in orthopedic surgery: report of four cases and review of the literature. Spine. 2002;27(22):E478–81.
112. Singh K, Phillips FM, Kuo E, Campbell M. A prospective, randomized, double-blind study of the efficacy of postoperative continuous local anesthetic infusion at the iliac crest bone graft site after posterior spinal arthrodesis. Spine. 2007;32(25):2790–6.
113. Zenner J, Hitzl W, Mayer M, Koller H. Analysis of postoperative pain at the anterior iliac crest harvest site: a prospective study of the intraoperative local administration of ropivacaine. Asian Spine J. 2015;9(1):39–46.
114. Wai EK, Sathiaseelan S, O'Neil J, Simchison BL. Local administration of morphine for analgesia after autogenous anterior or posterior iliac crest bone graft harvest for spinal fusion: a prospective, randomized, double-blind, placebo-controlled study. Anesth Analg. 2010;110(3):928–33.
115. Katz J, Jackson M, Kavanagh BP, Sandler AN. Acute pain after thoracic surgery predicts long-term postthoracotomy pain. Clin J Pain. 1996;12(1):50–5.
116. Gotoda Y, Kambara N, Sakai T, Kishi Y, Kodama K, Koyama T. The morbidity, time course and predictive factors for persistent post-thoracotomy pain. Eur J Pain. 2001;5(1):89–96.
117. Jabbary Moghaddam M, Barkhori A, Mirkheshti A, Hashemian M, Amir Mohajerani S. The effect of pre-emptive dexmedetomidine on the incidence of post-thoracotomy pain syndrome in patients undergoing coronary artery bypass grafting. Anesthesiol Pain Med. 2016;6(3):e36344.
118. Gerner P. Postthoracotomy pain management problems. Anesthesiol Clin. 2008;26(2):355–67., vii.
119. Andreae MH, Andreae DA. Regional anaesthesia to prevent chronic pain after surgery: a Cochrane systematic review and meta-analysis. Br J Anaesth. 2013;111(5):711–20.
120. Joshi GP, Bonnet F, Shah R, Wilkinson RC, Camu F, Fischer B, Neugebauer EAM, Rawal N, Schug SA, Simanski C, Kehlet H. A systematic review of randomized trials evaluating regional techniques for postthoracotomy analgesia. Anesth Analg. 2008;107(3):1026–40.
121. Baidya DK, Khanna P, Maitra S. Analgesic efficacy and safety of thoracic paravertebral and epidural analgesia for thoracic surgery: a systematic review and meta-analysis. Interact Cardiovasc Thorac Surg. 2014;18(5):626–35.
122. Yeung JH, Gates S, Naidu BV, Wilson MJ, Gao Smith F. Paravertebral block versus thoracic epidural for patients undergoing thoracotomy. In: Gao Smith F, editor. Cochrane database system reviews. Chichester: Wiley; 2016.
123. Davies RG, Myles PS, Graham JM. A comparison of the analgesic efficacy and side-effects of paravertebral vs epidural blockade for thoracotomy—a systematic review and meta-analysis of randomized trials. Br J Anaesth. 2006;96(4):418–26.
124. Mendola C, Cammarota G, Netto R, Cecci G, Pisterna A, Ferrante D, Casadio C, Della Corte F. S+ −ketamine for control of perioperative pain and prevention of post thoracotomy pain syndrome: a randomized, double-blind study. Minerva Anestesiol. 2012;78(7):757–66.
125. Dualé C, Sibaud F, Guastella V, Vallet L, Gimbert Y-A, Taheri H, Filaire M, Schoeffler P, Dubray C. Perioperative ketamine does not prevent chronic pain after thoracotomy. Eur J Pain. 2009;13(5):497–505.
126. Brulotte V, Ruel MM, Lafontaine E, Chouinard P, Girard F. Impact of Pregabalin on the occurrence of postthoracotomy pain syndrome. Reg Anesth Pain Med. 2015;40(3):262–9.
127. Khelemsky Y, Noto CJ. Preventing post-thoracotomy pain syndrome. Mt Sinai J Med. 2012;79(1):133–9.
128. Rosero EB, Joshi GP. Preemptive, preventive, multimodal analgesia: what do they really mean? Plast Reconstr Surg. 2014;134(4 Suppl 2):85S–93S.
129. Urman R, Vadivelu N, Mitra S, Kodumudi V, Kaye A, Schermer E. Preventive analgesia for postoperative pain control: a broader concept. Local Reg Anesth. 2014:17.
130. Justo D, Zeltser D. Torsades de pointes induced by antibiotics. Eur J Intern Med. 2006;17(4):254–9.
131. Woolf CJ. Recent advances in the pathophysiology of acute pain. Br J Anaesth. 1989;63(2):139–46.
132. Vadivelu N, Mitra S, Schermer E, Kodumudi V, Kaye AD, Urman RD. Preventive analgesia for postoperative pain control: a broader concept. Local Reg Anesth. 2014;7:17–22.

24 Transverse Abdominal Plane, Pectoral and Serratus Plane, and Quadratus Lumborum Blocks

Rita Merman, Vladislav Shick, and Vikram Bhasin

Transversus Abdominis Plane (TAP) Block

Introduction

In the past the transversus abdominis plane blocks were traditionally performed with the blind technique through the angle of Petit. Recently, the ultrasound-guided TAP block has developed, leading to improved localization and deposition of the local anesthetic as well as improved accuracy [1, 2]. The TAP block initially described by O'Donnell placed the local anesthetic in the plane between the internal oblique and the transversus abdominis muscles [2]. It has gained popularity as a method for pain control for abdominal surgery because it is less invasive than paravertebral blocks or epidurals and avoids many of the complications associated with neuraxial anesthesia. In recent years, several studies have demonstrated that the TAP block is very effective for postoperative pain control. A recent meta-analysis by Brogi et al. [3] looked at 51 trials and found a reduction in pain scores and morphine consumption after gynecological surgery, appendectomy, inguinal surgery, bariatric surgery, and urological surgery. The same study also looked at 12 studies that evaluated TAP block for cesarean delivery and found that the TAP block did not reduce pain scores at 6 hours but did reduce 12-h pain scores as well as mean 24-h opioid consumption.

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Anatomy

The abdominal wall is composed of three muscle layers: external oblique, internal oblique, and transversus abdominis (Fig. 24.1). The abdominal anterolateral wall is innervated by the anterior rami of T7–L1 spinal nerves. The intercostal nerves (T7–T11), the subcostal nerve (T12), the iliohypogastric nerve, and the ilioinguinal nerve (L1) supply the lower portion of the abdominal wall. These nerves enter the abdominal wall between transversus abdominis and internal oblique.

Position

The patient is placed supine (see Fig. 24.2).

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_24

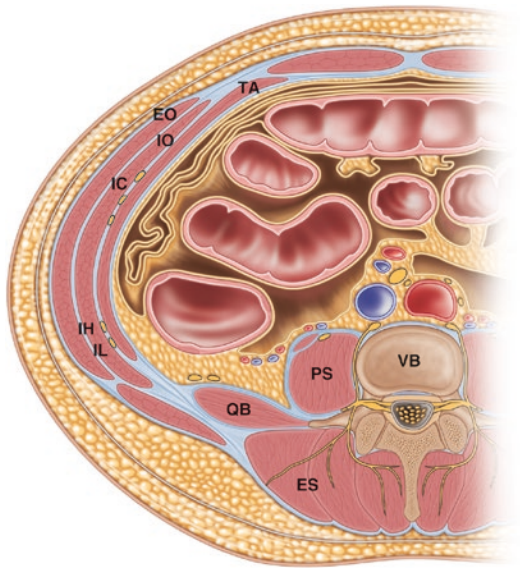

Fig. 24.1 The cross section of the abdominal wall. Please note that the nerves are not obvious on the ultrasound image and somewhat discrete between the plane of internal oblique and transversus abdominis muscles. *EO* external oblique muscle, *IO* internal oblique muscle, *TA* transversus abdominis muscle, *IC* intercostal nerves, *IL* ilioinguinal nerve, *IH* iliohypogastric nerve, *PS* psoas, *ES* erector spinae, *VB* vertebral body, *QB* quadratus lumborum

Fig. 24.2 Positioning of needle and probe for the TAP block. Note iliac crest caudad to the ultrasound probe

Needles

We use a 22-gauge 8-cm Tuohy needle. An 18-gauge Tuohy needle may be used for catheter placement.

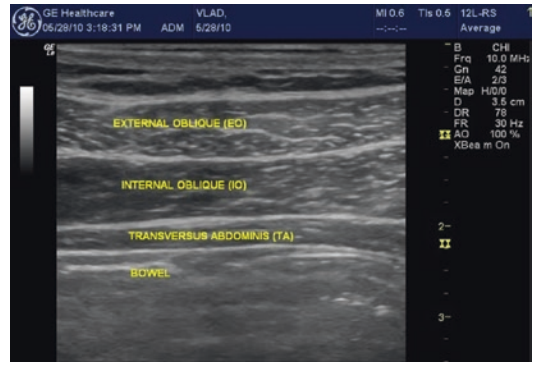

Fig. 24.3 The ultrasound anatomy of the abdominal wall at the anterior axillary line between the iliac crest and twelfth rib

Local Anesthetic

For a rapid onset of block, 1% lidocaine 20 cm^3 can be used. We usually use 20–30 cm^3 of 0.5% ropivacaine.

Probe: 40–60-mm curved array at a frequency of 3–8 MHz.

Technique

The procedure can be done preoperatively or postoperatively. Because the anatomy of the abdominal wall is intact preoperatively, we prefer to perform this block before the operation. This procedure must be done under extreme aseptic technique due to the possible penetration of the peritoneum.

The patient is positioned supine, and the probe is placed above the iliac crest at the anterior axillary line (Fig. 24.1). The muscle layers are identified on the ultrasound image (Fig. 24.2). Then, the needle is inserted in-plane.

The needle movement is carefully observed until the tip is positioned between the plane of transversus abdominis and internal oblique (Figs. 24.3 and 24.4).

The local anesthetic is injected, and the appearance of the plane between the internal oblique and transversus abdominis is observed (Fig. 24.4).

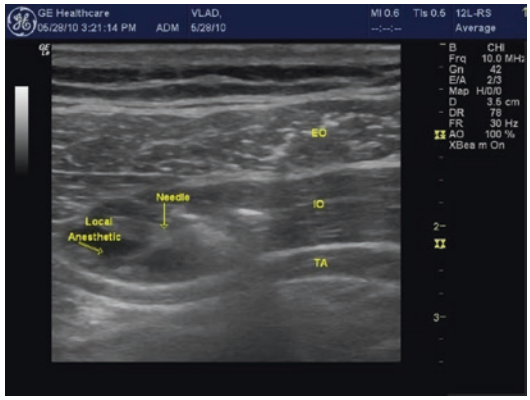

Fig. 24.4 The local anesthetic injection in the facial plane between internal oblique and transversus abdominis muscles. Note the lifting up of internal oblique muscle with local anesthetic injection. *EO* external oblique, *IO* internal oblique, *TA* transversus abdominis muscles

Precautions and Fine Points

- This block requires hydrodissection with normal saline of the plane between the internal oblique and transversus abdominis muscles prior to placing the local anesthetic between the internal oblique and transversus abdominis.
- The needle could easily penetrate into the peritoneal space, so be very careful with needle adjustments and depth.
- Never deposit the local anesthetic into the muscle tissue. The block will be ineffective.
- The plane between internal oblique and transversus abdominis will appear once the normal saline has been injected. After finding the correct plane, inject local anesthetic into the lacuna.

Pectoral and Serratus Plane Blocks

Introduction

Chest wall blocks such as between the pectoralis major and minor muscles, as well as between the pectoralis and serratus muscles, have seen increased use for post-thoracotomy pain, breast surgery, and chest trauma such as rib fractures. The nerves targeted include the medial and lateral pectoral nerves, the lateral intercostal nerves, the long thoracic nerve, and the thoracodorsal nerve. These blocks are particularly appealing because they have many of the benefits of thoracic paravertebral blocks without some of the undesirable side effects, such as pneumothorax. In addition, paravertebral blockade does not adequately cover the anterior chest. The initial Pecs block was described by Blanco [4]. Building on that, he described the initial serratus plane block [5].

There are three types of blocks involving the muscle layers of the chest wall, named "Pecs I," "Pecs II," and serratus plane block. The Pecs I block involves deposition of local anesthetic between pectoralis major and pectoralis minor. The Pecs II block involves deposition of local anesthetic between the pectoralis minor and serratus muscle at the level of the third rib and targets the lateral branches of the intercostal nerves and the long thoracic nerve. The serratus anterior plane block involves deposition of local anesthetic either superficial or deep to the serratus anterior muscle, at the level of the fifth rib in the midaxillary line. It is similar to the Pecs II block except that the needle is placed more caudally and posteriorly [6].

Anatomy

The pectoralis major is the most superficial muscle of the chest wall. Pectoralis minor lies deep to pectoralis major, and serratus anterior lies deep to pectoralis minor (Fig. 24.5). The lateral pectoral nerves arise from C5 to C7 and are deep to pectoralis major. The medial pectoral nerves arise from C8 to T1 and are deep to pectoralis minor. Other relevant nerves include the long thoracic nerve and the thoracodorsal nerve.

Positioning

For Pecs I and II blocks, the patient is placed in a supine position. For the serratus anterior plane block, the patient is placed in a lateral decubitus position.

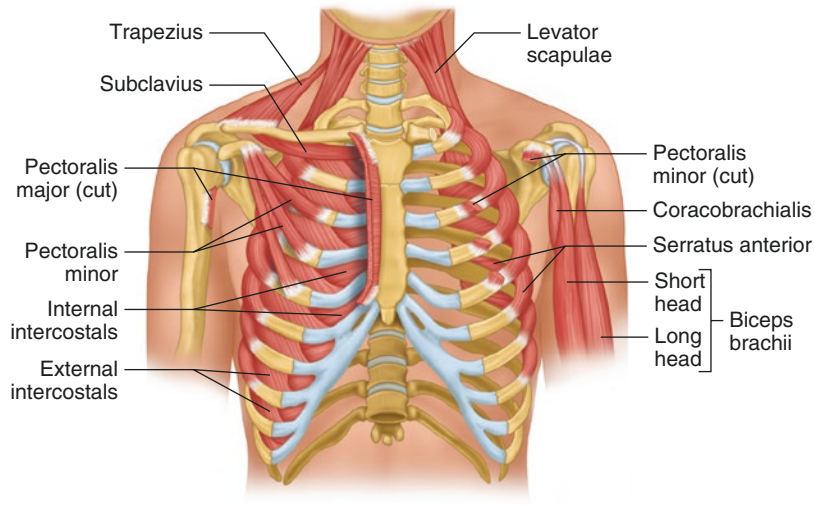

Fig. 24.5 The pectoralis minor lies underneath pectoralis major, and serratus anterior lies deep to pectoralis minor

Needles and Catheters

We use a 22-gauge 8-cm Tuohy needle. An 18-gauge Tuohy needle may be used for catheter placement.

Local Anesthetic

Procedure

The Pecs I block is performed as follows: With the patient in the supine position, place a linear probe at the midclavicular line, pointing inferolaterally. Pectoralis major is identified as the most superficial muscle and the thoracoacromial artery is identified between the pectoralis muscles. The lateral pectoral nerves are generally adjacent to the artery [4]. Move the probe laterally to identify pectoralis minor and serratus anterior. With the needle pointed from medial to lateral, advance the needle in-plane until it is between pectoralis major and pectoralis minor. For the Pecs II block, advance the needle further until it is in the plane between pectoralis minor and serratus and inject local anesthetic. For the serratus block, the probe is moved further caudally and laterally toward the fifth rib. The serratus anterior is identified over the fifth rib and local anesthetic is injected in a medial to lateral direction, either superficial or deep to the serratus muscle. An ultrasound image of the serratus muscle in the area of the eighth and ninth ribs is shown in Fig. 24.6.

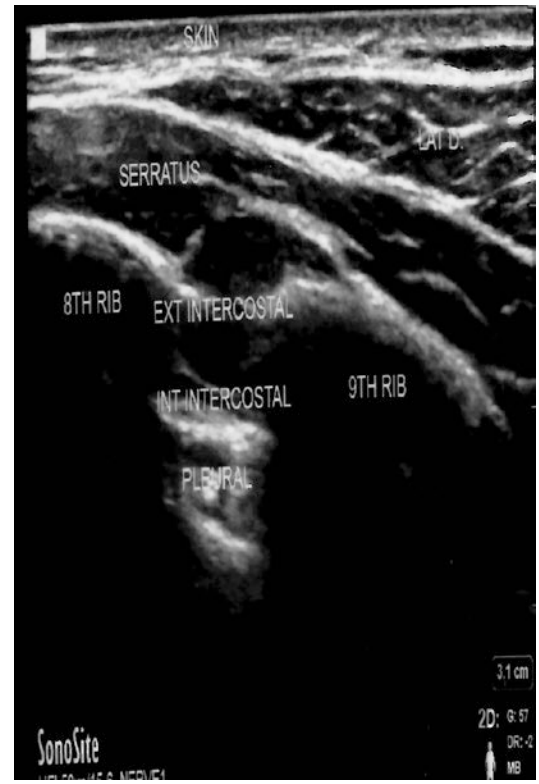

Fig. 24.6 Serratus muscle in the area of the eighth and ninth ribs

Precautions

The main concern for these blocks is pneumothorax, but the risk is significantly lower than for paravertebral nerve blocks.

Quadratus Lumborum Block

Introduction

The quadratus lumborum (QL) block has recently seen a significant increase in its use. Dr. Blanco at first described the block technique at the European Society of Regional Anesthesia Meeting in 2007 (unpublished). Compared to the traditional approach to the TAP block, the posterior approach to the TAP block resulted in increased number of dermatomes blocked.

The QL block technique requires deposition of local anesthetic on either the anterolateral or posterior border of the quadratus lumborum muscle. Recently, the paravertebral spread of contrast has been observed from T5 to L1 using MRI [7]. This has led to the use of the QL block for abdominal surgeries, hip surgeries (ORIF and replacement), and iliac crest bone harvesting. It has also been researched for analgesia after cesarean section. It is thought that the spread of anesthetic within fascial planes, described below, allows for a significantly greater dermatomal spread.

There are three common approaches to the QL block. The most common naming convention terms them as "QL 1," "QL 2," and "QL 3," respectively. The "QL 1" may be seen as a posterior TAP block, in which local anesthetic is injected along the anterolateral border of the QL muscle and the thoracolumbar fascia formed by the external oblique, internal oblique, and transversus abdominis muscles.

The QL 2 block involves injection of local anesthetic along the posterolateral border of the QL muscle. The QL 3 block is the most posterior and involves deposition of local anesthetic in-plane between the QL and psoas muscle groups.

Currently, there is limited but emerging clinical data regarding the QL block. Blanco and colleagues evaluated the QL block for postoperative analgesia after c-section in two separate studies. The first one compared QL block to placebo and noted decreased morphine use and significant analgesia [8]. The second study compared the QL block with TAP block [9] and, overall, noted decreased morphine use up to the first 48 h after c-section. Another study by Parras and colleagues evaluated the QL block for femoral neck fracture surgery and noted a decrease in VAS score in patients with the QL block [10]. Several other studies are currently being pursued regarding the QL block. At our institution, we are conducting a retrospective study comparing the QL block with the lumbar plexus block for total hip arthroplasty.

Anatomy

The quadratus lumborum muscle originates at the iliac crest and inserts on the lower border of the twelfth rib and transverse processes of the upper four lumbar vertebrae. Posterolaterally, the QL is located near the latissimus dorsi muscle. Posteromedially, the QL muscles is located near the sacrospinalis muscles. Anteromedial to the QL is the psoas major muscle. Lateral to the QL are the three major abdominal muscles, the external oblique, internal oblique, and transversus abdominis (Fig. 24.7).

Because the QL block is a fascial plane block, it is also important to consider the various tissue layers surrounding the QL muscle (Fig. 24.8). The thoracolumbar fascia (TLF) encases the muscles of the back, from the thoracic to the lumbar spine. The thoracolumbar fascia forms from the aponeurosis of the transversus abdominis and internal oblique muscles. The anterior layer of the thoracolumbar fascia is anterior to the QL muscle. The middle layer is between the QL and the erector spinae, and the posterior layer is posterior to the erector spinae [11].

Positioning

The quadratus lumborum block may be performed in the supine or lateral decubitus position. The QL 2 and QL 3 blocks are more ideally approached with the patient in the lateral decubitus position.

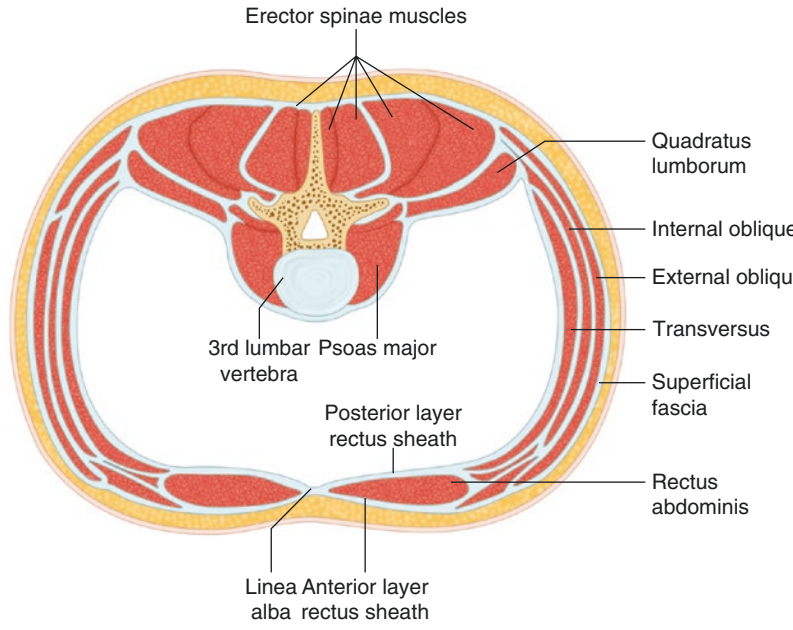

Fig. 24.7 Coronal view of the lower abdomen. The quadratus lumborum is bordered by the major abdominal muscles laterally, the psoas major anteriorly, and the erector spinae muscles muscle posteriorly

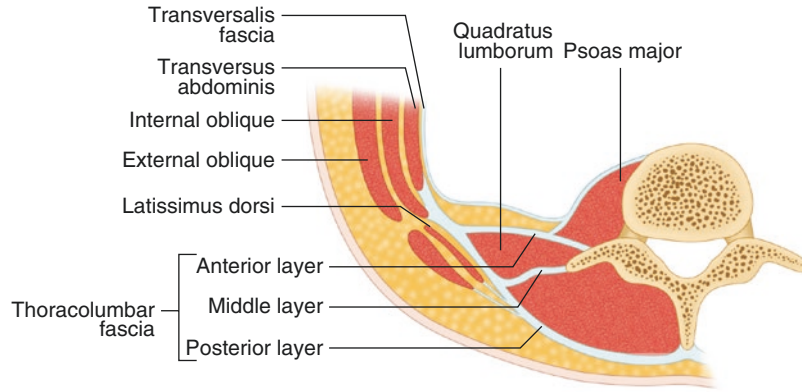

Fig. 24.8 The thoracolumbar fascia begins at the aponeurosis of the external oblique, internal oblique, and transversus abdominis

Needles and Catheters

We use a 22-gauge 8-cm Tuohy needle. An 18-gauge Tuohy needle may be used for catheter placement.

Local Anesthetic

Due to the "tissue/fascial plane" block, we recommend injecting 20 cm^3 of local anesthetic per side. At our institution, we use 20 cm^3 of 0.5% ropivacaine. Additives include 4 mg of dexamethasone and 20–30 μg of dexmedetomidine. Epinephrine may also be added.

Procedure

With the patient in the supine or lateral decubitus position, the ultrasound probe should be placed in midaxillary line between the twelfth rib and superior to the iliac crest. The external oblique, internal oblique, and transversus abdominis should be identified. The probe should then be moved posterior until the aponeurosis and the quadratus lumborum are identified. At this point, the thoracolumbar fascia should be identified as a hyperechoic structure. The thoracolumbar fascia is an important landmark in separating the muscle layers from the peritoneum. In addition, the peritoneum can

usually be identified by the intraperitoneal bowel peristalsis. The kidney and perinephric fat must be identified in order to avoid injection of local anesthetic into the kidney. The needle should enter in medial to lateral direction. For a QL 1 block, the needle should be positioned between the anterior border of the QL muscle and its fascia. The QL 2 block is performed by placing the needle along the posterolateral aspect of the QL muscle. The QL 3 block is performed by placing the needle between the planes of QL and psoas muscle groups.

Precautions

Given the location of the QL muscle, there is a risk of kidney injury, puncture of the bowel, and puncture of blood vessels.

Also, because the QL and ES muscles are part of postural muscles, the blood supply to these muscle groups is extensive. The volume of the injectate should be minimized in order to avoid arterial and venous absorption of local anesthetic and subsequent local anesthetic systemic toxicity.

Review Questions

1. Where does the QL block technique require deposit of local anesthetic?
(a) Just below transversalis fascia
(b) On either the anterolateral or posterior border of the quadratus lumborum muscle
(c) On medial border of the quadratus lumborum muscle
(d) Into the quadratus lumborum muscle
(e) Between quadratus lumborum muscle and transversus abdominis muscle

2. What is the patient's position for QL block?
(a) Sitting
(b) Supine
(c) Prone
(d) Lateral decubitus

3. The lateral pectoral nerves arise from which nerves:
(a) C2–C4
(b) C5–C7
(c) T1–T2
(d) T3–T6
(e) T7

4. The lower portion of the abdominal wall is innervated by:
(a) The subcostal nerve (T12), the iliohypogastric nerve, and the ilioinguinal nerve (L1)
(b) The intercostal nerves (T5–T11) and the subcostal nerve (T12)
(c) The iliohypogastric nerve and the ilioinguinal nerve (L1)
(d) The intercostal nerves (T7–T11), the subcostal nerve (T12), the iliohypogastric nerve, and the ilioinguinal nerve (L1)
(e) The subcostal nerve (T12) and the iliohypogastric nerve

5. Why is it not recommended to deposit the local anesthetic into the muscle tissue?
(a) The block will be ineffective.
(b) It may case tissue necrosis.
(c) It may cause local anesthetic toxicity from extensive absorption.
(d) High chance of seizures from intravascular injection
(e) Very slow local anesthetic absorption from the muscle.

Answers
1. b
2. b and d
3. b
4. d
5. a

References

1. Hebbard P, Fujiwara Y, Shibata Y, et al. Ultrasound-guided transversus abdominis plane (TAP) block. Anaesth Intensive Care. 2007;35(4):616–7.
2. O'Donnell BD, McDonnell JG, McShane AJ. The transversus abdominis plane (TAP) block in open retropubic prostatectomy. Reg Anesth Pain Med. 2006;31(1):91.

3. Brogi E, Kazan R, Cyr S, Giunta F, Hemmerling TM. Transversus abdominal plane block for postoperative analgesia: a systematic review and meta-analysis of randomized-controlled trials. Can J Anaesth. 2016 Oct;63(10):1184–96.
4. Blanco R. The 'pecs block': a novel technique for providing analgesia after breast surgery. Anaesthesia. 2011 Sep;66(9):847–8.
5. Blanco R, Parras T, McDonnell JG, Prats-Galino A. Serratus plane block: a novel ultrasound-guided thoracic wall nerve block. Anaesthesia. 2013 Nov;68(11):1107–13.
6. Chakraborty A, Khemka R, Datta T. Ultrasound-guided truncal blocks: a new frontier in regional anaesthesia. Indian J Anaesth. 2016 Oct;60(10):703–11.
7. Carney J, Finnerty O, Rauf J, Bergin D, Laffey JG, Mc Donnell JG. Studies on the spread of local anaesthetic solution in transversus abdominis plane blocks. Anaesthesia. 2011 Nov;66(11):1023–30.
8. Blanco R, Ansari T, Girgis E. Quadratus lumborum block for postoperative pain after caesarean section: a randomised controlled trial. Eur J Anaesthesiol. 2015 Nov;32(11):812–8.
9. Blanco R, Ansari T, Riad W, Shetty N. Quadratus lumborum block versus transversus abdominis plane block for postoperative pain after cesarean delivery: a randomized controlled trial. Reg Anesth Pain Med. 2016;41(6):757–62.
10. Parras T, Blanco R. Randomised trial comparing the transversus abdominis plane. Block posterior approach or quadratus lumborum block type I with femoral block for postoperative analgesia in femoral neck fracture, both ultrasound-guided. Rev Esp Anestesiol Reanim. 2016 Mar;63(3):141–8.
11. Carline L, McLeod GA, Lamb C. A cadaver study comparing spread of dye and nerve involvement after three different quadratus lumborum blocks. Br J Anaesth. 2016;117(3):387–94.

Part V

Regional Anesthesia and Subspecialties

Outpatient Surgery

25

Kelly R. Mercer

Clinical Case Study

A 65-year-old male with a history of end-stage renal disease (ESRD), congestive heart failure (CHF) with reduced ejection fraction, and hypertension (HTN) presents for left upper extremity arteriovenous (AV) fistula formation.

Introduction

The volume of outpatient surgical cases performed in the United States and worldwide continues to expand, paralleling advances in surgical and anesthetic techniques. Today, upward of 60–70% of all surgical procedures in the United States are performed on an outpatient basis [1]. This increase in outpatient volume offers an opportunity for healthcare providers to not only increase patient satisfaction but also provide a vehicle for cost containment in an era of ever-increasing healthcare expenses. Both anesthesiologists and surgeons are tasked with the challenge of expediting patient discharge. This is accomplished by optimizing recovery and decreasing side effects of surgery and anesthesia such as postoperative pain, nausea, vomiting, and oversedation [2, 3]. Regional anesthesia, within the constructs of a multimodal analgesic regimen, helps address this challenge and can help expedite the transition from the operating room to home.

Literature suggests that regional blocks can provide beneficial health and economic consequences for our patient population. First, regional blocks may help decrease time to discharge by reducing systemic opioid use, minimizing nausea/vomiting, and avoiding postoperative respiratory depression [4–6]. By improving the side effect profile of the anesthetic and increasing patient alertness, a well-functioning block may also increase patient satisfaction with their anesthetic [6]. Furthermore, regional anesthesia may have economic advantages. By diminishing the amount of anesthesia-dedicated time (i.e., induction and emergence from anesthesia), peripheral nerve blocks help reduce operating costs for a surgical suite [6].

Nerve blocks are effective in treating postoperative pain for both upper and lower extremity surgeries, as well as surgery on the trunk, breast, and groin [7, 8]. The block technique chosen depends not only the anatomic site but also upon other factors such as the anticipated length of the procedure and the ambulatory requirements after surgery. This chapter will discuss the implementation of an outpatient regional anesthesia program, utilizing both "single-shot" techniques and continuous perineural catheter techniques. The use of continuous catheter techniques has been

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_25

utilized successfully in both academic and private practice settings [9, 10]. Essential needs for any practice implementing an outpatient regional service are summarized below.

	Essential needs for a practice implementing an outpatient regional service
1.	Start small by focusing on one or two surgeons' patients. This allows a practice to examine the effectiveness of the block program and engage in early quality improvement measures. It also helps with the initial stages of patient follow-up.
2.	Patient selection is important. Patients who have the cognitive ability to understand the goal of the block, as well as manage a catheter, will often report higher satisfaction with their block. This in turn will help with "buy-in" from surgeons and ancillary staff.
3.	Communicate with patients about their expectations for the blocks. Impress on them that the block is only one aspect of their multimodal pain regimen.
4.	Organize/create a dedicated block area in order to expedite patient care in a safe manner. By streamlining preoperative nursing needs, blocks may be performed more efficiently.
5.	Dedicate a block nurse to help manage the block area. Ideally, this/these personnel are able to perform preoperative nursing documentation, execute time-outs, sedate patients, and help with the actual block placement.
6.	Work with scrub nurses to develop ways to drape patients without removing indwelling catheters to prevent inadvertent catheter removal/ suboptimal block duration.
7.	Follow up with patients (e.g., via phone call) to assess the efficacy of the block. This helps with rapport and quality improvement.
8.	Ensure that patients have a way to get in touch with an on-call physician/nurse. At our institution we have a dedicated number of patients that can call to get in touch with our block team.

The primary goal in clinical practice is to provide each patient with a quick, safe, and effective block while maintaining efficient operating room utilization. At our institution, we perform our blocks preoperatively in a dedicated block area so that the patient is prepared for surgery prior to the assigned start time. We create and stress a culture of open communication between the anesthesia team, surgical team, and patient. By doing this, all parties are invested in patient-centered care. For example, a surgical oncologist that plans to resect a mass with nerve encasement usually desires to perform intraoperative or postoperative nerve assessment. Given this situation, we frequently place an indwelling perineural catheter preoperatively and subsequently assess and bolus the catheter postoperatively pending surgical clearance. Finally, we communicate the goal of the block to the patient and to the surgical team caring for them. The goal of the block is frequently to *decrease* rather than eliminate pain. With this understanding in place preoperatively, patients and surgeons will often be more satisfied with the results of the block.

Postoperative follow-up of block performance as well as surveillance for any potential complications serves a valuable role in any institution. At our institution we make brief daily postoperative phone calls (pt's are informed preoperatively of this) – either for the duration of a continuous nerve block or until resolution of a single-shot block. Among other things, we always ask the following questions to our patient population:

1. How is your pain intensity? (Utilizing a 0–10 pain scale)
2. When were you able to start moving your extremity? Do you have any existing weakness that was not present preoperatively?
3. When did your numbness completely resolve? Do you have any existing numbness or abnormal sensations?
4. If you had to have this surgery again, would you request a nerve block?

These calls allow the anesthesiologist/healthcare provider to suggest adjustments to that patient's regimen (i.e., in the setting of a continuous perineural catheter with infusion pump). They also allow providers to obtain suggestions for improvements in their practice. Finally, the calls detect possible block complications such as continued sensory or motor disturbances that may require continued follow-up and evaluation. Once the calls are completed, the information obtained is easily transcribed into a preformatted electronic medical record note that is attached to the patient's chart.

Meticulous preoperative and postoperative assessment of blocks requires increased work and energy on the block team. However, it also leads to better outcomes such as lower pain scores, shorter PACU stays, and higher satisfaction with the perioperative experience.

Neuraxial Techniques

Epidural and spinal anesthesias have been successfully utilized for a variety of ambulatory procedures involving the lower abdominen, perineum, and lower extremity. Neuraxial anesthesia provides good operating conditions with minimal patient discomfort, in addition to other benefits. However, careful consideration of drug dosing is paramount to provide both ample duration of anesthesia with a low side effect profile and fast resolution of block. A recent meta-analysis concluded that although patients' opioid usage in the PACU was diminished and VAS scores lower with neuraxial anesthesia, the incidence of nausea, PACU stay, and discharge from hospital were unchanged [6]. Furthermore, side effects such as urinary retention, transient neurologic symptoms, postdural puncture headaches, epidural hematoma/abscess, and back pain all have to be weighed alongside the benefits of these blocks.

Single-Shot Techniques

It is important to consider the following for both single-shot and continuous perineural catheter techniques: patient selection, patient education, block selection, medication selection, postoperative care, and follow-up.

Patient selection is of obvious importance with all regional anesthesia techniques. First, the patient must provide consent for the block after being educated regarding its risks and benefits and the steps involved in block placement. To provide a streamlined perioperative experience, our institution obtains consent and provides education regarding block placement in our preoperative assessment clinic (PAC). By doing this, the patient is well prepared to receive the block on their operative day, allowing the block anesthesiologist to more effectively utilize time toward completing blocks.

Depending on the site of the surgery, the anesthesiologist must choose an appropriate block by taking into account coexisting disease (i.e., lung disease, preexisting neuropathy), provider experience, and patient-specific characteristics such as BMI, absence of psychological impairment, willingness to comply with block placement, and appropriate anatomy. After choosing the block, we convey expectations regarding the block to patients and their caregivers. Although much of this is covered in our preoperative clinic, it is still useful for the block anesthesiologist to briefly reiterate important points. For example, patients and caregivers need to understand that as the block recedes, it will be necessary to begin analgesics, as their exposure to the surgical pain will increase. We frequently recommend that patients start taking stronger analgesics (i.e., hydrocodone or oxycodone) prior to going to sleep the evening of surgery (unless long-acting adjuvants were added to the block solution). If the patient fails to anticipate this, their pain level upon awakening can be quite high, and their satisfaction with the block will be decreased [11, 12].

We provide our patients with an education form to reiterate many of these topics. This form outlines expections for the block and the need for preemptive analgesia and provides a way for patients to contact the healthcare team if there is a problem. Additionally, the form discusses weight-bearing precautions and instructs patients to go to the emergency room if they experience shortness of breath following an upper extremity nerve block (secondary to pneumothorax or phrenic nerve paresis). An example of this sheet is included at the end of the chapter.

Some blocks may not be appropriate for certain patient populations [13]. For example, a femoral nerve block creates significant quadricep weakness, predisposing to postoperative falls in patients without significant caregiver support. When placing a femoral nerve catheter, it is of great importance to impress upon the patient (and their caregiver) that they will have weakness and

will need a great deal of assistance in ambulating until the block has resolved. Another example is selection of an interscalene nerve block in the setting of advanced pulmonary disease, as the risk of phrenic nerve paresis may compromise a patient's respiratory status. This can prolong the time to PACU discharge or necessitate an inpatient stay for supplemental oxygen.

After selecting an appropriate block, determine which local anesthetic to use and whether or not it will be necessary to use adjuvant medications with the local. Longer-acting local anesthetics, such as bupivacaine or ropivacaine, are frequently used for postoperative pain control. The use of adjuvant agents such as epinephrine, clonidine, and dexamethasone has been described to increase block duration [14–16].

Postoperatively, it is important to evaluate the patient's pain level and augment their analgesic regimen with systemic medications as needed. Occasionally, additional blocks may need to be performed, particularly saphenous blocks in foot and ankle surgery to supplement a popliteal block. Following the achievement of a satisfactory pain score, the patient may then be discharged to a same-day surgery unit for subsequent discharge home.

Follow-up should occur until resolution of the block as discussed previously. Question each patient about the duration of numbness and motor block. Also, knowing the time to first oral analgesic consumption can be helpful for determining efficacy of block. It is valuable to find out the reason for the first oral analgesic, as patients may take them due to surgical pain and block site pain, or they may be simply taking them as directed. Following the resolution of the block, it is insightful to ask their satisfaction scores, as this can help determine which techniques are working well in a given practice.

Continuous Block Techniques

Patient selection is paramount for these blocks [11]. It is necessary to have a patient who can both understand the fundamentals of a nerve block and be motivated to follow instructions regarding their postoperative care. In the event that a patient cannot be trusted or understand/execute instructions well, it is often beneficial to avoid the placement of a catheter.

In addition to the education provided for single-shot blocks, patients and caregivers must be informed about the nerve catheter, its insertion site, delivery device, and postop management. Discussion with both the patient and a caregiver is important with perineural catheters, as the patient may not be able to adequately visualize and keep the catheter insertion site well preserved, especially with interscalene or posterior popliteal approaches. Catheter care, occlusive dressings, and management of the infusion device are all important topics to cover before the patient is discharged. Again, we cover much of this education in our preoperative assessment clinic. Finally, patients must understand how long their infusion will last and how to remove the catheter at the conclusion of the infusion. An example of a patient education form for continuous techniques is included at the end of the chapter.

Block selection is vital as with single-shot techniques. A distinguishing characteristic for continuous techniques is the need for the insertion site and dressing to be isolated from the surgical field. For example, it can be difficult to secure an interscalene catheter properly while also allowing the surgeon adequate exposure for the procedure. Use of a benzoin type of preparation can increase the adherence of the dressing to the skin. Lastly, communication with the surgical and nursing team regarding the continuous catheter is very beneficial to develop methods to prevent accidental dislodgement during placement and removal of the surgical drapes.

The necessity for longer-acting local anesthetics is trumped by the ability to continuously deliver meds via the indwelling catheter. The use of less cardiotoxic local anesthetics is thus important, as the risk of an unwitnessed cardiac arrest following discharge is present, albeit extremely unlikely. The medicolegal ramifications of this alone could warrant the use of more expensive local anesthetics with less cardiotoxic properties

(such as ropivacaine). A bolus of local anesthetic after placement of the catheter, with a small amount of epinephrine as a test dose, may be used to demonstrate the lack of immediate intravascular absorption.

After assessing a patient's pain postoperatively, boluses of local anesthetic can be considered in this setting. Also, examination of the catheter insertion site and the integrity of the dressing is important prior to discharge as reinforcement may be needed to avoid inadvertent catheter removal.

During follow-up of perineural catheters, the rate of infusion (depending on the delivery device) may be adjusted to meet analgesic needs and mitigate any side effects such as respiratory compromise from an interscalene catheter. Postoperative communication is thus very important for these patients, not only in terms of analgesic management but also in terms of assessing the catheter site. Presence of erythema can be the first sign of infection, and removal of the catheter may be warranted, as well as starting antimicrobial therapy. Finally, following removal of the catheter, it is important for the patient to verify that the tip is intact so that no foreign body is left in situ.

Complications

In addition to the aforementioned complications of regional anesthesia that are discussed in other chapters, the most frequent complication of outpatient peripheral nerve blocks is inadequate analgesia. Options for treating this include increasing the rate of infusion in the instance of a continuous block or using a multimodal backup (including opioids). If none of these measures work, the final option is to instruct the patient to present to an emergency department for pain control.

Another complication unique to outpatient blocks is local infection which can frequently be prevented by observing aseptic technique during block placement, limiting the duration of infusion, and tunneling the catheter. Furthermore, avoidance of the femoral and axillary continuous blocks is helpful [17]. Finally, the other major complication of continuous peripheral nerve blocks is dislodgement of the catheter. The best way to prevent this is adequate securing of the dressing and patient education to prevent excessive wear to the dressing.

Instructions for nerve blocks
You have received a peripheral nerve block. This is an injection of medication next to a nerve that will decrease the amount of pain you have after surgery. This is information about your nerve block.
The medicine will wear off over the next day or two, and you will begin having pain. Begin taking your pain medicine when you start to have pain from your surgical site. The nerve block is only one part of your pain therapy.
If within 2 days you do not have sensation and/or strength in that area, we would like for you to call us, so that we will know this and be able to help you.
If you had arm or shoulder surgery, and begin having shortness of breath at home, go immediately to the emergency room.
If you need help immediately, call hospital paging at (555)555-1234 and ask the operator to page the anesthesia doctor who is on call.
The doctor on call will not prescribe, call in, or refill any narcotic/pain medications.

Instructions for nerve block catheter
You are being sent home with a continuous infusion of medicine through a catheter that is placed near the nerves that supply sensation to your surgical site. Please read the following instructions carefully regarding the care and removal of this catheter.
Begin taking your pain medicine when you start to have pain from your surgical site. The nerve block is only one part of your pain therapy.
The rate of the medicine infusion is dialed to _ on your pump dial. Do not change this rate without discussion from your anesthesia provider.
If you had surgery on an arm or shoulder and your catheter is located in your neck or shoulder area, and you experience any trouble breathing, use the white clamp between the pump and the dial to clamp the tubing, and call the anesthesia doctor who is on call (contact information below).
DO NOT inject anything into the catheter or tubing. This may result in severe limb injury or loss.
You should remove the catheter when the pain pump is empty or if instructed by your medical team. To remove the catheter:
1. Clamp the tubing from the pump with the white clamp between the pump and the dial

Instructions for nerve block catheter
2. Leave it clamped for 2 h
3. Peel off all adhesive parts, gently pull the white catheter out, and dispose of the entire apparatus (including the On-Q medicine ball)
The catheter should come out easily, and you should not experience pain during removal. If you meet any resistance, turn your head or bend your knee and try again. If you still have pain or resistance, leave catheter in place, and call the anesthesia doctor on call (contact information below). Once the catheter is out, examine the end – there should be a small (3–5 mm) metal tip. If not, call the anesthesia doctor who is on call, and save the catheter for examination.
If at any point the dressing breaks down and the catheter becomes exposed to air, remove the catheter. If you have redness or pus that develops around the catheter site, or if you develop a fever greater than 101 °F, call the anesthesia doctor who is on call.
If you need help immediately, call hospital paging at (555)555-1234, and ask the operator to page the anesthesia doctor who is on call.
The doctor on call WILL NOT prescribe, call in, or refill any narcotic/pain medications.

Clinical Pearls

- A successful outpatient regional program relies on quality equipment to provide a continuous infusion of local anesthetic for perineural catheters. Pump selection is thus very important to the success of a program [17, 18]. Ideal characteristic of a pump is one that is simple to operate, allows for multiple infusion rates, is small (but with a large reservoir), and offers a very low failure rate. Simplicity of operation allows for ease of communication between the anesthesiologist and patient over the phone regarding the state of the pump. Multiple infusion rates allow for flexibility in dosing but also have a downside in that the patient may alter the pump rate without the direction of the anesthesiologist. Small size allows ease of movement and ambulation while the pump is connected, while a large reservoir allows for a longer infusion time.
- Drug selection for the pumps can be influenced by several factors. For the author, the simpler the system, the less likely that complications can occur. I choose to limit the drug to only a local anesthetic, and it is always the same drug at the same concentration. This allows for few errors, particularly on block days that are busy and also when a patient calls about their block.
- Selection of a single-shot versus a continuous catheter technique can be important for reasons other than desire for a longer block or patient compliance. In the case of a patient with pulmonary disease, it may be wise to place an interscalene catheter rather than performing either a single shot or no block at all. The advantage of the catheter technique is that the patient's block can be incrementally bolused, titrating to effect. A smaller titrated volume of local anesthetic is less likely to cause phrenic nerve paresis. Also, the presence of the block can decrease the amount of opioid that is needed in the postoperative setting, which can reduce the chances of complications from systemic opioid medications.

Clinical Case Study Revisited

A 65-year-old male with a history of ESRD, CHF with reduced ejection fraction, and HTN presents for left upper extremity AV fistula formation.

- A well-executed brachial plexus block can mitigate several risks of general anesthesia, such as hemodynamic instability, perioperative airway complications, and postop nausea and vomiting in a patient with cardiopulmonary disease.
- For this gentleman, it is important to communicate with the surgery team regarding where the fistula will be placed and if there is a possibility the upper arm will be manipulated during the surgery.
- If the surgical team plans on placing the fistula near the antecubital fossa and/or below the elbow, supraclavicular, infraclavicular, and axillary blocks (with musculocutaneous coverage) are all viable options for surgical anesthesia. At our institution, we often place a supraclavicular block with ultrasound guidance, using approx. 20–30 ml of 1.5% mepivicaine, along with an intercostobrachial

and medial brachial cutaneous nerve field block (if necessary).
- If the surgeon desires to place the fistula/graft well into the upper arm, an interscalene block may be needed to help cover shoulder and upper arm dermatomes.

Review Questions

1. Postoperative follow-up of block performance as well as surveillance for any potential complications is important in any practice. Questions regarding block performance should include which of the following topics?

(a) Pain intensity
(b) Motor function
(c) Sensory function
(d) All of the above

2. Characteristics of patients who are likely to successfully manage a perineural catheter from home include…

(a) Adequate cognitive ability with a good support/caregiver network
(b) Poor cognitive ability
(c) Multiple comorbidities/medications
(d) Absence of adequate social support

3. Which of the following blocks may precipitate respiratory distress via partial diaphragmatic paralysis in a susceptible patient?

(a) Axillary block
(b) Infraclavicular block
(c) Interscalene block
(d) Femoral block

Answers

1. d
2. a
3. c

References

1. Hall MJ, Lawrence L. Ambulatory surgery in the United States, 1996. Adv Data. 1998;359:1–16.
2. Chung F, Ritchie E, Su J. Postoperative pain in ambulatory surgery. Anesth Analg. 1997;85:808–16.
3. Rawal N, Hylander J, Nydahl PA. Survey of postoperative analgesia following ambulatory surgery. Acta Anaesthesiol Scand. 1997;41:1017–22.
4. Brown AR, Weiss R, Greenberg C, et al. Interscalene block for shoulder arthroscopy: comparison with general anesthesia. Arthroscopy. 1993;9:295–300.
5. White P, Issioui T, Skrivanek G, Early J, Wakefield C. The use of a continuous popliteal sciatic nerve block after surgery involving the foot and ankle: does it improve the quality of recovery? Anesth Analg. 2003;97:1303–9.
6. Liu SS, Strodtbeck WM, Richman JM, Wu CL. A comparison of regional versus general anesthesia for ambulatory anesthesia: a meta-analysis of randomized controlled trials. Anesth Analg. 2005;101:1634–42.
7. Ilfeld B, Morey T, Wright T, Chidgey L, Enneking FK. Continuous interscalene brachial plexus block for postoperative pain control at home: a randomized, double-blinded, placebo-controlled study. Anesth Analg. 2003;96:1089–95.
8. Williams B, Kentor M, Vogt M, Williams J, Chelly J, Valalik S, et al. Femoral-sciatic nerve blocks for complex outpatient knee surgery are associated with less postoperative pain before same-day discharge. Anesthesiology. 2003;98:1206–13.
9. Macaire P, Gaertner E, Capdevila X. Continuous post-operative regional analgesia at home. Minerva Anestesiol. 2001;67:109–16.
10. Fredrickson M, Ball C, Dalgleish A. Successful continuous interscalene analgesia for ambulatory shoulder surgery in a private practice setting. Reg Anesth Pain Med. 2008;33:122–8.
11. Ilfeld B, Morey T, Enneking FK. Continuous infraclavicular brachial plexus block for postoperative pain control at home. Anesthesiology. 2002;96:1297–304.
12. Ilfeld B, Morey T, Wang RD, Enneking FK. Continuous popliteal sciatic nerve block for postoperative pain control at home. Anesthesiology. 2002;97:959–65.
13. Rawal N. Postoperative pain treatment for ambulatory surgery. Best Pract Res Clin Anesthesiol. 2007;21:129–48.
14. Klein SM, Buckenmaier CC. Ambulatory surgery with long acting regional anesthesia. Minerva Anestesiol. 2002;68:833–47.
15. Cucchiaro G, Ganesh A. The effects of clonidine on postoperative analgesia after peripheral nerve blockade in children. Anesth Analg. 2007;104:532–7.
16. Casati A, Magistris L, Fanelli G, Beccaria P, Cappelleri G, Aldegheri G, et al. Small-dose clonidine prolongs postoperative analgesia after sciatic-femoral nerve block with 0.75% Ropivicaine for foot surgery. Anesth Analg. 2000;91:388–92.
17. Capdevila X, Bringuier S, Borgeat A. Infectious risk of continuous peripheral nerve blocks. Anesthesiology. 2009;110:182–8.
18. Liu S, Salinas FV. Continuous plexus and peripheral nerve blocks for postoperative analgesia. Anesth Analg. 2003;96:263–72.

Regional Anesthesia in the Critical Care Setting

26

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Introduction

Pain control in the critically ill population is quite challenging, particularly with the lack of a reliable clinical tool that can be used for an objective assessment of pain. Adequate pain control is essential in reducing the stress response during critical illness. Conventional opioid therapy runs the risk of developing respiratory depression, altered mental status, and reduced bowel function. In all these aspects, regional anesthesia can benefit the patient significantly in the critical care environment.

Most published data on this topic is in the form of recommendations based on cohort studies, case reports, expert opinion, and studies focused on intraoperative and postoperative intensive care unit (ICU) use of continuous regional and neuraxial analgesia. This chapter provides a summary of these best practice recommendations.

Epidural Analgesia

The use of epidural analgesia during and after major surgery remains highly variable and dependent on factors including institution-specific protocols, surgeon experience and preferences, and anesthesiologist experience and skills. There is a lack of strong evidence-based studies demonstrating major benefits with epidurals in spite of the abundance of studies performed over the past three decades. However, epidural analgesia is still one of the most commonly used regional anesthesia procedures in the ICU setting. It is mainly used in the ICU to treat conditions such as rib fractures [1, 2], flail chest, cardiothoracic surgery, abdominal surgery, major vascular surgery, major orthopedic surgery, pancreatitis, paralytic ileus, and intractable angina.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_26

Epidural use in the context of rib fractures has been controversial. There are some studies which recommend thoracic epidurals in patients with multiple rib fractures [1, 2]. According to these studies, thoracic epidurals decrease the rate of nosocomial pneumonia and the duration of mechanical ventilation. However, the latest Eastern Association for the Surgery of Trauma (EAST) guidelines [3, 4] did not find convincing evidence regarding decreased rates of pneumonia or duration of mechanical ventilation.

Thoracic epidurals have been used more frequently in patients undergoing cardiac surgeries. Monaco et al. reported that a thoracic epidural along with general anesthesia provides good hemodynamic stability and postoperative analgesia. They also reported decreased rates of intraoperative and postoperative cardiovascular complications in elderly patients undergoing cardiac surgery for mitral regurgitation [5].

In addition, thoracic epidurals have been widely used to relieve pain after thoracic surgery [6]. A meta-analysis performed by Popping et al. reported decreased rates of postoperative pulmonary complications in patients with thoracic epidurals undergoing thoracic surgeries [7]. Benefits of epidurals for urological and gynecological surgeries are limited owing to advances in surgical techniques that involve smaller incision sites. It is well accepted that epidural analgesia after major surgery results in early mobility, decreased risk of venous thromboembolism, decreased cardiopulmonary complications, and superior pain control. However, having an epidural potentially increases the risk of hypotension, motor block, urinary retention, and neurological injury. It should be noted that ketamine for rib fractures and other critically ill patients is becoming commonplace and a component of a multimodal approach in the intensive care setting.

The effect of epidural analgesia on mortality remains a controversial subject. Some studies [8–10] have shown improved rates of mortality. Other studies, including the MASTER (Multicenter Australian Study of Epidural Anesthesia) trial [11] and the Veteran Affairs study [12], reported no decrease in mortality rates in patients with epidurals undergoing major abdominal surgery. In this regard, a 2014 systemic review and meta-analysis by Popping et al. reported a mortality benefit in patients having postoperative analgesia by an epidural.

Sepsis with positive blood cultures might be a contraindication in an ICU patient. Other comorbidities common to the ICU patient, including depressed level of consciousness, spinal cord injury, coagulopathy, hemodynamic instability, sedation, and positioning, may preclude an ICU patient from receiving an epidural. Strategies implemented to guide epidural placement in high-risk patients include measuring serum markers such as procalcitonin and C-reactive protein with suspected bacteremia [13] and tunneling of epidural catheters in order to reduce infection [14]. Motor evoked potentials (MEP) and somatosensory evoked potentials (SSEP) of the tibia can also be used to monitor patients with altered mental status. Current ASRA guidelines should be followed while placing epidurals on patients receiving anticoagulation.

Peripheral Nerve Block for Upper Extremity

There are few completed studies evaluating the use of upper extremity blocks for patients in the critical care setting. In the trauma ICU, continuous upper extremity nerve catheters can be used in patients with severe trauma to the upper extremity, particularly in patients with coexisting traumatic brain injury and in whom using opioids might interfere with the neurological exam [15]. Use of peripheral nerve blocks and catheters has been shown to decrease opioid consumption and pain scores, in turn prompting earlier mobilization and rehabilitation which leads to higher patient satisfaction [16–18]. Trauma ICU patients might further benefit from continuous nerve catheters related to sympathectomy-associated vasodilation and improved blood flow, especially in patients undergoing revascularization and reimplantation procedures [19].

Compartment syndrome can occur in patients with continuous upper extremity nerve catheters. Keeping the rate of infusion as low as possible while providing sensory blockade may not lessen the rate at which compartment syndrome occurs

but perhaps allows for earlier detection. Breakthrough pain in a patient receiving continuous upper extremity analgesia from a functional nerve catheter may serve as a red flag, prompting further clinical investigation into possible compartment syndrome [17, 20].

Neurological impairment and sedation can impede the placement of continuous upper extremity nerve catheters due to concerns for nerve injury. Nerve stimulators and ultrasound, used as a guide for catheter placement, decrease rates of injury to the nerves and surrounding structures [21]. Interscalene catheters carry a particular concern for accidental phrenic nerve block and subsequent hemidiaphragm dysfunction. Post-pressure ventilation does not carry any increased risk in these patients, but weaning of the ventilator may incite respiratory compromise and warrants close monitoring [15]. Lower volumes of local anesthetic lessen the chance of a phrenic nerve block in this scenario [22, 23]. The proximity of interscalene catheters to the tracheostomy site increases risk of infection, so daily monitoring of the catheter site should be performed.

Continuous supraclavicular, infraclavicular, and axillary catheters can be used to provide good postoperative analgesia to the patients with injuries to the arm, elbow, and hand. Local anesthetic can be bolused to provide surgical anesthesia to patients who need dressing changes or debridement for burns and other soft tissue wounds [15]. In general, there is an increased risk of pneumothorax with both supraclavicular and infraclavicular nerve catheter placements. With this risk in mind, placing a prophylactic chest tube on the side of the nerve catheter placement can be beneficial. When pneumothorax is of concern in a high-risk patient with pulmonary compromise, an axillary nerve catheter should be performed [15].

Peripheral Nerve Blocks for the Lower Extremities

There are a number of blocks available for lower extremity analgesia, with multiple targets present from the lumbar plexus to the ankle. Most patients requiring acute pain control in the ICU are those involved in trauma or crush injury and require surgery with systemic support. Typically, femoral catheters are used for anesthesia and analgesia in combination with other lower extremity blocks for various surgical procedures and analgesia [24]. 20mL of anesthetic is adequate for femoral single-injection block, while 8–10 mL/h of 0.25% bupivacaine or 0.2% ropivacaine is adequate for analgesia in the postoperative period; this can be titrated down in concentration as tolerated, but 0.5% is typical for anesthesia [24]. Femoral catheters can be combined with sciatic blocks for total lower extremity analgesia [24]. Lumbar plexus blocks, also known as the psoas block, can combine with the sciatic block as a continuous catheter. This block is the most proximal and reliable block for the femoral, obturator, and lateral femoral cutaneous nerves [25]. It is an advanced block, requiring the use of nerve stimulation in addition to ultrasound guidance [25]. The sciatic block is usually combined with other blocks including femoral, lateral femoral cutaneous, obturator, etc., as it is rarely used alone [26]. It is generally indicated in ankle fractures and tibia fractures [26]. When combined with the above techniques, adequate pain control should result. Approximately 20–25 mL of anesthetic can be used in the block, so care must be taken with other blocks to prevent toxic dosing [26]. Typically, 0.5% of bupivacaine and 0.5–0.75% ropivacaine provide adequate motor blockade [26]. The subgluteal and midthigh approaches are most commonly used and performed under ultrasound guidance; confirmation with nerve stimulation is preferable [26]. Ayling et al., in a large retrospective chart review, described adequate pain control with decreased opioid requirements in those patients undergoing lower limb amputation via continuous perineural infusions of local anesthetic [27].

Other Regional Analgesic Techniques

A multitude of other regional injections and infusions are available in the ICU setting, each with potential to reduce opioid requirements and

long-term dependence. The transverse abdominal plane (TAP) block can be used for those who cannot tolerate neuraxial anesthesia, hemodynamically unstable, or coagulopathic patients. TAP catheters are also helpful for postoperative analgesia. The limitations of the TAP block involve sensation in the pelvic floor as well as visceral pain [16]. Other abdominal fascial plane blocks and thoracic fascial pain blocks are available for a variety of surgeries in unstable trauma patients [16]. Celiac plexus blocks are useful in the prevention of visceral cancer-related pain, particularly in the gastric and pancreatic areas [28], and are typically combined with TAP or intercostal blocks in abdominal surgery. The intercostal blocks are typically used for analgesia following upper abdominal, thoracic surgery, and rib fractures, and are also frequently useful during the placement of chest tubes and gastrostomy tubes [29]. In addition, the paravertebral block and catheter infusions are excellent in unilateral chest trauma and procedures, including thoracotomies, nephrectomies, and breast surgery, and can be used if an epidural catheter fails or if analgesia is inadequate [30]. Clinicians may also employ local infiltration in the ICU setting for small bedside procedures, including central line insertion, arterial line insertion, lumbar punctures, and other bedside interventions.

Systemic Effects and Complications of Local Anesthesia in Critically Ill Patients

Local anesthesia is a therapy aimed at disrupting the afferent neuronal propagation of pain action potentials initiated in the periphery before they reach the pain processing center in the central nervous system. Local tissue infiltration, peripheral nerve blockade, and neuraxial anesthesia are the three major modalities for the application of local anesthesia. When included as part of a multimodal approach to pain control, local anesthesia has shown numerous benefits such as improved patient satisfaction, decreased opiate use, decreased ICU admissions, and decreased length of hospitalization [12]. Also, presumably as a result of a decreased sympathetic response to surgery, the use of regional anesthesia, when compared to general anesthesia, may in fact reduce perioperative mortality [3–5]. These beneficial effects, even when small, may be of significant importance for the critically ill patient. On the other hand, in the same critically ill patient, the generally benign systemic effects of local anesthetics could be just as disastrous and even fatal. Further, despite the overall rarity of major complications from local anesthesia, the critically ill patient may be comparatively at an elevated risk.

Known systemic effects and complications of local anesthesia include, but are not limited to, central nervous system (CNS) toxicity, cardiotoxicity, hypotension, allergic reaction, methemoglobinemia, bleeding, infection, pneumothorax, and hemidiaphragmatic paralysis [6]. Each patient and all of their individual comorbidities must always be considered when assessing a patient's risk for one of many potential complications of local anesthetics. This is especially true in the critical care unit where certain pertinent comorbidities such as advanced age, coronary artery disease, cardiomyopathies, dysrhythmias, pulmonary disease, coagulopathies, and bacteremia are prevalent.

One of the most feared complications of local anesthetics is local anesthetic systemic toxicity (LAST). LAST is a life-threatening culmination of CNS toxicity and cardiotoxicity resulting from elevated plasma levels of local anesthetics either from increased absorption of local anesthetics from the periphery into the systemic circulation or by an inadvertent intravascular injection. The block site, choice of anesthetic, and amount of drug administered are major determinants of the speed and degree of systemic absorbance. However, certain patient characteristics have been found to be associated with an increased risk of systemic toxicity. These include age over 70, cardiac arrhythmias, and preexisting cardiac, renal, or hepatic dysfunction [7]. A dose reduction may be prudent in patients when presenting with one or more of these risk factors. Surprisingly, though critically ill patients can often be frail and emaciated, a dose reduction based on body mass index (BMI) alone has not

been shown to correlate with local anesthetic plasma levels. Finally, it may be difficult to recognize LAST in the critically ill patient; if suspected, the definitive therapy is an infusion of 20% lipid emulsion [8].

Neuraxial anesthesia in particular warrants special consideration with regard to the critically ill. One may want to avoid spinal anesthesia use in the hypotensive patient, since a sympathectomy with vasodilation can be expected. If deemed necessary, a bolus of 20 cm^3/kg crystalloid 20 min prior to the procedure has been shown to decrease the incidence of hypotension [9]. Patients in the critical care unit often develop coagulopathies such as thrombocytopenia or are given anticoagulant medications to prevent or treat thrombosis. Coagulopathies increase the risk of developing an epidural hematoma, which if untreated results in permanent paralysis.

There are also some interesting systemic effects of local anesthetics that have been observed, but not yet completely understood. Continuous infusion catheters may serve as pathways for infection or, at the very least, be implicated as a source in a patient found to be bacteremic. However, in vitro studies have found bupivacaine to possess intrinsic antibacterial activity [10]. In vitro studies also suggest that lidocaine, but not ropivacaine or bupivacaine, may unfortunately impair the molecular processes involved in wound healing [11]. Lastly, while still inconclusive, it has been suggested that the use of regional anesthesia may decrease cancer recurrence in oncologic surgery; and there are ongoing studies at this time to evaluate the validity of this [12].

General Management Aspects of Continuous Regional Anesthesia Catheters in Critically Ill Patients

Critically ill patients can present numerous barriers to effective monitoring of regional anesthesia administration via continuous catheters in the ICU. The presence of endotracheal tubes, altered levels of consciousness, and administration of sedative drugs can often hinder communication, making it difficult to assess the effectiveness of pain control or be verbally alerted to complications the patients may be experiencing. Therefore, it is imperative that the ICU personnel not only be attentive and detailed but well trained on the maintenance, complications, and assessment of continuous catheters. Personnel should be educated on how to best handle catheters to increase longevity and decrease errors in order to alleviate infection risk [31]. Critical care personnel should also be cognizant of the cardinal signs of potential complications and able to effectively execute protocols to ensure the resolution of such complications. In addition, there should be effective means for assessing and monitoring the efficacy of analgesia administration, and personnel should be competent at said assessments and making necessary alterations to achieve optimal analgesia [18]. To achieve these means, the ICU and anesthesia teams must work together seamlessly.

In regard to maintenance of continuous catheters, catheter-related infections and dislodgement remain primary concerns. While tunneling catheters and strict adherence to aseptic technique have been mainstays in reducing the risk of infection, Bomberg suggested that a prophylactic dose of antibiotics was associated with decreased numbers of both peripheral and epidural catheter infections in his study [32]. While catheters are characteristically left in place 2–7 days postoperatively, a study by Compere was unable to find a correlation between prolonged use of tunneled catheters and increased risk of infection or inflammation, suggesting a longer duration of use may be feasible [33]. A study conducted by Pacenta, in which a peripheral nerve block ran continuously for a total of 88 days in an immunocompromised patient, without signs of infection, further supports this assertion [33].

In addition to reducing infection risk, tunneling of catheters has also shown to reduce the chance of dislodgement. Furthermore, dressing technique has proven to play a critical role in ensuring the catheter is secure. A study by Borg demonstrated that the use of an anchoring device may increase the strength of the dressing's integrity and decrease rates of premature dislodgment [34].

As previously proved by Langevin, when catheters are disconnected and an observable fluid column is static within, the proximal 25 cm of the catheter may be placed in disinfectant, cut, and then reconnected to a sterile connector. However, this technique is not appropriate for stimulating catheters as the metal spiral wire may be unwound. Though previous studies have shown high incidence of colonization of femoral catheters without septic complications, the decision to reconnect or remove the catheter altogether must be evaluated using one's clinical judgment based on the circumstances of each particular case [31].

With regard to morbidity and mortality and postoperative regional, recent data has been sparse. Studies previously conducted by Moen and associates, as well as Auroy and co-workers, have produced results consistent with low risk of permanent neurological damage or death [31].

Case Study

A 19-year-old sickle cell patient on long-standing opioids is involved in a major car accident at a high rate of speed. His injuries are extensive to his left lower extremity. He is admitted to the trauma critical care unit. Surgery is successfully performed on a badly fractured left femur. Despite being placed on patient-controlled analgesia with hydromorphone, the patient is unable to achieve any type of significant relief. A femoral catheter is placed with a combination of ropivacaine and fentanyl with a drop in his pain scores from 9–10/10 to 2–3/10. He is later discharged from the unit on day 4 and is able to go home on day 9.

Summary

As in many other clinical environments, a multimodal approach to pain control is recommended for the ICU setting. The use of regional and neuraxial analgesia can play a valuable role in this approach to achieve optimal pain relief, thereby reducing physiologic and psychologic stress. In addition, reduction in the utilization of opiate therapy decreases the risk for withdrawal syndrome, mental status changes, delirium, nausea and vomiting, and reduced gastrointestinal motility.

A structured team-wise approach with highly qualified nursing care and well-trained physicians is essential to the safe use of these techniques in the ICU setting. The recommendations in this chapter are based on small series, uncontrolled trials, and extrapolated conclusions. Further research in this area is needed before definitive guidelines can be produced.

Review Questions

1. The addition of epinephrine to local anesthetics for regional blockade prolongs the action of the blockade.
   (a) True
   (b) False
2. Peripheral nerve blocks can enhance the detection of compartment syndrome.
   (a) True
   (b) False
3. What is the first sign of local anesthetic toxicity in a critically ill patient?
   (a) Hypotension
   (b) Circumoral numbness
   (c) Tachycardia
   (d) Delirium
4. What is the first step in the treatment of local anesthetic toxicity?
   (a) Airway control with 100% oxygen
   (b) Seizure control
   (c) Reversal with 20% intralipid
   (d) None of the above
5. What is the 20% lipid emulsion dose for local anesthetic toxicity?
   (a) Continuous infusion of 0.25 mL/kg/min
   (b) Bolus of 2 mL/kg/min and then continuous infusion of 0.5 mL/kg/min
   (c) Bolus of 1.5 mL/kg/min then continuous infusion of 0.25 mL/kg/min
   (d) Continuous rate of 1 mL/kg/min

Answers:

1. a
2. a
3. b
4. a
5. c

References

1. Bulger EM, Edwards T, Klotz P, Jurkovich GJ. Epidural analgesia improves outcome after multiple rib fractures. Surgery. 2004;136(2):426–30.
2. Jensen CD, Stark JT, Jacobson LL, Powers JM, Joseph MF, Kinsella-Shaw JM, Denegar CR. Improved outcomes associated with the liberal use of thoracic epidural analgesia in patients with rib fractures. Pain Med. 2017;18(9):1787–94.
3. Galvagno SM Jr, Smith CE, Varon AJ, Hasenboehler EA, et al. Pain management for blunt thoracic trauma: a joint practice management guideline from the eastern Association for the Surgery of trauma and trauma anesthesiology society. J Trauma Acute Care Surg. 2016;81(5):936–51.
4. Carrier FM, Turgeon AF, Nicole PC, Trépanier CA, Fergusson DA, Thauvette D, Lessard MR. Effect of epidural analgesia in patients with traumatic rib fractures: a systematic review and meta-analysis of randomized controlled trials. Can J Anaesth. 2009;56(3):230–42. https://doi.org/10.1007/s12630-009-9052-7.
5. Monaco F, Biselli C, De Luca M, Landoni G, Lembo R, Zangrillo A. Thoracic epidural anesthesia in elderly patients undergoing cardiac surgery for mitral regurgitation feasibility study. Ann Card Anaesth. 2012;15(2):164–5. https://doi.org/10.4103/0971-9784.95085.
6. Maxwell C, Nicoara A. New developments in the treatment of acute pain after thoracic surgery. Curr Opin Anaesthesiol. 2014;27(1):6–11. https://doi.org/10.1097/ACO.0000000000000029.
7. Pöpping DM, Elia N, Marret E, Remy C, Tramèr MR. Protective effects of epidural analgesia on pulmonary complications after abdominal and thoracic surgery: a meta-analysis. Arch Surg. 2008;143(10):990–9.; discussion 1000. https://doi.org/10.1001/archsurg.143.10.990.
8. Wijeysundera DN, Beattie WS, Austin PC, Hux JE, Laupacis A. Epidural anaesthesia and survival after intermediate-to-high risk non-cardiac surgery: a population-based cohort study. Lancet. 2008;372(9638):562–9. https://doi.org/10.1016/S0140-6736(08)61121-6. Epub 2008 Aug 8
9. Wu CL, Hurley RW, Anderson GF, Herbert R, Rowlingson AJ, Fleisher LA. Effect of postoperative epidural analgesia on morbidity and mortality following surgery in medicare patients. Reg Anesth Pain Med. 2004;29(6):525–33. discussion 515-9
10. Park WY, Thompson JS, Lee KK. Effect of epidural anesthesia and analgesia on perioperative outcome: a randomized, controlled veterans affairs cooperative study. Ann Surg. 2001;234(4):560–9. discussion 569-71
11. Rigg JR, Jamrozik K, Myles PS, Silbert BS, Peyton PJ, Parsons RW, Collins KS, Anaethesia Trial MASTER. Study group. Epidural anaesthesia and analgesia and outcome of major surgery: a randomised trial. Lancet. 2002;359(9314):1276–82.
12. Rodgers A, Walker N, Schug S, McKee A, et al. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomised trials. BMJ. 2000;321(7275):1493.
13. Bell K, Wattie M, Byth K, Silvestrini R, Clark P, Stachowski E, Benson EM. Procalcitonin: a marker of bacteraemia in SIRS. Anaesth Intensive Care. 2003;31(6):629–36.
14. Bomberg H, Kubulus C. Tunnelling of thoracic epidural catheters is associated with fewer catheter-related infections: a retrospective registry analysis. Br J Anaesth. 2016;116(4):546–53. https://doi.org/10.1093/bja/aew026.
15. Textbook of Regional Anesthesia and Acute Pain Management; editor, Admir Hadzic Chapter 67:Regional Analgesia in the Critically Ill, Sebastian Schulz-Stubner.
16. Ehieli E, Yalamuri S, Brudney CS, Pyati S. Analgesia in the surgical intensive care unit. Postgrad Med J. 2017;93(1095):38–45. https://doi.org/10.1136/postgradmedj-2016-134047.
17. Aguirre J, Del Moral A, Cobo I, et al. The role of continuous peripheral nerve blocks. Anesthesiol Res Pract. 2012;2012:560879. https://doi.org/10.1155/2012/560879.
18. Guedes L, Rebelo H, Oliveira R, et al. Regional analgesia in intensive care. Rev Bras Anestesiol. 2012;62:719–30. https://doi.org/10.1016/S0034-7094(12)70170-8.
19. Chelly JE, Ghisi D, Fanelli A. Continuous peripheral nerve blocks in acute pain management. Br J Anaesth. 2010;105(Suppl 1):i86–96. https://doi.org/10.1093/bja/aeq322.
20. Mar GJ, Barrington MJ, McGuirk BR. Acute compartment syndrome of the lower limb and the effect of postoperative analgesia on diagnosis. Br J Anaesth. 2009;102:3–11. https://doi.org/10.1093/bja/aen330.
21. Le-Wendling L, Enneking FK. Continuous peripheral nerve blockade for postoperative analgesia. Curr Opin Anaesthesiol. 2008;21(5):602–9. https://doi.org/10.1097/ACO.0b013e32830a4be6.
22. Nadeau MJ, Lévesque S, Dion N. Ultrasound-guided regional anesthesia for upper limb surgery. Can J Anaesth. 2013;60(3):304–20. https://doi.org/10.1007/s12630-012-9874-6. Epub 2013 Feb 2
23. Lee JH, Cho SH, Kim SH, Chae WS, Jin HC, Lee JS, Kim YI. Ropivacaine for ultrasound-guided interscalene block: 5 mL provides similar analgesia but less phrenic nerve paralysis than 10 mL. Can J Anaesth.

2011;58(11):1001–6. https://doi.org/10.1007/s12630-011-9568-5. Epub 2011 Aug 20
24. Farag, Ehab and Brown, David L. Ch 14, Femoral Block. Brown's Atlas of Regional Anesthesia, 5th edition, 2017, 115–126.
25. Mounir-Soliman, Loran and Brown, David L. Ch 12 Lumbar Plexus Block, Brown's Atlas of Regional Anesthesia, 97–102.
26. Farag, Ehab and Brown, David L. Ch 13 Sciatic Block, Brown's Atlas of Regional Anesthesia, 103–113.
27. Ayling OG, et al. Continuous regional Anaesthesia provides effective pain management and reduces opioid requirements following major lower limb amputation. Eur J Vasc Endovasc Surg. 2014;48(5):559–64.
28. Brown, David L. Ch 51, Celiac Plexus Block, Brown's Atlas of Regional Anesthesia, 321–328.
29. Brown, David L. Ch 35, Intercostal Block, Brown's Atlas of Regional Anesthesia, 227–230.
30. Farag, Ehab. Ch 39, Paravertebral Block, Brown's Atlas of Regional Anesthesia, 245–248.
31. Bomberg H, Krotten D, Kubulus C, et al. Single-dose antibiotic prophylaxis in regional anesthesia: a retrospective registry analysis. Anesthesiology. 2016;125(3):505–15.
32. Borg L, Howard SK, Kim TE, Steffel L, Shum C, Mariano ER. A comparison of strength for two continuous peripheral nerve block catheter dressings. Korean J Anesthesiol. 2016;69(5):506–9.
33. Pacenta HL, Kaddoum RN, Pereiras LA, Chidiac EJ, Burgoyne LL. Continuous tunnelled femoral nerve block for palliative care of a patient with metastatic osteosarcoma. Anaesth Intensive Care. 2010;38(3):563–5.
34. Schulz-stübner S, Boezaart A, Hata JS. Regional analgesia in the critically ill. Crit Care Med. 2005;33(6):1400–7.

Part VI

Regional Anesthesia for Chronic Situations

Regional Anesthesia for Chronic Pain

27

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Introduction

Sympathetic blockade has become a mainstay of therapy in the treatment of a variety of cancer-related and chronic pain conditions. The advent of ultrasound-, fluoroscopy-, endoscopy-, and CT-guided techniques has brought this important area of pain control to the regional anesthesiologist and pain management specialist. Imaging has allowed precise placement of needles to deliver local anesthetics, steroids, and neurolytic substances to block various sympathetic ganglia. With these techniques, pain relief can potentially be achieved for a variety of cancer and noncancer pain conditions.

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Autonomic Nervous System

The autonomic nervous system is composed of both the sympathetic and parasympathetic nervous systems, which provide opposing actions to one another. The autonomic nervous system is primarily responsible for a variety of homeostatic mechanisms in the body. These are paramount in maintaining organ perfusion, function, and metabolism. Specific areas of action include maintaining vascular tone, cardiac conduction and inotropy, pulmonary bronchodilation and bronchoconstriction, smooth muscle tone, and the transmission of pain.

Elements of the autonomic system can be found at various levels of the spinal cord. The parasympathetic nervous system is composed of cranial nerves arising from the brainstem and the sacral portion of the spinal cord, and it is termed the "craniosacral" portion of the autonomic nervous system. The sympathetic nervous system is composed of neural fibers arising from the thoracic and lumbar areas of the spinal cord, and it is termed the "thoracolumbar" portion of the spinal cord. It is the sympathetic nervous system that is of interest to the pain specialist, as it is not only important for homeostatic function of the body, but it also acts as a conduit for afferent nociceptive impulses from the periphery and major organs (Fig. 27.1).

The cell bodies of the sympathetic nervous system are found in the intermediolateral column

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_27

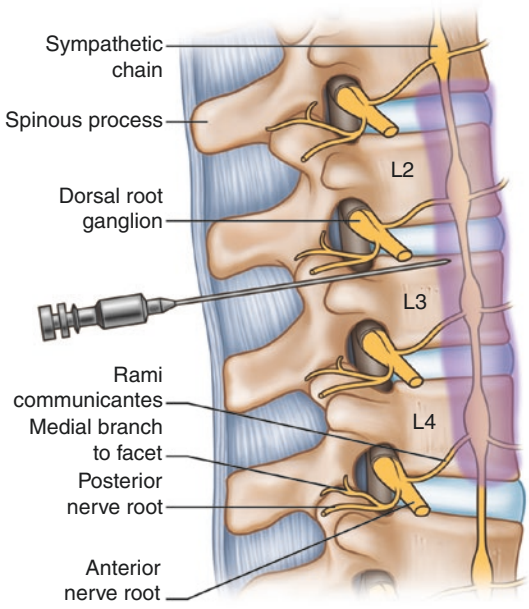

Fig. 27.1 Sympathetic chain and various ganglia

of the thoracolumbar portion of the spinal cord. The primary ventral ramus carries the preganglionic sympathetic fibers as it exits the neural foramen. White rami communicantes allows the sympathetic axons to exit the ventral ramus and enter the paravertebral sympathetic ganglia and chain, which are located at the anterolateral portion of the vertebral body. In the sympathetic ganglia, preganglionic fibers synapse with postganglionic neural cells. Postganglionic fibers either travel through the gray rami communicantes to the original ventral ramus to peripheral sites or travel directly to the organs they affect. A variety of painful conditions can be successfully treated with blockade of the sympathetic chain at various levels including the stellate ganglia, celiac plexus, lumbar sympathetic chain, and superior hypogastric plexus.

Chronic Pain States

CRPS I, CRPS II

Complex regional pain syndrome (CRPS) is a broad diagnosis based on different signs and symptoms. Pain is a presenting symptom in the vast majority of cases of CRPS, with the remaining diagnosis based primarily on history and physical exam. CRPS I (formerly known as reflex sympathetic dystrophy) describes a variety of painful conditions following an insult to an extremity that appears in a regional distribution with a distal predominance of abnormal findings. In CRPS I, a broad range of minor or major injuries to a limb precede the onset of symptoms. CRPS II (formerly known as causalgia) may potentially develop after a peripheral nerve injury. The presence of vasomotor changes (temperature changes, sweating abnormalities, edema, and vascular changes) suggests that sympathetic dysfunction plays a role in many aspects of the disease; this is termed sympathetic-mediated pain.

Complex Regional Pain Syndrome (CRPS)

1. Continuing pain, which is disproportionate to any inciting event.
2. Must report at least one symptom in *three of the four* following categories:
   (a) *Sensory:* reports of hyperalgesia and/or allodynia
   (b) *Vasomotor:* reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry
   (c) *Sudomotor/edema:* reports of edema and/or sweating changes and/or sweating asymmetry
   (d) *Motor/trophic:* reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)
3. Must display at least one sign at time of evaluation in *two or more* of the following categories:
   (a) *Sensory:* evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement)
   (b) *Vasomotor:* evidence of temperature asymmetry and/or skin color changes and/or asymmetry
   (c) *Sudomotor/edema:* evidence of edema and/or sweating changes and/or sweating asymmetry

(d) *Motor/trophic:* evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. There is no other diagnosis that better explains the signs and symptoms.

Subtypes of Complex Regional Pain Syndrome (CRPS)

1. CRPS 1 (old name: reflex sympathetic dystrophy)
2. CRPS 2 (old name: causalgia): defined with electrodiagnostic or other definitive evidence of a major nerve lesion
3. CRPS-NOS (not otherwise specified): partially meets CRPS criteria; not better explained by any other condition

While, primarily, the diagnosis of CRPS is based on clinical criteria, there are several diagnostic tests, with varying sensitivities and specificities, that can be used to support the findings. These tests include plain radiography, bone scan, quantitative sensory testing, temperature differences, MRI, and even skin biopsy. There are also tests analyzing sweat patterns such as thermoregulatory sweat test (TST) and quantitative sudomotor axon reflex test (QSART), the latter of which analyzes small nerve fibers which are linked to sweat glands.

The proposed mechanisms for CRPS involve a variety of physiologic and pathophysiologic factors. Theories involve possible changes to peripheral fibers, causing usual painless stimuli to elicit a painful response via low-threshold mechanoreceptors, resulting in allodynia; axonal injury may further potentiate this mechanism. In addition, communication between the somatic and sympathetic nervous system may result in abnormal sympathetic activity at the site of injury, causing alterations in skin blood flow, temperature regulation, sweating, as well as trophic changes. Furthermore, the somatic sensory nervous system may be more sensitive to circulating catecholamines. Finally, local inflammation and immobility of the affected area are not only proposed causes of CRPS but can also potentiate injury and may predispose to the development of chronic changes. Currently, there is some evidence that immune cell-mediated inflammation and autoimmune responses are involved in the pathogenesis of CRPS.

The management of CRPS is multifactorial. Many treatments have been proposed, but few have withstood rigorous scientific investigation. The mainstay of therapy should focus on early intervention, combined with functional rehabilitation of the extremity. If the patient does not respond rapidly to conservative therapy using NSAIDs and functional rehabilitation, early consultation with a pain specialist should be considered. A variety of therapies can be offered, including neuropathic medications (i.e., tricyclic antidepressants, gabapentin, pregabalin, etc.), sympathetic ganglion blocks for sympathetically maintained pain, intravenous regional sympatholysis, spinal cord stimulation, or even irreversible sympathectomy in selected cases. Patients with acute CRPS should receive sympathetic interventions as soon as possible in order to achieve the highest degree of pain relief and to facilitate physical therapy. In children, CRPS is generally rare; symptoms tend to resolve without invasive intervention.

In patients with CRPS (types I and II), blockade of the sympathetic system (stellate ganglion, lumbar sympathetic chain) can provide profound pain relief when combined with other complimentary methods (physical therapy, neuropathic medications, etc.). The pain component that is relieved by specific sympatholytic procedures is considered sympathetically maintained pain. The positive or negative effect of a sympathetic blockade is not essential for the diagnosis of CRPS; it is, though, the only approach that is used to classify the pain as sympathetically maintained. If pain persists despite sympathetic blockade, the term sympathetic-independent pain is used.

Visceral and Cancer Pain

The pain of cancer, inflammation, and solid viscus distension can be excruciating to many

patients. Classically, these patients tend to be terminal cancer patients, for which tumor-induced visceral pain can be very significant. The generation of visceral and cancer pain involves a complex interplay of local organ and tissue manifestations, the somatic nervous system, and the sympathetic nervous system. Together, this interplay results in transmission of a variety of pain stimuli from solid organs. Due to the diffuse, nonspecific nature that visceral pain (especially cancer visceral pain) can produce, it is essential to understand the basic etiology and mechanisms of pain transmission prior to the institution of therapy.

The characteristics of visceral pain tend to differ from the characteristics of somatic pain. Visceral pain tends to be more diffuse and nonspecific in nature, making the exact pain difficult to localize. In addition, much of the visceral pain can be referred to cutaneous structures, further impeding localization. Some stimuli that lead to visceral pain can be similar to those that cause somatic pain; these include inflammation and ischemia. Stimuli unique for visceral pain include smooth muscle spasm and hollow-organ distension.

Visceral afferent fibers tend to arise on or in close relationship to the innervated organs. While the afferent fibers themselves convey sensory information, these fibers tend to pass with efferent autonomic fibers of the sympathetic and parasympathetic nervous systems; these afferents tend to carry visceral nociceptive information from the organ of interest to the central nervous system. Visceral afferent fibers, while passing with sympathetic fibers, tend to have cell bodies in the dorsal root ganglia and terminate in the dorsal horn of the spinal cord, similar in nature to cutaneous nociceptive fibers. Two important differences in visceral versus nociceptive fibers are (1) visceral fibers tend to have thresholds for stimulation that respond only to noxious stimulation and (2) the number of visceral afferent fibers tends to comprise a smaller proportion as compared to cutaneous afferents.

The relationship of visceral afferent fibers to the autonomic nervous system tends to make autonomic blockade (i.e., celiac plexus block, superior hypogastric plexus block) an attractive option to treat visceral malignant and nonmalignant pain.

Neuropathic Pain

Neuropathic pain occurs due to pathology in the peripheral nerves themselves and is classified as either mononeuropathy, polyneuropathy, mononeuritis multiplex, or autonomic neuropathy. The most common form is (symmetrical) peripheral polyneuropathy, which generally affects the feet and legs since they are the longest nerves from the CNS and are therefore more prone to injury. The form of neuropathy may also be classified on the part of the nerve involved (axonal degradation versus a demyelinating lesion) or based on the size of predominant fiber that is involved (large fiber versus small fiber peripheral neuropathy). If the underlying cause of a neuropathy cannot be identified, it is designated idiopathic. Examples of neuropathic conditions include complex regional pain syndrome, diabetic peripheral neuropathy, postherpetic neuralgia, and phantom limb pain.

Neuropathy may be associated with numerous symptoms throughout the body, depending on the number and type of nerves involved; these symptoms include motor loss, sensory changes (paresthesias, numbness, etc.), and autonomic changes. Loss of muscle tone may be seen due to denervation atrophy. Fasciculations may also be seen, generally about 5 weeks following lower extremity denervation. Sensory symptoms include “negative” changes such as loss of sensation and “positive” changes such as tingling or pain. Uncontrolled diabetics, for example, often have a symmetric polyneuropathy described as a sensation in a “stocking and glove” distribution which feels like pins and needles. Loss of balance and coordination may also occur due to injury to the nerves involved in proprioception. Autonomic neuropathy leads to symptoms such as abnormal blood pressure, heart rate, sexual dysfunction, constipation, bladder control, and sweating [1].

Cranial Nerve and Cervicogenic Pain

Trigeminal neuralgia (TN), also known as tic douloureux, is a neuropathic disorder characterized by episodes of intense facial pain due to aberrant signals from one of the three branches of the trigeminal nerve [ophthalmic (V1), maxillary (V2), or mandibular (V3) branches]. It is estimated that the incidence of trigeminal neuralgia is 4.3 per 100,000 persons a year. Women have a slightly higher incidence (5.9 per 100,000) in comparison to men (3.4 per 100,000) [2]. Various treatment modalities exist for trigeminal neuralgia, including neuropathic agents such as carbamazepine, nerve blocks such as Gasserian ganglion and sphenopalatine ganglion blocks, radiofrequency rhizotomy of these ganglia, microvascular decompression of the trigeminal nerve from the superior cerebellar artery, and gamma knife radiation [3]. Occipital neuralgia is another painful condition due to pathology affecting the occipital nerve, which gets its distribution via the C2 and C3 nerves to form the greater and lesser occipital nerve (therefore, it is not technically a cranial nerve even though it affects the posterior portion of the cranium). Some experts advocate that cervicogenic headaches and occipital neuralgia may be adequately treated with blockade of the "third occipital nerve" at the C2/C3 facet joint itself [4]. The occipital nerves can be the target for nerve blocks and possible rhizotomy. Other causes of cranial nerve pain include herpes zoster ophthalmicus and various forms of headaches which manifest as pain in a cranial nerve distribution (cluster headache, etc.).

Herpes Zoster Pain

After the varicella-zoster-virus-mediated chickenpox has resolved, often during childhood, the virus can remain latent in the dorsal root ganglia where it can reemerge later in life as herpes zoster or shingles. Herpes zoster is a disease characterized by a transient rash in a dermatomal distribution that is usually painful. The term postherpetic neuralgia (PHN) is used if the pain persists after the rash has resolved. Older individuals and immunocompromised individuals are generally the ones that are at significant risk for reactivation of herpes zoster and the subsequent development of PHN. Studies have shown that peripheral and central demyelination in conjunction with neuron destruction may be involved. Both the vaccine against VZV (Varivax) and the vaccine against herpes zoster (Zostavax) may lead to substantial reductions in morbidity from herpes zoster and PHN in the future [5]. Multiple medications are often utilized in reducing the pain associated with PHN, including antidepressants, neuropathic agents, opioids, NMDA receptor antagonists, topical lidocaine, and capsaicin. Intrathecal corticosteroids may play a role in treating PHN, but this is only based on level B evidence and needs to be further studied. Sympathetic blockade and spinal cord stimulation may also play a role in treating the pain of herpes zoster or PHN [6]. Early stellate ganglion block combined with an antiviral agent dramatically decreases the intensity of pain, shortens the duration, and decreases the incidence of postherpetic neuralgia of the face [7].

Phantom Limb Pain and Stump Pain

Limb amputation can be associated with a myriad of symptoms, including phantom limb sensation, phantom limb pain, and stump pain. Phantom limb pain is the phenomenon of experiencing pain in an extremity after that extremity has been removed, either primarily (as in trauma) or secondarily (as in surgical amputations). Patients report experiencing a wide range of pain characteristics including burning, cramping, and tingling as well as lancinating electrical shocks, itching, stabbing, throbbing, and even a feeling of "pins and needles" [8]. Although phantom limb pain is experienced in both upper and lower limb amputees, it tends to be localized distally. The onset of phantom limb pain occurs within the first 24 h for about half of all patients and within a week for another 25% [9]. Both central and peripheral nervous system mechanisms have been proposed for phantom limb pain, and some experts suggest that phantom pain is a combination of both. There are varying treatment modalities with different levels

of evidence as far as efficacy for phantom limb pain. These include treatments which have level 1 evidence such as opioids and gabapentin; level 2 evidence such as amitriptyline, tramadol, tricyclic antidepressants, calcitonin, TENS units, mirror therapy, ketamine, and memantine; and level 3 evidence such as carbamazepine, mirtazapine, perioperative epidural infusions, clonidine, mexilitene, and acupuncture [10, 11]. Stump pain, on the other hand, is the experience of pain at the site of the amputation itself and is often due to a neuroma which has formed at the incision site during the healing process. Stump pain, also called residual limb pain, is often treated with a combination of neuropathic medications, local anesthetic creams, and opioids; occasionally, more invasive modalities such as neuroma resection and muscle reimplantation are also utilized [12].

Review of Blocks

Stellate Ganglion Block

Introduction

The stellate ganglion is, essentially, a fusion of the superior thoracic sympathetic ganglion and the inferior cervical sympathetic ganglion. This fusion is present in about 80% of the population. The stellate ganglion is oval in shape and is about 2–3 cm long and 0.5–1 cm wide. Cell bodies for the sympathetic fibers that supply the head, neck, and upper extremity arise from T1 to T8, sometimes including T9. Preganglionic fibers travel to the sympathetic chain and travel cephalad to synapse in the inferior, intermediate, or superior cervical ganglia. Postganglionic fibers either travel along the gray rami communicantes to join the ventral rami comprising the cervical and brachial plexus, while the remaining postganglionic fibers travel from the ganglia directly to the head, neck, and upper extremity as perivascular structures. Some sympathetic fibers bypass these ganglia completely and course with the vertebral artery; thus, blockade of these ganglia can sometimes produce inconsistent and incomplete sympathetic blockade to structures in the head and neck, rendering this block of little value in such cases. The stellate ganglia are separated by loose connective tissue, allowing local anesthetic spread to superior and inferior sympathetic structures; this also allows local anesthetic spread to nonrelated structures, such as the brachial plexus.

The stellate ganglion block has been used successfully to treat a variety of sympathetically maintained syndromes of the upper extremity. It has long been one of the cornerstones of therapy (along with physical therapy and neuropathic medications) for complex regional pain syndrome (types I and II) of the upper extremity. Stellate ganglion block has also been used for a variety of upper extremity neuropathic pain syndromes, including postherpetic neuralgia and phantom limb pain. In addition, stellate ganglion blocks have been used successfully to treat syndromes of vascular insufficiency, including embolic disease, Raynaud's disease, vasospasm, and even angina pectoris.

Block Technique

A variety of fluoroscopy- and ultrasound-guided techniques are utilized for stellate ganglion blockade. This text will describe blockade of the stellate ganglion using both of these modalities, depending on operator preference. Prior to performance of the block, it is mandatory that monitoring, IV supplies, airway equipment, and resuscitative drugs are immediately available. In addition, it is important that personnel trained in dealing with any immediate complications of the blockade are readily available.

The anterior approach is generally used to access the stellate ganglion.

1. The patient is placed supine, with a roll placed underneath the patient's shoulders; this facilitates extension of the patient's neck.
2. The transverse process of C6 is identified with fluoroscopy. The transverse process joins the vertebral body inferior to the uncinate process of the vertebral body.
3. Next, the skin is prepped with an antiseptic solution.
4. A skin wheal is placed over the tubercle, and a small needle (23–25 gauge, 2 in.) is advanced in a coaxial plane to contact the tubercle.

5. Once on the tubercle, the needle is withdrawn 2 mm.
6. Tubing is connected to the needle, the syringe is steadied, and aspiration is carried out.
7. A test dose of 1 cc of local anesthetic solution should be injected while watching for complications of intravascular or intrathecal injection.
8. Local anesthetic (generally 8–10 cm^3 of 0.25% bupivacaine) is given slowly with frequent aspiration, and attention is paid to keeping the needle in a stable position.

Signs of successful blockade include clinical recognition of the presence of Horner's syndrome (miosis, ptosis, enophthalmos, conjunctival injection, and hemianhidrosis). However, this does not always indicate complete sympathetic blockade of the upper extremity. Assessment of sympathetic blockade can be done clinically by examination of the extremity for venodilation, vasodilation, and warmth (a temperature increase of 1–3 °C is typically seen). Alternative methods include measuring skin resistance (sympathogalvanic response), Doppler flow measurements, microneurography, and the sweat test.

Fluoroscopy-/Ultrasound-Guided Approach

Depending on operator comfort and preference, a fluoroscopy- or ultrasound-guided approach can also be used for confirmation of needle placement and local anesthetic spread, with appropriate needle localization and placement on Chassaignac's tubercle with direct visualization. While performing the fluoroscopic technique, contrast dye can be used to confirm appropriate placement prior to local anesthetic injection. Ultrasound-guided stellate ganglion blockade may decrease the risk of soft tissue and vascular injury as it allows better visualization of these structures [13]. The patient is positioned as above, and the ultrasound probe is oriented transversely, lateral to the cricoid cartilage. This allows visualization of the thyroid gland, vertebral artery, esophagus, pleura, nerve roots, and longus colli muscle. An in-plane technique is utilized (both lateral and medial approaches have been described) to contact Chassaignac's tubercle, withdrawing 2 mm and injecting local anesthetic. Appropriate spread anterolateral to the longus colli muscle, deep to the prevertebral fascia, and superficial to the fascia investing the longus colli muscle can be seen [13].

