Danilo Jankovic
Philip Peng

Regional Nerve Blocks in Anesthesia and Pain Therapy

Traditional and Ultrasound-Guided Techniques

Fourth Edition

Springer

Regional Nerve Blocks in Anesthesia and Pain Therapy

Danilo Jankovic • Philip Peng

Regional Nerve Blocks in Anesthesia and Pain Therapy

Traditional and Ultrasound-Guided Techniques

Fourth Edition

Springer

Danilo Jankovic
Pain Management Center
Cologne-Hürth
Nordrhein-Westfalen
Germany

Philip Peng
Department of Anesthesia
Toronto Western Hospital
University of Toronto
Toronto
Ontario
Canada

ISBN 978-3-319-05130-7 ISBN 978-3-319-05131-4 (eBook)
DOI 10.1007/978-3-319-05131-4

Library of Congress Control Number: 2015945349

Springer Cham Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

I dedicate this book to my wife Lydia, and my children Lara and Aleks.
Their love, support, and encouragement have made this book possible.

Danilo Jankovic

This book is dedicated to my wife, Carol, for her continued support,
encouragement and understanding; to my children, Julia and Michael,
who fill me with joy and love; and to my sister, Rita, who keeps
reminding me to be strong and assertive.
Without them, this book would be impossible.

Philip Peng

Preface

In recent years, the field of regional anesthesia, and in particular peripheral and neuraxial nerve blockade, has entered an unprecedented renaissance. This renaissance is due primarily to the widespread introduction of ultrasound-guided regional anesthesia. The ability to visualise the anatomy of interest, the needle-nerve relationship, and the spread of the local anesthetic has resulted in significant growth of interest in the use of peripheral and neuraxial nerve blocks. Although ultrasound guidance eventually may become the most prevalent method of nerve blockade, most procedures world-wide are still performed using the methods of peripheral nerve stimulation and/or surface landmarks, particularly in the developing world. Because this book has been one of the important teaching sources internationally, we decided to retain the section on the traditional techniques of nerve blockade in addition to the new section on ultrasound-guided regional anesthesia.

The book contains precise anatomical drawings, illustrations and self-made native anatomical preparations in full-colour throughout, and also provides detailed instructions on how to apply regional anesthesia. The descriptions of anatomy and sonoanatomy are highly relevant to the regional blocks and the clear illustrations helps to better understanding of each block.

We made every effort that the overall style of presentation is methodical, thorough and precise. The description of each block is broken down into headings: definition; anatomy; indications; contraindications; technique (ultrasound-guided and traditional); drug choice and dosage; side effects; complications (and how to avoid them or treat them) and medicolegal documentation. The information given in a checklist record for each technique helps in proper documentation of the performed nerve blocks. The book focuses on each area of the body, describes its anatomy and sonoanatomy, and then explains the needed supplies for each nerve block and the details of how to do it.

This book is practically oriented; the book could almost be taken to the operating room and used as a guide. Topics are consistently organized, very detailed and simple to read. This book is intended for practicing anesthesiologists and all specialties engaged in the field of pain therapy (such as pain specialists, general surgeons, orthopaedic surgeons, neurosurgeons, neurologists, and general practitioners).

The book comprises 73 chapters, organized in 12 sections, covering US-guided and traditional nerve blocks in anesthesia and interventional pain management:

Part I reviews the General Considerations (Use of Local Anesthetics in Regional Anesthesia and Pain Management, Basics of Ultrasound Imaging, Use of Nerve Stimulation and Stimulating Catheters in the Ultrasound Era, and Complications of Peripheral and Neuraxial Nerve Blocks).

Part II covers the Head and Neck Region (Regional Anesthesia in Ophthalmology, Facial Nerve Block, Anesthesia of the Airways, Phrenic Nerve Block, Trigeminal Nerve Block (classic and neurodestructive procedures), Occipital Nerves Block, Stellate Ganglion Block, Superior Cervical Ganglion Block, and Deep and Superficial Cervical Plexus Block).

Part III focuses on Blocks in Cervical Region (Cervical Interlaminar Epidural Block, Cervical Facet Nerve Blocks (Medial Branch and Third Occipital Nerve), (Pulsed) Radiofrequency

Treatment Adjacent to the Cervical Dorsal Root Ganglion, and Cervical Percutaneous Facet Denervation).

Part IV reviews the Shoulder Region (Suprascapular Nerve Block, Glenohumeral and Acromioclavicular Joints, Subacromial Subdeltoid Bursa, Long Head of Biceps Tendon, Treatment of Calcific Tendinitis, and Rotator Muscles and Subscapular Nerve Injections).

Part V covers the detailed Blocks of the Upper Extremity (Brachial Plexus and Intravenous Regional Anesthesia).

Part VI reviews region of the Elbow and Wrist.

Part VII focuses on the Thoracic Region (Thoracic Paravertebral Block, Intercostal Nerve Block).

Part VIII reviews Lumbosacral Spine (Neuraxial Blocks, Neuraxial Analgesia in Obstetrics, Caudal Epidural Injections, Lumbar Sympathetic Blocks, Lumbar Facet Joint and Nerve Injection, Lumbar Percutaneous Facet Denervation, Sacroiliac Joint Injection, Sacral Nerve Root Block, Lumbosacral Epiduroscopy, and Percutaneous Epidural Neuroplasty).

Part IX focuses on Abdominal and Pelvic Region (Celiac Plexus Block, Nerve Blocks of the Abdominal Wall, Ilioinguinal, Iliohypogastric and Genitofemoral Nerve Blocks, Injection for Piriformis Syndrome, Pudendal Nerve Block, Superior Hypogastric and Ganglion Impar Block, Paracervical Block).

Part X reviews detailed Lower Extremity Blocks.

Part XI covers Lower Extremity Musculoskeletal Injections (common Joint and Bursa Injections).

Part XII reviews detailed Regional Blocks for Children (Upper and Lower Limb and Trunk and Neuraxial Blocks).

This book owes a great deal to a number of well-recognized clinicians, academicians and regional anesthesia teams from around the globe. Many thanks to numerous international collaborators, who supported this project.

We would like to express our special thanks to colleagues and friends who have contributed to this edition:

Ban C.H. Tsui, Michael Barrington, James Heavner, Vincent Chan, Ki Jinn Chin, Andrew Gray, Andre'van Zundert, Olaf Rohof, Robert Jan Stolker, Stephen Halpern, Jens Kessler, Jan van Zundert, Admir Hadzic, Andreas Siegenthaler, Chin-Wern Chan, Daquan Xu, Peter Cheng, Chandra Kumar, Hariharan Shankar, Jeffrey Ghassemi, Antony Hade, Herman Sehmbi, Anahi Perlas, Gloria Seah, John Hanlon, Faraj Abdallah, Geoff Bellingham, Paul Gray, Sara Cheung, Heather Ting, Dario Bugada, Paul Tumber, Steven Renes, Santine van den Heuvel, Geert-Jan van Geffen, Derek Dillane, Anuj Bhatia, Hemmo Bosscher, Gabor Racz, Miles Day, Battista Borghi and Amit Pawa.

We express our sincere thanks to all Springer editorial staff for their expertise and help in editing this book and their constant support and expert assistance.

Cologne-Hürth, Nordrhein-Westfalen, Germany — Danilo Jankovic
Toronto, ON, Canada — Philip Peng

Contents

Part VIII Lumbosacral Spine

Collaborators

Faraj W. Abdallah Department of Anesthesia, St. Michael's Hospital, University of Toronto, Toronto, ON, Canada

Michael J. Barrington, MBBS, FANZCA, PhD Department of Anesthesia and Acute Pain Medicine, St. Vincent's Hospital, Melbourne, Fitzroy, VIC, Australia

Faculty of Medicine, Dentistry and Health Sciences, Melbourne Medical School, University of Melbourne, Melbourne, VIC, Australia

Geoff Bellingham Department of Anesthesia and Perioperative Medicine, St. Joseph's Health Care London, University of Western Ontario, London, ON, Canada

Anuj Bhatia Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada

Battista Borghi Anesthesia Research Unit, Instituti Orthopedici Rizzoli, Bologna, Italy

Hemmo Bosscher, MD Texas Tech University Health Sciences Center, Lubbock, TX, USA

Dario Bugada, MD Department of Anesthesia and Intensive Care-Pain Therapy Service, IRCCS Foundation Policlinico S. Matteo, University of Pavia, Pavia (PV), Italy

Vincent W. Chan, MD, FRCPC Department of Anesthesia, Toronto Western Hospital, University Health Network, University of Toronto, Toronto, ON, Canada

Chin-Wern Chan Department of Pain Medicine, Sir Charles Gairdner Hospital, Nedlands, WA, Australia

Peter Cheng, MD Southern California Permanent Medical Group, Walnut, CA, USA

Sara S.K. Cheung, MBBS, FHKCA, FHKAM Department of Anesthesia, Pamela Youde Nethersole Eastern Hospital, Hong Kong, China

University of Alberta, Edmonton, AB, Canada

Ki Jinn Chin, MBBS, MMed., FANZCA, FRCPC Department of Anesthesia, Toronto Western Hospital, Toronto, ON, Canada

Miles Day, MD Texas Tech University Health Sciences Center, Lubbock, TX, USA

Derek Dillane, MB BCh, FRCA Department of Anesthesiology and Pain Medicine, University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada

P. Van Elderen, MD, FIPP Department of Anesthesiology and Multidisciplinary Pain Centre, Ziekenhuis Oost-Limburg, Genk, Belgium

Geert-Jan van Geffen Department of Anesthesiology, Pain and Palliative Medicine, Radboud University Nijmegen Medical Center, Nijmegen, The Netherlands

Jeffrey Ghassemi, MD, MPH Department of Anesthesiology & Perioperative Care, University of California, San Francisco, CA, USA

Andrew T. Gray, MD, PhD Department of Anesthesia and Perioperative Care, University of California, San Francisco General Hospital, San Francisco, CA, USA

Paul Gray Department of Anesthesia, Princess Alexandra Hospital, Woolloonabba, Australia

Gerbrand Groen Division of Perioperative Medicine, Department of Anesthesiology & Emergency Care, University Medical Centre Utrecht, Utrecht, The Netherlands

Antony Hade Faculty of Medicine, Dentistry and Health Sciences, Melbourne Medical School, University of Melbourne, Melbourne, VIC, Australia

Admir Hadzic, MD, PhD Department of Anesthesology, Ziekenhuis Oost-Limburg, Genk, Belgium

John G. Hanlon Department of Anesthesia, St. Michael's Hospital, University of Toronto, Toronto, ON, Canada

Stephen Halpern, MD, MSC, FRCPC Department of Anesthesia, Sunnybrook Health Sciences Centre, Toronto, ON, Canada

James Heavner, DVM, PhD Department of Anesthesiology, Texas Tech University Health Sciences Center, Lubbock, TX, USA

Santine A.S. van den Heuvel Department of Anesthesiology, Pain and Palliative Medicine, Radboud University Nijmegen Medical Center, Nijmegen, The Netherlands

Danilo Jankovic, MD Pain Management Centre, Colgne-Huerth, Germany

Jens Kessler, MD Department of Anesthesiology, University Hospital Heidelberg, Heidelberg, Germany

Chandra M. Kumar, FFARCS, FRCA, MSc Khoo Teck puat Hospital, Singapore, Singapore

Amit Pawa Guy's and St. Thomas' NHS Foundation Trust, London, UK

Philip W.H. Peng Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada

Anahi Perlas, MD, FRCPC Department of Anesthesia, Toronto Westarn Hospital, University of Toronto, Toronto, ON, Canada

Martine Puylaert, MD, FIPP Department of Anesthesiology and Multidisciplinary Pain Centre, Ziekenhuis Oost-Limburg, Genk, Belgium

Gabor B. Racz, MD Department of Anesthesiology, Texas Tech University Health Sciences Center, Lubbock, TX, USA

Steven H. Renes, MD, PhD, FIPP Department of Anesthesiology, Pain and Palliative Medicine, Radboud University Nijmegen Medical Center, Nijmegen, The Netherland

Olav Rohof, MD Pain Clinic, Atrium Orbis Location Sittard Geleen, Sittard Geleen, The Netherlands

Gloria J. Seah Department of Anesthesia and Acute Pain Medicine, St. Vincent's Hospital, Melbourne, VIC, Australia

Herman Sehmbi, MBBS, MD, EDAIC, EDRA Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada

Hariharan Shankar, MD Pain Clinic, Department of Anesthesiology, Clement Zablocki VA Medical Center, Milwaukee, WI, USA

Department of Anesthesiology, Medical College of Wisconsin, Milwaukee, WI, USA

Andeas Siegenthaler Department of Anesthesiology and Pain Therapy, University of Bern, Inselspital, Bern, Switzerland

Robert Jan Stolker, MD, PhD Department of Anesthesiology, Erasmus MC, Rotterdam, The Netherlands

Hans van Suijlekom Department of Anesthesiology, ICU and Pain Therapy, Catharina-Ziekenhuis, Eindhoven, The Netherlands

Heather Y.Z. Ting, MD, FRCPC University of Alberta Hospital, Edmonton, AB, Canada

Ban C.H. Tsui, MSc, MD, FRCPC Department of Anesthesiology and Pain Medicine, University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada

Paul Tumber Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada

P. De Vooght, MD Department of Anesthesiology and Multidisciplinary Pain Centre, Ziekenhuis Oost-Limburg, Genk, Belgium

Daquan Xu, MD, MSc, MPH New York School of Regional Anesthesia (NYSORA), St. Luke's – Roosevelt Hospitals, New York, NY, USA

André van Zundert, MD, PhD, FRCA, EDRA, FANZA Faculty of Medicine and Biomedical Sciences, Royal Brisbane and Women's Hospital-Ned, The University of Queensland, Brisbane, QLD, Australia

Jan van Zundert, MD, PhD, FIPP Department of Anesthesiology and Multidisciplinary Pain Management Centre, Ziekenhuis Oost-Limburg, Genk, Belgium

Department of Anesthesiology and Pain Management, University Hospital Maastricht, Maastricht, The Netherlands

About the Authors

Danilo Jankovic

Dr. Danilo Jankovic is currently Director of the Regional Pain Management Centre DGS in Cologne-Huerth. Dr. Jankovic's main areas of interest include regional nerve blocks in anesthesia and interventional pain management, regional anesthesia anatomy, treatment of pain by developing new techniques designed and published for the rapid resolution of musculoskeletal pain and dysfunction, medicolegal documentation in regional anesthesia e.g.

Dr. Jankovic is a member of numerous professional associations and has lectured extensively both nationally and internationally.

Dr. Jankovic has been awarded with Rudolf Frey Award 2000 and German Pain Association Award 2007 for his contribution in the field of Regional Anesthesia and Pain Management. Dr. Jankovic has authored the book *Regional Nerve Blocks and Infiltration Therapy* (1st–4th ed.), which has been translated into six languages and awarded "book of the year" 2005 by the Society of Authors & The Royal Society of Medicine in London. He has also contributed many chapters to textbooks edited by colleagues.

Philip Peng

Dr. Philip Peng is a Professor in the Department of Anesthesia of University of Toronto. He is the Director of the anesthesia chronic pain program of University Health Network, the largest teaching hospital of Canada.

He has played an important role in the education of pain medicine and established major teaching courses for Pain in Canada such as National Pain Refresher Course, Canadian Pain Interventional Course, and Ultrasound for Pain Medicine Course. Royal College of Physicians and Surgeons of Canada (RCPSC) honored him with the Founder designation in pain medicine for his role in establishing pain medicine subspecialty in Canada. Besides, he currently serves as the Chair of Exam committee in Pain Medicine in RCPSC, and previously served as the Chair of the Education Special Interest Group (SIG) of Canadian Pain Society and the founding executive of Pain Education SIG of International Association for the Study of Pain (IASP). He has been honored with numerous teaching awards at national and regional levels.

Dr. Philip Peng is also a leader and pioneer in the application of ultrasound for pain medicine. Being one of the founding fathers for Ultrasound for Pain Medicine (USPM) SIG for ASRA (American Society of Regional Anesthesia), he was involved in the establishment of the guideline for Education and Training for USPM, which was adopted by five continents. He has been the chair or main organizer for various major teaching courses for USPM, including satellite meeting of World Congress of Pain, International Pain Congress, satellite meeting for combined Canadian and British Pain Society Conference, International Symposium of Ultrasound for Regional Anesthesia and Canadian Pain Interventional Course.

Part I
General Considerations

Chapter 1

Use of Local Anesthetics in Regional Anesthesia and Pain Therapy

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_1,

Local anesthetics produce reversible blockage of sodium channels in the nerve cell membrane, thereby interrupting stimulus conduction.

Chemical Structure and Physicochemical Properties [1]

All local anesthetics in common clinical use have three characteristic molecular sections in their chemical structure:

An aromatic residue, which basically determines the lipophilic properties of the agent. Substitutions in the aromatic group allow the pKa and lipid solubility of the substance to be influenced.

An intermediate chain, which in local anesthetics of the ester type (Table 1.1) contains a relatively unstable ester bond (CO–O) that can be broken down hydrolytically by pseudocholinesterases. Local anesthetics of the amide type (Table 1.2) are much more stable, since the amide bond (NH–CO) in their intermediate chain cannot be broken down in plasma. The length of the chain between the aromatic residue and the substituted amino group has an influence on the intensity of effect of the local anesthetic. The agent's protein-binding capacity and lipid solubility can be altered by substitution in the intermediate chain.

A substituted amino group, the protonization of which determines the ratio of the cationic to the basic form. Only the free base is capable of penetrating lipoprotein membranes. However, to be able to affect the nerve membrane, the local anesthetic must be available as a cation. The type of amino group substitution affects the distribution coefficient, the plasma protein binding, and the intensity and duration of the drug's action.

Clinical Significance of the Physicochemical Properties

Local anesthetics differ with regard to their molecular weight, their lipid and water solubility, pKa, and protein-binding characteristics. These factors in turn have a substantial influence on the potency of the drug's local anesthetic effect on the onset of the effect and on its duration (Tables 1.3a and 1.3b).

Local Anesthetic Potency [2]

The combined effect of factors such as protein binding, stereoisomeric structure, and lipophilia determines the potency of a local anesthetic agent. To achieve a blocking effect, the local anesthetic has to diffuse across the cell membrane into the interior of the cell (importance of lipophilia for membrane diffusion) so that, from the cytosol (appropriate hydrophilic properties), it can occupy the sodium channel in its then protonated form (Table 1.4).

A high degree of lipophilia is associated with good membrane permeation, and a high degree of hydrophilia is associated with good solubility in the cytosol. Local anesthetics therefore have to have both of these properties in a favorable ratio.

However, the clinical distinction that is made in local anesthetics between those of mild potency (procaine), medium potency (lidocaine, prilocaine, mepivacaine), and high potency (ropivacaine, bupivacaine, levobupivacaine, etidocaine) does not conform to these correlations in all respects.

The onset of effect in the isolated nerve, at physiological pH, depends on the pKa value of the local anesthetic. The lower this value is, the more local anesthetic base can diffuse toward the membrane receptors, and the shorter the time will be to the onset of the nerve block. Higher concentrations of local anesthetic accelerate onset.

The duration of effect depends on the dosage and concentration of the local anesthetic, its binding to the membrane receptors (protein-binding capacity), and its reabsorption from the tissue into the blood.

Table 1.1 Local anesthetics with an ester bond

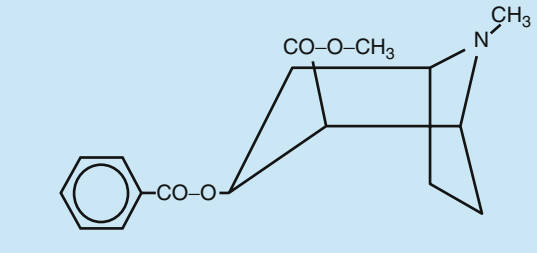
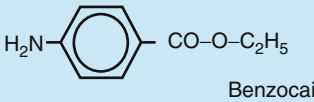
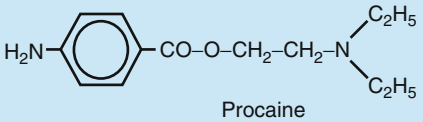
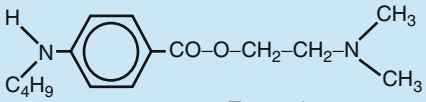
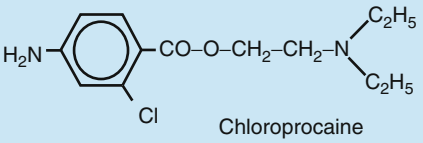
Aromatic residue	Intermediate chain	Substit. amino group	Year introduced
			
Cocaine			1884
			
Benzocaine			1900
			
Procaine			1905
			
Tetracaine			1930
			
Chloroprocaine			1955

Table 1.2 Local anesthetics with an amide bond

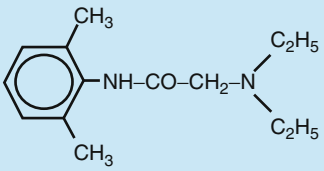
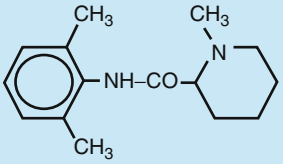
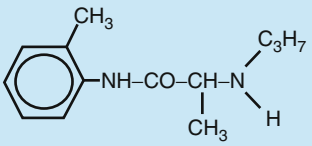
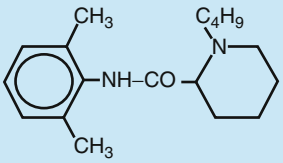
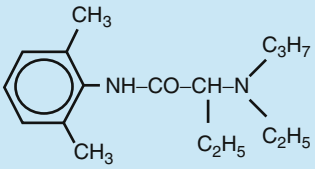
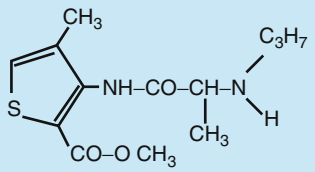
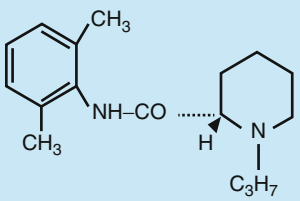
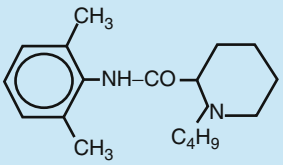
Aromatic residue	Intermediate chain	Substit. amino group	Year introduced
		Lidocaine	1944
		Mepivacaine	1957
		Prilocaine	1960
		Bupivacaine	1963
		Etidocaine	1972
		Carticaine	1974
		Ropivacaine	1996
		Levobupivacaine	2000

Table 1.3a A physicochemical and pharmacological parameters

Agent	Molecular weight	pKa (25°)	Distribution coefficient (lipid/water)	Protein binding (%)	Potency in vitro (isolated nerve)
Procaine	236	8.9	0.02	5.8	1
Lidocaine	220	7.7	2.9	64–70	4
Mepivacaine	234	7.7	0.9	77–80	3–4
Prilocaine	246	7.6	0.8	55	3–4
Bupivacaine	288	8.1	27.5	95	16
Etidocaine	276	7.7	141	95	16
Ropivacaine	274	8.1	9	95	16
Levobupivacaine	288	8.09	27.5	97	16

Table 1.3b Local anesthetic potency and duration of effect

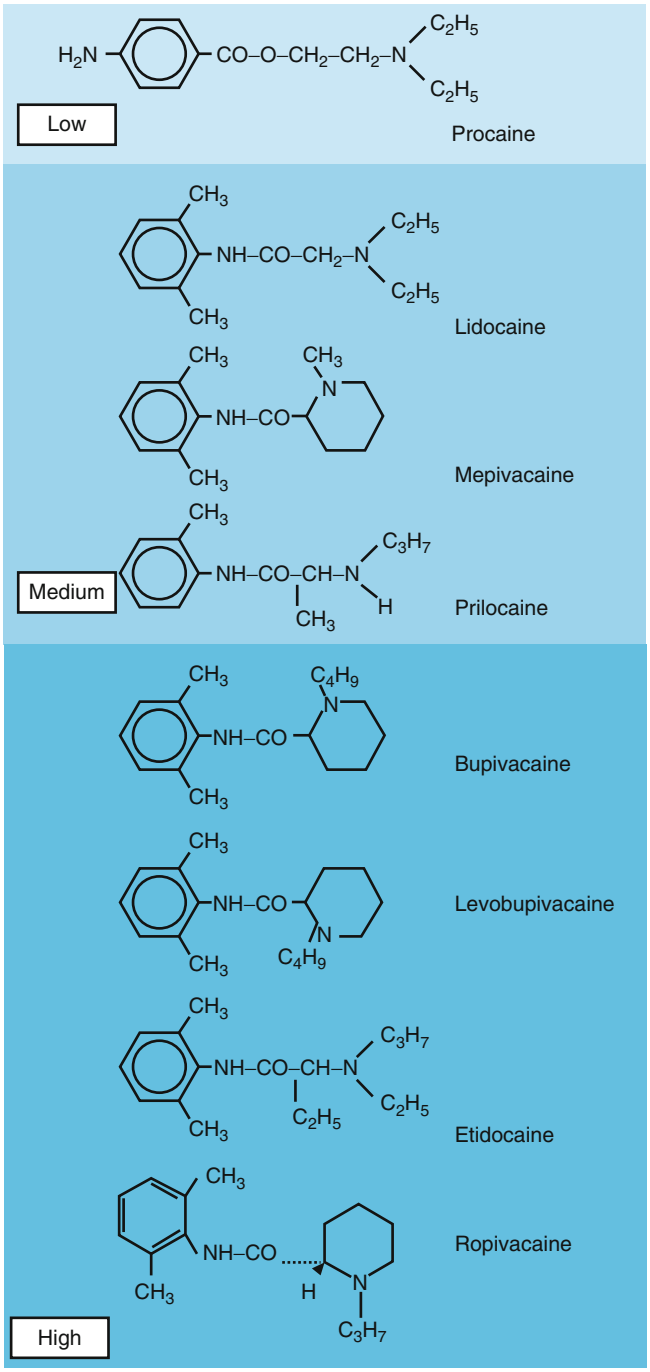
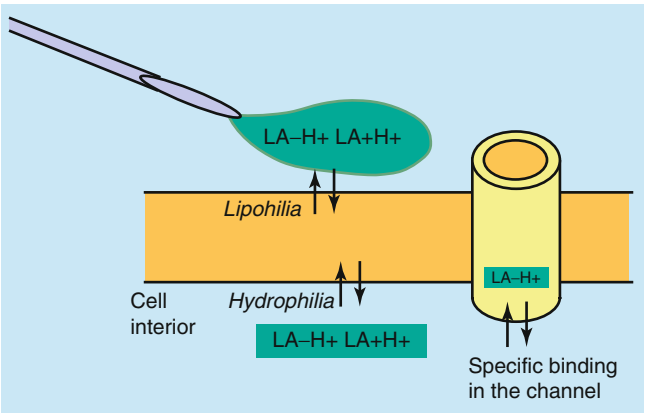


Table 1.4 Chemical requirements of a local anesthetic local anesthetics must combine lipophilic and hydrophilic properties in a favorable ratio with each other hydrophilia, soluble in cytosol; lipophilia, overcoming the cell membrane



Equipotent Concentrations

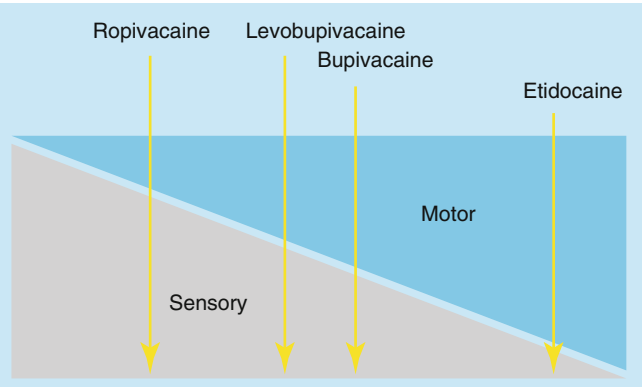
Medium-duration local anesthetics have more or less the same clinical potency (except perhaps for lidocaine—due to stronger vasodilation, this local anesthetic is resorbed more readily from the site of action, and this can affect the duration and intensity of the block).

Equipotent concentrations of long-acting local anesthetics cannot be demonstrated in the same way, since the three local anesthetics mentioned have completely different block profiles: etidocaine (highest lipophilic capacity) produces a mainly motor block, ropivacaine has a mainly sensory effect, and bupivacaine has both motor and sensory effects. Anesthetic concentrations of bupivacaine and ropivacaine are equipotent (one to one).

Block Profile (Table 1.5)

The block profile shows the relation between sensory and motor block. Physicochemical properties determine the block profile. At high anesthetic concentrations—so far as these are toxicologically permissible—the excess quantity of the agent can also block fibers not primarily affected (motor or sensory fibers). On the other hand, the block profile is not altered by low concentrations. A reduced motor block is obtained at the cost of reduced analgesic quality, and this is why opioid supplementation is usually necessary with dilute concentrations of local anesthetic.

Table 1.5 Relative block profile of long-acting local anesthetics



Incompatibility

Local anesthetics can precipitate after dilution with alkaline solutions and should therefore not be diluted with or injected simultaneously with sodium bicarbonate.

Side Effects and Systemic Effects (Tables 1.6 and 1.7)

When assessing the safety and tolerability of a local anesthetic, not only its central nervous system and cardiovascular effects need to be taken into account, but also its allergenic potential and toxic degradation products that may form as it is metabolized.

Table 1.6 Toxicity of clinical dosages of local anesthetics

Local anesthetic	Central nervous system	Heart
Lidocaine	++	+
Mepivacaine	++	+
Prilocaine	+	+/–
Bupivacaine	+++	++++++[a]
Levobupivacaine	++	++++
Ropivacaine	++(+)	+++

[a]Clinical dose can be equivalent to a lethal dose when incorrectly administered

Table 1.7 Symptoms of intoxication due to local anesthetics

Central nervous system	Cardiovascular system
Stimulation phase, mild intoxication	
Tingling of lips, tongue paresthesias, perioral numbness, ringing in the ears, metallic taste, anxiety, restlessness, trembling, muscle twitching, vomiting	Cardiac palpitation, hypertonia, tachycardia, tachypnea, dry mouth
Stimulation phase, moderately severe intoxication	
Excitation phase, moderate toxicity Speech disturbance, dazed state, sleepiness, confusion, tremor, choreoid movements, tonic-clonic cramp, mydriasis, vomiting, polypnea	Tachycardia, arrhythmia, cyanosis and pallor, nausea and vomiting
Paralytic phase, severe toxicity	
Stupor, coma, irregular breathing, respiratory arrest, flaccidity, vomiting with aspiration, sphincter paralysis, death	Severe cyanosis, bradycardia, drop in blood pressure, primary heart failure, ventricular fibrillation, hyposystole, asystole

Systemic Effects

Adverse systemic effects of local anesthetics can occur when their plasma concentration is high enough to affect organs with membranes that can be irritated.

Toxic plasma levels can be reached as a result of:

- Inadvertent intravascular or intrathecal/epidural injection
- Overdosing, particularly in areas with good perfusion and correspondingly high resorption
- Failure to adjust the dosages (mg/kg body weight), particularly in patients with hepatic or renal disease

The severity of intoxication depends on the absolute plasma level, as well as on the strength of the local anesthetic's effect. While anesthetic dosages of short-acting local anesthetics (prilocaine, mepivacaine, lidocaine) can trigger clear CNS symptoms in a range extending to generalized cramp, cardiotoxic reactions are also possible with long-acting local anesthetics. In particular, cases of cardiac arrest have been reported with bupivacaine with comparatively small intravascular injections (50 mg; not treatable in half of the cases).

Cardiac symptoms and cardiac arrest can also occur with ropivacaine after inadvertent intravascular injections. However, these can be treated effectively and only occur at higher dosages. The following sequence of increasing systemic toxicity applies to the most frequently used local anesthetics: procaine < prilocaine < mepivacaine < lidocaine < ropivacaine < levobupivacaine < bupivacaine.

CNS toxicity: Central reactions predominate in terms of frequency and clinical significance. The symptoms of these are listed in Table 1.7 in order of severity and toxicity. For speedy and appropriate treatment, it is important to observe and react immediately when even the preconvulsive signs of CNS intoxication are seen—particularly numbness of the tongue and perioral region.

Cardiovascular toxicity: Toxic effects on the cardiovascular system usually occur after the administration of very high doses. They are seen in the form of conduction disturbances in the autonomic cardiac and vascular nerve fibers, depression of cardiac function, and peripheral vasodilation (Tables 1.6 and 1.7).

Local Anesthetic Systemic Toxicity (LAST) [3–7]

Diagnosing (Table 1.7)

Classic descriptions of LAST depict a progression of subjective symptoms of *CNS excitement* (agitation, auditory changes, metallic taste, or abrupt onset of psychiatric symptoms) followed by seizures or *CNS depression* (drowsiness, coma, or respiratory arrest). Near the end of this continuum, initial signs of *cardiac toxicity* (hypertension, tachycardia, or ventricular arrhythmias) are supplanted by cardiac depression (bradycardia, conduction block, asystole, decreased contractility) [3]. However, there is substantial variation of this classic description, including the following:

- Simultaneous presentation of CNS and cardiac toxicity
- Cardiac toxicity without prodromal signs and symptoms of CNS toxicity

Thus, the practitioner must be vigilant for atypical or unexpected presentation of LAST. The timing of LAST presentation is variable: Immediate (<60 s) presentation suggests intravascular injection of LA with direct access to the brain, while presentation that is delayed 1–5 min suggests intermittent or partial intravascular injection, delayed circulation time, or delayed tissue absorption. Because LAST can present >15 min after injection, patients who receive potentially toxic doses of LA should be closely monitored for at least 30 min after injection.

Caution

The onset of LAST is usually very rapid, following a single LA injection by 50 s or less in half of the cases and occurring before 5 min in ¾ of the cases [3]. The most important first step in improving patient outcome is to have a low threshold for considering the diagnosis (atypical presentation was reported in approximately 40 % of published cases of LAST) [6].

Prevention [6]

Prevention is the most important measure in reducing the frequency and severity of LAST. No single intervention has been identified that can reliably eliminate risk. Central to prevention is limiting the opportunity for intravascular injection or tissue uptake to local anesthetic, which is best accomplished by early detection of intravascular needle or catheter placement.

Local anesthetic dose reduction may be particularly important for those patients thought to be at greater risk of LAST.

Risk Reduction

Local anesthetic blood levels are influenced by the site of injection, and those factors that can increase the likelihood of LAST include:

1. Those patients at extremes of age (<4 months or >70 years).
2. Heart failure, history of ischemic heart disease, and cardiac conduction abnormalities.
3. Metabolic (e.g., mitochondrial) disease.
4. Liver disease.

5. Low plasma protein concentration.
6. Metabolic or respiratory acidosis.
7. Medications that inhibit sodium channels.
   Neither body weight nor body mass index correlates with local anesthetic plasma levels after a specific dose in adults; the correlation is more accurate in children.

Caution

1. Use incremental injection of local anesthetics before and during the injection (after each 4–5 mL—aspiration should be carried out repeatedly, pausing 15–30 s between each injection) observing for signs and querying frequently for symptoms of toxicity between each injection recognizing that there is ~2 % false-negative rate for this diagnostic intervention [5].
2. Maintain verbal contact with the patient.
3. Monitor the patient during and after completing the injection, as clinical toxicity can be delayed up to 30 min (or longer after tumescent procedures).

Intravascular Marker

When injecting potentially toxic doses of local anesthetic, use of an intravascular marker is recommended [5]. Although imperfect, intravascular test dosing remains the most reliable marker of intravascular injection. Of the various options described, only fentanyl and epinephrine meet suggested standards for reliability and applicability [4, 6].

Intravascular injection of epinephrine 10–15 μg/mL in adults produces a ≥10-beat HR increase or a ≥15-mmHg SBP increase in the absence of ß-blockade, active labor, advanced age, or general/neuraxial anesthesia. Intravascular injection of epinephrine 0.5 μg/kg in children produces a ≥15-mmHg increase in SBP [8]. Nevertheless, epinephrine test doses are unreliable in the elderly or in patients who are sedated, taking ß-blockers, or anesthetized with general or neuraxial anesthesia. Fentanyl 100 μg produces sedation if injected intravenously in laboring patients [5].

Ultrasound

Ultrasound guidance may reduce the frequency of intravascular injection, but actual reduction of LAST remains unproven in humans.

Recommendations for Treatment of LAST (Table 1.8) [3, 6, 7]

Table 1.8 Recommendation for treatment of LAST

(a)
1. Be prepared. Establish a plan and checklist for managing
2. If signs and symptoms of LAST occur, prompt and effective airway management (ventilate with 100 % oxygen) is crucial to preventing hypoxia and acidosis
▼
3. Immediate treatment of convulsions within 15–30 s of their onset especially correcting hypoxia and acidosis is not associated with cardiac catastrophe [7]
4. Get help
(b)
If seizures occur
1. Benzodiazepines are preferred
2. If benzodiazepines are not readily available,
▼
Small doses of propofol (0.5–1.5 mg/kg) or thiopental (1–2 mg/kg) are acceptable
Although propofol can stop seizures, large doses further depress cardiac function
(c)
▼
If seizures persist despite
Benzodiazepines/small doses of succinylcholine (0.5–1 mg/kg)
(d)
▼
If cardiac arrest occurs
Standard and advanced cardiac life support
Small initial doses of epinephrine (10- to 100-μg boluses in adult) are preferred. There is laboratory evidence that epinephrine can impair resuscitation from LAST and reduce the efficacy of lipid rescue. Therefore, it is recommended to avoid high doses of epinephrine and use smaller doses, for example, 1 μg/kg, for treating hypotension [6]

Caution

1. Avoid vasopressin.
2. Treatment with local anesthetics (lidocaine or procainamide) is not recommended.
3. Avoid calcium channel blockers and beta-adrenergic receptor blockers.
   If ventricular arrhythmias develop, amiodarone is preferred.
4. Alert the nearest facility having cardiopulmonary bypass capability.

Table 1.9 Lipid emulsion therapy

1. 5 mL/kg (lean body mass) 20 % lipid emulsion bolus intravenously over 1 min (ca. 100 mL)
▼
Continuous infusion of 0. 25 mL/kg/min (ca. 18 mL/min; adjust by roller clamp), continued for at least 10 min after circulatory stability is attained
▼
If circulatory stability is not attained, consider giving another bolus and increasing infusion to 0.5 mL/kg/min
Approximately 10 mL/kg lipid emulsion over 30 min is recommended as the upper limit for initial dosing

Lipid Emulsion Therapy (Table 1.9) [7]
Timing of lipid infusion in LAST is controversial. The most reasonable approach is to implement lipid therapy on the basis of clinical severity and rate of progression of LAST.

Dosing [7] (Table 1.9)

Caution
1. Propofol is not a substitute for lipid emulsion.
2. Failure to respond to lipid emulsion and vasopressor therapy should prompt institution of cardiopulmonary bypass.
3. Prolonged monitoring (≥12 h) is recommended after any signs of cardiac toxicity because cardiovascular depression due to LAST can persist or recur after treatment.

Substance-Specific Side Effects [1]

One specific side effect of prilocaine is the increased methemoglobin level caused by the metabolite o-toluidine. Clinically, cyanosis, headache, cardiac palpitation, and vertigo can be expected at methemoglobin levels of 10–20 % and loss of consciousness, shock, and death when the level is 60 % or more. This does not call into question the beneficial toxicological properties of prilocaine, since clinically relevant methemoglobinemia can only occur at dosages of more than 600 mg, which is much more than clinically used doses of mepivacaine or lidocaine. A clinically harmful methemoglobin level can be treated within a few minutes by the intravenous administration of 2–4 mg/kg toluidine blue (or, alternatively, 1–2 mg/kg methylene blue). Because of this specific side effect, prilocaine is not indicated in patients with congenital or acquired methemoglobinemia, in patients who are anemic or have a history of heart disease, in obstetrics (e.g., for pudendal nerve or paracervical block), or in children under the age of 6 months.

Allergenic Potential

There are no reliable data regarding the frequency of allergic reactions after the administration of local anesthetics. There is no doubt that these are extremely rare, although the symptoms can range from allergic dermatitis to anaphylactic shock. Occasional cases of allergic reactions to ester local anesthetics have been reported, and the preservative substances which the various preparations contain (e.g., parabens) and the antioxidant sodium bisulfide in epinephrine-containing solutions are also under discussion as potential causes. In patients with suspected intolerance of local anesthetics, intracutaneous testing with 20 pL of the agent can be conducted.

When the result is positive, subcutaneous provocation tests at increasing dosages (0.1 mL diluted to 1:10,000, 1:1,000, and 1:10; undiluted at 0.1, 0.5, and 1 mL) can be considered. When these tests are being carried out, it is vital to prepare all the necessary safety measures in case of a severe reaction.

Recommended maximum doses without epinephrine, according to specialist information:

Lidocaine	Mepivacaine	Prilocaine
200 mg	300 mg	400 mg

Table 1.10 Functional distinctions between nerve fibers

Fiber type	Function
$A\alpha$	Motor, touch, pressure, depth sensation
$A\beta$	Motor, touch, pressure, depth sensation
$A\gamma$	Regulation of muscle tone
$A\delta$	Pain, temperature, touch
B	Preganglionic sympathetic function
C	Pain, temperature, touch, postganglionic sympathetic function

Table 1.11 Overview of drugs

Drug	Potency	Duration of effect (h)	Toxicity	Half-life	Vdiss
Lidocaine	1	2	1	96′	91
Mepivacaine	1	2–3	1.2	114′	84
Prilocaine	1	2–3	0.5	93′	261

Selection of Suitable Substances for Regional Block

When surgical interventions are being carried out under regional anesthesia, priority must go to shutting off both sensory and motor systems, and knowledge of the expected length of the operation is vital to the choice of anesthetic. The onset of effect and the toxicity of the drug used play important parts, but not decisive ones. In the context of pain therapy, in which the fast-conducting A delta fibers and the slow-conducting C fibers (Table 1.10) are the target of the block, toxicity is much more important than the duration of the effect.

In diagnostic and therapeutic blocks, in which there is a risk of intravascular injection—e.g., in a stellate ganglion block or superior cervical ganglion block—prilocaine should be selected, as it is the medium-duration local anesthetic with the lowest toxicity (mepivacaine and lidocaine are alternatives) (Table 1.11).

Bupivacaine has an important role in regional blocks, being a longer-duration local anesthetic that provides high-quality analgesia and an easily controlled motor block. Its anesthetic potency is about four times that of local anesthetics with medium-duration effects (such as prilocaine). When the lower dosage required in pain therapy than in regional anesthesia is taken into account, bupivacaine can be used for practically all pain therapy procedures in spite of its relatively high toxicity.

Ropivacaine is the most recently introduced long-duration local anesthetic in the amino-amide series. The differential block is even more marked than with bupivacaine, and the drug is associated with much lower CNS toxicity and cardiac toxicity. These characteristics make it particularly suitable for regional anesthesia procedures in which higher dosages or concentrations are required. Ropivacaine provides good-quality analgesia while largely maintaining motor activity (up to 80 % of patients have no measurable motor block on the Bromage scale). At a dosage of 2 mg/mL, the drug is therefore the local anesthetic of choice for epidural obstetric analgesia and for postoperative analgesia (Table 1.5). With its pharmacological profile, ropivacaine is the first local anesthetic with primarily analgesic effects, and it is therefore particularly suitable for pain therapy indications.

Every anesthetist and pain therapy physician who uses anesthetic methods for temporary interruption of stimulus conduction in a ganglion, nerve, or neural plexus should be familiar with the properties and potential applications of the following agents:

Short-Acting Local Anesthetics

Procaine (Novocain®) (Tables 1.1, 1.3a, and 1.3b)

In 1905, Einhorn in Germany succeeded in synthesizing a new local anesthetic, which he called "procaine." Heinrich Braun introduced procaine into clinical practice on the same year, as a 4.5 and 5 % solution.

Class of drug: Local anesthetic of the ester type.
Single threshold dose: 500 mg without epinephrine in adults.
LD_{50} (mouse): 52.2–60.0 mg/kg body weight i.v.
Plasma half-life: <0.14 h.
Latency: Medium.
Duration of effect: 0.5–1 h, depending on the area of application and the concentration used.
Metabolism: Procaine is broken down in plasma by pseudocholinesterase into p-aminobenzoic acid—a naturally occurring component of folic acid synthesis—and into diethylaminoethanol. The metabolites are excreted in the urine or broken down in the liver.
Tolerability and control: Procaine is one of the local anesthetics that have the lowest toxicity. Due to its short half-life, procaine is easily controlled.

Clinical uses: It is not so much its local anesthetic potency that predominates in procaine, but rather its muscle-relaxing properties and vasodilatory effect—which are of primary importance in infiltration therapy and trigger point treatment.
In the therapeutic field, very good results can be obtained with superior cervical ganglion block. However, procaine's high allergenic potency in comparison with amide local anesthetics argues against its use.
Dosage: Procaine is administered at concentrations of 0.5–2 %. Precise dosages are described in the relevant sections of this book.

2-Chloroprocaine (Table 1.1)

2-Chloroprocaine, an ester local anesthetic, is a chlorinated derivative of procaine and is the most rapidly metabolized local anesthetic currently used. Although the potency of chloroprocaine is relatively low, it can be used for epidural anesthesia in large volumes in a 3 % solution because of its low systemic toxicity. The duration of action is between 30 and 60 min. This agent enjoyed its greatest popularity for epidural analgesia and anesthesia in obstetrics because of the rapid onset and low systemic toxicity in both mother and fetus. However, frequent injections are needed to provide adequate pain relief in labor, and it is more usual to establish analgesia with chloroprocaine and then change to a longer-acting agent such as ropivacaine or bupivacaine.

The use of chloroprocaine declined because of reports of prolonged neurological deficit following accidental subarachnoid injection. This toxicity was ascribed to the sodium metabisulfite used in the past as preservative. However, there are no reports of neurotoxicity with newer preparations of chloroprocaine which contain disodium ethylenediaminetetraacetic acid (EDTA) as the preservative. Nevertheless, these preparations are not recommended for intrathecal administration. However, since then, a number of reports of back pain have appeared. The incidence of back pain appears to be related to the large volume (greater than 40 ml) of drug injected. Chloroprocaine has also proved of value for peripheral nerve blocks and epidural anesthesia when the duration of surgery is not expected to exceed 30–60 min.

Tetracaine (Table 1.1)

Tetracaine is a long-acting amino ester. It is significantly more potent and has a longer duration of action than procaine or 2-chloroprocaine. Tetracaine remains a very popular drug for spinal anesthesia in the United States. This drug possesses excellent topical anesthetic properties, and solutions of this agent were commonly used for endotracheal surface anesthesia. Because of its slow onset and high toxicity, tetracaine is rarely used in peripheral nerve blocks.

Medium-Term Local Anesthetics

Lidocaine (Xylocaine®, Lignocaine) (Tables 1.2, 1.3a, 1.3b, 1.6, and 1.11)

Löfgren and Lundqvist in Sweden isolated a new substance in 1943 that was given the working name of "LL 30." It was later renamed "lidocaine." Following extensive pharmacological studies by Goldberg, the first clinical tests in dentistry using lidocaine were carried out in 1947. Torsten Gordh, the father of Swedish anesthesia, carried out the first investigations of lidocaine in humans.

Class of drug: Lidocaine is a medium-duration local anesthetic of the amide type.

Single threshold dose: 200 mg without epinephrine in adults/70 kg body weight. After injection of a maximum dose, subsequent injections should not be given for 90 min. The second dose must not exceed a maximum of half of the first dose.

LD_{50} (mouse): 31.2–62.2 mg/kg body weight i.v.

Plasma half-life: ca. 1.6 h.

Latency: Fast.

Duration of effect: 1–2 h, depending on the area of application and the concentration used.

Metabolism: Lidocaine is metabolized in hepatic microsomes. Only about 3 % of the drug is excreted unchanged via the kidney.

Tolerability and control: Lidocaine is one of the local anesthetics with moderate relative toxicity. It is characterized by a medium-term duration of effect and good distribution characteristics.

Lidocaine causes vasodilation, which may be less than that of procaine. When the medium-duration local anesthetics are compared, the strengths of the associated vasodilatory effects show the following sequence: lidocaine > mepivacaine > prilocaine. Lidocaine is therefore often used with epinephrine.

Clinical uses: Lidocaine is widely used in clinical practice, particularly in neural and segmental therapy. It is also suitable for infiltration anesthesia, for peripheral nerve block, for epidural anesthesia, and for mucosal surface anesthesia (2 % gel, Emla®).

Dosage: Lidocaine is mainly administered as a 0.5–1 % (1.5) % solution. Specific doses are given in the relevant chapters of this book.

Emla® Cream

Emla® (a mixture of 2.5 % lidocaine and 2.5 % prilocaine) is a topical local anesthetic that penetrates intact skin and reaches an anesthetic depth of up to 5 mm. The onset of effect is approximately 1 h. When the effect takes place, the vessels in the skin show vasoconstriction initially, followed by vasodilation when higher concentrations are reached. This form of administration of this local anesthetic mixture has proved particularly useful in pediatric anesthesia before intravenous access placement and for minor surgical procedures on the skin surface.

Lidocaine Plaster

Lidocaine, administered in various forms (i.v., i.m., or transdermally), relieves pain associated with postherpetic neuralgia (PHN) [8–13]. The analgesia is based on the blockade of neuronal sodium channels. However, intravenous administration of lidocaine can lead to plasma concentrations associated with antiarrhythmic effects. Topical application of lidocaine in the form of a gel or plaster avoids high plasma concentrations. This type of lidocaine plaster was developed in the United States, where it has been licensed since 1999 for pain treatment in postherpetic neuralgia (Lidoderm®, Endo Pharmaceuticals Ltd., Chadds Ford, PA). The plaster consists of a soft, stretchable polyester base connected to an adhesive layer that contains 5 % lidocaine. The plaster is 10 × 14 cm in size.

The systemic absorption of lidocaine has been shown in preclinical and clinical studies to be minimal (3 %) in both volunteers and patients with PHN. Treatment with lidocaine plaster has been investigated in comparison with a placebo in three randomized, double-blind clinical studies including a total of 217 patients with PHN [8, 12–14]. A significant reduction in pain intensity and allodynia was observed. Lidocaine plaster therefore represents a treatment option with a relatively low risk of adverse systemic events or drug interactions [15].

In Europe, clinical testing of the plaster for use in postherpetic neuralgia is currently taking place, and its licensing for this indication can be expected within the next 2 or 3 years.

Mepivacaine (Scandicaine®, Mebeverine®) (Tables 1.3a, 1.3b, 1.6, and 1.11)

In 1956, Bo af Ekenstam, a Swedish scientist, developed mepivacaine. It was introduced into clinical practice by K. G. Dhunér in Sweden in 1957.

Class of drug: Mepivacaine is a medium-duration local anesthetic of the amide type.
Single threshold dose without epinephrine in adults (70 kg body weight): 200 mg in the ENT field and 300 mg in other applications.
LD_{50} (mouse): 40.3 ± 3.2 mg/kg body weight i.v.
Plasma half-life: ca. 1.9 h.
Latency: Fast.
Duration of effect: 1–3 h, depending on the area of application and the concentration used.
Metabolism: Mepivacaine is metabolized in the hepatic microsomes.

After intravenous administration, up to 16 % of the agent is excreted unchanged via the kidney. Degradation in the liver mainly produces m-hydroxymepivacaine and p-hydroxymepivacaine. These metabolites are conjugated with glucuronic acid and excreted in the urine. Another metabolite, pipecoloxylidide, collects in bile and passes through the enterohepatic circulation with its degradation products. No 2,6-xylidine is produced when mepivacaine is metabolized, and there is no evidence that either the agent or its metabolites have mutagenic or carcinogenic properties.

Tolerability and control: Mepivacaine is another of the local anesthetics with moderate relative toxicity. It is characterized by a medium-term duration of effect, with good distribution properties and some vasodilatory effect.
Clinical uses: Mepivacaine is the local anesthetic of choice when a medium-duration effect is required for diagnostic and therapeutic blocks in pain therapy—particularly in outpatients. It is suitable for infiltration anesthesia, intravenous regional anesthesia, peripheral nerve block and ganglion block, and epidural anesthesia. Mepivacaine cannot be recommended in obstetrics due to its long elimination half-life in the neonate.
Dosage: Mepivacaine is mainly used as a 1 % (1.5 %) or 0.5 % solution. Specific doses are given in the relevant chapters of this book.

Prilocaine (Xylonest®) (Tables 1.3a, 1.3b, 1.6, and 1.11)

Class of drug: Prilocaine is a medium-duration local anesthetic of the amide type.
Single threshold dose: 400 mg (with or without vasopressor) in adults/70 kg body weight.
LD_{50} (mouse): 62 mg/kg b.w. i.v.
Plasma half-life: ca. 1.5 h.
Latency: Fast.
Duration of effect: 2–3 h, depending on the area of application and the concentration used.
Metabolism: Prilocaine is mainly metabolized in hepatic microsomes, but also in the kidney and lungs. During degradation, the metabolite ortho-toluidine is produced. At doses higher than 600 mg, the body's reduction systems may become exhausted. At doses higher than 800 mg, noticeable methemoglobinemia can be expected (see the section on substance-specific side effects). Fast elimination from the blood leads to low systemic toxicity.
Tolerability and control: Among the amide local anesthetics, prilocaine shows the best ratio between anesthetic potency and toxicity. Due to its high distribution volume and marked absorption in the lungs, plasma levels are significantly lower than those of mepivacaine and lidocaine (by a factor of 2–3). It has a medium-term duration of effect.

Clinical uses: Due to its comparatively low toxicity, prilocaine is particularly suitable for regional anesthesia techniques that require a single injection of a large volume or a high anesthetic dosage. The increasing use of prilocaine (2 % isobaric solution) for spinal anesthesia is relatively new. Comparative studies in recent years have shown good tolerability, while transient neurological symptoms (TNS; see Chap. 41) were observed more often with lidocaine and mepivacaine. Prilocaine—like other medium-duration agents—is not suitable for continuous blocks. Due to the possibility of raised methemoglobin levels, prilocaine should not be used in anemic patients, in children under the age of 6 months, or in obstetrics.

Dosage: Depending on the area of application, a 0.5–2 % solution is used. Specific doses are given in the relevant chapters of this book.

Long-Acting Local Anesthetics

Ropivacaine (Naropin®) (Tables 1.3a, 1.3b, 1.5, and 1.11)

Class of drug: Local anesthetic of the amide type, pure S-enantiomer.

Single threshold dose:

Anesthesia:

Epidural: 0.5–1 %, 200 mg.

Plexus blocks: 0.75 %, 300 mg.

Conduction and infiltration anesthesia: 0.5–0.75 %, 225 mg.

Injection at myofascial trigger points: 0.2 % (1–2 mL per trigger point).

Continuous procedures: 0.2 %, up to 14 mL/h. Increased doses may be required during the early postoperative period—up to 0.375 %, 10 mL/h (maximum 37.5 mg/h). When it is administered over several days, the resulting concentrations are well below potentially toxic plasma levels.

A dosage of 300 mg should be regarded as a guideline value, as this dosage has been confirmed as tolerable by various pharmacological studies.

LD_{50} (mouse): ca. 11.0–12.0 mg/kg b.w. i.v.

Plasma half-life: ca. 1.8 h.

Duration of effect: Epidural anesthesia ca. 7 h (analgesia); ca. 4 h (motor block), 10 mg/mL.

Plexus anesthesia (brachial plexus, lumbosacral plexus): 9–17 h, 7.5 mg/mL.

Infiltration anesthesia: Postoperative analgesia after inguinal herniorrhaphy >7 h (5–23 h), 7.5 mg/mL. Peripheral nerve blocks in pain therapy: 2–6 h (0.2–0.375 mg/mL).

Latency: Medium (decreasing latency at increasing concentrations).

Metabolism: Ropivacaine is metabolized in the liver, mainly through aromatic hydroxylation. Only about 1 % of the drug is excreted unchanged in the urine. The main metabolite is 3-hydroxyropivacaine.

Tolerability: Ropivacaine provides relatively low toxicity for a long-term local anesthetic. Compared with bupivacaine, it has a lower arrhythmogenic potential, and the margin between convulsive and lethal doses is wider. Ropivacaine has more favorable receptor kinetics ("fast in, medium out") in cardiac sodium channels and in comparison with bupivacaine has only slight depressant effects on the energy metabolism of the mitochondria in cardiac muscle cells.

Clinical uses: The first clinical tests were carried out in 1988. Ropivacaine (Naropin®) has been in use since 1996. It is the first local anesthetic with a primary analgesic effect and is therefore of particular interest in pain therapy (postoperative and obstetric, as well as therapeutic blocks).

Ropivacaine is the most comprehensively documented and the most widely approved local anesthetic today. It is the most frequently used long-acting local anesthetic throughout the world. It should be noted that it has been approved, with clinical relevance, for use in continuous therapy for acute pain (epidural and peripheral continuous nerve blocks). Approval for administration in children, including continuous epidural administration, was extended to neonates in 2007. It is the first local anesthetic with primarily analgesic effects and is therefore of particular interest for pain therapy (postoperative and obstetric and therapeutic blocks). In comparison with bupivacaine, it has fewer toxic side effects (CNS and, in particular, cardiac toxicity). High doses are needed before toxic effects develop. CNS symptoms appear well before cardiac symptoms, which in the clinical situation provides time for the local anesthetic injection to be stopped and for early treatment steps to be taken. In an animal model, the chances of successful resuscitation were also found to be better than with bupivacaine (90 % vs. 50 %) [16]. In addition, ropivacaine shows marked differential blocking in epidural analgesia and peripheral blocks. With a good quality of analgesia, up to 80 % of patients have no measurable motor block on the Bromage scale. Epidural combinations (e.g., with sufentanil, dosage range 0.5–1 pg/mL) are possible. In view of the increased use of peripheral blocks and infiltrations at painful trigger points, evidence of higher muscular tissue tolerance in comparison with bupivacaine is also of interest [17]. The relatively low toxicity of ropivacaine means that high concentrations can be given (e.g., 10 mg/mL solution for epidural anesthesia)—providing more intense motor block, a higher success rate, and better-quality analgesia than 0.5 % bupivacaine, for example (Table 1.5 and 1.6).

Dosage: Ropivacaine is administered at concentrations of 2 mg/mL (0.2 %), 7.5 mg/mL (0.75 %), and 10 mg/mL (1 %). Use for continuous epidural infusion has been

approved (Naropin® 2 mg/mL polybag, 100 and 200 mL infusion solution). Cumulative daily doses of up to 675 mg (see specialist information) are well tolerated in adults. Precise information on doses is given in the following chapters.

Levobupivacaine (Chirocaine®) (Tables 1.3a, 1.3b, 1.5, and 1.6)

Class of drug: Local anesthetic of the amide type. A pure S-enantiomer of bupivacaine.
Single threshold dose without epinephrine in adults: 150 mg.
LD_{50} (mouse): 10.6 mg/kg b.w.
Plasma half-life: 80 ± 22 min. Plasma protein binding of levobupivacaine in humans has been assessed in vitro and was more than 97 % at concentrations of 0.1–1.0 pg/mL.
Latency: Medium (between ropivacaine and bupivacaine).
Duration of effect: 8–24 h, depending on the area of application and the concentration used.
Metabolism: Levobupivacaine is extensively metabolized, and unaltered levobupivacaine is not found in the urine or feces. 3-Hydroxylevobupivacaine, one of the principal metabolites of levobupivacaine, is excreted via the urine as a glucuronic acid and sulfate ester conjugate. In vitro studies have shown that levobupivacaine is metabolized via CYP3A4 isoforms and CYP1A2 isoforms into desbutyl levobupivacaine or 3-hydroxylevobupivacaine. The studies showed that the degradation of levobupivacaine and bupivacaine is similar. After intravenous administration of levobupivacaine, the recovery rate within 48 h averaged ca. 95 %, quantitatively measurable in urine (71 %) and feces (24 %). There is evidence of in vivo racemate formation with levobupivacaine.
Tolerability and control: Experimental animal studies have demonstrated a lower risk of CNS and cardiovascular toxicity with levobupivacaine than with bupivacaine. In volunteers, fewer negative inotropic effects were observed after intravenous administration of more than 75 mg levobupivacaine in comparison with bupivacaine. QT interval changes only occurred in a very few cases.
Clinical uses: There is little experience as yet with levobupivacaine in clinical practice. The numbers of published controlled clinical studies are also comparatively small. Available in vitro, in vivo, and controlled patient studies comparing levobupivacaine and bupivacaine have shown similar potency for neural blocks. After epidural administration of levobupivacaine, the same quality of sensory and motor block as with bupivacaine was seen. However, a significant differential block, as provided by ropivacaine, cannot be expected, as the drug has the same degree of lipophilia as bupivacaine. Levobupivacaine has not been approved for use in Germany.
Dosage: 0.125–0.75 %. Precise information on doses is given in the following chapters.

Bupivacaine (Carbostesin®, Marcaine®) (Tables 1.3a, 1.3b, 1.5, and 1.6)

The first clinical studies of a long-acting local anesthetic, bupivacaine, were carried out in 1965/1966.

Class of drug: Local anesthetic of the amide type.
Single threshold dose: 150 mg without epinephrine in adults.
LD_{50} (mouse): 7.8 ± 0.4 mg/kg b.w. i.v.
Plasma half-life: ca. 2.7 h.
Latency: Medium.
Duration of effect: 2.5–20 h, depending on the area of application and the concentration used. A mean duration of effect of 3–6 h can be assumed.
Metabolism: Bupivacaine is broken down in hepatic microsomes at a high rate. The predominant metabolization involves dealkylation to pipecoloxylidide (desbutyl bupivacaine). There is no evidence that either the agent or its metabolites have mutagenic or carcinogenic properties.
Tolerability and control: Bupivacaine is one of the local anesthetics that has a high relative toxicity. Its anesthetic potency is about four times greater than that of mepivacaine. It is characterized by a slower onset of effect and by a long duration of effect.
Clinical uses: Bupivacaine is indicated as a long-duration local anesthetic, particularly for regional anesthesia in the surgical field, in postoperative analgesia, and in therapy for various pain conditions.
It is suitable for infiltration anesthesia, peripheral nerve block, ganglion block and plexus block, as well as all forms of neuraxial anesthesia.
The marked cardiac toxicity of bupivacaine has been known since publications dating from the late 1970s, and severe and fatal adverse effects are still reported. Strict observation of safety standards is therefore of fundamental importance for the safe use of this drug at high doses.
Dosage: Depending on the indication, bupivacaine is administered as a 0.125–0.5 % solution. A 0.75 % solution is still being marketed. Higher concentrations are not required in pain therapy. Specific doses are given in the following chapters.

Examination and Patient Preparation

Before regional anesthesia, the same type of examination of the patient should be carried out as for general anesthesia. Contraindications must be excluded, as well as neurological abnormalities, and when there are relative

contraindications—e.g., hemorrhagic diathesis, stable systemic neurological disease, or local nerve damage—a careful assessment of the risk–benefit ratio needs to be made.

Particular attention needs to be given to anatomical relationships, palpation of the landmarks, and precise localization and marking of the needle insertion point.

To ensure cooperation, the patient should be given detailed information about the aim of the block, its technical performance, and possible or probable paresthesias and their significance. The patient should also be informed about potential adverse effects and complications of the block, and outpatients in particular should be familiarized with guidelines on behavior after anesthesia. The patient information session should be documented using a consent form signed by the patient.

In general, premedication and the administration of sedatives or analgesics should be avoided, particularly in outpatient pain therapy. Constant verbal contact should be maintained with the patient during the block, so that potential side effects or complications can be recognized immediately. In addition, any sedation that is not adjusted individually can lead to respiratory and circulatory complications, which may be mistaken for the early symptoms of local anesthetic toxicity.

Documentation of Treatment

The patient history, including investigations at other centers, and diagnostic results should be documented just as carefully as the preparation, implementation, and success of the block. The checklists and record forms used in our own pain center have been adapted for each individual block technique and are included in the following chapters.

References

1. Weninger E. Pharmakodynamik der Lokalanästhetika. Anästhesiologie Intensivmedizin. 1996;5(37):249–67.
2. Borchard U, Niesel HC. Grundlagen der Pharmakologie der Lokalanästhetika. In: Niesel HC, editor. Regionalanästhesie, Lokalanästhesie, Regionale Schmerztherapie. Stuttgart/New York: Georg Thieme Verlag; 1994.
3. Di Gregorio G, Neal MJ, Rosenquist RW, Weinberg LG. Clinical presentation of local anesthetic systemic toxicity. A review of published cases, 1979 to 2009. Reg Anesth Pain Med. 2010;35:181–7.
4. Guay J. The epidural test dose: a review. Anesth Analg. 2006;102:921–9.
5. Mulroy MF, Hejtmanek MR. Prevention of local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35:177–80.
6. Neal MJ, Bernards MC, Butterworth FJ, Di Gregorio G, Drasner K, Hejtmanek M, Mulroy MF, Rosenquist RW, Weinberg GL. ASRA practice advisory on local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35:152–61.
7. Weinberg LG. Treatment of local anesthetic systemic toxicity (LAST). Reg Anesth Pain Med. 2010;35:188–93.
8. Rowbotham MC, Reisner-Keller LA, Fields HL. Both intravenous lidocaine and morphine reduce the pain of postherpetic neuralgia. Neurology. 1991;41:1024–8.
9. Baranowski AP, De Courcey J, Bonelle E. A trial of intravenous lidocaine on the pain and allodynia of postherpetic neuralgia. J Pain Symptom Manage. 1999;17(6):429–33.
10. Basler HD, Ernst A, Flöter Th, Gerbershagen HU, Hankemeier U, Jungck D, Müller-Schwefe G, Zimmermann M. Gemeinsame Richtlinien der Deutschen Gesellschaft zum Studium des Schmerzes e. V. (DGSS) und des SCHMERZtherapeutischen Kolloquiums e. V. (StK) für die Zusatzweiterbildung zum Algesiologen DGSS/STK. SCHMERZtherapeutisches Kolloquium. 1995;11:3–5.
11. Rowbotham MC, Davies PS, Galer BS. Multicenter, double-blind, vehicle-controlled trial of long term use of lidocaine patches for postherpetic neuralgia (abstract). In: 8th World Congress on Pain. Vancouver: IASP Press; 1996. p. 274.
12. Rowbotham MC, Davies PS, Fields HL. Topical lidocaine gel relieves postherpetic neuralgia. Ann Neurol. 1995;37:246–53.
13. Rowbotham MC, Davies PS, Verkempinck C. Lidocaine patch: double-blind controlled study of a new treatment method for postherpetic neuralgia. Pain. 1996;65:39–44.
14. Galer BS, Rowbotham MC, Perander J. Topical lidocaine patch relieves postherpetic neuralgia more effectively than a vehicle topical patch: results of an enriched enrollment study. Pain. 1999;80:533–8.
15. Gammaitoni AR, Alvarez NA, Galer BS. Safety and tolerability of the lidocaine patch 5%, a targeted peripheral analgesic: a review of the literature. J Clin Pharmacol. 2003;43(2):111–7.
16. Groban L, Deal DD, Vernon JC, James RL, Butterworth J. Cardiac resuscitation after incremental overdosage with lidocaine, bupivacaine, levobupivacaine, and ropivacaine in anesthetized dogs. Anesth Analg. 2001;92:37–43.
17. Zink W, et al. Bupivacaine but not Ropivacaine Induces Apoptosis in Mammalian Skeletal Muscle Fibers. Poster Discussion, Local Anesthesia and Pain, Basic Science II, ASA. 2002:A-971

Chapter 2

Regional Nerve Blocks and Infiltration Therapy in Clinical Practice

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_2, © Springer International Publishing Switzerland 2015

Introduction

Regional anesthesia means the interruption of impulse conduction in the nerves using specific, reversibly acting drugs (local anesthetics). This interruption of impulse conduction can be carried out in every region of the body in which the nerves are accessible for external injection.

The indications for regional anesthesia include:

1. Clinical anesthesia

   Particularly in the fields of traumatology, orthopedics, urology, and gynecology, as well as in large-scale abdominal surgery with continuous procedures for epidural or spinal anesthesia
2. Obstetrics
3. Postoperative analgesia

   There is no postoperative analgesia procedure that is more appropriate than regional anesthesia. This field also includes the classic indications for a combination of local anesthetics with opioids or other substances.

   Optimal patient care can only be achieved using a multimodal approach (effective pain therapy, early mobilization, early enteral nutrition, and emotional and psychological care). Effective pain therapy (e.g., with catheter analgesia procedures) plays a central role here, as it can substantially reduce the perioperative stress response (Table 2.1).
4. Pain therapy

In 1979, a commission set up by the International Association for the Study of Pain (IASP) defined pain as "… an unpleasant sensory and emotional experience, linked to actual or potential tissue damage" [1].

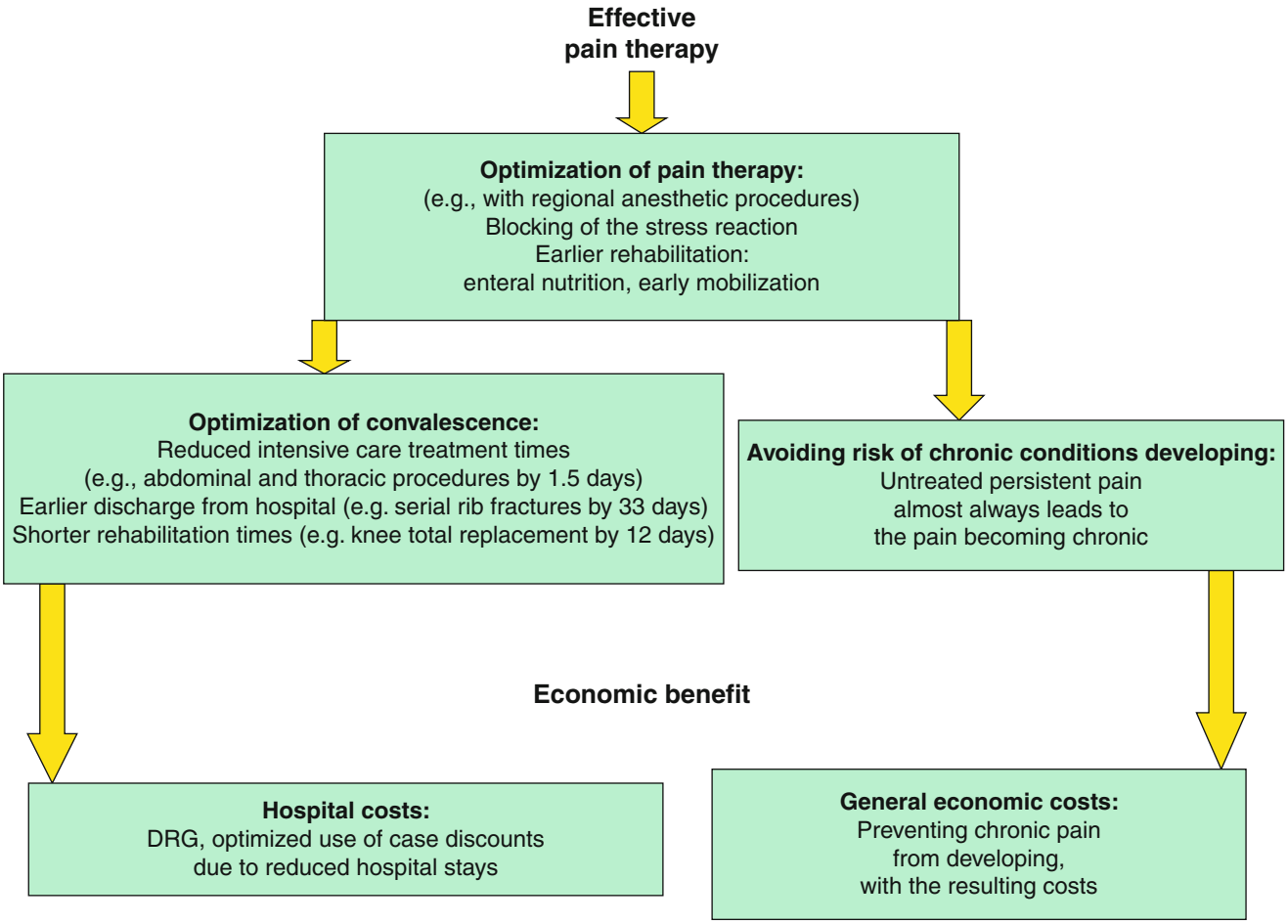
Acute pain is caused by stimulation of pain receptors. This stimulation is transient and sets in motion biologically useful protective mechanisms. Ideally, pain can be relieved by treating the cause. Chronic pain is regarded as a pathological response on the part of the body. The relief of acute pain should be viewed as a "human right." In this context, there are a significant number of patients who will not obtain effective pain relief without access to potent neural blockade techniques.

It arises due to constant stimulation of nociceptive afferents or can develop as neuropathic pain after injury or damage to the peripheral nociceptive system [2, 3, 5, 6].

Chronic pain can often lead to alterations in patients' living habits, physical abilities, and personality and requires a coordinated interdisciplinary approach. This in turn presupposes a clear diagnosis, based on a full general history and pain history, physical examination, and functional assessment of the patient's musculature, locomotor apparatus, autonomic nervous system, and neurological and angiological situation.

In addition to medical treatment for pain, nerve blocks have a firmly established place in pain therapy—alongside physical and manual procedures, neurological and neurosurgical methods, physiotherapy, and the psychosocial management of patients. In quantitative terms, regional anesthesia procedures play only a minor part in the management of chronic pain, but qualitatively they can produce very good results when used with the correct indications.

Table 2.1 Importance of effective pain therapy as part of a multimodal approach to treatment



Nerve Blocks in Surgery and Pain Therapy (Table 2.2)

Table 2.2 Important rules to observe when administering regional anesthesia or therapeutic nerve blocks

Before the block
Patient 1. **Preoperative information** Explain the procedure Discuss potential side effects and complications Advise the patient about what to do after the procedure Document the discussion 2. **Determine the patient's neurological status** Exclude neurological abnormalities 3. Exclude *contraindications* 4. Avoid *premedication* in outpatients (particularly in blocks in which there is an increased risk of intravascular injection—e.g., stellate ganglion or superior cervical ganglion)
Anatomy, complications, and side effects 1. With rarely used regional blocks, the *anatomic* and *technical* aspects should always be studied again beforehand 2. Detailed knowledge of potential *complications* and *side effects* of a regional block and how to avoid them 3. *Ability to control* potential complications and side effects 4. Select the *correct block techniques* 5. *Manual skill* and *good training* on the part of the anesthetist
Preparation
1. Ensure *optimal positioning* of the patient 2. Always secure *intravenous access* 3. Check that *emergency equipment* is complete and fully functioning 4. *Added vasopressors* are *contraindicated* in pain therapy 5. Observe *sterile precautions*
Safety standards when injecting larger doses of local anesthetics
1. Carry out *aspiration tests* before and during the injection 2. Administer a *test dose* 3. Inject local anesthetics in *incremental doses* (several test doses) 4. Maintain *verbal contact* with the patient 5. *Cardiovascular monitoring* 6. Keep careful *notes* of the block

The application of the anesthesiological methods described in the subsequent chapters of this book for temporary interruption of stimulus conduction in a nerve or nerve plexus requires the use of strictly established indications and the implementation of a coordinated therapeutic approach. In principle, these blocks can be administered for surgery, diagnosis, prognosis, and therapy [1].

Surgical blocks are administered with high-dose local anesthetics for targeted isolation of a specific body region in order to carry out an operation.

Diagnostic blocks using low-dose local anesthetics are appropriate for the differential diagnosis of pain syndromes. They allow the affected conduction pathways to be recognized and provide evidence regarding the causes of the pain. Diagnostic blocks can also be used to clarify the question of whether the source of the pain is peripheral or central.

Prognostic blocks allow predictions to be made regarding the potential efficacy of a longer-term nerve block, neurolysis, or surgical sympathectomy. They should also be used to prepare the patient for the effects of a permanent block.

Therapeutic blocks are used in the treatment of a wide variety of pain conditions. Typical examples of these are post-traumatic and postoperative pain, complex regional pain syndrome (CRPS) types I and II (reflex sympathetic dystrophy and causalgia), joint mobilization, postherpetic neuralgia, and tumor pain.

Nerve Blocks and Chronic Pain [4]

A multimodal treatment approach to chronic pain is essential for successful treatment. The use of nerve blocks as part of this approach presupposes that the following steps have been taken:

1. Careful analysis of the pain
2. Correct diagnosis and establishment of the indication
3. Assessment of the pain chronicity stage
4. Well-selected patient groups

Important preconditions for the application of nerve blocks in chronic pain include:

1. A good knowledge of anatomy
2. Attention to and control of potential side effects and complications
3. Choice of the correct block techniques
4. Manual skill and good training on the part of the therapist

The most important tasks facing us include conducting more double-blind, randomized, and well-controlled studies on the use of nerve blocks in chronic pain conditions and developing a consistent standard for carrying out nerve blocks. The answers to the two questions need to be found:

1. Selection criteria to identify which patients are suitable for nerve blocks
2. The number of nerve blocks to be used in the treatment of chronic pain

Technical Requirements

Carrying out temporary nerve blocks and regional anesthetic procedures in surgery and pain therapy requires the appropriate basic technical equipment and experience in the use of all of the instruments concerned.

The conditions for patient positioning, the aseptic conditions required, and the syringes, needle types, and other supplies needed are discussed alongside the individual block techniques described in this book. Complete and properly functioning equipment must be available both for primary care and in case of adverse events and complications, as well as treatment monitoring.

Accessories for Primary Care

Emergency Equipment (Figs. 2.1 and 2.2)

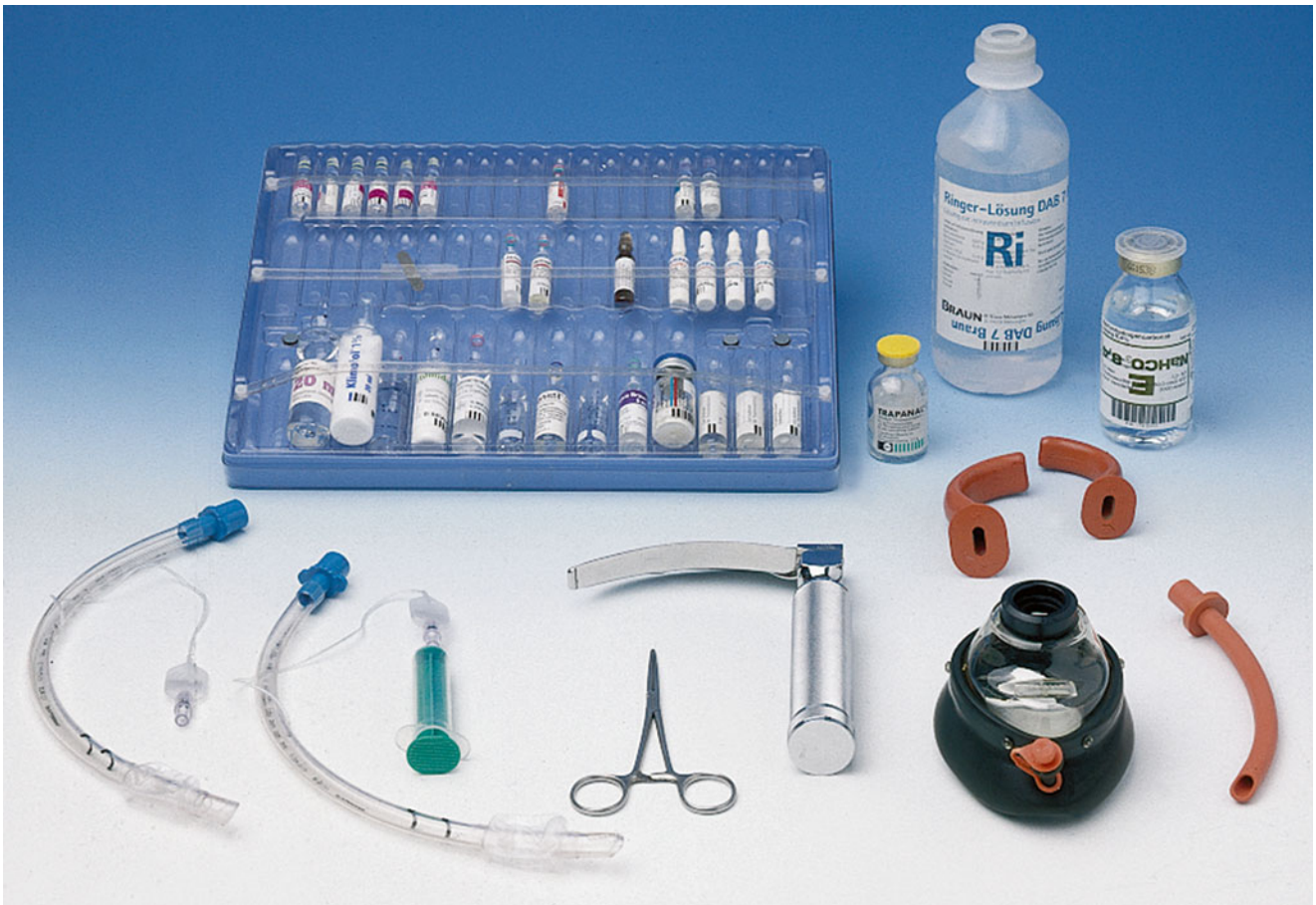


Fig. 2.1 Emergency equipment

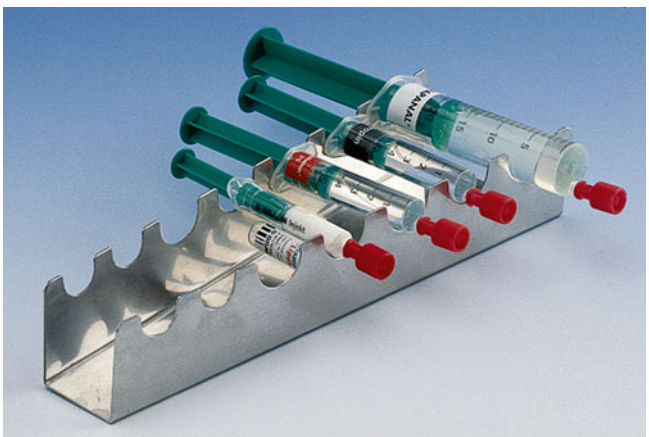


Fig. 2.2 Emergency drugs

- Intubation and ventilation facilities
- Oxygen source (breathing apparatus)
- Ventilation bag with two masks (large, medium)
- Guedel tubes nos. 3, 4, and 5
- Wendel tubes nos. 26–32
- Endotracheal tubes nos. 28–36
- Tube clamp and blocker syringe (10 mL)
- Laryngoscope with batteries (replacement batteries and replacement bulbs) and spatula
- Magill forceps, mouth wedge, and 1 tube of 2 % lidocaine gel
- Suction device
- Infusion equipment
- Two sets of infusion instruments
- Five plastic indwelling catheters
- Syringes (2L, 5, 10 mL), plaster, and gauze bandages

- Infusion solutions
- 1 bottle each of Ringer's solution, plasma expander, and 8.4 % sodium bicarbonate (100 mL)
- Defibrillator (Fig. 2.3)
- Drugs for emergency treatment

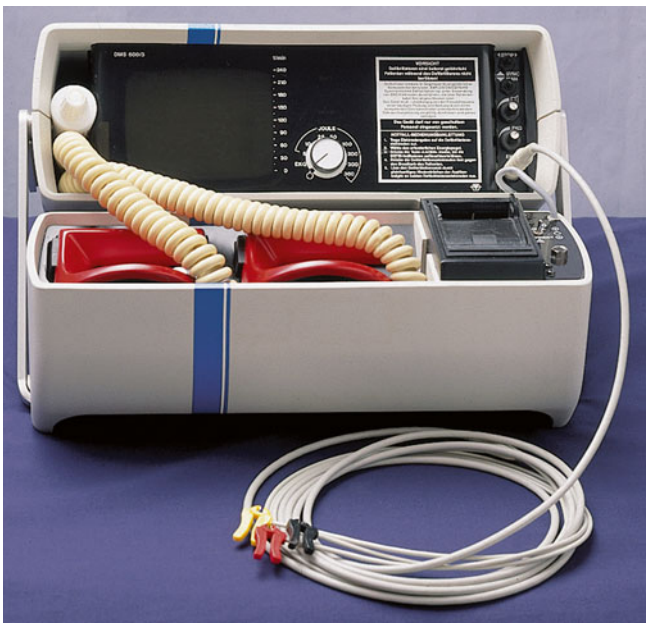


Fig. 2.3 Defibrillator

When blocks are being administered, a sedative (Diazepam), a vasopressor, and a vagolytic (atropine) should be available for immediate injection. All other emergency medications should also be on hand:

- 5 ampoules of atropine
- 3 ampoules of 0.1 % epinephrine (1:1,000)
- 2 prepared syringes of epinephrine (1:10 000, 10 mL)
- 1 ampoule of 10 % calcium gluconate
- 1 ampoule of dimethindene maleate (Fenistil®)
- Methylprednisolone (Solu-Decortin®) (50, 250, 1,000 mg)
- 5 ampoules of 0.9 % sodium chloride
- 5 ampoules of diazepam (10 mg)
- 2 ampoules of midazolam (Dormicum®) (5 mg)
- 1 ampoule of clonazepam (Rivotril®) (1 mg)
- 1 injection bottle of thiopental sodium
- 2 ampoules of propofol
- 2 ampoules of succinylcholine (suxamethonium chloride)
- 20 % lipid emulsion

Anesthetic Machine

For neuraxial anesthesia, ganglion blocks, intravenous regional anesthesia, and plexus anesthesia, an anesthesia trolley with facilities for intubation is also required (Fig. 2.4).

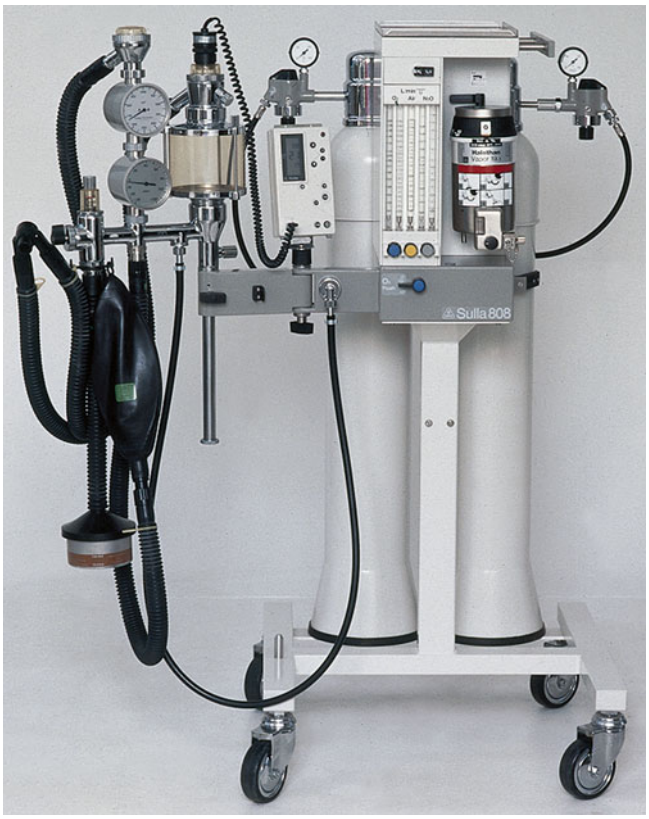


Fig. 2.4 Anesthetic machine

Monitoring

- Electrocardiogram (ECG).
- Pulse oximeter (Fig. 2.5).
- Electrostimulator (Fig. 2.6). Peripheral nerve stimulation is a valuable aid in clinical practice and has considerable advantages in combination with an atraumatic catheter technique.

Fig. 2.5 ECG and pulse oximeter

Fig. 2.6 Electrostimulation device

The safety functions that a modern nerve stimulator should have include:

- A display showing differences between the actual and target current. An alarm alerts the user that the stimulation current that has been set is not flowing at the full level due to high resistance in the system and can therefore no longer be used as a reliable indicator of the distance between the nerve and the tip of the needle (screenshot 1).
- Alarm for the threshold current value. The stimulator should warn the user when the current falls below the threshold value (e.g., 0.30 mA at 0.1 ms), to avoid the risk of nerve injury by the needle tip or even intraneural injection (screenshot 2).
- Adjustment of current when the impulse duration is altered.
- The stimulator should automatically reduce the impulse amplitude—e.g., when the impulse duration is increased—in order to avoid a sudden increase in the energy released into the patient's body.
- Electrodes that can be connected to nerve stimulators, such as the Stimuplex Pen (B. Braun Melsungen), are suitable for transcutaneous stimulation of superficial nerves such as the femoral nerve. This is particularly useful during the training period for specialists, but it can also be used for preliminary localization immediately before puncture (Fig. 2.7).
- Temperature sensor, touch-free miniature infrared skin thermometer (Fig. 2.8).

Ultrasound (Fig. 2.9)

In recent years, the field of regional anesthesia, and in particular peripheral nerve blockade, has entered an unprecedented renaissance. This renaissance is due primarily to the widespread introduction of ultrasound-guided regional anesthesia. The ability to visualize the anatomy of interest, the needle–nerve relationship, and the spread of the local anesthetic has resulted in significant growth of interest in and use of peripheral, sympathetic, and neuraxial blocks.

The treatment of side effects and severe complications—e.g., after inadvertent intravascular, epidural, or subarachnoid injection of a local anesthetic—is discussed here together with the individual block techniques.

Fig. 2.7 The Stimuplex Pen for transcutaneous nerve stimulation

Fig. 2.8 Temperature sensor

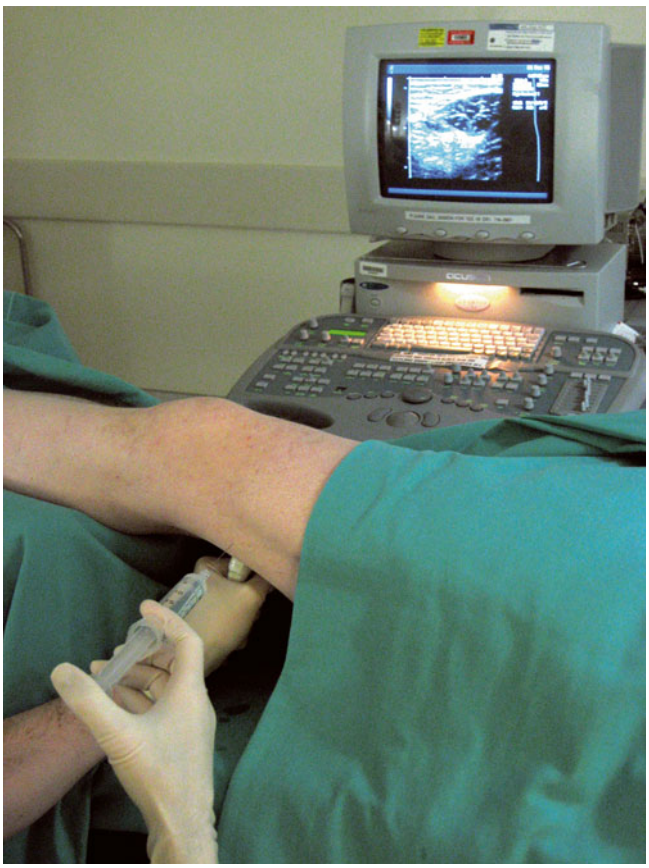


Fig. 2.9 Ultrasound machine

References

1. Bonica JJ, Buckley PF. Regional analgesia with local anesthetics. In: Bonica JJ, editor. Management of pain. 2nd ed. Philadelphia/ London: Lea & Febiger; 1990.
2. Campbell JN, Raja SN, Cohen RH, Manning D, Khan A, Meyer RA. Peripheral neural mechanisms of nociception. In: Wall PD, Melzack R, editors. Textbook of pain. 2nd ed. Edinburgh: Churchill Livingstone; 1989.
3. Devor M. Central changes mediating neuropathic pain. In: Dubner R, Gebhart GF, Bond MR (editors). Proceedings of the Vth World Congress on Pain. Pain Research and Clinical Management. Amsterdam: Elsevier; 1988. p. 7.
4. Jankovic D. New approaches to chronic pain: nerve blocks in the head. In: Van Zundert A, Rawal N (editors). Highlights in regional anesthesia and pain therapy. XI (Special Edition: World Congress on Regional Anaesthesia and Pain Therapy, Barcelona); 2002. p. 258–69.
5. Weninger E. Pharmakodynamik der Lokalanästhetika. Anästhesiologie Intensivmedizin. 1996;5(37):249–67.
6. Zimmermann M. Physiologie von Nozizeption und Schmerz. In: Zimmermann M, Handwerker HO, editors. Schmerz-Konzepte und ärztliches Handeln. Berlin: Springer; 1984.

Chapter 3

Basics of Ultrasound Imaging

Herman Sehmbi, MBBS, MD, EDAIC, EDRA • Anahi Perlas, MD, FRCPC (✉)
Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada
e-mail: hermansehmbi@gmail.com; anahi.perlas@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_3,

Basics of Ultrasound Physics

Introduction

Since the initial description of the use of ultrasound to assist brachial plexus block by Dr. LaGrange in 1978, there has been a vast expansion in the field of "ultrasound-guided regional anesthesia (UGRA)" [1]. The last decade has seen major strides in both technological advances and its wide clinical acceptance [2]. Ultrasound provides real-time image guidance for many regional anesthesia and pain interventional procedures. Ultrasound is the most practical tool as it is easy to learn, portable, moderately priced, and devoid of radiation risks [3]. It has been shown to hasten block onset, to improve success rate, and to be cost-effective [4, 5]. This chapter provides a brief overview of some of the fundamental principles and physics underlying ultrasound technology that are relevant to regional anesthesiologists, as well as some of its limitations and pitfalls.

Characteristics of an Ultrasound Wave

An ultrasound wave is a *non-audible sound wave* (with frequencies above 20 kHz). A sound wave is a pressure wave of mechanical energy that is transmitted through a medium by vibration of molecules. It can be transmitted as a *longitudinal wave* (Fig. 3.1), which refers to the oscillation of molecules in a linear direction (in mediums such as gases, liquids, or plasma), or *transverse wave* (Fig. 3.2), which refers to oscillation of molecules perpendicular to the direction of the propagated wave (such as in solids) [6]. Unlike light, it cannot travel through vacuum.

An ultrasound wave has the following basic parameters (Fig. 3.2) [7]:

(a) Wavelength (λ) is the spatial period of the wave, i.e., the distance over which the wave's shape repeats. It is determined by measuring the distance between two consecutive corresponding points of the same phase. It is expressed in *meters* (m).
(b) Amplitude (A) is a measure of the height of the wave, i.e., maximal particle displacement. It is expressed in *meters* (m).
(c) Period (T) is the time taken for one complete wave cycle to occur. The unit of period is *seconds* (s).
(d) Frequency (f) is the number of completed cycles per second. Thus, it is the inverse of the period (T) of a wave. The unit of frequency is *hertz* (Hz). Medical imaging uses high-frequency waves (1–20 MHz).
(e) Velocity (c) is the speed of propagation of a sound wave through a medium. The velocity (c) of a wave is the product of its frequency (f) and wavelength (λ) of a sound wave. The unit of velocity is *meters per seconds* (m/s). The speed of sound in biological medium is assumed to be constant at 1,540 m/s for practical purposes. However, it varies greatly, being as low as 330 m/s in air and as high as 4,000 m/s through bone.
(f) Energy (E) of a sound wave is proportional to the square of its amplitude (A). This means that as the amplitude of a wave decreases (such as with deeper penetration), the energy carried by the wave reduces drastically.
(g) Power (P) of a sound wave is the energy (E) delivered per unit time (t).

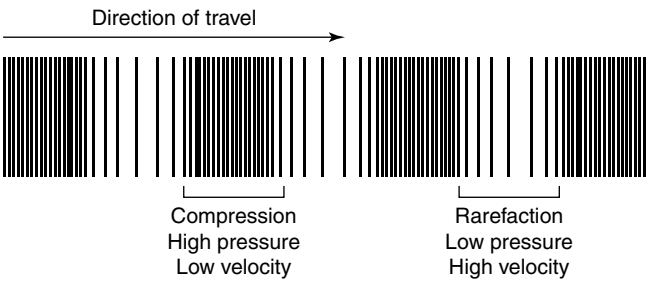


Fig. 3.1 Characteristics of a longitudinal sound wave (Reproduced with permission from www.regionalfortrainees.com)

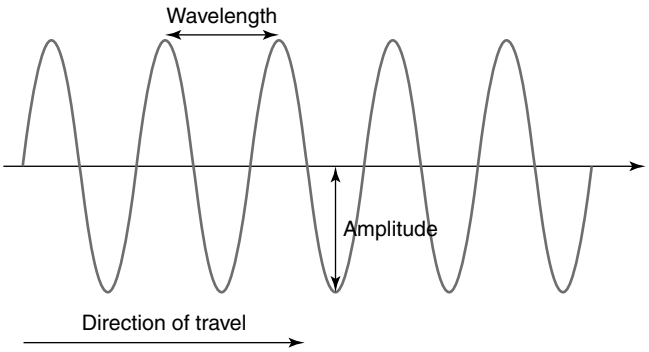


Fig. 3.2 Characteristics of a transverse sound wave (Reproduced with permission from www.regionalfortrainees.com)

Generation of Ultrasound Waves

Piezoelectric crystals (e.g., quartz) in an ultrasound transducer produce a small potential difference across them when compressed or under tension. The ability to covert mechanical energy into electrical energy is termed the *piezoelectric effect* (Fig. 3.3a) [8]. This was first demonstrated by *Pierre Curie* and *Jacques Curie* in 1880 [8]. *This effect is used in image generation when returning ultrasound waves apply mechanical energy to the crystals which in turn generate an electrical signal.*

The converse, i.e., the application of electrical energy across a piezoelectric crystal, causes mechanical distortion of the crystals resulting in their vibration and generation of sound waves. This phenomenon, termed the *reverse piezoelectric effect* (Fig. 3.3b), was first proposed by *Gabriel Lippmann* in 1881. *This property is utilized to generate ultrasound waves from transducers* [9].

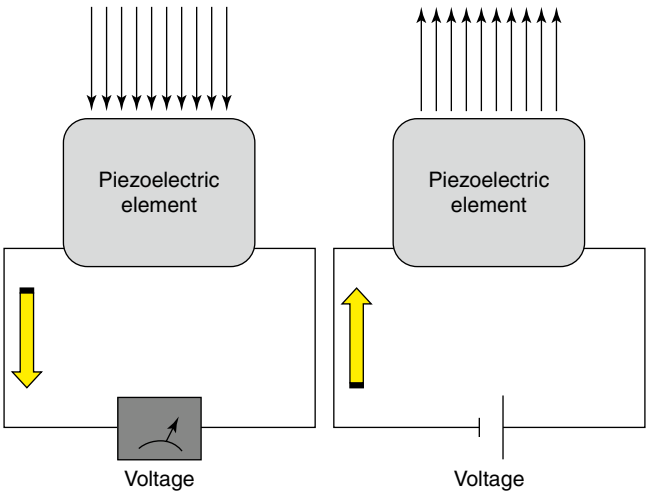


Fig. 3.3 (**a**) The piezoelectric effect. (**b**) The reverse piezoelectric effect (Reproduced with permission from www.regionalfortrainees.com)

Structure of an Ultrasound Transducer

An ultrasound transducer has a dual functionality. It is responsible for both the production of ultrasound waves and, after a set period of time, the reception of waves reflected from the tissues. This is called *pulsed ultrasound*. The *pulse repetition frequency* (PRF) is the number of pulses emitted by the transducer per unit of time [10]. The PRF for medical imaging devices ranges from 1 to 10 kHz (Fig. 3.4).

The ultrasound transducer has the following layers (Fig. 3.5) [11]:

(a) *Backing material*: located behind the piezoelectric element, it serves to prevent excessive vibration. This causes the element to generate ultrasonic waves with a shorter pulse length, improving axial resolution in images.
(b) *Piezoelectric elements*: they generate ultrasonic waves and also generate images. Piezoelectric ceramic (PZT: lead zirconate titanate) is most commonly used because of their high conversion efficiency [12].
(c) *Acoustic matching layer*: this reduces the acoustic impedance mismatch between the transducer and the object, minimizing reflection off the interface [13]. This is usually made up of a resin.
(d) *Acoustic lens*: the acoustic lens prevents the ultrasonic waves from spreading and focuses them in the slice direction to improve the resolution.

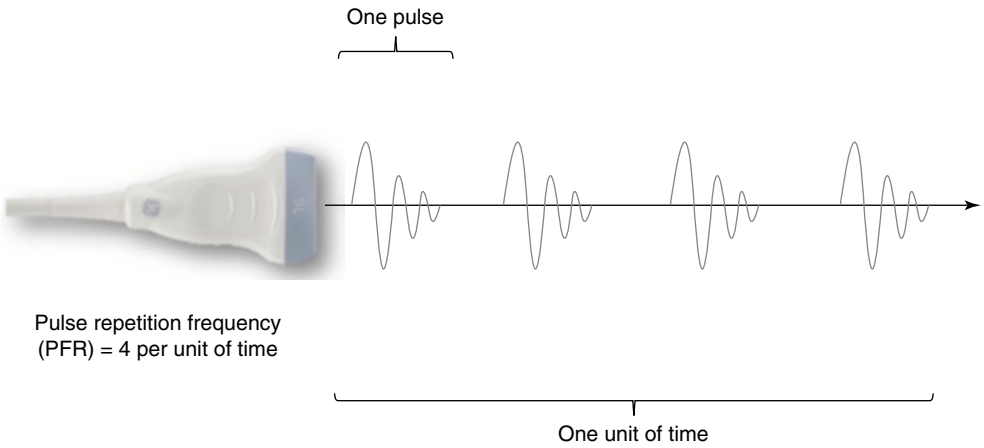


Fig. 3.4 Pulse repetition frequency (Reproduced with permission from www.regional-fortrainees.com)

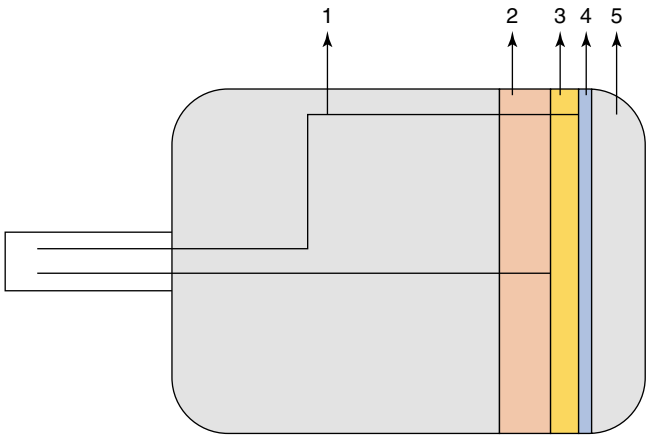


Fig. 3.5 Layers of an ultrasound transducer (Reproduced with permission from www.regionalfortrainees.com)

Interaction of Ultrasound with Tissues

The final image on the screen of an ultrasound machine is the result of the interaction of ultrasound waves with the tissues being examined. As the ultrasound wave travels through the tissues, it loses amplitude (and hence energy). This reduction in amplitude is called *attenuation*. Attenuation is the summative effect of absorption (conversion of acoustic energy to heat), reflection, and refraction of ultrasound waves [7].