Complications

Complications of the stellate ganglion block include mechanical, infectious, bleeding, and pharmacologic ones. Mechanical complications constitute direct nerve and visceral injury during insertion/manipulation of the needle. These include brachial plexus injury, tracheal injury, esophageal injury, pneumothorax, hemothorax, and chylothorax. Bleeding complications are generally caused from a vascular injury with the needle, resulting in local hematoma or more significant perivascular bleeding. Infectious complications include local abscess, cellulitis, or osteomyelitis. Pharmacologic complications include blockade of the recurrent laryngeal nerve (resulting in hoarseness), blockade of the phrenic nerve (resulting in respiratory dysfunction), brachial plexus blockade (resulting in upper extremity weakness), vertebral artery injection (resulting in seizures), or possible epidural/intrathecal injection (resulting in a high spinal block).

Celiac Plexus Block

Introduction

The celiac plexus is located in the retroperitoneum at the T12–L1 level and surrounds major vascular structures, including the abdominal aorta and branching arteries. It is a diffuse network of nerve fibers, composed of both sympathetic fibers from the anterolateral horn of the spinal cord from T5 to T12 (greater, lesser, and least splanchnic nerves) and parasympathetic fibers from the vagus nerve. Autonomic innervation is supplied to major gastrointestinal organs, such as the liver, gallbladder, pancreas, stomach, small bowel, and ascending and transverse colon. Nociceptive impulses from the abdominal viscera travel with the sympathetic nerves.

Local anesthetic and neurolytic blockade of the celiac plexus have been used for both malignant and chronic abdominal visceral pain. Blocks with variable success rate have been used for management of acute or chronic inflammatory pain (i.e., pancreatitis). Neurolytic celiac plexus blocks have most commonly been used for management of malignant intra-abdominal pain, particularly pancreatic cancer-related pain. The block can achieve dramatic pain relief and eliminate the need for high-dose opioid therapy (and its inherent side effects) in the management of end-of-life malignant cancer pain. In one meta-analysis study of pain relief for cancer pain following celiac plexus block, good to excellent pain relief was reported in 89% of patients 2 weeks after the neurolytic celiac plexus blockade was performed. The study also revealed there was partial to complete pain relief in 90% of patients 3 months post block and in 70–90% of patients until death, even beyond the 3-month timeline.

Block Technique

Due to the presence of major vascular and neural structures encountered during block placement, it is recommended that an imaging modality (most commonly fluoroscopy- or CT-guided technique) be chosen to confirm appropriate needle placement. Three approaches to the treatment of visceral intra-abdominal malignancy pain include the retrocrural approach to block the celiac plexus (classic approach), the anterocrural approach to block the celiac plexus (transaortic approach), or a block of the splanchnic nerves (not described here, but involves advancement to the anterolateral portion of T12–L1).

1. An imaging modality should be chosen, emphasizing operator preference and comfort; generally, fluoroscopy is chosen for the interventional pain physician.
2. The patient should be placed in prone position, and the skin prepped with an antiseptic solution. The area should be draped and sterile technique utilized.
3. Radiographic guidance should verify the location of the 12th rib and the L1 vertebral body.
4. An entry point is chosen 5–7 cm left of the midline and 1–2 cm below the inferior margin of the 12th rib; a local anesthetic wheal is made at the entry point.
5. Using radiographic guidance, a 22-gauge spinal needle is advanced to the anterolateral margin of the L1 vertebral body.
6. A second needle is advanced on the right side using the same approach; the needles are passed no further than 0.5 cm anterior to the anterior border of L1.
7. For the anterocrural approach, the original (left-sided) needle is advanced 2–3 cm beyond the vertebral body while continuously aspirating; when blood is encountered (indicating likely intra-aortic placement), the needle should be advanced until there is negative aspiration, placing of the needle anterior to the aorta.
8. Needle position can be verified using a small amount of radiocontrast media; this will not only confirm proper needle placement but will also rule out intravascular needle placement.
9. For the retrocrural approach, 20–25 cm^3 of solution are used per side; for the anterocrural technique, only 8–10 cm^3 of solution are required.
10. Local anesthetic is chosen for temporary blockade (e.g., 0.25% bupivacaine), while alcohol and phenol are chosen for neurolytic blockade. The need for higher volumes precludes the use of phenol in the retrocrural approach.

Complications

The adverse effects of celiac plexus blockade can be divided into expected physiologic side effects and complications. Expected physiologic side effects include diarrhea, abdominal cramping, and hypotension. These effects are generally transient and are due to sympathetic blockade. In a meta-analysis study, the most common adverse effects were local pain (38%), diarrhea (44%), and hypotension (38%). In addition, the side effects vary with the approach chosen. Hypotension is more common with the retrocrural technique, while diarrhea is more common with the anterocrural technique.

Complications of celiac plexus block include injury to adjacent structures [kidney injury (resulting in hematuria), lung injury (resulting in pneumothorax), vascular injury (resulting in aortic dissection or retroperitoneal hemorrhage)], intravascular injection, and paraplegia. Hemorrhage can be caused by bleeding into the retroperitoneum or bleeding into abdominal viscera. Damage to vascular structures, although rare, has been reported, including dissection of the abdominal aorta. It is recommended that the transaortic technique be avoided in patients with aortic pathology or atherosclerosis. Intravascular injection can occur with local anesthetic or neurolytic substances. Local anesthetic levels can reach high enough levels to cause toxicity with the high volumes required for the retrocrural approach to celiac plexus blockade. Intravascular injection of phenol can cause symptoms similar to local anesthetic toxicity.

The most feared complication of celiac plexus neurolysis is paraplegia. This is thought to occur because of spasm or necrosis of lumbar segmental arteries that perfuse the spinal cord. In many patients, the artery of Adamkiewicz (with a variable level of appearance) is the dominant blood supply to the anterior two-thirds of the spinal cord; a spasm can cause complete paralysis. Other factors are likely responsible as well, such as direct vascular injury and possible retrograde spread of neurolytic agent to the spinal cord.

Lumbar Sympathetic Block

Introduction

Lumbar sympathetic block (LSB) is a modality available to aid in the inhibition of sympathetically mediated pain in the lower extremities (just as stellate ganglion block is used for such pain in the upper extremities). The LSB can be used to both help in the diagnosis and treatment of sympathetically maintained pain in conditions such as CRPS type I and type II as well as phantom limb pain. The lumbar sympathetic chain consists of three to five ganglia which lie anterior to the L2, L3, and L4 vertebral bodies. It is located anterior to the psoas muscle and posterior to the vena cava on the right side and to the aorta on the left. Based on anatomic studies, the number of rami which connect to each ganglia varies between zero and six, with the vast majority having just one ramus [1].

Block Technique

1. The patient is generally placed in prone position with a pillow under the abdomen to decrease the lumbar lordosis.
2. Using fluoroscopic guidance, the spinous processes of L2, L3, and L4 are identified and marked.
3. The fluoroscope is then rotated obliquely toward the side to be blocked, visualizing the transverse process overlying that particular lumbar segment.
4. The area of proposed needle entry is then infiltrated with local anesthetic, and then, a 10-cm 20-gauge needle is inserted and advanced until it comes in contact with the transverse process. The needle depth is noted and then withdrawn slightly, angling caudad, and walked inferiorly off the transverse process. It should be avoided to enter the skin more than 7 cm lateral from the midline and inadvertently contact visceral structures during needle advancement (kidney, liver, spleen, etc.).
5. A slight medial angulation is used to contact the vertebral body at that level. This distance should also be noted. Then the fluoroscope should be rotated lateral.
6. The needle is advanced anteriorly to walk off that body (the tip thereby remaining close to the vertebra) until its anterolateral border is reached.
7. Injection of contrast solution at this point should demonstrate linear spread in a cephalocaudal direction.
8. This technique can either be performed at three individual levels (L2, L3, and L4; L3 with 5 mL of 0.25% bupivacaine at each level) or simply at L3 (with 15 mL of 0.25% bupivacaine).

To determine efficacy, temperature probes should be placed on both extremities prior to pro-

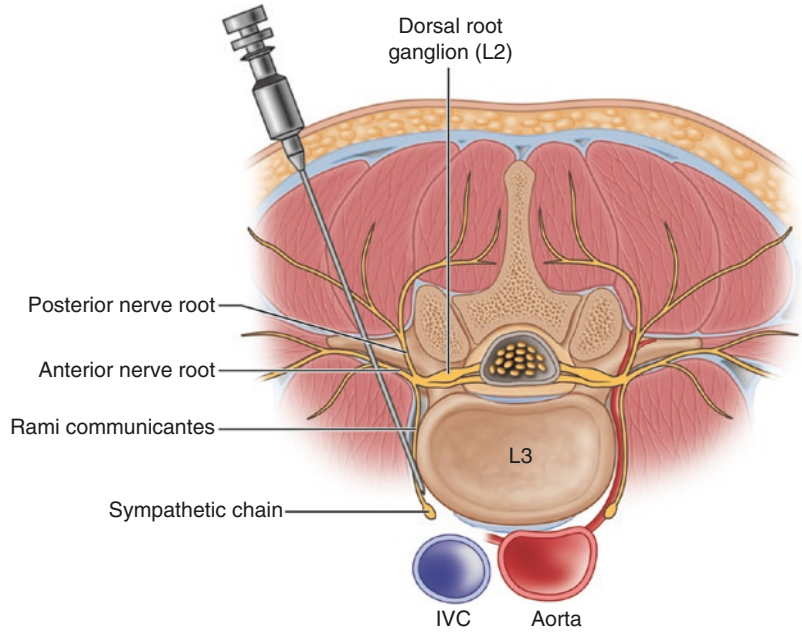

Fig. 27.2 Lumbar sympathetic block, axial diagram

ceeding. After a successful block, an approximate increase in temperature of 3 °C should be noted on the blocked side. It has been demonstrated that the distal lower extremity ipsilateral to the LSB had the greatest magnitude (8.7 °C ± 0.8 °C) and rate (1.1 °C ± 0.2 °C/min) of temperature change. It has been also shown that the great toe temperature was within 3 °C of core temperature within 35 min after LSB. The patient should initiate physical therapy at this time to perform range of motion and strengthening exercises since the pain involved in performing such exercises should be decreased after a successful block has been performed (Fig. 27.2).

Superior Hypogastric Plexus Block

Introduction

The superior hypogastric block is useful for the treatment of pelvic pain, which is either nonmalignant or malignant in nature. The superior hypogastric plexus is situated in the retroperitoneum, bilaterally extending from the lower third of the fifth lumbar vertebral body to the upper third of the first sacral vertebral body. Therefore, the target for needle insertion is anterior to the body of the L5 vertebra at the L5–S1 junction. The plexus contains both postganglionic sympathetic fibers and afferent pain fibers. The plexus gives rise to the innervation of the rectum, bladder, perineum, vulva, vagina, prostate, and uterus. Pain originating from any of these pelvic structures could theoretically be treated by blocking this plexus. It has been demonstrated that this block is effective in reducing pain scores in 70% of patients with pelvic pain associated with cancer; the majority of which in their particular study had cervical cancer.

Block Technique

1. The block can be performed under CT or fluoroscopic guidance, with the goal being to place the tip of the needles anterior to the L5–S1 junction. The technical difficulty of this block arises from the fact that the iliac crest oftentimes blocks adequate access to needle advancement in a purely oblique view, thereby necessitating a fairly lateral and slightly cephalad approach.
2. Once the needles are in position bilaterally, a lateral view should reveal appropriate contrast spread in a smooth posterior contour corresponding to the anterior psoas fascia [4].

3. This is subsequently followed by injection of 5–10 mL of local anesthetic (e.g., 0.25% bupivacaine) via both needles for the prognostic block and an equal volume of neurolytic agent (either alcohol or phenol) for the subsequent therapeutic block.

Conclusion

Various regional modalities exist to treat pain associated with chronic painful conditions including sympathetic-mediated pain, cancer pain, and nonmalignant visceral pain. A firm understanding of the autonomic nervous system, spinal anatomy, and radiographic visualization is required to perform these blocks successfully.

Clinical Pearls

- The autonomic nervous system is primarily responsible for a variety of homeostatic mechanisms in the body; these functions are important in maintaining organ perfusion, function, and metabolism.
- It is the sympathetic nervous system that is of interest to the pain specialist, as it is not only important for homeostatic function of the body but also acts as a conduit for afferent nociceptive impulses from the periphery and major organs.
- CRPS I (formerly known as reflex sympathetic dystrophy) describes a variety of painful conditions following an insult to an extremity that appears in a regional distribution with a distal predominance of abnormal findings; CRPS II (formerly known as causalgia) may potentially develop after a peripheral nerve injury.
- In patients with CRPS (types I and II), blockade of the sympathetic system (stellate ganglion, lumbar sympathetic chain) can provide profound pain relief when combined with other complimentary methods.
- The generation of visceral and cancer pain involves a complex interplay of local organ and tissue manifestations, the somatic nervous system, and the sympathetic nervous system.
- Neuropathy may be associated with numerous symptoms throughout the body including motor loss, sensory changes, and autonomic changes.
- Herpes zoster is a disease characterized by a transient rash in a dermatomal distribution that is usually painful. The term postherpetic neuralgia (PHN) is used if the pain persists after the rash has resolved. Older individuals and immunocompromised individuals are at significant risk for reactivation of herpes zoster and the subsequent development of PHN.
- Phantom limb pain is the phenomenon of experiencing pain in an extremity after that extremity has been removed, either primarily (as in trauma) or secondarily (as in surgical amputations). Patients report experiencing a wide range of pain characteristics including burning, cramping, tingling, and feelings of electrical shocks.
- The stellate ganglion block has been used successfully to treat a variety of sympathetically maintained syndromes of the upper extremity. It has long been one of the cornerstones of therapy (along with physical therapy and neuropathic medications) for complex regional pain syndrome (types I and II) of the upper extremity.
- Local anesthetic and neurolytic blockade of the celiac plexus have been used for both malignant and chronic abdominal visceral pain. Blocks with variable success rates have been used for management of acute or chronic inflammatory pain (i.e., pancreatitis).
- Lumbar sympathetic block (LSB) is a modality available to aid in the inhibition of sympathetically mediated pain in the lower extremities (just as stellate ganglion block is used for such pain in the upper extremities). LSB can be used to both help in the diagnosis and treatment of sympathetically maintained pain in conditions such as CRPS type I and type II, as well as phantom limb pain.
- The superior hypogastric block is useful for the treatment of pelvic pain, which is either nonmalignant or malignant in nature. The superior hypogastric plexus gives rise to the innervation of the rectum, bladder, perineum, vulva, vagina, prostate, and uterus.

Acknowledgment The authors wish to thank Dr. Ruben Koshy for his contribution to this chapter in the prior edition.

Review Questions

1. Celiac plexus block does not alleviate pain originating in the following viscera:
(a) Liver
(b) Pancreas
(c) Uterus
(d) Ascending colon

2. Which of the following statements is correct?:
(a) Alcohol has a delayed onset compared to phenol.
(b) Alcohol is more prone to cause vasospasm.
(c) Alcohol is isobaric compared to CSF.
(d) Nerve regeneration with the use of alcohol is faster when compared to the one with phenol.

3. Regarding phantom limb pain:
(a) It is a nociceptive type of pain.
(b) A-beta fibers are involved.
(c) Spontaneous dysesthesias are absent.
(d) Allodynia is absent.

Answers
1. c
2. b
3. b

References

1. "Peripheral Neuropathy Fact Sheet," NINDS. NIH Publication No. 04-4853.
2. Obermann M. Treatment options in trigeminal neuralgia. Ther Adv Neurol Disord. 2010;3(2):107–15.
3. Linskey ME, Ratanatharathorn V, Peñagaricano J. A prospective cohort study of microvascular decompression and gamma knife surgery in patients with trigeminal neuralgia. J Neurosurg. 2008;109(Suppl):160–72.
4. Bogduk N, Govind J. Cervicogenic headache: an assessment of the evidence on clinical diagnosis, invasive tests, and treatment. Lancet Neurol. 2009;8:959–68.
5. Christo PJ, Hobelmann G, Maine DN. Drugs post-herpetic neuralgia in older adults: evidence-based approaches to clinical management. Aging. 2007;24(1):1–19. Review
6. Benzon HT, et al. Evidence-based case report: the prevention and management of postherpetic neuralgia with emphasis on interventional procedures. Reg Anesth Pain Med. 2009;34(5):514–21. Review
7. Makharita MY, Amr YM, El-Bayoumy Y. Effect of early stellate ganglion blockade for facial pain from acute herpes zoster and incidence of postherpetic neuralgia. Pain Physician. 2012;15(6):467–74.
8. Weinstein SM. Phantom limb pain and related disorders. Neuropathic Pain Syndr. 1998;16:919–35.
9. Carlen PL, Wall PD, Nadvorna H, et al. Phantom limbs and related phenomena in recent traumatic amputations. Neurology. 1978;28:211–7.
10. Nikolajsen L, Ilkjaer S, Kroner K, et al. The influence of preamputation pain on postamputation stump and phantom pain. Pain. 1997;72:393–405.
11. Weeks SR, et al. Phantom limb pain; theories and therapies. Neurologist. 2010;16:277–86.
12. Lewin-Kowalik J, et al. Prevention and management of painful neuroma. Neurol Med Chir. 2006;46(2):62–8.
13. Narouze S. Ultrasound-guided stellate ganglion block; safety and efficacy. Curr Pain Headache Rep. 2014;18(6):424.

28 Regional Anesthesia Considerations for Chronic Noncancer Pain

Lauren K. Eng, Lindsey K. Okada, and Matthew R. Eng

Definition

Chronic noncancer pain is commonly defined as chronic pain lasting more than 3 months that is not directly attributable to cancer. Common examples of chronic noncancer pain include chronic low back pain, arthritis, headache/migraine, fibromyalgia, sickle cell pain, and peripheral neuropathy. The source of the chronic pain can be categorized as musculoskeletal, headache-related, neuropathic, or inflammatory. The origin of chronic noncancer pain is complex, with pathophysiologic, cognitive, social, and psychological components. Many of these patients have pain which is out of proportion to the pathophysiology and/or pain with no known pathophysiologic origin.

A biopsychosocial approach to the management of patients with chronic noncancer pain is ideal since many of these patients cannot be managed with pharmacologic or interventional treatment alone. In this approach, the pain must be addressed in consideration of psychological, biological, and social conditions [1]. The biopsychosocial approach was first described as an alternative view of practicing medicine, but has since been suggested as a model for approaching chronic pain [2]. In this model, the treating physicians take into consideration a patient's life circumstances. Efforts must be focused on understanding a patient's community, relationships, and psychological state in order to provide a multidimensional treatment. A multidisciplinary team consisting of a pain management physician, physical therapist, occupational therapist, and psychologist work together to treat the needs of such a patient.

Management Strategies

The perioperative management of a patient with chronic noncancer pain should also be multimodal. Such an approach is the utilization of multiple analgesic techniques and drug regimens simultaneously, resulting in a synergistic effect greater than any single agent or technique alone [3, 4]. A patient with chronic noncancer pain often presents underlying psychologic and social components to their pain and participates in ongoing counseling, behavioral modification therapy, pharmacologic therapies, and procedural intervention therapies. Thus it is of key importance for the anesthesiologist to be aware of any ongoing treatments when devising an anesthetic plan.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_28

Opioid Analgesic Therapy

With recent legislation and regulatory changes by healthcare organizations such as the Joint Commission on Accreditation of Healthcare Organizations (JCAHO), hospitals are now required to assess pain as a vital sign. These efforts have perhaps led to unintended consequences, including the oversedation of patients and adverse side effects of opioid medications [5]. Furthermore, the increase in assessment parameters has not been correlated with an improved quality of pain management [6]. Additional adverse effects of high-dose opioid therapy in these patients include constipation, nausea and vomiting, pruritus, sedation, and respiratory depression. In patients on chronic dosages of opioids, the challenge of addressing their pain without causing unwanted side effects can be complex.

Postoperative pain management in chronic noncancer pain is predictably greater than patients without chronic noncancer pain. Predictors of postoperative pain include age, gender, type of surgery, anxiety, and preoperative pain [7, 8]. In addition to pre-existing chronic pain, the impact of chronic opioid medications on postoperative pain and perioperative opioid requirements is significant. In a retrospective analysis of patients with chronic cancer and noncancer pain, the patients required a threefold increased postoperative opioid regimen to achieve pain control [9]. The increasing dosage required to achieve the same pain control in patients is well known to be opioid tolerance. More recently, however, it has been suggested that the acute and chronic exposure to opioids may induce opioid-induced hyperalgesia (OIH). Paradoxically, OIH may contribute to escalating opioid requirements, as it may cause increased sensitivity to pain as a result of consuming opioids [10].

Perioperative Management

In managing patients with chronic pain, careful planning and optimization is necessary in the preoperative, intraoperative, and postoperative management. Preoperatively, all chronic opioid medications should be converted to intravenous equianalgesic dosages to be used during the perioperative period. It should also be noted that in patients with a psychologic component, the reporting of home opioid consumption may be unreliable. Therefore, the history should be cross-referenced with any available documentation when available. Non-opioid analgesic adjuvants should be considered, and regional techniques should be considered when appropriate. The provider should also assess and address anxiety about the operation as well as discuss the plan for pain management with the patient. Intraoperatively, it is critical that the patient's home dosage of opioid medication is continued to prevent withdrawal. Non-opioid analgesic adjuvants and wound infiltration techniques should be employed as a multimodal strategy. In the postanesthesia care unit, the patient's pain must be carefully managed while guarding against oversedation and withdrawal. The patient should be closely observed with respiratory monitors, pulse oximetry, blood pressure monitors, and electrocardiogram monitoring.

Opioid-Sparing Techniques

Adjuvants are important to reducing the dose escalation of opioids and intolerable side effects of high-dose opioids. Nonsteroidal anti-inflammatory drugs (NSAIDs), cyclooxygenase-2 (COX-2) inhibitors, NMDA antagonists, anticonvulsants, and other non-opioid adjuvants have been effective in delivering pain relief while reducing opioid requirements. Other strategies in reducing opioid requirements include regional anesthesia nerve blocks and local anesthesia infiltration.

NSAIDs, COX-2 Inhibitors, and Acetaminophen

Perioperative administration of nonsteroidal anti-inflammatory drugs, ibuprofen, cyclooxygenase-2 inhibitors, and acetaminophen has

demonstrated decrease in opioid requirements and overall opioid-related side effects. Ketorolac, an NSAID, has profound analgesic qualities that offers comparable pain relief to morphine following limb surgery [11]. Due to the nonselective cyclooxygenase-inhibiting properties of NSAIDs, there has been concern regarding gastrointestinal bleeding, kidney injury, and platelet impairment. COX-2 inhibition is the therapeutic pathway of analgesia for NSAIDs. COX-2 inhibitors have become suitable analgesic alternatives where NSAIDs may be contraindicated [12]. Celecoxib, a COX-2 inhibitor, and ibuprofen have both demonstrated significant surgical pain relief while reducing opioid requirements, improving recovery times, and reducing opioid side effects [13]. Another adjuvant, intravenous acetaminophen, was approved by the US FDA in 2010. The adjuvant has potent analgesic properties with an established safety and tolerability profile. When administered either at the beginning of surgery or prior to PACU transfer, intravenous acetaminophen decreased the incidence of opioid-related side effects, namely, postoperative nausea and vomiting [14].

NMDA Receptor Antagonists

Ketamine is a noncompetitive NMDA receptor antagonist that was first used for induction and maintenance of anesthesia. When administered in subhypnotic dosages, it may prevent central mediated sensation of pain through receptors located on the dorsal horn. The well-known hypnotic side effects of ketamine may be avoided by using subhypnotic doses (<150 ug/kg). In these dosages, ketamine may be used as an excellent analgesic adjuvant. When administered in knee arthroscopy operations, morphine requirements were decreased, and passive knee mobilization was improved at 24 h [15]. Perhaps even more important in chronic noncancer patients on chronic opioids, subhypnotic doses of ketamine have been demonstrated to attenuate opioid-induced acute tolerance [16].

Anticonvulsants

Gabapentinoids, gabapentin, and pregabalin are also useful non-opioid analgesic adjuvants that have demonstrated opioid-sparing benefits. While associated with a favorable tolerability and safety profile, some patients may experience dose-related sedation with this class of medications.

Regional Anesthesia Techniques

Regional anesthesia techniques can potentially offer the silver bullet solution to chronic noncancer pain patients who are consuming chronic opioids. While there is no strong evidence favoring regional anesthesia techniques over general anesthesia techniques, a regional block may ideally eliminate the need for any increase in opioid consumption in patients who may have intensified pain in response to surgical insult. Regional techniques include neuraxial blockade or catheters, peripheral nerve blockade or catheters, wound infiltration, intra-articular injection, and fascial plane infiltration. There is still a lot of work to be done in demonstrating the benefits of regional anesthetic techniques in patients consuming chronic opioids; however, the benefits of opioid-sparing analgesics can be inferred by utilizing regional anesthetic techniques. Any chronic opioid consumption must be converted to equianalgesic equivalents and administered throughout the perioperative period irrespective of anesthetic technique.

Wound Infiltration

Whenever peripheral nerve block or neuraxial techniques are not applicable, local anesthetics should be infiltrated into the surgical wound site. In a study of 180 gynecologic patients, patients who received bupivacaine 0.5% 20 mL as a local wound infiltration consumed 17 times less morphine over the first 24 h than patients who received an infiltration of saline placebo. Over the past few years, liposomal bupivacaine has

been developed and approved for wound infiltration. Liposomal bupivacaine utilizes a novel drug delivery system that encapsulates the local anesthetic with a honeycomb network of lipid-based particles. The pharmacokinetics of liposomal bupivacaine indicates active concentrations of bupivacaine for up to 96 hours. In total hip replacement operations, liposomal bupivacaine was compared to the routine wound infiltration using bupivacaine and ketorolac. Patients in the routine infiltration group required 2.64 times more doses of morphine than patients in the liposomal bupivacaine infiltration group [17]. Improvement in wound infiltration technique and types of local anesthetics will improve outcome of patients with chronic noncancer pain by reducing their opioid requirements postoperatively.

Upper Extremity Peripheral Nerve Blocks

Patients with chronic noncancer pain undergoing upper extremity or shoulder operations may be ideal candidates for an upper extremity nerve block or catheter. The brachial plexus may be blocked at the interscalene, supraclavicular, infraclavicular, or axillary level depending on the type of operation and the anatomical considerations of the patient. Single-shot upper extremity nerve blocks can be very effective in providing surgical analgesia and postoperative analgesia for up to 24–30 h. In a patient with chronic noncancer pain, intensified postoperative pain may be anticipated, and a peripheral nerve catheter may provide extended analgesia beyond 24–30 h. In patients undergoing shoulder surgery, an interscalene peripheral nerve catheter was demonstrated to be superior to patient-controlled analgesia with respect to pain control and opioid-related side effects.

Lower Extremity Peripheral Nerve Blocks

Lower extremity peripheral nerve blocks can provide partial to complete surgical analgesia for patients undergoing lower extremity surgery. Interventions include femoral, saphenous, sciatic, fascia iliaca, and lumbar plexus nerve blocks. The goals of postoperative recovery must be addressed in orthopedic surgery procedures where early ambulation is encouraged. In knee replacements, regional techniques that avoid interfering with any motor function have been advocated to improve surgical outcome [18]. However, in patients with significant chronic noncancer pain and high chronic opioid consumption, a multidisciplinary plan must be established which accounts for the patient's pain control. As with upper extremity operations, patients with chronic noncancer pain may benefit from the prolonged duration of a peripheral nerve catheter that can infuse local anesthetics for an extended period of time.

Truncal Blocks

With the proliferation of ultrasound-based nerve blocks, there have been some new techniques over the past few years in truncal blocks. The truncal blocks are numerous: paravertebral, transversus abdominis plane, rectus sheath, ilioinguinal/iliohypogastric, quadratus lumborum, and transversalis fascia block. The most commonly performed block is the transversus abdominis plane block, indicated for postoperative pain management of abdominal incisional pain. In a meta-analysis of 641 patients, morphine consumption was reduced by 9.1 mg in the first 24 h and 5 mg in the second 24 h, and opioid-related side effects were also reduced [19]. Although truncal blocks are only adjuvants in reducing pain, they are opioid sparing and can be beneficial to patients with chronic noncancer pain.

Neuraxial Anesthesia

Chronic noncancer pain patients on chronic opioids have been shown to respond to epidural opioids very differently than opioid naive patients. In a study of 116 patients with chronic cancer receiving epidural infusions, the patients who were chronically consuming over 50 mg morphine daily for

3 months required triple the dose of epidural morphine for three times the duration of the opioid naive patients [20]. This suggests that there is some cross-tolerance that exists in opioids that are consumed by the epidural route. A further study by the same research group demonstrated that chronic opioid-tolerant patients who did not respond to epidural morphine responded to epidural sufentanil with greater pain relief [21]. A hydrophilic opioid, morphine, is not absorbed as quickly as a lipophilic opioid, sufentanil, suggesting that potent lipophilic opioids are more appropriate to use in patients with chronic noncancer pain.

Conclusions

Patients with chronic noncancer pain can be challenging with psychological, social, and biological sources of pain and are often consuming high chronic doses of opioids. Accounting for the analgesic needs perioperatively can be challenging. Preventing withdrawal, preventing overdose, and avoiding an escalation of opioid consumption should be the goals of the regional anesthesiologist. Utilizing non-opioid analgesics and regional anesthetic techniques are effective anesthetic plans when patients present with these challenging medical conditions.

Case Study

A 57-year-old man with a history of hypertension, diabetes, depression, and chronic low back pain presents for a sigmoid colectomy for diverticular disease. He reports that he takes lisinopril for his blood pressure. He is taking lantus insulin for his diabetes and reports that his most recent HgbA1C is 5.5. He takes fluoxetine and alprazolam for his anxiety. For his low back pain, he takes oxycontin 30 mg TID for the past 2 years, and for diabetic neuropathy, he takes gabapentin 300 mg TID for the past 12 months. He denies smoking cigarettes, but drinks 2–3 shots of whiskey per night. The patient is 300 pounds, 5′10″. He is well mannered, but extremely nervous about his operation.

Review Questions

1. The patient's pain is best explained by which of the following:
   (a) Degenerative disc disease
   (b) Work-related stress
   (c) Diabetic neuropathy
   (d) Social isolation
   (e) All of the above
2. Which of the following is the most important therapy in the analgesic plan for this patient?
   (a) Performance of bilateral transversus abdominis plane blocks prior to PACU transfer
   (b) Administration of IV Acetaminophen 1000 mg prior to PACU transfer
   (c) Administration of equianalgesic dosage of home oxycontin
   (d) Wound infiltration of liposomal bupivacaine by surgeon
   (e) Administration of intraoperative subhypnotic dose of ketamine
3. Assuming the surgeon has requested an epidural for postoperative pain management and you were able to place the epidural successfully, which of the following infusions would be most appropriate for this patient?
   (a) Bupivacaine + morphine
   (b) Ropivacaine + morphine
   (c) Bupivacaine + hydromorphone
   (d) Ropivacaine + sufentanil
   (e) Bupivacaine + diamorphine

Answers:

1. e. All of the above

This patient's pain has biopsychosocial causes. In a patient with chronic pain, the pain may have biomedical causes (degenerative disc disease, diabetic neuropathy), psychologic causes (work-related stress), and social causes (social isolation).

2. c. Administration of equianalgesic dosage of home oxycontin

This patient is on a high dosage of chronic opioid medication. The patient is at high risk for withdrawal, and this is the most critical issue for

this patient. Analgesic adjuvants, regional techniques, and wound infiltration are important in reducing the patient's opioid requirements perioperatively. However, the most important therapy is to prevent opioid withdrawal.

3. d. Ropivacaine + sufentanil

In a patient who has been taking chronic opioids, neuraxial lipophilic opioids are absorbed systemically better than lipophobic opioids. Morphine and hydromorphone are considered to be lipophobic opioids, while sufentanil, fentanyl, and alfentanil are considered to be lipophilic opioids.

References

1. Feinberg SD. The pain medicine specialist as a physician-healer. Clin J Pain. 1996;12(1):3–5.
2. Engel GL. The clinical application of the biopsychosocial model. Am J Psychiatry. 1980;137(5):535–44.
3. Kehlet H. Multimodal approach to control postoperative pathophysiology and rehabilitation. Br J Anaesth. 1997;78(5):606–17.
4. Kehlet H, Dahl JB. The value of "multimodal" or "balanced analgesia" in postoperative pain treatment. Anesth Analg. 1993;77(5):1048–56.
5. Mularski RA, White-Chu F, Overbay D, Miller L, Asch SM, Ganzini L. Measuring pain as the 5th vital sign does not improve quality of pain management. J Gen Intern Med. 2006;21(6):607–12.
6. Vila H Jr, Smith RA, Augustyniak MJ, Nagi PA, Soto RG, Ross TW, et al. The efficacy and safety of pain management before and after implementation of hospital-wide pain management standards: is patient safety compromised by treatment based solely on numerical pain ratings? Anesth Analg. 2005;101(2):474–80.
7. Kalkman CJ, Visser K, Moen J, Bonsel GJ, Grobbee DE, Moons KGM. Preoperative prediction of severe postoperative pain. Pain. 2003;105(3):415–23.
8. Ip HYV, Abrishami A, Peng PWH, Wong J, Chung F. Predictors of postoperative pain and analgesic consumption: a qualitative systematic review. Anesthesiology. 2009;111(3):657–77.
9. Rapp SE, Ready LB, Nessly ML. Acute pain management in patients with prior opioid consumption: a case-controlled retrospective review. Pain. 1995;61(2):195–201.
10. Angst MS, Clark JD. Opioid-induced hyperalgesia: a qualitative systematic review. Anesthesiology. 2006;104(3):570–87.
11. Rainer TH, Jacobs P, Ng YC, Cheung NK, Tam M, Lam PK, et al. Cost effectiveness analysis of intravenous ketorolac and morphine for treating pain after limb injury: double blind randomised controlled trial. BMJ. 2000;321(7271):1247–51.
12. Jackson LM, Hawkey CJ. COX-2 selective nonsteroidal anti-inflammatory drugs: do they really offer any advantages? Drugs. 2000;59(6):1207–16.
13. White PF, Tang J, Wender RH, Zhao M, Time M, Zaentz A, et al. The effects of oral ibuprofen and celecoxib in preventing pain, improving recovery outcomes and patient satisfaction after ambulatory surgery. Anesth Analg. 2011;112(2):323–9.
14. Apfel CC, Turan A, Souza K, Pergolizzi J, Hornuss C. Intravenous acetaminophen reduces postoperative nausea and vomiting: a systematic review and meta-analysis. Pain. 2013;154(5):677–89.
15. Menigaux C, Fletcher D, Dupont X, Guignard B, Guirimand F, Chauvin M. The benefits of intraoperative small-dose ketamine on postoperative pain after anterior cruciate ligament repair. Anesth Analg. 2000;90(1):129–35.
16. Kissin I, Bright CA, Bradley EL Jr. The effect of ketamine on opioid-induced acute tolerance: can it explain reduction of opioid consumption with ketamine-opioid analgesic combinations? Anesth Analg. 2000;91(6):1483–8.
17. Emerson RH, Barrington JW, Olugbode O, Lovald S, Watson H, Ong K. Comparison of local infiltration analgesia to bupivacaine wound infiltration as part of a multimodal pain program in Total hip replacement. J Surg Orthop Adv. 2015;24(4):235–41.
18. Perlas A, Kirkham KR, Billing R, Tse C, Brull R, Gandhi R, et al. The impact of analgesic modality on early ambulation following total knee arthroplasty. Reg Anesth Pain Med. 2013;38(4):334–9.
19. Abdallah FW, Laffey JG, Halpern SH, Brull R. Duration of analgesic effectiveness after the posterior and lateral transversus abdominis plane block techniques for transverse lower abdominal incisions: a meta-analysis. Br J Anaesth. 2013;111(5):721–35.
20. de Leon-Casasola OA, Myers DP, Donaparthi S, Bacon DR, Peppriell J, Rempel J, et al. A comparison of postoperative epidural analgesia between patients with chronic cancer taking high doses of oral opioids versus opioid-naive patients. Anesth Analg. 1993;76(2):302–7.
21. de Leon-Casasola OA, Lema MJ. Epidural bupivacaine/sufentanil therapy for postoperative pain control in patients tolerant to opioid and unresponsive to epidural bupivacaine/morphine. Anesthesiology. 1994;80(2):303–9.

Malignant Pain

29

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Introduction

Malignant pain is a common and frequently debilitating experience shared by many individuals diagnosed with a neoplastic disease. It is reported by about 90% of cancer patients during different stages of their disease trajectory [1]. Moderate to severe pain is present in approximately 80% of individuals with an advanced neoplastic disease [2]. Pain syndromes are broken down into those arising from a direct effect of a neoplasm on nearby tissues and structures (85%), side effect of a treatment (17%), pain due to disease progression (9%), and pain from other causes not related to malignancy [3]. It is broadly categorized as nociceptive somatic (71.6%), nociceptive visceral (34.7%), and neuropathic (39.7%) [1].

The World Health Organization (WHO) analgesic stepladder has guided the treatment of cancer-related pain since its inception in 1986 by Italian physician and researcher, Dr. Vittorio Ventafridda [4]. The original algorithm included oral opioids of increasing potency and adjuvant pharmacological agents for the management of malignant pain of different severity. Opioid medications, which are the cornerstone of cancer-related pain management, provide satisfactory pain control in many cancer patients. However, an inadequate analgesia is still reported by 50% of cancer patients [5]. The pain-reducing benefits should be weighed against potentially deleterious side effects and complications in this fragile population. Safe opioid regimen at minimal possible doses which might be a goal of every prescriber is unfortunately difficult to attain. A gap between the harmful and effective opioid-dosing regimen narrows at the advanced stages of the disease. Other treatment modalities are needed to alleviate the pain, such as adjunctive non-opioid medications or interventional pain management techniques. Opinions regarding the management of cancer-related pain continue to change. As a result, some propose adopting a modified WHO analgesic construct, which includes the interventional pain management strategies as the fourth step of the stepladder (Fig. 29.1) [6]. Typically, the interventional techniques were reserved for patients with intense pain after pharmacological options have either failed or are sufficient to bring the pain under control. While this holds true for selected interventional pain strategies, it might be advisable to utilize peripheral nerve block techniques before the initiation or in conjunction with oral or parenteral medications to

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_29

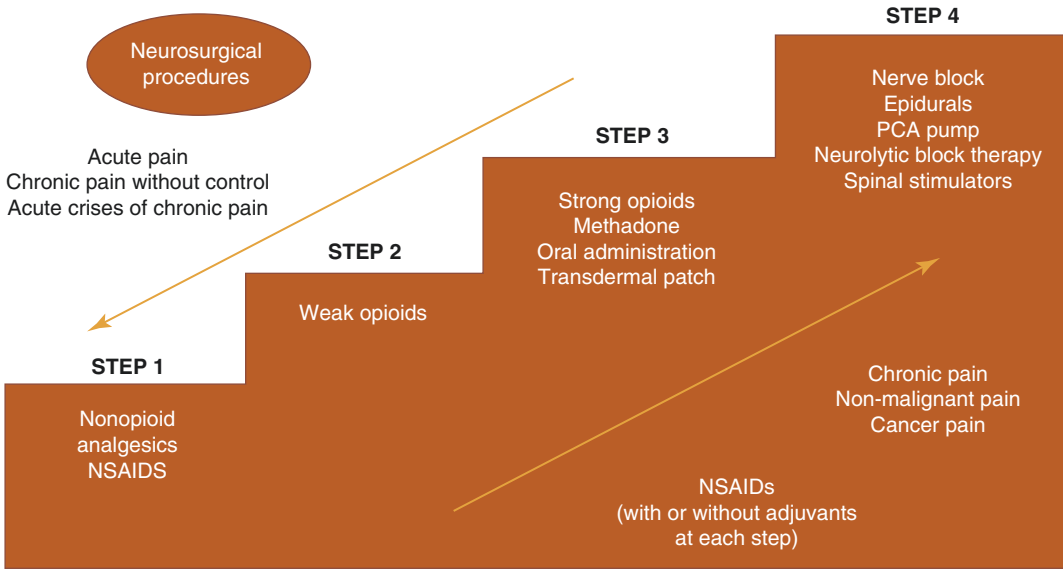


Fig. 29.1. New adaptation of the analgesic ladder. Reproduced with permission from Vargas-Schaffer G. Is the WHO analgesic ladder still valid? Twenty-four years of experience. Canadian Family Physician. Le Médecin de famille canadien. Vol 56: June 2010

limit the opioid usage, reduce the risk of opioid-related side effects, and maximize the pain control. This approach might be especially beneficial for the surgical oncologic patients in the perioperative and postsurgical period.

Peripheral Nerve Blocks

Peripheral nerve blocks (PNBs) are utilized to provide palliation for acute pain in the immediate postsurgical period or to relieve persistent oncologic pain in instances when conventional oral analgesics fail to provide appreciable pain relief or when such treatment is complicated by intolerable side effects. A systematic review conducted by the European Association for Palliative Care (EAPC) to assess the efficacy of major peripheral nerve blocks in adult cancer patients, which included 16 publications and 79 cases, showed a successful use of PNBs (intercostal, brachial plexus, paravertebral, and others) in controlling intractable cancer-related pain [7]. The authors concluded that PNBs might be underutilized despite their potential to provide good pain control while lowering the doses of oral analgesics and minimizing medication-induced side effects. Opioid medications, which are currently used as first-line agents for postsurgical pain, are known to suppress the immune system in vulnerable individuals [8]. Therefore, PNBs might be a valuable modality in alleviating postsurgical pain while supporting immune defenses to aid recovery and prevent relapse of the disease. This section discusses most commonly used peripheral nerve blocks; possible complications are listed in Table 29.1. A long-term pain relief can be achieved in selected patients with radiofrequency ablation (RFA) techniques after a positive response to peripheral nerve blocks with local anesthetic.

Intercostal nerve block (ICNB) is used to diagnose and treat painful entities involving the anterior chest wall and upper abdominal area innervated by the intercostal nerves. It can be helpful in treating acute pain after thoracic or upper abdominal surgeries and long-standing painful intercostal neuropathy, post mastectomy, and post-thoracotomy pain. A medication is deposited into the intercostal space about

Table 29.1. Summary of potential complications associated with peripheral nerve blocks

Summary of complications of selected peripheral nerve blocks		
Intercostal nerve block	Paravertebral nerve block	Brachial plexus block
Pneumothorax	Vascular puncture	Hematoma formation
	Hypotension	Infection
Infection	Epidural injection	Anesthetic toxicity
	Intrathecal injection	Brachial plexus injury
Local anesthetic toxicity	Pleural puncture	Phrenic nerve injury[a]
	Pneumothorax	Hemidiaphragmatic paresis[a]
Spinal anesthesia	Harlequin syndrome	Recurrent laryngeal nerve injury[a]
	Horner's syndrome	
Visceral injury	Infection	Horner's syndrome[a]
		Pneumothorax[a]
		Subcutaneous emphysema[a]
		Spinal block[a]
		Intravascular injection[b]

Data from [25–29]
[a]Possible with the techniques above the clavicle; interscalene and supraclavicular
[b]Most likely with the axillary technique

6–10 cm from the midline at the inferior rib margin. Due to a potential risk of pneumothorax, the needle should be advanced no deeper than 2–3 mm from the rib margin [9]. A recent randomized controlled trial compared the level of postoperative analgesia with ICNB plus intravenous patient-controlled morphine versus patient-controlled morphine in 60 patients undergoing video-assisted thoracoscopic surgery for mediastinal lymph node biopsy [10]. The treatment group received superior analgesia and required less morphine compared to the control group in the first 6 h after the surgery. Retrospective study by Wong et al. (n = 25) shows appreciable pain relief in 80% and reduction of opioids in 56% oncological patients [11]. The pain relief lasted between 5 and 158 days, while 32% of patients remain pain-free until the end of their lives. The technique has also been applied to control pain after esophageal surgery in 80 patients with esophageal cancer [12]. A positive response to local anesthetic might be followed by chemical neurolysis using phenol or alcohol for a more sustained pain control. Matchett describes successful use of diagnostic ICNB followed by chemical neurolysis with phenol in 11 patients with intractable cancer-related chest wall pain [13]. Ultrasound guidance may improve visualization of pulmonary structures while minimizing pneumothorax risk, but this remains hypothetical.

Paravertebral nerve block (PVB) is an alternative technique used to alleviate procedural or chronic pain of the chest wall and upper abdomen for the treatment of acute and chronic pain involving the chest and upper abdomen. Medication is injected into the paravertebral space, which is a wedge-shaped area lateral to the vertebral column surrounded by the parietal pleura anteriorly, the superior costotransverse ligament posteriorly, vertebral unit medially, and the distal ends of ribs superiorly and inferiorly. The paravertebral space houses thoracic spinal nerves, sympathetic chains, and rami communicantes, which can be blocked at specific dermatomal levels with single injections or continuous catheter infusions. A systematic review analyzed the evidence presented by 24 randomized trials with a total of 1,822 patients and showed that PVBs diminish the immediate postoperative pain and chance of chronic pain at 6 months post-surgery, decrease opioid consumption intra- and post-operatively, and reduce nausea and vomiting [14].

Pectoral nerve block (pecs block) might be a valuable addition to multimodal analgesia in treatment of both acute and chronic

malignancy-related chest wall pain syndromes. Local anesthetic solution is deposited between the pectoralis major and pectoralis minor muscles to block the pectoral, 3rd to 6th intercostal, intercostobrachial, and long thoracic nerves. Favorable outcomes following pecs blocks have been demonstrated in a RCT involving 120 surgical female patients undergoing radical mastectomy under general anesthesia [15]. The patients who received the pecs blocks prior to the surgery reported lower postsurgical pain scores; had lower opioid requirements intra- and postoperatively; experienced less sedation, nausea, and vomiting; and had quicker discharge rates from the PACU compared to females in the control group. Two independent RCTs compared the effectiveness of ultrasound-guided pecs blocks with paravertebral blocks (PVBs) in 60 and 40 female patients, respectively, scheduled for radical breast surgeries and arrived at similar conclusions [16, 17]. The authors of both studies showed superior analgesia and lower morphine use in the postoperative period in the pecs block group compared to the PVBs. Wahba et al. have also shown decreased requirement for fentanyl during the surgery in the pecs block group versus the PVB group [16]. No adverse events have been reported in either of the groups. Therefore, a conclusion can be made that pecs block technique might be safer and easier to perform alternative to the PVB technique. Pecs block technique has also been successfully applied to provide a substantial level of analgesia, reduce dysesthesias, and improve sleep in a pilot study involving nine female patients who have undergone breast surgeries and subsequently suffered from a debilitating chronic neuropathic pain of the anterior chest wall due to surgically severed pectoral nerves [18]. This pilot study illustrated a significant analgesia lasting for 7 days, reduced sensory disturbances, and improved sleep in nine female patients suffering from a debilitating chronic pain after a breast surgery.

Regional anesthesia has also been employed to treat intractable cancer-related neuropathic pain. *Intercostobrachial nerve block (ICBNB)* has been used to alleviate painful intercostobrachial neuralgia secondary to an axillary lymph node dissection or breast surgery [19, 20]. Upper extremity neuropathic pain secondary to neoplastic brachial plexopathy from a pulmonary or breast neoplasm can be successfully treated with single or continuous *brachial plexus block (BPB) techniques.* Multiple case reports exist of patients with various types of cancer who were treated with BPBs and derived pain relief lasting between 2 and 31 weeks [21–24].

Neurolytic Plexus Blocks

Neurolysis (rhizolysis, rhizotomy) entails a deliberate destruction of neural pathways to alleviate sympathetically mediated pain due to a neoplastic disease. Neurolytic blockade is performed by injecting phenol or alcohol solution near the sympathetic ganglia at discrete sites, which blocks the transmission of the stimuli delivered by the visceral afferent nerve fibers at a corresponding anatomic location. The technique is offered to carefully selected candidates who failed to respond to conventional treatment modalities, but who had positive prognostic blocks with local anesthetic.

A systematic review of 27 controlled studies conducted by the European Association for Palliative Care Research Network (EAPCRN) to assess the benefits of sympathetic blocks in reducing visceral cancer pain revealed improved pain control, decreased opioid requirements, and lower rates of opioid-induced side effects after celiac plexus block (CPB) and diminished pain and opioid usage but not the rates of opioid-related side effects after superior hypogastric plexus block (SHPB) [30]. Consequently, the European Association for Palliative Care (EAPC) has provided a strong recommendation for CPB and weak recommendations for SHPB in pancreatic cancer patients. Below is an overview of selected neurolytic plexus blocks. Table 29.2 includes indications for the blocks and possible complications associated with each of the blocks.

Celiac plexus block (CPB) neurolysis can be used for refractory visceral upper abdominal pain due to malignancy of the pancreas and other vis-

Table 29.2. Summary of indications and complications for neurolytic sympathetic plexus block techniques

Neurolytic sympathetic plexus blocks		
Block	Indications	Complications
Celiac plexus block	Upper abdominal pain due to malignancy of pancreas, stomach, hepatobiliary system, small intestine, spleen, ascending colon, adrenal glands	Hypotension Diarrhea Spinal cord damage
Superior hypogastric block	Pelvic pain due to malignancy of large colon, ovaries, uterus, cervix, bladder, prostate, rectum	Pain at the injection site Localized bleeding Infection Nerve injury Rectal perforation Puncture of vessels or internal organs Urinary and bowel problems Distal ischemia if iliac artery penetrated
Lumbar plexus block	Lower extremity pain Rectal pain Intractable back pain	Genitofemoral neuralgia Retrograde ejaculation Epidural, subdural, or intrathecal injection Hypotension Aortic or vena caval injection Femoral nerve neuropathy Psoas muscle injection Peritoneal puncture Renal subcapsular hematoma Thrombosis, embolization Bladder, bowel, sexual dysfunction Local anesthetic toxicity Death
Ganglion impar block	Perineal pain due to rectal, bladder, or perineal cancer	Pain at the injection site Rectal perforation Puncture of internal vessels or organs Infection Bleeding and hematoma formation Neural injury Paralysis Death

Data from [46, 47]

A common contraindication for all the blocks is a presence of coagulopathy, anticoagulant and/or antiplatelet therapy, history of allergy to iodine-based contrast agents, presence of infection at the injection site, and inability to remain stable during the procedure.

ceral organs (Fig. 29.2). The celiac plexus is located under the diaphragm at the T12 and L1 vertebral level in the retroperitoneal space. It receives sympathetic input from greater, lesser, and least splanchnic nerves and parasympathetic input from the vagus nerve. CPB can be accessed with transcrural, transaortic, retrocrural, anterior transabdominal, and transesophageal endoscopic approaches under CT or fluoroscopic guidance. No significant variations in terms of efficacy and morbidity have been reported between the different approaches [31].

The efficacy of CPB has been extensively studied and demonstrated by different peer-reviewed publications [30–38]. A double-blinded randomized controlled trial comparing the effectiveness of percutaneous CPB to opioid analgesia in 100 patients with unresectable pancreatic cancer showed 53% and 40% pain reduction in the CPB group compared to 27% and 14% in the

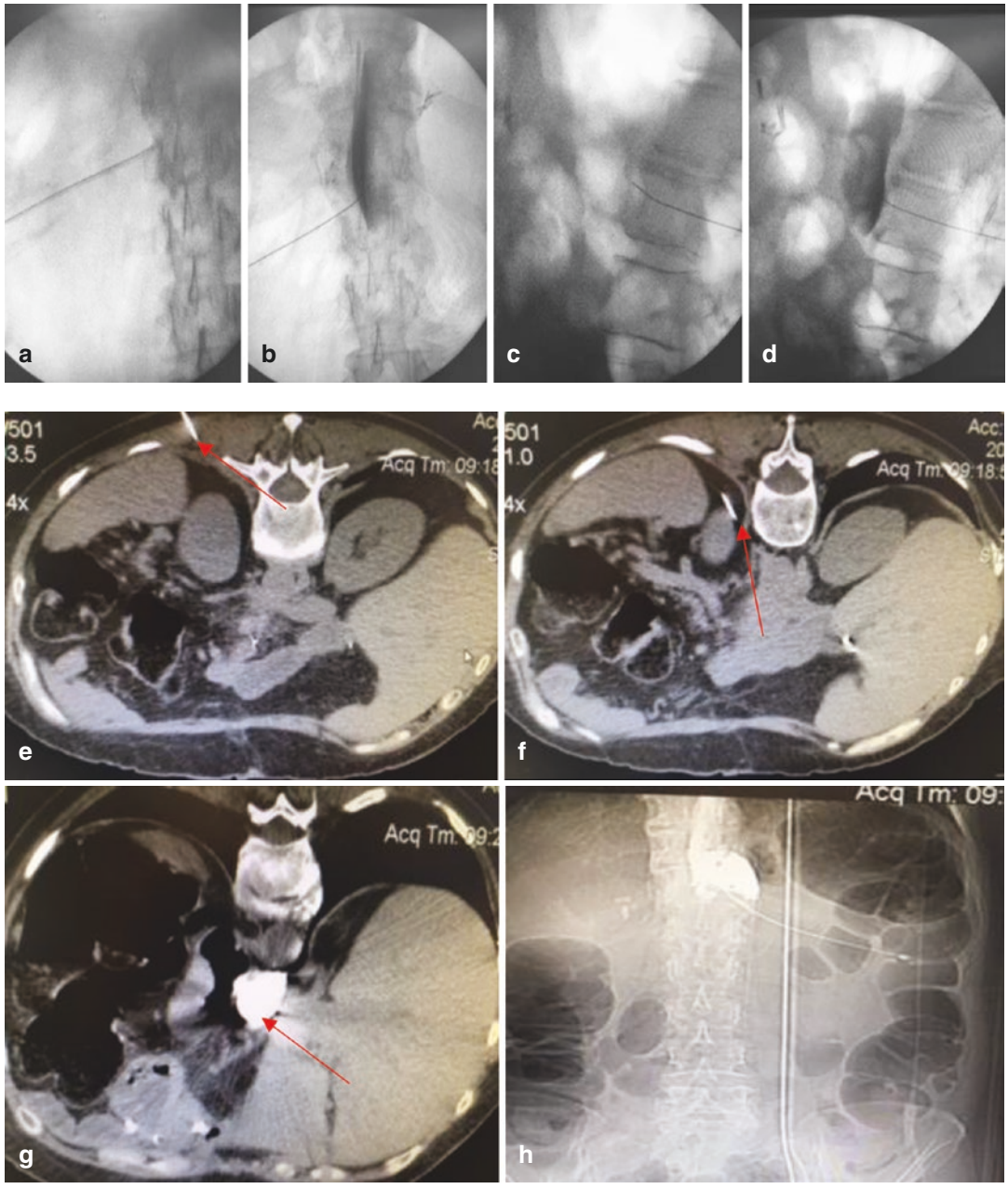


Fig. 29.2. Diagnostic celiac plexus block: (**a**) A-P view showing needle approaching spine at T12-L1 from the left side, (**b**) A-P view showing contrast proceeding in a cephalad direction from T12 to L1, (**c**) lateral view showing needle passing anterior margin of L1 vertebral body, (**d**) contrast injection under lateral view showing para-aortic spread without intravascular uptake or spread posteriorly. **Therapeutic celiac plexus block**: (**e**) arrow points to needle entering tissues posteriorly 8 cm from dorsal midline on left side, (**f**) arrow points to needle advancing past kidney and along lateral wall of L1 vertebral body, (**g**) spread of injected contrast (5 mL) hugging para-aortic gutter without extension into vascular structure or toward the spine, (**h**) A-P scout film showing transcrural spread of contrast cephalad from T12 to L1 blocking not only the celiac plexus but also the splanchnic nerve (Images courtesy of Kenneth D. Candido, MD)

control group at 1- and 6-week follow-up [34]. No significant differences have been observed in regard to opioid use, opioid-related side effects, quality of life, and survival time between the groups. Different double-blinded randomized controlled trials compared an early application of CPB to pharmacological therapy in 96 patients with an inoperable pancreatic cancer [37]. Diminished pain was noted in the treatment group at 1 and 3 months compared to increased pain intensity and morphine use in the control group at both time points. No differences in quality of life or overall survival have been observed. Reduced pain and opioid use following CPB have been supported by two large systematic reviews of relevant publications (6 studies, $n = 358$; 66 studies, $n = 295$) [31, 38] and meta-analysis of 7 RCTs ($n = 196$) [36].

Superior hypogastric plexus neurolysis (SHPB) is applied to ameliorate cancer-related visceral and sympathetic pain arising from the large colon and reproductive structures (Fig. 29.3). The superior hypogastric plexus is a bilateral paired structure situated between the L4 and S1 segments in the retroperitoneal cavity. Anterior percutaneous, posterior percutaneous, and transdiscal approaches have been described. The efficacy of the SHPB has been documented by various investigators [39–41]. Plancarte et al. examined the efficacy of the technique in 227 patients with pelvic pain and reported pain relief in 79% of patients after the diagnostic block and in 72% after neurolysis with 10% phenol [41]. Moreover, the patients treated with the neurolytic blocks decreased their opioid consumption by 43%. Improved pain control, decreased opioid utilization, and improved quality of life were demonstrated by another study of 60 cancer patients suffering from pelvic and abdominal pain [39]. A recent randomized controlled trial of 50 patients with gynecological cancer shows notable pain relief in the SHPB compared to the control group, but no statistical differences in morphine consumption or improved functioning between the two groups [40].

Lumbar sympathetic neurolysis (LSN) involves destruction of any of the four-paired lumbar sympathetic ganglia supplying innervation to the lower body (Fig. 29.4). LSN has been applied to treat neuropathic pain in the lower extremities secondary to arterial occlusive disease, vasospastic disorders, complex regional pain syndrome,

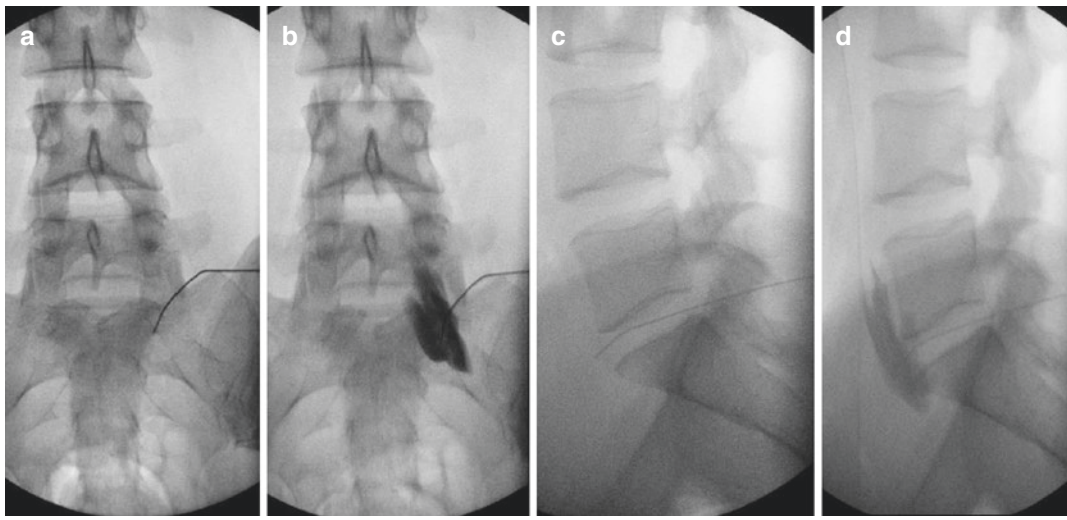


Fig. 29.3. Neurolytic superior hypogastric plexus block, right sided: (**a**) A-P view of needle, (**b**) A-P view of needle after injection of 5 ml of contrast, (**c**) lateral view of needle, (**d**) lateral view of needle after injection of 5 ml of contrast (Images courtesy of Kenneth D. Candido, MD)

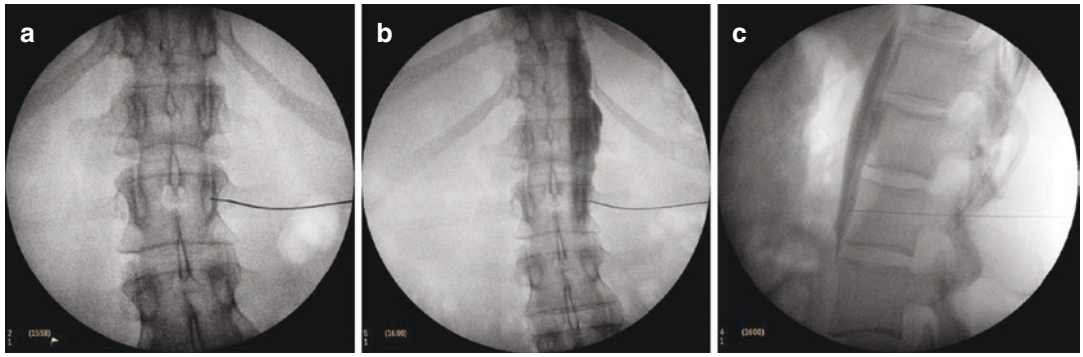


Fig. 29.4. Lumbar sympathetic plexus neurolytic block: (**a**) A-P view of needle placed anterior and lateral to the right L2 vertebral body at the level of pedicle, (**b**) A-P view demonstrating right-sided spread of contrast (5 ml) at the right L2 vertebral body injected prior to phenol neurolysis, (**c**) lateral view showing linear spread of contrast along anterior margin of multiple vertebral bodies (Images courtesy of Kenneth D. Candido, MD)

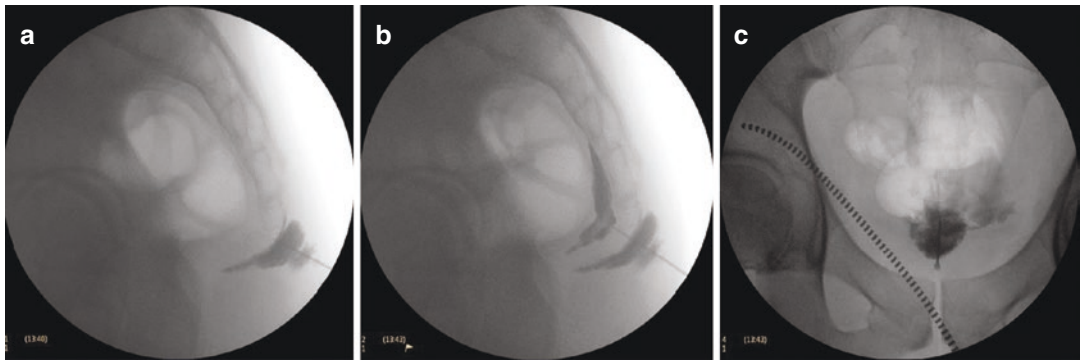


Fig. 29.5. Neurolytic ganglion impar block: (**a**) lateral view of needle at the sacrococcygeal junction and contrast injected outside of the junction, (**b**) lateral view of needle going through sacrococcygeal junction and additional contrast demarcating anterior sacral margin, (**c**) A-P view of contrast spread (Images courtesy of Kenneth D. Candido, MD)

and peripheral neuralgia [42]. It is also utilized in the setting of malignancy to abolish lower extremity pain from tumor invasion of the spinal canal or of the peripheral tissues. It has also been used with good outcomes to relieve bladder spasms in patients diagnosed with bladder cancer [43] and to treat lower extremity lymphedema in patients with gynecological cancers [44].

Ganglion impar neurolysis (GIB) is used to treat pain of the perineal or pelvic area due to neoplasms of the distal GI tract, urogenital system, external genital organs, and perineum not amenable to conventional treatment options (Fig. 29.5). The ganglion impar (the ganglion of Walther) is the end point of the sympathetic chain located in the retroperitoneum anterior to the sacrococcygeal junction and posterior to the rectum. Evidence supporting the use of this technique in cancer pain treatment is limited; however, it shows favorable outcomes and >50% pain reduction in the examined participants [45].

Neuraxial Neurolytic Blocks

Neuraxial neurolytic blocks have a long-standing history of providing an excellent pain relief to patients suffering from recalcitrant chronic pain (Fig. 29.6). The blocks are also an invaluable tool in managing cancer patients (Table 29.3). This

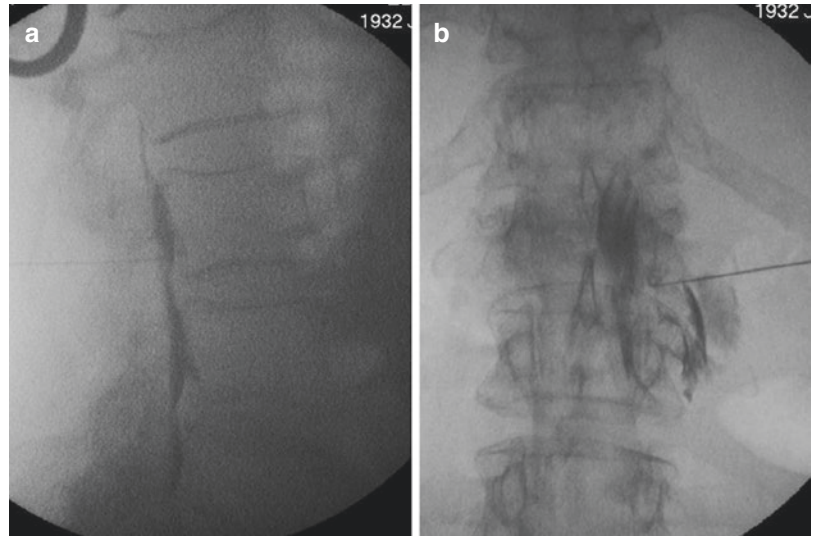


Fig. 29.6. Neurolytic transforaminal phenol injection in a patient with history of decompression laminectomies and tumor growth preventing placing the patient in lateral decubitus position required for neurolytic subarachnoid block. (**a**) Lateral view of needle and contrast spread, (**b**) A-P view of needle and contrast spread (Images courtesy of Kenneth D. Candido, MD)

Table 29.3. Summary of indications, contraindications, and complications for neuraxial neurolytic blocks

Neuraxial neurolytic blocks		
Indications	Contraindications	Complications
Well-established diagnosis Failure to respond to analgesic therapy Short life expectancy (6–12 months) Pain limited to 2 or 3 dermatomes Pain somatic in origin Unilateral pain Pain relieved with diagnostic block with local anesthetic	Neuraxial metastases Pathology in the spinal canal Coagulopathy Skin infection at the intended puncture site Active systemic infection Extensive or poorly localized Sympathetic or neuropathic pain Failure to respond to 2 diagnostic blocks	Paresis or paralysis Dysesthesias Urinary retention Bowel incontinence Aseptic/septic meningitis Epidural abscess Spinal cord injury Post-dural puncture headache

Data from [46, 48]

approach might specifically benefit patients willing to engage in physical therapy and rehabilitation, as the neuraxial neurolysis provides analgesia by blocking sensory fibers while sparing the motor fibers [46, 48]. Transforaminal phenol neurolysis was performed in a patient experiencing thoracolumbar pain with lower extremity radiculopathy secondary to stage IV metastatic disease, who failed to respond to other measures, including surgical intervention [49]. He derived an outstanding pain relief but also attained a substantial improvement of cognitive function. The intervention enabled the patient to discontinue all his analgesic medications and allowed him to enjoy meaningful family interactions in his final days. A different case report describes a complete resolution of an incapacitating leg pain in a terminal cancer patient after a chemical neurolysis of the L2, L3, and L4 lumbar nerve roots lasting for 6 weeks until his death [50].

Neuromodulation

The idea behind using stimulation of the nociceptive pathways to abolish pain perception came into existence in the 1960s. It is derived from the gate control theory formulated by Melzack and Wall in 1965 who postulated that stimulation of the Aβ nerve fibers in the dorsal horn of the spinal cord interrupts transmission of the ascending pain signals [51]. This evoked tonic stimulation classically elicits paresthesias in the affected territory perceived by the patient in place of the

painful sensation. Shealy et al. first attempted spinal cord stimulation (SCS) implantation in 1967 in an animal model [52], and 3 years later introduced it into the clinical practice [53].

Over the ensuing years, new types of SCSs have been developed, and presently non-tonic burst and dorsal root ganglion-specific (DRG) neuromodulation devices are used to treat select pain syndromes. SCS has been successfully used to treat pain due to failed back surgery syndrome [54], complex regional pain syndrome [55], ischemic limb pain [56], phantom limb pain [57], postherpetic neuralgia [58], chronic mesenteric ischemia [59], and refractory angina [60]. More recently, a dramatic pain relief has been achieved by patients with HIV-related polyneuropathy [61]. Neuromodulation technology is considered an effective alternative treatment strategy by the Neuromodulation Appropriateness Consensus Committee (NACC) of the International Neuromodulation Society (INS), which is not associated with the adverse reactions seen with pharmacotherapeutic strategies [62]. It is a minimally invasive and relatively safe procedure. A retrospective review of 707 cases of SCS shows that the most common complications include hardware malfunctioning, lead migration (22.6%), lead connection failure (9.5%), and lead breakage [63]. Other reported complications include pain at the generator site (12%) and infection (4.5%). The rate of infection in diabetic patients was 9 vs. 4% in the nondiabetics. Clinical reports illustrate that cancer-related neuropathic pain, such as chemotherapy-induced pain, is amenable to the treatment with SCS [64, 65].

At the present time, the literature assessing the success of this intervention for the management of cancer-type pain is limited to a small number of case series studies describing treatment of cancer-related chest wall [66, 67], low back [68], testicular [69], chemotherapy-induced [64], and other persistent neuropathic [70] pain syndromes. Despite those promising results, Cochrane systematic review by Peng et al. did not find sufficient evidence to establish the value of SCS in managing refractory malignancy-related pain [71]. The results presented by the few available studies are undoubtedly encouraging and lay a foundation for a more extensive application of this modality in treating cancer-related pain. The absence of high-quality clinical trials should not deter nor prohibit the use of neuromodulation in appropriately selected patients, especially those who have exhausted other conventional treatment options (Fig. 29.7).

Implantable Drug Delivery Systems (IDDS)

This approach is intended for patients on long-term opioid therapy who derived a suboptimal pain control despite dose escalation, or for those who benefit from the medications but side effects prohibits continuation of the therapy. Intrathecal pumps help to optimize pain control while bypassing systemic absorption, thereby reducing the risk of unpleasant and unnecessary side effects (Fig. 29.8). Additionally, with appropriately tailored dosing to the patient's needs, IDDS may ultimately reduce or completely withdraw oral opioid medications. Use of IDDS requires a carefully selected patient population, cautious dosing, and titration of the medication with an ongoing patient monitoring and pump management [72]. Indications, contraindications, and possible complications are presented in Table 29.4.

The success of IDDS has been demonstrated by prospective and randomized controlled studies [73–75]. Most recently, Zheng et al. [75] evaluated the clinical efficacy of the IDDS in a prospective cohort study in 53 patients with intractable cancer pain due to advanced malignancy on a high dose of oral opioids (mean 452.90 mg/day) and reported >50% pain reduction in almost 80% of patients at 1-month interval and 64% at 3-month interval. Both basal and breakthrough opioids decreased during the follow-up period with 90% patients discontinuing the oral opioid analgesics. Different pharmacological agents have been used with good results, including morphine, clonidine, or baclofen, although combinations of morphine and clonidine provide better analgesia than either of the medications alone [76].

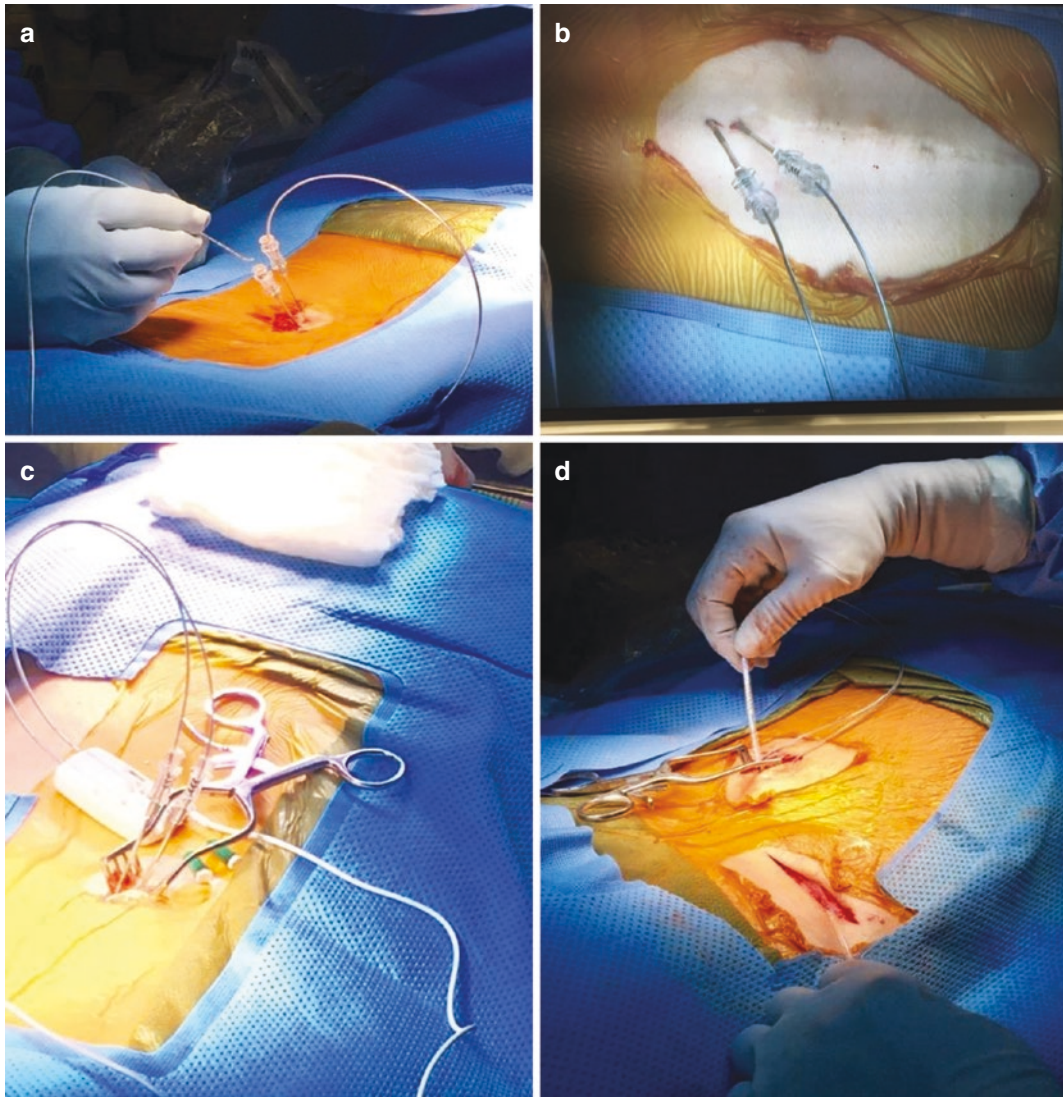


Fig. 29.7. Placement of spinal cord stimulator for the treatment of cancer-related neuropathic pain. (**a**) Two contiguous needles in the L1-L2 interlaminar space with eight-contact leads in epidural space, (**b**) leads extending from epidural needles, (**c**) creating dorsal anchoring site, (**d**) passage of leads from medial site, (**e**) fluoroscopic A-P view of epidural needle at L1-L2 and epidural lead extending from the needle tip, (**f**) A-P view of leads side by side in epidural space, (**g**) A-P view of bilateral eight-contact leads in epidural space (Images courtesy of Kenneth D. Candido, MD)

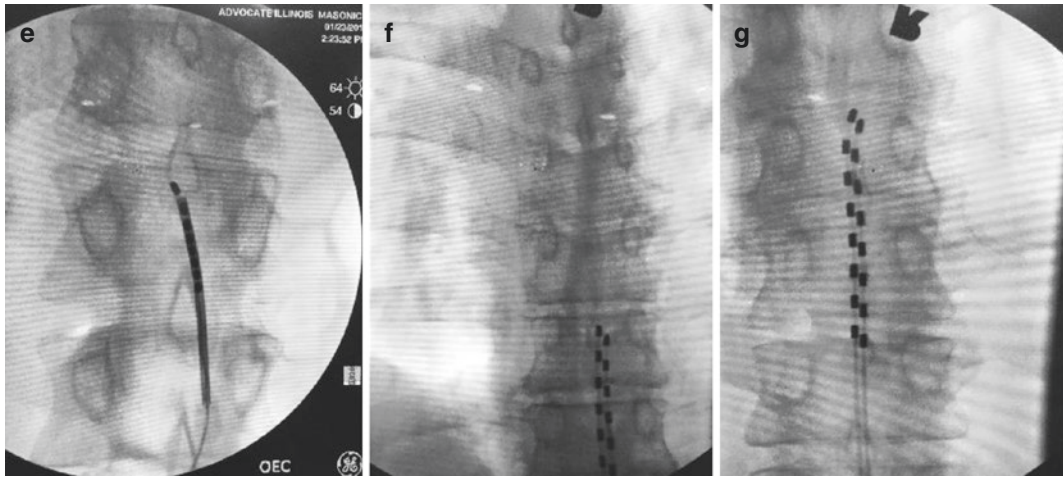


Fig. 29.7. (continued)

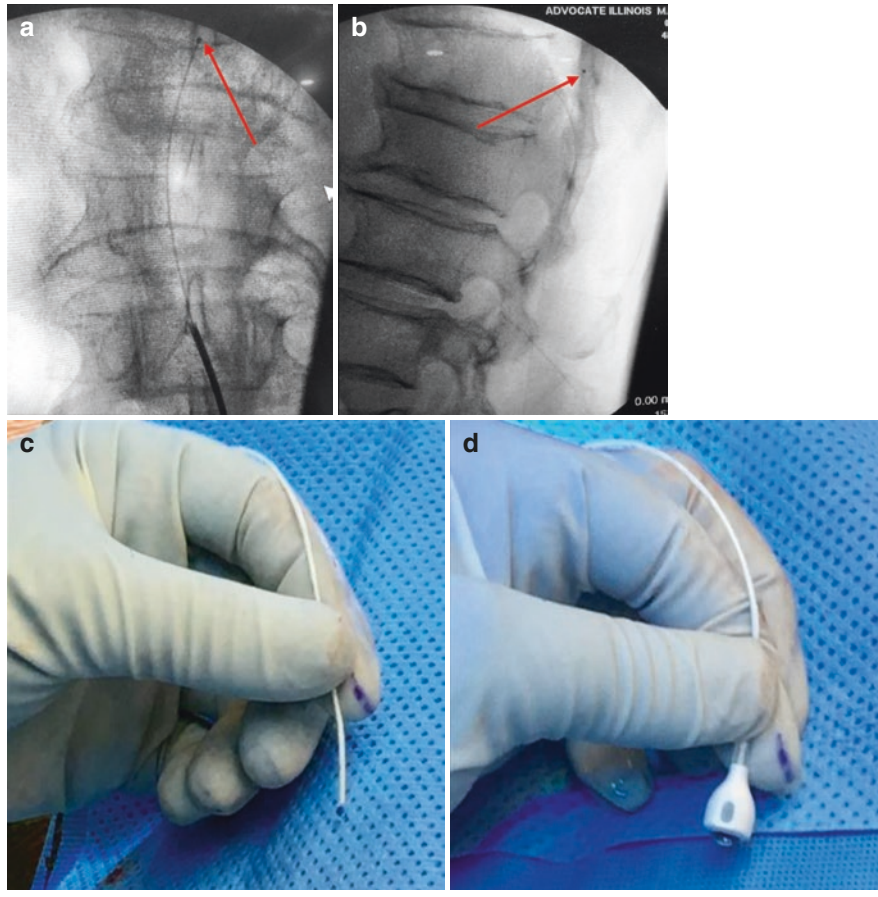
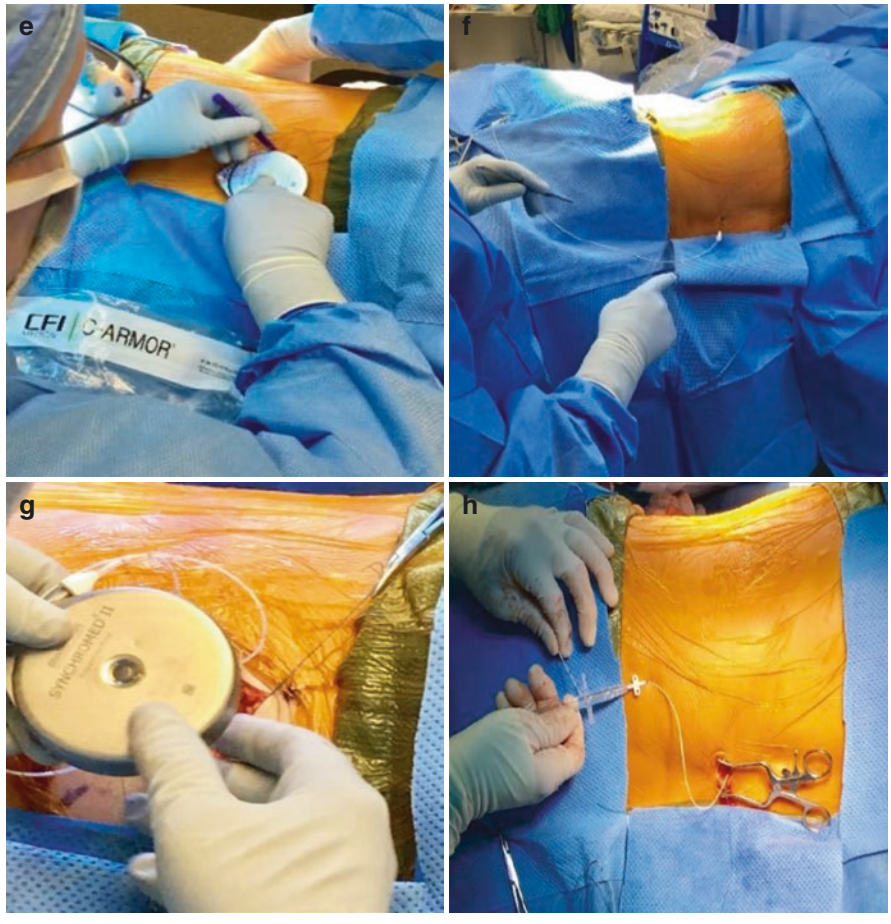


Fig. 29.8. Images depicting the steps of implantable intrathecal drug delivery system placement. (**a**) A-P fluoroscopic view showing needle in place and IDDS catheter tip (red arrow), (**b**) lateral fluoroscopic view of catheter in intrathecal space (red arrow), (**c**) clear, free flow of CSF confirmed from catheter tip, (**d**) CSF dripping from catheter tip indicating the presence in intrathecal space, (**e**) outlining pump pocket site, (**f**) measuring catheter length, (**g**) pump reservoir 20 ml or 40 ml, (**h**) aspirating CSF using syringe, (**i**) embedded IDDS catheter, (**j**) passage of catheter from back to abdomen, (**k**) wound closure of reservoir site (Images courtesy of Kenneth D. Candido, MD)



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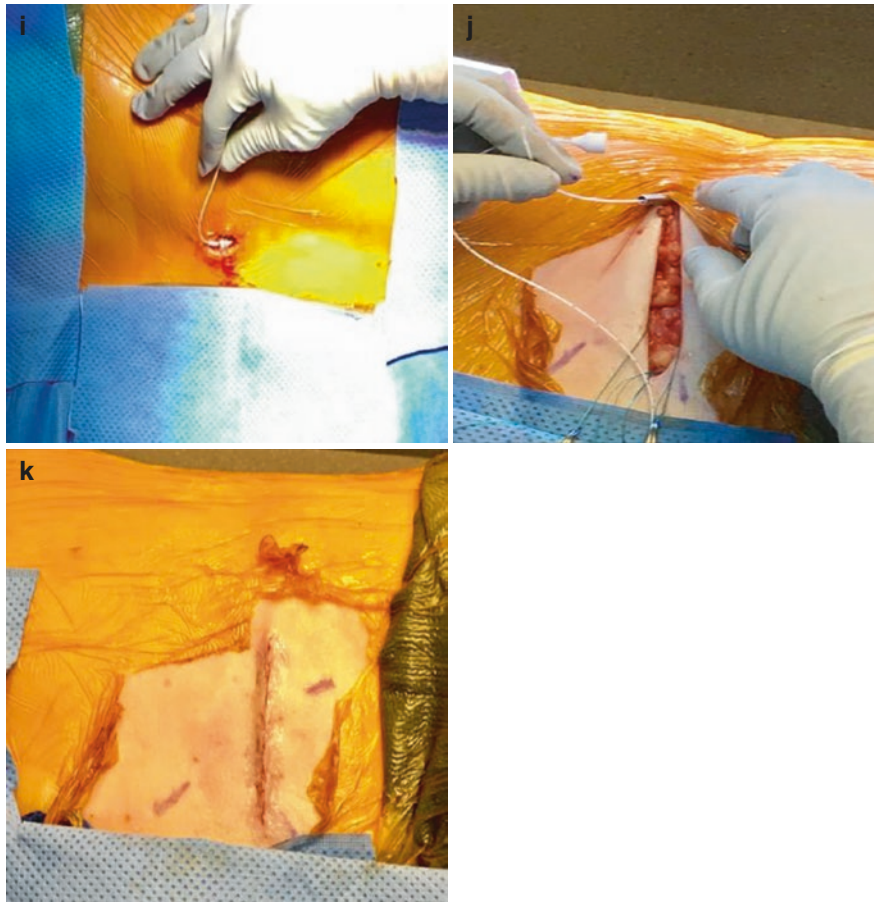

Fig. 29.8. (continued)

Table 29.4. Summary of indications, contraindications, and possible complications of intrathecal drug therapy

Intrathecal drug therapy		
Indications	Contraindications	Complications
Established diagnosis Nociceptive or neuropathic pain Well-localized pain End-of-life pain control Positive response to systemic opioids Moderate-severe intractable pain Inadequate analgesia with oral opioids Prohibitive side effects of oral opioids Morbid obesity or sleep apnea	Widespread pain Neuraxial metastases Pathology in the spinal canal/column Inability to implant catheter/device Coagulopathy Active systemic infection Inadequate family/caregiver support Patient unable to comply with the therapy	Surgical site infection Pump pocket infection Seroma of pump pocket Post-dural puncture headache Meningitis Pump/catheter malfunction Catheter tip granuloma Hematoma Medication-related side effects

Data from [47, 72, 77]

Vertebral Augmentation Procedures (VAP)

Bone metastatic lesions are a common occurrence in patients with advanced cancers. Incidence of spinal metastases reaches 70% in patients with breast, lung, and prostate cancers [78]. Vertebral compression fractures (VCFs) due to metastatic tumor compromise the stability of the vertebral column and can be a source of severe and debilitating pain for many cancer patients. Vertebral augmentation denotes two minimally invasive percutaneous interventions, vertebroplasty and kyphoplasty, used for symptom management and restoration of vertebral stability in patients with VCFs (Fig. 29.9). The procedures are done in the operating room under fluoroscopic or CT imaging guidance, with either local anesthetic and sedative or general anesthesia on board.

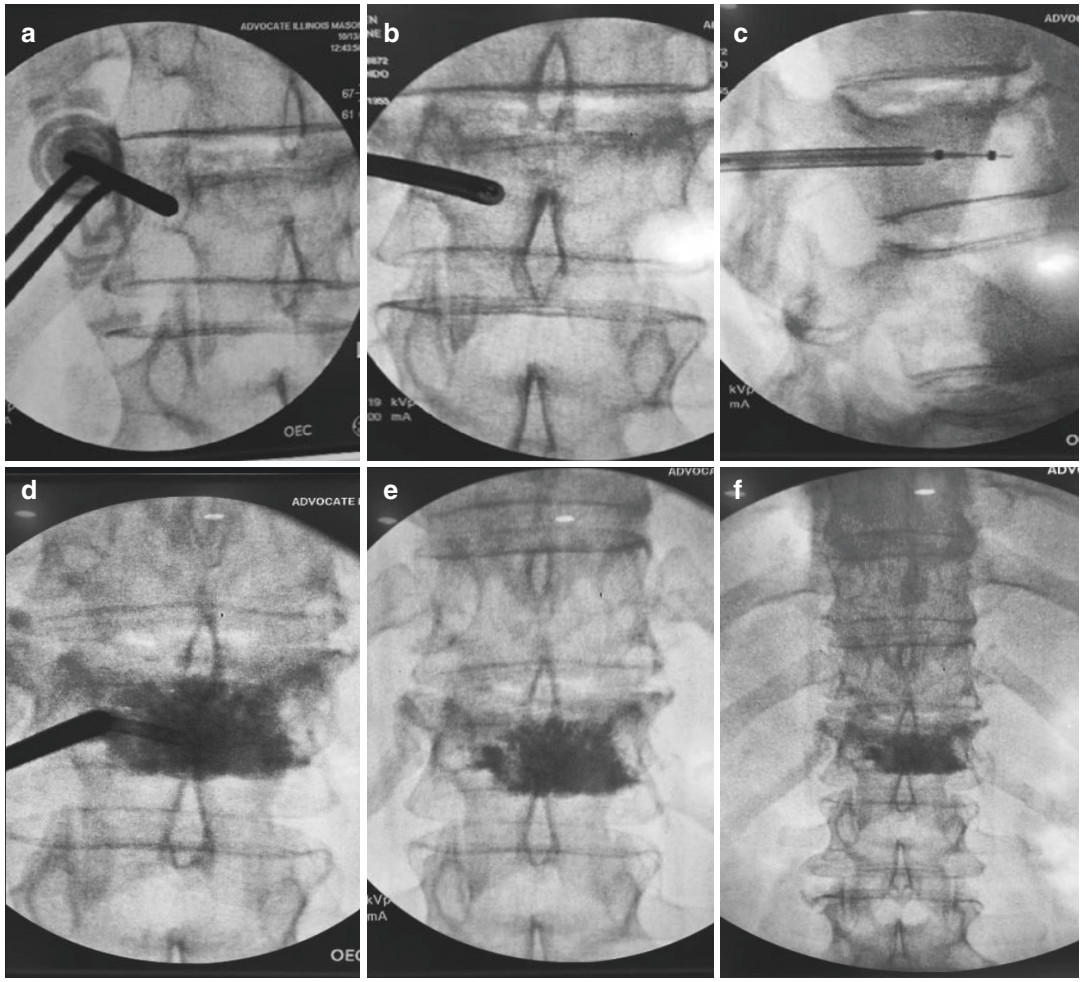

Fig. 29.9. Fluoroscopic images depicting the technique of vertebral augmentation. (**a**) A-P view of the trocar positioned at pedicle of T12, (**b**) A-P view showing the unipedicular approach at T12 toward midline, (**c**) lateral view with kyphoplasty balloon deflated protruding through the trocar tip, (**d**) A-P view showing barium-infused methyl methacrylate spreading from pedicle to pedicle, (**e**) trocar removed without track, (**f**) expanded A-P view showing MMA in T12 vertebral body (Images courtesy of Kenneth D. Candido, MD)

Table 29.5. Summary of the indications, contraindications, and complications related to vertebral augmentation

Vertebral augmentation		
Indications	Contraindications	Complications
VCFs due to primary bone tumors or metastatic infiltration Persistent pain despite conservative measures Inability to ambulate despite analgesic therapy Inability to participate in physical therapy despite analgesic therapy Presence of prohibitive side effects of pharmacological therapy	Uncorrectable coagulopathy Unstable spinal fractures Pregnancy Active site or systemic infection Asymptomatic fractures Improvement on medical therapy Pain unrelated to VCFs Allergy to PMMA or contrast dye Difficulty to lie prone Tumor extension into the spinal canal or cord compression Diffuse metastases Fracture of the posterior column Spinal stenosis Radicular pain	Cement extravasation Spinal cord or nerve compression Radiculopathy Paraplegia Pulmonary embolus of PMMA Spinal or paraspinal hematoma Pain at the injection site Infection Epidural abscess Adjacent vertebral fracture Rib or pedicle fracture Allergic reaction to PMMA or contrast dye

Data from [82, 83]

Percutaneous vertebroplasty (PVP) involves injection of bone cement polymethylmethacrylate (PMMA) into the affected vertebral body to preserve the stability of the spine and diminish pain due to metastatic spine tumors. Kyphoplasty is used with a goal to "restore the height of the collapsed vertebral body" by introducing an inflatable balloon catheter into the given vertebra and filling it with bone cement. Indications, contraindications, and potential complications of vertebral augmentation are listed in Table 29.5. Injection of acrylic cement in order to treat angioma-related spinal complications was first performed by Galibert and Deramond et al. in [79]. Shortly thereafter, the procedure has been adopted around the globe in patients with metastatic pheochromocytoma [80]. Recently, cervical vertebroplasty has been successfully applied to relieve neck pain in cancer patients with cervical metastases [81]. Use of vertebroplasty combined with radiofrequency ablation has been reported in 18 patients with metastatic vertebral fractures who experienced on average a five-point drop in VAS index score at the 6-month follow-up [82].

Conclusion

Malignant pain is a prevalent and debilitating symptom in the cancer patient population. The dynamic course and frequently poorly responsive nature of the malignant pain to pharmacologic agents requires reaching for other advanced therapeutic modalities. Interventional pain management techniques are an invaluable treatment strategy proven to provide relief for pain that is difficult to treat or unresponsive to the conservative management. The advantages of these modalities frequently exceed the ones provided by commonly employed oral analgesics in many cancer patients. An increased use of interventional pain techniques can alter pain management and influence outcomes for many cancer patients.

Case Study

A 64-year-old female patient with history of insulin-dependent diabetes mellitus, hyperlipidemia, and advanced pancreatic cancer status post Whipple procedure and chemoradiation therapy presented with chronic severe intractable abdominal pain. She has been receiving palliative care therapy with high-dose extended and immediate-release opioids, tricyclic antidepressants, and lidocaine patches for pain, as well as dexamethasone and ondansetron for nausea control. Despite the instituted regimen, the patient experiences continuous incapacitating pain. The patient's family inquires about other available pain management options.

Discussion

Optimal pain control is a critical, yet frequently challenging, component of the oncologic treatment. The WHO analgesic stepladder, encompassing non-opioids, opioids of varying potency, and other adjuvant pharmacologic agents (Fig. 29.1), has guided the treatment of malignant pain for the past three decades. Although, the algorithm successfully addresses the pain in a large number of cases, many cancer patients require the application of interventional pain management techniques for a satisfactory pain control. Neurolytic sympathetic blocks are an effective and proven therapeutic adjunct for the management of visceral type cancer-related pain (Table 29.2; Figs. 29.1, 29.2, 29.3, and 29.4).

Celiac plexus block (CPB) is utilized to relieve visceral upper abdominal pain due to the neoplasms of the pancreas or other abdominal organs (Fig. 29.1). The celiac plexus consists of sympathetic and parasympathetic nerve fibers and is situated in the retroperitoneal cavity under the diaphragm at the T12 and L1 vertebral level. Blockade of the celiac plexus is accomplished by depositing up to 20 mL of a neurolytic agent (usually absolute ethanol) in the para-aortic area at the lateral wall of the first lumbar vertebra after a positive response to a diagnostic block with a local anesthetic. CPB helps to ameliorate severe pain refractory to pharmacologic agents and allows reduction of the usage of narcotic medications and minimize the risk of undesirable side effects from the opioid therapy. These benefits may have a favorable effect on the function of the digestive tract permitting improved nutrition and increased caloric intake. Appropriate patient selection is essential due to a narrow risk-benefit ratio. Hypotension, increased gastric motility, aortic dissection, retroperitoneal hematoma, and spinal cord damage are some of the potential complications related to celiac plexus block technique.