Absorption

Absorption is the process of transfer of the ultrasound beam's energy to the medium through which it travels through *heat generation* and it accounts for most of the wave attenuation.

Reflection

When an incident ultrasound pulse encounters an interface of two body tissues with different acoustic impedances, the sound energy is reflected back to the transducer. When the size of the object is smaller than the wavelength of the beam (e.g., red blood cells) and/or the surface of a structure not smooth, only a part of it is reflected back to the transducer with a proportion of the waves being scattered in multiple directions (*diffuse scattering*). However, when the size of the object exceeds the wavelength of the incident beam and the surface is smooth (e.g., a fascial plane or the surface of the pleura), most of the beam is reflected back to the transducer (*specular reflection*, Fig. 3.6).

If the incident ultrasound wave approaches the linear interface at 90°, almost all of the generated echo will travel back to the transducer. However, if the angle of incidence with the specular boundary is less than 90°, the echo will not return to the transducer but rather be reflected at an angle equal to the angle of incidence. The returning echo will potentially miss the transducer and not be detected. This explains why needles inserted at a steep angle may not be well visualized on the ultrasound image (Fig. 3.7). Some needle manufacturers have introduced needles with "cornerstone reflectors" in their design, such that the indentations on their surface maximize the proportion of waves that are reflected back to the transducer. This makes such needles more "echogenic" [14, 15].

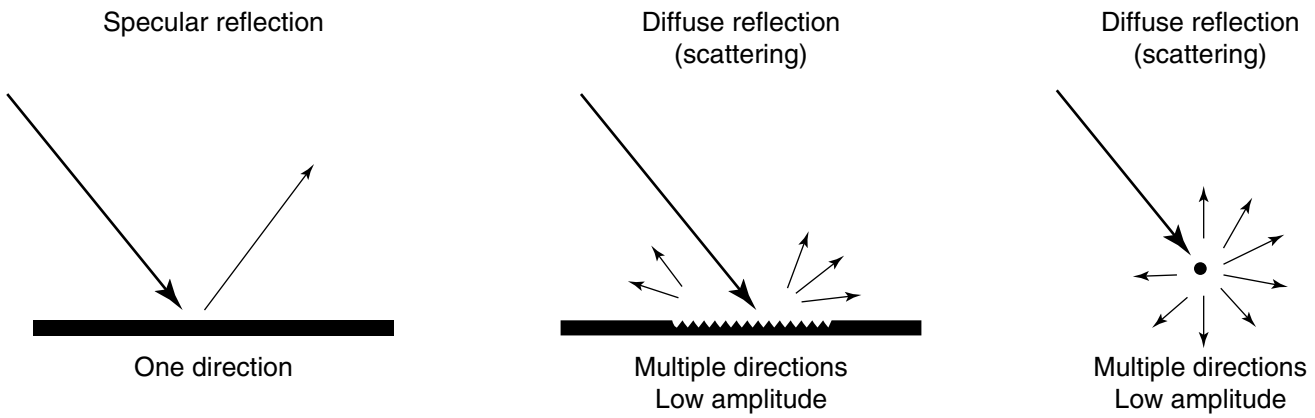


Fig. 3.6 Types of reflection (Reproduced with permission from www.usra.ca)

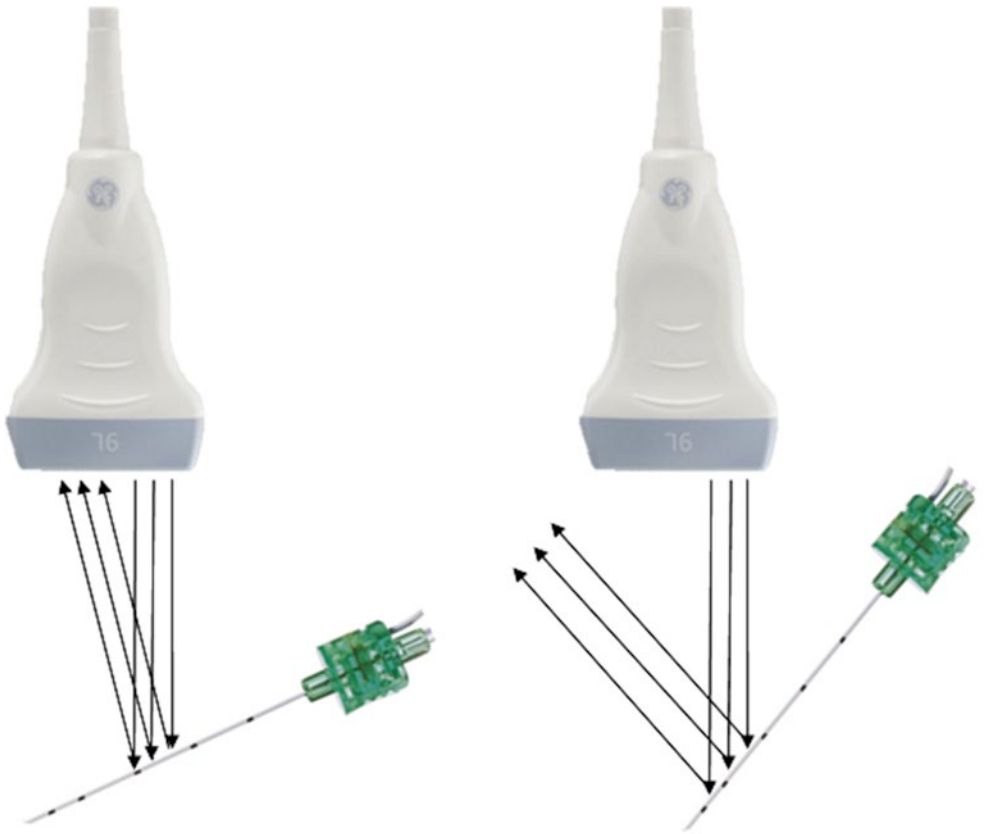


Fig. 3.7 Effect of angle of needle insertion on acquisition of reflected ultrasound waves (Reproduced with permission from www.regionalfortrainees.com)

Refraction

Refraction refers to a change in the direction of the sound after reaching an interface of two tissues with different speeds of sound transmission. It is dependent upon the *change in velocity* and not due to differences in acoustic impedance (Fig. 3.8). Refraction is one of the most frequent causes of artifacts on an ultrasound image. It is described by *Snell's law*, where *θi* is the angle of incidence, *θt* is the angle of transmission, *Ci* is the velocity in the first medium, and *Ct* is the velocity in the second medium such that [16]

$$\frac{\sin\theta_i}{\sin\theta_t} = \frac{C_i}{C_t}$$

Attenuation

The degree to which a given ultrasound wave is attenuated depends on a number of factors:

(i) The *type of tissue*: attenuation is greater for denser tissues such as bone and lower for fluid mediums such as blood- or fluid-containing cysts or cavities.

(ii) *Frequency* of the ultrasound wave: the degree of attenuation for a given unit of distance is greater for waves of higher frequency. This explains why higher-frequency transducers (commonly up to 13–15 MHz) are mostly used to image superficial structures (up to 5–6 cm deep) while lower-frequency transducers are required to image deeper structures.

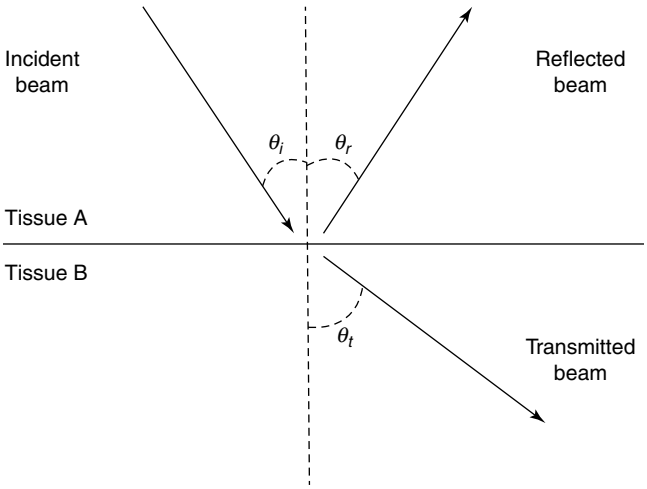


Fig. 3.8 Refraction across two mediums (Reproduced with permission from www.regionalfortrainees.com)

(iii) *Distance* traveled: for any ultrasound wave, the degree of attenuation is directly proportional to the distance traveled (Fig. 3.9).

Acoustic Impedance

The *acoustic impedance* (*Z*) is the degree to which medium particles would resist change due to mechanical disturbance and is a product of the density (ρ) of the medium and the acoustic velocity (c) of the ultrasound wave [17]. The amount of reflection depends upon the relative changes in the acoustic impedance (resistance at the interface) between the two tissues (or mediums). This is determined by the *reflection coefficient* (*R*). Large difference in acoustic impedance occurs at air/soft tissue and bone/soft tissue interface causing very high reflection at such sites. This makes bone cortex appear white while air appears black. This is also why a gel is needed at the transducer to eliminate air/soft tissue interface to obtain any image.

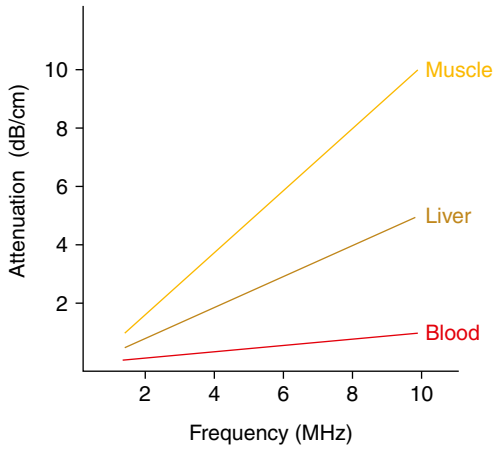


Fig. 3.9 Relationship of attenuation with tissue type and frequency (Reproduced with permission from www.usra.ca)

Image Acquisition and Processing

The echo signals returning from the tissues reach the crystals and produce an electric current (piezoelectric effect). This is then converted to a pixel on the image, with the energy of the returning wave being proportional to the brightness of the pixel dot on the image. However, this involves these additional processes [18]:

(a) *Amplification* (*gain*): the amplitude of the returning echo signals is very small to be properly displayed on a screen. Hence, it needs amplification. However, amplification adds to "background noise."
(b) *Time gain compensation*: because the ultrasound waves undergo attenuation, the intensity (and hence brightness) of the returning waves from deeper tissues is much lower than the ones returning from the nearer tissues. This is overcome by preferential enhancement of signals returning from deeper tissues, the so-called time gain compensation such that echoes returning from similar reflectors are represented by the same shade of gray regardless of their depth (Fig. 3.10a, b) [19].
(c) *Analog to digital conversion*: this eliminates external interference and, more importantly, allows for signal processing to improve image quality.
(d) *Post-processing*: this involves techniques such as frame averaging, gray-level transfer curves, edge enhancement, and adaptive image processing to yield the final image on a display.

The final image allows structures to be represented according to their brightness or echogenicity as the following [10]:

(a) *Hyperechoic*: areas with returning echoes of higher energy than their surrounding structures, appearing whiter (such as peripheral nerves and muscle tendons) (Fig. 3.11a)
(b) *Hypoechoic*: areas with returning echoes of lower energy than their surrounding structures, appearing as gray (such as proximally located spinal nerve roots) (Fig. 3.11b)
(c) *Anechoic*: areas with no returning echoes, appearing as black (such as the fluid within vessels) (Fig. 3.11c)

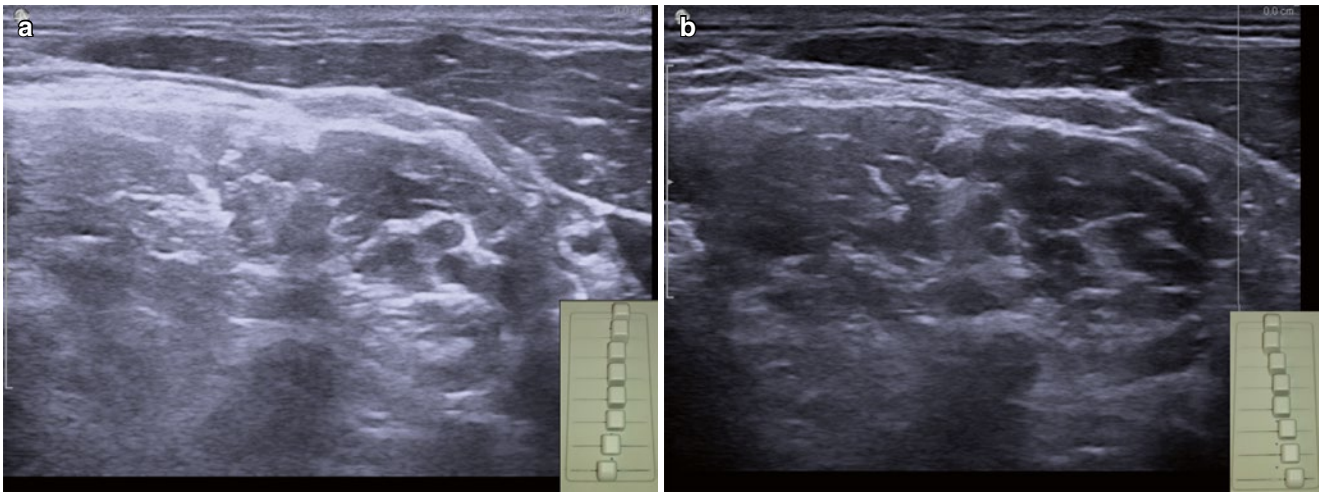


Fig. 3.10 Time gain compensation (**a**) not applied (**b**) and correctly applied (Reproduced with permission from www.regionalfortrainees.com)

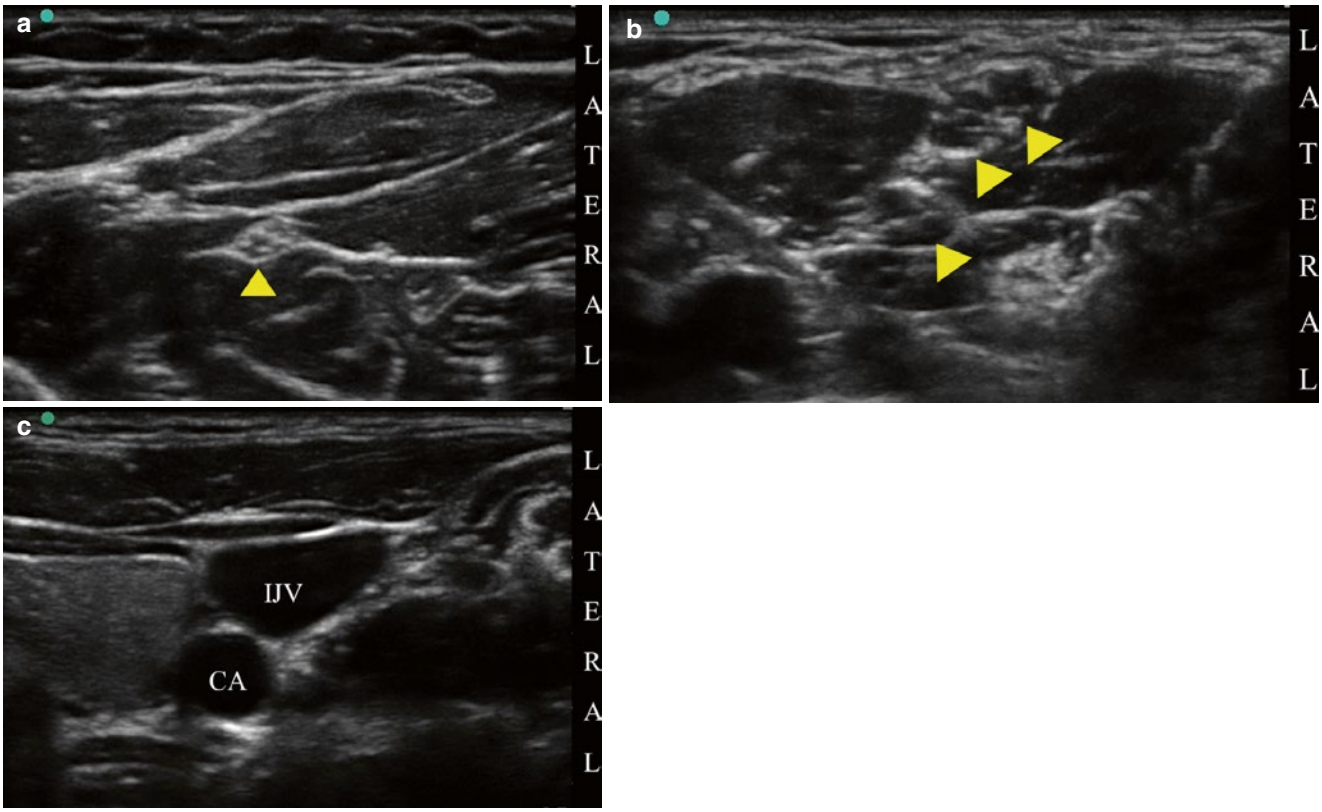


Fig. 3.11 Structures of different echogenicity. (**a**) Hyperechoic peripheral nerves (*yellow arrowhead* median *n*). (**b**) Hypoechoic cervical nerve roots (*yellow arrowhead* cervical nerve roots). (**c**) Anechoic vein and artery (*IJV* internal jugular vein, *CA* carotid artery in the neck) (Reproduced with permission from www.regionalfortrainees.com)

Ultrasound Transducer Characteristics

Ultrasound transducers can be classified by their shape. The most commonly used probes for UGRA are linear array and curved array probes [20].

Linear Array Probe

These have a linear footprint (Fig. 3.12a). They are high-frequency probes (8–12 MHz) and best reserved for imaging superficial structures (such as the brachial plexus). They provide a square or rectangular field of view (Fig. 3.12b).

Curved Array Probes

These have a curved footprint (Fig. 3.13a). They are low-frequency probes (1–5 MHz) and best reserved for imaging deeper structures (such as the sciatic nerve in the gluteal area). They provide a sectorial field of view (Fig. 3.13b).

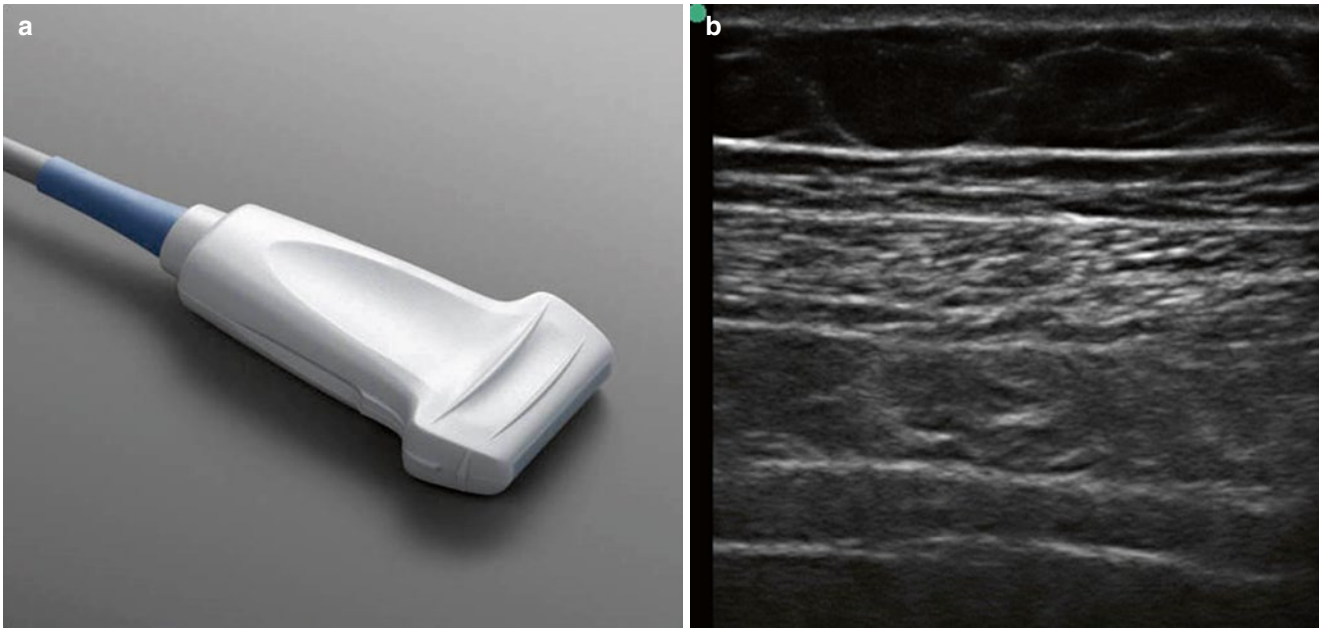


Fig. 3.12 (**a**) A linear array probe. (**b**) Field of view of a linear probe (Reproduced with permission from www.regionalfortrainees.com)

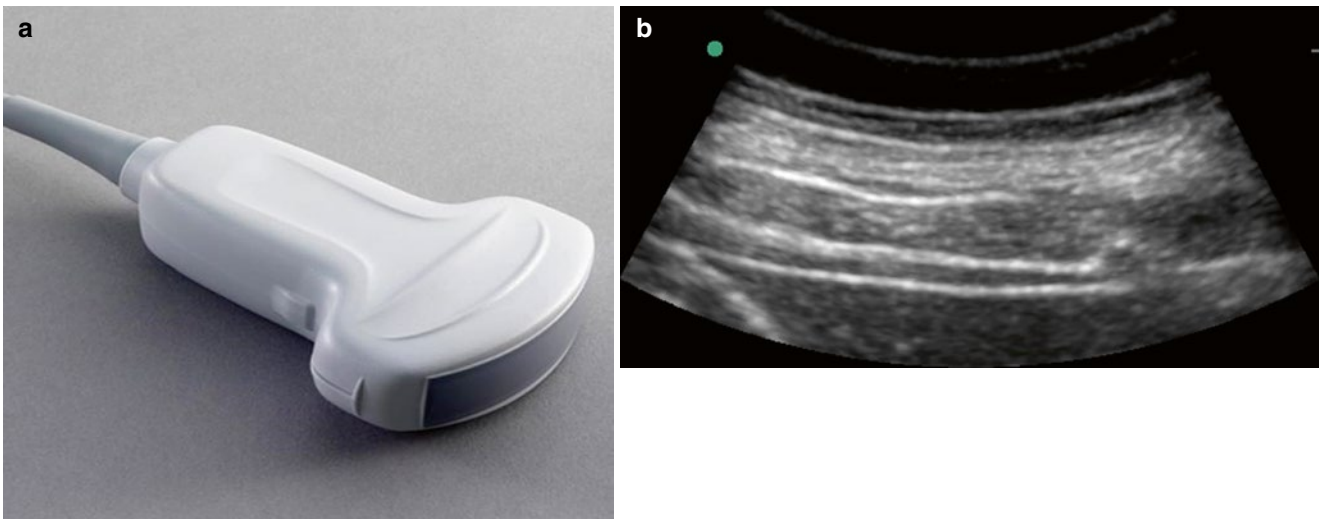


Fig. 3.13 (**a**) A curved array probe. (**b**) Field of view of a curved array probe (Reproduced with permission from www.regionalfortrainees.com)

Resolution

Resolution of an ultrasound machine is its ability to distinguish two close objects as separate. In simple terms, it provides clarity to the ultrasound image [21–23]. Different types of resolution important for a regional anesthesiologist are:

(a) *Axial resolution* is the ability of the ultrasound machine to separate two structures lying at different depths, parallel to the direction of the ultrasound wave (Fig. 3.14a). It is half of the pulse width. Thus, high-frequency probes (shorter pulse width) have better axial resolution. However, they also have higher attenuation, resulting in poor penetration.

(b) *Lateral resolution* is the ability of the ultrasound machine to separate two structures lying at the same depth, perpendicular to the direction of the ultrasound wave (Fig. 3.14b). It is inversely related to the width of the ultrasound beam, which is lower with high-frequency probes.

(c) *Temporal resolution* is directly related to the frame rate of the ultrasound machine. To limit its impact, needle manipulation and injection of local anesthetic should be slow to minimize blur.

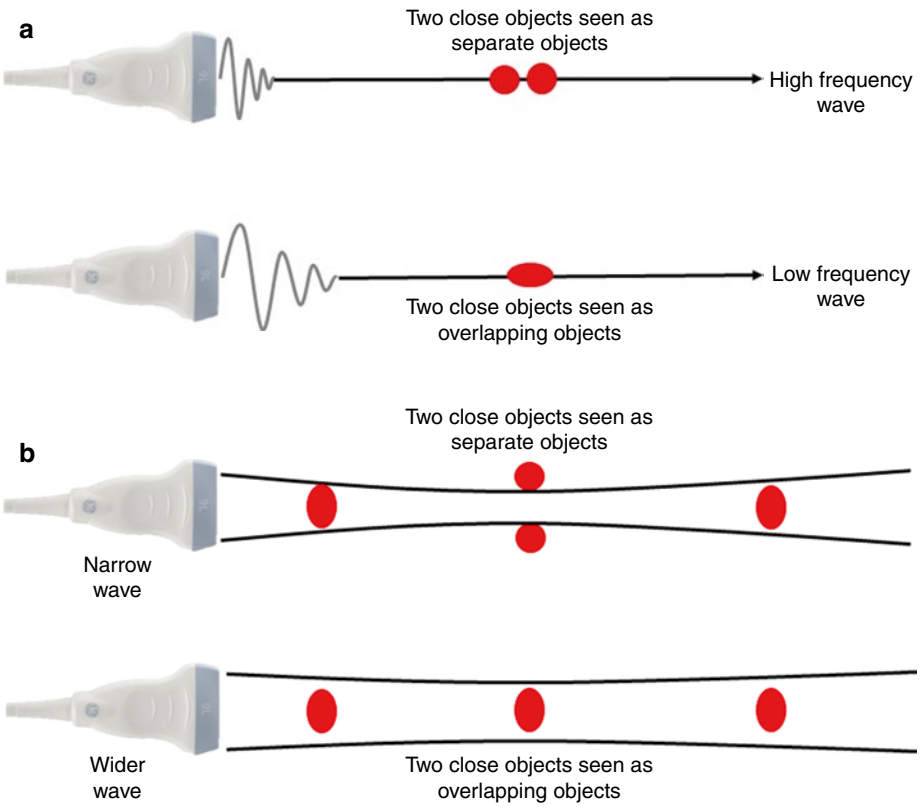


Fig. 3.14 (**a**) The effect of frequency of the transducer on the axial resolution (Reproduced with permission from www.regionalfortrainees.com). (**b**) The effect of width of the ultrasound beam on lateral resolution (Reproduced with permission from www.regionalfortrainees.com)

Image Optimization

The following steps can be considered to optimize ultrasound image acquisition:

Probe Selection

As discussed earlier, linear probes lend themselves to most superficial nerve blocks (such as brachial plexus block), while curvilinear probes are helpful to perform blocks at deeper sites (such as sciatic block at the subgluteal region). The footprint size of a given probe is also an essential consideration. While smaller footprint probes (e.g., 25-mm linear transducers) are better for use in pediatric population or smaller adults, they also reduce the field of view (Fig. 3.15). Curvilinear probes are also available in different footprint sizes.

Fig. 3.15 Smaller footprint linear transducer (Reproduced with permission from www.regionalfortrainees.com)

Frequency

Most probes are broadband transducers and are capable of allowing adjustment of their frequency (5–15 MHz). Selecting a higher frequency provides superior lateral resolution but at the expense of increased attenuation and poor penetration. Lowering the frequency of a given probe allows better visualization of deeper structures at the cost of resolution (Fig. 3.16).

Default Pre-optimized Modes

Most ultrasound units provide preset modes that can be customized for the user for commonly encountered clinical settings [24]. They represent the most common machine settings (depth, gain, focus, multi-beam, frequency range, frame rate) that are useful for a specific area.

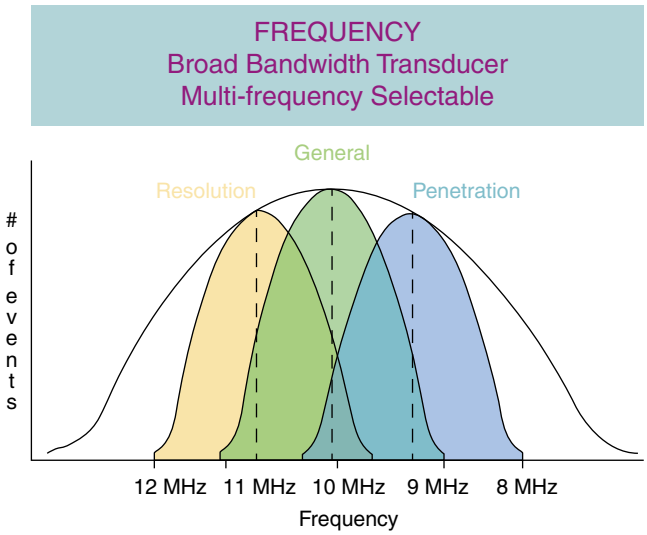


Fig. 3.16 The frequencies generated by linear broadband transducers (Reproduced with permission from www.usra.ca)

Gain

The gain setting allows the operator to change the brightness of the image. This happens through amplification (increasing gain) or dampening (decreasing gain) of the returning signals rendering the image more hyperechoic or hypoechoic, respectively. However, it also increases the background noise which may result in generation of artifacts (Fig. 3.17a–c).

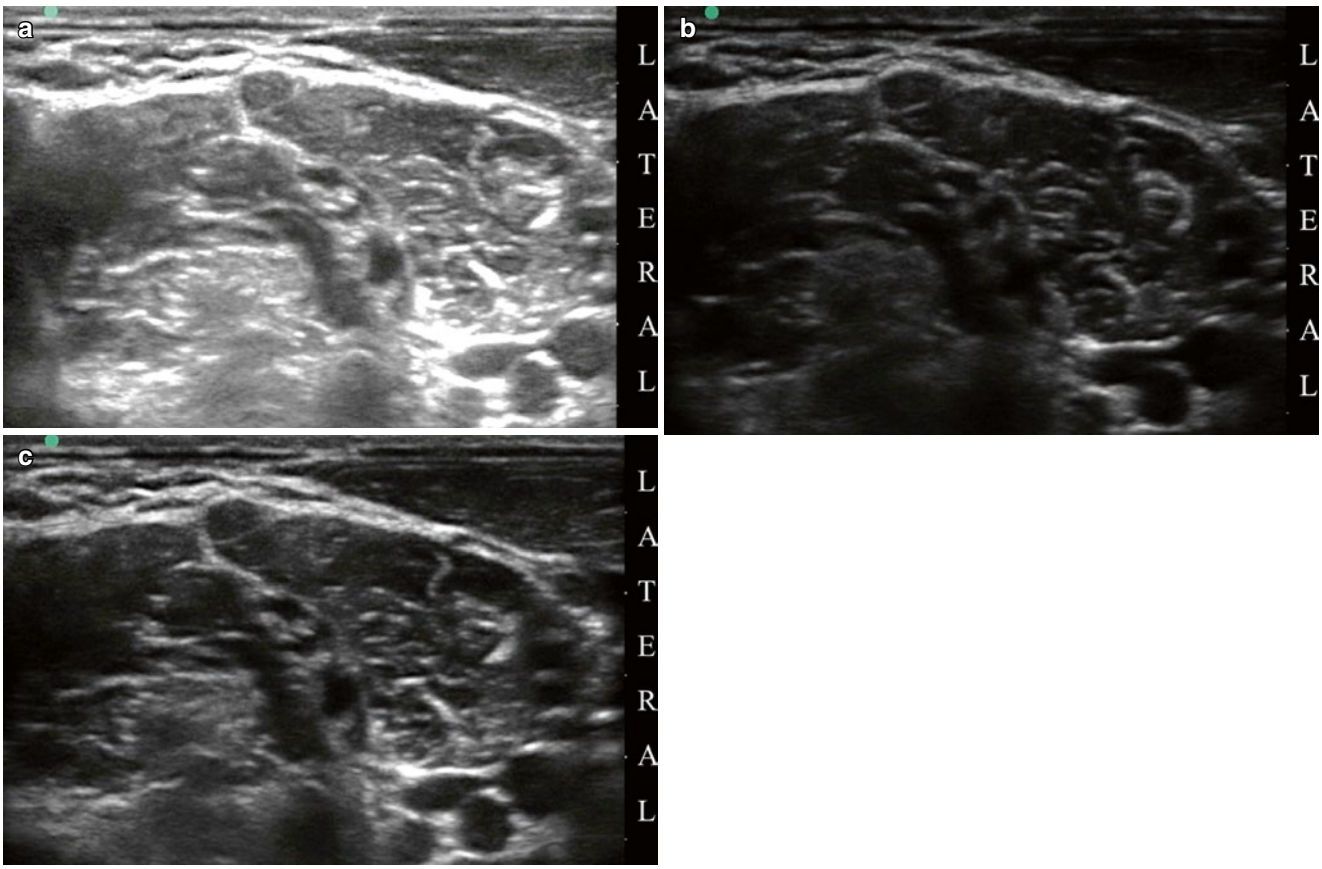


Fig. 3.17 Effects of too high (**a**), too low (**b**), and optimal gain (**c**) settings on the view at interscalene brachial plexus (Reproduced with permission from www.regionalfortrainees.com)

Time Gain Compensation (TGC)

While the signals returning from a superficial target undergo less attenuation, those returning from deeper structures undergo greater attenuation (Fig. 3.18). Therefore, an unoptimized image is generally brighter (representing superficial tissues) at the top than at the bottom (representing deeper tissues). "Time gain compensation" allows for selective amplification of signals at various depths [19]. Thus, increasingly, optimization of gain with increasing depth allows for a properly optimized image. Some machines have individual slots for TGC, while others allow adjustment of far/near/overall gain.

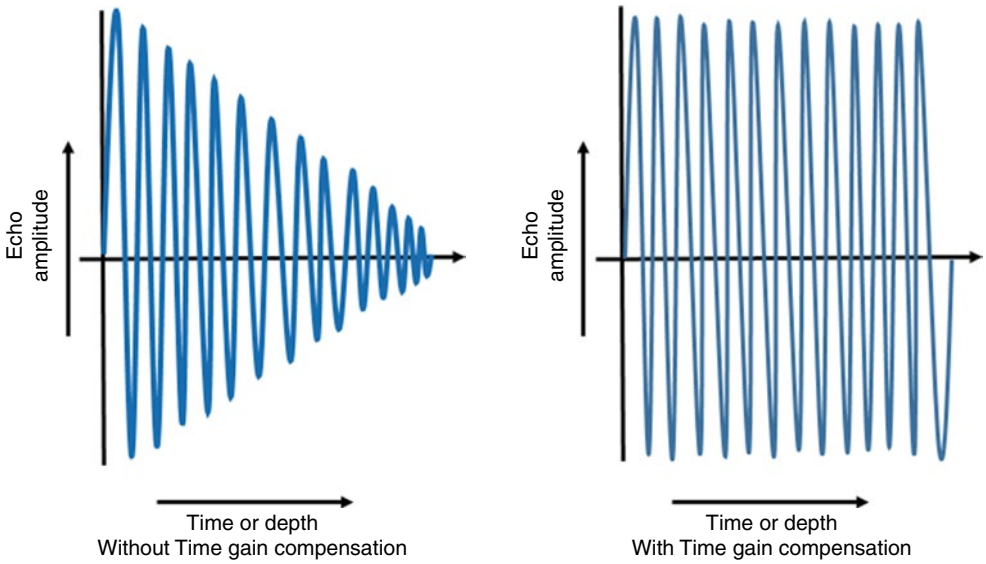


Fig. 3.18 Time gain compensation (Reproduced with permission from www.regionalfortrainees.com)

Depth

The optimal depth selection (Fig. 3.19a) allows for visualization of the target structure while minimizing the wasted space deeper to the target. Choosing a higher depth than needed may reduce the size of target (Fig. 3.19a), while too shallow a depth may obscure the target altogether (Fig. 3.19a). Optimal depth also optimizes temporal resolution.

Focus

The ultrasound beams can be focused to improve the lateral resolution in the "focal zone." Both the number of areas of focus and their depth can be optimized (Fig. 3.20).

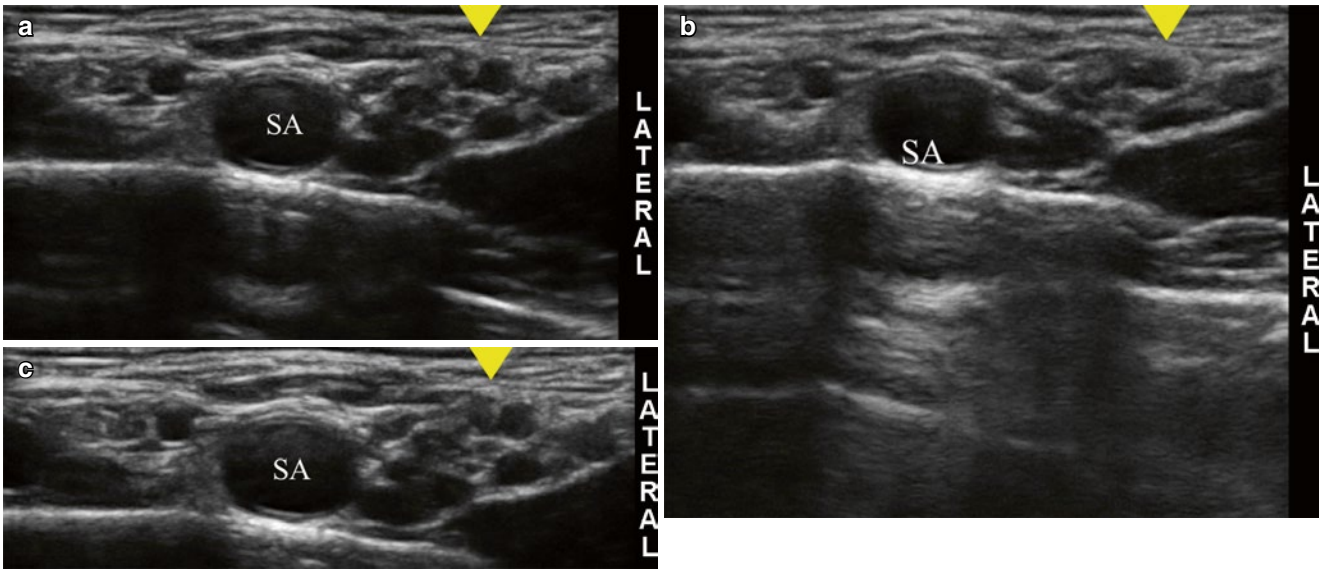


Fig. 3.19 Depth setting in supraclavicular block: (**a**) optimal, (**b**) too deep, and (**c**) too shallow (Reproduced with permission from www.regionalfortrainees.com)

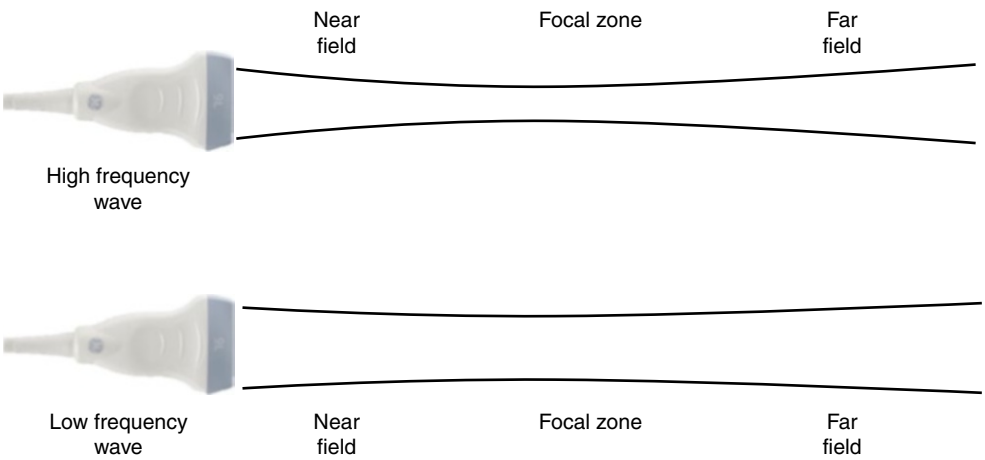


Fig. 3.20 Focal zones and focal length of an ultrasound beam (Reproduced with permission from www.regionalfortrainees.com)

Color-Flow Doppler

The application of "Doppler effect" to medical ultrasound allows for identification and quantification (velocity and direction) of blood flow within structures [7]. The blood moving toward the probe creates returning waves of higher frequency, while that moving away returns waves of lower frequency. This is represented as a red (moving toward the transducer) or blue (moving away from the transducer) color spectrum (Fig. 3.21a, b). Color-flow Doppler mode is recommended to identify vascular structures within the intended needle trajectory.

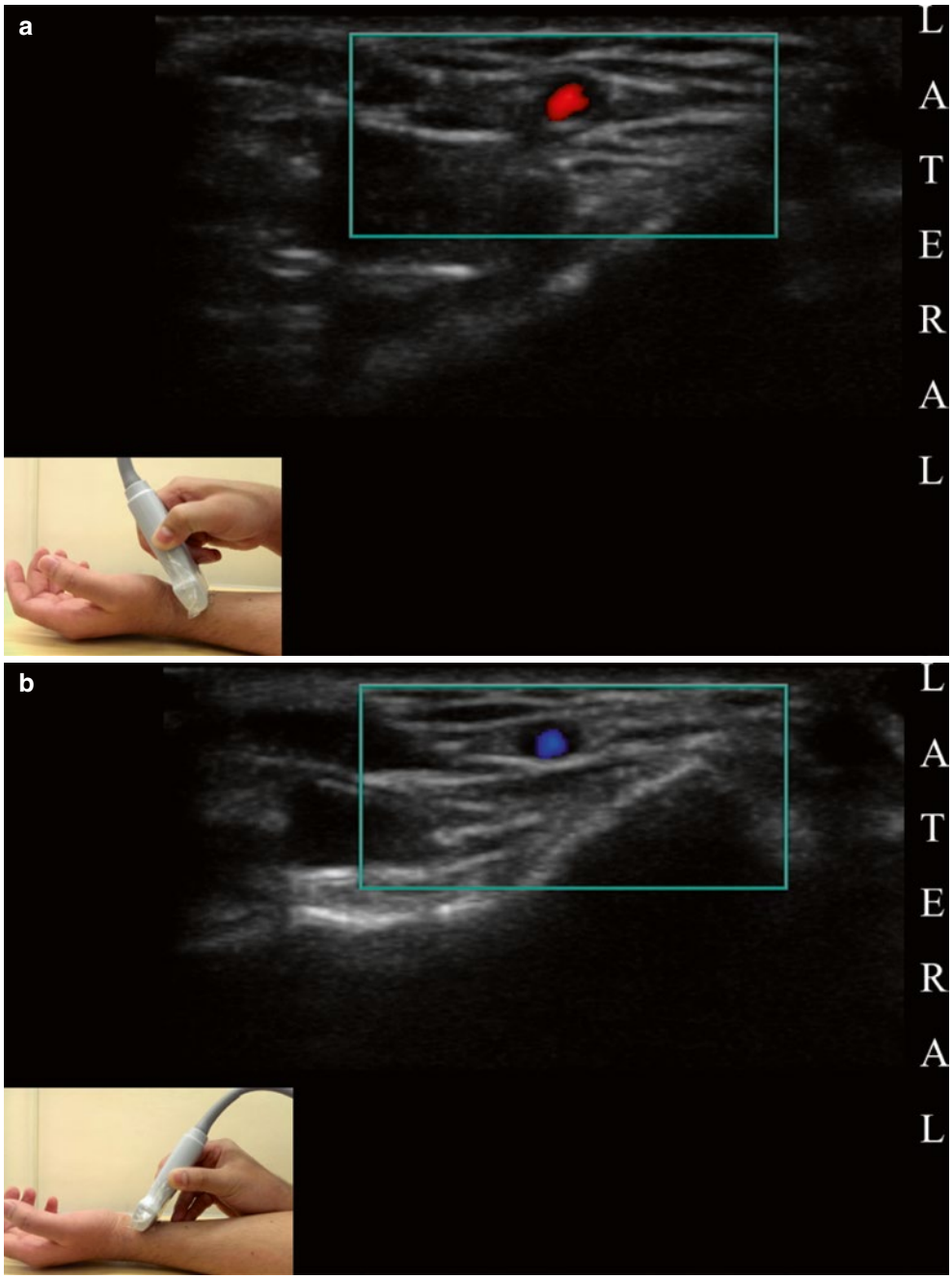


Fig. 3.21 Use of color-flow Doppler for detection of blood flow in radial artery: (**a**) *red*, toward the transducer; (**b**) *blue*, away from the transducer (Reproduced with permission from www.regionalfortrainees.com)

Pulse-Wave Doppler

This mode provides flow data from a small preselected area along the ultrasound beam. The displayed pulse-wave information is displayed along with generated sound allowing differentiation of arterial from venous pulsation (Fig. 3.22) [7].

Other Optimization Modes

(i) *Compound imaging*: this involves capturing multiple images of the tissue under examination from different angles and their combination yielding an image of better quality and reduced artifacts [25].

(ii) *Tissue harmonic imaging*: echoes are reflected by the examined tissue not only at the original frequency but also at various multiples of the original frequency [26]. Tissue harmonic imaging utilizes these harmonic multiples producing better imaging via reduced scatter and artifacts [27]. However, this may negatively impact needle visibility.

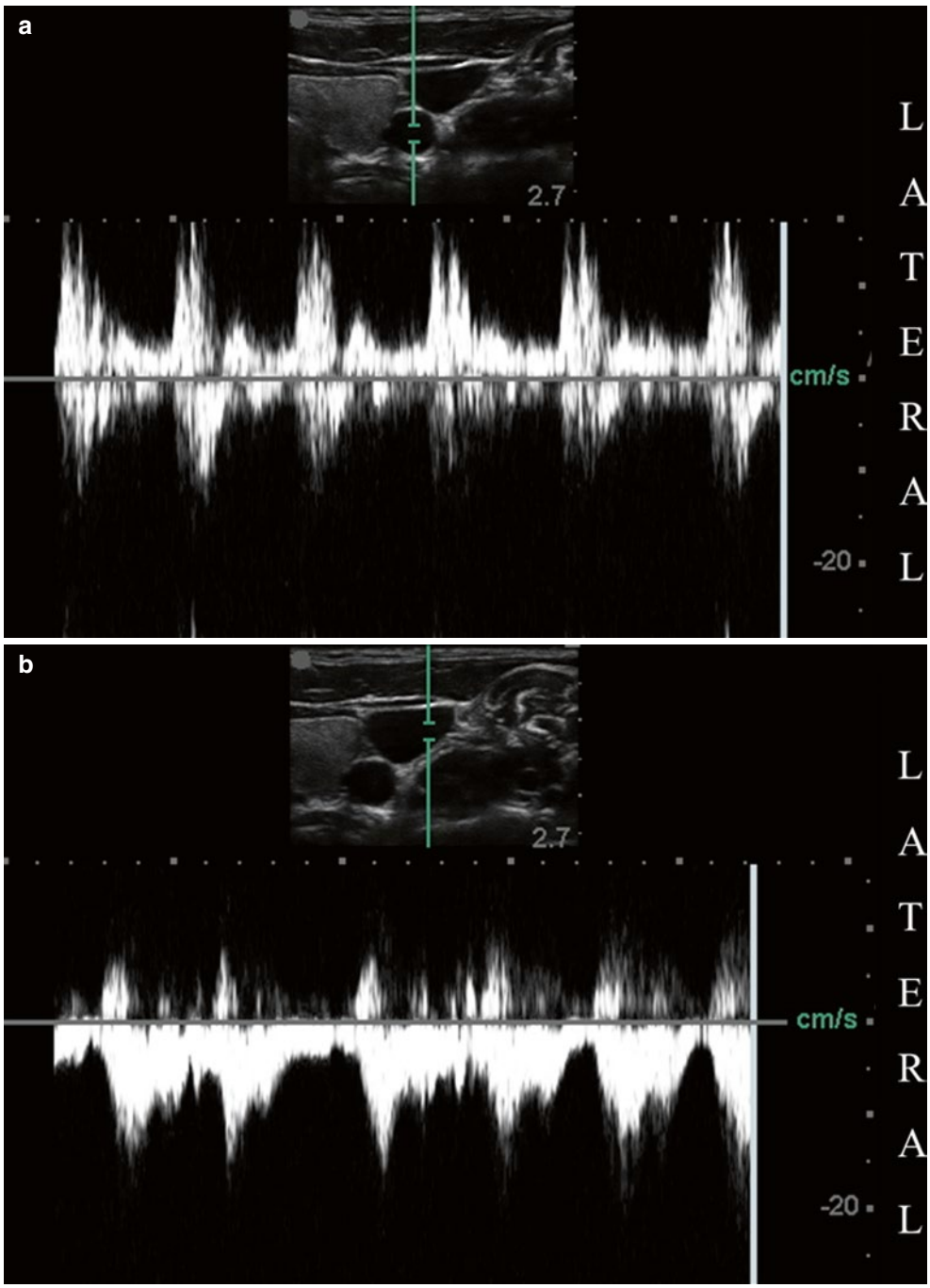


Fig. 3.22 Pulse-wave Doppler showing an arterial (**a**) and a venous waveform (**b**) in femoral artery and vein, respectively (Reproduced with permission from www.regionalfortrainees.com)

Artifacts

An artifact is an image, or a part of an image, that does not correspond with the anatomy of the structure under examination. This causes either falsely assuming the existence of a structure or failing to recognize its disguise as an artifact. Artifacts are generated because the imaging system makes a number of *assumptions* about ultrasound propagation in tissue. This includes assuming a fixed speed of ultrasound wave in tissues, assuming that the axis of the beam is straight, that the attenuation the beam undergoes is constant, and that the pulse travels only to the structures in the path of the beam and returns to the transducer [28]. Whenever there is a significant deviation from these assumptions, visible image artifacts are likely to occur.

These artifacts can be classified into four types as *acoustic*, *anatomic*, *optical illusions*, and *electrical noise artifacts* [29]. For a regional anesthesiologist, acoustic and anatomic artifacts are most important. Some of these artifacts are considered below.

Over-gain and Under-gain Artifact

Inappropriately low-gain settings can result in "missing structure" artifact, while a very high gain can obscure existing structures. A correctly applied "time gain compensation" allows appropriate image production and attenuation reduction (Figs. 3.17a–c and 3.18).

Acoustic Shadowing

This occurs when a structure with high attenuation coefficient lies above a structure with lower attenuation coefficient. This causes the underlying structure to appear far less echogenic [29]. For example, the tip of a needle can be obscured by the transverse process while performing a paravertebral block [30].

Acoustic Enhancement Artifact

This occurs when the ultrasound waves returning from a structure behind a weak attenuator (such as a fluid-containing organ) result in stronger echoes. This type of artifact is called "posterior acoustic enhancement" (Fig. 3.23).

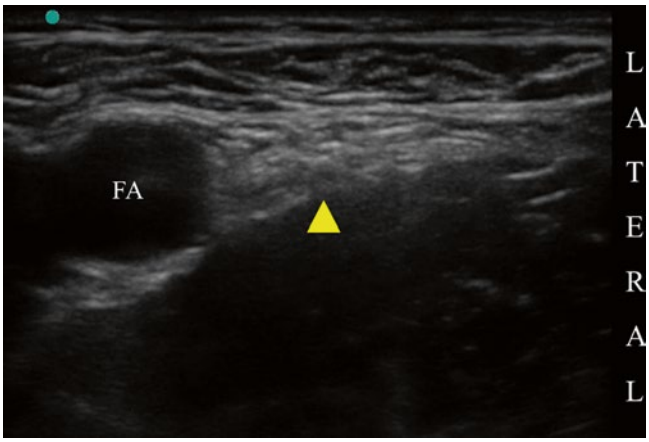


Fig. 3.23 Posterior acoustic enhancement deep to the femoral artery (*FA*) must not be mistaken for the femoral nerve (*yellow arrowhead*) which lies lateral to the artery (Reproduced with permission from www.regionalfortrainees.com)

Lateral Resolution Artifact

Lateral resolution implies the ability of the system to differentiate between two objects lying side by side at the same depth (perpendicular to the direction of the incident beam). It is best attained at the focal length of an ultrasound beam and depends upon the frequency of the array probe (being lower for high-frequency transducers). Using an incorrect frequency (with a different focal length) can cause the two objects to appear as one. Choosing an appropriate probe or frequency will help eliminate this artifact.

Reverberation Artifact

Reverberation occurs when ultrasound contacts a *strong specular reflector* (such as a needle or the surface of pleura). In the case of a needle, a part of the ultrasound wave is reflected back creating an initial image. The remaining part passes through the shaft to be reflected back by the back surface. As this continues, the strength of the returning echoes continually decreases, while the time taken to return to the transducer is prolonged. This results in an appearance of multiple needles of decreasing brightness, a "reverberation artifact" (Fig. 3.24) [31].

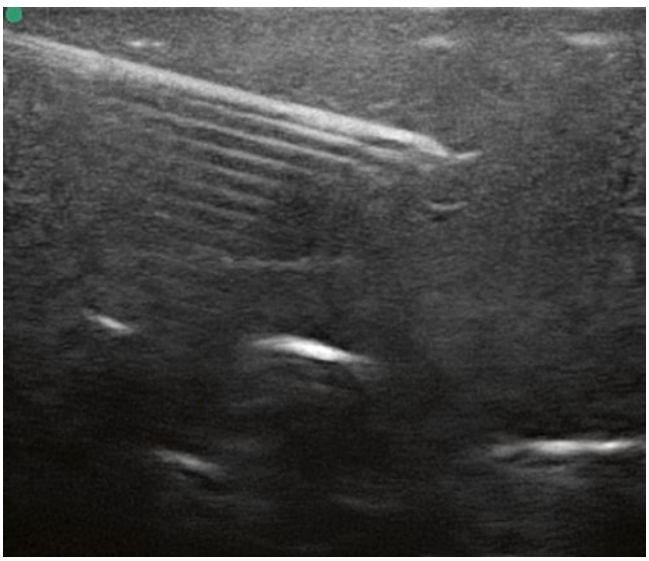


Fig. 3.24 Reverberation artifact with a Tuohy needle (Reproduced with permission from www.regionalfortrainees.com)

Anisotropy

Anisotropy means angle dependence of the appearance of a structure (such as a nerve) on an image. This results because reflection is maximum when the transducer (and the incident waves) is perpendicular to the structure under examination. Any change on this incidence angle dramatically reduces the returning echo, causing the structure to "disappear" out of the image (Fig. 3.25a, b). Manipulation of the transducer (so as to direct the incident beam perpendicular to the structure of interest) and beam-steering can help address anisotropy [32, 33].

In conclusion, having a high degree of suspicion, a good knowledge of anatomy, rotating transducer from transverse to longitudinal view (or vice versa), using appropriate gain, using tissue compression, and observing the structure under examination from another window are a few ways to properly identify and interpret commonly occurring artifacts.

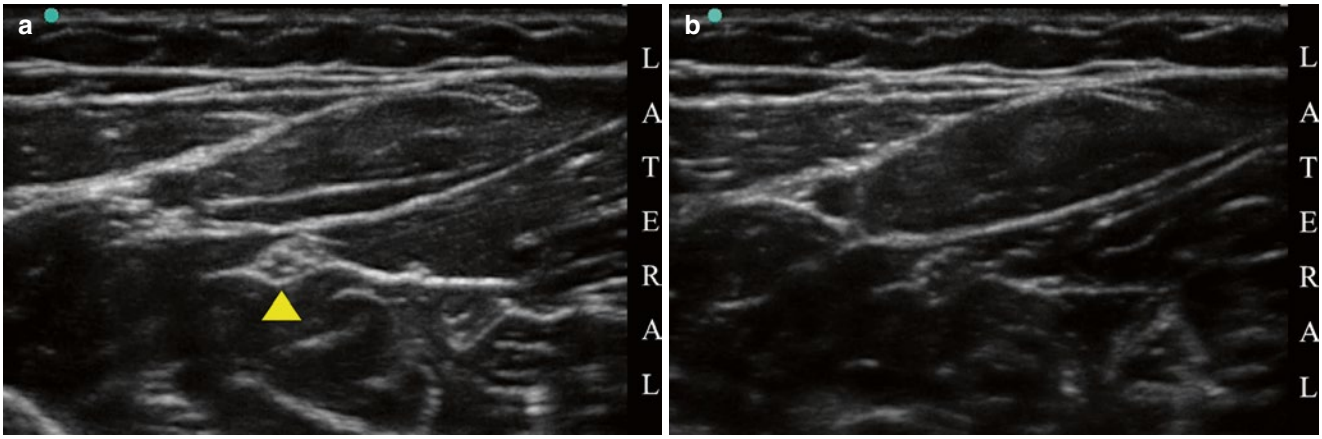


Fig. 3.25 (**a**) Median nerve (*yellow arrowhead*) when the transducer is perpendicular to the nerve. (**b**) Disappearance of the nerve on tilting the probe (Reproduced with permission from www.regionalfortrainees.com)

Practical Scanning and Needling Methodology

Procedural Ergonomics

Ergonomics is the study (or science) of the interaction between humans and their working environment [34]. Recent years have witnessed an increasing application of optimal procedural ergonomics in regional anesthesia in an effort to improve outcomes [35]. Poor ergonomics may not only lead to suboptimal performance of a procedure but may contribute to work-related musculoskeletal discomfort [36].

A sound application of the principles of ergonomics to regional anesthesia includes the consideration of the following factors:

Positioning and Care of the Patient

The patient must be placed in a comfortable position for the block. This position may vary with the type of block being performed. For example, the patient may need a supine position for an upper limb block, a prone position for a popliteal block, and a sitting position for a spinal anesthetic. Additionally, the position of the limb may be adjusted to assist the procedure. For example, performing an interscalene block may need a semi-recumbent positioning of the patient with the face turned toward the contralateral side. The head pillow is removed for a better access. Similarly, placing a cushion under the lower leg may assist in performing an ankle block.

Positioning of the Physician

Maintaining a good position with respect to the patient helps to ensure operator comfort and allows optimal bloc k performance (Fig. 3.26). This includes the following:

(a) Adjusting the *height* of patient bed to an appropriate level for the operator.
(b) Assuming a good posture, by choosing to *stand or sit down* (on a chair).
(c) Performing the block from the side being blocked to *avoid reaching over* the patient.

Positioning the Equipment

The ultrasound machine must be placed such that the operator, the target site, and the screen of the ultrasound machine are in a straight alignment. Often this is achieved by placing the ultrasound machine on the opposite side to the one that the operator assumes. This allows for a smooth coordinated scanning and needling, without the need for excessive movements on the part of the operator.

In addition, the ultrasound machine must be placed sufficiently close to the operator such that it can be reached by the operator or their assistant if any manipulation of the settings is required. The ultrasound machine must have brake pedals locked to avoid its movement relative to the operator. Similarly, when using a peripheral nerve stimulator, it should be placed sufficiently close to allow its operation during the procedure. The screen of the monitoring equipment must be turned to face the operator during the block to allow a prompt recognition of any significant change of vital signs.

Position of the Assistant

An assistant may be needed both to operate the ultrasound machine and inject the local anesthetic solution. This may be achieved by standing opposite to the operator and near the ultrasound machine.

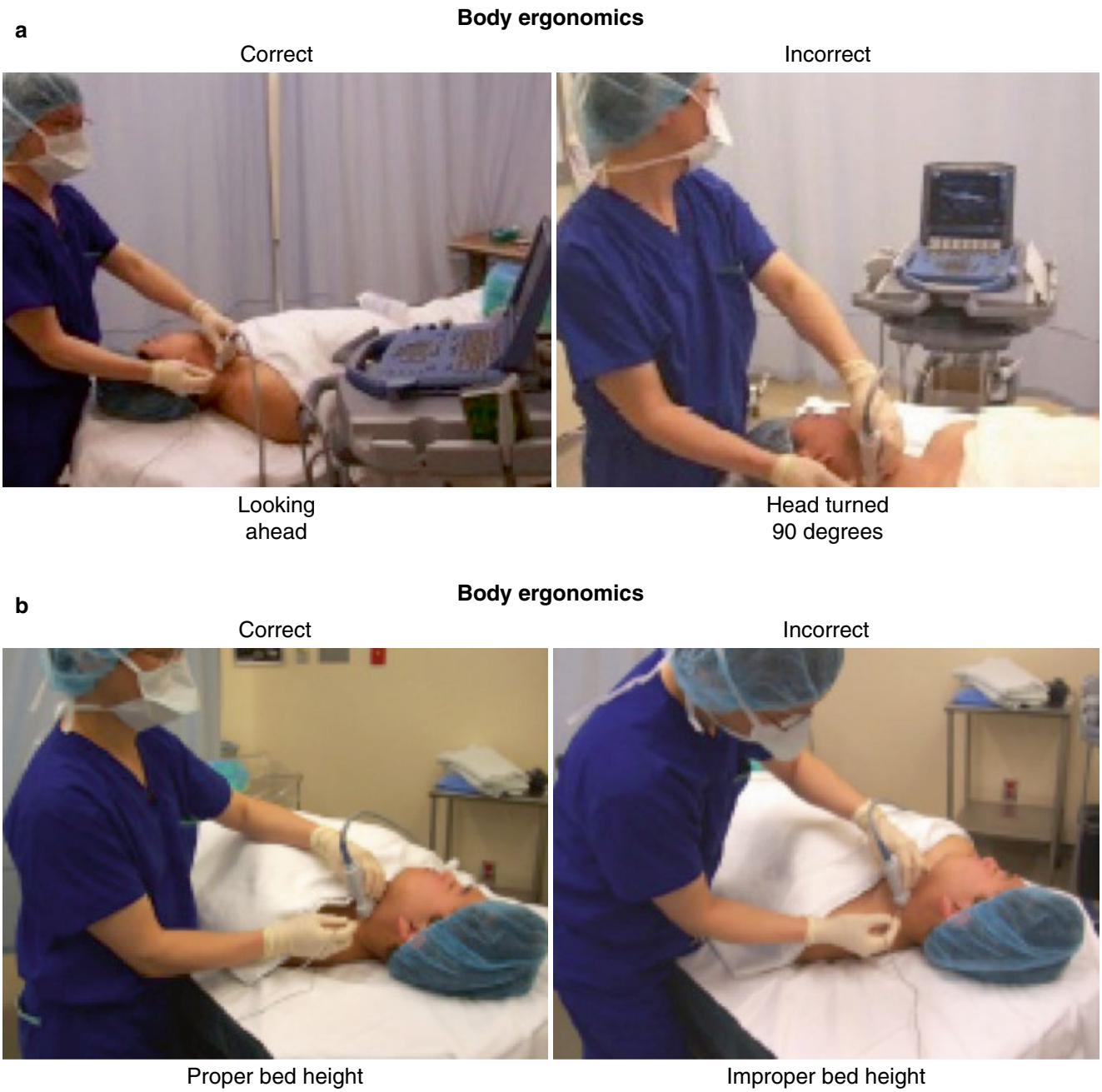


Fig. 3.26 (**a**, **b**) Optimal position of the operator, equipment, and the assistant during an upper limb block (Reproduced with permission from www.usra.ca)

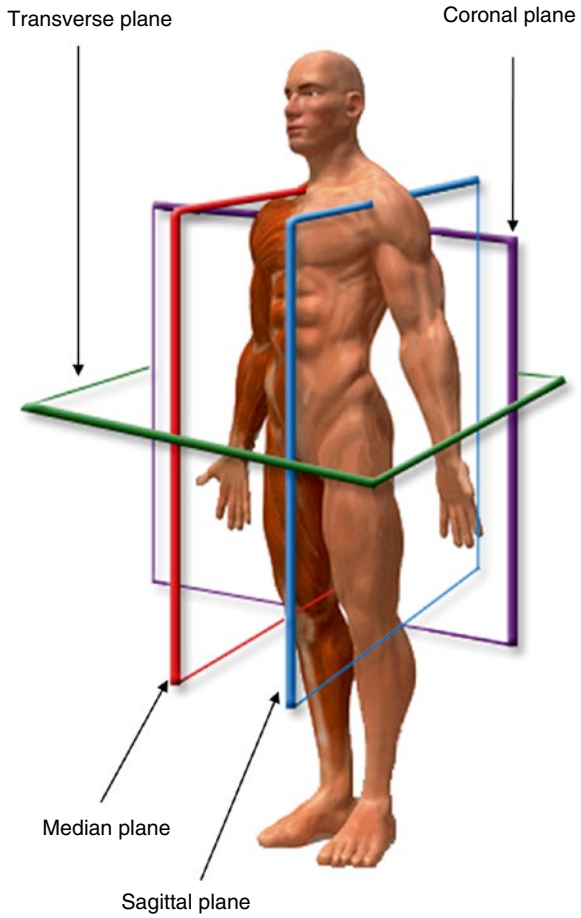


Fig. 3.27 The standard anatomical planes of the body (Reproduced with permission from www.usra.ca)

Scanning Methodology

Anatomical Planes

While scanning, it is common to refer to the *anatomical plane* of the scan. These include the axial (or transverse) plane, the sagittal plane, and the coronal plane. They are all perpendicular to one another (Fig. 3.27):

(a) The *axial* (*or transverse*) *plane* is an imaginary plane that divides the body into superior and inferior parts.
(b) The *sagittal plane* is an imaginary plane that divides the body into left and right halves.
(c) The *coronal plane* is a vertical plane that divides the body into ventral and dorsal sections.

In addition, a body part may be scanned in an oblique manner (e.g., *parasagittal oblique* or *transverse oblique planes*) [37].

Transducer Orientation

All ultrasound transducers have an *orientation marker* (Fig. 3.28a). By convention, the orientation marker is placed on the *right* of the patient in a transverse orientation and *cephalad* in a longitudinal orientation. The marker is usually placed on the *left top corner* of the screen on the ultrasound machine (Fig. 3.28b) but can be placed at any corner upon the choice of the operator. The orientation of the transducer can be correctly identified by touching one side of the transducer after gel has been applied and observing the screen for movement. Another way to identify the correct orientation is to move the transducer to one side while scanning and observing the direction of the movement of anatomical structures on the screen.

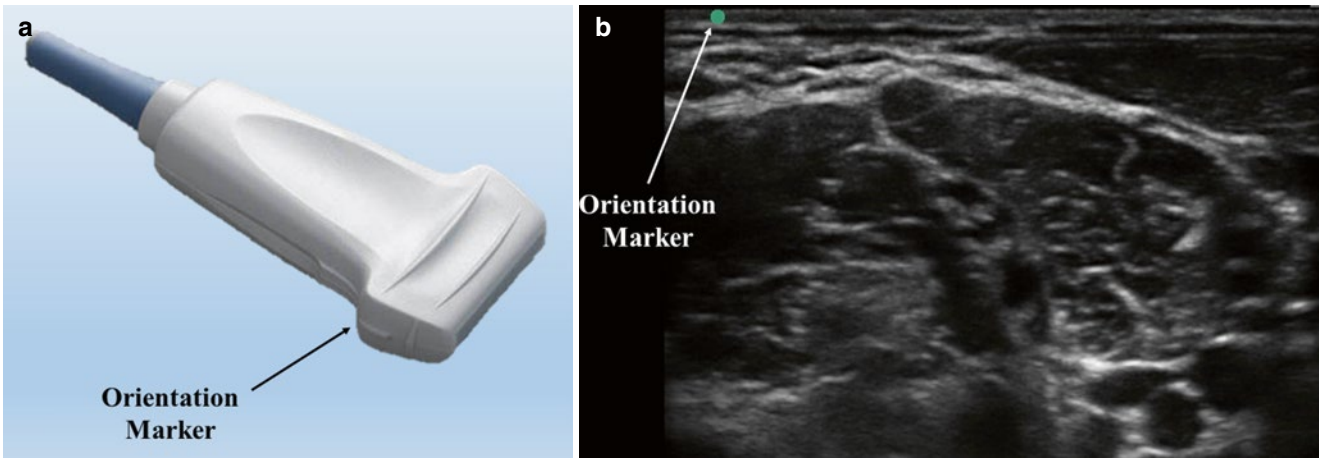


Fig. 3.28 (**a**) Orientation marker on a linear probe. (**b**) Orientation marker on the top left corner of the ultrasound screen (Reproduced with permission from www.regionalfortrainees.com)

Axis of Scanning

Nerves and vessels may be scanned along their transverse axis, obtaining a cross-sectional view of the structure (Fig. 3.29a), or along their longitudinal axis (Fig. 3.29b). The scans so obtained are commonly referred to as a "transverse scan" and a "longitudinal scan." Transverse scans are commonly used for many ultrasound-guided peripheral nerve blocks, while longitudinal scans are more often used for confirmation of perineural or intravascular catheter placements [38, 39].

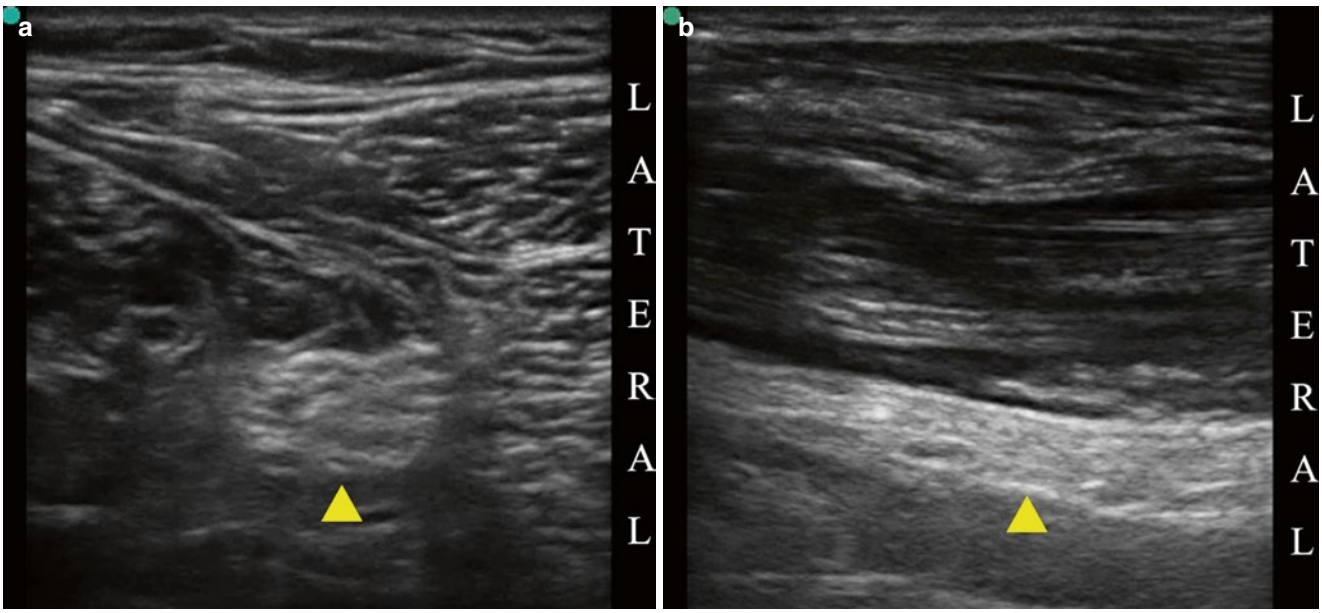


Fig. 3.29 (**a**) Transverse scanning of sciatic nerve at popliteal fossa. (**b**) Longitudinal scanning of sciatic nerve at popliteal fossa (Reproduced with permission from www.regionalfortrainees.com)

Handling the Transducer

(a) Holding the transducer

A linear probe is usually held between the thumb and the fingers, with the heel of the palm or ulnar aspect of the hand placed on the patient providing stability while scanning (Fig. 3.30). A curvilinear probe may be held by wrapping fingers around it, supported by the ulnar aspect of the scanning hand.

(b) Transducer movements

Obtaining a good image of a structure under examination requires a systemic scanning approach, involving both major and minor movements. These include pressure, alignment, rotation, and tilt (commonly called the *PART* maneuver) [40, 41].

(i) *Pressure*: applying adequate pressure while scanning improves the image quality by shortening the distance of the target from the probe (Fig. 3.31a). Additionally, it may allow compressing a vein (helping in its identification). One-sided pressure may also allow directing the ultrasound waves at a desired angle [42]. However, excessive pressure may also cause underestimation of the target depth.

(ii) *Alignment* (*or sliding*): this allows the target structure to be centralized in the image as this provides the best resolution (Fig. 3.31b). Sliding along the long axis also allows to follow the structure of interest proximally or distally (scout scan).

(iii) *Rotation*: rotating the probe clockwise or counterclockwise helps identify a true transverse plane (Fig. 3.31c). Additionally, rotating the probe from a transverse plane to a longitudinal plane allows a better examination of the target and the best plane for needle trajectory.

(iv) *Tilt*: tilting the probe along its vertical axis allows an examination of the target (along its long axis) without the need for sliding the probe proximally or distally (Fig. 3.31d). Tilting is very useful to track the needle tip.

Identifying Signposts

Structures that help in orienting an operator to the underlying anatomy are referred to as "signposts." They also provide a starting point for a systematic scan of the target area. For example, the subclavian artery is an important signpost while scanning for the brachial plexus in the supraclavicular region.

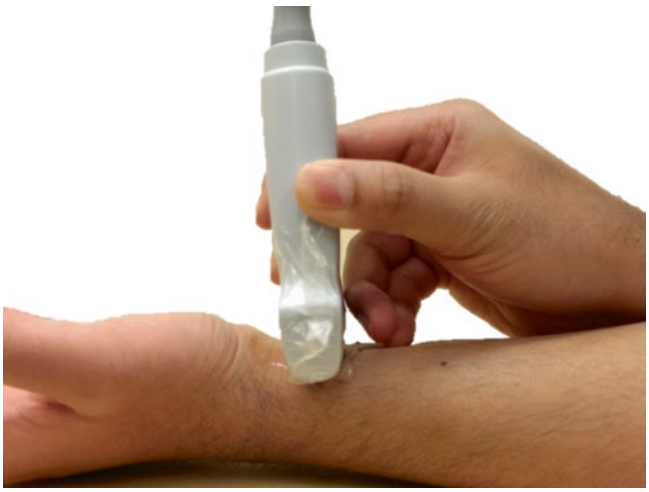


Fig. 3.30 Holding the linear transducer (Reproduced with permission from www.regionalfortrainees.com)

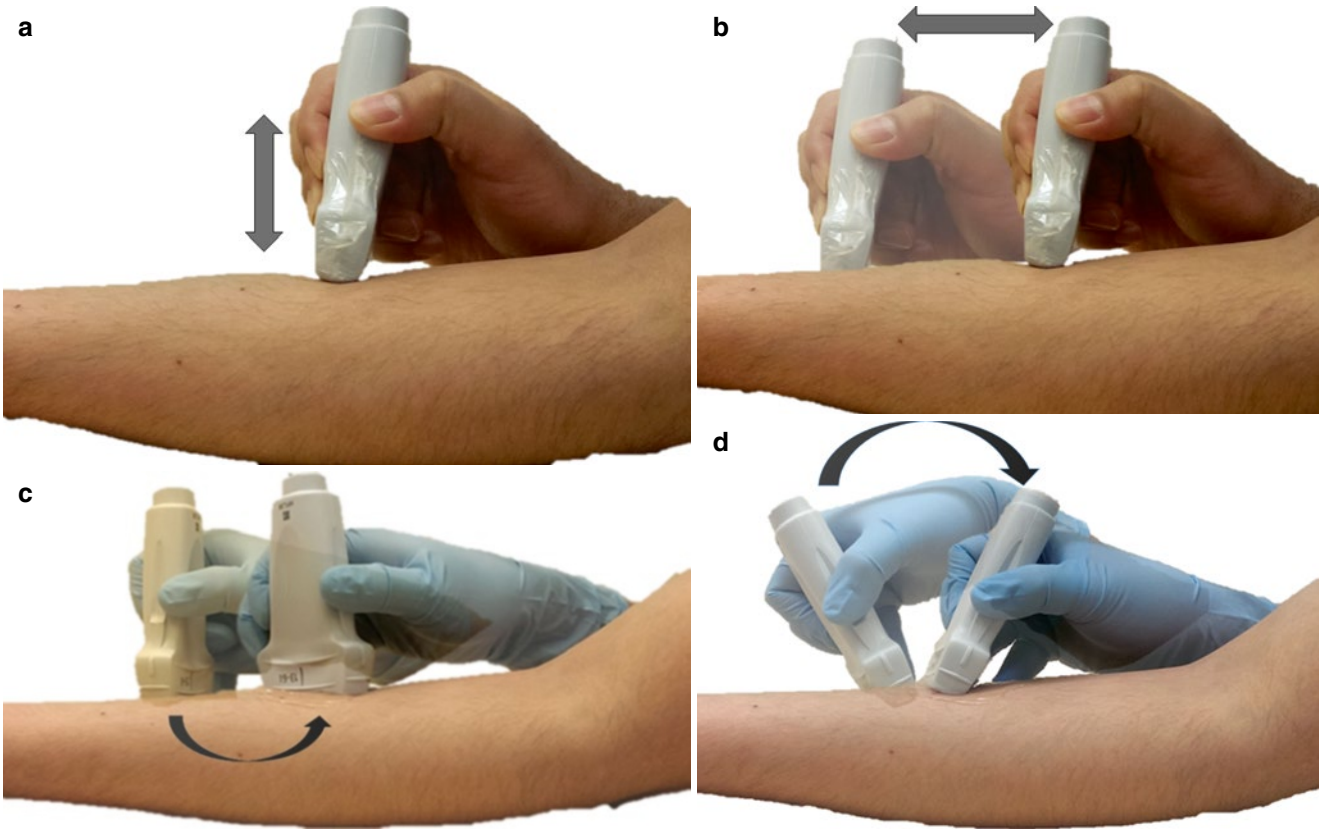


Fig. 3.31 Transducer movements (PART): (**a**) pressure, (**b**) alignment, (**c**) rotation, and (**d**) tilt (Reproduced with permission from www.regionalfortrainees.com)

Needling Techniques

Needle Insertion Technique

The "in-plane" and "out-of-plane" needle approaches are most commonly used:

(a) *In-plane needle insertion*: the needle is advanced in the plane of the ultrasound beam with the intention to visualize the needle from the shaft to its tip (Fig. 3.32a). A shallower trajectory will result in better visibility [42, 43].
(b) *Out-of-plane needle insertion*: here, the needle trajectory is perpendicular to the ultrasound probe such that the needle tip or its shaft is seen as a hyperechoic dot on the screen (Fig. 3.32b). This approach offers a shorter route to the target structure (nerve or vessel), but it can prove more challenging to follow the exact needle tip position in real time, especially for novice users.

In combination with the transverse (short) axis or the longitudinal (long) axis, the following combinations are possible:

(a) Short-axis in-plane needle insertion
(b) Short-axis out-of-plane needle insertion
(c) Long-axis in-plane needle insertion
(d) Long-axis out-of-plane needle insertion

Short-axis in-plane needling and short-axis out-of-plane needling are commonly used in regional anesthesia. Short-axis out-of-plane needling is particularly useful while placing perineural catheters. There is no evidence to suggest that one method is better than the other, and it may be prudent to follow the most familiar method [44].

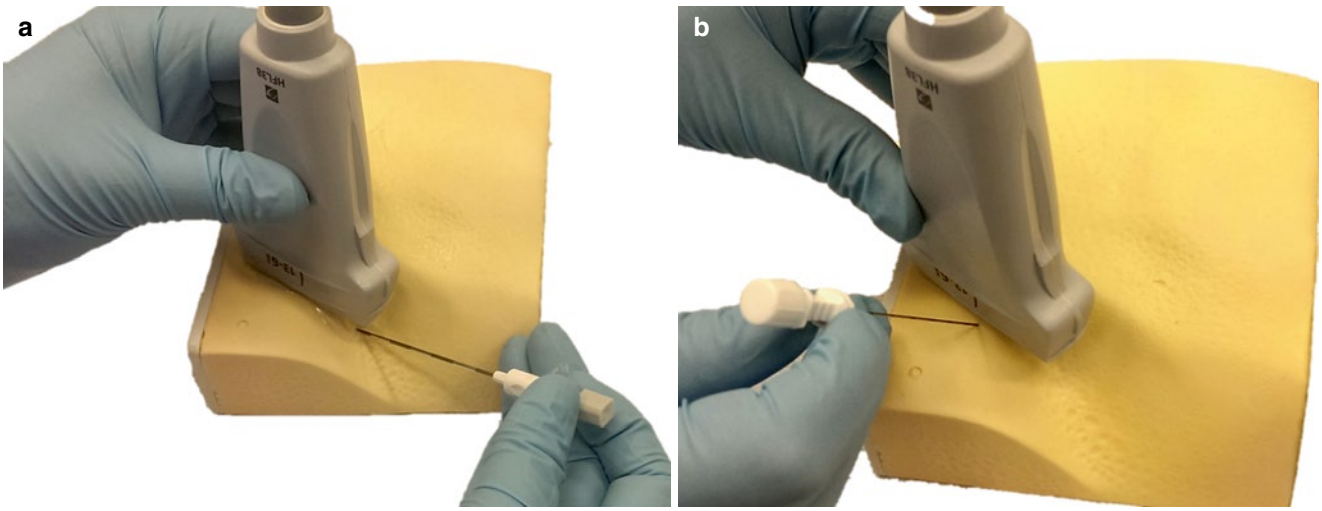


Fig. 3.32 Needling techniques. (**a**) In-plane needle insertion. (**b**) Out-of-plane needle insertion (Reproduced with permission from www.regionalfortrainees.com)

Needle Advancement

Correct needle advancement techniques allow tracking of needle tip from the point of insertion to the target structure:

(a) *In-plane needling*: following the needle is easier in in-plane needling. The needle is inserted initially at a shallow angle to allow visualization (Fig. 3.33, point A). It is then redirected deeper toward the target structures, keeping the advancing needle in view at all times (Fig. 3.33, point B). If the needle is directed slightly obliquely, only a part of it may be visualized. Using "PART" maneuvers for transducer manipulation, one can then attempt to visualize the entire needle.

(b) *Out-of-plane needling*: this is more challenging and can be achieved using two methods:

(i) *Sliding the probe*: after inserting the needle in an anticipated trajectory, the probe is first brought closer to the needle until the needle tip can be seen as a hyperechoic dot on the screen (Fig. 3.34a, plane A). Then the probe is moved away from the needle, followed by advancement of the needle till its tip reappears on the screen (Fig. 3.34a, plane B). Using such sliding motions of the probe, the needle tip can be followed and guided up to the target structure.

(ii) *Tilting the probe*: here, the probe is kept stationary. The needle is inserted out of plane in an anticipated trajectory, and the probe is tilted toward the needle to allow visualization of its tip on the screen (Fig. 3.34b, plane A). Next, the probe is tilted away from the needle such that the needle tip disappears. The needle is advanced until the tip reappears (Fig. 3.34b, plane B). This motion is repeated until the needle tip approached the target structure (Fig. 3.34b, plane C). This method may cause difficulty if the underlying structure is quite anisotropic (as a minor tilt may result in a poor image of the structure of interest).

Fig. 3.33 In-plane needle advancement (Reproduced with permission from www.regionalfortrainees.com)

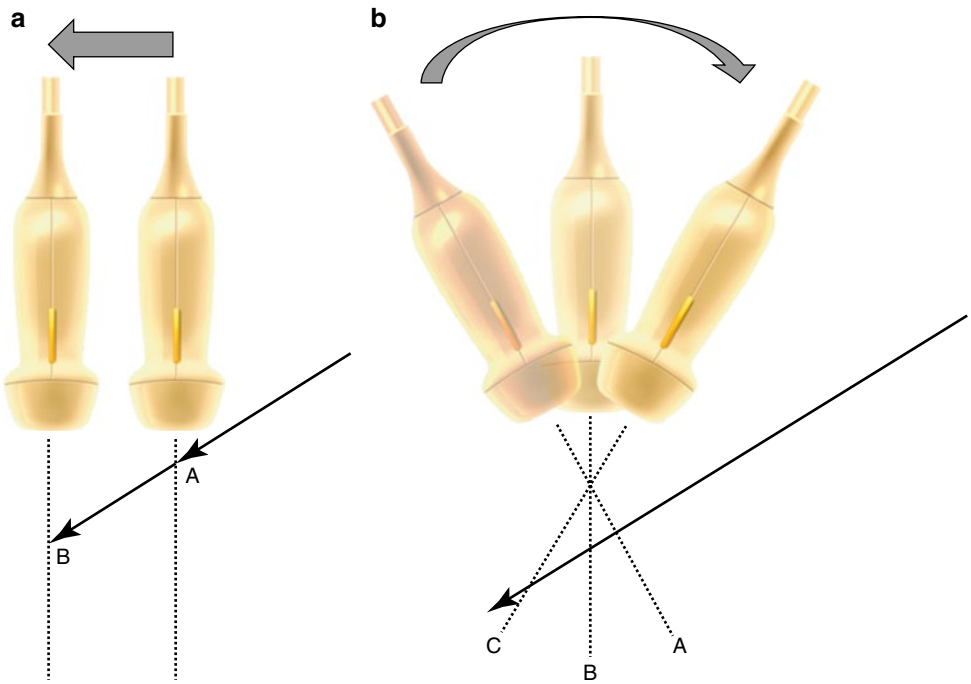


Fig. 3.34 Out-of-plane needle advancement. (**a**) Sliding the probe. (**b**) Tilting the probe (Reproduced with permission from www.regionalfortrainees.com)

References

1. La Grange P, Foster PA, Pretorius LK. Application of the Doppler ultrasound bloodflow detector in supraclavicular brachial plexus block. Br J Anaesth. 1978;50(9):965–7.
2. Marhofer P, Willschke H, Kettner S. Current concepts and future trends in ultrasound-guided regional anesthesia. Curr Opin Anaesthesiol. 2010;23(5):632–6.
3. Cory PC. Concerns regarding ultrasound-guided regional anesthesia. Anesthesiology. 2009;111(5):1167–8.
4. Liu SS, Ngeow J, John RS. Evidence basis for ultrasound-guided block characteristics: onset, quality, and duration. Reg Anesth Pain Med. 2010;35(2 Suppl):S26–35.
5. Neal JM, Brull R, Chan VWS, Grant SA, Horn J-L, Liu SS, 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.
6. Martin K, Ramnarine K. Physics. In: Hoskins PR, Martin K, Thrush A, editors. Diagnostic ultrasound: physics and equipment. 2nd ed. Cambridge: Cambridge University Press; 2010. p. 4–22.
7. Murthi SB, Ferguson M, Sisley AC. Ultrasound physics and equipment. In: Frankel HL, deBoisblane BP, editors. Bedside procedures for the intensivist. New York: Springer; 2010. p. 57–80.
8. Manbachi A, Cobbold RSC. Development and application of piezoelectric materials for ultrasound generation and detection. Ultrasound. 2011;19(4):187–96. SAGE Publications.
9. Szabo TL, Lewin PA. Piezoelectric materials for imaging. J Ultrasound Med. 2007;26(3):283–8.
10. Case TD. Ultrasound physics and instrumentation. Surg Clin North Am. 1998;78(2):197–217.
11. Martin K. Basic equipment, components and image production. In: Allan PL, Baxter GMWM, editors. Clinical ultrasound. 3rd ed. London: Elsevier; 2011. p. 16–30.
12. Lancée CT, Souquet J, Ohigashi H, Bom N. Transducers in medical ultrasound: part one: Ferro-electric ceramics versus polymer piezoelectric materials. Ultrasonics. 1985;23(3):138–42.
13. Persson HW, Hertz CH. Acoustic impedance matching of medical ultrasound transducers. Ultrasonics. 1985;23(2):83–9.
14. Guo S, Schwab A, McLeod G, Corner G, Cochran S, Eisma R, et al. Echogenic regional anaesthesia needles: a comparison study in Thiel cadavers. Ultrasound Med Biol. 2012;38(4):702–7.
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(1):143–8.
16. Noce JP. Fundamentals of diagnostic ultrasonography. Biomed Instrum Technol. 1990;24(6):456–9.
17. Gregg EC, Palagallo GL. Acoustic impedance of tissue. Invest Radiol. 1965;4(6):357–63.
18. Martin K. B-Mode instrumentation. In: Hoskins PR, Martin K, Thrush A, editors. Diagnostic ultrasound: physics and equipment. 2nd ed. Cambridge: Cambridge University Press; 2010. p. 47–63.
19. Pye SD, Wild SR, McDicken WN. Adaptive time gain compensation for ultrasonic imaging. Ultrasound Med Biol. 1992;18(2):205–12.
20. Whittingham T, Martin K. Transducers and beam forming. In: Hoskins PR, Martin K, Thrush A, editors. Diagnostic ultrasound: physics and equipment. 2nd ed. Cambridge: Cambridge University Press; 2010. p. 23–46.
21. Brull R, Macfarlane AJR, Tse CCH. Practical knobology for ultrasound-guided regional anesthesia. Reg Anesth Pain Med. 2010;35(2 Suppl):S68–73.
22. Martin K. Properties, limitations and artefacts of B-mode images. In: Hoskins PR, Martin K, Thrush A, editors. Diagnostic ultrasound: physics and equipment. 2nd ed. Cambridge: Cambridge University Press; 2010. p. 64–74.
23. 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 I: understanding the basic principles of ultrasound physics and machine operations. Reg Anesth Pain Med. 2007;32(5):412–8.
24. Barr RG, Grajo JR. Dynamic automatic ultrasound optimization: time savings, keystroke savings, and image quality. Ultrasound Q. 2009;25(2):63–5.
25. Entrekin RR, Porter BA, Sillesen HH, Wong AD, Cooperberg PL, Fix CH. Real-time spatial compound imaging: application to breast, vascular, and musculoskeletal ultrasound. Semin Ultrasound CT MR. 2001;22(1):50–64.
26. Tranquart F, Grenier N, Eder V, Pourcelot L. Clinical use of ultrasound tissue harmonic imaging. Ultrasound Med Biol. 1999;25(6):889–94.
27. Blaivas M, DeBehnke D, Sierzenski PR, Phelan MB. Tissue harmonic imaging improves organ visualization in trauma ultrasound when compared with standard ultrasound mode. Acad Emerg Med. 2002;9(1):48–53.
28. Antonakakis JG, Sites B. The 5 most common ultrasound artifacts encountered during ultrasound-guided regional anesthesia. Int Anesthesiol Clin. 2011;49(4):52–66.
29. Kremkau FW, Taylor KJ. Artifacts in ultrasound imaging. J Ultrasound Med. 1986;5(4):227–37.
30. Reusz G, Sarkany P, Gal J, Csomos A. Needle-related ultrasound artifacts and their importance in anaesthetic practice. Br J Anaesth. 2014;112(5):794–802.
31. Saranteas T, Karabinis A. Reverberation: source of potential artifacts occurring during ultrasound-guided regional anesthesia. Can J Anaesth. 2009;56(2):174–5. Springer.
32. AIUM technical bulletin. Transducer manipulation. American Institute of Ultrasound in Medicine. J Ultrasound Med. 1999;18(2):169–75.
33. Connolly DJ, Berman L, McNally EG. The use of beam angulation to overcome anisotropy when viewing human tendon with high frequency linear array ultrasound. Br J Radiol. 2001;74(878):183–5.
34. Stone R, McCloy R. Ergonomics in medicine and surgery. BMJ. 2004;328(7448):1115–8.
35. 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(8):1277–80.
36. Roll SC, Selhorst L, Evans KD. Contribution of positioning to work-related musculoskeletal discomfort in diagnostic medical sonographers. Work. 2014;47(2):253–60.
37. Chin KJ, Ramlogan R, Arzola C, Singh M, Chan V. The utility of ultrasound imaging in predicting ease of performance of spinal anesthesia in an orthopedic patient population. Reg Anesth Pain Med. 2013;38(1):34–8.
38. Tokumine J, Lefor AT, Yonei A, Kagaya A, Iwasaki K, Fukuda Y. Three-step method for ultrasound-guided central vein catheterization. Br J Anaesth. 2013;110(3):368–73.
39. Ilfeld BM, Fredrickson MJ, Mariano ER. Ultrasound-guided perineural catheter insertion: three approaches but few illuminating data. Reg Anesth Pain Med. 2011;35(2):123–6.
40. Ihnatsenka B, Boezaart AP. Ultrasound: basic understanding and learning the language. Int J Shoulder Surg. 2010;4(3):55–62.
41. 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(1):40–6.
42. Chin KJ, Perlas A, Chan VWS, Brull R. Needle visualization in ultrasound-guided regional anesthesia: challenges and solutions. Reg Anesth Pain Med. 2008;33(6):532–44.
43. Maecken T, Zenz M, Grau T. Ultrasound characteristics of needles for regional anesthesia. Reg Anesth Pain Med. 2007;32(5):440–7.
44. Fredrickson MJ, Danesh-Clough TK. Ultrasound-guided femoral catheter placement: a randomised comparison of the in-plane and out-of-plane techniques. Anaesthesia. 2013;68(4):382–90.

Chapter 4

Use of Nerve Stimulation and Stimulating Catheters in the Ultrasound Era

Derek Dillane, MB BCh, FRCA
Department of Anesthesiology and Pain Medicine, University of Alberta and University of Alberta Hospital, Edmonton, AB, Canada
e-mail: dillane@ualberta.ca

Ban C.H. Tsui, MSc, MD, FRCPC (✉)
Department of Anesthesiology and Pain Medicine, University of Alberta and University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada
e-mail: btsui@ualberta.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_4, © Springer International Publishing Switzerland 2015

Introduction

Approaches to nerve localization, from the century-old, landmark-based paresthesia techniques through to electrical nerve stimulation and latterly ultrasound guidance, strive for accuracy leading to successful anesthesia without nerve damage. A reduction in volume and dose of local anesthetic agents, a greatly increased percentage of successful blocks, and the ability to block nerves that were difficult to locate were listed among the benefits of the electrical nerve stimulator when it was introduced into clinical practice half a century ago. This was an admirable assertion, but for the most part, regional anesthesia remained in the confines of the specialist practitioner, more of an art form than a science. The introduction and widespread acceptance of ultrasound as a localization device promises to negate the limitations of landmark, paresthesia, and nerve stimulation techniques. Rather than relying on a surrogate measure of needle-to-nerve proximity, it is now possible to visualize the anatomy of the neural target, nearby structures to be avoided, the needle trajectory, and spread of local anesthetic.

There has been a growing body of randomized, controlled trials over the past decade comparing ultrasound-guided regional anesthesia with other forms of nerve localization techniques [1, 2]. In particular, many investigators have attempted to demonstrate the superiority of ultrasound guidance over nerve stimulation. All of which leads to the contentious issue of whether electrical nerve stimulation has been superannuated by a safer and more effective technique [1].

Electrophysiology and Practicalities of Electrical Nerve Stimulation

Direct electrical current flowing between two electrodes on a given nerve will stimulate the nerve at the *cathode* (negative electrode). Negative current from the cathode alters the resting membrane potential of the neuronal cell causing depolarization that results in an action potential (Fig. 4.1). The cathode is usually attached to the stimulating needle/catheter, while the anode is attached to the patient's skin as a returning electrode. This results in either muscle contraction or paresthesia in the pertinent nerve distribution and is dependent on nerve fiber type, i.e., motor or sensory.

Needles used for nerve stimulation are insulated with a nonconducting material. This directs the current density to a sphere around the uncoated needle tip. The use of nonelectrolyte/nonconducting injectates, for example, dextrose 5 % in water (D5W), reduces the conductive area around the needle tip and increases the current density resulting in maintenance *or even augmentation* of the motor response at a low current (<0.5 mA).

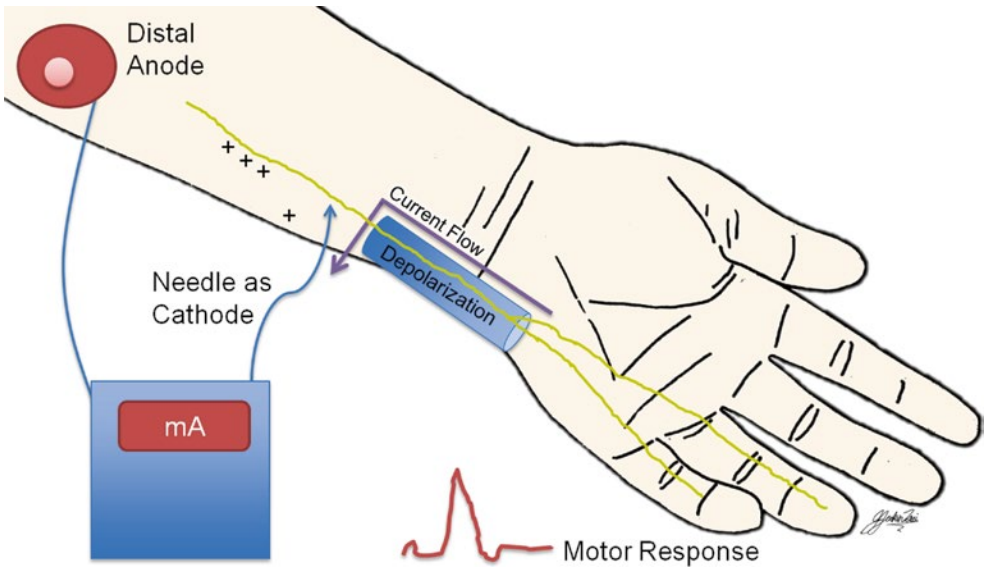


Fig. 4.1 Ulnar nerve stimulation. Electron flow is toward the needle with the needle as the cathode. This causes an area of depolarization around the needle tip and a subsequent action potential causing a motor response

Important Adjustable Features of Modern Nerve Stimulators

Current: Coulomb's law describes the relationship between distance and current intensity:

$$I = k\left(i / r^2\right)$$

I current required, k constant, i minimal current, r distance from nerve

Consequently, as the distance between needle and nerve decreases, a lower current intensity should be required to initiate a motor response.

Pulse width: The duration of the pulse enables selective stimulation of sensory or motor nerves. Motor nerves are more easily targeted with shorter pulse widths (e.g., 50–150 μs).

Frequency of stimulation: Low frequencies may cause the target nerve to be missed due to poor timing. Most operators utilize a frequency of 2 Hz.

Concerns Regarding Nerve Stimulation in the Ultrasound Era

Ultrasound imaging, while having enhanced our understanding of the needle–nerve relationship, has created ambivalence regarding long-held tenets of nerve stimulation. As we have seen in the above section, as distance between the needle and nerve decreases, a lower current intensity should be required to initiate a motor response. However, when observed under ultrasound guidance, a motor response to nerve stimulation is frequently not seen until the needle tip is advanced into an intraneural location [3]. On occasion, with an intraneural needle tip location, a high stimulating current may be required to generate a motor response. A stimulating current as high as 1.5 mA has been found *not* to produce a motor response when the needle tip is located in the intraneural space. This contradicts previously held electrophysiological principles upon which safe practice in peripheral nerve blockade was based. Current evidence suggests that a motor response to a stimulating current of 0.2 mA always signifies an intraneural position [4, 5]. The distillation of human and animal studies into clinically useful guidelines advocates that nerve stimulation has higher specificity than sensitivity for detecting intraneural needle placement. In other words, a response to stimulation at a low current ($\leq$0.2 mA) confirms intraneural placement, but lack of a response does not necessarily rule it out.

Mechanisms of Nerve Injury: Is It Possible to Detect Intraneural Injection Using Ultrasound?

To answer this important question, it is first necessary to review the basic histology of the nerve fiber. The axon is a projection of the nerve cell body. Electrical impulses are conducted through the axon away from the cell body. Axons may be coated with a myelin sheath which increases the speed of conduction. A peripheral nerve is composed of multiple nerve axons arranged into fascicles. Neural microarchitecture consists of a complex network of nerve tissue enclosed within concentric layers of protective connective tissue (Fig. 4.2). Individual axons are surrounded by a connective tissue layer called the *endoneurium*. The fascicle itself is enclosed in a tough and mechanically resistant sheath called the *perineurium*. A collection of fascicles, along with blood supply and fatty tissue, is surrounded by a third, and final, layer of connective tissue comprised of collagen and adipose tissue called the *epineurium*.

The ratio of fascicular-to-epineurial tissue varies between 30 and 70 % of the total nerve area [6], a relationship which deviates not only between different nerves but also along individual nerves. The ratio of neural-to-nonneural tissue is greater closer to the nerve root for both the sciatic nerve and the brachial plexus, i.e., more fascicular tissue relative to surrounding connective tissue at the proximal sciatic nerve and interscalene brachial plexus, respectively [7, 8]. Fortunately, the path of least resistance for an intraneurally placed needle may be through the more compliant adipose tissue of the interfascicular epineurium rather than through the fascicles. However, significant and lasting nerve injury is thought to occur only when injection of solution occurs inside the fascicle.

Ultrasonographic detection of intraneural injection is largely dependent on the surrogate measure of nerve expansion upon injection. Indeed, ultrasonographic nerve expansion has been equated with intraneural injection as confirmed by histologic analysis in porcine studies [9, 10]. However, while ultrasound guidance may permit a rudimentary assessment of nerve diameter, the prohibitive resolution of available ultrasound technology precludes consistent differentiation between intrafascicular and extrafascicular injection. A 15-MHz transducer, in the high end of most practitioner's armamentarium, only permits visualization of one-third of sciatic nerve fascicles as compared with light microscopy [11]. Regardless of its technological limitations, ultrasound images must be interpreted by the operator. Ultrasonographic evidence of nerve expansion may not always be obvious, and two recent cautionary case reports regarding brachial plexus blockade by experienced practitioners are testament to the imperfections of the technology [12, 13].

The overall incidence of late neurologic deficit is such a rarity that it precludes statistical substantiation by randomized controlled trials. Nevertheless, it is instructive to scrutinize the incidence of serious nerve injury associated with regional anesthesia before and after the introduction of ultrasound guidance. In one of the largest reports to date, a prospective survey in France recorded an incidence of late neurologic injury of 0.2/1,000 in over 150,000 regional anesthesia procedures [14]. In the ultrasound era, the Australasian Regional Anesthesia Collaboration reports a similar incidence of late neurologic deficit of 0.4/1,000 [15].

There is no evidence to suggest that nerve stimulation is any better at detecting intrafascicular needle placement than ultrasound guidance. However, the two modalities may be complimentary and serve to compensate for their respective deficits.

The authors, when performing blocks under ultrasound guidance, use a dual ultrasound–nerve stimulation technique. A constant stimulating current of 0.25 mA is used. This ensures patient comfort while improving the safety of nerve localization.

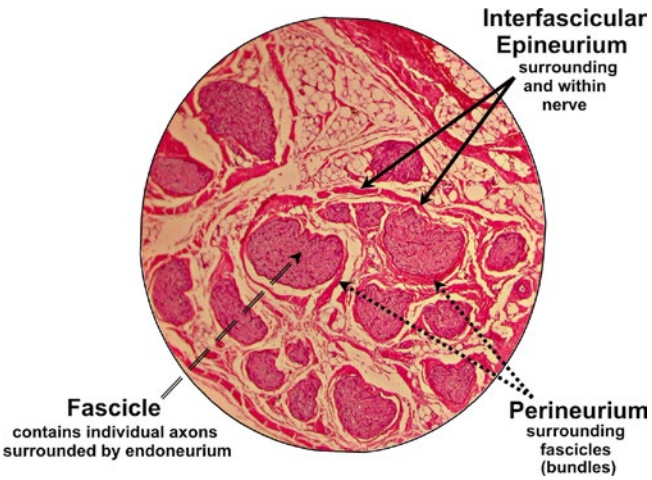


Fig. 4.2 Cross section of a nerve at 25× magnification. Fascicles are surrounded by protective connective tissue layers (perineurium and interfascicular epineurium). Note that the endoneurium is too fine to be seen with light microscopy at this magnification

Nerve Stimulation and the Potential for Patient Injury

There appears little doubt than when electrical nerve stimulation is used as a location device in conjunction with ultrasound guidance that block performance times are lengthened without an improvement in success rates [16, 17]. This suggests a greater number of needle passes and associated patient discomfort. Moreover the lower dose and volume of local anesthetic solution permitted by the highly accurate perineural placement of local anesthetic solution under ultrasound guidance alone results in a significant reduction of local anesthetic systemic toxicity [2].

A longer block performance time may indeed be required if an anatomic and neurophysiologic endpoint are sought. However, when compared with an ultrasound guidance technique alone, the procedure time may be equivalent when using a dual ultrasound–nerve stimulation approach for the exclusion of intraneural needle placement rather than for confirmation of nerve location.

Use of Nerve Stimulation for Training Novices

While expertise in recognition and location of the pertinent sonoanatomy can be procured with time, haptic perception and consistent hand–eye coordination are more challenging skills to acquire. Failure to maintain needle tip visualization is the most common error observed in residents learning ultrasound-guided regional anesthesia [18]. Other common sources of error during novice practice and beyond include failure to appreciate the nuances between acoustic artifact and nerve and failure to distinguish between adjacent isoechoic structures, e.g., tendon and nerve.

The use of a dual nerve stimulation–ultrasound technique may improve block efficiency and efficacy while preventing injection of local anesthetic at a nonneural location. Even the experienced practitioner may benefit from the reassurance provided by nerve stimulation when faced with a challenging obese patient where target neural structures may be difficult to identify with precision at a deep location.

Stimulating Catheters

Peripheral Nerve Blocks

When compared with a single-shot technique, continuous regional anesthesia has the potential to improve the quality and duration of analgesia [19] and to cause fewer systemic side effects including local anesthetic toxicity. In addition, it may be associated with a less profound motor block and improved functional recovery. However, catheter techniques may be technically difficult and subsequently are subject to either primary or secondary failure, the latter occurring when the catheter tip is dislodged, either partially or fully, so that it is no longer in proximity to the nerve target. Historically, nerve block catheters were placed by identifying the neural target using an insulated stimulating needle through which a catheter was blindly threaded a variable distance with the expectation that it would follow the path of the nerve. A less arbitrary method involves the insertion of a perineural catheter which conducts current to its tip, the stimulating catheter. Though some studies do report a reduction regarding the need for rescue analgesia with stimulating catheters, results are not as consistent as expected with respect to pain scores and functional recovery [20].

Ultrasound-guided perineural catheter insertion appears to offer several advantages related to insertion time, pain score, opioid use, and patient comfort during the block procedure [21, 22]. However, unless certain steps are taken to help with identification of a frequently obscured catheter tip, an ultrasound technique may lead to an indiscriminate catheter tip location. Injection and subsequent aspiration of agitated D5W or air may be used with or without Doppler guidance. This can be done with the transducer placed along the length of the catheter.

A catheter-over-needle assembly may prevent many of the potential problems associated with accurate placement and dislodgment [23]. The catheter is almost indistinguishable from a regular intravenous cannula in appearance and operation (Fig. 4.3). The procedure for insertion is simple and comparable to that for a single-shot nerve block. Once the needle tip has been accurately placed in an appropriate perineural location, the needle is withdrawn. The tip of the catheter is now at an identical location to the original needle tip as seen on ultrasound. To strengthen the overall assembly and prevent subsequent kinking, an inner catheter is then passed through and Luer-locked to the outer catheter. Because there is a tighter fit between the skin and catheter in comparison to a catheter through needle technique, leakage and dislodgement are infrequent.

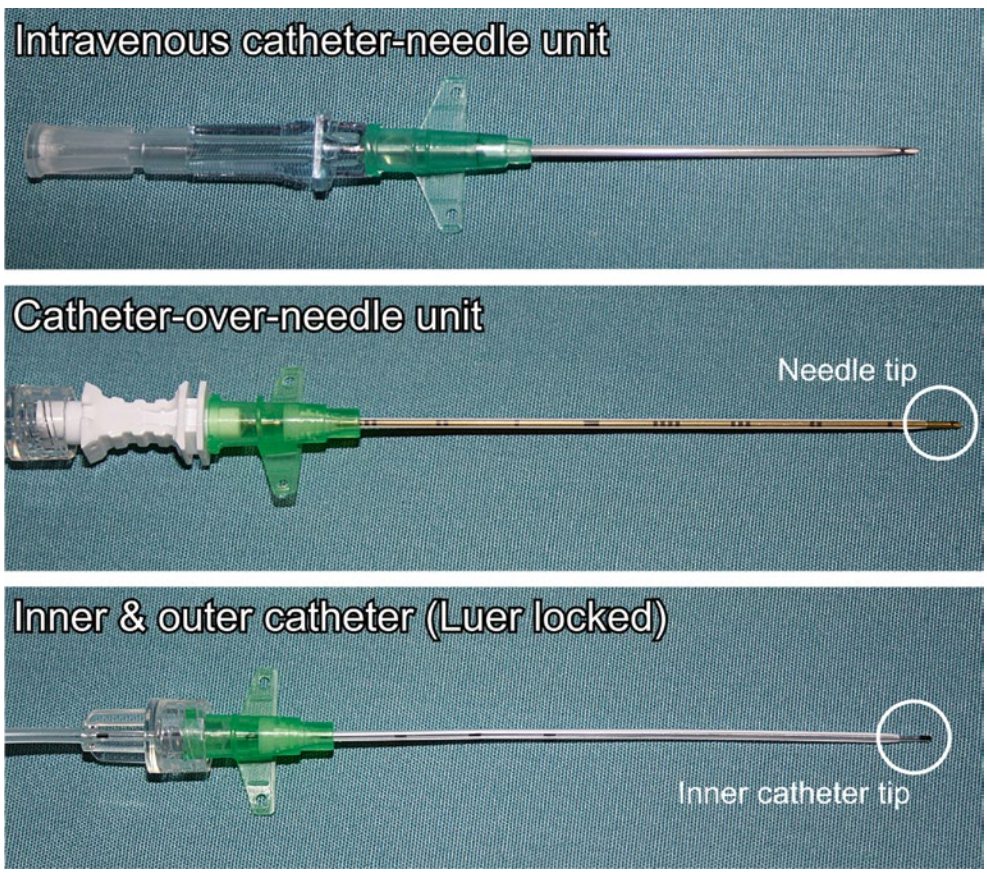


Fig. 4.3 *Top*, standard intravenous assembly consisting of needle within catheter. *Middle*, peripheral nerve block catheter-over-needle assembly (Multiset UPK NanoLine 21156-40E, Pajunk, Germany) consisting of an outer catheter preloaded over a needle. When the needle tip is in an ideal perineural location, the needle is removed, and the inner catheter is threaded through and Luer-locked onto the outer catheter (*bottom panel*)

Central Neuraxial Blocks

The same electrical principles that apply to peripheral nerve blocks can be applied to central neuraxial blocks. Using a specialized stimulating epidural catheter or regular epidural catheter via a saline bridge, electrical current can be conducted to the tip of the catheter to stimulate epidural nerve roots, eliciting a corresponding motor response [24, 25]. This approach has promise as a useful alternative to radiological imaging or ultrasound to monitor or confirm correct placement of epidural catheters [26]. Importantly, the epidural stimulation test appears able to detect most potential locations of the catheter; in addition to the desired epidural space, the test can also detect catheter tip placement in the intrathecal space, against a nerve root, and subcutaneously, depending on the threshold current (Table 4.1). In addition, the epidural stimulation test can also be used to guide the catheter or needle to a specific target epidural location [27].

Table 4.1 Motor responses and currents associated with catheter location during electrical epidural stimulation test

Catheter location	Current	Motor response
Subcutaneous	N/A	No motor response
Subdural	<1 mA	Bilateral (many segments)
Subarachnoid	<1 mA	Unilateral or bilateral
Epidural space		
Non-intravascular	1–15 mA (threshold increases upon local anesthetic injection)[a]	Unilateral or bilateral
Intravascular	1–15 mA (no change in threshold upon local anesthetic injection)	Unilateral or bilateral
Against nerve root	<1 mA	Unilateral

[a]These currents are more reliable for caudal and lumbar placement; thoracic placement may require higher upper limits (e.g., 17 mA). Lower threshold limits apply to both catheter and needle placement

Conclusions

Ultrasound guidance when compared with electrical nerve stimulation appears to be more successful less painful and significantly reduces the incidence of local anesthetic systemic toxicity. It has strengthened our understanding of the needle–nerve relationship and shed new light on highly regarded tenets of electrical nerve stimulation. However, the use of ultrasound guidance does not neutralize the risk of serious nerve injury. Minimum stimulating current may still provide valuable information in this regard. A dual ultrasound–nerve stimulation approach utilizing a low current may combine the superior nerve locating aspects of ultrasound guidance with the discerning quality of nerve stimulation safeguarding both safety and success without compromising efficiency. In terms of its use in placing epidural catheters, the electrical epidural stimulation test has the potential to improve both the safety and success of epidural anesthesia when used in conjunction with a loss-of-resistance technique.

Acknowledgements The authors thank Jenkin Tsui for providing original artwork for the figures, Alex Kwan for assistance with creating and organizing the figures, and Dr. Gareth Corry for assistance with manuscript preparation and management.

Disclosure statement: Dr. Tsui has a licensing agreement with Pajunk for a stimulating catheter epidural kit.

References

1. 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. PubMed.
2. Barrington MJ, Kluger R. Ultrasound guidance reduces the risk of local anesthetic systemic toxicity following peripheral nerve blockade. Reg Anesth Pain Med. 2013;38(4):289–97. PubMed.
3. 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(2): 673–7. PubMed.
4. Tsai TP, Vuckovic I, Dilberovic F, Obhodzas M, Kapur E, Divanovic KA, et al. Intensity of the stimulating current may not be a reliable indicator of intraneural needle placement. Reg Anesth Pain Med. 2008;33(3):207–10. PubMed.
5. 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(6):1844–6. PubMed.
6. Sala-Blanch X, Ribalta T, Rivas E, Carrera A, Gaspa A, Reina MA, et al. Structural injury to the human sciatic nerve after intraneural needle insertion. Reg Anesth Pain Med. 2009;34(3):201–5. PubMed.
7. Moayeri N, Bigeleisen PE, Groen GJ. Quantitative architecture of the brachial plexus and surrounding compartments, and their possible significance for plexus blocks. Anesthesiology. 2008; 108(2):299–304. PubMed.
8. Moayeri N, Groen GJ. Differences in quantitative architecture of sciatic nerve may explain differences in potential vulnerability to nerve injury, onset time, and minimum effective anesthetic volume. Anesthesiology. 2009;111(5):1128–34. PubMed.
9. Lupu CM, Kiehl TR, Chan VW, 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(2):132–9. PubMed.
10. 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(2): 203–6. PubMed.
11. Silvestri E, Martinoli C, Derchi LE, Bertolotto M, Chiaramondia M, Rosenberg I. Echotexture of peripheral nerves: correlation between US and histologic findings and criteria to differentiate tendons. Radiology. 1995;197(1):291–6. PubMed.
12. Cohen JM, Gray AT. Functional deficits after intraneural injection during interscalene block. Reg Anesth Pain Med. 2010;35(4): 397–9. PubMed.
13. Reiss W, Kurapati S, Shariat A, Hadzic A. Nerve injury complicating ultrasound/electrostimulation-guided supraclavicular brachial plexus block. Reg Anesth Pain Med. 2010;35(4):400–1. PubMed.
14. 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(5):1274–80. PubMed.
15. 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(6):534–41. PubMed.
16. Chan VW, Perlas A, McCartney CJ, Brull R, Xu D, Abbas S. Ultrasound guidance improves success rate of axillary brachial plexus block. Can J Anaesth. 2007;54(3):176–82. PubMed.
17. Dingemans E, Williams SR, Arcand G, Chouinard P, Harris P, Ruel M, et al. Neurostimulation in ultrasound-guided infraclavicular block: a prospective randomized trial. Anesth Analg. 2007;104(5): 1275–80, tables of contents. PubMed.
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. PubMed.
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(5):1303–9. PubMed.
20. Morin AM, Kranke P, Wulf H, Stienstra R, Eberhart LH. The effect of stimulating versus nonstimulating catheter techniques for continuous regional anesthesia: a semiquantitative systematic review. Reg Anesth Pain Med. 2010;35(2):194–9. PubMed.
21. 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. PubMed.
22. 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(5):480–5. PubMed.
23. Ip VH, Tsui BC. The catheter-over-needle assembly facilitates delivery of a second local anesthetic bolus to prolong supraclavicular brachial plexus block without time-consuming catheterization steps: a randomized controlled study. Can J Anaesth. 2013;60(7): 692–9. PubMed.

24. Tsui BC, Gupta S, Finucane B. Confirmation of epidural catheter placement using nerve stimulation. Can J Anesth. 1998;45(7): 640–4.
25. Abdallah FW, Chan VW. From the journal archives: practical applicability of the epidural stimulation test. Can J Anesth. 2014; 61(9):881–5.
26. Morley-Foster PK, Abotaiban A, Ganapathy S, Moulin DE, Leung A, Tsui B. Targeted thoracic epidural blood patch placed under electrical stimulation guidance (Tsui test). Can J Anesth. 2006;53(4):375–9.
27. Tsui BC, Wagner A, Cave D, Kearney R. Thoracic and lumbar epidural analgesia via the caudal approach using electrical stimulation guidance in pediatric patients: a review of 289 patients. Anesthesiology. 2004;100(3):683–9.

Chapter 5

Complications of Peripheral Nerve Blocks

Daquan Xu, MD, MSc, MPH (✉)
New York School of Regional Anesthesia (NYSORA), St. Luke`s – Roosevelt Hospitals, New York, NY, USA
e-mail: daquan.xu@gmail.com

Admir Hadzic, MD, PhD
Professor Anesthesiology, Director NAICE, Director NYSORA, Consultant, Department of Anesthesiology, Ziekenhuis oost-Limburg, Genk, BE, USA
e-mail: admir@nysora.com

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_5, © Springer International Publishing Switzerland 2015

Anesthesia after most peripheral nerve blocks (PNBs) wears off to complete return to normal nerve function. However, a small percentage of patients develop transient or persisting deficits of motor or sensory performance or a pain syndrome. Similarly, some patients develop systemic toxicity of local anesthetics. These rare but potentially devastating complications should not be surprising since local anesthetics are applied through sharp needles in considerable concentrations close to nerves or blood vessels. The goal of this chapter is to provide a concise overview of the incidence, mechanism of action, and means of prevention of complications associated with the use of PNBs as well as to forecast future developments in preventing such complications.

Neurological Complications After PNBS

There are relatively few published reports of anesthesia-related nerve injury associated with the use of peripheral nerve blocks (PNBs); the commonly cited incidence (0.02–0.4 %) of severe injury is underestimated owing to underreporting [1, 2]. Most complications of peripheral nerve blocks were reported with upper extremity blocks; the less frequent clinical application of lower extremity nerve blocks may be the main reason that there are even fewer reports of anesthesia-related nerve injury associated with lower extremity PNBs as compared with upper extremity PNBs [3].

Mechanisms of Peripheral Nerve Injury

There are four basic etiologic categories of peripheral nerve injury related to the use of PNBs (Table 5.1) [4]. Laceration results when the nerve is cut partially or completely, such as by a scalpel or a large-gauge cutting needle. Stretch injuries to the nerves may result when nerves or plexuses are stretched in a nonphysiologic or exaggerated physiologic position, such as during shoulder manipulation under an interscalene block (or general anesthesia). Pressure, as a mechanism of nerve injury, is relatively common. Typical example of this mechanism is chronic compression of the nerves by neighboring structures, such as fibrous bands, scar tissue, or abnormal muscles where they pass through fibro-osseous spaces if the space is too small, such as the carpal tunnel. Such chronic compression syndromes are called entrapment neuropathies. The pressure may be repeated and have a cumulative effect (e.g., an ulnar neuropathy resulting from habitually leaning on the elbow). Such a scenario is conceivable, for instance, in a patient who positions the anesthetized arm (e.g., long-acting or continuous brachial plexus block) in a nonphysiologic position for a few hours. Another example of pressure-related nerve injury is prolonged use of a high-pressure tourniquet. Similarly, an intraneural injection may lead to sustained high intraneural pressure, which exceeds capillary occlusion pressure, and leads to nerve ischemia. Vascular nerve damage after nerve blocks can occur when there is acute occlusion of the arteries from which the vasa nervora are derived or from a hemorrhage within a nerve sheath. With injection injuries, the nerve may be directly impaled and the drug injected directly into the nerve, or the drug may be injected into adjacent tissues, causing an acute inflammatory reaction or chronic fibrosis, indirectly involving the nerve. Chemical nerve injury is the result of tissue toxicity of injected solutions (e.g., local anesthetic toxicity, neurolysis with alcohol or phenol, etc.)

Table 5.1 Mechanism of peripheral nerve injury related to peripheral nerve blocks

Mechanical—acute
Laceration
Stretch
Intraneural injection
Vascular
Acute ischemia
Hemorrhage
Pressure
Extraneural
Intraneural
Compartment syndrome
Chemical
Injection of neurotoxic solutions

Severity of Acute Nerve Injuries

Classification of acute nerve injuries is useful when considering the physical and functional state of damaged nerves. In his classification, Seddon introduced the terms neurapraxia, axonotmesis, and neurotmesis (Table 5.2).

Neurapraxia refers to nerve dysfunction lasting several hours to 6 months after a blunt injury to the nerve. In neurapraxia, the nerve axons and connective tissue structures remain intact. The nerve dysfunction probably results from several factors, of which focal demyelination is the most important abnormality. Intraneural hemorrhage, changes in the vasa nervora, disruption of the blood–nerve barrier and axon membranes, and electrolyte disturbances all may add to the impairment of nerve function. Because the nerve dysfunction is rarely complete, clinical deficits are partial and recovery usually occurs within 6–8 weeks, although some neurapraxic lesions (with minimal or no axonal degeneration) may take several months to recover. *Axonotmesis* consists of physical interruption of the axons but within intact Schwann cell tubes and intact connective tissue structures of the nerve (i.e., the endoneurium, perineurium, and epineurium). Sunderland subdivided this group, depending on which of the three structures were involved. With axonotmesis, the nerve sheath remains intact, enabling regenerating nerve fibers to find their way into the distal segment. Consequently, efficient axonal regeneration can take 2–18 months. *Neurotmesis* refers to a complete interruption of the entire nerve including the axons and all connective tissue structures (epineurium included). Clinically, there is total nerve dysfunction. With both axonotmesis and neurotmesis, axonal disruption leads to Wallerian degeneration, from which recovery occurs through the slow process of axonal regeneration. However, with neurotmesis, the two nerve ends may be completely separated, and the regenerating axons may not be able to find the distal stump. For these reasons, effective recovery does not occur unless the severed ends are sutured or joined by a nerve graft.

Table 5.2 Classification systems for nerve injury

Classification	Pathology	Prognosis
Neurapraxia	Myelin injury or ischemia	Excellent recovery in weeks to months
Axonotmesis	Axon loss Variable stromal disruption	Good to poor Depending upon the integrity of supporting structures and distance to muscle
	Endoneurial tubes intact Perineurium intact Epineurium intact	Good Depending upon distance to muscle
	Endoneurial tubes disrupted Perineurium intact Epineurium intact	Poor Axonal misdirection Surgery may be required
	Endoneurial tubes disrupted Perineurium disrupted Epineurium intact	Poor Axonal misdirection Surgery usually required
Neurotmesis	Axon loss Endoneurial tubes severed Perineurium severed Epineurium severed	No spontaneous recovery Surgery required Prognosis after surgery guarded

Mechanical Nerve Injury

Intraneural Injection

Intraneural injection has the potential to create structural damage to the fascicle(s) that is more extensive and less likely to heal as compared to a relatively clear injury caused by a needle. Indeed, the devastating sequelae of sensory and motor loss after injection of various agents into peripheral nerves has been well documented [5, 6]. Nearly all experimental studies on this subject have demonstrated that the site of injection is critical in determining the degree and nature of injury. In order for peripheral nerve injury to occur with an intraneural injection, local anesthetic probably need be injected intrafascicularly; even intentionally, intraneural injections may not invariably lead to neurologic injury [7].

In clinical practice of peripheral nerve blockade (PNB), injection of local anesthetic is typically followed by a latency of 10–30 min for the blockade to develop. In contrast, injections of the same LA for the same PNBs occasionally result in nearly instantaneous, dense, and unusually long-lasting nerve blockade. It is almost certain that such blocks are the result of intraneural injections and the consequent intimate exposure of neural tissue to even low concentration and small volume of local anesthetics [7, 8]. However, intraneural injections can be extrafascicular or intrafascicular. The intraneural–extrafascicular injections are characterized by a diffuse spread of the injectate within the epineurium with escape of the fluid into the extraneural space (Figs. 5.1 and 5.2). Such injections do not necessarily result in nerve injury [5–10]. In contrast, intrafascicular injections almost invariably lead to some degree of neurologic impairment [5] and possibly a substantial proximal spread of the injectate toward the neuraxis [11, 12].

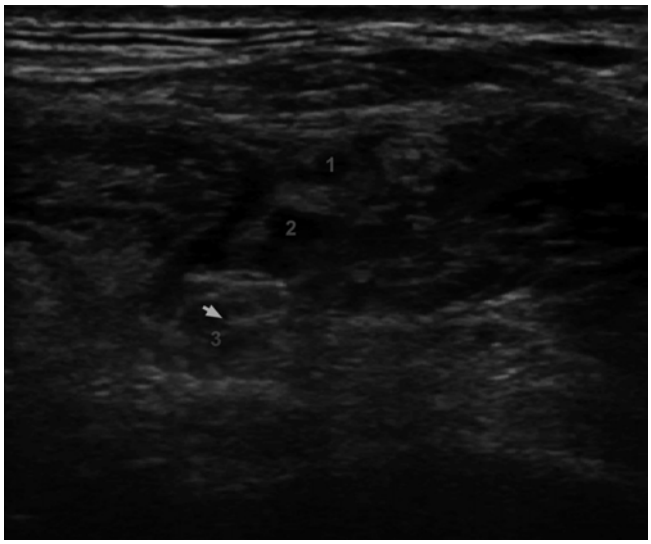


Fig. 5.1 Ultrasonographic image of the brachial plexus during interscalene blockade. Shown are the superior (*1*), middle (*2*), and inferior (*3*) cords of the brachial plexus. The off-line review of the image demonstrated needle insertion in the inferior cord of the brachial plexus (*arrowhead*)

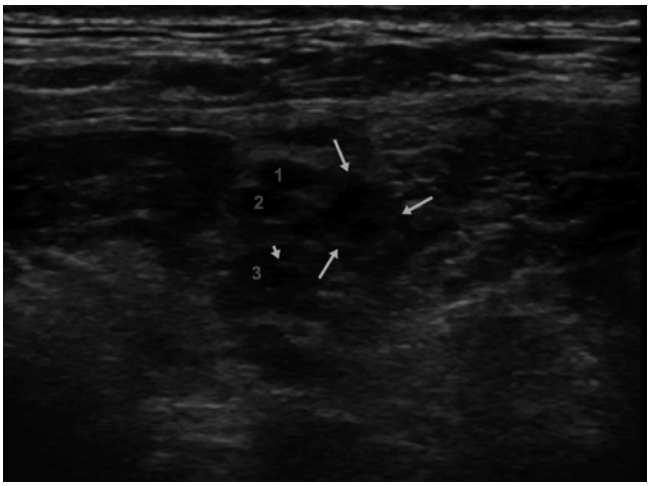


Fig. 5.2 Inadvertent intraneural injection. Shown are the superior (*1*), middle (*2*), and inferior (*3*) cords of the brachial plexus. The tip of the needle is seen within the inferior cord of the brachial plexus (*arrowhead*), while the injection of local anesthetic resulted in escape of the local anesthetic into the interscalene groove (*arrows*)

Histologic features of injury after intraneural injection are rather nonspecific and range from simple mechanical disruption and delamination to fragmentation of the myelin sheath and marked cellular infiltration (Fig. 5.3). Using a variety of animal models of nerve injury, a vast array of cellular changes following peripheral nerve trauma have been documented [8]. The extent of actual neurological damage after an intrafascicular injection can range from neurapraxia with minimal structural damage to neurotmesis with severe axonal and myelin degeneration, depending upon the needle–nerve relationship, agent injected, and dose of the drug. Injury to primary sensory neurons may cause a shift in membrane channel expression, sensitivity to algogenic substances, neuropeptide production, and intracellular signal transduction, both at the injury site and in the cell body in the dorsal root ganglion. These events may lead to increased excitability and the occurrence of acute or chronic pain or dysesthesia often experienced by patients with neurologic injury.

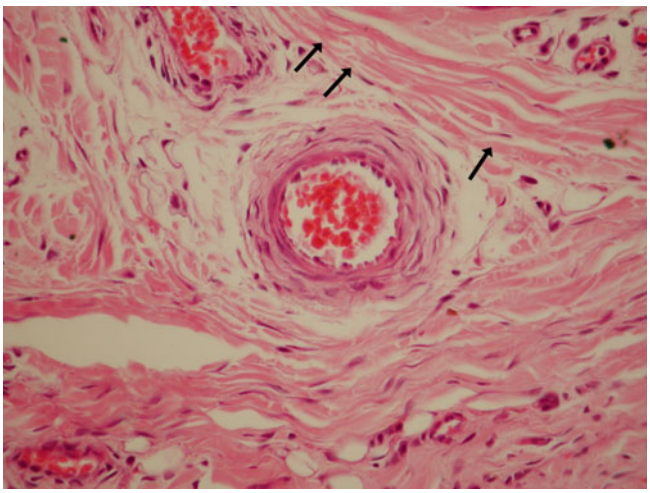


Fig. 5.3 Pathohistologic changes in the sciatic nerve of the dog after an intraneural injection of 2 % lidocaine. Shown are perineurial delamination (*arrows*) and fascicular and epineurial cellular (inflammatory) infiltration

Signs, Symptoms, and Methods to Reduce the Risk of Intraneural Injection

Our ability to monitor and avoid intrafascicular injection during PNBs has been limited. The discussion below focuses on methods commonly used in clinical practice to reduce the risk of intraneural injection.

Pain on Injection

Pain with injection has long been thought of as the cardinal sign of intraneuronal injection; consequently, it is commonly suggested that blocks be avoided in heavily premedicated or anesthetized patients. However, numerous case reports have suggested that pain may not be reliable as a sole warning sign of impending nerve injury, and it may present in only a minority of cases [13]. Fanelli and colleagues have reported unintended paresthesia in 14 % of patients in their study; however, univariate analysis of potential risk factors for postoperative neurologic dysfunction failed to demonstrate paresthesia as a risk factor. In addition, the sensory nature of the pain or paresthesia can be difficult to interpret in clinical practice. For instance, a certain degree of discomfort on injection ("pressure paresthesia") is considered normal and affirmative of impending successful blockade because it is thought that this symptom indicates that injection of local anesthetic has been made in the vicinity of the targeted nerve [14]. In clinical practice, however, it can be difficult to discern when pain–paresthesia on injection is "normal" and when it is the ominous sign of an intraneural injection. Moreover, it is unclear how pain or paresthesia on injection, even when present, can be used clinically to prevent the development of neurologic injury. For instance, in a prospective study on neurological complications of regional anesthesia by Auroy and colleagues, although the participating anesthesiologists did not continue to inject local anesthetic when pain on injection occurred, neurologic injuries after paresthesia still ensued [1].

Minimal Intensity of the Stimulating Current

The optimal current intensity resulting in accurate localization of a nerve has been a topic of controversy. Methods in most recently published reports have reported obtaining nerve stimulation with currents of 0.2–0.5 mA (100 μs) prior to injecting local anesthetics, believing that motor response with current intensities lower than 0.2 mA may be associated with intraneural needle placement. A recent study in an animal model suggested that the ability to obtain motor response to nerve stimulation using current intensity of <0.2 mA may result in an intraneural injection and inflammation of the nerve [15].

Paresthesia Versus Nerve Stimulation

While it is clear that needle trauma can result in nerve damage, it is uncertain whether block techniques that seek to elicit mechanical contact paresthesias during block needle insertion increase the risk of lasting injury. One study demonstrates that seeking paresthesias may increase postoperative lesions [16], but a contrasting study [17] shows only a 0.36 % rate of neuropathy from brachial plexus blocks done with intentional production of paresthesias. It is unresolved whether using electrical stimulation through the needle reduces the incidence of nerve damage. However, advancement of a needle beyond the depth that produces a motor response by current stimulation will typically cause a mechanical paresthesia by contact [18], indicating that electrical nerve localization occurs at a somewhat greater distance than mechanical paresthesia [19]. Nonetheless, the stimulator technique cannot guarantee safety, since it has been shown that the needle may enter the nerve without producing a detectable motor response [1, 20, 21].

Ultrasound-Guided Nerve Blocks

Real-time monitoring of needle placement by US is useful, but of inadequate resolution to avoid intrafascicular injection [10]. During ultrasound-guided nerve blocks, some clinicians first inject a small volume (2–3 ml) of local anesthetic as a precautionary measure to avoid an intrafascicular injection. However, fascicles are small structures, and injury may occur even with minute volumes of LA (≤0.5 mL) [5, 8, 11, 22]. Injections into fascicles are characterized by high opening injection pressure (≥20 psi), followed by a rapid decrease of injection pressure to normal as the perineurium ruptures and local anesthetic leaks out perineurally (Fig. 5.4) [8, 11, 22]. Therefore, intraneural injection of even small volumes of local anesthetic may be hazardous. For success and safety of PNBs, a combination of real-time US needle guidance along with in-line injection pressure monitoring [23] and avoidance of injection with stimulation of <0.2 mA [15] may prove to be the ultimate monitoring during PNBs (Fig. 5.5) [8, 23, 24].

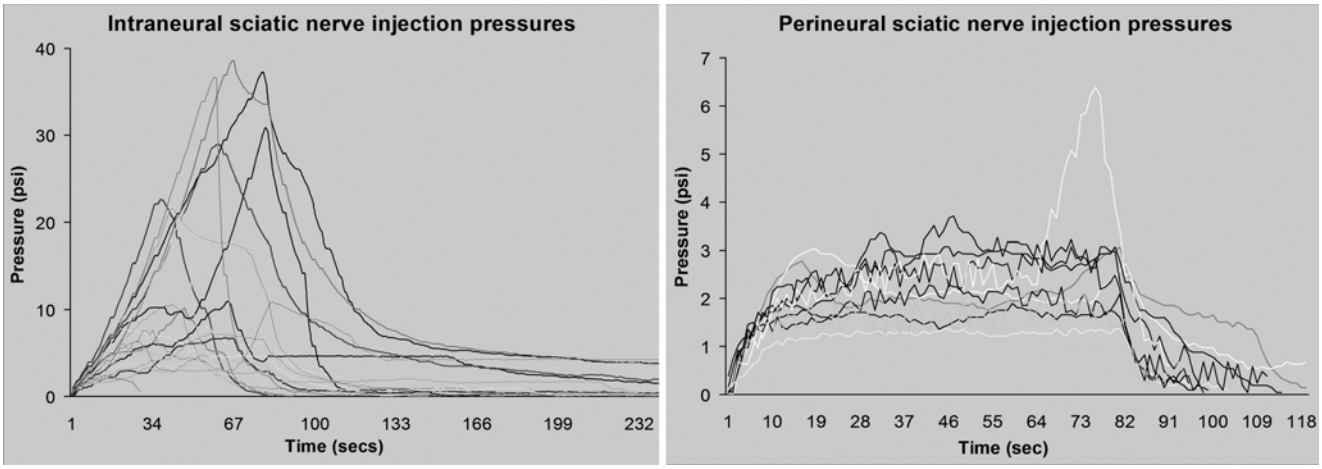


Fig. 5.4 Injection pressure during intraneural (intrafascicular) and perineural application of local anesthetics.Intraneural injection of local anesthetic results in significantly higher injection pressures (Kapur E et al. [8]. With permission of Blackwell publishing)

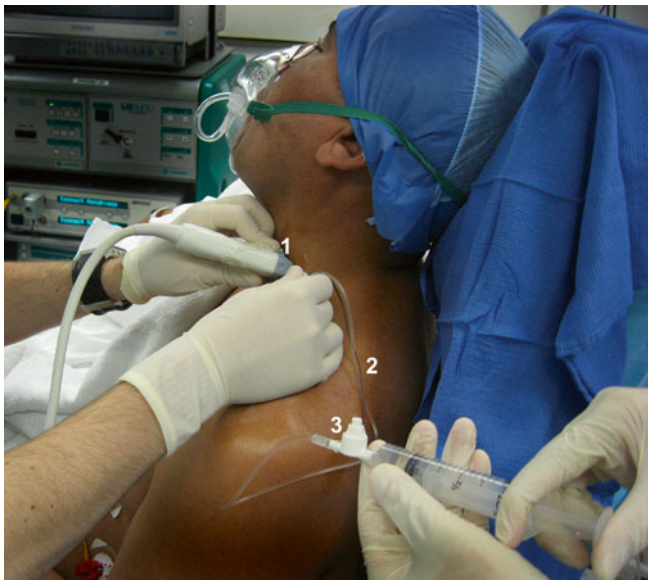


Fig. 5.5 Administration of interscalene brachial plexus block with ultrasound guidance (*1*), electrical nerve stimulation (*2*), and in-line injection pressure monitoring (*3*) to avoid injection pressure >20 psi which may be indicative of an intraneural intrafascicular injection

Needle Design and Direct Needle Trauma

Needle tip design and risk of neurologic injury have been matters of considerable debate. Nearly 30 years ago, Selander and colleagues suggested that the risk of perforating a nerve fascicle was significantly lower when a short-bevel (e.g., 45°) needle was used instead of a long-bevel (12–15°) needle [25]. The result of their work is largely responsible for the current trend of using short-bevel needles (i.e., angles 30–45°) for the majority of major peripheral nerve conduction blocks. However, the more recent work of Rice and McMahon suggested that when placed intraneurally, short-beveled needles cause more mechanical damage than the long-beveled needles [26]. In a rat model, where deliberate penetration of the largest fascicle of the sciatic nerve with 12- to 27°-beveled needles was studied, short-beveled needles resulted in the greatest degree of neural trauma. Their work suggests that sharp needles produce clean, more-likely-to-heal cuts, whereas blunt needles produced noncongruent cuts and more extensive damage on microscopic images. In addition, the cuts produced by the sharper needles were more likely to recover faster and more completely than were the irregular; more traumatic injuries caused by the blunter, short-beveled needles. Although the data on needle design and nerve injury have not been clinically substantiated, the theoretical advantage of short-beveled needles in reducing the risk of nerve penetration has influenced both practitioners and needle manufacturers. Consequently, whenever practical, most clinicians today prefer to use short-beveled needles for major conduction blocks of the peripheral nerves and plexuses. Long-bevel, small-gauge needles, however, continue to be used routinely for many nerve block procedures, such as axillary transarterial brachial plexus block, wrist and ankle blocks, cutaneous nerve block, and others.

Toxicity of Injected Solutions

Local anesthetics produce a variety of cytotoxic effects in cell cultures, including inhibition of cell growth, motility, and survival, and they may also produce morphologic changes [27]. The extent of these effects is proportionate to the duration of exposure to the local anesthetic solution, and they can occur using local anesthetic concentrations in the range used clinically. Within this range, the cytotoxic changes are greater as concentrations increase. Relevant to the clinical setting, the exact site of the local anesthetic deposition plays a critical role in determining the pathogenic potential. After application of local anesthetics outside a fascicle, the regulatory function of the perineurial and endothelial blood–nerve barrier is only minimally compromised. The normally hypertonic endoneural fluid becomes hypotonic, with the accumulation of edema, increased perineural permeability, and increased fluid pressures within the fascicles [28]. Inflammatory changes, as well as myelin and Schwann cell injury, have been identified [28–30].

High concentrations of extrafascicular anesthetics produce axonal injury independent of edema formation and elevated endoneurial fluid pressure [31]. The duration of exposure and concentration of local anesthetic determine the degree and incidence of local anesthetic-induced residual paralysis [32–34]. The importance of these changes after extrafascicular injections in contributing to clinical cases of nerve injury has not been determined, but it is prudent to use the minimum necessary local anesthetic concentrations. Since small-fiber neurons are more sensitive to chemical damage, the manifestations of local anesthetic nerve damage may include spontaneous paresthesias and deficits in pain and temperature perception but not loss of motor, touch, or proprioceptive function [35].

Injection of local anesthetic within a nerve fascicle has been invariably neurotoxic in numerous animal models [5]. Intrafascicular injection of saline alone, lidocaine 1 %, and bupivacaine 0.5 % results in evidence of axonal degeneration and barrier changes. Findings are progressively worse with increasing concentrations of both agents, especially in concentrations above the clinically used range [36, 37].

In addition to local anesthetics themselves, various adjuvant agents injected together with the local anesthetics for neural blockade may play a role in causing nerve damage. Epinephrine amplifies the vasocompression effects of local anesthetics [38]. The addition of epinephrine has been shown to increase the neurotoxicity of bisulfite-containing chloroprocaine solutions [29] and to increase the axonal degeneration that follows intrafascicular bupivacaine injection [36, 37]. Nerve blood flow, especially in the large epineural feeding arteries, is sensitive to the vasoconstrictive effect of injected epinephrine at usual concentrations [39]. Regardless of these laboratory findings, however, the clinical use of epinephrine is common, and its potential to cause clinical neurotoxicity remains controversial.

Nerve Ischemia

The earliest response of the peripheral sensory neuron to ischemia is depolarization and generation of spontaneous activity, perceived by the subject as paresthesias. This is followed by blockade of slow-conducting myelinated fibers and eventually all neurons [21], possibly through accumulation of excess intracellular calcium [40], which accounts for the loss of sensation with initiation of limb ischemia.

Nerve function returns within six hours if ischemic times are less than two hours [41], and ischemic periods of up to 6 h may fail to produce permanent structural changes in the nerves [42]. However, more detailed pathological examination after 3 h of reperfusion can show edema and fiber degeneration that lasts for 1–2 weeks, followed by a phase of regeneration lasting 6 weeks [43]. In addition to neuronal damage, oxidative injury associated with ischemia and reperfusion also affects the Schwann cells, initiating apoptosis

[44]. Recently, sensory testing of rats 2–4 h after a 3-h period of hind paw ischemia demonstrated hypersensitivity to cold and innocuous or nociceptive mechanical stimuli reminiscent of human hyperalgesic syndromes [45].

Tourniquets may cause nerve damage either by ischemia or mechanical deformation. The initial effect of direct compression of the nerve by the tourniquet is failure of transmission by fast conducting myelinated fibers [46]. Prolonged nerve dysfunction results from damage to the portion of the nerve under the edge of the pneumatic cuff, where the mechanical distortion of the nerve is maximal. Irreversible damage, including substantial distortion of myelin lamellae and axonal shrinkage, may ensue as early as 2–4 h after tourniquet inflation [47] and predominantly affects large diameter neurons [48]. Thus, the main findings of tourniquet-induced neuropathy are motor loss and diminished touch, vibration, and position sense, with preserved senses of heat, cold, and pain and the absence of spontaneous paresthesias [49]. One may minimize nerve damage by using wide cuffs and inflation pressures just adequate for arterial occlusion [50], but periodic deflation of the cuff (even as much as 10 min down every hour) has no beneficial effects on the compression trauma [51]. Alternating between the two cuffs of a double-cuff tourniquet may allow prolonged blood flow interruption with diminished mechanical damage to the nerves, since each site is compressed for only half the total duration [52].

Systemic Toxicity of Local Anesthetics
(See also Chap. 1)

Systemic toxicity of local anesthetics is manifested in either the central nervous system (CNS) or the cardiovascular system (CVS); CNS toxicity occurs at lower plasma concentrations than CVS toxicity.

Central Nervous System Toxicity

The reported incidence of seizures during regional anesthesia is between 0.1 and 1 per thousand [1, 53]. Local anesthetic plasma concentrations high enough to cause seizures can be reached either by inadvertent intravascular (venous or arterial) injection, systemic absorption from the perineural or epidural injection site, or a combination of the two.

Human studies of local anesthetic CNS toxicity demonstrate that if plasma concentrations rise slowly enough, subjects will progress through a fairly stereotypic series of CNS symptoms prior to developing seizures [54–56]. The early CNS symptoms of rising local anesthetic plasma concentration include tongue or circumoral numbness followed by "lightheadedness" and later visual or auditory disturbances. One could reasonably argue that an awake patient would be able to warn a clinician of developing CNS symptoms prior to seizures and that if the local anesthetic is being injected slowly enough, the injection could be aborted before a dose large enough to cause seizures (or worse) is administered. Consistent with this argument are studies demonstrating that un-premedicated subjects can detect an intravenous bolus of lidocaine (1.5 mg/kg), 2-chloroprocaine (90 mg), or bupivacaine (25 mg) with 100 % sensitivity but that the sensitivity drops to between 60 and 80 % with as little as 1.5–2.8 mg midazolam and 60–96 μg fentanyl [57, 58]. However, CNS toxicity from systemic absorption of local anesthetic generally occurs after all of the local anesthetic has been injected; thus, premonitory symptoms will occur too late to prevent a toxic dose from being administered. In addition, seizures that occur as a result of inadvertent local anesthetic injection into the carotid or vertebral arteries during blocks in the neck (e.g., stellate ganglion, interscalene) have occurred after less than 1.5 ml of local anesthetic was injected [59, 60]. Consequently, these patients have seizures too rapidly to be able to signal a warning. Similarly, patients in whom local anesthetic is inadvertently injected rapidly intravenously may well develop seizures before they have time to appreciate CNS warning symptoms and interject to prevent administration of a toxic dose. Therefore, keeping patients unanesthetized or unsedated for the purpose of avoiding severe CVS toxicity may not provide any discernable benefit and may increase the risk of CNS and potentially cardiovascular toxicity.

The addition of epinephrine or isoproterenol produces characteristic changes in heart rate, blood pressure, or T-wave amplitude that are sensitive indicators of intravascular injection, even in subjects who are beta-blocked or anesthetized [61]. Fentanyl has also been shown to be a sensitive indicator of intravascular injection in obstetrics [62]. Similarly, air (1 ml) has been successfully used to detect intravascular injection by monitoring the heart with a precordial Doppler to detect the characteristic "mill wheel" murmur of air in the right atrium [63, 64]. Importantly, the dose of epinephrine/isoproterenol and the diagnostic criteria for considering a cardiovascular response to be positive are different in anesthetized vs. awake patients (and in "elderly" patients for that matter), but the sensitivity is still 100 % if the appropriate test dose and criteria are used [65–67]. The patient's report of CNS symptoms, however, is not 100 % sensitive because of the large number of patients incapable of either sensing or adequately expressing their symptoms (e.g., young children, demented patients, developmentally delayed patients, patients with a language barrier, etc.) [68, 69]. Because an appropriately applied and monitored test dose is 100 % effective at detecting intravascular injection in "all" patients (whether they are awake, sedated, or anesthetized), the test dose, and not patient report, may be considered the optimum method to detect and prevent intravascular injections.

Apart from issues related to detecting intravascular injections, appropriate sedation can actually decrease the risk of seizures. Specifically, because sedative hypnotics like benzodiazepines, barbiturates, and propofol significantly raise the threshold for local anesthetic-induced seizures (i.e., increase the plasma concentration at which seizures occur), their use can increase the safety margin for local anesthetic CNS toxicity. Seizure prevention is likely greater for general anesthesia because the magnitude of CNS depression is significantly greater than that achieved by "sedation." However, when sedation results in significant respiratory depression (e.g., if opioids are a part of the sedative "mix"), the risk of seizures may actually be increased by a pharmacokinetic mechanism.

Cardiovascular Toxicity

Unlike CNS toxicity, which can occur from absorption of local anesthetic properly deposited at the intended block site, higher local anesthetic concentrations required to produce significant cardiovascular toxicity, particularly cardiac arrest, can only be reached by intravascular injection. Consequently, prevention of cardiovascular toxicity rests primarily on the ability to prevent significant intravascular injection of local anesthetic. Perhaps the most effective method to prevent intravascular injection is by slow, incremental injection of a local anesthetic solution containing a marker of intravascular injection (e.g., epinephrine, isoproterenol, air, etc.) while simultaneously monitoring for the appropriate objective cardiovascular response.

However, heavy sedation/anesthesia may modulate the manifestation of cardiovascular toxicity. Animal studies document that intralipid can raise the threshold for bupivacaine-induced cardiovascular toxicity [70] and that it is also effective for resuscitation from bupivacaine-induced cardiovascular collapse [71–73]. A recent human case report of successful resuscitation after severe systemic toxicity of bupivacaine is consistent with the experimental animal data [74]. Intralipid's salutary effects appear to be the result of a change in blood lipophilicity that results in bupivacaine partitioning out of the heart and into the blood stream. Because propofol is emulsified in intralipid, it is conceivable that propofol sedation/anesthesia may reduce the risk of bupivacaine cardiovascular toxicity. In fact, propofol anesthesia raises the threshold for seizures and attenuates dysrhythmias, bradycardia, and hypotension, although not asystole, in rats receiving i.v. bupivacaine [70]. Whether this incomplete cardioprotective effect of propofol is also the result of a shift in bupivacaine partitioning between the myocardium and blood is unknown.

Volatile anesthetics (sevoflurane) alter the manifestations of bupivacaine cardiovascular activity. In rats, sevoflurane raised the dose of bupivacaine necessary to cause dysrhythmias even more so than did propofol [70]. Sevoflurane also increased the dose of bupivacaine required to produce a 50 % reduction in heart rate but had no effect on the dose of bupivacaine required to produce hypotension or asystole. Similarly, benzodiazepine premedication alters the manifestations of bupivacaine cardiovascular toxicity in pigs [75]. Both diazepam and midazolam increased the dose of bupivacaine required to produce cardiac dysrhythmias and prevented the early hypertension and tachycardia experienced by the control animals. However, benzodiazepine neither altered the bupivacaine dose nor plasma concentration at which cardiovascular collapse occurred. Interestingly, while all of the control animals in the Bernards et al. study developed tonic–clonic seizures prior to cardiovascular collapse, only 2 of the 20 animals premedicated with a benzodiazepine developed seizures [75]. However, the mechanism by which general anesthesia or benzodiazepine-mediated sedation alters the early hemodynamic manifestations of bupivacaine toxicity remains unknown.

In summary, the risk of severe complications associated with administration of peripheral nerve blocks is probably similar to that with general anesthesia. However, complications related to the nerve blocks fall in two categories—a neurologic injury and systemic toxicity of local anesthetics. While these complications are relatively rare, they have occurred even in expert hands due to the lack of precise monitoring during nerve localization and administration of local anesthetics. The recent introduction of real-time monitoring of needle placement, injection pressure, and minimal stimulating current monitoring will likely go a long way in transforming regional anesthesia into a more objective, reproducible, and safer discipline.

References

1. Auroy Y, Benhamou D, Bargues L. Major complications of regional anesthesia in France: The SOS regional anesthesia hotline service. Anesthesiology. 2002;97:1274–80.
2. 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.
3. Hadzic A, Vloka J. Kuroda M, et a: The practice of peripheral nerve blocks in the United States: a national survey. Reg Anesth Pain Med. 1998;23:241–6.
4. Hadzic A, Vloka J. Complications of peripheral nerve blocks. 1st ed. New York: McGrawHill; 2004.
5. Mackinnon S, Hudson A, Llamas F, Dellon A, Kline D, Dan A, Hunter R. Peripheral nerve injury by chymopapain injection. J Neurosurg. 1984;61:1–8.
6. Mackinnon S, Dellon A. Classification of nerve injuries as the basis of treatment. In: Mackinnon S, editor. Surgery of the peripheral nerve. New York: Thieme Medical Publishers Inc; 1988. p. 35–63.
7. 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.

8. Kapur E, Dilberovic F, Zaciragic A, Cosovic E, Vuckovic I, Divanovic KA, Mornjakovic Z, Mirsad B, Borgeat A, Thys DM, Hadzic A. Neurologic and histologic outcome after intraneural injections of lidocaine in canine sciatic nerves. Acta Anaesthesiol Scand. 2006;51(1):101–7.
9. Sala–Blanch X, Pomes J, Matute P, Valls–Sole J, Carrera A, Tomas X, Garcia–Diez AI. Intraneural injection during anterior approach for sciatic nerve block. Anesthesiology. 2004;101:1027–30.
10. Schafhalter–Zoppoth I, Zeitz I, Gray A. Inadvertent femoral nerve impalement and intraneural injection visualized by ultrasound. Anesthesiology. 2004;99:620–30.
11. Selander D, Sjostrand J. Longitudinal spread of intraneurally injected local anesthetics. An experimental study of the initial neural distribution following intraneural injections. Acta Anesth Scand. 1978;22:622–34.
12. Dutton R, Eckhardt 3rd W, Sunder N. Total spinal anesthesia after interscalene blockade of the brachial plexus. Anesthesiology. 1994;80:939–41.
13. Bhananker S, Domino K. What actions can be used to prevent peripheral nerve injury. In: Evidence–based practice of anesthesiology. Philadelphia: Elsevier Inc; 2004. p. 228–35.
14. Winnie A. Interscalene brachial plexus block. Anesth Analg. 1970;49:455–66.
15. Voelckel W, Klima G, Krismer C, Halsonger C, Stadlbauer H, Wenzel V, von Goedecke A. Signs of inflammation after sciatic nerve block in pigs. Anesth Analg. 2005;101:1844–6.
16. Selander D, Edshage S, Wolff T. Paresthesiae or no paresthesiae? Acta Anaesthesiol Scand. 1979;23:27–33.
17. Winchell SW, Wolfe R. The incidence of neuropathy following upper extremity nerve blocks. Reg Anaesth. 1985;10:12–5.
18. 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.
19. Karaca P, Hadzic A, Yufa M, Vloka JD, Brown AR, Visan A, Sanborn K, Santos AC. Painful paresthesiae are infrequent during brachial plexus localization using low–current peripheral nerve stimulation. Reg Anesth Pain Med. 2003;28:380–3.
20. 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.
21. Urmey WF, Stanton J. Inability to consistently elicit a motor response following sensory paresthesia during interscalene block administration. Anesthesiology. 2002;6:552–4.
22. Hadzic A, Dilberovic F, Shah S, Kulenovic A, Kapur E, Zaciragic A, Cosovic E, Vuckovic I. 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.
23. Tsui B, Li L, Pillay J. Compressed air injection technique to standardize block injection pressures. Can J Anesth. 2006;53:1098.
24. Gebhardt R, Ghelber O, Warters R, Pivalizza E, Szmuk P. Local Anesthetic Injection–Pressure Monitoring during Nerve Blocks with the Compuflo™ Injection Pump. ASA Annual Meeting Abstracts Session. 2006;A1136.
25. Selander D, Dhuner K, Lundborg G. Peripheral nerve injury due to injection needles used for regional anesthesia. Acta Anaesthesiol Scand. 1977;21:182–9.
26. Rice A, McMahon S. Peripheral nerve injury caused by injection needles used in regional anaesthesia: Influence of bevel configuration, studied in a rat model. Br J Anaesth. 1992;9:433–8.
27. Sturrock JE, Nunn JF. Cytotoxic effects of procaine, lignocaine and bupivacaine. Br J Anaesth. 1979;51:273–81.
28. Myers RR, Kalichman MW, Reisner LS, Powell HC. Neurotoxicity of local anesthetics: altered perineurial permeability, edema, and nerve fiber injury. Anesthesiology. 1986;64:29–35.
29. Barsa J, Batra M, Fink BR, Sumi SM. A comparative in vivo study of local neurotoxicity of lidocaine, bupivacaine, 2–chloroprocaine, and a mixture of 2–chloroprocaine and bupivacaine. Anesth Analg. 1982;61:961–7.
30. Powell HC, Kalichman MW, Garrett RS, Myers RR. Selective vulnerability of unmyelinated fiber Schwann cells in nerves exposed to local anesthetics. Lab Invest. 1988;59:271–80.
31. Kalichman MW, Powell HC, Myers RR. Pathology of local anesthetic–induced nerve injury. Acta Neuropathol (Berl). 1988;75:583–9.
32. Li DF, Bahar M, Cole G, Rosen M. Neurological toxicity of the subarachnoid infusion of bupivacaine, lignocaine or 2–chloroprocaine in the rat. Br J Anaesth. 1985;57:424–9.
33. Ready LB, Plumer MH, Haschke RH, Austin E, Sumi SM. Neurotoxicity of intrathecal local anesthetics in rabbits. Anesthesiology. 1985;63:364–70.
34. Lambert LA, Lambert DH, Strichartz GR. Irreversible conduction block in isolated nerve by high concentrations of local anesthetics. Anesthesiology. 1994;80:1082–93.
35. Woolley EJ, Vandam LD. Neurological sequelae of brachial plexus nerve block. Ann Surg. 1959;149:53–60.
36. Gentili F, Hudson AR, Hunter D, Kline DG. Nerve injection injury with local anesthetic agents: a light and electron microscopic, fluorescent microscopic, and horseradish peroxidase study. Neurosurgery. 1980;6:263–72.
37. Selander D, Brattsand R, Lundborg G, et al. Local anesthetics: Importance of mode of application, concentration and adrenaline for the appearance of nerve lesions. Acta Anaesth Scand. 1979;23:127–36.
38. Partridge BL. The effects of local anesthetics and epinephrine on rat sciatic nerve blood flow. Anesthesiology. 1991;75:243–50.
39. Myers RR, Heckman HM. Effects of local anesthesia on nerve blood flow: studies using lidocaine with and without epinephrine. Anesthesiology. 1989;71:757–62.
40. Duchen MR. Effects of metabolic inhibition on the membrane properties of isolated mouse primary sensory neurones. J Physiol. 1990;424:387–409.
41. Lundborg G. Structure and function of the intraneural microvessels as related to trauma, edema formation, and nerve function. J Bone Joint Surg Am. 1975;57:938–48.
42. Tountas CP, Bergman RA. Tourniquet ischemia: ultrastructural and histochemical observations of ischemic human muscle and of monkey muscle and nerve. J Hand Surg [Am]. 1977;2:31–7.
43. Iida H, Schmelzer JD, et al. Peripheral nerve ischaemia: reperfusion injury and fiber regeneration. Exp Neurol. 2003;184:997–1002.
44. Iida H, Schmeichel AM, Wang Y, Schmelzer JD, Low PA. Schwann cell is a target in ischemia–reperfusion injury to peripheral nerve. Muscle Nerve. 2004;30:761–6.
45. Coderre TJ, Xanthos DN, Francis L, Bennett GJ. Chronic post–ischemia pain (CPIP): a novel animal model of complex regional pain syndrome–type I (CRPS–I; reflex sympathetic dystrophy) produced by prolonged hindpaw ischemia and reperfusion in the rat. Pain. 2004;112:94–105.
46. Fern R, Harrison PJ. The contribution of ischaemia and deformation to the conduction block generated by compression of the cat sciatic nerve. Exp Physiol. 1994;79:583–92.
47. Ochoa J, Fouler TJ, Gilliatt RW. Anatomical changes in peripheral nerve compressed by a pneumatic tourniquet. J Anat. 1972;113:433–55.
48. Nitz AJ, Matulionis DH. Ultrastructural changes in rat peripheral nerve following pneumatic tourniquet compression. J Neurosurg. 1982;57:660–6.
49. Mullick S. The tourniquet in operations upon the extremities. Surg Gynecol Obstet. 1978;146:821–6.
50. Moore MR, Garfin SR, Hargens AR. Wide tourniquets eliminate blood flow at low inflation pressures. J Hand Surg [Am]. 1987;12:1006–11.

51. Mohler LR, Pedowitz RA, Myers RR, Ohara WM, Lopez MA, Gershuni DH. Intermittent reperfusion fails to prevent posttourniquet neurapraxia. J Hand Surg [Am]. 1999;24:687–93.
52. Dreyfuss UY, Smith RJ. Sensory changes with prolonged double–cuff tourniquet time in hand surgery. J Hand Surg [Am]. 1988;13:736–40.
53. 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.
54. Scott B. Acute intravenous tolerance of etidocaine. Acta Anaesthesiol Scand Suppl. 1975;60:117–20.
55. Scott DB. Evaluation of clinical tolerance of local anaesthetic agents. Br J Anaesth. 1975;47(Suppl):328–31.
56. Scott DB, Lee A, Fagan D, Bowler GM, Bloomfield P, Lundh R. Acute toxicity of ropivacaine compared with that of bupivacaine. Anesth Analg. 1989;69:563–9.
57. 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.
58. 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.
59. Mahli A, Coskun D, Akcali DT. Aetiology of convulsions due to stellate ganglion block: a review and report of two cases. Eur J Anaesthesiol. 2002;19:376–80.
60. Korevaar WC, Burney RG, Moore PA. Convulsions during stellate ganglion block: a case report. Anesth Analg. 1979;58:329–30.
61. Guay J. The epidural test dose: a review. Anesth Analg. 2006;102:921–9.
62. Yoshii WY, Miller M, Rottman RL, Kotelko DM, Wright WC, Stone JJ, Rasmus KT, Rosen PJ. Fentanyl for epidural intravascular test dose in obstetrics. Reg Anesth. 1993;18:296–9.
63. Leighton BL, Topkis WG, Gross JB, Arkoosh VA, Lee SH, Huffnagle HJ, Huffnagle SL. Multiport epidural catheters: does the air test work? Anesthesiology. 2000;92:1617–20.
64. Leighton BL, Gross JB. Air: an effective indicator of intravenously located epidural catheters. Anesthesiology. 1989;71:848–51.
65. Tanaka M, Goyagi T, Kimura T, Nishikawa T. The efficacy of hemodynamic and T wave criteria for detecting intravascular injection of epinephrine test doses in anesthetized adults: a dose–response study. Anesth Analg. 2000;91:1196–202.
66. Tanaka M, Nishikawa T. The combination of epinephrine and isoproterenol as a simulated epidural test dose in isoflurane–anesthetized adults. Anesth Analg. 1998;86:1312–7.
67. Tanaka M, Nishikawa T. A comparative study of hemodynamic and T–wave criteria for detecting intravascular injection of the test dose (epinephrine) in sevoflurane–anesthetized adults. Anesth Analg. 1999;89:32–6.
68. Tanaka M, Nishikawa T. Efficacy of simulated intravenous test dose in the elderly during general anesthesia. Reg Anesth Pain Med. 1999;24:393–8.
69. Tanaka M, Takahashi S, Kondo T, Matsumiya N. Efficacy of simulated epidural test doses in adult patients anesthetized with isoflurane: a dose–response study. Anesth Analg. 1995;81:987–92.
70. Ohmura S, Ohta T, Yamamoto K, Kobayashi T. A comparison of the effects of propofol and sevoflurane on the systemic toxicity of intravenous bupivacaine in rats. Anesth Analg. 1999;88:155–9.
71. Weinberg G, Ripper R, Feinstein DL, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine–induced cardiac toxicity. Reg Anesth Pain Med. 2003;28:198–202.
72. Weinberg GL, Ripper R, Murphy P, Edelman LB, Hoffman W, Strichartz G, Feinstein DL. Lipid infusion accelerates removal of bupivacaine and recovery from bupivacaine toxicity in the isolated rat heart. Reg Anesth Pain Med. 2006;31:296–303.
73. Weinberg GL, VadeBoncouer T, Ramaraju GA. Garcia–Amaro MF, Cwik MJ: Pretreatment or resuscitation with a lipid infusion shifts the dose–response to bupivacaine–induced asystole in rats. Anesthesiology. 1998;88:1071–5.
74. 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;105:217–8.
75. Bernards CM, Artru AA. Effect of intracerebroventricular picrotoxin and muscimol on intravenous bupivacaine toxicity. Evidence supporting central nervous system involvement in bupivacaine cardiovascular toxicity. Anesthesiology. 1993;78:902–10.

Part II
Head and Neck Region

Chapter 6

Regional Anesthesia in Ophthalmology

André van Zundert, MD, PhD, FRCA, EDRA, FANZA (✉)
Professor and Chairman, Discipline of Anesthesiology, Chair, The University of Queensland-Faculty of Medicine and Biomedical Sciences, Royal Brisbane and Women's Hospital-Ned Hanlon Building level 4, Herston Campus-Brisbane, Queensland 4029, Australia
e-mail: vanzundertandre@gmail.com

Chandra M. Kumar, FFARCS, FRCA, MSc
Senior Consultant in Anesthesia, Khoo Teck Puat Hospital, 90 Yishun Central, 768828, Singapore
e-mail: chandra.kumar2406@gmail.com

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_6, © Springer International Publishing Switzerland 2015

The pioneering work of Jankovic [1] on the anesthetic effect of cocaine in the context of ophthalmic surgery was the historical starting point for local and regional anesthesia. Local anesthesia is commonly used today for majority of ophthalmic surgical procedures. Cataract surgery is the most common surgery and local anesthesia is the norm, but the provision of anesthesia varies worldwide but exact frequency of the use of a particular technique is not known. Patient comfort, safety, and low complication rates are the essentials of local anesthesia. The technique is chosen to provide anesthesia with or without reduction of eye movements. Ophthalmic blocks can be achieved by inserting a needle or a blunt cannula.

Orbital Anatomy

The globe or bulbus oculi lies in front of the orbit and is covered by eyelids. The orbit is an irregular four-sided pyramid with its apex pointing posteromedially and its base facing anteriorly (Figs. 6.1, 6.2, and 6.3). The base is formed by the surface of the cornea, the conjunctiva, and the lids. The medial wall of orbit is parallel to the sagittal plane, but the lateral wall is angled inward at 45°. The roof is horizontal but the floor slopes upward, front to back by 10°. The globe is closer to the roof than the floor of the orbit. The length of orbit varies from 42 to 54 mm. One-fourth of the orbit is filled by the globe (7 ml), and the remainder is filled with fatty tissue, vessels, lacrimal gland, connective tissue, nerves, and extraocular muscles. The transparent cornea lies on the anterior surface of the eyeball (Fig. 6.4).

Underneath this lies the crystalline lens which is located in front of the iris, with its central opening, the pupil. The conjunctiva covers relatively the tough sclera. The optic nerve enters the globe on the hind surface of the globe slightly medial to the axis. The anterior chamber of the eye is bounded by the cornea, the iris, and the lens. The posterior chamber of the eye encircles the lens in a ringlike shape, and the posterior chamber of the eye contains the vitreous body.

The movements of the globe are made possible by four straight muscles, inferior rectus (oculomotor nerve), lateral rectus (abducent nerve), medial rectus (oculomotor nerve), and superior rectus (oculomotor nerve), and two oblique, superior oblique muscle (trochlear nerve) and inferior oblique muscle (oculomotor nerve) (Figs. 6.2, 6.3, and 6.5). The rectus muscles arise from the annulus of Zinn near the apex of the orbit and insert anterior to the equator of the globe thus forming an incomplete cone. Thus, the orbit is divided into two compartments although not completely separate, an extraconal compartment and an intraconal compartment. Injected local anesthetics are easily able to cross the barrier between two compartments by diffusion. Within the annulus and the muscle cone lie the optic nerve (II), the oculomotor nerve (III containing both superior and inferior branches), the abducent nerve (VI nerve), the nasociliary nerve (a branch of V nerve), the ciliary ganglion, and vessels.

The sensory supply of the orbit is provided by lacrimal, frontal, and nasociliary branches of the ophthalmic division of the trigeminal nerve. (See Chap. 10 Trigeminal Nerve, pp. 135–7) Autonomic fibers run from the ciliary ganglion situated within the cone near to the orbital apex (Figs. 6.2 and 6.3). The ciliary ganglion, a tiny collection of nerve cells, lies in the posterior part of the orbit between the optic nerve and the lateral rectus muscle. The sensory and sympathetic roots of the ciliary ganglion are provided by the nasociliary nerve and the neural network around the internal carotid artery but do not always connect to the ciliary ganglion. Their fibers can reach the eye directly via the ciliary nerves. The sympathetic fibers which are already postganglionic after they have switched to the cervical sympathetic trunk ganglia can accompany the ophthalmic artery and its branches on the way to their destination. Stimuli from the cornea, iris, choroid, and intraocular muscles are conducted in the sensory fibers.

The eye and orbital contents receive their main arterial supply from the ophthalmic artery (Fig. 6.6). The ophthalmic artery is a branch of the internal carotid artery. In the orbital cavity, the artery runs forward for a short distance lateral to the optic nerve and medial to the lateral rectus muscle, the abducent and oculomotor nerves, and the ciliary ganglion. The artery then turns medially and crosses above the optic nerve, accompanied by the nasociliary nerve. The venous blood of the orbit is drained by the superior and inferior ophthalmic veins which in turn drain into the cavernous sinus. The superior ophthalmic vein crosses the optic nerve with the ophthalmic artery. The veins of the orbit are tortuous and freely anastomose with one another, and they have no valves. These vessels are known to be damaged when a long needle is inserted deep into the apex.

Tenon capsule (fascial sheath) is a thin membrane that envelops the eyeball and separates it from the orbital fat. The inner surface is smooth and shiny and is separated from the outer surface of the sclera by a potential space the sub-Tenon space. Crossing the space and attaching the fascial sheath to the sclera are numerous delicate bands of connective tissue (Fig. 6.7). Anteriorly, the fascial sheath is firmly attached to the sclera about 3–5 mm posterior to the corneoscleral junction. Posteriorly, the sheath fuses with the meninges around the optic nerve and with the sclera around the exit of the optic nerve. The tendons of all six extrinsic muscles of the eye pierce the sheath as they pass to their insertion on the globe. At the site of perforation, the sheath is reflected back along the tendons of these muscles to form a tubular sleeve. The local anesthetic is injected beneath this part of the sub-Tenon space.

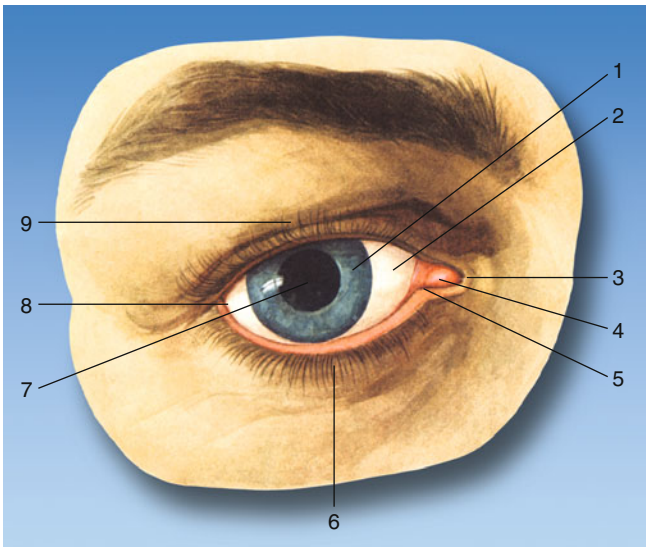


Fig. 6.1 Eyelids and lacrimal apparatus. (*1*) Cornea, (*2*) conjunctiva, (*3*) medial angle of the eye, (*4*) lacrimal caruncle, (*5*) lacrimal papilla, (*6*) inferior eyelid, (*7*) pupil, (*8*) lateral angle of the eye, (*9*) superior eyelid (With permission from D. Jankovic)

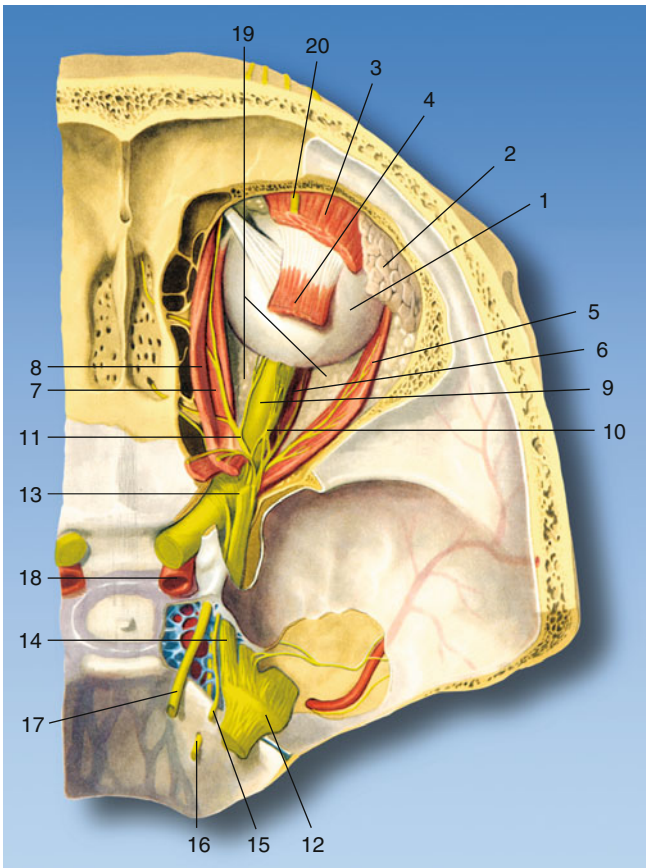


Fig. 6.2 Anatomy of the eye. (*1*) Eyeball, (*2*) lacrimal gland, (*3*) levator palpebrae superioris muscle, (*4*) superior rectus muscle, (*5*) lateral rectus muscle, (*6*) inferior rectus muscle, (*7*) medial rectus muscle, (*8*) superior oblique muscle, (*9*) optic nerve, (*10*) ciliary ganglion, (*11*) nasociliary nerve, (*12*) trigeminal ganglion, (*13*) frontal nerve, (*14*) ophthalmic nerve, (*15*) trochlear nerve, (*16*) abducent nerve, (*17*) oculomotor nerve, (*18*) internal carotid artery, (*19*) retrobulbar fat, (*20*) supraorbital nerve (With permission from D. Jankovic)

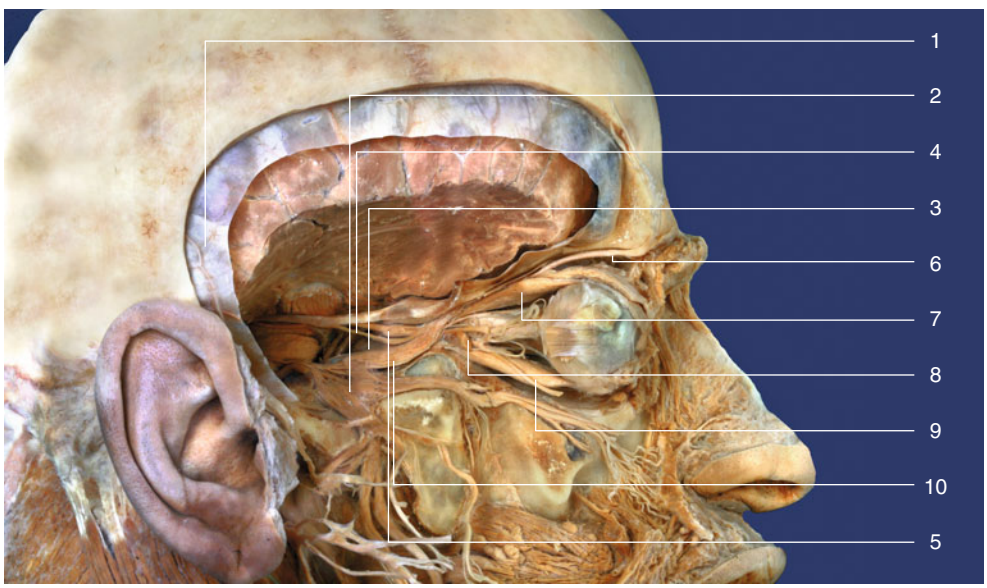


Fig. 6.3 Anatomy. (*1*) Spinal dura mater, (*2*) trigeminal ganglion, (*3*) internal carotid artery, (*4*) trochlear nerve, (*5*) oculomotor nerve, (*6*) supraorbital nerve, (*7*) superior rectus muscle, (*8*) ciliary ganglion, (*9*) inferior rectus muscle, (*10*) ophthalmic nerve (With permission from D. Jankovic)

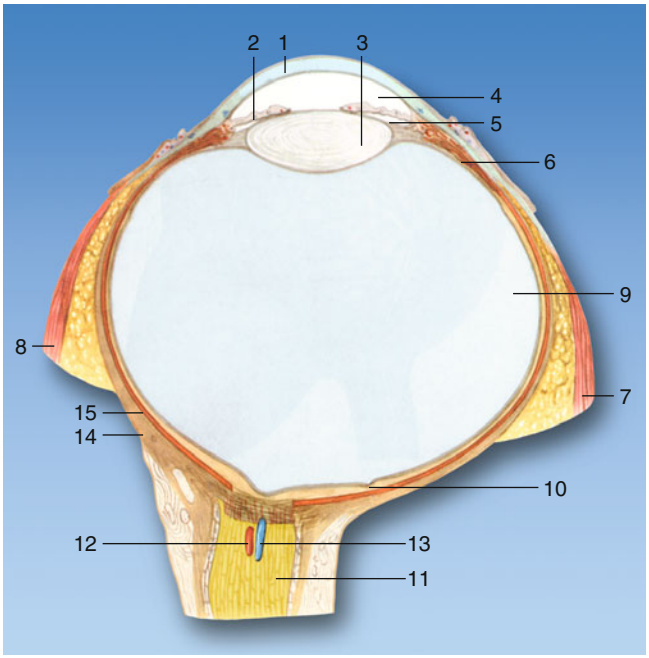


Fig. 6.4 Horizontal section through the eyeball. (*1*) Cornea, (*2*) iris, (*3*) lens, (*4*), anterior chamber of the eyeball, (*5*) posterior chamber of the eyeball, (*6*) ciliary body, (*7*) lateral rectus muscle, (*8*) medial rectus muscle, (*9*) vitreous body, (*10*) central retinal fovea, (*11*) optic nerve, (*12*) central retinal artery, (*13*) central retinal vein, (*14*) sclera, (*15*) choroid (With permission from D. Jankovic)

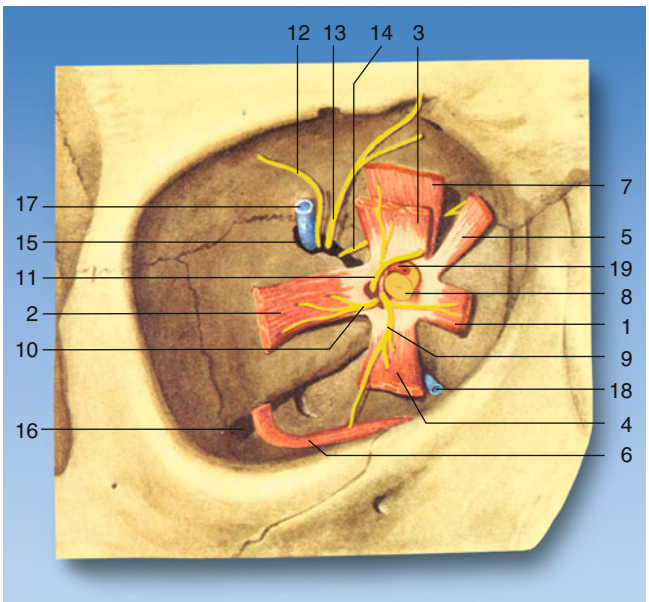


Fig. 6.5 Eye: muscles, nerves, and vessels. (*1*) Medial rectus muscle, (*2*) lateral rectus muscle, (3) superior rectus muscle, (*4*) inferior rectus muscle, (*5*) superior oblique muscle, (*6*) inferior oblique muscle, (*7*) levator palpebrae superioris muscle, (*8*) optic nerve, (*9*) oculomotor nerve, (*10*) abducent nerve, (*11*) nasociliary nerve, (*12*) lacrimal nerve, (*13*) frontal nerve, (*14*) trochlear nerve, (*15*) superior orbital fissure, (*16*) inferior orbital fissure, (*17*) superior ophthalmic vein, (*18*) inferior ophthalmic vein, (*19*) ophthalmic artery (With permission from D. Jankovic)

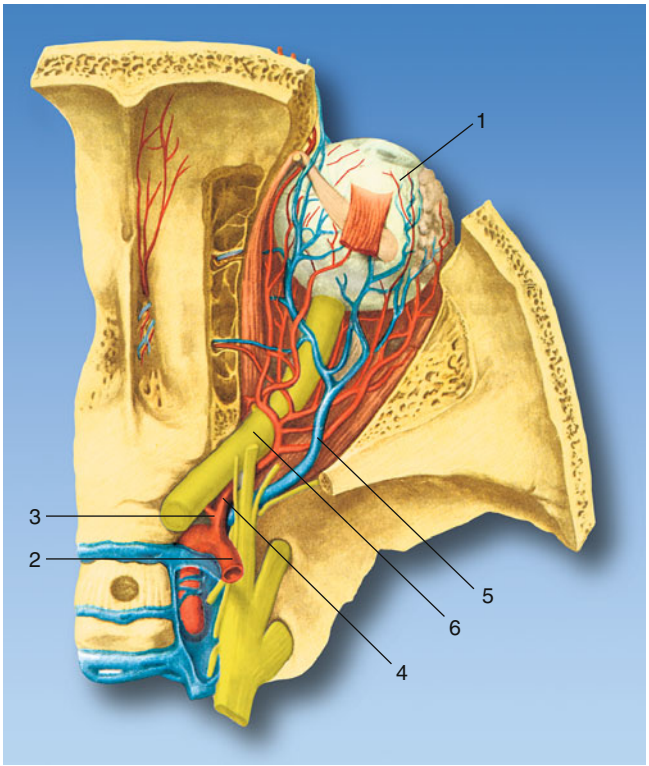


Fig. 6.6 Blood supply in the eye. (*1*) Eyeball, (*2*) internal carotid artery, (*3*) central retinal artery, (*4*) ophthalmic artery, (*5*) superior ophthalmic vein, (*6*) optic nerve (With permission from D. Jankovic)

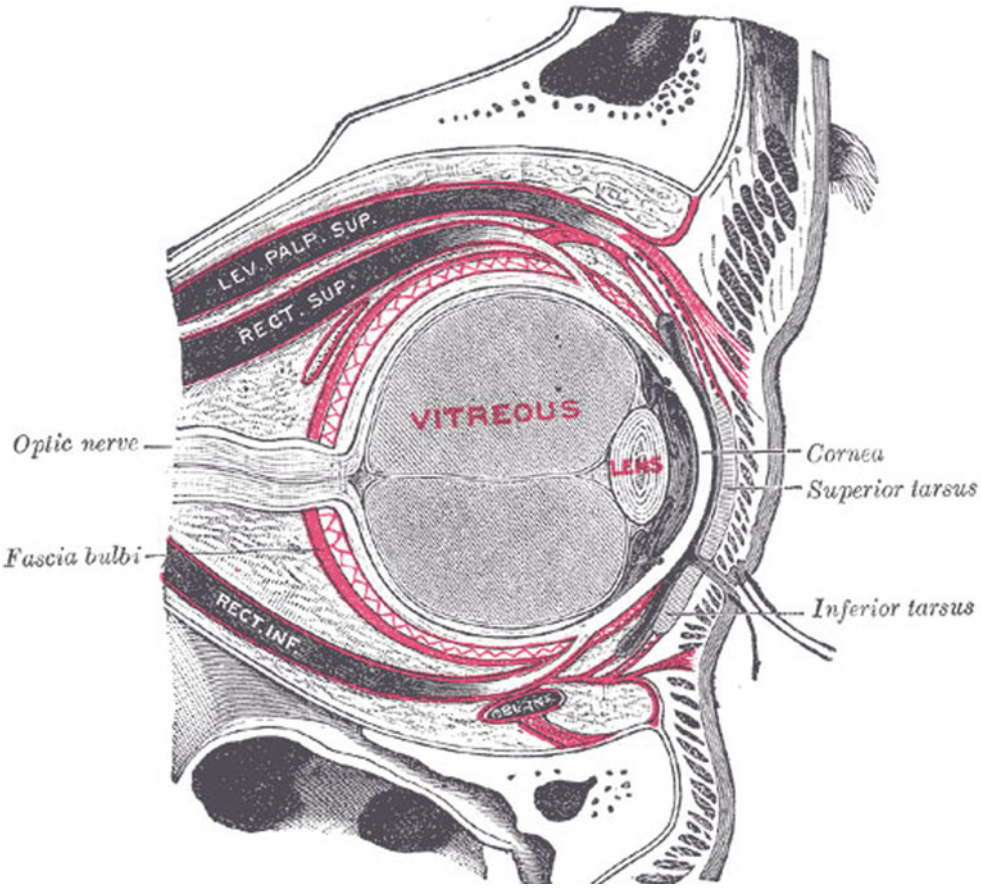


Fig. 6.7 Sub-Tenon space showing multiple connective tissue bands (With kind permission from www.bartleby.com)

Anatomy Relevant to Blocks

The terminology used for regional orbital blocks is controversial. An intraconal (retrobulbar) block involves the injection of a local anesthetic agent into the muscle cone behind the globe formed by four recti muscles and the superior and inferior oblique muscles. An extraconal (peribulbar) block involves the injection of local anesthetic outside the muscle cone. Many studies confirm that there are multiple communications between two compartments, and it is sometimes difficult to differentiate if the injection is intraconal or extraconal. A combination of intraconal and extraconal blocks is described as combined retroperibulbar block.

The average distance between the orbital margin and the ciliary ganglion is approximately 38 mm (range 32–44 mm). If a 38-mm needle is used, it may reach the optic nerve in a large number of patients leading to serious catastrophic events. A needle *<31 mm* must be used to protect the globe, optic nerve, and other major structures behind the globe during both extraconal and intraconal blocks. In cadaver studies, Karampatiakis et al. found that when a retrobulbar block was carried out with a needle 40 mm long, the needle tip would reach the posterior optic nerve in 100 % of cases; even with needles 35 mm long, the covering of the optic nerve would be touched [2].

Average axial length (anteroposterior distance of the globe) of the eye varies from 22 to 24 mm. Eye with axial lengths >26 mm are more prone to globe damage. The axial length measurement is available during cataract surgery. If a patient is scheduled for non-cataract surgery, spherical power of the spectacles may offer some clue. If a patient has very thick prescription glasses, it indicates high myopia. The risk of damage to the globe and optic nerve is also greater when the globe is rotated during injection. Therefore, it is recommended that the globe should be in the neutral gaze during the injection. It is safer to introduce a needle as far lateral as possible because if it is introduced at the junction of medial 2/3rd and lateral 1/3rd of the inferior orbital rim, the inferior rectus and inferior oblique muscles and their nerves may be damaged.

In sub-Tenon block, local anesthetic agent is injected under the Tenon capsule. This block is also known as parabulbar block, pinpoint anesthesia, and medial episcleral block.

Physiology

The physiological pressure (intraocular pressure) interior of the eye is between 10 and 20 mmHg. It is higher in patients with a large-diameter eyeball and in the recumbent position. It is higher in the morning than in the evening. It increases during coughing, physical exertions, and vomiting. An increase in the plasma concentration of carbon dioxide and decrease in oxygen concentration increase the intraocular pressure. Intraocular pressure increases after the needle technique, and this is due to increase in the volume behind the globe. There is little or no increase in intraocular pressure after sub-Tenon block. The anterior and posterior chambers of the eye contain 250 μl of an aqueous liquid (rate of synthesis ca. 2.5 μl/min).

Indications and Contraindications

Both intraocular procedures (cataract extraction, vitrectomy) and extraocular procedures (strabismus surgery, retinal detachment) can be carried out under local anesthesia in suitable patients. There are only a few absolute contraindications such as patient refusal to accept local anesthesia, allergy to local anesthetic agent, local infection, excessive abnormal body movements, severe breathlessness, psychiatric conditions, and in situations where it is difficult to establish a good communication between patients and health-care workers, but the list is shrinking and local anesthesia is increasingly used even in the above group.

Patient's Assessment and Preparation

Most ophthalmic surgical procedures are carried out as outpatient basis. There is debate as to the degree of preoperative assessment and investigation required and they vary worldwide. In the UK, the Joint Colleges Working Party Report recommended that routine investigations are unnecessary and the patients are not fasted [3]. Routine investigation of patients undergoing cataract surgery under regional anesthesia is not essential because it neither improves the

health nor improves the outcome of surgery, but tests can be done to improve the general health of the patient if required [4].

The preoperative assessment should always include a specific enquiry about bleeding disorders and related drugs. There is an increased risk of hemorrhage, and this requires that a clotting profile is available (and recorded) prior to injection (see later) [5, 6]. Patients receiving anticoagulants are advised to continue their medications. Clotting results should be within the recommended therapeutic range. Currently, there is no recommendation for patients receiving antiplatelet agents. Blood pressure should be controlled in hypertensive patients. Diabetic patients are allowed to take their usual medications, and active control of blood sugar is not necessary. There is no need for antibiotic prophylaxis in patients with valvular heart disease, and surgery is not performed if patient has suffered myocardial infarction during the last 3 months [3]. The presence of a long eye, staphyloma, or enophthalmos, the use of faulty technique, a lack of appreciation of risk factors, an uncooperative patient, and the use of unnecessarily long needles are some of the contributing causes. Knowledge of axial length measurement is essential. A precise axial length measurement is usually available for intraocular lens diopter power calculation before cataract surgery [7]. If the block is performed for other surgery and the axial length measurement is not known, close attention to the diopter power of patients' spectacles or contact lenses may provide valuable clues to globe dimension (patients usually provide this history).

General Considerations and Preparation Before Block

The patient and the person performing the block must be involved in full discussion of the proposed technique. The anesthetic and surgical procedures are explained to the patients. Patient is placed in a comfortable position, and oxygen is administered via a suitable device. All monitoring and anesthetic equipments in the operating environments should be fully functional. Blood pressure, oxygen saturation, and ECG leads are connected, and baseline recordings are obtained. Insertion of an intravenous line is a prerequisite for needle block, but its use has been questioned during sub-Tenon injections [2]. It is not unusual to observe adverse medical events in elderly patient, and a working intravenous line could only be a good clinical practice.

Sedation and Analgesia During Ophthalmic Blocks

The use of sedation varies in different parts of the world and may or may not be used. Sedation is used during block procedure, surgical procedure, or during block and surgery. However, sedation is common during topical anesthesia. Selected patients, in whom explanation and reassurance have proved inadequate, may benefit from sedation. Short-acting benzodiazepines, opioids, and small doses of intravenous anesthetic induction agents are favored, but the dosage must be minimal. An increased incidence of adverse intraoperative events is anticipated with sedation in elderly [6].

It is recommended that one of the following drugs is given immediately before carrying out regional anesthesia:

- Midazolam 1 mg (+ remifentanil 0.33 μg/kg)
- Propofol 0.5 mg/kg

Deep sedation should not accompany regional anesthesia; the patient should be capable of cooperating.

Conduction and Infiltration Block

Needle Techniques

Atkinson's classical retrobulbar block involves insertion of a 38-mm-long needle through the skin at the junction of the medial 2/3rd and lateral 1/3rd of the inferior orbital margin in a rotated eye toward the orbital apex. A facial nerve block (7th nerve block) is essential (see Chap. 7).

The classical retrobulbar block has now been superseded by a higher-volume modern retrobulbar and peribulbar blocks.

Modern Retrobulbar Block

The needle is deliberately directed toward the apex and within the muscle cone (see above) while the globe is in neutral gaze.

Indications

Both intraocular and extraocular surgery may include cataract surgery, vitreoretinal surgery, panretinal photocoagulation, trabeculectomy, optic nerve sheath fenestration, delivery of drugs (steroids, neurolytic agents), and sometimes strabismus surgery in willing patients. Retrobulbar block is also performed for the delivery of neurolytic agents for the treatment of chronic orbital pain.

Preparation

See above.

Materials

Equipments such as local anesthetic eyedrop, local anesthetic agent, needle, and syringe (Fig. 6.8) are essential for needle block. Other materials such as syringe, small needle, oculocompression device, gauge swab, hyaluronidase, epinephrine, and balanced salt solution (BSS) may be required.

The short and sharp needles are favored because they reduce the discomfort on insertion but at the expense of a reduced tactile feedback, hence a higher risk of failing to recognize a globe perforation. The blunt or dull needles are favored because it is believed that blood vessels are pushed rather traumatized and tissue planes could be more accurately defined, but these are more likely to cause greater damage when misplaced [8].

Patient Positioning

The best position for the patient is a semi-recumbent position (45°).

Injection Technique

Topical local anesthetic drops and antiseptic drops are instilled to obtain surface anesthesia (Fig. 6.9). A dilute local solution (add 2 ml of concentrated local anesthetic agent to 13 ml of BSS) is helpful before the injection of concentrated local anesthetic agent. Of this dilute solution, 1.5–2 ml is injected through the conjunctiva under the inferior tarsal plate in the inferotemporal quadrant.

A 27-gauge, 1-cm-long needle (Fig. 6.10) is inserted under the inferior tarsal plate through the conjunctiva to deliver dilute local anesthetic. The needle can be introduced through the skin or conjunctiva. A 25-G, 31-mm-long needle is inserted through the skin in the inferotemporal quadrant as far lateral as possible (7 o'clock position on the right eye or the 5 o'clock position on the left eye) below the lateral rectus muscle (Fig. 6.11) while the patient's eye is in the neutral gaze position. It is important that the needle hub is visible and the skin is not indented. The initial direction of the needle is tangential to the globe, then passed below the globe, and, once passed the equator as gauged by axial length of the globe, is allowed to go upward and inward along the floor of the orbit to enter the central space just behind the globe (Figs. 6.12 and 6.13). The globe is continuously observed during the needle placement.

Motility testing of the eye must be carried out before the procedure.

Dosage

- Oxybuprocaine HCl 0.4 % for surface anesthesia if the transconjunctival access route is chosen.
- 3–5 ml local anesthetic (e.g., 0.75 % ropivacaine, 0.5 % bupivacaine, 2 % prilocaine, 2 % mepivacaine, 2 % lidocaine) or combinations of these.
- Possible adjuncts to the local anesthetic:

  Epinephrine (5 μg/ml or 1:200,000)
  Hyaluronidase (150 IU) [7]

The addition of hyaluronidase to local anesthetics leads to improved diffusion and thus to a faster onset. This provides very good conditions for surgical procedures in the eye. Akinesia and anesthesia usually follow but they are dose dependent. If the amount injected is small, anesthesia may follow but akinesia may not occur at all. On the other hand, higher volume can generally guarantee anesthesia and akinesia, but intraocular pressure rises and other complications such as chemosis may occur. Sometimes a supplementary injection such as a medial peribulbar block is required (see later).

Advantages

Anesthesia and akinesia are very predictable and quick. It is easy to learn the technique provided knowledge of basic sciences relevant to block are achieved.

Disadvantages

There are reports of serious complications both sight- and life-threatening.

Side Effects and Complications

1. Hemorrhage in the very well-vascularized orbit, with a frequency of 0.7–1.7 % (known as "compartment syndrome"), can lead to blindness.
2. Conjunctival hemorrhage (20–100 %).
3. Retinal detachment and vitreous hemorrhage after perforation of the eyeball can lead to loss of vision.
4. Subperiosteal hemorrhage due to contact between the needle and the orbital floor.
5. Chemosis (25–40 %) due to fast injection of larger volumes of a local anesthetic.
6. Perforation of the eyeball and intraocular injections can occur, particularly in severely myopic patients. To avoid these, patients should gaze straight ahead throughout the block procedure.
7. Injury to the optic nerve or intraneural injection (immediate blindness).
8. Subarachnoid injection is a severe complication.
9. Intravascular injection, with serious CNS intoxication. See Chap. 1.
10. Oculocardiac reflex: bradycardia due to the vasovagal reflex is observed in younger and frail patients and may be seen both during the block procedure and also intraoperatively.
11. Injury to the extraocular muscles (usually the inferior oblique muscle or inferior rectus muscle) can lead to muscle necrosis, contractions, or disturbances of healing.

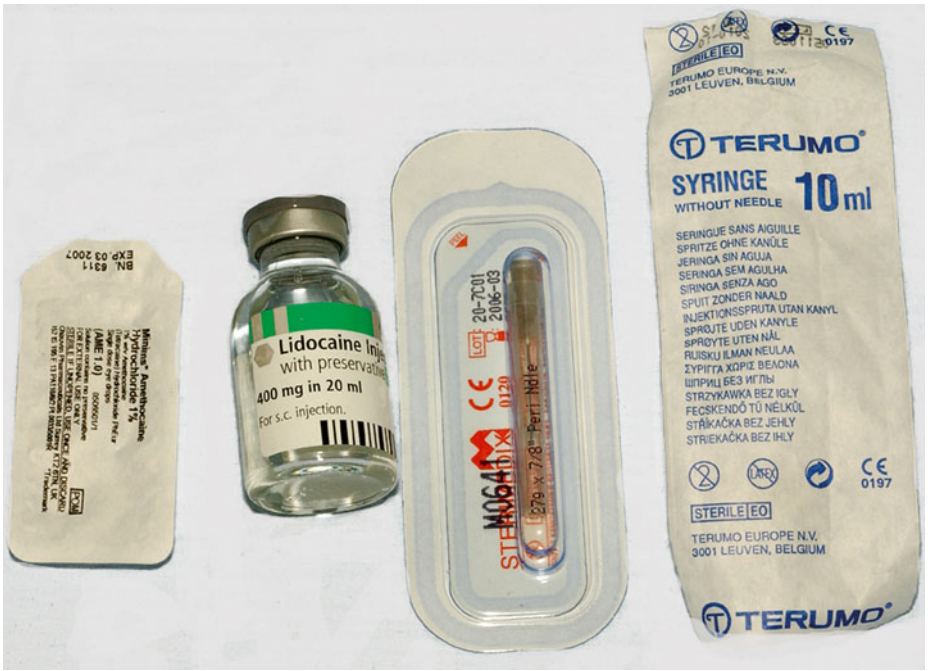


Fig. 6.8 Essential equipments for needle block

Fig. 6.9 Surface anesthesia

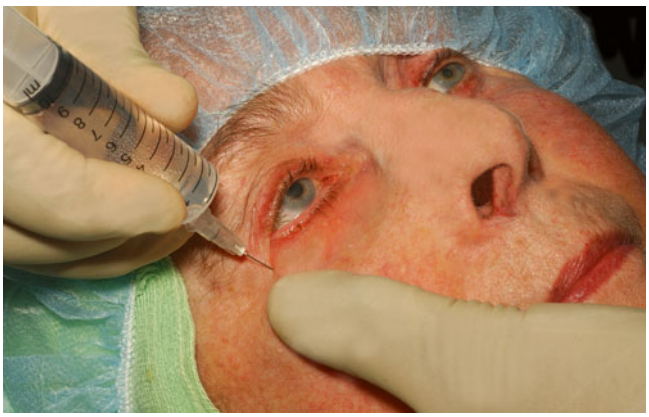


Fig. 6.12 Initial direction of needle (tangential to the globe) during modern retrobulbar block

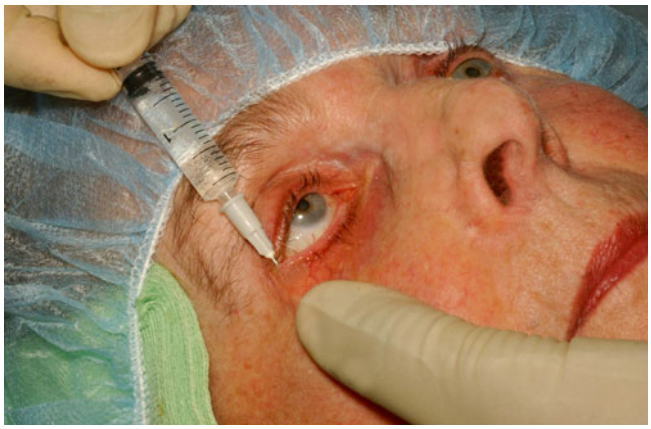


Fig. 6.10 27-G, 1-cm-long needle inserted under the inferior tarsal plate through the conjunctiva to deliver dilute local anesthetic

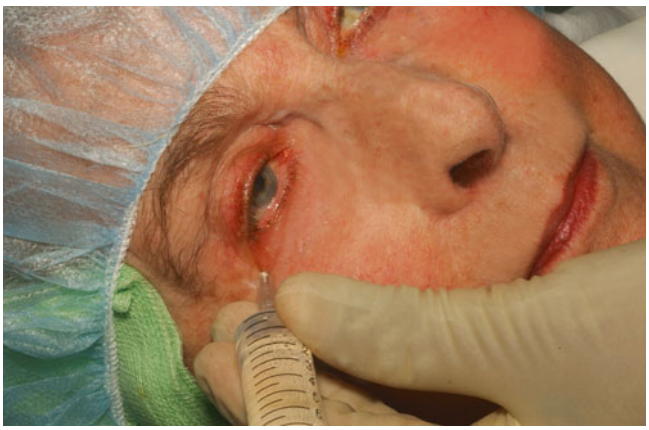


Fig. 6.13 The needle is directed upward and inward during modern retrobulbar block

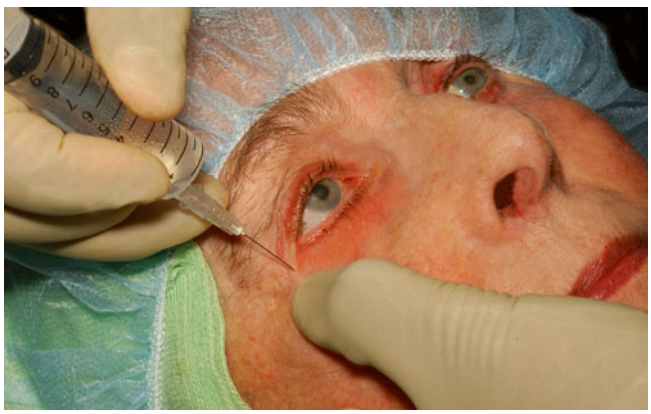


Fig. 6.11 27-G, 2-cm-long needle is inserted at *the extreme* inferolateral quadrant percutaneously during intended modern retrobulbar block

Peribulbar Block

Inferotemporal Peribulbar Block

This block is used as a main injection alternative to modern retrobulbar block.

Indications, Preparation, and Materials
See above

Patient Positioning
See above.

Injection Technique
Possible approaches of peribulbar injection are presented in Fig. 6.14.

A 25-G, 31-mm-long needle is inserted through the conjunctiva as far laterally as possible in the inferotemporal quadrant. Once the needle is under the globe, it is not directed upward and inward, but it is directed along the orbital floor. Five milliliter of local anesthetic agent of choice is injected. Many patients require a supplementary injection [8, 9].

Medial Peribulbar Injection: The Medial Canthus (Caruncular) Single Injection

A medial peribulbar block is usually performed to supplement inferotemporal, retrobulbar, or peribulbar injection, when akinesia is not adequate. A 27-G, 2.5-cm-long needle is inserted in the blind pit between the caruncle and the medial canthus (Figs. 6.14, 6.15, 6.16, and 6.17) to a depth of 15–20 mm. The needle is inserted at the medial side of the caruncle, at the extreme medial side of the palpebral fissure, and directed at a 5° angle away from the sagittal plane toward the medial orbital wall. The needle should never be introduced more than 20 mm for the caruncular puncture. This technique may require supplementary injection [10]. A medial peribulbar with a 25-G, 31-mm-long needle is used as primary technique in patients with long axial length which may be associated with higher incidence of staphyloma [11].

Dosage
Three to five milliliter of local anesthetic agent of choice is injected if this technique is used as a supplementary injection. However, if this technique is used as a primary injection technique, a volume of 8 ml may be required and this may be very painful during injection.

Advantages of the Caruncular Technique
1. The medial canthus single injection of peribulbar anesthesia is less painful as -efficient as the classic double-injection peribulbar technique.
2. Less local anesthetic agent is required.
3. Reduce the number of punctures and, thus, reduce the risk of injuring an important structure of the eye.
4. Short onset of block.
5. Less volume acquired, less ocular pressure.
6. The diffusion space of anesthetic agents around the globe is segmented by a network of numerous tiny aponeuroses, which may be less dense in the medial canthus region, and this might explain better diffusion and low-volume requirement. Further extraconal space in this compartment is relatively large and avascular, and this may reduce the risk of hematoma or intravascular injection.

Disadvantages
Multiple injections are required during peribulbar block. Caution is required during supplementary injection as the first injection is likely to alter the position of the globe in the orbit.

Complications of Needle Blocks
There are many complications of needle blocks, ranging from simple to serious, that have been reported and published [7]. The complications may be limited to the orbit or may be systemic. Orbital complications include failure of the block, corneal abrasion, chemosis, conjunctival hemorrhage, vessel damage leading to retrobulbar hemorrhage, globe perforation, globe penetration, optic nerve damage, and extraocular muscle damage. The systemic complications, such as local anesthetic agent toxicity, brain stem anesthesia, and cardiorespiratory arrest, may be due to intravenous or intrathecal injections or the spread or misplacement of drug in the orbit during or immediately after injection.