Review Questions

1. Which of the following is the least likely adverse effect related to celiac plexus block?
   (a) Hiccups
   (b) Orthostatic hypotension
   (c) Constipation
   (d) Motor paralysis
2. The benefits of celiac plexus block include which of the following?
   (a) Decreased opioid consumption
   (b) Reduced risk of medication-related side effects
   (c) Improved appetite and nourishment
   (d) All of the above
3. Which of the following serve as contraindications to performing celiac plexus block?
   (a) Hypovolemia
   (b) Intestinal obstruction
   (c) Severe thrombocytopenia
   (d) Localized skin tissue infection
   (e) All of the above

Answers:

1. c
2. d
3. e

References

1. Caraceni A, Portenoy RK. An international survey of cancer pain characteristics and syndromes. IASP task force on cancer pain. International Association for the Study of Pain. Pain. 1999;82(3):263–74.
2. Bruera E, Kim HN. Cancer pain. JAMA. 2003;290(18):2476–9.
3. Grond S, Zech D, Diefenbach C, Radbruch L, Lehmann KA. Assessment of cancer pain: a prospective evaluation in 2266 cancer patients referred to a pain service. Pain. 1996;64(1):107–14.
4. Organization WH. Cancer Pain Relief. 1986:1–70.
5. Deandrea S, Montanari M, Moja L, Apolone G. Prevalence of undertreatment in cancer pain. A review of published literature. Ann Oncol. 2008;19(12):1985–91.
6. Vargas-Schaffer G. Is the WHO analgesic ladder still valid? Twenty-four years of experience. Can Fam Physician. 2010;56(6):514–7. e202-5
7. Klepstad P, Kurita GP, Mercadante S, Sjogren P. Evidence of peripheral nerve blocks for cancer-related pain: a systematic review. Minerva Anestesiol. 2015;81(7):789–93.
8. Vallejo R, de Leon-Casasola O, Benyamin R. Opioid therapy and immunosuppression: a review. Am J Ther. 2004;11(5):354–65.
9. Stone JB, Klein MA, Furman MB. Intercostal Blockade. In: Furman MB, editor. Atlas of

image-guided spinal procedures. Philadelphia, PA: Elsevier Saunders; 2013. p. 231–5.
10. Ahmed Z, Samad K, Ullah H. Role of intercostal nerve block in reducing postoperative pain following video-assisted thoracoscopy: a randomized controlled trial. Saudi J Anaesth. 2017;11(1):54–7.
11. Wong FC, Lee TW, Yuen KK, Lo SH, Sze WK, Tung SY. Intercostal nerve blockade for cancer pain: effectiveness and selection of patients. Hong Kong Med J. 2007;13(4):266–70.
12. Peng J, Wang SD. Effect of anesthesia combined with intercostal nerve block on analgesia for esophageal cancer after operation. Eur Rev Med Pharmacol Sci. 2015;19(12):2293–6.
13. Matchett G. Intercostal nerve block and Neurolysis for intractable cancer pain. J Pain Palliat Care Pharmacother. 2016;30(2):114–7.
14. Terkawi AS, Tsang S, Sessler DI, Terkawi RS, Nunemaker MS, Durieux ME, et al. Improving analgesic efficacy and safety of thoracic paravertebral block for breast surgery: a mixed-effects meta-analysis. Pain Physician. 2015;18(5):E757–80.
15. Bashandy GM, Abbas DN. Pectoral nerves I and II blocks in multimodal analgesia for breast cancer surgery: a randomized clinical trial. Reg Anesth Pain Med. 2015;40(1):68–74.
16. Wahba SS, Kamal SM. Thoracic paravertebral block versus pectoral nerve block for analgesia after breast surgery. Egyptian J Anaesth. 2013;30:129–35.
17. Kulhari S, Bharti N, Bala I, Arora S, Singh G. Efficacy of pectoral nerve block versus thoracic paravertebral block for postoperative analgesia after radical mastectomy: a randomized controlled trial. Br J Anaesth. 2016;117(3):382–6.
18. Wijayasinghe N, Andersen KG, Kehlet H. Analgesic and sensory effects of the Pecs local anesthetic block in patients with persistent pain after breast cancer surgery: a pilot study. Pain Pract. 2016;
19. Wisotzky EM, Saini V, Kao C. Ultrasound-guided intercostobrachial nerve block for intercostobrachial neuralgia in breast cancer patients: a case series. PM R. 2016;8(3):273–7.
20. Wijayasinghe N, Duriaud HM, Kehlet H, Andersen KG. Ultrasound guided intercostobrachial nerve blockade in patients with persistent pain after breast cancer surgery: a pilot study. Pain Physician. 2016;19(2):E309–18.
21. Vranken JH, Zuurmond WW, de Lange JJ. Continuous brachial plexus block as treatment for the Pancoast syndrome. Clin J Pain. 2000;16(4):327–33.
22. Buchanan D, Brown E, Millar F, Mosgrove F, Bhat R, Levack P. Outpatient continuous interscalene brachial plexus block in cancer-related pain. J Pain Symptom Manag. 2009;38(4):629–34.
23. Sato S, Yamashita S, Iwai M, Mizuyama K, Satsumae T. Continuous interscalene block for cancer pain. Reg Anesth. 1994;19(1):73–5.
24. Zinboonyahgoon N, Vlassakov K, Abrecht CR, Srinivasan S, Narang S. Brachial plexus block for cancer-related pain: a case series. Pain Physician. 2015;18(5):E917–24.
25. Naja Z, Lonnqvist PA. Somatic paravertebral nerve blockade. Incidence of failed block and complications. Anaesthesia. 2001;56(12):1184–8.
26. Burlacu CL, Buggy DJ. Coexisting harlequin and Horner syndromes after high thoracic paravertebral anaesthesia. Br J Anaesth. 2005;95(6):822–4.
27. Lenters TR, Davies J, Matsen FA 3rd. The types and severity of complications associated with interscalene brachial plexus block anesthesia: local and national evidence. J Shoulder Elb Surg. 2007;16(4):379–87.
28. Candido KD. Brachial plexus blocks: techniques above the clavicle. In: Benzon HT, editor. Essentials of pain medicine and regional anesthesia. 2nd ed. Philadelphia, PA: Elsevier; 2005. p. 607–21.
29. Candido KD. Yaghmour E. Brachial plexus blocks: techniques below the clavicle. In: Benzon H, editor. Essentials of pain medicine and regional anesthesia. 2nd ed. Philadelphia, PA: Elsevier Churchill Livingstone; 2005. p. 622–35.
30. Mercadante S, Klepstad P, Kurita GP, Sjogren P, Giarratano A, European Palliative Care Research C. Sympathetic blocks for visceral cancer pain management: a systematic review and EAPC recommendations. Crit Rev Oncol Hematol. 2015;96(3):577–83.
31. Nagels W, Pease N, Bekkering G, Cools F, Dobbels P. Celiac plexus neurolysis for abdominal cancer pain: a systematic review. Pain Med. 2013;14(8):1140–63.
32. Kaufman M, Singh G, Das S, Concha-Parra R, Erber J, Micames C, et al. Efficacy of endoscopic ultrasound-guided celiac plexus block and celiac plexus neurolysis for managing abdominal pain associated with chronic pancreatitis and pancreatic cancer. J Clin Gastroenterol. 2010;44(2):127–34.
33. Staats PS, Hekmat H, Sauter P, Lillemoe K. The effects of alcohol celiac plexus block, pain, and mood on longevity in patients with unresectable pancreatic cancer: a double-blind, randomized, placebo-controlled study. Pain Med. 2001;2(1):28–34.
34. Wong GY, Schroeder DR, Carns PE, Wilson JL, Martin DP, Kinney MO, et al. Effect of neurolytic celiac plexus block on pain relief, quality of life, and survival in patients with unresectable pancreatic cancer: a randomized controlled trial. JAMA. 2004;291(9):1092–9.
35. Erdek MA, Halpert DE, Gonzalez Fernandez M, Cohen SP. Assessment of celiac plexus block and neurolysis outcomes and technique in the management of refractory visceral cancer pain. Pain Med. 2010;11(1):92–100.
36. Zhong W, Yu Z, Zeng JX, Lin Y, Yu T, Min XH, et al. Celiac plexus block for treatment of pain associated with pancreatic cancer: a meta-analysis. Pain Pract. 2014;14(1):43–51.
37. Wyse JM, Carone M, Paquin SC, Usatii M, Sahai AV. Randomized, double-blind, controlled trial of early endoscopic ultrasound-guided celiac plexus neurolysis to prevent pain progression in patients with newly diagnosed, painful, inoperable pancreatic cancer. J Clin Oncol. 2011;29(26):3541–6.

38. Arcidiacono PG, Calori G, Carrara S, McNicol ED, Testoni PA. Celiac plexus block for pancreatic cancer pain in adults. Cochrane Database Syst Rev. 2011;3:CD007519.
39. de Oliveira R. Dos Reis MP, Prado WA. The effects of early or late neurolytic sympathetic plexus block on the management of abdominal or pelvic cancer pain. Pain. 2004;110(1–2):400–8.
40. Mishra S, Bhatnagar S, Rana SP, Khurana D, Thulkar S. Efficacy of the anterior ultrasound-guided superior hypogastric plexus neurolysis in pelvic cancer pain in advanced gynecological cancer patients. Pain Med. 2013;14(6):837–42.
41. Plancarte R, de Leon-Casasola OA, El-Helaly M, Allende S, Lema MJ. Neurolytic superior hypogastric plexus block for chronic pelvic pain associated with cancer. Reg Anesth. 1997;22(6):562–8.
42. Zechlinski JJ, Hieb RA. Lumbar sympathetic Neurolysis: how to and when to use? Tech Vasc Interv Radiol. 2016;19(2):163–8.
43. Gulati A, Khelemsky Y, Loh J, Puttanniah V, Malhotra V, Cubert K. The use of lumbar sympathetic blockade at L4 for management of malignancy-related bladder spasms. Pain Physician. 2011;14(3):305–10.
44. Woo JH, Park HS, Kim SC, Kim YH. The effect of lumbar sympathetic ganglion block on gynecologic cancer-related lymphedema. Pain Physician. 2013;16(4):345–52.
45. Toshniwal GR, Dureja GP, Prashanth SM. Transsacrococcygeal approach to ganglion impar block for management of chronic perineal pain: a prospective observational study. Pain Physician. 2007;10(5):661–6.
46. Candido KD. Neurolytic blocks. In: Diwan S, editor. Atlas of Pain Medicine Procedures: MCGraw-Hill Professional; 2014.
47. Sindt JE, Brogan SE. Interventional treatments of cancer pain. Anesthesiol Clin. 2016;34(2):317–39.
48. Candido K, Stevens RA. Intrathecal neurolytic blocks for the relief of cancer pain. Best Pract Res Clin Anaesthesiol. 2003;17(3):407–28.
49. Candido KD, Philip CN, Ghaly RF, Knezevic NN. Transforaminal 5% phenol neurolysis for the treatment of intractable cancer pain. Anesth Analg. 2010;110(1):216–9.
50. De Pinto M, Naidu RK. Peripheral and neuraxial chemical neurolysis for the management of intractable lower extremity pain in a patient with terminal cancer. Pain Physician. 2015;18(4):E651–6.
51. Melzack R, Wall PD. Pain mechanisms: a new theory. Science. 1965;150(3699):971–9.
52. Shealy CN, Taslitz N, Mortimer JT, Becker DP. Electrical inhibition of pain: experimental evaluation. Anesth Analg. 1967;46(3):299-305.
53. Shealy CN, Mortimer JT, Hagfors NR. Dorsal column electroanalgesia. J Neurosurg. 1970;32(5):560–4.
54. Frey ME, Manchikanti L, Benyamin RM, Schultz DM, Smith HS, Cohen SP. Spinal cord stimulation for patients with failed back surgery syndrome: a systematic review. Pain Physician. 2009;12(2):379–97.
55. Grabow TS, Tella PK, Raja SN. Spinal cord stimulation for complex regional pain syndrome: an evidence-based medicine review of the literature. Clin J Pain. 2003;19(6):371–83.
56. Simpson EL, Duenas A, Holmes MW, Papaioannou D, Chilcott J. Spinal cord stimulation for chronic pain of neuropathic or ischaemic origin: systematic review and economic evaluation. Health Technol Assess. 2009;13(17):iii–x.
57. Viswanathan A, Phan PC, Burton AW. Use of spinal cord stimulation in the treatment of phantom limb pain: case series and review of the literature. Pain Pract. 2010;10(5):479–84.
58. Harke H, Gretenkort P, Ladleif HU, Koester P, Rahman S. Spinal cord stimulation in postherpetic neuralgia and in acute herpes zoster pain. Anesth Analg. 2002;94(3):694–700.
59. Ceballos A, Cabezudo L, Bovaira M, Fenollosa P, Moro B. Spinal cord stimulation: a possible therapeutic alternative for chronic mesenteric ischaemia. Pain. 2000;87(1):99–101.
60. Taylor RS, De Vries J, Buchser E, Dejongste MJ. Spinal cord stimulation in the treatment of refractory angina: systematic review and meta-analysis of randomised controlled trials. BMC Cardiovasc Disord. 2009;9(13)
61. Knezevic NN, Candido KD, Rana S, Knezevic I. The use of spinal cord Neuromodulation in the management of HIV-related polyneuropathy. Pain Physician. 2015;18(4):E643–50.
62. Deer TR, Mekhail N, Provenzano D, Pope J, Krames E, Leong M, et al. The appropriate use of neurostimulation of the spinal cord and peripheral nervous system for the treatment of chronic pain and ischemic diseases: the Neuromodulation appropriateness consensus committee. Neuromodulation. 2014;17(6):515–50. discussion 50
63. Mekhail NA, Mathews M, Nageeb F, Guirguis M, Mekhail MN, Cheng J. Retrospective review of 707 cases of spinal cord stimulation: indications and complications. Pain Pract. 2011;11(2):148–53.
64. Cata JP, Cordella JV, Burton AW, Hassenbusch SJ, Weng HR, Dougherty PM. Spinal cord stimulation relieves chemotherapy-induced pain: a clinical case report. J Pain Symptom Manag. 2004;27(1):72–8.
65. Abd-Elsayed A, Schiavoni N, Sachdeva H. Efficacy of spinal cord stimulators in treating peripheral neuropathy: a case series. J Clin Anesth. 2016;28:74–7.
66. Wininger KL, Bester ML, Deshpande KK. Spinal cord stimulation to treat postthoracotomy neuralgia: non-small-cell lung cancer: a case report. Pain Manag Nurs. 2012;13(1):52–9.
67. Yakovlev AE, Resch BE, Karasev SA. Treatment of cancer-related chest wall pain using spinal cord stimulation. Am J Hosp Palliat Care. 2010;27(8):552–6.
68. Yakovlev AE, Resch BE. Spinal cord stimulation for cancer-related low back pain. Am J Hosp Palliat Care. 2012;29(2):93–7.
69. Nouri KH, Brish EL. Spinal cord stimulation for testicular pain. Pain Med. 2011;12(9):1435–8.

70. Yakovlev AE, Ellias Y. Spinal cord stimulation as a treatment option for intractable neuropathic cancer pain. Clin Med Res. 2008;6(3–4):103–6.
71. Peng L, Min S, Zejun Z, Wei K, Bennett MI. Spinal cord stimulation for cancer-related pain in adults. Cochrane Database Syst Rev. 2015;6:CD009389.
72. Pope JE, Deer TR, Bruel BM, Falowski S. Clinical uses of Intrathecal therapy and its placement in the pain care algorithm. Pain Pract. 2016;16:1092.
73. Rauck RL, Cherry D, Boyer MF, Kosek P, Dunn J, Alo K. Long-term intrathecal opioid therapy with a patient-activated, implanted delivery system for the treatment of refractory cancer pain. J Pain. 2003;4(8):441–7.
74. Smith TJ, Staats PS, Deer T, Stearns LJ, Rauck RL, Boortz-Marx RL, et al. Randomized clinical trial of an implantable drug delivery system compared with comprehensive medical management for refractory cancer pain: impact on pain, drug-related toxicity, and survival. J Clin Oncol. 2002;20(19):4040–9.
75. Zheng S, He L, Yang X, Li X, Yang Z. Evaluation of intrathecal drug delivery system for intractable pain in advanced malignancies: a prospective cohort study. Medicine (Baltimore). 2017;96(11):e6354.
76. Siddall PJ, Molloy AR, Walker S, Mather LE, Rutkowski SB, Cousins MJ. The efficacy of intrathecal morphine and clonidine in the treatment of pain after spinal cord injury. Anesth Analg. 2000;91(6):1493–8.
77. Deer TR, Smith HS, Burton AW, Pope JE, Doleys DM, Levy RM, et al. Comprehensive consensus based guidelines on intrathecal drug delivery systems in the treatment of pain caused by cancer pain. Pain Physician. 2011;14(3):E283–312.
78. Wibmer C, Leithner A, Hofmann G, Clar H, Kapitan M, Berghold A, et al. Survival analysis of 254 patients after manifestation of spinal metastases: evaluation of seven preoperative scoring systems. Spine (Phila Pa 1976). 2011;36(23):1977–86.
79. Galibert P, Deramond H, Rosat P, Le Gars D. Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplasty. Neurochirurgie. 1987;33(2):166–8.
80. Cai S, Kong X, Yan C, Liu Y, Zhou X, Qiu G. Successful treatment of metastatic pheochromocytoma in the spine with cement augmentation. Medicine (Baltimore). 2017;96(4):e5892.
81. Bao L, Jia P, Li J, Chen H, Dong Y, Feng F, et al. Percutaneous vertebroplasty relieves pain in cervical spine metastases. Pain Res Manag. 2017;2017:3926318.
82. Maugeri R, Graziano F, Basile L, Guli C, Giugno A, Giammalva GR, et al. Reconstruction of vertebral body after radiofrequency ablation and augmentation in dorsolumbar metastatic vertebral fracture: analysis of clinical and radiological outcome in a clinical series of 18 patients. Acta Neurochir Suppl. 2017;124:81–6.
83. Health Quality O. Vertebral augmentation involving Vertebroplasty or Kyphoplasty for cancer-related vertebral compression fractures: a systematic review. Ont Health Technol Assess Ser. 2016;16(11):1–202.

Regional Anesthesia and Cardiovascular Disease

30

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Introduction

In 2010, approximately 7.4 million surgeries involving the cardiovascular system were performed in the United States, of which 395,000 were cardiac bypass surgeries [1]. Patients who present for such procedures have significant comorbidities involving the pulmonary, cardiac, and peripheral vascular system. Thus, this cohort of patients is at a significant risk perioperatively. Studies have shown that several chronic cardiac conditions such as coronary artery disease increase the risk for cardiac complications after surgery [2].

Surgical stress can result in major adverse cardiac events, and this accounts for significant morbidity and mortality [3]. Surgical stress response encompasses a wide range of endocrinal, immunological, and hematological effects that lead to untoward effects, including potential organ dysfunction which can culminate in undesirable patient outcomes [4]. Effective regional analgesia with local anesthetic agents prevents the endocrine and metabolic responses to surgery. For example, epidural block from dermatomal segment T4 to S5, established before the start of surgery, has been shown to prevent increases in cortisol and glucose concentrations in response to hysterectomy [4]. Both afferent inputs from the operative site to the central nervous system and efferent autonomic neuronal pathways are blocked, minimizing or negating a "surgical stress response."

In addition to alleviating surgical stress, more effective analgesia provided by regional block (neuraxial, paravertebral, and peripheral nerve block) can allow for improved postoperative function improving recovery times and reducing potential complications.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_30

Neuraxial Block

In patients undergoing cardiac surgery, respiratory complications have been reported to be more likely the cause of death than those related to cardiac complications [5]. Adequate perioperative analgesia enables full expansion of the chest, reducing the risk of splinting, atelectasis, and pneumonia in spontaneously breathing patients. An effective thoracic epidural or intrathecal catheter may, thus, reduce morbidity, hospital costs, and length of stay, therefore, improving the patient's quality of life and reducing the incidence of chronic pain.

Physiologic Effects of Neuraxial Block

Neuraxial anesthesia is a well-known tool for anesthesia and pain control in obstetrics; however, it is also an effective anesthetic for many common surgeries including general, vascular, and orthopedic surgeries. Given the aging and increased survival of the overall population, as well as the widespread incidence of cardiovascular disease among the general population, it is essential to be familiar with the cardiovascular effects of neuraxial block to guide appropriate patient selection and management.

In humans, the two autonomic outflow tracts from the central nervous system, the parasympathetic and the sympathetic system, exit as the craniosacral outflow and thoracolumbar outflow, respectively.

The parasympathetic portion of the autonomic nervous system (ANS) consists of a cranial output arising from the cranial nerves 3, 7, 9, and 10. The sacral output arises from the sacral spinal cord. In contrast, the sympathetic thoracolumbar output has its preganglionic neuron origin in the intermediolateral column of the thoracic and upper lumbar spinal cord. White communicating rami from the sympathetic preganglionic neurons synapse with sympathetic neurons in the paravertebral sympathetic chain. Thus, it is quite evident that any thoracic spinal cord block would block sympathetic output leading to a parasympathetic dominance. Part of the physiologic effects of a thoracic and lumbar epidural block can be partly explained by this parasympathetic dominance.

Preganglionic sympathetic nerve fibers originate in the thoracolumbar spine. These nerve fibers regulate vasomotor tone throughout the body. Neuraxial block leads to a "sympathectomy," whereby transmission from these nerve fibers is inhibited, leading to vasodilatory effects. Significant vasodilatation can occur as local anesthetics can spread to higher thoracic levels and lead to vasodilatation of the splanchnic veins (T5–L3), depending on volume and concentration of administered local anesthetic. This sympathectomy and resulting vasodilation affect both arterial and venous blood vessels; however, given that a disproportionately larger volume of blood resides in the venous system, the decrease in venous return to the heart can be profound. The Bezold-Jarisch reflex may be elicited if the patient is hypovolemic, resulting in bradycardia to allow increased diastolic filling time of the heart. This bradycardia may result in vagal symptoms and possibly cardiac arrest in even healthy patients; thus ensuring adequate preload is essential [6, 7].

Neuraxial block is also known for decreasing the release of catecholamines, and thus the stress response, related to the trauma of surgery. The block that extends to at least L1 and higher decreases the transmission of efferent sympathetic fibers to the adrenal medulla. It also blocks afferent impulses that signal a noxious stimulus to the adrenal medulla and the resulting catecholamine release. This is beneficial to patients with cardiac disease including coronary artery disease and aortic stenosis, where catecholamine release can lead to tachycardia and increased myocardial oxygen demand causing perioperative acute coronary syndrome or arrhythmias [6–10].

Intrathecal Anesthesia

Spinal Block in Adult Cardiac Surgery

Leading experts have described the advantages of single-dose spinal anesthesia in addition to general anesthesia for patients undergoing cardiac

surgery [7]. As opinions in the management of cardiac patients evolve, so do anesthetic techniques. Over the years, the technique of high-dose intravenous opioid use for cardiac stability has changed to a balanced technique for fast tracking and earlier extubation [8]. A more contemporary approach to caring for cardiac patients includes the minimizing of immunosuppression and humoral/metabolic responses to surgical stimulation. These goals may all be provided by a sympathectomy via spinal anesthesia: positive myocardial oxygen balance (hypodynamic circulation), protection of myocardium and abdominal organs (redistribution of blood flow within the myocardium or abdominal organs), and protection against β-receptor downregulation [9].

A meta-analysis in 2004 by Liu et al. reviewed the risks vs. benefits of intrathecal analgesia for patient's undergoing coronary artery bypass grafting (CABG). Seventeen RCTs with 668 patients met inclusion criteria. This review did not show any significant effect on incidences of mortality, myocardial infarction, dysrhythmias, nausea/vomiting, or time to tracheal extubation [10].

Intrathecal opioids alone may offer potential mechanisms for improved outcomes secondary to improved analgesia and reduced stress response [10]. In a recent evaluation of high-quality studies on the use of single-dose intrathecal morphine, Richardson et al. concluded that intrathecal morphine reduces postoperative pain scores, increases the time to first IV morphine dose, and reduces the overall postoperative IV morphine dose required [11]. Intrathecal morphine has been demonstrated to cause comparable opioid-related complications to intravenously administered morphine. Significant reductions in time to extubation, reductions in ICU stay, and improvements in postoperative lung function were found only in small retrospective studies. Despite the use of anticoagulation, no spinal hematomas were reported in any of the studies evaluated.

Intrathecal catheters are not used as frequently as epidural catheters related to concerns of inadvertently overdosing the intrathecal catheter. There is also a risk of development of a spinal hematoma following heparinization during cardiac surgery. However, the spinal technique has been associated with a lower risk of hematoma formation compared to the epidural technique [7].

Spinal Block in Cardiac Patients Undergoing Non-cardiac Surgery

Intrathecal anesthesia, usually used as a combined spinal and epidural catheter, is frequently used for non-cardiac surgery as the primary anesthetic. It can also be used combined with general anesthesia for many benefits, such as to decrease intraoperative opioid use and to improve postoperative pain.

Fleron et al. studied 217 patients undergoing abdominal aortic surgery, demonstrating that the group which received intrathecal opioid provided more intense analgesia than the patient-controlled analgesia (PCA) group during the first 24 h postoperatively ($p < 0.05$). However, intrathecal opioids do not improve any major incidence of cardiovascular, respiratory, or renal complications when compared to the general anesthetic group [12].

The concern for hemodynamic instability from the sympathectomy following spinal anesthesia may deter anesthesia providers from using intrathecal anesthesia, especially in patients with preexisting cardiac diseases. Juelsgaard et al. studied 43 patients with coronary artery disease undergoing hip surgery [13]. They found that the incidence of myocardial ischemia and hypotension was lowest in the group receiving incremental doses of spinal anesthesia compared to single-dose injection and general anesthesia without spinal anesthesia. However, there was no significant difference in mortality rates among the three groups after 1 month.

In a retrospective study by Rashid et al. 194 elderly patients underwent open reduction internal fixation (ORIF) of intertrochanteric femur fractures, either under spinal or general anesthesia. Spinal anesthesia reduced operating room (OR) time. However, there were no statistically significant differences in the rates of wound infections, the length of hospital stay, postoperative ambulation status, intraoperative blood loss, postoperative complications, and mortality between the two groups [14].

In Acute Pain Management

Epidural catheters can be used in the postoperative setting, whereas an intrathecal dose of opioid lasts 24 h. Catheters, however, are not commonly used for cardiac surgery related to the risk of hematoma formation due to heparinization for cardiopulmonary bypass. Latham et al. compared the effects of an intravenous remifentanil infusion plus intrathecal morphine with intravenous sufentanil infusion [15]. Both groups also underwent a general anesthetic. There were no significant differences in time to extubation; however, following extubation, patients in the remifentanil infusion plus intrathecal morphine had significantly lower visual analog scale (VAS), reduced PCA requirements, and greater satisfaction with postoperative pain management when compared to the group that only received a sufentanil infusion.

Epidural Block

Epidural analgesia and anesthesia have been well established as the gold standard in pain management for a variety of surgical procedures. Epidural anesthesia has also been successfully used as a sole anesthetic in both healthy patients and patients with severe cardiorespiratory disease.

Cited benefits of epidural anesthesia include the avoidance of the need for airway instrumentation, especially beneficial in patients with a potentially difficult airway, avoidance of general anesthesia, and effects on cardiovascular function, which may be beneficial in patients with severe cardiac disease. Additionally, reduction in perioperative opioid requirements has been described as well as superior analgesia which is beneficial in many patient populations such as those with opioid dependence or tolerance.

However, epidural anesthesia and analgesia are not without issues. Potential complications and side effects include the physiologic effects of sympathetic block, neurological injury, and infection. In the presence of coagulopathy, placement or presence of an epidural catheter is greatly discouraged to minimize the chance of an epidural hematoma which can potentially compress the spinal cord.

It is recommended, therefore, that epidural block is placed at the level of the intended site of action. In this regard, low lumbar epidural placement is not advisable when coverage is required in thoracic dermatomes.

Epidural Block in Cardiac Surgery

Pain leading to surgical stress response has significant undesirable effects perioperatively. In patients undergoing cardiac surgery, pain has been shown to be a significant factor contributing to chronic post-sternotomy pain. Adequate pain control has the added benefit of improved hemodynamics and myocardial oxygenation [16]. Thus, thoracic epidural analgesia as a part of a general anesthetic regimen may provide significant benefits in patients undergoing cardiac surgery.

A recent Cochrane review of randomized control trials (e.g., 31 trials in 3047 patients, 1578 patients with general anesthesia, and 1469 patients with general anesthesia and thoracic epidurals) demonstrated a significant benefit for a reduction in arrhythmias and pulmonary complications [17]. A benefit was also shown for a reduction in mortality, myocardial infarction, and stroke but was not statistically conclusive related to the scarcity of these events and the inadequacy of sample size. Zhang et al. recently performed a similar meta-analysis and came to a similar conclusion [18]. Additionally, Zhang et al. found a significant reduction in intensive care unit (ICU) and hospital stay, significantly better pain control, and earlier time to tracheal extubation in those patients with a thoracic epidural. In a recent retrospective review of 288 patient charts comparing general anesthesia with general anesthesia plus thoracic epidural analgesia in cardiac surgery, Porizka et al. concluded there were no major differences in early postoperative outcome data between the two groups [19]. However, patients in the thoracic group exhibited superior pain relief, shorter time to extubation, and earlier hospital discharge.

In off-pump coronary artery bypass, the degree of heparinization is greatly reduced. In a two-center, open, parallel-group randomized control trial, Caputo et al. evaluated 266 patients, 109 of which were randomly assigned to general anesthesia plus thoracic epidural anesthesia and 117 to only general anesthesia [20]. Time to discharge in the epidural group was significantly shorter by 1 day. There was also a significant reduction in new arrhythmias and median intubation time in the thoracic epidural group. As expected, pain scores were also significantly reduced postoperatively. There were no instances of epidural hematoma despite heparinization.

Epidural Block in Cardiac Patients Undergoing Non-cardiac Surgery

Epidural anesthesia has mainly been used in vascular and orthopedic surgery and is of benefit particularly in patients with cardiovascular disease where general anesthesia presents an undue risk. A good example would be patients undergoing aortic and peripheral vascular surgery.

In a 2016 Cochrane meta-analysis of epidural analgesia versus systemic opioid-based pain relief for abdominal aortic surgery [21], Guay et al. evaluated 15 trials involving 1498 patients. Authors concluded from their analysis that epidural analgesia provided better pain management, reduced myocardial infarction, reduced time to tracheal extubation, reduced postoperative respiratory failure, reduced gastrointestinal bleeding, and reduced intensive care unit length of stay compared with systemic opioid-based drugs. However, there was no difference in 30-day mortality. Bardia et al. [22], in a retrospective chart analysis, found a benefit when looking at mortality over 5 years, as opposed to 30 days. They compared general anesthesia (GA) with general anesthesia plus epidural anesthesia (EA-GA) in aortic abdominal aortic aneurysmal surgery. A total of 1540 patients were identified, 980 of which received EA-GA. At 5 years, overall survival rates were 74% (95% CI, 72–76%) and 65% (95% CI, 62–68%) in the EA-GA and GA-alone groups, respectively ($p < 0.01$). Patients receiving EA-GA also had lower odds of a 30-day surgical re-intervention (odds ratio [OR], 0.65; 95% CI, 0.44–0.94; $p = 0.02$) as well as postoperative bowel ischemia (OR, 0.54; 95% CI, 0.31–0.94; $p = 0.03$), pulmonary complications (OR, 0.62; 95% CI, 0.41–0.95; $p = 0.03$), and dialysis requirements (OR, 0.44; 95% CI, 0.23–0.88; $p = 0.02$). No significant differences were noted for the odds of wound and cardiac complications. However, based on such a data registry alone, it remains unclear as to whether the long-term survival was a direct consequence of epidural usage as an adjunct.

Epidural Block and Epidural Hematoma in Cardiovascular Surgery

Lack of widespread acceptance of epidural analgesia in cardiac surgery is related to a major concern for the possibility of an epidural hematoma, especially with the use of anticoagulation required for cardiopulmonary bypass.

Few studies have examined the benefits of the use of epidural block in patients undergoing cardiac surgery. In a recent meta-analysis of 66 trials involving 6383 patients, Landoni et al. found a significant reduction in mortality in those patients who had an epidural placed for cardiac surgery (59/3123 [1.9%] vs. 108/3260 [3.3%],RR 0.65 [95% CI 0.48–0.86], $p = 0.003$, NNT = 70) [23]. No epidural hematoma was reported in these 66 trials (3320 epidurals). After evaluating other literature (case reports, case series, and randomized trials not fulfilling inclusion criteria) and the results of a web-based international study, the authors estimated the risk of an epidural hematoma following epidural placement for cardiac surgery to be 1:3552.

The risk of epidural hematoma in patients undergoing non-cardiac surgery has been more extensively studied. In a retrospective study involving 7430 patients over a 10-year period at a tertiary teaching hospital, investigators determined the incidence of serious adverse events (epidural abscess, persistent neurological damage, cardiac arrest) to be 0.014% and identified no cases of an epidural hematoma [24]. In another

meta-analysis of randomized trials (125 trials involving 9044 patients), investigators found there were no reports of severe neurologic complications because of hematoma, infection, or trauma [25]. In a more comprehensive study, the United Kingdom National Health Service audit of 707,455 neuraxial techniques performed over a 1 year period identified 5 cases of epidural hematoma, all of which occurred in patients undergoing an epidural placement (97,925 patients). The rate of incidence of epidural hematoma was calculated as being 1:5000 [26]. Such a rare event as an epidural hematoma, with a calculated incidence of about 0.0002%, would require an extremely large randomized control trial to more precisely estimate the risk.

Evidence so far has revealed that the risk of an epidural hematoma following epidural catheter placement in patients undergoing cardiac surgery to be similar (0.0002%) to those patients undergoing non-cardiac surgery. Strict adherence to international protocols may have contributed to the lack of epidural hematoma formation in these groups of patients.

Benefits of Epidural Block and Its Role in Enhanced Recovery Protocols

In a review comparing neuraxial to general anesthesia and looking specifically at postoperative mortality, Rodgers and colleagues looked at 9559 patients undergoing general surgery, gynecologic surgery, and orthopedic surgery. There were 247 deaths within 1 month of surgery, and less than a third of those deaths were in the group that received neuraxial anesthesia [27]. The role of epidural anesthesia in anesthetic management is expanding due to the increase in enhanced recovery after surgery (ERAS) programs. These programs emphasize comprehensive perioperative management of the patient, including thorough preoperative education and planning, multimodal pain regimen intraoperatively to reduce the stress response to surgery, and early ambulation and aggressive therapy postoperatively. Although not all ERAS programs have shown a decrease in the length of stay, they have consistently shown a reduction in complications, earlier return of bowel function, and improved cardiopulmonary function. Multiple specialties are involved in the development of an ERAS program, including gynecology, urology, and colorectal surgery [28–30]. As the population continues to age, non-cardiac surgery is performed on patients with a multitude of diseases, most concerning of which are cardiovascular disease. The ability of epidural anesthesia to repeatedly demonstrate decreased morbidity and mortality when used either alone or as an adjunct makes it very beneficial and significant in many aspects.

Paravertebral and Intercostal Nerve Blocks

Paravertebral Block in Cardiac and Thoracic Surgery

Cardiac surgery patients are at particularly increased risk for the development of postoperative pulmonary complications related to changes in respiratory mechanics, a problem which is exacerbated by the intense pain [31]. Adequate pain management is imperative to “fast track” these patients, leading to early extubation and reducing the hospital length of stay and further complications. Analgesia for these patients can include a variety of management strategies including intercostal and thoracic paravertebral nerve blocks (PVB). Systemic reviews have established equivalence between thoracic epidural anesthesia (TEA) and PVB with respect to pain scores and morphine consumption [32, 33]. Compared to TEA, there is a reduced incidence of hypotension, urinary retention, pruritus, PONV, and respiratory complications with PVB [33, 34]. PVB are less likely to be associated with epidural hematoma formation following anticoagulation which is an important consideration for cardiac surgery. Contraindications to thoracic epidural may not preclude the placement of a PVB catheter.

Canto et al. conducted a prospective observational study to assess the use of bilateral paravertebral catheters in patients undergoing traditional median sternotomy, “on-pump” cases. They concluded that this technique is

feasible and safe, provides good hemodynamic stability and excellent analgesia, and allows for earlier extubation [28].

Minimally invasive cardiac surgery has become increasingly popular. This surgical approach often includes multiple lateral thoracotomy incisions. Since lateral thoracotomy is more painful than midline sternotomy, pain control is imperative to minimize complications and promote early recovery. A prospective study by Neuburger and colleagues compared general anesthesia alone to general plus a single-shot paravertebral block for patients undergoing robotic mitral valve repair. Those with PVB had significantly less postoperative pain and required less narcotic intraoperatively and postoperatively. They reported higher satisfaction with anesthesia but did not have shorter hospital stays compared to those without blocks [29]. A prospective randomized trial was also conducted to compare paravertebral blocks to thoracic epidurals in 41 patients undergoing minimally invasive direct coronary artery bypass surgery. They reported no significant difference between the two groups for pain scores and supplemental analgesic requirements. They noted hypotension and a backache in the epidural group and no adverse side effects in the paravertebral group [30].

Paravertebral and Intercostal Block in Cardiovascular Patients Undergoing Non-cardiac Surgery

As widespread use of postoperative opiate continues to be a problem relevant to the current practice of anesthesia, alternative methods of achieving adequate analgesia are vital. Pain management strategies which incorporate the use of regional anesthetic techniques are an appealing means of approaching this problem, especially in patients with cardiovascular or pulmonary disease which may be prone to the negative respiratory effects of opiates [35]. Paravertebral blocks and intercostal blocks have been used for a wide variety of cases including thoracic surgery, breast surgery, and upper abdominal surgery.

Thoracic surgery is associated with a significant reduction in functional residual capacity and vital capacity. Uncontrolled pain is a major contributing factor to the reduction in pulmonary capacities [32]. A 2008 systematic review by Joshi et al. considered the use of peripheral nerve blocks, specifically paravertebral catheters as well as single-shot injection intercostal nerve blocks for postoperative analgesia after thoracotomy. The authors concluded that continuous paravertebral blocks are an effective alternative to thoracic epidural anesthesia and that intercostal injections may be a suitable alternative as well (although limited by duration) [36].

More recent studies have investigated the role of liposomal bupivacaine, as a means for producing satisfactory prolonged effect from perioperative intercostal nerve block [37, 38]. These studies demonstrate that intercostal nerve block may be a safe and effective alternative to thoracic epidural anesthesia. It has been suggested that the use of liposomal bupivacaine and the extended duration of neural block may increase the use of intercostal nerve blocks for the management of acute postoperative pain after thoracotomy, especially when indwelling catheters may be contraindicated [34]. PVB also reportedly decrease the incidence of chronic post-thoracotomy pain [39].

Awake thoracic surgery has been described using an intercostal block as the sole anesthetic. The surgery was conducted via video-assisted thoracoscopy (VATS) using a single incision [40]. Thoracic paravertebral blocks have also been described as the sole anesthetic for VATS [41].

Paravertebral blocks have been used successfully for postoperative pain following the Nuss procedure for pectus excavatum repair. When retrospectively reviewing 20 patients, there was no statistically significant difference in pain scores or postoperative opioid consumption between those patients who had an epidural compared with the paravertebral group [42]. Loftus et al. conducted a retrospective cohort study of 137 patients who underwent either the Nuss or Ravitch procedure and found the hospital length of stay was shorter in both the intercostal and paravertebral groups when compared to those

who had a thoracic epidural. This reduced length of stay did not translate into a reduced total cost. Pain scores were higher in the intercostal and paravertebral groups at day 1 but equivalent by day 3 [43].

Paravertebral blocks have been used with tremendous success for breast surgery. They have been demonstrated to decrease postoperative opioid consumption and pain scores. Studies have also been done to look at the effect of paravertebral blocks on chronic pain following breast surgery. Kairaluoma et al. performed a prospective, randomized, placebo-controlled, and single-blinded study of 60 patients undergoing breast surgery for cancer and followed them for 12 months. They reported those who had PVB had a lower prevalence and severity of pain up to a year out. The PVB group reported less motion-related pain and less pain at rest [37]. pain and fewer symptoms in patients who received paravertebral blocks [38]. In a retrospective study of 132 patients undergoing unilateral and bilateral breast reconstruction, those with intercostal nerve blocks had significantly reduced the length of hospital stay and opiate consumption and increased cost saving per patient [44]. There has also been a retrospective analysis to suggest that paravertebral blocks for breast cancer surgery reduce the risk of recurrence and metastasis during the initial years of follow-up. To further investigate this conclusion, prospective trials are warranted [45].

Certain open upper abdominal surgeries have been successfully managed with intercostal nerve blocks perioperatively. After open cholecystectomy, they have been shown to provide pain control and improve respiratory function [46, 47]. In geriatric patients after open distal gastrectomy, they had reduced analgesic use and offered more stable hemodynamics when general anesthesia was combined with an intercostal block [48].

In these beneficial effects of paravertebral and intercostal nerve block on reducing perioperative opioid requirements, improvement in postoperative respiratory function and reduced stress response may prove beneficial in cardiac patients undergoing non-cardiac surgery.

Paravertebral and Intercostal Block for Acute Pain Management

The use of intercostal nerve blocks has been investigated in the treatment of a variety of pain conditions, ranging from acute traumatic chest pain to the treatment of chronic pain syndromes. In patients who have suffered acute rib fractures, the resulting detrimental effects on respiratory mechanics have been demonstrated, and pain management has a significant impact on patients' recovery [49]. A 2004 study by Osinowo et al. demonstrated that when intercostal nerve blocks were performed in patients with rib fractures, there is a significant improvement in respiratory mechanics as demonstrated by increased arterial oxygen saturation and peak expiratory flow rates [50]. Paravertebral blocks can also provide effective pain control for patients with multiple rib fractures. However, regional anesthesia has not been incorporated into the guidelines for pain management in these patients [51]. The reason cited is the lack of evidence supporting routine use, as well as the potential for complications including pneumothorax. The risk of a significant pneumothorax is reportedly less than 0.01% [52]. The use of ultrasound-guided techniques to aid regional anesthesia may increase the safety of intercostal nerve blocks, although more research needs to be completed to demonstrate this safety profile [53].

Intercostal nerve blocks have been proposed as a means of treating and diagnosing a variety of chronic pain conditions. They may be used as a means for predicting the success of intercostal nerve cryoablation as an interventional pain management strategy [54]. For patients with persistent pain after breast cancer surgery, neural block with intercostal nerve block may be a useful strategy to guide further surgical or ablative procedures [46].

Peripheral Nerve Block in Patients with Cardiac Disease Undergoing Non-cardiac Surgery

Cardiovascular disease poses a perioperative impact on anesthetic management, and in the age of multimodal analgesia, peripheral nerve blocks and peripheral nerve catheters have been shown to have reduced opioid consumption and have less hemodynamic instability during general anesthesia [47]. The overall poor health of patients with cardiovascular disease requires alternatives to the use of general anesthesia for access procedures include upper and lower extremity peripheral nerve blocks. Upper and lower extremity peripheral nerve blocks may have potential advantages when compared with general or neuraxial anesthesia such as avoidance of intubation, shorter hospital stays, reduced opioid consumption [55], and, with respects to neuraxial anesthesia, lack of concern about anticoagulation. Peripheral nerve blocks have few cardiovascular or pulmonary side effects. But there are risks associated with single-shot and continuous catheters [56].

Brachial plexus blocks can be utilized to provide anesthesia for the creation of new arteriovenous fistulae and other upper extremity surgeries in patients with end-stage renal disease or cardiac disease [57, 58].

Specific examples of the utility of upper extremity regional anesthesia are described in a case report of a cardiac patient with a Neer type III fracture of the surgical neck of the humerus. The patient had multiple valvular abnormalities such as severe aortic stenosis, mild aortic regurgitation, and mild tricuspid and mitral regurgitation. This patient had a five- to sevenfold increase in mortality for non-cardiac surgery with an aortic valve area of 0.7 cm^2 and a transvalvular gradient of 58 mmHg. Regional anesthesia via interscalene block with 30 mL of 0.5% ropivacaine was adequate for the procedure. Regional anesthesia provided the patient with optimal hemodynamics including normal sinus rhythm and adequate systemic vascular resistance and was able to avoid tachycardia and maintain a balance of myocardial oxygen demand [59].

Lower extremity nerve blocks are commonly utilized for lower extremity surgery. The combined femoral-sciatic nerve block is one of the most useful anesthetic procedures for lower limb surgeries where neuraxial anesthesia is contraindicated [60]. This can be used for lower limb surgeries without any major complications. It can be used in critically ill patients where both GA and central neuraxial block carries a high risk of mortality. There has been a successful coronary artery bypass grafting in awake patients using a combination of high thoracic epidural and femoral nerve block in a case series of 15 total patients undergoing off-pump coronary artery bypass grafting. There were 3 patients who required conversion to general anesthesia related to insufficient analgesia, need for cardiopulmonary bypass, and the third for profound respiratory movements. A single-shot femoral nerve block was used for saphenous vein harvesting, and this combined with the high thoracic epidural provides sufficient analgesia for off-pump coronary artery bypass grafting in a case report of 15 patients [61].

Summary

In summary, the literature demonstrates clear cardiovascular benefits with the use of different regional techniques for both cardiac and non-cardiac patients. The aging population and a lack of better understanding of therapeutics in the management of major diseases in our population resulting in older and sicker patients will continue to further challenge anesthesiologists not only intraoperatively but postoperatively. Short- and long-term benefits can be gained and appreciated through many regional techniques; therefore, anesthesiologists selecting regional techniques for carefully selected patient populations undergoing surgical interventions may assist in ensuring the best possible outcomes.

Case Study

A case of a 99-year-old patient using clopidogrel undergoing regional anesthesia for surgical treatment of hip fracture without complications was described by Brasileiro and Imbelloni [62].

Review Questions

1. Potential benefits of regional block in cardiac patients undergoing surgery include all of the following except:
   (a) More effective analgesia
   (b) Improvements in postoperative function
   (c) Alleviation of surgical stress
   (d) Reduction in incidence of wound infection
   (e) Decreased length of stay
2. Which of the following is true about the autonomic nervous system?
   (a) Parasympathetic autonomic outflow originates from the thoracolumbar spinal cord segments.
   (b) Sympathetic autonomic outflow originates from the cranial nerves (3,7,9,10) and sacral spinal cord segments.
   (c) Parasympathetic outflow originates from the cranial nerves (3,7,9,10) and sacral spinal cord segments.
   (d) Sympathetic outflow originates from the thoracolumbar spinal cord segments.
3. Benefits of bilateral paravertebral block over an epidural block for patients undergoing thoracic surgery include:
   (a) Decrease in postoperative opioid requirements
   (b) Decrease in intraoperative blood loss
   (c) Reduced incidence of hypotension
   (d) Decreased hospital length of stay
   (e) Decreased incidence of post-thoracotomy pain syndrome
4. Enhanced recovery after surgery protocols have consistently shown which of the following improvements in patient care:
   (a) Reduction in complications
   (b) Earlier return of bowel function
   (c) Improved cardiopulmonary function
   (d) Decreased hospital length of stay
5. Analgesic options for patients undergoing cardiac or thoracic surgery include all of the following except:
   (a) Epidural block
   (b) Spinal block
   (c) Paravertebral block
   (d) Intercostal nerve block
   (e) Peripheral nerve block

Answers:

1. d
2. c, d
3. c
4. a, b, c
5. e

References

1. N. Center for Health Statistics. Number of all-listed procedures for discharges from short-stay hospitals, by procedure category and age: United States, 2010; 2010.
2. Devereaux PJ, Sessler DI. Cardiac complications in patients undergoing major noncardiac surgery. N Engl J Med. 2015;373(23):2258–69.
3. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, Sugarbaker DJ, Donaldson MC, Poss R, Ho KK, Ludwig LE, Pedan A, Goldman L. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation. 1999;100(10):1043–9.
4. Desborough JP. The stress response to trauma and surgery. Br J Anaesth. 2000;85(1):109–17.
5. Welsby IJ, Bennett-Guerrero E, Atwell D, White WD, Newman MF, Smith PK, Mythen MG. The association of complication type with mortality and prolonged stay after cardiac surgery with cardiopulmonary bypass. Anesth Analg. 2002;94(5):1072–8.
6. Veering BT. Cardiovascular and pulmonary effects of epidural anaesthesia. Minerva Anestesiol. 2003;69(5):433–7.
7. Brull SJ, Greene NM. Zones of differential sensory block during extradural anaesthesia. Br J Anaesth. 1991;66(6):651–5.
8. Buggy DJ, Smith G. Epidural anaesthesia and analgesia: better outcome after major surgery?. Growing evidence suggests so. BMJ. 1999;319(7209):530–1.
9. Moraca RJ, Sheldon DG, Thirlby RC. The role of epidural anesthesia and analgesia in surgical practice. Ann Surg. 2003;238(5):663–73.

10. Wang J, Guo W, Wu Q, Zhang R, Fang J. Impact of combination epidural and general anesthesia on the long-term survival of gastric cancer patients: a retrospective study. Med Sci Monit. 2016;22:2379–85.
11. Kowalewski R, Seal D, Tang T, Prusinkiewicz C, Ha D. Neuraxial anesthesia for cardiac surgery: thoracic epidural and high spinal anesthesia - why is it different? HSR Proc Intensive Care Cardiovasc Anesth. 2011;3(1):25–8.
12. Cheng DC, Karski J, Peniston C, Asokumar B, Raveendran G, Carroll J, Nierenberg H, Roger S, Mickle D, Tong J, Zelovitsky J, David T, Sandler A. Morbidity outcome in early versus conventional tracheal extubation after coronary artery bypass grafting: a prospective randomized controlled trial. J Thorac Cardiovasc Surg. 1996;112(3):755–64.
13. Lee TWR, Grocott HP, Schwinn D, Jacobsohn E, Winnipeg High-Spinal Anesthesia Group. High spinal anesthesia for cardiac surgery: effects on beta-adrenergic receptor function, stress response, and hemodynamics. Anesthesiology. 2003;98(2):499–510.
14. Liu SS, Block BM, Wu CL. Effects of perioperative central neuraxial analgesia on outcome after coronary artery bypass surgery: a meta-analysis. Anesthesiology. 2004;101(1):153–61.
15. Richardson L, Dunning J, Hunter S. Is intrathecal morphine of benefit to patients undergoing cardiac surgery. Interact Cardiovasc Thorac Surg. 2008;8(1):117–22.
16. Fléron M-H, Weiskopf RB, Bertrand M, Mouren S, Eyraud D, Godet G, Riou B, Kieffer E, Coriat P. A comparison of intrathecal opioid and intravenous analgesia for the incidence of cardiovascular, respiratory, and renal complications after abdominal aortic surgery. Anesth. Analg. 2003;97(1):2–12.
17. Juelsgaard P, Sand NP, Felsby S, Dalsgaard J, Jakobsen KB, Brink O, Carlsson PS, Thygesen K. Perioperative myocardial ischaemia in patients undergoing surgery for fractured hip randomized to incremental spinal, single-dose spinal or general anaesthesia. Eur J Anaesthesiol. 1998;15(6):656–63.
18. Rashid RH, Shah AA, Shakoor A, Noordin S. Hip fracture surgery: does type of anesthesia matter? Biomed Res Int. 2013;2013:1–5.
19. Latham P, Zarate E, White PF, Bossard R, Shi C, Morse LS, Douning LK, Chi L. Fast-track cardiac anesthesia: a comparison of remifentanil plus intrathecal morphine with sufentanil in a desflurane-based anesthetic. J Cardiothorac Vasc Anesth. 2000;14(6):645–51.
20. Bigeleisen PE, Goehner N. Novel approaches in pain management in cardiac surgery. Curr Opin Anaesthesiol. 2015;28(1):89–94.
21. V. Svircevic, M. M. Passier, A. P. Nierich, D. van Dijk, C. J. Kalkman, and G. J. van der Heijden, "Epidural analgesia for cardiac surgery," in Cochrane Database of Systematic Reviews, no. 6, V. Svircevic, Ed. Chichester: Wiley, Ltd, 2013, p. CD006715.
22. Zhang S, Wu X, Guo H, Ma L. Thoracic epidural anesthesia improves outcomes in patients undergoing cardiac surgery: meta-analysis of randomized controlled trials. Eur J Med Res. 2015;20(1):25.
23. Porizka M, Koudelkova K, Kopecky P, Porizkova H, Dohnalova A, Kunstyr J. High thoracic anesthesia offers no major benefit over general anesthesia in on-pump cardiac surgery patients: a retrospective study. Springerplus. 2016;5(1):799.
24. Caputo M, Alwair H, Rogers CA, Pike K, Cohen A, Monk C, Tomkins S, Ryder I, Moscariello C, Lucchetti V, Angelini GD. Thoracic epidural anesthesia improves early outcomes in patients undergoing off-pump coronary artery bypass surgery. Anesthesiology. 2011;114(2):380–90.
25. Guay J, Kopp S. Epidural pain relief versus systemic opioid-based pain relief for abdominal aortic surgery. In Guay J. (ed.) Cochrane database of systematic reviews. 1, Chichester: Wiley; 2016.
26. Bardia A, Sood A, Mahmood F, Orhurhu V, Mueller A, Montealegre-Gallegos M, Shnider MR, Ultee KHJ, Schermerhorn ML, Matyal R. Combined epidural-general anesthesia vs general anesthesia alone for elective abdominal aortic aneurysm repair. JAMA Surg. 2016;151(12):1116.
27. Landoni G, Isella F, Greco M, Zangrillo A, Royse CF. Benefits and risks of epidural analgesia in cardiac surgery. Br J Anaesth. 2015;115(1):25–32.
28. Pöpping DM, Elia N, Van Aken HK, Marret E, Schug SA, Kranke P, Wenk M, Tramèr MR. Impact of epidural analgesia on mortality and morbidity after surgery. Ann Surg. 2014;259(6):1056–67.
29. Rodgers A, Walker N, Schug S, McKee A, Kehlet H, van Zundert A, Sage D, Futter M, Saville G, Clark T, MacMahon S. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomised trials. BMJ. 2000;321(7275):1493.
30. Poon JTC, Fan JKM, Lo OSH, Law WL. Enhanced recovery program in laparoscopic colectomy for cancer. Int J Color Dis. 2011;26(1):71–7.
31. Melnyk M, Casey RG, Black P, Koupparis AJ. Enhanced recovery after surgery (ERAS) protocols: time to change practice? Can Urol Assoc J. 2011;5(5):342–8.
32. de Groot JJ, Maessen JM, Slangen BF, Winkens B, Dirksen CD, van der Weijden T. A stepped strategy that aims at the nationwide implementation of the Enhanced Recovery After Surgery programme in major gynaecological surgery: study protocol of a cluster randomised controlled trial. Implement Sci. 2015;10(1):106.
33. Gerner P. Postthoracotomy pain management problems. Anesthesiol Clin. 2008;26(2):355–67.
34. Davies RG, Myles PS, Graham JM. A comparison of the analgesic efficacy and side-effects of paravertebral vs epidural blockade for thoracotomy--a systematic review and meta-analysis of randomized trials. Br J Anaesth. 2006;96(4):418–26.
35. Joshi GP, Bonnet F, Shah R, Wilkinson RC, Camu F, Fischer B, Neugebauer EAM, Rawal N, Schug SA, Simanski C, Kehlet H. A systematic review of

randomized trials evaluating regional techniques for Postthoracotomy analgesia. Anesth Analg. 2008;107(3):1026–40.
36. Daly DJ, Myles PS. Update on the role of paravertebral blocks for thoracic surgery: are they worth it? Curr Opin Anaesthesiol. 2009;22(1):38–43.
37. Cantó M, Sánchez MJ, Casas MA, Bataller ML. Bilateral paravertebral blockade for conventional cardiac surgery. Anaesthesia. 2003;58(4):365–70.
38. Neuburger PJ, Ngai JY, Chacon MM, Luria B, Manrique-Espinel AM, Kline RP, Grossi EA, Loulmet DF. A prospective randomized study of paravertebral blockade in patients undergoing robotic mitral valve repair. J Cardiothorac Vasc Anesth. 2015;29(4):930–6.
39. Dhole S, Mehta Y, Saxena H, Juneja R, Trehan N. Comparison of continuous thoracic epidural and paravertebral blocks for postoperative analgesia after minimally invasive direct coronary artery bypass surgery. J Cardiothorac Vasc Anesth. 2001;15(3):288–92.
40. Lee LA, Caplan RA, Stephens LS, Posner KL, Terman GW, Voepel-Lewis T, Domino KB. Postoperative opioid-induced respiratory depression: a closed claims analysis. Anesthesiology. 2015;122(3):659–65.
41. Gosselink R, Schrever K, Cops P, Witvrouwen H, De Leyn P, Troosters T, Lerut A, Deneffe G, Decramer M. Incentive spirometry does not enhance recovery after thoracic surgery. Crit Care Med. 2000;28(3):679–83.
42. Rice DC, Cata JP, Mena GE, Rodriguez-Restrepo A, Correa AM, Mehran RJ. Posterior intercostal nerve block with liposomal bupivacaine: an alternative to thoracic epidural analgesia. Ann Thorac Surg. 2015;99(6):1953–60.
43. Khalil KG, Boutrous ML, Irani AD, Miller CC, Pawelek TR, Estrera AL, Safi HJ. Operative intercostal nerve blocks with long-acting bupivacaine liposome for pain control after thoracotomy. Ann Thorac Surg. 2015;100(6):2013–8.
44. Saby A, Swaminathan K, Pangarkar S, Tribuzio B. Alleviating thoracotomy pain with intercostal liposomal bupivacaine: a case report. PM&R. 2016;8(11):1119–22.
45. Romero A, Garcia JEL, Joshi GP. The state of the art in preventing Postthoracotomy pain. Semin Thorac Cardiovasc Surg. 2013;25(2):116–24.
46. Piccioni F, Langer M, Fumagalli L, Haeusler E, Conti B, Previtali P. Thoracic paravertebral anaesthesia for awake video-assisted thoracoscopic surgery daily. Anaesthesia. 2010;65(12):1221–4.
47. Hall Burton DM, Boretsky KR. A comparison of paravertebral nerve block catheters and thoracic epidural catheters for postoperative analgesia following the Nuss procedure for pectus excavatum repair. Pediatr Anesth. 2014;24(5):516–20.
48. Loftus PD, Elder CT, Russell KW, Spanos SP, Barnhart DC, Scaife ER, Skarda DE, Rollins MD, Meyers RL. Paravertebral regional blocks decrease length of stay following surgery for pectus excavatum in children. J Pediatr Surg. 2016;51(1):149–53.
49. Kairaluoma PM, Bachmann MS, Rosenberg PH, Pere PJ. Preincisional paravertebral block reduces the prevalence of chronic pain after breast surgery. Anesth Analg. 2006;103(3):703–8.
50. Karmakar MK, Samy W, Li JW, Lee A, Chan WC, Chen PP, Ho AM-H. Thoracic paravertebral block and its effects on chronic pain and health-related quality of life after modified radical mastectomy. Reg Anesth Pain Med. 2014;39(4):289–98.
51. Shah A, Rowlands M, Krishnan N, Patel A, Ott-Young A. Thoracic intercostal nerve blocks reduce opioid consumption and length of stay in patients undergoing implant-based breast reconstruction. Plast Reconstr Surg. 2015;136(5):584e–91e.
52. Exadaktylos AK, Buggy DJ, Moriarty DC, Mascha E, Sessler DI. Can anesthetic technique for primary breast cancer surgery affect recurrence or metastasis? Anesthesiology. 2006;105(4):660–4.
53. Murphy DF. Continuous intercostal nerve blockade for pain relief following cholecystectomy. Br J Anaesth. 1983;55(6):521–4.
54. Pourseidi B, Khorram-Manesh A. Effect of intercostals neural blockade with Marcaine (bupivacaine) on postoperative pain after laparoscopic cholecystectomy. Surg Endosc. 2007;21(9):1557–9.
55. Chen J, Lin J, Chen X, Lin C. Efficacy of an intercostal nerve block administered with general anesthesia in elderly patients undergoing distal gastrectomy. Clin Invest Med. 2015;38(6):E351–7.
56. Easter A. Management of patients with multiple rib fractures. Am J Crit Care. 2001;10(5):320–9.
57. Galvagno SM, Smith CE, Varon AJ, Hasenboehler EA, Sultan S, Shaefer G, K. B. To, Fox AD, Alley DER, Ditillo M, Joseph BA, Robinson BRH, Haut ER. Pain management for blunt thoracic trauma: a joint practice management guideline from the eastern Association for the Surgery of trauma and trauma anesthesiology society. J Trauma Acute Care Surg. 2016;81(5):936–51.
58. Moore DC, Bridenbaugh LD. Intercostal nerve block in 4333 patients: indications, technique, and complications. Anesth Analg. 1962;41:1–11.
59. Abrahams M, Derby R, Horn J-L. Update on ultrasound for Truncal blocks. Reg Anesth Pain Med. 2016;41(2):275–88.
60. Hingorani AP, Ascher E, Gupta P, Alam S, Marks N, Schutzer RW, Multyala M, Multyala M, Shiferson A, Yorkovich W, Jacob T, Salles-Cunha S. Regional anesthesia: preferred technique for venodilatation in the creation of upper extremity arteriovenous fistulae. Vascular. 14(1):23–6.
61. Flaherty J, Horn J-L, Derby R. Regional anesthesia for vascular surgery. Anesthesiol Clin. 2014;32(3):639–59.
62. Brasileiro B, Imbelloni LE. Regional anesthesia in patients of aged 99 years in Clopidogrel use. Anesth Essays Res. 2017;11(1):257–9. https://doi.org/10.4103/0259-1162.186869.

31 Regional Anesthesia in the Patient with Preexisting Neurological Disease

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Introduction

Regional anesthesia has always provided benefits for patients undergoing surgical procedures. However, in patients with preexisting neurological diseases, there has been controversy surrounding whether regional anesthesia is beneficial or harmful. Using regional anesthesia with these individuals often places them at greater risk for neurological injury, and predisposes the patient to worsening disease processes, which can potentially be debilitating for the patient [1]. There are a wide range of causes related to postoperative neurologic deficits that may be due to the surgical trauma, neural ischemia, prolonged labor, local anesthetic effect, tourniquet pressure, and improper positioning [2]. Furthermore, a secondary insult may worsen or increase neurological injury further [3]. Consequently, many practitioners avoid regional anesthetics in these patients, and they are often treated conservatively. To date, there is still a paucity of data that supports regional anesthesia in patients with a preexisting neurologic condition due to the small number of documented regional anesthesia performed on these patients. The decision to proceed with regional anesthesia is dependent on the physician's comfort level, understanding the preexisting disease process and the patient's disease severity. It is equally important for providers to recognize risk factors for potential compromise and worsening of the preexisting disease with perioperative nerve injury. The goal of this chapter is to summarize the evidence to date in order to guide physicians in their decision-making and to provide the best care possible to patients with preexisting neurologic conditions undergoing regional anesthesia.

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Multiple Sclerosis

Multiple sclerosis (MS) is a rare immune-mediated degenerative demyelinating disorder of the central nervous system (CNS) that manifests clinically as periodic attacks of varied neurologic symptoms that may eventually progress to fixed neurologic deficits. It is described as a chronic degenerative disease that causes demyelination in the spinal cord and brain. The etiology of MS is not fully understood, but multiple factors may be involved. MS has a genetic predilection particularly with afflicted first-degree relatives with growing evidence that MS most likely involves

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_31

the T cells. There is a possibility that T cells in the peripheral blood become stimulated and attack foreign antigens while simultaneously attacking brain proteins that share similar molecular region of the antigen. It has also been associated with environmental factors and exposure to bacterial and viral agents. Lastly, there is a correlation of low vitamin D levels with MS [4]. Demyelination and glial scarring are characteristics of MS and are consequences of the inflammatory processes which subsequently may resemble a local anesthetic blockade, i.e., conduction blockade occurs along the neural pathways wherein the sodium channels are affected [5]. The demyelination results in "waxing and waning" of MS symptoms that are attributable to the fluctuating conduction blockade which is characteristic of this disease.

There are three main types of MS and clinically present as:

- Relapsing-remitting (RRMS): Symptoms are episodic with periods of remissions.
- Secondary progressive (SPMS): Progressive neurologic deterioration with remissions.
- Primary progressive (PPMS): Progressive neurologic deterioration without remissions.

MS patients present as a challenge to anesthesiologists. Regardless of the anesthetic technique, MS patients may have worsening neurologic function postoperatively and the mechanism remains ambiguous. Certain factors have been shown to predispose MS patients to neurologic deterioration, including emotional stress, infection, and hyperpyrexia [6]. Current literature is limited mostly to anecdotal case reports which describe the use of general anesthesia (GA), spinal anesthesia, and epidural anesthesia with low concentrations of local anesthetics which have shown to be safe with MS patients [4, 5]. With respect to spinal anesthesia, there is some evidence that has shown new or worsening neurologic symptoms after spinal anesthetic blocks [3, 7]. Peripheral nerve blocks have proven to be relatively safe for MS patients; however one case report did show severe brachial plexopathy following an interscalene block for shoulder surgery. The mechanism of injury was unclear though given the patient's history of peripheral neuropathy and underlying MS, this may have led to the development of peripheral autoimmune injury leading to brachial neuritis [8]. Recent studies have shown that there is a 5–47% incidence of peripheral demyelination that may occur as a result of MS [9, 10]. Finucane and colleagues showed that demyelination may lead to prolongation of local anesthetic effects. They presented a case report in which a patient underwent a paravertebral nerve block with an extended duration of neural blockade that may have been related to an abnormal uptake of local anesthetics into the spinal cord in the presence of demyelination [11].

In conclusion, there is no strong data that supports or refutes the use of regional anesthesia in patients with MS. Patient risks and benefits should be clearly explained, and patients must be informed of the potential risk of developing new diseases or worsening of their existing disease process after the use of regional anesthesia [3, 12].

Case Study

A 21-year-old woman, 42 weeks age of gestation, G1P0, with a history of MS presented in labor. The patient had a history of lower extremity limb weakness that presented as relapsing-remitting type with movement-induced muscle spasms. Her physical and neurologic examinations were normal other than decreased vision in both eyes. The patient had requested and received epidural analgesia for labor. During labor the baby started to show persistent late decelerations, and the patient required an emergent caesarian section. The patient was consented for a spinal anesthetic and it was performed without difficulty. The medications administered neuraxially were 1.6 ml of 0.75% bupivacaine with 50 mcg of fentanyl. The delivery was uneventful; however, the patient did present with persistent leg weakness that lasted for 12 h. These symptoms were attributed to potential abnormal uptake of local anesthetic likely secondary to the presence of demyelination within the spinal cord.

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) or Lou Gehrig's disease is a rapidly progressive, a fatal degenerative neurological disease involving the upper motor, lower motor, and motor brainstem neurons. The progressive degeneration of motor neurons causes muscular weakness, atrophy, fasciculations, spasticity, and hyperreflexia. The etiology of the disease is unknown, but it afflicts males between 40 and 60 years of age. Typically, death results 3–5 years after diagnosis, generally from myocardial or respiratory failure.

A concerning problem with ALS is general anesthesia; these patients may have extreme hemodynamic responses with progressive weakness, muscle atrophy, and risk for aspiration and ventilator dependence [13]. General anesthesia carries an increased risk of respiratory complications and may result in prolonged intubation. With mechanical ventilation and inhalational anesthetics, these patients could experience increases in loss of muscle tone and worsening respiratory functions. Therefore, weaning these patients from a ventilator could potentially be difficult. ALS patients have better outcomes when general anesthesia can be avoided entirely; therefore, regional anesthesia should be considered whenever possible. Epidural anesthesia may be a reasonable choice in selected cases but has the potential of impairing respiratory function by producing intercostal muscle weakness. There have been several cases reported of successful epidural anesthesia in patients suffering from ALS [14]. Thampi et al. reported a case in which lumbar epidural anesthesia was used successfully in a patient without any complications by avoiding endotracheal intubation and potential ventilator complications [15]. Even though epidural anesthesia has been successfully described in patients with ALS [14], one should be cognizant of the possibility of a high epidural or spinal block [15]. Regardless of the technique, ALS patients are still susceptible to postoperative neurologic deterioration. The decision in proceeding with regional anesthesia should be based on a thorough discussion of risks and benefits for each individual patient [3].

Post-Polio Syndrome

Polio or poliomyelitis is a crippling disease caused by the poliovirus. The virus is extremely infectious and can be spread from person to person contact via the fecal-oral route, often spread through contaminated water or food. The virus destroys nerve cells within the spinal cord specifically attacking the anterior horn motor neurons leading to muscle wasting and potential limb paralysis. Viral infection is easily preventable with the vaccine for poliovirus. The injectable vaccine was first developed by Salk in 1955 and the oral vaccine by Sabin in 1961. Since the development of the polio vaccines, its occurrence is extremely rare in the United States, and in 2015 there were zero cases reported. Currently, new cases of polio are mostly restricted to Africa, Southeast Asia, and the Middle East [16].

Post-polio syndrome (PPS) is a condition that affects polio survivors' years after recovery from an initial acute attack of the poliomyelitis virus. The onset of symptoms may occur up to 30 years after the initial episode of poliomyelitis. PPS is the most prevalent progressive neuromuscular disease reported in North America [17] with a greater rate in women than in men [18]. The most common symptoms include slowly progressive muscle weakness, fatigue, and gradual muscle atrophy starting in the muscles previously affected by the virus. Although rare, life-threatening symptoms may occur, with most symptoms significantly interfering with a person's ability to function independently [19].

Preoperatively, a thorough history of the patient's previous poliomyelitis illness, patient's age at the time of illness, severity, and the amount of recovery are all important and helpful in determining which patients are more likely to exacerbate their PPS symptoms. Other important aspects to note during preoperative assessments include the presence of chronic pain syndromes, contractures, spinal deformities, detailed respiratory evaluation, a history of sleep apnea or hypoventilation syndrome, the presence of dysphagia, and reflux disease [20].

It is important to consider if regional anesthesia will be utilized preoperatively. Patients with PPS have fewer motor neurons than patients without PPS, and it is difficult to know if the remaining motor neurons are more heavily affected by the local anesthetics. The largest series ($n = 79$) of PPS patients to receive neuraxial anesthesia documented no worsening of symptoms. However, lack of data on these patients suggests that the risk and benefit of a neuraxial technique should be balanced against that of general anesthesia [12]. Thus, the use of general versus regional anesthesia should be evaluated on a case-by-case basis, based on each individual patient.

To avoid confounding factors for further neurologic insults, the patient should be positioned comfortably and limbs supported with padding as necessary. Also, the use of warming devices such as blankets, head covers, forced-air warming blankets, and warm ambient air should be used to combat cold intolerance [21].

The lack of significant clinical data relating to PPS and regional anesthesia prevents clear recommendations from being made regarding the safety of neuraxial anesthesia or peripheral nerve blockade with PPS [12]. Caution with the use of sedating medications and opioids is advised due to the inherent sensitivity of PPS patients when it comes to these medications.

Peripheral Nervous System Disorders

These disorders comprise neurological disease states that involve the cell body, axon, neuromuscular junction, and myelin sheath. These are all in the periphery and do not involve the brain or spinal cord. These patients suffer from the disruption of axons with distal degeneration or segmental demyelination caused by Schwann cell degeneration which will result in peripheral neuropathies [20, 22]. These can be caused by genetics, infections, malignancy, toxins, or environmental and metabolic derangements. Such conditions typically start distally and spread in a proximal fashion leading to the classic "glove and stocking" distribution of decreased sensation, weakness, and diminished reflexes. Diagnosis is often supported with screening for metabolic derangements, serology, viral titers, and autoimmune assays. Electromyogram and nerve conduction velocity studies will often reveal evidence of denervation and decreased nerve conduction velocity [20].

Hereditary Peripheral Neuropathy

There is a whole host of inherited neuropathies with a wide range of genotypes presenting with subclinical, mild, and severe debilitating neurologic conditions. The clinical presentation can often appear insidious in onset and prolonged, ranging for years and even decades, each with its own pathophysiologic symptomatology and prognosis. The most common of these hereditary neuropathies are a group of disorders known as Charcot-Marie-Tooth (CMT) disease. Patient presentation with CMT often includes severe motor weakness, muscle wasting within the distal lower extremities, gait abnormalities, loss of tendon reflexes, and numbness within the lower limbs. There are two reported cases of patients with Charcot-Marie-Tooth (CMT) disease who underwent regional anesthesia followed by a delayed recovery from local anesthetics. It was concluded that in both cases, the use of higher concentration of local anesthetics likely caused the prolonged effect [20, 23, 24]. Due to the rarity of these disease states, definitive recommendations on the conduct of safe regional anesthesia cannot be made due to a lack of clinical data. Utmost caution and minimizing other surgical and anesthetic risk factors for perioperative nerve injury should be employed if contemplating the use of regional anesthesia on this subset of patients [3].

Acquired Peripheral Neuropathy

Diabetic Polyneuropathy (DPN)

Diabetes mellitus is associated with several types of neuropathy, the most common of which is distal symmetric sensorimotor polyneuropathy. Though uncommon, mononeuropathy causing transient pain and weakness in an isolated nerve

distribution can occur. Most of these patients are often asymptomatic, but the majority will have evidence of abnormal nerve conduction studies [25]. Patients frequently present with lower extremity paresthesia, weakness, and sensory changes that are commonly worse at night. These patients may also have an underlying autonomic neuropathy that is associated with increased risk of intraoperative dynamic instability and increased risk of cardiac arrest [20, 25].

The mechanism of diabetic polyneuropathy (DPN) is poorly understood and likely multifactorial [3]. Some proposed mechanisms for axonal degeneration include sorbitol deposition in the nerve secondary to high glucose levels. The extra glucose is shifted into a polyol pathway, thus leading to formation of sorbitol. Other mechanisms include local tissue ischemia, abnormal tissue repair, oxidative stress, and disturbed biochemical processes leading to mitochondrial dysfunction [26, 27]. There is a growing body of evidence in animal studies that support the findings that diabetic nerve fibers are more susceptible to local anesthetic neurotoxicity. Chronic ischemic hypoxia of the nerves and decreased perineural blood flow may also explain the predilection of diabetic nerve in comparison to nondiabetic nerves from local anesthetics especially if given in large doses. In their studies, Williams and Kalichman concluded that preexisting subclinical neuropathy may lead to substantial prolongation of the duration of blockade in diabetic rats [12, 28, 29]. However, it is still unclear whether these findings are clinically relevant in humans. In a single institutional study, Hebl et al. showed that diabetic patients who had spinal anesthesia were not a greater risk than the general population with neuraxial anesthesia. The authors concluded that the risk with neuraxial anesthesia in patients with preexisting CNS disorders may not be as frequent as once thought and should not be considered an absolute contraindication within this patient population [7]. Diabetic patients have also shown decrease sensitivity to electrical stimulation and may pose a risk for intraneural needle puncture when a nerve stimulator is used for nerve localization. With the advent and use of ultrasound for peripheral nerve blocks, nerve injury may lessen and prove beneficial for neuraxial or peripheral nerve blockade. The use of ultrasound may improve needle visualization and prevent unintentional nerve contact and reduce the amount local anesthetic injectate needed in diabetic patients [12, 30]. Like any other peripheral neuropathies, the use of regional anesthesia in these patients should depend on a thorough analysis of its risks and benefits. With an underlying autonomic instability, and potential cardiovascular complications, diabetic patients would substantially benefit from regional anesthesia. It has been reported that the use of spinal and epidural anesthesia prior to surgical incision inhibits the surgical stress response providing better glycemic control [22]. Conversely, it is also apparent that these patients potentially may develop an exaggerated response to local anesthetics causing hypotension and possible myocardial depression. Therefore, it is advocated to avoid large doses of local anesthetic for neuraxial anesthesia [20, 31]. Diabetic patients also present with a risk of double-crush syndrome; strategies to avoid double-crush injury with patients include avoidance of adjuvant epinephrine [12] and perhaps use of ultrasound guidance to facilitate real-time visualization of the needle.

Chemotherapy-Induced Neuropathy

The widespread use of chemotherapeutic agents for cancer patients places this population at a risk for neuropathy. Even though the incidence of perioperative neuropathy is uncommon, it is more prevalent in this subset of patients, and it can potentially be catastrophic and debilitating. The incidence of neuropathy with cancer patients receiving chemotherapy is about 30–40%, and the toxicity is dependent on the chemotherapeutic agent, duration given, and total dose received [32].

When performing regional anesthesia on these patients, physicians' caution should be taken with respect to medications used for regional anesthetic. Local anesthetic toxicity is a possibility and can potentially worsen patient symptoms. Many of these patients have a subclinical neuropathy and may manifest as neurologic insult after regional anesthesia is administered. In a single-center cohort study performed by Abcejo

et al., they reported an overall 2.2% incidence of perioperative nerve injury in patients with previous systemic chemotherapy undergoing upper extremity surgery with an associated peripheral nerve block [33]. This data from Abcejo and colleagues was not different from the baseline risk established in larger cohort studies [34]. Chemotherapy-induced peripheral neuropathy is not an uncommon side effect, and the mechanism of neuropathy is still not clearly delineated. However, it has been proposed that possible causes may be direct toxic effects on the nervous system, damage to microtubules, or mitochondrial disruption or indirectly due to drug-induced metabolic derangements [3, 33].

Case Study

A 65-year-old male with history of diabetic neuropathy, multiple allergies, and difficulty emerging from anesthesia was scheduled for open reduction and internal fixation of the left ankle. After a thorough review of the patient's history, a lumbar spinal anesthetic was performed at the L4–L5 level. The medications delivered into the subarachnoid space consisted of 3 ml of 0.75% bupivacaine and 50 mcg of fentanyl. A successful sensory and motor block was achieved to the T12 dermatome level. The surgery was uneventful; however 8 hours postoperatively, the patient regained all motor function but complained of persistent numbness and tingling over the bilateral lower extremities. The patient was referred to a neurologist, whereby further testing was done and included magnetic resonance imaging (MRI) and nerve conduction studies. The MRI revealed normal results with no evidence of cord or nerve root compression. Conversely, nerve conduction velocity (NCV) and electromyography (EMG) studies showed distal symmetrical sensorimotor polyneuropathy consistent with diabetic neuropathy. The patient had persistent paresthesia that was considered to be worsening diabetic neuropathy likely secondary to the local spinal anesthetics used.

Inflammatory Neuropathies

Guillain-Barre Syndrome

Guillain-Barre syndrome (GBS) is an acute, inflammatory, demyelinating disease that occurs as an autoimmune response following a gastrointestinal or respiratory viral infection. It is characterized by acute onset, rapid progression, symmetric muscle weakness, and unstable ambulation, with hyporeflexia and/or areflexia. Weakness is usually predominantly distal, and it frequently presents as ascending paralysis with weakness in the legs spreading to the upper limbs and face [35].

The annual incidence of GBS is reported to be 1.2–2.3 per 100,000. Most studies have found that incidence increases linearly with age and men are about 1.5 times more likely to be affected than women [36]. The mechanism of GBS is believed to be an inflammatory neuropathy due to cross-reactivity between neural antigens and antibodies induced by specific infections [37]. Campylobacter jejuni is the most widely reported infection and is found in 25–50% of adult patients. Other infections include Cytomegalovirus, Epstein-Barr virus, measles, influenza A virus, Mycoplasma pneumoniae, as well as enterovirus D68 and Zika virus [35]. Many reports have also documented the occurrence of GBS shortly after vaccinations, operations, or stressful events [36].

The main clinical feature associated with GBS is rapidly progressive weakness. Most commonly the disease progression reaches its maximum phase of weakness within 2 weeks. However, a plateau phase (ranging from days to weeks or months) can occur in patients in varying duration. Despite treatment, 20% of severely affected patients are unable to walk after 6 months, and many patients remain otherwise disabled or severely weakened [36]. Guillain-Barre symptoms are usually treated with supportive and symptomatic management. These patients may also present with autonomic dysfunction making anesthetic management challenging. Plasmapheresis and intravenous immunoglobulin are usually efficacious for symptoms. The majority of patients will have resolution of most symptoms,

while 20% will develop persistent residual neurologic deficits [20].

Most reports of successful neuraxial anesthesia in GBS are in the obstetric population that have had regional neuraxial anesthesia for labor analgesia and caesarian section [3, 38–40]. However, there are concerns about local anesthetics interacting with peripheral myelin or direct nerve trauma [3] or that neurologic symptoms can worsen after neuraxial anesthesia [41]. Some studies suggest that regional anesthesia may precipitate or reactivate GBS within hours to weeks after surgery [3, 42–44]. There are some case reports that utilized epidural opioids without any complication to control acute painful paresthesias [45, 46]. Since local anesthetics can be neurotoxic, opioids can then be used as an alternative. With this patient population potentially developing autonomic dysfunction, caution should be practiced with regional techniques because of an exaggerated response to indirect acting vasopressors such as ephedrine [20].

The decision to perform neuraxial anesthesia in patients with GBS requires individual consideration of risks versus benefits. Literature has shown an equal number of cases with and without neurologic deterioration after neuraxial blockade, with no clear evidence in favor of or against it [47]. Thus, careful evaluation and documentation of a patient's baseline status, including thorough discussion of the potential risks as well as benefits of regional anesthesia, should be foremost on patients with GBS.

Spinal Canal Pathology

As the population growth continues, spinal canal pathology has become more prevalent secondary to aging and multiple pathologies such as spinal canal stenosis, lumbar degenerative disk disease, scoliosis, spondylosis, and history of prior spine surgery. Many of these patients may require surgical interventions and possibly require neuraxial anesthesia for primary anesthetic or postoperative pain control. This specific population of patients can be challenging and at times not good candidates for regional neuraxial anesthetics secondary to a presumed increased risk for neurologic complications. The mechanism of injury can be multifactorial and include ischemic, mechanical trauma, and/or local anesthetic toxicity. In particular, patients who have had previous back surgery may be susceptible for further neurologic injury and block failure, and the administration of neuraxial anesthesia may be technically challenging due to anatomical changes or scarring of the epidural space or central canal. In patients with spinal stenosis, the preexisting narrowing of the central spinal canal may limit the use of local anesthetic volume or even placement of a small-diameter epidural catheter without creating considerable pressure on the spinal cord. This pressure on the spinal cord could potentially lead to mechanical compression and the potential for a devastating injury. Following back surgery, many of the posterior elements of the spine may have been violated or obliterated. Some patients may have instrumentation, fusions, and/or bone grafting contributing to increase incidence of block failure and inability to access epidural space properly [3, 12]. Adhesions often develop in these patients, leading to patchy or incomplete neuraxial blocks. The authors highly recommend that when performing neuraxial anesthesia, physicians should avoid any segments of the spine that have had surgical treatments. Not only would it be difficult to access the epidural space in these patients, there could be a loculation effect with injectable medications leading to cord compression. There have been reported cases of postsurgical fusion patients receiving neuraxial anesthesia in previously fused segments that developed postoperative cauda equina syndrome and paresis after uneventful spinal anesthesia [48].

Several case series have described the successful use of epidural anesthesia with catheter placement. However, false loss of resistance, subsequent dural puncture, inadequate epidural anesthesia, and traumatic needle placement are

common problems encountered that have been described [49–51]. Hebl and colleagues performed a retrospective analysis on 937 patients undergoing surgery with a neuraxial blockade with a previous history of spinal surgery, lumbar spinal stenosis, lumbar radiculopathy, and peripheral neuropathy. The goal of this study was to determine if patients with spinal pathology are at increased risk of neurological complications after neuraxial anesthesia. The authors concluded that patients with preexisting spinal canal pathology have a greater occurrence of neurologic complications after neuraxial blockade than that previously reported for patients without such underlying pathology. The authors also could not determine whether the higher incidence of neurologic injury was due to the surgical procedure, the anesthetic technique, the natural history of spinal pathology, or a combination of each [48].

In the 2015 ASRA guidelines [12], it stated that although previous spinal surgery should not be considered, a contraindication to neuraxial or interventional pain medicine techniques, extreme consideration should be given prior to the performance of the regional anesthetic by having preprocedure imaging that will help recognize aberrant anatomy, existing spine deformity, and/or the presence of surgical implants. More importantly, history and physical examination with thorough neurologic examination should not be overlooked.

Case Study

A 65-year-old male with a past surgical history significant for L4–L5 and L5–S1 laminectomy presented for a right total hip arthroplasty. After reviewing the patient's medical and surgical history, it was decided that a regional neuraxial anesthetic would be performed. Given the patient's surgical history, an L3–L4 lumbar epidural was attempted above the level of the previous surgery. The attempt yielded an inadvertent dural tap which was subsequently used to advance an epidural catheter within the intrathecal space for intraoperative and postoperative pain control. Subsequently, the catheter was employed, and 0.125% ropivacaine was infused at a rate of 6 ml/h. The patient had an unremarkable perioperative course other than a prolonged block for 6 h after removal of the catheter on the third day. The patient continued to have fatigue with muscle stiffness of the lower back and occasionally bilateral lower extremity and an area of dysesthesia on the right thigh. An MRI of the lumbar spine taken after 3 months was interpreted as having clumped nerve roots posteriorly in the thecal sac consistent with arachnoiditis. It was surmised that the administration of the local anesthetic led to a neurotoxic effect and may have contributed to the persistent dysesthesia.

Spinal Cord Injury

There are two different stages of spinal cord injury: acute and chronic. Initial injuries occur over a 1- to 3-week period associated with flaccid paralysis, loss of temperature regulation, and loss of spinal cord reflexes below the injury. This is commonly referred to as "spinal shock." During this initial phase, there is hemodynamic instability, and regional anesthesia is not used due to the nature of the evolving neurologic injury. On the other hand, the chronic stage presents with spasticity and return of spinal and autonomic reflexes below the level of the injury.

Autonomic hyperreflexia (AH) is a dangerous clinical syndrome that develops as a sequela to spinal cord injury. Approximately 85% of spinal cord injury patients are at risk for developing hyperreflexia symptoms if the lesion involves the T6 spinal cord level or higher [20]. This injury can produce acute uncontrolled hypertension with the introduction of a noxious stimulus below the level of the lesion, leading to a peripheral sympathetic response producing vasoconstriction below the injured level. Although a noxious trigger is the most common cause, it can also occur via non-noxious stimuli. The leading trigger that stimulates AH is typically distention of the urinary bladder or colon. The pathophysiology involves an imbalanced

reflex sympathetic discharge that involves uninhibited spinal reflexes that lead to hypertension and reflex bradycardia. Patients can present with intracranial hemorrhages, seizures, coma, myocardial ischemia, and death. Early recognition, prevention, and aggressive management should be initiated to avoid debilitating consequences. Regional anesthesia has been used successfully in patients with autonomic hyperreflexia. Particularly, spinal anesthesia has shown to inhibit spinal reflexes below the lesion which inhibits the associated autonomic reflex. These patients are a particularly challenging group whether a regional or general anesthetic is chosen. A thorough history of previous anesthetics performed, time since injury, and cardiovascular assessment should be done preoperatively. If a regional technique is chosen, it is imperative that an adequate level of blockade is achieved, by comparing the sensory level with surgical field to prevent AH. Whether general or neuraxial anesthesia is chosen, the goal is to diminish the inciting event and manage any hypertensive events that may occur. Each anesthetic technique has the potential for complications, and medications to treat severe hypertension should be readily available.

Conclusion

The use of regional anesthesia in patients with preexisting neurologic disease is associated with many concerns. The difficulty is determining if the progression or new onset of neurologic dysfunction will worsen with the use of regional anesthetics. To date, there are no robust clinical studies that support or refute this concern. Some of these patients will benefit from perioperative pain control and decreasing morbidity associated with general anesthesia if regional anesthesia is used. The patient should always be given an informed consent, and preoperative neurologic status should be fully documented. Conscious efforts should be made to decrease neural injury in the perioperative period by proper positioning, decreased tourniquet time, and avoidance of constrictive dressings to decrease pressure on vulnerable sites. Ultimately, new neurologic deficits should not be taken for granted but should have a proper evaluation by a neurologist and should undergo further testing as necessary. Likewise, long-term follow-up should be provided.

Review Questions

1. A 67-year-old female, obese and diabetic with peripheral neuropathy, is scheduled for an exploratory laparotomy and total abdominal hysterectomy with lymph node dissection. The patient requests an epidural for postoperative pain control. Which of the following factors would put this patient at increased risk for postoperative neurologic injury?
   (a) Using bupivacaine 0.125% infusion at 5 ml/h.
   (b) Performing epidural anesthesia prior to extubation for postoperative pain control.
   (c) Utilization of ultrasound for localization of the intervertebral space.
   (d) Padding lower extremity and placement of TED hoses for DVT prophylaxis.
2. Which of the following is the preferred imaging modality to diagnose spinal canal pathology?
   (a) Computed tomography.
   (b) Radiographic imaging such as X-ray.
   (c) Magnetic resonance imaging.
   (d) Myelography.
3. Which of the following statement is true regarding regional anesthesia and peripheral nerve injury (PNI)?
   (a) Intrafascicular injections are associated with higher opening injection pressures and risk of PNI compared with perineural injections.
   (b) The use of maximum concentration of local anesthetics is not associated with an increased risk of PNI in patients with preexisting neurologic injury.
   (c) The radial nerve at the elbow and the posterior tibial nerve are at increased risk of PNI.
   (d) The use of peripheral nerve blocks (PNB) is associated with PNI after total knee arthroplasty.

4. Which of the following patient-specific risk factors is not associated with perioperative nerve injury in patients with preexisting neurologic disorder?
   (a) Diabetes mellitus.
   (b) Female gender.
   (c) Obesity.
   (d) Advanced age.
5. Multiple sclerosis (MS) is a chronic degenerative disease that causes demyelination in the spinal cord and brain. Since the etiology of MS is multifactorial, which is the least likely cause of multiple sclerosis?
   (a) MS has a genetic predilection particularly with afflicted first-degree relatives with growing evidence that MS most likely involves the B cells.
   (b) T cells in the peripheral blood become stimulated and attack foreign antigens while simultaneously attacking brain proteins that share similar molecular region of the antigen.
   (c) Environmental factors and exposure to bacterial and viral agents may lead to MS.
   (d) Low vitamin D levels may be associated with MS.
6. Amyotrophic lateral sclerosis is a rapidly progressive disease with progressive degeneration of motor neurons that causes muscular weakness, atrophy, fasciculations, spasticity, and hyperreflexia. With respect to neuraxial anesthesia in patients with ALS, which answer is correct?
   (a) Patents are at greater risk for hemodynamic instability compared to general anesthesia.
   (b) Neuraxial anesthesia is not associated with any respiratory comprise when compared to general anesthesia.
   (c) Neuraxial anesthesia should be avoided in patients with ALS.
   (d) Neuraxial anesthesia may be a reasonable choice but has the potential of impairing respiratory function.
7. Post-polio syndrome (PPS) is a condition that affects polio survivors from an initial acute attack of the poliomyelitis virus. Which of the following statements is not true?
   (a) The onset of symptoms occurs up to 30 years after the initial episode of poliomyelitis.
   (b) PPS is the most prevalent progressive neuromuscular disease reported in North America.
   (c) PPS affects men greater than women.
   (d) The most common symptoms include progressive muscle weakness, fatigue, and muscle atrophy starting in the muscles previously affected by the virus.
8. Peripheral nervous system disorders comprise neurological disease states that involve the cell body, axon, neuromuscular junction, and myelin sheath. Which answer is most correct?
   (a) These disorders are mostly in the periphery and involve the brain or spinal cord.
   (b) These patients suffer from the disruption of axons with distal degeneration or segmental demyelination caused by Schwann cell degeneration.
   (c) These conditions typically start proximally and spread in a distal fashion leading to the classic "glove and stocking" distribution of decreased sensation, weakness, and diminished reflexes.
   (d) Electromyogram and nerve conduction velocity studies typically will not reveal evidence of denervation and any changes in nerve conduction velocity.
9. Autonomic hyperreflexia (AH) is a dangerous clinical syndrome that develops as a sequela to spinal cord injury. Which of the following statements is true?
   (a) Eighty-five percent of spinal cord injury patients are at risk for developing hyperreflexia symptoms if the lesion involves the T8 spinal cord level or lower.
   (b) Spinal cord injury can produce acute uncontrolled hypertension with the introduction of a noxious stimulus above the level of the lesion.
   (c) The leading trigger that stimulates AH is typically distention of the urinary bladder or colon.
   (d) The pathophysiology involves an imbalanced reflex sympathetic discharge that

involves inhibited spinal reflexes that leads to hypertension and reflex bradycardia.

(e) General anesthesia has been shown to inhibit spinal reflexes better than spinal anesthesia below the lesion which inhibits the associated autonomic reflex.

Answers

1. (b) Patients under general anesthesia or deep sedation lack the ability to verbalize or report warning signs and can place a patient at risk for neurologic injury. Warning signs such as paresthesia or pain on injection of local anesthetics would indicate needle proximity to neuraxis. The use of dilute local anesthetics and appropriate patient positioning with adequate padding help prevent further neurologic compromise. Even though the use of ultrasound in neuraxial blockade has not been shown to reduce the risk of peripheral nerve injury, it would beneficial to aid proper anatomic localization.
2. (c) Magnetic resonance imaging is the diagnostic modality of choice in patients suspected with neuraxial lesions. However, computed tomography should be used for rapid diagnosis if MRI is not immediately available especially if neuraxial compression injury is suspected.
3. (a) Local anesthetic toxicity is time- and concentration-dependent. The ulnar nerve and the common peroneal nerve are at increased risk of PNI. PNB is not associated with PNI after TKA.
4. (b) There is a plethora of factors that can contribute to PNI especially in patients with preexisting neurologic disorder. These include diabetes mellitus, extremes of habitus, male gender, and advanced age.
5. (a) The etiology of MS is not fully understood, but multiple factors may be involved. MS has a genetic predilection particularly with afflicted first-degree relatives with growing evidence that MS most likely involves the T cells. There is a possibility that T cells in the peripheral blood become stimulated and attack foreign antigens while simultaneously attacking brain proteins that share similar molecular region of the antigen. It has also been associated with environmental factors and exposure to bacterial and viral agents. Lastly, there is a correlation of low vitamin D levels with MS.
6. (d) Amyotrophic lateral sclerosis (ALS) leads to progressive degeneration of motor neurons and causes muscular weakness, atrophy, fasciculations, spasticity, and hyperreflexia. A concerning problem with ALS is general anesthesia; these patients may have extreme hemodynamic responses with progressive weakness, muscle atrophy, and risk for aspiration and ventilator dependence. ALS patients have better outcomes when general anesthesia can be avoided entirely; therefore, regional anesthesia should be considered whenever possible. Epidural anesthesia may be a reasonable choice in selected cases but has the potential of impairing respiratory function by producing intercostal muscle weakness.
7. (c) The onset of symptoms may occur up to 30 years after the initial episode of poliomyelitis. PPS is the most prevalent progressive neuromuscular disease reported in North America with a greater rate in women than in men. The most common symptoms include slowly progressive muscle weakness, fatigue, and gradual muscle atrophy starting in the muscles previously affected by the virus.
8. (b) Peripheral nervous system disorders occur in the periphery and do not involve the brain or spinal cord. These patients suffer from the disruption of axons with distal degeneration or segmental demyelination caused by Schwann cell degeneration which will result in peripheral neuropathies. Such conditions typically start distally and spread in a proximal fashion leading to the classic "glove and stocking" distribution of decreased sensation, weakness, and diminished reflexes. Electromyogram and nerve conduction velocity studies will often reveal evidence of denervation and decreased nerve conduction velocity.
9. (c) Approximately 85% of spinal cord injury patients are at risk for developing hyperreflexia symptoms if the lesion involves the

T6 spinal cord level or higher. This injury can produce acute uncontrolled hypertension with the introduction of a noxious stimulus below the level of the lesion, leading to a peripheral sympathetic response producing vasoconstriction below the injured level. The leading trigger that stimulates AH is typically distention of the urinary bladder or colon. The pathophysiology involves an imbalanced reflex sympathetic discharge that involves uninhibited spinal reflexes. Regional anesthesia has been used successfully in patients with autonomic hyperreflexia. Particularly, spinal anesthesia has shown to inhibit spinal reflexes below the lesion which inhibits the associated autonomic reflex.