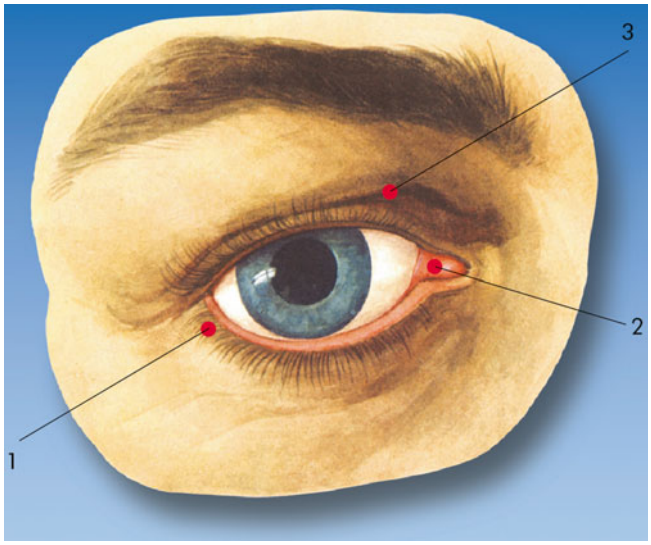


Fig. 6.14 Peribulbar block. Possible approaches: (*1*) inferotemporal, (*2*) medial caruncular, (*3*) superonasal (With permission from D. Jankovic)

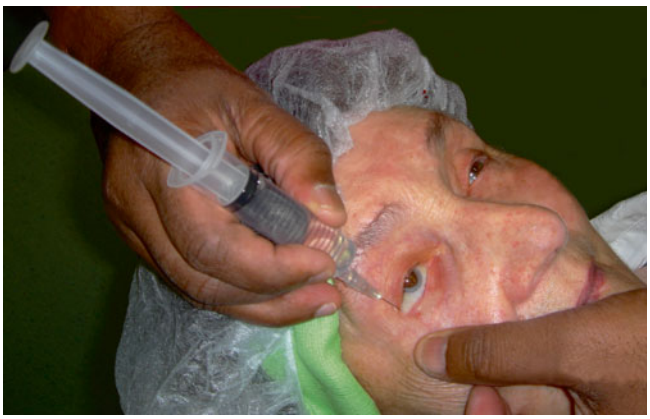


Fig. 6.15 Inferotemporal peribulbar block—a needle inserted through the conjunctiva in the extreme inferotemporal quadrant below the lateral rectus muscle

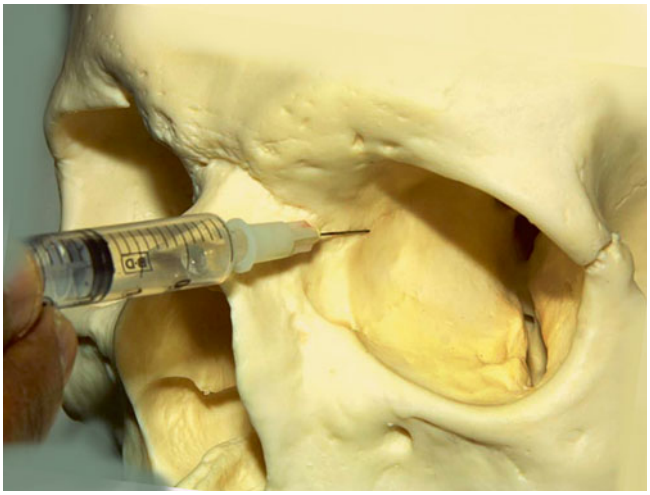


Fig. 6.16 A photography of the skull showing direction of needle during medial (caruncular) peribulbar block

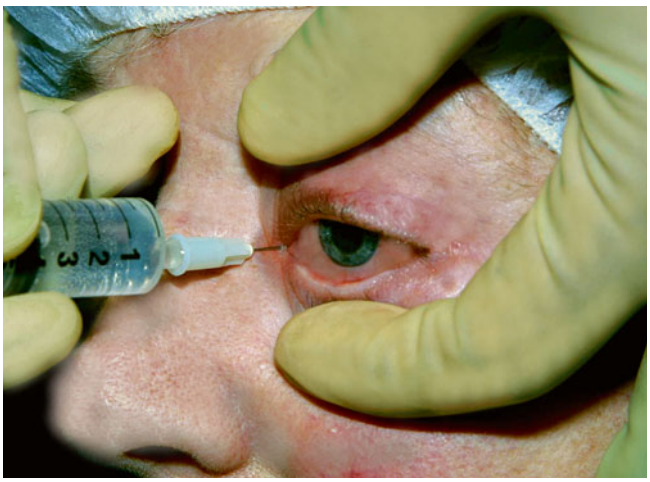


Fig. 6.17 Medial (caruncular) block: 27-G, 2-cm-long needle is inserted between the caruncle and medial canthus during medial peribulbar block

Sub-tenon Block

This block was introduced as a simple, safe, and effective alternative to needle blocks. The technique involves gaining access to the sub-Tenon space, the insertion of a blunt cannula, and the administration of local anesthetic agent into the sub-Tenon space [12].

Anatomy Relevant to Block

Orbital blocks are performed to achieve anesthesia and akinesia during orbital regional blocks. Injection of local anesthetic agent under the Tenon capsule diffuses into the intraconal space. Anesthesia is obtained when the branches of short ciliary nerve are blocked as they pass through the Tenon capsule to the globe. Akinesia occurs when branches of the motor nerves are blocked in the intraconal compartment.

The sub-Tenon space can be accessed from all four quadrants, but the inferonasal quadrant is the most commonly reported site of access in the published studies as the placement of cannula in this quadrant allows good fluid distribution superiorly while avoiding the area of access for surgery and damage to the vortex veins.

Indications

Sub-Tenon block is a versatile and effective technique. Its use has been advocated primarily for cataract surgery but is also effective for vitreoretinal surgery, panretinal photocoagulation, trabeculectomy, strabismus surgery, optic nerve sheath fenestration, and the delivery of drugs. This technique is also increasingly favored in patients who are on anticoagulants, aspirin, and nonsteroidal anti-inflammatory drugs (NSAIDs) [13, 14]. Complications are less severe and relatively fewer.

Materials

Topical local anesthetic agent, povidone iodine (5 %), scissors, forceps, local anesthetic agent, blunt sub-Tenon cannula, and syringe are required (Fig. 6.18). Additional materials such as speculum (Clarke or Screw speculum), gloves, cotton buds, hyaluronidase, and epinephrine may be helpful.

Preparation

Surface Anesthesia and Speculum Placement

All topical local anesthetic drops have been used, but preservative-free topical preparations in single-dose containers (0.4 % oxybuprocaine or tetracaine eyedrop) are usually preferred but they all produce stinging on initial application. Surface anesthesia can also be achieved by the application of a cotton bud soaked with topical agent in the area of dissection. The procedure is carried out under sterile conditions, and no-touch technique is advocated as infections are known to occur. Conjunctiva is cleaned with aqueous 5 % povidone iodine. Some prefer to use a speculum, but if not available, the lower lid is retracted by an assistant.

Position of the Patient and Technique

Anesthetist can approach from the head end or from the side [13]. The patient is positioned supine with comfortable pads and sponges. The patient is asked to look upward and outward (Fig. 6.19). If sedation is used, dissection can be performed in a neutral gaze position. Under sterile conditions, the conjunctiva and Tenon capsule are gripped with non-toothed forceps 5–10 mm away from the limbus (Fig. 6.20a). A small incision is made through these layers with scissors to expose the white area, and a sub-Tenon cannula (2.54 cm curved and blunt) is inserted along the curvature of the globe (Fig. 6.20a, b). It is not unusual to meet a resistance during this procedure especially when the Tenon capsule is not dissected properly or the cannula does not advance because resistance is met by tissues (connective tissue bands, muscles etc.); the cannula should be repositioned or inserted but force is never applied.

Dosage

Three to five milliliter of local anesthetic (2 % lidocaine, 0.5 % bupivacaine, 0.5 % levobupivacaine, 0.75 % ropivacaine) can be used. Addition of hyaluronidase is a subject of debate (see later). The volume of local anesthetic agent for sub-Tenon block varies from 1.5 to 11 ml but 3–5 ml is common.

Disadvantages

It is an invasive and surgical technique. Asepsis is of paramount importance. The technique could be difficult to learn initially but gets easier with experience. Muscle and eyelid akinesia are variable and are volume dependent. Pain during injection and conjunctival hemorrhage are frequently seen. Loss of local anesthetic during this procedure may occur. A syringe should contain at least 5 ml of local anesthetic agent of choice with or without adjuvant.

- About 10 % of patients require supplementation with an additional block of the facial nerve (4 %), surface anesthesia (2.6 %), or retrobulbar block (0.8 %).
- Pain during injection.

Complications

Although sub-Tenon block was introduced as a very safe technique over the years, a number of complications both minor (see above) and major have been reported [2, 13, 14]. Major complications include orbital and retrobulbar hemorrhage, rectus muscle paresis and trauma, globe perforation, the central spread of local anesthetic, orbital cellulitis, and others. Most of these complications have occurred following the use of a 2.54-cm metal cannula, but the exact mechanism and their incidence are not known. Smaller or flexible cannulae appear to be safer, but the incidence of minor complications increases.

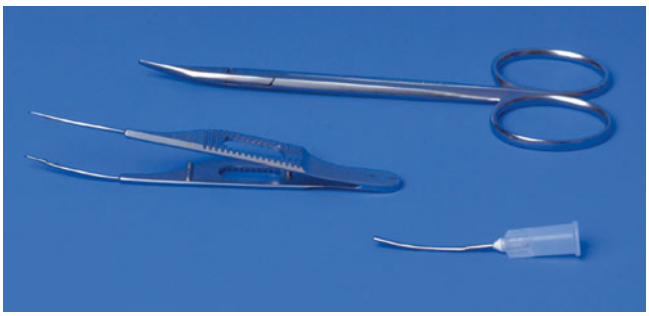


Fig. 6.18 Materials for a sub-Tenon block (With permission from D. Jankovic)

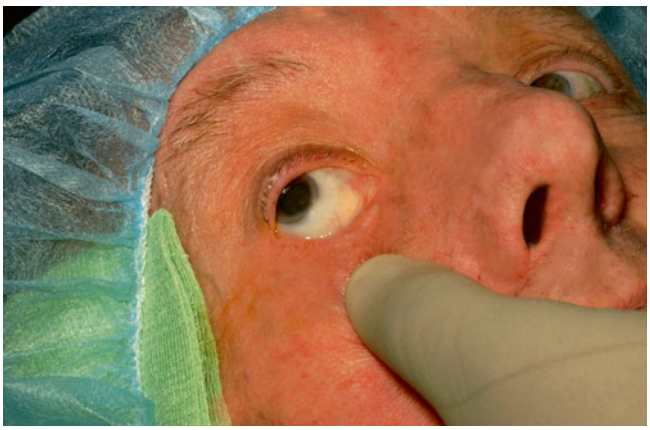


Fig. 6.19 Gaze of the globe (upward and outward) during dissection for inferonasal access for sub-Tenon block. Upward and outward rotation helps to expose the area of dissection

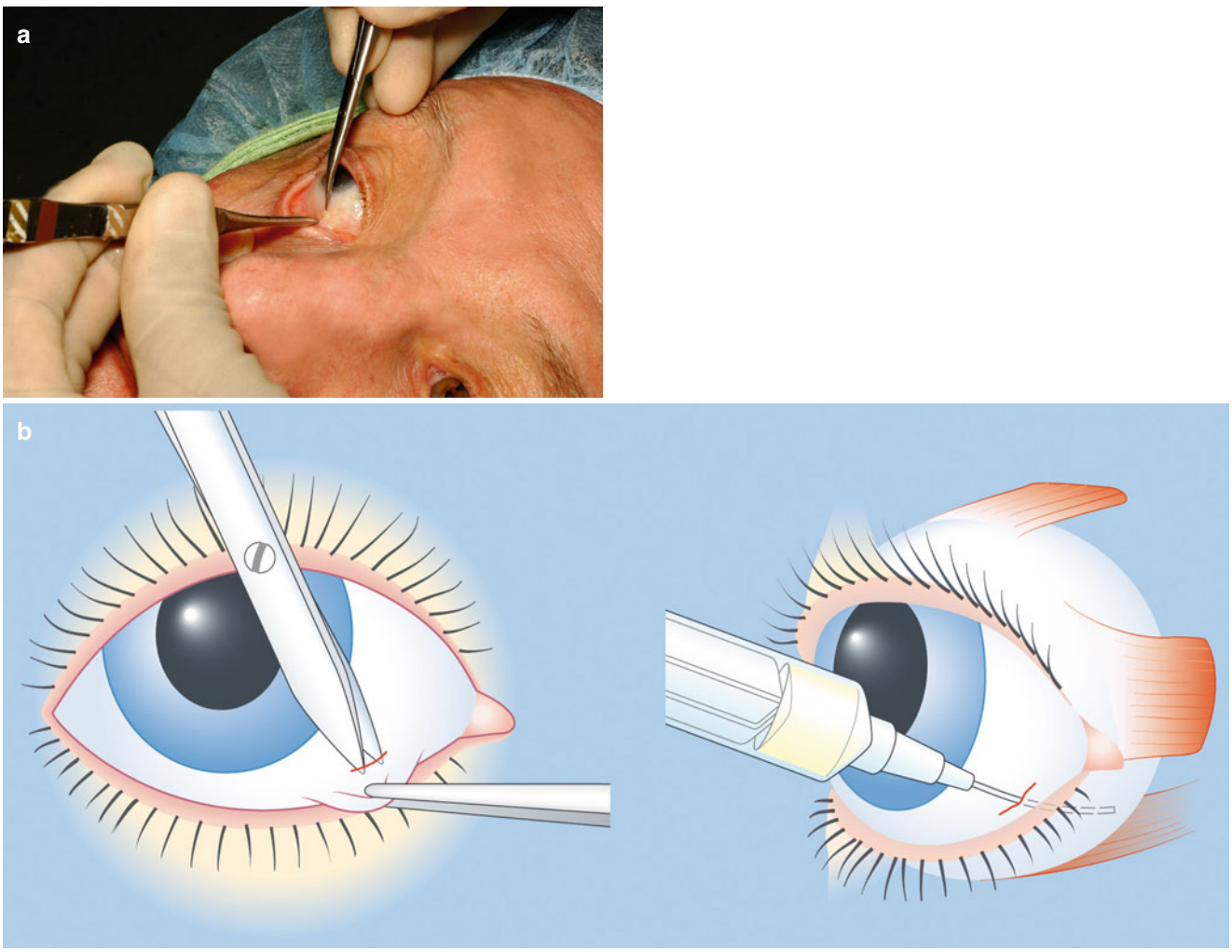


Fig. 6.20 (**a**) Inferonasal dissection for sub-Tenon block with Westcott scissors and Moorfields forceps. (**b**) Carrying out a sub-Tenon block. (**a**) The conjunctiva and Tenon capsule are opened with Westcott scissors. (**b**) The sub-Tenon needle is introduced into the posterior sub-Tenon space (With permission from D. Jankovic)

Topical Anesthesia

Topical anesthesia is increasingly used and preferred by ophthalmic surgeons or non-medical health-care professionals. This is not an invasive technique and different considerations apply. Readers are advised to consult relevant texts.

Checking Adequacy of Anesthesia

There is no objective method of checking anesthesia. It is anticipated that if akinesia is complete or near complete, anesthesia would accompany. If akinesia is not adequate or surgeon prefers complete akinesia, a repeat injection is considered.

Anticoagulation and Eye Block

There is an increased incidence of hemorrhage in patients who receive aspirin, nonsteroidal anti-inflammatory drugs, oral anticoagulants, and newer antiplatelet agents. Clotting profile should be checked and values must be in the therapeutic range. If patient is receiving warfarin, it should not be stopped. INR must be checked. If INR is higher than 3.5, the injection technique is better avoided. Long needles with sharp tips increase the risk of hemorrhage. Smaller needles and single-shot injections appear to be safer, and the sub-Tenon technique is apparently safer still [3].

Local Anesthetic Agents for Ophthalmic Blocks

The ideal agent for ophthalmic block should be safe, painless to inject, and produce a rapid onset of dense motor and sensory block, the duration of which must be sufficient for surgery yet not excessively prolonged. A technique based on higher volume can lead to an increased incidence of chemosis. The speed of onset is partially determined by the properties of the anesthetic, but more directly by the proximity to the nerves. All modern, high-potency local anesthetic agents are suitable for ophthalmic blocks and numerous studies have shown little difference in the quality of anesthesia, analgesia, and akinesia [15]. A complete blockade of nerves may not occur, and this is manifested by various visual sensations and experiences reported during ophthalmic regional anesthesia. Muscles regain their full activities after a few hours, and double vision may be experienced. It is important to inform the patient that while local anesthetic is present, it is important to avoid rubbing the eye.

Role of Vasoconstrictor

Vasoconstrictor (epinephrine) is commonly mixed with local anesthetic solution to increase the intensity and duration of block and minimize bleeding from small vessels. A concentration of 1:200,000 has no systemic effect. However, epinephrine may cause vasoconstriction of the ophthalmic artery, compromising the retinal circulation [15]. The use of epinephrine-containing solutions is avoided in elderly patients suffering from cerebrovascular and cardiovascular diseases. If anesthesia for longer duration is required, an agent which lasts longer such as bupivacaine, ropivacaine, or levobupivacaine should be used.

Role of Hyaluronidase

Hyaluronidase is an enzyme, which reversibly liquefies the interstitial barrier between cells by depolymerization of hyaluronic acid to a tetrasaccharide, thereby enhancing the diffusion of molecules through tissue planes. It is available as a powder readily soluble in local anesthetic solution. Hyaluronidase has been shown to improve the effectiveness and the quality of needle as well as sub-Tenon block, but its use remains controversial. The amount of hyaluronidase used varies from 5 to 150 IU/ml. The UK data sheet limits the concentration to 15 IU/ml [16]. Orbital swelling and allergic reactions can occur. Excellent blocks can be achieved without hyaluronidase, but there are reports of muscle dysfunction when it is not used during needle block. It is generally believed that local anesthetic agents stay in contact with thin muscles for a longer period leading to myotoxicity [7, 16].

Intraocular Pressure and Ophthalmic Blocks

Rise in intraocular pressure is observed immediately after retrobulbar and peribulbar injections, but no such rise is reported after sub-Tenon block [8]. Oculocompression devices such as Honan's balloon and McIntyre mercury bag may be necessary to reduce the intraocular pressure. However, when an oculocompression device is used, the pressure should not exceed 25 mmHg and it should be removed every 5 min.

Retained Visual Sensations During Ophthalmic Blocks

Many patients experience intraoperative visual sensations that include light, colors, movements, and instruments during surgery under all forms of local ophthalmic anesthesia

[17]. Although the majority of patients feel comfortable with the visual sensations they experience, a small proportion found the experience to be unpleasant or frightening. Therefore, patients receiving orbital blocks should receive preoperative advice as this may alleviate an unpleasant experience and the sedative may stop recall.

Intraoperative Care

The patient should be comfortable and soft pads are placed under the pressure areas. All patients undergoing major eye surgery under local anesthesia are monitored with pulse oximetry, ECG, noninvasive blood pressure measurement, and the maintenance of verbal contact. Patients should receive an oxygen-enriched breathing atmosphere to prevent hypoxia at a flow rate enough to prevent rebreathing and the ensuing hypercarbia once draped. ECG and pulse oximetry should be continued. Once the patient is under the drapes, verbal and tactile contacts are maintained [3].

Advantages and Disadvantages of Different Techniques

There are conflicting reports on the relative effectiveness of akinetic blocks [4]. The evidence indicates that peribulbar and retrobulbar anesthesia produce equally good akinesia and equivalent pain control during cataract surgery. There is insufficient evidence in the literature to make a definite statement concerning the relative effectiveness of sub-Tenon block in producing akinesia when compared with peribulbar or retrobulbar block. However, there was moderate evidence that sub-Tenon block produced better pain control than retrobulbar and peribulbar block. Finally, there was weak evidence that sub-Tenon block produces better pain control than topical anesthesia.

Choosing a Technique

There are numerous studies illustrating the diversity of preference for anesthetic technique by surgeons. The choice of the technique will always depend on a balance between the patient's wishes, the operative needs of the surgeon, the skills of the anesthetist, and the place where such surgery is being performed [8]. The practice of local anesthesia varies around the world. Although akinesia is not desirable during modern phacoemulsification surgery, other ophthalmic surgeries will require a still complete and anesthetized eye (viscocanalostomy). Needle blocks are single short techniques, and anesthesia and akinesia will depend on the choice of local anesthetic agent used. However, a sub-Tenon block can be repeated through the initial dissection should the need arise.

Conclusion

Eye blocks provide excellent anesthesia for ophthalmic surgery and success rates are high. Satisfactory anesthesia and akinesia can be obtained with both needle and cannula. At present, there is no perfect technique. Although rare, orbital injections may cause severe local and systemic complications. Knowledge of orbital anatomy and training are essential for the practice of safe orbital regional anesthesia.

Conduction anesthesia for intraocular procedures

Block ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgery*

Needle: ☐ _____ *G* ☐ *Sharp* ☐ *Blunt*

☐ *Other* ☐ _____ *G* _____ *mm long*

i. v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Sitting* ☐ *Semi-Sitting*

Approach: ☐ *Retrobulbar* ☐ *Peribulbar* ☐ *Sub-Tenon*

☐ *Surface anesthesia* ☐ *Other (facial nerve, trigeminal nerve)*

Sedoanalgesia before block: ☐ *Midazolam* _____ *mg* ☐ *Propofol* _____ *mg/kg*

☐ *Remifentanil* _____ *µg/kg* ☐ *Other* _____

Local anesthetic: _____ *mL* _____ *%* _____

Patient's remarks during injection:

Addition to injection solution: ☐ *No* ☐ *Yes*

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Objective block effect after 15 min:

☐ *Akinesia* ☐ *Mydriasis* ☐ *Exophthalmos* ☐ *Incomplete block*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

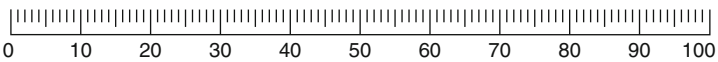
Time of discharge: _____

Complications: ☐ *None*

☐ *Yes (hematoma, intravasculatr injection, other)*

_____

VISUAL ANALOG SCALE



Special notes:

_____

_____

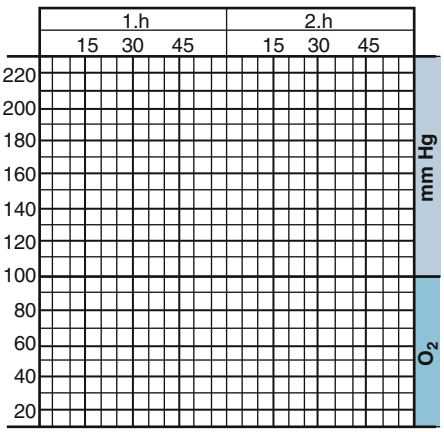
_____

_____

_____

With permission from Danilo Jankovic

Record and checklist



References

1. Jankovic D. Die Geschichte der Regionalanaesthesie. In. Jankovic D, editor. Regional Blockaden & Infiltrationtherapie. 4. Auflage. Berlin: ABW Wissenschaftsverlag; 2008. p.1–14.
2. Karampatiakis V, Natsis K, Gigis P, Stangos NT. Orbital depth measurements of human skulls in relation to retrobulbar anesthesia. Eur J Ophthalmol. 1998;8:118–20.
3. Local anaesthesia for intraocular surgery. The Royal College of Anaesthetists and The Royal College of Ophthalmologists; 2001.
4. Agency for Healthcare Research and Quality. Evidence report/technology assessment: Number 16: Anaesthesia management during cataract surgery. Available at http://www.ahcpr.gov/clinic/epcsums/anestsum.htm. Accessed on 11 July 2006.
5. Konstantatos A. Anticoagulation and cataract surgery: a review of the current literature. Anaesth Intensive Care. 2001;29:11–8.
6. Katz J, Feldman MA, Bass EB, Lubomski LH, Tielsch JM, Petty BG, et al. Study of Medical Testing for Cataract Surgery Team. Risks and benefits of anticoagulant and antiplatelet medication use before cataract surgery. Ophthalmology. 2003;110:1784–8.
7. Kumar CM, Dowd TC. Complications of ophthalmic regional blocks: their treatment and prevention. Ophthalmologica. 2006;220:73–82.
8. Kumar CM, Dodds C, Faanning GL, editors. Ophthalmic anaesthesia. Lisse: Swets and Zeitlinger; 2002.
9. Kumar CM, Dodds C. Ophthalmic regional block. Ann Acad Med Singapore. 2006;35:158–67.
10. Deruddre S, Benhamou D. Medial canthus single-injection peribulbar anaesthesia: a prospective randomized comparison with classic double-injection peribulbar anaesthesia. Reg Anesth Pain Med. 2005;30:255–9.
11. Vohra SB, Good PA. Altered globe dimensions of axial myopia as risk factors for penetrating ocular injury during peribulbar anaesthesia. Br J Anaesth. 2000;85:242–5.
12. Stevens JD. A new local anesthesia technique for cataract extraction by one quadrant sub-Tenon's infiltration. Br J Ophthalmol. 1992;76:670–4.
13. Kumar CM, Williamson S, Manickam B. A review of sub-Tenon's block: current practice and recent development. Eur J Anaesthesiol. 2005;22:567–77.
14. Kumar CM, Dodds C. Sub-Tenon's anesthesia. Ophthalmol Clin North Am. 2006;19:209–19.
15. McLure HA, Rubin AP. Review of local anaesthetic agents. Minerva Anestesiol. 2005;71:59–74.
16. British National Formulary. A joint publication of the British Medical Association and the Royal Pharmaceutical Society of Great Britain, London, 2002.
17. Tan CS, Eong KG, Kumar CM. Visual experiences during cataract surgery: what anaesthesia providers should know. Eur J Anaesthesiol. 2005;22:413–9.

Chapter 7

Facial Nerve Block

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_7,

In the majority of intraocular procedures, it is necessary to prevent eyelid movement (orbicularis oculi muscle). This can be achieved by a distal infiltration block of the nerve endings of the facial nerve that provide the motor supply to the orbit.

Anatomy [3, 8] (Figs. 7.1 and 7.2)

The seventh cranial nerve carries motor fibers for the muscles of facial expression and—in the intermediate nerve, a nerve fascicle emerging separately from the brain stem—gustatory fibers and visceral efferent secretory (parasympathetic) fibers. Both sections of the nerve pass through the internal acoustic meatus and emerge as a neural trunk in the facial canal. The geniculate ganglion is located at the bend in the nerves in the petrous bone. The facial canal then courses via the tympanic cavity and turns caudally toward the stylomastoid foramen, through which the nerve exits from the skull. In the parotid gland, it divides into its end branches (parotid plexus). Before entering the parotid gland, the facial nerve gives off the posterior auricular nerve and branches to the posterior belly of the digastric muscle and to the stylohyoid muscle. From the parotid plexus emerge the temporal branches, zygomatic branches, buccal branches, and marginal mandibular branch and the cervical branch to the platysma. These branches supply the muscles of facial expression.

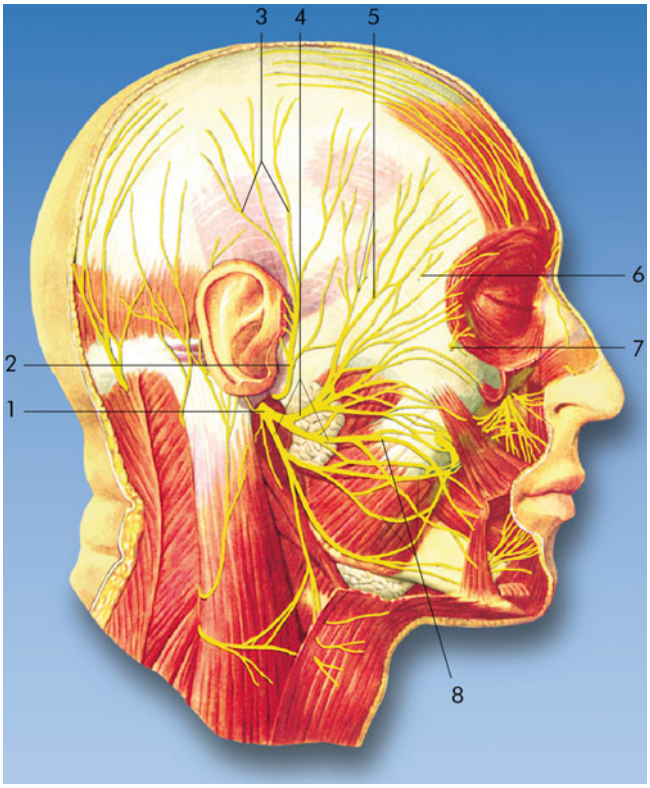


Fig. 7.1 Anatomy of the facial nerve. (*1*) Facial nerve, (*2*) auriculotemporal nerve, (*3*) superficial temporal branches, (*4*) parotid plexus, (*5*) temporal branches, (*6*) zygomaticotemporal branch of the zygomatic nerve, (*7*) zygomaticofacial branch of the zygomatic nerve, (*8*) buccal branches of the facial nerve (Reproduced with permission from Danilo Jankovic)

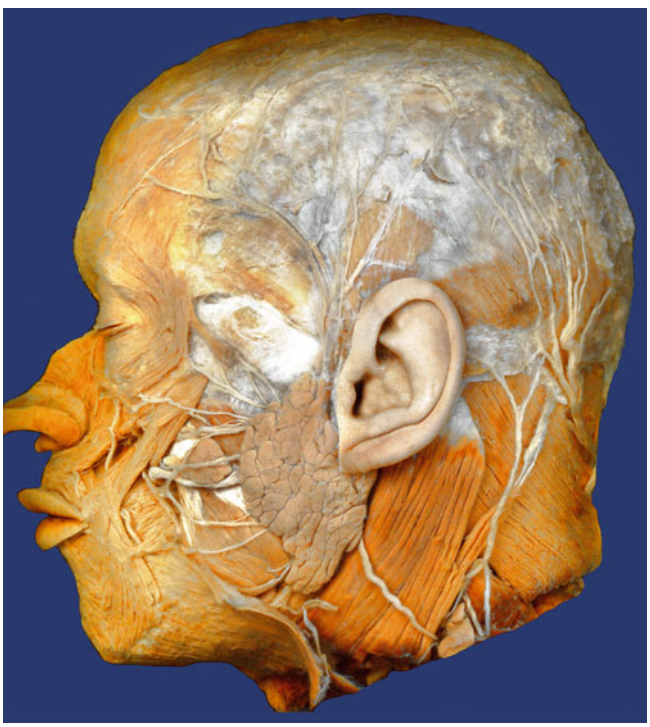


Fig. 7.2 Facial nerve. Anatomic specimen (Reproduced with permission from Danilo Jankovic)

Block Techniques

The facial nerve can be blocked using various techniques along its course to the orbit.

Van Lint Method [5] (Fig. 7.3)

The injection is carried out temporally from the exterior margin of the eyelid or classically just below it temporally. A 23-G needle 30–40 mm long is initially introduced until bone contact is made. After careful aspiration, the injection is carried out medially and downward and then medially and upward. Fan-shaped injection is carried out as the needle is advanced (dosage, 1.5–2 mL local anesthetic).

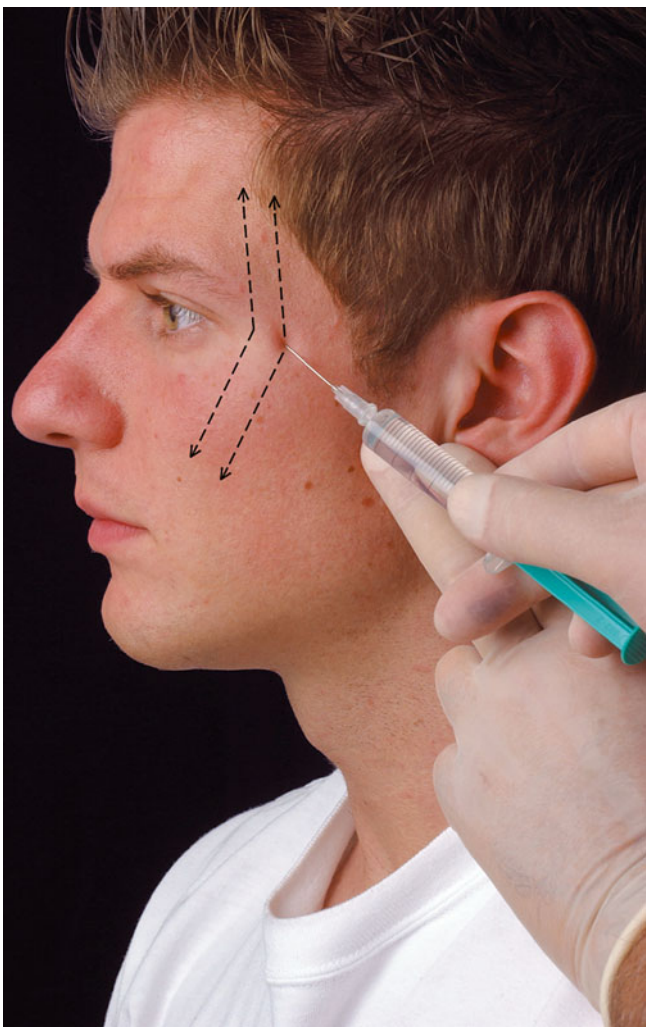


Fig. 7.3 Block for eyelid anesthesia. Van Lint method (Reproduced with permission from Danilo Jankovic)

O'Brien Method [4] (Fig. 7.4)

The facial nerve trunk is blocked just above the condylar process of the mandible. It is helpful for the patient to open and close the mouth. After palpation of the condylar process of the mandible, a 25-G needle 30–40 mm long is introduced until bone contact is made, and 1 mL local anesthetic is injected. The needle is then withdrawn, followed by injections first in the caudal direction and then in the cranial direction (dosage, 2–3 mL local anesthetic).

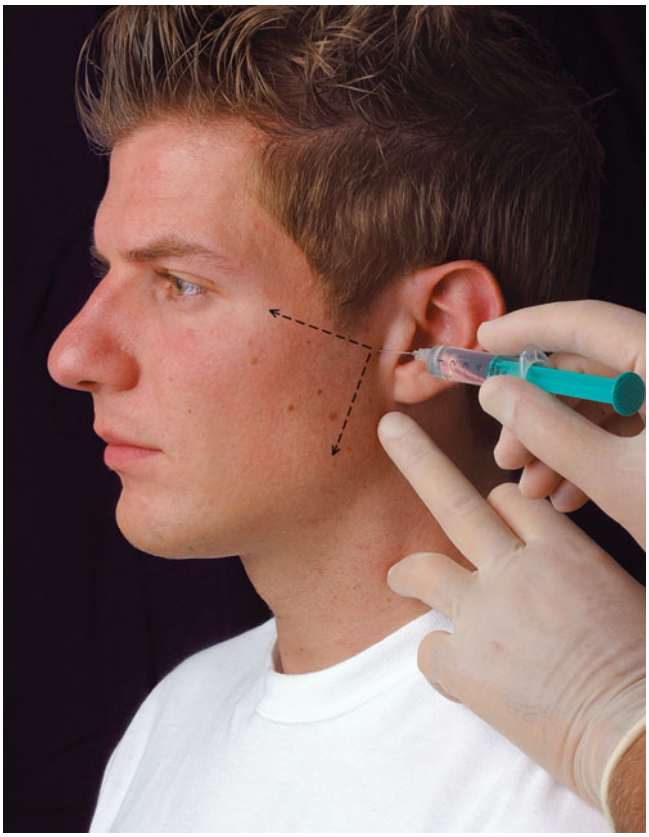


Fig. 7.4 Block for eyelid anesthesia. O'Brien method (Reproduced with permission from Danilo Jankovic)

Atkinson Method [1] (Fig. 7.5)

A 23-G needle 30–40 mm long is introduced below the exterior angle of the eye at the level of the zygomatic arch and is moved upward and outward (dosage, 3 mL local anesthetic).

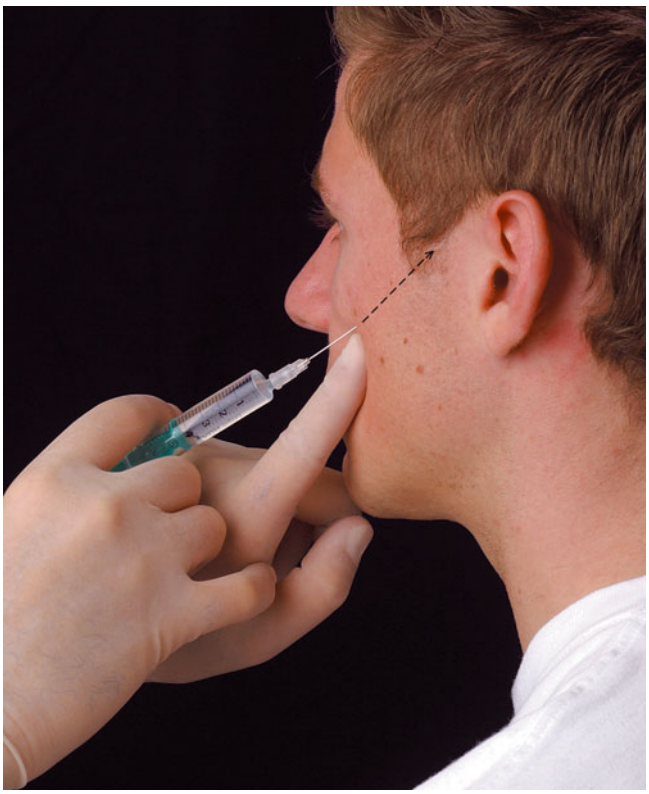


Fig. 7.5 Block for eyelid anesthesia. Atkinson method (Reproduced with permission from Danilo Jankovic)

Nadbath–Rehmann Method [2, 6, 7] (Fig. 7.6)

The blocking of the main trunk of the facial nerve is carried out directly underneath the mastoid process. A 25-G needle 30 mm long is introduced vertically to a depth of 1.5–2 cm (dosage, 2–3 mL local anesthetic).

Fig. 7.6 Block for eyelid anesthesia. Nadbath–Rehmann method (Reproduced with permission from Danilo Jankovic)

References

1. Atkinson WS. Akinesia of the orbicularis. Am J Ophthalmol. 1953;36:1255.
2. Nadbath RP, Rehman I. Facial nerve block. Am J Ophthalmol. 1963;55:143.
3. Netter FH. Autonomes Nervensystem. Autonome Innervation von Kopf un Hals. In: Krämer G, Hrsg: Farbatlanten der Medizin. (Band 5). Nervensystem I: Neuroanatomie und Physiologie. Stuttgart/New York: Georg Thieme Verlag; 1987.
4. O'Brien CS. Akinesia during cataract extraction. Arch Ophthalmol. 1929;1:447.
5. van Lint A. Paralysie palpebrale temporaire provoqueé par I'ope'ration de la cataracte. Ann Ocul (Paris). 1914;151:420.
6. Cofer HF. Cord paralysis after Nadbath facial nerve block. Arch Ophthalmol. 1986;104:337.
7. Wilson CA, Ruiz RS. Respiratory obstruction following the Nadbath facial nerve block. Arch Opthalmol. 1985;103:1454.
8. Jankovic D. Additional blocks for eyelid akinesia. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. 3rd ed. Blackwell, Malden, Massachusetts; Oxford; Carlton, Victoria, Australia; 2004. p. 26–8.

Chapter 8

Airway

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Hürth, Germany
e-mail: danilo@jankovic1.de

Peter Cheng, MD (Ultrasound Section)
Partner Emeritus, Southern California, Permanente Medical Group, 21131 E.Lariat Court, Walnut, CA 91789, USA
e-mail: peterc0426@yahoo.com

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_8, © Springer International Publishing Switzerland 2015

Anesthesia of the Airways

General Considerations

If there is one set of regional block that an anesthesiologist should master, it is airway block. Even anesthesiologists who prefer to use general anesthesia for most of their cases are faced with the need to provide airway blocks before anesthetic induction in patients who may have airway compromise, trauma to the upper airway, or unstable cervical vertebrae.

Anatomy [2, 7]

Air passes to the pulmonary alveoli via the airways in the nose and nasal cavity, pharynx, larynx, trachea, and bronchial tree, which divides at multiple levels. The upper airways are mainly located in the head. The lower airways are located in the neck and chest and include the larynx, trachea, and all the branches of the bronchial tree. The functional anatomy of the airways is illustrated in Figs. 8.1 and 8.2a, b.

The *nasal cavity* is divided by the nasal septum into right and left halves. The sensory innervation of the skin of the external nose is provided by the branches of the *ophthalmic nerve* and *maxillary nerve* (Fig. 8.1; cf. Chaps. 6 and 10), and the motor innervation of the facial muscles around the nose is from the *facial nerve* (cf. Chap. 7).

Each nasal cavity opens via a posterior choana into the upper level of the *pharynx*, the nasopharynx (or epipharynx). The nasopharynx connects with the choanae and is enclosed by the skull base superiorly and by the pharyngeal wall posteriorly. Inferiorly, the soft palate forms the boundary to the middle level of the pharynx (oropharynx). The *larynx* is an air-conducting organ that stretches from the lower pharyngeal space (laryngopharynx) as far as the trachea. In adult males, the larynx is located at the level of the C1–C3 vertebrae (Fig. 8.3), while in women and children, it lies further up. The laryngeal mucosa is innervated as far as the vocal folds by the purely sensory *internal branch* of the *superior laryngeal nerve* and below that by the *recurrent laryngeal nerve*, a branch of the *vagus nerve*. The inner laryngeal muscles are all supplied by the (*inferior*) *recurrent laryngeal nerve*. The only exterior laryngeal muscle, the *cricothyroid*, is innervated by the external branch of the *superior laryngeal nerve* (Fig. 8.4).

The *tongue* is basically a powerful muscle body that is covered with a mucosal layer, the mucosa of the tongue. The lingual mucosa in the anterior (presulcal) part of the tongue receives its sensory innervation from the lingual nerve (a branch of the mandibular nerve; cf. Chap. 10) and (with the exception of the vallate papillae) from the *chorda tympani* (branching from the intermediofacial nerve). The posterior (postsulcal) part of the tongue receives its sensory innervation from the *glossopharyngeal nerve* (with the exception of the epiglottic valleculae, which are supplied from the vagus nerve). The exterior lingual muscles (with the exception of the palatoglossus) are innervated from the *hypoglossal nerve* (Fig. 8.4).

The trachea is 10–12 cm long and stretches from the cricoid to the tracheal bifurcation. The smooth trachealis muscle is innervated from the recurrent laryngeal nerve, which is also responsible for the sensory and secretory innervation.

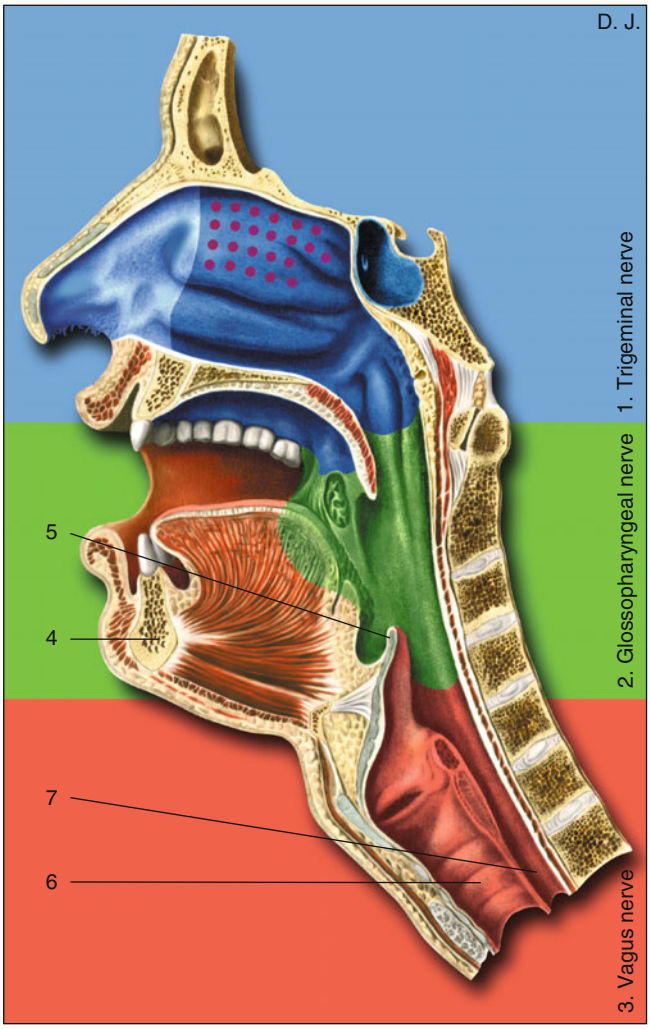


Fig. 8.1 Functional anatomy of the airways. (*1*) Trigeminal nerve (V) with the ophthalmic and maxillary nerves. *Dotted red area*: field of sensory olfactory innervation via the olfactory nerve. (*2*) The sensory innervation area of the glossopharyngeal nerve (IX), (*3*) the sensory innervation area of the vagus nerve (X), (*4*) mandible, (*5*) epiglottis, (*6*) trachea, (*7*) esophagus (With permission from Danilo Jankovic)

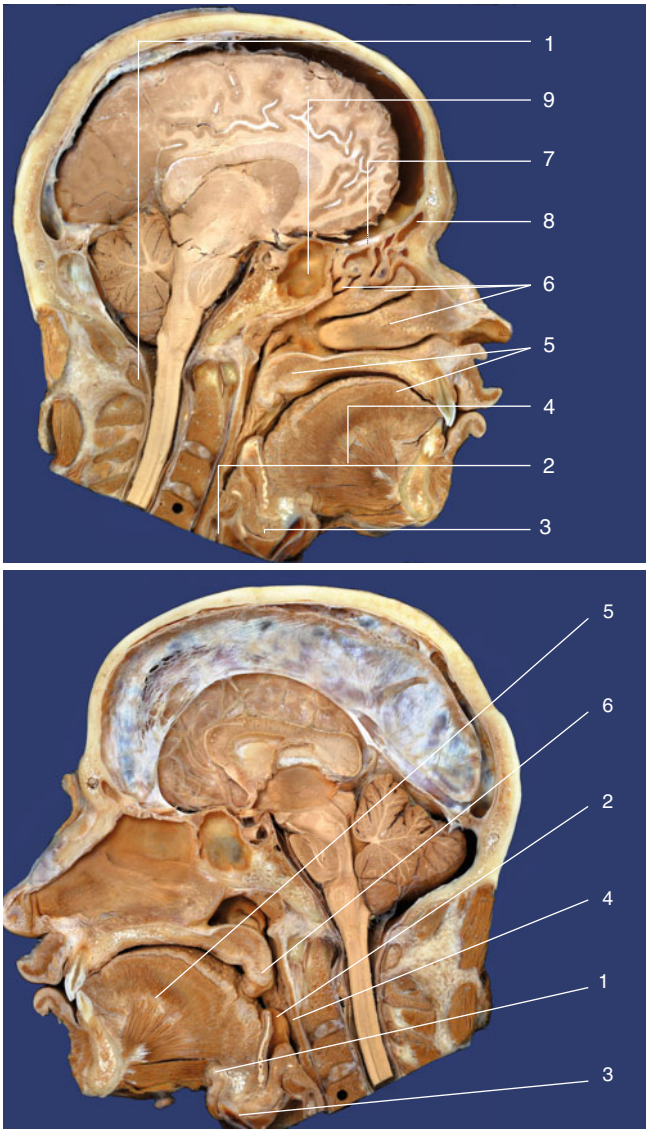


Fig. 8.2 (**a**) Functional anatomy of the airways. Median section through the head. (*1*) Posterior arch of the atlas, (*2*) cricoid cartilage, (*3*) geniohyoid, (*4*) genioglossus, (*5*) soft/hard palate, (*6*) nasal concha (superior, middle, inferior), (*7*) olfactory tract, (*8*) frontal sinus, (*9*) sphenoid sinus (With permission from Danilo Jankovic). (**b**) Functional anatomy of the airways. Median section through the head. (*1*) Hyoid bone, (*2*) epiglottis, (*3*) thyroid cartilage, (*4*) posterior pharyngeal wall, (*5*) genioglossus, (*6*) uvula (With permission from Danilo Jankovic)

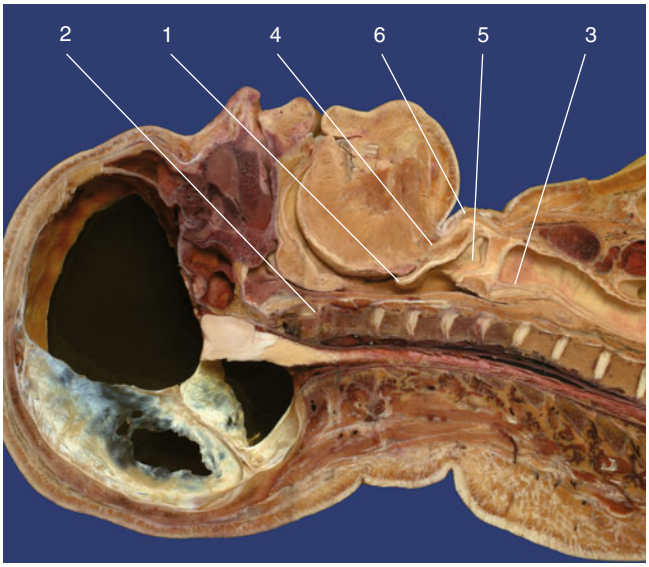


Fig. 8.3 Anatomic specimen. Paramedian sagittal section. In adults, the larynx is located over the C3–C6 vertebrae. (*1*) Epiglottis, (*2*) atlantodental joint, (*3*) trachea, (*4*) hyoid bone, (*5*) laryngeal ventricle, (*6*) thyroid cartilage (With permission from Danilo Jankovic)

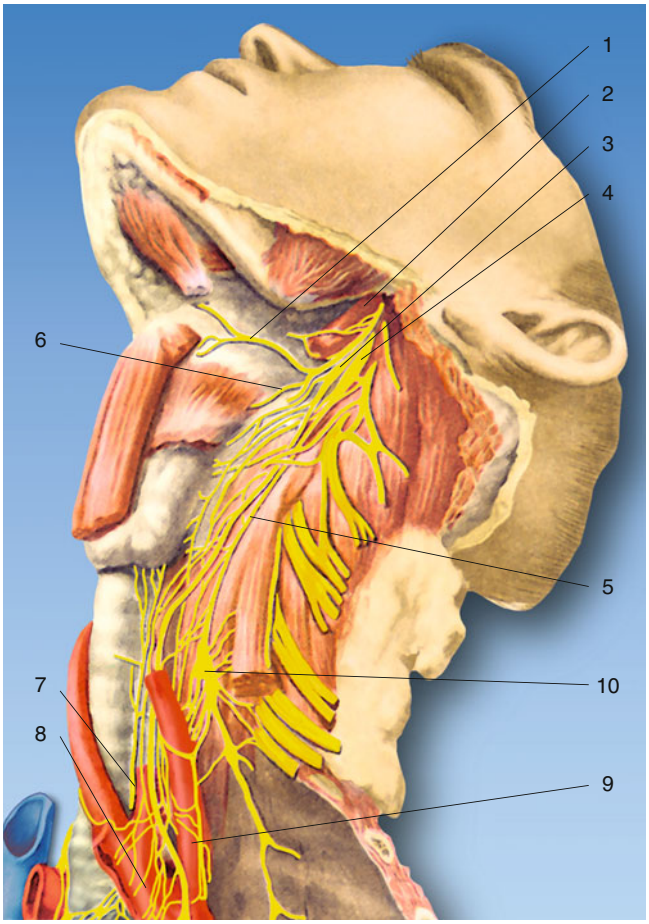


Fig. 8.4 Anatomy. (*1*) Hypoglossal nerve, (*2*) internal carotid artery,(*3*) inferior ganglion of the vagus nerve, (*4*) inferior cervical ganglion, (*5*) vagus nerve, (*6*) superior laryngeal nerve, (*7*) recurrent laryngeal nerve, (*8*) aortic arch, (*9*) left subclavian artery, (*10*) cervicothoracic ganglion (With permission from Danilo Jankovic)

Anesthesia of the Larynx and Trachea

Various procedures can be used for anesthesia of the laryngeal and tracheal mucosa: spraying and advancement with the fiber endoscope, injection of local anesthetic through the cricothyroid membrane, bilateral blockade of the superior laryngeal nerve, and aerosol inhalation.

Fiberoptic Intubation [1–6, 8, 9]

The importance of fiberoptic devices for anesthesia was already recognized in the late 1960s by Murphy and Kronschwitz. Taylor and Towey were the first to report the use of the fiber bronchoscope, developed in 1968 by Ikeda, for intubation.

Indications

The principal indication for using flexible endoscopes is difficult intubation:

- Limited mobility (jaw, head, cervical spine)
- Malformations and acquired anomalies (face, jaw, oral cavity, neck)
- Space-occupying lesions (oral cavity, pharynx, larynx, trachea)
- Patient history (including failure with conventional techniques)
- Plastic surgery in the facial region
- Mobility disturbances in the head and nucha
- Reintubation in high-risk patients
- To avoid the need for contraindicated drugs (e.g., muscle relaxants)
- When the patient has a full stomach as well as hindrances to intubation

Sedatives and Analgetics for Endoscopic Intubation

Benzodiazepines (Table 8.1) are used in combination with opioids (Table 8.2) for sedation and attenuation of the laryngeal reflexes. Benzodiazepines and opioids have synergistic effects, and the combination is highly effective at low dosage.

Table 8.1 Pharmacology of benzodiazepines [9]

	Dosage (mg/kg)	Clearance (mg/kg/min)	Elimination half-life (h)
Diazepam	0.3–0.5	0.2–0.5	21–37
Midazolam	0.15–0.3	6–8	1–4
Lorazepam	0.05	0.7–1.0	10–20

Table 8.2 Pharmacology of opioids [9]

	Analgetic strength	Clearance (mg/kg/min)	Elimination half-life (h)
Morphine	1	15–23	114
Fentanyl	75–125	11–21	185–219
Sufentanil	500–1,000	13	148–164
Alfentanil	25	5.0–7.9	70–98

Local Anesthesia for Endoscopic Intubation

Adequate topical anesthesia of the mucosa in the upper respiratory tract is required for fiber-endoscopic intubation, in order to subdue coughing, swallowing movements, laryngospasm, and excessive secretion. Lidocaine (Xylocaine®) and cocaine are usually used for topical anesthesia in the respiratory tract for endoscopic intubation in conscious patients.

Lidocaine (Xylocaine®) is administered as a 4 % solution for mucosal anesthesia. A 10 % pump spray is also used for the oropharynx and nasopharynx. Gargling with a 2 % viscous lidocaine solution can also induce oropharyngeal anesthesia. Anesthesia of the nose can be achieved with a 2 % gel.

Bleeding in the region of the nasopharynx and mesopharynx can become a severe hindrance to endoscopic intubation. In the nasal procedure, an attempt therefore needs to be made to achieve not only good mucosal anesthesia (with lidocaine spraying) but also bleeding prophylaxis using vasoconstrictive agents. The local anesthetic and vasoconstrictive effects of *cocaine* are therefore advantageous during nasal endoscopic intubation. The synthetic analogs lack cocaine's vasoconstrictive action. Cocaine nose drops (4–10 %, 0.5 mL into each nostril) have been found to be very useful in practice. The maximum dosage of cocaine is 100–200 mg. Alternatively, a mixture of lidocaine (4 %) and phenylephrine (1 %) at a proportion of 3:1 can be used in the nasal procedure. In the oral procedure, pretreatment of the mesopharynx with a lidocaine spray is necessary.

Possible alternatives to initial treatment with local anesthetics and mucosal decongestants in the nasal airways include:

- 10 % cocaine solution (1-mL ampullae)
- 10 % lidocaine spray + xylometazoline 0.1 % (Otrivin®)
- 4 % lidocaine + phenylephrine 1 % (Neo-Synephrine®)
- Lidocaine spray + naphazoline nitrate 0.05 % (Privine®)

Before anesthesia in the oropharynx, an anticholinergic (atropine) should be injected to dry out the mucosa. Oropharyngeal anesthesia is administered with two or three spray shots of 10 % lidocaine or by gargling with 2–4 mL of a viscous solution of 2 % lidocaine for 30–40 s.

Practical Procedure for Nasal Endoscopic Intubation in a Conscious Patient

The anesthetist usually stands at the patient's head, as this position provides easier orientation for conventional intubation. Steps in endoscopic intubation:

- Adequate preoxygenation (5–10 min).
- Secretory inhibition: atropine 0.5 mg i.m.
- Inhibition of laryngeal reflexes: fentanyl 0.1 mg i.v. (alfentanil 1–2 mg in adults) combined with a sedative (e.g., midazolam)
- Vasoconstriction, mucosal anesthesia: 10 % cocaine (0.5 mL into each nostril).
- Preparation of the tube and flexible endoscope (Xylocaine® gel, antifogging agent) (Fig. 8.5a). The endotracheal tube is advanced over the flexible part of the endoscope and attached with adhesive plaster.
- Introduction of the flexible endoscope via a nostril (under vision).
- Adjustment of the laryngeal inlet (Fig. 8.5b)
- Mucosal anesthesia via the biopsy channel (5-mL Xylocaine® 2 %). The onset of action should be awaited.
- Introduction of the flexible part of the endoscope into the trachea.
- Induction of anesthesia.
- Advancement of the tube over the flexible endoscope, using it as a guide rail.

Practical Procedure for Nasal Endoscopic Intubation in an Anesthetized Patient

- Atropine for secretory inhibition
- 10 % cocaine nose drops (1 mL) (vasoconstriction local anesthesia)
- Preoxygenation
- Induction of anesthesia (etomidate, fentanyl)
- Introduction of a spiral tube into the nasopharynx
- Introduction of the endoscope, lubricated with gel, into the tube, after applying antifogging agent to the lens
- Inspection of the laryngeal inlet and vocal folds
- Introduction of the scope into the trachea with visual guidance, as far as the bifurcation
- Advancement of the tube over the endoscope, using it as a guide rail
- Checking of tube position
- Continuation of anesthesia

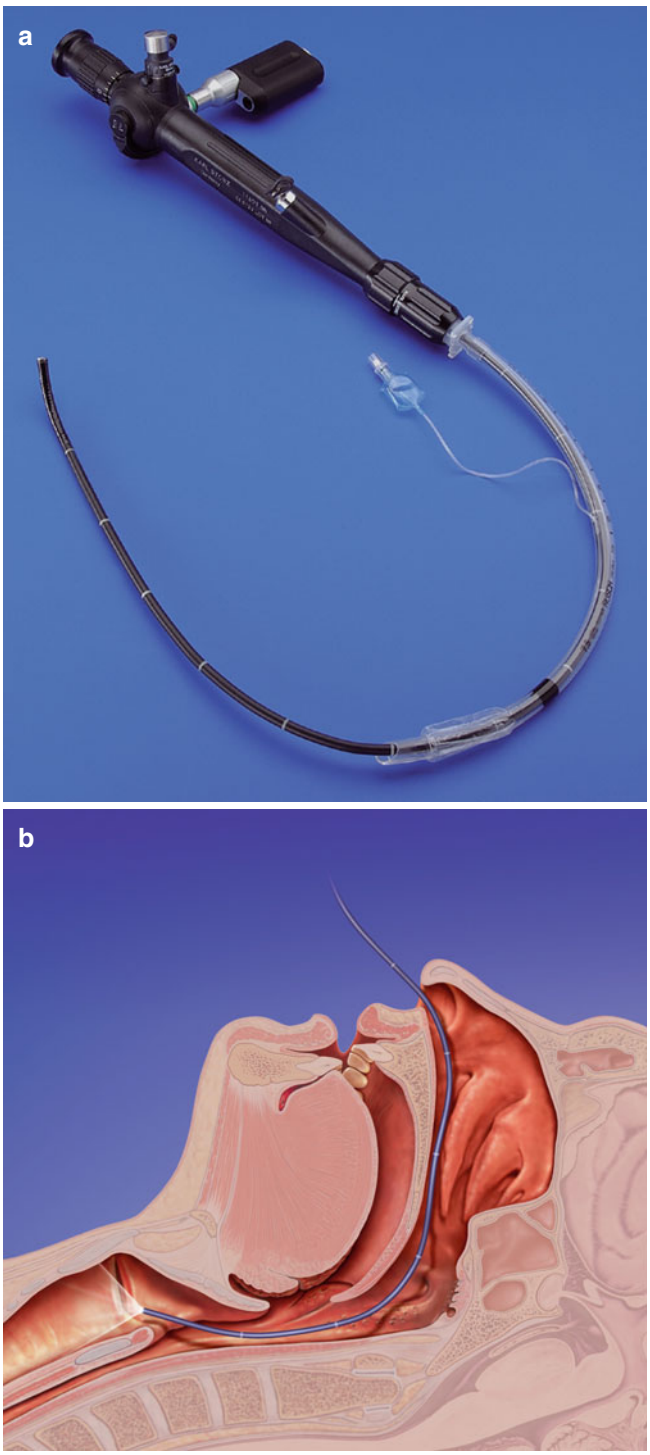


Fig. 8.5 (**a**) Flexible fiber endoscope for intubation (5.2 mm) with LED battery light source and tube switch attached (Karl Storz, Tuttlingen). (**b**) Introducing the flexible fiber endoscope into the trachea and positioning of the flexible endotracheal tube

Techniques for Blocking Individual Nerves of the Airway

Danilo Jankovic

The following nerve blockades are used in the region of the upper airways:

- Superior laryngeal nerve blockade—above the vocal folds
- Translaryngeal block—larynx and trachea below the vocal folds
- Glossopharyngeal nerve blockade—oropharynx

Vagus Nerve, Superior Laryngeal Nerve, and Recurrent Laryngeal Nerve

Anatomy [11, 14–18, 21–23]

The *vagus nerve* (cranial nerve X) not only supplies areas of the head but also descends into the thoracic and abdominal space. It is the largest *parasympathetic nerve* and carries motor fibers (pharyngeal muscles), exteroceptive sensory fibers, visceromotor fibers, viscerosensory fibers, and gustatory fibers. The fibers emerge immediately behind the oliva, combine into a nerve trunk, and exit from the skull through the *jugular foramen*.

The nerve forms the superior ganglion of the vagus nerve (jugular ganglion) within the foramen and after passing through it forms the much larger inferior ganglion of the vagus nerve (nodose ganglion) (Fig. 8.6).

- The *superior ganglion of the vagus nerve* is connected to the *superior cervical ganglion* and also to the *facial nerve, glossopharyngeal nerve*, and *accessory nerve* (Fig. 8.7a, b).
- The *inferior ganglion* of the *vagus nerve* has connections with the cerebral part of the *accessory nerve* and with the *superior cervical ganglion*, the *hypoglossal nerve*, and the loop between the first and second spinal nerves.

In the area of the head, the vagus nerve gives off a *meningeal branch* (for sensory supply to the dura in the posterior cranial fossa) and also an *auricular branch*.

The *vagus nerve* descends along with the *internal carotid artery, common carotid artery*, and *internal jugular vein*, enclosed in a common connective-tissue sheath, down through the neck and passes along with them through the superior thoracic aperture. It gives off four branches in the cervical area:

1. *Pharyngeal branches*. The motor vagus fibers innervate muscles in the soft palate and throat: the muscles of the tonsillar niche, the levator veli palatini, and the constrictor muscles of the pharynx.
2. *Superior laryngeal nerve*. This arises below the inferior ganglion (nodose ganglion) and divides at the level of the hyoid bone into an external branch (the motor branch for the cricothyroid muscle) and an internal branch (the sensory branch for the laryngeal mucosa as far as the vocal folds) (Figs. 8.6 and 8.7a).
3. *Recurrent laryngeal nerve*. This divides in the chest after the vagus nerve has passed over the *aortic arch* on the *left side* and over the *subclavian artery* on the *right side*. Between the trachea and the esophagus, where the nerve gives off the tracheal branches and esophageal branches, it passes to the larynx. Its terminal branch, the *inferior laryngeal nerve*, provides the motor supply for all of the laryngeal muscles except for the cricothyroid and the sensory supply for the laryngeal mucosa below the vocal folds.

After the inferior ganglion, the two vagus nerves course caudally in each ipsilateral carotid sheath to the superior thoracic aperture. Fibers are exchanged on both sides with the cervical part of the sympathetic nerve, so that from the neck downward, the vagus nerves are actually mixed with parasympathetic and sympathetic nerves (Figs. 8.6 and 8.7a, b).

N.B.
The blockade technique for the vagus nerve is practically identical with that for the glossopharyngeal nerve (see below) and the superior cervical ganglion (cf. Chap. 14).

The present chapter describes blockades of the branches of the vagus nerves (the superior laryngeal nerve and recurrent laryngeal nerve). The use of a vagus nerve blockade as a therapeutic option in the treatment of bronchial asthma has been reported.

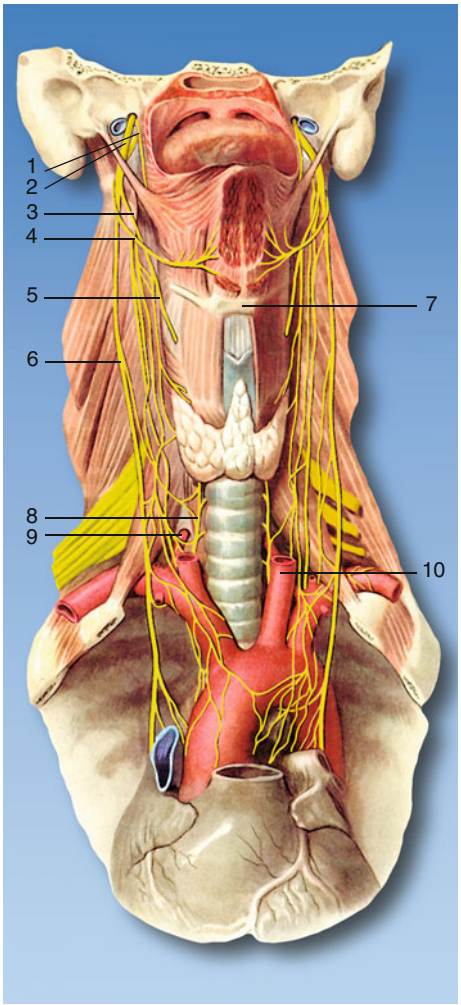


Fig. 8.6 Anatomy. (*1*) Glossopharyngeal nerve, (*2*) inferior ganglion of the vagus nerve, (*3*), superior cervical ganglion, (*4*) hypoglossal nerve, (*5*) superior laryngeal nerve, (*6*) vagus nerve, (*7*) hyoid bone, (*8*) recurrent laryngeal nerve, (*9*) vertebral artery, (*10*) left common carotid artery (With permission from Danilo Jankovic)

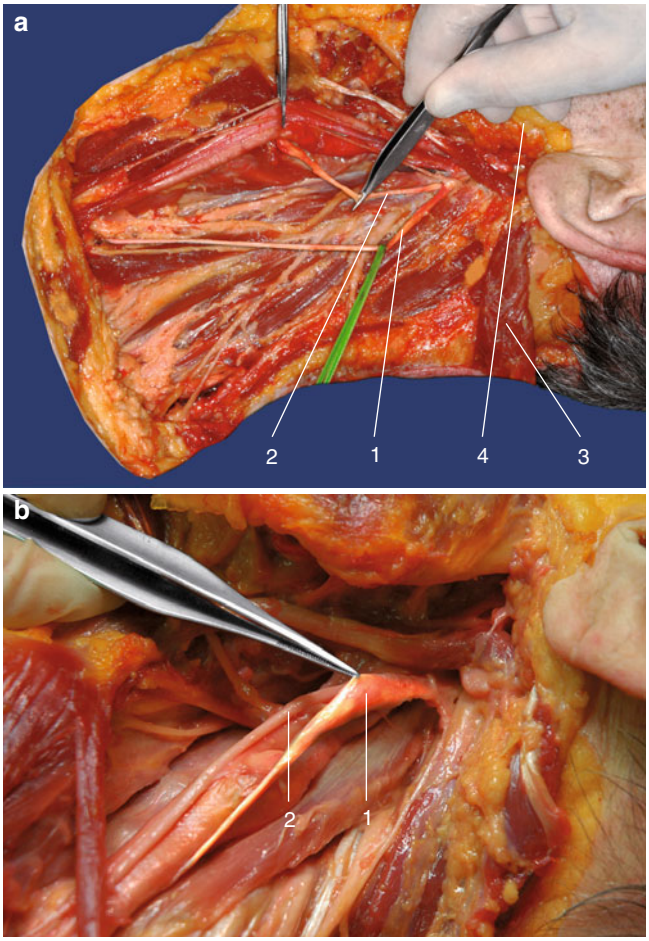


Fig. 8.7 (**a**) Anatomic specimen. (*1*) Division of the vagus nerve. (*2*) Branching of the superior laryngeal nerve, (*3*) sternocleidomastoid (reflected back), (*4*) mandible (With permission from Danilo Jankovic). (**b**) Anatomic specimen. (*1*) Superior cervical ganglion and (*2*) vagus nerve (With permission from Danilo Jankovic)

Superior Laryngeal Nerve Block [10, 12, 13, 19, 20, 22, 24]

Indications

1. Pain and swallowing symptoms or neuralgia in the innervation area: superior laryngeal neuralgia is characterized by pain in the proximal laryngeal area and hyoid bone, as well as the lateral neck region. Swallowing, coughing, and yawning are reported as triggering factors. The symptoms are usually not accompanied by any major neurological deficits.
2. Anesthesia of the airways for intubation of the conscious patient.
3. Laryngoscopy, esophagoscopy, and transtracheal echocardiography.

Contraindications

1. When declined by the patient
2. Local infections and skins diseases at the injection site
3. Anticoagulant treatment
4. Second-degree atrioventricular block

Procedure

This blockade should be carried out by experienced anesthetists or under their supervision.

Preparations

An information discussion with the patient is absolutely necessary.

Check that the emergency equipment is complete and in working order.

Sterile precautions.

Intravenous access, ECG monitoring, ventilation facilities, and pulse oximetry.

Materials

A 25–30-mm-long 23 (25)-G needle

Patient Positioning

Supine, with slightly extended neck

Landmarks (Fig. 8.8a, b)

Greater horn of the hyoid bone
Thyrohyoid membrane

Injection Technique

This branch of the vagus nerve can be easily blocked at the caudal boundary of the hyoid bone (easily palpable, even in obese patients) (Fig. 8.8b). The hyoid bone is pushed with the thumb of the left hand toward the side that is to be blocked, to make the greater horn easier to palpate (Fig. 8.9). The short puncture needle is introduced until there is contact with the greater horn and then withdrawn slightly and advanced at a more oblique angle by approximately 2 mm over the caudal edge of the hyoid bone in the direction of the thyrohyoid ligament. Perforation of the thyrohyoid ligament is noted when a loss of resistance occurs. The needle is then located between the thyrohyoid ligament and the laryngeal mucosa and must not be advanced any deeper.

Following aspiration to exclude an intralaryngeal location, 2 mL of local anesthetic is administered. As the needle is being withdrawn, a further 1 mL of local anesthetic is injected into the neighboring tissue.

The *area of anesthesia* covers the caudal region of the epiglottis, the larynx, and the glottis. A higher concentration of the local anesthetic (e.g., lidocaine 1 %) produces a motor blockade of the cricothyroid.

Complications

1. Laryngeal perforation (intralaryngeal injection)
2. Intravascular injection

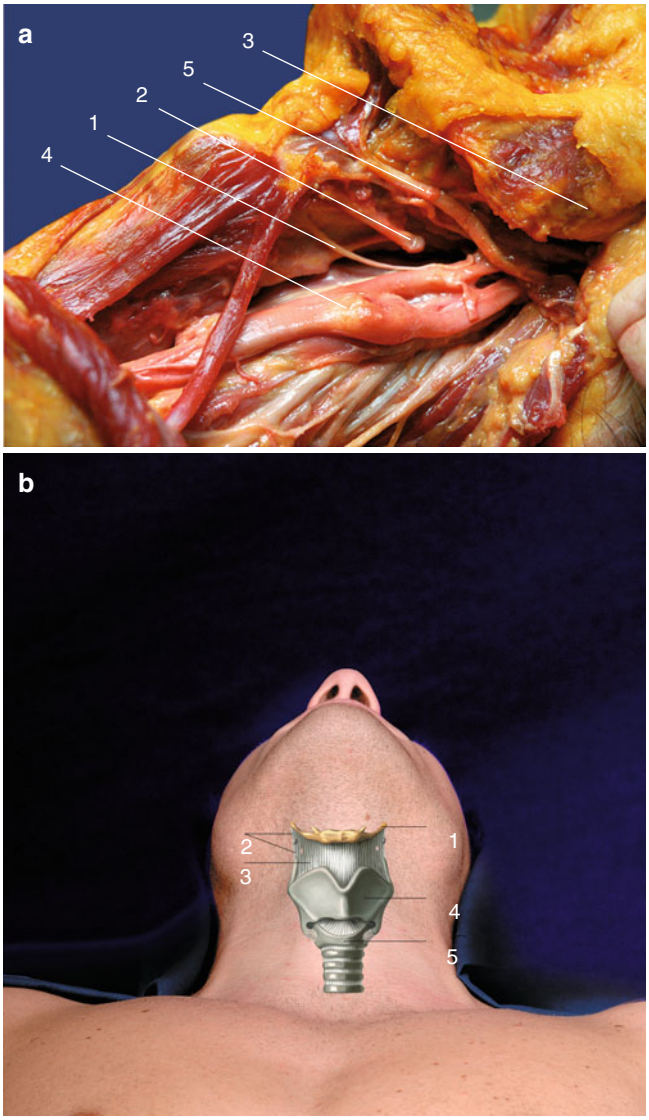


Fig. 8.8 (**a**) Anatomic specimen. (*1*) Superior laryngeal nerve, (*2*) greater horn of the hyoid bone, (*3*) mandible, (*4*) common carotid artery, (*5*) hypoglossal nerve (With permission from Danilo Jankovic). (**b**) Blockade of the superior laryngeal nerve. Landmarks. (*1*) Greater horn of the hyoid bone, (*2*) thyrohyoid ligament, (*3*) thyrohyoid membrane, (*4*) thyroid cartilage, (*5*) cricoid cartilage (With permission from Danilo Jankovic)

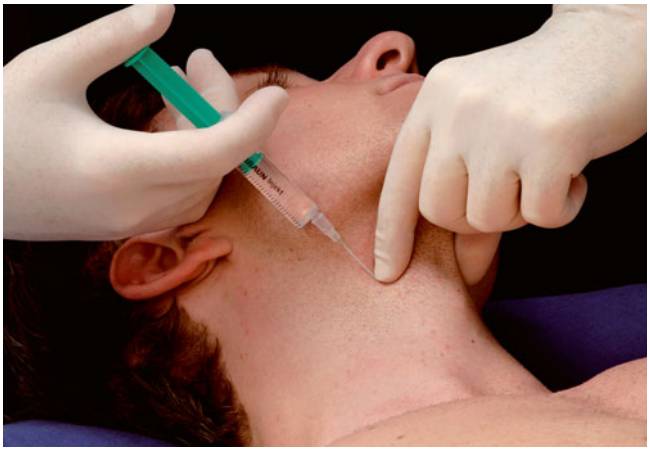


Fig. 8.9 Blockade of the superior laryngeal nerve. The puncture needle is introduced until there is contact with the greater horn of the hyoid bone (With permission from Danilo Jankovic)

Ultrasound-Guided Superior Laryngeal Nerve Block

The cartilages of the larynx are presented in Fig. 8.10. A high-frequency linear US probe (6–15 MHz) is used.

The superior laryngeal nerve (SLN) bifurcates near the pharynx into the external and internal sensory branches. The bilateral SLN block can be used to obtain airway anesthesia, using a percutaneous approach based on anatomic landmarks (greater cornu of the hyoid bone and the thyroid cartilage) (Figs. 8.8 and 8.10). These landmarks may be difficult to locate in some patients. The anatomical study confirmed the presence of a space (named SLN space) containing the internal branch of the superior laryngeal nerve, with hyoid bone superiorly, the thyroid cartilage inferiorly, the thyrohyoid muscle anteriorly, the external carotid artery posteriorly, and the thyrohyoid membrane as the floor (Figs. 8.10 and 8.11) [11, 15–18, 21, 23]. The thyroid cartilage and hyoid bone were identified first, with a probe in a parasagittal plane. The probe was then moved laterally to visualize from the skin to the larynx (Fig. 8.11):

1. Thyrohyoid muscle—large hypoechoic band, inserted on the hyoid bone and passing over the thyroid cartilage (TC) (Fig. 8.11)
2. Thyrohyoid membrane (hyperechoic layer, marking the interface with the hypoechoic pre-epiglottis space) (Fig. 8.11)

The quality of the ultrasound image was rated optimal when all the following were seen: thyrohyoid muscle, pre-epiglottis space, hyoid bone (HB), thyroid cartilage (TC), and thyroid membrane (Fig. 8.11).

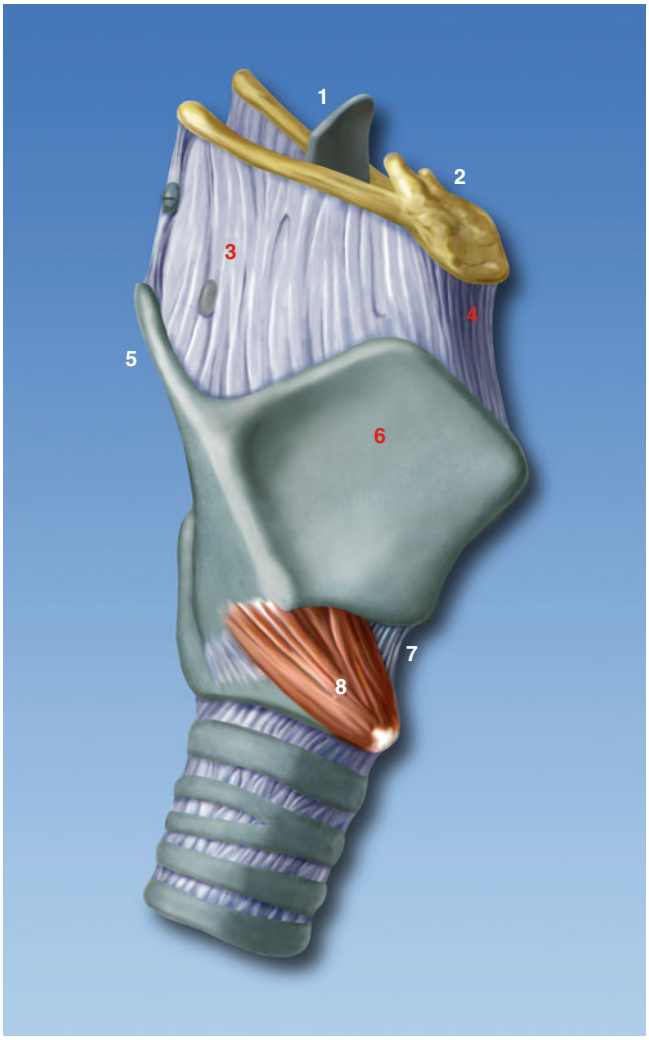


Fig. 8.10 Cartilages of the larynx. (*1*) Epiglottis, (*2*) hyoid bone, (*3*) thyrohyoid membrane, (*4*) thyrohyoid ligament, (*5*) superior horn of thyroid cartilage, (*6*) thyroid cartilage lamina, (*7*) cricothyroid ligament, (*8*) cricothyroid muscle (With permission from Danilo Jankovic)

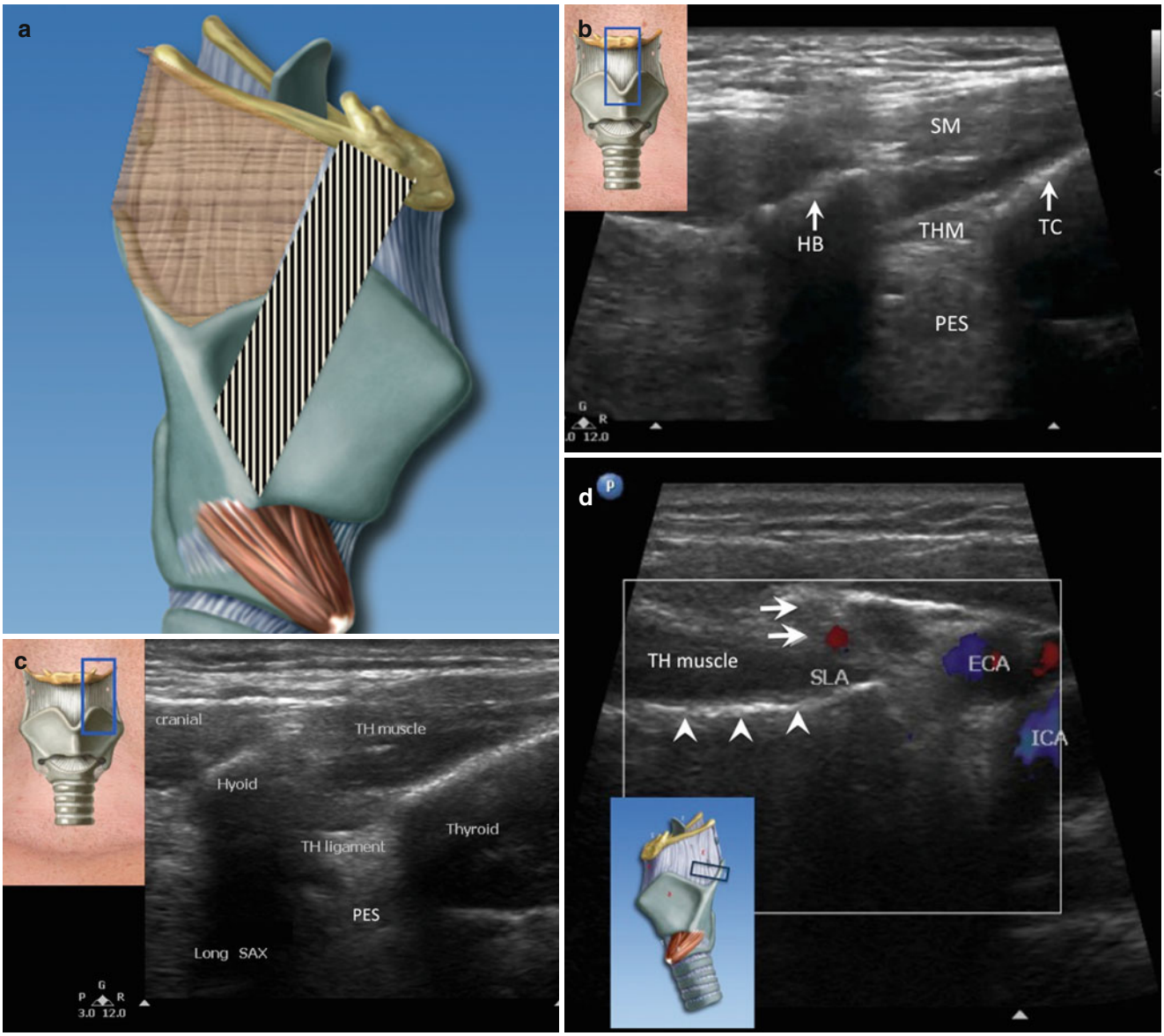


Fig. 8.11 Sonoanatomy of the superior laryngeal nerve. (**a**)The *brown* area, the superior laryngeal nerve (SLN) space, is bounded by the hyoid bone superiorly, thyroid cartilage inferiorly, thyrohyoid muscle (black and white striated area) anteriorly, and external carotid artery posteriorly (not shown); (**b**) Sonoanatomy of the anterior aspect of thyrohyoid region. *HB* hyoid bone, *THM* thyrohyoid membrane, *SM* strap muscle, *TC* thyroid cartilage, *PES* pre-epiglottis space (fat). The position of the probe is shown in the inset. (**c**) Parasagittal scan of the thyrohyoid region. *TH ligament* (thyrohyoid ligament or membrane), (**d**) Short-axis scan of the thyrohyoid membrane. The superior laryngeal nerve SLA is seen here (*arrows*). In case the nerve is not seen, the location can be approximated by the thyrohyoid (*TH*) muscle anteriorly, external carotid artery (*ECA*) posteriorly, and the thyrohyoid membrane (*arrowheads*) deep to the nerve. *ICA* internal carotid artery (Reproduced with permission from Peter Cheng)

Injection

In-plane is the preferred technique. The SLN may not be visualized well in ultrasound scan. With a 23-G needle, 2–3 mL of local anesthetics was injected into the surface of the thyrohyoid membrane, anterior to the external carotid artery and posterior to the thyrohyoid muscle under ultrasound guidance (Fig. 8.12). This area correlates with the location of the internal branch of the superior laryngeal nerve.

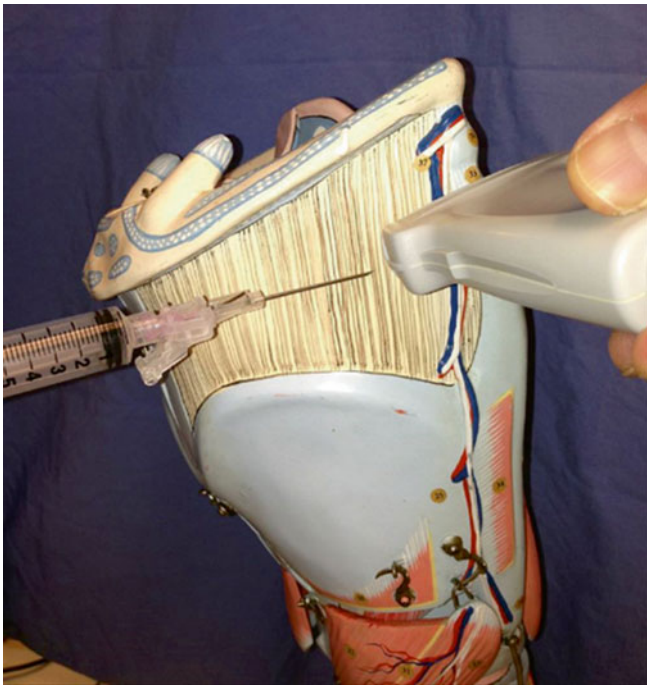


Fig. 8.12 Diagram showing the probe position (similar to Fig. 8.11d) and the direction of the needle insertion for the injection of the internal branch of the superior laryngeal nerve (Reproduced with permission from Peter Cheng)

Recurrent Laryngeal Nerve Block (Transtracheal Injection) [10, 13, 14, 19, 20, 22]

Indications
Local anesthesia in the lower pharynx, larynx, and trachea for procedures such as bronchoscopy, esophagoscopy, and endotracheal intubation and for prophylaxis against laryngospasm

Contraindications
Patients with a short neck, goiter, or severely limited movement in the cervical spine

Procedure

Positioning
Supine, with maximally extended neck

Landmarks (Fig. 8.13)
1. Thyroid cartilage
2. Cricoid cartilage
3. Median cricothyroid ligament

Injection Technique
The patient must not swallow, speak, cough, or move. Sedation with fentanyl and midazolam is recommended to prevent heavy coughing attacks.

The anesthetist stands at the patient's head. The puncture is carried out in the *midline* of the neck, as this area has the poorest vascularization. The *cricothyroid membrane* is palpated with the index and *middle fingers* of the left hand (Fig. 8.13).

Vertically to the examination table, a fine 30–40-mm-long 23-G needle with a local anesthetic syringe attached is introduced through the cricothyroid membrane into the lumen of the trachea (Fig. 8.14).

After air has been aspirated (to confirm the intratracheal position), the local anesthetic is rapidly injected into the tracheal lumen. The needle is then quickly removed again. Strong coughing accelerates the spread of the local anesthetic.

Dosage
2–4 mL 2–4 % lidocaine. A higher concentration (2–4 %) accelerates the onset of effect.

N.B.
1. External blockade of the recurrent laryngeal nerve is rarely indicated (e.g., for administering neurolytics in carcinoma of the glottis).
2. The recurrent laryngeal nerve lies in a fossa between the esophagus and the trachea and can be blocked underneath the cricoid cartilage. The injection is carried out at the posterolateral edge of the first tracheal ring.
3. Unintentional blockade of the recurrent laryngeal nerve occurs very often after blockades of the stellate ganglion (cf. Chap. 13 stellate ganglion block).

Complications
1. Intravascular injection, with toxic reactions due to rapid resorption of the (high-dose) local anesthetic
2. Esophageal perforation

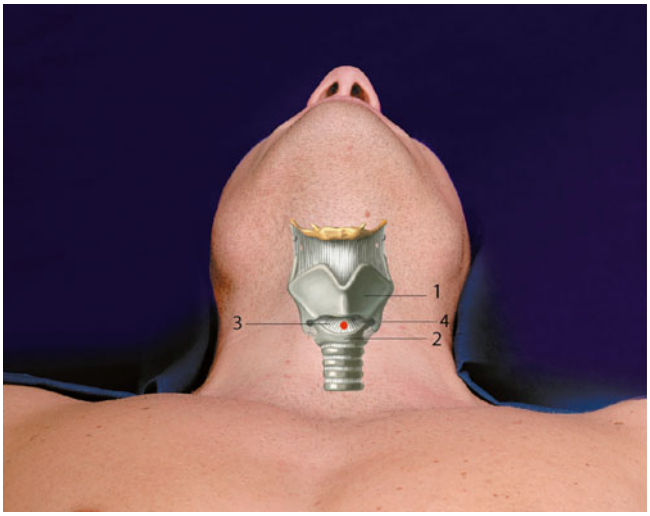


Fig. 8.13 Transtracheal injection. Landmarks. (*1*) Thyroid cartilage, (*2*) cricoid cartilage, (*3*) median cricothyroid ligament, (*4*) puncture site (*red*) (With permission from Danilo Jankovic)

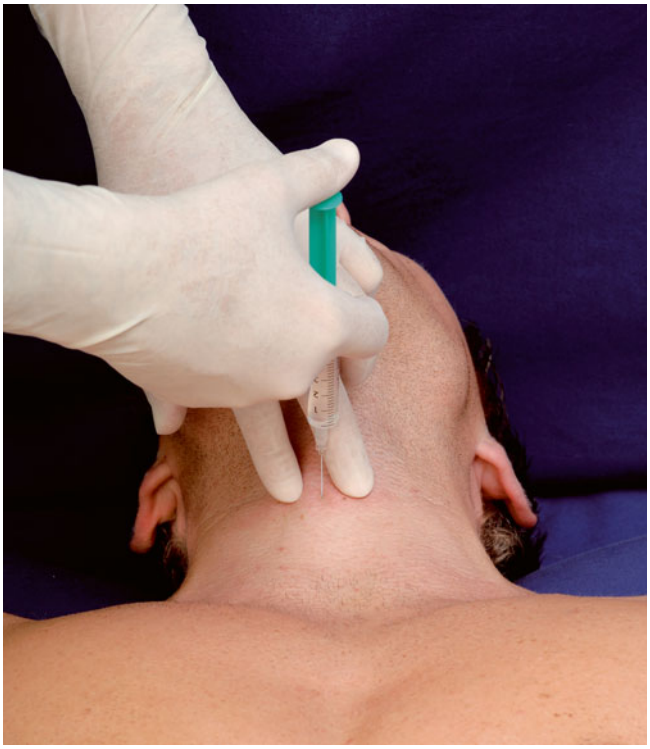


Fig. 8.14 Transtracheal injection. Puncture in the midline of the neck, through the cricothyroid membrane, vertical to the examination table (With permission from Danilo Jankovic)

Glossopharyngeal Nerve Block

Danilo Jankovic

Anatomy [25–28]

Cranial nerve IX (the glossopharyngeal nerve) is closely related to the vagus nerve (X), both topographically and functionally (Fig. 8.15). It provides the *sensory* innervation for the *middle ear*, for areas of the tongue and *pharynx*, and for *motor muscles* of the pharynx. It contains motor, visceromotor (parasympathetic), viscerosensory, and gustatory fibers.

Behind the oliva, it passes directly above the vagus nerve out of the medulla and exits the skull along with the vagus nerve through the *jugular foramen*. Within the foramen, it forms the *superior ganglion* and after passing through it forms the larger *inferior ganglion* (the *petrosal ganglion*). Lateral to the internal carotid artery and the pharynx, it follows an arching course toward the base of the tongue, where it divides into several terminal branches. The *tympanic nerve* provides the sensory supply for the mucosa of the tympanic cavity and pharyngotympanic (auditory) tube. The secretory fibers pass as the lesser petrosal nerve to the otic ganglion. In addition to connections to the vagus nerve, facial nerve, and sympathetic nerve, the *carotid branch of the glossopharyngeal nerve* is also given off from the inferior ganglion. Also given off are the *pharyngeal branches*, which along with part of the vagus nerve form the *pharyngeal plexus* and are involved in the sensory and motor supply to the pharynx, the *stylopharyngeal branch* (stylopharyngeus muscle), the *tonsillar branches* (to the tonsils and soft palate), and the *lingual branches* (posterior third of the tongue).

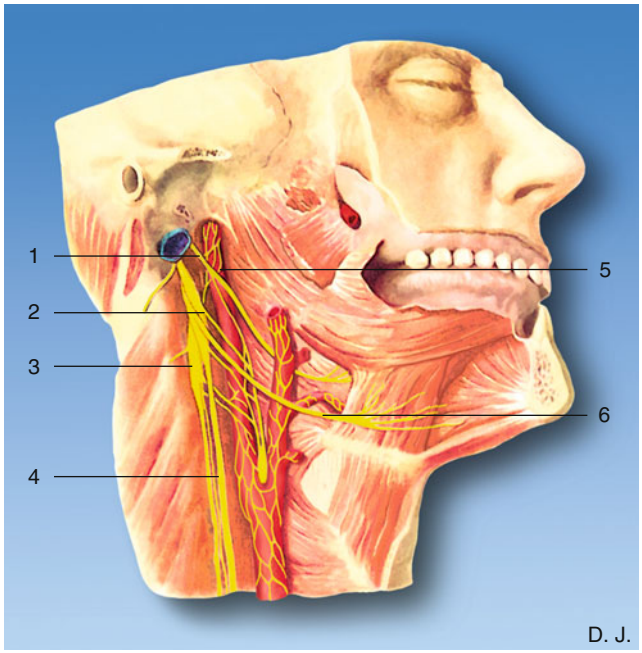


Fig. 8.15 Anatomy. (*1*) Glossopharyngeal nerve, (*2*) inferior ganglion of the vagus nerve, (*3*) superior cervical ganglion, (*4*) vagus nerve, (*5*) internal carotid artery, (*6*) hypoglossal nerve (With permission from Danilo Jankovic)

Indications

Diagnostic

- Differential diagnosis of trigeminal neuralgia (anterior two thirds of the tongue) and glossopharyngeal neuralgia (posterior third of the tongue)
- Before neurodestructive procedures

Therapeutic

- Glossopharyngeal neuralgia [26]. Glossopharyngeal neuralgia is characterized by strictly unilateral, fulminant paroxysms of severe stabbing and burning pain (oropharynx, tonsillar niches, more rarely in the area of the ear—due to co-involvement of the tympanic branch—or mandibular angle). The first report of this clinical picture was by Sicard and Robineau in 1920. In 10–30 % of cases, there is simultaneous trigeminal neuralgia. The symptoms are unilateral in 75 % of cases. Triggering factors include chewing, speaking, swallowing, coughing, yawning, and cold food and drinks. The glossopharyngeal nerve can sometimes be paralyzed along with the vagus nerve and accessory nerve due to a tumor or aneurysm in the posterior cranial fossa.
- Tumor pain (neurodestructive procedures—e.g., injection of neurolytic agents)

Contraindications

- When declined by the patient
- Second-degree atrioventricular block
- Anticoagulant treatment
- Simultaneous bilateral blockade
- Contralateral paresis of the glossopharyngeal and adjacent nerves

Procedure

This blockade should only be carried out by experienced anesthetists or under their supervision.

Preparations

Check that emergency equipment is complete and in working order; sterile precautions; intravenous access; ECG monitoring; ventilation facilities; and pulse oximetry. An information discussion with the patient is absolutely necessary.

Materials

- A 40-mm-long 23-G needle

Patient Positioning

- Supine, with the head rotated to the contralateral side by approximately 30°

Landmarks (Fig. 8.16)

- Angle of the mandible.
- Mastoid process.
- Styloid process. The variable anatomy and depth of the styloid process need to be taken into account during the puncture procedure.

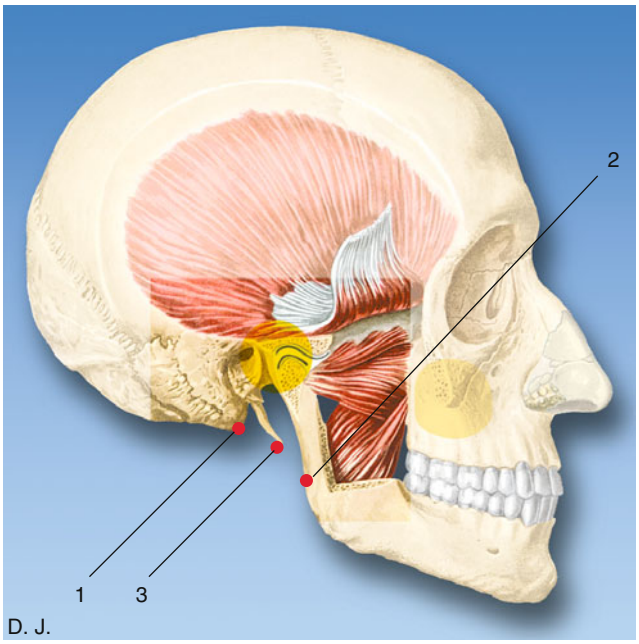


Fig. 8.16 Landmarks. (*1*) Mastoid process, (*2*) angle of the mandible, (*3*) styloid process (With permission from Danilo Jankovic)

Injection Technique

Extraoral Access (Lateral Cervical Technique) [25–27]

The needle is introduced at the midpoint between the mastoid tip and the angle of the mandible, vertically to the skin, until there is bone contact with the styloid process (Fig. 8.17). This is usually after approximately 3 cm. The needle is then withdrawn slightly and, after loss of bone contact with the styloid process, is introduced posteriorly at a depth of 0.5–1.0 cm.

After aspiration, fractionated administration of a local anesthetic is carried out.

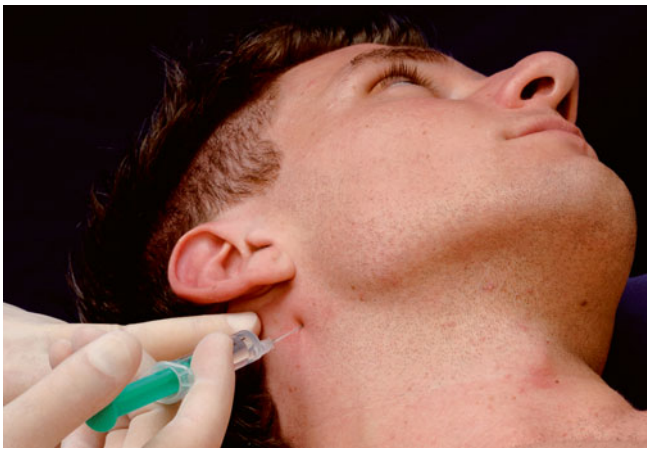


Fig. 8.17 Glossopharyngeal nerve. Injection technique vertical to the skin, toward the styloid process

Intraoral Access

Following topical anesthesia of the tongue, the patient's mouth is opened widely and the posterior tonsillar pillar (palatopharyngeal fold) identified by using a Macintosh laryngoscope blade. In an aseptic manner, the stylet of the 22G spinal needle should be removed from the disposable spinal needle and discarded. Subsequently, utilizing the sterile container that the 22G spinal needle was packed in, the distal 1 cm of the needle is bent to allow more controlled submucosal insertion. The needle tip is inserted submucosally, and following careful aspiration for blood, 2–3 mL of local anesthetic is injected. The block is then repeated on the contralateral side (Fig. 8.18).

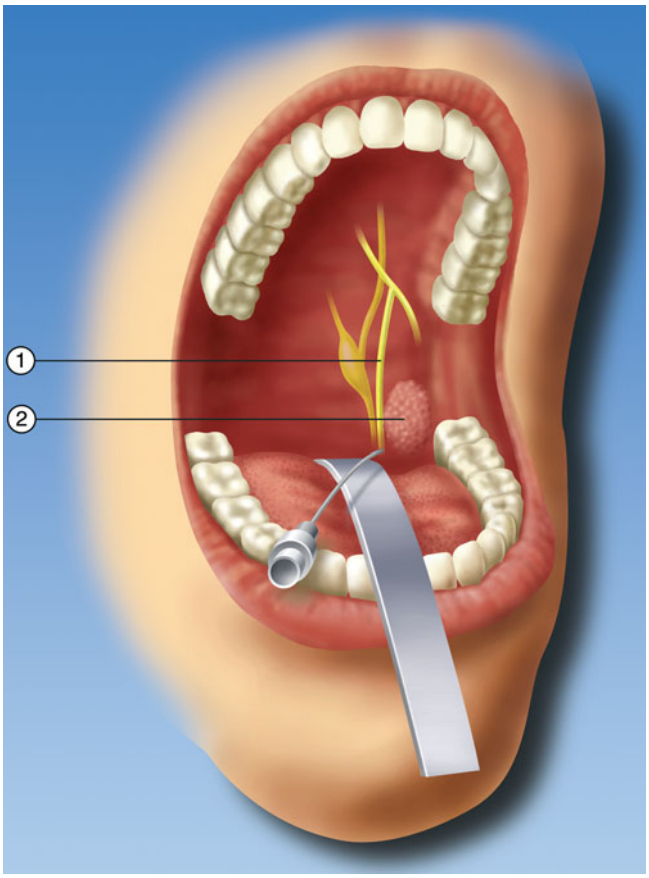


Fig. 8.18 Intraoral glossopharyngeal nerve block. (*1*) Glossopharyngeal nerve, (*2*) palatine tonsil

Potentials Problems

Effects of the Blockade

Globus sensation in the neck, swallowing difficulties, loss of sensation in the pharynx and mucosa, retropharyngeal and tonsillar paresthesias are signs that the glossopharyngeal nerve is blocked. The vagus nerve, which is in the vicinity of the glossopharyngeal nerve, and the superior cervical ganglion and hypoglossal nerve are always anesthetized as well.

Dosage

Two to three milliliters of local anesthetic—e.g., procaine 0.5–1.0 %, prilocaine 0.5–1.0 %, ropivacaine 0.2 %, bupivacaine 0.125 % (levobupivacaine 0.125 %); if appropriate, mixed with 80 mg methylprednisolone.

Complications

- Intravascular injection, with toxic reactions (internal carotid artery, internal jugular vein) (cf. Chap. 13 stellate ganglion)
- Hematoma formation
- Intraoral approach
- With the intraoral approach, the terminal branches of the glossopharyngeal nerves are closely related to the internal carotid arteries, which lie immediately lateral to the needle tips if they are correctly positioned.

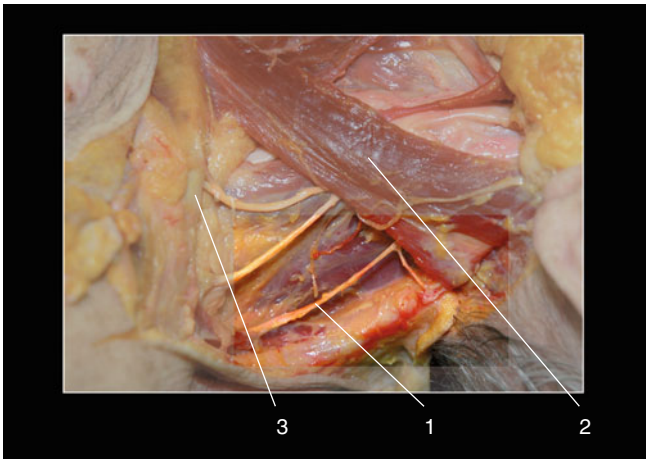


Fig. 8.19 Anatomic specimen. (*1*) Course of the accessory nerve, (*2*) sternocleidomastoid, (*3*) clavicle (With permission from Danilo Jankovic)

Side Effects

Simultaneous anesthetization of the following nerves:

- Vagus nerve (paresis of the vagus nerve is characterized by the collection of saliva in the piriform fossa when there is paresis of the posterior cricoarytenoid muscle—hoarseness, accompanied by nasal vocalization) (Fig. 8.15).
- Hypoglossal nerve (the outstretched tongue deviates toward the paralyzed side) (Fig. 8.15)
- Accessory nerve (weakness in the area of the trapezius) (Fig. 8.19)
- Superior cervical ganglion

These side effects usually arise after the injection of larger amounts of local anesthetic (cf. Chap. 14).

Suggested Reading

Anesthesia of the Airways

1. Ikeda S. Flexible bronchofiberscope. Keio J Med. 1968;17:1.
2. Jankovic D. Anästhesie der luftleitenden Wege. In: Jankovic D, Hrsg. Regionalblockaden & Infiltrationstherapie. Berlin: ABW Wissenschaftsverlag; 2007. p. 49–61.
3. Kleemann PP. Die Intubation mit dem flexiblen Fiberbronchoskop. In: Sehhati-Chafai G, Hrsg. Praxis der Anästhesiologie und Intensivmedizin, Band 1. Bochum: Winkler; 1988.
4. Kronschwitz H. Die Nasotracheale Intubation mit einem Intubation-Fiberscop. Anaesthesist. 1969;18:58.
5. Murphy P. A fiberoptic endoscope used for nasal intubation. Anaesthesia. 1967;22:489.
6. Ovassapian A. Fiberoptic airway endoscopy in anesthesia and critical care. New York: Raven; 1990.
7. Pernkopf E. Atlas der topographischen und angewandten Anatomie des Menschen, 1. Bd. Kopf und Hals. 2. Aufl. München/Wien/Baltimore:Urban Schwarzenberg; 1980.
8. Simmons ST, Schleich AR. Airway regional anesthesia for awake fiberoptic intubation. Reg Anesth Pain Med. 2002;27(2):180–92.
9. Taylor PA, Towey RM. The broncho-fiberscope as an aid to endotracheal intubation. Br J Anaesth. 1972;44:611.

Techniques for Blocking Individual Nerves of the Airway

10. Brown DL. Atlas of regional anesthesia. 3rd ed. Philadelphia: Saunders, Elsevier; 2006.
11. Gervasio A, Mujahed I, Biasio A, Alessi S. Ultrasound anatomy of the neck: the infrahyoid region. J Ultrasound. 2010;13:85–9.
12. Goel AC. Auricular nerve block in bronchial asthma. J Indian Med Assoc. 1981;76:132.
13. Hahn MB, McQuillan PM, Sheplock GJ. Regional anesthesia. St. Louis: Mosby; 1996.
14. Jankovic D. Luftleitende Wege-Nervenblockaden. In: Jankovic D, Hrsg. Regionalblockaden & Infiltrationstherapy. 4 Auflage. Berlin: ABW-Wissenschaftsverlag; 2007. p. 53–6.
15. Kiray A, Naderi S, Ergur I, Korman E. Surgical anatomy of the internal branch of the superior laryngeal nerve. Eur Spine J. 2006;15:1320–5.
16. Lida T, Suzuki A, Kunisawa T, Iwasaki H. Ultrasound –guided superior laryngeal nerve block and translaryngeal block for awake tracheal intubation in a patient with laryngeal abscess. J Anesth. 2013;27:309–10.
17. Melamed H, Harris M, Awasthi D. Anatomic considerations of superior laryngeal nerve during anterior cervical spine procedures. Spine. 2002;27(4):83–6.
18. Monfared A, Kim D, Jaikumar S, Gorti G, Kam A. Microsurgical anatomy of the superior and recurrent laryngeal nerves. Neurosurgery. 2001;49(4):925–31.
19. Moore DC. Regional block. 4th ed. Springfield: Charles Thomas; 1981.
20. Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 4th ed. Philadelphia/New York: Lippincott-Raven; 1998.
21. Paraskevas G, Raikos A, Ioannidis O, Brand-Saberi B. Topographic anatomy of the internal laryngeal nerve: surgical considerations. Head Neck. 2012;34:534–40.
22. Pernkopf E. Atlas der topographischen und angewandten Anatomie des Menschen, 1.Bd. Kopf und Hals. 2. Aufl. München/Wien/Baltimore Urban Schwarzenberg; 1980.
23. Stockwell M, Lozanoff S, Lang S, Nyssen J. Superior laryngeal nerve block: an anatomical study. Clin Anat. 1995;8:89–95.
24. Victor M, Adams RD. Krankheiten der Hirnnerven. In: Harrison's principles of internal medicine. 8th ed. New York: Mc Graw-Hill; 1983.

Glossopharyngeal Nerve Block

25. Brown DL. Atlas of regional anesthesia. 3rd ed. Philadelphia: Saunders, Elsevier; 2006.
26. Jankovic D.Nervus glossopharingeus. In. Jankovic D, Hrsg. Regionalblockaden & Infiltrationstherapy. Berlin: ABW-Wissenschaftsverlag; 2007. p. 57–8.
27. Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 4th ed. Philadelphia/New York: Lippincott-Raven; 1998.
28. Pernkopf E. Atlas der topographischen und angewandten Anatomie des Menschen, 1. Bd. Kopf und Hals. 2. Aufl. München/Wien/Baltimore: Urban Schwarzenberg; 1980.

Chapter 9

Phrenic Nerve Blockade

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, Toronto Western Hospital, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_9,

Anatomy [2, 4, 5]

The *phrenic nerve* (*C3*, *C4*) receives fibers from the fourth cranial nerve and often also from the third.

It crosses the *scalenus anterior* and enters the superior thoracic aperture in front of the subclavian artery (Figs. 9.1 and 9.2). The course of the phrenic nerve on the scalenus anterior muscle (more lateral in the superior part and medial in the inferior part) is regarded as a landmark zone.

The nerve then passes through the mediastinum as far as the diaphragm, giving off fine branches—the pericardiac branches—along the way, to provide the sensory supply to the pericardium (Fig. 9.3). On the surface of the diaphragm, it divides and supplies the entire diaphragmatic musculature. Fine branches provide the sensory supply to the skin adjoining the diaphragm, to the pleura cranially, and caudally to the diaphragmatic peritoneum and the peritoneal coat of the upper abdominal organs.

When the nerve is paralyzed unilaterally, pulmonary capacity declines by approximately 200–500 mL or 400–600 mL. At resting respiration, this represents 37–48 %.

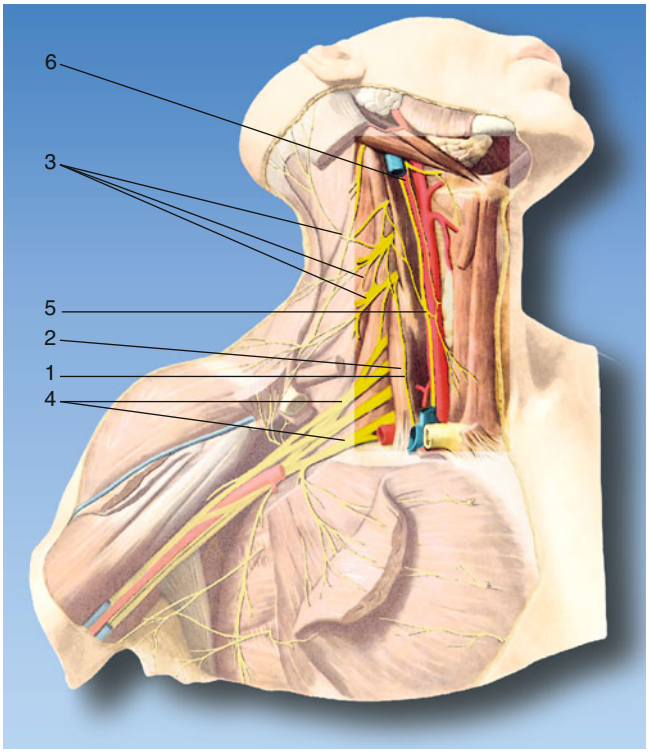


Fig. 9.1 Anatomy. (*1*) Phrenic nerve, (*2*) scalenus anterior, (*3*) cervical plexus (C3, C4), (*4*) brachial plexus, (*5*) vagus nerve, (*6*) internal carotid artery (With permission from Danilo Jankovic)

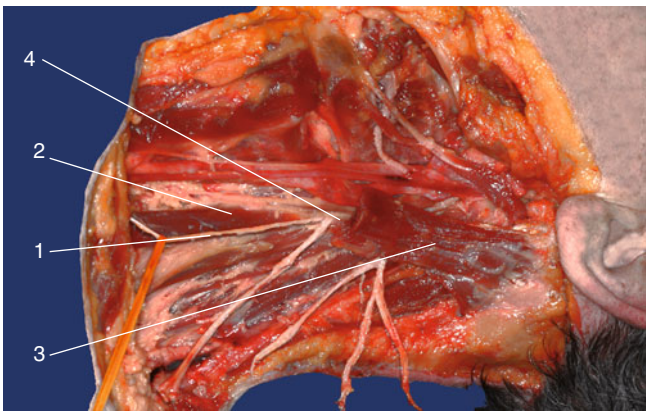


Fig. 9.2 Anatomic specimen. (*1*) Phrenic nerve, (*2*) scalenus anterior, (*3*) sternocleidomastoid (partly resected), (*4*) deep cervical plexus (C4). The course of the phrenic nerve on the scalenus anterior is regarded as a landmark zone (With permission from Danilo Jankovic)

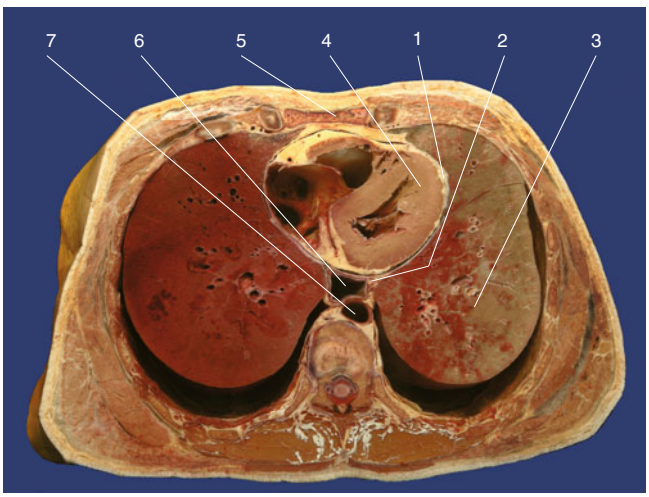


Fig. 9.3 Transverse section through the chest at the level of T9. (*1*) Left phrenic nerve, (*2*) vagus nerve, (*3*) lung, (*4*) left ventricle, (*5*) sternum, (*6*) esophagus, (*7*) aorta (With permission from Danilo Jankovic)

Indications

1. Treatment-resistant hiccup of various etiologies (after exclusion of central causes and nonresponse to drug therapy). Treatment-resistant hiccup can have numerous causes [5]

   Idiopathic
   Psychogenic
   Organic

   - Central nervous system (neoplasms, multiple sclerosis, cerebrovascular insults, trauma)
   - Peripheral nervous system (irritation of the phrenic nerve or vagus nerve)—tumors (stomach, pancreas, lung)
   - Renal causes (uremia) or hepatic diseases
   - Pericarditis
   - Tumors in the head and neck area
   - Hiatus hernia

   Drug-induced/metabolic:

   - Intravenous administration of glucocorticoids, general anesthesia, administration of pethidine (Dolantin®), pentazocine (Fortral®)
   - Infections (sepsis, malaria, tuberculosis)
   - Electrolyte imbalance (hypocalcemia, hyponatremia)

2. Pain in the innervation area (e.g., caused by tumors in the lung, mediastinum, or diaphragm or by metastases). Signs of irritation of the phrenic nerve include epigastric pain radiating into the shoulder area.

Contraindications

1. When declined by the patient
2. Local infections (skin diseases) at the injection site
3. Anticoagulant treatment
4. Severe asthma/emphysema
5. Contralateral paresis of the phrenic nerve or recurrent laryngeal nerve
6. Contralateral pneumothorax
7. No simultaneous bilateral blockade

Procedure

This blockade should only be carried out by experienced anesthetists or under their supervision.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions. Intravenous access, ECG monitoring, ventilation facilities, and pulse oximetry. An information discussion with the patient is absolutely necessary.

Materials

- 30–40-mm-long 22 (23)-G needle
- Electrostimulation
  - Nerve stimulator
  - 35-mm 22-G (15°) needle with injection tubing

Patient Positioning

Supine, with head rotated toward the contralateral side.

Landmarks (Figs. 9.1 and 9.2)

- Lateral edge of the sternocleidomastoid (clavicular part)
- Clavicle
- Scalenus anterior

Injection Technique [1, 5–7]

The patient is asked to turn his or her head toward the contralateral side and raise it slightly, so that the posterior edge of the sternocleidomastoid clearly stands out. Using the thumb and index finger, the sternocleidomastoid is pulled medially in order to draw the carotid artery away from the nerve. The scalenus can be palpated with the thumb.

Approximately 2–3 cm above the clavicle, at the lateral edge of the sternocleidomastoid, at right angles to the body axis and almost parallel to the clavicle, the needle is introduced 2–3 cm deep in the medial direction. The index finger, positioned medially, is used to check the needle position.

Following careful aspiration, 3–5 mL of a local anesthetic is injected (Fig. 9.4).

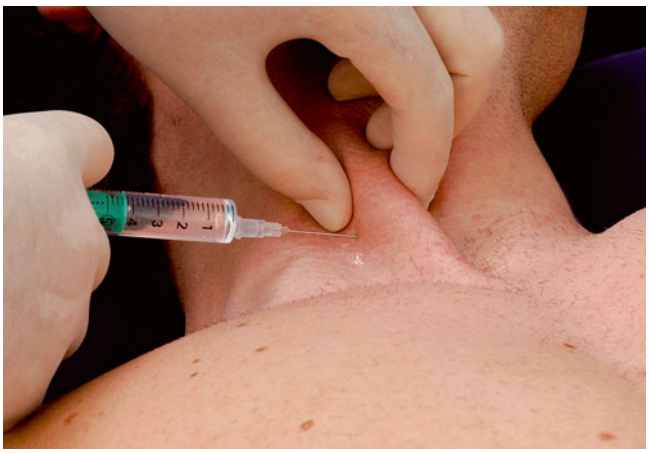


Fig. 9.4 Palpation of the sternocleidomastoid. Introduction of the needle medial and transverse to the body axis (With permission from Danilo Jankovic)

Ultrasound-Guided Injection Technique

The scan is similar to the scan of the interscalene block (refer to Chap. 29). The scan should reveal the sternocleidomastoid, scalenus anterior, and medius muscles as well as the interscalene groove. Usually the level is around C6. With this, a fascia expansion is seen between the scalenus anterior and sternocleidomastoid muscles (Fig. 9.5). An in-plane technique with the needle inserted from lateral to medial is suggested. Caution should be exercised because of the superficial location and the proximity to the cervical nerve root. Therefore, the needle should be visualized right after insertion to avoid inadvertent nerve root contact. Because the space is confined, 1–2 mL of local anesthetic is sufficient.

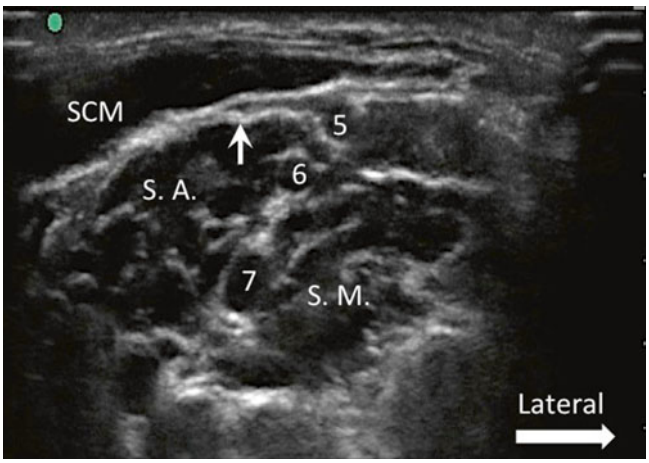


Fig. 9.5 Sonography of the phrenic nerve. The nerve is small and is usually hypoechoic (*arrow*). *SCM* sternocleidomastoid muscle, *SA* scalenus anterior muscle, *SM* scalenus medius muscle. Cervical nerve roots 5–7 are numbered accordingly as *5–7* (Reproduced with permission from Philip Peng Educational Series)

Effects of the Blockade

Paralysis of diaphragm movement.

Dosage

Three to five milliliters local anesthetic—e.g., prilocaine 1 %. For repeat blockades, ropivacaine 0.375 % and bupivacaine 0.125–0.375 % (levobupivacaine). A contralateral blockade must only be carried out after an interval of approximately 30–60 min.

Complications

1. Intravascular injection, with toxic reactions (cf. Chap. 1)
2. Pneumothorax

Side Effects

1. Ipsilateral paralysis of diaphragmatic movement
2. Partial blockade of the stellate ganglion (Horner syndrome)
3. Simultaneous anesthesia of the recurrent laryngeal nerve (hoarseness and a foreign-body sensation in the neck)

Suggested Reading

1. Brown DL. Atlas of regional anesthesia. 3rd ed. Philadelphia: Saunders Elsevier; 2006.
2. Frotcher M. Kahle W. Nervensystem und Sinnesorgane. In: Taschenatlas der Anatomie, Band 3. Stuttgart/New York: Thieme; 2001.
3. Grehl H, Reinhardt F, Neuberger J. Checkliste Neurologie, 2. Aufl. Stuttgart/New York: Thieme; 2002.
4. Hahn MB, McQuillan PM, Sheplock GJ. Regional anesthesia. St. Louis: Mosby; 1996.
5. Jankovic D. Blockade des Nervus phrenicus. In: Jankovic D, editor. Regionalblockaden & Infiltrationstherapy, 4. Auflage. Berlin: ABW-Wissenschaftsverlag; 2007. p. 59–61.
6. Moore DC. Regional block. 4th ed. Springfield: Charles Thomas; 1981.
7. Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 4th ed. Philadelphia/New York: Lippincott-Raven; 1998.

Chapter 10

Trigeminal Nerve

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Ban C.H. Tsui, MSc, MD, FRCPC (Ultrasound Section)
Department of Anesthesiology and Pain Medicine, University of Alberta, Edmonton, AB, Canada
University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada
e-mail: btsui@ualberta.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_10,

Anatomy

The trigeminal nerve, the largest of the cranial nerves, exits from the pons with a small motor root (the portio minor) and a large sensory root (portio major).

In the semilunar cavity of the dura mater, the sensory root expands to become the trigeminal ganglion (semilunar ganglion). The motor root runs along the medial side of the ganglion to the mandibular nerve. The trigeminal ganglion lies on the dorsal surface of the petrous bone. The three main branches originate from its anterior margin (Fig. 10.1): the ophthalmic nerve, maxillary nerve, and mandibular nerve [9–11, 13].

Ophthalmic Nerve (Figs. 10.1 and 10.2)

The optic branch is purely sensory and passes lateral to the cavernous sinus and abducent nerve to the superior orbital fissure. It draws sympathetic fibers from the internal carotid plexus and in turn gives off sensory fibers to the oculomotor nerve, trochlear nerve, and abducent nerve. Before entering the fissure, the ophthalmic nerve branches into the lacrimal nerve, nasociliary nerve, and frontal nerve.

The frontal nerve runs along the levator palpebrae superioris muscle to behind the center of the orbital cavity. There it divides into the supraorbital nerve, which passes to the supraorbital notch, and the supratrochlear nerve, which runs in a medial direction toward the trochlea. The branches of the supratrochlear nerve supply the upper eyelid, the root of the nose, and the adjoining skin of the forehead (upper end branch), as well as the skin and conjunctiva of the medial canthus (lower end branch).

Maxillary Nerve (Figs. 10.1 and 10.2)

The second branch of the trigeminal nerve is also purely sensory. It emerges from the skull through the round foramen and enters the pterygopalatine fossa.

From here, it gives off the zygomatic nerve to the orbit and the pterygopalatine nerves—two very short nerves that connect with the pterygopalatine (sphenopalatine) ganglion (Fig. 10.12). As a continuation of its trunk, the infraorbital nerve penetrates through the inferior orbital fissure to the base of the orbit, to the infraorbital groove and infraorbital canal. After passing through the infraorbital foramen, it reaches the facial surface of the maxilla. Here it divides into three groups of branches, which supply the side of the nose, the lower eyelid, and the upper lip.

Mandibular Nerve (Figs. 10.1 and 10.2)

As the largest branch of the trigeminal nerve, the mandibular nerve contains the sensory parts of the trigeminal ganglion and takes up the motor root of the trigeminal nerve. After passing through the oval foramen, the mandibular nerve forms a short, thick nerve trunk, on the medial side of which lies the otic ganglion. In its later course, the mandibular nerve divides into an anterior trunk with mainly motor fibers and a posterior trunk with nerve branches and end fibers mainly consisting of sensory fibers. The most important nerves and areas of supply in the posterior trunk are:

- Mental nerve (skin and mucosa of the lower lip and chin)
- Inferior alveolar nerve (molar and premolar teeth of the mandible)
- Lingual nerve (floor of the mouth, mucosa of the anterior two thirds of the tongue)
- Auriculotemporal nerve (ear, skin, and fascia of the temple)

The sensory branch of the anterior trunk, the buccal nerve, supplies the skin and mucosa in the area of the buccinator muscle.

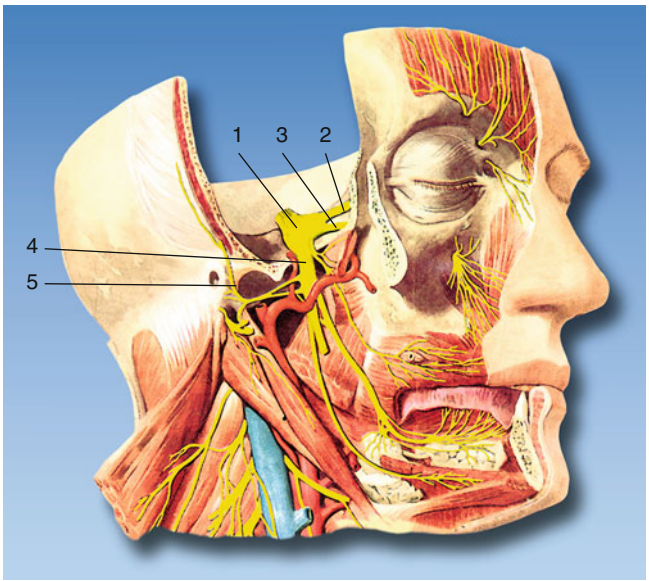


Fig. 10.1 Sensory supply to the face. (*1*) Trigeminal ganglion, (*2*) ophthalmic nerve, (*3*) maxillary nerve, (*4*) mandibular nerve, and (*5*) auriculotemporal nerve (With permission from Danilo Jankovic)

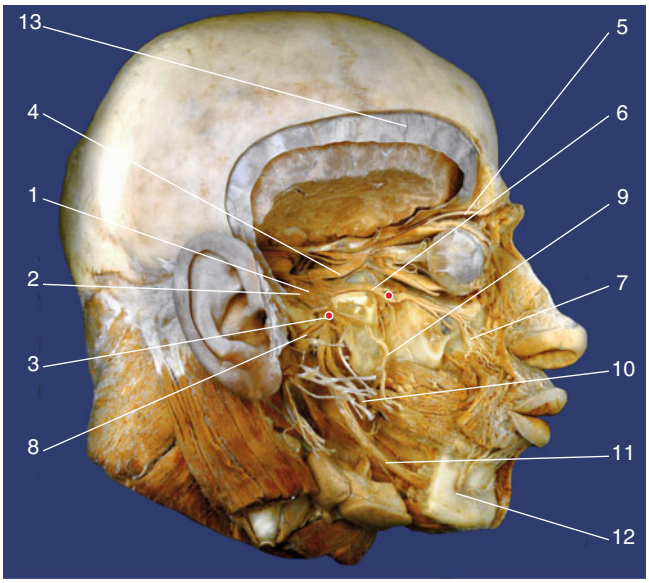


Fig. 10.2 Trigeminal nerve. (*1*) Trigeminal (Gasserian, semilunar) ganglion, (*2*) mandibular nerve in the oval foramen, (*3*) mandibular nerve and otic ganglion (*red point*), (*4*) ophthalmic nerve, (*5*) frontal nerve, (*6*) maxillary nerve and pterygopalatine ganglion, (*7*) infraorbital nerve,(*8*) auriculotemporal nerve,(*9*) buccal nerve, (*10*) lingual nerve, (*11*) alveolar inferior nerve, (*12*) mental nerve, (*13*) dura mater (With permission from Danilo Jankovic)

Peripheral Branches of the Trigeminal Nerve [2, 4, 7, 9–11, 13]

The supraorbital foramen, infraorbital foramen, and mental foramen lie on a single line running about 2.5 cm lateral to the midfacial line and passing through the pupil (Fig. 10.3).

Block of Ophthalmic Nerve Branches, Supraorbital and Supratrochlear Nerves

The end branches of these two nerves provide the sensory supply for the skin of the forehead, the top of the nose, and the skin and conjunctiva of the medial canthus (Figs. 10.4 and 10.30).

Indications

Diagnostic

- Differential diagnosis of hyperalgesic zones, e.g., the frontal part of the occipitofrontalis muscle

Therapeutic

- Trigeminal neuralgia of the first branch and postherpetic neuralgia
- Postoperative and post-traumatic pain
- Minor surgical interventions (note higher doses) along the surface of the innervated area, e.g., removal of cysts and atheromas, wound care

Specific Contraindications

None.

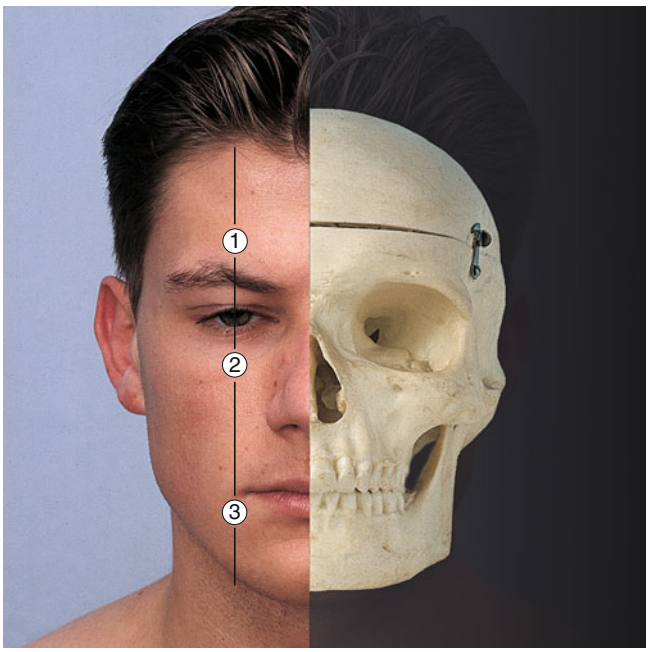


Fig. 10.3 The supraorbital foramen (*1*), infraorbital foramen (*2*), and mental foramen (*3*) lie on a single line running about 2.5 cm lateral to the midfacial line and passing through the pupil (With permission from Danilo Jankovic)

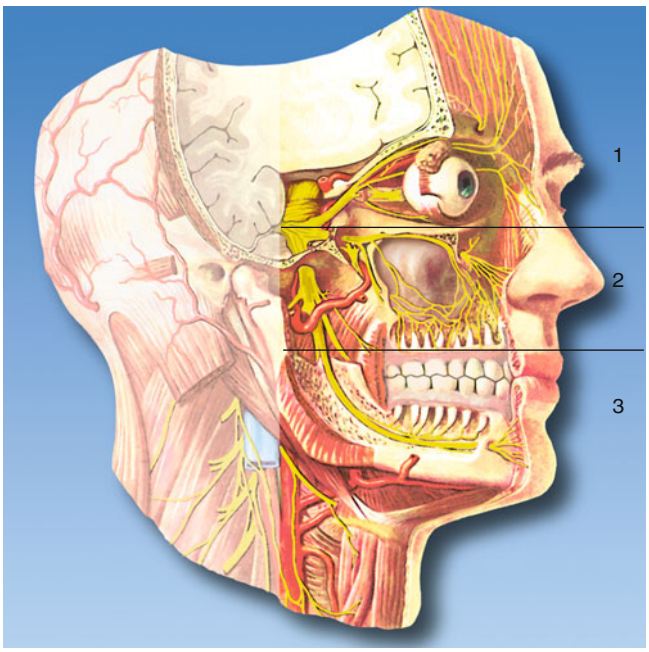


Fig. 10.4 (*1*) Supraorbital and supratrochlear nerves, (*2*) infraorbital nerve, (*3*) mental nerve (With permission from Danilo Jankovic)

Procedure

Preparations
Check that the emergency equipment is complete and in working order. Sterile precautions.

Materials
2-mL syringes, fine 26-G needles (25 mm), disinfectant, and swabs for compression.

Skin Prep
For all blocks.

Patient Positioning
Supine.

Landmarks
Supraorbital foramen and upper angle of the orbit.

Supraorbital nerve: palpation of the supraorbital foramen at the orbital margin.

Supratrochlear nerve: palpation of the upper angle of the orbit on the medial side of the root of the nose.

Injection Techniques
Supraorbital Nerve

After palpation of the supraorbital foramen, a swab is laid on the eyelid to prevent uncontrolled spread of the local anesthetic. The needle is introduced as far as the supraorbital foramen (bone contact), slightly withdrawn, and after aspiration the injection is carried out slowly (Figs. 10.3 and 10.5).

Supratrochlear Nerve

After palpation of the upper angle of the orbit, a swab is laid on the eyelid to prevent uncontrolled spread of the local anesthetic. The needle is introduced at the upper internal angle of the orbit (Fig. 10.6) and minimally withdrawn after bone contact. Slow injection of the local anesthetic follows after careful aspiration.

> It is not necessary to elicit paresthesias. Look for bone contact, withdraw the needle slightly, aspirate, and inject.

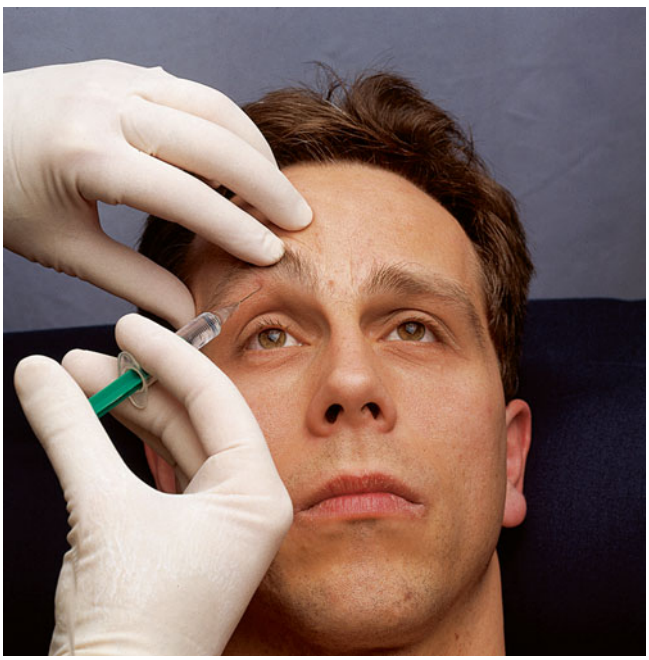


Fig. 10.5 Anesthetizing the supraorbital nerve (With permission from Danilo Jankovic)

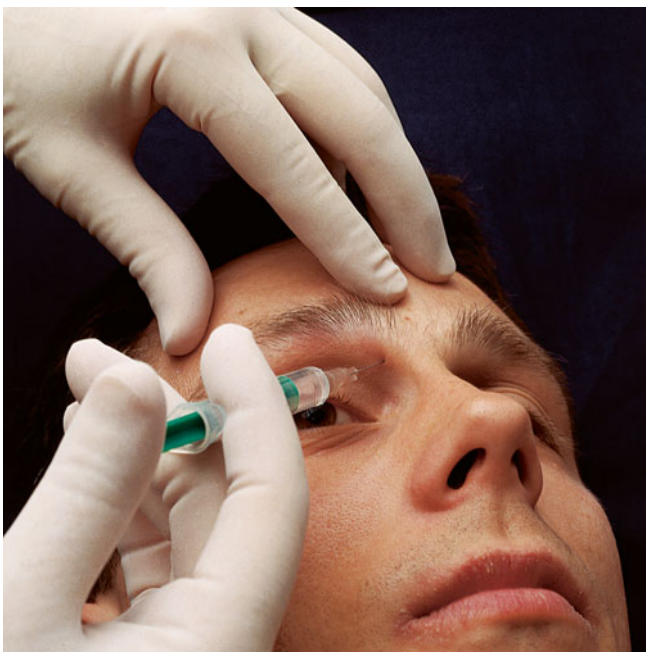


Fig. 10.6 Anesthetizing the supratrochlear nerve (With permission from Danilo Jankovic)

Dosage

Diagnostic

0.5–1 mL local anesthetic, e.g., 0.5–1 % prilocaine, mepivacaine, and lidocaine.

Therapeutic

0.5–1 mL local anesthetic, e.g., 0.5–0.75 % ropivacaine and 0.25–0.5 % bupivacaine.

Surgical

Up to 5 mL local anesthetic.

Shorter procedures: e.g., 1 % prilocaine or 1 % mepivacaine.

Longer procedures: 0.75 % ropivacaine or 0.5 % bupivacaine (0.5 % levobupivacaine).

After the injection, carry out thorough compression (massaging in) to prevent hematoma formation and to encourage the local anesthetic to spread.

Side Effects

Possible hematoma formation (prophylactic compression).

Complications

Risk of blood vessel and nerve damage with injections into the foramina and bone channels.

No injections should be made into the supraorbital foramen due to the risk of nerve injury.

Block of Branches of Maxillary Nerve, Block of the Infraorbital Nerve

The infraorbital nerve, the end branch of the maxillary nerve, emerges about 1 cm below the middle of the lower orbital margin through the infraorbital foramen (Figs. 10.3, 10.4, and 10.32).

Indications

Diagnostic

- Differential diagnosis of trigger zones

Therapeutic

- Trigeminal neuralgia in the second branch and postherpetic pain
- Facial pain in the innervation area of the infraorbital nerve, post-traumatic pain, and pain after dental extraction
- Minor surgical procedures on the surface of the area of distribution (note higher dosages)

Specific Contraindications

None.

Procedure

Preparation and Materials (See Supraorbital Block)

Skin Prep

For all blocks.

Patient Positioning

Supine.

Landmarks

Infraorbital foramen and orbital margin (Figs. 10.3 and 10.4).

Extraoral Injection

Palpation of the infraorbital foramen, about 1 cm below the middle of the lower orbital margin.

Intraoral Injection

Palpation of the lower orbital margin.

Injection Techniques

Extraoral Injection

After palpating the infraorbital foramen, the needle is introduced cranially just below the palpation point until bone contact is made (Fig. 10.7) and then withdrawn slightly.

Intraoral Injection

The center of the lower orbital margin is palpated and marked with the middle finger. The upper lip is raised with a spatula or with the thumb and index finger. The needle is introduced above the second premolar tooth toward the infraorbital foramen, until bone contact is made, and then withdrawn slightly (Fig. 10.8).

For both of these techniques, it is important that slow injection of the local anesthetic should only be carried out after careful aspiration. Afterward, thorough compression should be carried out to prevent hematoma formation and to obtain better distribution of the local anesthetic.

No injections should be made into the infraorbital canal due to the risk of nerve injury.

Fig. 10.7 Extraoral technique for blocking the infraorbital nerve (With permission from Danilo Jankovic)

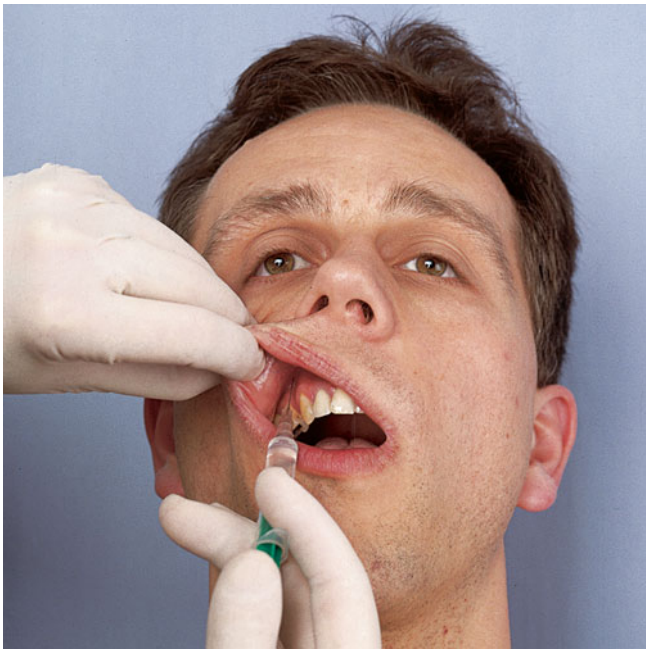


Fig. 10.8 Intraoral technique for blocking the infraorbital nerve (With permission from Danilo Jankovic)

Dosages

Diagnostic

0.5–1 mL local anesthetic, e.g., 0.5–1 % prilocaine, mepivacaine, and lidocaine.

Therapeutic (Extraoral Technique)

0.5–1 mL local anesthetic, e.g., 0.5–0.75 % ropivacaine and 0.5 % bupivacaine (0.5 % levobupivacaine).

Surgical

Up to 5 mL local anesthetic extraorally.

Shorter procedures: 1 % prilocaine or 1 % mepivacaine. Longer procedures: 0.75 % ropivacaine or 0.5 % bupivacaine (0.5 % levobupivacaine).

Intraorally: 2–3 mL local anesthetic.

Side Effects

Potential hematoma formation (prophylactic compression).

If the needle is advanced too far, penetration of the orbit can occur.

Symptom: temporary double vision.

Complications

Injection into the bone canal carries a risk of nerve damage.

Block of Branches of the Mandibular Nerve, Block of the Mental Nerve

The mental nerve, the sensory end branch of the mandibular nerve, emerges from the mental foramen at the level of the second premolar (Figs. 10.3 and 10.9).

It provides the sensory supply of the skin and mucosa of the lower lip and chin (Fig. 10.33).

Indications

Diagnostic

- Differential diagnosis of trigger points and hyperalgesic zones

Therapeutic

- Trigeminal neuralgia of the third branch
- Post-traumatic pain and pain in the innervation area of the mental nerve
- Dental treatment of the canine tooth and first premolars and incisors of the lower jaw
- Postdental extraction pain (intraoral technique)
- Surgical procedures on the surface of the lower lip (note higher dosages)

Specific Contraindications

None.

Procedure

Preparation and Materials (See Supraorbital Nerve Block)

Skin Prep

In all blocks.

Patient Positioning

Supine.

Landmarks

Mental foramen (Figs. 10.3 and 10.4).

Extraoral and intraoral injection

Palpation of the mental foramen at the level of the second premolar.

Injection Techniques

Extraoral Injection

After palpation of the mental foramen, the needle is inserted about 2.5 cm lateral to the midline until bone contact is made (Fig. 10.9).

Intraoral Injection

After palpation of the mental foramen, the lower lip is pressed downward using a spatula. The needle is inserted between the first and second premolars, into the lower reflection of the oral vestibule, in the direction of the neurovascular bundle (Fig. 10.10).

> For both of these techniques, it is important that slow injection should only be carried out after careful aspiration. Afterward, thorough compression should be carried out to prevent hematoma formation and to obtain better distribution of the local anesthetic.

Dosage

Diagnostic

0.5–1 mL local anesthetic, e.g., 0.5–1 % prilocaine, mepivacaine, and lidocaine.

Therapeutic (Extraoral Technique)

0.5–1 mL local anesthetic, e.g., 0.5–0.75 % ropivacaine and 0.5 % bupivacaine (0.5 % levobupivacaine).

Surgical

Up to 5 mL local anesthetic extraorally.

Shorter procedures: 1 % prilocaine or 1 % mepivacaine.

Longer procedures: 0.75 % ropivacaine or 0.5 % bupivacaine (0.5 % levobupivacaine).

Intraorally: 2–3 mL local anesthetic.

Side Effects

Potential hematoma formation (prophylactic compression).

Complications

Injection into the bone canal carries a risk of nerve damage.

Injections should never be made into the mental canal, due to the risk of nerve injury.

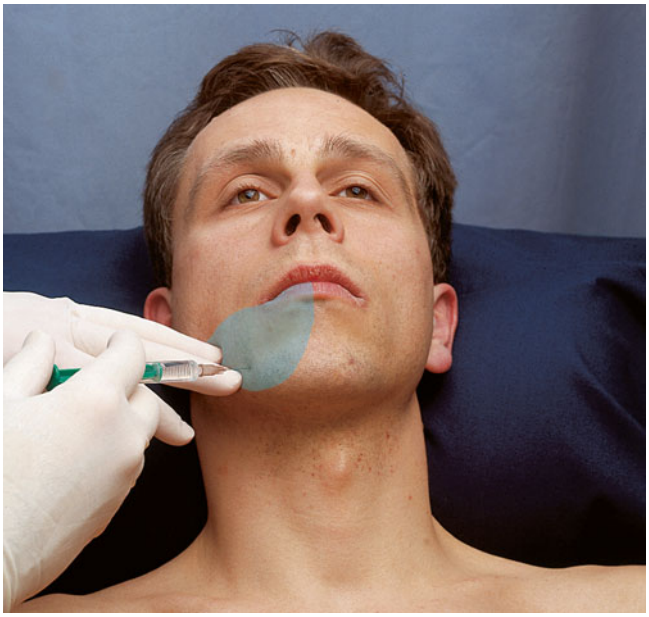


Fig. 10.9 Extraoral technique for blocking the mental nerve (With permission from Danilo Jankovic)

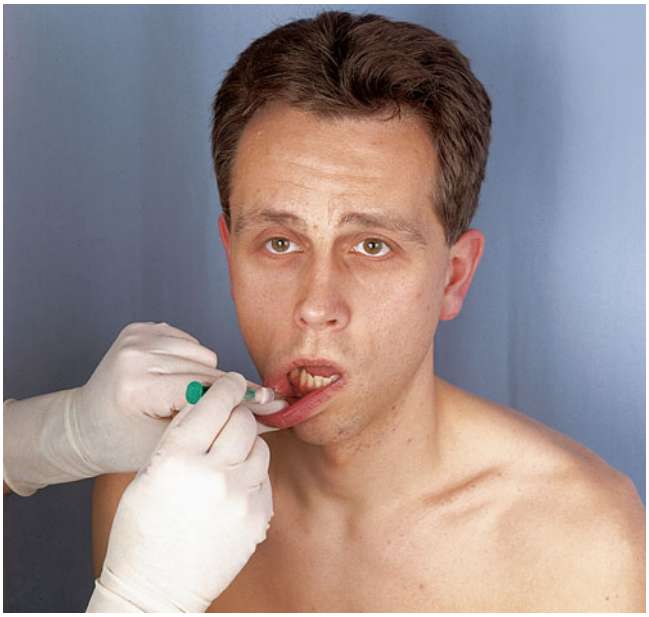


Fig. 10.10 Intraoral technique for blocking the mental nerve (With permission from Danilo Jankovic)

Ultrasound Imaging for Peripheral Branches of the Trigeminal Nerve

Ban C.H. Tsui

Ultrasound imaging is a common, noninvasive modality to facilitate many regional nerve blocks and to visualize nerves and surrounding soft tissue. However, there is limited available literature describing the use of ultrasound for superficial and deep trigeminal nerve block. This is because the most comprehensive deep trigeminal nerve blocks target the central ganglion. Because the ganglion is deep, blockade is often performed under fluoroscopic guidance or computerized tomography.

While blockade of deep structures is inaccessible to ultrasound, superficial branches of the trigeminal nerve (i.e., supraorbital, infraorbital, and mental nerves) are too small to visualize with most common ultrasound machines. In fact, the superficial branches can be performed easily by palpating their respective foramina. Although these bony foramina are usually easy to palpate physically, they can also be identified by observing a disruption or discontinuity within the bony hyperechoic line when imaging with ultrasound [22], as described below. Due to the superficial nature of these blocks, most experts find it more practical to use ultrasound imaging for landmarking the foramina rather than for real-time guidance.

Scanning Technique

1. As described in the previous section, all three foramina are typically located approximately 2.5 cm lateral to the midfacial line passing through the pupil in the sagittal plane on each side of the face.
2. Using a high-frequency linear transducer, the foramina can be identified dynamically by subtle movement of the probe position after placing it in the usual vicinity of the foramina; this technique will allow the user to capture breaks in the linear hyperechogenicity of the bony surface caused by the foramina:
   (a) To locate the supraorbital landmark, the probe is positioned transversely above the roof of the orbital rim and in alignment with the pupil. The bone is scanned slowly in a cephalad-to-caudad direction to identify the supraorbital foramen.
   (b) The infraorbital foramen can be located by scanning in a sagittal plane from medial to lateral along the lower orbital margin or inferior rim of the orbit. This foramen is usually located about 1 cm below the middle of the lower orbital margin.
   (c) The mental foramen can be localized using a transverse plane and scanning in a cephalad direction from the inferior border of the mandible. The nerve typically lies inferior to the outer lip at the level of the second premolar, midway between the upper and lower borders of the mandible.

As shown in Fig. 10.11, the image of the bony surface will change from a definite linear border to a border with a hypoechoic break caused by the foramen during dynamic scanning. It is important to note that the location of each orifice is associated with its respective blood vessels, which can be illuminated using color Doppler. However, the relatively small size of the arteries upon exiting the foramen renders them difficult to identify.

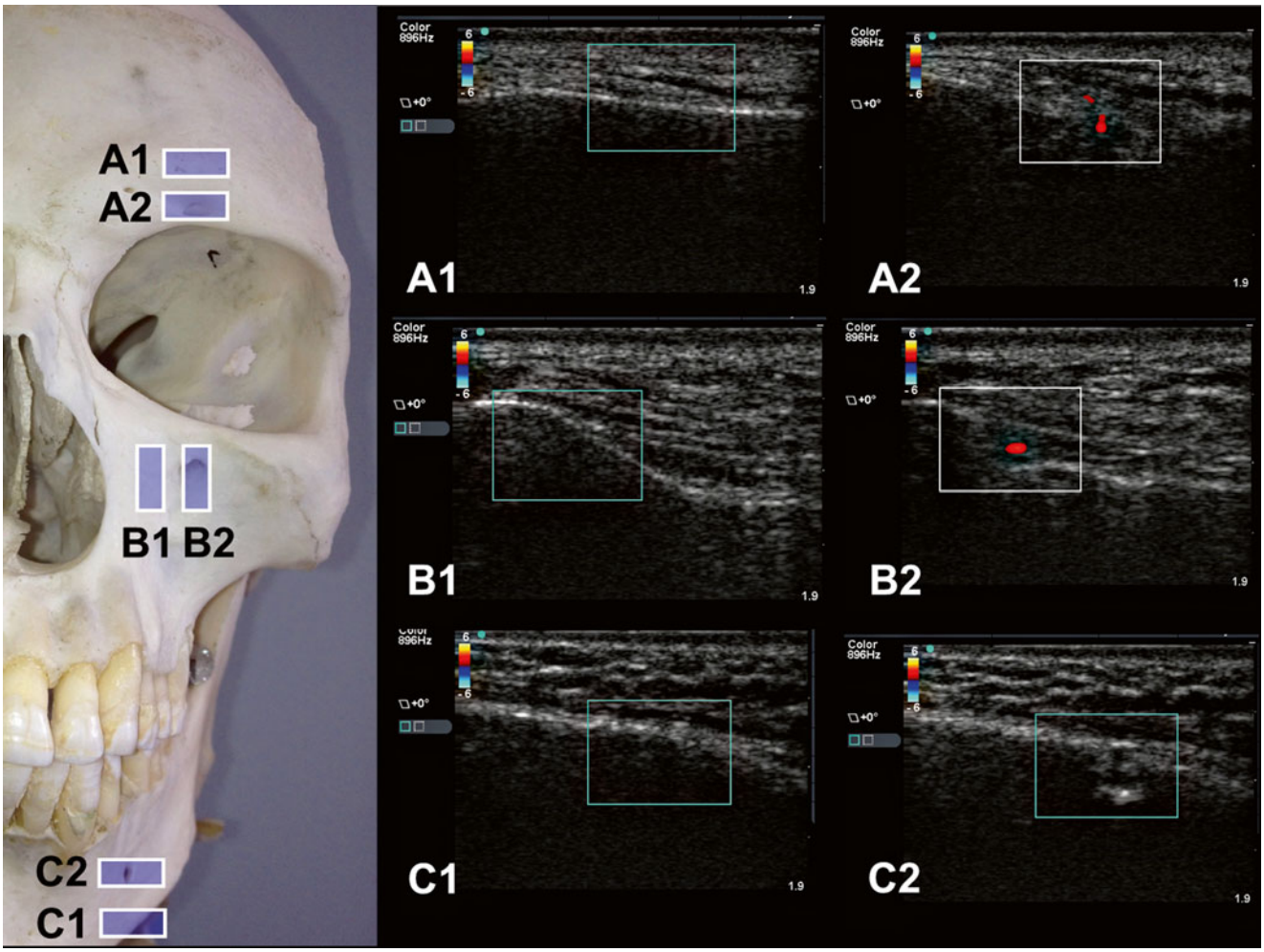


Fig. 10.11 *Left*, ultrasound scanning locations to identify supraorbital (*A1*, *A2*), infraorbital (*B1*, *B2*), and mental (*C1*, *C2*) foramina. *A1*, *B1*, and *C1* represent starting position of probe; *A2*, *B2*, and *C2* represent position of probe over the foramen. *Right*, ultrasound images corresponding to probe positions. Doppler imaging (far right) allows identification of supraorbital and infraorbital arteries (With permission from Ban Tsui)

Trigeminal Nerve: Deep Block

Maxillary Nerve, Block of the Main Trunk in the Pterygopalatine (Pterygomaxillary) Fossa

The maxillary nerve emerges from the skull through the round foramen. It connects with the pterygopalatine (sphenopalatine) ganglion in the pterygopalatine fossa (Figs. 10.12, 10.13, and 10.14). The nerve and ganglion are responsible for sensory and autonomic supply to the central area of the face and head. The pterygopalatine (sphenopalatine) ganglion, which lies in the pterygopalatine fossa (sphenomaxillary fossa), is triangular in shape; extending to ca. 5 mm, it is the largest neuronal conglomerate outside of the brain. The ganglion has three types of nerve fiber and is connected to the trigeminal nerve via sensory fibers. It is linked to the facial nerve, internal carotid plexus, and superior cervical ganglion via sympathetic fibers; the motor fibers have parasympathetic (visceromotor) connections. There is also direct contact between the anterior horn of the spinal cord and the neurohumoral axis (adenohypophysis) [15, 20, 21].

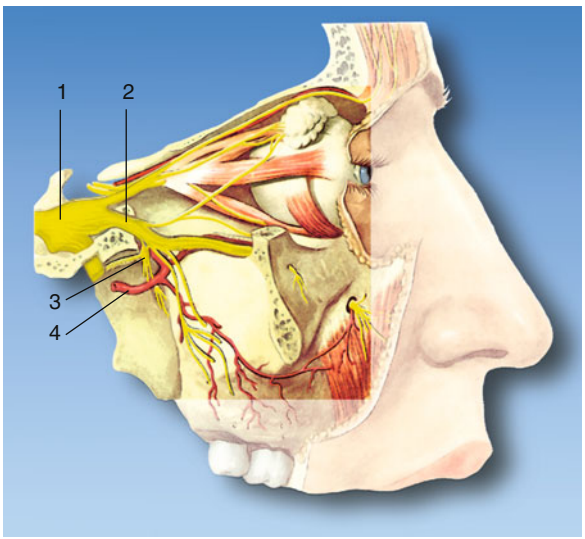


Fig. 10.12 (*1*) Trigeminal ganglion (Gasserian ganglion) and (*2*) pterygopalatine fossa with the maxillary nerve, (*3*) pterygopalatine ganglion, and (*4*) maxillary artery (With permission from Danilo Jankovic)

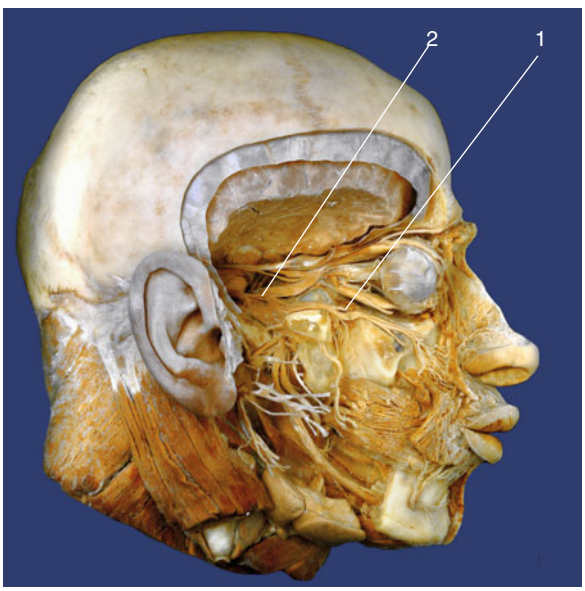


Fig. 10.13 (*1*) Maxillary nerve and pterygopalatine ganglion, (*2*) trigeminal ganglion (Gasserian ganglion) (With permission from Danilo Jankovic)

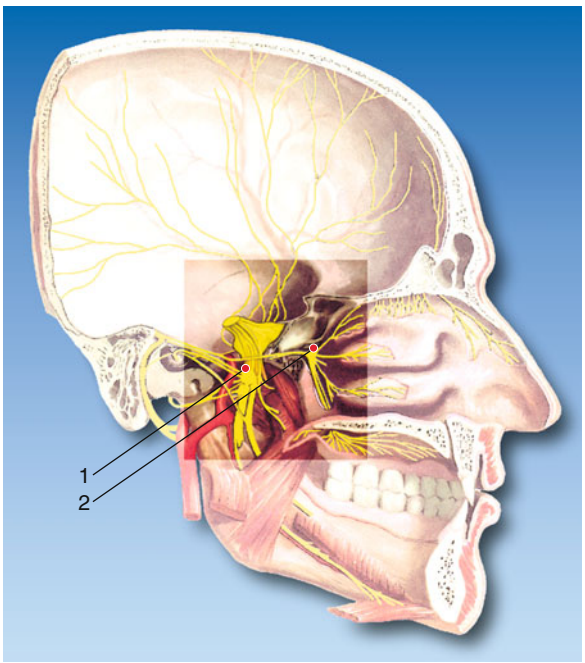


Fig. 10.14 Nerves and ganglia in the vicinity: (*1*) Otic ganglion, (*2*) pterygopalatine ganglion (With permission from Danilo Jankovic)

Indications

Diagnostic

- Differential diagnosis of facial pain

Therapeutic

- Trigeminal neuralgia in the second branch, postherpetic neuralgia
- Cluster headache [6], histamine headache, Sluder's neuralgia [19]
- Facial pain in the area of supply
- Pain in the eye region (iritis, keratitis, corneal ulcer), root of the nose, upper jaw, and gums
- Postoperative pain in the area of the maxillary sinus and teeth
- Pain after dental extraction

Neural Therapy

- Hay fever, vasomotor rhinitis
- Diseases of the oral mucosa
- Localized paresthesias

Specific Contraindications

Bleeding diathesis and anticoagulation treatment.

Procedure

These blocks should only be carried out only with appropriate experience. It is absolutely necessary to have a detailed discussion with the patient before the procedure.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions. Intravenous access.

Materials

2-mL syringe and 22-G needle (40 mm) for the intraoral technique and 5-mL and 10-mL syringe and 23-G needle (60 mm) for the extraoral technique. Disinfectant, spatula for the intraoral technique, compresses, cooling element available, and emergency drugs.

Skin Prep

In all blocks.

Intraoral Technique

Patient Positioning

The patient should be sitting, leaning back slightly and with the head tilted back.

Landmarks

Posterior edge of the upper seventh tooth (second maxillary molar) (Fig. 10.15).

Injection Technique

Using a 22-G needle (40 mm), the puncture is made medial to the posterior edge of the upper seventh tooth (second maxillary molar) through the greater palatine foramen. The needle is introduced at an angle of about 60°. The vicinity of the ganglion is reached at a depth of 3.5–4 cm. The greater palatine canal is about 3.4 cm long in adults. After careful aspiration at various levels, the local anesthetic is injected (Fig. 10.16).

Intraoral access is associated with fewer complications.

Dosage

Therapeutic

Intraorally: 1–2 mL local anesthetic, e.g., 0.75 % ropivacaine and 0.5 % bupivacaine (0.5 % levobupivacaine).

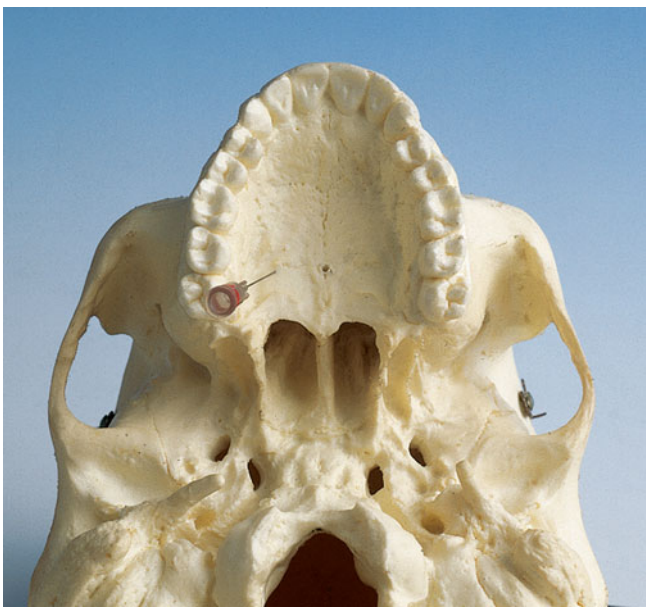


Fig. 10.15 Intraoral technique: orientation (With permission from Danilo Jankovic)

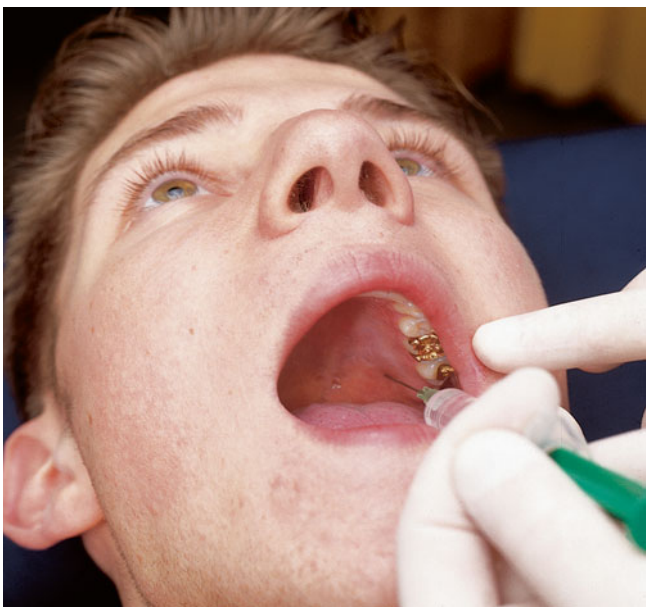


Fig. 10.16 Intraoral block of the pterygopalatine ganglion (With permission from Danilo Jankovic)

Extraoral Technique

Above the Zygomatic Arch (Suprazygomatic Technique)

Injection above the zygomatic arch is more comfortable for the patient.

Patent Positioning

Sitting, with face to the side and with the mouth slightly opened. Alternative: supine.

Landmarks

Center of the upper margin of the zygomatic arch.

Injection Technique

A skin injection is made directly above the middle of the zygomatic arch. A 6-cm-long needle is introduced at an angle of ca. 45° in the direction of the pterygopalatine fossa (contralateral molar teeth) (Fig. 10.17). After paresthesias have been elicited in the area of the nostril, the upper lip, and the cheek, the needle is withdrawn slightly and aspirated carefully at various levels, and the local anesthetic is administered slowly in several small doses. Repeated aspiration at various levels must be carried out during this procedure.

Separate blocking of the maxillary nerve and pterygopalatine region is rarely possible with this method.

Fig. 10.17 Orientation for injections above the zygomatic arch (With permission from Danilo Jankovic)

Below the Zygomatic Arch (Infrazygomatic Technique)

Patient Positioning

Supine or sitting, face to the side with the mouth slightly open.

Landmarks

Mandibular fossa.

Injection Technique

The most important requirement for carrying out this block successfully is accurate location of the mandibular fossa between the condylar and coronoid processes of the mandible. It is helpful for the patient to open and close the mouth. After skin infiltration, a 6-cm needle is introduced at an angle of 45° in the direction of the back of the eyeball (Fig. 10.18). After ca. 4–4.5 cm, the lateral part of the pterygoid process is reached and the needle is withdrawn slightly and lowered into the pterygopalatine fossa (about 0.5 cm medial to the pterygoid). After the paresthesias described above have been elicited and after careful aspiration at various levels, the local anesthetic is carefully injected in several small doses. If pain occurs in the region of the orbit, the procedure should be stopped.

Dosage

Diagnostic

Up to 5 mL local anesthetic, e.g., 0.5 % prilocaine, mepivacaine, and lidocaine.

Fig. 10.18 Extraoral technique beneath the zygomatic arch (mandibular fossa) (With permission from Danilo Jankovic)

Therapeutic

Extraorally: 5–10 mL local anesthetic, e.g., 0.5 % ropivacaine and 0.25 % bupivacaine (0.25 % levobupivacaine).

In acute conditions, with 1–2 mg dexamethasone added.

Surgical

Extraorally: 5–10 mL local anesthetic, e.g., 0.75 % ropivacaine, 0.5 % bupivacaine (0.5 % levobupivacaine), 1 % prilocaine, and 1 % mepivacaine.

Block series.

A sequence of six to eight blocks is recommended for the extraoral technique.

Side Effects

- Transient visual weakness (extremely rare).
- Horner's syndrome, extremely rare and usually with high doses. There are connections with the superior cervical ganglion via the pterygoid canal, deep petrosal nerve, and greater superficial petrosal nerve.
- Hematoma in the cheek or orbital cavity due to blood vessel puncture. Immediate outpatient treatment: alternating ice pack and heparin ointment, depending on the spread of the hematoma, for ca. 1 h. This can be continued at home, with the patient also taking coated Reparil® tablets (sodium aescinate) if appropriate. Resorption of the hematoma, which is harmless but visually uncomfortable for the patient, occurs within 2 weeks at the most.

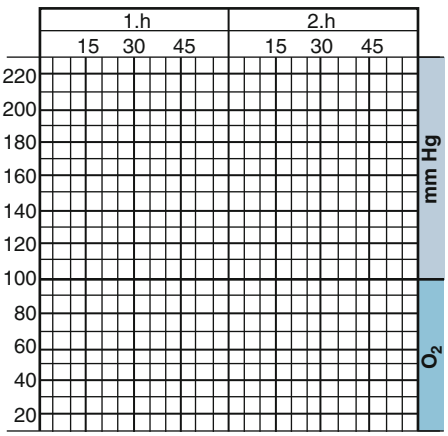
Complications

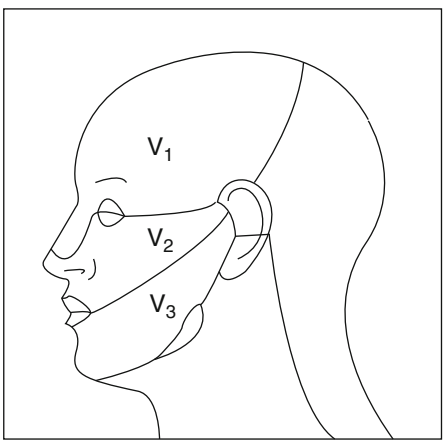
- Intravascular injection (maxillary artery and maxillary vein) (Fig. 10.12)
- Epidural or subarachnoid injection

Both of these complications are extremely rare. Immediate treatment: see Chap. 1, p. 8.

The maxillary artery and vein lie in the immediate vicinity.

Record and checklist





Maxillary nerve and pterygopalatine ganglion

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: ☐ *22 G* ☐ *40 mm long* ☐ *60 mm long*

i.v. access: ☐ *Yes* ☐ *No*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Sitting*

Approach: ☐ *Above the zygomatic arch* ☐ *Intraoral*

☐ *Below the zygomatic arch (mandibular fossa)*

Local anesthetic: _____ *ml* _____ *%* _____

Test dose: _____ *ml*

Addition to injection solution: ☐ *No* ☐ *Yes* _____

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Nerve region _____ _____ _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* ___ *°C left* ___*°C*

☐ *Numbness (V_2)*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____

Complications: ☐ *None*

☐ *Yes (hematoma, intravascular, injection, other)*

_____

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

With permission from Danilo Jankovic

Nasal Block of the Pterygopalatine Ganglion

Indications

Greenfield Sluder [19] drew attention to the significance of this ganglion as long ago as 1903. In 1918, he described a number of symptoms capable of being treated by injection or topical application of a local anesthetic or cocaine, with the associated anesthesia of the pterygopalatine ganglion: headache; pain in the eyes, mouth, or ears; lumbosacral pain; arthritis; glaucoma; and hypertension. Similar observations were reported by Ruskin [17], Byrd and Byrd [5], and Amster [1].

More recent studies [3, 8, 14, 16] have shown that nasal local anesthesia of the ganglion can be used with good success rates in the treatment of:

- Acute migraine
- Acute or chronic cluster headache
- Various types of facial neuralgia
- Tumor pain in the nasal and pharyngeal area [12, 18]

Specific Contraindications

None.

Procedure

Materials (Fig. 10.19)

2-mL syringe, plastic part of a plastic indwelling catheter (for self-administration in tumor pain), nasal speculum, and applicators (cotton buds).

Patient Positioning

Supine or sitting, with head tilted back.

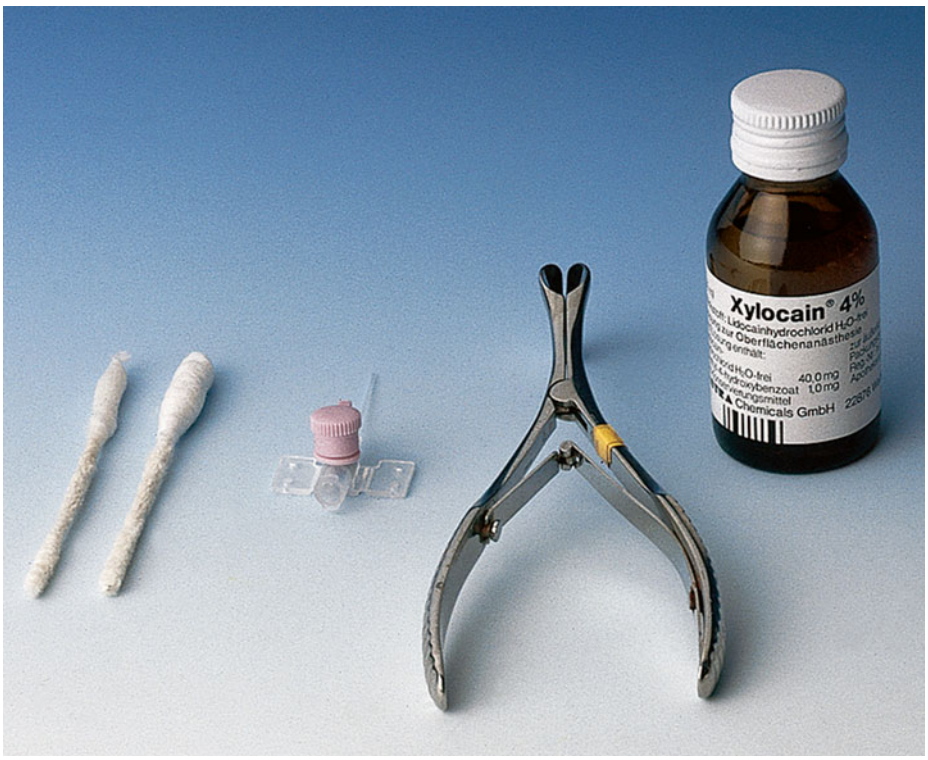


Fig. 10.19 Materials (With permission from Danilo Jankovic)

Application

An applicator, preferably a cotton bud, soaked in local anesthetic, e.g., 2 % lidocaine gel or a 4 % aqueous lidocaine solution, is carefully advanced along the inferior nasal concha as far as the posterior wall of the nasopharynx (Fig. 10.20) and left in place for 20–30 min (Fig. 10.21). In patients with cancer pain, the plastic part of a plastic indwelling catheter can be advanced as far as possible into the nasal cavity, and the local anesthetic, e.g., 0.5 % bupivacaine, can be instilled with a 2-mL syringe. The block can be carried out bilaterally.

> To prevent trauma, the applicator should not be advanced forcefully if resistance is encountered.

Dosage [12, 18]

Local anesthetics: 2 % lidocaine gel, 1.5–2 mL 4 % lidocaine (aqueous solution), or 1.5–2 mL bupivacaine. Disadvantage: the onset of effect is slightly slower.

10 % cocaine: at a dosage of 0.2–0.4 mL, there is no reason to fear adverse CNS effects [3]. Advantage: very fast onset of effect.

If the recommended doses are used, there is no difference between these substances with regard to effectiveness and resorption.

Block Series

In acute pain, one or two applications are recommended. In chronic conditions, one to three applications can be given over a period of up to 3 weeks.

In cancer pain, applications may be indicated three times per day over a longer period.

Side Effects

The method is not very invasive and has minimal side effects. Effects that may occur include a sense of pressure in the nose, sneezing, short-term lacrimation due to irritation of branches of the lacrimal gland, a bitter taste, and slight numbness in the oral and pharyngeal cavity.

Complications

Very occasionally, toxic effects may occur as a result of the absorption of the local anesthetic into very well-vascularized tumor tissue. In long-term treatments, erosions may sometimes lead to spinal absorption of the local anesthetic. To prevent this, periodic rinsing with a physiological saline solution can be carried out.

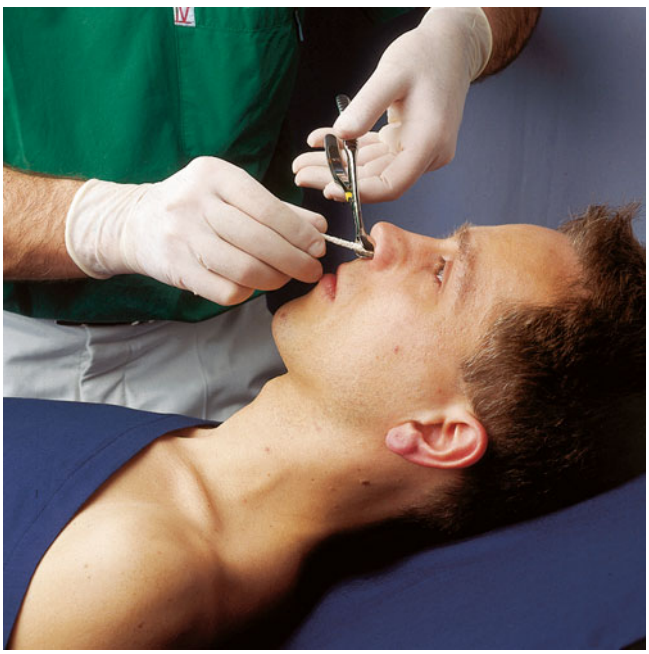


Fig. 10.20 Nasal application (With permission from Danilo Jankovic)

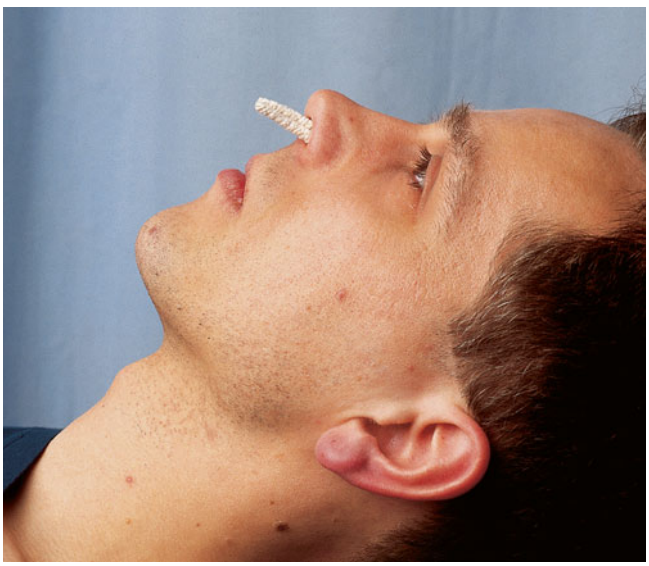


Fig. 10.21 The anesthetic should be allowed 20–30 min to take effect (With permission from Danilo Jankovic)

Mandibular Nerve, Block of the Mandibular Nerve and Otic Ganglion in the Infratemporal Fossa [2, 4, 7, 9, 11, 13]

After passing through the oval foramen, the mandibular nerve forms a short, thick nerve trunk, with the otic ganglion lying on the medial side of it. Its most important branches (Fig. 10.22) are the buccal nerve, lingual nerve, inferior alveolar nerve, mental nerve, and auriculotemporal nerve.

Indications

Diagnostic

- Differential diagnosis of trigeminal neuralgia (anterior two thirds of the tongue) and glossopharyngeal neuralgia (posterior third of the tongue)

Therapeutic

- Tinnitus (the otic ganglion has connections with the chorda tympani, the nerves of the pterygoid canal, and the medial pterygoid nerve)
- Trigeminal neuralgia in the third branch
- Trismus after dental extraction
- Dental surgery and maxillary surgery (higher dosages required)
- Temporomandibular joint dysfunction syndrome (in collaboration with an orthodontist), if infiltration of the trigger points of the temporalis muscle, lateral pterygoid muscle, and masseter muscle is unsuccessful

Specific Contraindications

Bleeding diathesis and anticoagulation treatment.

Procedure

This block should only be carried out only with appropriate experience. It is absolutely necessary to have a detailed discussion with the patient before the procedure.

Preparation and Materials (See Above)

Skin Prep

In all blocks.

Patient Positioning

Supine, with face to the side.

Landmarks

Mandibular fossa, zygomatic arch, and tragus (the needle insertion point lies ca. 2 cm laterally) (Fig. 10.23).

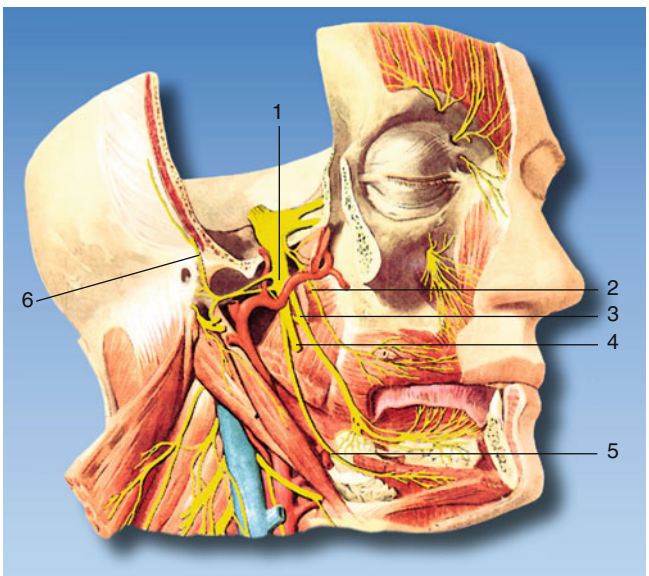


Fig. 10.22 Distribution areas of the (*1*) mandibular nerve, (*2*) buccal nerve, (*3*) lingual nerve, (*4*) inferior alveolar nerve, (*5*) mental nerve, (*6*) auriculotemporal nerve (With permission from Danilo Jankovic)

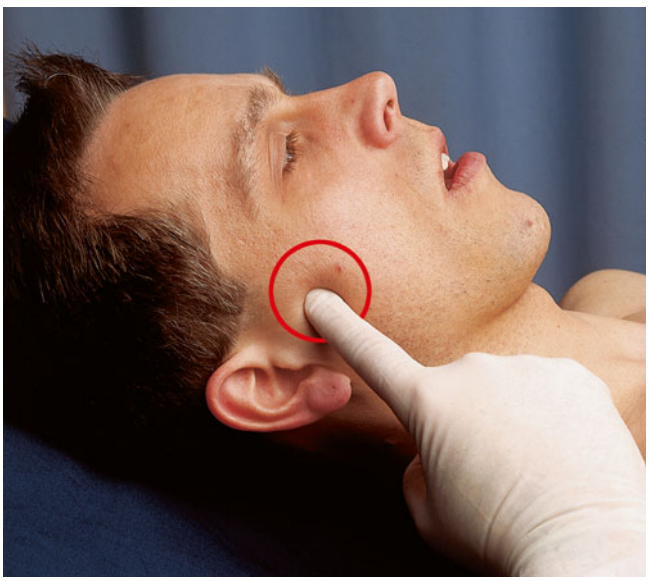


Fig. 10.23 The most important requirement is that the mandibular fossa should be identified precisely. It lies between the condylar process and the coronoid process of the mandible and is easiest to localize when the patient opens and closes his or her mouth (With permission from Danilo Jankovic)

Injection Technique

After skin infiltration, a 60-mm needle is introduced into the skin perpendicularly (Fig. 10.24). Paresthesias in the lower jaw region, lower lip, and lower incisors occur when the needle reaches a depth of ca. 4–4.5 cm. After paresthesias have clearly developed, the needle is withdrawn slightly and aspirated carefully at various levels, and the local anesthetic is slowly injected in several small doses. Aspiration should be repeated several times at different levels as this is done. There is a delayed onset of the desired effect in the area of the auriculotemporal nerve.

> If contact is made with the pterygoid process when the needle is being introduced, withdraw the needle 0.5–1 cm and redirect dorsally.

Distribution of the Block

The otic ganglion (Fig. 10.25), which lies directly under the oval foramen, is always anesthetized along with the nerve.

The area supplied by the mandibular nerve is shown in Fig. 10.33.

Dosage

Diagnostic

Up to 5 mL local anesthetic, e.g., 5 % prilocaine, mepivacaine, and lidocaine.

Therapeutic

5–10 mL local anesthetic, e.g., 0.5 % ropivacaine and 0.25 % bupivacaine (0.25 % levobupivacaine).

In acute conditions, with 1–2 mg dexamethasone added.

Surgical

10 mL local anesthetic, e.g., 0.75 % ropivacaine, 0.5 bupivacaine, 1 % prilocaine, and 1 % mepivacaine.

Block Series

A series of six to eight blocks is recommended. When there is evidence of symptomatic improvement, further blocks can also be carried out.

Side Effects

- Transient facial paralysis caused by injecting too superficially.
- Hematoma in the cheek due to vascular puncture. These harmless hematomas can take up to two weeks to resolve. Immediate treatment: see the section on blocks of the maxillary nerve and pterygopalatine ganglion.

Complications

- Intravascular injection (middle meningeal artery and maxillary artery).
- Epidural or subarachnoid injection. Immediate treatment: see Chap. 1, p. 8.

The middle meningeal artery and maxillary artery lie in the immediate vicinity.

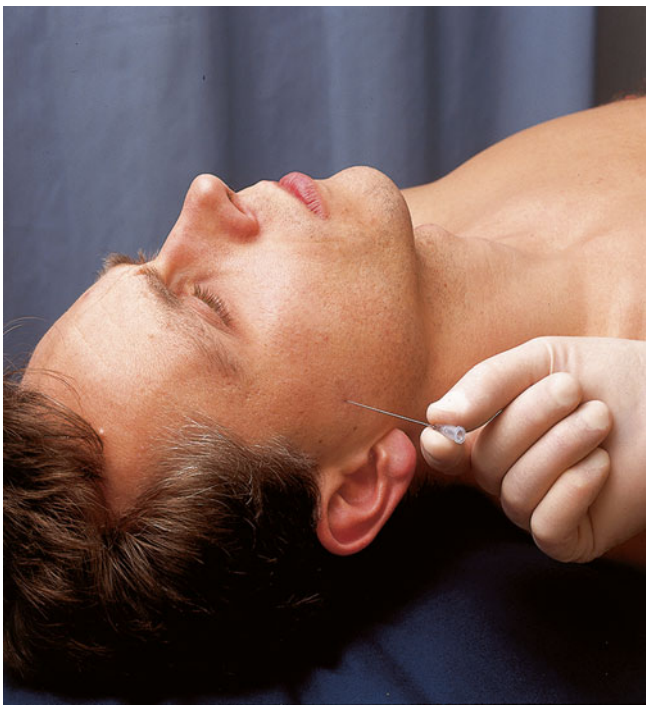


Fig. 10.24 Needle insertion technique: the needle is directed at an angle of 90° (With permission from Danilo Jankovic)

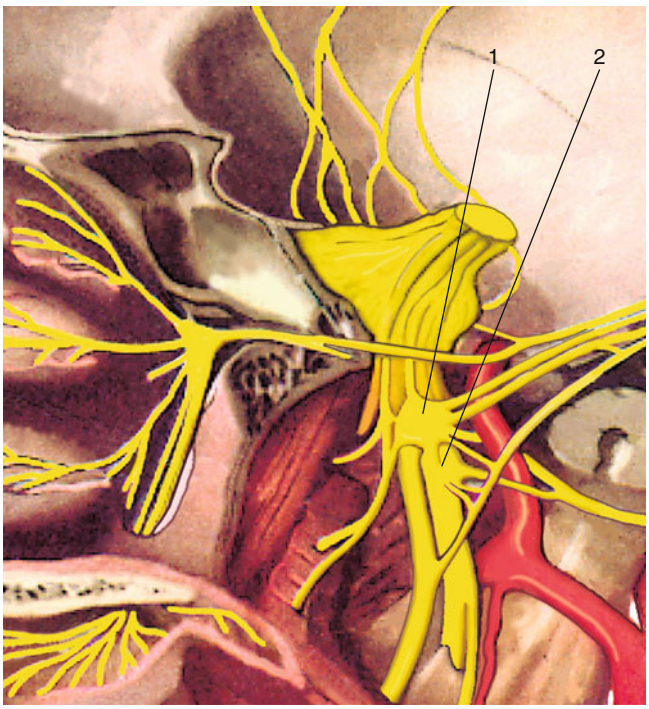


Fig. 10.25 There are close anatomical connections between the otic ganglion (*1*) and the mandibular nerve (*2*) (With permission from Danilo Jankovic)

Mandibular nerve and otic ganglion

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: ☐ *22 G* ☐ *50 mm long* ☐ *60 mm long*

i.v. access: ☐ *Yes* ☐ *No*

Monitoring: ☐ *ECG* ☐ *Pulse oxymetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs*): ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Sitting*

Approach: ☐ *Mandibular fossa*

Local anesthetic: _____ *ml* _____ *%* _____

Testdose: _____ *ml*

Addition to injection solution: ☐ *No* ☐ *Yes* _____

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Nerve region _____ _____ _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* ___ *°C left* ___ *°C*

☐ *Numbness (V_3)*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____

Complications: ☐ *None*

☐ *Yes (hematoma, intravascular, injection, other)*

_____

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

_____

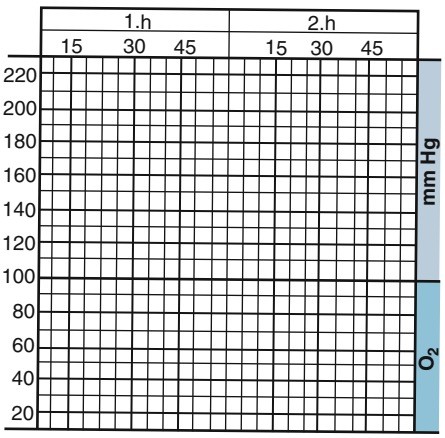
_____

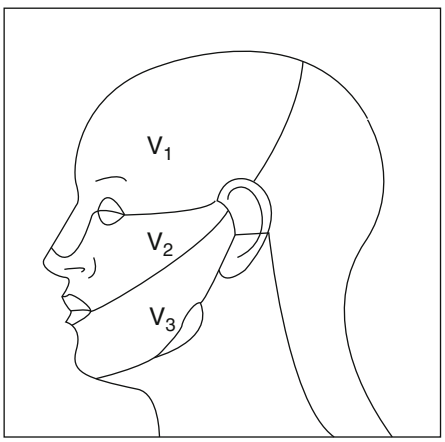
_____

_____

_____

Record and checklist





With permission from Danilo Jankovic

Gasserian Ganglion Block

The trigeminal ganglion (semilunar ganglion, Gasserian ganglion) lies on the dorsal surface of the petrous bone. The intracranial Gasserian ganglion lies medially in the middle cranial fossa; lateral to the cavernous sinus, internal carotid artery, and cranial nerves III–VI; and posterior and superior to the oval foramen, through which the mandibular nerve exits from the intracranial cavity (Figs. 10.1, 10.2, and 10.26). All of these structures can be injured when the ganglion is blocked. The average size of the ganglion is ca. 1–2 cm. Part of the ganglion (the posterior two thirds) is located within the trigeminal cave (Meckel cavity), a duplication of the dura that encloses the ganglion. The oval foramen is a channel ca. 5 mm long and its largest diameter is ca. 8 mm.

Indications

Local Anesthetics

Diagnostic, before neurodestructive procedures.

Neurodestructive Procedures

Neurodestructive methods—particularly radiofrequency lesions of the ganglion and more rarely glycerol rhizolysis, alcohol injection, corticosteroid injection, or balloon compression of the ganglion—are used in pain conditions that are unbearable and cannot be influenced using other conservative measures:

- Cancer pain
- Trigeminal neuralgia
- Cluster headache
- Pain in the eye region
- Postherpetic neuralgia

Specific Contraindications

Local infection, sepsis, hemorrhagic diathesis, anticoagulation treatment, and significantly increased intracranial pressure.

Procedure

This block should only be carried out by highly experienced specialists. A very good knowledge of anatomy, manual skill, radiographic guidance when conducting the procedure, and strictly aseptic conditions are required. It is necessary to have a detailed discussion with the patient before the procedure.

Premedication

This method is painful, and preoperative administration of 0.05 mg fentanyl is therefore recommended.

Preparations

The completeness and functioning of the emergency equipment should be checked. Sterile precautions. Intravenous access, ECG monitoring, ventilation facilities, and pulse oximetry.

Materials

A fine 22-G spinal needle 80 mm long, 2-mL and 5-mL syringes, disinfectant, sterile compresses, emergency medication, intubation kit, and cooling element should be ready in hand.

Skin Prep

In all blocks.

Patient Positioning

Supine; the head is raised with a cushion.

Landmarks (Fig. 10.27 a,b)

- Medial edge of the masseter muscle, ca. 3 cm lateral from the angle of the mouth at the level of the second molar tooth.
- Ipsilateral pupil.
- Center of the zygomatic arch and articular tubercle (external acoustic meatus).

The following should be noted during puncture:

- The operator should stand on the side on which the block is being carried out.
- Radiographic guidance for the puncture is indispensable.
- An intraoral location should be excluded after introduction of the needle (risk of contamination).
- There is a risk of perforating the dural cuff (subarachnoid injection).
- Frequent aspiration and fractionated injection of the smallest possible fractions (blood, CSF?).

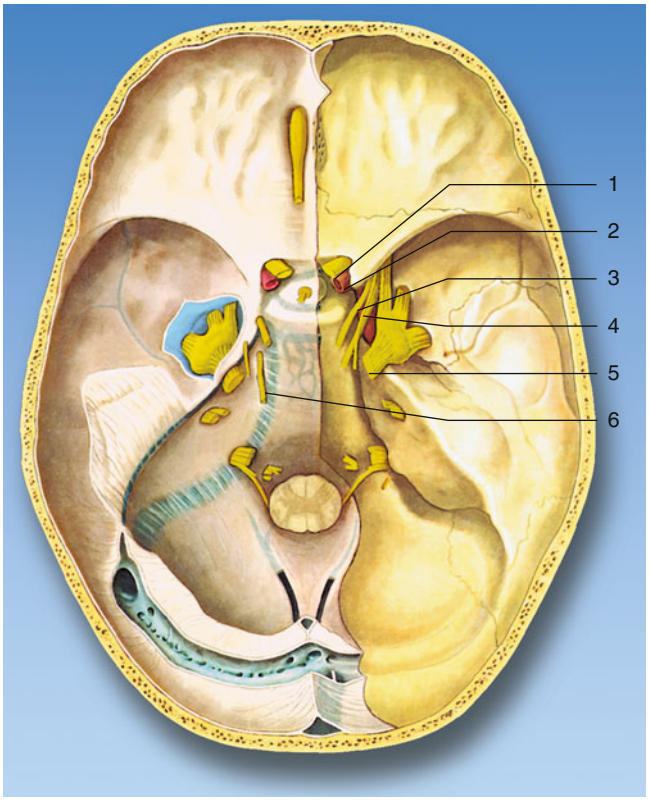


Fig. 10.26 The trigeminal ganglion and the neighboring cranial nerves and internal carotid artery. (*1*) Optic nerve, (2) internal carotid artery, (*3*) oculomotor nerve, (*4*) trochlear nerve, (*5*) trigeminal nerve, (*6*) abducent nerve (With permission from Danilo Jankovic)

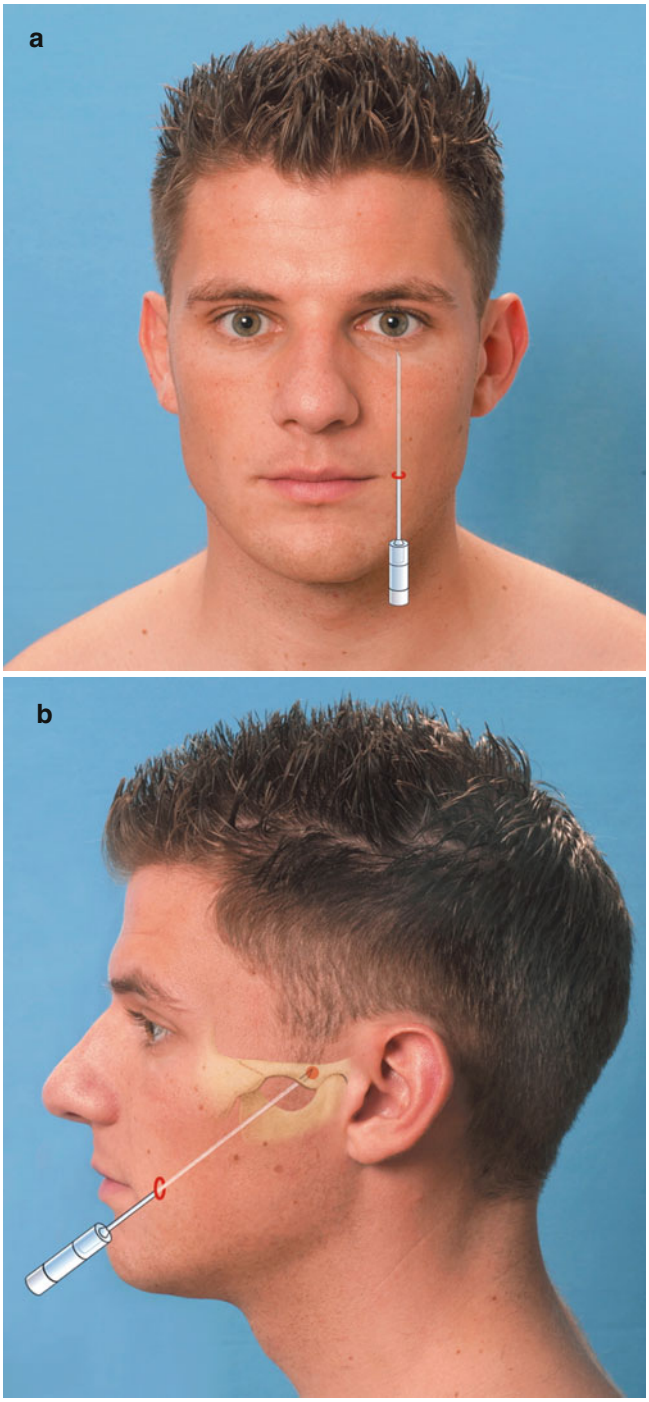


Fig. 10.27 (**a**, **b**) Landmarks: medial edge of the masseter muscle, ipsilateral pupil, center of the zygomatic arch (With permission from Danilo Jankovic)

Needle Insertion Technique [2, 4, 7, 9, 11, 13]

Local anesthesia at the needle insertion site is carried out ca. 3 cm from the angle of the mouth (medial edge of the masseter muscle). The patient is asked to gaze straight ahead and focus on a marked point on the wall. The needle should be directed toward the forward-gazing pupil when seen from the front and toward the articular tubercle of the zygomatic arch or external acoustic meatus when viewed from the side (Fig. 10.27).

The needle is then introduced at the level of the second molar tooth, through the previous skin injection in the direction indicated. An intraoral location of the needle must be excluded (risk of contamination). After 4.5–6 cm, bone contact should be made (infratemporal surface of the large wing of the sphenoid bone, directly in front of the upper boundary of the oval foramen) (Fig. 10.28). The needle is now withdrawn slightly, and the path to the oval foramen (ca. 1–1.5 cm away from the first bone contact) (Fig. 10.28) is probed millimeter by millimeter by advancing and withdrawing the needle. If the tip of the needle is located in the oval foramen, the patient will report pain and paresthesias in the area of distribution of the mandibular nerve (mandible). The needle is now slowly advanced for a further 0.5–1 cm. A small test dose of 0.1–0.2 mL local anesthetic is carefully administered. The remaining dose of 1–1.5 mL is injected in small fractions with constant aspiration. Particular attention should be given to possible subarachnoid or intravascular positioning of the needle. The sensory distribution of the block is shown in Fig. 10.29.

Dosage

1–2 mL local anesthetic, e.g., 1 % lidocaine, 0.5–0.75 % ropivacaine, or 0.5 % bupivacaine.

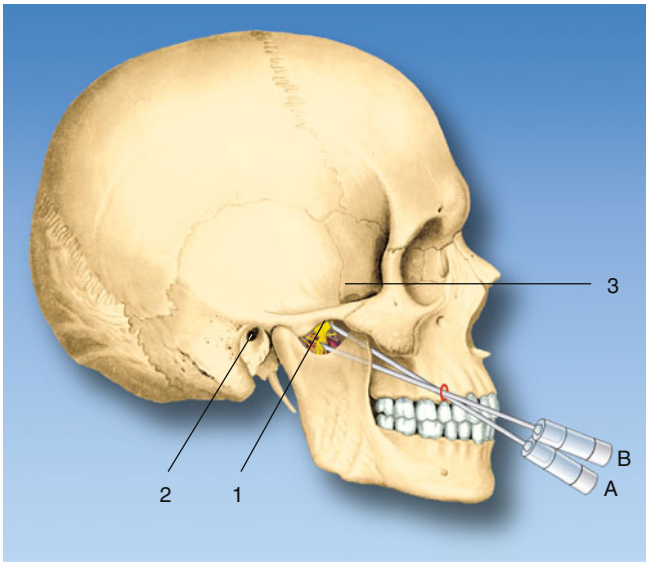


Fig. 10.28 Injection point (level of the second molar tooth). Needle position *A*: bone contact, infratemporal. Needle position *B*: entrance into the oval foramen. (*1*) Zygomatic arch, (*2*) external acoustic pore, (*3*) temporal fossa (With permission from Danilo Jankovic)

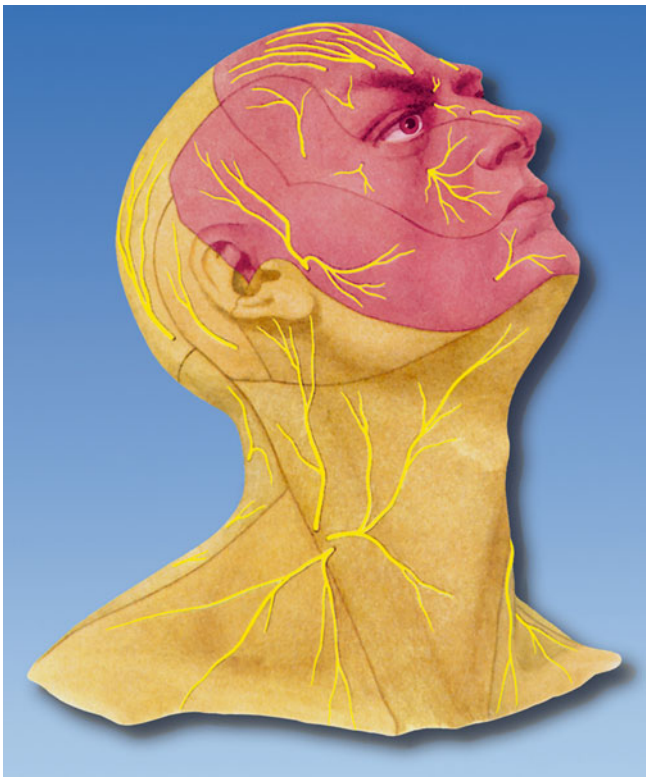


Fig. 10.29 Sensory deficit after blocking of the trigeminal ganglion (With permission from Danilo Jankovic)

Complications

1. Subarachnoid injection (total spinal anesthesia).
   Immediate measures: see Chap. 1, p. 8.
   Important prophylactic measures:
   - Very good knowledge of anatomy
   - Precise execution of the procedure (radiographic guidance)
   - Careful dosage
   - Constant aspiration and injection in the tiniest fractions of 0.1 mL local anesthetic (several test doses)
   - No time pressure
2. Intravascular injection
   Intravascular injection (middle meningeal artery) is always possible (in this highly vascularized region).
3. Hematoma in the cheek or orbit due to vascular puncture.
4. Transient visual weakness or blindness.
   Optic nerve; extremely rare.

Trigeminal Nerve: Comparison of Analgesia Zones

Figures 10.29, 10.30, 10.31, 10.32, 10.33, and 10.34 provide schematic illustrations of the areas supplied by the individual nerves. During blocks, the anesthetic spread may overlap.