References

1. Warner MA, Martin JT, Schroeder DR, Offord KP, Chute CG. Lower-extremity motor neuropathy associated with surgery performed on patients in a lithotomy position. Anesthesiology. 1994;81(1):6–12.
2. Bajaj P. Regional anaesthesia in the patient with preexisting neurological dysfunction. Indian J Anaesth. 2009;53(2):135–8.
3. Kopp SL, Jacob AK, Hebl JR. Regional anesthesia in patients with preexisting neurologic disease. Reg Anesth Pain Med. 2015;40(5):467–78.
4. Dorotta IR, Schubert A. Multiple sclerosis and anesthetic implications. Curr Opin Anaesthesiol. 2002;15(3):365–70.
5. Perlas A, Chan VW. Neuraxial anesthesia and multiple sclerosis. Can J Anaesth. 2005;52(5):454–8.
6. Korn-Lubetzki I, Kahana E, Cooper G, Abramsky O. Activity of multiple sclerosis during pregnancy and puerperium. Ann Neurol. 1984;16(2):229–31.
7. Hebl JR, Horlocker TT, Schroeder DR. Neuraxial anesthesia and analgesia in patients with preexisting central nervous system disorders. Anesth Analg. 2006;103(1):223–8. table of contents
8. Koff MD, Cohen JA, McIntyre JJ, Carr CF, Sites BD. Severe brachial plexopathy after an ultrasound-guided single-injection nerve block for total shoulder arthroplasty in a patient with multiple sclerosis. Anesthesiology. 2008;108(2):325–8.
9. Pogorzelski R, Baniukiewicz E, Drozdowski W. Subclinical lesions of peripheral nervous system in multiple sclerosis patients. Neurol Neurochir Pol. 2004;38(4):257–64.
10. Misawa S, Kuwabara S, Mori M, Hayakawa S, Sawai S, Hattori T. Peripheral nerve demyelination in multiple sclerosis. Clin Neurophysiol. 2008;119(8):1829–33.
11. Finucane BT, Terblanche OC. Prolonged duration of anesthesia in a patient with multiple sclerosis following paravertebral block. Can J Anaesth. 2005;52(5):493–7.
12. Neal JM, Barrington MJ, Brull R, Hadzic A, Hebl JR, Horlocker TT, et al. The second ASRA practice advisory on neurologic complications associated with regional anesthesia and pain medicine: executive summary 2015. Reg Anesth Pain Med. 2015;40(5):401–30.
13. Forbes RB, Colville S, Swingler RJ, Scottish Motor Neurone Disease Research G. Frequency, timing and outcome of gastrostomy tubes for amyotrophic lateral sclerosis/motor neurone disease--a record linkage study from the Scottish Motor Neurone Disease Register. J Neurol. 2004;251(7):813–7.
14. Kochi T, Oka T, Mizuguchi T. Epidural anesthesia for patients with amyotrophic lateral sclerosis. Anesth Analg. 1989;68(3):410–2.
15. Thampi SM, David D, Chandy TT, Nandhakumar A. Anesthetic management of a patient with amyotrophic lateral sclerosis for transurethral resection of bladder tumor. Indian J Anaesth. 2013;57(2):197–9.
16. Lambert DA, Giannouli E, Schmidt BJ. Postpolio syndrome and anesthesia. Anesthesiology. 2005;103(3):638–44.
17. Elrod LM, Jabben M, Oswald G, et al. Vocational implications of post-polio syndrome. Work. 2005;25(2):155–61.
18. Ragonese P, Fierro B, Salemi G, et al. Prevalence and risk factors of post-polio syndrome in a cohort of polio survivors. J Neurol Sci. 2005;236(1–2):31–5.
19. National Institute of Neurological Disorders and Stroke, NINDS Post-Polio Syndrome Fact Sheet. http://www.strokenindsnihgov/disorders/post_polio/detail_post_poliohtm. Accessed 8 Jan 2017
20. Kattula A, Angelini G, Arndt G. Regional anesthesia in the presence of neurologic disease. In: Finucane BT, editor. Complications of regional anesthesia. New York, NY: Springer New York; 2007. p. 373–85.
21. Schwartz A, Bosch LM. Anesthetic implications of postpolio syndrome: new concerns for an old disease. AANA J. 2012;80(5):356–61.
22. Stoelting R, Dierdorf SF. Diseases of the nervous system. In: Anesthesia and coexisting disease. 3rd ed. New York: Churchill-Livingstone; 1993. p. 218.
23. Schmitt HJ, Muenster T, Schmidt J. Central neural blockade in Charcot-Marie-Tooth disease. Can J Anaesth. 2004;51(10):1049–50.
24. Tanaka S, Tsuchida H, Namiki A. Epidural anesthesia for a patient with Charcot-Marie-Tooth disease, mitral valve prolapse syndrome and IInd degree AV block. Masui. 1994;43(6):931–3.
25. Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach JM, et al. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology. 1993;43(4):817.
26. Hebl JR, Kopp SL, Schroeder DR, Horlocker TT. Neurologic complications after neuraxial anesthe-

sia or analgesia in patients with preexisting peripheral sensorimotor neuropathy or diabetic polyneuropathy. Anesth Analg. 2006;103(5):1294–9.
27. Sinnreich M, Taylor BV, Dyck PJ. Diabetic neuropathies. Classification, clinical features, and pathophysiological basis. Neurologist. 2005;11(2):63–79.
28. Kalichman MW, Calcutt NA. Local anesthetic-induced conduction block and nerve fiber injury in streptozotocin-diabetic rats. Anesthesiology. 1992;77(5):941–7.
29. Williams BA. Toward a potential paradigm shift for the clinical care of diabetic patients requiring perineural analgesia: strategies for using the diabetic rodent model. Reg Anesth Pain Med. 2010;35(4):329–32.
30. Sites BD, Gallagher J, Sparks M. Ultrasound-guided popliteal block demonstrates an atypical motor response to nerve stimulation in 2 patients with diabetes mellitus. Reg Anesth Pain Med. 2003;28(5):479–82.
31. Lucas LF, Tsueda K. Cardiovascular depression after brachial plexus block in two diabetic patients with renal failure. Anesthesiology. 1990;73(5):1032–5.
32. Pachman DR, Barton DL, Watson JC, Loprinzi CL. Chemotherapy-induced peripheral neuropathy: prevention and treatment. Clin Pharmacol Ther. 2011;90(3):377–87.
33. Abcejo AS, Sviggum HP, Mauermann ML, Hebl JR, Mantilla CB, Hanson AC, et al. Perioperative nerve injury after peripheral nerve block in patients with previous systemic chemotherapy. Reg Anesth Pain Med. 2016;41(6):685–90.
34. Jacob AK, Mantilla CB, Sviggum HP, Schroeder DR, Pagnano MW, Hebl JR. Perioperative nerve injury after total hip arthroplasty: regional anesthesia risk during a 20-year cohort study. Anesthesiology. 2011;115(6):1172–8.
35. Esposito S, Longo MR. Guillain-Barre syndrome. Autoimmun Rev. 2017;16(1):96–101.
36. van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barre syndrome. Lancet Neurol. 2008;7(10):939–50.
37. Walling AD, Dickson G. Guillain-Barre syndrome. Am Fam Physician. 2013;87(3):191–7.
38. Kocabas S, Karaman S, Firat V, Bademkiran F. Anesthetic management of Guillain-Barre syndrome in pregnancy. J Clin Anesth. 2007;19(4):299–302.
39. Alici HA, Cesur M, Erdem AF, Gursac M. Repeated use of epidural anaesthesia for caesarean delivery in a patient with Guillain-Barre syndrome. Int J Obstet Anesth. 2005;14(3):269–70.
40. Vassiliev DV, Nystrom EU, Leicht CH. Combined spinal and epidural anesthesia for labor and cesarean delivery in a patient with Guillain-Barre syndrome. Reg Anesth Pain Med. 2001;26(2):174–6.
41. Wiertlewski S, Magot A, Drapier S, Malinovsky JM, Pereon Y. Worsening of neurologic symptoms after epidural anesthesia for labor in a Guillain-Barre patient. Anesth Analg. 2004;98(3):825–7. table of contents
42. Olivier N, Laribi H, Pages M, Camu W, Juntas Morales R. Recurrent Guillain-Barre syndrome after surgery. Rev Neurol (Paris). 2010;166(6–7):644–7.
43. Mangar D, Sprenker C, Karlnoski R, Puri S, Decker D, Camporesi E. Rapid onset of guillain-barre syndrome after an obstetric epidural block. A A Case Rep. 2013;1(1):19–22.
44. Vinay B, Sonia B, Bhadrinarayan V. Hyperacute onset of Guillain Barre Syndrome in the immediate postpartum period following Caesarean section under spinal anaesthesia. Indian J Anaesth. 2015;59(6):391–2.
45. Connelly M, Shagrin J, Warfield C. Epidural opioids for the management of pain in a patient with the Guillain-Barre syndrome. Anesthesiology. 1990;72(2):381–3.
46. McGrady EM. Management of labour and delivery in a patient with Guillain-Barre syndrome. Anaesthesia. 1987;42(8):899.
47. Wipfli M, Arnold M, Luginbuhl M. Repeated spinal anesthesia in a tetraparetic patient with Guillain-Barre syndrome. J Clin Anesth. 2013;25(5):409–12.
48. Hebl JR, Horlocker TT, Kopp SL, Schroeder DR. Neuraxial blockade in patients with preexisting spinal stenosis, lumbar disk disease, or prior spine surgery: efficacy and neurologic complications. Anesth Analg. 2010;111(6):1511–9.
49. Daley MD, Rolbin SH, Hew EM, Morningstar BA, Stewart JA. Epidural anesthesia for obstetrics after spinal surgery. Reg Anesth. 1990;15(6):280–4.
50. Crosby ET, Halpern SH. Obstetric epidural anaesthesia in patients with Harrington instrumentation. Can J Anaesth. 1989;36(6):693–6.
51. Hubbert CH. Epidural anesthesia in patients with spinal fusion. Anesth Analg. 1985;64(8):843.

Sympathetic Blockade

32

Miles Day, Rafael Justiz, Audra Day, and Maxim S. Eckmann

Indications and Contraindications

Specific indications will be listed with the individual blocks. Absolute and relative contraindications are listed below. For patients taking anticoagulant medications, check with the prescribing physician prior to holding any of these medications for a procedure.

Absolute (A) and Relative (R) Contraindications

Patient refusal—A
Local infection and sepsis—A
Allergy to medications used—A and R
Coagulopathy—R
Anticoagulant therapy—R
History of facial trauma (SPG)—R
Pre-existing neurological deficits—R
History of previous surgery in the region of the block—R
Altered mental status—R

Evidence

The evidence for each block will be briefly reviewed. The grade of recommendation is based on Guyatt et al.'s [1] evidence-based medicine guidelines.

Sphenopalatine Ganglion Block

Indications

- Sphenopalatine neuralgia
- Trigeminal neuralgia
- Migraine headaches
- Cluster headaches
- Post-traumatic headaches
- Persistent idiopathic facial pain
- Cancer of the tongue and floor of the mouth
- Herpes zoster ophthalmicus

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_32

Anatomy

- The sphenopalatine ganglion (SPG) resides in the pterygopalatine fossa. The SPG is bordered medially by the palatine bone, cephalad by the sphenopalatine sinus, anteriorly by the posterior wall of the maxillary sinus, and posteriorly by the medial pterygoid plate.
- The pterygomaxillary fissure allows passage of a needle into the fossa, while the pterygopalatine foramen is located medial to the ganglion and is just posterior to the middle turbinate. The fossa is approximately 1-cm wide and 2-cm high and resembles the letter "V" on a lateral fluoroscopic image. A large venous plexus overlies the fossa. Foramen rotundum and the pterygoid canal are located on the superolateral and inferomedial aspect of the fossa, respectively. The maxillary artery resides in the fossa.
- The ganglion is "suspended" from the maxillary nerve by the pterygopalatine nerves and is medial to the maxillary division of the trigeminal nerve. Posteriorly the ganglion is connected to the Vidian nerve which is formed by the deep petrosal (sympathetic from the upper thoracic spinal cord) and greater petrosal (parasympathetic from the superior salivatory nucleus) nerves.
- The ganglion has efferent branches and forms the superior posterior lateral nasal and pharyngeal nerves. Caudally, the greater and lesser palatine nerves exit the ganglion. Sensory fibers arise from the maxillary nerve, pass through the SPG, and innervate the upper teeth, nasal membranes, soft palate, and some parts of the pharynx. A small number of motor nerves are believed to travel with the sensory trunks.

Procedure

There are multiple techniques for blockade of the SPG. This chapter will focus on two techniques: the intranasal approach and the infrazygomatic approach.

Intranasal Approach

The intranasal SPG block can be safely performed in an office setting with appropriate monitoring. The location of the SPG in relation to the middle turbinate as well as the lateral nasal mucosa allows absorption of local anesthetic from one or two cotton-tipped applicators inserted into the naris. The local anesthetic of choice is 4% cocaine secondary to its inherent vasoconstrictor property. This may help prevent epistaxis. If this is not available or there is a contraindication to using cocaine, 1–2% lidocaine, 0.25–0.50% bupivacaine, or 0.2–0.5% ropivacaine can be used. If these are chosen, the practitioner can pretreat the nares with Neo-Synephrine to produce vasoconstriction.

- Place the patient in the supine position. Obtain baseline vital signs.
- Estimate the depth of insertion. Measure the distance from the opening of the naris to the mandibular notch beneath the zygoma. Place a mark corresponding to this depth on the shaft of the cotton-tipped applicator. Soak the applicators in the local anesthetic for several minutes.
- Slowly insert the applicator into the naris and advance in a line parallel to the zygoma with the tip angled laterally. Do not advance the applicator in a cephalad direction. This may cause epistaxis. The end point should be the depth marked on the applicator.
- Place a second applicator into the naris using the same technique, except advance it approximately 0.5–1.0 cm deeper and superior to the first. If resistance is encountered, slightly withdraw and redirect the applicator. The second applicator is not an absolute necessity. The nares of some patients may not accommodate it.
- Leave the applicator(s) in for 30–45 min. Signs of a successful block of the SPG include ipsilateral tearing, conjunctival injection, and nasal congestion (parasympathetic features).
- If the SPG is a pain generator or transmitter, analgesia should be apparent. If after 20–30 min there are no signs of a block or the patient has not received any pain relief, additional local anesthetic may be needed and can be trickled down the shaft of the applicator.

- Remove the cotton-tipped applicators after 45 min even if there are no signs of a block or analgesia. If there are no signs of a block or analgesia, the SPG may be too deep, i.e., too lateral, to be blocked by this technique or is not involved in the transmission of pain. Regardless, the infrazygomatic approach should be performed to rule out both of the aforementioned scenarios.

Infrazygomatic Approach

This approach to SPG blockade can be challenging. Fluoroscopic guidance is highly recommended as this will anecdotally improve the success of the block and the speed at which it is performed and decrease potential complications. Noninvasive monitors should be used. Light sedation with midazolam and fentanyl can be used, but on occasion, deeper sedation with propofol may be necessary. Place the patient in the supine position, and sterilely prep and drape the appropriate side of the face.

- Palpate the mandibular notch and anesthetize the skin. If the notch is not palpable, identify the notch on a lateral fluoroscopic view.
- Identify the pterygopalatine fossa (appears as a "V") on the lateral image and superimpose the right and left fossae. This is accomplished by manipulating the C-arm or the head. The block can be performed with a 3.5-in., 22-gauge, short-bevel needle with the distal tip bent at a 30° angle away from the notch of the stylet or with a curved, blunt, 10-cm, 20- or 22-gauge needle. The technique description will reflect the use of a blunt needle.
- Anesthetize the skin and insert a 1.25-in., 16- or 18-gauge angiocatheter through the skin, and advance until it is just medial to the ramus of the mandible. This can be checked on an AP image.
- Pass the block needle through the angiocatheter and advance it medial, anterior, and slightly cephalad. Obtain a lateral image to check the direction of the needle. Your target is the upper midportion of the pterygopalatine fossa (Fig. 32.1).
- Get an AP view, and advance the needle toward the middle turbinate, stopping when the tip is adjacent to the palatine bone (Fig. 32.2). If resistance is encountered at any point, withdraw and redirect the needle. Given the small size of the fossa, frequent AP and lateral images are may be required to redirect the needle.
- Once in the fossa, inject 0.5–1.0 ml nonionic, water-soluble contrast, and observe for intravascular spread and/or intranasal placement of the needle.
- Once correct placement has been confirmed, inject 2 ml of local anesthetic (1–2% lidocaine, 0.25–0.50% bupivacaine, or 0.2–0.5% ropivacaine), with or without steroids.

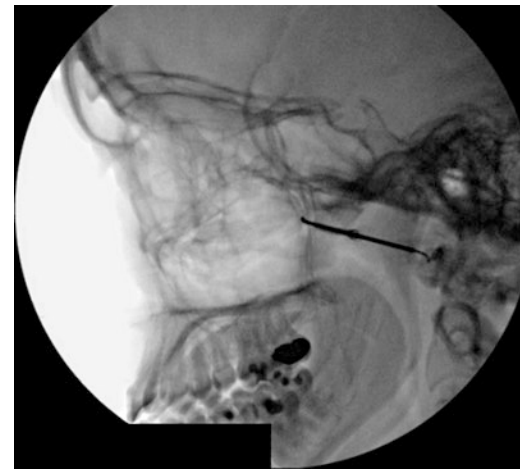

Fig. 32.1 Lateral fluoroscopic image of a curved, blunt needle in the pterygopalatine fossa

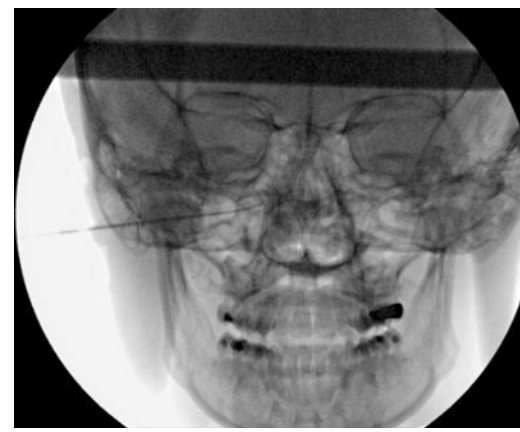

Fig. 32.2 AP fluoroscopic image of a curved, blunt needle adjacent to the middle turbinate

- An alternative approach is to insert the angiocatheter below the zygoma, but just cephalad to the coronoid process. Using a coaxial technique, the block needle can be advanced straight into the pterygopalatine fossa. This technique can only be used if the coronoid process is low lying and does not obstruct the path of the needle to the fossa.

Radiofrequency Thermocoagulation and Pulsed Radiofrequency

After a successful diagnostic block, two therapeutic choices are available: conventional radiofrequency thermocoagulation (RFTC) and pulsed electromagnetic field radiofrequency (P-EMF). An insulated RF needle with a 2–3-mm active tip is placed using the infrazygomatic approach. This prevents inadvertent lesioning of other nerves around the SPG.

- Once in place, sensory stimulation is performed at 50 Hz up to 1 V. If the tip of the needle is adjacent to the SPG, the patient should perceive a paresthesia at the root of the nose at less than 0.3 V. If the paresthesia is felt in the hard palate, the needle should be redirected cephalad and medial. A paresthesia in the upper teeth indicates stimulation of the maxillary nerve, and the needle should be more caudal and medial. Motor stimulation is not necessary.
- After appropriate sensory stimulation, RFTC can be performed at 67–80 °C for 90 s times two cycles. Before lesioning, 2–3 ml of local anesthetic (1–2% lidocaine, 0.25–0.50% bupivacaine, or 0.2–0.5% ropivacaine) should be injected.
- For P-EMF, the size of the active tip is not important as the electromagnetic field is projected from the tip of the needle and not from the shaft. There is not a general consensus in the literature as to the number of 120-s pulse cycles that are necessary, but originally it was two to four. One of the authors performs two cycles each in the 12, 3, 6, and 9 o'clock positions on the ganglion. Local anesthetic is not required for P-EMF but is helpful for post-procedure pain.
- The choice of whether to do a RFTC or a P-EMF lesion after a successful block is up to the discretion of the pain management practitioner. The authors prefer P-EMF as it is a non-neurodestructive procedure.

Complications

Complications include bruising, bleeding, infection, damage to nerves, proptosis from retrobulbar hematoma, dysesthesias, paresthesias, and/or numbness from RFTC. Bradycardia ("Konen" reflex) has been described during RFTC and P-EMF and can be prevented with pretreatment with atropine or glycopyrrolate [2].

Evidence

Day published an article on the current evidence for sympathetic blocks [3]. For the sphenopalatine ganglion block, ten articles (six case reports, three case series, and one retrospective review) were mentioned for the indications listed above. All were graded as 1C: strong recommendation and low-quality or very low-quality evidence. A case series on RFTC of the sphenopalatine ganglion for chronic cluster headaches by Narouze et al. also receives a 1C recommendation [4].

Stellate Ganglion Block

Indications [5–7]

- Complex regional pain syndromes types I and II
- Sympathetic maintained pain
- Phantom limb pain
- Postherpetic neuralgia
- Angina pectoris
- Arterial insufficiency
- Hyperhidrosis
- Meniere's disease
- Chronic facial pain
- Cervical pain
- Atypical vascular-type headaches
- Hot flashes
- Frostbite
- Post-traumatic stress disorder

Anatomy

- The stellate ganglion (SG) is the most inferior ganglion of the cervical sympathetic chain. It is most often formed when the inferior cervical ganglion fuses with the first thoracic ganglion forming an oval, dumbbell, or inverted "L" mass 2.5-cm long, 1-cm wide, and 0.5-cm thick [8–10]. In approximately 20% of the population, the two ganglia remain separated [9].
- The SG is located in the posterior region of the superior thorax 5 mm anterolateral to bony structures [8, 11]. It is positioned anterior to and between the base of the seventh cervical transverse process and the neck of the first rib. In cases where fusion is absent, the inferior cervical portion is positioned anterior to the tubercle of the seventh cervical vertebra, while the first thoracic ganglion lies directly superior to the neck of rib number one [9].
- It is medial to the scalene muscles, anterolateral to the lateral border of the longus coli muscle, and lateral to the esophagus, trachea, and recurrent laryngeal nerve [8–10].
- The vertebral artery lies anterior to the ganglion as it originates from the subclavian artery [8]. Additional vascular landmarks include the common carotid artery which is located medially and the inferior thyroid artery which is anterior and lateral [8, 9]. The inferior thyroid artery has a variable and unpredicted anatomy and has a very tortuous serpentine course which may make it more susceptible to needle puncture [12].
- The postganglionic fibers exit the ganglion through the gray rami communicantes and provide sympathetic efferent impulses to the following [10]:
  - Cardiac plexus located posterior to the aortic arch
  - Vascular smooth muscle of the subclavian arteries, vertebral arteries, and brachiocephalic trunk
  - Skin sweat glands and erector pili muscles

Relevant Soft Tissue Structures and Sonoanatomy (Fig. 32.3)

- The ultrasound probe is placed on the skin after a warmed acoustic compatible gel is applied over the transducer. Orientation is transverse to the body axis, lateral of midline in the cervical region near the level of the cricoid cartilage. If a procedure is planned, the skin should have been prepped, drapes placed, and a sterile cover placed over the probe. The lateral edge of the probe is rotated slightly superiorly (e.g., counterclockwise if on the patient's left side) and sometimes tilted slightly cephalad.
- Platysma and sternocleidomastoid muscles overly the anterior neck in this region. Muscle tissue is hyperechoic compared to fluid/blood and may have relatively hyperechoic internal stranding and outer connective tissue.
- Thyroid tissue overlies anterior access to the stellate ganglion, often extending from the fifth cervical vertebral level to the first thoracic level. Notably, this tissue is rich in vessels including the inferior thyroid artery; therefore positive identification can assist in avoidance of puncture and hematoma risk [9]. It is anterior and laterally adjacent to the trachea on either side. General appearance is a

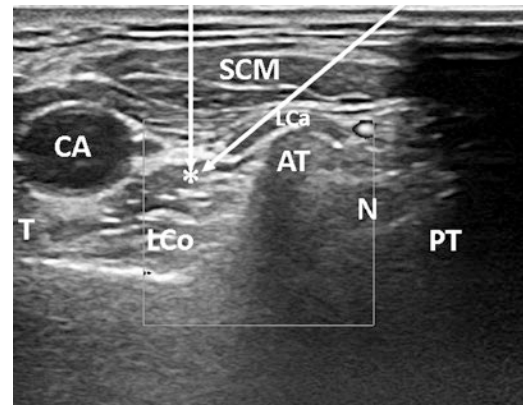

Fig. 32.3 Performance of the cervical sympathetic chain block at C6. *AT* anterior tubercle, *PT* posterior tubercle, *SCM* sternocleidomastoid m., *LCa* longus capitis m., *LCo* longus colli m., *CA* carotid artery, *N* nerve root, *T* thyroid, *CA* carotid artery

solid homogenous structure hyperechoic in comparison to muscle and adipose.
- The esophagus is deep to and slightly leftward of the trachea; it can be augmented dynamically by having the patient swallow, revealing an air artifact that highlights the lumen. Diverticula and other anatomic abnormalities may be seen and avoided.
- Lateral to the thyroid and anterior to the prevertebral fascia lie the carotid artery and internal jugular vein. Even more laterally, the external jugular vein may be visible. The veins may be compressed during ultrasound exam, while the carotid artery will generally remain patent. Vessels have a pulsatile motion in real time and are internally hypoechoic.
- Color Doppler can be used to highlight vascular structures. However, vessels may not enhance well when flow is purely perpendicular to the plane of the ultrasound image.
- More laterally and just beneath the prevertebral fascia, the anterior tubercle of the transverse process is readily identified as a prominent hyperechoic peak which casts an ultrasound shadow beneath. Its hyperechoic cortical signature is continuous with the anterior body of the cervical vertebra. At C6, this tubercle is very prominent, and the longus capitis can be seen just overlying the peak as a thin layer as it is just beginning its distal attachment point here. At higher levels, this muscle becomes more prominent. Below C6 it should usually no longer be visible. A vertebral artery signature should not be seen in this area at C6 but can be seen at C7 and other levels [13]. At C7 there is no anterior tubercle, but there is a visible posterior tubercle [14].
- Deep to the prevertebral fascia, just medial to the anterior tubercle, and just anterior to the vertebral body is the longus colli muscle, oval shaped in cross-section. The cervical sympathetic chain lies anterior to this muscle and deep to the prevertebral fascia. The muscle attaches from thoracic bodies as low as T2 or T3 and ascends to the upper cervical transverse processes and the arch of the atlas.
- Lateral to the anterior tubercle, a round hypoechoic structure signifies the cervical nerve root.
- Further laterally the posterior tubercle can be seen as a broader and more posterior hyperechoic peak with shadow effect.

Procedure: Fluoroscopically Guided

There are several techniques for blockade of the stellate ganglion. This section will focus on two techniques: the anterior paratracheal approach at C6 and C7. Correct needle placement can be attained at each approach by different methods, palpating specific landmarks or using fluoroscopy. The authors prefer fluoroscopy as the landmarks are not easily palpable, and it provides a safer and more accurate method of performing these injections.

Patient Position

Place the patient in a supine position with the head flat on the table without a pillow and folded sheet under the shoulders for a slight neck extension. Allow a slight jaw opening to relax the skin and musculature tension over the targeted area in the neck. Sterilely prep and drape the neck region from the base of the chin to the upper sternum.

Anterior C6 Approach

- Palpating the anterior neck, identify the cricoid cartilage and carotid artery. Palpate laterally with firm pressure until the anterior C6 tubercle (Chassaignac's) is identified, or identify using fluoroscopy.
- A 22-gauge, 1.5-in. B-bevel needle is advanced, while the structures underneath are gently displaced posteriorly and laterally. Alternatively, a 22- or 25-gauge, 3.5-in. Quincke needle can be used.
- The needle is advanced in a perpendicular plane until bony contact is made at the C6 transverse process.
- Once bony contact is attained, the needle is slightly withdrawn 0.5 cm to remove it from the longus coli muscle or periosteum.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble

contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the C6 vertebral body. Failure of contrast spread caudad or cephalad typically suggests injection of contrast into the longus coli muscle, and instantaneous dissipation of contrast agent indicates intravascular placement. If this occurs, the needle needs to be repositioned.

- Obtain an AP and lateral image. The final position should be with the needle tip touching the C6 transverse process in both views.
- Once appropriate position is confirmed, inject 10 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration with or without steroids.
- A successful block is indicated by an increase in temperature of 1.5–3 °C.
- Alternatively, this procedure can be performed blindly in the same steps as described above without the addition of radiocontrast agent.

Anterior C7 Approach

- Once the anterior C6 tubercle (Chassaignac's) is identified, move one finger breadth inferiorly to locate your target, the C7 tubercle, or identify using fluoroscopy.
- Under fluoroscopy in an AP view, the junction of the C7 transverse process with the vertebral body is identified as the target.
- A 22-gauge, 1.5-in. B-bevel needle is advanced, while the structures underneath are gently displaced posteriorly and lateral. Alternatively, a 22- or 25-gauge, 3.5-in. Quincke needle can be used.
- Correct needle position is confirmed with fluoroscopy in both the AP and lateral views with the needle tip at the junction of the C7 transverse process and the vertebral body.
- Once bony contact is attained, the needle is slightly withdrawn 0.5 cm to remove it from the longus coli muscle or periosteum.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the C7–T1 plane in a superior and inferior direction (Fig. 32.4). Failure of contrast spread caudad or cephalad typically suggests injection of contrast into the longus coli muscle, and instantaneous dissipation of contrast agent indicates intravascular placement. If this occurs, the needle needs to be repositioned.
- Obtain an AP and lateral image. An oblique image can also be obtained (Fig. 32.5). The final position should be with the needle tip

Fig. 32.4 AP fluoroscopic image of contrast spread at C7 along the longus colli muscle

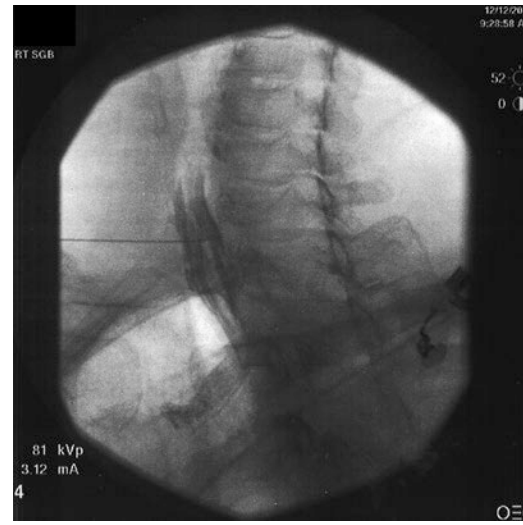

Fig. 32.5 Oblique fluoroscopic image of contrast spread at C7 along the longus colli muscle

touching the junction of the C7 transverse process and vertebral body in both views.
- Once appropriate position is confirmed, inject 5 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration with or without steroids.
- Alternatively, this procedure can be performed blindly in the same steps as described above without the addition of radiocontrast agent.

Procedure: Ultrasound-Guided Approach

There are several approaches for blockade of the cervical sympathetic chain under ultrasound guidance. This section will focus on two techniques: the anterior paratracheal approach at C6 and the oblique approach at C6. The block can be performed at C7 as well; however, the practitioner should be well practiced in the ability to locate the needle tip as well as in identifying deep vascular structures (i.e., the vertebral artery), and the final needle location should remain anterior to the longus colli muscle.

General Considerations

Ultrasound confers advantages in performance of the stellate ganglion block, particularly in the ability to avoid soft tissue structures not visible under fluoroscopy [9, 13–15]. Often, the ultrasound technique requires "three hands" to manipulate all the tools during the procedure. An assistant with sterile gloves may be employed to handle the syringe on flexible tubing to aspirate and deliver controlled boluses of injectate. Alternatively, the advanced practitioner may elect to "palm" the syringe in hand while manipulating the needle, intermittently letting go of the needle to deliver injectate. The key disadvantage to this single-provider modification is that the needle may move out of an optimal visual plane during this process. The authors recommend against using a syringe directly connected to the needle hub as stable needle positioning while injection is occurring becomes more difficult. We recommend instead using extension tubing to relieve strain from the needle.

The needle may be advanced adjacent or "out-of-plane" to the transducer in a vertical fashion or "in-plane" from a starting point lateral to the transducer and then into the field of view directly. Full visualization of the needle can be challenging due to the very thin ultrasound plane, and the echogenicity of needles oriented in a steep or vertical angle is very low. Echogenic needles with modified surface geometry can enhance visualization. Additionally, the use of small amounts of saline injection during the procedure can help confirm needle tip location through hydro-dissection.

Unlike in fluoroscopic techniques, there exists no positive contrast confirmation of intravascular injection. However, blood vessels can be avoided with positive sonolocation. Similarly, confirmation of injection spread in the desired location can be seen immediately in the plane of view, but tracing the injectate down to thoracic levels is not feasible with ultrasound. If such confirmation is desired, the practitioner may consider a combined technique with fluoroscopy.

Patient Position

Place the patient in a supine position with the head flat on the table without a pillow and folded sheet under the shoulders for a slight neck extension. The chin can be rotated somewhat contralaterally to accommodate placement of the transducer. Sterilely prep and drape the neck region from the base of the chin to the upper sternum.

Anterior C6 Approach

- Palpating the anterior neck, identify the cricoid cartilage or estimate its level.
- Apply a sterile-prepped ultrasound probe and sterile gel over the intended vertebral level, lateral to the trachea, in a transverse orientation to the axis of the body. Rotate the probe such that the lateral edge moves slightly cephalad.

- Scan for the optimal view of the C6 anterior tubercle, which should be the largest and most prominent of cervical levels. If the longus capitis muscle becomes more prominent with cephalad movement, then the probe is passing cranially to C6. If it disappears with caudad movement, then the probe is passing below C6.
- Identify relevant soft tissue structures with special attention to the trachea, esophagus, inferior thyroid artery (if seen), carotid artery, vertebral artery (if present), nerve root, internal jugular vein, and external jugular vein. These structures should not be traversed although the veins can be traversed with a small gauge needle and subsequent manual pressure for several minutes after the procedure is complete. A safe anterior-posterior vector can be identified.
- Identify the longus colli muscle anterior to the vertebral body, medial to the anterior tubercle, lateral to the carotid artery, deep to a hyperechoic line of prevertebral fascia.
- Needle insertion can be started along this vector in an "out-of-plane" technique, directed to the lateral aspect of the longus colli muscle. Saline hydro-dissection may be required to discern the location of the needle tip.
- A 22-gauge, 1.5-in. B-bevel needle is a good option; it is advanced, while the structures underneath are gently displaced posteriorly and lateral. Alternatively, a 22- or 25-gauge, 3.5-in. Quincke needle can be used.
- The needle is advanced until it pierces the prevertebral fascia, just shallow to the longus colli muscle.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of saline to confirm location. Appropriate spread should be in subtle tissue movement or fluid pocket formation with injection.
- Once appropriate position is confirmed, inject 5–10 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration with or without steroids.
- A successful block is indicated by an increase in temperature of 1.5–3 °C.

Anterior C7 Approach

- This procedure proceeds in the same fashion as the C6 technique.
- Rather than a prominent anterior tubercle, as seen at the C6 level, a relatively flat transverse process is seen at C7 more medially, and the posterior tubercle more laterally as expected, with the nerve root exposed just medially to the posterior tubercle [14].
- The vertebral artery should be identified, often deep and lateral to the longus colli muscle.
- Extra care with needle tip control should be used as deep vascular structures may be difficult to visualize.

Oblique C6/C7 Approach

- This procedure proceeds in much the same fashion as the anterior techniques described above.
- The advantage of this approach is that it allows for total needle visualization and does not pass as closely to central critical structures. However, with aberrant placement, nerve root and foraminal entry is possible. Also, phrenic nerve injury could theoretically be more likely if the needle passes through the anterior scalene muscle [15].
- The view should be optimized so that the anterior tubercle and nerve roots are positively identified.
- The planned path of advancement is "in-plane" with the ultrasound image, starting lateral to the transducer and moving medially in an oblique angle. The path should pass just superficial to the anterior tubercle on the way to the injection target (Fig. 32.3).
- The external and internal jugular vein may be in the path; therefore, pressure may be needed after injection and needle removal to reduce the chance of hematoma.
- The needle is maintained in continuous view along its full length as the procedure progresses to ensure that the tip is visualized. Saline hydro-dissection is still a useful tool in the event that small translational or rotational changes in probe orientation make full needle visualization difficult.

Fig. 32.6 Using ultrasound as an adjunct to fluoroscopically guided stellate ganglion block

Of note is that fluoroscopy and ultrasound can be used in conjunction to perform blockade of the stellate ganglion using all of the approaches previously described (Fig. 32.6).

Radiofrequency Thermocoagulation [16]

For RFTC of the stellate ganglion, place the needles as in the C7 approach except for the following changes:

- Once the target is identified, a 20- or 22-gauge, 5-mm active tip radiofrequency probe is advanced until bony contact is made at the junction of the transverse process and the vertebral body.
- Correct needle placement is confirmed by AP and lateral views followed by radiocontrast dye injection.
- This is followed by sensory and motor stimulation trials to ensure that the phrenic nerve and recurrent laryngeal nerve are not lesioned.
- Sensory stimulation is performed at 50 Hz and 0.9 V, while motor stimulation is done at 2 Hz and 2.0 V.
- During motor stimulation, the patient is asked to vocalize the vowels "aaa" and "eee" to ensure that the vocal cord function is not affected.
- This is followed by injection of 0.5 ml of local anesthetic for lesioning. Ropivacaine is preferred by the authors since it provides a greater sensory block than motor block. After waiting 2–3 min, radiofrequency current is applied for 60–90 s at 80–90° for one cycle.
- The needle is then redirected more medially in the same plane, and the same process is performed there for one cycle.
- Following the second lesion, the needle is again redirected medially and superiorly toward the upper junction of the transverse process and the vertebral body, and the final lesion is performed with the same parameters as before.

Chemical Neurolysis

The administration of a neurolytic agent can be performed via any approach desired. We will describe administration via the anterior C6 and C7 approaches.

- Utilizing the anterior approach at C6 or C7, the operator will perform the procedure in the same fashion.
- Once the needle is correctly placed, the operator should inject 2–3 ml of nonionic, water-soluble contrast under live fluoroscopy. This is to ensure that the spread of the dye is around the vertebra and not intravascular, intrathecal, epidural, or along the longus coli muscle.
- If the dye spread is satisfactory, then a solution containing a mixture of local anesthetic, phenol, and steroid is injected. The total volume of 5 ml should consist of 2.5 ml of 6% phenol in saline, 1 ml of 40-mg triamcinolone, and 1.5 ml of 0.5% ropivacaine (the total 5-ml dose contains a final mixture of 3% phenol) [17].
- After the injection, the patient remains supine with the head elevated slightly for approximately 30 min to prevent complications.

- Patients should be advised of potential complications such as permanent Horner's syndrome or even recurrent laryngeal nerve paralysis.
- It has been advocated that a local anesthetic block prior to the neurolysis may help prevent these complications by observing the patient for 15–30 min, and if the patient develops the Horner's syndrome, the neurolytic block should not be performed. However, even the local anesthetic block cannot predict the outcome of the neurolytic injection. The patient should always be forewarned of the possibility of these complications before stellate ganglion block using the vertebral body approach.

Complications

The proximity of major vascular and neural structures can lead to potential complications as the needle is inserted and the medicines are injected. Complications include the block of neural structures such as the brachial plexus, vagus nerve, recurrent laryngeal nerve, and phrenic nerve. Intravascular injection leading to seizures and loss of consciousness is possible. Retropharyngeal hematoma is possible [18]. Pneumothorax is another potential complication that can occur. Horner's syndrome (ptosis, miosis, and facial anhidrosis or enophthalmos) is another potential complication [19]. In reality, it is more of an expected side effect that occurs with blockade of the upper cervical sympathetics and will resolve within a couple of hours unless neurolysis is performed.

Evidence

According to Day, 11 articles were reviewed on the stellate ganglion block [3]. Ten articles (four case reports, five case series, and one retrospective review) received a 1C recommendation. The eleventh article received a 1B recommendation: strong recommendation, moderate-quality evidence. A recent randomized controlled trial by Salviggio et al. [20] comparing stellate ganglion blocks versus oral medication for facial pain receives a grade of 1B. Kumar et al. performed a randomized, double-blind, placebo-controlled study comparing the analgesic efficacy of preoperative stellate ganglion block on postoperative pain relief after upper limb orthopedic surgery and receives a 1B rating [21].

T_2 and T_3 Sympathetic Block

Indications

- Complex regional pain syndrome type 1
- Complex regional pain syndrome type 2
- Sympathetically mediated pain of the thorax, chest wall, thoracic and upper abdominal viscera
- Hyperhidrosis
- Ischemic pain
- Herpes zoster
- Postherpetic neuralgia

Anatomy

- The second thoracic (T_2) ganglion is commonly found at the head of the second rib near the costovertebral junction. It may also be slightly lateral within the intercostal space at the level of the intervertebral space near the upper border of the third rib [22, 23].
  - The majority of the ganglion is posterior to the parietal pleura; on the left side, it is within close approximation to the aortic arch [22].
- The third thoracic (T_3) ganglion is 17–20 mm dorsal to the ventral surface of the T_3 vertebral body and 2 mm rostral of vertebral bodies near the head of the third rib [24].
- The Kuntz nerve runs 2.3–15.7 mm lateral to the main body of the T_2 sympathetic ganglion [23]. Additional sympathetic connections are formed by the intrathoracic rami and rami communicantes found lateral, posterolateral, or posteromedial to the second and third ventral rami [25].
- Postganglionic fibers leave the sympathetic ganglion and enter the brachial plexus to provide sympathetic innervations to the distal regions of the upper extremity [25].

Procedure

This description will reflect the use of an introducer cannula and blunt, curved block needle. A sharp needle can be used as an alternative without the introducer.

- Place the patient in the prone position. Attach appropriate monitors.
- Sterilely prep and drape the upper thoracic region from the base of the neck to the inferior aspect of the scapula.
- Identify the spinous process of the T_2 and angle the C-arm in the cephalocaudal (image intensifier cephalad) direction to square the superior and inferior end plates. This opens up the rib space to allow visualization of the lateral aspect of the vertebral body below the transverse process of T_2. Mark the skin over the spinous process.
- Oblique the C-arm to the appropriate side approximately 10–15°. The skin entry site is just lateral to the shadow of the bottom half of the vertebral body below the transverse process of T_2 and must not be greater than 4 cm lateral to the spinous process of T_2 viewed in the AP plane. This will theoretically decrease the chance of pneumothorax.
- Using a coaxial technique, anesthetize the skin and insert the introducer cannula. Using spot fluoroscopic images, advance the introducer until it is engaged in tissue.
- Obtain a lateral fluoroscopic image to check the depth of the introducer. Advance the introducer until it is just posterior to the T_2–T_3 foramen. Additional local anesthetic may be required during advancement of the introducer, but do not inject anymore once the introducer reaches the foramen. Local anesthetic at this level may anesthetize the nerve root, and if a sharp needle is used, the patient may not respond should the needle pierce the nerve root.
- Return to the oblique image and check the direction of the needle. Maintaining coaxial technique with the curved tip pointing medially, advance the needle past the foramen until periosteum is touched.
- Recheck the depth of the needle with a lateral image. Rotate the needle tip cephalad and advance the needle. On a lateral image, the final needle tip position should be at the midpoint of the T_2 vertebral body in the cephalocaudal direction and at the junction of the middle and posterior thirds of the T_2 vertebral body in the anteroposterior direction (Fig. 32.7).
- Obtain an AP image. The final position should be with the needle tip touching an imaginary line drawn through the midpoint of the T_2 pedicle shadow.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the T_2 vertebral body and should not change position with respiration (Fig. 32.8). If the location of the contrast ascends and descends with respiration, the needle tip is lateral and needs to be repositioned more medially.
- Once appropriate position is confirmed, inject 3–5 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary but is not contraindicated.

Fig. 32.7 Lateral fluoroscopic image of needles in place for RFTC of the T2 and T3 sympathetic ganglia. Contrast can be seen spreading cephalad and caudad near the tip of the needles

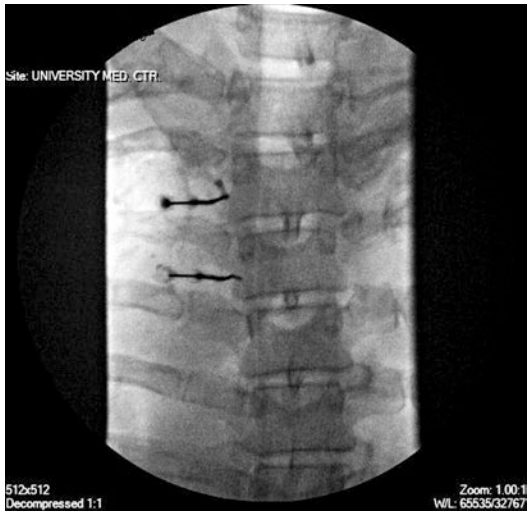

Fig. 32.8 AP fluoroscopic image of needles in place for RFTC of the T2 and T3 sympathetic ganglia

- If there is caudal spread to T_3 ganglion, inject an additional 3–5 ml of the local anesthetic mixture. This will block the T_3 ganglion.
- If there is no caudal spread to T_3, block the T_3 ganglion using the same technique.

Radiofrequency Thermocoagulation

For RFTC of the T_2 and T_3 ganglion, place the needles as above except for the following changes:

- Using a 10-mm active tip, the final needle tip position in the anteroposterior direction should be at the junction of the anterior and middle third of the vertebral body. This allows the active portion of the needle to span the middle third of the vertebral body. This will avoid potential lesioning of the ventral root of T_2 and T_3. The position in the cephalocaudal direction is the same as the block.
- Inject contrast under live fluoroscopy and observe for appropriate spread.
- Once the needle is in correct position, perform sensory (50 Hz up to 0.9 V) and motor (2 Hz up to 2 V) stimulation. No root stimulation should be perceived. The patient may feel a pressure deep in the chest with sensory stimulation. This is normal.
- If root stimulation is felt at a low voltage, the needle needs to be advanced 1–2 mm. Repeat sensory stimulation.
- After appropriate stimulation, inject 3 ml of the local anesthetic solution. Steroid can be included in the mixture to decrease the incidence of neuritis. Higher volumes may spread caudally and block the T_3 ganglion which will make stimulation at that level difficult. Wait 2–3 min and lesion at 80 °C for 90 s. With a curved needle, two lesions are made, one in the cephalomedial direction and one in the caudomedial direction.

Complications

Complications include bruising, bleeding, infection, damage to nerves/spinal cord, neuraxial injection, and pneumothorax. This block should never be performed on a patient with a contralateral pneumothorax or pneumonectomy.

Evidence

There is a paucity of evidence for this block. Two articles for the percutaneous approach received 1C and 2C recommendations [3]. A recent article by Rocha et al. receives a 1B recommendation [26].

Splanchnic Nerves and Celiac Plexus Block

Indications

- Cancer-related pain from the stomach to mid-transverse colon, including the gall bladder, pancreas, spleen, testicle, kidney, and upper ureter
- Nonmalignant pain from the stomach to mid-transverse colon, including the gall bladder, pancreas, spleen, testicle, kidney, and upper ureter

Contraindications

These procedures should not be performed on patients with partial or complete obstruction or perforation of the small or large bowels. Blockade of the sympathetics will allow unopposed parasympathetic outflow, which will increase peristalsis and potentially convert a bowel obstruction into a perforation.

Anatomy

Splanchnic Nerves

- The *greater splanchnic nerve* is the most rostral, originating around the level of the fifth through ninth thoracic vertebrae. It originates from the four roots of the thoracic sympathetic ganglia, descends obliquely, pierces the crus of the diaphragm at a 90° angle, and joins to the celiac plexus [27–29].
- The *lesser splanchnic nerve* is formed by the rami of the ninth through eleventh thoracic sympathetic ganglia. After exiting the ganglia, it travels inferomedial to the vertebral bodies at this level, passes through the crus of the diaphragm, and terminates at the celiac plexus [27, 29].
- The *least splanchnic nerve* is the most caudal, originating from the twelfth thoracic sympathetic ganglion. It travels medially across the vertebral body, passes through the crus of the diaphragm, and terminates at the celiac plexus. Interestingly, this nerve was found to be absent in 43% of dissections, bilaterally [27, 29].
- Individually, each of these nerves contains preganglionic visceral sympathetic efferent neurons from the thoracic sympathetic chain plus postganglionic visceral afferent neurons that provide pain sensation from the upper abdominal organs to the central nervous system [28, 29].

Celiac Plexus

- This large network of nerves, including a right and left ganglia, is a region where pre- and postganglionic neurons from the sympathetic, parasympathetic, and visceral sensory divisions synapse [28, 29].
- This large star- or oval-shaped plexus is 0.5–4.5 cm in diameter and spans a region from the twelfth thoracic vertebra disc space to the middle vertebral body of the second lumbar vertebra [29].
- The main body lays anterolateral to the aorta but also surrounds the celiac artery and superior mesenteric artery [29].
- Afferent fibers concerned with nociception pass diffusely through the celiac plexus and represent the main target of celiac plexus blockade.

Procedure

The techniques for blockade and neurolysis of the splanchnic nerves and the celiac plexus will be described separately. Pretreat the patient with 500 ml of normal saline or lactated Ringer's solution to decrease the incidence of post-procedure postural hypotension from dilation of the abdominal vasculature.

Splanchnic Nerve Block

This is a retrocrural block and involves blocking the greater, lesser, and least splanchnic nerves at the level of the T_{11} vertebra. Depending on the spread of the contrast caudally, a supplemental block may be needed at the level of the T_{12} vertebra in order to block the least splanchnic nerve.

- Identify the T_{11} vertebra, and square the superior and inferior end plates with a caudocephalad tilt of the C-arm.
- Identify the spinous process in the midline and mark.
- Oblique the C-arm to the appropriate side approximately 10–15°. The skin entry site is just lateral to the shadow of the bottom half of the vertebral body below the transverse process of T_{11} and must not be greater than 4 cm lateral to the spinous process of T_{11} viewed in the AP plane. This will theoretically decrease the chance of pneumothorax.
- Using a coaxial technique, anesthetize the skin and insert the introducer cannula. Using

spot fluoroscopic images, advance the introducer until it is engaged in tissue.

- Obtain a lateral fluoroscopic image to check the depth of the introducer. Advance the introducer until it is just posterior to the T_{11}–T_{12} foramen. Additional local anesthetic may be required during advancement of the introducer but do not inject anymore once the introducer reaches the foramen. Local anesthetic at this level may anesthetize the nerve root, and if a sharp needle is used, the patient may not respond should the needle pierce the nerve root.
- Return to the oblique image and check the direction of the needle. Maintaining coaxial technique with the curved tip pointing medially, advance the needle past the foramen until periosteum is touched.
- Recheck the depth of the needle with a lateral image. Rotate the needle tip cephalad and advance the needle. On a lateral image, the final needle tip position should be at the midpoint of the T_{11} vertebral body in the cephalocaudal direction and at the junction of the anterior and middle third of the T_{11} vertebral body in the anteroposterior direction (Fig. 32.9).

Fig. 32.9 Lateral fluoroscopic image of a block needle at T11 for a splanchnic nerve block. Contrast is spreading along the middle of the vertebral body

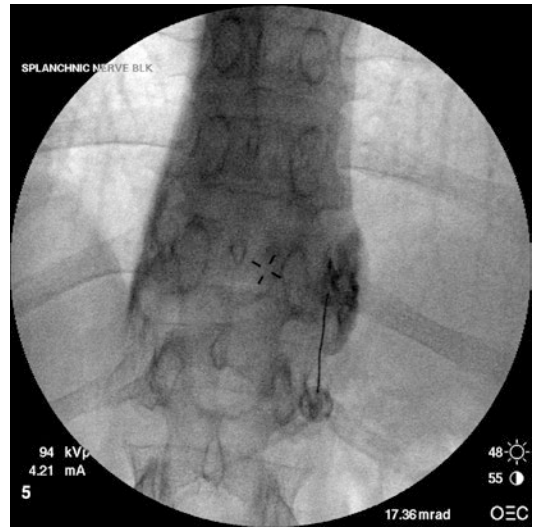

Fig. 32.10 AP fluoroscopic image of a block needle at T11 for a splanchnic nerve block. The contrast is spreading along the lateral aspect of the vertebral body

- Obtain an AP image. The final needle tip position should be medial to the medial aspect of the T_{11} pedicle shadow.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the T_{11} vertebral body and should not change position with respiration (Fig. 32.10). If the location of the contrast ascends and descends with respiration, the needle tip is lateral and needs to be repositioned more medially.
- Once appropriate position is confirmed, inject 5 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.
- If there is caudal spread to T_{12} vertebral body, inject an additional 5 ml of the local anesthetic mixture. This will block the least splanchnic nerve.
- If there is no caudal spread to T_{12} vertebral body, block the least splanchnic nerve at T_{12} vertebral body using the same technique (Figs. 32.11 and 32.12).

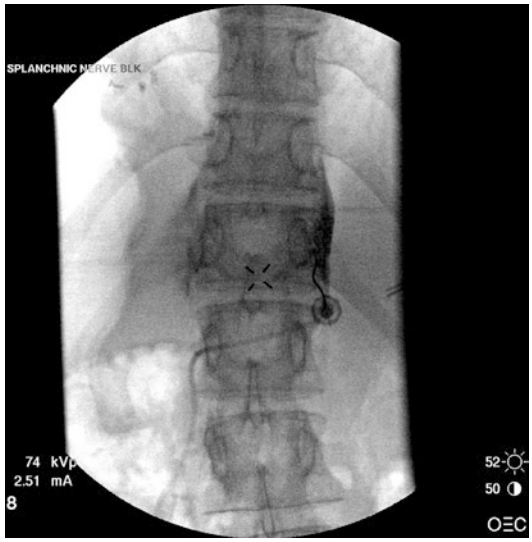

Fig. 32.11 Lateral fluoroscopic image of a block needle at T12 for a splanchnic nerve block

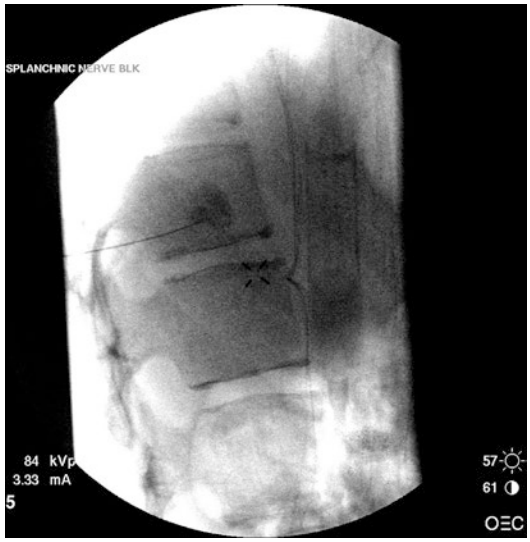

Fig. 32.12 AP fluoroscopic image of a block needle at T12 for a splanchnic nerve block

Radiofrequency Thermocoagulation

For RFTC of the greater, lesser, and least splanchnic nerves, place the needles at T_{11} and T_{12} except for the following changes:

- Using a 10-mm active tip, the final needle tip position in the anteroposterior direction should be across the junction of the anterior and middle third of the vertebral body. This will avoid potential lesioning of the ventral root of T_{11} and T_{12}. The position in the cephalocaudal direction is the same as the block.
- Inject contrast under live fluoroscopy and observe for appropriate spread.
- Once the needle is in correct position, perform sensory (50 Hz up to 0.9 V) and motor (2 Hz up to 2 V) stimulation. No root stimulation should be perceived. The patient may feel a pressure, deep in the abdomen with sensory stimulation. This is normal.
- If root stimulation is felt at a low voltage, the needle needs to be advanced 1–2 mm. Repeat sensory stimulation.
- After appropriate stimulation, inject 5 ml of the local anesthetic solution. Steroid can be included in the mixture to decrease the incidence of neuritis. Higher volumes may spread caudally and block the least splanchnic nerve which will make stimulation at that level difficult. Wait 2–3 min and lesion at 80 °C for 90 s. With a curved needle, two lesions are made, one in the cephalomedial direction and one in the caudomedial direction.

Celiac Plexus Block

There are several approaches used to block the celiac plexus. Prone approaches include retrocrural, anterocrural, and transaortic. The retrocrural and anterocrural approaches require bilateral needles, while the transaortic approach requires only one needle. There is also an anterior approach using ultrasound and CT guidance, which will not be described.

Retrocrural Approach

- Place the patient in the prone position. After sterile prep and drape, identify the tip of the twelfth rib on an AP fluoroscopic image, and raise a skin wheal with local anesthetic.
- Insert the introducer cannula, and angle toward the lower third of the L_1 vertebral body. If a sharp needle is used, this step is omitted.
- A 15-cm, 22- or 20-gauge, curved, blunt block needle is inserted and advanced until

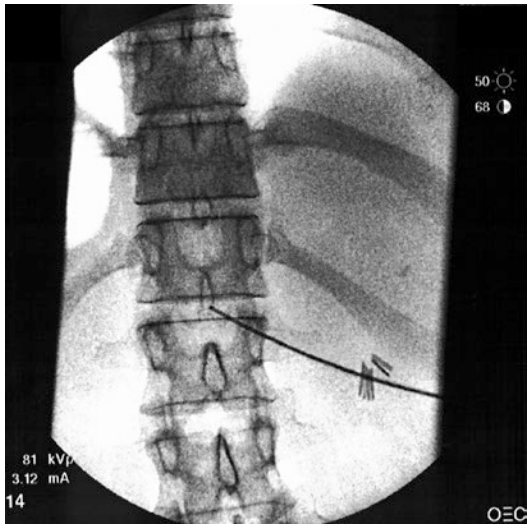

Fig. 32.13 AP fluoroscopic image at L1 for celiac plexus block

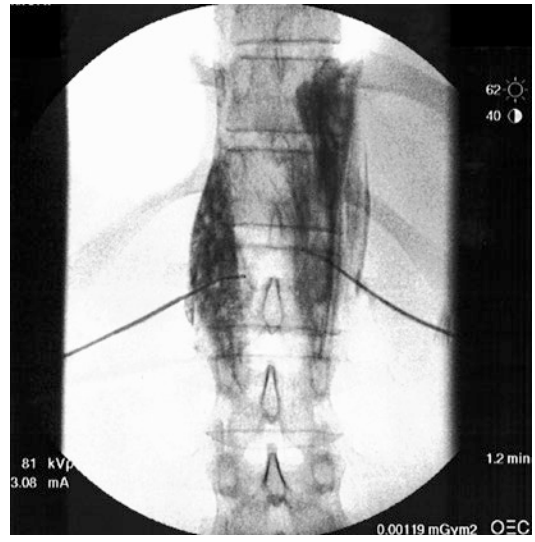

Fig. 32.14 AP fluoroscopic image for celiac plexus block. The contrast is spreading linearly from T11 to L1

the lower third of the L_1 vertebral body is touched. Check the position on a lateral image to make sure the needle is not entering the L_1–L_2 foramen. If not, inject 2–3 ml of local anesthetic.

- Withdraw the needle slightly, and using a steepened angle, advance along the lateral border of the L_1 vertebral body until the needle slips off of the anterior border (Fig. 32.13).
- Using the same technique, insert another needle on the opposite side.
- On a lateral image, advance the right needle 2 cm and the left needle 1 cm past the anterior border of the L_1 vertebral body until aortic pulsations are felt.
- Check the needle position on an AP image. The right needle should be more toward the midline than the left needle as it is more anterior.
- After negative aspiration for blood, urine, and cerebrospinal fluid, inject 2–3 ml of a nonionic, water-soluble contrast through each needle. On the AP image, the contrast should be off of midline over the L_1 vertebral body (Fig. 32.14). The contrast should spread in a linear fashion along the L1 vertebral body on a lateral image (Fig. 32.15).

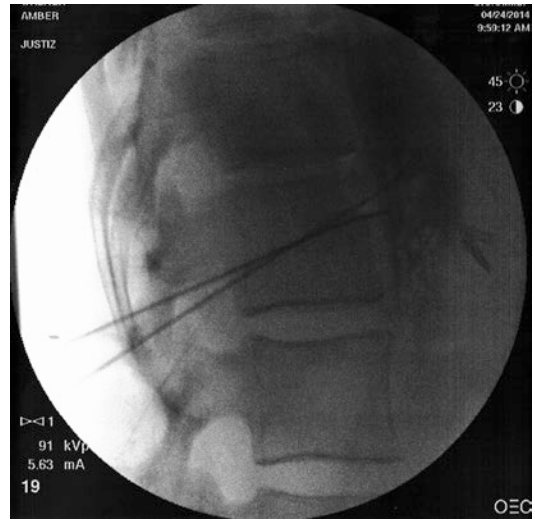

Fig. 32.15 Lateral fluoroscopic image of the final position for celiac plexus block. The right needle is more anterior than the left needle. The contrast is anterior to the L1 vertebral body

- Using 1–2% lidocaine, 0.2% ropivacaine, or 0.25% bupivacaine, inject a 2-ml test dose through each needle to rule out intravascular or intrathecal injection.
- After a negative test dose, 10–15 ml of the aforementioned local anesthetic is injected through each needle.

Transaortic Approach

This is a single-needle approach from the left side. It is best to use a B-bevel needle such as a 20- or 22-gauge Chiba.

- Place the left-sided needle 1 cm past the L_1 vertebral body until aortic pulsations are felt as described in the retrocrural approach.
- Remove the stylet of the needle, and swiftly advance it through the posterior wall, lumen, and anterior wall of the aorta. The needle tip will be in the preaortic area where the celiac plexus lies. To decrease bleeding, a loss of resistance syringe filled with 5 ml of preservative-free normal saline can be attached to the needle and advanced using loss of resistance technique until the preaortic area is entered.
- Check needle tip position with AP and lateral fluoroscopic images. Inject 3–5 ml of nonionic, water-soluble contrast after negative aspiration. It should fill the periaortic area and should not spread retrocrural.
- Once needle tip position is confirmed, inject 10–15 ml of the aforementioned local anesthetics after negative aspiration for blood.

Neurolytic Celiac Plexus Block

After a positive diagnostic block with local anesthetic, a neurolytic block can be performed. Phenol in 6–10% concentrations and anhydrous alcohol in 50–100% concentrations have been used. A total volume of 30–40 ml (15–20 ml on each side) is common for a two-needle technique. The transaortic approach only requires 15 ml of either neurolytic. The injection of alcohol is painful and requires the injection of 5–10 ml of 0.2% ropivacaine or 0.25% bupivacaine prior to its injection. Prior to removal of the needle/s, 2–3 ml of preservative-free normal saline should be used to flush the needle/s.

Failure to flush the needle/s could result in tissue sloughing along the tract of the needle.

Other Techniques

Ultrasound guidance celiac plexus block for cancer patients has been described by multiple authors using and endoscopic approach with the assistance of ultrasound. However, there is a scarcity of literature using US approach percutaneously for the celiac plexus block. Tadros and Elia conducted a 21-patient single institutional study looking at the use of US-guided neurolytic celiac plexus block for the treatment of upper abdominal pain associated with cancer. Patients received 50% ethanol injections and were assessed at weeks 1, 4, and 12 and maintained significant pain relief 3 months post neurolysis. The authors concluded US guidance was a safe effective tool in the assistance of performing a neurolytic celiac plexus block in patients suffering from upper abdominal cancer with no major complications and high success rates [30].

Side Effects and Complications

The difference between a side effect of the procedure and a complication of the procedure must be explained to the patient beforehand. Side effects secondary to sympathetic blockade include hypotension from dilation of the abdominal vasculature and diarrhea from unopposed parasympathetic outflow. Complications include backache, bruising, bleeding (superficial and retroperitoneal), infection, damage to nerves/spinal cord, neuraxial injection, paralysis, pneumothorax, and tissue sloughing from the use of neurolytics.

Evidence

The evidence is strong for use of the splanchnic nerve/celiac plexus block/neurolysis for cancer-related pain. Fifteen articles were identified consisting of one case report; six case series; two retrospective reviews; two prospective, randomized, controlled studies; one randomized, double-blind study; one prospective, randomized, double-blind, controlled study; one prospective, randomized, single-blind, controlled study; and two meta-analysis [3]. The case report, case

series, and retrospective reviews received 1C recommendations, while the rest of the articles received 1B recommendations.

Lumbar Sympathetic Block

Indications [31]

- Complex regional pain syndromes types I and II
- Sympathetic maintained pain
- Phantom limb pain
- Diabetic gangrene
- Phlegmasia
- Arterial insufficiency
- Hyperhidrosis
- Alba dolens
- Erythromelalgia
- Acrocyanosis
- Intractable urogenital pain
- Trench foot
- Frostbite

Anatomy

- Preganglionic afferent neurons synapse to autonomic afferent fibers which innervate the lower extremity and give off visceral branches to the lumbar splanchnic nerves.
- There is considerable variation in size, number, and position on the vertebral bodies of the ganglia. On average this chain contains 3–4 ganglia and is usually located between the second and fourth lumbar vertebra [32–34].
- The ganglia are cylindrical or elliptical in shape and are intertwined within the fascicles of the medial border of the psoas major muscle [35–37].
- In the horizontal plane, the ganglia reside 0–0.5 cm posterior to the anterior border and 1.8–3.0 cm laterally from the center of the third lumbar vertebra [35].
- On the right side of the vertebral column, the chain lies posterior to the inferior vena cava, while on the left side, it is posterior to the para-aortic nodes. As the chain continues caudally, it passes posterior to the common iliac artery [37].

Procedure

Traditionally, two approaches have been described for the lumbar sympathetic block: the classic approach described by Kappis and Mandal and the lateral approach described by Mandal and Reed. With the advent of fluoroscopy, there is now a new so-called modern approach. Secondary to its safety and ease of performing, we will describe the modern (oblique-view) approach. This description will reflect the use of an introducer cannula and blunt, curved block needle. A sharp needle can be used as an alternative without the introducer.

Modern Approach

- Place the patient in the prone position. Attach appropriate monitors.
- Sterilely prep and drape the lumbar region.
- Identify the vertebral body of interest in an AP view, and square off the vertebral end plates. For a single-needle approach, L_3 is usually the target, but if the pain is located in the lower leg or foot, L_4 or sometimes L_5 may need to be the targeted level.
- Oblique the C-arm to the appropriate side approximately until the transverse process is situated just lateral to the vertebral body.
- Once the target is identified, a small skin wheal with local anesthetic is raised.
- Using a coaxial technique, anesthetize the skin and insert the introducer cannula. Using spot fluoroscopic images, advance the introducer until it is engaged in tissue.
- Obtain a lateral fluoroscopic image to check the depth of the introducer. Advance the introducer until it is just posterior to the vertebral body foramen. Additional local anesthetic may be required during advancement of the introducer.
- Return to the oblique image and place the blunt, curved block needle. Maintaining coax-

ial technique with the curved tip pointing medially, advance the needle past the foramen until periosteum is touched.

- Recheck the depth of the needle with a lateral image. Rotate the needle tip medial or lateral and advance the needle. On a lateral image, the final needle tip position should be at the anterior vertebral body.
- Obtain an AP image. The final position should be with the needle tip touching an imaginary line drawn through the medial aspect of the pedicle shadow at that level.
- After negative aspiration for air, blood, urine, and CSF, inject 2–5 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the vertebral body (Fig. 32.16). On lateral view, the solution should spread caudad and cephalad anterior to the vertebral body (Fig. 32.17). On an AP image, the contrast should appear to spread medially, cephalad, and caudad directions while hugging the vertebral body (Fig. 32.16). If the needle tip is too lateral, contrast may spread into the origins of the psoas muscle, and the needle will need to be repositioned more medially.

Fig. 32.16 AP fluoroscopic image of a block needle at L3 for a lumbar sympathetic block. Contrast is spreading along the anterolateral vertebral body

Fig. 32.17 Lateral fluoroscopic image of a block needle at L3 for a lumbar sympathetic block. Contrast is spreading along the anterolateral vertebral body. Calcifications in the aorta can be seen anterior to the vertebral bodies

- Once appropriate position is confirmed, inject 10–20 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration [38]. Steroid is not necessary but is not contraindicated.

Radiofrequency Thermocoagulation

For RFTC of the lumbar sympathetic chain, place the needles as described above using the modern approach except for the following changes. The needle position will vary at each independent vertebral level with the location of the appropriate ganglions. Lesion will be performed at the inferior one third of the L_2 vertebral body, upper one third of the L_3 vertebral body, and middle of the L_4 vertebral body [34, 35, 39–41].

- Using a 10-mm active tip on a curved, blunt electrode, the final needle tip position in the lateral fluoroscopic view should be at the anterior portion of each of the respective vertebral bodies intended to be blocked. In an AP image, the needle tips should lie on an imaginary line at or near the medial aspect of the pedicle.
- Inject 2 ml of contrast under live fluoroscopy and observe for appropriate spread.

- Once the needle is in correct position, perform sensory (50 Hz up to 1.0 V) and motor (2 Hz up to 2 V) stimulation.
- With sensory stimulation, the patient may feel a pressure or discomfort in the lumbar region with 0.2–0.5 V; this is normal. If paresthesia is elicited in the groin region, the needle must be repositioned as it is situated too close to the genitofemoral nerve.
- Motor stimulation should not elicit any motor response in the lower extremities.
- 1–2 ml of local anesthetic (lidocaine 2%) is given; wait 2–3 min before lesioning.
- With a curved needle, two lesions are made, one in the cephalomedial direction and one in the caudomedial direction. Rotate the electrode tip cephalad and medial direction and lesion for 60–90 s at 80–90° for one cycle. Then, rotate the electrode tip caudal and medial direction, and perform another cycle for 60–90 s at 80–90°.
- Lesions should be performed at the inferior one third of the L_2 vertebral body, the superior one third of the L_3 vertebral body, and the middle of the L_4 level vertebral body.

Neurolytic Lumbar Sympathetic Plexus Block

Chemical neurolysis can be accomplished in a similar fashion to the local anesthetic block with same-needle placement technique. Needle placement is confirmed with nonionic, water-soluble contrast prior to injection of the neurolytic. This is followed by 2–3 ml of 6–12% phenol per level when using multiple-needle technique or a larger volume of 15–20 ml through a single needle. Prior to removing the needle/s, 2–3 ml of preservative-free normal saline should be used to flush the needle/s to prevent tracking of the neurolytic agent [40, 42].

Complications

Like many procedures performed, a host of complications can occur. With proper and safe technique, many of these complications can be avoided. The most common complications will be post-procedural discomfort in the lumbar region for 3–5 days, retrograde ejaculation (bilateral sympathectomy), genitofemoral neuralgia [43], kidney or ureteral damage [44], and intravascular and subarachnoid injections.

Evidence

Continuing with Day's evidence-based article, 11 papers were graded. Four case reports and five case series earned 1C recommendations [3]. The remaining two articles, one a prospective randomized trial and the other a prospective randomized controlled trial, received 1B recommendations.

Superior Hypogastric Plexus Block

Indications

- Cancer-related pain in the bladder, vagina, penis, rectum, anus, and perineum
- Pain related to nonmalignant conditions such as endometriosis, pelvic adhesions, pelvic inflammation, interstitial cystitis, irritable bowel syndrome, proctalgia fugax, vulvodynia

Anatomy

- These plexuses contain efferent pre- and postganglionic sympathetic, preganglionic parasympathetic, and afferent visceral sensory nerve fibers.
- The *superior hypogastric plexus* is embedded in connective tissue anterior to the mid body of the fifth lumbar vertebra and sacral promontory, positioned anterior to the aortic bifurcation, left common iliac vein, and medial sacral vessels [45]. It is in close proximity to the roof of the sigmoid colon mesentery with the attachment point of the mesocolon left of the plexus [37]. The plexus then branches to form the left and right hypogastric nerves. Postganglionic sympathetic and parasympathetic fibers innervate the pelvic organs as they exit the superior plexus.

- The *inferior hypogastric plexus* forms a triangular structure with the following landmarks: (1) the cephalad edge runs parallel to the hypogastric artery, (2) the caudal edge stretches from the fourth sacral root to the ureter entry point at the broad ligament, and (3) the dorsal edge runs along the ventral surface of the sacrum close to the S_2–S_4 nerve roots [46]. Postganglionic sympathetic, parasympathetic, and visceral sensory nerves provide innervations to the bladder, prostate, penis, uterus, vagina, and rectum [46, 47].

Procedure

As with the other sympathetic blocks, there are several approaches for blocking the superior hypogastric plexus: traditional, medial, and transdiscal. The trans-sacral foramina approach actually blocks the inferior hypogastric plexus and will not be described. The hypogastric plexus block needs to be performed with fluoroscopic, CT, and ultrasound guidance. Only fluoroscopic techniques will be described in this chapter. A 15-cm, 20- or 22-gauge, curved, sharp, or blunt needle is used. The patient is placed in the prone position with pillows placed under the lower abdomen to reverse the lumbar lordosis.

Traditional Two-Needle Approach

- Sterilely prep and drape the lower lumbar and sacral region.
- Identify the L_4–L_5 interspace, and tilt the C-arm in the cephalocaudal direction to square the inferior end plate of L_4 and the superior end plate of L_5.
- Raise a skin wheal with local anesthetic approximately 5–7 cm lateral to the L_4–L_5 interspace.
- Insert an introducer cannula through the skin wheal, angling 30–45° medially and caudally. If using a sharp needle, this step is omitted.
- Insert the block needle through the cannula, and advance toward the inferior, anterolateral aspect of the L_5 vertebral body. Check the depth of the needle with a lateral image.
- Adjust the angle of the needle until the tip walks off the anterior edge of the L_5–S_1 interspace. The transverse process of L_5 may sometimes be encountered and requires the initial angle of the needle to be steeper.
- On an AP image, the needle tip should be medial to an imaginary line drawn through the medial aspect of the lumbar pedicle shadows and extending caudally through the sacrum.
- Repeat the procedure on the opposite side using the same technique.
- Under continuous lateral fluoroscopy and after negative aspiration for blood and CSF, inject 2 ml of nonionic, water-soluble contrast through each needle. The contrast should spread caudally in a curvilinear fashion over the anterior aspect of the L_5–S_1 disc and sacral promontory (Fig. 32.18). The AP view should show contrast over the upper portion of the sacrum extending caudally (Fig. 32.19).
- The block is performed with 8–10 ml of 1–2% lidocaine or 0.2% ropivacaine or 0.25% bupivacaine.

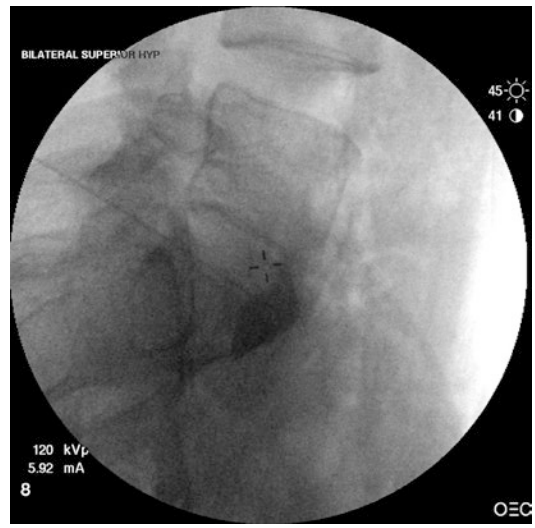

Fig. 32.18 Lateral fluoroscopic image of a bilateral superior hypogastric plexus block using the classic approach. The contrast is spreading over the sacral promontory

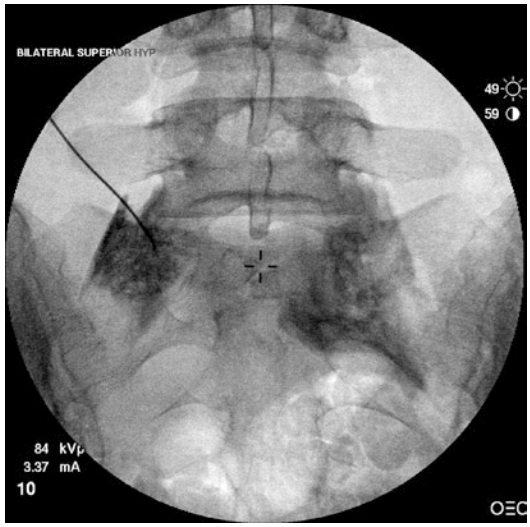

Fig. 32.19 AP fluoroscopic image of a bilateral superior hypogastric plexus block using the classic approach. The contrast is spreading caudally

Fig. 32.20 AP fluoroscopic image of a needle in place for the superior hypogastric plexus block using the L5 paramedian approach

Paramedian Two-Needle Approach

This approach is very similar to blockade of the L5 sympathetic ganglion, except the target is the inferior aspect of the L5 vertebral body at the L_5–S_1 disc. This technique can be used if the practitioner attempts the traditional approach and has difficulty maneuvering past the L_5 transverse process.

- Square the inferior end plate of L_5 and the superior aspect of the sacrum.
- Oblique the C-arm ipsilaterally, stopping just before the shadow of the inferior, lateral aspect of the L_5 vertebral body overlaps the iliac crest.
- Raise a skin wheal with local anesthetic over the inferior, lateral aspect of L_5.
- Insert the introducer cannula in a coaxial fashion, and check the depth with a lateral image.
- Insert the curved, blunt block needle through the introducer.
- Return to the oblique view, and advance the block needle checking its direction with spot images.
- Once bone is touched, turn the tip caudally and advance the needle on a lateral image.
- As the needle is advanced, turn the tip medially to confirm that the needle is still on bone. Advance the needle until the tip is just past the inferior, anterolateral edge of the L_5 vertebral body.
- On the AP view, the tip of the needle should be medial to the medial aspect of the L_5 pedicle (Fig. 32.20).
- Repeat the procedure on the opposite side using the same technique.
- Under continuous lateral fluoroscopy and after negative aspiration for blood and CSF, inject 2–3 ml of nonionic, water-soluble contrast through each needle. The contrast should spread caudally in a curvilinear fashion over the L_5–S_1 disc and sacral promontory (Fig. 32.21). The AP view should show contrast over the upper portion of the sacrum extending caudally (Fig. 32.22).
- The block is performed with 8–10 ml of 1–2% lidocaine or 0.2% ropivacaine or 0.25% bupivacaine.

Transdiscal Approach

This is a one-sided (left) approach. A double-needle technique is used.

- Square the inferior end plate of L_5 and the superior aspect of the sacrum with a cephalocaudal tilt of the C-arm.

Fig. 32.21 Lateral fluoroscopic image of a needle in place for the superior hypogastric plexus block using the L5 paramedian approach. The contrast is spreading over the sacral promontory

Fig. 32.22 AP fluoroscopic image of a needle in place for the superior hypogastric plexus block using the L5 paramedian approach. The contrast is spreading caudally

- Oblique the C-arm toward the left until an inverted triangle is created with the shadows of the superior end plate of L5, the lateral aspect of the superior articular process of the sacrum, and the iliac crest.
- Raise a skin wheal over the shadow of the lateral aspect of the superior articular process of the sacrum.
- Insert the introducer cannula in a coaxial fashion at the midpoint of the lateral aspect of the superior articular process of the sacrum.
- Insert the curved, sharp, or blunt block needle through the introducer cannula, and advance in a coaxial fashion toward the disc using spot images.
- Check a lateral image and advance until the tip of the needle is posterior to the L_5–S_1 foramen.
- Return to the oblique view and check to make sure the needle is coaxial. If not, withdraw the needle slightly and redirect medial.
- On a lateral view, advance the needle into and through the disc until the needle tip just exits the anterior portion of the disc.
- Check an AP image. The needle tip should be in the same position as described in the aforementioned techniques.
- Inject contrast and observe for proper spread (Fig. 32.23). The block is performed with

Fig. 32.23 Lateral fluoroscopic image of a needle in place for the superior hypogastric plexus block using the L5–S1 transdiscal approach. The contrast is spreading over the sacral promontory

8–10 ml of 1–2% lidocaine or 0.2% ropivacaine or 0.25% bupivacaine.
- As the needle is withdrawn, inject antibiotic (cefazolin is common) into the disc in order to avoid discitis.

Neurolytic Hypogastric Plexus Block

Chemical neurolysis can be accomplished with 5–8 ml of 6–10% phenol or 50–100% anhydrous alcohol. Confirm proper needle placement with nonionic, water-soluble contrast prior to the injection of either neurolytic. Prior to removal of the needle/s, 2–3 ml of preservative-free normal saline should be used to flush the needle/s. Failure to flush the needle/s could result in tissue sloughing along the tract of the needle.

Ultrasound Hypogastric Plexus Block

US guidance for the superior hypogastric plexus block has been described by several authors. Although we won't describe the actual technique in this section, we will present data with relevance to the procedure and its use in the superior hypogastric plexus block. Gofeld et al. in an experimental cadaver study describes successful placement and spread of injectate using US-guided approach in a supine position. Bilateral spread was attained with real-time observation of injection. Final needle location and spread of a radiopaque contrast was confirmed by fluoroscopy. Gofeld concluded that a modified US technique resulted in a similar spread of injectate as the traditional fluoroscopy-guided technique and in a clinical scenario would offer complete block of the superior hypogastric plexus [48].

Mishra and colleagues performed a study on 50 patients diagnosed with pelvic cancer pain to assess the efficacy of anterior US-guided superior hypogastric plexus block with neurolytic. The study assessed narcotic treatment vs superior hypogastric plexus neurolysis. There was a significant decrease in pain in the neurolytic block group compared to the group that received narcotics only. The conclusion of authors was that US-guided injection is a useful technique that avoids radiation exposure; however, the procedure requires technical expertise when being performed [49].

Complications

The proximity of the iliac vessels increases the potential for vessel puncture or intravascular injection. Other complications include backache, bruising, bleeding (superficial and retroperitoneal), infection, damage to nerve roots, neuraxial injection, paralysis, discitis, rectal puncture, and tissue sloughing from the use of neurolytics.

Evidence

Six articles were identified for pelvic cancer pain [3]. One case report and five case series were graded as 1C, and a prospective randomized trial earned the grade of 1B. There were three case reports and one case series for noncancer-related pelvic pain, and all were graded as 1C.

Ganglion Impar Block (Ganglion of Walther)

Indications [50–54]

- Indications for ganglion impar block
- Perineal pain
- Pain secondary to endometriosis
- Phantom limb pain
- Complex regional pain syndromes types I and II
- Proctalgia fugax
- Radiation enteritis
- Rectal pain
- Pelvic pain
- Genital pain

Anatomy

- The ganglion impar receives preganglionic fibers from the sacral portion of the sympathetic trunk, while postganglionic fibers carry sympathetic outflow to the perineum via the pudendal nerve [55]. Additional innervations

by the parasympathetic and visceral sensory nervous systems have been reported.
- It is a single midline structure located anterior to the level of the sacrococcygeal junction, or in some cases, it can be more caudal at the midpoint of the coccyx [55–57].
- The ganglion can be oval, irregular, triangular, or rectangular with a mean length of 0.7–4.4 mm depending on the shape [56].
- In a cadaveric study with an $n = 50$, 18% of ganglia were located at the sacrococcygeal junction, while 46% of the ganglia were located from 20 to 30 mm from the tip of the coccyx [56].

Procedure

There are several techniques for blockade of the ganglion impar. This section will focus on two techniques with various approaches: the lateral and prone.

Lateral Technique [58]

- Patient is placed in the lateral decubitus with knees flexed toward the chest.
- C-arm is placed in an AP direction to the table and rotated accordingly until the sacrum is visualized in a true lateral view. The target will be located at the fifth sacral vertebrae just cephalad to the sacrococcygeal junction.
- A 22-gauge, 3.5-in. spinal needle with a small bend distally is advanced toward the sacral-coccyx junction midline and toward the sacrum to avoid the posterior rectal wall.
- AP view is taken to ensure the needle tip is midline. After negative aspiration for blood, inject 2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a cephalad and caudal fashion along the S_5 vertebral body in the precoccygeal space.
- Once appropriate position is confirmed, inject 4–6 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.
- This technique is useful when there is calcification of the sacrococcygeal ligament.

Prone Technique

Trans-Sacrococcygeal Junction Approach [59, 60]

- Patient is placed in the prone position, and an AP view is used to visualize the sacrococcygeal junction.
- A 22-gauge, 3.5-in. spinal needle is advanced in an AP view in coaxial fashion through the sacrococcygeal ligament junction.
- A lateral view is obtained, and the needle position should lie just past the sacrococcygeal ligament (Fig. 32.24).
- After negative aspiration for blood, inject 2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a cephalad and caudal fashion along the S_5 vertebral body in the precoccygeal space (Fig. 32.25).
- Once appropriate position is confirmed, inject 4–6 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.

Paramedian Approach [61, 62]

- Patient is placed in the prone position, and an AP view is used to visualize the sacrococcygeal junction and the coccyx.
- A 22-gauge, 3.5-in. spinal needle with a small bend (20–30°) distally or multiple bends is

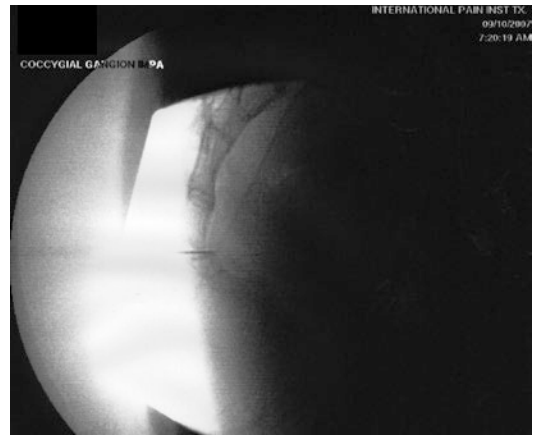

Fig. 32.24 Lateral fluoroscopic image of a needle through the sacrococcygeal juncture for ganglion impar block

Fig. 32.25 Contrast spread in the shape of a "comma" along the anterior edge of the sacrum

placed inferior to the sacrococcygeal junction just lateral and inferior to the junction of the transverse process and body of coccyx in an AP view [63].

- Once bony contact is made, the needle is rotated laterally and advanced slightly until it slips off the coccygeal body.
- A lateral view is then taken, and the needle tip should lie on the posterior one third of the vertebral body. The needle is then rotated in a caudal and medial direction and advanced until the tip is just anterior to the coccyx. At this point, the needle should lie within the precoccygeal space.
- Another AP image is taken to ensure the needle tip is positioned near midline. After negative aspiration for blood, inject 2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a cephalad and caudal fashion along the S_5 vertebral body in the precoccygeal space.
- Once appropriate position is confirmed, inject 4–6 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.
- This technique is useful when there is calcification of the sacrococcygeal ligament.

Radiofrequency Thermocoagulation and Pulsed Radiofrequency

For radiofrequency thermocoagulation (RFTC) [64] and pulsed electromagnetic field radiofrequency (P-EMF) [65] of the ganglion impar, any of the aforementioned techniques can be performed and modified with the use of a 20- or 22-gauge, 5-mm active tip radiofrequency probe. The trans-sacrococcygeal approach, lateral approach, paramedian approach, or a combination of two can be used. For simplicity, we will describe the trans-sacrococcygeal approach.

- Once the target is identified, a 22-gauge, 5-mm active tip radiofrequency probe is advanced in an AP view in coaxial fashion through the sacrococcygeal junction.
- Correct needle placement is confirmed by AP and lateral views followed by radiocontrast dye injection.
- This is followed by sensory and motor stimulation trials. Sensory stimulation is performed at 50 Hz and 1.0 V, while motor stimulation is done at 2 Hz and 3.0 V.
- This is followed by injection of 1.0 ml of local anesthetic for lesioning. After waiting 2–3 min, radiofrequency current is applied for 60–90 s at 80–90° for two to three cycles. With each cycle, the needle tip should be rotated 90°.
- For P-EMF, the size of the active tip is not important as the electromagnetic field is projected from the tip of the needle and not from the shaft. With P-EMF lesioning, two to four 120-s lesions are performed at 45 V. Local anesthetic is not required for P-EMF.

Neurolytic Ganglion Impar Block

The administration of a neurolytic agent can be performed via any approach desired. We will describe administration via the trans-sacrococcygeal approach.

- Utilizing the trans-sacrococcygeal approach, the operator will perform the procedure in the same fashion.
- Once the needle is correctly placed, the operator should inject 1–2 ml of nonionic, water-

soluble contrast under live fluoroscopy. This is to ensure that the spread of the dye is around the desired area.

- If the dye spread is satisfactory, then a solution containing a mixture of local anesthetic, phenol, and steroid is injected. The total volume of 5 ml should consist of 2.5 ml of 6% phenol in saline, 1 ml of 40-mg triamcinolone, and 1.5 ml of 0.5% ropivacaine or 0.5% bupivacaine. The total 5-ml dose contains a final mixture of 3% phenol [17]. Alternatively, similar amounts of 6–10% phenol or anhydrous alcohol can be used.

Other Approaches for Ganglion Impar Block

This section has included traditional approaches and treatments for the performance of the ganglion impar block; however, there are a variety of new and different approaches that will be briefly discussed. Traditionally, the ganglion impar block has been performed under fluoroscopy, but with the advent of ultrasound and other imaging techniques such as magnetic resonance imaging (MRI) and computer tomography (CT) into interventional pain, these techniques have proved successful in the treatment of various pain conditions associated with the ganglion of Walther. The use of ultrasound (US) guidance technique for ganglion impar block has become more common place given the multiple advantages of less radiation exposure, and ease of access over other modalities. Lin and colleagues describe the use of US-guided technique in 15 patients that the sacrococcygeal ligament was not easily visible via fluoroscopy. All injections were accurately and successfully performed with US guidance; needle depth and contrast spread was confirmed with fluoroscopy [66].

The ganglion impar block has also been described with the use MRI on cadaveric studies. Six MRI-guided injections were successfully performed on human cadavers. The study showed that the ganglion impar was easily visualized in 66% of cases and was accessed successfully 100% revealed by appropriate periganglionic dispersal and filling of the presacrococcygeal space. The authors concluded that interventional MRI can visualize and directly target the ganglion impar for accurate needle placement and successful periganglionic injection with the additional benefit of no ionizing radiation exposure to patient and staff [67].

CT-guided approaches have been demonstrated for the treatment of coccydynia. Datir and colleagues performed ganglion impar blocks on eight subjects with coccydynia. Technically, all patients had a successful ganglion impar block without any complications, and the procedure was tolerated well. The authors determined that CT-guided injection of the ganglion impar block was more accurate, less risk of complications compared to fluoroscopic approaches [57].

Complications

The proximity of the ganglion impar to the adjacent structures can lead to potential complications as the needle is inserted. The rectum lies just anterior to the precoccygeal space, and inadvertent puncture can lead to perforation and fistula formation. Other possible complications include epidural spread of agent, neurolytic injection into nerve roots or the rectum, neuritis, cauda equina syndrome, tracking of contaminants back through the needle, and infection.

Evidence

Two case reports for cancer-related pain were graded as 1C [3]. One case report and one prospective case series for nonmalignant pain were also graded as 1C. In 2008, Agarwal et al. [55] published a case series of 43 patients who received CT-guided blocks and chemical neurolysis of the ganglion impar for both malignant and nonmalignant pain. It is graded as 1C. In the article, 12 additional ganglion impar blockade articles were reviewed which included the 4 mentioned in Day's paper. All were case reports and case series and were graded as 1C evidence.

Conclusion

Sympathetic blocks can be useful tools in the management of chronic malignant and nonmalignant pain. Neurolytic and radiofrequency procedures can provide longer relief when the diagnostic blocks have been successful.

The majority of the available evidence is case series and case reports. The charge to current and future pain physicians is to implement well-designed clinical studies that support the need and use of these blocks.

Clinical Pearls

The practitioner should proceed with the block once an appropriate diagnosis is made, and the patient is an acceptable candidate. In certain patients, coagulation parameters should be checked. Knowledge of the anatomy around the targeted ganglia/ganglion is key. Proper needle placement is paramount to increase the success of the block and to decrease the incidence of untoward events. This includes observing for appropriate contrast spread and the absence of intravascular or neuraxial spread.

Review Questions

1. Prior to radiofrequency of the splanchnic nerves, sensory stimulation is carried out. Where will the patient perceive the stimulation?
   (a) Lower ribs in a dermatomal distribution
   (b) Pelvis
   (c) Deep in the abdomen
   (d) Sacrum
2. Which of the following is a side effect of a splanchnic/celiac block?
   (a) Backache
   (b) Neuraxial injection
   (c) Pneumothorax
   (d) Diarrhea
3. Sensory stimulation of the sphenopalatine ganglion will produce a paresthesia where?
   (a) Root of the nose
   (b) Posterior pharynx
   (c) Lower teeth
   (d) Hard palate
4. The target for a thoracic sympathetic block at T2 and T3 is:
   (a) The midpoint to posterior one third of the vertebral body in the anterior-posterior direction
   (b) The junction of the middle and lower third of the vertebral body in the cephalocaudal direction
   (c) The midpoint of the vertebral body in the cephalocaudal direction
   (d) The junction of the anterior and middle third of the vertebral body in the anterior-posterior direction
5. The appropriate contrast pattern on a lateral fluoroscopic view for a hypogastric plexus block is
   (a) Cephalad toward the L4–L5 interspace
   (b) Over the anterior aspect of the L5 vertebral body
   (c) Over the sacral promontory at the L5–S1 interspace
   (d) Caudal towards S2–S3
6. The final needle tip position for the transsacrococcygeal approach for the ganglion impar is:
   (a) Just anterior to the sacrococcygeal junction
   (b) Just posterior to the sacrococcygeal junction
   (c) 1 cm anterior to the sacrococcygeal junction
   (d) In the sacrococcygeal joint
7. The parasympathetic component of the sphenopalatine ganglion originates from what brainstem nucleus?
   (a) Superior salivatory nucleus
   (b) Inferior salivatory nucleus
   (c) Nucleus ambiguus
   (d) Nucleus caudalis

8. The anterior tubercle of C6 is commonly referred to as:
   (a) Anderson's tubercle
   (b) Chassaignac's tubercle
   (c) Chavira's tubercle
   (d) Silverman's tubercle
9. Which of the following is a component of a Horner's syndrome?
   (a) Exophthalmos
   (b) Mydriasis
   (c) Tongue deviation
   (d) Ptosis
10. Kuntz fibers are located at which vertebral body levels?
   (a) T1 and T2
   (b) T3 and T4
   (c) T2 and T3
   (d) C7 and T1
11. For a splanchnic nerve block at T11, the entry site of the needle should not be greater than 4 cm from the midline to avoid what complication?
   (a) Puncture of the aorta
   (b) Pneumothorax
   (c) Nerve root injury
   (d) Neuraxial injection
12. Because of its anatomical location, which of the following structures is more likely to be punctured when performing a left stellate ganglion block as compared to a right stellate ganglion block?
   (a) Trachea
   (b) Esophagus
   (c) Thyroid gland
   (d) Carotid artery
13. The transaortic neurolytic block of the celiac plexus requires what volume of neurolytic?
   (a) 5 ml
   (b) 10 ml
   (c) 15 ml
   (d) 20 ml
14. To perform a radiofrequency neurotomy of the L2, L3, and L4 sympathetic ganglia, where on the vertebral bodies should the needle tips be placed?
   (a) Middle of L2, middle of L3, and middle of L4
   (b) Upper one third of L2, lower one third of L3, and middle of L4
   (c) Lower one third of L2, upper one third of L3, and lower one third of L4
   (d) Lower one third of L2, upper one third of L3, and middle of L4
15. While performing sensory stimulation prior to radiofrequency lesioning of the L3 sympathetic ganglion, the patient feels a paresthesia in the groin. What is the appropriate next step?
   (a) Reposition the needle as it is close proximity to the genitofemoral nerve and retest.
   (b) Place local anesthetic and proceed with the lesioning as this is an expected response.
   (c) Proceed with lesioning as the sympathetic ganglion is not a sensory nerve and does not require local anesthetic.
   (d) Proceed with motor stimulation and if no motor response, proceed with lesioning.

Answers

1. c
2. d
3. a
4. b
5. c
6. a
7. a
8. b
9. d
10. c
11. b
12. b
13. c
14. d
15. a

References

1. Guyatt G, Gutterman D, Bauman M, et al. Grading strength of recommendation and quality of evidence in clinical guidelines: report from an American College of Physician's Task Force. Chest. 2006;129:174–81.
2. Konen A. Unexpected effects due to radiofrequency thermocoagulation of the sphenopalatine ganglion: two case reports. Curr Rev Pain. 2000;10:30–3.

3. Day M. Sympathetic blocks: the evidence. Pain Pract. 2008;8:98–109.
4. Narouze S, Kapural L, Casanova J, Mikhail N. Sphenopalatine radiofrequency ablation for the management of chronic cluster headache. Headache. 2009;49:571–7.
5. Lipov EG, Joshi JR, Sanders S, Slavin KV. A unifying theory linking the prolonged efficacy of the stellate ganglion block for the treatment of chronic regional pain syndrome (CRPS), hot flashes, and posttraumatic stress disorder (PTSD). Med Hypotheses. 2009;72:657–61.
6. Peterson RC, Patel L, Cubert K, Gulati A. Serial stellate ganglion blocks for intractable postherpetic itching in a pediatric patient: a case report. Pain Physician. 2009;12:629–32.
7. Chester M, Hammond C, Leach A. Long-term benefits of stellate ganglion block in severe chronic refractory angina. Pain. 2000;87:103–5.
8. Ateş Y, Asik I, Ozgencil E, et al. Evaluation of the longus coli muscle in relation to stellate ganglion block. Reg Anesth Pain Med. 2009;34:219–23.
9. Narouze S, Vydyanathan A, Patel N. Ultrasound-guided stellate ganglion block successfully prevented esophageal puncture. Pain Physician. 2007;10:747–52.
10. Pather N, Partab P, Singh B, Satyapal KS. Cervicothoracic ganglion: its clinical implications. Clin Anat. 2006;19:323–6.
11. Feigl GC, Rosmarin W, Stelzl A, et al. Comparison of different injectate volumes for stellate ganglion block: an anatomic and radiologic study. Reg Anesth Pain Med. 2007;32(3):203–8.
12. Narouze S. Letter to the editor: Beware of the "serpentine" inferior thyroid artery while performing stellate ganglion block. Anesth Analg. 2009;109:289–90.
13. Bhatia A, Flamer D, Peng PW. Evaluation of sonoanatomy relevant to performing stellate ganglion blocks using anterior and lateral simulated approaches: an observational study. Can J Anaesth. 2012;59:1040–7.
14. Gofeld M, Bhatia A, Abbas S, Ganapathy S, Johnson M. Development and validation of a new technique for ultrasound-guided stellate ganglion block. Reg Anesth Pain Med. 2009;34:475–9.
15. Joeng ES, Jeong YC, Park BJ, Kang S, Yang SN, Yoon JS. Sonoanatomical change of phrenic nerve according to posture during ultrasound-guided stellate ganglion block. Ann Rehabil Med. 2016;40:244–51.
16. Anderson SR. T2 and T3 sympathetic block and neurolysis. In: Raj PP, Lou L, Erdine S, Staats PS, Waldman SD, editors. Radiographic imaging for regional anesthesia and pain management. Philadelphia, PA: Churchill Livingstone; 2003. p. 132–7.
17. Racz G. Techniques of neurolysis. Boston: Kluwer Academic; 1989.
18. Higa K, Hirata K, Hirota K, Nitahara K, Shono S. Retropharyngeal hematoma after stellate ganglion block. Anesthesiology. 2006;105:1238–45.
19. Hoşten T, Gürkan Y, Solak M, Toker K. A case of Horner's syndrome following lateral sagittal infraclavicular block. Agri. 2008;20:45–8.
20. Salviggio I, Adducci E, Dell'Aquila L, Rinaldi S, Marini M, Zappia L, et al. Facial pain: a possible therapy with stellate ganglion block. Pain Med. 2008;9:958–62.
21. Kumar N, Thapa D, Gombar S, Ahuja V, Gupta R. Analgesic efficacy of pre-operative stellate ganglion block on postoperative pain relief: a randomized controlled trial. Anaesthesia. 2014;69:954–66.
22. Singh B, Ramsaroop L, Partab P, Moodley J, Satyapal K. Anatomical variations of the second thoracic ganglion. Surg Radiol Anat. 2005;27:119–22.
23. Ramsaroop L, Partab P, Singh B, Satyapal KS. Thoracic origin of a sympathetic supply to the upper limb: the 'nerve of Kuntz' revisited. J Anat. 2001;199:675–82.
24. Yarzebski JL, Wilkinson HA. T2 and T3 sympathetic ganglia in the adult human: a cadaver and clinical-radiographic study and its clinical application. Neurosurgery. 1987;21:339–42.
25. Cho HM, Lee DY, Sung SW. Anatomical variations of rami communicantes in the upper thoracic sympathetic trunk. Eur J Cardiothorac Surg. 2005;27:320–4.
26. Rocha R, Teixeira M, Yeng L, Cantara M, Faria V, Liggieri V, et al. Thoracic sympathetic block for the treatment of complex regional pain syndrome type 1: a double blind randomized controlled study. Pain. 2014;155:2274–81.
27. Gest TR, Hildebrandt S. The pattern of the thoracic splanchnic nerves as they pass through the diaphragm. Clin Anat. 2009;22:809–14.
28. Rathmell JP, Gallant JM, Brown DL. Computed tomography and the anatomy of celiac plexus block. Reg Anesth Pain Med. 2000;25:411–6.
29. Loukas M, Klaassen Z, Merbs W, et al. A review of the thoracic splanchnic nerves and celiac ganglia. Clin Anat. 2010;23:512–22.
30. Tadros M, Elia R. Ultrasound-guided celiac plexus neurolysis in advanced upper abdominal cancer pain. Egypt J Radiol Nucl Med. 2005;46:993–8.
31. Seishima M, Kanoh H, Izumi T, et al. A refractory case of secondary erythermalgia successfully treated with lumbar sympathetic ganglion block. Br J Dermatol. 2000;143:868–72.
32. Bonica JJ. The management of pain. Philadelphia: Lee and Febiger; 1953.
33. Bradley KS. Observations on the surgical anatomy of the thoraco-lumbar sympathetic system. Aust N Z J Surg. 1951;20:272–7.
34. Umeda S, Arai T, Hatano Y, et al. Cadaver anatomic analysis of the best site for chemical lumbar sympathectomy. Anesth Analg. 1987;66:643–6.
35. Rocco AG, Palombi D, Raeke D. Anatomy of the lumbar sympathetic chain. Reg Anesth. 1995;20:13–9.
36. Murata Y, Takahashi K, Yamagata M, et al. Variations in the number and position of human lumbar sympathetic ganglia and rami communicantes. Clin Anat. 2003;16:108–13.
37. Mirilas P, Skandalakis JE. Surgical anatomy of the retroperitoneal spaces, part V: surgical applications and complications. Am Surg. 2010;76:358–64.

38. Cousins MJ, Reeve TS, Glynn CJ, et al. Neurolytic lumbar sympathetic blockade: duration of denervation and relief of rest pain. Anaesth Intensive Care. 1979;7:121–35.
39. Haynsworth RF Jr, Noe CE. Percutaneous lumbar sympathectomy: a comparison of radiofrequency denervation versus phenol neurolysis. Anesthesiology. 1991;74:459–63.
40. Manjunath PS, Jayalakshmi TS, Dureja GP, Prevost AT. Management of lower limb complex regional pain syndrome type 1: an evaluation of percutaneous radiofrequency thermal lumbar sympathectomy versus phenol lumbar sympathetic neurolysis – a pilot study. Anesth Analg. 2008;106:647–9.
41. Datta S, Pai U. Paradiscal extraforaminal technique for lumbar sympathetic block: report of a proposed new technique utilizing a cadaver study. Pain Physician. 2004;7:53–7.
42. Stanton-Hicks M. Lumbar sympathetic nerve block and neurolysis. In: Waldman SD, editor. Interventional pain management. 2nd ed. Philadelphia: W.B. Saunders; 2001. p. 485–92.
43. Sayson SC, Ramamurthy S, Hoffman J. Incidence of genitofemoral nerve block during lumbar sympathetic block: comparison of two lumbar injection sites. Reg Anesth. 1997;22:569–74.
44. Dirim A, Kumsar S. Iatrogenic ureteral injury due to lumbar sympathetic block. Scand J Urol Nephrol. 2008;42:395–6.
45. Plancarte R, de Leon-Casasola OA, El-Helaly M, et al. Neurolytic superior hypogastric plexus block for chronic pelvic pain associated with cancer. Reg Anesth. 1997;22:562–8.
46. Mauroy B, Demondion X, Bizet B, et al. The female inferior hypogastric (= pelvic) plexus: anatomical and radiological description of the plexus and its afferences – applications to pelvic surgery. Surg Radiol Anat. 2007;29:55–66.
47. Ali M, Johnson IP, Hobson J, et al. Anatomy of the pelvic plexus and innervation of the pros- tate gland. Clin Anat. 2004;17:123–9.
48. Gofeld M, Lee CW. Ultrasound-guided superior hypogastric plexus block: a cadaveric feasibility study with fluoroscopic confirmation. Pain Pract. 2017;17(2):192–6.
49. Mishra S, Bhatnagar S, Rana SP, et al. Efficacy of the anterior ultrasound-guided superior hypogastric plexus neurolysis in pelvic cancer pain in advanced gynecological cancer patients. Pain Med. 2013;14:837–42.
50. Lim SJ, Park HJ, Lee SH, Moon DE. Ganglion impar block with botulinum toxin type a for chronic perineal pain – a case report. Korean J Pain. 2010;23:65–9.
51. Ellinas H, Sethna NF. Ganglion impar block for management of chronic coccydynia in an adolescent. Paediatr Anaesth. 2009;19:1137–8.
52. Foye PM. Ganglion impar injection techniques for coccydynia (coccyx pain) and pelvic pain. Anesthesiology. 2007;106:1062–3.
53. Yeo SN, Chong JL. A case report on the treatment of intractable anal pain from metastatic carcinoma of the cervix. Ann Acad Med Singap. 2001;30:632–5.
54. Eker HE, Cok OY, Kocum A, et al. Transsacrococcygeal approach to ganglion impar for pelvic cancer pain: a report of 3 cases. Reg Anesth Pain Med. 2008;33:381–2.
55. Agarwal-Kozlowski K, Lorke DE, Habermann CR, et al. CT-guided blocks and neuroablation of the ganglion impar (Walther) in perineal pain: anatomy, technique, safety, and efficacy. Clin J Pain. 2009; 25:570–6.
56. Oh CS, Chung IH, Ji HJ, Yoon DM. Clinical implications of topographic anatomy on the ganglion impar. Anesthesiology. 2004;101:249–50.
57. Datir A, Connell D. CT-guided injection for ganglion impar blockade: a radiological approach to the management of coccydynia. Clin Radiol. 2010;65:21–5.
58. Plancarte R, Amescua C, Patt RB, Allende S. Presacral blockade of the ganglion of Walther (ganglion impar). Anesthesiology. 1990;73:A751.
59. Wemm K Jr, Saberski L. Modified approach to block the ganglion impar (ganglion of Walther). Reg Anesth. 1995;20:544–5.
60. Toshniwal GR, Dureja GP, Prashanth SM. Transsacrococcygeal approach to ganglion impar block for management of chronic perineal pain: a prospective observational study. Pain Physician. 2007;10:661–6.
61. Huang JJ. Another modified approach to the ganglion of Walther block (ganglion of Impar). J Clin Anesth. 2003;15:282–3.
62. Foye PM, Patel SI. Paracoccygeal corkscrew approach to ganglion impar injections for tailbone pain. Pain Pract. 2009;9:317–21.
63. Nebab EG, Florence IM. An alternate needle geometry for interruption of the ganglion impar. Anesthesiology. 1997;86:1213–4.
64. Reig E, Abejón D, del Pozo C, Insausti J, Contreras R. Thermocoagulation of the ganglion impar or ganglion of Walther: description of a modified approach. Preliminary results in chronic, nononcological pain. Pain Pract. 2005;5:103–10.
65. Usta B, Gozdemir M, Sert H, et al. Fluoroscopically guided ganglion impar block by pulsed radiofrequency for relieving coccydynia. J Pain Symptom Manag. 2010;39:e1–2.
66. Lin CS, Cheng JK, Hsu YW, Chen CC, Lao HC, Huang CJ, et al. Ultrasound-guided ganglion impar block: a technical report. Pain Med. 2010;11:390–4.
67. Marker DR, U-Thainual P, Ungi T, Flammang A, Fichtinger G, Iordachita II, et al. MR-guided perineural injection of the ganglion impar: technical considerations and feasibility. Skelet Radiol. 2016;45:591–7.

Part VII

Outcomes Studies in Regional Anesthesia

33 Outcome Studies and Infection Control in Regional Anesthesia

Joshua Ryan Dooley and Stuart Alan Grant

Introduction

Regional anesthesia began half a century after ether was first used for general anesthesia. Each branch of anesthesia has waxed and waned in popularity since their introduction, but now, with the advent of evidence-based medicine, true comparisons of outcome following general or regional anesthesia should be available.

Regional anesthesia holds a certain promise as our population continues to age and acquire comorbid conditions and may represent a gentler mode of providing care for patients who may not tolerate the insult of general anesthesia.

Regional anesthesia has many theoretically beneficial effects both intraoperatively and in the postoperative period, although studies have alternatively demonstrated or refuted these effects. The majority of evidence pertains to neuraxial regional anesthesia, though there are increasing numbers of studies examining peripheral nerve blockade. The outcome data on regional anesthesia is difficult to generalize because of many variables: insertion site, medication delivered, duration of use, congruency to surgical site and risks, and complications of a given surgical procedure. For this reason, when data is discussed, the entire perioperative environment must be considered.

When looking at outcomes, there are many disparate outcomes that are measured. Traditional outcomes include morbidity and mortality, cardiovascular, pulmonary, and GI endpoints. Alternative outcomes measured include overall opioid use, length of stay, quality of life, and patient satisfaction. Traditional outcomes such as myocardial infarction, pneumonia, and mortality have decreased through innovation and best clinical practices to the point that large randomized controlled trials for each type of surgery are needed to prove a significant difference, making the measurement of such outcomes more challenging. Opioid use is easily tracked and compared, though it is dependent on the assumption that fewer opioids are always better. Length of stay and cost analysis are among the outcome measurements that emphasize the relative stresses different types of anesthesia place on our healthcare system. As healthcare increasingly emphasizes a patient-centered approach, other outcome measurements, such as quality of life, quality of recovery, and patient satisfaction, deserve attention as well.

Intraoperative Effects of Regional Anesthesia

The surgical stress response is divided into the endocrine, metabolic, and inflammatory pathways, although, in reality, there are a myriad of

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_33

interactions among the three. Surgery-induced inflammation incites [1, 2] an increase in cytokines and a hypercoagulable state both intra- and postoperatively [3, 4]. In addition to the disturbance of coagulation, it can lead to activation of immune cells in the CNS. The surgery-induced neuroinflammation may be partly responsible for postoperative cognitive dysfunction [5].

Neuraxial anesthesia has been shown to blunt the stress response and should therefore theoretically improve morbidity and mortality associated with these pathways.

The neuroendocrine response to stress has been well described by Charmandari et al. [6]. The perioperative period is characterized by increased epinephrine, cortisol, and inflammatory mediators, all a function of the neuroendocrine response to surgical injury. This, coupled with other elements of the perioperative period such as inhaled anesthetics, decreased level of activity, and glucocorticoid therapy, leads to hyperglycemia. Hyperglycemia and the stress response lead to decreased immune function, increased oxidative stress, endothelial dysfunction, a procoagulant state, fluid shifts, electrolyte fluxes, and increased inflammatory mediators and mitogens. For the patient, the consequences can be delayed wound healing, increased infection, potential end-organ dysfunction, and delayed recovery [7]. This has been a driving force in the investigation of a multimodal approach to blunt these responses, of which regional anesthesia plays a key role.

Blood Loss and Transfusion Requirements

Intraoperative and perioperative blood loss may be significantly impacted by the type of anesthesia. There are a variety of theories as to the etiology of decreased blood loss. The most prevalent theory is that neuraxial anesthesia provides a lower and more stable blood pressure, resulting in decreased blood loss [8].

Intraoperative blood loss during total hip and total knee replacement was reviewed in a meta-analysis in 2010 that included 880 patients in 12 studies [9]. Results were mixed, with five studies showing no significant difference in blood loss between the regional anesthesia and general anesthesia. Of the remaining seven studies in the meta-analysis, five showed reduced blood loss with regional anesthesia, and another two studies showed increased blood loss with regional anesthesia.

When looking strictly at total hip arthroplasty, the pooled data from ten of the studies demonstrated a statistically significant decrease in blood loss with regional anesthesia. In the meta-analysis, two trials were excluded from the regional anesthesia arm for significant reductions in blood pressure. Given that hypotension is often a desired effect of regional anesthesia, it is reasonable to include the results of these studies in clinical decision-making for a particular patient. In addition, five of the studies in this meta-analysis included data on intraoperative transfusion. Overall, the incidence of transfusion was reduced by more than half with the use of regional anesthesia for total hip replacement.

The importance of decreasing the transfusion rates is important. A recent meta-analysis reviewed six studies with more than 20,000 patients showed that receiving an allogenic blood transfusion after total hip or knee arthroplasty dramatically increased surgical site infections [10].

Cancer Recurrence and Regional Anesthesia

Surgical stress places the body in an immunocompromised state, affecting the ability of natural killer (NK) cells to function. Previously animal studies have shown a dose-related decrease in NK cells by opioids [11]. More recently human studies have shown similar results. A prospective study evaluated patient for 48 h post-surgery showed that fentanyl, when compared to a non-steroidal, suppressed natural killer cell cytotoxicity [12]. NK cells are the primary defense we have against cancer. NK cells recognize and kill tumor cells in the body (Table 33.1).

It has long been a concern that surgery itself promotes the growth of dormant metastases and accelerates the growth of previously slow-growing masses, with opioids for postoperative pain control only exacerbating the problem. Regional

Table 33.1 Immune function changes with general and regional anesthesia

	General	Regional
Immunosuppression	↓	↔
Natural killer cell function	↓	↔
Catecholamine levels	↑	↔
ACTH	↑	↑/↔
Cortisol	↑	↑/↔

anesthesia has been shown to decrease the stress response to surgery and preserves more immunologic function. Regional anesthesia causes a smaller reduction of NK cell function than general anesthesia [13, 14]. The mechanism of this regional anesthesia-mediated preservation of NK function is the blockade of afferent transmission of noxious stimuli to the central nervous system and the blockade of efferent activation of the sympathetic nervous system, thereby both diminishing the body's response to surgery and decreasing the amount of anesthesia necessary for surgery. Spinal anesthesia added to halothane general anesthesia significantly decreased the surgical promotion of lung metastases in rats [15].Wada et al. demonstrated that spinal anesthesia added to sevoflurane general anesthesia decreased the surgical promotion of liver metastases in a murine model with the surgical insult being laparotomy. They demonstrated that the tumoricidal function of liver mononuclear cells and the improved T1–T2 helper cell ratio were instrumental in the better outcomes [16]. The first human study of a possible influence of regional anesthesia on long-term cancer outcomes was published by Exadaktylos et al. [17]. This was a retrospective study of 129 patients receiving mastectomy and axillary node dissection with an average follow-up of 32 months. Fifty patients received general anesthesia and a paravertebral block, while 79 patients received general anesthesia and postoperative morphine analgesia. On follow-up, recurrence and metastasis-free survival was 94% and 77% at 3 years for the paravertebral and morphine analgesia groups, respectively. This was a retrospective study, with inherent weaknesses, and the methodology of the study has been criticized for its poor bias control. More recently, the timing of epidural anesthesia has been evaluated on the effect of ovarian cancer recurrence [18]. de Oliveira et al. compared groups who received epidural anesthesia intraoperatively and postoperatively, postoperatively, or no epidural. The recurrence time for the intraoperative epidural group was 73 months. The postoperative epidural group and the no epidural group had 33 months and 38 months, respectively, time to recurrence of cancer.

Combined cervical epidural and general has also been shown to increase cancer-free survival and overall survival for head and neck cancer patients [19]. The combined group had a 5-year 68% cancer-free survival rate compared with the general alone 5-year 37% cancer-free survival. Combined group also had an overall 5-year survival rate of 59% compared with 41% for the general alone group.

Despite the early apparent positive association with regional anesthesia and tumor recurrence, contemporary studies have not been favorable. Myles et al. performed a prospective randomized controlled clinical trial to compare recurrence of cancer and survival of patients having major abdominal surgery for cancer [20]. They evaluated 503 patients placed into either an epidural group or a non-epidural group. The median time to recurrence of cancer was 2.6 years for the epidural group and 2.8 years for the non-epidural group. Recurrence-free survival and overall mortality were also nearly identical for the two groups. Epidural analgesia has also not shown to be beneficial for recurrence-free survival during open radical prostatectomy for advanced prostate cancer [21].

While future research may provide some clarity as to the role of regional anesthesia and cancer recurrence, current mixed data does not support regional anesthesia as a protective measure.

Postoperative Effects of Regional Anesthesia

Mortality

Short-term mortality is positively affected by regional anesthesia and postoperative epidural pain control, and this improvement has been demonstrated in various surgical interventions [22]. One Medicare claims database analysis

demonstrated an improved survival using regional anesthesia with an odds ratio of 0.52 for mortality at day 7 and 0.74 at postoperative day 30, with significant 95% CI and *P* values [23]. The analysis was from years 1997 to 2001, and it would be beneficial, given the change in approach to the perioperative care of the patient in the last decade, to see if this mortality benefit was sustained. Currently, no such study has been done.

Long-term mortality is an all-encompassing outcome and therefore difficult to attribute to a single factor in the majority of surgical procedures. Surgical mortality has been decreased by a multimodal approach to modulating the body's response to surgical stress, an approach to this problem studied by Kehlet [2, 24]. This comprehensive accelerated recovery pathway is more important than any single factor (such as regional anesthesia) within the pathway. Given the relative rarity of mortality in many surgical procedures, it is difficult to determine the effect of one aspect of a multimodal approach. One way to compensate for this is to select a single operation and use this as a benchmark of the effect of regional anesthesia on long-term mortality. This benchmarking has been done using the example of hip surgery in the elderly. There are obvious difficulties with this assumption, the first being that the body of an elderly person with a fractured hip is responding the same to stress as a patient who is responding to the pain of a surgical incision. Also, it is incorrect to compare an elderly patient who is undergoing an elective hip arthroplasty with a patient who has suffered a hip fracture. Although studies from 1986 to 1987 [25, 26] demonstrated a short-term benefit without a long-term benefit to regional anesthesia in patients with fractured hips, a Cochrane Database review from 2016 [27] found no difference in 1-month mortality. This recent review did, however, highlight that without prophylaxis with anticoagulant drugs, the risk of deep venous thrombosis was less with neuraxial block. Another review of ten trials involving 2201 patients having surgery with general anesthesia alone or with an epidural found that patients with an epidural had a decreased risk of death of 3.1% vs 4.9% [28].

The most recent and comprehensive evaluation of the effect of anesthesia technique evaluated more than 300 thousand patients utilizing the US National Surgical Quality Improvement database [29]. Both surgical procedures and ASA physical status were matched. The primary outcome evaluated was 30-day postoperative mortality. The modalities studied were regional anesthesia and general anesthesia. Despite the group's hypothesis that regional anesthesia would have a higher survival rate, the data showed that after adjusting for clinical and patient cofounders, there was no improvement in mortality with regional anesthesia compared to general anesthesia.

Cardiovascular Outcomes

Postoperative myocardial infarction (PMI) is a leading cause of postoperative morbidity, and so, investigation of methods of reducing postoperative myocardial infarction will not only have great benefit for individual patients but also for the healthcare system as a whole.

For specific surgeries and patient populations that are high risk, some data suggest there might be benefit to using a neuraxial technique. A recent review of 15 trials [30] evaluating epidural versus opioid-based pain relief for abdominal aortic surgery concluded that the epidural group had a reduced number of myocardial infarctions, but no mortality difference. This is vastly different from results pooled from a wide variety of surgeries. In 20 studies in this review [22], there was found to be decreased 30-day mortality in patients who received a neuraxial block. Based on six studies evaluated by the group, there was found to be no difference in myocardial infarction.

A review published in the *BMJ* in 2000 involving 9559 patients in 141 trials looked at multiple postoperative outcomes. A total of 104 myocardial infarctions were reported, with approximately one third fewer events in the patients with neuraxial anesthesia [31]. A meta-analysis was published looking specifically at the risk of postoperative myocardial infarction [32]. A total of

1173 patients in 17 studies were included. This meta-analysis concluded that at least 24 h of postoperative epidural analgesia reduced the rate of postoperative myocardial infarction by 40% in patients with a thoracic epidural. It further suggested that high-risk cardiac patients should have postoperative epidural analgesia but calls for further studies for a more certain determination. Lumbar epidural analgesia did not have as great a benefit on cardiac morbidity as thoracic epidural analgesia.

The results are only significant using a fixed-effects model and not the random effects model of analysis, and as the model becomes more conservative, the studies lack the power to show significance.

Since the advent of a multimodal approach to the care of orthopedic patients and standardization of DVT prophylaxis, there is no significant difference in the incidence of DVT postoperatively, regardless of the method of anesthesia selected. The most recent meta-analysis on this reviewed ten studies reporting incidence of DVT and eight reporting incidence of pulmonary embolism [9]. Provided the patient receives DVT prophylaxis, there exists no significant difference in the incidence of deep vein thrombosis or pulmonary embolism based on the choice of anesthesia. It may be beneficial to consider a regional anesthetic technique in a patient at high risk for a thromboembolic event in order to maximize all factors for its prevention, but there is no data to support this decision.

Both nationally and internationally, the number of people requiring treatment for end-stage renal disease continues to increase. Treatment often includes hemodialysis via an arteriovenous fistula. The formation of these fistulae is plagued by an extremely high early failure rate. A recent prospective randomized study evaluated the type of anesthesia as it related to patency of arteriovenous fistulae at 3 months [33]. Sixty-three patients were randomized to receive local anesthetic, and 63 patients were randomized to receive a single-injection brachial plexus block. The patency of the fistula in the single-injection brachial plexus group was 84% versus 62% in the local anesthesia group.

The effect of neuraxial anesthesia on hypercoagulability combined with the vasodilation from sympatholysis is more notable in vascular surgery. This was demonstrated in the PIRAT study of the early 1990s. In this study, it was demonstrated that neuraxial anesthesia resulted in a significantly decreased number of graft failures due to thrombosis. Fibrinogen, plasminogen activator inhibitor-1, and D-dimer were followed in the study, and the regional anesthetic appeared to prevent the postoperative inhibition of fibrinolysis, resulting in fewer graft failures. This is in direct contrast to a recent retrospective study [34] of 822 patients that showed no difference in graft patency between general versus epidural anesthesia techniques.

Pulmonary Outcomes

Surgery results in a multifactorial etiology of pulmonary complications, with the incidence of postoperative pulmonary complications varying widely depending on the surgical intervention. Surgery may disrupt normal respiratory activity, and anesthesia has an impact on respiratory responses to acid–base changes and hypoxia postoperatively depending on the medications given. The mechanisms of respiratory impairment include a reflex inhibition of the phrenic nerve, surgical dressings affecting the mechanics of respiration, and uncontrolled pain that may result in a change of respiratory mechanics [35] (Table 33.2).

Postoperative pulmonary complications include pneumonia, aspiration pneumonitis, respiratory failure, reintubation, weaning failure, atelectasis, and bronchospasm. In addition to increasing hospital costs, postoperative pulmo-

Table 33.2 Pulmonary function changes with general and regional anesthesia

	General	Regional
Pneumonia	↔/↑	↔/↓
Respiratory failure	↑	↓
Respiratory function	↓	↑
Pain score	↑	↓

nary complications increase ICU admission rates, postoperative length of stay, and mortality. The mean increase in length of stay due to pulmonary complications can be up to 8 days [36]. In 2015 the National Surgery Quality Improvement Program evaluated pulmonary complications after major abdominal surgery and concluded that esophageal procedures and advanced American Society of Anesthesiology classification were the strongest predictors of postoperative pulmonary complications [37].

Guidelines from the American College of Physicians underscore the continued significance of postoperative pulmonary complications [38]. In fact, cardiac and pulmonary complications have an equal incidence postoperatively and may have the same increased risk of mortality and increase in length of stay for non-cardiac procedures.

The largest meta-analysis ($n = 9559$) comparing neuraxial blockade with general anesthesia was published in 2000 and found that neuraxial blockade in mixed surgical procedures demonstrated a significantly decreased risk of pneumonia (3.1 versus 6%, OR 0.61 with 95% CI 0.48–0.76) [31]. This finding is in agreement with an earlier meta-analysis examining pulmonary complications following thoracic epidurals [39]. There is a great degree of variance in the incidence of pulmonary complications based on the surgery considered. When considering specific surgical interventions, thoracic epidural analgesia in the setting of coronary artery bypass surgery, although controversial, was shown to decrease the incidence of postoperative pulmonary complications, with a relative risk of 0.68 [40]. Open abdominal aortic surgery with a thoracic epidural for analgesia also demonstrated a significantly reduced risk of respiratory failure, but a relative risk reduction in pneumonia postoperatively was not statistically significant. Two large, randomly controlled trials found a significant decrease in respiratory failure in high-risk patients, but no significant decrease in respiratory failure when all patients receiving thoracic epidurals were included [41, 42]. The high-risk patient findings were the result of subgroup analysis, which is a weakness of these studies.

It has been demonstrated that epidural analgesia provides superior analgesia to intravenous patient-controlled analgesia (PCA), and epidurals improve respiratory function, and thoracic epidurals are considered the gold standard for esophagectomy [43, 44]. Standardized multimodal perioperative care pathways for abdominal and pelvic surgery that include thoracic epidurals have shown to improve outcomes [45], and thoracic epidurals are included in the Enhanced Recovery After Surgery Society guidelines [46]. Currently, there is sufficient evidence in the literature to support the use of thoracic epidural analgesia for pulmonary risk reduction in the case of high-risk patients, especially for major abdominal surgery, open abdominal aortic surgery, or coronary artery bypass grafting.

Gastrointestinal Outcomes

The use of fast-track protocol in gastrointestinal surgery and a multimodal approach to the perioperative care of the patient has become standard practice of medicine which includes the use of an epidural corresponding to the site of surgical intervention, both for intraoperative anesthesia and postoperative analgesia. Patients undergoing major abdominal surgery including colon resection have superior analgesia and decreased postoperative ileus with a thoracic epidural. However, most of these studies are in the setting of a fast-track protocol that include multimodal pain regimen, early and advanced feeding, early removal of drains and catheters, and enforced mobilization. It is difficult to parse out the exact role that epidurals play within this new milieu, but wise to acknowledge they are a vital piece in a very precarious puzzle (Table 33.3).

A meta-analysis of 16 randomized controlled trials of thoracic epidurals versus general anesthesia and systemic opioid analgesia for colorectal surgeries focused on length of stay with secondary outcomes of postoperative pain control, duration of postoperative ileus, incidence of anastomotic leak, incidence of postoperative nausea and vomiting, pruritis, sedation and respira-

Table 33.3 Postoperative patient endpoint changes with general and regional anesthesia

	General	Regional
Length of stay	↔	↔
Postoperative ileus	↑	↓
Hypotension	↓	↑
Visual analog pain scale	↑	↓
Postoperative nausea and vomiting	↔	↔

tory depression, incidence of cardiac and pulmonary complications, as well as hypotension and motor blockade [47]. When data was not available in the published article, the authors were contacted. Only two of the included trials were laparoscopic; the rest were open colorectal surgery. All but two trials done after 2000 had patients enrolled in a fast-track program. Ultimately, it was found that thoracic epidural analgesia does not significantly diminish length of hospital stay, although it does diminish duration of postoperative ileus by an average of 36 h and provides significantly better visual analog pain scales. Patients with a thoracic epidural showed increased incidence of pruritis 21% versus 5% and urinary retention 10% versus 1% for epidural analgesia and systemic analgesia, respectively, but showed no significant difference in degree of sedation or postoperative nausea and vomiting. There were no significant differences in motor blockade, anastomotic leakage, or cardiopulmonary events. There was an increase in the incidence of hypotension with epidurals, although there was no comment on the clinical significance of this finding.

Although there is a clear role for thoracic epidural anesthesia and analgesia in large open abdominal cases, the data regarding the use of a thoracic epidural in laparoscopic abdominal surgery is less clear, with positive findings on pain relief, dietary intake, and length of stay that have not always proven reproducible, perhaps due to decreased invasiveness and surgical stress response compared to open procedures. Taqi et al. compared systemic opioids to thoracic epidural analgesia with local anesthetic and fentanyl using 50 consecutive patients undergoing laparoscopic colon resection with a standard non-accelerated perioperative care plan [48]. It was demonstrated that there was a significant difference in reduction of postoperative ileus of 1–2 days, as well as a quicker return to full diet and better pain control. However, there was no significant difference in readiness to discharge or length of stay. A prospective observational study published by Zingg et al. in 2009 enrolled 76 patients to compare the effects of general anesthesia with systemic opioids and thoracic epidural analgesia using a combination of local anesthetic and opioid on postoperative pain control and ileus [49]. The thoracic epidural group required fewer analgesics, had a mean opioid use of 12 mg of morphine compared to 103 mg of morphine, and had significantly lower visual analog pain scale and time to gastrointestinal recovery of 2.96 versus 3.81 days ($P = 0.025$). This correlates well with open procedures that have shown decreased opioid use and decreased ileus. While the study did not find a significant difference in nasogastric tube reinsertion or postoperative vomiting, the investigators noted that this may have been due to liberal metoclopramide use in the systemic opioid group. Clear benefits of a thoracic epidural extended as far out as postoperative day 7 in this study, which did follow a multimodal approach to perioperative care, though no specific fast-track regimen was followed. No differences in surgical and anesthetic morbidity or mortality were noted between the two arms of the study. Weaknesses of the study include the fact that the patients in this study were a subset of a larger study, and there was no standardized postoperative opioid regimen.

Postoperative nausea and vomiting is also of significant concern when considering anesthetic technique. PONV can result in delayed discharge, unplanned admissions, and diminished patient satisfaction. A review published in *Anesthesiology* in 2003 offers a comprehensive discussion of PONV and regional anesthesia. The majority of the literature regarding PONV is in the setting of general anesthesia. When comparing regional to general anesthesia, the preponderance, though not all, of the literature in this review supports the belief that regional anesthesia has a lower incidence of PONV than general anesthesia [50].

More specifically, epidural has the potential to decrease PONV as well. However, an updated review performed by Guay et al. evaluated epidural local anesthetics versus opioid-based analgesic regimens on gut motility and PONV for abdominal surgery [51]. This review included 94 trials with over 5000 participants. The data revealed that return of gastrointestinal transit was improved by 17 h in the epidural group and noted that this was based on high quality of evidence. A moderate quality of evidence existed for epidural offering better pain relief than opioids, and there was no difference in PONV or anastomotic leak.

In 2014 Pöpping et al. performed a systematic review and meta-analysis of randomized controlled trials to evaluate the impact that epidural analgesia had on morbidity and mortality. The effect epidural analgesia was positive for a wide variety of gastrointestinal symptoms. The data collected and analyzed showed that time to first defecation, start of bowel function, time to first flatus, and decreased ileus all favored epidural [28].

With data-driven support, epidural has become a cornerstone in most enhanced recovery after surgery protocols for open colorectal surgery. As technology and expertise advance, more surgeries are performed laparoscopically and the role that epidural may play in postoperative care of the patient is unclear at this time. A recent meta-analysis of five randomized controlled clinical trials attempted to evaluate the influence of epidural analgesia following laparoscopic colorectal surgery within an ERAS program [52]. The review found that the epidural group had a longer hospital stay, not a shorter one, compared to the group without the epidural. There was no difference in postoperative complications or readmission rates.

Rehabilitation and Length of Stay

The pharmacodynamics of regional anesthesia are variable and depend on whether there is use of a single shot or continuous catheter blockade, the concentration of the local anesthetic used, and the presence of adjuvants such as opioids. All of this affects the motor block that may be seen after a peripheral nerve block. Motor block must be considered when choosing the type of block and local anesthetic solution. Motor block may affect time to rehabilitation, frequency of complications, and time to recovery. Recently, it was demonstrated that a weakness in the muscles around the knee can reduce stability during rotations and direction changes several hours after surgery for patients with continuous lumbar plexus nerve block compared with single-injection block or no block can lead to increased falls [53]. Contrarily, a review of inpatient falls in 2014 did not show an association of increased falls with peripheral nerve block compared to no nerve block for total knee arthroplasty [54]. The same study showed that patients with increased falls were older, had a higher comorbidity burden, and had more complications. A recent meta-analysis of studies comparing peripheral nerve blocks to an epidural technique reviewed 12 studies and demonstrated that peripheral nerve blocks and epidural are equivalent for pain control for total knee arthroplasty [55]. For abdominal surgery, a Cochrane Database review concluded that epidurals with local anesthetics can decrease hospital length of stay, although with very low quality of evidence [51].

In addition to length of stay for the original procedure, the incidence of readmission must also be considered. ERAS protocols are not always complied with 100% of the time, but the use of epidural has the highest compliance, and this has led to shorter length of stays, but also unfortunately a higher rate of readmission [56]. Length of stay in the hospital has been a useful metric, but also of significance is the length of stay in the post-anesthesia care unit (PACU). Regional anesthesia has the potential to decrease PACU times and thus allow for increased throughput. Indeed, Grauman et al. performed a retrospective study that showed that patients that received a brachial plexus block for upper limb surgery had a much shorter stay in PACU (99 min) versus patients receiving only general anesthesia (171 min) [57]. In addition to this decreased time spent in PACU, the regional anesthesia group also received no additional opiates and had 18% less occurrence of PONV and

administration of antiemetic. Utilization of regional anesthesia can also decrease length of stay for patients undergoing carotid endarterectomy (CEA) [58]. Siu et al. did a retrospective review of 346 patients undergoing CEA and showed that the regional anesthesia group had a length of stay of 1.2 days versus the general anesthetic group with a length of stay of 2.0 days. Additionally, the overall cost for the general anesthetic group was more than 3000 dollars more than the regional group. The use of femoral and sciatic nerve blocks for total knee arthroplasty has also been shown to decrease length of stay by 19 h [59].

Postoperative Pain Relief

Postoperative pain control is an important outcome, not only for patient-centered reasons but also because of its impact on length of stay, time to beginning rehabilitation, and recovery of function, as well as minimization of atelectasis and pneumonia in thoracic procedures. Through randomized controlled trials and meta-analysis of RCTs, it has been shown that continuous epidural analgesia provides superior postoperative analgesia than intravenous patient-controlled analgesia (PCA) [60]. In addition, single-injection peripheral nerve blocks are limited by the duration of the local anesthetic infused, lasting from 10 to 24 h at most. These results were shown in many different types of surgery, although the predominance of the data is in orthopedic surgery. These benefits have been demonstrated in studies of continuous nerve catheters in the hospital setting as well as at home [61]. This superior analgesia resulted in an earlier time to walking or movement and an improved side-effect profile. When considering epidural anesthesia versus peripheral nerve blockade, a meta-analysis published in 2008 found that the level of pain relief, measured by visual analog pain scales, was equivalent, although in two of three studies, patients with continuous peripheral nerve catheters rated their satisfaction with the anesthesia higher, and all had less hypotension, pruritis, and urinary retention and did not incur the risk of central nervous system complications [62]. Peripheral nerve blockade has also been shown to be equivalent epidural for pain management after total knee arthroplasty with a reduced amount of complication in the peripheral nerve block group [55]. Interestingly, a recent study found that patients who received a single-injection brachial plexus block for wrist fracture repair had an increase in unplanned healthcare resource utilization due to pain [63]. These results should not be any surprise; patients often have an extensive amount of pain after the single-injection nerve block wears off and are generally not educated in how to treat this pain [64, 65].

Regional anesthesia does have several caveats to consider with these results. First, the surgical site must be concordant with the area of analgesia. If the incision or area of surgical trauma extends beyond the dermatomes covered by the epidural or peripheral nerve catheter, then it will obviously be less than fully functional for analgesia. In addition to catheter/surgical site congruency, the choice of local anesthetic or opioid, the duration of the infusion, and a multimodal approach to pain control are critical elements to optimizing postoperative pain control.

Chronic Pain

Chronic pain is a potential consequence of surgery that has long-lasting implications, severely affecting the quality of life of the patient. Chronic pain is not an uncommon problem, with approximately 25% of all patients reporting surgery as the source of their chronic pain [66]. The intensity of postoperative pain seems to affect how the central nervous system remodels itself in response to the surgical insult, and therefore, it is assumed that blunting this acute pain would decrease central sensitization and would affect the incidence of postsurgical chronic pain. This hypothesis is, as of yet, unproven and has mixed data behind it. Chronic postsurgical pain syndrome can delay recovery and return to normal daily living [67].

In the field of chronic pain, it has been well established that the degree of pain suffered, as well as the duration, has significant bearing on

whether or not a patient will develop a chronic pain syndrome following their procedure. What is not yet as clear is if regional anesthesia can improve upon general anesthesia's incidence of chronic pain. Currently, the data are mixed. For example, in thoracic surgery, there is no clear data that states whether epidurals or paravertebral blocks are preferable from a long-term outcome standpoint, primarily due to a lack of long-term data on patients who had a paravertebral block. Paravertebral blocks have a better side-effect profile than thoracic epidural analgesia with equal control of immediate postoperative pain [68]. Although chronic pain in thoracic surgery has several possible etiologies, using regional techniques for analgesia are thought to minimize chronic post-thoracotomy pain [69]. There is no consensus on the time of initiation of thoracic epidural analgesia (TEA) in thoracotomy in terms of prevention of chronic pain. In a mixed surgical population, despite evidence of better at home pain control and mobility in patients discharged to home with peripheral nerve catheters, there are no reliable data showing a significant difference in chronic pain compared to patients with general anesthesia and intravenous and oral pain control [70]. The primary predictor of chronic pain syndrome is the degree of postoperative pain control and regional anesthesia decreases postoperative pain; yet regional techniques risk nerve damage with the development of a subsequent chronic pain syndrome. Further study with long-term follow-up data is required to provide satisfactory evidence for an answer to this question.

In 2013 Andreae and Andreae performed a systematic review and meta-analysis to determine if regional anesthesia can prevent chronic pain after surgery [70]. They identified 23 randomized controlled trials and pooled data from 250 participants from three trials after thoracotomy with outcomes at 6 months. This review showed that epidural anesthesia for the prevention of pain after surgery had an odds ratio of 0.33. For paravertebral block after breast surgery, they pooled data from 89 participants. This data showed that outcomes at 6 months were favored in the paravertebral group with an odds ratio of 0.37. The authors summarize the data by stating that one out of every four or five patients treated could benefit with decreased risk of chronic pain.

In an effort to elucidate the role of epidurals in enhanced recovery programs for colorectal surgery, McIsaac et al. performed a scoping review evaluating evidence that epidurals improve outcomes [71]. In 36 studies 58% found that the addition of an epidural was associated with significant improvement in pain. No studies demonstrated that the addition of an epidural had worse pain scores.

Phantom limb pain is very frequent particularly among amputee patients and can have devastating effects on their quality of life. Many pharmacologic interventions have been attempted, but to date there has been limited efficacy. A recent Cochrane Database review evaluating interventions for treating pain highlights the possible future role of calcitonin, local anesthetics, and dextromethorphan [72]. Epidural and peripheral nerve blocks are generally in use around surgical time and only remain in place for the in hospital stay. Ambulatory nerve catheters are a fairly new modality at treating pain and have the possibility of remaining in situ after the patient is discharged from the hospital. Indeed, Ilfeld et al. performed a crossover pilot study to evaluate the treatment of phantom limb pain with high dose local anesthetic and ambulatory catheters (ropivacaine 0.5% for 6 days) [73]. While this study was small, the patients who received local anesthetic had complete resolution of symptoms during the infusion and reduced pain scores even after the perineural catheter was removed.

Despite the limitation of utilization of epidurals to patients who are in hospital, this does not mean they have no value. A double-blind randomized multicenter study involving a cohort of 60 patients studied the value of adding calcitonin to the epidural infusion [74]. One group had bupivacaine and fentanyl, and the other group had bupivacaine, fentanyl, and calcitonin. The authors demonstrated a significant improvement in phantom limb pain in the calcitonin group at 12 months after surgery, with the no calcitonin group having a higher degree of allodynia and hyperalgesia.

Postoperative Cognitive Decline

Postoperative cognitive decline (POCD) spans a wide spectrum of characteristics and severity, with impairment in cognitive function, memory, and consciousness being the three primary areas assessed. Impairment in cognitive function is assessed by the ability of the patient to perform simple mental tasks when asked to do so.

Risk for postoperative cognitive decline is exacerbated by increasing age, medical comorbidities, preexisting cognitive dysfunction, and type of surgery. Patients at higher risk for immediate postoperative cognitive dysfunction also demonstrate an increased risk of long-term cognitive dysfunction. Postoperative cognitive dysfunction, while an important outcome itself, is also a predictor of poor patient outcomes, prolonged recovery from surgery, and impaired quality of life in the longer term [75].

There is a surprisingly high incidence of postoperative cognitive dysfunction (POCD), especially in high-risk patients. A large international multicenter trial of approximately 1200 patients over the age of 60 found 25.8% of patients had POCD 1 week after surgery and 9.9% after 3 months, compared to 3.4% at 1 week and 2.8% at 3 months for nonsurgical controls [76]. While the percentage of postoperative cognitive dysfunction in our patient population is unlikely to change, the population as a whole is aging, and a greater number of older patients with multiple comorbidities are presenting for surgery, increasing the overall burden to our healthcare system that POCD presents. In 1986, acute confusional states were estimated conservatively to cost Medicare at least $2 billion, and in 1999, estimates of the cost of inpatient delirium to the healthcare system were $4 billion, with an accompanying increase in inpatient days of 17.5 million annually [77].

Risk factors for postoperative cognitive dysfunction following non-cardiac surgery have been well elucidated and are divided into preoperative, intraoperative, and postoperative factors. Cardiac bypass presents its own cognitive dysfunction and is outside of the purview of this discussion. Of the preoperative factors, age features most prominently, with other factors such as preexisting cerebral, cardiac, or vascular disease and alcohol abuse [78]. Up to 65% of elderly patients may suffer from some sort of cognitive dysfunction after surgery [79]. Despite POCD being a common occurrence, preoperative cognition is rarely measured, and a decrease in cognition after surgery is scarcely ever discussed with the patient or the patient's family. Intraoperative risk factors include the specific surgical intervention especially orthopedic surgery, aortic aneurysm repair and cardiac procedures, and duration of surgery and anesthesia. The effects of profound sustained hypotension, hypoglycemia, anemia, and hypoxia on POCD are not certain. Postoperative use of meperidine, benzodiazepines, or anticholinergic medications is associated with increased POCD. Other postoperative risk factors include infection, respiratory complication, and increased postoperative pain.

Given these risk factors, one would expect that regional anesthesia would be protective against POCD. In 2003, the International Study of Postoperative Cognitive Dysfunction (ISPOCD) randomized 438 patients at 12 different institutions to regional or general anesthesia for a range of non-cardiac procedures and found no significant difference in the incidence of POCD at 3 months [80]. The next year, a systematic review of 24 trials that considered POCD as an outcome found that the choice of regional or general anesthesia had no bearing on the incidence of POCD [81]. One of the methodological issues with this review was that the postoperative analgesic regimen was not standardized across studies. With improvements in peripheral nerve catheters and ultrasound technology, this is becoming a promising area for clinical impact. Although there was another systematic review of postoperative analgesia and its effect on outcomes in 2007, it was determined that more definitive research was needed on this aspect of regional anesthesia [82]. The improved postoperative analgesia as well as the improved sleep and decreased postoperative fatigue with peripheral nerve catheters are expected to be major contributors to a decrease in postoperative cognitive decline [83]. Despite some of these earlier reviews showing regional

anesthesia to have very little effect on POCD, specific populations may still benefit from either peripheral nerve or neuraxial blockade. More recently, a recent study examined the use of epidurals versus general anesthetic for patients receiving a total hip arthroplasty [84]. The study included 100 patients divided into the two groups. Preoperative mini-mental state exams (MMSE) were equivalent between the groups; postoperatively the general anesthesia group had a POCD occurrence of 36% compared to 16% in the epidural group. These differences were less at the third postoperative day, with only 10% of the general anesthesia group suffering from POCD compared with 6% in the epidural group. For urologic surgeries, a systematic review concluded that there is insufficient data to pool to show any difference between neuraxial versus general techniques in regard to POCD [85].

Patient Satisfaction

While clinical outcomes remain important, patient-oriented outcomes have taken on an increasing prominence both in the literature and in the impact of daily practice. Patient-oriented outcomes include postoperative pain control, quality of life, and patient satisfaction. Ultimately, a good deal of patient satisfaction is dependent on the management of the expectations of the patient and conveying your genuine concern for them as their healthcare provider.

Patient satisfaction influences the interaction of society and the individual with the healthcare community and is also used as a benchmark of service and for marketing. Whether or not patient satisfaction is a true indicator of quality of care remains controversial and is beyond the scope of this chapter [86]. Of interest, higher patient satisfaction scores do not necessarily lead to improved outcomes. Fenton et al. evaluated patient satisfaction scores in a nationwide sample and showed that higher patient satisfaction scores are associated higher overall healthcare expenditures and increased mortality [87]. Regardless of validity or reproducibility, patient satisfaction is an acknowledged endpoint of outcomes research, which is ultimately designed as a patient-centered assessment.

A review article looking at patient satisfaction with regard to regional anesthesia was published by Wu and colleagues in 2001 [88]. Patient satisfaction is multidimensional, involving sociodemographic, cognitive, and affective elements [83, 88]. This makes patient satisfaction very hard to standardize. Many different theories have been advanced, but few have been sufficiently tested and validated. Many patient satisfaction theories are modifications of customer satisfaction and marketing theories and may be classified into three broad categories: intra-patient comparisons, patient–provider comparisons, and inter-patient comparisons.

There are several methodological issues with the measurement of patient satisfaction. Many patient surveys have not undergone psychometric construction to evaluate such a multivariate outcome as patient satisfaction. In addition, many surveys are unable to discriminate between portions of care the patient found to be satisfactory and those the patient found unsatisfactory. Many surveys lack reliability and validity, and bias may be introduced in many aspects of the survey process itself.

Bearing in mind the limitations expressed above, in 2007, a systematic review of the literature comparing postoperative regimens found that postoperative regional analgesia, particularly with local anesthetics resulted in significantly lower visual analog pain scores, and yet, there was a paucity of data on patient satisfaction and few validated instruments to reliably measure satisfaction [82]. Even with improved pain control with a regional technique, improved satisfaction scores were not always seen. The determination of the true effect of regional anesthesia on patient satisfaction will require large multicenter RCTs with validated instruments for measuring satisfaction and strict methodological controls.

Neurologic Complications

Neurologic complications of peripheral nerve blockade are a major concern to both patients and providers. The instance of nerve lesion varies based on the site of blockade. In a review of 32 studies, the rate of neuropathy after spinal and

epidural anesthesia was 3.78:10,000 and 2.19:10,000, respectively, with permanent neurologic injury rates of 0–4.2:10,000 and 0–7.6:10,000 for spinal and epidural anesthesia, respectively [89]. The rates of neuropathy after interscalene brachial plexus block, axillary brachial plexus block, and femoral nerve block were 2.84:100 for interscalene, 1.48:100 for axillary, and 0.34:100 for femoral. There was only one permanent neuropathy reported in 16 studies of neurologic complications of peripheral nerve block. In 1997 and 2002, Auroy et al. reported the incidence of nerve lesions in 21,278 and 50,223 peripheral nerve blocks, with a combined incidence of 0.02% in each study [90, 91]. In the 2002 study, the incidence of nerve injury in blocks with adverse neurologic sequelae ranged from 0.03% in femoral blockade to 0.31% in popliteal blocks. Of the nerve injuries seen, seven persisted for greater than 7 months. To complicate the picture, the risk of femoral nerve injury in total hip arthroplasty is cited as 0.1–0.4%. Deficits from these lesions all resolved, although the longest duration injury took 10 months to return to baseline. Though the incidence of neural complication was low, making the determination of independent risk factors for neurologic injury difficult, intensive care unit hospitalization was positively associated with nerve injury, and the use of bupivacaine was associated with increased paresthesia and dysesthesia. Even though the risk of nerve damage is extremely low with regional nerve blocks, the proceduralist needs to be aware of what kind of injury constitutes immediate imaging, neurologic or neurosurgical evaluation or treatment versus an injury that can managed with observation and follow-up. Specific guidelines and an evaluation algorithm have been established by the American Society of Regional Anesthesia in 2015 and should be used as a template when a nerve injury is suspected [92].

Multiple sclerosis (MS) is the most widespread immune-mediated disabling neurological disease of young adults with most people being diagnosed between the ages of 20 and 40 years of age. Neuraxial anesthesia in patients with MS is controversial. Stress, surgical or other, is a well-known risk factor for the onset or relapse of MS. Postsurgical management of pain via an epidural has the potential to be beneficial to surgical patients with MS. A recent systematic review of the available literature investigated 11 studies and 26 case reports [93]. There were two prospective studies evaluating epidurals in patients with MS in an obstetric setting [94, 95]. Each one of these studies independently concluded that epidural anesthesia had no correlation with postpartum relapses or disability.

Block Site Infectious Complications

Like many other outcome measurements, there are many variables to infectious complications including patient history, duration of peripheral nerve catheter, site of blockade, and infection control precautions taken. In a study of 700 patients with interscalene catheters placed for upper extremity surgery, six patients showed clinical evidence of infection, one at 3 days after surgery, four at 4 days, and one at 5 days [96]. The catheters were removed and cultured, with three showing coagulase-negative *Staphylococcus*, one colonized with *Staphylococcus aureus* and two that grew no bacteria in culture. All catheters were scanned by ultrasound to look for a fluid collection in the setting of clinical infection. Of the six, five had no fluid collection, and all five were treated with antibiotics and had no further complications. The one patient with evidence of a fluid collection had this surgically drained, an antibiotic course administered, and there was no further complication. In summary, out of 700 patients with an interscalene catheter, 0.8% demonstrated clinical evidence of infection, with only 0.1% requiring surgical drainage of a fluid collection, and no long-term sequelae were found related to infection at 1, 3, or 6 months follow-up. In a prospective trial by Capdevila that involved a range of peripheral nerve catheters, the insertion technique was a standardized aseptic technique with cap, mask, sterile gown and gloves, and sterile draping of the surrounding area [97]. There were 256 interscalene catheters, 126 axillary catheters, 20 lumbar plexus catheters, 683 femoral catheters, 94 fascia iliaca catheters, 32 sciatic catheters, 167 popliteal catheters, and 32 cubital or median

nerve catheters in the study. In their 969 catheters, 278 or 28.7% had positive bacterial colonization on testing. The most common organism was coagulase-negative *Staphylococcus*, and 242 of the colonized catheters were single-organism cultures. Only 3% of all patients demonstrated clinical signs of inflammation and of these, only 44% had positive cultures. Of catheters that had no clinical sign of inflammation, 18.6% were culture positive. The one major infectious adverse event reported was a *S. aureus* psoas muscle abscess and cellulitis in a diabetic woman with a femoral catheter for total knee replacement. This corresponds to other case reports of adverse infectious events, the majority of which were *S. aureus* infections in diabetics. Additional risk factors for local inflammation or infection in this study were found to be postoperative monitoring in the intensive care, catheter duration longer than 48 h, male sex, and lack of prophylactic antibiotics.

Data on infection rates in neuraxial techniques are similarly broad in range, with a study of 170,000 epidural and 550,000 spinal anesthetics between 1987 and 1993, citing a rate of 1.1 infections to the spine or central nervous system per 100,000 blocks [98]. While there remains a wide reported range of both infectious and neurologic complications with a multiplicity of variables among studies making studies less than completely comparable with one another, there is agreement in the severity of these complications to individual patients and the importance of striving to minimize these unfortunate occurrences.

Infection Control

Infection control should be a focus of the anesthesia provider from the planning of the block until after the patient removes the catheter at home. Given the variability in how regional anesthesia was being performed and the potential for devastating complications, as well as the many clinical unknowns surrounding regional anesthesia and infectious complications, the American Society of Regional Anesthesia recognized the need for an updated consensus statement on infection control and the importance of aseptic technique. In 2015 the society released the Second Practice Advisory on Neurologic Complications [92]. This provides a peer-reviewed set of guidelines for aseptic technique during regional procedures, regional anesthesia in the setting of an immunocompromised patient, and regional anesthesia in the infected or febrile patient.

Regional Anesthesia and Aseptic Technique [99]

Infection sources related to the patients' health such as immunosuppression, trauma, malignancy, or pregnancy are classified as intrinsic sources, whereas skin invasion through a needle tract, contaminated needles, syringes, catheter hubs, or breaches in sterile technique are extrinsic sources. A survey of Australian obstetric anesthesiologists indicated that there was a broad range of what was considered essential for adherence to strict aseptic technique [100]. Given the broad range of methodologies listed in the literature, one may assume that this variance of opinion exists in more than Australia. Infections can occur even when aseptic techniques are used however [101].

Based on extrapolation from surgical data, it is recommended that an alcohol-based antimicrobial scrub for hand washing be used prior to a regional procedure, although there are no randomized controlled studies of hand washing and regional anesthesia nor are there likely to be in the future. Bacteria counts are higher on the hands of physicians who do not remove their rings, increasing the probability of nosocomial infection, and the necessity of removing of wristwatches is a view held by many infection control experts. A majority of NHS trusts in the United Kingdom have instituted a program called "Bare Below the Elbow" for all physicians to limit nosocomial infections [102].

There are no studies of micro-contamination after use of sterile gloves in a sterile procedure. Sterile gloves, however, never negate the need for hand washing. Between nonsterile vinyl and latex gloves, vinyl gloves were almost nine times as

likely to have leaks after use than latex gloves, with micro-contamination on the hands of 13% of the healthcare providers tested before and after nonsterile procedures. This is material evidence that bacteria traveled through leaks in the gloves or that there was perforation of the gloves during the procedure. Hospital acquired infections are of serious concern, and hands of hospital personnel have been identified as the most important route of transmission of pathogens [103]. It is estimated that 648,000 patients in the United States will be afflicted with hospital acquired infection which can lead to increased costs, length of stay, as well as morbidity and mortality [104]. Despite good evidence that hand hygiene is vitally important, compliance continues to be poor. Improvement in compliance specifically for regional anesthesia team members can be accomplished by implementing personal alcohol-based gel dispensers [105].

The issue of wearing masks as a method of infection control has been controversial, with some studies stating that it may increase surgical infection with a posited mechanism of action being friction against the face resulting in scaling of epidermal tissue into the sterile field. The methodology of the study resulting in these data was widely criticized, and a more rigorous study resulted in data indicating no difference in surgical infection rate with or without the use of surgical masks. Nonetheless, there are case reports of a cluster of streptococcal meningitis from patients who had spinal anesthesia from a provider who was being treated for recurrent tonsillitis, did not wear a mask and spoke throughout the procedures, and a reported case of epidural abscess with a strain of *S. aureus* shown to be from the strain colonizing the nose of the healthcare provider who placed the epidural [106, 107]. Historically wearing a mask during some procedures has been controversial. Due to an increase in reported cases of bacterial meningitis in patients who had a neuraxial procedure, the Centers for Disease Control and Prevention has made specific recommendations that healthcare providers wear masks during these procedures [108]. There are insufficient data regarding the use of gowns during regional block to make a recommendation, but, like masks, the gown may provide an important piece of protective gear for the healthcare provider. A study of more than 200 parturients showed there was no difference in epidural catheter colonization rate if a gown was worn during the procedure or not [109]. Ultimately, the degree of severity of infectious complications would argue for an enhanced sensitivity to aseptic technique despite the lack of conclusive evidence from randomized controlled trials, which would have ethical issues in their methodology.

Hub contamination and bacterial filters are also to be considered with regional techniques. Micropore filters are designed to filter bacteria out that may exist in the infusing solution as well as prevent foreign material from gaining access to the epidural space, but there have been documented epidural abscesses in the presence of antibacterial filters [99]. There are other observational findings that also show that bacterial colonization can occur with micropore filters [110]. There are several possible mechanisms for this occurrence. There may be hematogenous spread of bacteria from a distant source, the bacteria may migrate along the tract outside the catheter, the filter may have diminished function after a period of time, or there may be direct contamination of the hub while changing the filter.

Any antiseptic solution used as a prep for regional anesthesia procedures must be broad spectrum, with fast onset, long duration, minimal toxic skin effects, and not be inactivated by biological fluids. The majority of the literature reviews povidone-iodine and chlorhexidine gluconate.

Chlorhexidine is effective against Gram-positive and Gram-negative bacteria and yeast, alters cell wall permeability, precipitates cell membrane and cytoplasm components, and adheres to the stratum corneum for prolonged effect. The addition of isopropyl alcohol potentiates its bactericidal effects. It remains effective in the presence of blood and other body fluids, produces few skin reactions, and has few pathogens resistant to it [99]. It is currently FDA approved for surgical skin preparation. There is insufficient testing for acquisition of an FDA approval for preparation for a regional anesthesia

procedure. Currently there are no reports of neurologic or central nervous system adverse events due to chlorhexidine used as a skin antiseptic preparation.

Povidone-iodine is also effective against most Gram-positive and Gram-negative bacteria, though its mechanism is dependent on its continued release of iodine, which disrupts protein synthesis. Because of its mechanism, povidone-iodine requires several minutes for maximum effectiveness. Release of iodine is accelerated by isopropyl alcohol, but it is inactivated with organic material such as blood or pus. Some patients may have acute skin or systemic reactions to iodine, and certain strains of *S. aureus* have developed resistance to it. It is currently FDA approved for preparation of the surgical site and does not have an FDA indication for preparation of a site for a regional anesthesia procedure due to lack of clinical data. Addition of alcohol to povidone-iodine may increase its efficacy. A recent study showed that the addition of alcohol to povidone-iodine was superior to povidone-iodine alone in preventing surgical site infections [111]. A recent Cochrane Database Systematic Review found some evidence that preoperative skin preparation with 0.5% chlorhexidine in methylated spirits was associated with lower rates of surgical site infections compared with alcohol-based povidone-iodine [112]. However, they also conclude that there is overall, very limited data.

In addition to the type of antiseptic solution used, it is important to consider using single use bottles/packets of the antiseptic and not multi-use bottles. Contamination of multi-use bottles of povidone-iodine has been demonstrated [113].

Chlorhexidine dressings have been shown to significantly reduce the number of epidural catheters colonized on removal and reduce the overall bacterial count by a factor of 100 compared to non-medicated dressings [113, 114]. A prospective randomized study was performed that showed a significant reduction in bacterial colonization of the tip of the catheter and at the insertion site for epidural and in peripheral nerve catheters [115]. Despite the decreased colonization, there were no reductions in local infections.

Regional Anesthesia in the Immunocompromised Patient [116]

Regional anesthesia is also complicated by the patient whose immune system is compromised, as his or her susceptibility to infection is increased, and both the frequency and severity of infection are increased; however, it has been shown that regional anesthesia diminishes the suppression of immune function caused by surgical stress [117]. Horlocker and Wedel published the findings of the Practice Advisory Panel on the Infectious Complications regarding regional anesthesia in these patients [116].

The primary barrier to infection, the skin, will be breeched by both surgery and the regional anesthesia, enforcing the importance of aseptic technique. Both cellular and humorally mediated immunity are suppressed for several days after surgery. Neuraxial anesthesia has been shown to significantly diminish the surgical stress response, although it must be continued in the postoperative period. Therefore, the patient population that would have a significant benefit in preserving what remained of their immune system also represents a greater risk of meningitis, epidural abscess, and site infection. In 2002, there was a limited study comparing peripheral and neuraxial techniques and their effect on surgical stress. This showed that epidural was superior to peripheral nerve blocks for suppression of stress hormones, although pain scores were equivalent [118]. Therefore, the techniques that may limit risks of epidural abscess or meningitis may also be less effective at preserving the remnant of immune response that remains in the immunosuppressed patient. The consequences of meningitis and epidural abscess are sufficiently dire that great precautions are merited to avoid these sequelae. Untreated, bacterial meningitis has a 100% mortality rate, and even with appropriate and timely antibiotic therapy, mortality remains at 30%. Epidural abscesses are primarily bacteria, although fungal and mycobacterial abscesses may present. Immunosuppression is an independent risk factor for the formation of epidural abscess. Complete recovery is reported in less

than 40% of cases and most often when surgical intervention is undertaken in less than 36 h.

Central nervous system infections due to neuraxial anesthesia are rare. A Finnish study evaluated serious complications associated with spinal and epidurals from the year 2000 to 2009 [119]. During the study period, 1.4 million neuraxial blocks were performed in Finland. From a closed claim database, four patients had an epidural abscess, all four of which recovered, two with conservative therapy and two with surgical intervention. Eight patients suffered from meningitis, seven recovered, and one died. The overall calculated rate of fatality was 1:233,000 and serious complications at 1:35,000.

Although it has been shown that immunocompromised patients are at greater risk for CNS infection with neuraxial blockade, there is little data on the exact incidence of complications within a given immunocompromised population. A review of 1620 pediatric patients demonstrated one report of a *Candida tropicalis* epidural collection in a child with metastatic cancer with complete resolution of neurologic symptoms after surgical decompression [120].

Two separate viral infections must be considered in terms of neuraxial anesthetic management. There is a theoretical concern in herpes simplex virus type 2 (HSV-2) of introduction into the central nervous system resulting in aseptic meningitis as the presenting clinical picture. Multiple studies have been done, primarily in the obstetric population where the majority of patients had recurrent HSV-2, and there remains insufficient data to state the risk of CNS infection due to neuraxial anesthesia during a primary infection. Epidural and spinal anesthesia in patients with HSV-2 recurrences appears to be safe [116, 121–123].

Human immunodeficiency virus presents a different set of concerns. Since CNS involvement occurs during the first few weeks of infection and 90% of HIV patients have neuropathic abnormalities on autopsy [124], the introduction of the virus into the CNS is a moot point in HIV. The concern is that there are many different factors which may contribute to neuropathy: the virus itself, opportunistic infections, retroviral medications, an increased risk of worsening of neurologic deficits in the perioperative period due to regional anesthesia, the surgical intervention, and surgical positioning. The many factors contributing to neuropathy provide a confusing picture, decreasing the ability to divine the specific cause of a neurologic deficit. A retrospective analysis evaluated 90 patients with HIV comparing general anesthesia, local anesthesia, and combined spinal epidural. They found that complication and infection rate was equivalent among the groups [125]. The overall conclusion was that all three kinds of anesthesia could be used with considerable safety and selected based on clinical need, not HIV status. Although few in number, other studies have also had little to no problems with neuraxial anesthesia and HIV with broad consensus that epidurals are safe for these patients [126–128].

The etiology of immunosuppression in a patient brings up the possibility of secondary effects with complications occurring primarily in two categories: hemorrhagic and neurologic. With immune system compromise comes opportunistic infection, and the most common disorder of hemostasis during an infection is thrombocytopenia, although one must also be wary for disseminated intravascular coagulation. In addition, some anti-infectious agents will result in thrombocytopenia through bone marrow suppression or further immune system compromise. If patients have evidence of petechiae or purpura, coagulation studies and a platelet count should be obtained. Currently, given the risk of significant infectious and hemorrhagic complications, there is no role for neuraxial anesthesia in the patient with an active untreated infection unless extraordinary circumstances may mitigate the significant risks. In the face of cancer, there are both hemorrhagic and thromboembolic risks, as well as considerations for certain types of cancer. Solid tumors predispose patients to thromboembolic events, and hemorrhagic complications occur more frequently with acute leukemia, although there is a spectrum of events with any tumor. Ninety percent of patients with metastatic disease demonstrate laboratory evidence of DIC, although a much smaller percentage has clinical evidence of the dysregula-

tion of their hemostasis [129]. It is estimated that in 10–15% of patients with metastatic solid tumors have signs of DIC and 15% of acute leukemia patients have some degree of clinical DIC [130]. Patients in acute DIC are far more likely to have a hemorrhagic complication, while those patients with a fulminating chronic DIC are far more likely to have a prothrombotic complication. Given the far higher thrombotic risk during the perioperative period for patients with cancer, aggressive thromboprophylaxis is required and is important to consider when discussing possible anesthetic management plans. The most common cause of bleeding for cancer patients is thrombocytopenia, which may be due to decreased production, sequestration, or increased destruction, both by the malignant process and its treatment regimen. If a patient has recently had chemotherapy or has a myeloproliferative process, a targeted evaluation of their hemostasis is warranted. Specifically, in the presence of circulating leukemic cells, neuraxial techniques should be avoided. Dural puncture in acute lymphoblastic leukemia patients may seed the CNS with blast cells, significantly worsening the probable outcome.

Patients who are immunocompromised often have a preexisting neurologic deficit either due to their disease, the treatment for their disease, or both. Any further insult, in the form of needle trauma, local anesthetic toxicity, or ischemia due to blood vessel spasm, has a synergistic effect, either exacerbating old neurologic damage or creating new neurologic deficits. If considering a spinal or epidural anesthetic, a review of recent radiographs is necessary to rule out vertebral metastases at the desired level of entry, and a clinical exam is required to rule out spinal cord compression. Peripheral neuropathy is a common complication of chemotherapy. It is seen in 100% of people who take vincristine, 85% of those who have at least 300 mg/m^2 of cisplatin, 60% of patients who have taken at least 250 mg/m^2 of paclitaxel, and is common in several other chemotherapeutic agents [116].

Solid organ transplant is becoming more common, and thus the need to understand the infectious complications due to post-transplant immunosuppression is paramount [131]. The risk of infection in these patients is primarily due to epidemiologic exposure or the patients overall state of immunosuppression.

Regional Anesthesia in the Febrile or Infected Patient [132]

Although dural puncture has been considered a risk for development of meningitis, central neuraxial infections are very rare, with a series of 65,000 spinal anesthetics yielding only three cases of meningitis, a review of 50,000 epidurals yielding no CNS infection, and a multicenter prospective study of 40,640 spinal and 30,413 epidural anesthetics yielding no infectious complications [90, 133]. Moen et al. reviewed 1.26 million spinals and 450,000 epidurals done in Sweden between 1990 and 1999 and found 29 cases of meningitis and 13 epidural abscess cases [134]. Given the low incidence of these complications, very large data sets are required to extract meaningful information. The limited data available suggests that a patient with bacteremia receiving an epidural or spinal anesthetic is at greater risk for CNS infection.

Data from studies in humans examining meningitis has been mixed. Studies with no significant difference in the incidence of meningitis, those with an association and no clear causal relationship, and those with a significantly different incidence in meningitis have been published with weaknesses associated with each study [135–137]. Most reports of meningitis after dural puncture are related to either a break in sterile procedure or due to unusual or nosocomial organisms [132]. Epidural anesthesia may also result in meningitis without epidural abscess. Two parturients demonstrate this in the literature, one patient with an area of cellulitis at the insertion site and meningitis from *Streptococcus faecalis* and the other patient with CSF, urine, blood, and vaginal cultures positive for *Staphylococcus uberis* [138]. The first patient's most likely cause was cellulitis at the insertion site, though other causes could not be excluded, and it was posited that the second parturient acquired meningitis from hematogenous seeding from the vagina during delivery.

As mentioned previously, epidural abscesses are a very rare though serious CNS complication of lumbar puncture. Although uncommon, spinal epidural abscesses have increased from 1975 rate of 0.2–1.2 per 10,000 admissions to a rate of 2.5–3.0 per 10,000 admissions in 1998 [139]. Another patient population expected to incur additional risk of epidural abscess would be parturients with chorioamnionitis, 8% of whom are bacteremic. A recent nationwide inpatient sample was analyzed in order to describe the incidence of and risk factors for spinal hematoma and abscess associated with epidural analgesia in adult obstetric and non-obstetric populations in the United States [140]. More than three million epidural procedures were identified, and the incidence of spinal abscess in obstetric patients was zero. In the non-obstetric population, the rate of epidural abscess was 7.2 per 100, 000. It is proposed that, although pregnancy is a relatively immunosuppressed state, there is a short duration for the epidural catheter, and the patients generally have fewer comorbidities to predispose them to opportunistic infection. Overall, it is considered safe to administer regional anesthesia to women with chorioamnionitis, with or without antibiotics [141].

Ultimately, the decision of whether to perform a regional anesthesia technique on a febrile patient must be made based on evaluation of that patient. The experimental and epidemiological data does suggest an association between dural puncture during bacteremia and meningitis, although this association is primarily from the pediatric population, which has a much higher incidence of meningitis. Animal models of dural puncture during untreated bacteremia frequently had bacterial counts far higher than are clinically relevant. Nevertheless, given the possible association, expert opinion would advise not performing neuraxial anesthesia in a floridly bacteremic patient without some very persuasive extenuating circumstances. If a patient however has received appropriate antibiotics to treat systemic infection and demonstrated an appropriate response, all data suggests that it is safe to perform spinal anesthesia provided there is antibiotic dosing prior to dural puncture. Epidural anesthesia data under the same circumstances is reassuring, but limited, and no recommendation for epidural anesthesia under these circumstances is made by the Advisory Panel. A summary of the recommendations for prevention of infection by the Practice Advisory Panel on the Infectious Complications for regional anesthesia procedures is provided below [142]:

- Before performing neuraxial techniques, conduct a history and physical examination relevant to the procedure, and review relevant laboratory studies in order to identify patients who may be at risk of infectious complications.
- Consider alternatives to neuraxial techniques for patients at high risk.
- When neuraxial techniques are selected in a known or suspected bacteremic patient, consider administering pre-procedure antibiotic therapy.
- Select neuraxial technique on a case-by-case basis, including a consideration of the evolving medical status of the patient.
- Avoid lumbar puncture in the patient with a known epidural abscess.
- Use aseptic techniques during preparation of equipment (e.g., ultrasound) and the placement of neuraxial needles and catheters, including:
  - Removal of jewelry (e.g., rings and watches).
  - Hand washing.
  - Wearing of caps.
  - Wearing of masks covering both mouth and nose.
  - Consider changing masks before each new case.
  - Use of sterile gloves.
  - Sterile draping of the patient.
  - Use individual packets of antiseptics for skin preparation.
  - Use an antiseptic solution (e.g., chlorhexidine with alcohol) for skin preparation, allowing for adequate drying time.
  - Use sterile occlusive dressings at the catheter insertion site.
  - Bacterial filters may be considered during extended continuous epidural infusion.
  - Limit the disconnection and reconnection of neuraxial delivery systems in order to minimize the risk of infectious complications.

- Consider removing unwitnessed accidentally disconnected catheters.
- Catheters should not remain in situ longer than clinically necessary.

Conclusions

In conclusion, the choice of regional anesthesia is still an individual one tailored to the comorbidities and desires of each patient, balanced with a risk profile specific to the case. Some of the theoretical benefits posited by the physiologic effect of regional anesthesia have been shown to be true, while others do not seem to have the expected clinical effect. There are distinct pulmonary benefits, especially in thoracic and large open abdominal procedures. These benefits are less apparent in minimally invasive procedures. Decreases in postoperative myocardial infarction can be seen for patients undergoing cardiac or vascular procedures, but only if they are in a high-risk category for myocardial infarction and if neuraxial analgesia is continued at least 24 h postoperatively. There is ample evidence that epidurals improve time to first ambulation for large abdominal surgeries, time to reach physical therapy goals, and time to discharge in orthopedics. Epidurals also decrease time to return of bowel function and reduce length of stay in colorectal surgery. Most importantly, regional anesthesia plays a pivotal role in any enhanced recovery after surgery protocol.

Clinical Pearls

Blood Loss and Transfusion Requirements

- Regional anesthesia decreases blood loss in total hip replacement by the mechanism of systemic blood pressure control.

Cancer Recurrence and Regional Anesthesia

- Regional anesthesia decreases postoperative immunosuppression and prevents intraoperative catecholamine and stress hormone surges. This preserves natural killer cell function, which has been shown to improve long-term survival in several different cancers. Controlled trial results are pending with the possibility of a major impact on the survival rates of many cancers.

Mortality

- The biggest effect on long-term mortality has been the multimodal approach to patient care with a comprehensive enhanced recovery pathway, of which regional anesthesia is only one aspect.

Cardiovascular Outcomes

- Thoracic epidurals may reduce postoperative myocardial infarction, although some studies demonstrating this benefit were less rigorous. The previous benefit of decreased DVTs with neuraxial analgesia has been obviated by thrombosis prophylaxis. Regional anesthesia does seem to have some advantages in vascular surgery, through sympatholysis, vasodilation, and decreased fibrinolysis. Regional anesthesia results in fewer graft failures than with general anesthesia.

Pulmonary Outcomes

- Regional anesthesia does not impair respiratory mechanics; in fact, it improves them. Thoracic epidurals have been shown to decrease respiratory failure and pneumonia in high-risk patients.

Gastrointestinal Outcomes

- Regional anesthesia as part of a multimodal approach to patient care has been shown to provide superior pain control, decreased opiate requirements, and reduced duration of postoperative ileus. Reduced

PONV is supported by some studies, though the findings are not uniform in the literature.

Rehabilitation and Length of Stay

- Regional anesthesia permits earlier mobilization, but the possibility of motor block and residual weakness must also be managed. Peripheral nerve blocks may have an advantage in decreasing postoperative motor block. Regional anesthesia is also associated with a decreased rate of readmission in complex knee surgery.

Postoperative Pain Relief

- Pain control in continuous peripheral nerve catheters and epidurals is equivalent, but patient satisfaction and side-effect profile are better in continuous peripheral nerve catheters. Peripheral nerve catheters can be run in patients discharged home. Both provide superior analgesia compared to PCA.

Chronic Pain

- Although the argument that regional anesthesia should decrease chronic pain is theoretically persuasive, there are mixed data as to whether this is seen clinically.

Postoperative Cognitive Decline

- Regional anesthesia and general anesthesia have an equivalent incidence of POCD. Emerging data indicates that there may be a decrease in POCD with postoperative regional analgesia for pain control.

Patient-Oriented Outcomes

- Patient-oriented outcomes are important considerations when planning an anesthetic. Patient satisfaction is improved with regional anesthesia and analgesia compared to placebo or with regional anesthesia compared to general anesthesia and PCA.

Neurologic Complications

- The incidence of neurologic complications is very low but differs by block. Permanent nerve injury is far less common than transient neurologic symptoms.
- ICU patients are at increased risk for nerve injury.

Infectious Complications

- Many continuous peripheral nerve catheters are colonized with bacteria and yet have no active signs of infection. Diabetes may present an increased risk for infectious complications, as does lack of prophylactic antibiotics, catheter duration >48 h, male sex, and ICU monitoring.

Review Questions

1. The following may be at higher risk of developing a postoperative infection at the peripheral nerve catheter site except:
   (a) Diabetes
   (b) Catheter duration >48 h
   (c) Female sex
   (d) ICU monitoring
   (e) Lack of prophylactic antibiotics
2. A decrease in mortality rates has been proven with which of the following:
   (a) Spinal
   (b) A multimodal approach including regional anesthesia
   (c) Brachial plexus nerve catheter
   (d) Epidural
   (e) General anesthesia
3. Which of the following offers the best postoperative pain relief?
   (a) Oral opiates
   (b) PCA
   (c) Nurse administered IV opiates as needed

(d) Peripheral nerve catheters
(e) NSAIDs

Answers

1. c
2. b
3. d

References

1. Prondzinsky R, Knupfer A, Loppnow H, Redling F, Lehmann DW, Stabenow I, Witthaut R, Unverzagt S, Radke J, Zerkowski HR, Werdan K. Surgical trauma affects the proinflammatory status after cardiac surgery to a higher degree than cardiopulmonary bypass. J Thorac Cardiovasc Surg. 2005;129(4):760–6.
2. Kehlet H. Multimodal approach to control postoperative pathophysiology and rehabilitation. Br J Anaesth. 1997;78(5):606–17.
3. Sembo J. Influence of thoracotomy and pulmonary surgery on coagulation and fibrinolysis in the intra and postoperative period—experimental and clinical study. Nihon Geka Gakkai Zasshi. 1989;90(2):199–209.
4. Rosenfeld BA. Benefits of regional anesthesia on thromboembolic complications following surgery. Reg Anesth. 1996;21(6 Suppl):9–12.
5. Steinberg BE, Sundman E, Terrando N, Eriksson LI, Olofsson PS. Neural control of inflammation: implications for perioperative and critical care. Anesthesiology. 2016;124(5):1174–89.
6. Charmandari E, Tsigos C, Chrousos G. Endocrinology of the stress response. Annu Rev Physiol. 2005;67:259–84.
7. Akhtar S, Barash PG, Inzucchi SE. Scientific principles and clinical implications of perioperative glucose regulation and control. Anesth Analg. 2010;110(2):478–97.
8. Modig J. Regional anaesthesia and blood loss. Acta Anaesthesiol Scand Suppl. 1988;89:44–8.
9. Hu S, Zhang ZY, Hua YQ, Li J, Cai ZD. A comparison of regional and general anaesthesia for total replacement of the hip or knee: a meta-analysis. J Bone Joint Surg Br. 2009;91(7):935–42.
10. Kim JL, Park JH, Han SB, Cho IY, Jang KM. Allogeneic blood transfusion is a significant risk factor for surgical-site infection following total hip and knee arthroplasty: a meta-analysis. J Arthroplast. 2017;32(1):320–5.
11. Shavit Y, Ben-Eliyahu S, Zeidel A, Beilin B. Effects of fentanyl on natural killer cell activity and on resistance to tumor metastasis in rats. Dose and timing study. Neuroimmunomodulation. 2004;11(4):255–60.
12. Narahara H, Kadoi Y, Hinohara H, Kunimoto F, Saito S. Comparative effects of flurbiprofen and fentanyl on natural killer cell cytotoxicity, lymphocyte subsets and cytokine concentrations in post-surgical intensive care unit patients: prospective, randomized study. J Anesth. 2013;27(5):676–83.
13. Li JM, Shao JL, Zeng WJ, Liang RB. General/epidural anesthesia in combination preserves NK cell activity and affects cytokine response in cervical carcinoma patients undergoing radical resection: a cohort prospective study. Eur J Gynaecol Oncol. 2015;36(6):703–7.
14. Buckley A, McQuaid S, Johnson P, Buggy DJ. Effect of anaesthetic technique on the natural killer cell anti-tumour activity of serum from women undergoing breast cancer surgery: a pilot study. Br J Anaesth. 2014;113(Suppl 1):i56–62.
15. Bar-Yosef S, Melamed R, Page GG, Shakhar G, Shakhar K, Ben-Eliyahu S. Attenuation of the tumor-promoting effect of surgery by spinal blockade in rats. Anesthesiology. 2001;94(6):1066–73.
16. Wada H, Seki S, Takahashi T, Kawarabayashi N, Higuchi H, Habu Y, Sugahara S, Kazama T. Combined spinal and general anesthesia attenuates liver metastasis by preserving TH1/TH2 cytokine balance. Anesthesiology. 2007;106(3):499–506.
17. Exadaktylos AK, Buggy DJ, Moriarty DC, Mascha E, Sessler DI. Can anesthetic technique for primary breast cancer surgery affect recurrence or metastasis? Anesthesiology. 2006;105(4):660–4.
18. de Oliveira GS Jr, Ahmad S, Schink JC, Singh DK, Fitzgerald PC, McCarthy RJ. Intraoperative neuraxial anesthesia but not postoperative neuraxial analgesia is associated with increased relapse-free survival in ovarian cancer patients after primary cytoreductive surgery. Reg Anesth Pain Med. 2011;36(3):271–7.
19. Merquiol F, Montelimard AS, Nourissat A, Molliex S, Zufferey PJ. Cervical epidural anesthesia is associated with increased cancer-free survival in laryngeal and hypopharyngeal cancer surgery: a retrospective propensity-matched analysis. Reg Anesth Pain Med. 2013;38(5):398–402.
20. Myles PS, Peyton P, Silbert B, Hunt J, Rigg JR, Sessler DI. Perioperative epidural analgesia for major abdominal surgery for cancer and recurrence-free survival: randomised trial. BMJ. 2011;342:d1491.
21. Wuethrich PY, Thalmann GN, Studer UE, Burkhard FC. Epidural analgesia during open radical prostatectomy does not improve long-term cancer-related outcome: a retrospective study in patients with advanced prostate cancer. PLoS One. 2013;8(8):e72873.
22. Guay J, Choi P, Suresh S, Albert N, Kopp S, Pace NL. Neuraxial blockade for the prevention of postoperative mortality and major morbidity: an overview of Cochrane systematic reviews. Cochrane Database Syst Rev. 2014;(1):Cd010108.
23. Wu CL, Hurley RW, Anderson GF, Herbert R, Rowlingson AJ, Fleisher LA. Effect of postoperative epidural analgesia on morbidity and mortality following surgery in medicare patients. Reg

Anesth Pain Med. 2004;29(6):525–33. discussion 515–29
24. Kehlet H. Acute pain control and accelerated postoperative surgical recovery. Surg Clin North Am. 1999;79(2):431–43.
25. Valentin N, Lomholt B, Jensen JS, Hejgaard N, Kreiner S. Spinal or general anaesthesia for surgery of the fractured hip? A prospective study of mortality in 578 patients. Br J Anaesth. 1986;58(3):284–91.
26. Davis FM, Woolner DF, Frampton C, Wilkinson A, Grant A, Harrison RT, Roberts MT, Thadaka R. Prospective, multi-centre trial of mortality following general or spinal anaesthesia for hip fracture surgery in the elderly. Br J Anaesth. 1987;59(9):1080–8.
27. Guay J, Parker MJ, Gajendragadkar PR, Kopp S. Anaesthesia for hip fracture surgery in adults. Cochrane Database Syst Rev. 2016;(2):Cd000521.
28. Popping DM, Elia N, Van Aken HK, Marret E, Schug SA, Kranke P, Wenk M, Tramer MR. Impact of epidural analgesia on mortality and morbidity after surgery: systematic review and meta-analysis of randomized controlled trials. Ann Surg. 2014;259(6):1056–67.
29. Saied NN, Helwani MA, Weavind LM, Shi Y, Shotwell MS, Pandharipande PP. Effect of anaesthesia type on postoperative mortality and morbidities: a matched analysis of the NSQIP database. Br J Anaesth. 2017;118(1):105–11.
30. Guay J, Kopp S. Epidural pain relief versus systemic opioid-based pain relief for abdominal aortic surgery. Cochrane Database Syst Rev. 2016;(1):Cd005059.
31. Rodgers A, Walker N, Schug S, McKee A, Kehlet H, van Zundert A, Sage D, Futter M, Saville G, Clark T, MacMahon S. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: results from overview of randomised trials. BMJ. 2000;321(7275):1493.
32. Beattie WS, Badner NH, Choi P. Epidural analgesia reduces postoperative myocardial infarction: a meta-analysis. Anesth Analg. 2001;93(4):853–8.
33. Aitken E, Jackson A, Kearns R, Steven M, Kinsella J, Clancy M, Macfarlane A. Effect of regional versus local anaesthesia on outcome after arteriovenous fistula creation: a randomised controlled trial. Lancet. 2016;388(10049):1067–74.
34. Wiis JT, Jensen-Gadegaard P, Altintas U, Seidelin C, Martusevicius R, Mantoni T. One-week postoperative patency of lower extremity in situ bypass graft comparing epidural and general anesthesia: retrospective study of 822 patients. Ann Vasc Surg. 2014;28(2):295–300.
35. Beaussier M, Genty T, Lescot T, Aissou M. Influence of pain on postoperative ventilatory disturbances. Management and expected benefits. Ann Fr Anesth Reanim. 2014;33(7–8):484–6.
36. Sabate S, Mazo V, Canet J. Predicting postoperative pulmonary complications: implications for outcomes and costs. Curr Opin Anaesthesiol. 2014;27(2):201–9.
37. Yang CK, Teng A, Lee DY, Rose K. Pulmonary complications after major abdominal surgery: National Surgical Quality Improvement Program analysis. J Surg Res. 2015;198(2):441–9.
38. Qaseem A, Snow V, Fitterman N, Hornbake ER, Lawrence VA, Smetana GW, Weiss K, Owens DK, Aronson M, Barry P, Casey DE Jr, Cross JT Jr, Fitterman N, Sherif KD, Weiss KB. Risk assessment for and strategies to reduce perioperative pulmonary complications for patients undergoing noncardiothoracic surgery: a guideline from the American College of Physicians. Ann Intern Med. 2006;144(8):575–80.
39. Ballantyne JC, Carr DB, deFerranti S, Suarez T, Lau J, Chalmers TC, Angelillo IF, Mosteller F. The comparative effects of postoperative analgesic therapies on pulmonary outcome: cumulative meta-analyses of randomized, controlled trials. Anesth Analg. 1998;86(3):598–612.
40. Svircevic V, Passier MM, Nierich AP, van Dijk D, Kalkman CJ, van der Heijden GJ. Epidural analgesia for cardiac surgery. Cochrane Database Syst Rev. 2013;(6):Cd006715.
41. Park WY, Thompson JS, Lee KK. Effect of epidural anesthesia and analgesia on perioperative outcome: a randomized, controlled veterans affairs cooperative study. Ann Surg. 2001;234(4):560–569; discussion 569-571.
42. Rigg JR, Jamrozik K, Myles PS, Silbert BS, Peyton PJ, Parsons RW, Collins KS. Epidural anaesthesia and analgesia and outcome of major surgery: a randomised trial. Lancet. 2002;359(9314):1276–82.
43. Gruber EM, Tschernko EM, Kritzinger M, Deviatko E, Wisser W, Zurakowski D, Haider W. The effects of thoracic epidural analgesia with bupivacaine 0.25% on ventilatory mechanics in patients with severe chronic obstructive pulmonary disease. Anesth Analg. 2001;92(4):1015–9.
44. Carney A, Dickinson M. Anesthesia for esophagectomy. Anesthesiol Clin. 2015;33(1):143–63.
45. Shen L, Huang YG. Role of postoperative multimodal analgesia in abdominal and pelvic enhanced recovery after surgery. Zhongguo Yi Xue Ke Xue Yuan Xue Bao. 2016;38(4):458–63.
46. Feldheiser A, Aziz O, Baldini G, Cox BP, Fearon KC, Feldman LS, Gan TJ, Kennedy RH, Ljungqvist O, Lobo DN, Miller T, Radtke FF, Ruiz Garces T, Schricker T, Scott MJ, Thacker JK, Ytrebo LM, Carli F. Enhanced Recovery After Surgery (ERAS) for gastrointestinal surgery, part 2: consensus statement for anaesthesia practice. Acta Anaesthesiol Scand. 2016;60(3):289–334.
47. Marret E, Remy C, Bonnet F. Meta-analysis of epidural analgesia versus parenteral opioid analgesia after colorectal surgery. Br J Surg. 2007;94(6):665–73.
48. Taqi A, Hong X, Mistraletti G, Stein B, Charlebois P, Carli F. Thoracic epidural analgesia facilitates the restoration of bowel function and dietary intake in

patients undergoing laparoscopic colon resection using a traditional, nonaccelerated, perioperative care program. Surg Endosc. 2007;21(2):247–52.
49. Zingg U, Miskovic D, Hamel CT, Erni L, Oertli D, Metzger U. Influence of thoracic epidural analgesia on postoperative pain relief and ileus after laparoscopic colorectal resection: benefit with epidural analgesia. Surg Endosc. 2009;23(2):276–82.
50. Borgeat A, Ekatodramis G, Schenker CA. Postoperative nausea and vomiting in regional anesthesia: a review. Anesthesiology. 2003;98(2):530–47.
51. Guay J, Nishimori M, Kopp S. Epidural local anaesthetics versus opioid-based analgesic regimens for postoperative gastrointestinal paralysis, vomiting and pain after abdominal surgery. Cochrane Database Syst Rev. 2016;(7):Cd001893.
52. Borzellino G, Francis NK, Chapuis O, Krastinova E, Dyevre V, Genna M. Role of epidural analgesia within an ERAS program after laparoscopic colorectal surgery: a review and meta-analysis of randomised controlled studies. Surg Res Pract. 2016;2016:7543684.
53. Johnson RL, Kopp SL, Hebl JR, Erwin PJ, Mantilla CB. Falls and major orthopaedic surgery with peripheral nerve blockade: a systematic review and meta-analysis. Br J Anaesth. 2013;110(4):518–28.
54. Memtsoudis SG, Danninger T, Rasul R, Poeran J, Gerner P, Stundner O, Mariano ER, Mazumdar M. Inpatient falls after total knee arthroplasty: the role of anesthesia type and peripheral nerve blocks. Anesthesiology. 2014;120(3):551–63.
55. Gerrard AD, Brooks B, Asaad P, Hajibandeh S, Hajibandeh S. Meta-analysis of epidural analgesia versus peripheral nerve blockade after total knee joint replacement. Eur J Orthop Surg Traumatol. 2017;27(1):61–72.
56. Ahmed J, Khan S, Lim M, Chandrasekaran TV, MacFie J. Enhanced recovery after surgery protocols – compliance and variations in practice during routine colorectal surgery. Color Dis. 2012;14(9):1045–51.
57. Grauman S, Boethius J, Johansson J. Regional anaesthesia is associated with shorter postanaesthetic care and less pain than general anaesthesia after upper extremity surgery. Anesthesiol Res Pract. 2016;2016:6308371.
58. Siu A, Patel J, Prentice HA, Cappuzzo JM, Hashemi H, Mukherjee D. A cost analysis of regional versus general anesthesia for carotid Endarterectomy. Ann Vasc Surg. 2017;39:189–94.
59. Mears SC, Edwards PK, Barnes CL. How to decrease length of hospital stay after total knee replacement. J Surg Orthop Adv. 2016;25(1):2–7.
60. Liu H, Hu X, Duan X, Wu J. Thoracic epidural analgesia (TEA) vs. patient controlled analgesia (PCA) in laparoscopic colectomy: a meta-analysis. Hepatogastroenterology. 2014;61(133):1213–9.
61. Klein SM, Grant SA, Greengrass RA, Nielsen KC, Speer KP, White W, Warner DS, Steele SM. Interscalene brachial plexus block with a continuous catheter insertion system and a disposable infusion pump. Anesth Analg. 2000;91(6):1473–8.
62. Fowler SJ, Symons J, Sabato S, Myles PS. Epidural analgesia compared with peripheral nerve blockade after major knee surgery: a systematic review and meta-analysis of randomized trials. Br J Anaesth. 2008;100(2):154–64.
63. Sunderland S, Yarnold CH, Head SJ, Osborn JA, Purssell A, Peel JK, Schwarz SK. Regional versus general anesthesia and the incidence of unplanned health care resource utilization for postoperative pain after wrist fracture surgery: results from a retrospective quality improvement project. Reg Anesth Pain Med. 2016;41(1):22–7.
64. Ding DY, Manoli A 3rd, Galos DK, Jain S, Tejwani NC. Continuous popliteal sciatic nerve block versus single injection nerve block for ankle fracture surgery: a prospective randomized comparative trial. J Orthop Trauma. 2015;29(9):393–8.
65. Soto Mesa D, Del Valle Ruiz V, Fayad Fayad M, Cosio Carreno F, Blanco Rodriguez I, Gonzalez Castano R, Bermejo Alvarez MA. Control of postoperative pain in knee arthroplasty: single dose femoral nerve block versus continuous femoral block. Rev Esp Anestesiol Reanim. 2012;59(4):204–9.
66. Reddi D. Preventing chronic postoperative pain. Anaesthesia. 2016;71(Suppl 1):64–71.
67. Baratta JL, Schwenk ES, Viscusi ER. Clinical consequences of inadequate pain relief: barriers to optimal pain management. Plast Reconstr Surg. 2014;134(4 Suppl 2):15s–21s.
68. Ding X, Jin S, Niu X, Ren H, Fu S, Li Q. A comparison of the analgesia efficacy and side effects of paravertebral compared with epidural blockade for thoracotomy: an updated meta-analysis. PLoS One. 2014;9(5):e96233.
69. Gottschalk A, Cohen SP, Yang S, Ochroch EA. Preventing and treating pain after thoracic surgery. Anesthesiology. 2006;104(3):594–600.
70. Andreae MH, Andreae DA. Regional anaesthesia to prevent chronic pain after surgery: a Cochrane systematic review and meta-analysis. Br J Anaesth. 2013;111(5):711–20.
71. McIsaac DI, Cole ET, McCartney CJ. Impact of including regional anaesthesia in enhanced recovery protocols: a scoping review. Br J Anaesth. 2015;115(Suppl 2):ii46–56.
72. Alviar MJ, Hale T, Dungca M. Pharmacologic interventions for treating phantom limb pain. Cochrane Database Syst Rev. 2016;(10):Cd006380.
73. Ilfeld BM, Moeller-Bertram T, Hanling SR, Tokarz K, Mariano ER, Loland VJ, Madison SJ, Ferguson EJ, Morgan AC, Wallace MS. Treating intractable phantom limb pain with ambulatory continuous peripheral nerve blocks: a pilot study. Pain Med. 2013;14(6):935–42.
74. Yousef AA, Aborahma AM. The preventive value of epidural calcitonin in patients with lower limb amputation. Pain Med. 2017;18(9):1745–51.