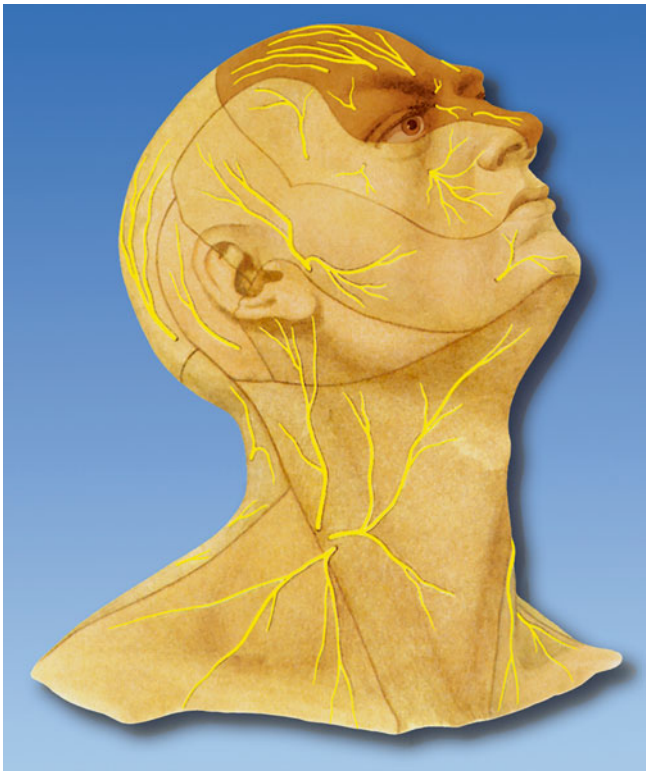


Fig. 10.30 Ophthalmic nerve (With permission from Danilo Jankovic)

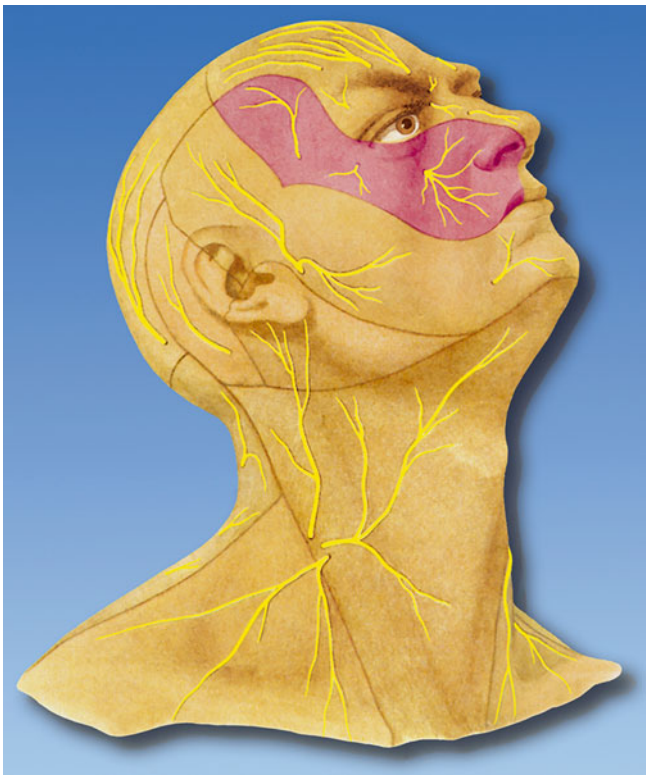


Fig. 10.31 Maxillary nerve (With permission from Danilo Jankovic)

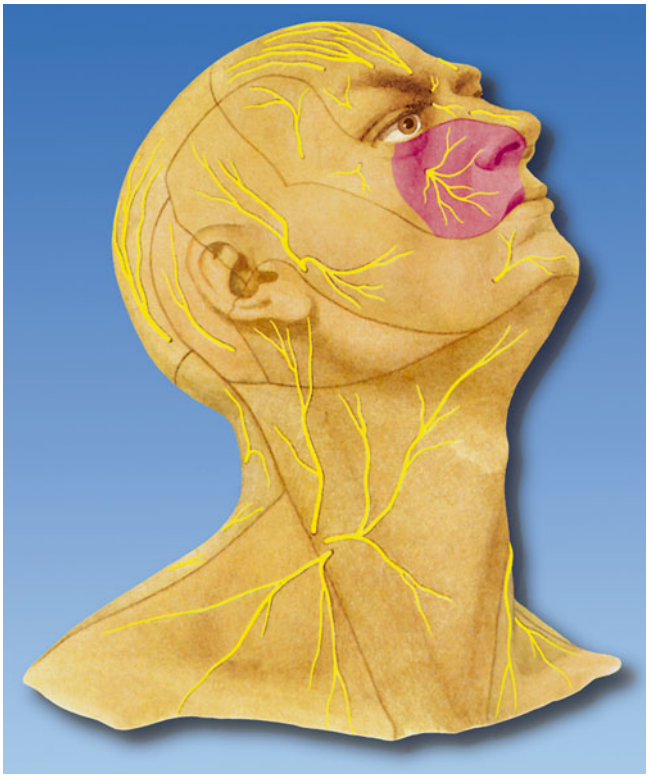


Fig. 10.32 Infraorbital nerve (With permission from Danilo Jankovic)

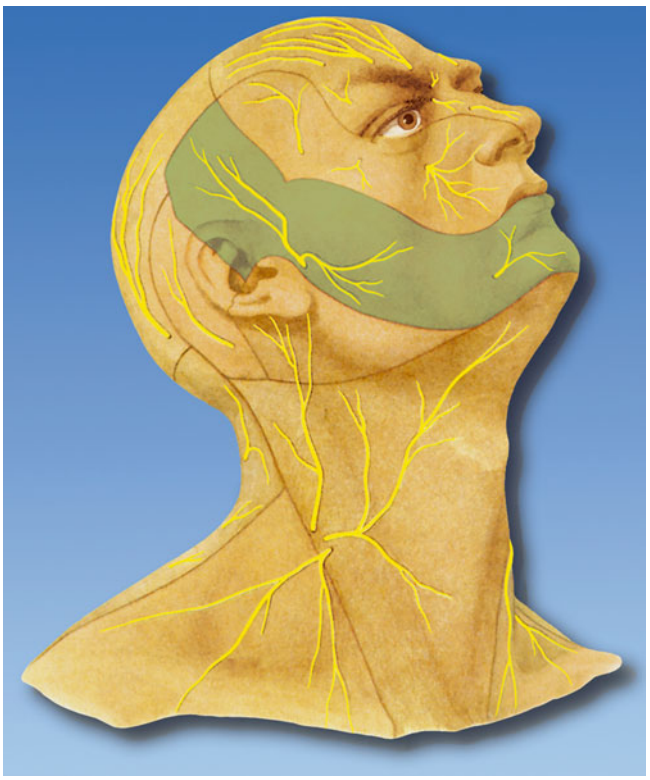


Fig. 10.33 Mandibular nerve (With permission from Danilo Jankovic)

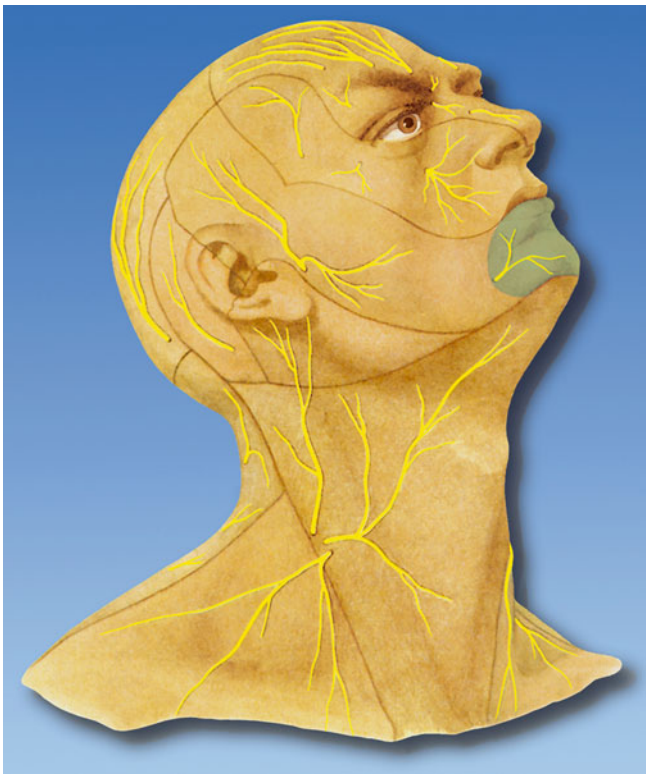
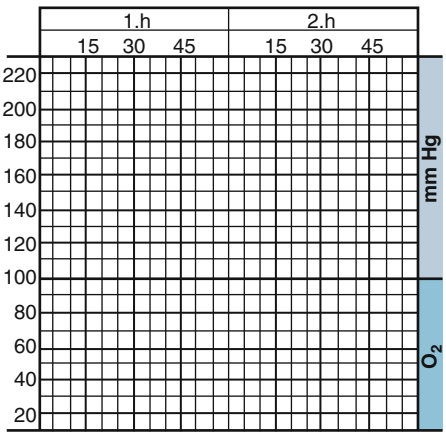
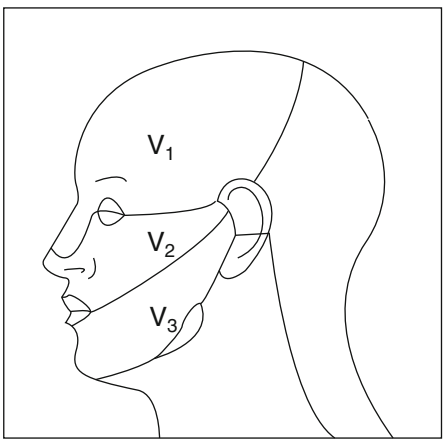


Fig. 10.34 Mental nerve (With permission from Danilo Jankovic)

Record and checklist





Trigeminal ganglion (Gasserian ganglion)

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: ☐ *22 G* ☐ *80 mm long* ☐ *100 mm long*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs*): ☐ *Checked* ☐ *X-ray*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Other*

Local anesthetic: _____ *ml* _____ *%* ☐ *Fractionated*

Test dose: _____ *ml*

Addition to injection solution: ☐ *No* ☐ *Yes* _____

☐ *Radiofrequency lesion*

☐ *Neurolysis* _____ *ml* _____ *%*

☐ *Other* _____

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Nerve region _____ _____ _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* ___ *°C left* ___ *°C*

☐ *Numbness (V_1,V_2,V_3)*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____

Complications: ☐ *None* ☐ *Amblyopia* ☐ *Hematoma* ☐ *Dural puncture*
☐ *Intravascular injection* ☐ *Postdural puncture headache*
☐ *Subarachnoidal injection* ☐ *Respiratory disturbance*
☐ *Total spinal anesthesia* ☐ *Neurological complications*

_____

Subjective effects of block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

With permission from Danilo Jankovic

References

1. Amster LJ. Sphenopalatine ganglion block for the relief of painful vascular and muscular spasm with special reference to lumbosacral pain. N Y State J Med. 1948;48:2475–80.
2. Auberger HG. Niesel HC. Gesichtsschädel: Proximale Leitungsanästhesie im Bereich des N. trigeminus. In: Auberger HG, Niesel HC, Hrsg. Praktische Lokalanästhesie. 4 Auflage. Stuttgart/New York: Georg Thieme Verlag; 1982.
3. Berger JJ, Pyles ST, Saga-Rumley SA. Does topical anesthesia of the sphenopalatine ganglion with cocaine or lidocaine relieve low back pain? Anesth Analg. 1986;65:700–2.
4. Bonica JJ. Block of cranial nerves. In: Bonica JJ, editor. The management of pain. 2nd ed. Philadelphia: Lea & Febiger; 1990.
5. Byrd H, Byrd W. Sphenopalatine phenomena: present status of knowledge. Arch Intern Med. 1930;46:1026–38.
6. Devogel JC. Cluster headache and sphenopalatine block. Acta Anesthesiol Belg. 1981;32:101–7.
7. Jenkner FL. Nervenblockaden auf pharmakologischem und auf elektrischem Weg. Wien: Springer; 1980.
8. Lebovits AH, Alfred H, Lefkowitz M. Sphenopalatine ganglion block: clinical use in the pain management clinic. Clin J Pain. 1990;6:131–6.
9. Moore DC. Regional block. 4th ed. Springfield: Charles Thomas; 1976.
10. Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988.
11. Netter FH. Nervengeflechte und periphere Nerven. In: Krämer G, Hrsg. Farbatlanten der Medizin. (Band 5). Nervensystem I: Neuroanatomie und Physiologie. Stuttgart/New York: Georg Thieme Verlag; 1987.
12. Prasanna A, Murthy PSN. Sphenopalatine ganglion block and pain of cancer. J Pain. 1993;8(3):125.
13. Petren T. Anatomie des Nervus trigeminus. In: Eriksson E, Hrsg. Atlas der Lokalanästhesie. 2. Auflage. Berlin/Heidelberg/New York: Springer; 1980.
14. Reder, Hymanson AS, Reder M. Sphenopalatine ganglion block in treatment of acute and chronic pain. In: Hendle NH, Long DM, Wise TN, editors. Diagnosis and treatment of chronic pain. Boston: John Wright; 1982.
15. Rosen S, Shelesnyak MC, Zacharias LR. Naso-genital relationship II. Pseudopregnancy following extirpation of sphenopalatine ganglion in rat. Endocrinology. 1940;27:463–8.
16. Ruskin AP. Sphenopalatine (nasal) ganglion: remote effects including "psychosomatic"symptoms, rage reaction, pain and spasm. Arch Phys Med Rehabil. 1979;60:353–8.
17. Ruskin SL. The neurologic aspects of nasal sinus infections. Headaches and systemic disturbances of nasal ganglion origin. Arch Otolaryng. 1929;4(10):337–82.
18. Saade E, Paige GB. Patient administrated sphenopalatine ganglion block. Reg Anesth. 1996;21(1):68–70.
19. Sluder G. Injection of the nasal ganglion and comparison of methods. In: Nasal neurology, headaches and eye disorders. St. Louis: CV Mosby; 1918.
20. Waldman SD. Sphenopalatine ganglion block – 80 years later. Reg Anesth. 1990;18:274–6.
21. Zacharias LR. Further studies in naso-genital relationship: anatomical studies of perihypophyseal region in rat. J Comp Neurol. 1941;74:421–45.
22. Tsui BC. Ultrasound imaging to localize foramina for superficial trigeminal nerve block. Can J Anesth. 2009;56:704–6.

Chapter 11

Trigeminal Nerve: Neurodestructive Procedures

Olav Rohof, MD (✉)
Pain clinic, Atrium Orbis Location Sittard Geleen, H. van der Hoffplein 1, Sittard Geleen, 6162 BG, The Netherlands
e-mail: o.rohof@orbisconcern.nl

Robert Jan Stolker, MD, PhD
Professor and Chair, Department of Anesthesiology, Erasmus MC, P.O. Box 2040, Rotterdam, 3000 CA, The Netherlands
e-mail: r.stolker@erasmusmc.nl

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_11,

Radiofrequency Lesioning of the Sphenopalatine Ganglion

Anatomy

The sphenopalatine (or pterygopalatine) ganglion (SPG) is mainly a parasympathetic ganglion in the upper part of the pterygopalatine fossa; its postsynaptic fibers supply the lacrimal, nasal, palatine, and pharyngeal glands. It is closely related to the maxillary nerve and lies just beneath the pterygopalatine (sphenopalatine) fossa (Fig. 11.1). The ganglion has a flat conical shape that can extend to a length of 5 mm. The fossa has a triangular shape with its basis at the floor of the sphenoid sinus with a length of approximately 2 and 1 cm width. On a lateral fluoroscopic view, the fossa resembles an "inverted vase" (Fig. 11.2). The fossa is bordered anteriorly by the posterior wall of the maxillary sinus, posteriorly by the pterygoid process and the ala major of the sphenoid bone, medially by the perpendicular plate of the palatine bone, and superiorly by the sphenoid sinus; laterally it communicates with the infratemporal fossa. The fossa is anteriorly connected to the orbit through the inferior orbital fissure, medially to the nasal cavity through the pterygo(spheno)palatine foramen, postero-superiorly to the middle skull "groove" through the foramen rotundum (which contains the maxillary nerve) and to the mouth through the palatine canal, and posteriorly to the nasopharynx. The fossa also contains the maxillary artery and its multiple branches.

The ganglion consists of three types of nerve fibers (Fig. 11.3):

1. Preganglionic parasympathetic (visceromotor) fibers to the lacrimal, nasal, and palatine glands, reaching the ganglion from the facial nerve through the greater superficial petrosal nerve and the nerve of the pterygoid canal.
2. Sympathetic fibers coming from the superior cervical ganglion and the internal carotid plexus reaching the ganglion via the deep petrosal nerve which joins the greater superficial petrosal nerve to form the Vidian nerve.
3. Many afferent fibers crossing the ganglion, originating from the nasal cavity, the soft palate, and the pharynx on their way to the Gasserian ganglion through the maxillary nerve. There is also direct contact between the anterior horn of the spinal cord and the neuro-humeral axis (adenohypophysis) [1, 2]. As the SPG plays a role in the innervation of the brain vascularization, and as such has been described in the "trigeminal vascular system," it has been suggested that it is probably involved in several forms of vascular headaches and atypical facial pains [3].

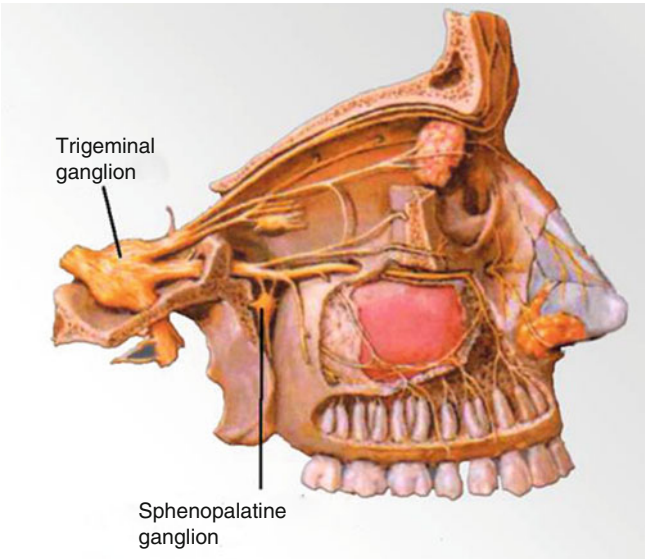


Fig. 11.1 Anatomy of the sphenopalatine ganglion (From Gauci C. Manual of RF techniques, 2004, FlivoPress, Meggen (LU), Switzerland)

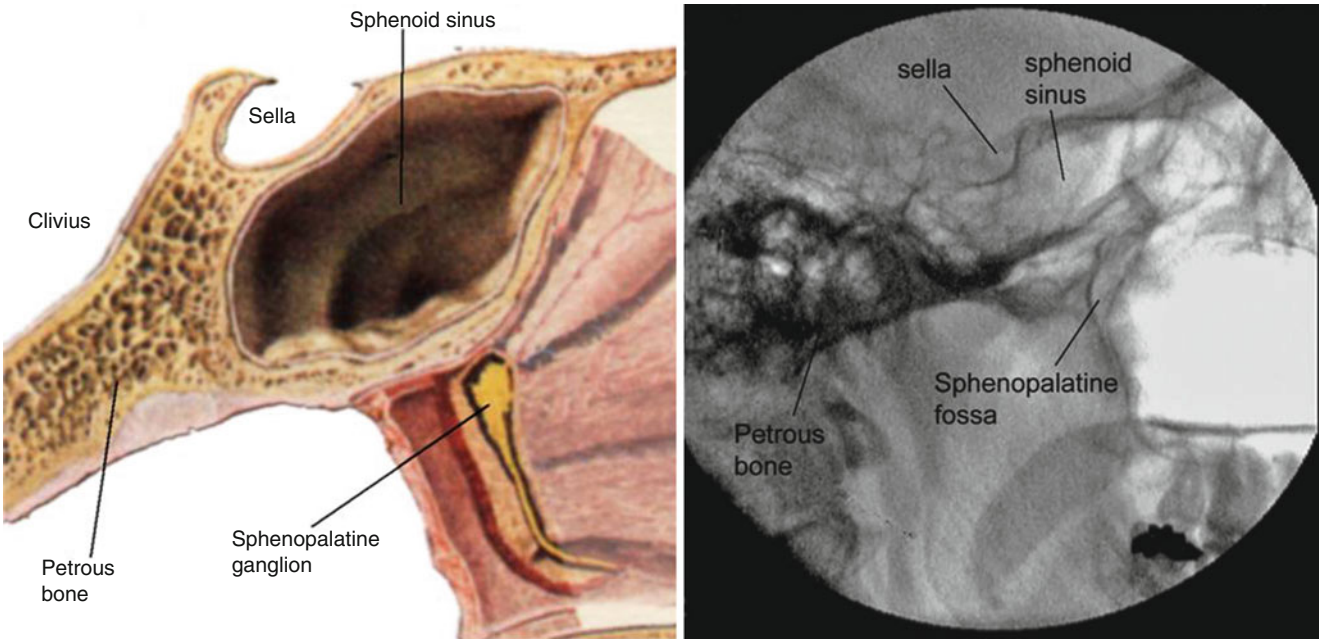


Fig. 11.2 Relevant anatomy around the sphenopalatine ganglion (Adapted from Sluijter [8])

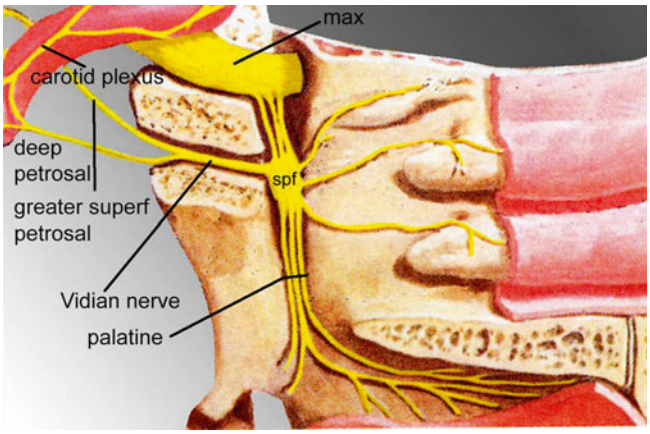


Fig. 11.3 Innervation of the sphenopalatine ganglion (From Sluijter [8])

Blockade of the Sphenopalatine Ganglion

Blockade of the SPG by local application or injection of a local anesthetic or cocaine has already been described as early as 1908 as an effective method in the treatment of headache; pain in the eyes, mouth, and ears; lumbosacral pain; arthritis, glaucoma; and hypertension [4]. SPG block with local anesthetics may be used for the treatment of acute migraine headache, acute cluster headache, and a variety of facial neuralgias including Sluder's, Vail's, and Gardner's syndromes. This treatment is also useful in the management of status migrainosus and chronic cluster headache. Later on, neurolytic fluids such as alcohol have been used, but uncontrollable spread of fluids has limited the use of this procedure. RF lesioning is a focal neurolysis, making the lesion only around the electrode tip.

The lateral approach is recommended for neurodestructive procedures of the sphenopalatine ganglion in order to avoid lesioning of the maxillary nerve [5].

Radiofrequency lesioning of the sphenopalatine ganglion has predominantly been studied for the treatment of chronic cluster headache unresponsive to conventional management, e.g., drug therapy [6].

The SPG is situated in a richly vascularized region, responsible for a rapid evacuation of the applied heat. Moreover, the size of the ganglion exceeds the size of the RF lesioning. These anatomical characteristics form an explanation for the fact that total nerve destruction of the SPG is never achieved following RF conventional thermolesion using 22-G needles.

The adapted delivery method of the high-frequency current, termed pulsed radiofrequency, delivers the current in bursts of 20 ms followed by 480 ms downtime during which the generated heat is allowed to wash out. In this way, higher voltage, usually 45 V, can be used without generating the neurodestructive temperature (i.e., 43 °C) in the nerve tissue. Pulsed radiofrequency is generally considered as safer than conventional radiofrequency; up till now, no neurological side effect or complication has been mentioned with pulsed radiofrequency treatment of a wide variety of pain syndromes.

Indications for Sphenopalatine Radiofrequency Treatment

I. Major indication:
   1. Cluster headache
II. Minor indications:
   1. Atypical facial pain in the maxillary region
   2. Side-locked migraine
   3. Remaining frontal headache after treatment of cervicogenic headache
   4. Trigeminopathy with parasympathetic features such as lacrimation, rhinorrhea, or mucosal congestion

Cluster headache, the major indication for radiofrequency treatment of the sphenopalatine ganglion, is a relatively rare disorder compared with migraine headache. An attack is typically brief, lasting 15 min to 2 h. Attacks occur in groups at the rate of one to several attacks a day over a period of several weeks to many months. The series of attacks may occur twice a year with remission of up to 20 years. When no remission occurs for more than 1 year, the disorder is considered to be chronic. The pain is strictly unilateral and typically in the temporal and periorbital region. The intensity of the attacks is of such severity that some patients have termed cluster headache "suicide headache." Patients may also use the description of a hot poker being pushed into the eye.

Technique

It is essential that this technique be performed only with fluoroscopic control.

In case patients are using clopidogrel or ASA, they are asked to stop this treatment 5 days prior to the intervention. When patients use coumarin and its derivatives, the intake of these drugs should be interrupted and the INR controlled before the procedure. INR of 1.8 or less is appropriate.

Patient Positioning and Visualization of the Sphenopalatine Ganglion

The patient is lying supine with the head on a radiolucent rest. The head should be gently taped to the headrest in order to prevent movements. The C-arm of the fluoroscope must be positioned in the axis of the operating table.

For a good and consistent anatomical orientation, it is recommended to have the image on X-ray-screen in the same position as the patient is lying (Fig. 11.4).

To reduce the operator's and patient's exposure to the X-ray beam as much as possible, it is recommended to place the C-arm in lateral position prior to activating the fluoroscope. The position of the fluoroscope is then adjusted until both angles of the mandible are superimposed. This will be achieved by moving the C-arm in both the frontal and axial planes. Next, move the C-arm to a more cranial and ventral position to visualize the essential anatomical structures: the sella turcica, base of the skull, sphenoid sinus, petrous bone, and fossa pterygopalatina. The fossa should now become visible as a "vase" with its base on the floor of the sphenoid sinus. Readjustment of the C-arm is often required to get sharp walls of the fossa. The ganglion is situated in the superior aspect of the fossa.

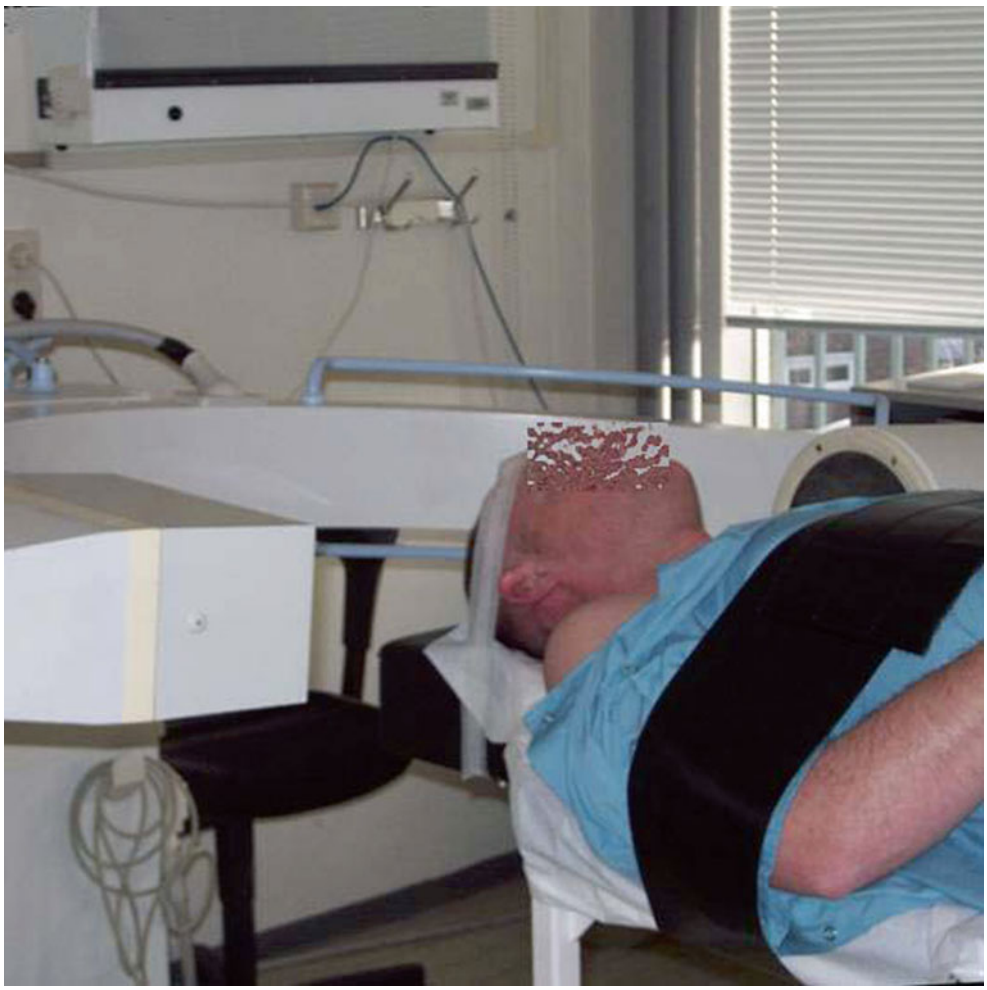


Fig. 11.4 Patient positioning for the radiofrequency procedure of the sphenopalatine ganglion, C-arm in the axis of the operating table. Image on screen in the same position as the patient

Needle (Cannula–Electrode) Insertion

In order to find the entry point of the electrode, a metal ruler is projected over the fossa on the side of the patient and a line is drawn over the skin (Fig. 11.5). Palpate the region and mark a point on the drawn line just at the inferior aspect of the zygomatic arch. The entry trajectory runs through the mandibular notch (space between anterior and posterior mandibular ramus (Fig. 11.6)). At first, with a 22-G 2–3-in. needle, local anesthesia is delivered through the skin and the mandibular notch to the deeper soft tissues. This entry point allows the controlling and is important for reaching the sphenopalatine fossa. If necessary, the entry point is corrected.

An SMK C-10 needle with a 5-mm active tip is then introduced infrazygomatically to the fossa in a slight anterior and cephalic direction and carefully advanced under lateral fluoroscopic guidance. In case of bone contact, the direction of the needle should be corrected to the target point. After insertion of about 4–5 cm or in case of doubt regarding the depth of the needle, a control in AP projection is performed. The final position is reached when the needle tip has passed the lateral wall of the nose (Fig. 11.7). Just before reaching the end position, the needle may hit the maxillary nerve causing a very unpleasant sensation for the patient. Patients should be informed prior to starting the procedure of this possibility and instructed to signal this straight away. If it occurs, stop advancing the needle immediately and inject carefully and slowly 0.5–1 ml local anesthetic. After waiting approximately one minute, the needle can be redirected to reach the final position.

On the AP projection, the nasal septum should be in the midline just over the dens. The needle tip must then be in the lateral wall of the nasal cavity (Fig. 11.8).

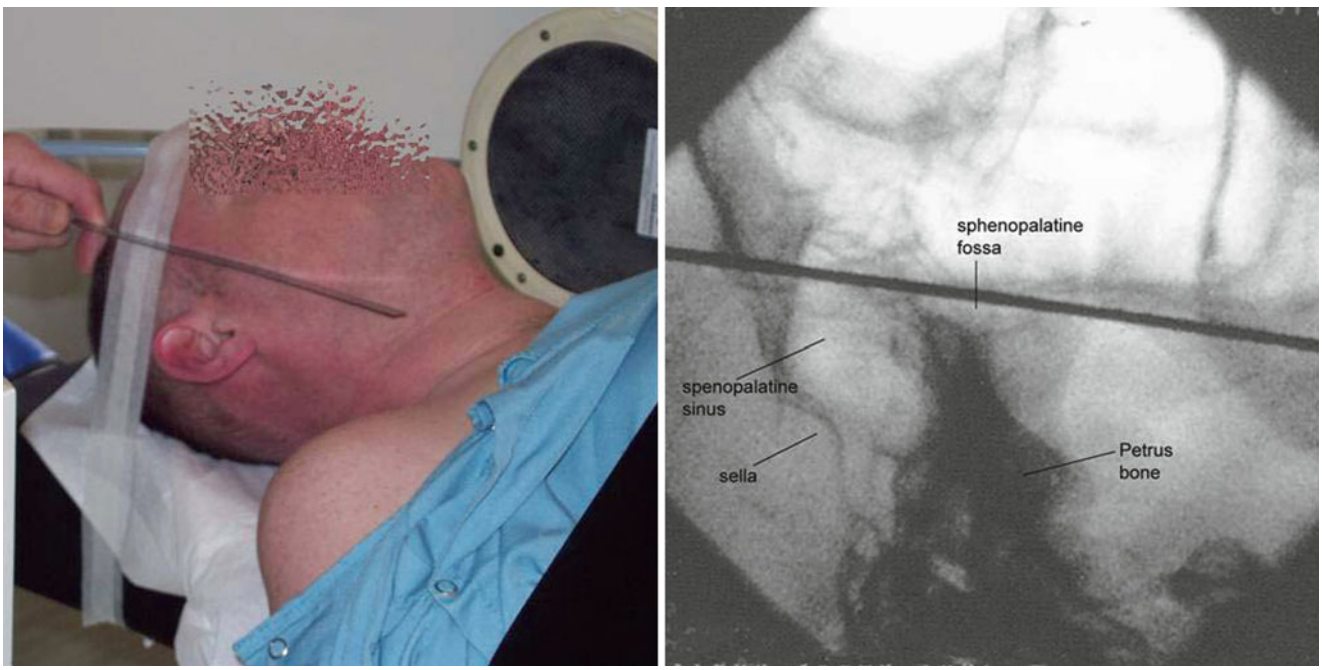


Fig. 11.5 Metal ruler projected over the fossa pterygopalatina, patient and fluoroscopic, X-ray lateral view

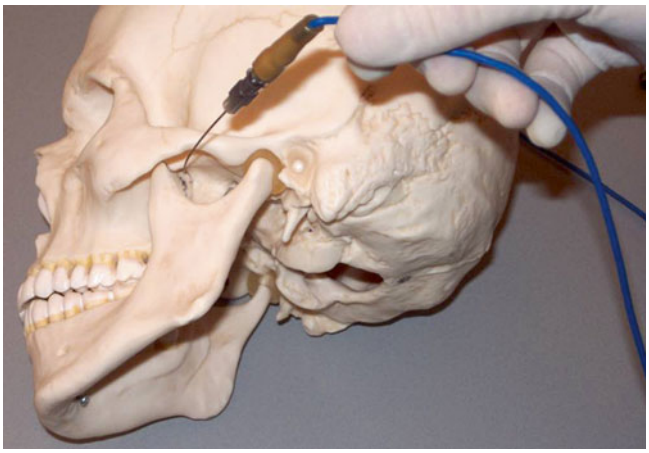


Fig. 11.6 Needle direction to the sphenopalatine ganglion on a model of the skull, infrazygomatic, through mandibular notch

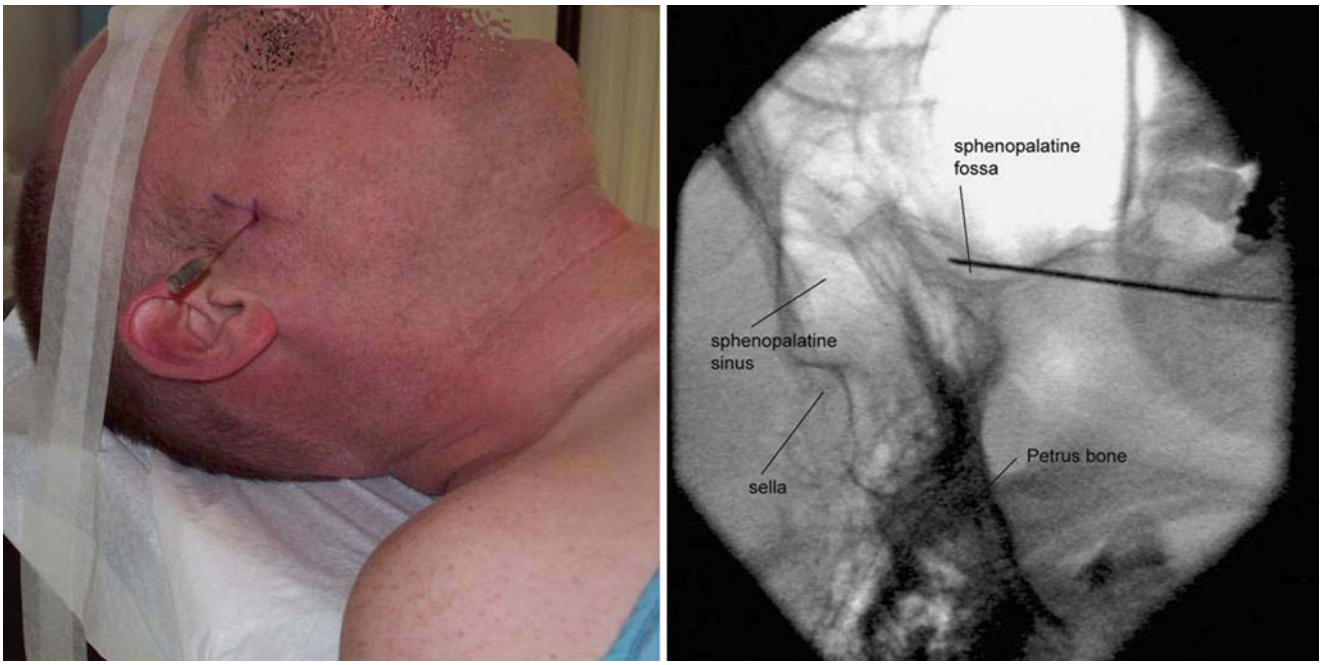


Fig. 11.7 Electrode positioning to the sphenopalatine ganglion, X-lateral view, patient and fluoroscopic

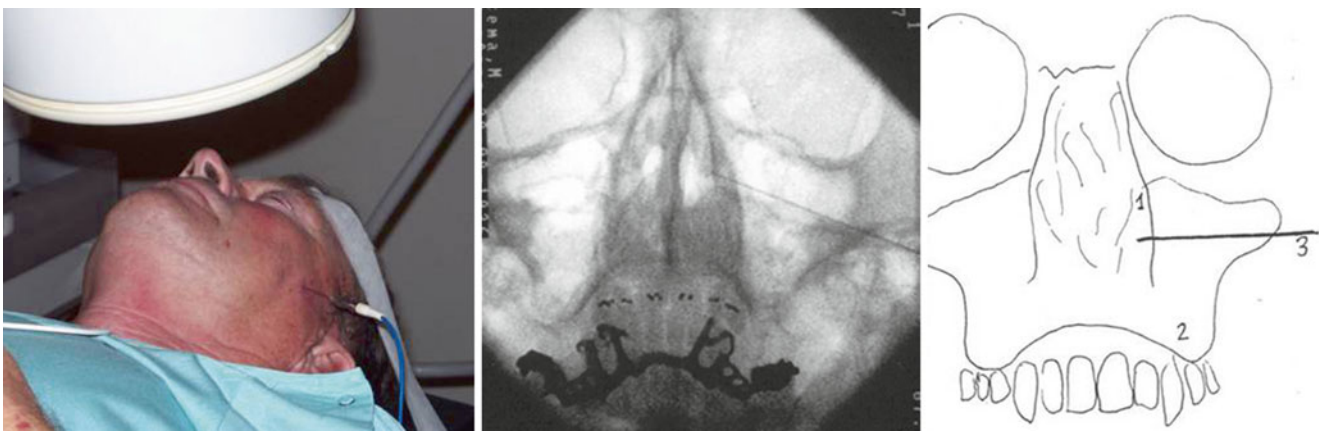


Fig. 11.8 Schematic and fluoroscopic visualization of the sphenopalatine ganglion in AP projection

Electrostimulation

At 50 Hz, paresthesia should be felt under 0.7–1.0 V (also depending on the amount of local anesthetic already given and on the duration of interval between administration of the local anesthetic and the electrostimulation). The patient must feel the stimulation in the nose or soft palate. If the paresthesia is felt in the upper teeth or lip, the cheek, or the outer part of the nose, the needle is too close to the maxillary nerve and the needle should be redirected more medially. If stimulation is felt in the hard palate, the needle point is too close to the palatine nerves and the needle must then be redirected more posteriorly and medially. If the position is correct (confirmed with fluoroscopy and electrostimulation), 1–2 ml of local anesthetic is injected. Part of it can flow in the nose and pharynx causing a bitter taste. After having waited for 1–2 min, an RF lesioning of the ganglion is performed.

Radiofrequency Lesion

Radiofrequency lesions are commonly performed in series of two or three, for 60 s at 70–80 °C. Between each lesion, the electrode is moved medially for a few mm until the septum has been touched.

Pulsed radiofrequency current is applied at 45 V for 2 min. After moving the needle to a more medial position, a second treatment is performed.

Complications

Total destruction of the SPG causes dryness of the eye; an "open nose," since the mucosa has less inclination to swell; and numbness of the palate [8]. Because of the size of the ganglion, the use of 22-G needles, and the high vascularization of the environment, total destruction of the SPG is never achieved.

Other side effects and complications are:

1. Unilateral sensory loss of the palate: this is usually transient, but you must warn the patient that incidentally it may persist for a longer period.
2. Epistaxis: in 3–5 % of cases. At the end of the procedure, the patient may sit up with a gauze under the nose. In case of a nasal bleeding, this stops within 10 min.
3. Hematoma of the cheek: < 1 %, can be treated with pressure and ice packs.
4. Denervation of the maxillary nerve. This is caused by a wrong technique! Reassure the patient and prescribe antiepileptics like pregabalin and/or clonazepam.
5. Facial nerve block or diplopia due to spread of local anesthetic to the facial nerve in front of the ear or to the orbit. Reassure the patient and let the patient go home after normalization.
6. A few weeks after the procedure, very slight intermittent bleeding from the nose may occur, however very seldom. Consultation at an ENT department is recommended.

Evidence for Radiofrequency Treatment of the Sphenopalatine Ganglion

Radiofrequency treatment is indicated when conventional treatment fails. Considering the severity of the pain and the relative low incidence of this syndrome, performing randomized controlled trials comparing radiofrequency treatment to sham intervention is hardly justifiable from an ethical point

of view. The comparison with the "gold standard" of conventional treatment may be considered, but for most patients, neurodestructive treatment is the last resort. These factors may explain why no controlled studies have been published up till now and will most likely not be performed.

Several retrospective studies report a long-term effect of radiofrequency treatment of the SPG. Sanders and Zuurmond [6] reported RF treatment of the SPG in 66 patients. Fifty-six patients suffered episodic cluster headache and 10 patients had the chronic form.

All previous pharmacological and surgical treatments had failed. All patients were treated with an RF lesioning of 70 °C three times for 60 s. Of the episodic CH patients, 60.4 % ($n=34$) and 30 % ($n=3$) of the chronic form reported complete pain relief during a mean follow-up period of 29 months.

In the group of 20 cluster headache patients treated by Stolker et al. [9, 10], 65 % ($n=13$) were pain-free, 15 % ($n=3$) had pain reduction of more than 50, and 20 % ($n=4$) had no result. Another group of 26 patients [9, 10], who suffered from cluster headache in combination with a cervical syndrome 31 % ($n=8$), were pain-free, 50 % ($n=13$) had 50 % or more relief, and 19 % ($n=9$) had insufficient result after initial treatment with an RF lesioning of the SPG only. When further treatment of the cervical syndrome was done by means of RF lesioning of the cervical medial branch and/or of the dorsal root ganglion, the result improved significantly: 73 % ($n=19$) were pain-free, 23 % ($n=6$) had 50 % or more pain relief, and in only 4 % ($n=1$), there was insufficient result.

Sluijter et al. [11] reported an excellent or good result in 78 % ($n=18$) of 23 patients.

Salar et al. [12] treated 7 patients with cluster headache with an RF lesioning of the SPG (65 °C, 60s). After a repeated RF lesioning of the SPG, all 7 patients were pain-free.

Recently, a case report of a patient suffering from post-traumatic headache who received pulsed radiofrequency treatment of the sphenopalatine ganglion reported pain relief for 17 months [13].

A retrospective analysis of the record cards of patients suffering from different types of facial pain showed complete pain relief in 21 % and mild to moderate pain relief in 65 % of the patients treated with pulsed radiofrequency [14].

Conclusions

Radiofrequency treatment may be considered for patients suffering from head and facial pain refractory to conventional treatment that can be attributed to the maxillary nerve. The procedure must be performed under careful fluoroscopic control, and correct electrode placement must be confirmed by electrostimulation. The results from retrospective studies indicate a high success rate with long-lasting pain relief. Though only very limited documentation on the pulsed radiofrequency treatment of the sphenopalatine ganglion is available, the initial findings suggest good results with this less neurodestructive technique.

Radiofrequency Treatment of the Gasserian Ganglion

Anatomy

The trigeminal ganglion (semilunar ganglion, Gasserian ganglion) lies on the dorsal surface of the petrous bone. The intracranial Gasserian ganglion lies medially in the middle cranial fossa; lateral to the cavernous sinus, internal carotid artery, and cranial nerves III–VI; and posterior and superior to the oval foramen, through which the mandibular nerve exits from the intracranial cavity (Fig. 11.9). The average size of the ganglion is 1–2 cm. Part of the ganglion (the posterior two thirds) is located within the trigeminal cave (Meckel's cavity), a duplicate of the dura that encloses the ganglion. The oval foramen is a channel ca. 5 mm long and its largest diameter is ca. 8 mm.

Anteriorly, the ganglion gives off three branches intracranially: the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerve. The two medial branches (V1 and V2) are sensory, whereas the most lateral branch (V3) also innervates the masticatory muscles and thus partly is a motor nerve.

The trigeminal ganglion shows a somatotopy: The ophthalmic [8] division is located dorsomedially and the mandibular (V3) division ventrolaterally, and the maxillary (V2) division has an intermediate position (Fig. 11.10). This has important implications for the targeted treatment of the ganglion in performing a (partial) RF lesioning in trigeminal neuralgia. The pars major is the sensory part, and the pars minor is the motor part—located more laterally.

The nucleus nervi trigemini is extending from the brain stem to the proximal part of the cervical myelum (Fig. 11.11). There are also many neural connections between the trigeminal nerve and the autonomous system (e.g., sphenopalatine ganglion, upper sympathetic trunk).

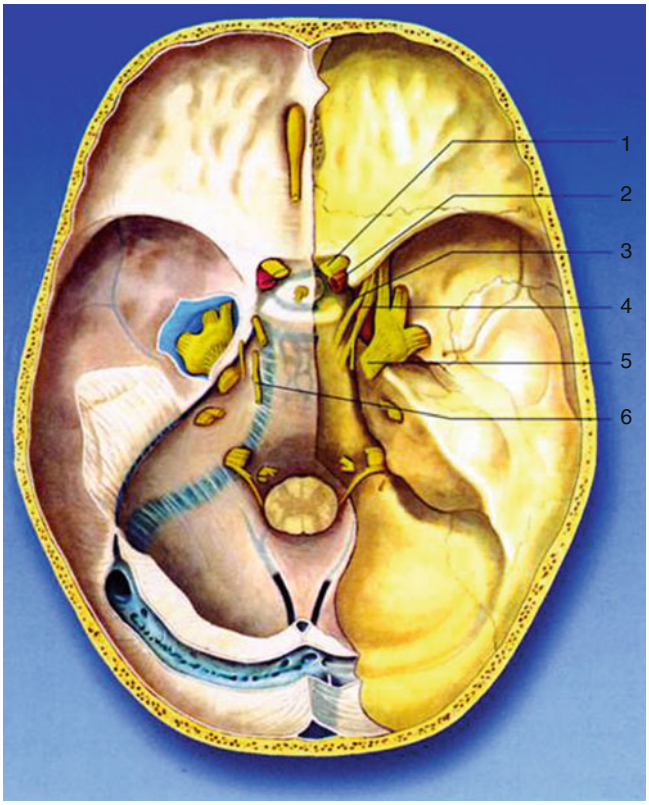


Fig. 11.9 Anatomy of the Gasserian ganglion (*1* optic nerve, *2* internal carotid artery, *3* oculomotor nerve, *4* trochlear nerve, *5* trigeminal nerve, *6* abducens nerve) (From *Regional Nerve Blocks and Infiltration Therapy*, Third Ed. (2004) Danilo Jancovic, Blackwell Publishing with permission of the editor)

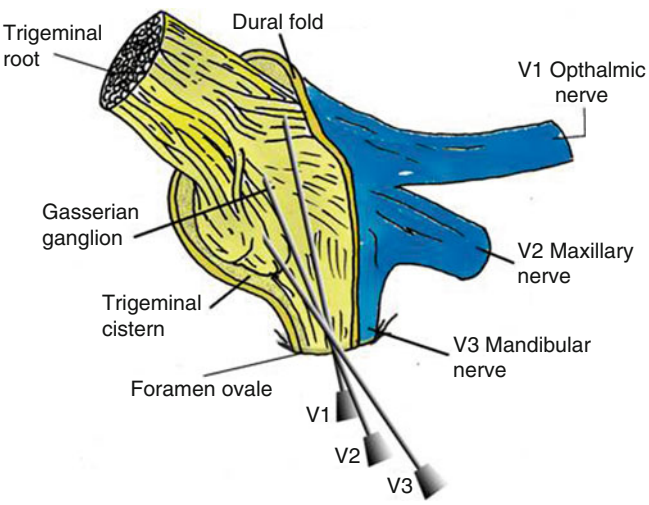


Fig. 11.10 Somatotopy of the trigeminal ganglion and schematic needle positioning for RF lesioning (Adapted from Sluijter [8], with permission of the editor)

Fig. 11.11 Anatomy of the nucleus nervi trigemini (Netter)

Facial Pain

Identifying the cause of facial pain in many cases is difficult and often requires consultation with the dentist, ophthalmologist, ENT doctor, oromaxillary surgeon, and neurologist to exclude specific pathologies.

In trigeminal neuralgia, the diagnosis is mostly clear: the patient is suffering from brief episodes of sharp lancinating pain in one or more of the trigeminal divisions, which should also be provocable by light touch of the skin, speaking, chewing, cleaning of the nose, or wind; both criteria should be met; otherwise, the diagnosis should be questioned.

The trigger areas need not necessarily be located in the division where the patient experiences the pain: many patients have pain in the first division and the trigger zone in the second division. It also frequently occurs that the trigger zone lies in the third and the pain is in the second and third division and the other way around. In the classical case, pain periods are followed by pain-free periods, but residual pain has been reported in 42 % of cases, mostly in patients suffering from trigeminal neuralgia for many years. This was described as a combination of trigeminal neuralgia and atypical facial pain [15], but trigeminal neuralgia with continuous pain is better described as "trigeminal neuropathy" because the typical characteristics of atypical facial pain are absent [8].

Trigeminal neuralgia has to be differentiated from atypical facial pain.

In atypical facial pain, one should always look for cervical trigger mechanisms, even if the patient does not complain about neck pain; on physical examination, tenderness over the cervical facet joints and/or over the occipital nerves at the occiput or mastoid process may be of great importance for the diagnosis of cervical facet pain and segmental nerve C2 or C3 involvement [8]. In these cases, referred pain from the cervical region may be the proper diagnosis instead of atypical facial pain.

In case of one or more autonomic symptoms such as unilateral lacrimation, rhinorrhea or nasal congestion, skin erythema, conjunctival injection, local edema, ptosis, or miosis are present, the sphenopalatine ganglion may be involved in the pain mechanism and become a successful target for treatment.

True atypical facial pain is mostly localized in the maxillary region. This condition usually occurs between the age of 30 and 50 years (usually younger patients than those suffering from trigeminal neuralgia). The female/male ratio is about 3:1. Many patients describe this pain as being constant, dull, and deep, often with a numb feeling, absolutely without any shooting pain episodes. Yet the trigeminal evoked potential is not disturbed, giving rise to suspicion of

underlying psychological disorders. Indeed, these patients show higher scores on the MMPI profile than patients with tension-type headache, neurovascular headaches, or temporomandibular joint dysfunction [8]. It is strongly recommended to have a psychological assessment of patients with atypical facial pain, before considering invasive treatment options.

Trigeminal neuralgia most frequently occurs in patients over 50 years; occasionally, it also occurs in very young patients. It is thought to be caused by vascular compression of the trigeminal root, which can however not always be objectified. That is why this form of trigeminal neuralgia is called the asymptomatic or idiopathic form. Trigeminal neuralgia may also be symptomatic (an expression of an underlying disease) such as multiple sclerosis, neuroma, or an intracerebral tumor. A proper neurological investigation including an MRI of the skull should therefore always be performed in order to diagnose eventual underlying pathologies and neurological diseases.

Radiofrequency treatment of the Gasserian ganglion should only be considered when conventional treatment fails or causes intolerable side effects. Conventional treatment consists of pharmacological treatment with antiepileptics as carbamazepine, phenytoin, gabapentin, pregabalin, clonazepam, etc. In younger patients, the neurosurgical (Jannetta) microvascular decompression (MVD) operation is the method of preference. In skilled hands, the complication rate is relatively low, but serious complications are described such as facial weakness, hearing loss, or more serious neurological damage; even mortality after this operation has been reported. In multiple sclerosis, the MVD operation only works in combination with a partial section of the trigeminal nerve. The pain-free period after MVD is longer than after RF lesioning of the ganglion. But if the pain recurs after MVD, recurrent vascular compression is seldom done during reoperation, and the incidence of complications is much higher. Also, the success rate of RF treatment of the ganglion is then reportedly less. In elderly patients, the method of preference is a point of discussion. Since the complication rate of the MVD operation is higher in the elderly and in case of recurrence of pain, further therapy may be problematic, and RF lesioning of the ganglion may be preferred. If properly informed, most elderly patients choose this treatment.

Nowadays, injection of glycerol [16] is rarely performed. Uncontrolled spread of the fluid can accidentally damage other (neural) structures.

More recently, Gamma Knife treatment (targeted cobalt irradiation) has been described. This procedure also has success for a limited time. Redo procedures are more difficult to perform and bear an increased risk of sensory damage.

Technique: RF Lesioning of the Gasserian Ganglion

Before treatment, informed consent is required, and all advantages and disadvantages of the procedure should be discussed with the patient and his/her relative(s). In case patients are using clopidogrel or ASA, they are asked to stop this treatment 5 days prior to the intervention. When patients use coumarin and its derivatives, the intake of these drugs should be interrupted and the INR controlled before the procedure. INR of 1.8 or less is appropriate.

Before treatment, the patient must be sober for at least 4 h.

The patient should have an intravenous access line and be connected to a cardiovascular and respiratory monitor to allow safe reversible patient sedation. Patient cooperation and communication with the doctor are mandatory during the stimulation phase, and during introduction of the needle, the contact with the ganglion is extremely painful requiring anesthesia. The authors prefer intravenous methohexital; however, propofol, alfentanil, remifentanil, or midazolam is also used.

The patient is placed in the supine position on a radiolucent headrest with a little pillow under the shoulders in order to obtain an extended head position, which is essential to provide a good fluoroscopic view. The head should be gently taped to prevent movements. The fluoroscope must be in line with the patient and the X-ray beam reversed from the normal configuration because most image intensifiers are too large to avoid contact with the thorax of the patient. The doctor should (always) stand at the right side of the patient.

With a tunnel vision technique in a submentovertex projection, the oval foramen should be made visible between the mandibular process on the lateral side and the maxilla at the medial side (Figs. 11.12 and 11.13). The C-arm should be adjusted to a position that the foramen has a true oval form because a too vertical X-ray direction gives the foramen a round configuration and a too horizontal direction a too flat configuration.

The entry point is marked over the oval foramen some centimeters lateral to the corner of the mouth; however, this point may be quite variable depending on the anatomical relations. The choice of the entry point is also dependent on the targeted division of the trigeminal nerve; for the third division, the end point is more lateral, more inferior, and more superficial and the end point for the first division more medial superior and deeper. The end point should always be chosen in the middle or medial one third of the oval foramen, for the second and first division always medial, for the third division a little more lateral (Fig. 11.10). A too lateral position results in a position in the pars minor.

The patient is sedated with methohexital 50–60 mg intravenously. The SMK C-10 needle with a tip of 2 mm is advanced through the skin and muscles of the cheek in the direction of the target (Fig. 11.14).

A finger of the free hand is placed in the patient's mouth to check potential penetration of the mucosa of the cheek, because if this happens, bacterial transfer may cause meningitis.

When the oval foramen is entered, the fluoroscope is changed to the lateral projection, and the electrode tip should be projected posterior to the sella turcica, having passed the base of the skull, but never deeper than the clivus (Fig. 11.15). Reflux of cerebrospinal fluid through the cannula is normal, signing the correct placement in Meckel's cave. Aspiration of blood indicates that a large vessel may be entered. In this case, the needle position should be controlled fluoroscopically, and the procedure must be aborted.

The patient is awakened and electrostimulation is carried out; with the 50-Hz sensory stimulation, the patient should feel paresthesia in the targeted division under 0.15 V; at 2 Hz, motor stimulation masticatory muscle contractions should be visible at a voltage that is at least 3 times the sensory stimulation threshold, thus over 0.45 V. If motor stimulation occurs <0.45 V, the needle tip is too close to the motor branch, and the needle position must be corrected to a deeper or a more medial position. Sensory stimulation should only occur in the division where the trigger, not the pain, is located.

When correct needle position is achieved, another i.v. dose of methohexital is given and the RF lesioning is performed. When the first division is targeted, treatment should start cautiously aiming at 55 °C; for the second and third division, the temperature is raised to 65 °C for 60 s. When the patient is sufficiently alert, testing of the sensory quality of the face is carried out by pinprick of the skin in all 3 divisions of the trigeminus, and the corneal reflex is tested for the first division; the patient's reactions at the left and right side are compared. On indication, a following second or third RF lesioning for 60 s with consecutive higher temperature of 70 and 76 °C is being made until a light pinprick hypalgesia has developed in the targeted division, securing the corneal reflex to remain positive. Especially treatment of the first division must be carried out more carefully using lower temperatures. After each lesion of 60 s, the patient is awakened, and sensory testing is carefully performed in order to prevent a too extensive lesion and subsequent anesthesia dolorosa or cornea anesthesia. It is not the aim of this technique to produce a deep sensory loss. There is a lower recurrence rate after a deep sensory facial loss [17–19]; the risk of the resulting annoying dysesthesia and even anesthesia dolorosa however is a too high price to pay. Therefore, the authors advise to try to produce a light pinprick hypalgesia and to take the higher recurrence rate for granted. Patients choose for the same option if informed properly. In choosing for this method of making a relatively small RF lesioning by using SMK C-10 needle with an active tip of 2 mm and stopping the procedure with the development of a light hypesthesia in the trigger zone, the complication rate is relatively low with good results on pain relief. The mean recurrence time was 2.3 years in the asymptomatic trigeminal neuralgia group ($n=265$) and 1 year and 3 months in the symptomatic group ($n=21$) [19]. There were no side effects in 48 % of the treated 286 patients and only mild side effects in the rest [19].

If a pulsed RF treatment of the ganglion is being performed, the procedure is identical until the electrostimulation. If the proper criteria are met, pulsed RF with a frequency of 2 Hz 45 V for 4–8 min is delivered under sedation. In case you have a NeuroTherm NT1000 generator, it may be better to produce a "pulsed dose" lesion, because with this treatment option, a consistent amount of electrical energy is being delivered in a standardized fashion.

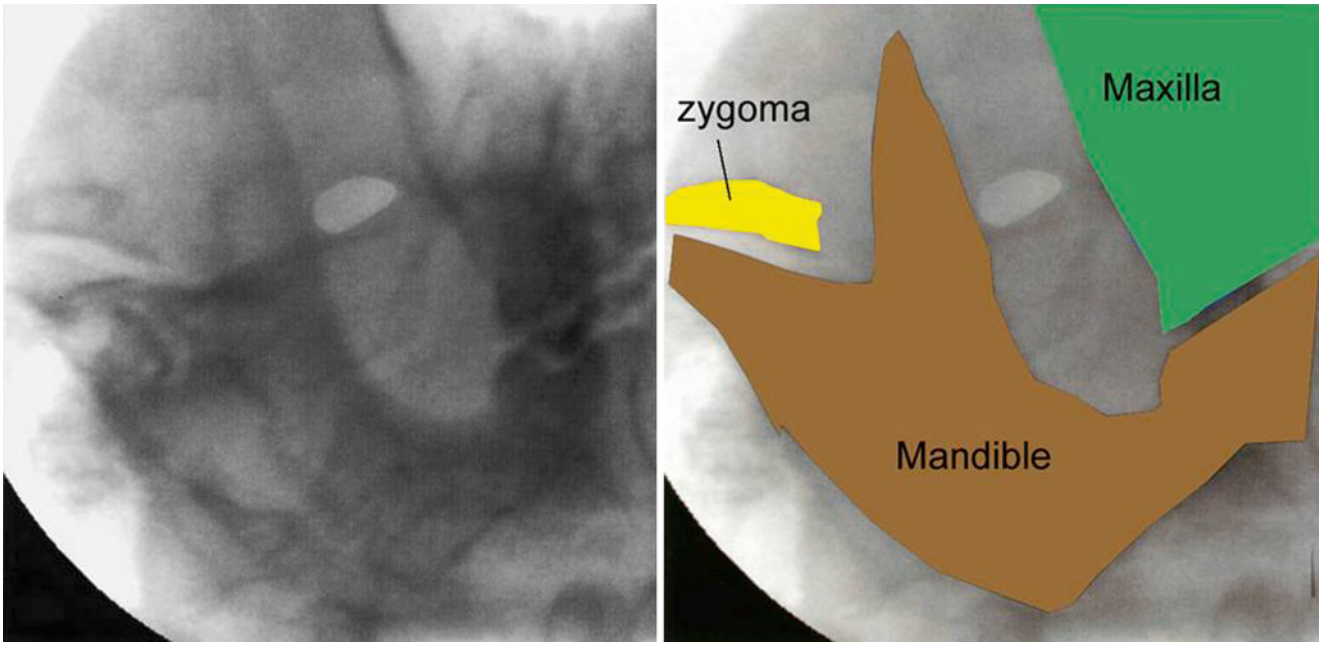


Fig. 11.12 Submentovertex projection with the oval foramen and other relevant structures (fluoroscopic and schematic) (From Sluijter [8], with permission of the editor)

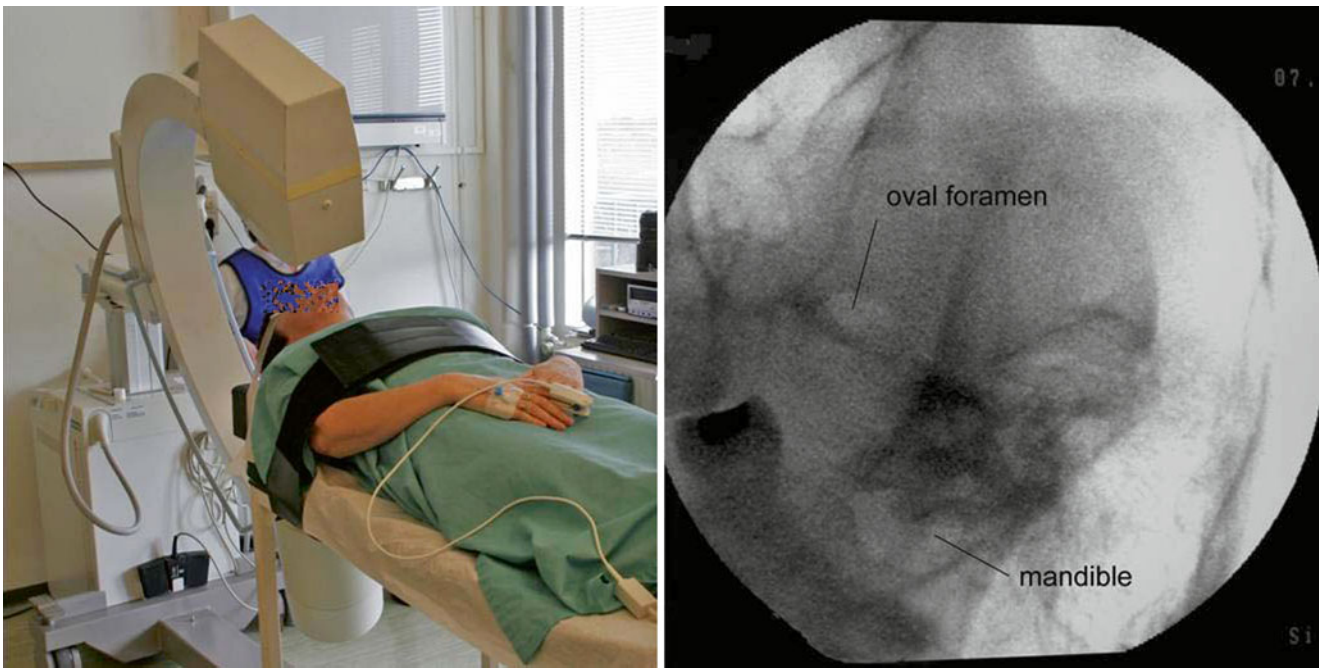


Fig. 11.13 Submentovertex projection, patient position and fluoroscopic localization

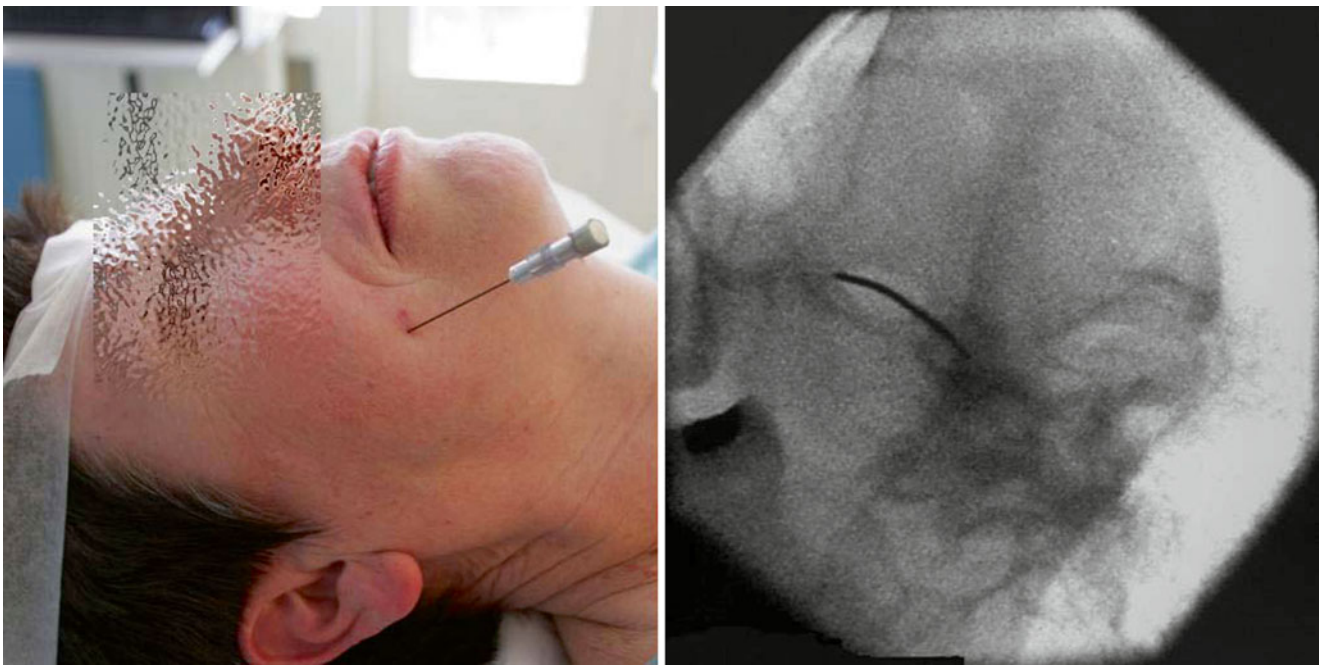


Fig. 11.14 Needle positioning, patient and fluoroscopic

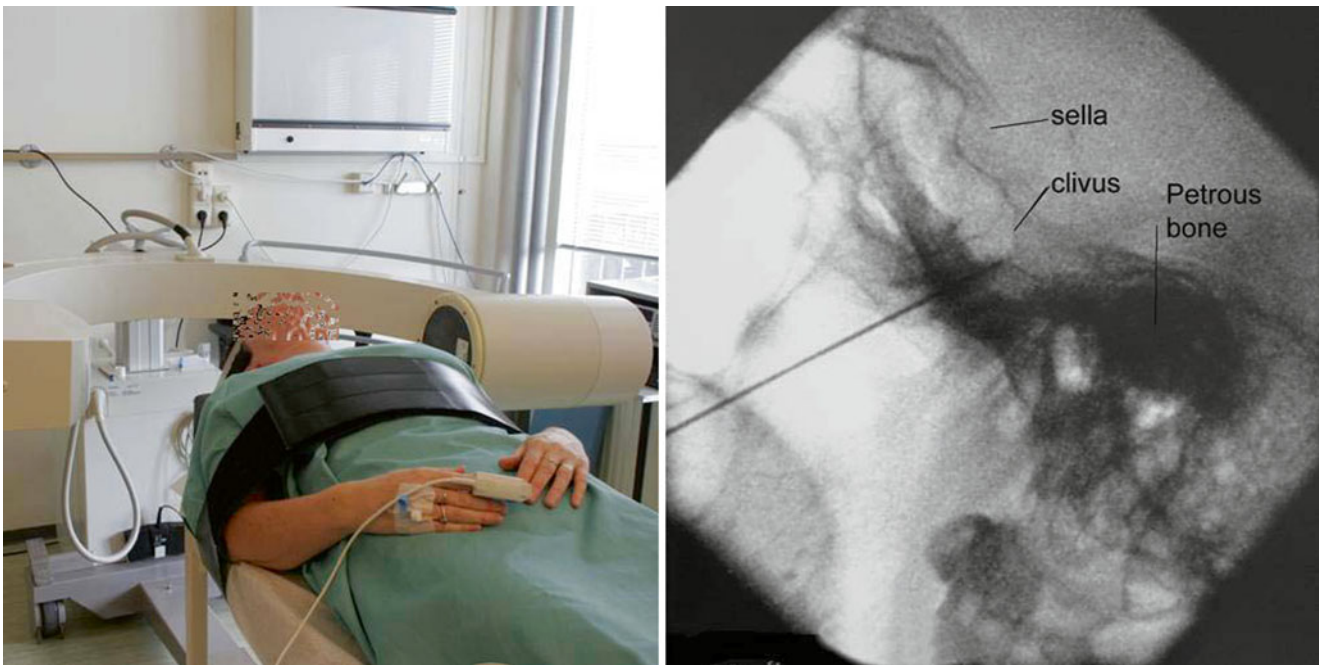


Fig. 11.15 Lateral projection, patient and fluoroscopic

Indications

The only indication for an RF lesioning of the Gasserian ganglion is trigeminal neuralgia. If this technique is performed with the incorrect diagnosis, it may lead to serious problems with intractable dramatic increase in pain. A tumor with intractable pain in the area of the nV may be an indication. In most cases, local tumor growth is the limitation of this indication.

Pulsed radiofrequency treatment is preferred over radiofrequency treatment for atypical facial pain, neuropathic pain in one or more divisions of the trigeminal nerve, postherpetic neuralgia of the trigeminal nerve, and intractable cluster headache. The indications and contraindications are summarized in Table 11.1.

The results of the PRF treatment are still inconclusive, but the authors have achieved some good results in very difficult cases. Further studies are needed.

Table 11.1 Indication and contraindications for (pulsed) radiofrequency treatment of the Gasserian ganglion

Radiofrequency lesioning of the Gasserian ganglion
Trigeminal neuralgia, especially also in MS patients, and in case of tumor growth in the area of nV
Pulsed RF treatment of the Gasserian ganglion
1. Atypical facial pain
2. Neuropathic pain in one or more divisions of the trigeminal nerve
3. Postherpetic neuralgia of the trigeminal nerve
4. Intractable cluster headache
Contraindications
Lack of cooperativeness
Bleeding disorders or use of anticoagulants
Signs of local infection
Signs of local malignancy
Allergy to local anesthetics

Complications and Results

The complication rate depends on experience, making a proper diagnosis, and meticulous performance of the technique. As stated above, annoying hypesthesia and dysesthesia are the most frequent complications. By choosing a 22-G SMK C-10 needle with an active tip of only 2 mm to produce the radiofrequency lesion, in 80 % of cases, there was an excellent result; after a second treatment session using an active tip of 2 or 5 mm, a 96 %; and after a third session, a 99 % success with a mean recurrence time of 2.3 years [19]. Other authors have comparable results; however, if more intense lesions are produced with the use of thicker needles and longer active tips, the complication rate is elevated, but the recurrence time is longer [20]. Other rare complications involve muscular weakness and paralysis, anesthesia dolorosa, keratitis, transient paralysis of cranial nerves III and IV, permanent palsy of the abducens nerve (nVI) [19]. The complications are listed in Table 11.2.

Because of the greater risk of sensory loss and loss of corneal reflex, the treatment of the first division requires more experience. Although the risk of meningitis is very low (<1 %), this is a very serious complication. Patients should be instructed to visit the emergency department without delay if signs of meningitis are present (neck and headache, fever, vomiting). Prompt treatment with i.v. antibiotics should start immediately.

Table 11.2 Reported side effects and complications

Annoying hypesthesia and dysesthesia (1.56 %)
Anesthesia dolorosa (0 %)
Keratitis (0 %)
Not annoying corneal hypesthesia (1.17 %)
Muscular weakness of masticatory muscles (4 %)
Transient paralysis of cranial nerves III and IV (0 %)
Palsy of the abducens nerve (permanent) (0 %)
Meningitis (0 %)
Cheek hematoma (2 %)

From Kanpolat et al. [20]

References

1. Rosen S, Shelesnyak M. Nasogenital relationship II. Pseudopregnancy following extirpation of sphenopalatine ganglion in rat. Endocrinology. 1940;27:463–8.
2. Zacharias L. Further studies in naso-genital relationship: anatomical studies of periphypophyseal region in rat. J Comp Neurol. 1941;74:421–45.
3. Drummond PD. Dissociation between pain and autonomic disturbances in cluster headache. Headache. 1990;30(8):505–8.
4. Sluder G. Injection of the nasal ganglion and comparison methods. In: Nasal Neurology, Headaches and Eye Disorders. Mosby CV, editor. St. Louis; 1918
5. Waldman S. Chapter 5. Sphenopalatine ganglion block: lateral approach. In: Waldman S, editor. Atlas of interventional pain management. 2nd ed. Philadelphia: Saunders; 2004.
6. Sanders M, Zuurmond WW. Efficacy of sphenopalatine ganglion blockade in 66 patients suffering from cluster headache: a 12- to 70-month follow-up evaluation. J Neurosurg. 1997;87(6):876–80.
7. Hildebrandt B, Wust P, Ahlers O, et al. The cellular and molecular basis of hyperthermia. Crit Rev Oncol Hematol. 2002;43(1):33–56.
8. Sluijter ME. Radiofrequency part 2. Meggen (LU): Flivopress, SA; 2003.
9. Stolker R, Kamphuis E, Rohof O, et al. Die Behandlung von Clusterkopfschmerzen mit radiofrequenten Laesionen. Der Schmerz. 1991;5194.
10. Vervest AC, Stolker R, Groen G, et al. Clusterkopfscmerz: Thermolaesion des Ganglion sphenopalatinum. Der Schmerz. 1992;69.
11. Sluijter M, Vercruysse P, Sterk W. Radiofrequency lesions of the sphenopalatine ganglion in the treatment of cluster headache. Der Schmerz. 1988;956–9.
12. Salar G, Ori C, Iob I, et al. Percutaneous thermocoagulation for sphenopalatine ganglion neuralgia. Acta Neurochir (Wien). 1987;84(1–2):24–8.
13. Shah RV, Racz GB. Long-term relief of posttraumatic headache by sphenopalatine ganglion pulsed radiofrequency lesioning: a case report. Arch Phys Med Rehabil. 2004;85(6):1013–6.
14. Bayer E, Racz GB, Miles D, et al. Sphenopalatine ganglion pulsed radiofrequency treatment in 30 patients suffering from chronic face and head pain. Pain Pract. 2005;5(3):223–7.
15. Juniper RP, Glynn CJ. Association between paroxysmal trigeminal neuralgia and atypical facial pain. Br J Oral Maxillofac Surg. 1999;37:444–7.
16. Hakanson S, Linderoth B. Injection of glycerol into the Gasserian cistern for the treatment of trigeminal neuralgia. In: Tasker R, editor. Textbook of stereotactic and functional neurosurgery. New York: McGraw-Hill; 1998. p. 1697–706.
17. Latchaw Jr JP, Hardy Jr RW, Forsythe SB, Cook AF. Trigeminal neuralgia treated by radiofrequency coagulation. J Neurosurg. 1983;59:479–84.
18. Moraci A, Buonaiuto C, Punzo A, et al. Trigeminal neuralgia treated by percutaneous thermocoagulation. Comparative analysis of percutaneous thermocoagulation and other surgical procedures. Neurochirurgia (Stuttg). 1992;35:48–53.
19. Rohof OJJM. Die percutane thermokontrollierte Radiofrequenzlaesion zur Behandlung der Trigeminusneuralgie- eine Langzeitbeobachtung Bonn, Lecture –University of Bonn (personal communication); 2002.
20. Kanpolat Y, Savas A, Bekar A, Berk C. Percutaneous controlled radiofrequency trigeminal rhizotomy for the treatment of idiopathic trigeminal neuralgia: 25- years experience with 1600 patients. Neurosurgery. 2001;48:524–34.

Chapter 12

Occipital Nerves

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng (✉)
Department of Anesthesia, Toronto Western Hospital, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_12, © Springer International Publishing Switzerland 2015

Anatomy

The anterior branches of the first four cervical spinal nerves form the cervical plexus, which is covered by the sternocleidomastoid muscle. The superficial branches of the cervical plexus, which penetrate the cervical fascia and pass to the skin, include the sensory lesser occipital nerve (anterior branch from C2 and C3). This emerges at the posterior edge of the sternocleidomastoid muscle, above its midpoint. It ascends steeply along the splenius capitis muscle and divides into several branches. The areas it supplies include the skin on the upper exterior side of the neck, the upper part of the auricle, and the adjoining skin of the scalp (Figs. 12.1, 12.2, 12.3, and 12.4).

After exiting from the lower edge of the obliquus capitis inferior (or inferior oblique capitis) muscle, the second cervical spinal nerve divides into anterior and posterior branches. The posterior branch of the second cervical spinal nerve passes in a dorsal direction around the obliquus capitis inferior muscle and runs between the occipitovertebral muscles and the semispinalis capitis muscle. Here it divides into three branches: an ascending branch, which supplies the longissimus capitis muscle; a descending branch, which anastomoses with the posterior branch of C3 (the third occipital nerve); and the medial greater occipital nerve (posterior branch of C2).

The sensory greater occipital nerve passes in a cranial direction, goes through the semispinalis capitis muscle and trapezius muscle, and reaches the skin about 2–3 cm away from the midline in the area of the superior nuchal line. It gives off several branches toward the top of the head and extends laterally as far as the ear. The course of its branches follows the branches of the occipital artery.

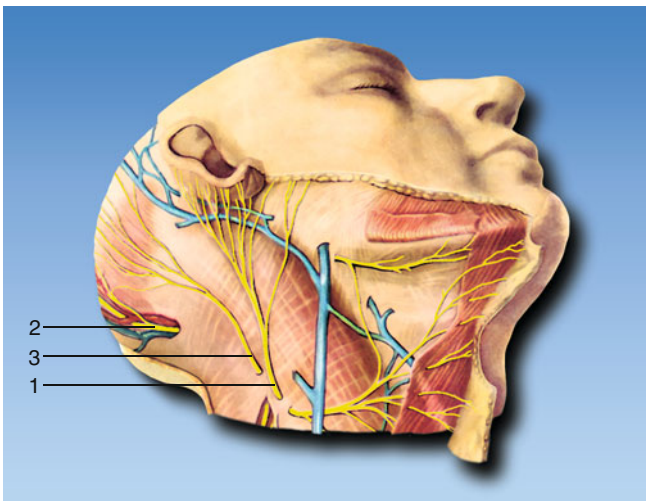


Fig. 12.1 Nerves supplying the surface of the back of the head: (*1*) great auricular nerve, (*2*) greater occipital nerve and occipital artery, and (*3*) lesser occipital nerve (With permission from Danilo Jankovic)

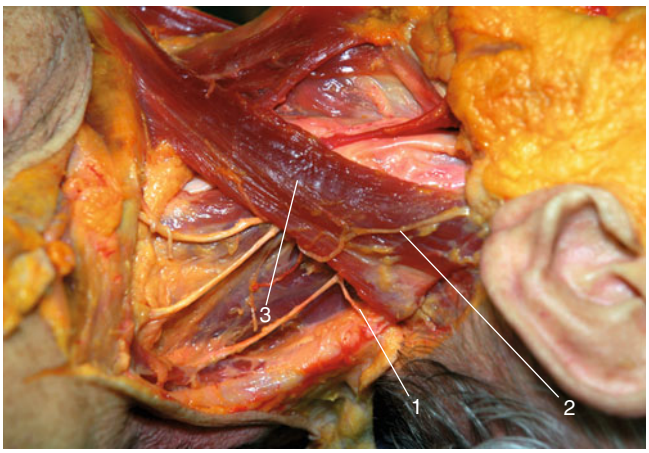


Fig. 12.2 (*1*) Lesser occipital nerve, (*2*) great auricular nerve, and (*3*) sternocleidomastoid muscle (With permission from Danilo Jankovic)

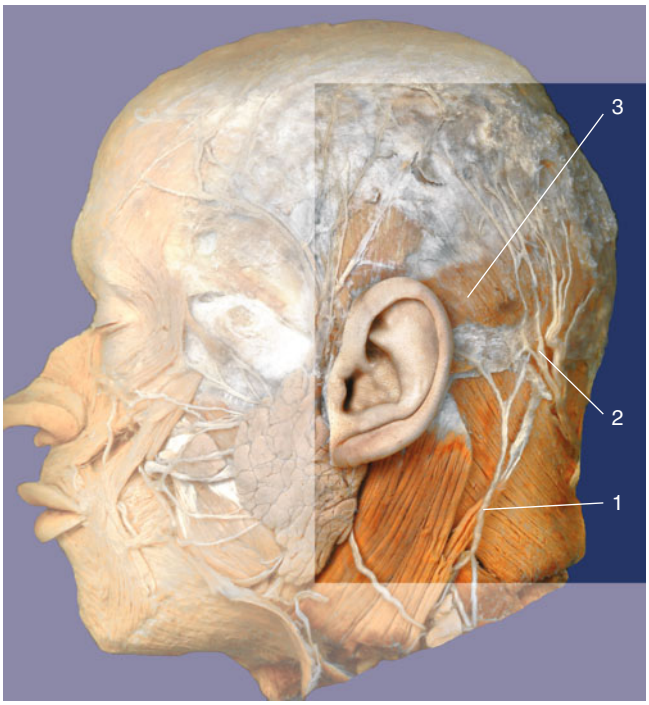


Fig. 12.3 Anatomy. (*1*) Lesser occipital nerve, (*2*) greater occipital nerve and occipital artery, and (*3*) occipital muscle (With permission from Danilo Jankovic)

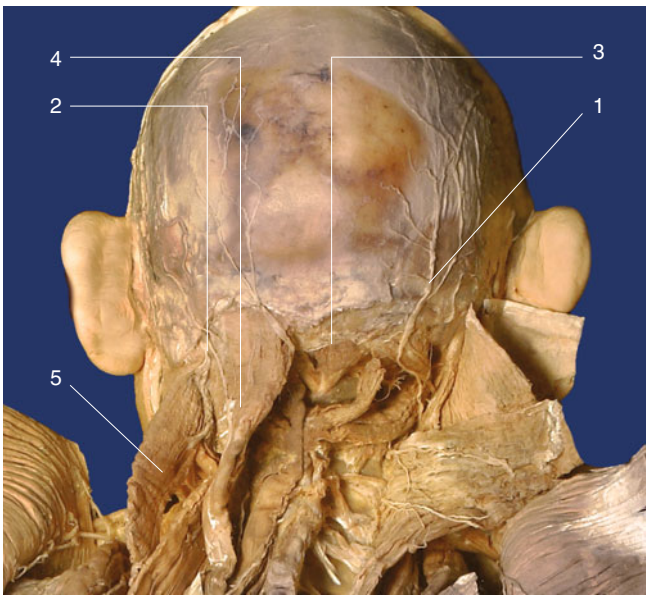


Fig. 12.4 Anatomy. (*1*) Greater occipital nerve, (*2*) the third occipital nerve, (*3*) inferior nuchal line, (*4*) semispinalis capitis muscle, and (*5*) splenius capitis muscle (With permission from Danilo Jankovic)

Sonoanatomy

At the superior nuchal line, the greater occipital nerve is not always seen as it may already branch out into small nerves. The surrogate marker is the greater occipital artery. A high frequency linear ultrasound probe (8–12 MHz) is applied just lateral to the greater occipital protuberance. Because it is very superficial, one should apply minimal pressure at this site to prevent compression of the occipital artery. The ultrasound probe may slide caudally just off the nuchal line, and the greater occipital artery is seen deep to the trapezius muscle (Fig. 12.5).

Other approach for greater occipital nerve is at the C2 level. At this level, the spinal process of C2 is well recognized for its bifid morphology (Fig. 12.6). The ultrasound probe is moved laterally to reveal the muscle layers from deep to superficial: obliquus capitis inferior, semispinalis capitis, splenius capitis, and trapezius muscles. To optimize the scan of the obliquus capitis inferior muscle, the lateral end of the probe is rotated toward the lateral mass of C1 to bring the probe along the long axis of the obliquus capitis inferior muscle. The greater occipital nerve is seen between the obliquus capitis inferior and semispinalis capitis muscles approximately 2–3 cm away from the midline (Fig. 12.7). The vertebral artery and dorsal root ganglion is usually revealed on the lateral side with Doppler scan (Fig. 12.8).

The lesser occipital nerve is the most cephalad branch of the cervical plexus coming out deep and posterior to the sternocleidomastoid muscle. By placing the ultrasound probe 1 fingerbreadth below the superior nuchal line between the sternocleidomastoid and trapezius muscle, the lesser occipital nerve can be seen embedded in the cervical fascia over the splenius capitis muscle (Fig. 12.9).

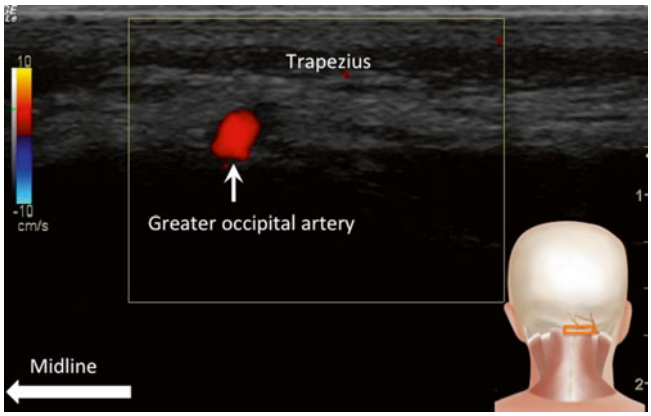


Fig. 12.5 Sonogram of occipital artery just caudal to the superior nuchal line. The artery indicates the plane where the greater occipital nerve lies. The probe position is indicated in the insert lower right corner (Reproduced with permission from Philip Peng Educational Series)

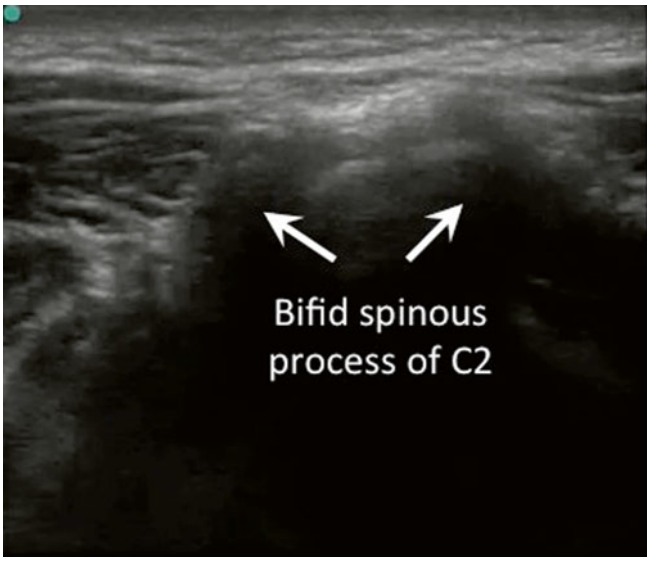


Fig. 12.6 Sonogram showing the bifid spinous process of C2 (Reproduced with permission from Philip Peng Educational Series)

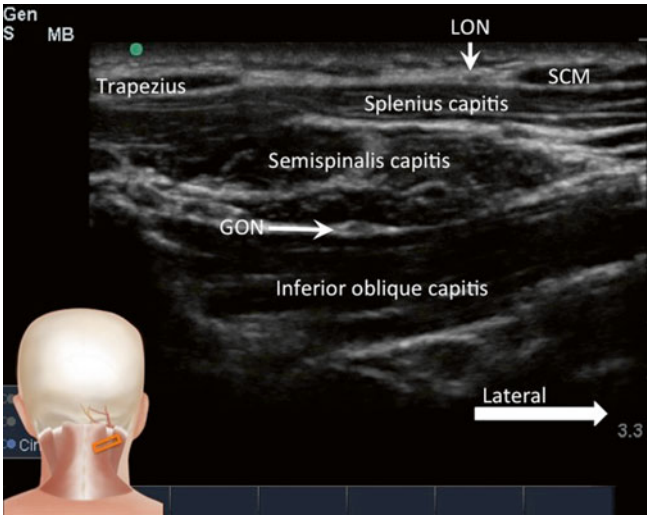


Fig. 12.7 Sonogram at the same level as Fig. 12.6 but the probe is moved in the lateral direction with the lateral end of the probe tilted toward C1 lateral mass. The *insert* shows the probe position. *GON* greater occipital nerve, *LON* lesser occipital nerve, *SCM* sternocleidomastoid muscle (Reproduced with permission from Philip Peng Educational Series)

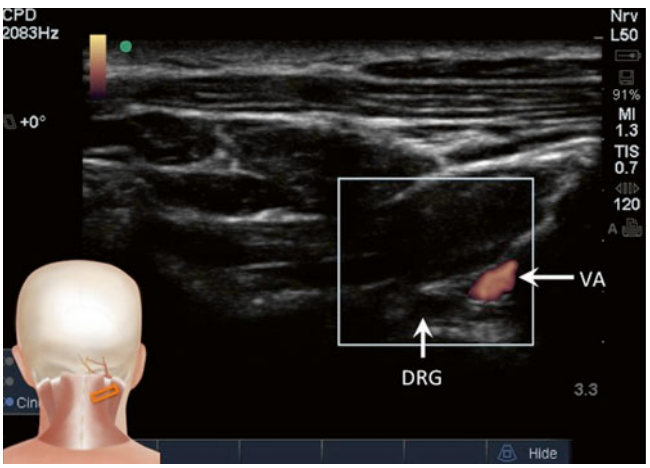


Fig. 12.8 Doppler scan of Fig. 12.7 showing the vertebral artery (*VA*) and the dorsal root ganglion (*DRG*) (Reproduced with permission from Philip Peng Educational Series)

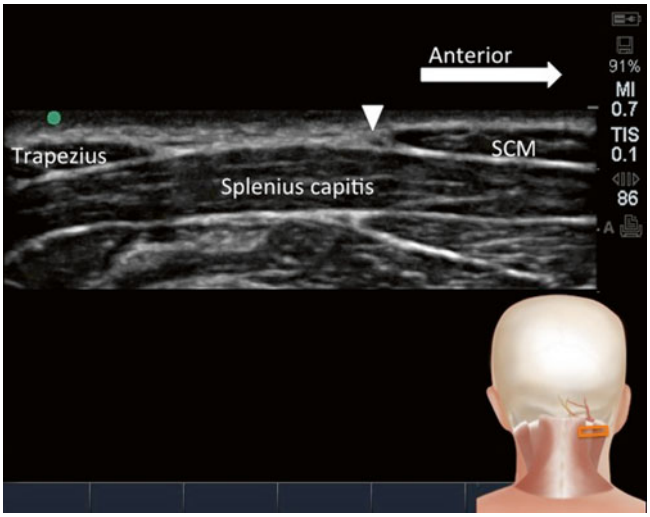


Fig. 12.9 Lesser occipital nerve (*arrow head*). The probe position is shown in the insert and is placed between sternocleidomastoid (*SCM*) and trapezius muscle one fingerbreadth caudal to the superior nuchal line (Reproduced with permission from Philip Peng Educational Series)

Indications

Blocks of the greater and lesser occipital nerves are carried out for prognostic, diagnostic, and therapeutic purposes in patients with painful conditions in the region of the back of the head. It is used as a diagnostic block in examining the differential diagnosis of pain at the back of the head, e.g., in suspected tumors of the posterior cranial fossa. It is commonly performed therapeutically for occipital neuralgia, characterized by pain in the suboccipital area and back of the head, or other headache.

Most of the studies evaluating the efficacy of the greater occipital nerve block in headache management are either of low quality or lack a control group. However, those trials showed that the duration of analgesic effects lasted far beyond the duration of action of the local anesthetic and sometimes the duration can be of few weeks. Efficacy was observed in migraine and cluster headache, but not in facial pain or tension-type headache.

Procedure

Landmark-Guided Technique

With emergency equipment available and sterile precautions for procedure, the patient is placed in sitting position with head tilted forward slightly. The key landmarks for greater occipital nerve injection are the occipital artery and superior nuchal line. The needle is inserted about 2.5 cm from the midline, directly medial to the easily palpable occipital artery. It is advanced at a slightly cranial angle (Fig. 12.10) between the insertions of the trapezius and semispinalis muscles until bone contact is made. After minimal withdrawal and aspiration, the local anesthetic is injected.

The injection of the lesser occipital nerve is carried out about 2.5 cm lateral to the puncture point described above (Fig. 12.11). Bone contact is also sought at a slightly cranial angle, and the needle is withdrawn a little, followed by aspiration and injection. The sensory areas supplied by these two nerves are shown in Fig. 12.12.

If the injection is used for diagnostic purpose, the volume in general is 0.5–1 mL local anesthetic (e.g. 0.5–1 % prilocaine, mepivacaine, or lidocaine). A higher volume (1–1.5 mL) is used for therapeutic block (e.g., 0.75 % ropivacaine, 0.5 % bupivacaine) and is often mixed with steroid (e.g., 1–2 mg dexamethasone or 20 mg triamcinolone). When there is evidence of improvement in the symptoms, a series of injections (e.g., 8–12 blocks) are indicated.

Ultrasound-Guided Technique

At the level of the superior nuchal line, the occipital artery is the surrogate landmark of the greater occipital nerve and a 1-in. 25-G needle is inserted out-of-plane to deposit the local anesthetic on either sides of the artery.

At the level of C2, a 22-G 3.5-in. needle is inserted in-plane from lateral to medial direction toward the fascial plane between the obliquus capitis inferior and semispinalis capitis muscles besides the greater occipital nerve. The nerve is usually associated with an artery and an absence of spread of injectate may suggest intravascular injection. Because the vertebral artery is deep on the lateral side, lateral-to-medial approach is advisable.

For the lesser occipital nerve, an in-plane approach from lateral to medial is used. The volume of injectate for both nerves is the same as landmark-guided technique.

Complications

Hematoma.
Inadvertent intra-arterial injection may occur, extremely rarely.

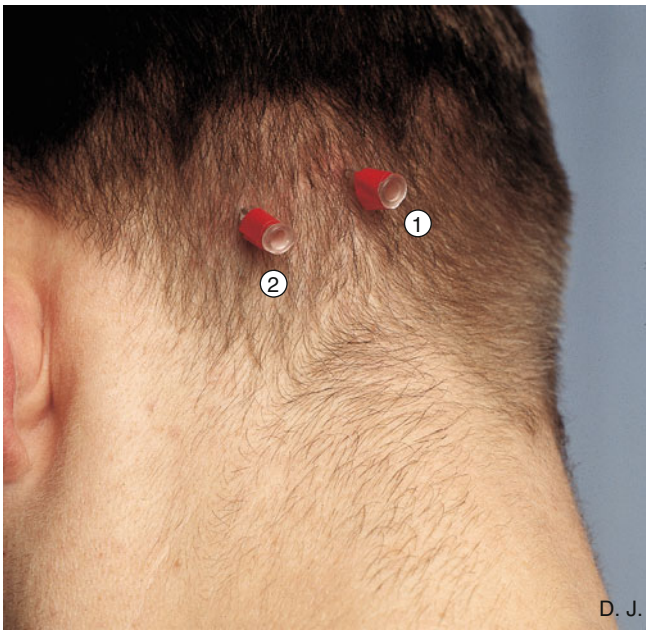


Fig. 12.10 Slightly cranial angle of the needles for blocks of the greater occipital nerve (*1*) and lesser occipital nerve (*2*) (With permission from Danilo Jankovic)

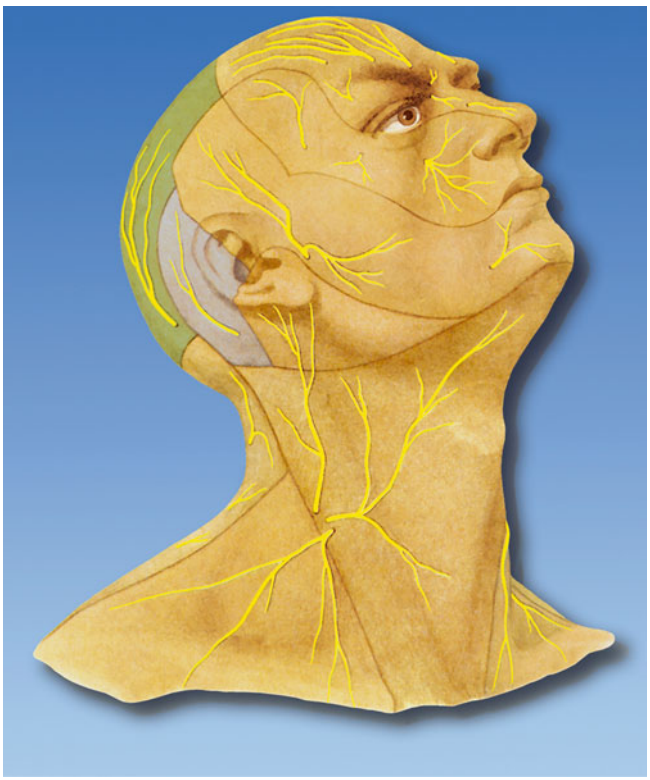


Fig. 12.12 Areas supplied by the greater occipital nerve (*green*) and lesser occipital nerve (*blue*) (With permission from Danilo Jankovic)

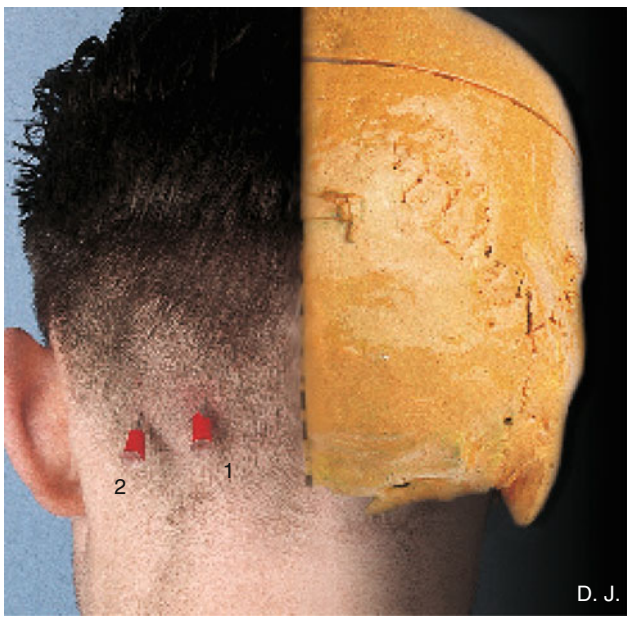


Fig. 12.11 Puncture points: (*1*) greater occipital nerve and (*2*) lesser occipital nerve (With permission from Danilo Jankovic)

Suggested Reading

1. Afridi SK, Shields KG, Bhola R, Goadsby PJ. Greater occipital nerve injection in primary headache syndromes–prolonged effects from a single injection. CA: Pain. 2006;122:126–9.
2. Ambrosini A, Vandenheede M, Rossi P, Aloj F, Sauli E, Pierelli F, Schoenen J. Suboccipital injection with a mixture of rapid- and long-acting steroids in cluster headache: a double-blind placebo-controlled study. Pain. 2005;118:92–6.
3. Ashkenazi A, Blumenfeld A, Napchan U, Narouze S, Grosberg B, Nett R, DePalma T, Rosenthal B, Tepper S, Lipton RB. Peripheral nerve blocks and trigger point injections in headache management – a systematic review and suggestions for future research. Headache. 2010;50:943–52.
4. Curatolo M. Greater occipital nerve. In: Peng, editor. Ultrasound for pain medicine intervention: a practical guide, Peripheral structures, vol. 1. Philip Peng educational series.1st ed. iBook, California CA: Apple Inc.; 2013. p. 46–50
5. Greher M, Moriggl B, Curatolo M, Kirchmair L, Eichenberger U. Sonographic visualization and ultrasound-guided blockade of the greater occipital nerve: a comparison of two selective techniques confirmed by anatomical dissection. Br J Anaesth. 2010;104:637–42.
6. Jankovic D. Occipital nerves. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Malden: Blackwell; 2004. p. 30–2.
7. Jurgens TP, Muller P, Seedorf H, Regelsberger J, May A. Occipital nerve block is effective in craniofacial neuralgias but not in idiopathic persistent facial pain. J Headache Pain. 2012;13:199–213.
8. Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988.
9. Peres MF, Stiles MA, Siow HC, Rozen TD, Young WB, Silberstein SD. Greater occipital nerve blockade for cluster headache. Cephalalgia. 2002;22:520–2.
10. Pernkopf E. Atlas der topographischen und angewandten Anatomie des Menschen. (1.Bd.) Kopf und Hals, (2.Aufl.). München/Wien/Baltimore: Urban, Schwarzenberg; 1980.
11. Tilscher H, Eder M. Infiltrationstherapie. Stuttgart: Hippokrates; 1994.
12. Young WB. Blocking the greater occipital nerve: utility in headache management. Curr Pain Headache Rep. 2010;14:404–8.

Chapter 13

Stellate Ganglion Block

Anuj Bhatia (✉)
Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada
e-mail: Anuj.bhatia@uhn.ca

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, Toronto Western Hospital, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_13, © Springer International Publishing Switzerland 2015

Introduction

Stellate ganglion refers to the confluence of inferior cervical and the first thoracic sympathetic ganglia. Stellate ganglion block (SGB) is performed for the management of a variety of pain conditions, including complex regional pain syndrome, refractory angina, and ischemic pain in the upper limb from peripheral vascular disease [7, 24, 25]. More recently, preoperative SGB has been shown to reduce postoperative pain and analgesic requirements [15].

Anatomy

The peripheral sympathetic nerve supply to the head and neck is derived from preganglionic neurons whose cell bodies are located in the anterior lateral horn of the first and second thoracic spinal cord segment. The axons pass via the anterior roots of the same spinal nerve levels through the rami communicantes to join the upper cervical sympathetic ganglia: superior, middle, intermediate, and inferior. From these ganglia the postganglionic axons pass upward along the internal and external carotid and vertebral arteries to the structures within the cranium (Figs. 13.1, 13.2, 13.3, 13.4, 13.5, 13.6, 13.7, 13.8, and 13.9). The axon may also join the gray rami communicantes; the latter join the cervical nerve supply to the neck and the upper extremity (the cervical portion of the brachial plexus). The stellate ganglion, formed by fusion of the inferior cervical and first thoracic ganglion, extends from the level of the head of the first rib to the inferior border of the transverse process of the seventh cervical vertebra (C7) and lies immediately adjacent to the dome of pleura and behind the subclavian artery. The postganglionic fibers from the stellate ganglion to the cervical nerves (seventh and eighth) and the first thoracic nerve provide sympathetic innervation to the upper limbs [8, 11, 27, 29]. The stellate ganglion is present in only 80 % of the population, so a more correct term for SGB is a cervicothoracic sympathetic trunk (CST) block [7]. The stellate ganglion is oval in shape and measures 2.5 cm long, 1 cm wide, and 0.5 cm thick. The stellate ganglion lies lateral to the longus colli muscle [12, 23]. The CST is located dorsal to the posterior fascia of the carotid sheath anteriorly and is embedded in the prevertebral fascia [6]. Since all the sympathetic flow to the head and neck structures either synapse here at the stellate ganglia or pass through it to the more cephalic sympathetic ganglia, SGB provides a more complete sympathetic denervation of the head and neck.