75. Zhou C, Zhu Y, Liu Z, Ruan L. Effect of dexmedetomidine on postoperative cognitive dysfunction in elderly patients after general anaesthesia: a meta-analysis. J Int Med Res. 2016;44(6):1182–90.
76. Moller JT, Cluitmans P, Rasmussen LS, Houx P, Rasmussen H, Canet J, Rabbitt P, Jolles J, Larsen K, Hanning CD, Langeron O, Johnson T, Lauven PM, Kristensen PA, Biedler A, van Beem H, Fraidakis O, Silverstein JH, Beneken JE, Gravenstein JS. Long-term postoperative cognitive dysfunction in the elderly ISPOCD1 study. ISPOCD investigators International Study of Post-Operative Cognitive Dysfunction. Lancet. 1998;351(9106):857–61.
77. Inouye SK, Schlesinger MJ, Lydon TJ. Delirium: a symptom of how hospital care is failing older persons and a window to improve quality of hospital care. Am J Med. 1999;106(5):565–73.
78. Rundshagen I. Postoperative cognitive dysfunction. Dtsch Arztebl Int. 2014;111(8):119–25.
79. O'Brien H, Mohan H, Hare CO, Reynolds JV, Kenny RA. Mind over matter? The hidden epidemic of cognitive dysfunction in the older surgical patient. Ann Surg. 2016;265(4):677–91.
80. Rasmussen LS, Johnson T, Kuipers HM, Kristensen D, Siersma VD, Vila P, Jolles J, Papaioannou A, Abildstrom H, Silverstein JH, Bonal JA, Raeder J, Nielsen IK, Korttila K, Munoz L, Dodds C, Hanning CD, Moller JT. Does anaesthesia cause postoperative cognitive dysfunction? A randomised study of regional versus general anaesthesia in 438 elderly patients. Acta Anaesthesiol Scand. 2003;47(3):260–6.
81. Wu CL, Hsu W, Richman JM, Raja SN. Postoperative cognitive function as an outcome of regional anesthesia and analgesia. Reg Anesth Pain Med. 2004;29(3):257–68.
82. Liu SS, Wu CL. The effect of analgesic technique on postoperative patient-reported outcomes including analgesia: a systematic review. Anesth Analg. 2007;105(3):789–808.
83. Richman JM, Liu SS, Courpas G, Wong R, Rowlingson AJ, McGready J, Cohen SR, Wu CL. Does continuous peripheral nerve block provide superior pain control to opioids? A meta-analysis. Anesth Analg. 2006;102(1):248–57.
84. Shi HJ, Xue XH, Wang YL, Zhang WS, Wang ZS, Yu AL. Effects of different anesthesia methods on cognitive dysfunction after hip replacement operation in elder patients. Int J Clin Exp Med. 2015;8(3):3883–8.
85. Barbosa FT, Castro AA. Neuraxial anesthesia versus general anesthesia for urological surgery: systematic review. Sao Paulo Med J. 2013;131(3):179–86.
86. Rubin HR. Can patients evaluate the quality of hospital care? Med Care Rev. 1990;47(3):267–326.
87. Fenton JJ, Jerant AF, Bertakis KD, Franks P. The cost of satisfaction: a national study of patient satisfaction, health care utilization, expenditures, and mortality. Arch Intern Med. 2012;172(5):405–11.
88. Wu CL, Naqibuddin M, Fleisher LA. Measurement of patient satisfaction as an outcome of regional anesthesia and analgesia: a systematic review. Reg Anesth Pain Med. 2001;26(3):196–208.
89. Brull R, McCartney CJ, Chan VW, El-Beheiry H. Neurological complications after regional anesthesia: contemporary estimates of risk. Anesth Analg. 2007;104(4):965–74.
90. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Samii K. Serious complications related to regional anesthesia: results of a prospective survey in France. Anesthesiology. 1997;87(3):479–86.
91. Auroy Y, Benhamou D, Bargues L, Ecoffey C, Falissard B, Mercier FJ, Bouaziz H, Samii K. Major complications of regional anesthesia in France: The SOS Regional Anesthesia Hotline Service. Anesthesiology. 2002;97(5):1274–80.
92. Neal JM, Barrington MJ, Brull R, Hadzic A, Hebl JR, Horlocker TT, Huntoon MA, Kopp SL, Rathmell JP, Watson JC. The second ASRA practice advisory on neurologic complications associated with regional Anesthesia and pain medicine: executive summary 2015. Reg Anesth Pain Med. 2015;40(5):401–30.
93. Bornemann-Cimenti H, Sivro N, Toft F, Halb L, Sandner-Kiesling A. Neuraxial anesthesia in patients with multiple sclerosis — a systematic review. Rev Bras Anestesiol. 2017;67(4):404–10.
94. Vukusic S, Hutchinson M, Hours M, Moreau T, Cortinovis-Tourniaire P, Adeleine P, Confavreux C. Pregnancy and multiple sclerosis (the PRIMS study): clinical predictors of post-partum relapse. Brain. 2004;127(Pt 6):1353–60.
95. Pasto L, Portaccio E, Ghezzi A, Hakiki B, Giannini M, Razzolini L, Piscolla E, De Giglio L, Pozzilli C, Paolicelli D, Trojano M, Marrosu MG, Patti F, La Mantia L, Mancardi GL, Solaro C, Totaro R, Tola MR, Di Tommaso V, Lugaresi A, Moiola L, Martinelli V, Comi G, Amato MP. Epidural analgesia and cesarean delivery in multiple sclerosis post-partum relapses: the Italian cohort study. BMC Neurol. 2012;12:165.
96. Borgeat A, Dullenkopf A, Ekatodramis G, Nagy L. Evaluation of the lateral modified approach for continuous interscalene block after shoulder surgery. Anesthesiology. 2003;99(2):436–42.
97. Capdevila X, Pirat P, Bringuier S, Gaertner E, Singelyn F, Bernard N, Choquet O, Bouaziz H, Bonnet F. Continuous peripheral nerve blocks in hospital wards after orthopedic surgery: a multicenter prospective analysis of the quality of postoperative analgesia and complications in 1,416 patients. Anesthesiology. 2005;103(5):1035–45.
98. Aromaa U, Lahdensuu M, Cozanitis DA. Severe complications associated with epidural and spinal anaesthesias in Finland 1987–1993. A study based on patient insurance claims [see comment]. Acta Anaesthesiol Scand. 1997;41(4):445–52.
99. Hebl JR. The importance and implications of aseptic techniques during regional anesthesia. Reg Anesth Pain Med. 2006;31(4):311–23.
100. Sellors JE, Cyna AM, Simmons SW. Aseptic precautions for inserting an epidural catheter: a

survey of obstetric anaesthetists. Anaesthesia. 2002;57(6):593–6.
101. Zhang Z, Xu X, Ni H. Disseminated Staphylococcus aureus infection following spinal anesthesia: a case report. J Clin Anesth. 2016;33:438–41.
102. Collins AM, Connaughton J, Ridgway PF. Bare below the elbows: a comparative study of a tertiary and district general hospital. Ir Med J. 2013;106(9):272–5.
103. Allegranzi B, Pittet D. Role of hand hygiene in healthcare-associated infection prevention. J Hosp Infect. 2009;73(4):305–15.
104. Magill SS, Edwards JR, Bamberg W, Beldavs ZG, Dumyati G, Kainer MA, Lynfield R, Maloney M, McAllister-Hollod L, Nadle J, Ray SM, Thompson DL, Wilson LE, Fridkin SK. Multistate point-prevalence survey of health care-associated infections. N Engl J Med. 2014;370(13):1198–208.
105. Parks CL, Schroeder KM, Galgon RE. Personal hand gel for improved hand hygiene compliance on the regional anesthesia team. J Anesth. 2015;29(6):899–903.
106. Schneeberger PM, Janssen M, Voss A. Alpha-hemolytic streptococci: a major pathogen of iatrogenic meningitis following lumbar puncture. Case reports and a review of the literature. Infection. 1996;24(1):29–33.
107. North JB, Brophy BP. Epidural abscess: a hazard of spinal epidural anaesthesia. Aust N Z J Surg. 1979;49(4):484–5.
108. Centers for Disease Control and Prevention. Bacterial meningitis after intrapartum spinal anesthesia – New York and Ohio, 2008–2009. MMWR Morb Mortal Wkly Rep. 2010;59(3):65–9.
109. Siddiqui NT, Davies S, McGeer A, Carvalho JC, Friedman Z. The effect of gowning on labor epidural catheter colonization rate: a randomized controlled trial. Reg Anesth Pain Med. 2014;39(6):520–4.
110. James FM, George RH, Naiem H, White GJ. Bacteriologic aspects of epidural analgesia. Anesth Analg. 1976;55(2):187–90.
111. Djozic H, Pandza H, Hasukic S, Custovic S, Pandza B, Krupalija A, Beciragic E. Efficiency of local antiseptic Alkosol (ethanol, isopropanol-30g and ortophenilphenol) and povidone iodide on the incidence of surgical site infection after inguinal hernioplasty. Med Arch. 2016;70(2):108–11.
112. Dumville JC, McFarlane E, Edwards P, Lipp A, Holmes A, Liu Z. Preoperative skin antiseptics for preventing surgical wound infections after clean surgery. Cochrane Database Syst Rev. 2015;(4):Cd003949.
113. Birnbach DJ, Stein DJ, Murray O, Thys DM, Sordillo EM. Povidone iodine and skin disinfection before initiation of epidural anesthesia. Anesthesiology. 1998;88(3):668–72.
114. Shapiro JM, Bond EL, Garman JK. Use of a chlorhexidine dressing to reduce microbial colonization of epidural catheters. Anesthesiology. 1990;73(4):625–31.
115. Kerwat K, Eberhart L, Kerwat M, Horth D, Wulf H, Steinfeldt T, Wiesmann T. Chlorhexidine gluconate dressings reduce bacterial colonization rates in epidural and peripheral regional catheters. Biomed Res Int. 2015;2015:149785.
116. Horlocker TT, Wedel DJ. Regional anesthesia in the immunocompromised patient. Reg Anesth Pain Med. 2006;31(4):334–45.
117. Grass JA. The role of epidural anesthesia and analgesia in postoperative outcome. Anesthesiol Clin North Am. 2000;18(2):407–28. viii
118. Adams HA, Saatweber P, Schmitz CS, Hecker H. Postoperative pain management in orthopaedic patients: no differences in pain score, but improved stress control by epidural anaesthesia. Eur J Anaesthesiol. 2002;19(9):658–65.
119. Pitkanen MT, Aromaa U, Cozanitis DA, Forster JG. Serious complications associated with spinal and epidural anaesthesia in Finland from 2000 to 2009. Acta Anaesthesiol Scand. 2013;57(5):553–64.
120. Strafford MA, Wilder RT, Berde CB. The risk of infection from epidural analgesia in children: a review of 1620 cases. Anesth Analg. 1995;80(2):234–8.
121. Bader AM, Camann WR, Datta S. Anesthesia for cesarean delivery in patients with herpes simplex virus type-2 infections. Reg Anesth. 1990;15(5):261–3.
122. Ramanathan S, Sheth R, Turndorf H. Anesthesia for cesarean section in patients with genital herpes infections: a retrospective study. Anesthesiology. 1986;64(6):807–9.
123. Crosby ET, Halpern SH, Rolbin SH. Epidural anaesthesia for caesarean section in patients with active recurrent genital herpes simplex infections: a retrospective review. Can J Anaesth. 1989;36(6):701–4.
124. Schifitto G, McDermott MP, McArthur JC, Marder K, Sacktor N, Epstein L, Kieburtz K. Incidence of and risk factors for HIV-associated distal sensory polyneuropathy. Neurology. 2002;58(12):1764–8.
125. Zhang WW, Wang YP, Wang YQ, Ji XM, Zhang MY. Effects of different anesthetic approaches on postoperative complications in HIV-infected patients. Genet Mol Res. 2015;14(3):9130–5.
126. Hughes SC, Dailey PA, Landers D, Dattel BJ, Crombleholme WR, Johnson JL. Parturients infected with human immunodeficiency virus and regional anesthesia. Clinical and immunologic response. Anesthesiology. 1995;82(1):32–7.
127. Avidan MS, Groves P, Blott M, Welch J, Leung T, Pozniak A, Davies E, Ball C, Zuckerman M. Low complication rate associated with cesarean section under spinal anesthesia for HIV-1-infected women on antiretroviral therapy. Anesthesiology. 2002;97(2):320–4.
128. Bremerich DH, Ahr A, Buchner S, Hingott H, Kaufmann M, Faul-Burbes C, Kessler P. Anesthetic regimen for HIV positive parturients undergoing elective cesarean section. Anaesthesist. 2003;52(12):1124–31.
129. Bick RL. Coagulation abnormalities in malignancy: a review. Semin Thromb Hemost. 1992;18(4):353–72.

130. Pi J, Kang Y, Smith M, Earl M, Norigian Z, McBride A. A review in the treatment of oncologic emergencies. J Oncol Pharm Pract. 2016;22(4):625–38.
131. Fishman JA, Issa NC. Infection in organ transplantation: risk factors and evolving patterns of infection. Infect Dis Clin N Am. 2010;24(2):273–83.
132. Wedel DJ, Horlocker TT. Regional anesthesia in the febrile or infected patient. Reg Anesth Pain Med. 2006;31(4):324–33.
133. Kane RE. Neurologic deficits following epidural or spinal anesthesia. Anesth Analg. 1981;60(3):150–61.
134. Moen V, Dahlgren N, Irestedt L. Severe neurological complications after central neuraxial blockades in Sweden 1990–1999. Anesthesiology. 2004;101(4):950–9.
135. Eng RH, Seligman SJ. Lumbar puncture-induced meningitis. JAMA. 1981;245(14):1456–9.
136. Smith KM, Deddish RB, Ogata ES. Meningitis associated with serial lumbar punctures and post-hemorrhagic hydrocephalus. J Pediatr. 1986;109(6):1057–60.
137. Teele DW, Dashefsky B, Rakusan T, Klein JO. Meningitis after lumbar puncture in children with bacteremia. N Engl J Med. 1981;305(18):1079–81.
138. Ready LB, Helfer D. Bacterial meningitis in parturients after epidural anesthesia. Anesthesiology. 1989;71(6):988–90.
139. Sendi P, Bregenzer T, Zimmerli W. Spinal epidural abscess in clinical practice. QJM. 2008;101(1):1–12.
140. Rosero EB, Joshi GP. Nationwide incidence of serious complications of epidural analgesia in the United States. Acta Anaesthesiol Scand. 2016;60(6):810–20.
141. Goodman EJ, DeHorta E, Taguiam JM. Safety of spinal and epidural anesthesia in parturients with chorioamnionitis. Reg Anesth. 1996;21(5):436–41.
142. The American Society of Anesthesiologists. Practice advisory for the prevention, diagnosis, and management of infectious complications associated with neuraxial techniques: an updated report by the American Society of Anesthesiologists Task Force on infectious complications associated with neuraxial techniques and the American Society of Regional Anesthesia and Pain Medicine. Anesthesiology. 2017;126(4):585–601.

Acute Pain Management

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Introduction

Postoperative pain is one area of anesthesia that has gained significant attention over the last 20 years. The use of regional anesthesia, ultrasound, improved drugs, and catheters has all aided in improvements. New studies continue to show the increased risk of morbidity and mortality that is associated with poor or ineffective pain control in postoperative settings [1]. Complications of postoperative pain include prolonged hospital stays, wound healing delays, prolonged rehabilitation, and poor outcomes of mental health. These negative outcomes in conjunction with the push for increased efficiency in the surgical process highlight reasons that an anesthesiologist should have an effective plan in place for postoperative pain management. Ideally, the plan should involve multimodal pain control strategies and utilize multiple agents that work through one or more different routes. This strategy would allow for different mechanisms of action ideally causing a synergistic nature for preventing pain. One important aspect of multimodal pain management is regional anesthesia. The combination of improved recovery times, reduced length of hospital stay, and relatively favorable side effect profile has resulted in better outcomes. This chapter, therefore, reviews various agents and techniques which can be used by physicians in a multimodal analgesic model.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_34

Single and Continuous Nerve Blocks

Indications

Peripheral nerve blocks (PNBs) offer various clinical postoperative advantages over opioid monotherapy, such as improved pain control, reduced side effects, improved patient satisfaction, shortened PACU stay, improved physical therapy, and earlier hospital discharge. These benefits can contribute to substantial cost savings for patients, institutions, and even entire health-care systems [2, 3]. The decision to perform a single-shot peripheral nerve block (SPNB) versus a continuous peripheral nerve block (CPNB) depends on many factors. The SPNB is a quicker and less complicated procedure to perform, which may improve OR efficiency. However, it has a shorter duration of action (12–24 h), usually with the abrupt return of pain. Based on the duration of analgesia alone, the CPNB seems like the obvious choice for the patient. However, the CPNB has its set of complications, including catheter obstruction, leakage of local anesthetic around the point of catheter insertion, and migration or inadvertent removal of the catheter. Although rare, infectious complications may also occur. Other considerations for ambulatory CPNB include appropriate patient selection, proper patient education, and need for access to a health-care provider 24/7 to address any concerns or complications [4].

Before discussing the particular indications of single-shot and continuous peripheral nerve blocks (CPNBs), the clinician must consider the following absolute contraindications:

1. Patient refusal
2. Infection at the injection/catheter placement site
3. Documented allergy to local anesthetic

Patients who present a clear indication for neural blockade include [5]:

1. Orthopedic surgeries:
   (a) Single-shot: All ORIFs, debridements, and upper or lower extremity soft tissue procedures
   (b) Continuous: All joint replacement surgeries, especially those requiring postoperative physical therapy, limb amputations, and any patient requiring multiple limb procedures over a short time period, such as serial debridements
2. Thoracic surgeries:
   (a) Single-shot: VATS and chest wall debridements
   (b) Continuous: Mastectomies and thoracotomies
3. Peripheral vascular surgeries:
   (a) Single-shot: A–V grafts, vein harvesting, and vascular reanastomosis or repairs
   (b) Continuous: Sympathectomy for thrombotic limb ischemia
4. Trauma patients:
   (a) Single-shot: Closed reduction of joint dislocations
   (b) Continuous: Rib fractures, traumatic amputations, or crushed limb injuries

Techniques

The appropriate technical selection must consider several key factors in addition to the patient's comorbidities and type of surgery. Many otherwise successful nerve blocks may fail to provide adequate anesthetic conditions when the duration and site of surgery, the need for tourniquet application, or complementary intraoperative sedation are neglected [3]. The following table (Table 34.1) provides a general guideline for the most common surgical procedures and appropriate nerve block selection.

Anesthetic Infusions and Adjuncts

The choice of local anesthetic for both single-shot PNBs and catheter infusions should be based on the following factors:

1. Duration of surgery
2. Patient disposition: ambulatory vs. inpatient
3. Postoperative physical therapy
4. Time of block placement: preop vs. post-op
5. Combination with general anesthesia

Table 34.1 Site/surgery-specific neural blockade

Surgical procedure	Nerve block technique	Notes
ENT surgery		
Carotid endarterectomy	Deep + superficial cervical plexus block or interscalene block + superficial cervical plexus	Intraoperative supplementation by the surgeon is often necessary to block neuronal innervation to the carotid artery from the glossopharyngeal nerve
Radical neck dissection (post-op pain)		
Cervical lymph node biopsy		
Upper limb surgery		
Shoulder arthroscopy or arthroplasty, rotator cuff repair, humeral neck fracture	Interscalene	Often spares the deltopectoral groove and posterior arthroscopic port incision. Supplement with superficial cervical plexus block
Mid-arm and elbow procedures, forearm surgery	Supraclavicular, infraclavicular, axillary nerve blocks	Supraclavicular block may exhibit limited or slow ulnar nerve coverage. Intercostobrachial nerve (T2) must be supplemented to block tourniquet pain
Hand or wrist surgery	Axillary or infraclavicular nerve blocks	
Chest/abdomen surgery		
Thoracotomy, vats, mastectomy, chest wall procedures	Intercostal, paravertebral nerve blocks	Follow ASRA guidelines for anticoagulated patients
Rib fractures, flail chest	Paravertebral nerve block	
Abdominal incisions, cesarean section (post-op pain)	Transversus abdominis plane nerve block, rectus sheath block	Requires bilateral blocks for midline incisions
Lower limb surgery		
Hip	Lumbar plexus	Follow ASRA guidelines in anticoagulated patients
Knee, patella, femur, thigh procedures	Lumbar plexus, femoral nerve block	Posterior knee will be spared due to sciatic innervations
Amputations	Combined femoral and sciatic nerve blocks	Blocks should be placed preoperatively to reduce incidence of phantom limb pain
Tibia, fibula, ankle, foot procedures	Sciatic nerve block	Femoral (saphenous) nerve block supplementation required if the medial aspect of the lower leg is involved

In general practice, the most common agents selected for neural blockade are mepivacaine and, for longer-acting effects, ropivacaine and bupivacaine. Ropivacaine provides additional safety in the event of intravascular injection when compared to bupivacaine. Our recommended injectates for common local anesthetics are as follows (Table 34.2):

Short procedure, preoperative PNB—1:1 mixture of 1.5% mepivacaine and 0.5% ropivacaine or 1:1 mixture of 2% lidocaine with epinephrine PF and 0.5% ropivacaine. This combination offers good intraoperative anesthetic conditions for 2–3 h while providing adequate postoperative analgesia for 8–12 h.

Long procedure, preoperative PNB—0.5% ropivacaine. For most procedures lasting more than 3 h, general anesthesia should be combined with the regional technique. This agent offers adequate intraoperative and postoperative analgesia for 10–16 h.

Table 34.2 Local anesthetic pharmacodynamics

Local anesthetic	Onset (min)	Anesthesia (h)	Analgesia (h)
3% 2-chloroprocaine	5–10	1.5	2
1.5% mepivacaine	5–15	2.4–4	3–6
2% lidocaine	5–15	3–6	5–8
0.5% ropivacaine	15–25	6–8	8–16
0.25% bupivacaine	20–30	3–4	4–10
0.5% bupivacaine	15–25	8–10	16–18

Postoperative PNB—for ambulatory patients, we often select 0.2% or 0.35% ropivacaine for its selective blockade of sensory transmission and motor sparing. This agent, therefore, minimizes the patient's inability to mobilize the operative limb at home. For inpatients, a higher concentration, such as 0.5%, can be utilized due to the availability of nursing staff who can assist the patient with ambulation.

CPNB—while any local anesthetic agent can be selected for an infusion as long as the appropriate),concentration and rate are selected to avoid toxicity, the objective of a continuous PNB is often to extend analgesia (rather than anesthesia) in the postoperative period. Ropivacaine's selective blockade of sensory transmission and motor sparing at low concentrations are ideal characteristics for continuous nerve block infusions. Depending on the block technique, we recommend ropivacaine 0.2% at the following infusion rates if a regimen with only a basal infusion is chosen (Table 34.3).

Local Anesthetic Adjuvants for PNB

Prolongation of peripheral neural blockade is best accomplished by placement of a continuous nerve block catheter. However, certain agents have been studied that may offer some degree of analgesic prolongation for cases requiring <24-h coverage [6]:

Epinephrine	Prolongs medium-acting agent anesthesia by 60% and speeds onset
• Dose	1:200,000 dilution, 5 mcg/mL
• Agents	Medium-acting—lidocaine, mepivacaine; not for use with ropivacaine
• Modality	Single-shot only; not supported for use in CPNBs
• Toxicity	Tachycardia, hypertension, and neuronal ischemia; avoid in patients with diabetes or peripheral vascular disease who may have preexisting neuropathy
Clonidine	Off-label use. Prolongs medium-acting agent analgesia by 100%. Speeds onset in areas of localized infection. No benefits for tourniquet pain
• Dose	0.1–0.5 mcg/kg up to 150 mcg total
• Agents	Medium-acting—lidocaine, mepivacaine
• Modality	Single-shot only
• Toxicity	Hypotension, bradycardia, and sedation (rare if dose <1.5 mcg/kg)

All other adjuvants, including ketamine, neostigmine, tramadol, dexamethasone, and opioids, are not supported by the literature for direct combination with local anesthetic solutions during neural blockade injections. Further studies are being done to justify use of other adjuvants for prolongation.

Postoperative Management of PNB and CPNB

Single-Shot Nerve Blocks

In the immediate postoperative period, some precautions must be taken to minimize and avoid injury in those patients who have received a peripheral nerve block. All patients who have received a block should be readily identifiable by all clinical support staff. In addition to the procedure note, we recommend the routine use of a disposable identification bracelet, placed on the same limb as the patient's medical ID band. The

Table 34.3 Continuous peripheral nerve block infusions

Ropivacaine 0.2% infusions	Nerve block technique	Rate (mL/h)
Brachial plexus	Interscalene	6–8
	Supraclavicular/infraclavicular/axillary	8–10
Paraneuraxial	Paravertebral	3–6
Lumbar plexus	Lumbar plexus	8–12
	Femoral/fascia iliaca	8–12
Sacral plexus	Sciatic (all techniques)	8–12

bracelet should clearly state "nerve block, regional," or similar, in large type and, preferably, on a colored background. This will allow for rapid identification of patients who require additional precautions in the PACU, hospital floor, and at home. The patient should be routinely assessed for [7]:

1. Pain score and presence of breakthrough pain
2. Motor and sensory block
3. Blocked limb protection—i.e., padding, splinting, etc.
4. Signs of infection or hematoma at the catheter site
5. Hemodynamic stability—for paraneuraxial blocks
6. Antithrombotic therapy (ASRA guidelines)

Continuous Nerve Blocks

Management of patients with CPNBs requires daily evaluation of their catheter-pump system as well as various physiologic and physical parameters. The clinician must focus on the pain score reported by the patient to make any necessary adjustments to the anesthetic infusion. Before implementing any changes to the infusion, however, the patient should be evaluated for:

1. Stable vital signs
2. A catheter site that is clean, dry, and void of any abnormalities around the site
3. A catheter-pump circuit that is unobstructed
4. Catheter depth that corresponds to the placement record
5. Pain pump that is filled and operational

When in the presence of hemodynamic instability, paraneuraxial blocks can worsen hypotension and should be titrated very carefully with low concentrations of the local anesthetic agent. Significant hypotension or other hemodynamic instabilities may warrant withholding the catheter infusion until hemodynamic stability occurs. Any catheter site that is painful, erythematous, warm, or purulent should bring attention to the provider. There should be a low threshold for removal of catheter if infection is suspected.

To determine that a patient's breakthrough pain is not the result of a malfunctioning catheter-pump system, the clinician should inspect the tubing for kinks, disconnections, and a functioning reservoir. To evaluate the catheter for proper placement or presence of a distal obstruction, a small bolus (5 mL) of a local anesthetic (such as 1% lidocaine) can be slowly injected after careful aspiration for blood or CSF (paraneuraxial catheters). If the patient is reporting pain relief associated with the local anesthetic bolus, the infusion rate should be increased by 20%. If there is no improvement after the bolus injection, the catheter can be withdrawn 1–2 cm and rebolused. Inability to bolus the catheter due to obstruction or failure to establish any sensory level warrants removal of the catheter.

Many patients report significantly decreased analgesic requirements by the third postoperative day or sooner, at which time the catheter is often removed, and the patient is transitioned to oral pain medication. It is not unusual, however, to use a CPNB for 5–7 days and as long as 35 days (reported by the US Military). The timing of catheter removal should be customized to each patient, taking into consideration the type of surgery, physical therapy, concomitant anticoagulation, the risk of infection, and patient satisfaction. Pain pump systems are designed to be tamper-proof and safely removed by patients at home. Disposition, therefore, should not impact on the timing of discontinuation. If a patient is discharged with a pain pump, daily follow-up should be maintained either by follow-up visits or via telephone.

Complication Prevention

Complications related to neural blockade are rare but not completely avoidable. The table below highlights a few of the complications associated with neural blockade (Table 34.4). For instance, the incidence of nerve injury associated with peripheral nerve blocks varies depending on the definition between 0.02% up to 10%. While more serious injury such as permanent loss of motor function is on the low

Table 34.4 Nerve block complications and their avoidance

Complication	Blocks involved	Strategy
Infection	All	Use strict sterile technique
Vascular puncture	All	Avoid multiple needle passes and always aspirate. Caution with anticoagulated patients. Use ultrasound color flow if available. Maintain compression for 5 min if puncture occurs
Hematoma	All	Avoid multiple needle passes. Use ultrasound guidance to visualize needle path and adjacent vessels. Exert caution in anticoagulated patients. Monitor patient after block for signs of bleeding
Last	All	Aspirate before each injection. Inject in 5 cc boluses between aspirations. Avoid highly vascularized injection sites. LA mixtures have additive toxicities. Remain within the recommended drug dosage. Avoid bupivacaine in clinical practice
Nerve injury	All	Avoid block performance under deep sedation/general anesthesia. Avoid forceful injections. Don't overcome resistance when injecting. Consider combining different nerve block modalities such as injection pressure monitoring, ultrasound, and nerve stimulation
Total spinal or epidural spread	Interscalene, cervical plexus, supra-clavicular, paravertebral, lumbar plexus	Avoid forceful injections. Inject slowly. Aspirate for CSF or blood before injections. Minimize drug volume

end of this range, neurologic symptoms such as tingling or residual paresthesias occur at a higher frequency. Intravascular injections of local anesthetics resulting in seizures are reported in 1:1000 cases. To prevent the occurrence of complications, some basic technical steps can be adopted regardless of the technique employed:

1. Aspirate before any injection.
2. Inject in small 5 cc boluses and continue to aspirate between injections.
3. Inject slowly.
4. Do not overcome resistance during the injection.
5. Do not inject if the pressure is greater than 20 psi (requires injection pressure monitoring).
6. Do not inject if the patient experiences a paresthesia or signs of toxicity, and, therefore, a reasonable level of consciousness is needed.

These simple maneuvers are designed for rapid and early identification of intraneural (intrafascicular) or intravascular needle-tip placement. The presence of any substance (blood, CSF, air, urine) or resistance upon injection warrants immediate withdrawal and repositioning of the needle. To respond rapidly and effectively to an accidental intravascular injection or local anesthetic systemic toxicity, the patient should be monitored using ASA standards during any nerve block procedure. We recommend the following protocol:

1. Oxygen via face mask
2. ECG, pulse oximetry, and noninvasive blood pressure monitoring and ETCO2 (for moderate to deep sedation)
3. A running IV
4. Resuscitation and intubation equipment/drugs nearby

In addition to the ASA standard monitors and resuscitation equipment, certain essential drugs should also be immediately available whenever placing a nerve block in order to rapidly treat

central nervous system and cardiovascular toxicity:

1. Midazolam—premedicate with 1–2 mg IV; if seizure develops, give 5 mg IV push.
2. Propofol—only use of midazolam is not available; large doses could exacerbate cardiovascular collapse and obtundation; if seizure develops, give 15–30 mg IV push.
3. Intralipid 20% emulsion—if signs of cardiotoxicity develop, give 1.5 mL/kg IV push.

Note: Intralipid is most effective against bupivacaine-induced cardiotoxicity [8–10].

In the event of local anesthetic systemic toxicity (LAST), the following guidelines should be followed [11]:

1. *Get help*.
2. *Initial focus*.
   (a) Airway management: ventilate with 100% oxygen.
   (b) Seizure suppression: benzodiazepines are preferred.
   (c) Basic and advanced cardiac life support (BLS/ACLS) may require prolonged effort.
   (d) Epinephrine dose should be *decreased* to 100–200 mcg IV push boluses as part of ACLS protocol due to its arrhythmogenic potential in LAST victims.
3. *Infuse* 20% lipid emulsion (values in parenthesis are for a 70-kg patient).
   (a) Bolus 1.5 mL/kg (lean body mass) intravenously over 1 min (~100 mL).
   (b) Continuous infusion at 0.25 mL/kg/min (~18 mL/min; adjust by roller clamp).
   (c) Repeat bolus once or twice for persistent cardiovascular collapse.
   (d) Double the infusion rate to 0.5 mL/kg/min if blood pressure remains low.
   (e) Continue infusion for at least 10 min after attaining circulatory stability.
   (f) Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 min.
4. *Avoid* vasopressin, calcium channel blockers, β-blockers, or local anesthetic.
5. *Alert* the nearest facility having cardiopulmonary bypass capability.
6. *Avoid propofol* in patients having signs of cardiovascular instability.

Epidurals

Indications

An epidural is an injection of a drug or contrast agent administered at the lower spine into the epidural space, the area outside of the dura mater of the spinal cord. Epidurals can result in the loss of sensation, such as the blockade of pain transmission, by inhibiting signal propagation thru nerve fibers in or around the spinal cord. These injections are frequently used for diagnostic purposes, such as determining specific nerve roots responsible for transmitting pain sensation, as well as for many therapeutic purposes. Epidurals are used therapeutically for the following reasons:

- Any major surgical procedure below T1
- Management of acute postoperative pain for thoracic procedures
- Management of acute traumatic pain due to bone fractures
- Management of radicular pain due to herniated nucleus pulposus with epidural steroid injection
- Management of radiculopathy due to lumbar spinal stenosis via steroid injection
- Discogenic pain via steroid injection
- Labor analgesia
- Cesarean section
- Prevention of phantom limb pain post-amputation

Contraindications

Because there are more nerves coming off of the spinal cord at higher levels, epidurals are more suitable for the lower body and regions of the spinal cord as high as T1. Epidurals are not possible for the head, and the epidural space is

(usually) more difficult and risky to access for neck and arm analgesia. Injections below T1 are safe, but the following absolute contraindications do exist:

- Patient refusal
- Infection at the injection site
- History of allergic reaction to any of the injected agents
- Local malignancy
- Acute spinal cord compression
- Hemodynamically significant hypovolemia or untreated bacteremia/sepsis
- Increased intracranial pressure [12]
- Anticoagulation not compatible with epidural catheter placement (ASRA guidelines)
- Patient inability to stay still during needle puncture
- Uncompensated congestive heart failure
- Uncontrolled diabetes
- Fluoroscopy injections during pregnancy

Although not considered contraindications, additional precautions should be taken in immunocompromised patients. The further risk-benefit analysis should also be considered when encountering the following factors that have been found to negatively affect outcomes: chronic pain syndrome, smoking/significant cardiopulmonary disease, axial-only pain, opioid dependence, diffuse pain, and disability claims.

Benefits (Evidence-Based)

Evaluation of the efficacy of epidurals has shown to yield the following benefits:

- Reduced autonomic hyperactivity
- Reduced cardiovascular stress
- Improved postoperative pulmonary function [13]
- Improved postoperative gastrointestinal motility
- Reduced hypercoagulability
- Short term relief for radicular pain via steroid injection

There also exists variable levels of evidence for longer-term relief for radicular pain, disability, axial pain, and avoidance of surgery [14].

Patient Preparation

Patients should be placed on ASA standard monitors as well as O_2 via face mask before any neuraxial block. Before the procedure, a working peripheral IV should be present, and a small 500 cc bolus infusion of isotonic fluids should be administered for intravascular compensation of the sympathectomy-associated hypotension that may occur. Resuscitation equipment and drugs, in particular, epinephrine and ephedrine, should be readily available to manage any cardiovascular complications, especially hypotension. Another issue that is important is sterility during this procedure. The most common bacterial cause of meningitis post neuraxial anesthesia comes from oral bacteria (Streptococcus viridans) suggesting that mask should be worn at all times during the procedure.

Epidural Injection Site Selection

The epidural injection site is critical to successful epidural analgesia and patient safety. Although poorly placed epidurals can be overcome by infusing larger doses of local anesthetic, this practice places the patient at undue risk for cardiovascular collapse, hypotension, and high neural blockade requiring intubation. Placing the epidural at a vertebral level that best approximates the center of the surgical incision minimizes both the dose and infusion rate required to achieve satisfactory analgesia and helps prevent adverse side effects.

Technique: Single-Shot Versus Catheter Versus CSE

When performing an epidural, the operator must be technically proficient to avoid any complications. As the needle passes thru the ligamentum

flavum, before entering into the epidural space, a popping sensation may be felt or heard. Air or saline is commonly used to identify correct entrance into the space. There are differing techniques that can be used to administer an epidural: single-shot, catheter, and combined spinal and epidural anesthesia (CSE). Regardless of the technique used, epidural placement is optimal if done before surgery. If the neuraxial block is placed and established before surgical incision, patient positioning, intraoperative hemodynamic stability, and postoperative analgesia are superior.

Special Situation: Epidurals and General Anesthesia

Practitioners commonly combine epidural analgesia with general anesthesia to reduce patient's needs for opioid analgesics. This practice is routinely done for a variety of surgeries, including gynecologic, orthopedic, vascular, and general surgeries. If combined with general anesthesia, catheters placed preoperatively should be tested before induction so that the patient is awake and able to report symptoms and anesthetic efficacy. The epidural infusion should ideally be used intraoperatively. If, however, epidural analgesia is withheld until the postoperative phase, a normal saline solution should be infused during surgery at 2–3 mL/h to maintain catheter patency. Typically, analgesics are administered postoperatively for a few days into the epidural space via the inserted catheter.

Epidural Infusions

Before administering an epidural infusion, the following parameters must be decided:

- Basal rate (mL/h)
- Bolus dose (mL)
- Bolus interval (min)
- Max dose (mL/h)

Regardless of the local anesthetic agent used, epidural infusion rates should be calculated using a systematic approach: basal rate dose (BRD) = levels required (L) × 1.5 mL per level × distribution factor (df). The location of the epidural determines the distribution factor (df) [10–12]:

$$\mathrm{BRD} = L \times 1.5\ \mathrm{mL} \times (\mathrm{df}) \cdot k$$

1. One milliliter of LA produces 1–1.5 dermatomes of analgesia—obese and term/near term pregnancy patients have reduced epidural spaces and should prompt a 1 mL LA = 1 dermatome conversion.
2. In the lumbar region, 2/3 of the injectate travels cephalad while 1/3 travels caudad. This is due to negative intrathoracic pressure generated during spontaneous ventilation. However, in an intubated patient, positive pressure ventilation counteracts cephalad extension; and spread may occur equally in the cephalad and caudal directions.
3. In the thoracic region, location is critical. In a spontaneously ventilating patient, the local anesthetic spread is greatest toward the midthoracic region:
   (a) High thoracic injections (C7–T2) result in preferential caudal spread.
   (b) Midthoracic injections (T3–T5) result in equally caudal and cephalad spread.
   (c) Low thoracic injections (T6–T12) result in preferential cephalad spread.
4. In the thoracic region, epidural injections move preferentially away from the thorax due to positive pressure ventilation reversing the above pattern. Example: Calculate the infusion rate for an L4–L5 epidural used to provide analgesia for an abdominal surgery with an incision from the pubic symphysis (L1) to the umbilicus (T10).

Dermatomes from catheter site to cephalad edge of incision = L4 to T10 = 5.

LA volume required to reach T10 = 1.5 mL/level × 5 levels = 7.5 mL.

LA distribution factor toward T10 = 2/3, hence 7.5 = 2/3 (bolus) → (bolus) = 7.5 × 1.5 = 11.25 mL.

Hence, 11.25 mL/h would be used as the basal rate, and 25–50% of this volume could be used as a bolus dose for breakthrough pain: approximately settings—basal rate = 12 mL/h,

Table 34.5 PCEA (patient-controlled epidural) infusion dosing table

Location	Basal rate (mL/h)	Bolus dose (mL)	Interval (min)	Total (mL/h)
Midthorax	4	1	15	8
Low thorax	6	2	15	14
Lumbar	12	4	15	28
Lumbar (labor)	6	6	10–15	42–30

bolus = 3 mL, interval = 15 min, total = 24 mL/h max.

When using patient-controlled epidural analgesia (PCEA), the bolus dose is typically 25–50% of the basal rate and is administered every 10–15 min. Based on the number of bolus doses demanded, the infusion should be adjusted after several hours (Table 34.5). The basal rate should be increased by 50% of the hourly bolus dose if patient need exceeds the number of boluses per hour. Conversely, the basal rate may be left unchanged while the frequency or dosage of the bolus is increased. To ensure patient safety and simplify evaluation of any medication changes, it is recommended that only one parameter be modified at a time

Local Anesthetics for Epidurals

The choice of local anesthetic for epidural blockade should be based on the following (Table 34.6) [15–17]:

1. Required speed of onset—elective vs. urgent/emergent situation
2. Required length of blockade—short vs. long procedures
3. Required intensity of blockade—anesthesia vs. analgesia
4. Use of adjuvants (i.e., opioids, epinephrine, etc.)

Epidural/Spinal Anesthetic Complications

Epidural Hematoma

Incidence = 1:168,000 (after epidural) 1:220,000 (after spinal) [18].

Symptoms: Localized back pain that may radiate, sensory and/or motor deficits in the lower extremities, and urinary/rectal incontinence.

Diagnosis: Spinal MRI (STAT) is the gold standard; spine CT is also acceptable.

Treatment: Surgical decompression and evacuation (emergently); neurosurgical consult should be sought upon ordering diagnostic imaging.

Risk factors: Anticoagulation, coagulopathy, traumatic needle placement, and multiple needle attempts.

Epidural Abscess

Incidence = 1:10,000 [19].

Symptoms: Localized back pain that may radiate, sensory and/or motor deficits in the lower extremities, and urinary/rectal incontinence. Symptoms are progressive and usually in the setting of fever, leukocytosis, and elevated ESR (unlike an epidural hematoma).

Diagnosis: Spinal MRI (stat) is the gold standard; spine CT is also acceptable.

Treatment: Surgical decompression and evacuation (emergently); neurosurgical consult should be sought upon ordering diagnostic imaging; in the absence of neurologic sequelae, IV antibiotics may be a treatment option.

Risk factors: IV drug abuse, concomitant non-spinal infections, and neurosurgical procedures.

Postdural Puncture Headache

Symptoms: Positional headache, often relieved by recumbency; may be bilateral, frontal, or occipital; may radiate to neck; described as throbbing and continuous; diplopia and tinnitus may be present; nausea and vomiting.

Onset: 12–72 h following a neuraxial procedure. Pneumocephalus should be suspected if symptoms are reported earlier.

Diagnosis: Clinical signs/symptoms.

Treatment: Caffeine 500 mg IV in 1 L over 2–3 h, may repeat once. Tramadol 50 mg PO q 4 h prn has shown 75% success in a recent pilot study involving PDPH patients. Generous IV and PO fluids are encouraged during the treatment phase. If conservative management fails, an epidural blood patch using 10–20 mL of

Table 34.6 Local anesthetics for epidural blockade

Drug	Concentration (%)	Onset (min)	Duration plain/+epinephrine (min)	Adjuvants
2-Chloroprocaine	3	10–15	45–60/60–90	Avoid w/opioids
Lidocaine	2	10–15	80–120/120–180	
Mepivacaine	1	15	90–160/160–200	
	2	15	Same	
Bupivacaine	0.25	15–20	160–220/180+	Avoid w/DepoDur
	0.375–0.5			Avoid alkalinization
				No prolongation w/epi
Etidocaine	1	15–20	120–200/150+	
Ropivacaine	0.5	15–20	140–180/150+	Avoid alkalinization
	0.6–0.75			No prolongation w/epi
Levobupivacaine	0.5	15–20	160–220/180+	No prolongation w/epi

sterile autologous blood will yield 90–95% symptomatic relief. A second dose will yield 95% efficacy in those patients who failed the first blood patch. Spontaneous resolution usually occurs within 7 days (72%) of cases and 87% by 6 months.

Opioid Analgesia

Patient-Controlled Analgesia

Opioid analgesia for postoperative pain management often relies on empiric dosing schedules that are dependent on a variety of patient clinical parameters: age, weight, previous opioid exposure, concurrent medications, clinical status, and pain severity [20]. Caution should be exercised with elderly, patients with renal failure, and those who are suspected to suffer from obstructive sleep apnea (OSA). When compared to parenteral administration of analgesics, patient-controlled analgesia is associated with higher opioid consumption and a higher incidence of pruritus, but better pain control and patient satisfaction [21]. Frequent patient monitoring (including patient pain assessment) and clinical judgment are imperative for the safe implementation of these agents (Table 34.7). It should be noted that fentanyl is the drug of choice in patients with hepatic insufficiency, renal insufficiency, or in patients with an intolerance to morphine or hydromorphone. In general, hydromorphone has become the most popular opioid for patient-controlled analgesia, and the use of the older agent meperidine has been discontinued at most facility in the United States.

Opioid Dosing Conversion: Step-by-Step (Table 34.8)

Table 34.7 Dosing tables for opioid-naïve patients

	Morphine PCA dosing table				
Bolus (mg)	0.5	1	1	2	2
Lockout interval (min)	6	10	6	15	10
Max boluses/h	10	6	10	4	6
Hydromorphone PCA dosing table					
Bolus (mg)	0.2	0.2	0.4	0.3	0.4
Lockout interval (min)	10	6	10	6	6
Max boluses/h	6	10	6	10	10
Fentanyl PCA dosing table					
Bolus (mg)	0.01	0.02	0.02	0.04	0.04
Lockout interval (min)	6	10	6	10	6
Max boluses/h	10	6	10	6	10
Remifentanil PCA dosing table					
Bolus (mcg/kg)	0.25	0.25	0.4	0.4	0.5
Lockout interval (min)	2	1	3	2	2
Max boluses/h	30	60	20	30	30

Table 34.8 Dosing and conversion chart for opioid analgesics in opioid-naïve patients

	Equianalgesic oral dose	Equianalgesic IV dose	Starting dose adults > 50 kg (oral)	Starting dose adults > 50 kg (IV)	Bioavailability	Starting dose adults < 50 kg (IV)
Opioid agonists						
Morphine	30 mg q 3–4 h	10 mg q 3–4 h	10–30 mg q 4 h	2.5–5 mg q 3–4 h	Oral: 0.17–0.33	0.1 mg/kg q 3–4 h
Codeine	130 mg q 3–4 h	75 mg q 3–4 h	15–60 mg q 3–4 h	60 mg q 2 h IM or SQ	Oral: 0.53	Not recommended
Fentanyl		0.1			Buccal film: 0.71 Buccal tablet: 0.65 Transdermal: 0.92	
Hydromorphone	7.5 mg q 3–4 h	1.5 mg q 3–4 h	Tablets: 2–4 mg q 4–6 h Liquid: 2.5–10 mg q 3–6 h	0.2–1 mg q 3–4 h	Oral: 0.24	0.015 mg/kg q 3–4 h
Hydrocodone	30 mg q 3–4 h	Not available	10 mg q 3–4 h	Not available		Not available
Levorphanol	4 mg q 6–8 h	2 mg q 6–8 h	4 mg q 6–8 h	0.04 mg/kg q 6–8 h	Oral: 0.7	0.02 mg/kg q 6–8 h
Meperidine	300 mg q 2–3 h	75 mg q 3 h	Not recommended	100 mg q 3 h	Oral: 0.5–0.6	0.75 mg/kg q 2–3 h
Methadone (acute)	20 mg q 6–8 h	10 mg q 6–8 h	2.5 mg q 8–12 h	2.5–10 mg q 8–12 h	Oral: 0.4–0.99	0.1 mg/kg q 6–8 h
Oxycodone	20 mg q 3–4 h	Not available	10 mg q 3–4 h	Not available	Oral: 0.6–0.87	0.1–0.2 mg/kg/dose q 4–6 h
Oxymorphone	10 mg	1 mg q 3–4 h	Immediate release: 5–10 mg q 4–6 h ER: 5 mg q 12 h	0.5 mg q 4–6 h	Oral: ~0.1	Not recommended
Opioid agonist-antagonist and partial agonist						
Buprenorphine	Not available	0.3–0.4 mg q 6–8 h	Not available	0.3 mg q 6–8 h		0.004 mg/kg q 6–8 h
Butorphanol	Not available	2 mg q 3–4 h	Not available	0.5–2 mg q 3–4 h		Not recommended
Nalbuphine	Not available	10 mg q 3–4 h	Not available	10 mg q 3–6 h	IV: 0.76–0.81	0.1 mg/kg q 3–4 h
Pentazocine	150 mg q 3–4 h	60 mg q 3–4 h	Not available	30 mg q 3–4 h	Oral: 0.2	Not recommended

- Total all oral or transdermal opioids currently taken by the patient in a 24-h period.
- Multiply the amount of each drug by its bioavailability.
- Convert each bioavailable dose to IV morphine equivalents
- Add any parenteral opioids currently taken as IV morphine equivalents.
- Total all IV morphine equivalents.
- Reduce the new total by 30% to accommodate for unknown cross-tolerance.
- Select new opioid and convert dose from IV morphine equivalent.

Neuraxial Opioids

The injection of opioids in the epidural or subarachnoid space as the sole analgesic agent or as an adjuvant to local anesthetics takes advantage of their direct action on the dorsal horn of the spinal cord, specifically on mu receptors within the substantia gelatinosa. The benefits afforded by this direct access to nociceptive receptors include:

1. Reduced dosing requirements
2. Decreased systemic side effects
3. Increased visceral analgesia for abdominal and thoracic procedures

Regarding onset and duration, opioid activity within the neuraxis is primarily determined by the lipid solubility of each medication and how each is transported throughout the CSF. Hydrophilic (or poorly lipid soluble) opioids, such as morphine, tend to exhibit a slower onset of action and greater duration. Hydrophobic (or highly lipid soluble) opioids, such as fentanyl and sufentanil, demonstrate much faster diffusion into neural tissue and, therefore, a faster onset of action and shorter duration:

1. Morphine (intrathecal dose = 0.25–1 mg; optimal epidural dose = 2.5–3.75 mg):
   (a) Poor lipid solubility (i.e., hydrophilic)
   (b) Slow onset of action = 45 min
   (c) Long duration = up to 24 h
   (d) Extensive cephalad spread = wide analgesic band
   (e) May produce delayed respiratory depression, and patients should be monitored for 24 h post-injection
2. Hydromorphone (intrathecal dose = 40–60 mcg; epidural dose = 0.25–1 mg):
   (a) Intermediate lipid solubility
   (b) Intermediate onset of action = 10–20 min
   (c) Intermediate duration = 8–12 h
   (d) Intermediate cephalad spread = intermediate analgesic band
3. Fentanyl (intrathecal dose = 10–15 mcg; epidural dose = 75–100 mcg) and sufentanil (intrathecal dose = 2–3 mcg; epidural dose = 10–15 mcg):
   (a) High lipid solubility (i.e., hydrophobic)
   (b) Fast onset of action = 6 min
   (c) Short duration = 2–6 h (sufentanil > fentanyl)
   (d) Limited cephalad spread = narrow analgesic band

The cephalad spread of neuraxial opioids within the CSF is inversely related to their degree of *lipid* solubility. In turn, this spread determines both the size of the analgesic band as well as the potential for serious side effects such as delayed respiratory depression (see Table 34.9).

Table 34.9 Neuraxial opioid side effects and treatment

Side effect	Frequency (%)	Treatment
Respiratory depression	<0.2	Naloxone bolus 0.2–2 mg IV
		Naloxone infusion 5 mcg/kg/h IV
Pruritus	20–60	Nalbuphine 3 mg IV
		Naloxone 0.04–0.08 mg IV
		Benadryl 25 mg IV
Nausea	6–50	Zofran 4 mg IV
		Check for hypotension: give IV fluid bolus PRN
Urinary retention	4–40	Catheterization

Continuous Epidural Infusions

Step 1: Test dose:

Before initializing or restarting an epidural infusion, all catheters should be tested for intravascular or intrathecal migration. A 3 cc test dose using lidocaine 1.5% with 1:200,000 epinephrine should be bolused through the catheter after negative aspiration for blood or CSF.

Positive test dose = IV or intrathecal catheter migration:

1. HR increase of 20%
2. BP increase of 20 mmHg
3. High anesthetic level (high spinal)

Step 2: Infusion:

Avoidance and Treatment of Neuraxial Opioid Side Effects

Monitoring: All patients who receive neuraxial opioids should be monitored for signs of respiratory depression.

1. May occur 6–18 h after initial injection.
2. Intrathecal morphine presents the greatest risk, and patient monitoring should be maintained for a minimum of 24 h.
3. Intrathecal fentanyl rarely produces respiratory depression after 2 h.

Risk factors:

1. Advanced age
2. Obstructive sleep apnea
3. Intrathoracic procedures
4. Concomitant systemic opioids or sedatives

Non-opioid Analgesia

Non-opioid agents are an integral component of multimodal analgesia and reduce the opioid requirements of patients while providing both a preemptive and complementary analgesic mechanism of action. This drug group is composed of various classes, including COX-2 inhibitors, NMDA agonists, acetaminophen, alpha-2 adrenergic agonists, and neuromodulators. In the postoperative management of acute pain, these agents offer an analgesic alternative that is devoid of the negative gastrointestinal, sedative, and respiratory side effects related to opioids. Optimal postoperative pain management with these compounds, however, begins with their administration preoperatively, an analgesic strategy termed "preemptive analgesia."

NSAIDs (Table 34.10)

Mechanism: This drug class blocks the synthesis of prostaglandins by inhibiting cyclooxygenase (COX) types I and II, thereby reducing inflammatory mediators of the acute pain response at the peripheral and central nervous system. They are characterized by anti-inflammatory, antipyretic, and analgesic activity.

Evidence-based analgesia: Numerous studies suggest that ketorolac and diclofenac offer the greatest analgesic equivalency to opioids when used as part of a multimodal analgesic regimen. By reducing the incidence of opioid-induced PONV, sedation, and respiratory depression via decreasing the opioid requirements of the postoperative patient, these agents also facilitate earlier discharge.

Adverse effects: Despite the numerous benefits of the NSAID drug class, they have the potential to cause gastrointestinal mucosal damage, renal tubular dysfunction, and platelet inhibition. As a result, these agents should be used with caution in patients with gastric ulcers, renal insufficiency, extremes of age, or active bleeding. Furthermore, controversy exists regarding the

Table 34.10 NSAIDs

NSAIDs	Dose (mg)	Route
Ketorolac	15–30	PO/IM/~~SQ~~/IV
Diclofenac	50–100 (max 150/day)	PO/~~IM~~/IV
Ibuprofen	300–800 (max 3200/day)	PO
Indomethacin	20–40	PO/~~PR/IM~~
Naproxen	250–500 (max 1000/day)	PO
Celecoxib	200–400	PO
Rofecoxib	25–50	PO

inhibitory effect of ketorolac on osteoblastic activity and the impact on postoperative bone healing following orthopedic surgery. Although only animal data support this finding, ketorolac regimens should be minimized to 72 h of continuous parenteral use while monitoring renal function.

Timing: Maximal benefit is achieved by preoperative administration followed by postoperative continuation through discharge and at home.

COX-2 Inhibitors (Table 34.11)

Mechanism: Selective inhibition of COX-2.

Evidence-based analgesia: Celecoxib 400 mg PO, rofecoxib 50 mg PO, and valdecoxib 40 mg PO demonstrate a 40–50% reduction in opioid requirements for postoperative pain management.

Adverse effects: COX-inhibitor activity on bone growth is dose-dependent and reversible; therefore, these agents should be used for no more than 3–5 days in the early postoperative period for orthopedic procedures. Due to their selective inhibition of COX-2, these agents minimize bleeding, renal tubular damage, and gastrointestinal damage compared to the nonselective NSAIDs. However, selective COX-2 inhibitors (like NSAIDS) should be avoided when possible in patients with a history of or an increased risk of cardiovascular disease because the coxibs increase the risk of hypertension, heart failure, arrhythmia, and ischemic cardiovascular disease. There is also a low, dose-dependent risk of myocardial infarction with use of a COX-2 inhibitor [22, 23].

Timing: Maximal benefit is achieved by preoperative administration followed by postoperative continuation for 3–5 days.

Acetaminophen

Mechanism: Selective inhibition of COX-2.

Evidence-based analgesia: Acetaminophen 35 mg/kg is equivalent to ketorolac 1 mg/kg IV. In adults, acetaminophen 2 g PO is equivalent to celecoxib 200 mg.

Adverse effects: Acetaminophen has minimal effects on postoperative bleeding or gastrointestinal mucosal damage, as well as no contraindications in the patient with preexisting cardiac disease. Overdosage, however, may result in hepatotoxicity and agranulocytosis. Avoid use in patients with G6PD deficiency, hepatic impairment, malnutrition, and severe renal impairment.

Timing: In adults, acetaminophen 1 g q 4 h as an adjuvant to patient-controlled analgesia (PCA) morphine has been shown to improve pain relief after major orthopedic procedures. The maximum dosage of 4 g daily should not be exceeded. In children, an initial preoperative dose of 30–40 mg/kg followed by a maintenance dose of 10–15 mg/kg q 4–6 h during the early postoperative period is recommended. For children, the maximum daily dosage is 75 mg/kg/day not to exceed 4 g per day.

NMDA Antagonists: Ketamine

Mechanism: Primarily, antagonism of the NMDA receptor, but also has muscarinic, voltage-sensitive calcium channel, and opioid mu

Table 34.11 COX-2 inhibitors

COX-2 drug	Route	Onset (min)	Duration (h)	COX2/COX1 activity	Side effect
Celecoxib	PO	30–50	4–8	8	Sulfa allergy, dyspnea, diarrhea
Rofecoxib (withdrawn)	PO	30–50	12–24	35	Leg edema, hypertension
Parecoxib	IM/IV	10–15	6–12	–	
Valdecoxib	PO	30–40	6–12	30	
Etoricoxib	PO	20–30	>24	106	

receptor antagonist properties. Limited local anesthetic activity is also demonstrated.

Evidence-based analgesia: Ketamine 0.1–0.2 mg/kg as an adjuvant intraoperative bolus dose has opioid-sparing effects with reduced incidence of adverse events. For sedation, a combination of ketamine 4–18 mg/kg/min with propofol 30–90 mg/kg/min greatly reduces the respiratory depression associated with the more commonly used sedative-opioid regimens. In the postoperative period, a 1:1 ratio morphine-ketamine PCA with a lockout interval of 8 min has shown positive results for analgesia after major orthopedic procedures.

Timing: In the chronic pain patient, a preoperative or adjuvant induction dose of ketamine 50 mg IV has been shown to improve postoperative pain scores via suspected central NMDA receptor antagonism. Otherwise, most analgesic regimens with ketamine are limited to the intraoperative setting as small adjuvant bolus doses or in combination with sedatives.

Neuraxial use: Although studies are limited [24], ketamine has demonstrated enhanced analgesia when 0.25 mg/kg is administered epidurally in combination with ropivacaine for total knee arthroplasty. Small epidural doses of 20–30 mg have also been shown to improve epidural morphine-induced analgesia after major abdominal surgery. Intrathecal ketamine (1 mg) has demonstrated a 50% reduction in the intrathecal morphine dose requirement while maintaining equivalent analgesia in cancer patients. All neuraxial administrations of ketamine should be limited to a few days due to the risk of subpial vacuolar myelopathy when used as a continuous infusion in doses >5 mg/day intrathecally.

Adverse effects: Include hypotension, diplopia and nystagmus, increased intraocular pressure, dysphoria, dizziness, nausea, vomiting, sialorrhea, bladder dysfunction, cystitis, dysuria, respiratory depression, and cardiac arrhythmias. Premedication with midazolam and glycopyrrolate reduces the incidence of neurologic side effects and hypersalivation. Patients should also be carefully screened for preexisting psychiatric conditions or history of drug abuse due to their predisposition for ketamine-induced psychosis.

Alpha-2 Adrenergic Agonists: Clonidine and Dexmedetomidine

Mechanism: Modulation of central alpha-2 receptors.

Evidence-based analgesia: Both clonidine and dexmedetomidine reduce opioid requirements while potentiating central analgesic activity. As discussed earlier, clonidine (up to 100 mcg) can greatly prolong the duration of medium-acting local anesthetics when used for peripheral neural blockade. Centrally, epidural (25–50 mcg/h) and intrathecal clonidine (75 mcg) improve postoperative analgesia when used as an adjuvant to the local anesthetic infusion with or without morphine. Premedication with oral clonidine has been shown to reduce the postoperative PCA morphine requirements by up to 50% after radical prostatectomy.

Dexmedetomidine also reduces postoperative pain and opioid analgesic requirements. When administered intraoperatively as 1 mg/kg followed by 0.4 mg/kg/h infusion, a 66% reduction in PCA morphine requirements has been demonstrated after major surgery. In patients managed with fentanyl PCA for postoperative analgesia, dexmedetomidine demonstrates synergy when given as an infusion, thereby enhancing analgesia while reducing respiratory depression.

Timing: Clonidine should be used in the preoperative or intraoperative setting, while dexmedetomidine can be administered throughout the perioperative period.

Adverse effects: Include nausea, atrial fibrillation, sedation, bradycardia, and hypotension. There have also been reports of refractory cardiogenic shock [25].

Neuromodulators: Gabapentin and Pregabalin

Mechanism: Anticonvulsants with structural analogy to GABA, however, do not bind directly to $GABA_A$ or $GABA_B$ receptors. The exact mechanism involves binding to the α2δ-1 subunit of voltage-gated calcium ion channels.

Evidence-based analgesia: Gabapentin (700–1200 mg) PO has been used to treat chronic neuropathic pain in escalating doses up to a maximum of 2400 mg PO per day. Preoperative gabapentin (1.2 g) has also been shown to significantly reduce opioid analgesic requirements by up to 66% without increasing side effects in patients undergoing arthroscopic knee surgery. Pregabalin is another related compound which has been studied for the treatment of neuropathic pain. Related to its greater potency, a dose of 75–100 mg PO per day, up to twice daily dosing, has demonstrated analgesic effects for chronic neuropathies of herpetic and diabetic origin. Gralise is a gastroretentive formulation of the identical compound gabapentin. Gralise provides slower release with peak levels at 9 h, once daily dosing, and improved side effect profiles when compared to older gabapentin or pregabalin. Gralise can be considered an extended release formulation compared to its immediate release formulation product Neurontin, which has been available commercially for over 20 years.

Timing: Preoperative and postoperative use are supported by the literature. Dosing before bedtime is recommended due to the sedative effects associated with these compounds.

Adverse effects: Include dizziness, drowsiness, fatigue, ataxia, peripheral edema, myalgias, and myoclonus, as well as first-degree heart block and hypotension. Some children taking gabapentin have been found to experience fever and viral infection as adverse effects.

Clinical Pearls

Epidurals

- Blood patches can be safely administered in an HIV-positive patient since the virus is neurotropic and already present in the CSF. It was thought that epidural blood patches are contraindicated in patients who have hematogenous neoplasms such as lymphoma or leukemia since the injection of autologous blood could seed the neuraxis with neoplastic cells. However, a recent study showed that epidural blood patch bears a low risk of cancer seeding when used to treat PDPH that is unresponsive to conservative treatments. Any potential diagnosis of PDPH should be made after ruling out meningitis.

Patient-Controlled Analgesia

- Avoid PCA basal rate infusions in opioid-naïve patients, OSA, and renal failure.
- Titrate doses slowly. Use smaller boluses initially and review opioid consumption per hour to adjust PCA settings. Establish a basal rate only after hourly bolus dose use has been evaluated (Table 34.7).
- Though hydromorphone has become the most popular patient-controlled analgesic agent, fentanyl and remifentanil demonstrate the lowest incidence of nausea, vomiting, constipation, and respiratory depression.
- When dosing opioids based on weight, use ideal body mass for lipophilic opioids (fentanyl, sufentanil) and total body mass for hydrophilic compounds (morphine, hydromorphone).
- PCA use is most effective when complemented by an alternative long-acting analgesic to establish a baseline level of analgesia. This can be achieved with NSAIDs, nerve blocks, epidurals, or other opioids.

Neuraxial Opioids

- In patients who are beta-blocked, look for an increase in systolic blood pressure. In the laboring obstetric patient, administer the test dose between contractions to discriminate any increase in BP from uterine pain.
- IV naloxone is the opioid antagonist of choice. In the setting of respiratory depression, however, a single IV dose is not sufficient due to naloxone's shorter duration of action than the neuraxial opioid. Respiratory depression can recur as early as 20 min following a dose of naloxone. Patients, therefore, should be transferred to a monitored unit, their epidural/intra-

thecal infusions withheld, and a naloxone infusion started at 5 mcg/kg/h (this dose will reverse respiratory depression with minimal analgesic antagonism).

Additional Future Considerations

A sublingual sufentanil preparation is currently being evaluated for acute pain management and provides certain pharmacokinetic advantages and efficacy with attractive low side effect profiles.

Exparel, a liposomal bupivacaine, which possesses effective duration of 3–4 days, is available commercially at present and will be presented with expanded indications in the near future to the FDA.

Acknowledgment We wish to thank Dr. Ralf E. Gebhard and Dr. Andres Missair for their significant contributions to the first edition of this chapter.

Review Questions

1. What fraction of the local anesthetic bolus will travel cephalad in a lumbar epidural catheter placed at the L4–L5 interspace in a spontaneously ventilating patient?
   (a) ½
   (b) 1/3
   (c) 2/3
   (d) ¾
2. How soon after a patient receives a prophylactic dose of Lovenox is it considered safe to place an epidural?
   (a) 2 h
   (b) 24 h
   (c) 12 h
   (d) No need to wait
3. What is the maximum INR that is considered safe for epidural placement?
   (a) 1
   (b) 2
   (c) 1.5
   (d) <1.5
4. The most important factor in determining anesthetic level after an epidural bolus is:
   (a) Concentration of local anesthetic
   (b) Volume of local anesthetic
   (c) Total dose of anesthetic
   (d) Patient position
5. The most important factor in determining the anesthetic level after a single-shot spinal is:
   (a) Volume of local anesthetic
   (b) Concentration of local anesthetic
   (c) Total dose of anesthetic
   (d) Baricity of local anesthetic
6. What nerve is typically spared after a single-shot interscalene block?
   (a) Median nerve
   (b) Musculocutaneous nerve
   (c) Radial nerve
   (d) Ulnar nerve
7. What steps are taken to avoid intravascular injection during nerve blockade?
   (a) Frequent aspiration before injection
   (b) Slow injection
   (c) Avoidance of injections when resistance is encountered
   (d) Adding epinephrine to the local anesthetic
8. What nerve must be blocked separately to ensure complete neural blockade during an axillary block?
   (a) Median nerve
   (b) Ulnar nerve
   (c) Radial nerve
   (d) Musculocutaneous nerve
9. A patient received an axillary single-shot nerve block for hand surgery and is complaining of tourniquet pain in the operative arm. Which nerve was missed?
   (a) Intercostobrachial
   (b) Musculocutaneous
   (c) Supraclavicular
   (d) Ulnar
10. The Bezold–Jarisch reflex is associated with which nerve block?
    (a) Axillary
    (b) Infraclavicular
    (c) Interscalene
    (d) Interscalene in the sitting position

11. Which local anesthetic has the potential for greatest cardiotoxicity if the dosage is held equal?
    (a) Lidocaine
    (b) Mepivacaine
    (c) Ropivacaine
    (d) Bupivacaine
12. Which opioid has the lowest incidence of respiratory depression when used for IV PCA?
    (a) Fentanyl
    (b) Meperidine
    (c) Hydromorphone
    (d) Morphine
13. When switching from IV to PO dosing, what conversion factor is required for hydromorphone?
    (a) 2×
    (b) 3×
    (c) 4×
    (d) 5×
14. Which non-opioid adjuvant exhibits synergy when coadministered with a fentanyl PCA?
    (a) Aspirin
    (b) Acetaminophen
    (c) Dexmedetomidine
    (d) Clonidine
15. Ketamine can be administered through which routes?
    (a) IV
    (b) IM
    (c) Epidural
    (d) All of the above

Answers

1. c
2. c
3. d
4. b
5. d
6. d
7. a
8. d
9. a
10. d
11. a
12. a
13. d
14. d
15. d

References

1. Lynch EP, Lazor MA, Gellis JE, Orav J, Goldman L, Marcantonio ER. The impact of postoperative pain on the development of postoperative delirium. Anesth Analg. 1998;86(4):781–5.
2. Richman JM, Liu SS, Courpas G, Wong R, Rowlingson AJ, McGready J, Cohen SR, Wu CL. Does continuous peripheral nerve block provide superior pain control to opioids? A meta-analysis. Anesth Analg. 2006;102(1):248–57.
3. Hadzic A, Vloka J. Peripheral nerve blocks: principles and practice. New York: McGraw-Hill; 2003.
4. Rosero EB, Joshi FP. Preemptive, preventive, multimodal analgesia: what do they really mean? Plast Reconstr Surg. 2014;134(4 Suppl 2):85S–93S.
5. Adriani J. Peripheral nerve blocks: principles and practice. In: Adriani J, Warring H, editors. Blocking of spinal nerves. 4th ed. St. Louis: Warren H. Green; 1985. p. 107–30.
6. Chelly J. Peripheral nerve blocks: a color atlas. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
7. Borgeat A, Ekatodramis G, Kalberer F, Benz C. Acute and nonacute complications associated with interscalene block and shoulder surgery: a prospective study. J Am Soc Anesthesiol. 2001;95(4):875–80.
8. Corman SL, Skledar SJ. Use of lipid emulsion to reverse local anesthetic-induced toxicity. Ann Pharmacother. 2007;41(11):1873–7.
9. Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB. Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. Anesthesiology. 2006;105(1):217–8.
10. Litz RJ, Popp M, Stehr SN, Koch T. Successful resuscitation of a patient with ropivacaine-induced asystole after axillary plexus block using lipid infusion. Anaesthesia. 2006;61(8):800–1.
11. Corcoran W, Butterworth J, Weller RS, Beck JC, Gerancher JC, Houle TT, Groban L. Local anesthetic-induced cardiac toxicity: a survey of contemporary practice strategies among academic anesthesiology departments. Anesth Analg. 2006;103(5):1322–6.
12. Hilt H, Gramm HJ, Link J. Changes in intracranial pressure associated with extradural anaesthesia. Br J Anaesth. 1986;58(6):676–80.
13. Pöpping DM, Elia N, Marret E, Remy C, Tramèr MR. Protective effects of epidural analgesia on pulmonary complications after abdominal and thoracic surgery. Arch Surg. 2008;143(10):990.

14. Friedrich JM, Harrast MA. Lumbar epidural steroid injections. Curr Sports Med Rep. 2010;9(1):43–9.
15. de Jong R. Physiology and pharmacology of local anesthesia. 2nd ed. Springfield, IL: Charles C. Thomas; 1970.
16. Axelsson K, Nydahl PA, Philipson L, Larsson P. Motor and sensory blockade after epidural injection of mepivacaine, bupivacaine, and etidocaine—a double-blind study. Anesth Analg. 1989;69(6):739–47.
17. Corke BC, Carlson CG, Dettbarn WD. The influence of 2-chloroprocaine on the subsequent analgesic potency of bupivacaine. Anesthesiology. 1984;60(1):25–7.
18. Vandermeulen EP, Van Aken H, Vermylen J. Anticoagulants and spinal-epidural anesthesia. Anesth Analg. 1994;79(6):1165–77.
19. Wang LP, Hauerberg J, Schmidt JF. Incidence of spinal epidural abscess after epidural analgesia: a national 1-year survey. J Am Soc Anesthesiol. 1999;91(6):1928.
20. Bonica JJ. Management of pain with regional analgesia. Postgrad Med J. 1984;60(710):897–904. PMCID: PMC2418093. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2418093/.
21. McNicol ED, Ferguson MC, Hudcova J. Patient controlled opioid analgesia versus non-patient controlled opioid analgesia for postoperative pain. In: McNicol ED, editor. Cochrane database of systematic reviews. John Wiley & Sons, Ltd: Chichester, UK; 2015.
22. Kearney PM, Baigent C, Godwin J, Halls H, Emberson JR, Patrono C. Do selective cyclooxygenase-2 inhibitors and traditional non-steroidal anti-inflammatory drugs increase the risk of atherothrombosis? Meta-analysis of randomised trials. BMJ. 2006;332(7553):1302–8.
23. Andersohn F, Suissa S, Garbe E. Use of first- and second-generation cyclooxygenase-2-selective nonsteroidal anti-inflammatory drugs and risk of acute myocardial infarction. Circulation. 2006;113(16):1950–7.
24. Kirdemír P, Özkoçak I, Demír T, Gögüş N. Comparison of postoperative analgesic effects of preemptively used epidural ketamine and neostigmine. J Clin Anesth. 2000;12(7):543–8.
25. Sichrovsky TC, Mittal S, Steinberg JS. Dexmedetomidine sedation leading to refractory cardiogenic shock. Anesth Analg. 2008;106(6):1784–6.

Regional Anesthesia for Chronic Disease States

35

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CNS Disorders

Historically, the use of regional anesthetic techniques in patients with preexisting central nervous system (CNS) disorders has been considered relatively contraindicated. Probably, the most conservative legal approach in these patients is to avoid regional anesthesia. The recommendations of Vandam and Dripps in 1956 were to avoid spinal anesthesia in patients with preexisting CNS disorders, and these recommendations have greatly influenced the clinical management of these patients for the last several decades. The cause of postoperative neurological disorders is multifactorial and is usually difficult to evaluate because of the many patients and surgical and anesthetic risk factors that may play a role [1]. Therefore, the abundance of contributing factors makes it extremely difficult for clinicians and investigators alike to reliably isolate the effect of anesthetic technique on neurologic outcome.

Contributing Factors to Deterioration in Preexisting Neurological Status

- Extremes of age/body habitus
- Surgical trauma
- Tourniquet inflation pressures/length of time for inflation
- Prolonged/difficult labor or normal vaginal delivery can result in a host of neurological deficits
- Improper patient positioning
- Anesthetic technique

However, high-risk patients, including those with significant cardiopulmonary disease, may benefit medically from regional anesthesia and analgesia. The decision to proceed with regional anesthesia in these patients should be made on a case-by-case basis. Meticulous regional anesthetic technique should be observed to minimize further neurological injury.

Increased ICP

Dural puncture is not recommended in patients with clinical or radiological signs of increased intracranial pressure, such as a patient with primary and metastatic brain tumors. CT or MRI evidences of high ICP are cerebral edema, lateral shift of the midline structures, and obliteration of

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_35

the fourth ventricle. The associated leakage of cerebrospinal fluid (CSF) following dural puncture decreases CSF pressure and may lead to transtentorail and cerebellar herniation. Epidural and caudal anesthesia are also contraindicated in patients with increased intracranial pressure because of the risk of accidental dural puncture and because the intracranial pressure may be further increased by injection of local anesthetic solution into the epidural space [1].

Intracranial Aneurysms and Arteriovenous Malformations

Patients with preexisting uncorrected and vascular lesions, such as saccular aneurysms or arteriovenous malformations, are at increased risk of neurological compromise during spinal or epidural anesthesia. Alterations in intracranial pressure and mean arterial pressure associated with neuraxial block may result in subarachnoid hemorrhage or cerebral infarction. On the other hand, neuraxial anesthesia in patients with a previous ischemic stroke is considered safe, but cerebral perfusion pressure should be maintained to prevent further ischemic damage [1].

Vascular malformations in the spinal cord, subdural, and epidural space which may be associated with congenital diseases such as in Von Hippel–Lindau and Klippel–Trenaunay syndrome may pose the risk of epidural bleeding or hematoma. Magnetic resonance imaging may allow verification of the possible safety of a neuraxial block. Neuraxial anesthesia should be avoided when such an examination is not available [2].

Seizure Disorders

Majority of seizures occurring in the perioperative period in patients with a preexisting seizure disorder are likely related to the patient's underlying condition and that regional anesthesia in these patients is not contraindicated. Furthermore, because the likelihood of a postoperative seizure is increased in patients with a recent seizure, it is essential to be prepared to treat seizure activity, regardless of the anesthetic and analgesic technique [3]. On the other hand, recent onset of seizure may represent pathologic intracranial conditions such as neoplasm, trauma, infection, or stroke. So in these situations, extra precaution should be taken due to primary pathological conditions causing seizure disorders.

Chronic Preexisting Central or Peripheral Nerve Conditions

Patients with a preexisting neurological condition may be at increased risk for regional anesthesia-related nerve injury on the basis of the "double crush," which hypothesizes that nerve fibers that are already compromised are also more vulnerable to injury at another site (Fig. 35.1) [4]. Anecdotal case reports and small case series suggest that neuraxial anesthesia and analgesia may be used in patients with stable neurologic symptoms without worsening their neurological deficits. However, definitive evidence supporting this practice is lacking. Therefore, a careful discussion regarding the potential risks and benefits of performing regional anesthesia in patients with preexisting neural compromise is strongly recommended (Class II) [5].

Multiple Sclerosis

Multiple sclerosis is an acquired central nervous system disease characterized by multiple sites of demyelination in the brain and spinal cord. Multiple sclerosis does not affect the peripheral nervous system. Demyelination of axons results in a slowing of sensory and motor conduction, which leads to widely variable clinical signs and symptoms specific to the sites of demyelination. Epidural and, more often, spinal anesthesia have been implicated in the relapse of multiple sclerosis, although the evidence is not strong [1]. Patients with multiple sclerosis may have exacerbations of their symptoms over time, which may occur in an unpredictable fashion. Anesthesiologists have been cautious because of concern that there could be an

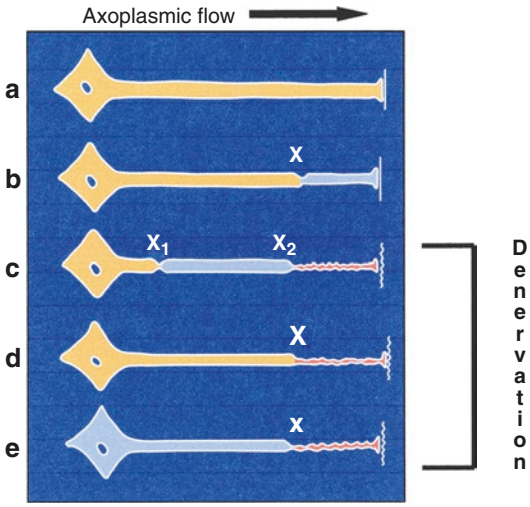

Fig. 35.1 The double-crush phenomenon. Axoplasmic flow is indicated by the *degree of shading*. Complete loss of axoplasmic flow results in denervation (**c**–**e**). (**a**) Normal neuron. (**b**) Mild neuronal injury at a single site (x) is insufficient to cause denervation distal to the insult. (**c**) Mild neuronal injury at two separate sites (x_1 and x_2) may cause distal denervation (i.e., double crush). (**d**) Severe neuronal injury at a single site (x) may also cause distal denervation. **(e)** Axon with a diffuse, preexisting underlying neurologic disease process (toxic, metabolic, and ischemic) may have impaired axonal flow throughout the neuron, which may or may not be symptomatic but predisposes the axon to distal denervation after a single minor neural insult at x (i.e., double crush)

exacerbation of the disease due to stress, fatigue, changes in temperature, and infection in perioperative period or due to natural course of disease, which could occur and even act as a coincidence. The mechanism by which spinal anesthesia may exacerbate multiple sclerosis is presumed to be direct local anesthetic toxicity. Epidural anesthesia has been recommended in preference to spinal anesthesia because the concentration of local anesthetic in the white matter of the spinal cord after epidural administration is one fourth of spinal anesthesia. A dilute solution of local anesthetic with spinal or epidural anesthesia is also advised [1]. Because multiple sclerosis is a disorder of the CNS, peripheral nerve blocks do not affect neurological function and are considered appropriate anesthetic techniques. The largest series of neuraxial anesthesia in patients with preexisting CNS conditions involved 139 patients [4]. Postpolio syndrome and multiple sclerosis were the most common CNS disorders in this study. There were no patients with new or worsening postoperative neurological deficits compared with preoperative findings. Most recent metanalysis over a period of 65 years of systematic literature search resulted in ten patients, in whom MS was worsened and 9 in whom MS or neuromyelitis optica was first diagnosed in a timely context with central neuraxial analgesia [6].

Amyotrophic Lateral Sclerosis

ALS is a degenerative disease with clinical features of both upper and lower motor neuron lesions with variability depending on the muscle groups involved. The clinical features of ALS involve progressive muscular atrophy with weakness and fasciculations of skeletal muscles. Bulbar muscle weakness often predominates with an associated risk of aspiration. Autonomic nervous system dysfunction is common with the associated risk of exaggerated hemodynamic responses during anesthesia. Epidural anesthesia has been successfully used in patients with ALS. However, a high epidural or spinal block can affect intercostal muscle function with detrimental effects in patients with minimal ventilatory reserve [7].

Chronic Spinal Cord Injury

Spinal cord injury results from trauma, bleeding, or a tumoral process. Possible consequences are bone decalcification, muscle spasms, pressure sores, deep venous thrombosis, thermoregulation problems, urological and renal complications, infections, metabolic disturbances, cardiovascular problems, and respiratory insufficiency. Lower extremity or abdominal surgery and also delivery may theoretically be performed without anesthesia if there is complete sensory loss at the operative site. However, depending on the level of cord transection, especially with lesions above T7, autonomic dysreflexia may occur after skin trauma, distention, or examination of hollow viscus. Subsequent hypertensive crisis may result in head-

ache, flushing, pupillary dilatation, convulsions, and intracranial bleeding. Prevention and early treatment of autonomic dysreflexia is critical. Regional anesthesia is preferred to general anesthesia to prevent autonomic dysreflexia. A titratable technique using a combined spinal–epidural (CSE), continuous spinal, or epidural should be considered rather than a single injection. Potential problems may be difficulty in placement, difficulty in control or examination of block level, and a potential increased risk of hypotension [2].

Previous Spine Surgeries

Previous spinal surgery has been considered to represent a relative contraindication to the use of regional anesthesia. This group of patient may vary from a simple lumbar laminectomy to a more extensive posterior spinal fusion and bone graft and placement of Harrington rod. Several postoperative anatomic changes make needle or catheter placement more difficult and complicated. Adhesions within or obliteration of the epidural space may affect the spread of epidural local anesthetic, producing an incomplete or "patchy" block, and may also increase the incidence of dural puncture. Thus, historically, it was concluded that epidural anesthesia may be successfully performed in patients who have had previous spinal surgery, but successful catheter placement may be possible on the first attempt in only 50% of patients, even by an experienced anesthesiologist. Although adequate epidural anesthesia is eventually produced in 40–95% of patients, there appears to be a higher incidence of traumatic needle placement, unintentional dural puncture, and unsuccessful epidural needle or catheter placement, especially if spinal fusion extends to between L-5 and S-1 [1]. In case of an extensive lumbar surgery, total epidural infusion volume required to produce adequate analgesia may need to be lower than normal, when the epidural is placed mid to low thoracic level. In this situations there would be high likelihood that epidural adhesions may hinder spread of local anesthetic to lumbar levels, and epidural level will spread more cephalad than usual.

Spinal Stenosis

Postoperative neurologic complications may be more likely or more severe in patients with preexisting severe spinal stenosis or other obstructive spinal canal pathology. In patients with known severe spinal stenosis or mass lesions within the spinal canal, a careful risk-to-benefit assessment of regional anesthesia to alternative perioperative anesthesia and analgesia techniques should be considered. In these patients, high local anesthetic volume neuraxial techniques (i.e., epidural anesthesia) may be associated with a higher risk of progressive mass effect when compared with low-volume techniques (i.e., spinal anesthesia) [8]. As stressed by most rescent ASRA practice advisory on neurologic complications associated with regional anesthesia, patients with spinal stenosis may have clinical or subclinical evidence of a preexisting neurological deficit because of neural compromise from the disease state. However, even moderately severe spinal stenosis is not always symptomatic; many patients (or their healthcare providers) are unaware that they have the condition (Class I) [5].

Peripheral Neurological Deficit

Patients with neuropathy or previously injured nerves (e.g., diabetes mellitus, severe peripheral vascular disease, or chemotherapy) may be at increased risk for block-related nerve injury. An abundance of animal data and limited clinical data support the concern that diabetic nerves are more sensitive to local anesthetics and perhaps more susceptible to injury [5].

Although current clinical evidence is only suggestive rather than definitive, consideration may be given to avoiding more potent local anesthetics, reducing local anesthetic dose and/or concentration, and avoiding or limiting vasoconstrictive additives in these patients [6]. There are no animal or human data to support the superiority of one nerve localization technique paresthesia, nerve stimulation, and ultrasound over another with regard to reducing the likelihood of nerve injury.

Mechanism of Neurological Injury Directly Related to Regional Anesthesia

- Direct needle- or catheter-induced trauma
- Paresthesia techniques
- The needle-bevel configuration, short/long bevel
- Prolonged exposure to high concentrations of local anesthetic solutions
- Neural ischemia may occur as a result of systemic or local vascular insufficiency

Guillain–Barré Syndrome

Guillain–Barré syndrome also involves the peripheral nervous system. Painful distal extremity paresthesias are common, and autonomic dysfunction occurs in a significant number of patients. Guillain–Barré usually resolves spontaneously over weeks to months, but approximately 20% of patients will have residual neurological deficits. Regional anesthesia is generally avoided in acute demyelinating phase of the disease. However, epidural narcotics have been used without complication in the acute phase of the disease in an attempt to control painful paresthesias. Epidural anesthesia has been used in parturients with some residual effects from an episode of Guillain–Barré in the past without adverse effects [5].

VP Shunt

VP shunt poses another challenge for neuraxial block, although formal contraindication for neuraxial block does not exist in the literature. It is common neurosurgical practice to perform lumbar puncture in patients with valveless shunts, as routine for fever investigation. There is a case report of successful spinal anesthesia for C/S in a patient with VPS without any neurologic changes secondary to the technique used [9]. Another series of five infants with VPS received spinal anesthesia for elective abdominal and perineal surgery. There was no report of complication due to spinal anesthesia [10].

Cardiovascular Disorders

The perioperative management, not just anesthetic technique, will dictate the outcome in patients with significant cardiovascular diseases. Adherence to properly drawn clinical protocols can positively influence outcome in this situations. If regional anesthesia is selected, strict control of blood pressure and heart rate during the perioperative period is required [9].

Ischemic Heart Disease

A large percentage of patients who undergo surgery in the United States have risk factors for or have known coronary artery disease, and cardiac morbidity is the primary cause of death after anesthesia and surgery. Regional anesthesia has favorable effects on ischemic heart [11, 12]. Some of these effects depend on dermatomal level of regional block and some do not. The choice of anesthesia is best left to the discretion of the anesthesia care team, which will consider the need for postoperative ventilation; cardiovascular effects, including myocardial depression; sympathetic blockade; and dermatomal level of the procedure. No one technique demonstrates a consistent advantage.

Effect of Regional Anesthesia on Myocardial Oxygen Supply and Demand

- Epidural anesthesia is associated with lower catecholamine levels
- Prevent sympathetically mediated decrease in myocardial oxygen supply (vasoconstriction and hypercoagulable state and thrombosis)

- Selective blockade of cardiac sympathetic innervation (T1–T5) can increase blood flow to ischemic regions of myocardium
- Improvement in regional distribution of myocardial blood flow by increasing the endocardial to epicardial blood flow ratio, although total coronary blood flow remains unchanged
- Increased the luminal diameter of stenotic epicardial coronary arteries without changing the diameter of non-stenotic segments

Chronic Heart Failure

The decision to use peripheral nerve block—neuraxial anesthesia as main anesthetic or adjunct to anesthesia or epidural for postoperative pain management—in patients with chronic heart failure depends on multiple factors, and generalization of recommendations is not possible. Yaeger et al. [13] reported that only 1 of 28 patients (3.6%) receiving epidural anesthesia (and "light levels of general anesthesia") and postoperative epidural analgesia developed CHF versus 10 of 25 patients (40%) given general anesthesia and postoperative parenteral narcotic analgesia, although other studies have concluded that the choice of anesthetic techniques does not significantly influence cardiac morbidity and overall mortality.

Valvular Heart Disease

Understanding of the pathophysiology of each valvular heart disease and the physiologic perturbations of neuraxial anesthesia is extremely important in management of such patients. Use of invasive monitoring should be considered in severe cases. Of the various valvular heart disease states, moderate and severe aortic stenosis (AS) is generally considered a contraindication to neuraxial anesthesia. The main reason is a risk of sudden and potentially profound decrease in systemic vascular resistance, which may precipitate life-threatening compromise in coronary perfusion. However, current evidence in the literature lacks the scientific validity provided by randomized clinical trials. The best information available are a few anecdotal observations that neuraxial anesthesia has been administered successfully in patients with significant AS while no contradictory evidence was found (i.e., adverse outcomes with neuraxial blockade in the same patient population). Benefits of regional anesthesia may outweigh the risks when the appropriate technique (continuous spinal or epidural) is selected and carefully conducted [14].

Pulmonary Hypertension

Primary pulmonary hypertension is a serious disease associated with high mortality during surgery and anesthesia. Literature on these patients is mostly limited to obstetrics case reports. The goal is to maintain right ventricular function with adequate preload, normal contractility, and sinus rhythm. Epidural anesthesia or continuous spinal anesthesia could be used in these patients as long as gradual dosing is done to avoid rapid hemodynamic changes. Spinal anesthesia is not recommended.

Pulmonary Disease

Neuraxial block reduces vital capacity (VC) and forced expiratory volume in 1 s (FEV1.0). In healthy patient under lumbar and low thoracic epidural anesthesia, these effects are negligible. Neuraxial block performed in higher levels can worsen these values by 30%. However, compared with postoperative lung function following abdominal or thoracic surgery in patients without epidural anesthesia, these effects are so small that the beneficial effects of epidural analgesia still lead to an improvement in postoperative lung function. These results can be explained by an improvement in pain therapy and diaphragmatic function and by early extubation.

In patients with COPD (chronic obstructive pulmonary disease), regional anesthesia tech-

niques lead to decreased narcotic requirements in the post-op period; however the use of thoracic epidural anesthesia has raised concerns about respiratory insufficiency due to motor blockade and the risk of bronchial constriction due to sympathetic blockade. In patients with severe asthma, thoracic epidural anesthesia decreases VC and FEV1.0 by 10% without increase in bronchial reactivity. Overall, epidural administration of local anesthetics not only provides excellent anesthesia and analgesia but also improves postoperative outcome and reduces postoperative pulmonary complications compared with anesthesia and analgesia without epidural anesthesia [15].

Interscalene brachial plexus block causes ipsilateral hemidiaphragmatic paresis in almost all patients due to the proximity of the phrenic nerve to the brachial plexus in the neck. This results in decrease in forced VC and FEV1.0. Several small studies have shown that ipsilateral phrenic nerve paralysis can also occur with the supraclavicular approach for the brachial plexus block presumably due to retrograde spread of local anesthetic within the brachial plexus sheath, but with a lower incidence. In conclusion brachial plexus block probably should not be performed in patients who are dependent on intact diaphragmatic function and are unable to tolerate reduction in pulmonary function.

Hepatic and Biliary Tract Disease

Delayed metabolism of local anesthetics and coagulopathic conditions are the main factors affecting regional anesthesia in patients with hepatic insufficiency. The pharmacokinetics of the majority of local anesthetics is affected by a poorly functioning liver associated with alterations in circulation and body fluids. All amide local anesthetics have hepatic metabolism, and less than 10% are excreted unchanged in the urine. When repeated doses or continuous infusions are used (epidural infusion or perineural catheter infusion), the accumulation of local anesthetics and their metabolites needs to be considered, and doses should be reduced accordingly. It should be kept in mind that patients with severe liver dysfunction may also have other diseases (such as nephropathy and cardiac disease), which may be even more important indications to reduce the dose of a drug. Conversely, in mild hepatic dysfunction related to alcoholism, there seems to be almost no alteration in the clearance of lidocaine.

In patients with hepatic dysfunction, single-dose blocks usually can be performed safely with normal doses of the local anesthetics. However, patients with severe liver disease often have renal dysfunction, which also requires dose reduction. The doses for repeat blocks within a short time period (<5 half-lives) and the doses for continuous infusion blocks need to be reduced markedly (10–50%) in patients with liver dysfunction mainly because of a significantly reduced clearance and accumulation of the local anesthetic and its metabolites in the blood [16].

Coagulopathy in Hepatic Insufficiency

Acute and chronic liver diseases are associated with coagulation disorders due to multiple factors. This can potentially cause spinal hematoma (in neuraxial block) or bleeding in deep tissue (in plexus block).

Coagulopathy Associated with Liver Diseases

- Decreased synthesis of clotting and inhibitor factors
- Decreased clearance of activated factors
- Quantitative and qualitative platelet defect and vitamin K deficiency
- Thrombocytopenia

Liver is the site of synthesis of all of the coagulation factors except vWF. Prolongation of prothrombin time (PT) and international normalized

ratio (INR) are indicators of severity of liver damage. Thrombocytopenia is seen in acute hepatitis with and without liver failure and is a common feature in chronic advanced liver disease. Splenomegaly due to portal hypertension is considered the main cause of low platelet count in cirrhosis [17].

Renal Disease

Pharmacokinetics of local anesthetics in renal insufficiency is affected by decreased clearance of lidocaine, bupivacaine, and ropivacaine and decreased clearance of ropivacaine metabolites: 2,6-pipecoloxylidide (PPX) and 3-OH-ropivacaine.

Factors Affecting Regional Anesthesia in Patients with Renal Dysfunction

- Clearance of local anesthetics
- Alteration in their hemostasis system
- Presence of uremic neuropathy
- Co-existing metabolic acidosis (may decrease the seizure threshold for local anesthetics)

Rapid rise in plasma concentration of local anesthetics due to enhanced absorption following brachial plexus block (due to the hyperdynamic circulation) has led to recommendation to reduce the dose of local anesthetics (by 10–20%). If repeating injection of local anesthetic within the time span of less than five half-lives, or performing continuous techniques, repeat doses, or infusion rates should also be reduced (by 10–20%) due to the risk of local anesthetic toxicity or its metabolites [16].

Increased bleeding tendency in chronic renal insufficiency should be considered before regional anesthetic. Factors associated with hemostasis abnormality in uremic patients include the following:

1. Thrombocytopenia is a common finding in uremic patients. Platelet count is rarely below 80,000/mm^3. Suggested causes for deceased numbers of circulating platelets: platelet consumption, inadequate production, compliment activation during hemodialysis, heparin-induced thrombocytopenia (when heparin in used as an anticoagulation regimen in hemodialysis).
2. Platelet dysfunction—in terminal renal insufficiency, cyclic adenosine monophosphate (cAMP) is elevated, and hemodialysis partially corrects this abnormality.
3. Platelet–vessel wall interaction due to substances in uremic blood that interacts with α2bβ3 receptor.

Dialysis improves platelet abnormalities and reduces the risk of hemorrhage although hemodialysis can contribute to the bleeding through the platelet activation induced by the interaction between blood and the artificial surfaces, and also, the anticoagulation used during hemodialysis might transiently enhance bleeding diathesis [18].

Coagulopathy and Thrombocytopenia

Patients with alteration in their coagulation status are at increased risk of bleeding-related complications following regional anesthesia. The coagulopathy could be related to systemic illness (e.g., hepatic failure) or due to use of medications that alters the coagulation system (e.g., warfarin).

Neuraxial block and peripheral nerve blocks are not recommended in patients with coagulation disorder. The risk of hematoma is higher with epidural than with subarachnoid techniques. Furthermore, concurrent spinal stenosis or some preexisting neurologic diseases may worsen injury severity in the presence of neuraxial hemorrhage. The American Society of Regional Anesthesia (ASRA) has published recommendations and practice advisory for "Regional Anesthesia in the Patient Receiving

Table 35.1 Summary of third ASRA recommendation and practice advisory for regional anesthesia in patients receiving antithrombotic or thrombolytic therapy

Drug	Neuraxial block placement, catheter removal
Thrombolytic therapy	No data available
Heparin IV	2–4 h after last dose, normalized PTT
Heparin SQ	2–4 h after last dose
LMWH (thromboprophylaxis dose)	10–12 h after last dose
LMWH (therapeutic dose)	24 h after last dose
Warfarin	5 days after last dose + normal INR
Ticlopidine	14 days after last dose
Clopidogrel	7 days after last dose
Abciximab	2–4 days after last dose
Eptifibatide	4–8 h after last dose
Ticagrelor	5–7 days after last dose
Prasugrel	7–10 days after last dose
Dabigatran	5 days after the last dose
Apixaban	3 days after the last dose
Rivaroxaban	3 days after the last dose

Antithrombotic or Thrombolytic Therapy". The summary of the recommendations is shown in Table 35.1 [5, 19, 20].

The minimum platelet count below which it is safe to place a regional anesthetic is unknown. Bromage has recommended not placing an epidural anesthetic in any patient whose platelet count is 100,000/mm^3. Other authors do not define a minimum platelet count but are similarly cautious. Based on the results of a survey by Beilin et al., most anesthesiologists (66% of those in academic practice and 55% of those in private practice) will place an epidural anesthetic when the platelet count is between 80,000 and 100,000/mm^3. In a study by Beilin on 15,919 women presenting for labor and delivery, 30 received an epidural while platelet count was 69,000–98,000/mm^3, and in 22 women, epidural catheter was placed when the count was >100,000/mm^3 but subsequently decreased to 58,000–99,000/mm^3. There was no documentation of any neurologic complications in the medical records [21].

Van Veen recommends that platelet count of 80,000/mm^3 and higher is safe for placing/removing an epidural or spinal anesthetic, provided that platelet count is stable, the PT and PTT are not prolonged, and the patient is not on an antiplatelet drug or anticoagulant. It is possible that lower platelet counts also may be safe, but there is insufficient published evidence to make recommendations for lower levels at this stage. For patients with platelet counts of 50,000–80,000/mm^3 requiring epidural or spinal anesthesia, an individual decision based on risks and benefits should be made [22].

Although the most significant hemorrhagic complication of regional anesthesia is spinal hematoma, the associated risk after deep plexus block (retroperitoneal hematoma following lumbar plexus block in anticoagulated patients) and peripheral nerve block in patients with coagulopathy is undefined. It is recommended that deep peripheral blocks be managed similar to neuraxial blocks [19].

Regional Anesthesia in the Immunocompromised Patient

Patients who have altered immune status because of diabetes, neoplasm, immunosuppression after solid organ transplantation, and chronic infection with human immunodeficiency virus (HIV) or herpes simplex virus (HSV) may be at greater risk of developing infectious complications after regional anesthesia [23].

The frequency of serious CNS infections such as arachnoiditis, meningitis, and abscess after spinal or epidural anesthesia is considered to be extremely low in patients with normal immune function. Peripheral nerve block and continuous catheter techniques mostly cause bacterial localization and local inflammation. However, incidence of more serious side effects like abscess formation and necrotizing fasciitis is unknown. The attenuated inflammatory response of an immunocompromised patient may diminish the clinical sign and symptoms often associated with infection and results in a delay in diagnosis and

treatment. Although regional anesthesia is not contraindicated in these patients, certain consideration is necessary.

Some Highlights of ASRA Practice Advisory for the Prevention, Diagnosis, and Management of Infectious Complications Associated with Neuraxial Techniques [24]

- Consider alternatives to neuraxial techniques for patients at high risk.
- Consider administering preprocedure antibiotic therapy.
- Consider removing unwitnessed accidentally disconnected catheters.
- Catheters should not remain in situ longer than clinically necessary.

Acute Compartment Syndrome

Thick layers of fascia separate different groups of muscles in upper and lower extremities. These confined and nonexpendable spaces or compartments contain muscles, nerves, and blood vessels. Compartment syndrome may happen after a simple trauma but mostly happens after a crush injury of the limbs. Surgery or a tight cast is possible but less common reasons for compartment syndrome. Permanent injury to the muscle and nerves could happen if the diagnosis is delayed. This is more common when injured person is unconscious or heavily sedated and cannot complain of the pain. Although the importance of pain in the diagnosis of compartment syndrome is controversial, virtually all analgesic modalities have been linked to a delayed diagnosis of compartment syndrome. Dense local anesthetic blocks can influence the assessment of pain and movement, making the diagnosis of compartment syndrome difficult without invasive pressure monitoring. Use of epidural anesthesia with dilute concentrations of local anesthetics to avoid motor and dense sensory blocks seems warranted. Whatever the mode of analgesia used, a high index of clinical suspicion, ongoing assessment of patients, and compartment pressure measurement are essential for early diagnosis [25].

Elderly Patient Considerations

Neuraxial block in the elderly might be anatomically more challenging due to loss of disk spaces, facet joints hypertrophy, narrower interlaminar space, osteophytes, and change in consistency of ligaments. Age is also a major determinant of duration of complete motor and sensory blockade with peripheral nerve block. The elderly patients have longer duration of complete sensory and motor blockade perhaps reflecting increased sensitivity to conduction failure from local anesthetic agents in peripheral nerves in the elderly population [26].

Aortic sclerosis is common in the elderly, and it could be associated with hemodynamically significant obstruction of left ventricular outflow. Severe aortic stenosis is considered as an independent risk factor for patients undergoing general anesthesia for noncardiac surgery. Although these patients will benefit from regional anesthesia, single-shot spinal anesthesia is generally considered unsafe in patients with severe aortic stenosis. Another comorbidity that is more common in elderly patients who require shoulder or upper extremity blocks and deserves attention is pulmonary compromise. Spread of local anesthetic to phrenic nerve causes ipsilateral hemidiaphragmatic paresis and could possibly lead to respiratory failure. Phrenic nerve block is very common with interscalene block and happens 40–60% of the time in supraclavicular block. In a patient with severe pulmonary compromise infraclavicular, axillary or paravertebral posterior approach to brachial plexus should be considered. Ultrasound-guided low-volume interscalene block might decrease the incidence of phrenic nerve block.

Clinical Pearls

A thorough preoperative evaluation is critical, and patients should also be informed about perioperative implications of anesthesia, surgery and

stress, and risk versus benefit of regional anesthesia. In difficult and complex cases with multiple comorbidities, decision should be made on an individual basis. A thorough history and physical with special attention to neurologic exam is very helpful in differential diagnosis of postprocedure neurological deficit. In case of presence of a preexisting severe neurological deficit, proper documentation by a third party is necessary. In certain preexisting conditions, paresthesia technique, addition of epinephrine, and high concentrations of local anesthetics should be avoided if a regional anesthetic is administered. Anesthesiologists should not automatically take all responsibility in cases of progressive or new deficits after the procedure. Finally, it is important to be aware of standard recommendations and guidelines related to regional anesthesia and patient comorbidities.

Review Questions

1. Which one of these statements are true in patients who are considered for regional anesthesia and had history of preexisting neurologic disease?
   (a) Regional anesthesia in patients with preexisting seizure disorder is not contraindicated.
   (b) Nerve fibers that are already compromised are also more vulnerable to injury at another site.
   (c) Postoperative neurologic complications may be more likely or more severe in patients with preexisting severe spinal stenosis or other obstructive spinal canal pathology.
   (d) All of the above.
2. Which one is *false* regarding The American Society of Regional Anesthesia (ASRA) Practice Advisory for Regional Anesthesia in the Patient Receiving Antithrombotic or Thrombolytic Therapy?
   (a) ASRA recommendations are intended for use by anesthesiologists. Other healthcare providers performing neuraxial and peripheral regional anesthetic/analgesic blockade do not need to follow the recommendations.
   (b) Therapeutic anticoagulation is not contraindicated while the patient has epidural catheter, as long as coagulation status is normalized 2 h prior to catheter removal.
   (c) Risk of neuraxial hematoma is the same following epidural and spinal anesthesia techniques.
   (d) All of the above.
3. Which one of the following statements is *false*?
   (a) Brachial plexus block probably should not be performed in patients with diaphragmatic dysfunction who are unable to tolerate reduction in pulmonary function.
   (b) Although epidural anesthesia reduces vital capacity, it is not contraindicated in patients with COPD or asthma.
   (c) Pulmonary hypertension is an absolute contraindication for epidural anesthesia.
   (d) Low thoracic epidural anesthesia has minimal effects in lung volume in healthy patients.

Answers

1. d
2. d
3. c

References

1. Horlocker TT. Regional anesthesia in the patient with pre-existing neurological dysfunction. In: Gullo A, editor. Anesthesia, pain, intensive care and emergency A.P.I.C.E. Milan: Springer; 2007. p. 325–36.
2. Heytens M, Vercauteren L. Anesthetic considerations for patients with a pre-existing neurological deficit: are neuraxial techniques safe? Acta Anaesthesiol Scand. 2007;51:831–8.
3. Kopp SL, Wynd KP, Horlocker TT, et al. Regional blockade in patients with a history of a seizure disorder. Anesth Analg. 2009;109:272–8.
4. Hebel JR, Horlocker TT, Schroeder DR. Neuraxial anesthesia and analgesia in patients with preexisting central nervous system disorders. Anesth Analg. 2006;103:223–8.

5. Neal JM, Barrington MJ, Brull R, Hadzic A, Hebl JR, Horlocker TT, Huntoon MA, Kopp SL, Rathmell JP, Watson JC. The second ASRA practice advisory on neurologic complications associated with regional anesthesia and pain medicine: executive summary 2015. Reg Anesth Pain Med. 2015;40(5):401–30.
6. Bornemann-Cimenti H, Sivro N, Toft F, Halb L, Sandner-Kiesling A. Neuraxial anesthesia in patients with multiple sclerosis – a systematic review (English edition). Braz J Anesthesiol. 2016. https://doi.org/10.1016/j.bjane.2016.09.001.
7. Kattula A, Angelini G, Arndt G. Regional anesthesia in the presence of neurologic disease. In: Brendan T, editor. Finucane: complications of regional anesthesia. 2nd ed. New York: Springer; 2007. p. 373–85.
8. Neal JM, Bernards CM, Hadzic A, et al. ASRA practice advisory on neurologic complications in regional anesthesia and pain medicine. Reg Anesth Pain Med. 2008;33:404–15.
9. Goulart AP, Moro ET, Rios Rde P, Pires RT. Subarachnoid blockade for cesarean section in a patient with ventriculoperitoneal shunt: case report. Rev Bras Anestesiol. 2009;59:471–5.
10. Kachko L, Platis CM, Livni G, et al. Spinal anesthesia in infants with ventriculoperitoneal shunt: report of five cases and review of literature. Paediatr Anaesth. 2005;16:578–83.
11. Go AS, Browner WS. Cardiac outcomes after regional or general anesthesia, do we have the answer? Anesthesiology. 1996;84:1–2.
12. Liu S, Carpenter R, Neal J. Epidural anesthesia and analgesia: their role in postoperative outcome. Anesthesiology. 1995;82:1474–506.
13. Yaeger M, Glass DD, Neft RK, et al. Epidural anesthesia and analgesia in high risk surgical patients. Anesthesiology. 1987;66:729–36.
14. McDonald SB. Is neuraxial blockade contraindicated in the patient with aortic stenosis? Reg Anesth Pain Med. 2004;29:496–502.
15. Groeben H. Epidural anesthesia and pulmonary function. J Anesth. 2006;20:290–9.
16. Rosenberg H, Veering BT, Urmey WF. Maximum recommended doses of local anesthetics: a multifactorial concept. Reg Anesth Pain Med. 2004;29:564–75.
17. Amitrano L, Guardascione MA, Brancaccio V, et al. Coagulation disorders in liver disease. Semin Liver Dis. 2002;22:83–96.
18. Boccardo P, Remuzzi G, Galbusera M. Platelet dysfunction in renal failure. Semin Thromb Hemost. 2004;30:579–89.
19. Horlocker TT, Wedel D, Rowlingson J, Enneking F, Kopp S, Benzon H, et al. ASRA practice advisory. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy. American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines, 3rd edition. Reg Anesth Pain Med. 2010;35:64–101.
20. Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, Kopp SL, Benzon HT, Brown DL, Heit JA, Mulroy MF, Rosenquist RW, Tryba M, Yuan C-S. Anticoagulation 3rd edition. Reg Anesth Pain Med. 2010;35(1):64–101.
21. Beilin Y, Zahn J, Comerford M. Safe epidural analgesia in thirty parturients with platelet counts between 69,000 and 98,000/mm^3. Anesth Analg. 1997;85:385–8.
22. Van Veen JJ, Nokes TJ, Makris M. The risk of spinal hematoma following neuraxial anesthesia or lumbar puncture in thrombocytopenic individuals. Br J Hematol. 2010;148:15–25.
23. Horlocker TT, Wedel DJ. Regional anesthesia in the immunocompromised patient. Reg Anesth Pain Med. 2006;31:334–45.
24. Practice advisory for the prevention, diagnosis, and management of infectious complications associated with neuraxial techniques: a report by the American Society of Anesthesiologists task force on infectious complications associated with neuraxial techniques. Anesthesiology. 2010;112:530–45.
25. Mar GJ, Barrington MJ, BR MG. Acute compartment syndrome of the lower limb and the effect of postoperative analgesia on diagnosis. Br J Anaesth. 2009;102:3–11.
26. Paqueron X, Boccara G, Bendahou M, Coriat P, Riou B. Brachial plexus nerve block exhibits prolonged duration in the elderly. Anesthesiology. 2002;97:1245–9.

Intravenous Regional Anesthesia

36

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Introduction

Intravenous regional anesthesia (IVRA) was first described in 1908 by Bier, who used procaine and injected it intravenously between two forearm tourniquets [1]. Holmes is credited with reintroducing the technique in 1963 [2]. The reintroduction was fueled by the invention of less toxic amino amides in the mid-twentieth century. Even though Bier's technique was created over 100 years ago, the basis for current IV regional blocks is still similar. Intravenous regional blocks continue to be in favor due to a combination of the straightforward technique, reliability, rapid onset, and decreased systemic toxicity. Historically, the success rate of IVRA, around 95%, compared favorably with brachial plexus block and also avoided the associated risks of pneumothorax, neuraxial spread of local anesthetic, and arterial puncture [3, 4]. Large studies have demonstrated an impressive safety record [5, 6].

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Mechanism of Action

The mechanism of action for intravenous local anesthetics in regional anesthesia is unclear; however, two theories have been widely accepted. The first theory is that local anesthetic injected into the venous system blocks the peripheral nerves adjacent to the veins. The second theory is that the local anesthetic leaves the venous system and blocks distal branches of the peripheral nerves. It is likely that a combination of these two theories is accurate [7].

Technique

- Select an appropriately sized double tourniquet. Check tourniquet function.
- Establish IV access in the nonoperative limb.

A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_36

- Place patient in resting supine position with surgical arm extend on arm board.
- Place a small-bore IV cannula as distal as possible in the surgical extremity.
- Attach standard monitors and give supplemental oxygen.
- Place tourniquet on the arm over protective padding (e.g., Webril).
- Elevate the limb and apply the Esmarch bandage to exsanguinate blood from the extremity.
- Inflate first the distal tourniquet, then inflate the proximal tourniquet. Deflate the distal tourniquet. Tourniquet pressure should be set to 100 mmHg over systolic pressure. Pressure should never exceed 300 mmHg.
- Remove the Esmarch bandage, then check for absence of the radial pulse and that the limb remains pale.
- Inject the local anesthetic over 30 s. The patient should be warned of a possible burning or cold sensation, which is short-lived. The cannula is then removed, and pressure is applied to the insertion site with a sterile pad, while the arm is prepped for surgery.
- It is normal for the arm to appear mottled. Surgical anesthesia is achieved after about 10 min.
- After 30 min, the patient may complain of tourniquet pain. At this time, the distal tourniquet cuff is inflated followed by deflation of the proximal cuff, which will relieve tourniquet pain for up to another 20 min.
- The tourniquet must remain inflated for a minimum of 30 min after injection. After the tourniquet is released, the patient must be closely observed for signs or symptoms of local anesthetic toxicity.
- Re-inflating the tourniquet several times for 1 min after a 10-s interval of deflation has been shown to reduce the rate of rise of systemic concentration of the local anesthetic but not to reduce its magnitude [7].
- The numbness will wear off within 10 min of tourniquet release. Local infiltration of the surgical site at the close of surgery increases patient comfort (Fig. 36.1).

Forearm Tourniquet

The use of a tourniquet on the forearm reduces the volume of anesthetic required and enhances the overall safety of the procedure. Twenty-five milliliters of anesthetic is commonly used. Concerns about the effectiveness of forearm tourniquets have not been substantiated [8]. The bulky nature of the tourniquet may impede surgical access.

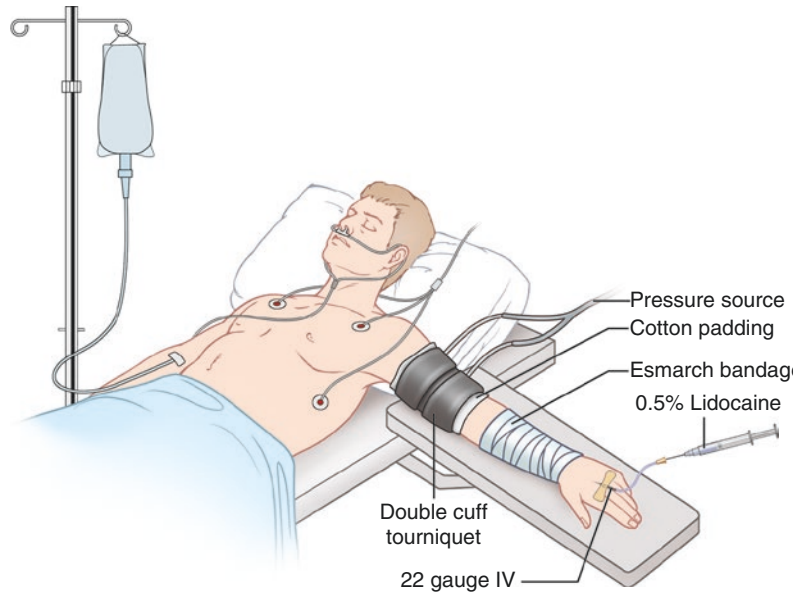

Fig. 36.1 Intravenous regional block (Bier block) of the upper extremity

IVRA for the Lower Limb

While IVRA can be used successfully for anesthesia of the lower limbs [9], higher volumes of local anesthetic are required, and the thigh tourniquet can cause the patient considerable discomfort. Discomfort is often the clinically limiting part of this technique. Placement of the tourniquet below the knee is associated with increased leakage under the tourniquet [9], and the common peroneal nerve is susceptible to injury at this site. IVRA using an ankle tourniquet has also been described for surgery on the foot; however, incomplete anesthesia may occur [10].

Choice of Local Anesthetic

There are many local anesthetic choices for IVRA (Table 36.1). The most commonly used anesthetics are lidocaine and prilocaine. Potent local anesthetics are not commonly used as there is a risk of a sudden release of a large volume of local anesthetic into the systemic circulation. IVRA using bupivacaine resulted in several deaths in the 1980s, with subsequent FDA withdrawal of approval for this use of bupivacaine [11].

Preservatives in anesthetics are associated with venous irritation and allergic reactions, and therefore preservative-free formulations are preferred [19].

Additives

Many additives to local anesthetics have been tried with the aim of improving anesthesia, ameliorating tourniquet pain, providing prolonged postoperative pain relief, and reducing anesthetic dose requirements. Clinically significant improvements have been achieved with NSAIDs, in particular, ketorolac, α-agonists such as clonidine and dexmedetomidine, and the 5-HT3 antagonist ondansetron [20–22]. Muscle relaxants have been demonstrated to improve motor block [23]. As mentioned above, additives or preservatives may increase adverse effects, such as postoperative nausea and vomiting, hypotension, and sedation [20].

IRVA and CRPS

IVRA with guanethidine or bretylium has been used in the treatment of complex regional pain syndromes. However, multiple recent reviews of

Table 36.1 Local anesthetics used in IVRA

Drug	Dose	Comment	References
Lidocaine	3 mg/kg 40 ml 0.5%	Most commonly used in the USA Cardiac arrest and death have been reported following IVRA	[12, 13]
Prilocaine	3 mg/kg 40 ml 0.5%	Less systemic toxicity than lidocaine. No reports of cardiac arrest Methemoglobinemia may occur with doses over 600 mg	[12–14]
Mepivacaine	5 mg/kg	Reported better intraoperative conditions than lidocaine	[15–17]
Ropivacaine	40 ml 0.2–0.25% (25 ml 0.375% with forearm tourniquet)	Longer postoperative analgesia than lidocaine	[11]
Bupivacaine	Not recommended	Five deaths reported following IVRA	[11]
Chloroprocaine	40 ml 0.5%	Urticaria/venous irritation Solutions containing preservative have been associated with thrombophlebitis	[14, 18]
Articaine	40 ml 0.5%	Rapid onset, rapid metabolism Skin rashes/urticaria common Not available preservative free in the USA	[12]

Table 36.2 Contraindications to IVRA

Absolute	Relative
Patient refusal	Cellulitis
True allergy to local anesthetics	Sickle cell disease
	Paget's disease of bone
	Arteriovenous fistula
	Compound fracture/ vascular injury

Table 36.3 Complications of IVRA

Major	Minor
Local anesthetic toxicity, including seizures, cardiac arrest, and death	Skin discoloration
Nerve injury	Petechiae, urticaria
Compartment syndrome, amputation	Pain on injection
	Thrombophlebitis

controlled trials, including a 2016 update to a Cochrane systematic review, have failed to show any significant benefit for this treatment modality [24–26].

Contraindications

Many of the contraindications to IVRA relate to the use of a tourniquet (Table 36.2).

Complications

The most feared complications of IVRA relate to systemic local anesthetic toxicity [27]. Systemic local anesthetic toxicity may lead to seizures, cardiac arrest, and death. Toxicity may be caused by accidental or inappropriately early release of the tourniquet. However, seizures after tourniquet time of 1 h and cardiac arrest after tourniquet time of 30 min have been reported. No cases of cardiac arrest have been reported following prilocaine IVRA (Table 36.3).

Clinical Pearls

- IVRA is unsuitable for surgery of more than 1 h, due to tourniquet pain after that time.
- It is best suited for superficial surgery of the forearm and hand which is not associated with severe postoperative pain.
- Use of a forearm tourniquet reduces the total dose of local anesthetic.
- Careful exsanguination is required to give a "dry" surgical field.
- The addition of ketorolac improves the quality of anesthesia.

Case Study

This study reports our experience with tourniquet deflation prior to 20 min with upper extremity IVRA [28].

Review Questions

1. Which of the following local anesthetics would be LEAST appropriate for IVRA?
   (a) Lidocaine
   (b) Bupivacaine
   (c) Prilocaine
   (d) All of the above are appropriate
2. All of the following local anesthetics are used in IVRA (intravenous regional anesthesia) *except*:
   (a) Lidocaine
   (b) Prilocaine
   (c) Mepivacaine
   (d) Bupivacaine
3. Clinically significant improvements to IVRA have been achieved by using additives including:
   (a) Ketorolac
   (b) Clonidine
   (c) Dexmedetomidine
   (d) Ondansetron
   (e) All of the above

Answers

1. b
2. d
3. e

References

1. Cave DA, Finegan BA. Complications of intravenous regional anesthesia. In: Complications of regional anesthesia. New York, NY: Springer New York; 2007. p. 211–23.
2. Holmes C. Intravenous regional analgesia a useful method of producing analgesia of the limbs. Lancet. 1963;281(7275):245–7.
3. Baranowski AP, Pither CE. A comparison of three methods of axillary brachial plexus anaesthesia. Anaesthesia. 1990;45(5):362–5.
4. Tuominen MK, Pitkänen MT, Numminen MK, Rosenberg PH. Quality of axillary brachial plexus block. Comparison of success rate using perivascular and nerve stimulator techniques. Anaesthesia. 1987;42(1):20–2.
5. Bartholomew K, Sloan JP. Prilocaine for Bier's block: how safe is safe? Arch Emerg Med. 1990;7(3):189–95.
6. Auroy Y, Narchi P, Messiah A, Litt L, Rouvier B, Samii K. Serious complications related to regional anesthesia: results of a prospective survey in France. Anesthesiology. 1997;87(3):479–86.
7. Farag E, Mounir-Soliman L, Preceded by (work): Brown, DL. Brown's atlas of regional anesthesia. 5th ed. Philadelphia, PA: Elsevier; 2017.
8. Coleman MM, Peng PW, Regan JM, Chan VW, Hendler AL. Quantitative comparison of leakage under the tourniquet in forearm versus conventional intravenous regional anesthesia. Anesth Analg. 1999;89(6):1482–6.
9. Brown EM, McGriff JT, Malinowski RW. Intravenous regional anaesthesia (Bier block): review of 20 years' experience. Can J Anaesth. 1989;36(3):307–10.
10. Kim DD, Shuman C, Sadr B. Intravenous regional anesthesia for outpatient foot and ankle surgery: a prospective study. Orthopedics. 1993;16(10):1109–13.
11. Heath ML. Deaths after intravenous regional anaesthesia. Br Med J (Clin Res Ed). 1982;285(6346):913–4.
12. Simon MAM, Gielen MJM, Alberink N, Vree TB, van Egmond J. Intravenous regional anesthesia with 0.5% articaine, 0.5% lidocaine, or 0.5% prilocaine: a double-blind randomized clinical study. Reg Anesth Pain Med. 1997;22(1):29–34.
13. Bader AM, Concepcion M, Hurley RJ, Arthur GR. Comparison of lidocaine and prilocaine for intravenous regional anesthesia. J Am Soc Anesthesiol. 1988;69(3):409–11.
14. Pitkänen MT, Suzuki N, Rosenberg PH. Intravenous regional anaesthesia with 0.5% prilocaine or 0.5% chloroprocaine. A double-blind comparison in volunteers. Anaesthesia. 1992;47(7):618–9.
15. Prieto-Alvarez P, Calas-Guerra A, Fuentes-Bellido J, Martínez-Verdera E, Benet-Català A, Lorenzo-Foz JP. Comparison of mepivacaine and lidocaine for intravenous regional anaesthesia: pharmacokinetic study and clinical correlation. Br J Anaesth. 2002;88(4):516–9.
16. Asik I, Kocum AI, Goktug A, Turhan KS, Alkis N. Comparison of ropivacaine 0.2% and 0.25% with lidocaine 0.5% for intravenous regional anesthesia. J Clin Anesth. 2009;21(6):401–7.
17. Niemi TT, Neuvonen PJ, Rosenberg PH. Comparison of ropivacaine 2 mg ml-1 and prilocaine 5 mg ml-1 for i.v. regional anaesthesia in outpatient surgery. Br J Anaesth. 2006;96(5):640–4.
18. Lavin PA, Henderson CL, Vaghadia H. Non-alkalinized and alkalinized 2-chloroprocainevs lidocaine for intravenous regional anesthesia during outpatient hand surgery. Can J Anesth. 1999;46(10):939–45.
19. Phillips JF, Yates AB, Deshazo RD. Approach to patients with suspected hypersensitivity to local anesthetics. Am J Med Sci. 2007;334(3):190–6.
20. Choyce A, Peng P. A systematic review of adjuncts for intravenous regional anesthesia for surgical procedures. Can J Anesth. 2002;49(1):32–45.
21. Memiş D, Turan A, Karamanlioğlu B, Pamukçu Z, Kurt I. Adding dexmedetomidine to lidocaine for intravenous regional anesthesia. Anesth Analg. 2004;98(3):835–40. table of contents
22. Honarmand A, Safavi M, Adineh-Mehr L. Effect of adding 8 milligrams ondansetron to lidocaine for Bier's block on post-operative pain. Adv Biomed Res. 2013;2:52.
23. Elhakim M, Sadek RA. Addition of atracurium to lidocaine for intravenous regional anaesthesia. Acta Anaesthesiol Scand. 1994;38(6):542–4.
24. Tran DQH, Duong S, Bertini P, Finlayson RJ. Treatment of complex regional pain syndrome: a review of the evidence. Can J Anesth. 2010;57(2):149–66.
25. Stanton TR, Wand BM, Carr DB, Birklein F, Wasner GL, O'Connell NE. Local anaesthetic sympathetic blockade for complex regional pain syndrome. Cochrane Database Syst Rev. 2013;(8):CD004598T.
26. O'Connell NE, Wand BM, Gibson W, Carr DB, Birklein F, Stanton TR. Local anaesthetic sympathetic blockade for complex regional pain syndrome. Cochrane Database Syst Rev. 2016;(7):CD004598.
27. Guay J. Adverse events associated with intravenous regional anesthesia (Bier block): a systematic review of complications. J Clin Anesth. 2009;21(8):585–94.
28. Gurich RW, Langan JW, Teasdall RJ, Tanner SL, Sanders JL. Tourniquet deflation prior to 20 minutes in upper extremity intravenous regional anesthesia. Hand (N Y); 2017 Jan 1:1558944716686214.

Pediatric Pain

37

Lori-Ann Oliver, Jodi-Ann Oliver, Hassan Rayaz, and J. Lance Lichtor

Introduction

Pain is classified as the fifth vital sign by the World Health Organization (WHO) [1]. Whether acute or chronic, it can be difficult to manage in children. Whether dealing with a child in pain after surgery or managing pain from a vaso-occlusive crisis in a child with sickle cell disease, the management of pain in children presents a daunting task for healthcare providers. There are many factors and misconceptions that contribute to this problem which must be addressed before effective pain management in the pediatric population can be understood and accomplished.

Historically, the management of pain in children has long been ignored. It was not considered a valuable part of patient care, and children either received no treatment or suboptimal pain treatment. This in part was due to a misconception concerning the ability of children to perceive pain: they were thought to have little or no perception of pain due to poorly developed neurophysiological pathways [1]. Newborns and premature infants were often subjected to painful procedures such as circumcision and pyloromyotomy, and they received little or no pain medication. To this day, in this country and worldwide, it is common practice in many neonatal intensive care units to have premature neonates intubated for prolonged periods without adequate sedation or pain control [2, 3].

A second reason for inadequate treatment of pain in children is because children, especially infants and toddlers, often lack the vocabulary or cognitive abilities to properly convey their emotions and feelings. This unfortunately has resulted in difficulty recognizing when these patients are actually in pain. As an example, post-anesthesia care unit (PACU) nurses, for neonates or infants who undergo minor procedures, thinking that a patient in pain is hungry, will give a bottle of Pedialyte instead of pain medication. Educating healthcare providers and caregivers about the symptoms of pain based on the age and developmental stage is crucial to allow for correct identification of and subsequent treatment of pain [4].

A third reason for inadequate pain treatment is the concern about opioids and the potential for addiction. Parents and other caregivers have often not administered pain medications as prescribed due to their own general attitude toward pain medications. Chronic undertreatment

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_37

of pain can result in a vicious cycle of worsening pain and behavioral changes such as agitation and aggressive and unpredictable behaviors [4, 5].

Fourthly, there's the fear of apnea, bradycardia, and even death from respiratory failure that has occurred in children who have received inappropriate amounts of opioids. Neonates and premature infants are at a greater risk for opioid overdose due to their low body weight, impaired drug metabolism, and poor renal clearance of certain drugs.

Finally, there is the mistaken perception that only pharmacologic agents make up the armamentarium of pain management strategies available for pain management in children. Regional anesthesia can be used to manage pain. Neuraxial techniques such as caudals have been the gold standard for reducing pain for a variety of urological and general surgical procedures such as circumcision, inguinal hernia repair, and pyeloplasty. With the advent of ultrasonography, the safety profile of peripheral nerve blocks has been significantly improved paving the way for advances in this field and increasing regional anesthesia popularity as a means of reducing postoperative pain in children [6].

Pain Assessment in Children

Assessing whether children are having pain is critical if pain is to be properly managed.

There are three general types of pain scales: self-reporting, observational, and physiological. Self-reporting pain is currently the gold standard for assessing pain in adults, but can be challenging, if not impossible, to use in children, particularly for infants, young children, and those with developmental delays, who cannot verbalize their pain. However, this type of pain scale has been used effectively in children 3 years or older. Two of the most commonly used pain scales are the Baker-Wong Faces Pain Rating Scale and the visual analog scale. With the Baker-Wong Faces Pain Rating Scale, children view images of faces and are asked to point to the face that best represents how they feel, as it relates to pain and discomfort. The score ranges from 0 to 10. The image portraying a face with no pain exhibits a smiling face. The practitioner evaluating the patient can then assign a pain score of 0. There is a progressive transition from a smile to a tearful and frowning face indicative of a patient in extreme pain where an evaluating practitioner can assess the pain score to be a 10. The visual analog scale is usually reserved for children 8 years and older, who are able to understand the concept of assigning a number similar to their pain [7, 8]. This scale also utilizes a 1–10 range with 1 being no pain and 10 being agonizing pain. Scores less than 5 are more indicative of milder discomfort. Scores above 5 are more indicative of dreadful and significant pain levels.

Observational pain scales are often used by the patient's medical providers, who are responsible for observing the child for any signs of pain based on objective criteria. One example of this is the FLACC (Face, Legs, Arms, Cry, Consolability) scale. The patient is assessed in each of five categories with a score applied to behaviors observed. The scores are totaled and the severity of pain is determined on a 0–10 numeric pain scale (Table 37.1). Behavioral changes are also important indicators of pain in children following surgical procedures. They include a decrease in physical activity; crankiness or irritability; nonverbal expressions of pain such as gasping, wincing, or frowning as well as physical signs such as flushing of the skin, rapid breathing, or sweating; and the tendency to favor or focus on one particular extremity [9, 10]. The behavioral observation scale (BOS) and the global rating scale (GRS) have been used with some success for the treatment of postoperative pain in children following surgery. The BOS focuses on documentation of specific behaviors indicative of pain in addition to physiologic measures such as changes in heart rate and blood pressure. The GRS includes a numerical scale as well as observation of behavior changes consistent with pain such as crying, wincing, or screaming [11, 12].

Table 37.1 FLACC scoring system for determining pain in infants and young children

Criteria	Score—0	Score—1	Score—2
Face	No particular expression or smile	Occasional grimace or frown, withdrawn, disinterested	Frequent to constant quivering chin, clenched jaw
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking or legs drawn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arched, rigid, or jerking
Cry	No cry (awake or asleep)	Moans or whimpers, occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured by occasional touching, hugging, or being talked to, distractible	Difficult to console or comfort

Based on Merkel SI, Voepel-Lewis T, Shayevitz JR, Malviya S. The FLACC: A behavioral scale for scoring postoperative pain in young children. Pediatr Nurs. 1997 May–Jun;23(3):293–7

Acute Pain

Management of acute perioperative pain is an essential part of any anesthetic plan and should be discussed with the patient and their family prior to surgery. This is especially true if peripheral nerve or neuraxial blocks are to be used. Perioperative pain management should also be discussed with the surgical team. When addressing acute pain management, one must also take into account several other factors such as age, gender, previous pain experience, temperament, cultural and family dynamics, and situational factors that can modify pain perceptions in children.

Age-Related Factors

The age and cognitive level of the patient plays an important role in creating and tailoring a pain regimen for each patient. Patients with significant developmental delay, no matter their age, especially require a pain management regimen that works for their cognitive level and disabilities. A multimodal approach is a good starting point across all age groups. Using different classes of pharmacological agents with the least amount of side effects to treat postoperative pain has been shown to have the best outcomes. This becomes extremely important in neonates and preterm infants, who are extremely prone to the deleterious side effects associated with opioids, such as respiratory depression from opioids. It is in this instance that use of non-opioid therapies is extremely important. Non-opioid options include the use of antipyretics such as acetaminophen and NSAIDS, anti-neuroleptics, and local anesthetics administered by wound infiltration, peripheral nerve blocks, and/or neuraxial blockade [13, 14]. While opioids are not completely contraindicated in this age group, patients under 1 month of age (especially preterm infants) will require vigilant monitoring for respiratory depression with the use of opioids [15]. Acetaminophen, a medication with a large therapeutic window and minimal side effect profile, has become useful to provide additional analgesia without causing respiratory depression often associated with using opioids. Infants and toddlers are less prone to respiratory depression from opioid therapy, but use of non-opioid therapy is still preferred [16].

In younger children with underdeveloped cognitive function or those with developmental delays who lack the cognitive ability to self-administer medication for pain, the use of nurse-controlled analgesia (NCA), low-dose infusions, or oral formulations of opioids are excellent options. As children grow into the preschool and school-aged years, patient-controlled analgesia with IV opioid medication can be used, as they are better able to understand and follow instructions regarding drug administration. This ability

to administer their own pain medications provides a sense of independence and control for these patients, which has been shown to not only decrease opioid consumption but also overall anxiety [17].

Opioid Pharmacology

Opioids are a mainstay for treatment of moderate-to-severe perioperative pain, sickle cell crisis, and cancer pain. They affect mu, kappa, sigma, and delta receptors in the brain, spinal cord, and peripheral nerves, thereby decreasing noxious stimulation to the CNS [18–20]. Mu receptor activation is the main function that is useful for pain management. Morphine, the most common opioid used in pediatrics, is mainly a mu receptor agonist. Other commonly used opioids like hydromorphone, fentanyl, and its subgroups, methadone, hydrocodone, and oxycodone, all have mu receptor activity with varying degrees of effect on other receptors.

Oral Formulations

Oral opioid formulations are useful alone to treat moderate pain in the perioperative setting and are also good adjuncts to the use of regional anesthesia in a multimodal regimen. Hydrocodone and oxycodone are often used. When an oral opioid medication is combined with acetaminophen, make sure the patient does not receive a potentially toxic dose of acetaminophen when acetaminophen is also used by itself or if acetaminophen is also combined with other drugs.

Codeine is another widely used oral formulation. The difference between codeine and other formulations is that it is a prodrug that requires demethylation to morphine. It is this dependence on metabolism that causes significant variability in efficacy and side effects in different patients. Codeine is not a very potent analgesic, so the risk/benefit profile is not very good on the benefit side (one-tenth as potent as morphine). The risks can be quite substantial, including death, since metabolism of the drug is variable [21]. Nausea, vomiting, constipation, and dysphoria can be severe. There has even been a case report about a rapid metabolizing mother taking codeine who transferred significant codeine metabolites to her breast-feeding child, with the child experiencing an overdose [22]. Codeine should not be used for patients after tonsillectomy and should generally be avoided in the pediatric population.

For children between 1 and 18 years old, oral methadone is available. It has a uniquely long half-life of 19 h (mean half-life) and can be used as an alternative to opioid infusions. Repeated dosing every 4–8 h will maintain stable plasma drug concentrations [23]. Furthermore, it is useful as a transition medication: weaning opioid-tolerant children off opioids as well as converting from IV to oral opioids [24–26]. When dosing methadone, however, it is important to understand the difference in its effect among opioid-naïve patients and opioid-tolerant patients. Methadone is approximately just as potent in IV morphine per dose in opioid-naïve children. It may be as low as one-tenth as potent as IV morphine in patient with significant opioid tolerance (e.g., sickle cell and cancer patients) [13–15]. As with all opioids monitoring after methadone administration is important, especially in light of its long half-life.

IV Administration

While there is a pro re nata (PRN) model of IV opioid bolus dosing as in adults, there is an increased use of different methods of opioid administration in children due to the younger, cognitively impaired, and physically disabled child's inability to follow his/her own PRN or patient-controlled analgesia (PCA) regimen. Continuous infusions allow for clinicians to manage the medication by ideally achieving an effective steady state. Rescue PRN doses of IV opioids are also available. Use of continuous pulse oximetry with consistent assessment is recommended when using IV opioid infusions [16]. Preterm and term infants are particularly sensitive to respiratory depression postoperatively due to decreased clearance [17]. Protocols for pump management and checks for accuracy of programming are important as well [16].

Morphine is the most commonly used IV opioid. Infusions ranging from 5 μg/kg/h for neonates to 16 μg/kg/h in 1–3-year-old children will achieve a target morphine concentration of 10 ng/mL [27]. Preterm infants have impaired morphine clearance (though this will improve as they grow) [27]. Term infants through 2 months of age also have impaired morphine clearance [27, 28]. This is more reason why preterm and term infants should be monitored carefully.

Respiratory depression is certainly a major side effect of IV opioids, especially in preterm and term infants receiving opiates for postoperative pain [29]. However, other side effects can also be detrimental to a child's recovery and hospital experience. Nausea/vomiting is the most common side effect of IV opioids. In one study, 42.5% of children experienced nausea/vomiting after morphine infusion [30]. Pruritus and constipation can also be persistent and frustrating for children and clinicians alike. This side effect profile is a major basis for the argument for a multimodal pain regimen that minimizes opioid use as much as possible in the hopes of improving analgesia with minimal side effects.

IV fentanyl is a useful substitute for morphine, especially in patients who don't tolerate the histamine release associated with morphine. It is 80–100 times more potent than morphine, so dosing is quite different [31, 32]. It is excreted via the kidneys. As with morphine, preterm and term infants have decreased clearance of fentanyl [31, 32]. Fentanyl has a side effect profile similar to other opioids: nausea/vomiting, pruritus, constipation, sedation, and dose-dependent respiratory depression [33].

IV hydromorphone is 3.5–7 times as potent as morphine. One study suggests a 5:1 conversion ratio of morphine dosage to hydromorphone dosage to be equianalgesic [34]. Conversions of 7:1 in children with bone marrow transplant underestimated hydromorphone requirements by 27% [34].

IV meperidine is one-tenth as potent as morphine and has an active metabolite, normeperidine. When normeperidine accumulates, there is an increased risk for seizures [35]. Meperidine is often used for treatment of postoperative shivering [36, 37].

For children older than 5–6 years, PCA is an option. A PCA allows patients to titrate the pain medication to their own satisfaction. It is an especially good tool for sickle cell and cancer patients who are admitted because of pain [34, 38, 39]. A PCA also grants a sense of autonomy in the middle of a hospital experience where much is out of the child's control. The child and his/her family should be coached on how to use the PCA and pain pumps to make sure that dosing is accurate. Problems such as human error, incorrect prescription, dispensing, administration, and equipment can all account for improper dosing with PCAs [40–42]. When a PCA regimen works well, it can lead to better analgesia with less total opioid use and side effects [43, 44].

NCA can also be used. A nurse assesses the patient and then gives the dose via the pain pump. Incidence of adverse events in opioid-naïve patients who received NCA was similar to patients receiving PCA for postoperative pain control [44]. The NCA group unfortunately required greater interventions such as airway management, escalation of management/monitoring, and opioid reversal. Cognitive impairment and first postoperative day were the most significant factors involved in these adverse events [44].

Overall, opioids are a mainstay in moderate-to-severe pain control. Their side effect profile is concerning enough that a multimodal pain management approach is recommended in treating pediatric pain. The hope is that with all of the tools at one's disposal, a clinician will be able to find the best balance of risk and benefit to manage the pain children experience.

Non-opioid Pharmacotherapy

Non-opioid medications are a good option for treating mild-to-moderate pain and can be used in conjunction with opioids to address more severe forms of pain. These non-opioid adjuncts not only decrease pain but also decrease total opioid consumption as well as the side effects associated with opioid therapy [45].

Acetaminophen

Acetaminophen works by blocking central and peripheral prostaglandin synthesis, limiting substance P and nitric oxide-induced noxious stimulus. Plasma levels of 5–20 μg per milliliter have been shown to provide adequate antipyretic as well as analgesic effect [45, 46]. The maximum recommended daily dose of acetaminophen is 45 mg/kg for preterm infants, 60 mg/kg for term infants, and 75 mg/kg for children [45, 47, 48].

Up to 10–15 mg/kg oral acetaminophen can be given every 4 h, with a maximum dose of 4 g in 24 h. Hepatotoxicity can occur when higher than recommended doses are administered [45, 49]. With the inclusion of oral acetaminophen in different types of over-the-counter as well as prescribed medications, one must be cognizant of the total dose of acetaminophen that's administered every 24 h.

Rectal acetaminophen has a less predictable rise in blood concentration. Plasma levels can peak anywhere from 60 to 180 min after initial drug administration [46, 49, 50]. In children undergoing orthopedic surgery, a loading dose of 40 mg/kg of rectal acetaminophen with repeat dosages of 20 mg/kg every 6 h produced peak plasma concentrations of 10–20 μg per milliliter with no evidence of accumulation over the first 24 h after surgery [50]. This may provide a good regimen that works for orthopedic and other types of painful procedures. First-pass acetaminophen metabolism is eliminated when acetaminophen is administered intravenously. Both uptake and peak CSF concentrations are higher when the drug is administered intravenously. Peak acetaminophen levels are reached after 57 min of drug administration, compared to 2–3 h when administered in the rectum or orally [51]. Yet rectal acetaminophen may be beneficial in prolonging the analgesic effect as shown in a randomized clinical trial comparing rectal acetaminophen 40 mg/kg and IV acetaminophen 15 mg/kg in children undergoing tonsillectomy. Those who received the rectal formulation required rescue analgesia much later compared to those who received the intravenous formulation. Though overall, in this study, both modes of administration were shown to provide good analgesia for the first 6 h in the postoperative period [51].

Nonsteroidal Anti-Inflammatory Medications

Peripherally, NSAIDs work by limiting the inflammation cascade at the site of tissue injury via inhibition of prostaglandin production at COX 1 or COX 2 receptors. NSAIDs also block glutamate and substance P activation at the spinal cord to attenuate transmission of noxious stimuli [52]. NSAIDs also cause platelet dysfunction, upset stomach for oral formulations, gastrointestinal bleeding, and renal dysfunction. Though NSAIDs cause platelet dysfunction, they are not always associated with excess bleeding. For example, a Cochrane review of patients undergoing tonsillectomy showed that the use of NSAIDs did not cause any significant increase in the number of patients who had to be taken back to the operating room due to excessive bleeding [53]. In that review, the authors also showed that postoperative nausea and vomiting was less and that NSAIDS improve pain children experience after surgery.

Ibuprofen is one of the most commonly used NSAIDs. The recommended dosage is 6–10 mg/kg every 6 h. It has been used to treat pain after surgery, trauma, arthritis, menstrual cramps, and sickle cell disease. A randomized, double-blinded study showed a greater decrease in VAS scores in children presenting to the ER with acute pain following musculoskeletal trauma after receiving ibuprofen compared to acetaminophen and codeine [54].

Diclofenac is another NSAID that can be used for perioperative pain control. The maximum dose is 1 mg/kg every 8 h when administered orally, 0.5 mg/kg rectally, and 0.3 mg/kg intravenously. Children receiving diclofenac for inguinal hernia repair had comparable analgesia to children receiving caudal bupivacaine or IV ketorolac [55, 56]. For children undergoing tonsillectomy and/or adenoidectomy, diclofenac has been shown to provide superior analgesia with better opioid sparing when compared to acetaminophen [57, 58].

Ketorolac is an intravenous NSAID that provides a level of postoperative analgesia similar to opioids, making it a particularly potent NSAID and a useful agent for minimizing overall opioid consumption [59, 60]. Its side effects are similar to those of NSAIDs overall but it also reduces renal blood flow [61]. Due to kidney effects, it's recommended that ketorolac administration should be administered no more than 48–72 h. The drug's use is effective in treating pain in neonates. One study of 37 infants and toddlers between 6 and 18 months of age showed no increased surgical drain output, renal dysfunction, hepatic lab derangement, or oxygen saturation when ketorolac and opioid were used after surgery [62]. Furthermore, a study of infants and children given ketorolac to supplement opioid therapy after open heart surgery showed no increase in renal dysfunction or complications from bleeding [63].

The role of ketorolac in tonsillectomy patients deserves special mention, as ketorolac can be beneficial in tonsillectomy patients who are at a higher risk for opioid-induced respiratory depression due to underlying obstructive sleep apnea. The concern of increased bleeding associated with ketorolac has made its use a bit controversial in this specific patient population. Most retrospective evidence reveals conflicting information regarding bleeding risk after tonsillectomy—several studies showed that perioperative administration of ketorolac is not associated with any increased postoperative bleeding risk, while others indicate quite the opposite [64, 65]. In one study, the number of patients who were brought back to the OR for hemostasis was so great that the study had to be stopped [36]. Needless to say, further research with larger randomized double-blinded studies is required before a final consensus can be reached on this matter.

Another controversial issue associated with the use of ketorolac, as with other NSAIDs, is the potential for deleterious effects on bone healing. If true, use of the drug can be problematic for patients who undergo orthopedic procedures. Prostaglandins play a major role in bone metabolism, resorption, and formation cycles, and since NSAIDs inhibit prostaglandin production, it's not surprising that several rabbit and human studies have reported a greater incidence of nonunion or pseudoarthrosis associated with the use of large doses of ketorolac. When measuring clinical outcomes for spinal fusion in patients with idiopathic scoliosis who were given ketorolac in the immediate postoperative period, there were no differences in curve progression, hardware failure, pseudoarthrosis, or the need for reoperation when compared to children and adolescents who were not given ketorolac for similar surgery [66–68]. Whether the findings are the same in patients with neuromuscular scoliosis still requires further elaboration and investigation.

Tramadol

Tramadol is a synthetic analog of codeine that has a weak affinity for mu receptors and inhibits serotonin as well as norepinephrine uptake. Adverse effects associated with the use of this medication include nausea/vomiting, pruritus, and rash [69]. Finkel et al. showed that children who received either tramadol 1 or 2 mg/kg for the purposes of transitioning from IV opioid therapy to an oral regimen showed no difference in side effect profile (sedation, oxygen desaturation) in either group but noted that those in the 2 mg/kg group required fewer doses of supplemental analgesics [69]. Khosravi et al. showed that the use of tramadol to the placement of ilioinguinal/iliohypogastric nerve blocks for pain control in children undergoing herniorrhaphy showed similar analgesic effects for both groups. The tramadol group, however, had higher incidence of nausea and vomiting [70]. Overall tramadol has a place as a lower potency opioid that can be used alone or in conjunction with other non-opioid adjuncts for improving analgesia while decreasing the total amount of opioids needed, depending on the severity of the pain.

Ketamine

Ketamine is an NMDA receptor antagonist that provides analgesia by modulating central sensitization (also referred to as also referred to "windup phenomenon") and has been used as part of a multimodal regimen due to its synergistic analgesic effects [71, 72]. In central sen-

sitization or "windup," pain increased with repeated impulses from noxious stimuli that are at least partially primed by NMDA receptor activity. Ketamine's NMDA receptor antagonist activity opposes this continuous winding up of pain signals [71, 72]. A case series involving children with refractory pain from advanced cancer showed that ketamine administration helped achieve better analgesia, improved overall functional status and reduced opioid requirement, and reduced associated side effects such as constipation, pruritus, and ileus [71].

Ketamine's ability to reduce acute postoperative pain is more nuanced. Aydin et al. showed that ketamine bolus with infusion in children undergoing tonsillectomy had decreased opioid requirements compared to ketamine bolus without infusion in children undergoing tonsillectomy [73]. Bazin et al., however, show no benefit of ketamine compared to placebo in children undergoing tonsillectomy, urologic, and orthopedic surgery [74]. There is more work to be done in determining the precise role that ketamine can have in non-cancer pediatric surgical patients.

Regional Anesthesia

Regional anesthesia is useful for all ages of children. The use of neuraxial blockade or peripheral nerve block can significantly decrease and/or prevent the side effects commonly associated with the administration of opioids and decrease the perception of pain. Peripheral nerve blocks are most commonly used for orthopedic cases. Truncal nerve blocks such as transverse abdominis plane (TAP)/rectus sheath and paravertebral blocks can be used for general surgery cases. Neuraxial blockade and paravertebral blockade can also be used for urological and thoracic cases, with thoracic epidurals in particular being important tools to limit the amount of opioid use postoperatively. For further detail, please refer to the Pediatric Regional Anesthesia chapter for more information.

Case #1: Lower Extremity Injury

A 17-year-old male with a complex past medical history significant for prematurity, intraventricular hemorrhages; hydrocephaly; Arnold-Chiari malformation, with resultant infantile cerebral palsy; malabsorption syndrome; gastroesophageal reflux disease; and scoliosis s/p T2-S2 posterior spinal infusion (PSIF) now presents for bilateral hip flexor releases and hamstring lengthening with left distal femoral extension osteotomy and left foot tibialis tendon transfer. The patient also has a history of seizures well managed with phenobarbital; the last seizure occurred approximately 4 years ago. He is also on baclofen 20 mg TID due to contractures from cerebral palsy. Due to his spinal fusion, the placement of a lumbar epidural was contraindicated, and the decision was made to place peripheral nerve blocks. Following the induction of general anesthesia, bilateral single-shot gluteal sciatic nerve blocks were placed using real-time ultrasonography, and bilateral lumbar plexus nerve blocks were placed using stimulation. 20 mL of 0.25% bupivacaine with 1:200,000 epinephrine and 5 mg of preservative-free dexamethasone were administered for both the left gluteal sciatic and lumbar plexus blocks and 10 mL of 0.25% bupivacaine with 1:200,000 epinephrine with 5 mg of preservative-free dexamethasone. Postoperatively in PACU, the patient was resting comfortably in bed and was noted to have bilateral motor and sensory blockade. He denied having any pain and did not require any additional opioids. On the ward, he was followed by the pediatric pain service where he was started on morphine NCA with IV morphine PRN for severe breakthrough pain. He was transitioned to oral oxycodone using a sliding scale, and his dose of baclofen was continued on postoperative day (POD) #1 once he was able to tolerate oral intake. The patient was discharged home on POD #4.

PNB and neuraxial blockade can both be effective measures of pain control for lower extremity surgery. Neuraxial techniques, most commonly epidurals, enable blockade of both lower extremities, while peripheral

nerve blocks will enable selection of one extremity.

Indications and contraindications for neuraxial and peripheral nerve blockade should be considered carefully. This patient has extensive spinal fusion, creating an environment of scar tissue and lack of available epidural space for the block. Patient/guardian refusal is an absolute contraindication. Coagulopathy is a contraindication and anesthesiologists should continue to refresh the ASRA guidelines for anticoagulants for neuraxial blockade along with evaluating the patient for symptomatic bleeding from a disorder. Increased intracranial pressure from an intracranial mass is also a contraindication [75]. Hemodynamic status should also be considered as the sympathectomy resulting from the epidural can cause hypotension in hypovolemic or hemodynamically unstable patients [76].

An epidural may be less effective in providing analgesia for foot/ankle procedures [77]. The popliteal sciatic and saphenous/adductor canal nerve blocks, in contrast, have a high success rate for foot/ankle procedures. The majority of lower extremity surgeries also involve just one lower extremity, making a peripheral nerve block a good choice.

Peripheral nerve blocks are not without their own risks. Infection, bleeding, nerve damage, and intravascular injection are all important risk factors. ASRA guidelines, broadly speaking, will also treat contraindications for coagulopathies for nerve blocks similarly to neuraxial blockade [78].

Local anesthetic dosing, in pediatrics is dosed as mg/kg and varies in accordance with the changing physiology of different age groups. The majority of local anesthetics used in pediatrics will be amides. Amide local anesthetics are metabolized by cytochrome p450 in the liver. The neonatal liver has limited metabolizing capability. Metabolism in the liver does not reach adult rates until 3–6 months of age [79–83]. This is to say that toxic levels of local anesthetic in patients up to 6 months of age may be lower than the adult levels. Furthermore, in regard to infusions, neonates also have less efficient clearance of amide local anesthetics compared to adults. This means that infusion rates should also be decreased and that translating adult mg/kg doses is not the only consideration when using local anesthetics in neonates and infants [84–86]. Amide anesthetics like bupivacaine and ropivacaine are highly bound to alpha-1 glycoprotein. The amount of nonprotein-bound bupivacaine, especially the d-isoform of bupivacaine, can determine toxicity. Infants have lower levels of alpha-1 glycoprotein to bind amides, and this provides another mechanism of potential toxicity in this age group [87, 88]. Concentrations of 0.25% bupivacaine and 0.2% ropivacaine are often used in the infant population for this reason. Ester local anesthetics are metabolized by plasma cholinesterase. Plasma cholinesterase levels are not as high in the infant compared to the adult, making infants more sensitive to the effects and potential toxicity of esters as well [89]. Maximum doses of ropivacaine and bupivacaine are 3 and 2.5 mg/kg, respectively. It is important to remember, however, that these doses are guidelines and that conservative dosing should be employed, especially in children under 6 months of age where doses should be reduced by about 30% [90–95].

Acetaminophen has been shown to be a good adjunct in a multimodal pain regimen. Please see the non-opioid section of this chapter for a more detailed look at acetaminophen. Within the context of this case, acetaminophen's blockade of prostaglandin production helps to diminish noxious stimuli. Perioperative use of acetaminophen can help patients decrease opioid use and side effects. NSAIDs work in a similar fashion (also covered in non-opioid section of this chapter) and are a major component of multimodal pain management that helps decrease opioid requirement, especially in the musculoskeletal surgery setting.

Baclofen is a muscle relaxant that is very useful in ameliorating muscle spasms perioperatively. Opioids do not directly address muscle spasms and muscle relaxants, and often GABA agonists like baclofen can provide centrally mediated muscle relaxation. Sedation and respiratory depression can occur as side effects, especially in patient with decreased renal clearance [96]. When used to treat muscle spasms with

proper monitoring of each patient's level of consciousness, muscle relaxants are a valuable component to multimodal pain management.

Case #2: Upper Extremity Injury

A *15-year-old, 6′2″ 80 kg male with no significant past medical history now presents for left open triangular fibrocartilage repair, which he sustained while snowboarding 6 months ago. Since the injury, the patient has complained of constant instability of the left wrist which has limited his ability to participate in sports or lift weights. The patient underwent an MR arthrogram that demonstrated the presence a peripheral triangular fibrocartilage complex tear. The pediatric regional/acute pain service was consulted to manage postoperative pain control by placement during anesthesia of a left single-shot brachial plexus nerve block of the left extremity* via *the axillary approach as well as an intercostal brachial nerve block both under ultrasound (US) guidance with nerve stimulation. The patient is currently taking over-the-counter acetaminophen with ibuprofen which he stopped taking approximately 1 week ago. At the time of examination, the patient denies numbness, tingling, or weakness in his left hand. His vitals are BP 143/67 HR 73 RR 21 and temperature 36.0 °C.*

Peripheral nerve blocks are often performed while children are under general anesthesia and therefore cannot express their discomfort with any paresthesias or cooperate in evaluation of intraneural injection. It is thought, however, that the risk of an uncooperative patient moving in the midst of the procedure is greater than the patient's lack of feedback and communication under general anesthesia. That is why conducting blocks under general anesthesia is more accepted in the pediatric population versus the adult population. Using ultrasound over landmark techniques may also help with decreasing dosage of local anesthetic given, more accurate placement of local anesthetics, increased block success rate, and better side effect profile. Ultrasound may also be used in combination with a nerve stimulator technique for improved success rate [97]. We recommend using an ultrasound technique whenever possible.

Axillary nerve blocks are the most commonly used upper extremity blocks in younger children as it has a better side effect profile when compared to other brachial plexus blocks such as interscalene nerve block. This peripheral nerve block is placed at the level of the terminal branches of the brachial plexus and is often used for surgeries involving the forearm and hands. Complications from axillary nerve blocks primarily include intravascular injection, hematoma formation with compression of surrounding structures, infection at the puncture site, and nerve damage [98, 99].

Infraclavicular nerve blocks are performed at the level of the cords of the brachial plexus, and the associated risks include pneumothorax, infection, bleeding, nerve damage, and intravascular injection. The placement of infraclavicular nerve block catheters is an excellent alternative for continuous nerve blocks in the pediatric patients when compared to axillary nerve blocks because the catheters have a lower rate of accidental dislodgment due to its location [100].

The supraclavicular nerve blocks are placed at the level of the divisions of the brachial plexus and can be used for shoulder procedures as well as procedures for the rest of the arm and can result in increased risk of bleeding, infection, nerve damage, intravascular injection, phrenic nerve involvement, Horner's syndrome, and pneumothorax [101, 102].

Interscalene nerve blocks consist of placement of local anesthetics at the root/trunk level of the brachial plexus. It provides excellent coverage for surgeries involving the shoulder. That being said, its side effect profile basically precludes its usage in younger children. The phrenic nerve is commonly blocked with the interscalene approach. The phrenic nerve innervates the ipsilateral diaphragm. In neonates, infants, and toddlers, blocking the phrenic nerve can cause significant respiratory depression as these children are more diaphragm dependent in their respiratory function. The risk of laryngeal nerve blockade from the interscalene approach will also cause ipsilateral vocal cord paralysis,

increasing airway resistance in younger children. Furthermore, infants have a more cranial location of their lung apices, predisposing them to increased risk of pneumothorax as well [103, 104]. Finally, case reports show that interscalene nerve blocks in the anesthetized patient can result in spinal anesthesia [105]. As a result of the number and severity of these side effects, interscalene nerve blocks are not routinely conducted in neonates, infants, and toddlers among other young children. Conceivably, interscalene nerve blocks can be done with similar risks to the adult population later in teenage years as anatomy and physiology start to resemble adult anatomy and physiology.

Conservative management through pain medication has its limitations. Opiates cause respiratory depression, nausea, constipation, drowsiness, and other side effects. Non-opioid medications have a maximum analgesic effect that may not completely cover moderate-to-severe postoperative pain (see non-opioid section of this chapter). A nerve block in addition to these measures completes a multimodal pain regimen that will help with perioperative pain management. Single-shot and continuous nerve blocks can be used in combination with conservative management to offer a more well-rounded pain regimen that minimized risks and maximizes efficacy [106].

Case #3: Abdominal Surgery

A 10-year-old male, otherwise healthy, with 3-month history of abdominal pain associated with nausea but no episode of vomiting. The patient has undergone placement of ear tubes in the past and received only rectal acetaminophen for pain control. His mother recalled that he developed significant postoperative nausea and vomiting (PONV) which was treated with ondansetron in the PACU. He was seen in the pediatric surgical office where he had enlarged outpouching of his umbilicus, which was reducible on examination. Ultrasound of the abdomen was performed at that time which confirmed umbilical hernia defect with small segment of the bowel present in the hernia. The hernia was easily reduced into the abdomen with no evidence of incarceration or strangulation. He subsequently was scheduled for umbilical hernia repair as an outpatient. The patient's mother also had an umbilical hernia and underwent repair as a child and recalls suffering from significant pain especially at her "belly button." She recalls significant PONV after receiving acetaminophen/codeine elixir in the recovery room and had to be admitted overnight for management of her pain and intractable nausea and vomiting. She is concerned that her son will have a similar experience and asked if there are other strategies or techniques available to help reduce his pain and to minimize opioid requirements after surgery.

The number of children undergoing surgeries has increased dramatically in the past few decades, due largely in part to the advancements in surgical techniques and improvement in outcomes. With the improvements in minimally invasive surgical techniques, a vast majority of abdominal procedures which once required hospitalization are now being done as outpatient surgery. A recent report from the American Society of Anesthesiologists (ASA) noted an almost 50% increase in the number of surgeries and anesthetics performed in children are done in ambulatory centers throughout our country. A similar trend can be seen worldwide in developed countries, where access to healthcare is readily available. The number of children that are admitted after elective surgeries is over 400,000 annually in the United States alone [107]. Poorly controlled pain and PONV)are among the most common reasons for hospitalizations after outpatient surgery in both children and adults. In addition to orthopedic surgeries and noncomplicated ENT procedures, general surgical and urological procedures such as the repair of abdominal wall defects and hernia repairs are now routinely performed in the outpatient setting in healthy children. The management of acute postoperative pain especially in the outpatient setting is critical, as uncontrolled or poorly controlled pain accounts for the vast majority of hospitalization and readmissions within the first 24 h after discharge. While opioids remain an important part in the manage-

ment of acute postoperative pain, adverse side effects such as PONV and respiratory depression can limit its effective use in children. Alternative strategies for pain management such as regional anesthetic techniques have been effectively used in both adults and children to reduce the amount of opioids needed in the perioperative period.

Umbilical and epigastric hernia repairs are common anterior abdominal wall procedures that are performed as an outpatient procedure in children, though after laparoscopic appendectomies and cholecystectomies, patients are still routinely hospitalized. Inguinal hernia repairs and circumcision are common outpatient urological procedures. Umbilical hernias are common in infants and children where the abdominal muscles fail to come together forming an umbilical ring, through which intestines protrude through an opening at the base of the umbilicus. These hernias often appear as outpouching of the belly button and are often reducible. These hernias can become incarcerated, resulting in reduced blood supply to involved sections of intestines trapped in this hernia defect. Symptoms such as abdominal pain and nausea with vomiting are common side effects associated with an incarcerated hernia, often prompting surgical repair. Strangulated hernia, with a complete cutoff of blood supply to intestines in the hernia defect, is a surgical emergency and requires immediate repair and hospitalization for further postoperative management.

With the advent of ultrasonography, peripheral nerve blocks in children have gained significant popularity and are being used increasingly for management of postoperative pain. A multimodal approach utilizing regional anesthesia or surgical infiltration of the operative site in addition to non-opioid adjuncts such as ketorolac and acetaminophen have been shown to reduce need for opioids and associated side effects. Ultrasound-guided bilateral rectus sheath blocks have been shown to provide adequate sensory coverage for all procedures involving the umbilicus such as umbilical hernia repair or in cases where the umbilicus has been accessed for trocar placement for laparoscopic surgeries. Studies have shown that rectus sheath blocks, when performed before surgical incision, have reduced both intraoperative and postoperative opioid requirements. The use of the non-opioid adjunct ketorolac 0.5 mg/kg up to 30 mg IV in conjunction with the rectus sheath blocks has been shown to provide analgesia superior to rectus sheath blocks alone. This combination has been used to effectively eliminate the need for postoperative opioids in the PACU. Surgical infiltration can also be used to reduce postoperative pain. However, Dingeman et al. showed superior pain control with ultrasound-guided rectus sheath blocks in comparison to surgical infiltration, with a lower median FACES scores and less opioid requirements in the rectus sheath block group [108].

Unlike umbilical hernias, epigastric hernias are small facial defects in the linea alba and can occur anywhere from the xiphoid to umbilicus. This difference in location is extremely important as it will impact the selection of the most appropriate peripheral nerve block. Transversus abdominis plane (TAP) block is most appropriate for postsurgical pain after epigastric hernia repair as it offers the appropriate sensory coverage. Innervation for the anterolateral wall is provided by the anterior rami of T6–L1 and intercostal nerves (T7–11), the subcostal nerve (T12), and the iliohypogastric and ilioinguinal nerves (L1), which travel between the transversus abdominis and internal oblique muscles and are best blocked at this level by the TAP block, whereas innervation for the umbilical area is provided by the right and left thoracoabdominal intercostal nerves, derived from the anterior rami of spinal roots T8-T12, traveling between the posterior rectus sheath and rectus abdominis muscle and best blocked by local anesthetic deposited along the posterior rectus sheath.

Inguinal hernias are common among infant and children and can be repaired by either pediatric general surgeons or pediatric urologists. These hernias form when the processus vaginalis fails to be obliterated, allowing the bowel or omentum to protrude. A hydrocele can develop as fluid from the abdominal cavity can freely enter into the scrotal sac. A higher incidence of

incarcerated or strangulated hernias has been seen in premature infants, particularly those who were mechanically ventilated. Regional anesthesia techniques such as neuraxial blocks and peripheral nerve blocks have been used to reduce the amount of opioids needed for pain control. The use of opioids, as previously discussed, is problematic when used in premature infants. Neuraxial techniques such spinal blocks have been successfully used as the sole anesthetic in premature infants undergoing inguinal hernia repair. The infant is awake for placement of the spinal block, after which soothing measures including sweeties are used to pacify infant during the repair. Unfortunately, the patient when sweeties are used is no longer NPO, which can be an issue if general anesthesia is needed, when the spinal blockade wears off [109]. Hyperbaric tetracaine 1 mg/kg has been shown to provide the longest duration for spinal block ranging from 45 to 60 min. Longer procedures will need a general anesthetic.

Single-shot caudal blocks are considered the gold standard for providing analgesia for children undergoing inguinal hernia repair, circumcision, and other uncomplicated urological procedures [110]. Bupivacaine is the most common local anesthetic agent used for caudal blocks, with dosing 1 mL/kg of 0.25% bupivacaine with epinephrine 1: 200,000 typically administered. 0.125% bupivacaine with epinephrine 1 mL/kg has been used as well and has been shown to provide equal analgesia with less motor blockade, which would be advantageous when caudals are performed in older children. Alternatively, ropivacaine 0.2% dosed at 1 mL/kg has been shown to provide comparable analgesia in children with the added benefit of being more cardioprotective than bupivacaine [111]. Adjuncts such as clonidine 1 mcg/kg can be added to prolong the duration of the caudal block [112]. Peripheral nerve blocks such as TAP and ilioinguinal/iliohypogastric (IL/IH) nerve blocks have been shown to provide adequate postoperative analgesia for these procedures [113, 114]. Penile block can also be used to provide analgesia for circumcision or meatoplasties and are usually performed by the pediatric urologist.

Case #4: Thoracic Surgery

A 16-year-old male recently developed pleuritic chest pain. Past medical history was significant for attention deficit hyperactivity disorder that was currently being managed on Adderall and Strattera. Medical evaluation showed the presence of an anterior mediastinal mass. Preoperative transthoracic echo was normal. He presented for biopsy with possible excision of the mass. Intraoperatively, biopsy confirmed the presence of a teratoma. The patient was subsequently positioned in the semi-recumbent right lateral decubitus position and underwent left thoracotomy with excision of a large teratoma. After surgical closure, a chest tube was placed.

After the procedure, placement of a thoracic epidural catheter would have been impossible due to positioning issues (proximity of the bean bag to the center of the spine). Paravertebral nerve blocks were performed in the operating room prior to extubation, while the patient, secured by bean bag, remained in the semi-recumbent right lateral decubitus position. Left T8 and T10 paravertebral catheters were placed with an 18 gauge Tuohy needle using the landmark technique. Through each catheter 10 mL of 0.5% ropivacaine was bolused.

Pain was well controlled with a 0.2% ropivacaine solution infusing at 10 mL/h through each paravertebral catheter in addition to IV acetaminophen and PCA. On POD#3, the patient was transitioned to oral pain medications. The paravertebral nerve blocks were removed on POD#4. Subsequent hospital course was uneventful.

Thoracic surgery when performed in children is associated with high levels of postoperative pain. If the pain is not properly managed, hospitalization can be due to complications such as atelectasis, pneumonia, and pulmonary embolism that are associated with decreased ambulation and the inability to participate in physical therapy or perform incentive spirometry. An aggressive approach to postoperative pain management continues to be the best and most successful strategy not only to control pain following surgery but also to prevent it. Thoracic surgery

procedures, particularly open procedures, where there is removal of a portion of the ribs or the chest wall have a high incidence of sympathetic mediated pain that can result in post-thoracotomy pain syndrome and neuropathic pain, which can be permanent if not managed early in the perioperative period. Due to the broad nature of thoracic surgery, this subsection will focus primarily on some of the more common thoracic procedures that are being performed in children and will present an overview of different strategies used for postoperative pain control.

Thoracic procedures are now being routinely performed in children to treat lung mass excision, perform pleurodesis for recurrent pneumothoraces due to apical blebs, and resect anterior mediastinal masses. These procedures can be done using either minimal invasive techniques, particularly thoracoscopy, or as an open procedure. The thoracoscopic approach or video-assisted thoracoscopic surgery is now being used more frequently because it has been shown to have fewer postoperative complications and has been associated with shorter hospital stays and faster recovery times. One of the main postoperative concerns these patients face following the procedure is pain, which is more severe following an open versus minimally invasive thoracoscopy. A detailed multimodal approach that is implemented early in the perioperative course is the most effective strategy for ensuring the patient has the best chance of the fastest recovery.

Thoracic epidurals have been shown to be the best modality for pain control following an open thoracotomy and continue to be the gold standard for postoperative pain control. Bupivacaine is the local anesthetic most commonly used for epidural infusions because it is the most effective local anesthetic; its prolonged duration of analgesia as well as its ability to provide varying levels of sensory blockade without profound motor blockade allows for earlier time to ambulation and faster discharge [115]. In addition to the use of a long-acting local anesthetic such as bupivacaine or ropivacaine, an opioid such as fentanyl or hydromorphone is often added to the epidural mixture to decrease the total dose of local anesthetic used per hour. Higher doses of epidural bupivacaine can result in motor blockade that can delay time to ambulation and increase the length of hospitalization. When epidural opioids are used, overall postoperative opioid consumption is reduced.

Local anesthetic-only epidural infusions can also be used to control postoperative pain despite concerns for potential motor blockade at higher doses. Indeed, some patients cannot tolerate opioid side effects even when they are administered through the epidural catheter. Paravertebral nerve block infusions through catheters have been shown to be as effective as epidural block in managing postoperative pain particularly since other side effects such as ileus, bladder incontinence, motor blockade, and pruritus that are associated with epidural blockade can be avoided [116, 117]. Other interventional procedures such as intercostal nerve blocks, local infiltration by the surgeon, and the placement of extrapleural catheters, commonly known as pain busters, are being more frequently used by thoracic surgeons particularly in adult patients [118]. However, these catheters have not been shown to be as efficacious when compared to paravertebral nerve block catheters [119]. These techniques can also be used in children though there continue to be concerns about local anesthetic toxicity with intercostal nerve block and extrapleural catheters due to increased vascular uptake of local anesthetics by the intercostal bundles below the ribs. Therefore, the use of these catheters are not routinely placed in children but can be considered an alternate option in teenagers or older children, where placement of either an epidural catheter or paravertebral nerve block is not desirable or feasible. The use of other analgesic adjuncts particularly anti-neuroleptics and antipyretics such as acetaminophen has been shown to provide effective pain control when compared to the use of epidural infusions alone [120].

Chronic Pain

Overview of Chronic Pain in Children

Chronic pain is defined as pain that persists beyond the initial injury and subsequent healing process. Traditionally the pain is greater than 6 months or

presents as a recurrence or an acute exacerbation of an underlying medical disease or pain syndrome. Examples include sickle cell crisis or a flare-up of inflammatory bowel disease. Exacerbation of chronic disease is often referred to as acute on chronic pain. It can present unique challenges to the providers, as there are behavioral components consistent with acute pain that could appear as drug-seeking behavior. Due to the broad and complex nature of chronic pain and its management, this section will focus on a subset of chronic pain and associated syndromes that have presented unique challenges to pain management in children. Neuropathic pain including phantom limb pain, CRPS Type 1 and 2, post-thoracotomy syndrome, sickle cell crisis, and cancer pain are becoming increasingly more common in children. Early intervention improves outcomes such as quality of life and functionality.

Neuropathic Pain

Neuropathic pain is defined as pain due to an intrinsic dysfunction of the central and/or peripheral nervous system or secondary to the presence of a lesion or injury to these areas. This pain often presents with a prodrome of symptoms including burning or electric-like sensations in specific areas of the body such as the hands or feet. The pain is often intermittent. There can also be sensory dysfunction including allodynia, hyperalgesia, hyperpathia, paresthesia, and dysesthesia.

Terminology of Dysfunctional Pain Responses

Allodynia: Severe pain triggered by benign stimuli such as light touch

Dysesthesia: An unpleasant sensation that can be spontaneous or evoked such electric-like sensations in the hands or feet

Hyperalgesia: Increased sensitivity to pain

Hyperpathia: Augmented response to normally painful stimuli

Paresthesia: An abnormal sensation that can be provoked or occur spontaneously

The management of neuropathic pain is often problematic in children. Children often lack the cognitive and verbal development to describe this sort of pain. For that reason, there often is underrecognition, inadequate, or no treatment for some patients.

Case #5: Complex Regional Pain Syndrome

A 15-year-old girl presented to the pediatrician complaining for the last 2 weeks of burning sensation in her left hand. Her mother stated that she slipped on ice while trying to catch the school bus. Later the school nurse stated that she did not sustain any bone fractures or major damage to the ligaments and muscles. The patient describes the sensation in her left hand as having a "fire hand" that is worse at night. Ice packs significantly reduce the pain. She is currently taking over-the-counter acetaminophen and ibuprofen as needed for pain. On examination, the patient's left hand appears erythematous, swollen, and painful to light touch.

Complex Regional Pain Syndrome (CRPS)

CRPS is associated with pain, hyperalgesia, allodynia, sudomotor, and/or vasomotor instability. Two types of CRPS have been classified: type 1 formally known as reflex sympathetic dystrophy (no direct history of illness or injury) and type 2 (based on direct nerve injury). CRPS is typically a diagnosis of exclusion and is more commonly reported in adults. The syndrome has three distinct phases, acute, dystrophic, and atrophic, which occur over time. The acute phase is characterized by pain with swelling in the affected extremity. The limb initially appears red, warm, and dry but eventually becomes cyanotic and cold. This phase typically occurs within the first 1–3 months after initial presentation and/or any inciting injury. During this period the patient has the best chance of making a full recovery if appropriate interventions are taken. The dystrophic or

second phase typically occurs within the next 3–6 months and is characterized by loss of hair with increased swelling and stiffness in the affected extremity. If these changes are not corrected, the patient will then progress to the third and final stage, the atrophic phase during which the effects of CRPS are irreversible. This phase typically occurs after 6 months and is characterized by severe atrophy with muscle wasting, contractures, and severe pain with functional limitations noted in the affected extremity [121]. The presentation and progression of these symptoms usually varies from patient to patient, and early intervention using occupational and physical therapy along with psychotherapy and medications such as anti-neuroleptics have been shown to be extremely effective in treating and even reversing the symptoms.

In children, this syndrome is quite common and is typically affected by such factors as gender, age, and certain locations within the body. There is currently a higher incidence noted in females compared to males, frequently involving the lower extremity, and the syndrome is more commonly seen in children greater than 6 years old [122]. Unlike adults, CRPS can have a variety of different presentations in children, which can make identification and ultimately treatment of the syndrome challenging. These patients often present with vague and nonspecific symptoms and have difficulty recalling the cause of the initial injury. Often, the only indication that there is something wrong can be seen in the child's performance, usually in school as opposed to home. These children can have difficulty participating in extracurricular activities due to difficulty walking or report having generalized weakness and malaise [123].

Early identification and treatment of this disease is critical in preventing progression and recurrence, which can be as high as 50%. Treatment must include aggressive occupational and physical therapy, cognitive behavioral psychotherapy, and sympathetic blockade involving the brachial or lumbosacral plexus, depending on the location of the lesion. In most cases, this treatment plan occurs on an outpatient basis and centers around occupational and physical therapy, involving active mobilization and strength training of the affected extremity. Although physical therapy is the first-line treatment for CPRS, studies have shown a recurrence rate of 20% with physical therapy alone [124]. This high incidence of recurrence is often attributed to the decreased ability for children to participate in physical therapy due to severe pain in the affected extremity. This is where the use of sympathetic blockade in the form of peripheral or central blockade has the greatest utility, as these techniques allow for improved pain control, which facilitates patient participation and improved outcomes. Sympathetic blockade is performed on an outpatient basis and should be performed daily followed by occupational and physical therapy, the same as what's been found to be successful in adults [125]. Younger children are unable to tolerate placement of the block with no or minimal sedation and often require hospitalization and general anesthesia for block placement and management. Currently use of peripheral nerve blocks or central blocks such as neuraxial catheters is preferred to repeated single-shot injections. In the case of lower extremities, the use of lumbar epidurals or peripheral nerve block catheters in addition to other analgesic adjuncts has been shown to be highly effective.

The decision to use either peripheral nerve blocks or neuraxial blockade depends primarily on the extent and location of the pain and autonomic dysfunction. If the lesion has a limited distribution, then the placement of peripheral nerve block catheter(s) would be preferred, as peripheral nerve blocks are not associated with side effects such as nausea, vomiting, urinary retention, ileus, or profound hypotension. Popliteal sciatic nerve block catheters are routinely placed for lesions below the knee [126]. The addition of either a femoral or adductor canal nerve block catheter should be considered if there is involvement in the distribution of the femoral nerve, which provides sensory innervation to the medial aspect of the foot. Saphenous nerve blocks in general have a failure rate of up to 10%, which makes utilization of this block less desirable [127]. If the injury is more proximal, then the placement of proximal sciatic with or without

lumbar plexus or femoral nerve block catheters would provide more complete sensory blockade that's required for treatment.

In the case of CRPS involving the upper extremity, supraclavicular or infraclavicular nerve block catheters are routinely used for treatment. Sympathetic blockade such as stellate ganglion blocks for upper extremity CRPS and lumbar sympathetic blocks for lower extremity CRPS are currently not used in children as a first-line treatment but rather as a last resort in patients who have failed the previously discussed therapies [128].

Dadure et al. showed that Bier blocks in conjunction to continuous PNB catheter can be used successfully to treat recurrent CRPS type 1 in children [129]. The study included 13 children between the age of 6–16 years old with recurrent CRPS type 1 that failed conventional treatment, which consisted of physical therapy and psychotherapy [129]. General anesthesia was used for both the Bier block and the placement of the PNB catheters. The patients were subsequently discharged home with continuous PNB catheters using disposable pumps. The pumps were monitored by their parents or guardians over a 96-h period. None of the patients had any symptoms of CRPS during the 2-month follow-up, and both the patients and their parents were extremely satisfied with the postoperative pain control. The children were able to participate in early mobilization and discharge home and had decreased anxiety, which decreased the psychological component of sympathetic mediated pain.

In addition to the use of regional anesthesia, other pharmacologic agents have been used with varying degrees of success for refractory cases. Anti-neuroleptics such as gabapentin or pregabalin despite not having been approved by the FDA for the treatment of chronic neuropathic pain are currently being used clinically with excellent results. Most of the studies involving the use of gabapentin have shown efficacy in managing painful diabetic neuropathy and postherpetic neuralgia in adults [130]. Pedemonte et al. did an analysis of seven children 7–15 years old with CRPS confirmed by imaging, such as X-ray which showed bone loss and MRI positive for soft tissue and muscle atrophy [131]. The patients were successfully treated with the physiotherapy, psychotherapy, and gabapentin or pregabalin. The study consisted of six girls and one boy with five of the cases involving the lower extremity and three due to previous trauma [131]. All of the patients had good clinical outcomes with no evidence of recurrence during follow-up from 4 to 30 months.

Gabapentin and pregabalin also have the added benefit of addressing the psychological component of CRPS, such as depression, by improving the patient's overall mood, which can dramatically improve the patients' symptomology.

Psychotherapy is essential since a significant component of pain is psychological. If this aspect of sympathetic-mediated pain is not properly addressed early in the treatment program particularly in children, the entire treatment plan can fail. Saltik et al. reported a case in which a 15-year-old girl presented with CRPS Type 1 of the left arm with recurrence of her symptoms following initial treatment [132]. One year later, her CRPS Type 1 was compounded by depression, which was refractory to treatment with selective serotonin reuptake inhibitors including sertraline and mirtazapine as well as gabapentin. Her symptoms were initially managed with the use of stellate ganglion blocks, antidepressants, and pregabalin, which allowed her to tolerate physical therapy. She subsequently had several episodes of recurrence despite taking the pregabalin and antidepressants as prescribed. The patient was then reevaluated by a psychiatrist due to suicidal ideations and was noted to have PTSD because she was physically abused by her father. The patient made a full recovery once she was removed from the home, and her psychiatric trauma was treated [132].

This case highlights the importance of having an initial psychological evaluation when formulating a treatment plan for these patients. There are a variety of different psychological techniques that address the psychological dysfunction such as anxiety and depression which is often seen in these patients. These techniques include hypnosis, relaxation therapy, biofeedback, and visual guided imagery. Cognitive behavior therapy

remains the first-line treatment and must include the patient's immediate family and caregivers [128]. The participation of the family unit in cognitive behavioral therapy will drastically decrease the patient's anxiety and improve outcomes. Children often look to their caregivers for direction and support. The caregivers will also ensure that the patient continues participation with the treatment regimen at home.

Case #6: Post-Thoracotomy Syndrome

A13-year-old male with a PMH significant for bipolar disorder currently on risperidone and osteosarcoma of the left leg. The patient presented with osteosarcoma and previously underwent left femur resection and reconstruction. The patient then received 4 months of chemotherapy. A metastasis to the right upper lung was then noted after which he underwent right thoracotomy with wedge resection. The tumor returned and the patient now presents for a second thoracotomy and wedge resection of the right lung. A thoracic epidural was placed for both of his prior surgeries, but because of block failure, his postoperative pain was poorly controlled. The option for placement of another epidural for this procedure was discussed with the patient's mother who was present at the bedside; she requested that "some other procedure" to manage pain be considered. He currently has chronic pain at the site of his previous thoracotomy incision which he describes as a burning pain that comes and goes and is worse at night. The patient is currently on gabapentin 300 mg TID, oxycodone 20 mg PO Q 12 h, and oxycodone sliding scale 5 or 10 mg every 3 h as needed. The patient took his morning dose of gabapentin before arriving at the hospital.

Post-Thoracotomy Syndrome

Post-thoracotomy syndrome (PTPS) is a chronic pain syndrome that occurs usually following thoracic surgery, particularly thoracotomies. Its incidence ranges from 20% to 67% [133]. The International Association for the Study of Pain defines PTPS as persistent or recurrent pain for at least 2 months following a thoracotomy. The pain is usually located at the site of the initial surgical incision [133]. The primary etiology of PTPS is unclear but is based on several surgical and nonsurgical factors. The type and size of the incision (muscle sparing vs muscle splitting), extent of surgical manipulation, and length of surgery as well as the approach used to enter the thoracic cavity are surgical factors that have been shown to contribute to the development of PTPS. In addition, factors such as age, psychosocial status, predisposition for neural injury, or the existence of pre-existing chronic pain or nerve damage have been shown to contribute to the incidence of PTPS [133]. Minimally invasive techniques such as video-assisted thoracic surgery (VATS) have been suggested to decrease the incidence of neuropathic pain and PTPS; however, the current literature is conflicting. Maguire et al. conducted a questionnaire to determine the prevalence of neuropathic pain following thoracic surgery and showed a similar incidence of PTPS irrespective of the surgical technique [134]. Shanthanna et al. showed that VATS was associated with a 35% incidence of PTPS compared to 54% for open cases [133]. There was also an increase incidence of PTPS among cancer patients and those with pre-existing pain. PTPS has both a neuropathic and myofascial component and is due in part to the direct damage of the intercostal nerves during surgery. This neural damage can lead to neuroma formation and lead to aberrant pain pathways during the regenerative process. This process also results in central CNS dysfunction, which accounts for the psychological component of pain that can be difficult to treat [135].

In addition to minimizing the surgical causes of PTPS, a variety of pharmacological and nonpharmacological interventions can be used to address both the peripheral and central CNS dysfunction central to this disease. The use of NSAIDs such as COX-2 inhibitors has been shown to decrease inflammation surrounding the

affected nerves caused by inflammatory mediators such as prostaglandins and leukotrienes, significantly reducing pain. Gabapentin and pregabalin have been successfully used to manage acute as well as chronic pain following thoracic surgery and are most effective when used preemptively prior to surgical incision [135]. Other non-opioid adjuncts such as ketamine infusions have been effectively used to modulate neuropathic pain in patients with recurrent PTPD. A ketamine infusion dose of 0.1–0.7 mg/kg/h has been shown to reduce PTPS [135].

The use of regional anesthesia such as thoracic epidurals and paravertebral and intercostal nerve blocks has been shown to decrease the PTPS incidence as a group. However, the placement of thoracic epidurals pre-incision has been shown to have mixed results in preventing PTPS. In fact, several studies have shown that epidural blockade did not decrease the incidence of PTPS [136]. Senturk et al. compared the placement of pre-incisional, post-incisional compared to only PCA use for patients undergoing thoracotomy. They showed that a pre-incisional epidural was associated with considerably lower postoperative pain and a decreased incidence of PTPS [137]. Paravertebral nerve blocks provide equal analgesia when compared with epidurals. Raveglia et al. directly compared thoracic epidurals to paravertebral nerve blocks for treating pain following thoracotomy [138]. The study was a randomized double-blind prospective study in which 52 patients were randomized into two groups receiving either a thoracic epidural or paravertebral catheter, and visual analog scales (VAS) were used to quantify postoperative pain. Both groups had similar VAS pain scores, but the paravertebral group had less side effects such as PONV, hypotension, and urinary retention associated with the epidural group [139].

Case #7: Sickle Cell Crisis

A14-year-old female 60 kg with PMH significant for sickle cell disease was admitted to the children's hospital in sickle cell crisis. She complained of significant pain including chest pain. The patient develops acute chest syndrome as well as pain in her lower extremities during each sickle cell crisis. Her pain has been managed with IV morphine PCA during her previous hospitalizations. She uses PO morphine at home, but she has developed a tolerance to this regimen. The pain service was consulted for recommendations on management of sickle cell crisis pain.

On admission, the patient is on a morphine PCA with a basal rate of 3 mg/h with demand dose of morphine 5 mg Q 15 min. She has been complaining of severe pain, and the nursing service reports that the patient has required breakthrough boluses of 2 mg morphine every hour. The pediatricians also ordered ketorolac 30 mg, and she received one dose 1 h before being evaluated by our service. Zofran 6 mg was also given as the patient reported nausea and had one episode of emesis. On evaluation, the patient is in significant distress, with HR 126 and BP 126/78 (82), receiving supplemental oxygen via *nasal cannula at 2 L/min with saturations ranging from 95% to 97% and decreasing to 91% on RA. Her respiratory rate is high 30s and has been as high as 48. She describes sharp pain in her right chest, right arm, and abdomen. The patient is febrile with high WBC and hematocrit 22. She is an athlete and was involved in rigorous training for an upcoming volleyball team competition. Her younger sister has been ill at home with a bout of gastroenteritis.*

Vaso-occlusive crisis is triggered by a complex pathophysiologic process in individuals afflicted with sickle cell disease (a hemoglobinopathy in patients with HbSS, HbSC, and select other variants) [140]. In the classic description, sickled erythrocytes clog microvasculature and cause ischemia. This results in end-organ damage, inflammation, vascular endothelial adhesion deficiencies, platelet dysfunction, and coagulation cascade abnormalities. They all combine to bring on vaso-occlusive crises like acute chest syndrome (ACS) [141–143]. Patients with ACS will classically present 2–3 days following a vaso-occlusive crisis, but may present sooner [144]. They can also exhibit acute respiratory

symptoms like tachypnea, cough, hypoxemia, and fever. Chest X-ray may also reveal infiltrates, commonly upper or middle lobe involvement, in the pediatric population (multilobular in the adult population). Infection is a major trigger for ACS for sickle cell patients, especially in patient with decreased splenic function [145].

In order to decrease the likelihood of ACS, there may be an increasing role for chronic antibiotic use at a younger age, preoperative transfusion, and hydroxyurea therapy. In the acute setting, the patient needs to be supported with appropriate temperature control to avoid hypothermia, hydration with intravenous fluid resuscitation, supplemental oxygen, and maintenance of physiologic pH, antibiotic therapy, respiratory support, and adequate pain control [146–148]. A multimodal approach using different pharmacologic agents targeting different receptors is the most effective strategy for managing pain in these patients. IV opioids, NSAIDs, and antineuropathic medications can be combined as part of a comprehensive plan that aggressively targets the different components of pain associated with vaso-occlusive crisis. Special attention should be paid to the patient's current renal function when determining the type of medications and correct doses to use, as acute renal insufficiency and/or failure is a potential sequela of sickle cell crisis due to vasoconstriction in the renal arteries.

NMDA antagonists such as ketamine have been used successfully in these patients especially in cases where high doses of opioids are required and there is respiratory depression occurring with poorly optimized pain control. Ketamine can be administered as a bolus dose of 0.5 mg/kg followed by infusion rate of 0.1–0.7 mg/kg/h. The bolus dose is often not routinely used in awake patients due to concerns of side effects associated with ketamine such as dissociative amnesia, and visual and auditory hallucination, which is dose dependent. Infusions usually start at a lower dose and are titrated upward for effect. Also benzodiazepines are added on a PRN basis for rescue or scheduled in cases where a higher dose of ketamine is needed to decrease opioid consumption [149].

In addition to pharmacological agents, regional anesthesia has been shown to decrease pain. In a patient with ACS who is hyperventilating and struggling to maintain adequate respiratory effort, a thoracic epidural is a good option. The neuraxial blockade can significantly decrease pain without increasing respiratory depression. Measures must be taken to ensure that the patient is adequately resuscitated prior to placement of an epidural, as epidurals can cause profound hypotension due to sympathectomy. Hypotension can worsen blood flow to vital organs such as the kidneys. In addition to maintaining euvolemia, slow administration of a less concentrated solution of local anesthetics with an opioid such as fentanyl has been shown to decrease the incidence of hypotension following epidural placement. Furthermore, a review of sickle cell data by experts in sickle cell treatment suggests that early treatment of acute crises may decrease conversion to chronic pain [145].

There is not an easy approach to patient suspected of drug-seeking behavior due to opioid addiction and/or dependence. While this tends to happen less in the pediatric population and is not common in the sickle cell population overall, it is still an issue that can take up major resources. If there is a concern that a patient is more concerned about specific doses rather than effect of therapy, illegal acquirement of narcotic medication, or other behavior that the clinician may ascertain to be alarming, then the hospital's addiction professionals should be notified.

Case #8: Phantom Limb Pain: Sarcoma Removal

A 16-year-old female with a PMH significant for asthma, seizures due to benzodiazepine withdrawal, and Ewing's sarcoma of the right tibia s/p, an above-the-knee amputation, and chemotherapy is now scheduled for right femoral biopsy. The patient is being evaluated for possible placement of a peripheral nerve block for postoperative pain control at the request of the primary service. The patient initially presented last year with 3–4-month duration pain in her right leg. She underwent an MRI which showed

the presence of a mass in the proximal right tibia. Due to concerns that the lesion might be Ewing's sarcoma, the patient underwent a proximal tibial resection with allograft and prosthetic hinged knee reconstruction arthroplasty a week later (9/2014). The patient was subsequently discharged home and started on chemotherapy. Unfortunately, the patient was readmitted 4 months later after presenting to the ED complaining of fever, chills, and significant pain in her right knee. She was noted to have a purulent discharge at the site of surgery. She subsequently underwent incision and drainage of the right the knee. Blood cultures were later positive for gram-negative Klebsiella infection of the prosthesis at which time she underwent an above-the-knee amputation with wound vacuum placement. Due to the patient's severe right knee pain, the pediatric regional and acute pain service was consulted and placed right gluteal sciatic and femoral nerve block catheters preoperatively for postoperative pain control. She is currently taking OxyContin 20 mg Q 12 h and oxycodone 15 mg every 3 h prior to her readmission.

Prior to PNB placement, the patient had severe anxiety and depression with allodynia and hyperalgesia. She complained of having a burning sensation in her right knee and a sensation that "her toes were curled under," which she found most upsetting because the lower portion of her right leg was removed along with the sarcoma. Following surgery, the patient was started on gabapentin, acetaminophen, and a hydromorphone PCA with hydromorphone 1 mg every 4 h PRN for severe breakthrough pain. Ropivacaine 0.2% at 10 mL/h was used for the peripheral nerve block infusion. She was later started on a ketamine infusion due to severe phantom limb pain and high opioid requirements. The patient was later discharged to a rehabilitation facility on gabapentin, OxyContin, and oxycodone sliding scale as needed. She presents now for a right femoral biopsy because she continues to complain of burning pain in her right upper thigh. A repeat MRI of the right femur shows the presence of a mass with concerns for recurrence.

In the pediatric population, trauma and disease such as cancer are two most common reasons for amputation. Traumatic amputations are the most common and can result in permanent physical and psychological disabilities. The incidence of traumatic amputations is higher among adolescent males, and more than 90% have single-limb involvement. Lower extremity amputation injuries account for 60% of these injuries. In terms of disease-related limb amputations, neoplasms are the common cause, with the highest incidence among patients between ages 12 and 21 years old [150]. Tumors such as osteogenic sarcoma, Ewing's sarcoma, and rhabdomyosarcoma are the most common neoplasms that have been associated with acquired amputations in the pediatric population. Due to advances in treatments such as chemotherapy and surgical techniques, the survival rate has improved to about 60–70%. In some cases, the affected limb can be salvaged, but success depends on factors such as aggressiveness and stage of tumor, responsiveness to neoadjuvant chemotherapy and chemotherapy, and the ability to obtain cancer-free margins [151]. Infections and other complications involving the salvaged limb can ultimately result in amputation.

Patients who have undergone limb amputation experience not only psychological and emotional trauma but also painful sensations coming from the part of the limb that is no longer there. This phenomenon is called phantom limb pain. It was described as early as 1871 by Mitchell [152]. The incidence of phantom limb pain in adults has been reported to be as high as 40–85%. However, in children, the incidence of phantom limb pain has been more difficult to determine due to a paucity of data and seems rare in children less than 10 years of age [153]. The mechanism for development of phantom limb is not clear, but several contributing factors have been identified. They include the degree of preoperative and intraoperative pain and psychological factors. Experimental studies indicate that increased activity involving parts of the sympathetic nervous system may be a potential cause. Sensitization of abnormal afferent output from damaged nerve fibers occurs by the sympathetic nervous system (SNS). Significant reorganization of parts of the central nervous system has been seen after amputation with the degree of reorganization corresponding to the magnitude of phantom limb pain [154].

Management of phantom limb is challenging. A multidisciplinary approach is considered the best approach for reduction of pain and improved functionality. Pharmacologic management includes neuropathic agents such as gabapentin, pregabalin, and tricyclic antidepressants such as nortriptyline. Non-pharmacological methods such as acupuncture and transcutaneous electrical nerve stimulation units have been used to downregulate pain. Regional anesthetic techniques when performed before amputation have been shown in some studies to reduce phantom limb pain, though studies show conflicting results [155].

Conclusion

In conclusion, the management of pain in children after surgery is now receiving the much needed attention that has allowed for advancements in the different modalities that are now available for use in this patient population. Providers are becoming more attentive and concerned with not just treatment of pain but also with its prevention. This new focus has increased the amount of regional and neuraxial procedures that are being performed for surgeries and other chronic pain syndromes. Even routine ambulatory procedures are now being managed with not just intravenous and oral opioids but also using this multimodal approach, using single-shot peripheral nerve blocks for better optimization of postoperative pain immediately following surgery as well as after discharge home.