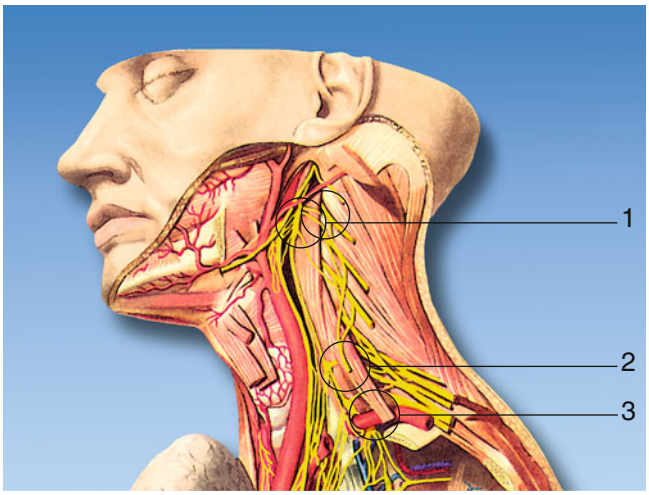


Fig. 13.1 The cervical ganglion trunk: (*1*) superior cervical ganglion, (*2*) middle cervical ganglion, and (*3*) cervicothoracic ganglion (With permission from Danilo Jankovic)

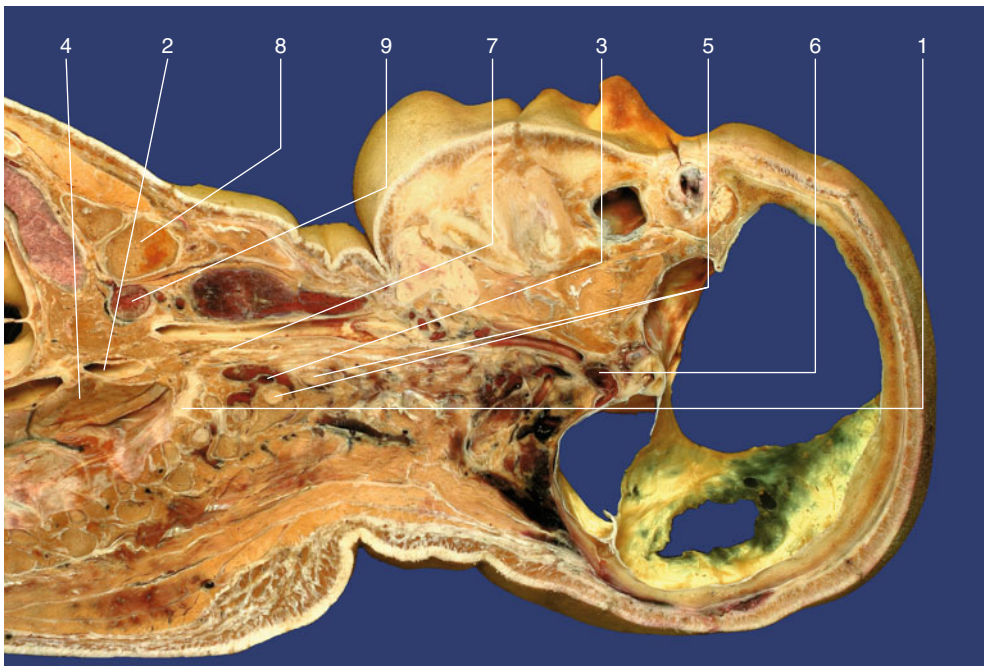


Fig. 13.2 Paramedian sagittal dissection (head and thorax): (*1*) stellate ganglion, (*2*) the subclavian artery, (*3*) the vertebral artery, (*4*) pleura, (*5*) the brachial plexus, (*6*) the carotid artery, (*7*) the vagus nerve, (*8*) clavicle, (*9*) V. brachiocephalica (With permission from Danilo Jankovic)

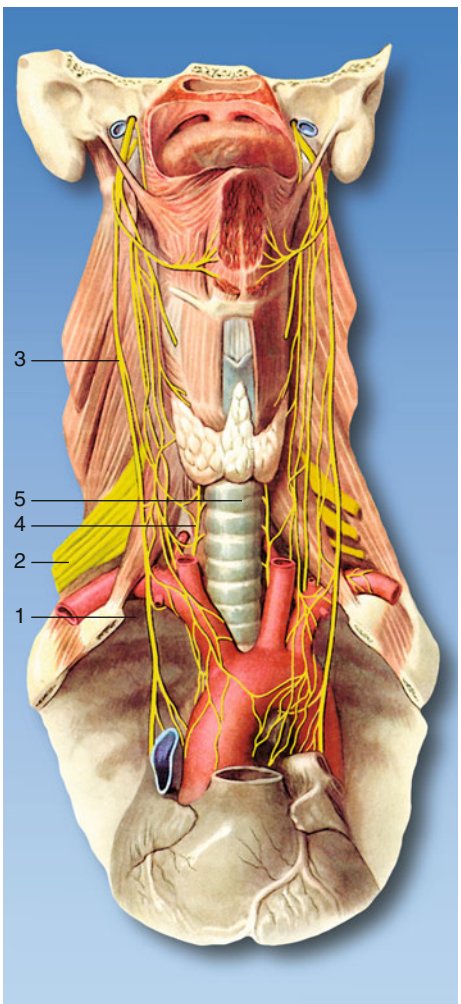


Fig. 13.3 The immediate vicinity of the stellate ganglion: (*1*) pleura, (*2*) brachial plexus, (*3*) vagus nerve, (*4*) recurrent laryngeal nerve, (*5*) trachea (With permission from Danilo Jankovic)

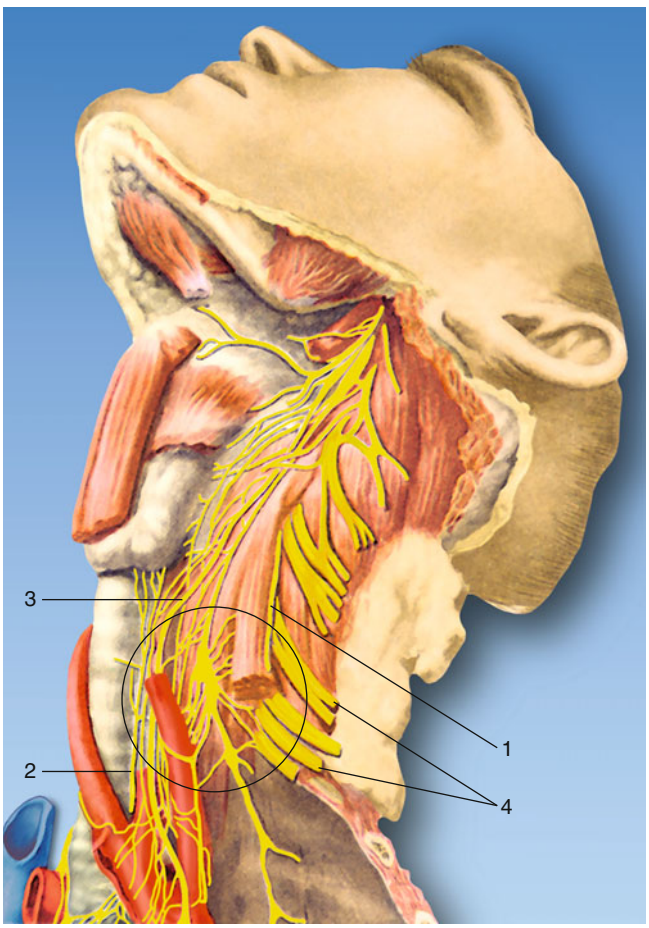


Fig. 13.4 Close anatomical connections in the ganglion trunk include those to (*1*) the phrenic nerve, (*2*) the recurrent laryngeal nerve, (*3*) the vagus nerve, and (*4*) the brachial plexus (With permission from Danilo Jankovic)

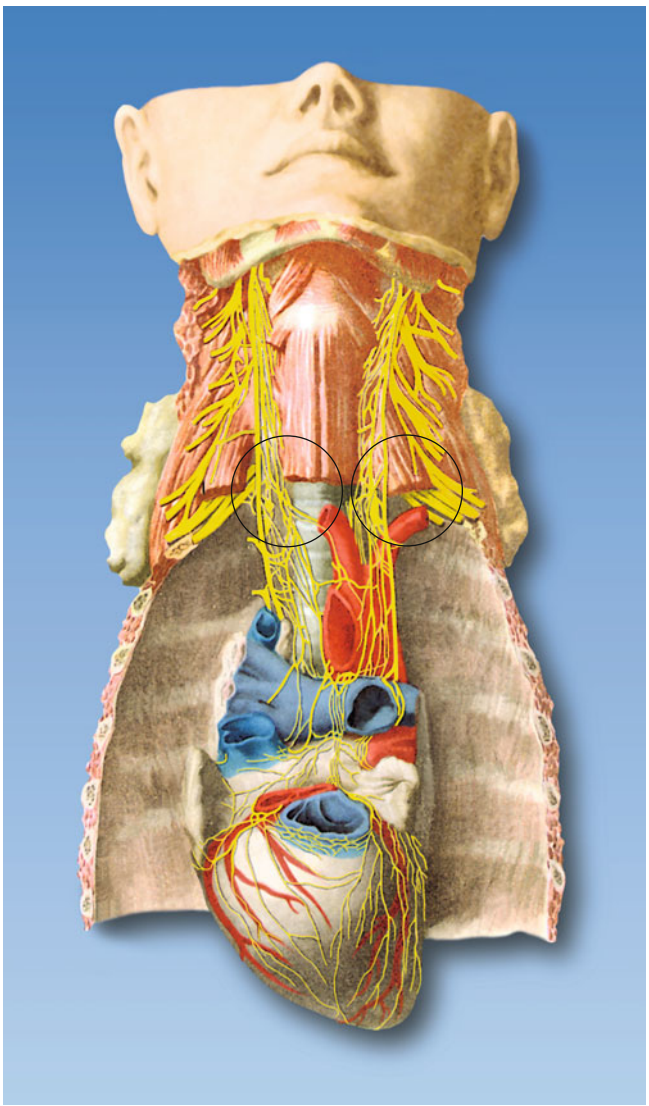


Fig. 13.5 Fibers from the gray rami communicantes (*circles*) supply the heart, esophagus, airways, and thymus (With permission from Danilo Jankovic)

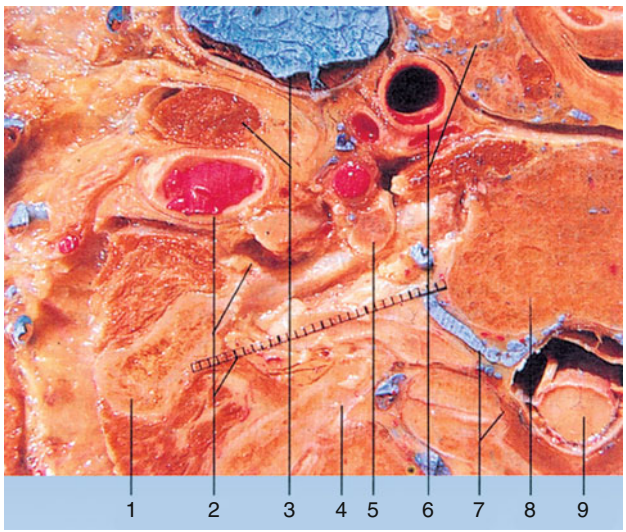


Fig. 13.6 The immediate vicinity of the ganglion (transverse section). (*1*) First rib, (*2*) subclavian artery and scalenus anterior muscle, (*3*) jugular vein, (*4*) second rib, (*5*) cervicothoracic ganglion, (*6*) common carotid artery and thyroid gland, (*7*) T2 intervertebral artery and zygapophyseal joint, (*8*) T2 vertebral body, (*9*) spinal medulla. The average size of the cervicothoracic ganglion is 25 mm × 3–10 mm × 5 mm (With permission from Danilo Jankovic)

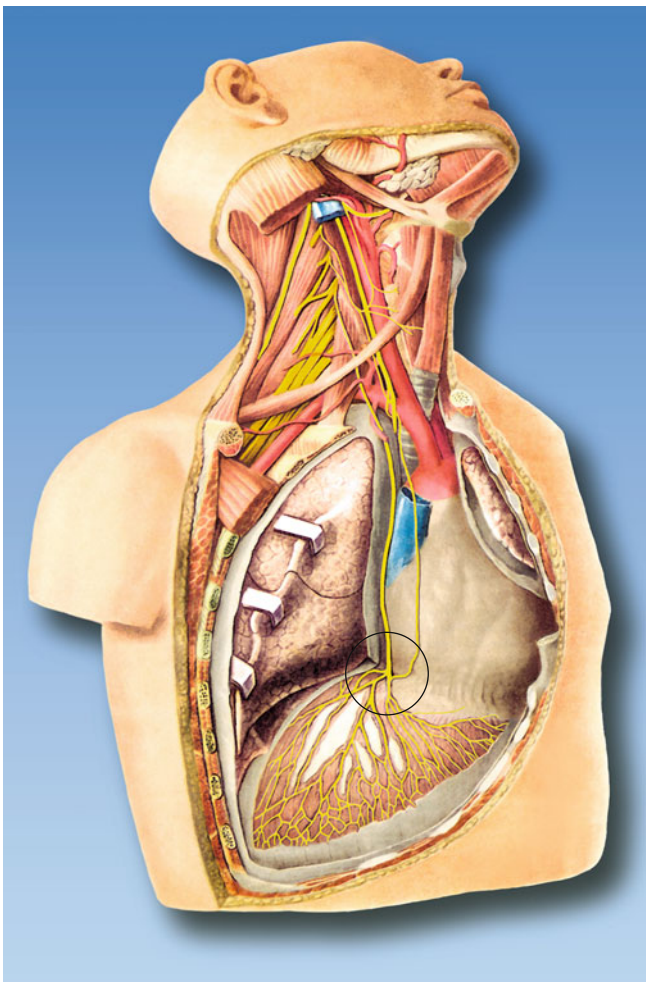


Fig. 13.7 Course of the phrenic nerve (*circle* shows the end position of the phrenic nerve) (With permission from Danilo Jankovic)

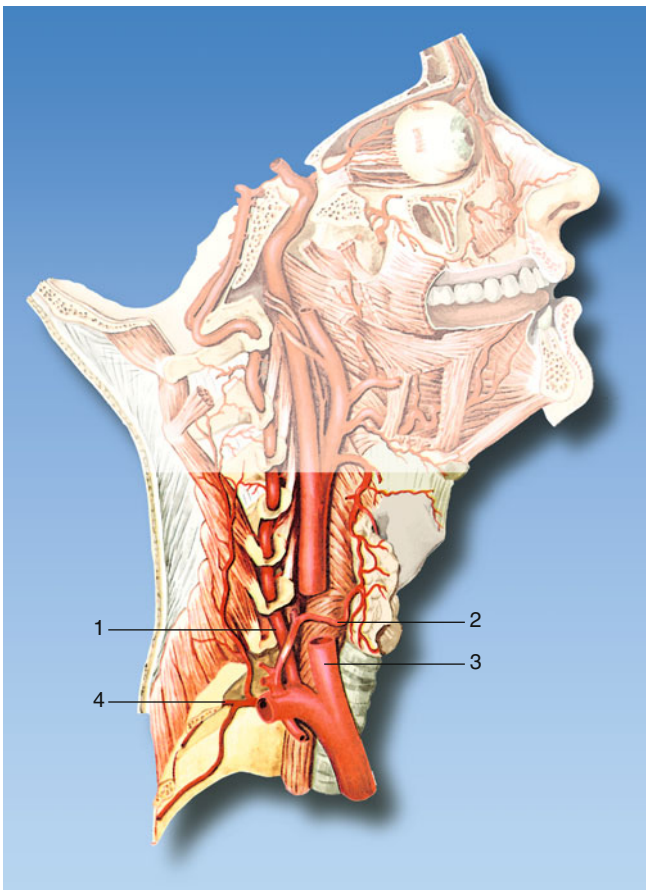


Fig. 13.9 Risk of intravascular injection into (*1*) the vertebral artery, (*2*) the inferior thyroid artery, (*3*) the carotid artery, and (*4*) first intercostal artery (With permission from Danilo Jankovic)

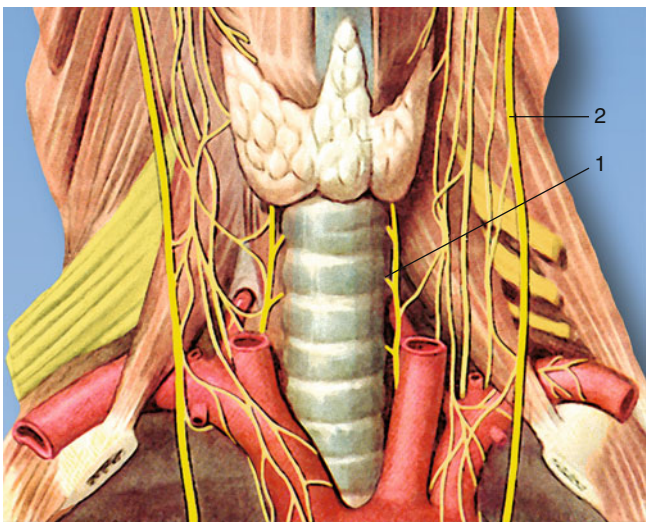


Fig. 13.8 Positions of (*1*) the recurrent laryngeal nerve and (*2*) the vagus nerve (With permission from Danilo Jankovic)

Traditional Approach for SGB

The most widely practiced approach to SGB is the anatomic-landmark- or fluoroscopy-guided paratracheal approach, in which the needle is inserted toward the anterior tubercle of the sixth (Chassaignac tubercle) cervical vertebra (Figs. 13.10 and 13.11). This approach is essentially a blockade of the cervical sympathetic chain in proximity to the middle cervical ganglion instead of the stellate ganglion, which is located opposite to the neck of the first rib. Thus, the classical approach is a cervical sympathetic trunk block rather than SGB.

There are significant limitations and potential hazards associated with traditional approaches. The cephalocaudal extent of the Chassaignac tubercle can be as narrow as 6 mm [13], and it can be easily missed with needle advancement with conventional techniques.

Possible consequences of non-ultrasound-guided approaches for SGB are:

1. *Potential for penetration of vascular structures and intravascular injection*:

   Retropharyngeal and cervicomediastinal hematomas after SGB has been reported despite negative aspiration of blood, and these can cause severe airway compromise [10, 20, 26]. Kapral et al., in one of the earliest papers examining US guidance, reported hematomas in three out of 12 patients who received SGB without US guidance [14]. Possibility of other arteries at risk (e.g. the ascending cervical branch of the inferior thyroid artery, transverse cervical artery) that traverse over the C6 anterior tubercle has also been mentioned [18]. Siegenthaler and colleagues found that the vertebral or other arteries were located in the needle path for traditional approach for SGB in over 28 % of subjects [22], while Bhatia and colleagues reported that a major vessel was observed in up to 29 and 43 % of patients at the C6 and C7 levels, respectively. It was also noted that the vertebral artery was outside the foramen transversarium in 7 % of subjects at the C6 level [2], and this has also been shown in other studies [4, 16]. A modified fluoroscopy-guided oblique approach has been proposed to reduce the risk of vertebral artery puncture as the needle is directed to the junction of the uncinate process and the vertebra body [1]. However, this technique directs the needle much closer to the esophagus (see below).
2. *Potential for penetration of esophagus*, *pleura*, *lateral lobes of thyroid gland*, *and cervical nerve roots*:

   Two recent studies have indicated that the esophagus is frequently located in the needle path of SGB performed using traditional approaches. The esophagus was located along the needle path in 37–50 % and 65–74 % of subjects at the C6 and C7 levels, respectively, in these studies [2, 22]. The risk of esophageal penetration is greater than on the left side because of the anatomical location of the esophagus. Esophageal puncture can result in mediastinitis especially if the patient has an unrecognized diverticulum.

   Pneumothorax is also a potential complication with anatomic-landmark- or fluoroscopy-guided techniques, especially if SGB is performed at the C7 level. Finally, a needle traversing through the thyroid gland can result in a hematoma, and exiting cervical nerve roots can also be traumatized during SGB.

Use of ultrasound enables the operator to visualize blood vessels, esophagus, pleura, nerve roots, and thyroid, and this can help avoid penetration of these structures [3, 17].

In addition to the risks of potential complications with traditional approaches for SGB, precision in deposition of the injectate and adequacy of its spread to the first and second thoracic vertebral levels are key considerations for ensuring efficacy. The location of CST is in the loose connective tissues of the prevertebral fascia. However, traditional approaches rely on contact with bony landmarks (transverse processes of C6 or C7) followed by withdrawal of the needle by a few millimeters and then injection. The spread of injectate with these approaches has been shown to be anterior to the prevertebral fascia and in the paratracheal space in most patients, without much caudal spread [12], whereas subfascial injection results in more caudal spread, higher rate of sympathetic block of the upper limbs, and lower incidence of blockade of vagus or recurrent laryngeal nerve (causing hoarseness) [5, 21].

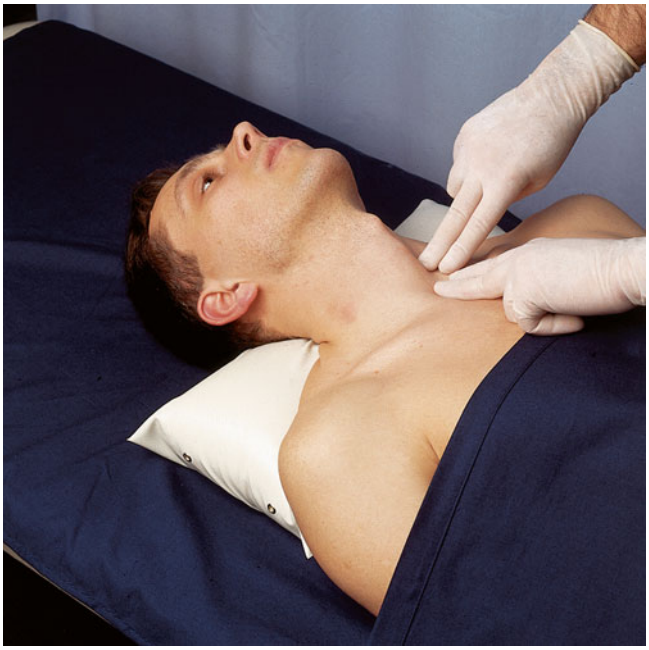


Fig. 13.10 Two-finger method of locating the level of C7 (With permission from Danilo Jankovic)

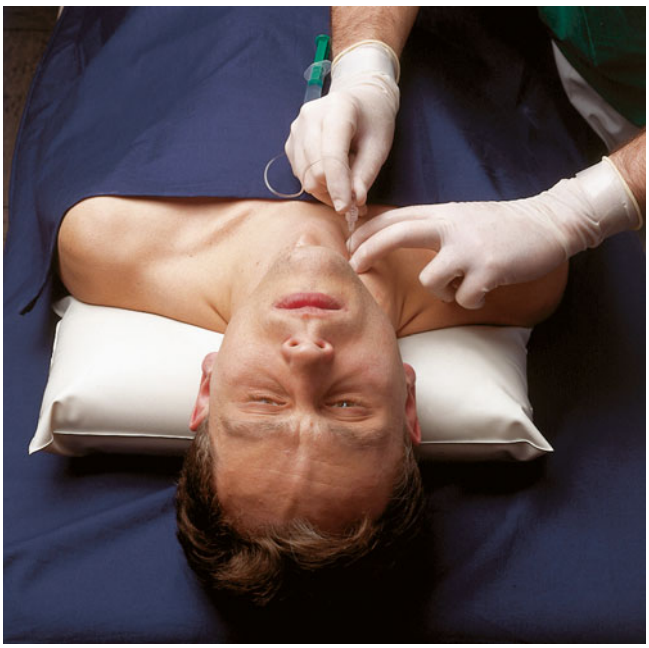


Fig. 13.11 Introducing the needle (With permission from Danilo Jankovic)

Sonoanatomy and Injection Technique for Ultrasound-Guided Stellate Ganglion Block

The patient is placed in the semi-lateral position with the procedure side nondependent and the neck in slight extension. A high-frequency linear US probe (6–15 MHz) is used and a probe with a small footprint is desirable. The probe placed transversely at the level of cricoid cartilage and the transverse process (TP) of the cervical vertebra is identified. If the TP has a prominent anterior tubercle and a smaller posterior tubercle, then this is likely to be the C6 level, but scanning should be continued in a caudal direction to allow recognition of the TP at C7 level that has only a posterior tubercle. The vertebral artery can easily be identified in cross section, its location being deeper and lateral to the common carotid artery at C7 level. Once the TP of the C6 vertebra has been identified, the longus colli muscle is identified in cross section (Fig. 13.12). This muscle is located anterior to the TP and medial to the anterior tubercle and is around 1 cm thick at this level [9]. The prevertebral fascia on the anterior surface of the muscle deep to the longus capitis muscle is then identified. Other important structures to recognize include the lateral lobe of the thyroid gland, blood vessels including the common carotid artery and the internal jugular vein, esophagus, exiting cervical nerve root, and any vessels that may be in the planned path of the injecting needle (Figs. 13.13 and 13.14).

The authors' preferred approach is the lateral, in-plane approach [9] in which the tip of the needle is directed to the prevertebral fascia between the carotid artery and the tip of C6 anterior tubercle. This needle path avoids traversing the lateral lobe of the thyroid gland. The US probe is adjusted (by putting more pressure on the medial end of the probe) so that the cervical nerve root is "removed" from the view – this reduces probability of encountering the nerve root while the needle is advanced. The internal jugular vein can be avoided by "pushing" away with the needle. A 25-gauge needle that is 4 cm in length or a spinal needle (8 cm length) can be used for this procedure. The alternative US-guided approach is an out-of-plane approach in which the patient is supine and the needle is directed at the prevertebral fascia on the surface of longus colli. This approach involves penetration of the thyroid gland and should be used only if the lateral approach is unsafe or nonviable because of anatomic variations. Irrespective of the approach, a pre-scan Doppler injection is advised to check for any vessels in the path of the needle.

Since there are two layers of prevertebral fascia and the cervical sympathetic chain is embedded inside the fascia, the needle tip should be placed deep to the prevertebral fascia (to avoid spread along the carotid sheath) but superficial to the fascia investing the anterolateral surface of the longus colli muscle (to avoid injecting into the muscle substance) [19]. Once the needle is in this position, hydrodissection with 0.9 % saline is recommended to ensure that the needle is in the correct plane (Fig. 13.15). This is followed by slow injection of a maximum of 5 mL of 0.5 % bupivacaine in 1:200,000 epinephrine. This volume has been shown to be adequate for spread from C4 to the first thoracic vertebral level [9].

Visualization of the spread of injectate under real-time scanning is important, as the absence of this may suggest unsuspected intravascular injection. Continuous hemodynamic and respiratory monitoring (ECG, blood pressure, pulse oximetry) is recommended during and for 5–10 min after the injection. A volume of 0.1 mL should be injected initially, and injection should be continued only if there is no evidence of intravascular spread (patient may report tinnitus, tingling or numbness around the lips and tongue, and lightheadedness, and signs include tachycardia, hypertension, and seizures). If the SGB is performed for relieving sympathetically mediated pain in the upper extremity, then skin temperature probes should be placed on both upper limbs prior to the procedure. An increase of 1–3 C is usually accepted as a sign of adequate sympathetic blockade though this has been contested in recent literature [28].

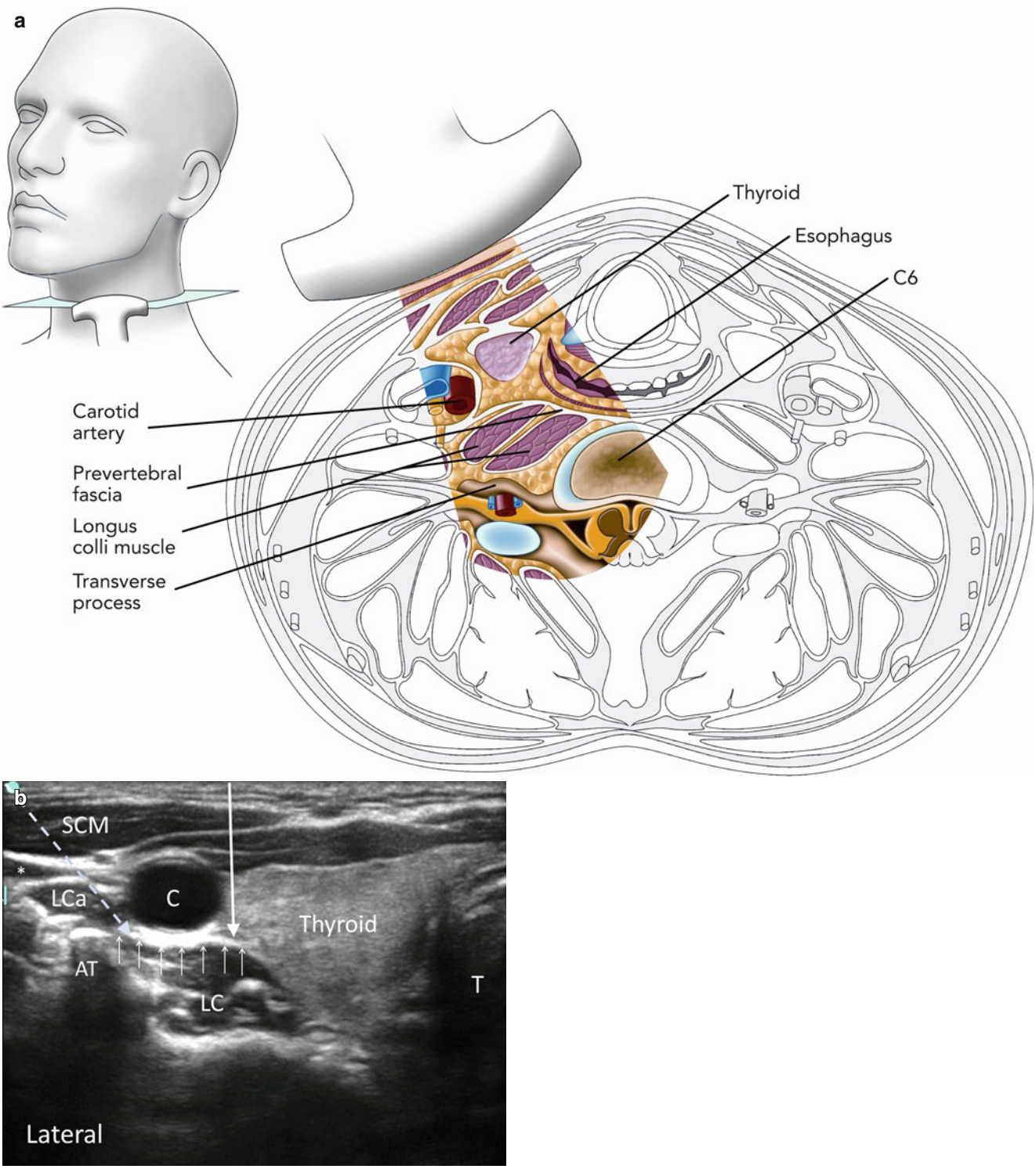


Fig. 13.12 (**a**) Cross section of the neck at the sixth cervical vertebral level correlating with the ultrasonographic image. (**b**) Ultrasonographic image of neck at C6. *C6* sixth cervical vertebra, *C* carotid artery, * internal jugular vein (compressed), *SCM* sternocleidomastoid muscle, *LC* longus colli muscle, *LCa* longus capitis muscle, *T* airway, *AT* anterior tubercle. The prevertebral fascia is marked by *small solid arrows*. The needle paths of anterior and lateral approach are marked by *long solid and dotted arrow*, respectively (Reproduced with permission from Philip Peng Educational Series)

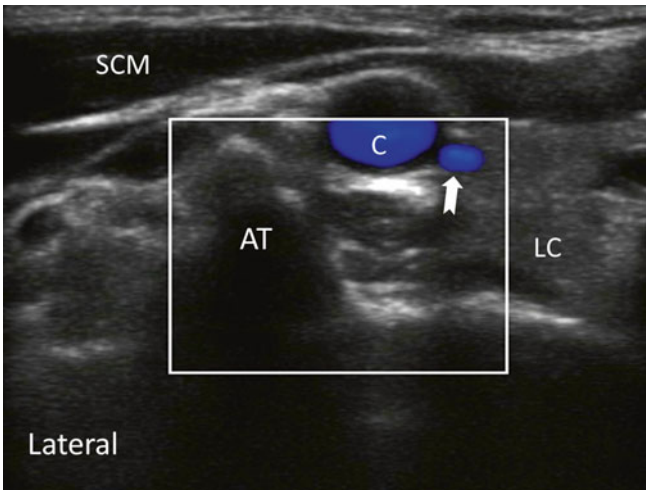


Fig. 13.13 Ultrasonographic image of neck at C6 level on the right side showing a vessel in the short axis (*bold arrows*). *LC* longus colli muscle, *C* carotid artery, *SCM* sternocleidomastoid muscle, *AT* anterior tubercle (Reproduced with permission from Philip Peng Educational Series)

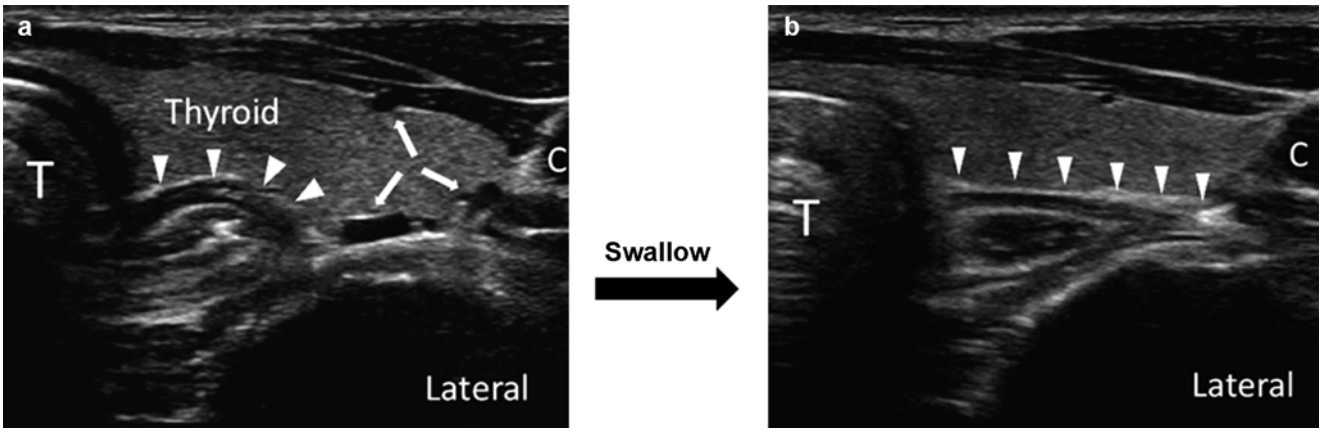


Fig. 13.14 Ultrasonographic image of neck at C7 level showing the variation of position of esophagus with swallowing. (**a**) Before swallowing, the esophagus (*arrow heads*) was seen covering half of the distance between trachea (*T*) and carotid artery (*C*); (**b**) during swallowing, the esophagus moved laterally toward the carotid artery, virtually covering the whole area between trachea and carotid artery. Note that the *bold arrows* showed the presence of three vessels in the pre-swallow scan. Swallowing action was evident by the increase in hyperechogenic shadow in the trachea (Reproduced with permission from Philip Peng Educational Series)

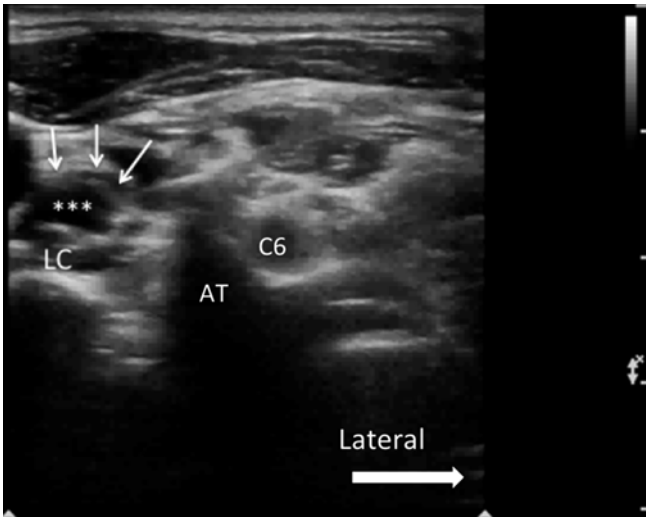
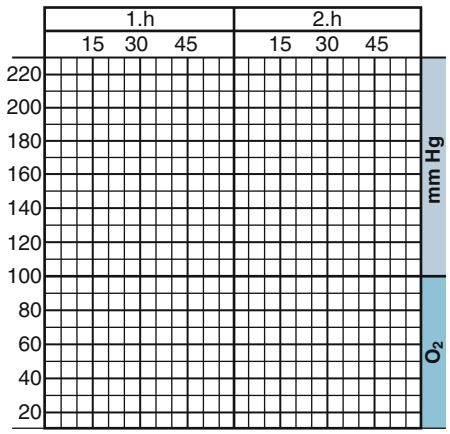


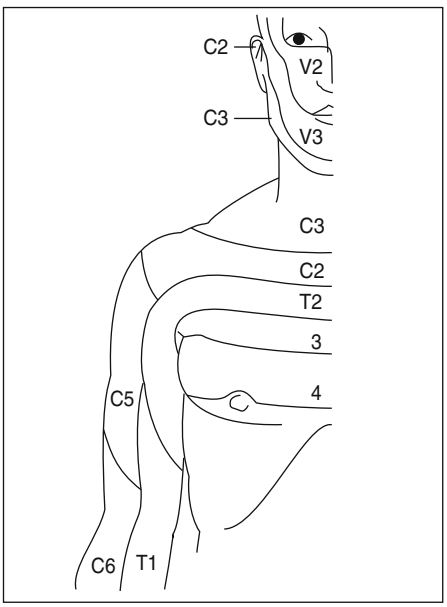
Fig. 13.15 Ultrasonographic image of neck at C6 level showing the local anesthetic spread within the prevertebral fascia following the injection. *C6* C6 nerve root, *AT* anterior tubercle of C6, *LC* longus colli muscle, *arrows* prevertebral fascia, *** local anesthetic (Reproduced with permission from Philip Peng Educational Series)

Conclusions

The use of ultrasound for SGB allows identification of important soft tissue structures relevant to the cervical sympathetic chain. Real-time visualization of the needle during advancement, ability to confirming spread of injectate in the appropriate fascial plane, and avoidance of exposure to radiation are other benefits of using US for this procedure. There is reasonable support in the published literature for performing US-guided SGB for enhancing accuracy, efficacy, and safety.

Record and checklist





With permission from Danilo Jankovic

Cervicothoracic ganglion (stellate ganglion)

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: ☐ *22 G* ☐ *40 mm long* ☐ *50 mm long*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipmet checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Neck extended*

Approach: ☐ *Paratracheal* ☐ *C6* ☐ *C7*
☐ *Ultrasound - guided*
☐ *Transducer* ☐ *Linear*
☐ *In plane* ☐ *Out of plane*

Local anesthetic: _____ *ml* _____ *%* _____

Test dose: _____ *ml*

Addition to
Injection solution: ☐ *No* ☐ *Yes* _____

Patient's remarks during injection:
☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Nerve region _____ _____ _____

Objective block effect after 15 min:
☐ *Cold test* ☐ *Temperature measurement right* ____ *°C left* ____ *°C*

Horner's syndrome: ☐ *Yes* ☐ *No*

Segment affected: ☐ *C2* ☐ *C3* ☐ *C4* ☐ *C5* ☐ *T* ____

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge _____

Complications: ☐ *None*
☐ *Yes (intravascular, epidural, subarachnoid injection, other)* _____

Side effects: ☐ *None*
☐ *Yes (recurrent laryngeal nerve, phrenic nerve, vagus* _____

Subjective effects of the block: *Duration:* _____
☐ *None* ☐ *Increased pain*
☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:
_____
_____
_____

References

1. Abdi S. A new and easy technique to block the stellate ganglion. Pain Physician. 2004;7:327–31.
2. 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.
3. Bhatia A, Brull R. Review article: is ultrasound guidance advantageous for interventional pain management? A systematic review of chronic pain outcomes. Anesth Analg. 2013;117:236–51.
4. Bruneau M, Cornelius JF, Marneffe V, Triffaux M, George B. Anatomical variations of the V2 segment of the vertebral artery. Neurosurgery. 2006;59:20–4.
5. Christie JM, Martinez CR. Computerized axial tomography to define the distribution of solution after stellate ganglion nerve block. J Clin Anesth. 1995;7:306–11.
6. Civelek E, Kiris T, Hepgul K, Canbolat A, Ersoy G, Cansever T. Anterolateral approach to the cervical spine: major anatomical structures and landmarks. J Neurosurg Spine. 2007;7:669–78.
7. Elias M. Cervical sympathetic and stellate ganglion blocks. Pain Physician. 2000;3:294–304.
8. Fitzgerald MJT. Neuroanatomy: basic and clinical. 3rd ed. London: WB Saunders; 1996.
9. 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.
10. Higa K, Hirata K, Hirota K, Nitahara K, Shono S. Retropharyngeal hematoma after stellate ganglion block. Anesthesiology. 2006;105:1238–45.
11. Hogan QH, Erickson SJ. MR imaging of the stellate ganglion: normal appearance. AJR Am J Roentgenol. 1992;158:655–9.
12. Hogan Q, Erickson S, Haddox D, et al. The spread of solution during stellate ganglion block. Reg Anesth. 1992;17:78–83.
13. Janik JE, Hoeft MA, Ajar AH, Alsofrom GF, Borrello MT, Rathmell JP. Variable osteology of the sixth cervical vertebra in relation to stellate ganglion block. Reg Anesth Pain Med. 2008;33:102–8.
14. Kapral S, Krafft P, Gosch M, Fleischmann M, Weinstabl C. Ultrasound imaging for stellate ganglion block: direct visualization of puncture site and local anesthetic spread. A pilot study. Reg Anesth. 1995;20:323–8.
15. Kumar N, Thapa D, Gombar S, Ahuja V, Gupta R. Analgesic efficacy of pre-operative stellate ganglion block on postoperative pain relief: a randomised controlled trial. Anaesthesia. 2014;69:954–60.
16. Matula C, Trattnig S, Tschabitscher M, Day JD, Koos WT. The course of the prevertebral segment of the vertebral artery: anatomy and clinical significance. Surg Neurol. 1997;48:125–31.
17. Narouze S, Vydyanathan A, Patel N. Ultrasound-guided stellate ganglion block successfully prevented esophageal puncture. Pain Physician. 2007;10:747–52.
18. Narouze S. Beware of the “serpentine” inferior thyroid artery while performing stellate ganglion block. Anesth Analg. 2009;109:289–90.
19. Narouze S. Ultrasound-guided stellate ganglion block: safety and efficacy. Curr Pain Headache Rep. 2014;18:424.
20. Okuda Y, Urabe K, Kitajima T. Retropharyngeal or cervicomediastinal haematomas following stellate ganglion block. Eur J Anaesthesiol. 2003;20:757–9.
21. Shibata Y, Fujiwara Y, Komatsu T. A new approach of ultrasound-guided stellate ganglion block. Anesth Analg. 2007;105:550–1.
22. Siegenthaler A, Mlekusch S, Schliessbach J, Curatolo M, Eichenberger U. Ultrasound imaging to estimate risk of esophageal and vascular puncture after conventional stellate ganglion block. Reg Anesth Pain Med. 2012;37:224–7.
23. Slapppendel F, Thijssen H, Crul BJ, et al. The stellate ganglion in magnetic resonance imaging, a quantification of anatomic variability. Anesthesiology. 1995;83:424–6.
24. Stanik-Hutt JA. Management options for angina refractory to maximal medical and surgical interventions. AACN Clin Issues. 2005;16:320–32.
25. Stanton-Hicks MD, Burton AW, Bruehl SP, et al. An updated interdisciplinary clinical pathway for CRPS: report of an expert panel. Pain Pract. 2002;2:1–16.
26. Takanami I, Abiko T, Koizumi S. Life-threatening airway obstruction due to retropharyngeal and cervicomediastinal hematomas following stellate ganglion block. Thorac Cardiovasc Surg. 2009;57:311–2.
27. Tubbs RS, Loukas M, Remy AC, Shoja MM, Salter EG. The vertebral nerve revisited. Clin Anat. 2007;20:644–7.
28. van Eijs F, Geurts J, van Kleef M, Faber CG, Perez RS, Kessels AG, Van Zundert J. Predictors of pain relieving response to sympathetic blockade in complex regional pain syndrome type 1. Anesthesiology. 2012;116:113–21.
29. Williams PL. Gray’s anatomy. 38th ed. New York: Churchill Livingstone; 1995.

Chapter 14

Superior Cervical Ganglion Block

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_14,

Anatomy

The superior cervical ganglion arises from the fusion of three or four upper cervical ganglia. It lies medial to the vagus trunk, in front of the longus capitis muscle and behind the internal carotid artery, and in the angle of the vertebrae and transverse processes of the second and third cervical vertebrae (Figs. 14.1 and 14.2). In the literature, its long, flat, or spindle-like extension is described as being 14–43 mm in length, 6–8 mm in breadth, and 3–5 mm in depth [1, 2] (Figs. 14.2 and 14.4). The superior cervical ganglion is thought to contain 760,000–1,000,000 nerve fibers in all, 5,000–12,000 of which are preganglionic. Some 5,000 of these fibers are myelinated [1, 2]. This underlines its importance as a switchpoint with numerous double or triple connections to neighboring ganglia, nerves, and vessels (Fig. 14.3). The superior cervical ganglion takes its preganglionic fibers mainly from the spinal nerves coursing thoracically, with only a few being drawn from the neighboring cervical nerve roots. An unknown number of these preganglionic fibers pass through the ganglion toward the higher carotid ganglia, without switching. Rami communicantes connect the superior cervical ganglion with numerous organs, vessels, muscles, bones, joints, the last four cranial nerves, the vertebral plexus, and also the phrenic nerve. It supplies the upper cervical spinal nerves with gray rami communicantes, and it sends off vascular fibers to the internal and external carotid arteries. Autonomic branches pass from the ganglion to the larynx, pharynx, heart, and—together with vascular plexuses—to the salivary and lacrimal glands, to the hypophysis, thyroid, and other glands. There are also contacts with the middle cervical ganglion and to the tympanic plexus. There are connections with the pterygopalatine ganglion via the nerve of the pterygoid canal, deep petrosal nerve, and greater superficial petrosal nerve. A variable number of fibers from the superior cervical ganglion pass to the inferior ganglion of the vagus nerve, to the hypoglossal nerve, and to the posterior root of the ansa cervicalis [1, 2]. The superior cervical cardiac nerve may be absent, more often on the right side. In these cases, it is replaced by a branch of the vagus nerve from the external branch of superior laryngeal nerve.

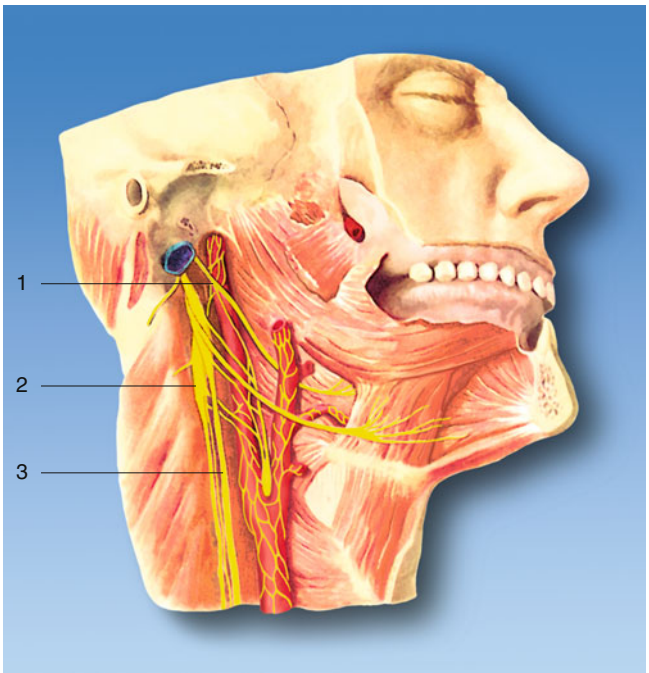


Fig. 14.1 Topographic position of the superior cervical ganglion: (*1*) glossopharyngeal nerve, (*2*) superior cervical ganglion, (*3*) vagus nerve (With permission from Danilo Jankovic)

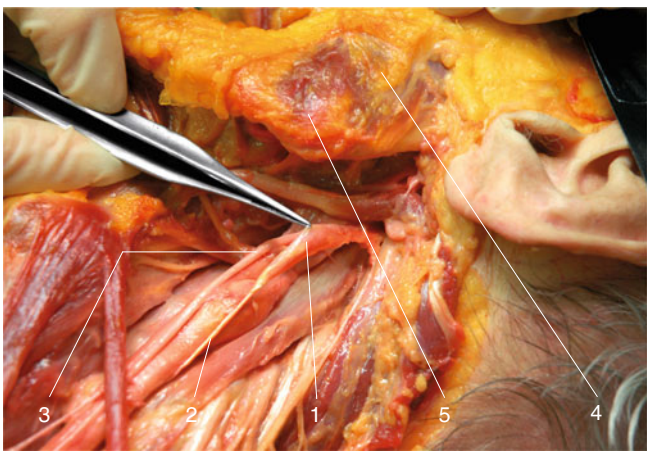


Fig. 14.2 The superior cervical ganglion has an average size of 26.6 mm (14–43 mm) ×7.2 mm × 3.4 mm. (*1*) Superior cervical ganglion, (*2*) sympathetic trunk, (*3*) vagus nerve with inferior vagal ganglion, (*4*) masseter muscle, (*5*) angle of the mandible (With permission from Danilo Jankovic)

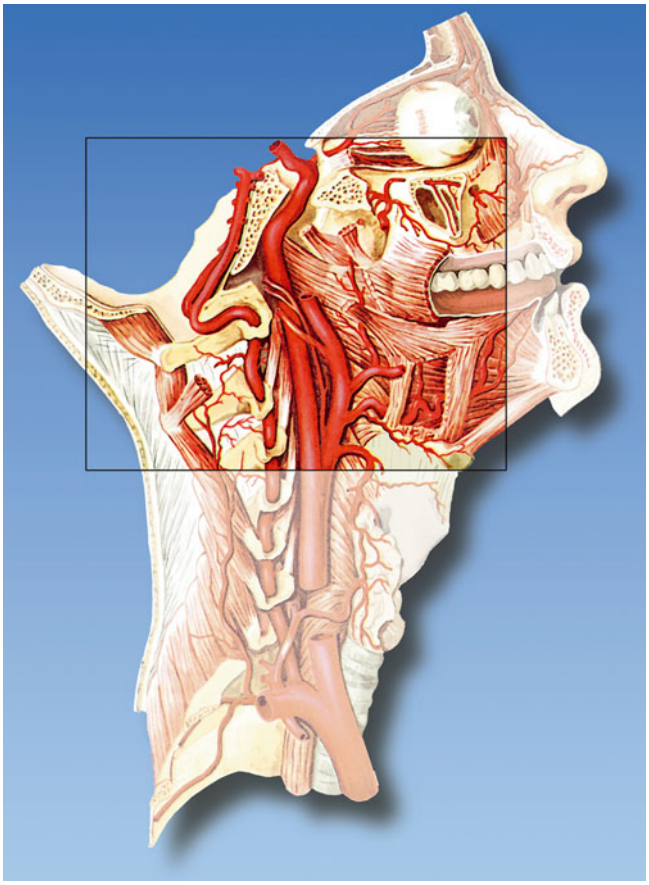


Fig. 14.3 Arteries in the immediate vicinity of the ganglion. Course of the vertebral artery and internal carotid artery (risk of intra-arterial injection) (With permission from Danilo Jankovic)

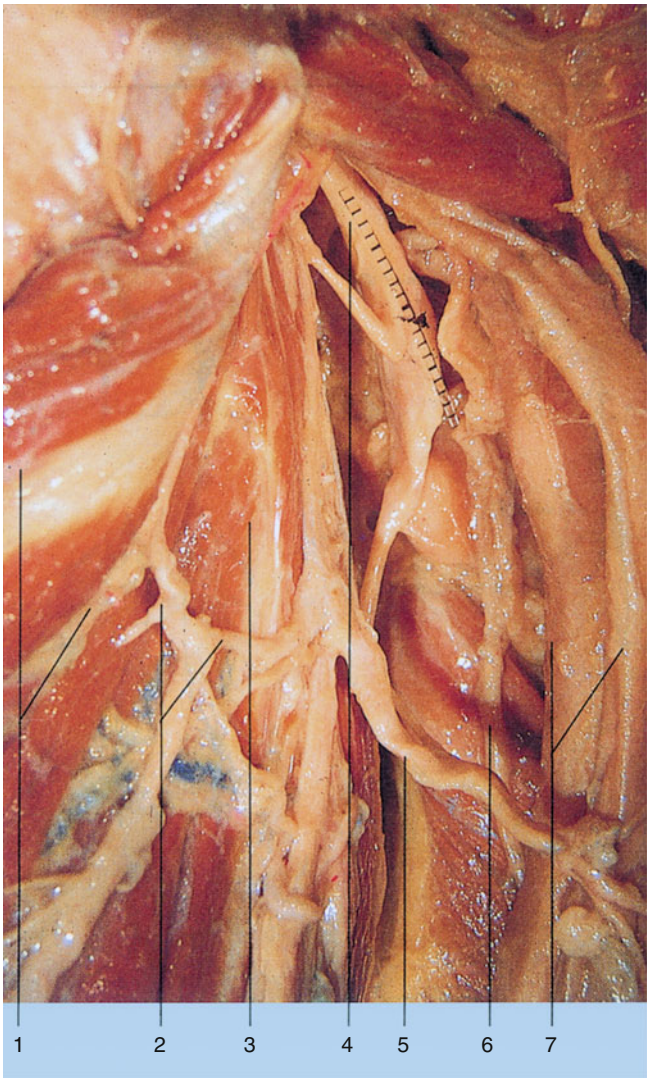


Fig. 14.4 Immediate vicinity of the superior cervical ganglion: (*1*) sternocleidomastoid muscle, repositioned dorsally, and the accessory nerve, (*2*) anastomosis between the C2 vertebral branch and nerve XII, (*3*) scalenus medius muscle, (*4*) superior cervical ganglion, (*5*) hypoglossal nerve, (*6*) external branch of the superior laryngeal nerve, (*7*) neurovascular fascicle, repositioned anteriorly (With permission from Danilo Jankovic)

Blocks of the Superior Cervical Ganglion

Indications

The areas of application are partly identical to those for the stellate block, but due to its marked cerebrofacial effects, the superior cervical ganglion block is particularly suitable for the head and facial region—although controlled studies are still lacking here.

Therapeutic [3]

1. Migraine [4], cluster headache, and headaches of cervical origin
2. Complex regional pain syndrome (CRPS) in the head region
3. Perfusion disturbances and vasospastic diseases
4. Central poststroke syndrome (contralateral block!)
5. Facial pain
6. Vertigo (of vertebral origin)
7. Peripheral facial paralysis
8. Trigeminal neuralgia in the first and second branches
9. Postherpetic neuralgias (otic, ophthalmic)
10. Sudden deafness and tinnitus
11. Hyperhidrosis in the head region
12. Positive effect on the immune system [5]

Neural Therapy

Asthma [6, 7], urticaria, vasomotor rhinitis, etc.

Specific Contraindications

- Grade 2 atrioventricular (AV) block, recent antithrombotic therapy after myocardial infarction or pulmonary embolism, anticoagulation treatment, contralateral paresis of the phrenic nerve, or recurrent laryngeal nerve.
- Simultaneous bilateral block.

Procedure

Lateral Extraoral Technique

This block should only be carried out by an experienced anesthetist. The patient should have a full explanation of the procedure before it is carried out.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions. Intravenous access, ECG monitoring, pulse oximetry, and ventilation facilities.

Materials

5-mL syringe, 23-G needle (60 mm), intubation kit, emergency drugs, and disinfectant.

Skin Prep

In all blocks.

Patient Positioning

Supine, with the head turned about 30–40° to the opposite side.

Landmarks

Mastoid process, angle of the mandible, and medial margin of the sternocleidomastoid muscle (Fig. 14.5). The angle of the mandible and the mastoid are marked with the index and middle finger. From the anterior margin of the mastoid process, a vertical line is drawn downward; about 1 cm above the angle of the mandible, a horizontal mark is applied. The intersection of these two lines defines the injection point (Fig. 14.6).

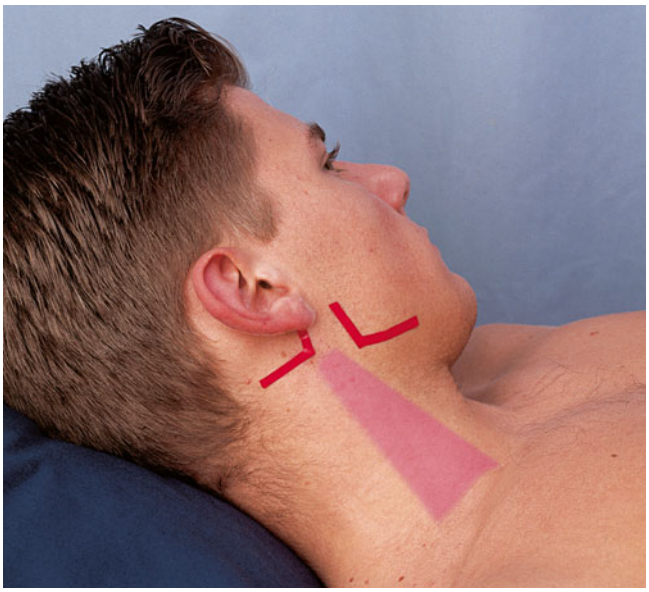


Fig. 14.5 Landmarks for locating the needle insertion position. Angle of the mandible, mastoid, medial margin of the sternocleidomastoid muscle (With permission from Danilo Jankovic)

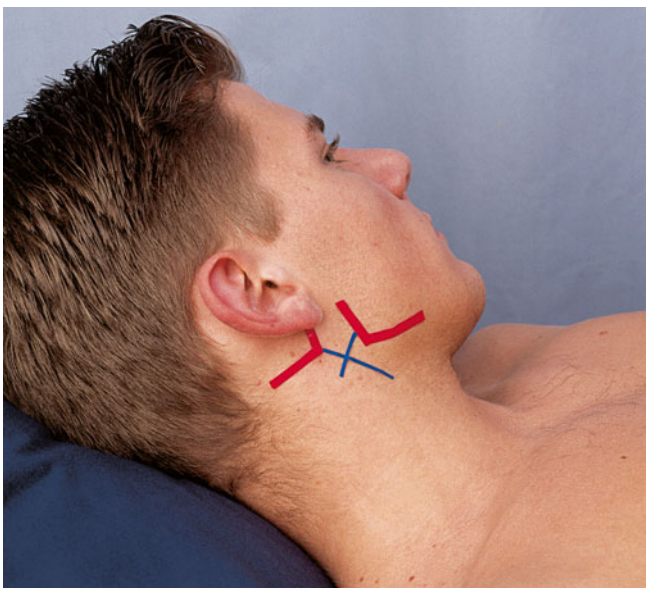


Fig. 14.6 Marking the injection site (With permission from Danilo Jankovic)

Injection Technique [8–10]

After skin infiltration, a 6-cm long needle is introduced in the direction of the contralateral mastoid at a craniodorsal angle of about 20° (Fig. 14.7). In normal anatomy, bone contact is made at about 3.5–5 cm, and careful aspiration is carried out at various levels after the needle has been minimally withdrawn. Only then can a test dose of 0.5 mL of the local anesthetic be administered. After about 1 min, slow injection of the remaining dose can be carried out. The patient's upper body is then raised.

- A single test dose by no means guarantees correct positioning of the needle. The remaining dose must never be injected quickly or carelessly. It must be administered slowly in small quantities (several test doses) with repeated aspiration.
- If the needle direction is incorrect, the patient will complain of pain and will resist the injection. In this case, the needle must be withdrawn to the subcutaneous tissues so that its position can be corrected.

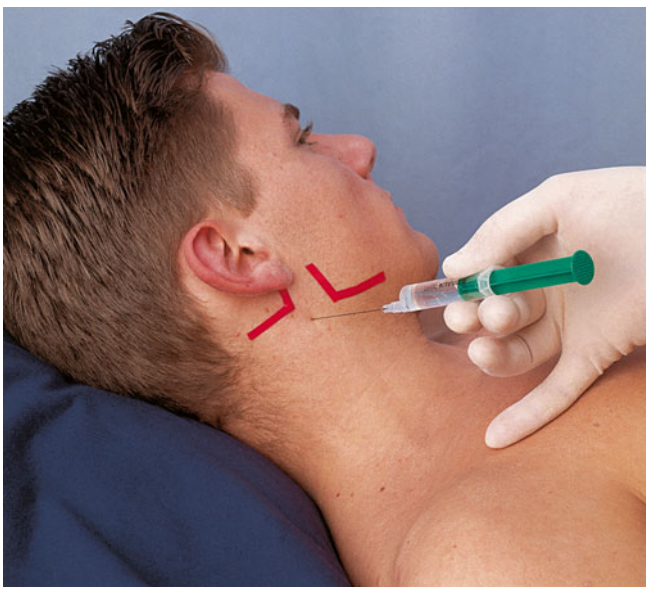


Fig. 14.7 Craniodorsal puncture in the direction of the contralateral mastoid (With permission from Danilo Jankovic)

Effects of the Block

Characteristic signs of a successful block are radiation and a warm sensation in the area of the back of the head, ear, eyes, and corner of the mouth and the ipsilateral half of the face (Figs. 14.8 and 14.9). Conjunctival injection, increased tear production, and ipsilateral nasal congestion are equally characteristic, as is Horner's syndrome—which is by no means restricted to stellate block but occurs in all blocks of the sympathetic cervical trunk.

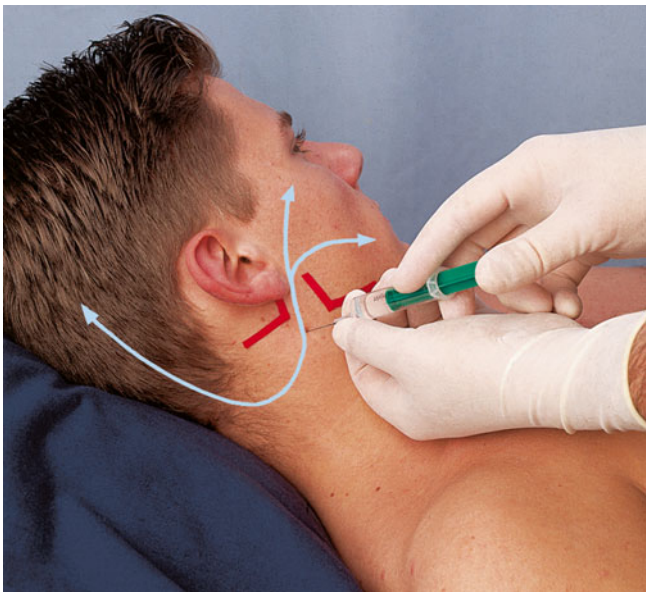


Fig. 14.8 Characteristic directions of radiation during the injection (With permission from Danilo Jankovic)

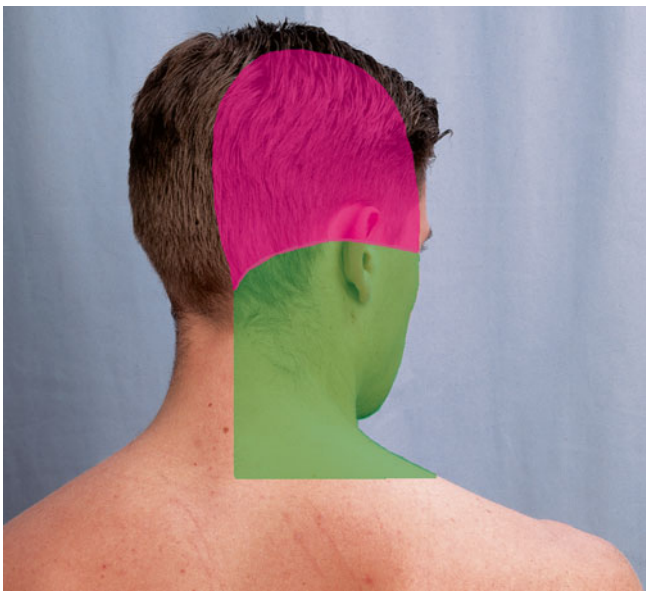


Fig. 14.9 Distribution of the block (With permission from Danilo Jankovic)

Dosage

Therapeutic

5 mL local anesthetic—e.g., 0.5–1 % procaine, 0.5–1 % prilocaine, and 0.5–1 % lidocaine, 0.2 % ropivacaine and 0.125 % bupivacaine (0.125 % levobupivacaine).

Block Series

A series of 6–10 blocks is appropriate for all indications. In difficult cases (e.g., herpes zoster), additional blocks can also be carried out when there is evidence of improvement.

Side Effects

1. Hematoma formation (harmless)
2. Block of the following nerves:
   - Phrenic nerve. Main symptom: dyspnea
   - Recurrent laryngeal nerve. Main symptoms: foreign-body sensation in the neck and hoarseness
   - Vagus nerve. Main symptoms: tachycardia, hypertension
   - Glossopharyngeal nerve. Main symptoms: numbness in the posterior third of the tongue, and paresis of the pharyngeal muscles
   - Partial anesthesia of the cervical plexus
   - Persistent coughing

When giving consent, the patient must be informed about these adverse effects and prepared for them.

Complications

Most complications arise when the local anesthetic is administered without prior bone contact.

Bilateral block of the superior cervical ganglion is contraindicated, since bilateral paralysis of the recurrent laryngeal nerve or phrenic nerve is life-threatening.

Intravascular Injection

There is a particular risk of injection into the vertebral artery (Fig. 14.3), the diameter of which is about 0.3 mm wider on the left side than on the right. Intra-arterial administration of a local anesthetic can produce toxic reactions very quickly.

Epidural or Subarachnoid Injection

There is a risk of perforating the dural membrane. Cerebrospinal fluid (CSF) pressure is very low in the cervical area, and it is almost impossible to aspirate CSF. The resultant high epidural anesthesia or high spinal anesthesia can lead to bradycardia, a drop in blood pressure, and possibly respiratory arrest and loss of consciousness.

Due to the potential complications, the patient must be monitored after the injection has been carried out—for at least 30 min after procaine administration and at least 60 min after administration of ropivacaine or bupivacaine.

Superior cervical ganglion blocks in pain therapy or as an option in depressive conditions

In my own clinical experience over many years with superior cervical ganglion block series (10–12 on average), there have been surprisingly good results in a large number of patients. These observations principally concern patients with pain-associated depression in chronic pain conditions (various types of headache, migraines, facial pain, postnucleotomy pain, fibromyalgia, etc.). In the superior cervical ganglion block, the usual volume of 5 mL local anesthetic (e.g., 1 % procaine) covers neighboring nerves such as the vagus nerve, for example. The superior cervical ganglion is often barely distinguishable from the vagus nerve. Left-sided vagus stimulation with an implantable electrode has been successfully used since 1938 to treat various neurological diseases such as epilepsy [11, 12], treatment-resistant depression anxiety states [13–16], sleep disturbances [17], and other conditions. Dysfunction of the autonomic nervous system is almost always present as an accompanying symptom of depression [3, 13]. The long-term analgetic effect of vagus stimulation was demonstrated in a study by Kirchner et al. [3]. Like the antiepileptic and antidepressive action of vagus stimulation, this is probably due to neurobiochemical effects. For example, patients receiving vagus stimulation of the cerebrospinal fluid show a significant increase in norepinephrine and serotonin levels and a significant decrease in proalgetic excitatory amino acids such as aspartate and glutamate. The same group of authors report marked symptomatic improvement during vagus stimulation in a patient with chronic tension headache. In this context, answers will have to be found in the future to the following questions: What role does the superior cervical ganglion play in this? Is the functioning of the superior cervical ganglion more important than that of the vagus nerve? It should not be forgotten that the superior cervical ganglion is the last station at which information from the body can be modulated before entering the CNS.

Superior cervical ganglion

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: ☐ *23 G* ☐ *50 mm* ☐ *60 mm* ☐ _____

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipmet checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Head to contralateral side*

Approach: ☐ *Extraoral (direction of C2 vertebra)*

Local anesthetic: _____ *mL* _____ *%* _____

Test dose: _____ *mL*

Addition to

Injection solution: ☐ *No* ☐ *Yes* _____

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Nerve region _____ _____ _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* ____ *°C left* ____ *°C*

Horner's syndrome: ☐ *Yes* ☐ *No*

Segments affected: ☐ *C2* ☐ *C3* ☐ *C4* ☐ *C5 (numbness, warmth)*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge _____

Complications:

☐ *None* ☐ *Yes (intravascular, epidural, subarachnoid injection; other)* _____

Side effects:

☐ *None* ☐ *Yes (recurrent laryngeal nerve, phrenic nerve, vagus nerve, glossopharyngeal nerve ...)* _____

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

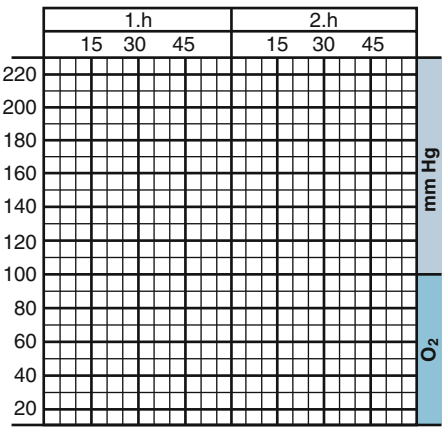
Special notes:

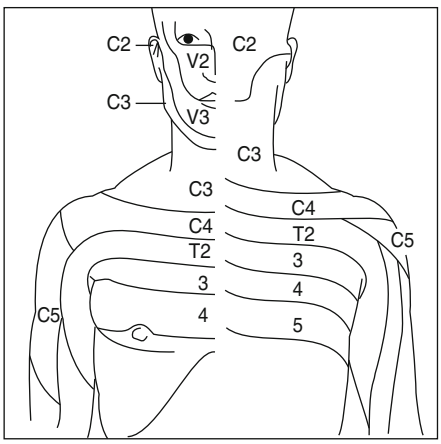
_____

_____

_____

Record and checklist





With permission from Danilo Jankovic

References

1. Lang J. Klinische Anatomie der Halswirbelsäule. Stuttgart/New York: Thieme; 1991.
2. Lang J. Einige Befunde zur Anatomie des Halssympathikus. Med Orth Techn. 1992;112:194–200.
3. Kirchner A, Birklein F, Stefan H, Handwerker HO. Vagusstimulation – Eine Behandlungsoption für chronische Schmerzen? Schmerz. 2001;15:272–7.
4. Harder HJ. Die Behandlung der Migraine Blanche und Ophthalmique mit Blockaden des Ganglion cervicale superius. Reg Anaesth. 1981;4:1–9.
5. Matsuoka H, Tokutomi Y, Muteki T, Yokojama MM. Influence of stellate ganglion block on the immune system. Masui, Jap. J Anesthesiol. 1985;34(7):917–23.
6. Goel AC. Auricular nerve block in bronchial asthma. J Indian Med Assoc. 1981;76:132.
7. Victor M, Adams RD. Krankheiten der Hirnnerven. In: Harrison's principles of internal medicine. 8th ed. New York: McGraw-Hill; 1983.
8. Jankovic D. Superior cervical ganglion. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Blackwell publishing, Malden, Massachusetts, Oxford, Carlton, Victoria Australia; 2008. p. 120–6.
9. Gross D. Therapeutische Lokalanästhesie des Halsgrenzstranges. In: Gross D, editor. Therapeutische Lokalanästhesie. Stuttgart: Hippokrates; 1972.
10. Jenkner FL. Blockade des Ganglion cervicale superius. In: Jenkner FL, editor. Nervenblockaden auf pharmakologischem und auf elektrischem Weg. Wien: Springer; 1980.
11. Amar AP, Heck CN, Levy ML, et al. An institutional experience with cervical vagus nerve trunk stimulation for medically refractory epilepsy: Rationale, technique and outcome. Neurosurgery. 1998;43:1265–80.
12. Ben-Menacham E, Manon-Espaillat R, Ristanovic R, et al. Vagus nerve stimulation for treatment of partial seizures: a controlled study of effect on seizures. Epilepsia. 1994;35:616–26.
13. Glassmann AH. Depression, cardiac death and the central nervous system. Neuropsychobiology. 1998;37:80–3.
14. Olfson M, Marcus S, Sackeim HA, et al. Use of ECT for the inpatient treatment of recurrent major depression. Am J Psychiatry. 1998;155:22–9.
15. Rosenbaum JF, Heninger G. Vagus nerve stimulation for treatment-resistant depression. Biol Psychiatry. 2000;47:273.
16. Rush AJ, George MS, Sackeim HA, et al. Vagus nerve stimulation (VNS) for treatment-resistant depressions: A multicenter study. Biol Psychiatry. 2000;47:277–86.
17. Vaugh BV, D'Cruz OF. Effect of vagal nerve stimulation on sleep (abstract). Epilepsia. 1999;40:137.

Chapter 15

Deep (and Superficial) Cervical Plexus

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_15,

Anatomy [4]

The anterior branches of the four upper cervical spinal nerves (C1 to C4) form the cervical plexus (Figs. 15.1 and 15.2), which is covered by the sternocleidomastoid muscle. The branches of the cervical plexus carry motor, sensory, proprioceptive, and autonomous fibers and divide into superficial cutaneous branches penetrating the cervical fascia and deeper muscular branches that mainly innervate the joints and muscles. The cutaneous branches of the cervical plexus are the lesser occipital nerve, great auricular nerve, transverse cervical (colli) nerve, and the supraclavicular nerves (Fig. 15.3). The lesser occipital nerve (from C2 and C3) passes on the splenius capitis muscle to its insertion area, where it fans out into several branches and supplies the skin on the upper side of the neck and upper part of the auricle and the adjoining skin of the scalp. The largest plexus branch is usually the great auricular nerve (from C2 and C3), which passes upward behind the external jugular vein and divides into a posterior and an anterior end branch. The posterior branch supplies the skin lying behind the ear and the medial and lateral surfaces of the lower part of the auricle. The anterior branch supplies the skin in the lower posterior part of the face and the concave surface of the auricle. The transverse cervical nerve (from C2 and C3) passes almost horizontally over the external surface of the sternocleidomastoid muscle in an anterior direction toward the hyoid bone, divides into superior and inferior branches, and supplies the skin over the anterolateral side of the neck between the mandible and the sternum. The common trunk of the supraclavicular nerves (from C3 and C4) appears at the posterior margin of the sternocleidomastoid muscle, just below the transverse cervical nerve; passes downward; and divides into anterior, medial, and posterior supraclavicular nerve branches. The areas supplied by the supraclavicular nerves include the skin over the caudal part of the neck and the skin above the shoulders and the lateral upper chest, as well as the skin covering the anterior part of the deltoid muscle and occupying the acromial region.

The muscular branches of the cervical plexus include segmentally arranged nerve branches supplying the deeper anterior neck muscles (the rectus capitis anterior and lateralis, longus colli, longus capitis and intertransverse, scalenus anterior and medius, and levator scapulae), as well as the inferior descending cervical nerve, the trapezius branch, and the phrenic nerve. The inferior descending cervical nerve (from C2 and C4) gives off several fibers to the carotid and jugular neural plexus and joins with the superior descending cervical nerve to form the ansa cervicalis. The area supplied includes the sternothyroid muscle, sternocleidomastoid muscle, thyrohyoid muscle, geniohyoid muscle, and omohyoid muscle.

The trapezius branch appears at the surface just below the accessory nerve and passes to the trapezius muscle. The phrenic nerve (from C4 and C3/5) is the motor nerve for the diaphragm, but it also contains sensory and sympathetic fibers that supply the fibrous pericardium, the mediastinal pleura, and the central part of the diaphragmatic pleura as the nerve courses through the thorax. Connections have been described between the phrenic nerve (left or right branch) or the phrenic plexus and the following structures: inferior and middle cervical ganglion, subclavian plexus, pulmonary plexus, inferior vena cava, esophagogastric junction, cardiac end of the stomach, hepatic portal, suprarenal cortex, etc.

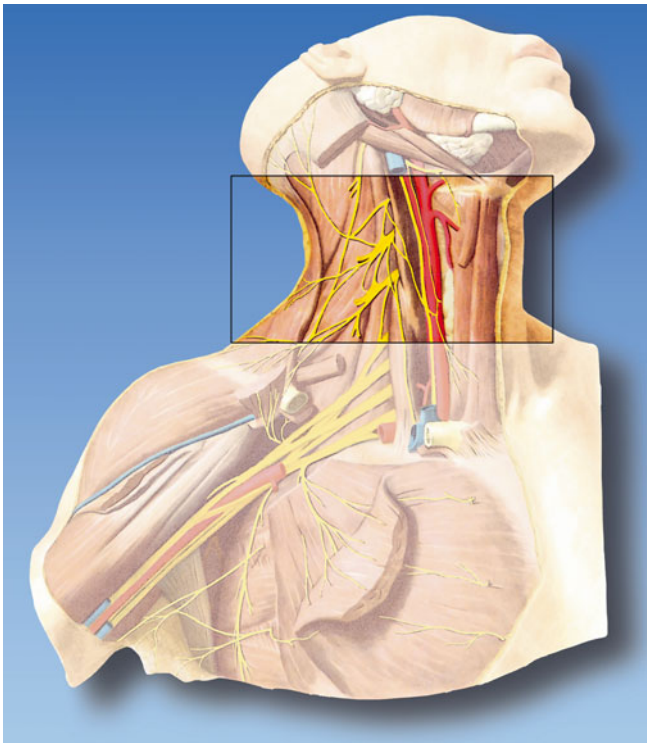


Fig. 15.1 Anatomy of the deep cervical plexus (With permission from Danilo Jankovic)

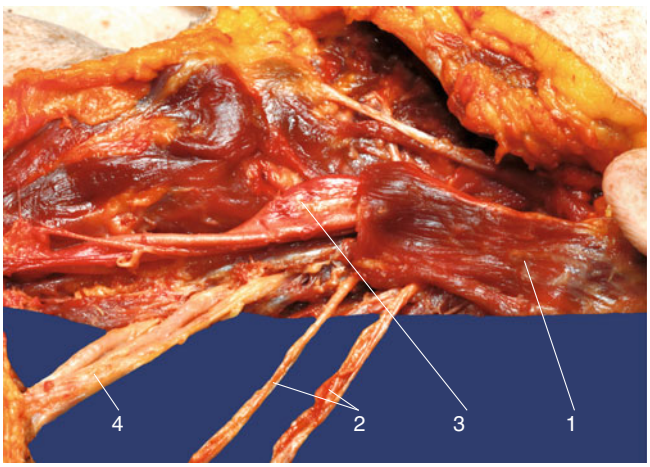


Fig. 15.2 Deep cervical plexus on the level of the C3, C4. (*1*) Sternocleidomastoid muscle (dissected), (*2*) deep cervical plexus from C3 and C4, (*3*) common carotid artery, and (*4*) trunks of the brachial plexus (With permission fom Danilo Jankovic)

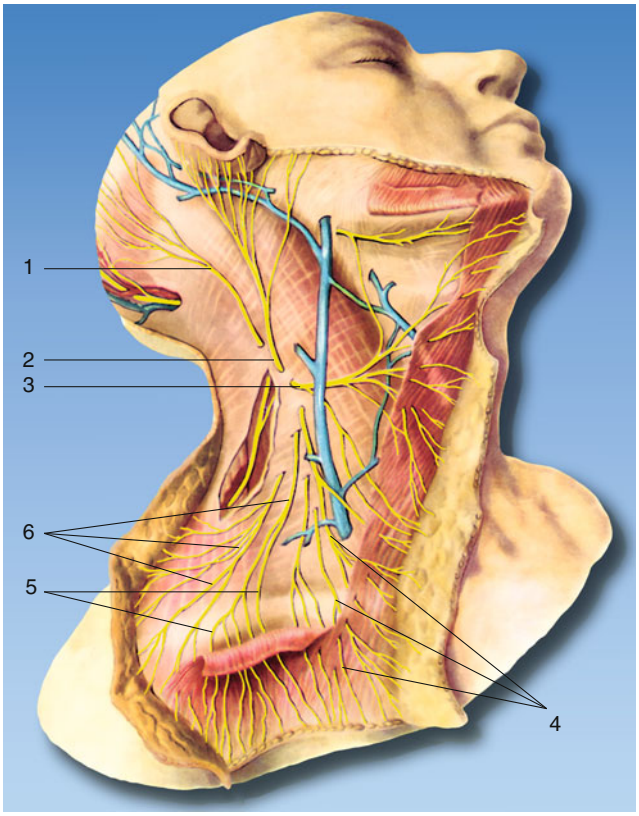


Fig. 15.3 Anatomy of the superficial cervical plexus. (*1*) Lesser occipital nerve, (*2*) great auricular nerve, (*3*) transverse cervical (colli) nerve, (*4*)medial supraclavicular nerves, (*5*) intermediate supraclavicular nerves, and (*6*) lateral supraclavicular nerves (With permission from Danilo Jankovic)

Block of the Superficial Cervical Plexus

Indications

1. Superficial procedure for submandibular, postauricular, and lower neck area
2. Combined with deep cervical plexus block for surgical procedures listed below
3. Isolated nerve (supraclavicular nerve) block adjunct to brachial plexus block for shoulder surgery

Procedure

With ultrasound, the typical morphology of cervical transverse processes from C2 to C7 can be easily revealed (Fig. 15.4a–g). At the level of C4 or C5, the superficial cervical plexus can be seen deep to the sternocleidomastoid muscle (Fig. 15.5). Both the in-plane and out-of-plane approaches can be performed, and a total of 5 mL of local anesthetic will be sufficient.

For the clavicle, blockade of the supraclavicular nerve of the superficial plexus is possible. The anatomical landmark is the supraclavicular area 2–3 cm above the clavicle, posterior to the sternocleidomastoid muscle superficial to the deep cervical investing fascia (Fig. 15.6). The nerve usually appears as a single nerve trunk before it branches into medial, intermediate, and lateral branches and is in intimate relation to the external jugular vein. To inject this nerve, only 2 mL of local anesthetic is sufficient.

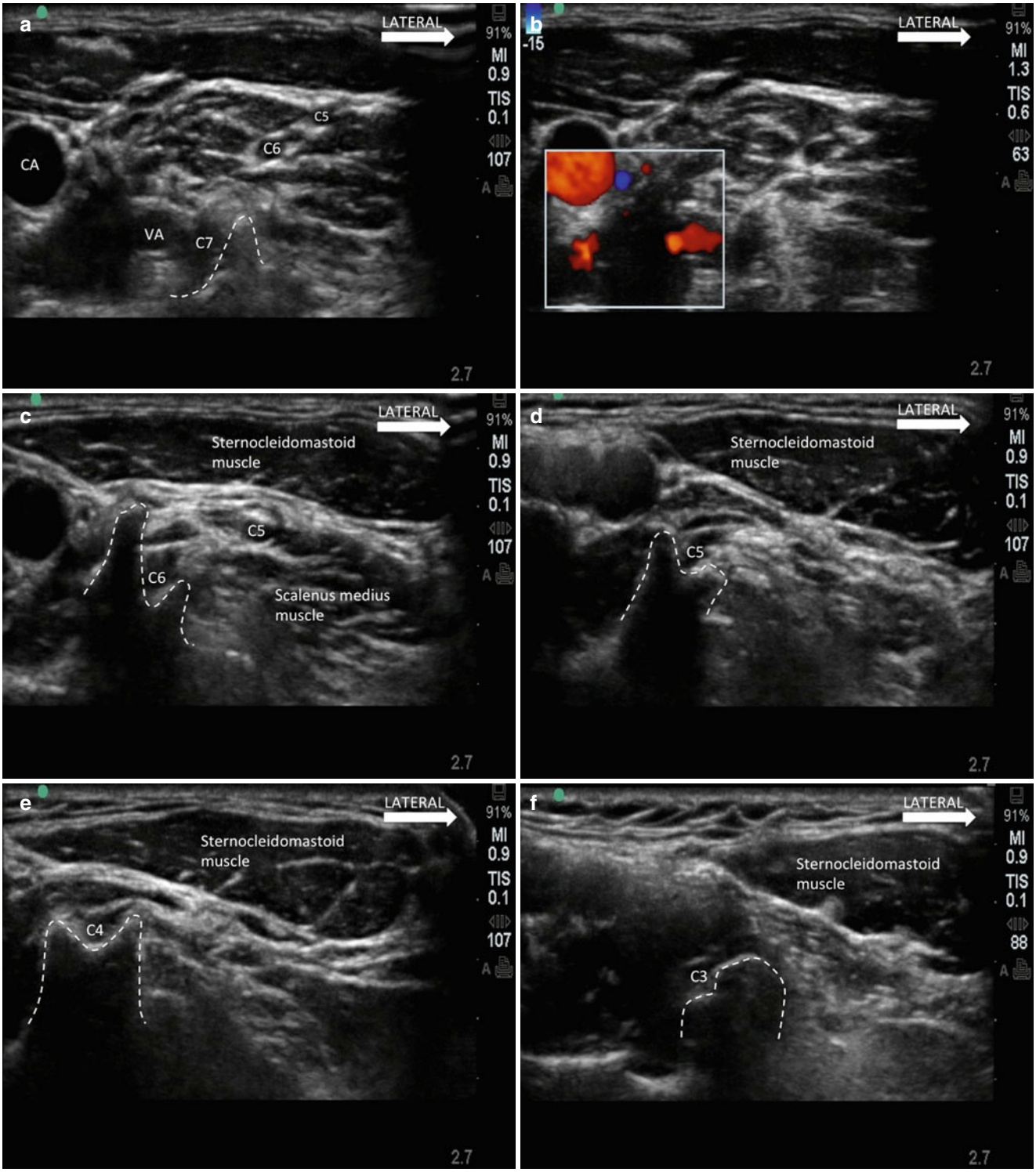


Fig. 15.4 (**a**) Sonogram showing transverse process of C7. Note that only the posterior tubercle is seen as the anterior tubercle is usually vestigial. The nerve roots C5 to C7 are seen aligned in the interscalene groove. The vertebral artery (*VA*) and carotid artery (*CA*) is seen in this sonogram and better shown in Doppler in (**b**). (**c**) Sonogram showing the transverse process of C6. Note the prominent anterior tubercle of C6. (**d**) Sonogram showing C5. Note the anterior and posterior tubercles are similar in size, contrast to that in C6. (**e**) Sonogram of C4. Note the morphology is similar to C5 but the tubercles are usually closer together than that of C5. (**f**) Sonogram of C3. Note the discrepancy in the shape of anterior and posterior tubercle. (**g**) Sonogram of C2. Note the prominence of the posterior tubercle (Reproduced with permission from Philip Peng Education Series)

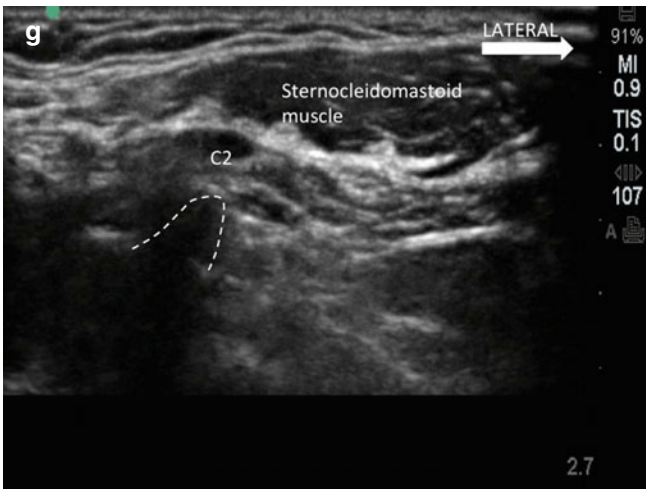


Fig. 15.4 (continued)

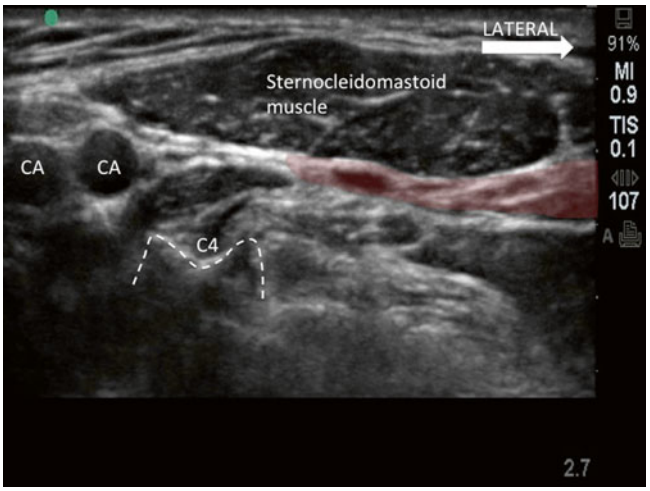


Fig. 15.5 Sonogram of the superficial cervical plexus (*shaded with purple color*) at the level of C4. Multiple nerves in short axis are seen sequestered in this space deep to the sternocleidomastoid muscle. CA, carotid artery, which bifurcates into internal and external carotid arteries (Reproduced with permission from Philip Peng Education Series)

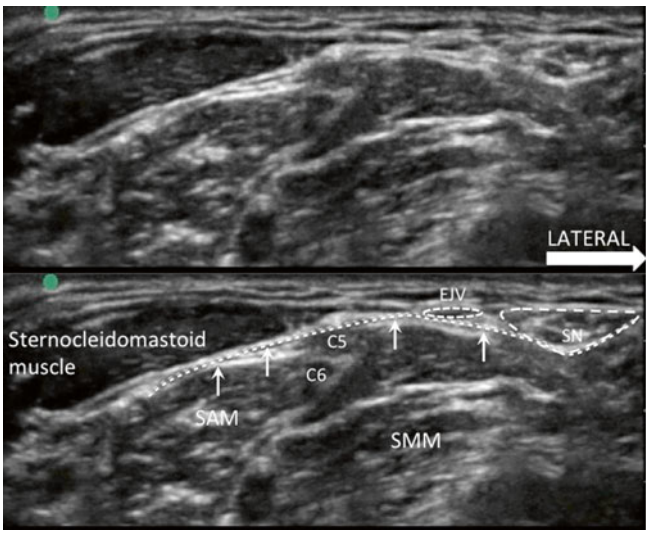


Fig. 15.6 Sonogram of the supraclavicular nerve (*SN*). The top picture is the unlabeled version of the bottom picture. Note the investing layer of the deep cervical fascia (*dotted line* indicated by *arrows*). The SN is superficial to this layer and is in close proximity with external jugular vein (*EJV*). *SAM* scalenus anterior muscle, *SMM* scalenus medius muscle (Reproduced with permission from Philip Peng Education Series)

Block of the Deep Cervical Plexus

Indications

Diagnostic

- Localization and differentiation of various types of neuralgia

Therapeutic

- Postherpetic neuralgia
- Occipital and cervicogenic headache
- Torticollis

Surgical

In combination with a block of the superficial cervical plexus:

- Carotid endarterectomy [1, 2]
- Excision of cervical lymph nodes
- Plastic surgery in the area of innervation

Specific Contraindications

- Grade 2 atrioventricular (AV) block, anticoagulant treatment, and contralateral paresis of the phrenic nerve or recurrent laryngeal nerve
- Simultaneous bilateral blocks

Procedure

This block should only be carried out by experienced anesthetists. It is absolutely necessary to have a detailed discussion with the patient before the procedure.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions. Intravenous access, ECG monitoring, pulse oximetry, and ventilation facilities.

Materials

5-mL syringes, 10-mL syringes, three fine 22-G needles (5 cm), intubation kit, emergency drugs, and disinfectant

Skin prep in all blocks.

Patient Positioning

Supine, with the head tilted slightly backward and turned about 45° to the opposite side.

Landmarks

Posterior edge of the sternocleidomastoid muscle, caudal part of the mastoid process, Chassaignac's tubercle (C6), and transverse processes of C2, C3, C4, and C5 (Figs. 15.7 and 15.8)

The patient is asked to turn the head toward the opposite side and to lift it slightly, making the posterior edge of the sternocleidomastoid apparent.

The transverse process of C6 and the caudal tip of the mastoid process are located. A line is drawn from the mastoid process along the posterior edge of the sternocleidomastoid muscle to the level of C6 (Figs. 15.9 and 15.10). The transverse process of C2 is palpated and marked on the skin. This lies about 1.5 cm caudal to the mastoid process and about 0.5–1 cm dorsal to the marked line. The transverse processes of C3, C4, and C5 are also palpated and marked. The distances between them are each ca. 1.5 cm, and like C2 they lie about 0.5–1 cm dorsal to the marked line.

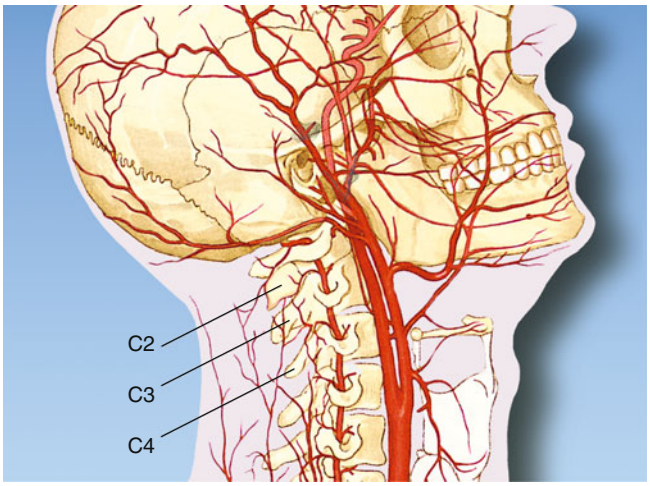


Fig. 15.7 Landmarks: transverse processes of C2 to C4 (With permission from Danilo Jankovic)

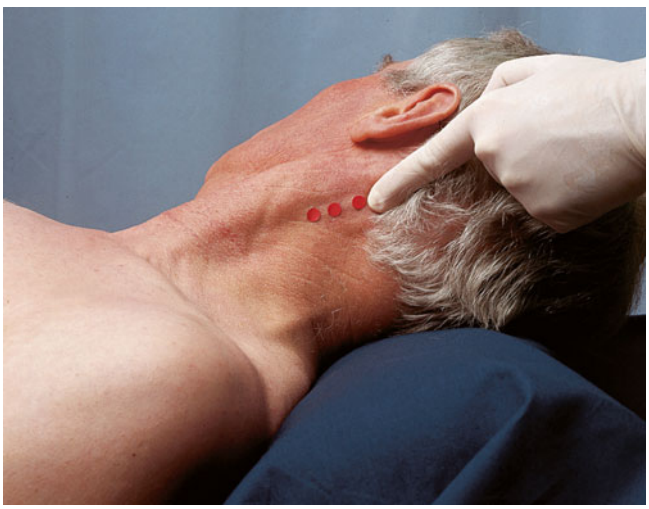


Fig. 15.8 Marking the guiding lines (With permission from Danilo Jankovic)

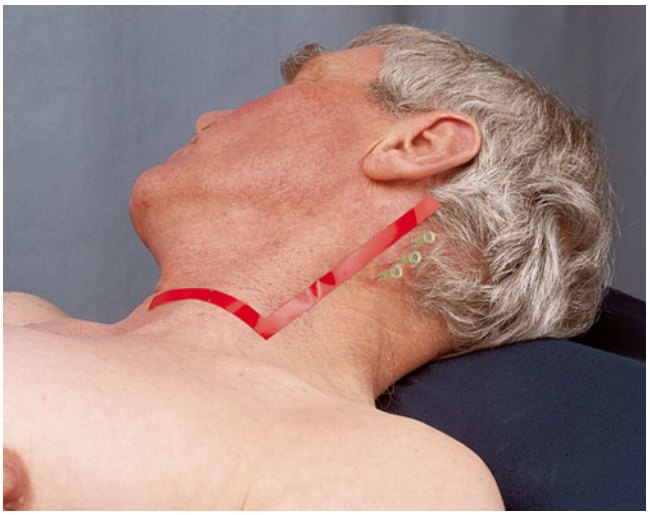


Fig. 15.9 Needle insertion in the area of the transverse processes of C2, C3, and C4 (With permission from Danilo Jankovic)

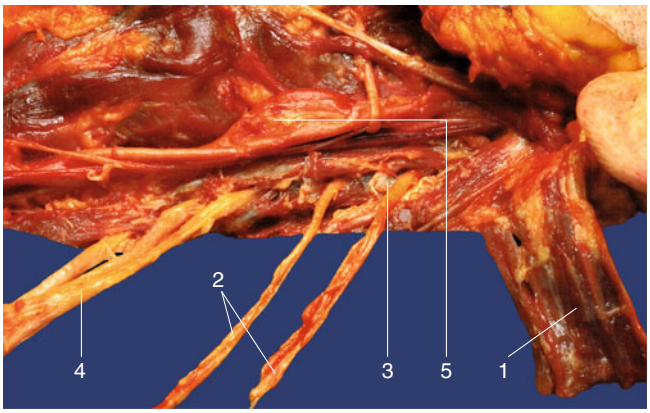


Fig. 15.10 Blockade target. Sulcus of the transverse processes. (*1*) Sternocleidomastoid muscle, (*2*) deep branches of the cervical plexus from C3 and C4, (*3*) sulcus of the transverse process, (*4*) trunks of the brachial plexus, and (*5*) common carotid artery (With permission from Danilo Jankovic)

Injection Technique [3, 4, 6, 7]

The aim is to block the anterior branches of the cervical plexus in the groove of the transverse process.

After thorough skin prep, skin infiltration is carried out at the marked areas of C2, C3, and C4 and the needles are introduced (Fig. 15.10). To do this, the anesthetist stands at the patient's head. In the sequence C2 to C4, the needles are directed perpendicular to the skin and advanced medially slightly caudal (ca. 30°) to contact the "gutters" in the superior surface of the transverse processes. In normal anatomy, the distance from the transverse processes to the skin varies between 1.5 and 3.5 cm. After clear bone contact and minimal withdrawal of the needle, careful aspiration needs to be carried out at various levels.

> The tip of the needle must reach the groove of the transverse process in order to ensure good anesthesia. Note that caudal direction is essential to avoid penetration of an intervertebral foramen, with possible injection into epidural space or dural sleeve.

Only then may the local anesthetic be injected in several small doses, with repeated aspiration (Fig. 15.11).

> An injection should never be carried out without definite bone contact. The local anesthetic must be slowly administered in small amounts (several test doses) and with repeated aspiration.

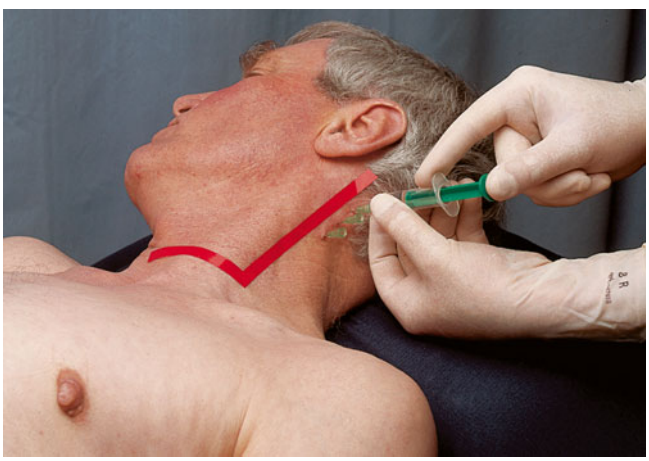


Fig. 15.11 Injection after aspiration

If the needle slips along the transverse process and enters an intervertebral foramen, there is a risk of dural puncture.

Effects of the Block

If the local anesthetic spreads in the direction of the superior cervical ganglion and/or the cervicothoracic ganglion, Horner's syndrome may develop (see Chap. 13).

Block Series

If there is improvement after two treatment sessions, a series of 8–12 therapeutic blocks is indicated.

Dosage

Diagnostic

2 mL local anesthetic per segment—e.g., 1 % prilocaine, mepivacaine, and lidocaine.

Therapeutic

3 mL local anesthetic per segment—e.g., 0.2–0.375 % ropivacaine and 0.125–0.25 % bupivacaine (0.125–0.25 % levobupivacaine).

Surgical

30 mL local anesthetic:

0.75 % ropivacaine or 0.25–0.5 % bupivacaine (0.25–0.5 % levobupivacaine) mixed with 1 % prilocaine or 1 % mepivacaine.

Of this: 10 mL for fan-like injection into the superficial cervical plexus (center of the posterior edge of the sternocleidomastoid muscle) (Figs. 15.3, 15.12, and 15.13) and 20 mL for anesthesia of the deep cervical plexus.

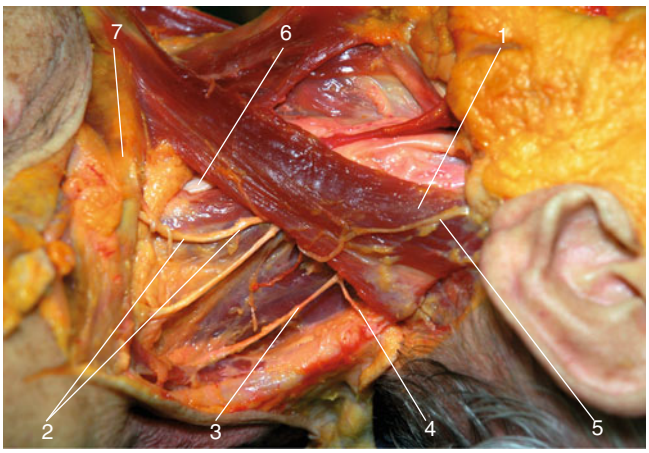


Fig. 15.12 Superficial cervical plexus. (*1*) Sternocleidomastoid muscle, (*2*) supraclavicular branches of the superficial plexus, (*3*) accessory nerve, (*4*) lesser occipital nerve, (*5*) great auricular nerve, (*6*) brachial plexus, and (*7*) clavicle (With permission from Danilo Jankovic)

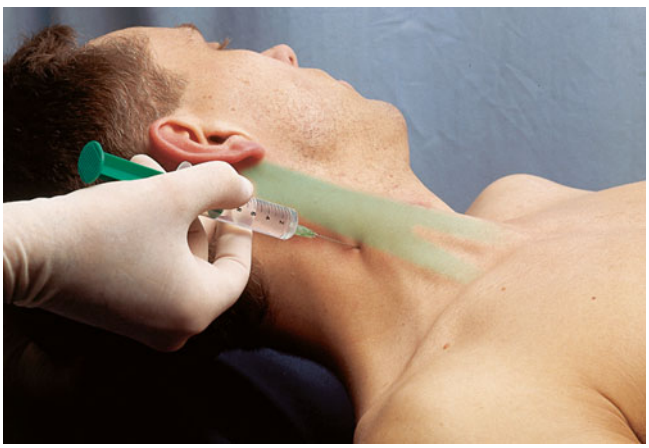


Fig. 15.13 Superficial cervical plexus block (With permission from Danilo Jankovic)

Side Effects [4, 5, 8]

Simultaneous block of the following nerves:

- Phrenic nerve, main symptom: unilateral paralysis of diaphragmatic movement
- Recurrent laryngeal nerve, main symptoms: hoarseness and foreign-body sensation in the throat
- Glossopharyngeal nerve, main symptoms: numbness in the final third of the tongue and paralysis of the pharyngeal muscles
- Vagus nerve, main symptoms: tachycardia and hypertension
- Partial block of the upper part of the brachial plexus

When giving consent, the patient must be informed about these adverse effects and prepared for them.

The patient must be monitored for 60 min after the block has been performed.

Complications

Intravascular Injection

There is always a risk of intravascular injection due to the rich vascular supply in this area. Particular attention should be given to avoiding puncture of the vertebral artery. Toxic reactions may occur after intravascular administration of local anesthetics, and the symptoms and treatment of these are outlined in Chap. 1, p. 8.