Review Questions

1. On pain rounds, a 2-year-old patient POD 1 exhibits occasional grimace, restless legs, squirming back and forth, crying steadily, and difficult to console. What is his FLACC score?
   (a) 7
   (b) 3
   (c) 11
   (d) 5
2. Which of the following choices are reasons why codeine may not be a good choice when prescribing oral opioids in the pediatric population?
   (a) Codeine is not a very potent analgesic.
   (b) Its efficacy is variable due to its dependence on metabolism.
   (c) Nausea/vomiting and dysphoria can be severe.
   (d) All of the above.
3. A 13-year-old patient was given IV ketorolac immediately postoperatively after undergoing a spinal fusion for idiopathic scoliosis. This patient is most likely to have:
   (a) Hardware failure
   (b) Improved pain control
   (c) Need for reoperation
   (d) Pseudoarthrosis
4. A 10-year-old patient is undergoing a right Achilles tendon repair. His mother mentions that he is sensitive to opioids and has profuse nausea/vomiting when taking them. What other pain option would best control his pain in the immediate postoperative period?
   (a) Thoracic epidural
   (b) Femoral nerve block
   (c) Popliteal sciatic and saphenous nerve blocks
   (d) Ankle block
5. A 3-year-old patient is undergoing a right hand procedure. Along with a multimodal pain regimen, what peripheral nerve block can be used to help with pain control?
   (a) Axillary nerve block
   (b) Infraclavicular nerve block
   (c) Interscalene nerve block
   (d) A and B
   (e) All of the above
6. Which of the following pain control options are best for reducing postoperative opioids for umbilical hernia repair?
   (a) Acetaminophen
   (b) Skin infiltration of local anesthetic
   (c) Ketorolac and ultrasound-guided rectus sheath block
   (d) Ketamine infusion

7. Paravertebral catheters may be used in patients undergoing thoracic procedures because
   (a) They are more efficacious than extrapleural catheters.
   (b) They do not cause bladder incontinence.
   (c) They have less vascular uptake when compared to intercostal nerve blocks.
   (d) All of the above.
8. Which of the following factors is associated with greater risk of CRPS in children?
   (a) Male gender
   (b) Greater than 6 years of age
   (c) Upper extremity involvement
   (d) None of the above
9. Peripheral nerve blocks have a role in multimodal pain regimens for the following conditions except:
   (a) CRPS
   (b) Sickle cell crisis
   (c) Neuropathic pain
   (d) Post-thoracotomy syndrome
   (e) Fibromyalgia

Answers

1. a
2. d
3. b
4. c
5. d
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References

1. Verghese ST, Hannallah RS. Acute pain management in children. J Pain Res. 2010;3:105–23.
2. Taddio A, Katz J. The effects of early pain experience in neonates on pain responses in infancy and childhood. Paediatr Drugs. 2005;7(4):245–57.
3. Taddio A, Katz J, Ilersich AL, Koren G. Effect of neonatal circumcision on pain response during subsequent routine vaccination. Lancet. 1997;349(9052):599–603.
4. DiLorenzo M, Pillai Riddell R, Holsti L. Beyond acute pain: understanding chronic pain in infancy. Children (Basel). 2016;3(4):E26.
5. Breivik H. Opioids in cancer and chronic non-cancer pain therapy-indications and controversies. Acta Anaesthesiol Scand. 2001;45(9):1059–66.
6. Ansermino M, Basu R, Vandebeek C, Montgomery C. Nonopioid additives to local anaesthetics for caudal blockade in children: a systematic review. Paediatr Anaesth. 2003;13(7):561–73.
7. Classification of chronic pain. Descriptions of chronic pain syndromes and definitions of pain terms. Prepared by the International Association for the Study of Pain, Subcommittee on Taxonomy. Pain Suppl 1986;3:S1–226.
8. Crellin D, Sullivan TP, Babl FE, O'Sullivan R, Hutchinson A. Analysis of the validation of existing behavioral pain and distress scales for use in the procedural setting. Paediatr Anaesth. 2007;17(8):720–33.
9. Babl FE, Crellin D, Cheng J, Sullivan TP, O'Sullivan R, Hutchinson A. The use of the faces, legs, activity, cry and consolability scale to assess procedural pain and distress in young children. Pediatr Emerg Care. 2012;28(12):1281–96.
10. von Baeyer CL, Spagrud LJ. Systematic review of observational (behavioral) measures of pain for children and adolescents aged 3 to 18 years. Pain. 2007;127(1–2):140–50.
11. Gomez RJ, Barrowman N, Elia S, Manias E, Royle J, Harrison D. Establishing intra- and inter-rater agreement of the face, legs, activity, cry, consolability scale for evaluating pain in toddlers during immunization. Pain Res Manag. 2013;18(6):e124–8.
12. Huguet A, Stinson JN, McGrath PJ. Measurement of self-reported pain intensity in children and adolescents. J Psychosom Res. 2010;68(4):329–36.
13. Trujillo KA, Akil H. Inhibition of morphine tolerance and dependence by the NMDA receptor antagonist MK-801. Science. 1991;251:85–7.
14. Ripamonti C, Groff L, Brunelli C, Polastri D, Stavrakis A, De Conno F. Switching from morphine to oral methadone in treating cancer pain: what is the equianalgesic dose ratio? J Clin Oncol. 1998;16(10):3216–21.
15. Berde CB, Sethna NF. Analgesics for the treatment of pain in children. N Engl J Med. 2002;347:11094–103.
16. Morton NS, Errera A. APA national audit of pediatric opioid infusions. Paediatr Anaesth. 2010;20:119–25.
17. Kart T, Christrup LL, Rasmussen M. Recommended use of morphine in neonates, infants and children based on a literature review: Part 1—Pharmacokinetics. Paediatr Anaesthesiol. 1997;7:5–11.
18. Pasternak GW, Standifer KM. Mapping of opioid receptors using antisense oligodeoxynucleotides:

correlating their molecular biology and pharmacology. Trends Pharmacol Sci. 1995;16:344–50.
19. Nagasaka H, Awad H, Yaksh TL. Peripheral and spinal actions of opioids in the blockade of the autonomic response evoked by compression of the inflamed knee joint. Anesthesiology. 1996;85:808–16.
20. Sabbe MB, Yaksh TL. Pharmacology of spinal opioids. J Pain Symptom Manage. 1990;5:191–203.
21. Ciszkowski C, Madadi P, Philips MS, Lauwers AE, Koren G. Codeine, ultrarapid-metabolism genotype, and postoperative death. N Engl J Med. 2009;361:827–8.
22. Koren G, Cairns J, Chitayat D, Gaedigk A, Leeder SJ. Pharmacogenetics of morphine poisoning in a breastfed neonate of a codeine-prescribed mother. Lancet. 2006;368:704.
23. Schechter NL, Berde CB, Yaster M. Opioid agonists and antagonists. 2nd ed. Philadelphia: Lipincott Williams and Wilkins; 2003.
24. Berde CB, Beyer JE, Bournaki MC, Levin CR, Sethna NF. Comparison of morphine and methadone for prevention of postoperative pain in 3- to 7-year-old children. J Pediatr. 1991;119:136–41.
25. Gourlay GK, Willis RJ, Wilson PR. Postoperative pain control with methadone: influence of supplementary methadone doses and blood concentration-response relationships. Anesthesiology. 1984;61:19–26.
26. Gourlay GK, Willis RJ, Lamberty J. A doluble-blind comparison of the efficacy of methadone and morphine in postoperative pain control. Anesthesiology. 1986;64:322–7.
27. Scott CS, Riggs KW, Ling EW, Fitzgerald CE, et al. Morphine pharmacokinetics and pain assessment in premature newborns. J Pediatr. 1999;135:423–9.
28. Lynn AM, Slattery JT. Morphine pharmacokinetics in early infancy. Anesthesiology. 1987;66:136–9.
29. Purcell-Jones G, Dormon F, Sumner E. The use of opioids in neonates. A retrospective study of 933 cases. Anaesthesia. 1987;42:1316–20.
30. Esmail Z, Montgomery C, Courtrn C, Hamilton D, Kestle J. Efficacy and complications of morphine infusions in postoperative paediatric patients. Paediatr Anaesthesiol. 1999;9:321–7.
31. Katz R, Kelly HW. Pharmacokinetics of continuous infusions of fentanyl in critically ill children. Crit Care Med. 1993;21:995–1000.
32. Santeiro ML, Christie J, Stromquist C, Torres BA, Markowsky SJ. Pharmacokinetics of continuous infusion fentanyl in newborns. J Perinatol. 1997;17:135–9.
33. Vaughn PR, Townsend SF, Thilo EH, McKenzie S, Moreland S, Denver KK. Comparison of continuous infusion of fentanyl to bolus dosing in neonates after surgery. J Pediatr Surg. 1996;31:1616–23.
34. Collins JJ, Geake J, Grier HE, Houck CS, Thaler HT, Weinstein HJ, Twum-Danso NY, Berde CB. Patient-controlled analgesia for mucositis pain in children: a three-period crossover study comparing morphine and hydromorphone. J Pediatr. 1996;129:722–8.
35. Kussman BD, Sethna NF. Pethidine-associated seizure in a healthy adolescent receiving pethidine for postoperative pain control. Paediatr Anaesthesiol. 1998;8:349–52.
36. Kranke P, Eberhart LH, Roewer N, Tramèr MR. Pharmacological treatment of postoperative shivering: a quantitative systematic review of randomized controlled trials. Anesth Analg. 2002;94:453–60.
37. De Witte J, Sessler DI. Perioperative shivering: physiology and pharmacology. Anesthesiology. 2002;96:467–84.
38. Rodgers BM, Webb CJ, Stergios D, Newman BM. Patient controlled analgesia in pediatric surgery. J Pediatr Surg. 1988;23:259–62.
39. Gaukroger PB, Tomkins DP, van der Walt JH. Patient-controlled analgesia in children. Anaesth Intensive Care. 1989;17:264–8.
40. Viscusi ER, Schechter LN. Patient-controlled analgesia: finding a balance between cost and comfort. Am J Health Syst Pharm. 2006;63:3–13.
41. Grissinger M. Safety and patient-controlled analgesia: Part 2: How to prevent errors. P T. 2008;33(1):8–9.
42. Monitto CL, Greenberg RS, Kost-Byerly S, Wetzel R, Billett C, Lebet RM, Yaster MCL. The safety and efficacy of parent-/nurse-controlled analgesia in patients less than six years of age. Anesth Analg. 2000;91:573–9.
43. Lehr VT, BeVier P. Patient-controlled analgesia for the pediatric patient. Orthop Nurs. 2003;22:298–304.
44. Voepel-Lewis T, Marinkovic A, Kostrzewa A, Tait AR, Malviya S. The prevalence and risk factors for adverse events in children receiving patient-controlled analgesia by proxy or patient-controlled analgesia. Anesth Analg. 2008;107:70–5.
45. Gibb IA, Anderson BJ. Paracetamol [acetaminophen] pharmacodynamics: interpreting the plasma concentration. Arch Dis Child. 2008;93:241–7.
46. van der Marel CD, van Lingen RA, Pluim MA, Scoones G, van Dijk M, Vaandrager JM, Tibboel D. Analgesic efficacy of rectal versus oral acetaminophen in children after major craniofacial surgery. Clini Pharmacol Ther. 2001;70:82–90.
47. Heubi JE, Barbacci MB, Zimmerman HJ. Therapeutic misadventures with acetaminophen: hepatoxicity after multiple doses in children. J Pediatr. 1998;132:22–7.
48. Savino F, Lupica MM, Tarasco V, Locatelli E, Garazzino S, Tovo PA. Fulminant hepatitis after 10 days of acetaminophen treatment at recommended dosage in an infant. Pediatrics. 2011;127:e494–7.
49. Birmingham PK, Tobin MJ, Fisher DM, et al. Initial and subsequent dosing of rectal acetaminophen in children: a 24-hour pharmacokinetic study of new dose recommendations. Anesthesiology. 2001;94:385–9.

50. Birmingham PK, Tobin MJ, Henthorn TK, et al. 24 hour pharmacokinetics of rectal acetaminophen in children: an old drug with new recommendations. Anesthesiology. 1997;87:244–52.
51. Kumpulainen E, Kokki H, Halonen T, et al. Paracetamol (acetaminophen) penetrates readily into the cerebrospinal fluid of children after intravenous administration. Pediatrics. 2007;119:766–71.
52. Malmberg AB, Yaksh TL. Hyperalgesia mediated by spinal glutamate or substance P receptor blocked by spinal cyclooxygenase inhibition. Science. 1992;257:1276–9.
53. Cardwell M, Siviter G, Smith A. Non-steroidal anti-inflammatory drugs and perioperative bleeding paediatric tonsillectomy. Cochrane Database Syst Rev. 2005;7.
54. Clark E, Plint AC, Correll R, Gaboury I, Passi B. A randomized, controlled trial of acetaminophen, ibuprofen, and codeine for acute pain relief in children with musculoskeletal trauma. Pediatrics. 2007;119:460–7.
55. Moores MA, Wandless JG, Fell D. Paediatric postoperative analgesia. A comparison of rectal diclofenac with caudal bupivacaine after inguinal herniotomy. Anaesthesia. 1990;45:156–8.
56. Ryhänen P, Adamski J, Puhakka K, Leppäluoto J, Vuolteenaho O, Ryhänen J. Postoperative pain relief in children. A comparison between caudal bupivacaine and intramuscular diclofenac sodium. Anaesthesia. 1994;49:57–61.
57. Baer GA, Rorarius MG, Kolehmainen S, Selin S. The effect of paracetamol or diclofenac administered before operation on postoperative pain and behaviour after adenoidectomy in small children. Anaesthesia. 1992;47:1078–80.
58. Tawalbeh MI, Nawasreh OO, Husban AM. Comparative study of diclofenac sodium and paracetamol for treatment of pain after adenotonsillectomy in children. Saudi Med J. 2001;22:121–3.
59. Lieh-Lai MW, Kauffman RE, Uy HG, Danjin M, Simpson PM. A randomized comparison of ketorolac tromethamine and morphine for postoperative analgesia in critically ill children. Crit Care Med. 1999;27:2786–91.
60. Papacci P, De Francisci G, Iacobucci T, Giannantonio C, De Carolis MP, Zecca E, Romagnoli C. Use of intravenous ketorolac in the neonate and premature babies. Paediatr Anaesth. 2004;14:487–92.
61. Cohen MN, Christians U, Henthorn T, et al. Pharmacokinetics of single dose intravenous ketorolac in infants aged 2011 months. Anesth Analg. 2011;112:655–60.
62. Lynn AM, Bradford H, Kantor ED, et al. Postoperative ketorolac tromethamine use in infants aged 6–18 months: the effect on morphine usage, safety assessment, and stereo-specific pharmacokinetics. Anesth Analg. 2007;104:1040–51.
63. Moffett BS, Wann TI, Carberry KE, Mott AR. Safety of ketorolac in neonates and infants after cardiac surgery. Paediatr Anaesth. 2006;16:424–8.
64. Krishna S, Hughes LF, Lin SY. Postoperative hemorrhage with nonsteroidal anti-inflammatory drug use after tonsillectomy: a meta-analysis. Arch Otolaryngol Head Neck Surg. 2003;129:1086–9.
65. Møiniche S, Rømsing J, Dahl JB, Tramèr MR. Nonsteroidal antiinflammatory drugs and the risk of operative site bleeding after tonsillectomy: a quantitative systematic review. Anesth Analg. 2003;96:68–77.
66. Munro HM, Walton SR, Malviya S, Merkel S, Voepel-Lewis T, Loder RT, Farley FA. Low-dose ketorolac improves analgesia and reduces morphine requirements following posterior spinal fusion in adolescents. Can J Anaesth. 2002;49:461–6.
67. Vitale MG, Choe JC, Hwang MW, Bauer RM, Hyman JE, Lee FY, Roye DP Jr. Use of ketorolac tromethamine in children undergoing scoliosis surgery. An analysis of complications. Spine J. 2003;2003:55–62.
68. Sucato DJ, Lovejoy JF, Agrawal S, Elerson E, Nelson T, McClung A. Postoperative ketorolac does not predispose to pseudoarthrosis following posterior spinal fusion and instrumentation for adolescent idiopathic scoliosis. Spine. 2008;33:1119–24.
69. Finkel JC, Rose JB, Schmitz ML, et al. An evaluation of the efficacy and tolerability of oral tramadol hydrochloride tablets for the treatment of postsurgical pain in children. Anesth Analg. 2002;94:1469–73.
70. Khosravi MB, Khezri S, Azemati S. Tramadol for pain relief in children undergoing herniotomy: a comparison with ilioinguinal and iliohypogastric blocks. Paediatr Anaesth. 2006;16:54–8.
71. Finkel JC, Pestieau SR, Quezado ZM. Ketamine as an adjuvant for treatment of cancer pain in children and adolescents. J Pain. 2007;8:515–21.
72. Conway M, White N, Jean CS, Zempsky WT, Steven K. Use of continuous intravenous ketamine for end-stage cancer pain in children. J Pediatr Oncol Nurs. 2009;26:100–6.
73. Aydin ON, Ugur B, Ozgun S, Eyigör H, Copcu O. Pain prevention with intraoperative ketamine in outpatient children undergoing tonsillectomy or tonsillectomy and adenotomy. J Clin Anesth. 2007;2007:115–9.
74. Bazin V, Bollot J, Asehnoune K, Roquilly A, Guillaud C, De Windt A, Nguyen JM, Lejus C. Effects of perioperative intravenous low dose of ketamine on postoperative analgesia in children. Eur J Anaesthesiol. 2010;27:47–52.
75. Wong C, editor. Epidural and spinal analgesia/anesthesia for labor and vaginal delivery. 5th ed. Philadelphia, PA: Elsevier Saunders; 2014.
76. Kunnumpurath S, Ramessur S, Fendius A, Vadivelu N. Neuraxial blockade: epidural anesthesia. In: Kaye A, Urman R, Vadivelu N, editors. Essentials of regional anesthesia. 1st ed. New York: Springer; 2012. p. 293–338.
77. Dadure SB, Nicolas F, Bromilow L, Raux O, Rochette A, Capdevila X. Continuous epidural block

versus continuous popliteal nerve block for postoperative pain relief after major podiatric surgery in children: a prospective, comparative randomized study. Anesth Analg. 2006;102:744–9.
78. Benzon H. Regional anesthesia in the anticoagulated patient. In: Hadzic A, et al., editors. Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd ed. New York: McGraw-Hill; 2012.
79. De Andrés J, Sala-Blanch X. Ultrasound in the practice of brachial plexus anesthesia. Reg Anesth Pain Med. 2002;27:77–89.
80. Tsui B, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents: a review of current literature and its application in the practice of extremity and trunk blocks. Anesthesiology. 2010;112:473–92.
81. Tsui BC, Suresh S. Ultrasound imaging for regional anesthesia in infants, children, and adolescents: a review of current literature and its application in the practice of neuraxial blocks. Anesthesiology. 2010;112:719–28.
82. Neal JM, Brull R, Chan VW, Grant SA, et al. The ASRA evidence -based medicine assessment of ultrasound-guided regional anesthesia and pain medicine: executive summary. Reg Anesth Pain Med. 2010;35:1–9.
83. Antonakakis J. Ultrasound-guided regional anesthesia for peripheral nerve blocks: an evidence-based outcome review. Anesthsiol Clin. 2011;29:179–91.
84. Berde C. Convulsions associated with pediatric regional anesthesia. Anesth Analg. 1992;75:164–6.
85. Luz G, Innerhofer P, Bachmann B, Frischhut B, Menardi G, Benzer A. Bupivacaine plasma concentrations during continuous epidural anesthesia in infants and children. Anesth Analg. 1996;82:231–4.
86. Luz G, Wieser C, Innerhofer P, Frischhut B, Ulmer H, Benzer A. Free and total bupivacaine plasma concentrations after continuous epidural anaesthesia in infants and children. Paediatr Anaesth. 1998;8:473–8.
87. Bardsley H, Gristwood R, Baker H, Watson N, Nimmo W. A comparison of the cardiovascular effects of levobupivacaine and racc-bupivacaine following intravenous administration to healthy volunteers. Br J Clin Pharmacol. 1998;46:245–9.
88. Huang YF, Pryor ME, Mather LE, Veering BT. Cardiovascular and central nervous system effects of intravenous levobupivacaine and bupivacaine in sheep. Anesth Analg. 1998;86:797–804.
89. Zsigmond EK, Downs JR. Plasma cholinesterase activity in newborns and infants. Can Anaesth Soc J. 1971;18:278–85.
90. Selden R, Sasahara AA. Central nervous system toxicity induced by lidocaine. Report of a case in a patient with liver disease. JAMA. 1967;202:908–9.
91. Reidenberg MM, James M, Dring LG. The rate of procaine hydrolysis in serum of normal subjects and diseased patients. Clin Pharmacol Ther. 1972;13:278–84.
92. De Jong RH, Heavner JE. Local anesthetic seizure prevention: diazepam versus pentobarbital. Anesthesiology. 1972;36:449–57.
93. Thomson PD, Melmon KL, Richardson JA, Cohn K, Steinbrunn W, Cudihee R, Rowland M. Lidocaine pharmacokinetics in advanced heart failure, liver disease, and renal failure in humans. Ann Intern Med. 1973;78:499–508.
94. Englesson S. The influence of acid-base changes on central nervous system toxicity of local anaesthetic agents. I. An experimental study in cats. Acta Anaesthesiol Scand. 1974;18:79–87.
95. Englesson S, Grevsten S. The influence of acid-base changes on central nervous system toxicity of local anaesthetic agents. II. Acta Anaesthesiol Scand. 1974;18:88–103.
96. Roberts JK, Westphal S, Sparks MA. Iatrogenic baclofen neurotoxicity in ESRD: recognition and management. Semin Dial. 2015;28:525–9.
97. Polaner DM, Drescher J. Pediatric regional anesthesia: what is the current safety record? Pediatr Anesth. 2011;21:737–42.
98. Suresh S, Sarwark JP, Bhalla T, Janicki J. Performing US-guided nerve blocks in the postanesthesia care unit (PACU) for upper extremity fractures: is this feasible in children? Paediatr Anaesth. 2009;19:1238–40.
99. Vandepitte C, Lopez AM, Jalil H. Ultrasound-guided axillary brachial plexus block. In: Hadzic A, et al., editors. Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd ed. New York: McGraw-Hill; 2012.
100. Wilson JL, Brown DL, Wong GY, Ehman RL, Cahill DR. Infraclavicular brachial plexus block: parasagittal anatomy important to the coracoid technique. Anesth Analg. 1998;87:870–3.
101. Bendtsen TF, Lopez AM, Vandepitte C. Ultrasound-guided supraclavicular brachial plexus block. In: Hadzic A, et al., editors. Hadzic's peripheral nerve blocks and anatomy for ultrasound-guided regional anesthesia. 2nd ed. New York: McGraw-Hill; 2012.
102. Brown DL, Spites BD, Spence BC. Supraclavicular block. In: Brown DL, editor. Atlas of regional anesthesia. 4th ed. Philadelphia, PA: Saunders Elsevier; 2010.
103. Sala-Blanch X, Lázaro JR, Correa J, Gómez-Fernandez M. Phrenic nerve block caused by interscalene brachial plexus block: effects of digital pressure and a low volume of local anesthetic. Reg Anesth Pain Med. 1999;24:231–5.
104. Ediale KR, Myung CR, Neuman GG. Prolonged hemidiaphragmatic paralysis following interscalene brachial plexus block. J Clin Anesth. 2004;16:573–5.
105. Tetzlaff JE, Yoon HJ, Dilger J, Brems J. Subdural anesthesia as a complication of an interscalene brachial plexus block. Case report. Reg Anesth. 1994;19:357–9.
106. Visoiu M. Paediatric regional anaesthesia: a current perspective. Curr Opin. 2015;28:577–82.

107. American Society of Anesthesiologists Task Force on Acute Pain Management. Practice guidelines for acute pain management in the perioperative setting: an updated report by the American Society of Anesthesiologists Task Force on Acute Pain Management. Anesthesiology. 2012;116(2):248–73.
108. Dingeman RS, Barus LM, Chung HK, Clendenin DJ, Lee CS, Tracy S, et al. Ultrasonography-guided bilateral rectus sheath block vs local anesthetic infiltration after pediatric umbilical hernia repair: a prospective randomized clinical trial. JAMA Surg. 2013;148(8):707–13.
109. Verghese ST, Hannallah RS. Postoperative pain management in children. Anesthesiol Clin North Am. 2005;23(1):163–84.
110. Dalens B, Hasnaoui A. Caudal anesthesia in pediatric surgery: success rate and adverse effects in 750 consecutive patients. Anesth Analg. 1989;68(2):83–9.
111. Wulf H, Peters C, Behnke H. The pharmacokinetics of caudal ropivacaine 0.2% in children. A study of infants aged less than 1 year and toddlers aged 1–5 years undergoing inguinal hernia repair. Anaesthesia. 2000;55(8):757–60.
112. Lee JJ, Rubin AP. Comparison of a bupivacaine-clonidine mixture with plain bupivacaine for caudal analgesia in children. Br J Anaesth. 1994;72(3):258–62.
113. Abdellatif AA. Ultrasound-guided ilioinguinal/iliohypogastric nerve blocks versus caudal block for postoperative analgesia in children undergoing unilateral groin surgery. Saudi J Anaesth. 2012;6(4):367–72.
114. Kendigelen P, Tutuncu AC, Erbabacan E, Ekici B, Koksal G, Altindas F, et al. Ultrasound-assisted transversus abdominis plane block vs wound infiltration in pediatric patient with inguinal hernia: randomized controlled trial. J Clin Anesth. 2016;30:9–14.
115. Bhalla T, Sawardekar A, Dewhirst E, Jagannathan N, Tobias JD. Ultrasound-guided trunk and core blocks in infants and children. J Anesth. 2013;27(1):109–23.
116. Chelly JE. Paravertebral blocks. Anesthesiol Clin. 2012;30(1):75–90.
117. Greengrass RA, Duclas R Jr. Paravertebral blocks. Int Anesthesiol Clin. 2012;50(1):56–73.
118. Elsayed H. Insertion of paravertebral block catheters intraoperatively to reduce incidence of block failure. Interact Cardiovasc Thorac Surg. 2012;14(5):648–9.
119. Helms O, Mariano J, Hentz JG, Santelmo N, Falcoz PE, Massard G, et al. Intra-operative paravertebral block for postoperative analgesia in thoracotomy patients: a randomized, double-blind, placebo-controlled study. Eur J Cardiothorac Surg. 2011;40(4):902–6.
120. Chen LM, Weinberg VK, Chen C, Powell CB, Chen LL, Chan JK, et al. Perioperative outcomes comparing patient controlled epidural versus intravenous analgesia in gynecologic oncology surgery. Gynecol Oncol. 2009;115(3):357–61.
121. Diwan SA, Malhotra V, Metha ND, Piracha MM. Chapter 48: Complex Regional Pain Syndromes. In: Yao F, Malhotra V, Fong J, Skubas N, editors. Yao and Artusio's Anesthesiology. 8th ed. Wolters: Kluwer; 2016. p. 912–23.
122. Berde C, Greco C. Chapter 33: Pain management in children. In: Gregory GA, Andropoulos DB, editors. Gregory's pediatric anesthesia. 5th ed. Oxford: Wiley-Blackwell; 2011. p. 133–7.
123. Berde C, Greco C. Chapter 33: Pain management in children. In: Gregory GA, Andropoulos DB, editors. Gregory's pediatric anesthesia. 5th ed. Oxford: Wiley-Blackwell; 2011. p. 138.
124. Sherry DD, Wallace CA, Kelley C, Kidder M, Sapp L. Short- and long-term outcomes of children with complex regional pain syndrome type I treated with exercise therapy. Clin J Pain. 1999;15(3):218–23.
125. Imani F, Hemati K, Rahimzadeh P, Kazemi MR, Hejazian K. Effectiveness of stellate ganglion block under fuoroscopy or ultrasound guidance in upper extremity CRPS. J Clin Diagn Res. 2016;10(1):UC09–12.
126. Hsu ES. Practical management of complex regional pain syndrome. Am J Ther. 2009;16(2):147–54. (the use of popliteal catheter for CRPS)
127. Montgomery SH, Shamji CM, Yi GS, Yarnold CH, Head SJ, Bell SC, et al. Effect of nerve stimulation use on the success rate of ultrasound-guided subsartorial saphenous nerve block: a randomized controlled trial. Reg Anesth Pain Med. 2017;42(1):25–31.
128. Kayode W, Guarino AM, Raja SN. Complex regional pain syndrome. In: Benzon HT, editor. Essentials of pain medicine. 3rd ed. London: Saunders; 2011. p. 351–7.
129. Dadure C, Motais F, Ricard C, Raux O, Troncin R, Capdevila X. Continuous peripheral nerve blocks at home for treatment of recurrent complex regional pain syndrome I in children. Anesthesiology. 2005;102(2):387–91.
130. Rho RH, Brewer RP, Lamer TJ, Wilson PR. Complex regional pain syndrome. Mayo Clin Proc. 2002;77(2):174–80.
131. Pedemonte Stall V, Medici Olaso C, Kanopa Almada V, Gonzalez Rabelino G. Complex regional pain syndrome type 1. Nuerologia. 2015;30:347–51. https://doi.org/10.1016/j.nrl.2013.12.016. Epub 2014 Jun 18
132. Saltik S, Sozen HG, Basgul S, Karatoprak EY, Icagasioglu A. Pregabalin treatment of a patient with complex regional pain syndrome. Pediatr Neurol. 2016;54:88–90.
133. Shanthanna H, Aboutouk D, Poon E, Cheng J, Finley C, Paul J, et al. A retrospective study of open thoracotomies versus thoracoscopic surgeries for persistent postthoracotomy pain. J Clin Anesth. 2016;35:215–20.
134. Maguire MF, Ravenscroft A, Beggs D, Duffy JP. A questionnaire study investigating the prevalence

of the neuropathic component of chronic pain after thoracic surgery. Eur J Cardiothorac Surg. 2006;29(5):800–5.
135. SSM R, Gadsden JM. Chapter 76: Preemptive analgesia. Regional anesthesia & the prevention if chronic postoperative pain. In: Hadic A, editor. Textbook of regional anesthesia and acute pain management. New York: McGraw-Hill; 2007. p. 1097–9.
136. Hu JS, Lui PW, Wang H, Chan KH, Luk HN, Tsou MY, et al. Thoracic epidural analgesia with morphine does not prevent postthoracotomy pain syndrome: a survey of 159 patients. Acta Anaesthesiol Sin. 2000;38(4):195–200.
137. Senturk M, Ozcan PE, Talu GK, Kiyan E, Camci E, Ozyalcin S, et al. The effects of three different analgesia techniques on long-term postthoracotomy pain. Anesth Analg. 2002;94(1):11–5. table of contents
138. Raveglia F, Rizzi A, Leporati A, Di Mauro P, Cioffi U, Baisi A. Analgesia in patients undergoing thoracotomy: epidural versus paravertebral technique. A randomized, double-blind, prospective study. J Thorac Cardiovasc Surg. 2014;147(1):469–73.
139. Wildgaard K, Ravn J, Kehlet H. Chronic postthoracotomy pain: a critical review of pathogenic mechanism and strategies for prevention. Eur J Cardiothorac Surg. 2009;36:170–80.
140. Platt OS, Brambilla DJ, Rosse WF, Milner PF, Castro O, Steinberg MH, Klug PP. Mortality in sickle cell disease. Life expectancy and risk factors for early death. N Engl J Med. 1994;330:1639–44.
141. Steinberg M. Management of sickle cell disease. N Engl J Med. 1999;340:1021–30.
142. Stuart MJ, Nagel RL. Sickle-cell disease. Lancet. 2004;364:1343–60.
143. Firth PG, Head CA. Sickle cell disease and anesthesia. Anesthesiology. 2004;101:766–85.
144. Vichinsky EP, Neumayr LD, Earles AN, Williams R, et al. Causes and outcomes of the acute chest syndrome in sickle cell disease. National Acute Chest Syndrome Study Group. N Engl J Med. 2000;342:1855–65.
145. Yawn BP, Buchanan GR, Afenyi-Annan AN, Ballas SK, et al. Management of sickle cell disease: summary of the 2014 evidence-based report by expert panel members. JAMA. 2014;312:1033–48.
146. Charache S, Terrin ML, Moore RD, Dover GJ, Barton FB, Eckert SV, RP MM, Bonds DR. Effect of hydroxyurea on the frequency of painful crises in sickle cell anemia. Investigators of the multicenter study of hydroxyurea in sickle cell anemia. N Engl J Med. 1995;323:1317–22.
147. Gladwin MT, Schechter AN. Nitric oxide therapy in sickle cell disease. Semin Hematol. 2001;38:333–42.
148. Buchanan GR, DeBaun MR, Quinn CT, Steinberg MH. Sickle cell disease. Hematology American Society of Hematology Education Program 2004;2004:35–47.
149. Okpala I, Tawil A. Management of pain in sickle-cell disease. J R Soc Med. 2002;86:456–8.
150. Hostetler SG, Schwartz L, Shields BJ, Xiang H, Smith GA. Characteristics of pediatric traumatic amputations treated in hospital emergency departments: United States, 1990–2002. Pediatrics. 2005;116(5):e667–74.
151. Nagarajan R, Neglia JP, Clohisy DR, Robison LL. Limb salvage and amputation in survivors of pediatric lower-extremity bone tumors: what are the long-term implications? J Clin Oncol. 2002;20(22):4493–501.
152. Nathanson M. Phantom limbs as reported by S. Weir Mitchell. Neurology. 1988;38(3):504–5.
153. Nikolajsen L, Jensen TS. Phantom limb pain. Br J Anaesth. 2001;87:107–16.
154. Karl A, Birbaumer N, Lutzenberger W, Cohen LG, Flor H. Reorganization of motor and somatosensory cortex in upper extremity amputees with phantom limb pain. J Neurosci. 2001;21(10):3609–18.
155. Borghi MD, White PF, et al. The use of prolonged peripheral neural blockade after lower extremity amputation: the effect on symptoms associated with phantom limb syndrome. Anesth Analg. 2010;111(5):1308–15.

Part VIII

Future Trends in Regional Anesthesia

Future Trends in Equipment

38

Neda Sadeghi and Jeff Gadsden

Abbreviations

EMF	Electromagnetic field
GPS	Global positioning satellite
pPNS	Percutaneous peripheral nerve stimulation
USB	Universal serial bus

Introduction

The growth of regional anesthesia in modern anesthetic practice has been fueled largely by innovations in regional anesthetic technology, most notably improvements in ultrasound machine resolution and ease of use. This chapter aims to highlight several trends in equipment used for regional anesthesia including the advent of ultraportable ultrasound machines, enhanced three-dimensional needle guidance features, and innovative technology for providing local analgesia using ultrasound-guided percutaneous placement of stimulating electrical filaments.

Pocket Ultrasound Machines

A steady trend in ultrasound machine manufacturing has been the progressive miniaturization of units in an effort to provide portability, affordability, and ease of use. At its outset, clinicians seeking to perform ultrasound-guided nerve blocks were forced to use large, bulky cart-based systems that were designed to be stationed in one place, such as the radiology suite or the cardiac operating theater. Over time, smaller cart-based systems and laptop-style devices permitted the user to move throughout the hospital easily, introducing the concept of point-of-care ultrasound. More recently, innovations in microprocessors and ultrasound transducer technology have ushered in an era of even smaller, ultraportable ("pocket") ultrasound machines [1]. These are handheld devices that are designed to be carried with the clinician and applied as needed for diagnostic or interventional purposes. Despite their smaller dimensions, these ultraportable units appear to maintain the high image quality expected from the laptop or cart-based machines for most procedural applications, although they may not yet provide the resolution required for detailed diagnostic tests such as those performed by radiologists [2].

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_38

Pocket ultrasound machines vary by manufacturer in their design, ergonomics, and pricing plan. They can be categorized broadly into two categories: those that are a self-contained unit where a transducer is fixed to a small screen and those where the transducer connects to an off-the-shelf tablet or phone via a wireless or universal serial bus (USB) connection. For example, the Vscan™ (GE Healthcare, Waukesha, WI), one of the earliest devices on the market, features a handheld, base with a cover that flips up to reveal the screen and a navigation wheel that can be controlled with the user's thumb [3]. The matrix-array transducer is affixed to the base via a flexible cord. Owing to the increased use of point-of-care ultrasound and the need to scan shallow as well as deep visceral structures, this company has developed a specialized transducer that has both matrix-array and linear-array heads on the same transducer. The transducer is attached to either a flip-style base or a touchscreen smartphone-style screen/interface (Fig. 38.1).

In contrast, the Lumify ultrasound system (Philips Healthcare, Andover, MA) does not have a proprietary screen but is instead composed of one of three transducers (matrix-array, linear-array and broad curved array) connected to a smart device (Android phone or tablet) via a cabled USB connection [4]. Similarly, the Clarius portable ultrasound system (Clarius, Burnaby, BC) connects a proprietary handheld transducer to a generic app-enabled smart device (Android or iOS) but does so wirelessly (Fig. 38.2) [5]. Currently this company has a combined convex and virtual matrix-array transducer, with plans to release a linear transducer in the future. All of these devices feature various combinations of presets for point-of-care.

Fig. 38.1 The Vscan Extend pocket ultrasound machine. The dual-headed transducer is hard-wired to the handheld screen unit. Figure provided by GE Healthcare and used with permission

Fig. 38.2 The Clarius pocket ultrasound machine. The handheld wireless transducer transmits to a generic smart device. Figure provided by Clarius Mobile Health and used with permission

Due to the nature of the specialty, internal medicine and critical care physicians have been leaders in demonstrating value in using pocket ultrasound machines. These devices have been used to enhance diagnosis and treatment in heart failure [6, 7], accurately diagnose forearm fracture and evaluate degree of reduction [8], and precisely measure the diameter of the optic nerve [9]. Pocket ultrasound machines are also well-suited for both undergraduate and graduate medical education, and some medical schools have outfitted entire classes of students with machines as part of a longitudinal ultrasound curriculum [10].

While innovative and intuitively attractive, there may be some limitations to pocket ultrasound systems. One important consideration is whether a system can permit the sharing, annotating, and uploading of images to the hospital image archiving system in HIPAA-compliant fashion. Another potential limitation to the use of pocket ultrasound machines is their reliance on battery power. Typically, these devices allow for 45–60 min of scanning time or 7–8 h of standby time. This may be acceptable for a hospitalist who uses the machine 3–4 times per day and is able to charge it in between consults. However, it may be inconvenient for a proceduralist who is scanning for much of an 8–12-h shift.

Indeed, what remains unclear is the utility of pocket ultrasound machines for use in regional anesthesia. It is likely that the image quality is sufficient, given the advances in that technology. Regional anesthesiologists require two hands to both grasp the transducer and the needle. The ergonomics and practicalities of where to place the "screen" with a pocket ultrasound machine in such a case may be tricky compared to using a laptop or cart-based system where the vertical screen is maintained in a certain position and is easily accessible. Indeed, there are no reports in the scientific literature of peripheral nerve blocks being facilitated with pocket ultrasonography. Notwithstanding, the extreme portability and accessibility of these devices have the potential to increase the role of ultrasound-guided regional anesthesia, perhaps into relatively novel areas of the hospital such as the critical care unit [11]. In addition, the price point (generally less than $5000) is attractive to centers that may not be willing or able to invest in a cart-based system.

In contrast to traditional ultrasonography, automated scanners are being introduced that provide the practitioner with useful information regarding depth, bony anatomy, and alignment. For example, the Accuro device (Rivanna Medical, Charlottesville, VA) is an "intelligent" handheld ultrasound scanner designed specifically for neuraxial imaging that permits the rapid identification of the correct craniocaudal needle insertion site, midline, and angle of insertion. A small prospective study of 47 women undergoing ultrasound-assisted labor epidural placement revealed that this device was simple to use and resulted in similar first-pass success rates and needle redirections as a traditional cart-based system [12].

Needle Visualization: Enhanced Needle and Guidance Features

Needle visualization during nerve block procedures can be a challenge, especially when the angle of incidence between the ultrasound probe surface and the needle exceeds 30–45° [13, 14]. Design features that result in enhanced needle visibility have the potential to improve block success and patient safety [15]. For example, in the last decade, the creation of echogenic needles has allowed for enhanced visualization of the needle by creating a "textured" needle with a strong reflective surface by laser-engraving serrated notches into the needle shaft. While sound waves striking a smooth needle at a 45° angle are likely to be directed off to the periphery, resulting in poor visualization, corner cubes impart discrete areas of 45° cutouts, so that sound waves are much more likely to strike the perpendicular face and reflect back to the transducer surface [16]. These needles have been shown to be have a significantly improved visibility compared to non-echogenic needles in both novice and experienced anesthesiologists [14, 15, 17]. Advances in materials related to both the metal needle itself and the coating used to insulate the needle may further improve the relative degree of echogenicity in the future.

Some investigators have sought to improve needle visualization through other electrical or mechanical strategies. Klein et al. attached simple, inexpensive piezoelectric actuators to the proximal ends of nerve block needles and polyamide catheters [18]. Using a cadaveric simulated sciatic nerve block model, a small oscillation applied at the proximal ends caused the tips of these devices to vibrate to a sufficient degree that the vibration was easily detected from the background image using color Doppler. This technological innovation has not been widely adopted to date, but due to its simplicity and low cost, it warrants further investigation. Beigi et al. reported an innovative solution to

difficult visualization at steep angles involving a needle-within-needle design: a large gauge (i.e., 17G Tuohy needle) was inserted into a meat phantom model, and the operator oscillated the inner stylet 2–3 times/s, moving it 3 cm with each movement, in order to create a visually apparent motion signal (without the use of Doppler imaging) [19]. This resulted in excellent estimation of the needle position, even at depths up to 9 cm and angles as steep at 80°, indicating that this may be a useful and underused technique, particularly as it does not require any additional equipment or expense.

A relatively new innovation in ultrasound technology is the advent of electronic needle tracking or guidance. Often described as "GPS" for regional anesthesia procedures, these machines use needle detection software so that the position of the needle and its trajectory can be inferred and displayed on the screen (Fig. 38.3). Perhaps most useful is the feature that the needle need not be directly underneath the beam. As long as the needle is in reasonable proximity to the transducer head, it can be detected, and its orientation projected on the screen. In this way, clinicians are no longer constrained by the need to use an in-plane orientation in order to feel comfortable with the needle position. It has been well-established that inadvertent forward movement of the needle without visualizing the needle tip is both a very common novice mistake and the cause of inadvertent puncture of target structures [13, 20]. By superimposing the needle tip position, depth, and/or trajectory onto the image of the underlying tissues, this may confer an additional margin of safety.

Ultrasound manufacturers that feature needle navigation have achieved this innovation using various methods. Most companies utilize the principle that electromagnetic interference—a metal needle entering an established electromagnetic field—will distort that field to a degree, and the precise pattern of distortion can predict position in space. Some companies utilize an external electromagnetic field receiver, whereas others use the transducer head itself as the source of the electromagnetic field. From a practical point of view, all of the current manufacturers display some type of real-time positional information on the screen during the procedure. The technical and display features of the current models available on the market are listed in Table 38.1 [21–26].

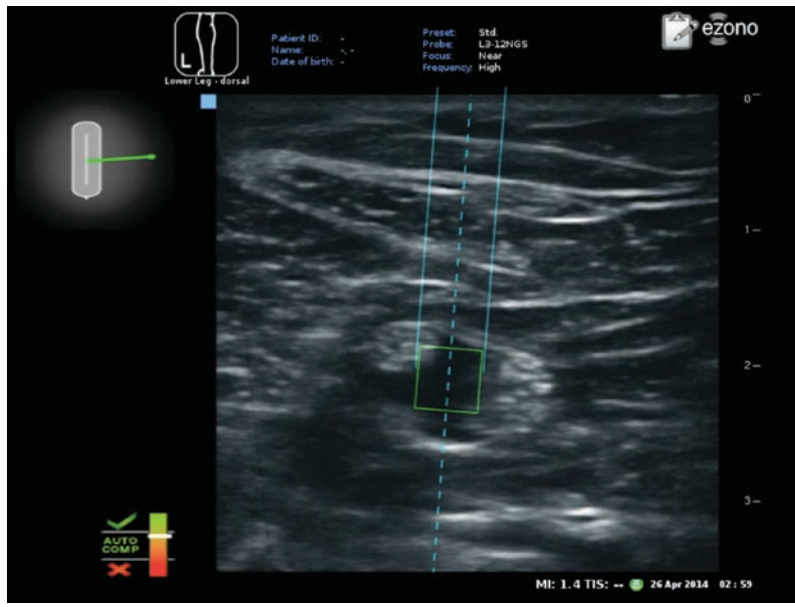

Fig. 38.3 An example of needle guidance technology used for a popliteal sciatic nerve block (eZono AG, Jena, Germany). The needle is approaching the transducer in an out-of-plane orientation (as signified by the graphic in the top-left corner) and is therefore not readily seen on the screen. The screen markings signifying trajectory (single dotted line) and depth (double solid lines) are easily visible. The square box shows where the needle will intersect with the plane of the beam and changes color depending on the proximity to that plane

Table 38.1 Ultrasound systems featuring needle navigation or tracking

System	Transducers	External EMF base?	Proprietary needle required?	Possible needle orientations	On screen display elements				Reference
					Transducer/ needle relationship	Trajectory	Needle depth	Needle proximity to plane of ultrasound beam	
SonixGPS (Analogic)	Linear and curved array	Required	Yes	360°; any needle approach	Yes	Yes	Yes	Only with in-plane; not progressive	[21]
Logiq E9 Needle Tracking (GE Healthcare)	Linear and curved array	Required	Yes—designed for interventional suite purposes (e.g. biopsy, ablation, aspiration)	In plane or out-of-plane	No	Yes	Yes	Yes, progressive and color-coded	[22]
eSieGuide Needle Tracking (Siemens)	Linear and curved array	Required	Yes—designed for interventional suite purposes (e.g. biopsy, ablation, aspiration)	360°; any needle approach	No	Yes	Yes	Yes	[23]
Virtual Navigator and Biopsy (Esaote)	Linear and curved array	Required	Yes—designed for interventional suite purposes (e.g. biopsy, ablation, aspiration)	360°; any needle approach	No	Yes	Yes	Yes	[24]
AxoTrack (SonoSite)	Specialized transducer for vascular access	No	Yes	Limited to in-plane	No	Yes	Yes	No	[25]
eZono 4000 (eZono AG)	Linear and curved array	No	No	360°; any needle approach	Yes	Yes	Yes	Yes, progressive and color-coded	[26]

EMF electromagnetic field

Needle navigation guidance appears to significantly improve performance during needle insertion tasks in phantom models versus conventional ultrasound. Groups allocated to guidance required fewer needle passes or redirection attempts, had greater proportion of task completion, and had a greater number of completions on the first attempt [27–30]. Interestingly, these results appear to be applicable to both inexperienced and expert practitioners of ultrasound-guided nerve blocks.

This technology may be useful for tasks that are both precise and are associated with potential for high-stakes complications (such as pleural puncture). In one study of 52 anesthesiologists, those randomized to use needle guidance during a simulated thoracic paravertebral block in a high-fidelity phantom model had a significantly reduced failure rate (failure defined as final needle tip position outside the paravertebral space) compared to those using conventional ultrasonography (OR 0.29, 95% CI 0.13–0.67, $p = 0.0042$) [28].

Finally, needle guidance features on ultrasound machines may be useful in education. Technical skills have been traditionally taught using an apprenticeship model (i.e., "see one, do one, teach one"). Clearly, with an increased emphasis on patient safety, there is a need to have trainees practice in a simulated environment. However, this often requires time and repeated training sessions to achieve competence. Levine et al. studied 50 anesthesiology and internal medicine trainees with no ultrasound experience. After an initial educational program familiarizing the trainees with ultrasound-guided procedures, subjects were randomized to a dedicated practice session of 20 simulated nerve blocks with the needle navigation feature turned on or off. Subjects then underwent a test of skill during a conventional (i.e., no navigation) ultrasound-guided task. Those who underwent the training session with needle navigation achieved a greater degree of competency and significantly shorter procedure time compared to those trained on conventional ultrasound. In other words, practice with needle navigation made those trainees better at ultrasound procedures—even when navigation was turned off—compared to the other cohort. The investigators could not say why the technology accelerated the learning curve for those trainees, but theorized that the real-time representation of the needle position on the screen sharpened the three-dimensional spatial and hand-eye coordination skills and permitted faster associations between needle movement and resulting position.

While this type of technology has been established in other areas of medicine such as percutaneous liver or breast biopsy [31, 32], regional anesthesiologists have only become interested in this during the last several years, and even then it is far from widespread. There appears to be an intuitive safety advantage to being able to infer where the needle tip is despite not observing it on the screen. Most of the studies that show an advantage in precision have been done in phantom models, and perhaps additional studies demonstrating an actual safety advantage in patients are required before this technology gains wider appeal.

Percutaneous Peripheral Nerve Stimulation

Many of the practical challenges related to peripheral nerve blocks of the lower limb are associated with the limitations of current local anesthetics: a certain mass of drug is required to provide adequate sensory blockade and analgesia, but even at low concentrations, some motor blockade may be present, thereby adversely affecting mobility and recovery. Ultrasound-guided percutaneous peripheral nerve stimulation (pPNS) is a novel technique that aims to provide targeted analgesia without some of the drawbacks of local anesthetic-based techniques. Nerve stimulation has been utilized for years in an effort to relieve pain. While there are several theories as to the mechanism of action, the gate theory of Melzack and Wall is the prevailing explanation: stimulation of large-diameter sensory fibers causes the inhibition of small-diameter pain fibers in the dorsal horn of the spinal cord. Nerve stimulation does not work well for acute pain since spinal and peripheral nerve stimulators

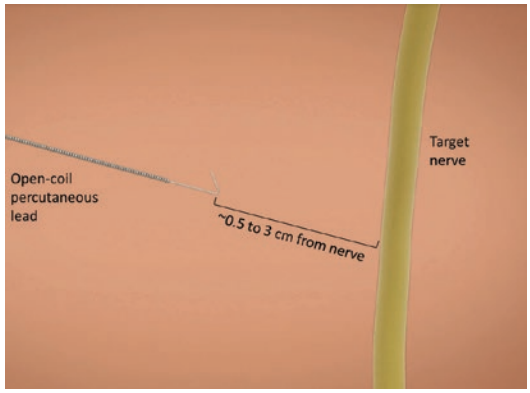

Fig. 38.4 Open-coil percutaneous nerve stimulation lead shown in proximity to target nerve. Figure provided by SPR Therapeutics, LLC and used with permission

require invasive surgery and transcutaneous electrical nerve stimulation (TENS) units are often associated with cutaneous discomfort at current intensities necessary to provide an effect [33].

Percutaneous peripheral nerve stimulation involves the ultrasound-guided deployment of a very small coiled wire lead (Sprint PNS™, SPR Therapeutics, Cleveland, OH) in the vicinity of a nerve or plexus, using a technique similar to placement of a perineural catheter (Fig. 38.4). A 20G needle is advanced under ultrasound guidance until it is within 0.5–3.0 cm of the target nerve; the pre-loaded 0.2 mm diameter wire lead is then deployed by withdrawing the needle. The lead is electrically stimulated (100 Hz, 15–200 μs, 0.2–20 mA), and an acceptable current is determined that provides a comfortable paresthesia sensation without muscular contraction. With the use of a battery-powered generator pack affixed to the patient's skin, these systems can be left in place for weeks and weeks. Overall, this system provides the theoretical advantage of being relatively noninvasive and anatomically precise, with little stimulation of cutaneous or other unrelated nerves. In addition, the current can be instantly turned off and on, making it fully and instantly reversible.

pPNS was recently used in five patients who had undergone a total knee arthroplasty and had significant pain within 60 days of surgery [34]. Leads were inserted on postoperative days 8–58, at the femoral and sciatic nerve locations. pPNS decreased pain at rest by 93% from baseline, with four of five subjects experiencing complete resolution of their pain. Pain during active knee range of motion was also decreased by 30% compared to baseline. Overall range of motion was not improved despite the pain relief.

The pPNS leads can be left in place for approximately 2 months. The infection rate is unknown given the small number of patients that have undergone this therapy, but a recent review of infection rates based on lead design showed that the open coil structure (e.g., similar to a spring shock absorber) is 25 times less likely to become infected than a solid, non-coiled design [35]. The reasons for this are unclear but may relate to the "pistoning" in and out of the skin that occurs with non-coiled catheters every time the patient moves, thereby dragging skin pathogens subcutaneously; in contrast, a coiled lead may stretch and compress with movement, leading to less pistoning at the skin surface.

While this technology has been investigated primarily for acute postoperative pain, there are potential uses in chronic pain. Kapural et al. reported two cases of patients with chronic low back pain treated with a 1-month course of pPNS therapy [36]. After the leads were withdrawn, both patients experienced significant and sustained (>4 months) improvements in pain, functional outcomes, and opioid consumption. Similar successful results have been obtained when used for hemiplegic shoulder pain [37].

pPNS is a technology that is both novel and promising. At present, there are several trials underway assessing efficacy for total knee arthroplasty, chronic post-amputation pain, and chronic back pain and more planned for total shoulder and ankle arthroplasty cases. While the data is scarce to date, this is an idea and a piece of equipment worth watching for the future.

Clinical Case Study

A 57-year-old female with breast cancer is scheduled for a bilateral mastectomy and placement of tissue expanders. She is concerned about postoperative pain management and postoperative nausea

and vomiting. Her past medical history is significant for BMI 39 and mild-moderate COPD. You plan to administer general anesthesia with positive pressure ventilation and also to perform preoperative, bilateral, paravertebral nerve blocks (four separate injections) for the management of postoperative pain. You are working with a resident who is inexperienced with regional anesthesia.

Discussion

Is there a way to improve the safety and success of an ultrasound-guided paravertebral blocks on this patient with difficult anatomy while minimizing her risk of pneumothorax?

Ultrasound-guided thoracic paravertebral block is an increasingly common anesthetic and analgesic procedure. However, visualization of both the paravertebral anatomy (due to bony vertebral structures) and the needle (due the angulation) frequently complicates attempts to precisely place the needle tip in the paravertebral space, especially in the obese population. These challenges make the ultrasound-guided paravertebral block challenging to both teach and perform. In this instance, one could benefit from the use of an ultrasound machine with a needle guidance feature that allows for precise determination of the needle tip position without the need for visualization. The three-dimensional needle guidance feature has been shown to improve the rate of successfully placing the needle in the paravertebral space when performed in a high-fidelity phantom model. While there are no human safety data, the ability to precisely infer where the needle is in space is certainly reassuring, especially for the novice clinician in what is a high-stakes procedure.

Review Questions

1. Which of the following is NOT a potential limitation of the use of portable "pocket" ultrasound machines
   (a) Ability to interact with hospital wireless and image archiving systems
   (b) Image quality
   (c) Battery life
   (d) Ergonomics during procedural applications
2. Three-dimensional electronic needle guidance ("GPS") is associated with:
   (a) Reduced incidence of local anesthetic systemic toxicity
   (b) Improved image quality
   (c) Reduced failure rate during simulated paravertebral block
   (d) The need for a second operator to operate the machine
3. Percutaneous peripheral nerve simulating leads may be left in place for:
   (a) 48 h
   (b) 3–5 days
   (c) 2 weeks
   (d) 2 months

Answers

1. b
2. c
3. d

References

1. Woo JSL, Magotti R, Benzie R. A futuristic vision of pocket ultrasound machines: watch this space. Australas J Ultrasound Med. 2014;17:110–2.
2. Stock KF, Klein B, Steubl D, Lersch C, Heemann U, Wagenpfeil S, et al. Comparison of a pocket-size ultrasound device with a premium ultrasound machine: diagnostic value and time required in bedside ultrasound examination. Abdom Imaging. 2015;40:2861–6.
3. General Electric Company. GE Healthcare: Vscan Family. http://www3.gehealthcare.com/en/products/categories/ultrasound/vscan_family. Accessed 29 Apr 2017.
4. Koninklijke Philips B.V. Philips: Lumify. https://www.lumify.philips.com/web/. Accessed 29 Apr 2017.
5. Clarius Mobile Health. https://www.clarius.me/. Accessed 29 Apr 2017.
6. Goonewardena SN, Gemignani A, Ronan A, Vasaiwala S, Blair J, Brennan JM, et al. Comparison of hand-carried ultrasound assessment of the inferior vena cava and N-terminal pro-brain natriuretic

peptide for predicting readmission after hospitalization for acute decompensated heart failure. JACC Cardiovasc Imaging. 2008;1:595–601.
7. Khandwalla RM, Birkeland KT, Zimmer R, Henry TD, Nazarian R, Sudan M, et al. Usefulness of serial measurements of inferior vena cava diameter by Vscan(TM) to identify patients with heart failure at high risk of hospitalization. Am J Cardiol. 2017;119:1631–6.
8. Lau BC, Robertson A, Motamedi D, Lee N. The validity and reliability of a pocket-sized ultrasound to diagnose distal radius fracture and assess quality of closed reduction. J Hand Surg Am. 2017;42(6):420–7.
9. Johnson GGRJ, Zeiler FA, Unger B, Hansen G, Karakitsos D, Gillman LM. Estimating the accuracy of optic nerve sheath diameter measurement using a pocket-sized, handheld ultrasound on a simulation model. Crit Ultrasound J. 2016;8:18.
10. Hoppmann RA, Rao VV, Bell F, Poston MB, Howe DB, Riffle S, et al. The evolution of an integrated ultrasound curriculum (iUSC) for medical students: 9-year experience. Crit Ultrasound J. 2015;7:18.
11. Haskins SC, Boublik J, Wu CL. Point-of-care ultrasound for the regional anesthesiologist and pain specialist: a series introduction. Reg Anesth Pain Med. 2017;42:281–2.
12. Seligman K, Weiniger C, Carvalho B. Accuracy of a handheld ultrasound device and a traditional ultrasound for neuraxial depth and landmark assessment. Abstract presented at the 42nd ASRA annual regional anesthesiology and acute pain medicine meeting, San Francisco, CA, Apr 2017.
13. Sites BD, Spence BC, Gallagher JD, Wiley CW, Bertrand ML, Blike GT. Characterizing novice behavior associated with learning ultrasound-guided peripheral regional anesthesia. Reg Anesth Pain Med. 2007;32:107–15.
14. Hocking G, Mitchell CH. Optimizing the safety and practice of ultrasound-guided regional anesthesia: the role of echogenic technology. Curr Opin Anaesthesiol. 2012;25:603–9.
15. Sviggum HP, Ahn K, Dilger JA, Smith HM. Needle echogenicity in sonographically guided regional anesthesia: blinded comparison of 4 enhanced needles and validation of visual criteria for evaluation. J Ultrasound Med. 2013;32:143–8.
16. Hebard S, Hocking G, Murray K. Two-dimensional mapping to assess direction and magnitude of needle tip error in ultrasound-guided regional anaesthesia. Anaesth Intensive Care. 2011;39:1076–81.
17. Hebard S, Hocking G. Echogenic technology can improve needle visibility during ultrasound-guided regional anesthesia. Reg Anesth Pain Med. 2011;36:185–9.
18. Klein SM, Fronheiser MP, Reach J, Nielsen KC, Smith SW. Piezoelectric vibrating needle and catheter for enhancing ultrasound-guided peripheral nerve blocks. Anesth Analg. 2007;105:1858–60.
19. Beigi P, Rohling R, Salcudean T, Lessoway VA, Ng GC. Needle trajectory and tip localization in real-time 3-D ultrasound using a moving stylus. Ultrasound Med Biol. 2015;41:2057–70.
20. Sites BD, Gallagher JD, Cravero J, Lundberg J, Blike G. The learning curve associated with a simulated ultrasound-guided interventional task by inexperienced anesthesia residents. Reg Anesth Pain Med. 2004;29:544–8.
21. BK Ultrasound. SonixGPS. https://bkultrasound.com/sonixgps/. Accessed 31 Aug 2014.
22. General Electric Company. GE Healthcare: LOGIQ E9 XDclear 2.0. http://www3.gehealthcare.com/en/products/categories/ultrasound/point_of_care_ultrasound/logiq_e9. Accessed 17 Aug 2017.
23. Siemens Healthcare GmbH. Siemens Healthineers: ACUSON S3000 Ultrasound System, HELX Evolution with Touch Control. https://www.healthcare.siemens.com/ultrasound/general-imaging/acuson-s3000-ultrasound-machine. Accessed 17 Aug 2017.
24. Esaote SpA. MyLab™Twice: Two Ways To Be Unique. http://www.esaote.com/ultrasound/ultrasound-systems/p/mylab-twice/. Accessed 17 Aug 2017.
25. FUJIFILM SonoSite, Inc. AXOTRACK. https://www.sonosite.com/products/axotrack. Accessed 17 Aug 2017.
26. eZono AG. http://www.ezono.com/. Accessed 31 July 2014.
27. Swenson JD, Klingler KR, Pace NL, Davis JJ, Loose EC. Evaluation of a new needle guidance system for ultrasound: results of a prospective, randomized, blinded study. Reg Anesth Pain Med. 2016;41:356–61.
28. Sadeghi N, Gadsden J. Needle guidance improves success rates of thoracic paravertebral block in a high-fidelity phantom model. Abstract presented at the 42nd ASRA annual regional anesthesiology and acute pain medicine meeting, San Francisco, CA, Apr 2017.
29. Johnson AN, Peiffer JS, Halmann N, Delaney L, Owen CA, Hersh J. Ultrasound-guided needle technique accuracy: prospective comparison of passive magnetic tracking versus unassisted echogenic needle localization. Reg Anesth Pain Med. 2017;42:223–32.
30. Tielens LKP, Damen RBCC, Lerou JGC, Scheffer G-J, Bruhn J. Ultrasound-guided needle handling using a guidance positioning system in a phantom. Anaesthesia. 2014;69:24–31.
31. Smith WL, Surry KJM, Kumar A, McCurdy L, Downey DB, Fenster A. Comparison of core needle breast biopsy techniques: freehand versus three-dimensional US guidance. Acad Radiol. 2002;9:541–50.
32. Hakime A, Deschamps F, Carvalho EGM, Barah A, Auperin A, Baere T. Electromagnetic-tracked biopsy under ultrasound guidance: preliminary results. Cardiovasc Intervent Radiol. 2012;35:898–905.
33. Ilfeld BM, Grant SA, Gilmore CA, Chae J, Wilson RD, Wongsarnpigoon A, et al. Neurostimulation for postsurgical analgesia: a novel system enabling

ultrasound-guided percutaneous peripheral nerve stimulation. Pain Pract. 2017;17(7):892–901.
34. Ilfeld BM, Gilmore CA, Grant SA, Bolognesi MP, Del Gaizo DJ, Wongsarnpigoon A, et al. Ultrasound-guided percutaneous peripheral nerve stimulation for analgesia following total knee arthroplasty: a prospective feasibility study. J Orthop Surg Res. 2017;12:4.
35. Ilfeld BM, Gabriel RA, Saulino MF, Chae J, Peckham PH, Grant SA, et al. Infection rates of electrical leads used for percutaneous neurostimulation of the peripheral nervous system. Pain Pract. 2017;17(6):753–62.
36. Kapural L, Gilmore CA, Chae J, Rauck RL, Cohen SP, Saulino MF, et al. Percutaneous peripheral nerve stimulation for the treatment of chronic low back pain: two clinical case reports of sustained pain relief. Pain Pract. 2018;18(1):94–103.
37. Chae J, Wilson RD, Bennett ME, Lechman TE, Stager KW. Single-lead percutaneous peripheral nerve stimulation for the treatment of hemiplegic shoulder pain: a case series. Pain Pract. 2013;13:59–67.

Future Trends in Regional Anesthesia Techniques

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Amanda Kumar and Jeff Gadsden

Abbreviations

Cm	Centimeters
IPACK	Interspace between the popliteal artery and the capsule of the knee
PACU	Postanesthesia care unit
PECS	Pectoral nerve block
QL	Quadratus lumborum
TAP	Transversus abdominis plane
TKA	Total knee arthroplasty
TTP	Transversus thoracic muscle plane

Introduction

Regional anesthesia is a rapidly progressive subspecialty with new and continually evolving approaches to peripheral nerve blockade. The introduction of ultrasound has particularly revolutionized and renewed this area. This chapter aims to highlight several novel trends in regional anesthesia including the growing use of fascial plane blocks as well as other peripheral nerve blocks that have been made possible through the use of ultrasound, the advent of long-acting local anesthetics, and innovative ergonomics.

Fascial Plane Blocks

Peripheral nerve blocks were previously defined by targeting a specific nerve or plexus. Fascial plane blocks involve the deposition of local anesthetic within an intermuscular or other fascial plane without identifying a specific nerve or plexus. Instead, the injectate spreads within this plane in a thin layer to reach the intended small peripheral nerves [1]. As a result, the effectiveness of plane blocks relies upon the spread and thus the volume of the local anesthetic. When local anesthetic spread is inconsistent, intramuscular, or confined to a limited "bolus" rather than widespread flooding of the plane, plane blocks tend to fail. For example, the transversus abdominis plane (TAP) block is a widely used plane block for analgesia after abdom-

Electronic supplementary material Supplementary material is available in the online version of this chapter at https://doi.org/10.1007/978-3-319-74838-2_39. Videos can also be accessed at http://www.springerimages.com/videos/978-3-319-74837-5.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2_39

inal procedures. Cutaneous sensory block spread after TAP block in healthy volunteers has been shown to be non-dermatomal with significant inter-subject variability [2]. Many of these blocks were originally performed through landmark technique, utilizing tactile feedback as an indicator of correct placement, which may have further limited the efficacy in non-expert hands. The use of ultrasound has made these plane blocks more feasible for all anesthesia practitioners [3]. Several examples of novel plane blocks are described below.

Quadratus Lumborum Block

The quadratus lumborum (QL) block was initially described as a posterior approach of the TAP block by Blanco in 2007 [4]. The quadratus lumborum muscle forms part of the posterior abdominal wall with insertion sites on the iliac crest and 12th rib, as well as the transverse processes of the upper four lumbar vertebrae. The ventral rami of the T12–L5 spinal nerve roots pass between the quadratus lumborum and the fascia transversalis before passing through the aponeurosis of the transversus abdominis muscle to lie in the transversus abdominis plane.

Due to the more posterior approach with potential spread to the paravertebral space, this block may provide visceral in addition to somatic pain relief of the upper and lower abdomen, although the mechanism of this reported effect remains unclear. One cadaveric study has shown that rostral spread within the paravertebral space may lead to blockade of the mid-thoracic spinal nerves [5]. The QL block appears to cover the T6 to L1 dermatomes with a prolonged duration of analgesia compared to TAP blocks [6]. Thus the QL block may be an ideal analgesic technique for abdominal procedures that requires fewer needle insertions compared to a combination of posterior TAP and subcostal TAP injections to achieve similar dermatomal coverage [7]. Since its initial description, several novel approaches to this block have been described (Fig. 39.1).

The QL type 1 block involves deposition of local anesthetic in the anterolateral border of the quadratus lumborum muscle at its junction with the transversalis fascia. Due to its close proximity to the peritoneum, Blanco et al. described an alternative approach that they reasoned may confer less risk of peritoneal puncture, termed the QL2 block [8]. This approach has a similar needle insertion site as a the QL type 1 block, but local anesthetic is deposited between the quadratus lumborum muscle and the erector spinae, latissimus dorsi, and the serratus posterior fascial layers. The QL type 3 block, also known as the transmuscular approach, is performed by depositing local anesthetic between the fascial layers of the quadratus lumborum and the psoas major muscles, with a posterior needle insertion site adjacent to the spinal midline [9]. There is no literature to date comparing the safety of the various approaches to the QL block.

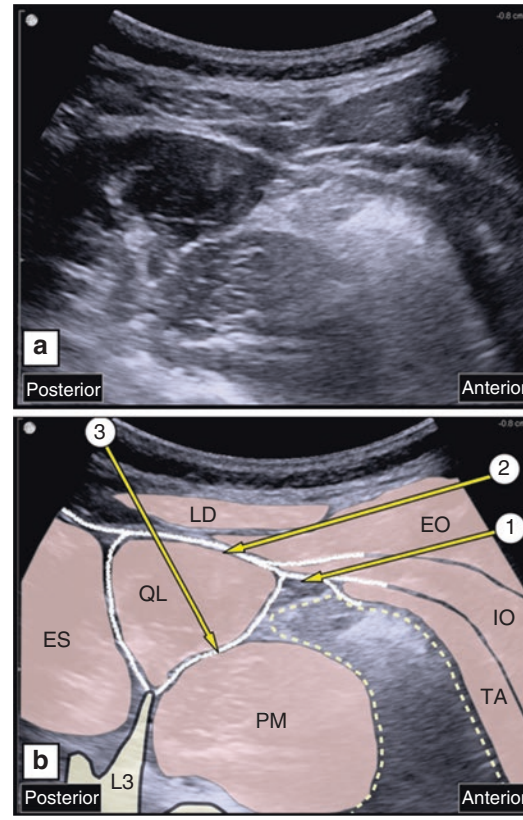

Fig. 39.1 Sonoanatomy of the QL block (**a** = unlabeled, **b** = labeled). *ES* erector spinae, *EO* external oblique muscle, *IO* internal oblique muscle, *L3* L3 transverse process, *LD* latissimus dorsi muscle, *PM* psoas major muscle, *QL* quadratus lumborum muscle, *TA* transversus abdominis muscle. Dotted line represents boundaries of the abdominopelvic cavity. Numbers represent the approaches to the QL block. 1, QL1; 2, QL2; 3, transmuscular or QL3 block

The QL block has been found to be more effective in reducing postoperative opioid consumption as compared to TAP block after cesarean delivery [8]. However there have been limited randomized controlled trials to assess this block's clinical efficacy. There have been no reported complications of quadratus lumborum blocks, but nearby structures may be susceptible to injury, including possible renal or intra-abdominal trauma. Vascular injury to the abdominal branches of the lumbar arteries is also possible.

Pectoral and Serratus Plane Blocks

Pectoral nerve blocks (PECS) are performed by infiltrating local anesthetic between the muscles of the thoracic wall. This technique was first described by Blanco in 2011 and has gained popularity as a method for chest wall analgesia when coagulopathy or hemodynamic instability may preclude the use of paravertebral or neuraxial techniques [10].

The PECS I block is a superficial plane block that is performed between the pectoralis major and pectoralis minor muscles at the level of the third rib [10]. This technique targets the medial and lateral pectoral nerves, both of which arise from the brachial plexus. The lateral pectoral nerve supplies innervation to the clavicular head of the pectoralis major muscle, acromioclavicular joint, subacromial bursa, periosteum of the clavicle, anterior articular capsule of the shoulder joint, and the costoclavicular ligaments, while the medial pectoral nerve supplies the pectoralis minor muscle, inferolateral part of the pectoralis major, and ventral aspects of the arm and chest wall, in addition to the intercostobrachial nerve [1]. Since these nerves primarily innervate chest wall musculature, the PECS I block is well suited for procedures such as submuscular breast implant or tissue expander placement.

The modified PECS block, or PECS II, involves infiltration of local anesthetic between the pectoralis minor and serratus anterior muscles at the level of the third or fourth rib, with the aim of spreading local anesthetic to the third to sixth intercostal nerves, long thoracic nerve, and thoracodorsal nerve [11]. This plane block provides anesthesia and analgesia to the T2 to T4 dermatomes and is better suited for wide breast excisions including mastectomies with or without axillary dissections.

Bashandy et al. showed that a combined PECS block resulted in significantly decreased pain scores, decreased opioid consumption in the intraoperative and postoperative period, and shorter PACU and hospital lengths of stay when compared to control [12]. Another randomized controlled trial by Kulhari et al. found that the combined PECS block resulted in significantly prolonged duration of analgesia as well as decreased opioid consumption when compared to a single-injection paravertebral block at T3 [13]. However, PECS II block, also referred to as pectoralis-serratus interfascial plane block, was shown to be inferior with regard to pain scores and opiate consumption when compared to multilevel paravertebral injection [14].

There have been no reported complications of PECS block to date, but pneumothorax and injury to vessels such as the thoracodorsal and thoracoacromial arteries are possible.

The serratus plane block was first described by Blanco in 2013 as a technique to provide complete paresthesia of the hemithorax [15]. This block involves the deposition of local anesthetic superficial or deep to the serratus anterior muscle at the level of the fifth rib in the midaxillary line. This approach also targets intercostal nerves in addition to the thoracodorsal and long thoracic nerves to provide sensory coverage of the T2 to T9 dermatomes. The serratus plane block is performed at the midaxillary line where the serratus plane is more superficial and easier to identify compared to the PECS II block.

A retrospective review showed that the serratus plane block provides effective regional anesthesia in breast surgery and seems to provide superior analgesia compared to wound infiltration [16]. Serratus plane blocks have also been reported to be effective for pain control after rib fractures, video-assisted thoracic surgery, implantable cardioverter-defibrillator insertion, and transaxillary robotic thyroidectomy [17–19].

Erector Spinae Plane Block

Erector spinae plane blocks are another option for planned or rescue analgesia of the thoracic and abdominal regions. Erector spinae blocks performed at the T5 transverse process have been used for thoracic analgesia with coverage of T2 to T8 vertebral levels [20]. Erector spinae blocks performed at T7 have provided adequate postoperative abdominal analgesia with coverage of T7 to T11 dermatomes [21]. These blocks have also been used successfully to treat pain associated with rib fractures [22]. Cadaveric studies have shown contrast spread from C5 to L2 after a single injection of 20 mL in the erector spinae plane at T7 as well as spread into the intercostal spaces [20]. After identifying the targeted level, a linear transducer is placed in a parasagittal orientation to identify the transverse process. Local anesthetic is injected between the erector spinae muscle and transverse process. The erector spinae block may provide an additional safety margin compared to a paravertebral injection given the easily identifiable landmarks as well as greater distance from pleura and neurovascular bundle [23].

Other Novel Blocks

The introduction of portable ultrasound machines has advanced the field of regional anesthesiology. Prior to the routine use of ultrasound, many peripheral nerve blocks could be accomplished through landmark or nerve stimulator techniques. However the advent of ultrasound-guided regional anesthesia has opened the doors to novel blocks that may not have been successfully achieved otherwise.

Parasternal Block

One such block that is now possible due to the safety margin provided by ultrasound visualization is the parasternal block. Targeting the anterior branches of the intercostal nerves, this particular block goes by many names and has many variations in approach [24, 25]. In 2005, McDonald et al. first described a parasternal block, which included a series of intercostal blocks just lateral to the sternal border in addition to a continuous line of local infiltration just above the periosteum along the lateral borders of the sternum [26]. In their study of 17 cardiac surgery patients, those who received the parasternal block had decreased opioid consumption and improved oxygenation at time of extubation. De la Torre et al. described the pecto-intercostal fascial block in 2014, which targets the area between the pectoralis major and external intercostal muscles 2 cm lateral to the sternum [27]. Similarly, Raza et al. described subpectoral interfascial plane catheters placed between the pectoralis major muscle and external intercostal muscle aponeurosis at a point 2 cm lateral to the sternal edge for a patient with sternal fractures [28]. In 2016, Hansen et al. described the parasternal PECS block, performed between the pectoralis major muscle and the sternum 3 cm below the jugular notch [29]. Ueshima et al. described the transversus thoracic muscle plane (TTP) block, which involves injection of local anesthetic between the transversus thoracic muscle and internal intercostal muscle between the third and fourth ribs at their connection with the sternum [30]. In case reports, the TTP block has been shown to be effective for median sternotomy, breast cancer resection, and implantable cardioverter-defibrillator implantation [31–33].

IPACK Block

The IPACK (interspace between the popliteal artery and capsule of the posterior knee) block is an ultrasound-guided technique for local anesthetic infiltration to provide posterior knee analgesia, most commonly performed for total knee arthroplasty (TKA). The block targets the genicular branches of the sciatic nerve to assist with analgesia while not contributing to motor block as it spares the main trunks of the tibial and peroneal nerves. An ultrasound probe is placed in the popliteal fossa to identify the femoral condyles (Fig. 39.2). The probe is then

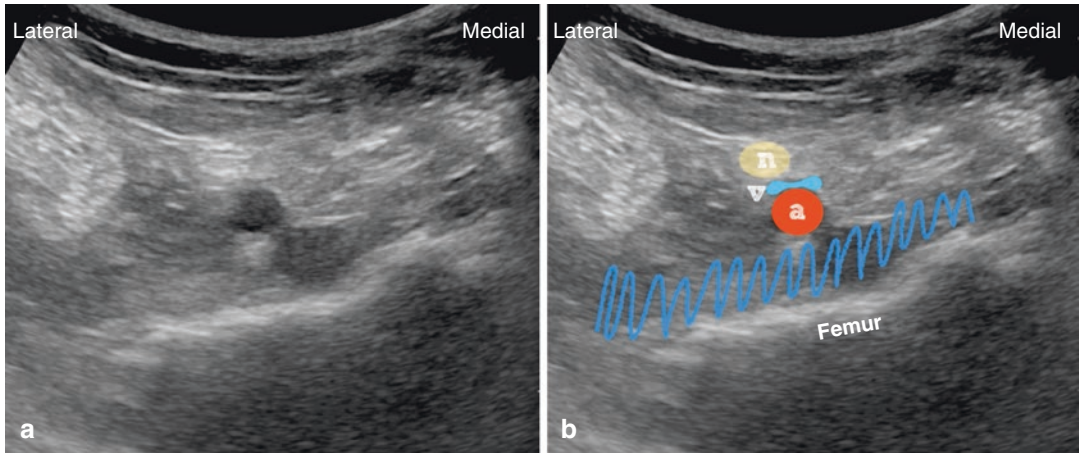

Fig. 39.2 Sonographic image (**a** = unlabeled, **b** = labeled) during the IPACK block. a, popliteal artery; n, tibial nerve; v, popliteal vein. The shaded area between the popliteal artery and vein represents the area to be infiltrated with 20–30 mL of local anesthetic

moved slightly cephalad to visualize the flat surface of the femur. The block is then inserted in-plane between the popliteal artery and the femur shaft, and 20–30 mL of dilute local anesthetic is infiltrated. A retrospective study showed that IPACK in combination with femoral catheter resulted in significantly decreased postoperative opioid consumption after TKA compared to patients who received a femoral catheter alone [34]. However this study did not find any difference in pain scores or physical therapy performance.

Long-Acting Anesthetics

There has been a great deal of recent investigation into long-acting local anesthetics, which may be able to extend the analgesic duration of local infiltration and peripheral nerve blocks. Should there be an approved and efficacious long-acting or extended-release local anesthetic, it may supplant the routine use of peripheral nerve catheters. Catheters, while effective for ongoing analgesia, can be associated with possible complications including the risk of infection, intravascular catheter migration, or catheter displacement requiring replacement or poor analgesia. Additionally, catheters in both inpatients and outpatients require close follow-up, which can prove to be a substantial time commitment for healthcare personnel.

Liposomal bupivacaine (Exparel®, Pacira Pharmaceuticals, Parsippany, NJ) is an extended-release, lipid-encapsulated form of bupivacaine that is designed to prolong the local anesthetic effect. Bupivacaine HCl is suspended in billions of phospholipid and cholesterol liposomes that, once deposited inside the tissue, degrade over a predictable time frame, typically 48–72 h [35]. The degraded lipid framework is metabolized by the liver and does not play an active role in the drug's effect. Several studies have shown that liposomal bupivacaine can result in significantly decreased pain the first 72 h postoperatively with reduced opioid requirements compared to placebo in hemorrhoidectomy, bunionectomy, local infiltration of the knee, and other surgical site infiltration [36–39]. However, the use of liposomal bupivacaine has led to unreliable dose-response relationships in femoral nerve blocks. Furthermore, the use of liposomal bupivacaine in the epidural space has shown limited prolongation of sensory effects [40]. Thus, its use is currently approved for surgical site infiltration, but further evaluation is necessary prior to approval in peripheral or neuraxial blocks. Liposomal bupivacaine has been used effectively in TAP blocks to prolong the duration of action of the abdominal wall sensory blockade. Studies in both

laparoscopic hand-assisted nephrectomy as well as robotic-assisted hysterectomy demonstrate superior analgesia and significant reductions in opioid use up to 72 h postoperatively [41, 42].

SABER®-bupivacaine (Posimir®, DURECT, Cupertino, CA) is an investigational extended-release local anesthetic that is similarly designed to relieve pain for up to 3 days after injection through its slow-release formulation. SABER® (sucrose acetate isobutyrate extended release) is a proprietary biodegradable water-insoluble matrix that can accommodate active drug (i.e., bupivacaine) at high concentrations and release it over 72 h following injection into the surgical site. Early clinical trials have shown SABER®-bupivacaine to be safe and effective in decreasing opioid requirements compared to placebo, but further studies are needed to elucidate its efficacy [43]. At the time of writing, SABER®-bupivacaine is in phase 3 clinical trial status.

Clinical Case Study

A 42-year-old female with chronic pain and a history of postoperative nausea and vomiting is scheduled for a laparoscopic hysterectomy. She receives bilateral TAP blocks with 0.2% ropivacaine after induction. The case is uncomplicated, and the patient is extubated and transported to the postanesthesia care unit (PACU). After 30 min, the patient complains of increasing abdominal pain, described as incisional pain at the port sites, as well as visceral discomfort. Unfortunately the pain does not respond to escalating doses of intravenous narcotics.

Discussion

Is there another appropriate regional analgesic option?

The quadratus lumborum (QL) block is another plane block that is performed further posterior than the TAP block and may provide both somatic and visceral pain relief. In order to prolong the analgesic effect, a long-acting local anesthetic such as liposomal bupivacaine may be used to extend the duration of analgesia. Liposomal bupivacaine is best combined with plain bupivacaine prior to injection in order to optimize onset while preserving the duration. One consideration that must be taken into account is that liposomal bupivacaine should not be co-administered with other amide local anesthetics, such as ropivacaine. This has been shown to increase the risk of premature breakdown of the liposomes, thereby releasing free bupivacaine into the tissue in an unexpectedly high dose. The choice of QL technique may be influenced by this—for example, liposomal bupivacaine administered via a QL2 or transmuscular QL would be far less likely to be in the same plane as ropivacaine delivered via a traditional lateral TAP block. Alternatively, an erector spinae plane block performed at T7 has been shown to provide somatic and visceral analgesia for some abdominal procedures. This could be performed using liposomal bupivacaine as well, as the tissue plane is quite remote from the TAP site.

Innovative Ergonomics

Regional anesthesia requires precision and procedural acumen to provide safe and efficient patient care. Ergonomics is the study of product, system, or process design intended to make the interaction between humans and their work environment more efficient. Several ergonomic techniques have been described to allow the provider to perform independently and to confirm continuous peripheral nerve catheter location.

The hand-on-syringe technique is a method for block placement where the syringe with saline or local anesthetic is connected directly to the block needle. The operator then holds the syringe when advancing the needle. This of course is not a novel technique, but is one that almost all but the most gray-haired of clinicians will remember. Almost 50 years after Winnie described and popularized the "immobile needle" [44], wherein a block needle is attached to the syringe via a length of flexible tubing, some practitioners are returning to the needle-syringe method, facilitated nowadays with the use of ultrasound.

The hand-on-syringe technique has the advantage of allowing a single operator to perform a block without assistance, which may be helpful in urgent situations or scenarios with limited staff, such as overnight block placement. In a study of emergency medicine trainees, the hand-on-syringe technique was shown to be comparable to the traditional hand-on-needle technique with respect to time to complete a block, needle visualization, or needle tip accuracy [45]. A possible safety advantage of the hand-on-syringe technique is that it allows the primary operator to experience the tactile feedback of resistance to injection, thus providing the potential to identify a possible intraneural injection.

A variation of this is the so-called Jedi grip, where the operator grasps the needle and tubing between the middle phalanges of his/her second and third fingers, while palming the syringe at a 90° angle (Fig. 39.3) [46]. This allows the operator to advance the needle, aspirate, and inject all with one hand. The group that innovated this approach cites a decreased tendency to lateral misalignment when attempting to guide the needle in-plane, although formal studies of this are lacking. Another purported advantage to a traditional two-person technique is the instantaneous tactile and visual feedback—since the operator knows exactly when and what volume of injectate is about to be administered, more precise delivery of local anesthetic with overall reduced volumes may result since less is "wasted" waiting/watching for the expansion on the ultrasound screen after the second injector has administered a certain volume.

Fig. 39.3 The Jedi grip. The operator holds both the needle and the syringe, eliminating the need for a second operator to inject

Ergonomics is critically important during continuous peripheral nerve block placement. Typically, the catheter is typically threaded through the needle once the operator confirms appropriate needle tip location. The operator often will thread the catheter through the needle blindly unless they have an assistant to hold the ultrasound probe. It is possible for a single operator to visualize the catheter leaving the tip of the needle in real time by holding the hub of the needle between the fourth and fifth fingers while advancing the catheter with the index finger and thumb. The ultrasound probe is held with the other hand (Video 39.1). This technique may be easier with stiffer nerve block catheters that facilitate threading past the end of the needle tip.

One problematic aspect of performing ultrasound-guided nerve blocks is maintaining needle-probe alignment while watching the screen image. Head-mounted display technology (MicroOptical CV-3, MyVu, Wellesley, MA) may provide a means for continuous real-time ultrasound imaging within the anesthesiologist's visual field allowing for optimization of needle, probe, and hand alignment [47]. While the use of this novel technology may come with an initial learning curve, this may alleviate significant limitations that are often present in cramped workspaces that prohibit ideal positioning of an ultrasound machine.

Review Questions

1. The goal of the transmuscular quadratus lumborum block (also known as QL3) is to deposit local anesthetic between:
   (a) Latissimus dorsi and erector spinae
   (b) Quadratus lumborum and erector spinae
   (c) Quadratus lumborum and psoas major
   (d) Quadratus lumborum and latissimus dorsi

2. The serratus plane block is intended to provide truncal coverage of which set of dermatomes:
   (a) T2–T4
   (b) T2–T6
   (c) T4–T10
   (d) T2–T9
3. Complications related to the PECS II block may include:
   (a) Intravascular injection
   (b) Bilateral spread
   (c) High spinal anesthesia
   (d) All of the above

Answers

1. c
2. c
3. a

References

1. Chakraborty A, Khemka R, Datta T. Ultrasound-guided truncal blocks: a new frontier in regional anaesthesia. Indian J Anaesth. 2016;60:703–11.
2. Støving K, Rothe C, Rosenstock CV, Aasvang EK, Lundstrøm LH, Lange KHW. Cutaneous sensory block area, muscle-relaxing effect, and block duration of the transversus abdominis plane block: a randomized, blinded, and placebo-controlled study in healthy volunteers. Reg Anesth Pain Med. 2015;40:355–62.
3. Gadsden J, Ayad S, Gonzales JJ, Mehta J, Boublik J, Hutchins J. Evolution of transversus abdominis plane infiltration techniques for postsurgical analgesia following abdominal surgeries. Local Reg Anesth. 2015;8:113–7.
4. Blanco R. Tap block under ultrasound guidance: the description of a "no pops" technique. Reg Anesth Pain Med. 2007;32:S1–130.
5. Dam M, Moriggl B, Hansen CK, Hoermann R, Bendtsen TF, Børglum J. The pathway of injectate spread with the transmuscular quadratus lumborum block: a cadaver study. Anesth Analg. 2017;125(1):303–12.
6. Murouchi T, Iwasaki S, Yamakage M. Quadratus lumborum block: analgesic effects and chronological ropivacaine concentrations after laparoscopic surgery. Reg Anesth Pain Med. 2016;41:146–50.
7. Chin KJ, McDonnell JG, Carvalho B, Sharkey A, Pawa A, Gadsden J. Essentials of our current understanding: abdominal wall blocks. Reg Anesth Pain Med. 2017;42:133–83.
8. Blanco R, Ansari T, Girgis E. Quadratus lumborum block for postoperative pain after caesarean section: a randomised controlled trial. Eur J Anaesthesiol. 2015;32:812–8.
9. Børglum J, Moriggl B, Jensen K, Lönnqvist PA, Christensen AF, Sauter AR, et al. Ultrasound-guided transmuscular quadratus lumborum blockade 2013. https://doi.org/10.1093/bja/el_9919
10. Blanco R. The "pecs block": a novel technique for providing analgesia after breast surgery. Anaesthesia. 2011;66:847–8.
11. Blanco R, Fajardo M, Parras Maldonado T. Ultrasound description of Pecs II (modified Pecs I): a novel approach to breast surgery. Rev Esp Anestesiol Reanim. 2012;59:470–5.
12. Bashandy GMN, Abbas DN. Pectoral nerves I and II blocks in multimodal analgesia for breast cancer surgery: a randomized clinical trial. Reg Anesth Pain Med. 2015;40:68–74.
13. Kulhari S, Bharti N, Bala I, Arora S, Singh G. Efficacy of pectoral nerve block versus thoracic paravertebral block for postoperative analgesia after radical mastectomy: a randomized controlled trial. Br J Anaesth. 2016;117:382–6.
14. Hetta DF, Rezk KM. Pectoralis-serratus interfascial plane block vs thoracic paravertebral block for unilateral radical mastectomy with axillary evacuation. J Clin Anesth. 2016;34:91–7.
15. Blanco R, Parras T, McDonnell JG, Prats-Galino A. Serratus plane block: a novel ultrasound-guided thoracic wall nerve block. Anaesthesia. 2013;68:1107–13.
16. Hards M, Harada A, Neville I, Harwell S, Babar M, Ravalia A, et al. The effect of serratus plane block performed under direct vision on postoperative pain in breast surgery. J Clin Anesth. 2016;34:427–31.
17. Durant E, Dixon B, Luftig J, Mantuani D, Herring A. Ultrasound-guided serratus plane block for ED rib fracture pain control. Am J Emerg Med. 2017;35:197.e3–6.
18. Corso RM, Maitan S, Russotto V, Gregoretti C. Type I and II pectoral nerve blocks with serratus plane block for awake video-assisted thoracic surgery. Anaesth Intensive Care. 2016;44:643–4.
19. Ueshima H, Hara E, Otake H. Successful cases of S-ICD implantation performed under the serratus plane block. J Clin Anesth. 2016;33:147–8.
20. Forero M, Adhikary SD, Lopez H, Tsui C, Chin KJ. The erector spinae plane block: a novel analgesic technique in thoracic neuropathic pain. Reg Anesth Pain Med. 2016;41:621–7.
21. Chin KJ, Adhikary S, Sarwani N, Forero M. The analgesic efficacy of pre-operative bilateral erector spinae plane (ESP) blocks in patients having ventral hernia repair. Anaesthesia. 2017;72:452–60.
22. Hamilton DL, Manickam B. Erector spinae plane block for pain relief in rib fractures. Br J Anaesth. 2017;118:474–5.
23. El-Boghdadly K, Pawa A. The erector spinae plane block: plane and simple. Anaesthesia. 2017;72:434–8.

24. Del Buono R, Costa F, Agrò FE. Parasternal, pectointercostal, Pecs, and transverse thoracic muscle plane blocks: a rose by any other name would smell as sweet. Reg Anesth Pain Med. 2016;41:791–2.
25. Ohgoshi Y, Ino K, Matsukawa M. Ultrasound-guided parasternal intercostal nerve block. J Anesth. 2016;30:916.
26. McDonald SB, Jacobsohn E, Kopacz DJ, Desphande S, Helman JD, Salinas F, et al. Parasternal block and local anesthetic infiltration with levobupivacaine after cardiac surgery with desflurane: the effect on postoperative pain, pulmonary function, and tracheal extubation times. Anesth Analg. 2005;100:25–32.
27. De la Torre PA, García PD, Alvarez SL, Miguel FJG, Pérez MF. A novel ultrasound-guided block: a promising alternative for breast analgesia. Aesthet Surg J. 2014;34:198–200.
28. Raza I, Narayanan M, Venkataraju A, Ciocarlan A. Bilateral subpectoral interfascial plane catheters for analgesia for sternal fractures: a case report. Reg Anesth Pain Med. 2016;41:607–9.
29. Hansen CK, Dam M, Poulsen TD, Lönnqvist PA, Bendtsen T, Børglum J. Ultrasound-guided parasternal pecs block: a new and useful supplement to Pecs I and serratus anterior plane blocks. Anaesthesia Cases. 2016; Published online at: https://www.anaesthesia-cases.org/case-reports/2016-0007
30. Ueshima H, Kitamura A. Blocking of multiple anterior branches of intercostal nerves (Th2-6) using a transversus thoracic muscle plane block. Reg Anesth Pain Med. 2015;40:388.
31. Ueshima H, Hara E, Marui T, Otake H. The ultrasound-guided transversus thoracic muscle plane block is effective for the median sternotomy. J Clin Anesth. 2016;29:83.
32. Ueshima H, Kimura S, Otake H. Bilateral breast cancer resection performed under the bilateral transversus thoracic muscle plane block. J Clin Anesth. 2016;33:413.
33. Ueshima H, Hiroshi O. A successful case of subcutaneous implantable cardioverter-defibrillator implantation performed under the transversus thoracic muscle plane block. J Clin Anesth. 2016;32:253–4.
34. Thobhani S, Thomas L, Osteen K, Nossaman B, Soberon J, Edelman J, et al. Effectiveness of local anesthetic infiltration between popliteal artery and capsule of knee (iPACK) for attenuation of knee pain in patients undergoing total knee arthroplasty. Abstract presented at the American Society of Regional Anesthesia and Pain Medicine 40th Annual Spring Meeting, Las Vegas, NV, 2015.
35. Gadsden J, Long WJ. Time to analgesia onset and pharmacokinetics after separate and combined administration of liposome bupivacaine and bupivacaine HCl: considerations for clinicians. Open Orthop J. 2016;10:94–104.
36. Gorfine SR, Onel E, Patou G, Krivokapic ZV. Bupivacaine extended-release liposome injection for prolonged postsurgical analgesia in patients undergoing hemorrhoidectomy: a multicenter, randomized, double-blind, placebo-controlled trial. Dis Colon Rectum. 2011;54:1552–9.
37. Dasta J, Ramamoorthy S, Patou G, Sinatra R. Bupivacaine liposome injectable suspension compared with bupivacaine HCl for the reduction of opioid burden in the postsurgical setting. Curr Med Res Opin. 2012;28:1609–15.
38. Golf M, Daniels SE, Onel E. A phase 3, randomized, placebo-controlled trial of DepoFoam® bupivacaine (extended-release bupivacaine local analgesic) in bunionectomy. Adv Ther. 2011;28:776–88.
39. Wu ZQ, Min JK, Wang D, Yuan YJ, Li H. Liposome bupivacaine for pain control after total knee arthroplasty: a meta-analysis. J Orthop Surg Res. 2016;11:84.
40. Tong YCI, Kaye AD, Urman RD. Liposomal bupivacaine and clinical outcomes. Best Pract Res Clin Anaesthesiol. 2014;28:15–27.
41. Hutchins J, Delaney D, Vogel RI, Ghebre RG, Downs LS, Carson L, et al. Ultrasound guided subcostal transversus abdominis plane (TAP) infiltration with liposomal bupivacaine for patients undergoing robotic assisted hysterectomy: a prospective randomized controlled study. Gynecol Oncol. 2015;138:609–13.
42. Hutchins JL, Kesha R, Blanco F, Dunn T, Hochhalter R. Ultrasound-guided subcostal transversus abdominis plane blocks with liposomal bupivacaine vs. non-liposomal bupivacaine for postoperative pain control after laparoscopic hand-assisted donor nephrectomy: a prospective randomised observer-blinded study. Anaesthesia. 2016;71:930–7.
43. Skolnik A, Gan TJ. New formulations of bupivacaine for the treatment of postoperative pain: liposomal bupivacaine and SABER-Bupivacaine. Expert Opin Pharmacother. 2014;15:1535–42.
44. Winnie AP. An "immobile needle" for nerve blocks. Anesthesiology. 1969;31:577–8.
45. Johnson B, Herring A, Stone M, Nagdev A. Performance accuracy of hand-on-needle versus hand-on-syringe technique for ultrasound-guided regional anesthesia simulation for emergency medicine residents. West J Emerg Med. 2014;15(6):641.
46. Pappin D, Christie I. The Jedi Grip: a novel technique for administering local anaesthetic in ultrasound-guided regional anaesthesia. Anaesthesia. 2011;66:845.
47. Udani AD, Harrison TK, Howard SK, Kim TE, Brock-Utne JG, Gaba DM, et al. Preliminary study of ergonomic behavior during simulated ultrasound-guided regional anesthesia using a head-mounted display. J Ultrasound Med. 2012;31:1277–80.

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A. D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
https://doi.org/10.1007/978-3-319-74838-2

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