Epidural or Subarachnoid Injection

When the needle slides along the transverse process and enters an intervertebral foramen, there is a risk of dural puncture and subarachnoid injection of local anesthetic. This can lead to a high spinal or high epidural block. The clinical picture and management of this complication is covered in Chap. 1, p. 8.

Deep (and superficial) cervical plexus

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No

☐ Yes (which?) _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: ☐ *22 G* ☐ *40 mm* ☐ *50 mm* ☐ *60 mm*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Head to contralateral side*

Needle technique: *3-needle technique (C2, C3, C4)*

☐ *Us-guided transducer* ☐ *Linear* ☐ *Curved*

☐ *In plane* ☐ *Out of plane*

Local anesthetic: _____ *ml* _____ *%* _____ *per segment*

Addition to
Injection solution: ☐ *No* ☐ *Yes* _____

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Nerve region _____ _____ _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* ____ *°C left* ____ *°C*

Horner's syndrome: ☐ *Yes* ☐ *No*

☐ *Sensory (C2, C3, C4, C5)* ☐ *Motor*

Segments affected: _____ *(numbness, warmth)*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____

Complications:

☐ *None* ☐ *Yes (intravascular, epidural, subarachnoid injection)*

Side effects:

☐ *None* ☐ *Yes (Horner's syndrome, phrenic nerve, recurrent laryngeal nerve, brachial plexus ...)* _____

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

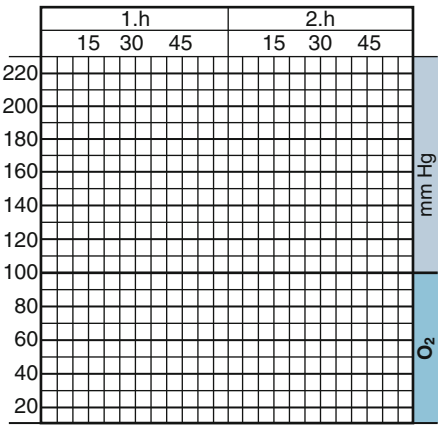
0 10 20 30 40 50 60 70 80 90 100

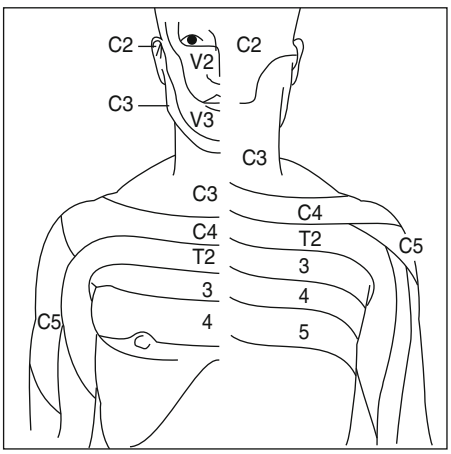
Special notes:

_____

_____

Record and checklist





With permission from Danilo Jankovic

References

1. Castresana EJ, Shaker IJ, Castresana MR. Incidence of shunting during carotid endarterectomy: Regional versus general anesthesia. Reg. Anesthesia. 1997; 22(2 Suppl).
2. Davies MJ, Silbert BS, Scott DA, Cook RJ, Mooney PH, Blyth C. Superficial and deep cervical plexus block for carotid artery surgery: a prospective study of 1000 blocks. Reg Anesth. 1997;22(5):442–6.
3. Hadzic A. Cervical plexus block. In: Hadzic A, editor. Hadzic`s Peripheral Nerve Blocks and Anatomy for Ultrasound-Guided Regional Anesthesia. 2nd ed. Mc Graw Hill; 2012. p. 140–8
4. Jankovic D. Deep and superficial plexus block. In: Jankovic A, Jankovic D, editors. Regional nerve blocks & infiltration therapy. Berlin: Blackwell Scientists; 2004. p. 76–80.
5. Johnson TR. Transient ischaemic attack during deep cervical plexus block. Br J Anesth. 1999;83:965–7.
6. Moore DC. Block of the cervical plexus. In: Moore DC, editor. Regional block. 4th ed. Springfield: Charles Thomas; 1976.
7. Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988. p. 489–514.
8. Weiss A, Isselhorst C, Gahlen J, et al. Acute respiratory failure after deep cervical plexus block for carotid endarterectomy as a result of bilateral recurrent laryngeal nerve paralysis. Acta Anaesthesiol Scand. 2005;49:715–9.

Part III
Cervical Region

Chapter 16

Cervical Interlaminar Epidural Block

Paul Gray (✉)
Department of Anesthesia, Princess Alexandra Hospital, 199 Ipswich Road, Woolloonabba, 4102, Australia
e-mail: pdgray@me.com

André van Zundert MD, PhD, FRCA, EDRA, FANZA
Professor and Chairman Discipline of Anesthesiology Chair, The University of Queensland-Faculty of Medicine and Biomedical Sciences, Royal Brisbane and Women`s Hospital-Ned Hanlon Building level 4, Herston Campus-Brisbane, Queensland 4029, Australia
e-mail: vanzundertandre@gmail.com

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_16, © Springer International Publishing Switzerland 2015

Indications

This technique was first described by Dogliotti in 1933 [1] and may be used for both surgical anesthesia and analgesia as well as for persistent pain states. The possible surgical indications include upper limb surgery [2], neck surgery [3], and breast surgery [4].

The persistent pain indication is usually restricted to the treatment of radicular pain originating from the cervical spine [5, 6]; however, it has also been recommended for chronic cervical pain [7] including post cervical surgery pain [8]. There have also been reports of using cervical epidural block for the management of complex regional pain syndrome of the upper limb [9].

Contraindications

Contraindications can be considered from a spectrum of relative to absolute. Systemic infection or local infection at the site of insertion can increase the risk of seeding bacteria into the epidural space which could result in an epidural infection, and this must be taken into account when considering the risk-to-benefit ratio of the procedure. Hereditary, acquired or induced coagulopathies are absolute contraindications as they increase the chances of epidural hematoma, which can have catastrophic consequences such as paralysis or death. Current therapy with anticoagulant medication such as warfarin or the antiplatelet agents including clopidogrel must be ceased prior to the procedure with the clinician deciding upon the need for bridging therapy based on the individual patient circumstances. Aspirin or other nonsteroidal anti-inflammatory drug therapy does not seem to increase the risk of epidural hematoma and can be continued without cessation. Special caution must be exercised when considering this procedure in patients on the newer factor Xa inhibitors (e.g., rivaroxaban) or direct thrombin inhibitors (dabigatran) as studies are lacking with regard to the optimal time period to withhold these medications. At our institution in patients with normal and hepatic function, we recommend withholding rivaroxaban for at least 3 days and dabigatran for 5 days. More detailed information on these newer agents is available from the ASRA guidelines [10]. Previous surgery of the cervical spine, particularly posteriorly, is a relative contraindication as the epidural space may be very thin if not obliterated, increasing the risk of dural puncture and spinal cord injury. In this situation the interventionist can consider accessing the space at a level below the surgical site and feeding a catheter up to the desired level of blockade. Severe spinal canal stenosis, reduced neck flexion, and ankylosing spondylitis are also relative contraindications as successfully accessing the epidural space may be significantly reduced.

Finally, caution should be exercised in those patients with a background of severe cardiovascular or respiratory disease [11].

Anatomy

An understanding of the anatomical differences at the cervical level will aid in performing the interlaminar epidural block as it allows the proceduralist to visualize the cervical spine in a three-dimensional manner and make informed choices of how best to acquire images and direct the needle under fluoroscopic or CT guidance (Figs. 16.1 and 16.2). Vertebra prominens (C7) is the most prominent of all the cervical spinous processes and aids in determining the level for injection. At the lower cervical spine, the spinous processes are quite horizontal in a similar way to the lumbar spinous processes and in contrast to the mid-thoracic region where the spinous processes are inclined (Fig. 16.3). Therefore, with flexion of the cervicothoracic junction, there is usually good separation of the spinous processes allowing access to the epidural space. The distance to the epidural space in the cervical region is similar to that in the lumbar region for that patient. It is influenced by the weight and neck size of the patient [12]. The ligamentum flavum is relatively thin when compared to the thoracic and lumbar regions resulting in a more subtle loss of resistance when the advancing needle penetrates the ligament and enters the epidural space. The approximate anterior–posterior distance of the epidural space is 1.5–2.0 mm at C7 but increases to 3.0–4.0 mm on flexion of the neck. This compares to 5.0–6.0 mm in the lumbar region. The joining of the periosteal and spinal dura at the foramen magnum forms the superior boundary of the cervical epidural space, and therefore an epidural injection should not enter the cranium. Epidural veins are concentrated in the anterolateral aspect of the epidural space; however, a blood tap is still possible with a posterior midline approach to the epidural space.

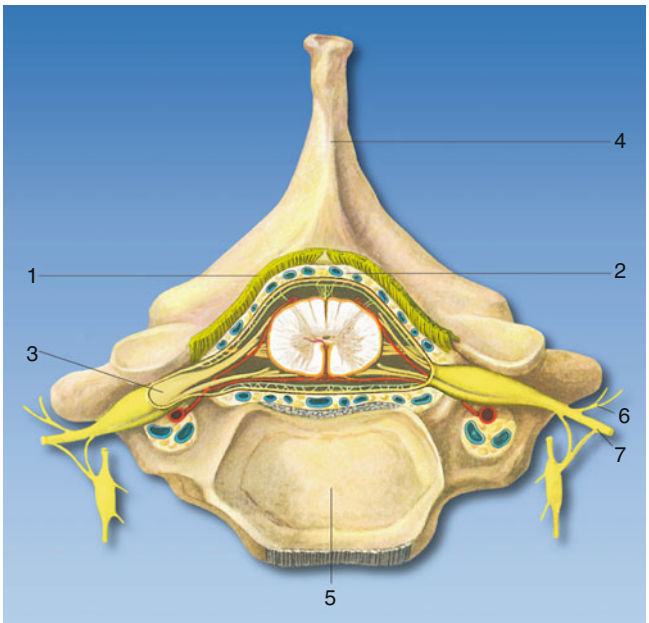


Fig. 16.1 Cross section of epidural space. (*1*) Ligamentum flavum, (*2*) epidural space with venous plexus, (*3*) spinal ganglion, (*4*) spinous process, (*5*) body of vertebra, (*6*) dorsal branch of spinal nerve, (*7*) ventral branch of spinal nerve (With permission from the Danilo Jankovic)

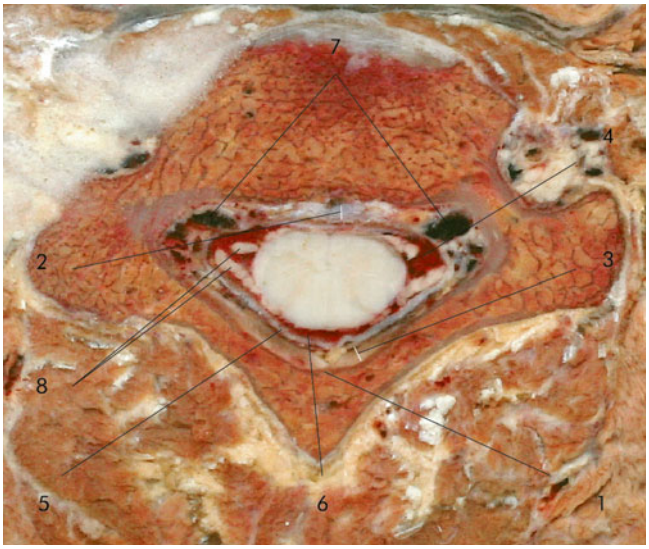


Fig. 16.2 Cross section at the level of the C6. (*1*) Ligamentum flavum, (*2*) anterior epidural space,(*3*) posterior epidural space, (*4*) subarachnoid space with the spinal cord,(*5*) spinal dura mater, (*6*) spinal pia mater, (*7*) epidural veins, (*8*) anterior and posterior spinal nerve roots (With permission from Danilo Jankovic)

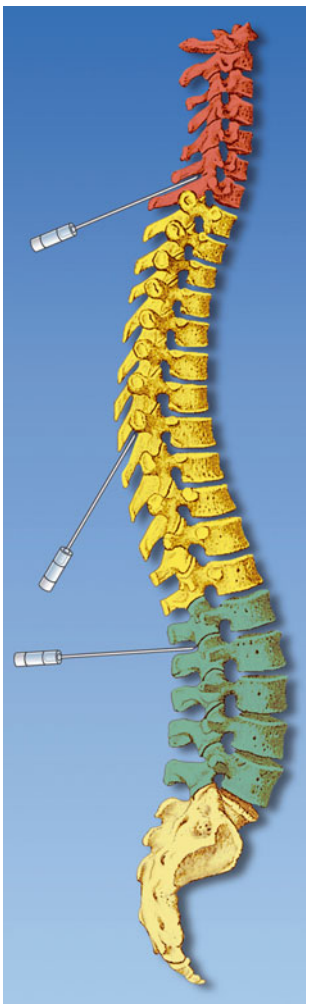


Fig. 16.3 Cervical, thoracic, and lumbar spine (With permission from Danilo Jankovic)

Technique

Informed consent for cervical interlaminar epidural block must be conducted prior to the procedure. This should include a discussion of the potential advantages and risks both general and specific to the patient. The cervical epidural interlaminar block should be reserved for the experienced interventionist who is proficient in epidural blockade at the lumbar level. Intravenous access should be established and the block performed in an area where staff, drugs, and equipment are available for resuscitation. Regular monitoring of blood pressure and pulse oximetry should occur during and for 60 min following the procedure. The block can be performed in the sitting (Fig. 16.4), prone, or lateral recumbent positions, with a supportive nurse stabilizing the patient. Pre-syncope and syncope may occur when the block is performed in the sitting position [13] with the added risk of misplacement of the needle. This risk could be reduced with intravenous loading with a crystalloid or colloid solution and doses of a vasopressor as needed. Due to this risk, the prone position is preferred and can be aided with fluoroscopy. Fluoroscopy is recommended as the use of loss of resistance alone has been shown to have a high false positive rate [14].

The cervical interlaminar epidural block is most commonly accessed between the C6/C7 and C7/T1 interspace. The volume of solution used is based on clinician preference with some authors recommending that sufficient spread is achieved with the use of 5 ml of solution [15]. Injection of similar volumes at either of these interspaces achieves a very similar spread of contrast solution [16].

When the cervical spine is in the extended position, there is the tendency for the ligamentum flavum to buckle toward the spinal cord and reduce the epidural space [17]. For this reason the patient is positioned with the cervical spine flexed as this maximizes the distance between the ligamentum flavum and the dura compared with the extended position. This flexion is achieved in the prone position through the patient having two pillows under their chest with the forehead resting on a rolled towel (Fig. 16.5).

The technique should be performed with full sterile technique using mask, sterile gloves, and gown. Due to the ligamentum flavum being thinner at the cervical region, the loss of resistance may be more reliably detected with the use of a 16-gauge Tuohy needle rather than an 18 gauge. Using loss of resistance with saline in the syringe is recommended as using loss of resistance to air can rarely result in pneumocephalus [18] in the event of a dural puncture. With the spinous processes being quite horizontal, the direction of the needle should be perpendicular to the skin and therefore perpendicular to the floor in the prone position. The right-handed interventionist would stand on the patient's left side when in the prone position. Ultimately fluoroscopy aids with direction and redirection as the needle is advanced.

On loss of resistance, careful aspiration of the needle should be performed as venous tap and dural puncture is generally only detected on aspiration of the needle. This is especially the case in the prone position due to the 10 cm of vertical height of the epidural needle.

A small volume (1–2 ml) of radiopaque contrast approved for epidural use can be injected to confirm the placement within the epidural space. Figure 16.6 demonstrates an epidurogram with a smaller volume of contrast solution, whereas Fig. 16.7 shows the normal appearance of an epidurogram with a larger volume of contrast.

If a longer-term unilateral block is required, an epidural catheter can be directed to the desired side by rotating the epidural needle 45° to that side and gently introducing the catheter [19]. This position can be confirmed with 1–2 ml of radiopaque solution approved for epidural use.

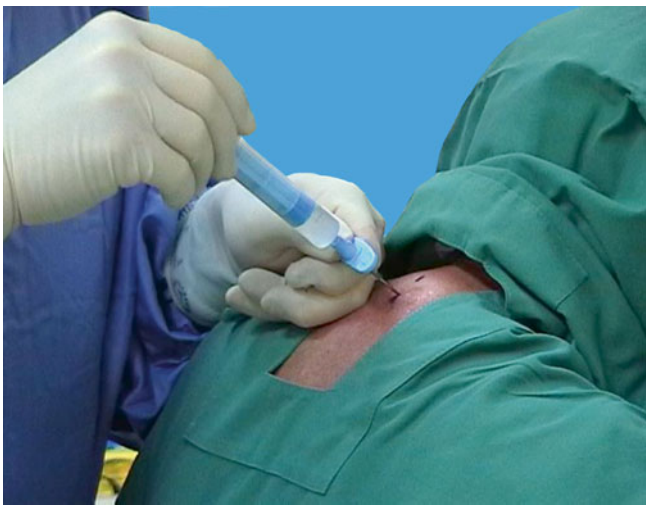


Fig. 16.4 Patient positioning for the sitting approach to the cervical epidural interlaminar block using loss of resistance to saline

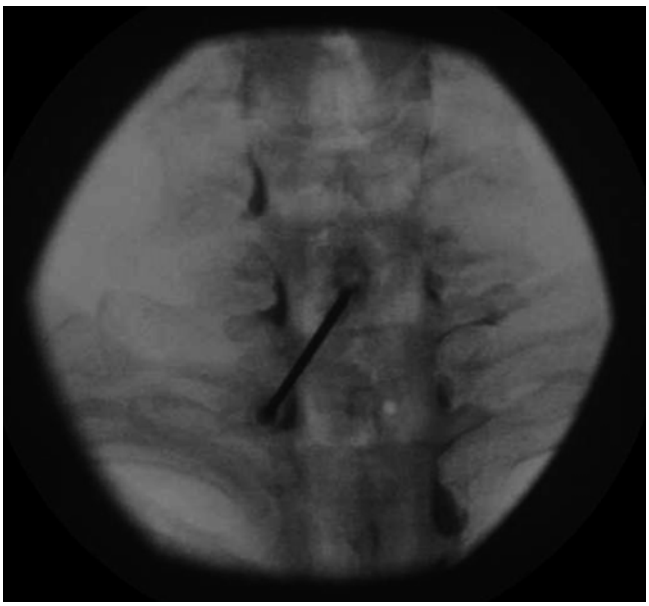


Fig. 16.6 Epidurogram of the cervical epidural space with a small volume of contrast solution. The epidural Tuohy needle is in position at the C7/T1 interspace

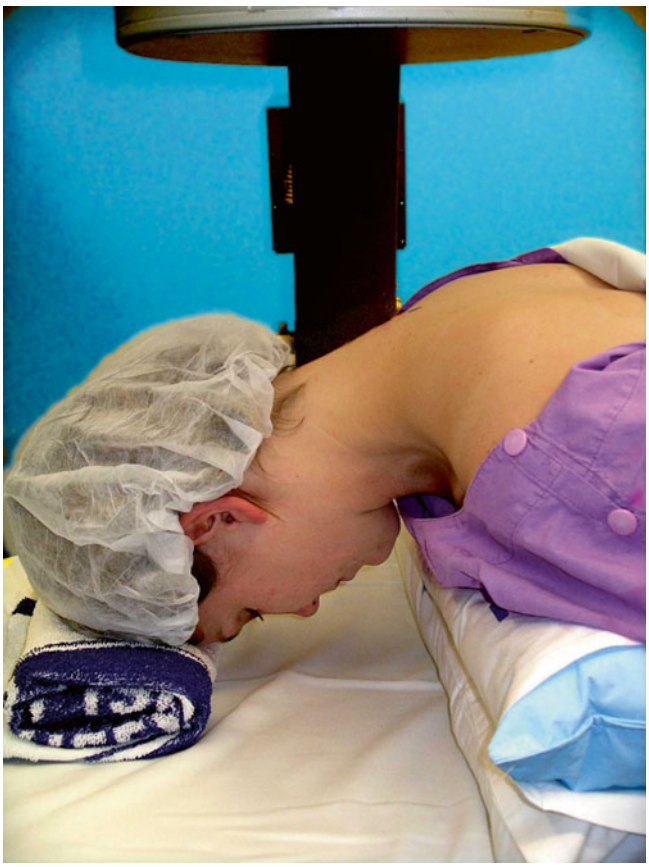


Fig. 16.5 Patient positioning for the preferred prone approach. Pillows under the chest achieve good flexion of the cervical spine while still allowing for a clear face to support breathing. Fluoroscopy can be easily performed with this position

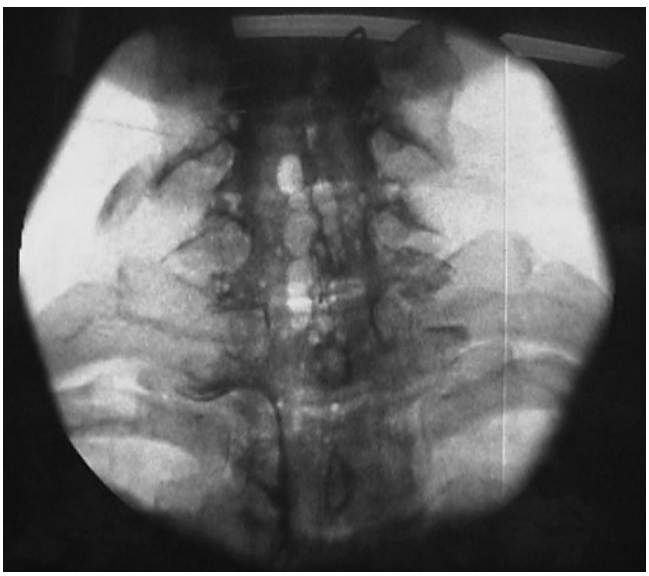


Fig. 16.7 Epidurogram of the cervical epidural space following a larger volume of contrast solution. Loss of resistance to air was used in this case with the bubbles of air visible in the epidural space following injection of the contrast solution

Complications

Injury to the spinal cord at the level of the cervical interlaminar epidural block can be devastating and may include paraplegia or death. This injury may occur via epidural hematoma, drug error, epidural infection, or direct trauma from the advancing epidural needle. However, reported cases of severe neurologic injury using the interlaminar technique are rare and significantly less when compared to transforaminal injections at the same level. The transforaminal risk is such that it has been suggested this approach be abandoned.

A case series of 394 patients described 2 inadvertent dural punctures and 6 blood taps [11]. Of the 6 venous taps, five were only detected on aspiration and one detected when the patient has a seizure following the injection of local anesthetic.

Inadvertent injections may occur into an epidural vein resulting in local anesthetic toxicity or injection into the subarachnoid space resulting in high spinal anesthesia, hence the importance of conducting the block in an environment where resuscitation can be performed.

More common adverse effects include those from the injected local anesthetic including hypotension secondary to the blocking of cardiac sympathetic fibers plus peripheral vasodilatation. This can be managed with supplemental oxygen, increased intravenous fluids, vasopressors, and atropine for the management of bradycardia and hypotension.

A mild reduction of respiratory function may be seen due to the effect of local anesthetic on the phrenic nerve (C3–C5) and therefore the contractility of the diaphragm. Of the 394 patients having carotid surgery under CEA in the case series mentioned previously, three patients developed respiratory failure requiring ventilation [11]. However, all of these patients had preexisting chronic obstructive airways disease, and the dose used in this study was a generous 15 ml of 0.5 % bupivacaine. It is also important to note that it is difficult to completely block the phrenic nerve. A cervical epidural injection of 15 ml of 2 % lignocaine at C7/T1 resulted in a 12–16 % reduction in FVC and FEV_1 but no change to maximum inspiratory pressure or SpO_2 [20].

The deposit of glucocorticosteroid may have several systemic effects. It has been reported to temporarily disrupt the hypothalamus–pituitary axis. Women may experience prolonged menses for 1 or 2 menstrual cycles. In addition, patients with diabetes mellitus may see several days of poorly controlled blood sugar levels, and it may be prudent to reduce the dose of steroid in this population.

Conclusions

The interlaminar cervical epidural block has been demonstrated to be of benefit in managing chronic radicular pain at this level secondary to a number of pathologies including disc herniation [7]. It should be reserved for the experienced interventionist, as the complications can be devastating and permanent. Patients should be carefully selected and provided with informed consent with respect to proposed advantages and potential adverse effects and complications.

Cervical epidural steroid injection

Block no.

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnosis* ☐ *Pain treatment*

Needle: ☐ *Tuohy G* ___ ☐ *Other* _____

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Sitting* ☐ *Lateral decubitus*

Access: ☐ *Median* ☐ *Paramedian*

Injection level: ☐ *C7/T1* ☐ *Other* _____

Injection technique: ☐ *Loss of resistance* ☐ *Other* _____

Test dose: ☐ *No* ☐ *Yes* _____ *mL* _____ *%*

Injection mixture: Steroid: _____ *mg*

NaCl 0.9%: _____ *mL*

Local anesthetic: _____ *mL* _____ *%*

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement before* ___ *°C after* ___ *°C*

☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 2 h* ☐ *> 2 h*

Time of discharge: _____

Abnormalities: _____

Complications:

☐ *None* ☐ *Vasovagal reactions* ☐ *Severe pain* ☐ *Fever*

☐ *Dural puncture* ☐ *Radicular symptoms* ☐ *Neurological complications*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

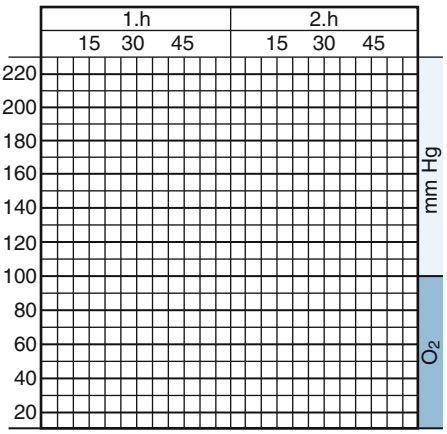
0 10 20 30 40 50 60 70 80 90 100

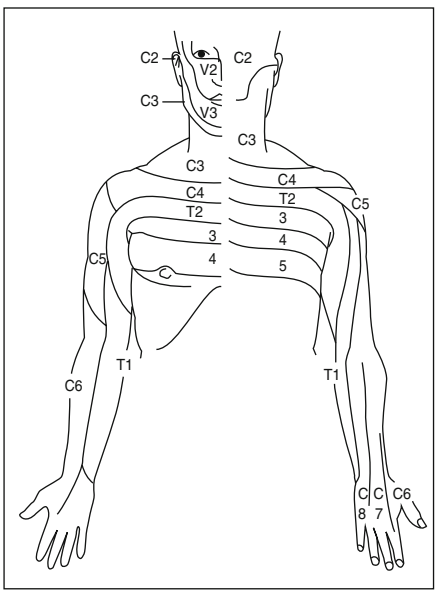
Special notes:

_____

_____

Record and checklist





With permission from Danilo Jankovic

References

1. Dogliotti AM. Segmental peridural spinal anesthesia. Am J Surg. 1933;20(1):107–18.
2. Nystrom UM, Nystrom NA. Continuous cervical epidural anesthesia in reconstructive hand surgery. J Hand Surg. 1997;22(5):906–12.
3. Ahsan SN, Faridi S. Cervical epidural anesthesia for sub-total thyroidectomy in a patient with aortic incompetence. J Pak Med Assoc. 1998;48(9):281–3.
4. Singh AP, Tewari M, Singh DK, Shukla HS. Cervical epidural anesthesia: a safe alternative to general anesthesia for patients undergoing cancer breast surgery. World J Surg. 2006;30(11):2043–7. doi:10.1007/s00268-006-0117-2; discussion 8–9.
5. Manchikanti L, Cash KA, Pampati V, Wargo BW, Malla Y. A randomized, double-blind, active control trial of fluoroscopic cervical interlaminar epidural injections in chronic pain of cervical disc herniation: results of a 2-year follow-up. Pain Physician. 2013;16(5):465–78.
6. Van Zundert J, Huntoon M, Patijn J, Lataster A, Mekhail N, van Kleef M, et al. 4. Cervical radicular pain. Pain Pract Off J World Inst Pain. 2010;10(1):1–17. doi:10.1111/j.1533-2500.2009.00319.x.
7. Benyamin RM, Singh V, Parr AT, Conn A, Diwan S, Abdi S. Systematic review of the effectiveness of cervical epidurals in the management of chronic neck pain. Pain Physician. 2009;12(1):137–57.
8. Manchikanti L, Malla Y, Cash KA, McManus CD, Pampati V. Fluoroscopic cervical interlaminar epidural injections in managing chronic pain of cervical postsurgery syndrome: preliminary results of a randomized, double-blind, active control trial. Pain Physician. 2012;15(1):13–25.
9. Kim C-T, Bryant P. Complex regional pain syndrome (type I) after electrical injury: A case report of treatment with continuous epidural block. Arch Phys Med Rehabil. 2001;82(7):993–5. doi:10.1053/apmr.2001.23955.
10. 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(1):64–101.
11. Bonnet F, Szekely B, Abhay K, Touboul C, Boico O, Saada M. Baroreceptor control after cervical epidural anesthesia in patients undergoing carotid artery surgery. J Cardiothorac Anesth. 1989;3(4):418–24.
12. Han KR, Kim C, Park SK, Kim JS. Distance to the adult cervical epidural space. Reg Anesth Pain Med. 2003;28(2):95–7.
13. Trentman TL, Rosenfeld DM, Seamans DP, Hentz JG, Stanek IP. Vasovagal reactions and other complications of cervical vs. lumbar translaminar epidural steroid injections. Pain Pract off J World Inst Pain. 2009;9(1):59–64. doi:10.1111/j.1533-2500.2008.00242.x.
14. Stojanovic MP, Vu TN, Caneris O, Slezak J, Cohen SP, Sang CN. The role of fluoroscopy in cervical epidural steroid injections: an analysis of contrast dispersal patterns. Spine. 2002;27(5):509–14.
15. Lee SE, Joe HB, Park JH, Yi IK, Choi YH, Han KR, et al. Distribution range of cervical interlaminar epidural injections: a comparative study with 2.5 mL, 5 mL, and 10 mL of contrast. Pain Physician. 2013;16(2):155–64.
16. Goel A, Pollan JJ. Contrast flow characteristics in the cervical epidural space: an analysis of cervical epidurograms. Spine (Phila Pa 1976). 2006;31(14):1576–9. doi:10.1097/01.brs.0000222020.45794.ac. 00007632-200606150-00012 [pii].
17. Young WF. Cervical spondylotic myelopathy: a common cause of spinal cord dysfunction in older persons. Am Fam Physician. 2000;62(5):1064–70, 73.
18. Simopoulos T, Peeters AC. Pneumocephalus after cervical epidural steroid injection. Anesth Analg. 2001;92(6):1576–7.
19. Buchheit T, Crews JC. Lateral cervical epidural catheter placement for continuous unilateral upper extremity analgesia and sympathetic block. Reg Anesth Pain Med. 2000;25(3):313–7.
20. Stevens RA, Frey K, Sheikh T, Kao TC, Mikat Stevens M, Morales M. Time course of the effects of cervical epidural anesthesia on pulmonary function. Reg Anesth Pain Med. 1998;23(1):20–4.

Chapter 17

Ultrasound-Guided Cervical Facet Nerve Blocks (Medial Branch and Third Occipital Nerve)

Andeas Siegenthaler
Department of Anesthesiology and Pain Therapy, University of Bern, Inselspital, Bern, Switzerland
e-mail: andisiegenthaler@gmail.com

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_17,

Introduction

The facet joints of the cervical spine are well-documented sources of chronic neck pain, with a prevalence among patients with chronic neck pain of around 40–50 % [1, 2]. To date, blocking the nerves of the facet joints with local anesthetic is the only validated method to diagnose facet joint pain [3]. In patients diagnosed with cervical facet joint-mediated pain, radiofrequency neurotomy is an effective therapy. It is supported by one high-quality randomized controlled trial [4] and several prospective observational studies [5–9]. In these trials, treatment has provided complete pain relief in 70–80 % of the selected patients for an average time of around 1 year.

Anatomy

The facet joints C3–4 to C6–7 are innervated by the medial branches of the spinal nerves' dorsal rami, with each joint being supplied by the nerve above and below the corresponding segment (Figs. 17.1 and 17.2). The nerves run across the center of the articular pillar, and injection of local anesthetic at this location is used to selectively block nociceptive input from a single joint for diagnostic purpose.

The superficial branch of C3 nerve root is well developed (the third occipital nerve, TON) and supplies C2–3 facet joint as it crosses the midportion of the C2–3 joint cleft. The medial branches' cervical dorsal rami are bound to the periosteum by an investing fascia and held against the articular pillar by tendons of semispinalis capitis (Fig. 17.2).

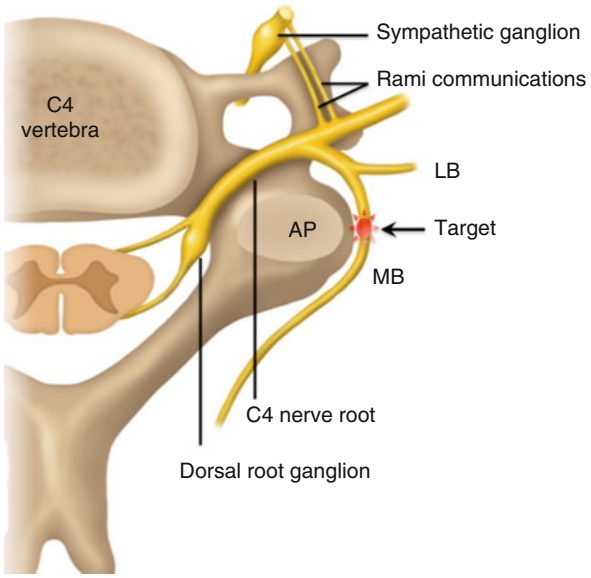


Fig. 17.1 The layout of the C4 cervical nerve root and the corresponding branch. *MB* medial branch, *LB* lateral branch, *AP* articular pillar (Reproduced with permission from Philip Peng Educational Series)

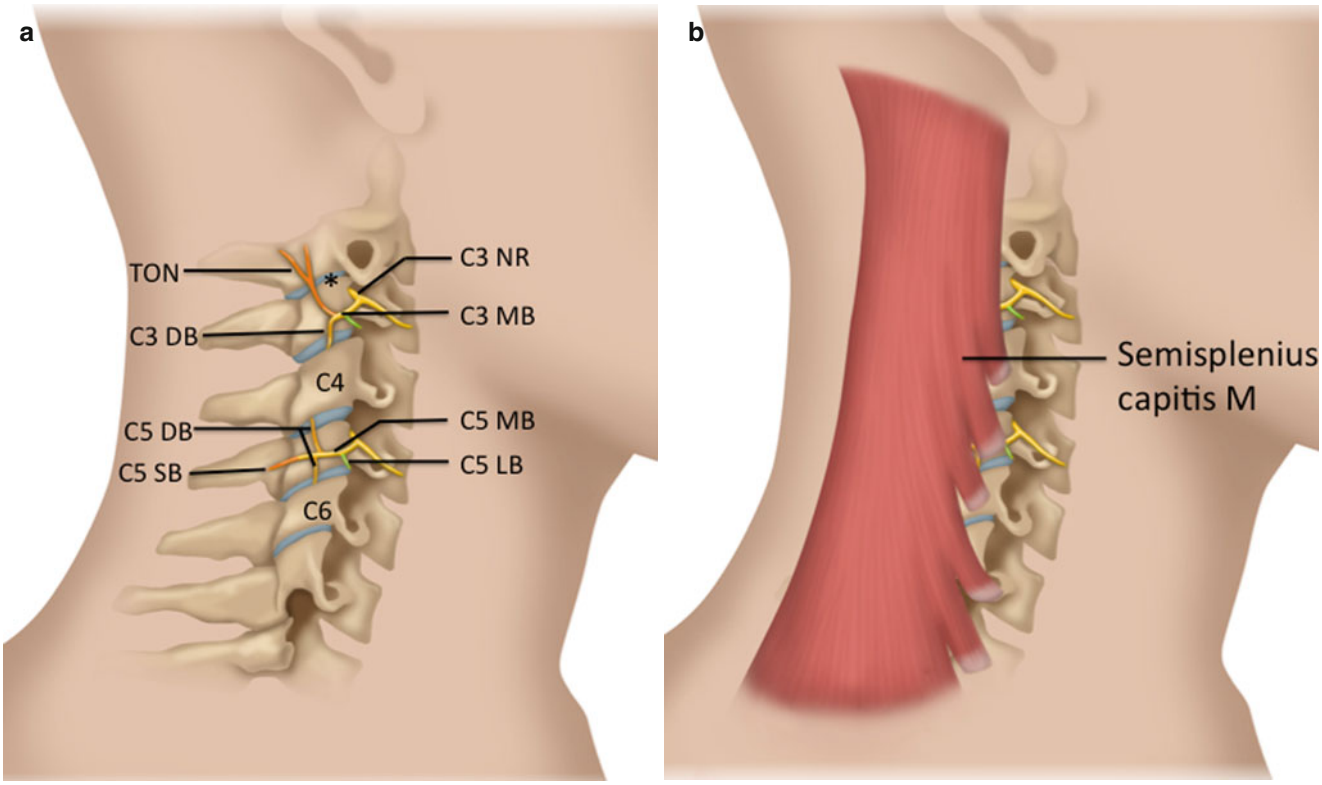


Fig. 17.2 (**a**) Schematic diagram to show the branches of cervical nerve root. *NR* nerve root, *MB* medial branch, *LB* lateral branch, *TON* third occipital nerve, *DB* deep branch of medial facet nerve, *SB* superficial branch of medial facet nerve. (**b**) Same diagram showing the overlying semispinalis capitis muscle (Reproduced with permission from Philip Peng Educational Series). * C2-3 facet joint

Technique

The classic technique of performing both diagnostic facet nerve blocks and RF neurotomy involves fluoroscopic needle guidance (Fig. 17.3).

Ultrasound imaging offers the advantage of being able to visualize the actual target nerves [10], which obviously is not possible with fluoroscopy. In a previous feasibility study performed on ten volunteers, the third occipital nerve was visible with ultrasound imaging and could reliably be anesthetized in most of the cases [11]. This opened the perspective of applying ultrasound imaging as an alternative to fluoroscopy in interventional diagnosis and treatment of neck pain. The important anatomic structures can reliably be identified with ultrasound imaging, even in a real chronic neck pain population, as determined in first exploratory study [10]. Not only the bony target can be identified, but also the medial branch itself, due to its superficial course (as opposed to the lumbar region). Further studies demonstrated the accuracy of ultrasound-guided cervical medial branch blocks, both in healthy volunteers [12] and in chronic neck pain patients [13–15].

Since the medial branch can be identified in most cases, it is expected that ultrasound imaging could facilitate the procedure of radiofrequency neurotomy in patients diagnosed with facet joint-mediated pain. The classic technique of radiofrequency neurotomy using fluoroscopy is to perform a matrix of about six thermal lesions in order to increase the chance of successful coagulation of the nerve [4], since the exact course of the nerve due to its variable course is not known. Being able to exactly determine the nerve course using ultrasound imaging is expected to decrease the number of thermal lesions needed. In 15 consecutive neck pain patients diagnosed with facet joint-mediated pain, radiofrequency neurotomy according to a shortened protocol based on prior ultrasound localization of the nerves reached the benchmark of the standard technique [8]. The author would like to emphasize that the radiofrequency procedure per se was performed solely under fluoroscopic imaging with a reduced number of thermal lesions performed according to the information of the exact nerve course gained by the prior ultrasound examination. Ultrasound is a subjective imaging technique, and verification of the exact electrode position, not least for legal reasons, is limited as compared to fluoroscopy.

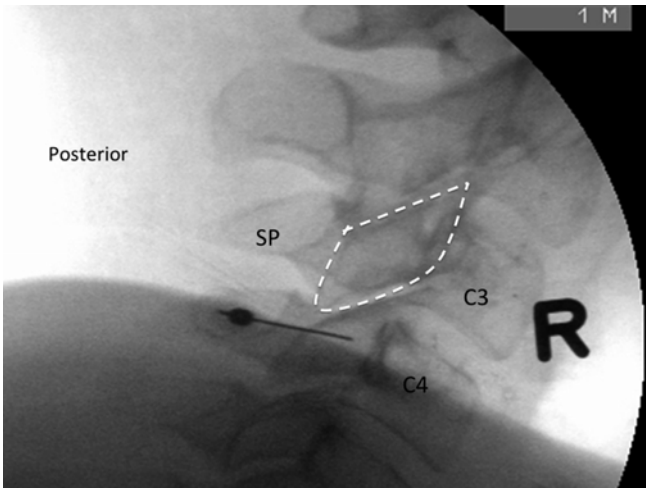


Fig. 17.3 X-ray picture showing the target of the facet medial branch block. The target is the intersection of the diagonal lines of the trapezoid (articular pillar). *SP* spinous process. (With permission of Dr. Andreas Siegenthaler)

Practical Aspects of Ultrasound-Guided Technique

With the patient placed in a lateral position, the lateral region of the neck first is scanned in a transverse plane in the area of the mastoid process, and by slowly moving the transducer in a caudal direction, the vertebral artery is identified and followed until it enters the transverse foramen of C2. Posterior the transverse foramen C2, the facet joint C2–3 is located. Here the transducer is turned 90° until it lies in a longitudinal plane to the neck. Here the typical image of the cervical facet joint region appears with each "hill" being a facet joint and each "groove" in between the joints corresponding to the articular pillar, where the medial branch can be identified as a hypoechoic structure, very much resembling a blood vessel (Fig. 17.4). The third occipital nerve (joint supplying nerve of the facet joint C2–3) is located on the joint cleft of the facet joint C2–3 (Fig. 17.5).

Counting the segments can be difficult in some patients. In this context it is helpful to know that the most cranial joint cleft identified in the typical longitudinal plane is always C2–3, since the more cranial joints C1–2 and C0–1 are located much more anterior. A further help is the region of C7. The transverse process of C7 has a unique anatomy (no anterior tubercle, very prominent posterior tubercle). This posterior tubercle is easily identified; hence the joint cleft located just cranial to this landmark will be C6–7 (Figs. 17.6 and 17.7).

Injection technique can be both in-plane (according to the publication of Finlayson and coworkers [13]) or out-of-plane. If an out-of-plane approach is chosen, it is recommended to always place the needle from anterior to posterior, since inexperienced practitioners will tend to place the needle too posteriorly (not dangerous), as opposed to a too anterior placement (i.e., toward the vertebral artery) if a posterior-to-anterior direction is chosen. The target for TON is the joint cleft of C2–3, while the targets for C3–6 medial branches are the grooves (articular pillars) where the medial branches are usually found between the articular pillar and investing layers of the semispinalis capitis. The volume of 0.3 mL of local anesthetic. For the C7 medial branch, the target is the apex of the superior articular process of C7, which is the caudal aspect of the C6–7 facet joint (Fig. 17.7).

For practitioners eager to start performing cervical facet joint blocks, it is recommended to perform the first ultrasound-guided cervical medial branch blocks with parallel fluoroscopic control. Correct level determination with ultrasound as described above may be difficult in the beginning, and the parallel use of fluoroscopy will help to develop a "feeling" for the procedure. It cannot be overemphasized that ultrasound-guided cervical facet blocks are demanding, so this book chapter ends with the citation of a critical editorial of Buvanendran and Rathmell which accompanied a publication cited above [12]: "We urge caution to practitioners eager to begin using ultrasound guidance immediately to perform cervical facet blocks in clinical practice" [16].

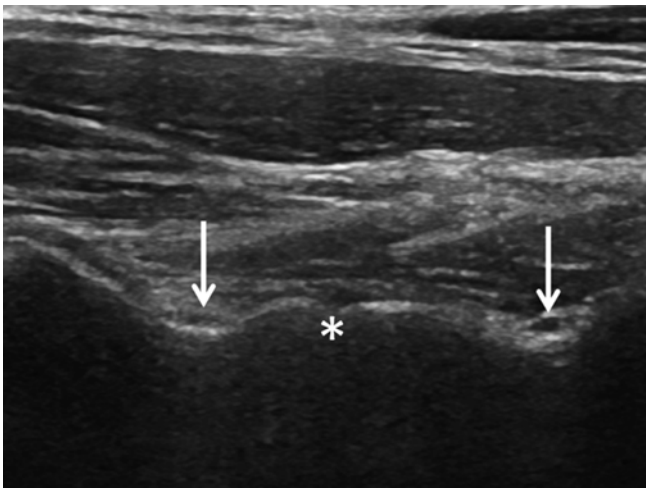


Fig. 17.4 Ultrasound anatomy of the cervical facet joint region cut in a longitudinal plane. * facet joint cleft. *Arrows* medial branch, which appears as a hypoechoic oval structure located in the middle of the "groove" between two facet joints. (With permission of Dr. Andreas Siegenthaler)

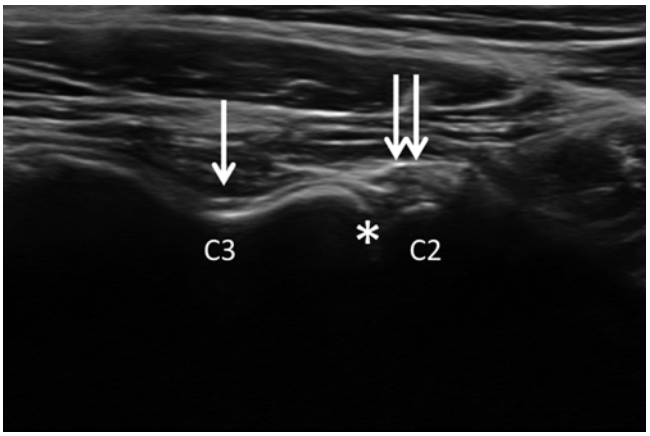


Fig. 17.5 Ultrasound image of the facet joint C2–3 in the longitudinal plane. * facet joint cleft of the joint C2–3. *Single arrow* medial branch C3. *Double arrow* third occipital nerve, which runs over the joint cleft of C2–3. (With permission of Dr. Andreas Siegenthaler)

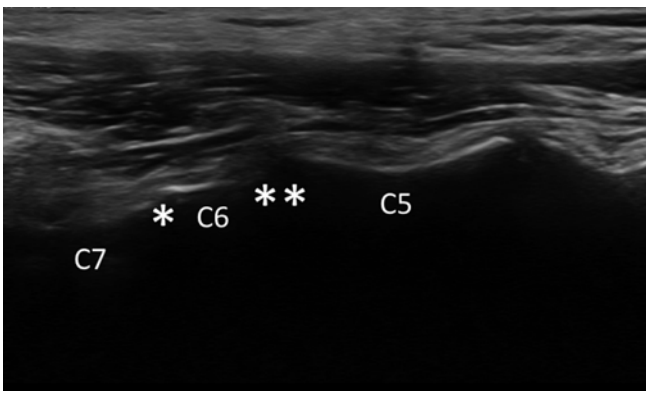


Fig. 17.6 Facet joint C6–7 (*) and C5–6 (**). (With permission of Dr. Andreas Siegenthaler)

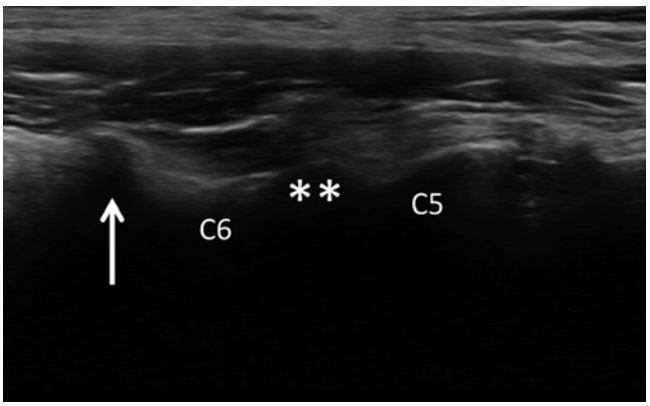


Fig. 17.7 The exactly same area in the same patient as in Fig. 17.6, with the probe tilted slightly posterior and the joint cleft of C6–7 is just not visible any more (as opposed to the joint C5–6, which still is visible **). The *arrow* marks the transverse process of C7 with its typical steep course. This helps us to determine the exact segment. (With permission of Dr. Andreas Siegenthaler)

References

1. Speldewinde GC, Bashford GM, Davidson IR. Diagnostic cervical zygapophyseal joint blocks for chronic cervical pain. Med J Aust. 2001;174:174–6.
2. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study. Spine. 1996;21:1737–45.
3. Curatolo M, Bogduk N. Diagnostic and therapeutic nerve blocks. In: Fishman SM, Ballantyne JC, Rathmell JP, editors. Bonica's management of pain. Philadelphia: Lippincott William & Wilkins; 2010. p. 1401–23.
4. Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N. Percutaneous radio-frequency neurotomy for chronic cervical zygapophyseal-joint pain. N Engl J Med. 1996;335:1721–6.
5. McDonald GJ, Lord SM, Bogduk N. Long-term follow-up of patients treated with cervical radiofrequency neurotomy for chronic neck pain. Neurosurgery. 1999;45:61–8.
6. Barnsley L. Percutaneous radiofrequency neurotomy for chronic neck pain: outcomes in a series of consecutive patients. Pain Med. 2005;6:282–6.
7. Govind J, King W, Bailey B, Bogduk N. Radiofrequency neurotomy for the treatment of third occipital headache. J Neurol Neurosurg Psychiatry. 2003;74:88–93.
8. Siegenthaler A, Eichenberger U, Curatolo M. A shortened radiofrequency denervation method for cervical zygapophysial joint pain based on ultrasound localisation of the nerves. Pain Med. 2011;12:1703–9.
9. MacVicar J, Borowczyk JM, MacVicar AM, Loughnan BM, Bogduk N. Cervical medial branch radiofrequency neurotomy in New Zealand. Pain Med. 2012;13:647–54.
10. Siegenthaler A, Schliessbach J, Curatolo M, Eichenberger U. Ultrasound anatomy of the nerves supplying the cervical zygapophyseal joints, an exploratory study. Reg Anesth Pain Med. 2011;36:606–10.
11. Eichenberger U, Greher M, Kapral S, Marhofer P, Wiest R, Remonda L, Bogduk N, Curatolo M. Sonographic visualization and ultrasound-guided block of the third occipital nerve: prospective for a new method to diagnose C2-C3 zygapophysial joint pain. Anesthesiology. 2006;104:303–8.
12. Siegenthaler A, Mlekusch S, Trelle S, Schliessbach J, Curatolo M, Eichenberger U. Accuracy of ultrasound-guided nerve blocks of the cervical zygapophysial joints. Anesthesiology. 2012;117:347–52.
13. Finlayson RJ, Gupta G, Alhujairi M, Dugani S, Tran de QH. Cervical medial branch block: a novel technique using ultrasound guidance. Reg Anesth Pain Med. 2012;37:219–23.
14. Finlayson RJ, Etheridge JP, Vieira L, Gupta G, Tran de QH. A randomized comparison between ultrasound- and fluoroscopy-guided third occipital nerve block. Reg Anesth Pain Med. 2013;38:212–7.
15. Finlayson RJ, Etheridge JP, Tiyaprasertkul W, Nelems B, Tran de QH. A prospective validation of biplanar ultrasound imaging for C5-C6 cervical medial branch blocks. Reg Anesth Pain Med. 2014;39:160–3.
16. Buvanendran A, Rathmell J. Ultrasound versus fluoroscopy in image-guided pain treatment: use caution. Anesthesiology. 2012;117:236–7.

Chapter 18

(Pulsed) Radiofrequency Treatment Adjacent to the Cervical Dorsal Root Ganglion

Jan Van Zundert, MD, PhD, FIPP (✉)
Department of Anesthesiology and Multidisciplinary Pain Centre, Ziekenhuis Oost-Limburg, Genk, Belgium
Department of Anesthesiology and Pain Management, University Hospital Maastricht, 6202 A2 Maastricht, The Netherlands
e-mail: Jan.VanZundert@zol.be

M. Puylaert, MD, FIPP • P. Van Elderen, MD, FIPP • P. De Vooght, MD
Department of Anesthesiology and Multidisciplinary Pain Centre, Ziekenhuis Oost-Limburg, Genk, Belgium

K. Van Boxem, MD, FIPP
Department of Anesthesiology and Pain Centre, AZ Nikolaas, St. Niklaas, Belgium

M. van Kleef, MD, PhD, FIPP
Department of Anesthesiology and Pain Management, University Hospital Maastricht, Maastricht, The Netherlands

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_18,

Cervical Radicular Pain

Radiofrequency (RF) treatment adjacent to the cervical dorsal root ganglion (DRG) is particularly indicated for the management of cervical radicular pain, refractory to conventional treatment. Cervical radicular pain is pain perceived in the arm, shooting or electric in quality, caused by irritation and/or injury of a cervical spinal nerve [1, 2]. It affects approximately 1 in 1,000 adults annually. There may be confusion between cervical radicular pain and cervical radiculopathy. The latter is a condition where an objective loss of sensory and/or motor function is present. Radicular pain and radiculopathy are therefore not synonymous, although they are frequently not differentiated in the literature. The former is a symptom caused by ectopic impulse generation. The latter also includes neurological signs. The two conditions may nonetheless coexist and may be caused by the same clinical entities, e.g., foraminal stenosis, intervertebral disc herniation, and radiculitis due to arteritis, infection, or inflammatory exudates [3]. The two syndromes can be part of a continuum, and radiculopathy may follow radicular pain as the underlying disease progresses.

A variety of treatment modalities for cervical radicular pain are described, but the optimal treatment approach remains unclear [1]. In clinical practice treatment is often started conservatively [4]. When conservative treatment fails to provide satisfactory outcome epidural steroid administration, (pulsed) radiofrequency or surgery may be considered as part of a multidisciplinary approach.

In cervical radicular pain the symptoms and signs are related to dysfunction of cervical spinal nerve roots and should be perceived along the distribution of the affected nerve root [2, 5, 6]. This distribution was verified in clinical experiments where radicular pain was elicited in the characteristic distributions by mechanical stimulation of cervical spinal nerves with a needle under fluoroscopic control. Bogduk summarized the distributions as follows: pain from C4 is restricted to the neck and suprascapular regions. Pain from C5 extends into the upper arm, while pain from C6 and C7 extends from the neck and shoulder into the forearm and hand. In both instances the pain covers the lateral border of the upper limb, but that of C7 extends more onto the dorsal aspect. Pain from successive spinal nerves overlaps considerably, and no particular region of the upper limb is characteristic of any particular segment [2]. Somatic referred pain from the zygapophyseal joint or from the cervical intervertebral disc can have similar distributions as radicular pain, when the pain is perceived in the proximal upper limb [7, 8]. However, when pain is distributed in the forearm and/or hand, it is far more likely to be radicular in origin. Nevertheless radicular pain should not be restricted to a dermatome and might be perceived in any of the structures innervated by the affected nerve, because cervical spinal nerves are also distributed to deep structures, such as muscles, joints, and ligaments as well as skin [2].

Diagnosis

As with other types of spinal pain, if cervical radicular pain is not resolved spontaneously within 3 months, vertebral column infections and cancer (e.g., pancoast tumor) should be ruled out before further symptomatic treatment is offered. Somatic referred pain and shoulder pathology should be excluded also, because their clinical presentation may be similar to radicular pain [2, 9]. Neurological examination of patients with cervical radicular pain includes testing of strength, muscle stretch reflexes, and sensation [10]. Five different clinical tests have been reported as useful for the diagnosis of cervical radicular pain: the neck compression test or Spurling test [11], shoulder abduction test [12], Valsalva's maneuver [13], axial manual traction test [14], and Elvey's upper limb tension test [15]. The validity of three tests, Spurling, axial manual traction, and shoulder abduction test, in the diagnosis of root compression in cervical disc disease was investigated regarding radicular pain, neurological signs, and root compression signs in myelography. All tests had a high specificity (81–100 %) but a low sensitivity (26–50 %) for the validity parameters [14]. Additionally, Spurling's test has also been validated in a controlled trial using electromyography as reference, with comparable results (sensitivity of 30 %; specificity of 93 %) [16]. Despite low sensitivity the three investigated tests are considered a valuable aid in the clinical diagnosis of a patient with neck and arm pain [14].

Besides, diagnosis of cervical radicular pain and radiculopathy requires a complete medical history, clinical diagnosis using standardized test methods of physical examination, imaging techniques, electrophysiological investigation, and determination of the symptomatic level by means of diagnostic selective nerve root blocks. Nevertheless, the discrepancy found in clinical practice between symptoms and pathology identified with imaging techniques and electrophysiological testing remains often striking. Moreover, recent research on genetically determined differences in sensitivity to pain highlights the problem of interindividual variability, which is often observed in clinical practice where pain symptoms and treatment effects vary among patients with similar clinical conditions [17, 18].

Treatment

Chronic cervical radicular pain is a complex syndrome that has a high impact on patients' quality of life [19]. An integrated approach involving psychological counseling, physical therapy, cognitive behavioral treatment, and symptomatic management of the pain is recommended [20]. The multidisciplinary evaluation also aims at providing guidance for the selection of any treatment [21, 22]. In this chapter we focus on radiofrequency/pulsed radiofrequency treatment.

The efficacy of RF treatment adjacent to the cervical DRG has been illustrated in two randomized clinical trials [23, 24]. Van Kleef et al. demonstrated a significant reduction in pain 8 weeks after RF at 67 °C compared to sham treatment [24]. Additionally, Slappendel et al. found that treatment with RF at 40 °C was equally effective as treatment at 67 °C [23]. According to two systematic reviews, there is currently limited evidence that radiofrequency treatment of the dorsal root ganglion is more effective than placebo in chronic cervical radicular pain [25, 26].

Recently pulsed radiofrequency (PRF) was introduced in clinical practice as a non- or minimal neurodestructive modification of conventional RF heat lesions [27]. A clinical audit [28] on the use of PRF treatment adjacent to the cervical DRG showed a positive outcome in 72 % of the patients after 8 weeks and in 33 % after one year. The first RCT on pulsed radiofrequency adjacent to the cervical DRG in patients with chronic cervical radicular pain was recently published. At 3 months the pulsed radiofrequency group showed a significantly better outcome with regard to the global perceived effect (>50 % improvement) and visual analog scale (20 point pain reduction). The need for pain medication was significantly reduced in the pulsed radiofrequency group after 6 months. No complications were observed during the study period [29].

The Procedure

Diagnostic Block

After the clinical diagnosis of cervical radicular pain is made, the segmental level that appears to be involved should be confirmed by diagnostic blocks at three separate levels. The technique used was previously described by van Kleef et al. [24, 30]. Overflow into the epidural space and intravascular injection can be avoided by careful observation of the spread of contrast medium by fluoroscopic real-time imaging. After the location of the cervical DRG is confirmed by injecting a small volume (approximately 0.5–1 ml) of iohexol contrast medium (Amersham Health, Cork, Ireland), an equal volume of 2 % lidocaine (AstraZeneca, Karlskoga, Sweden) should be slowly injected. Figures 18.1 and 18.2 show the spread of contrast medium.

Pain relief is assessed 10, 20, and 30 min after the procedure. A diagnostic block is considered positive if it results in a minimum of 50 % pain reduction, measured on the VAS within 30 min. The level that responds with the largest pain reduction is selected for intervention.

Fig. 18.1 Injection of contrast medium

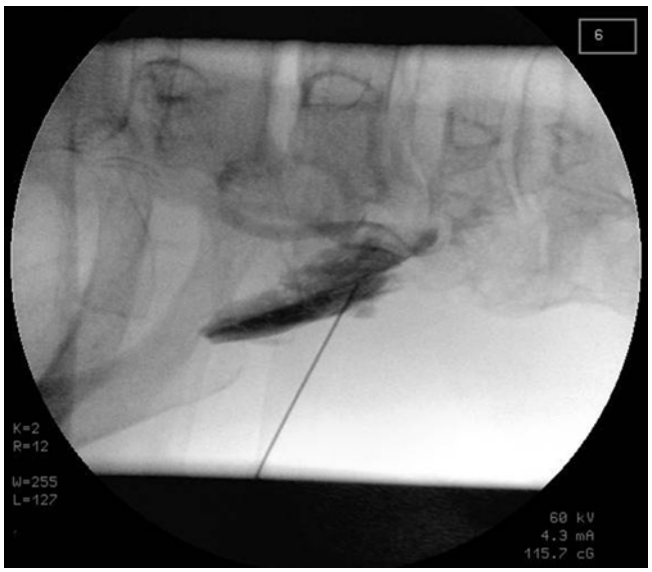


Fig. 18.2 Spread of contrast medium at C7 level

Radiofrequency Treatment

A technique similar to the one for performing diagnostic nerve blocks is used for the interventions [24, 30]. The C-arm of the fluoroscopy unit (Philips BV 25, Eindhoven, the Netherlands) is positioned with the beam parallel to the axis of the intervertebral foramen (25–35° anteriorly and 10° caudally). The entry point is located by projecting a metal ruler over the caudal and posterior part of the foramen (Fig. 18.3). The 22-G cannula (SMK Pole needle 54 mm with 4 mm active tip, Cotop International BV, Amsterdam, the Netherlands) is introduced parallel to the beam, and, if necessary, the position is corrected in the superficial subcutaneous layers until the cannula was projected on the screen as a single dot (Fig. 18.4). In practice, this dot should lie directly over the dorsal portion of the intervertebral foramen at the transition between the middle and most caudal third. This position is chosen to avoid possible injection into the vertebral artery, which runs anterior to the ventral part of the foramen. The fluoroscope is then adjusted to the anterior–posterior view, and the cannula is inserted further until the tip projects over the middle of the facetal column (Fig. 18.5).

The stylet of the cannula is then replaced by the RF probe (SMK-TC 5, Radionics, Burlington, MA). After checking the impedance, indicating a normal, closed electrical circuit, stimulation is started at a frequency of 50 Hz to obtain a sensory stimulation threshold.

A paresthesia is elicited along the tested cervical nerve root at less than 0.5 V and is considered to indicate adequate proximity of the DRG [31].

The treatment can be performed using either conventional radiofrequency or pulsed radiofrequency current.

Radiofrequency current is applied in order to increase the temperature at the tip slowly to 67 °C. This temperature is maintained for 60 s.

The PRF current is applied during 120 s from the lesion generator (Radionics RFG 3 C Plus, Burlington, MA) as described by Sluijter et al. [27]. With the use of PRF the output is set at 45 V so that the temperature at the electrode tip does not exceed 40 °C.

Fig. 18.3 Identification of the needle insertion point

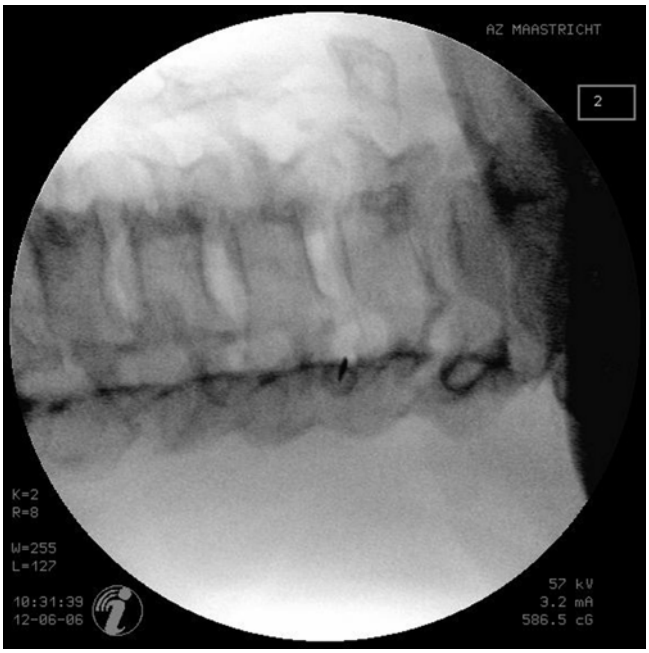


Fig. 18.4 Radiofrequency lesion adjacent to the dorsal root ganglion (RF-DRG) 20° oblique, 10° caudocranial projection. The needle is positioned in the posterior aspect of the foramen, at the junction of the middle and caudal third part. It is projected as a dot in tunnel vision

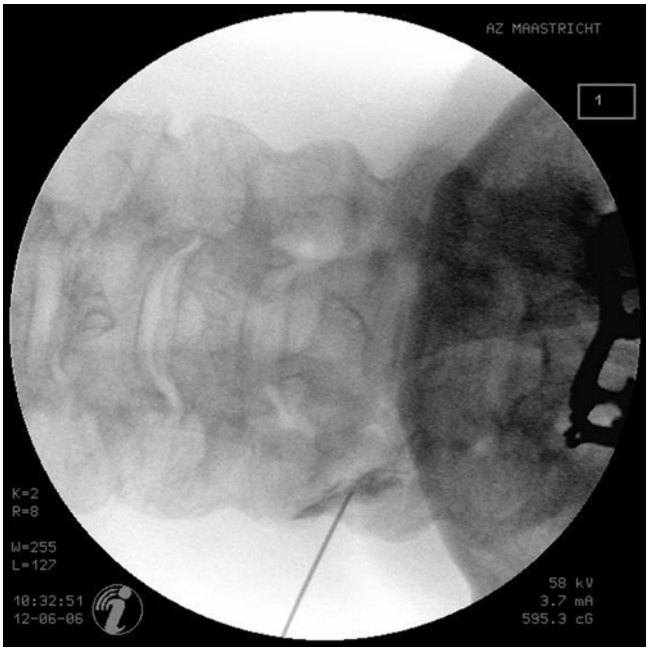


Fig. 18.5 RF-DRG AP view. The tip of the needle is projected over the facetal column

Side Effects and Complications

The most frequently reported side effect with conventional radiofrequency treatment is a vague burning sensation in the treated dermatome. This pain usually subsided spontaneously 6 weeks after treatment. Also hyposensibility may be observed in the treated dermatome. Though rarely occurring, the most troublesome complication of conventional radiofrequency treatment is the deafferentation pain.

In the current experience with pulsed radiofrequency treatment adjacent to the cervical DRG, no side effects and complications have been reported.

Conclusions

Cervical radicular pain may be refractory to pharmacological treatment and rehabilitation. Before offering symptomatic treatment, infection and cancer (e.g., pancoast) should be excluded. After careful neurological examination, to rule out shoulder pathology, the causative level should be confirmed by means of diagnostic blocks. Epidural corticosteroid administration, by intralaminar or transforaminal route, may be a treatment option. There is however no comparison between both techniques, though the transforaminal administration route has gained in interest because it is supposed to deliver the drug as close as possible to the inflammatory nerve root [1, 32, 33] Recently published case reports on serious adverse events after transforaminal steroid administration at the cervical level urge for a cautious use of this treatment. Surgical techniques for decompression with or without anterior interbody fusion are often performed to reduce the pain and disability, but are associated with a small but definite risk [34]. A randomized controlled trial indicates that a multidisciplinary treatment with cognitive behavioral therapy and psychological interventions are preferred over surgery [35]. Systematic reviews showed that there is little evidence for better efficacy of surgery compared to cognitive behavioral treatment, justifying the higher risk for complications [34, 36].

The available evidence on radiofrequency treatment adjacent to the cervical DRG suggests pain relief and improved patient satisfaction. The evidence on PRF adjacent to the cervical DRG indicates potentially similar clinical results. The perceived better safety of the latter technique justifies trying this minimal invasive procedure prior to other interventional treatment options.

References

1. Rathmell JP, Aprill C, Bogduk N. Cervical transforaminal injection of steroids. Anesthesiology. 2004;100(6):1595–600.
2. Bogduk N. Medical management of acute cervical radicular pain: and evidence-based approach. 1st ed. Newcastle: The Newcastle Bone and Joint Institute; 1999.
3. Merskey H, Bogduk N. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. 2nd ed. Seattle: IASP Press; 1994.
4. Saal JS, Saal JA, Yurth EF. Nonoperative management of herniated cervical intervertebral disc with radiculopathy. Spine. 1996;21(16):1877–83.
5. Devereaux MW. Neck and low back pain. Med Clin North Am. 2003;87(3):643–62.
6. Bland J. Cervical spine syndromes. J Muscoloskel Med. 1986;3:23–41.
7. Dwyer A, Aprill C, Bogduk N. Cervical zygapophysial joint pain patterns. I: A study in normal volunteers. Spine. 1990;15(6):453–7.
8. Schellhas KP, Smith MD, Gundry CR, Pollei SR. Cervical discogenic pain. Prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. Spine. 1996;21(3):300–11; discussion 11–2.
9. Date E, Gray L. Electrodiagnostic evidence for cervical radiculopathy ans suprascapular neuropathy in shoulder pain. Electromyogr Clin Neurophysiol. 1996;36:333–9.
10. Fager CA. Identification and management of radiculopathy. Neurosurg Clin N Am. 1993;4(1):1–12.
11. Spurling R, Scoville W. Lateral rupture of the cervical intervertebral discs: a common cause of shoulder and arm pain. Surg Gynecol Obstet. 1944;78:350–8.
12. Davidson RI, Dunn EJ, Metzmaker JN. The shoulder abduction test in the diagnosis of radicular pain in cervical extradural compressive monoradiculopathies. Spine. 1981;6(5):441–6.
13. Rothstein J, Roy S, Wolf S. The rehabilitation specialist's handbook. Philadelphia: FA Davis; 1991.
14. Viikari-Juntura E, Porras M, Laasonen EM. Validity of clinical tests in the diagnosis of root compression in cervical disc disease. Spine. 1989;14(3):253–7.
15. Elvey R. The investigation of arm pain: signs and adverse responses to the physical examination of the brachial plexus and related tissues. In: Boyling J, Palastanga N, editors. Grieve's modern manual therapy. New York: Churchill Livingstone; 1994. p. 557–85.
16. Tong HC, Haig AJ, Yamakawa K. The Spurling test and cervical radiculopathy. Spine. 2002;27(2):156–9.
17. Mogil JS. The genetic mediation of individual differences in sensitivity to pain and its inhibition. Proc Natl Acad Sci U S A. 1999;96(14):7744–51.
18. Mogil JS, Wilson SG, Bon K, Lee SE, Chung K, Raber P, Pieper JO, Hain HS, Belknap JK, Hubert L, Elmer GI, Chung JM, Devor M. Heritability of nociception II. 'Types' of nociception revealed by genetic correlation analysis. Pain. 1999;80(1–2):83–93.
19. Daffner SD, Hilibrand AS, Hanscom BS, Brislin BT, Vaccaro AR, Albert TJ. Impact of neck and arm pain on overall health status. Spine. 2003;28(17):2030–5.
20. Van Zundert J, Raj P, Erdine S, van Kleef M. Application of radiofrequency treatment in practical pain management: state of the art. Pain Pract. 2002;2(3):269–78.

21. Kerns RD, Habib S. A critical review of the pain readiness to change model. J Pain. 2004;5(7):357–67.
22. Wainner RS, Gill H. Diagnosis and nonoperative management of cervical radiculopathy. J Orthop Sports Phys Ther. 2000;30(12):728–44.
23. Slappendel R, Crul BJ, Braak GJ, Geurts JW, Booij LH, Voerman VF, de Boo T. The efficacy of radiofrequency lesioning of the cervical spinal dorsal root ganglion in a double blinded randomized study: no difference between 40 degrees C and 67 degrees C treatments. Pain. 1997;73(2):159–63.
24. van Kleef M, Liem L, Lousberg R, Barendse G, Kessels F, Sluijter M. Radiofrequency lesion adjacent to the dorsal root ganglion for cervicobrachial pain: a prospective double blind randomized study. Neurosurgery. 1996;38(6):1127–31; discussion 31–2.
25. Geurts J, van Wijk RM, Stolker R, Groen GJ. Efficacy of radiofrequency procedures for the treatment of spinal pain: a systematic review of randomized clinical trials. Reg Anesth Pain Med. 2001;26:394–400.
26. Niemisto L, Kalso E, Malmivaara A, Seitsalo S, Hurri H. Radiofrequency denervation for neck and back pain. A systematic review of randomized controlled trials. Cochrane Database Syst Rev. 2003;(1):CD004058.
27. Sluijter ME, Cosman ER, Rittman IIWB, van Kleef M. The effects of pulsed radiofrequency field applied to the dorsal root ganglion – a preliminary report. Pain Clin. 1998;11(2):109–17.
28. Van Zundert J, Lamé IE, de Louw A, Jansen J, Kessels F, Patijn J, van Kleef M. Percutaneous pulsed radiofrequency treatment of the cervical dorsal root ganglion in the treatment of chronic cervical pain syndromes: a clinical audit. Neuromodulation. 2003;6(1):6–14.
29. Van Zundert J, Patijn J, Kessels A, Lame I, van Suijlekom H, van Kleef M. Pulsed radiofrequency adjacent to the cervical dorsal root ganglion in chronic cervical radicular pain: a double blind sham controlled randomized clinical trial. Pain. 2007;127:173–82.
30. van Kleef M, Spaans F, Dingemans W, Barendse GAM, Floor E, Sluijter ME. Effects and side effects of a percutaneous thermal lesion of the dorsal root ganglion in patients with cervical pain syndrome. Pain. 1993;52:49–53.
31. Ford DJ, Pither C, Raj PP. Comparison of insulated and uninsulated needles for locating peripheral nerves with a peripheral nerve stimulator. Anesth Analg. 1984;63(10):925–8.
32. Rowlingson JC. Epidural steroids: do they have a place in pain management? Barcelona: V. Permanyer Publications; 1996.
33. Hession WG, Stanczak JD, Davis KW, Choi JJ. Epidural steroid injections. Semin Roentgenol. 2004;39(1):7–23.
34. Fouyas IP, Statham PF, Sandercock PA. Cochrane review on the role of surgery in cervical spondylotic radiculomyelopathy. Spine. 2002;27(7):736–47.
35. Persson LC, Lilja A. Pain, coping, emotional state and physical function in patients with chronic radicular neck pain. A comparison between patients treated with surgery, physiotherapy or neck collar–a blinded, prospective randomized study. Disabil Rehabil. 2001;23(8):325–35.
36. Jacobs W, Anderson P, Limbeek J, Willems P, Pavlov P. Single or double-level anterior interbody fusion techniques for cervical degenerative disc disease. Cochrane Database Syst Rev. 2004;(4):CD004958.

Chapter 19

Cervical Percutaneous Facet (Zygapophyseal Joint = ZA Joint) Denervation

Olav Rohof, MD, PhD, FIPP (✉)
Painclinic, Atrium Orbis location Sittard Geleen, H. van der Hoffplein 1, 6162 BG Sittard Geleen, The Netherlands
e-mail: o.rohof@orbisconcern.nl

Robert Jan Stolker, MD, PhD
Professor and Chair, Department of Anesthesiology, Erasmus MC, P.O. Box 2040, 3000 CA Rotterdam, The Netherlands
e-mail: r.stolker@erasmusmc.nl

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_19,

Anatomy

Zygapophyseal or facet joints are formed by the inferior and superior processes of two adjacent joints. These joints limit excessive mobility and distribute the axial loading over a broad area. The facet joints located at the dorsal side of the spine together with the intervertebral disc at the ventral side form the three moving parts of the motion segment. Compared to the lumbar facet joints, the cervical facet joints are relatively big joints. The facet joints are innervated by the medial branch of the dorsal ramus of the spinal nerve.

The dorsal and ventral roots come together to form the spinal nerve. This nerve splits into a ventral and a dorsal ramus or branch [1]. The dorsal ramus divides into a medial and a lateral branch. The dorsal or posterior ramus supplies the so-called dorsal compartment of the neck, which consists of structures of the neck situated behind the intervertebral foramen. This dorsal compartment contains muscles, ligaments, blood vessels, and the facet joints. Unique to the cervical spine is the vertebral artery, which passes through the transverse foramen of the transverse processes of the C6–C1 vertebrae (Fig. 19.1).

The facet joints are richly innervated by medial branches of the dorsal ramus from the corresponding level and by the medial branch of the dorsal ramus one level above. For example, the facet joint C3–C4 is innervated by the dorsal rami C3 and C4. Each dorsal ramus innervates two facet joints, and each facet joint is innervated from two levels. The innervation of the C2–C3 facet joint is more complex, since this joint is mainly supplied by the third occipital nerve, which is one of the two medial branches of the C3 dorsal ramus and to some degree also by the C2 dorsal ramus. The C2 medial branch is commonly known as the greater occipital nerve [1]. The practical implication of this observation is that for the treatment of facet joint pain at the level C2–C3, the third occipital nerve and the medial branch of the C2 dorsal ramus are required.

The medial branch innervates skin, ligaments, and muscles. The lateral branch innervates muscles. The medial branches have to be blocked in the concavity between the superior and inferior articular processes, in the "waist" of the vertebra. The medial branches are bound to the periosteum by an investing fascia and are held against the articular pillars by tendons of the semispinalis capitis muscle.

The technique of lumbar facet denervation was first described by Shealy in 1975 [2]. The large thermistor electrodes used at that time made the procedure difficult to perform at the cervical level. The introduction of the SMK system with a much smaller diameter made the application at the cervical level possible without the risk of mechanically induced tissue damage.

As the innervation of the joint is always from two levels, the procedure must always be done at two levels, in case of pain from two adjacent joints at three levels; in our practice we mostly perform RF lesioning at the C2 to C6 level in one session.

The procedure, which is mostly performed in an outpatient setting, can be done uni- or bilaterally, depending on the pain pattern and consequently the causative structures. Preferentially the procedure is performed in conscious patients allowing continuous communication. Therefore, the use of sedatives such as propofol, alfentanil, or remifentanil is rarely necessary, unless the patient is too anxious and the procedure is performed bilaterally in the same session.

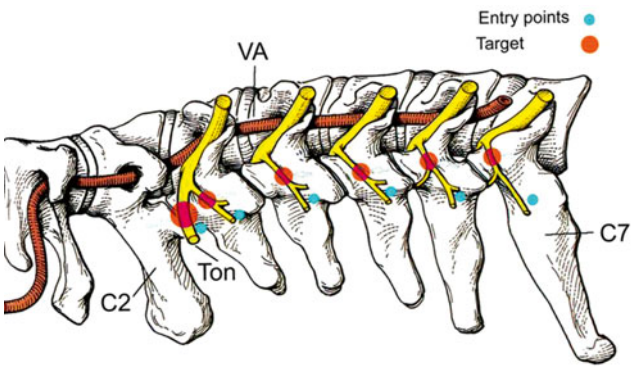


Fig. 19.1 Anatomy of the cervical spine with innervation; entry and target points are marked (*VA* vertebral artery, *TON* third occipital nerve)

Indications

Painful facet joints may cause pain in the neck, head, shoulder, arm, or interscapular region; the upper segments may also cause facial pain or may be a trigger for migraine and cluster headache. The upper zygapophyseal joints may play an important role in the development of cervicogenic headache. The indications listed below have been documented.

1. Pain from cervical zygapophyseal joint origin, which can give rise to neck pain, irradiation to the head (headache) and shoulder, the interscapular region, and the arm
2. Post-traumatic neck pain [3]
3. Cervicogenic headache [4]
4. Atypical facial pain and some forms of cluster headache and migraine caused by a cervicogenic trigger mechanism from the facet joints.

The possible signs and symptoms of facet pain in the cervical region are listed in Table 19.1

Uni- or bilateral pain lasting longer than 3–6 months not reacting to physical therapy and other conservative management could be an indication for percutaneous facet denervation (PFD). In the absence of a specific facet syndrome, most authors advocate to perform a test block prior to a PFD. This test block is a medial branch block or an intra-articular block with local anesthetics. Some authors add steroids in order to achieve a longer-lasting effect because steroids reduce potential joint inflammation, but in a placebo-controlled study, this strategy has proved to be ineffective on longer term.

Radiological findings per se, such as facet arthritis, are not an indication for a PFD. On the other hand, there may be facet pain without any radiological abnormality.

Contraindications for cervical facet denervation are summarized in Table 19.2.

Table 19.1 Possible signs and symptoms of facet pain in the cervical region

Neck pain with or without irradiation to the shoulder, arm, interscapular region, or face
Paravertebral tenderness, a sign of real facet pain in the majority of cases
Pain on rotation and ante-/retroflexion of the neck
Radiculopathy is absent (no neurological deficit)
Morning stiffness

Table 19.2 Contraindications for cervical facet denervation

Sensory loss
Lack of cooperativeness
Bleeding disorders or use of anticoagulants
Signs of local infection
Signs of local malignancy
Allergy to local anesthetics

Procedure

The material for cervical facet denervation is listed in Table 19.3.

This procedure should be performed by an experienced anesthesiologist or under his/her supervision and only with the use of a fluoroscope.

For the upper cervical facet joints (C2–C6), the patient lays in the supine position with his/her head on a special radiolucent rest, allowing free access to the neck using a fluoroscope. The fluoroscope is in line with the patient (Fig. 19.2). For the lower joints (C7 and in short necks C6), the prone position is advised, and the approach and fluoroscopic control is similar as for the lumbar and thoracic region.

For the upper facets, the C-arm is positioned to obtain a clear lateral view of the cervical spine; the image on the screen should be adjusted in such a way that the picture you see coincides with the patient's position: the spine in a horizontal position with the head at the upper end and the shoulder at the bottom and the cervical discs pointing upward (Fig. 19.2).

The insertion place is identified using a plastic caliper with a metallic ring. The ring should be visible at the lower end of the trapezium formed by the lamina; the needle insertion point is then marked on the skin.

The target point for the levels C3 to C6 is the middle of the trapezium formed by the lamina with a little proximal adjustment. The target for the C2–C3 level is the middle of the joint space and a few mm more cephalad and caudal (2–3 positions) (Figs. 19.3 and 19.4).

After disinfection of the skin, 0.5 ml lidocaine 1–2 % is (sub)cutaneously injected, the RF thermocouple needle is inserted for 1–2 cm, and its position is controlled by fluoroscopic viewing. The direction of the needle is, if necessary, corrected and the needle is advanced until bone contact at the middle of the lamina is made. After correcting the needle position slightly, the tip of the needle is advanced just a little more, keeping bone contact. The depth of the needle is controlled by a fluoroscopic oblique and AP view. In the oblique view (Fig. 19.4), the point of the needle should be at the proximal end of the pedicle and may not be deeper than the line connecting the posterior aspects of the intervertebral foramina.

Table 19.3 Material for cervical facet denervation

Two to six 5-cm 22-G radiofrequency needles with a 4-mm uninsulated tip or two to six 6-cm TOP XE or CXE needles with a 4-mm uninsulated tip
Local anesthetic (e.g., lidocaine 2 %)
Thermocouple electrode of 5 cm length
Radiofrequency lesion generator
Ground plate
Connecting wires

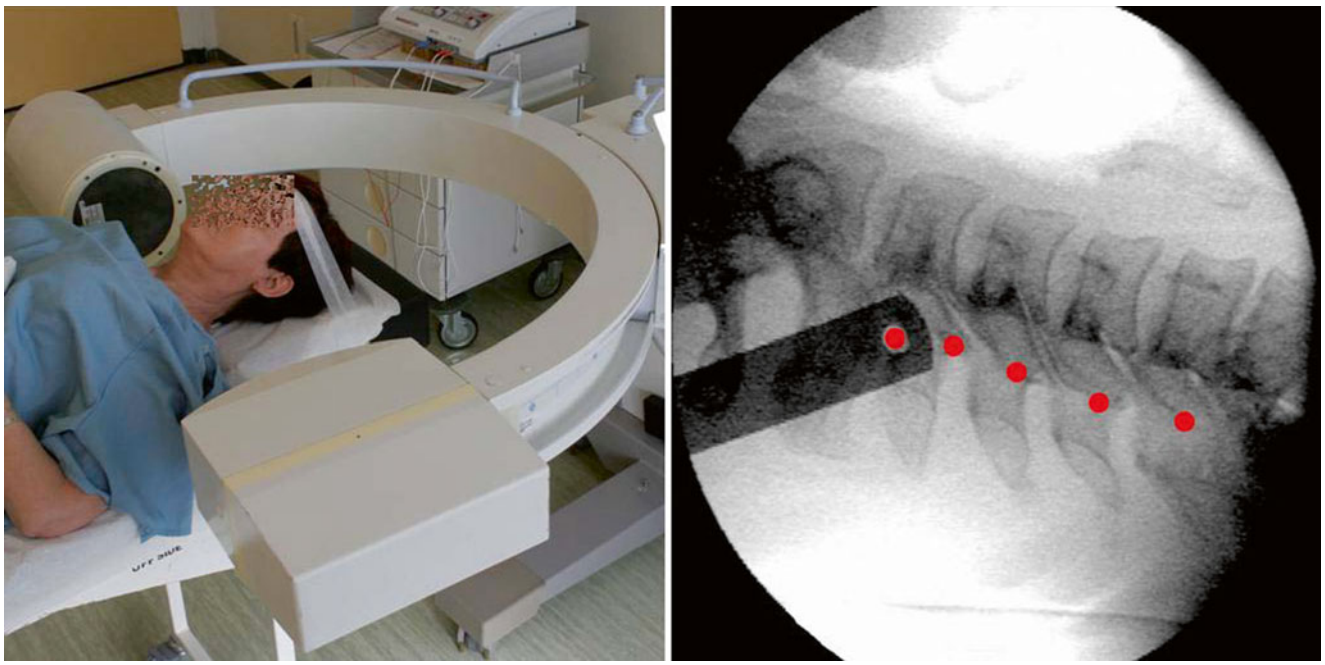


Fig. 19.2 Patient and fluoroscope positioning, lateral view, marking the entry points

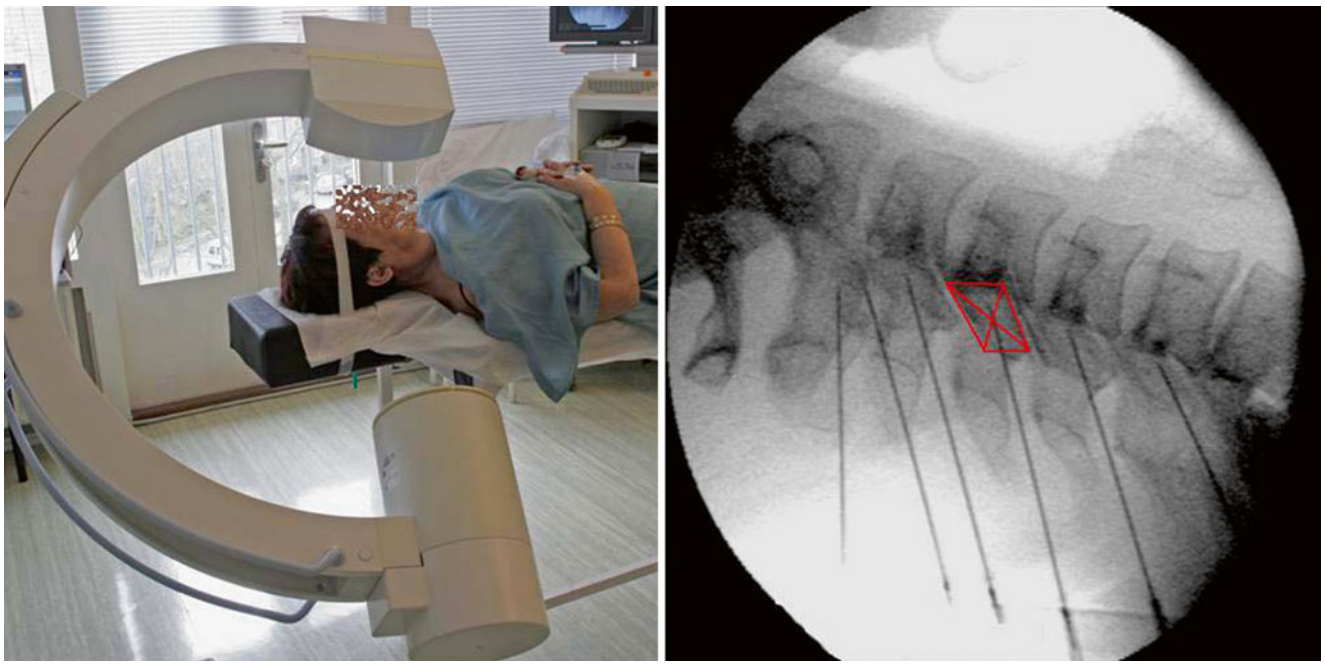


Fig. 19.3 Fluoroscopic transverse view for percutaneous facet denervation C2–C6, target at the cross point at the trapezium of lamina C4 is marked, needles in place

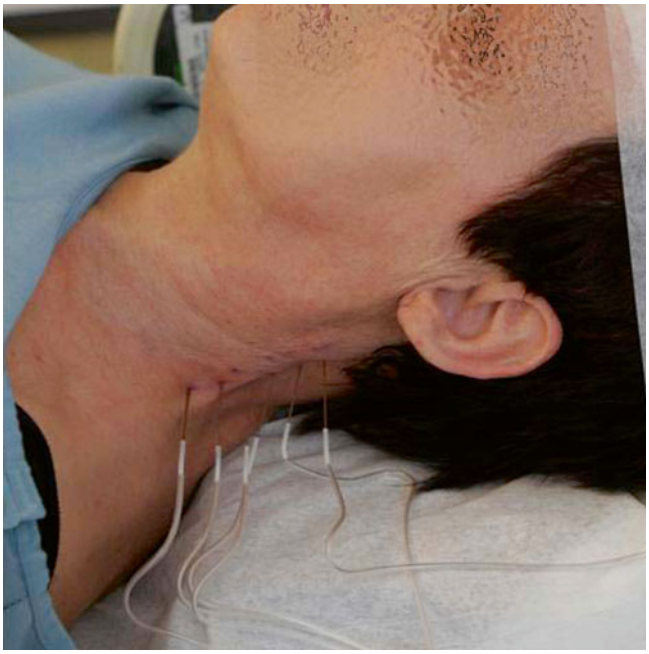


Fig. 19.4 X-oblique view, RF needles in place at the proximal end of pedicle C2–C6, bone contact

In the AP view, the tips should be adjacent to the spine in the concavity ("waist") between the articular processes (Fig. 19.5).

After obtaining the correct anatomical position, the stylet of the needle is removed, and a thermocouple electrode inserted. The electrode has to be connected to the lesion generator. The ground plate is connected. Stimulation with a 50 Hz is performed, and the patient is asked to signal when he feels tingling in the neck. Stimulation threshold must be less than 1 V. The lower the threshold, the closer the needle is to the nerve. A tingling feeling in the arm refers to a too anterior position of the needle (too close to the spinal nerve), which has to be withdrawn. The same stimulation procedure can be done at 2 Hz. As the medial branch is a mixed nerve, a response at the same threshold (or 0.1 V higher) is expected. Local contractions will occur. Contractions of the arm or diaphragm (C4) are a sign of a too anterior position, close to the spinal nerve. A threshold for motor response of the arm at 2 V or less is unacceptable and requires repositioning.

If these thresholds are met, 0.5 ml lidocaine must be injected after careful aspiration in each needle in order to anesthetize the site of the thermolesion.

After waiting for 1–2 min, the radiofrequency lesion at 80 °C for 60 s can be made.

RF thermocouple needles require multiple manipulations, which may result in displacement of the needle tip especially in thin patients. This may result in neurological complications or in making an insufficient lesion. The author prefers using TOP XE or CXE needles as no further manipulation after carefully controlled needle placement is required. Lesions of 20 V for 60 s are made (without temperature control) because 20 V coincides mostly with 80 °C.

It was however found that 20 V lesions may result in a temperature of 100 °C, thus causing severe postoperative discomfort and sensory skin dysesthesia. After modifying the lesion parameters, the incidence of postoperative discomfort was considerably reduced, but the clinical outcome remained good. We now start the procedure at 20 V but reduce the voltage guided on the patient's report of pain during the lesioning. We sometimes end the procedure at 13 V.

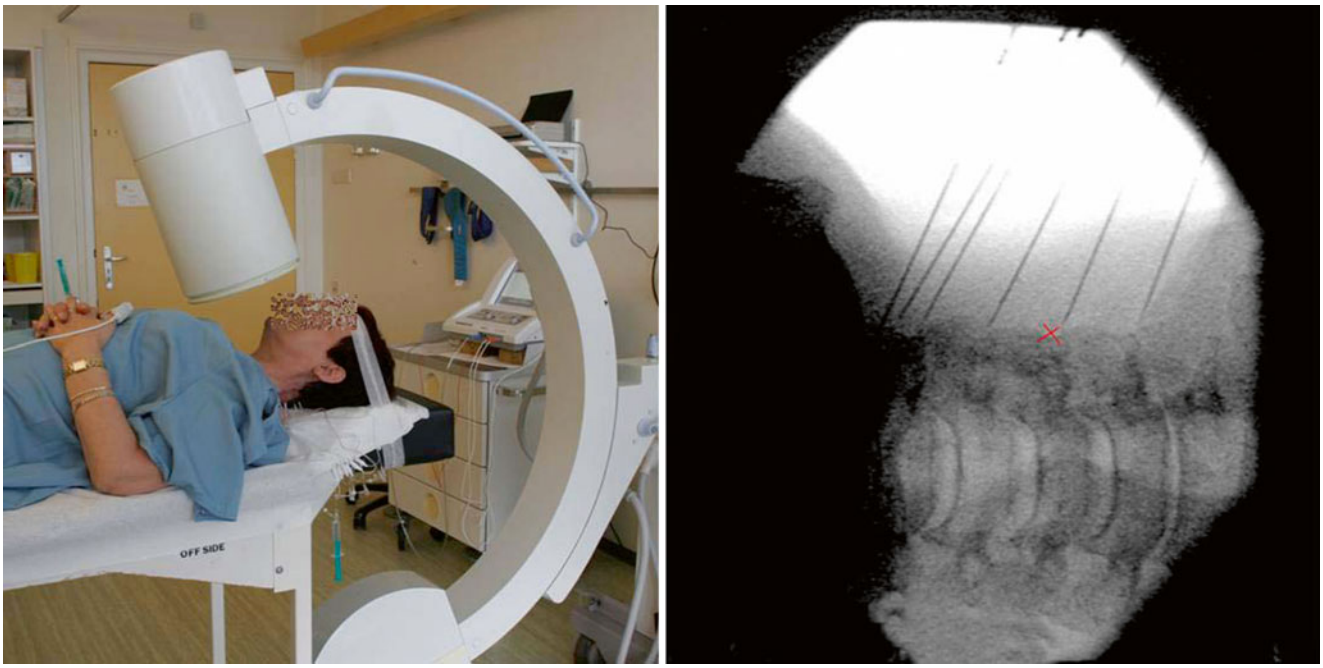


Fig. 19.5 AP view, needle tips in the "concavities," bone contact

Results

Systematic reviews of the literature relative to radiofrequency treatment following the Cochrane review guidelines [5, 6] conclude that there is limited evidence for the efficacy of radiofrequency neurotomy of the cervical zygapophyseal joint pain; a similar level of evidence is only obtained for the RF treatment of the cervical dorsal root ganglion.

In a prospective placebo-controlled study [3], the efficacy of cervical PFD has been proven in post-traumatic patients, but in nontraumatic patients the method is equally effective [7]. Inclusion criteria, the use of medial branch or intra-articular blocks with local anesthetics as selection for the procedure, are still subject of discussion. Some authors use single test blocks and others double blocks, whereas others perform no blocks at all as selection for the PFD. Their patient selection is solely based on clinical signs and symptoms.

Furthermore technical differences, such as exact positioning, size of the electrodes [8], used temperature, and exposure time, may be the cause of a different outcome. Finally the criteria for success are not equal in the published studies (VAS score, use of medication, inability scales, global perceived effect, or combinations of these).

There is discussion on the mode of action; the heat of the RF lesioning and thereby the denervation of the joint may not be the mode of action. By using the lateral approach with 23-G needles, it is not possible to fully denervate the joint and yet there is a good clinical outcome. So, only partial denervation is required, or it is not the exposure to heat but the electrical field caused by the RF current which is the explanation for the clinical effect. This explanation led to the development of pulsed RF [7]. There is a clinical study reporting good results from the pulsed RF treatment of the cervical medial branch [9]; however, no RCT is available yet.

Complications and Side Effects

The main complication is postoperative pain. Not only pain at the needle insertion point, lasting for a few days, but also serious burning pain may occur in 20 % of the patients. Patients should be informed that they can experience pain starting 7–9 days after the procedure that in some cases may last for a couple of weeks. This pain will always subside within 4–6 weeks. Bruising and hematomas in the neck occur in a low percentage and are rarely serious. Neurological damage is very rare if the positioning and stimulation have been performed carefully, taking the precautions described. During the procedure, prevention of neurological deficits happens at three precise points: (1) the lateral view (although not completely reliable), (2) electrostimulation, and (3) the injection of a small amount of local anesthetic (0.5 ml lidocaine) which is too small to anesthetize a spinal nerve when the electrode is accidentally placed too close. Sometimes the patient may be dizzy for a few hours or even a few days after the procedure due to the influence of the local anesthetics on the neck muscles. This will subside spontaneously within a few hours or days.

Instructions to Patients

Patients have to give their informed consent.

Patients using clopidogrel or ASA are asked to stop this treatment 5 days prior to the intervention. When patients use coumarin and its derivatives, the intake of these drugs should be interrupted and the INR controlled before the procedure. INR of 1.8 or less is appropriate.

It is recommended to let the patient eat a light meal before the procedure. Diabetic patients should not change their habits. Intake of food is free after the procedure. Because of possible temporary dizziness and/or weakness, it is recommended the patients stay in day care observation for at least one hour following the procedure. Sometimes discharge must be postponed if the patient is unable to walk properly. In all cases the patient should not participate in the traffic (e.g., drive a car) during the day of the intervention. He will only be allowed to leave the hospital when accompanied by a competent adult who can take care of transportation. The patient should be informed of the liability of driving a car on the day of the intervention.

Conclusions

A selected group of patients with disabling neck pain, headache, cervicobrachialgia, facial pain, and some forms of cluster headache and migraine, not relieved by conservative measures, attributed to the cervical facet joints can be successfully treated by facet joint denervation. Adequate patient selection and the use of an accurate lesioning technique under fluoroscopic control are essential elements for good long-term results. Pulsed RF treatment of the medial cervical branch may also be effective but there is still only little experience. (Pulsed) radiofrequency treatment of the cervical medial branch is a minimally invasive technique that is safe when performed by a trained anesthesiologist under fluoroscopic guidance. It is recommended to acquire sufficient experience for the PDF at the lumbar level. By sticking to the rules of the technique as described in this chapter, the RF treatment of the cervical medial branch of the dorsal ramus was demonstrated to be effective and safe.

References

1. Raj P, Lou L, Erdine S, Staats PS. Radiographic imaging for regional anesthesia and pain management. Philadelphia: Churchil Livingstone; 2003.
2. Shealy CN. Technique for percutaneous facet rhizotomy. Burlington: Radionics Corp; 1975.
3. Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N. Percutaneous radio-frequency neurotomy for chronic cervical zygapophyseal-joint pain. N Engl J Med. 1996;335(23):1721–6.
4. van Suijlekom JA, van Kleef M, Barendse G, Sluijter ME, Sjaastad O, Weber WEJ. Radiofrequency cervical zygapophyseal joint neurotomy for cervicogenic headache. A prospective study in 15 patients. Funct Neurol. 1998;13:297–303.
5. Geurts J, van Wijk RM, Stolker R, Groen GJ. Efficacy of radiofrequency procedures for the treatment of spinal pain: a systematic review of randomized clinical trials. Reg Anesth Pain Med. 2001;26:394–400.
6. Niemisto L, Kalso E, Malmivaara A, Seitsalo S, Hurri H. Radiofrequency denervation for neck and back pain: a systematic review within the framework of the cochrane collaboration back review group. Spine. 2003;28(16):1877–88.
7. Sluijter ME. Radiofrequency. Part 2. Meggen: Flivopress, SA; 2003.
8. van Kleef M, van Suijlekom JA. Treatment of chronic cervical pain, brachialgia, and cervicogenic headache by means of radiofrequency procedures. Pain Pract. 2002;2(3):214–23.
9. Mikeladze G, Espinal R, Finnegan R, Routon J, Martin D. Pulsed radiofrequency application in treatment of chronic zygapophyseal joint pain. Spine J. 2003;3(5):360–2.

Part IV
Shoulder

Chapter 20

Suprascapular Nerve Block

Chin-Wern Chan (✉)
Department of Pain Medicine, Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands WA 6009, Australia
e-mail: cchan89@hotmail.com

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_20,

Introduction

The suprascapular nerve (SSN) is the major sensory nerve supplying 70 % of the shoulder. It provides sensory innervation to the posterior and superior aspects of the shoulder joint [1]. Due to its consistent course through the suprascapular notch, the suprascapular nerve is an accessible target for neural blockade.

Blockade of the suprascapular nerve has been performed to assist in the diagnosis of suprascapular neuropathy [2], as well as in the management of acute [3, 4] and chronic shoulder pain [5].

There have been numerous variations and refinements in the technique of SSNB since its introduction [6]. Techniques based on anatomical landmarks and radiological guidance have been described in the literature. The more recent implementation of ultrasound guidance has attempted to improve the accuracy of SSNB while reducing complications [7–9]. In addition, use of lesioning techniques such as pulsed radiofrequency to provide sustained analgesia has also been described [10, 11].

Anatomy of the Suprascapular Nerve

The SSN is a large peripheral nerve possessing both motor and sensory fibers. It originates from the ventral rami of the fifth and sixth cervical nerve roots [12, 13]. There may also be a variable contribution from the fourth cervical nerve root [12, 14]. Following its formation, the nerve emerges from the lateral aspect of the upper trunk of the brachial plexus. It then travels through the posterior triangle of the neck, courses deep to the trapezius and omohyoid muscles, and enters the supraspinous fossa via the suprascapular notch underneath the superior transverse scapular ligament (STSL) (Fig. 20.1a). In the majority of cases, the suprascapular artery runs above (78.3 %) the STSL [15].

Shortly after passing through the suprascapular notch, the SSN emits two branches: one is the motor nerve for the supraspinatus muscle [16–18] and the other is known as the superior articular branch. The latter nerve is sensory and supplies the coracoclavicular, coracohumeral ligaments, the acromioclavicular joint, the glenohumeral joint (posterior and superior aspects), and the subacromial bursa [12, 19, 20]. The main trunk then exits the suprascapular fossa by curving around the lateral border of the scapula spine through a fibro-osseous tunnel formed by the spinoglenoid ligament and the spine of the scapula and terminates in motor branches to the infraspinatus muscle [18, 20] (Fig. 20.2).

The anatomy of the suprascapular notch is important for several reasons. The nerve is susceptible to injury and impingement at the level of the notch as it passes beneath the STSL [21, 22]. This site represents an attractive region for SSN blockade as the nerve has not divided yet. The variable shape of the notch has been described and has been categorized into different types [23, 24] (Fig. 20.3). In the adult, the most common type is a U-shaped or semicircular notch (Type 1 and 2 in Fig. 20.3) [23]. The notch is absent or converted into a foramen by the ossified STSL in 15 % of the specimens [24].

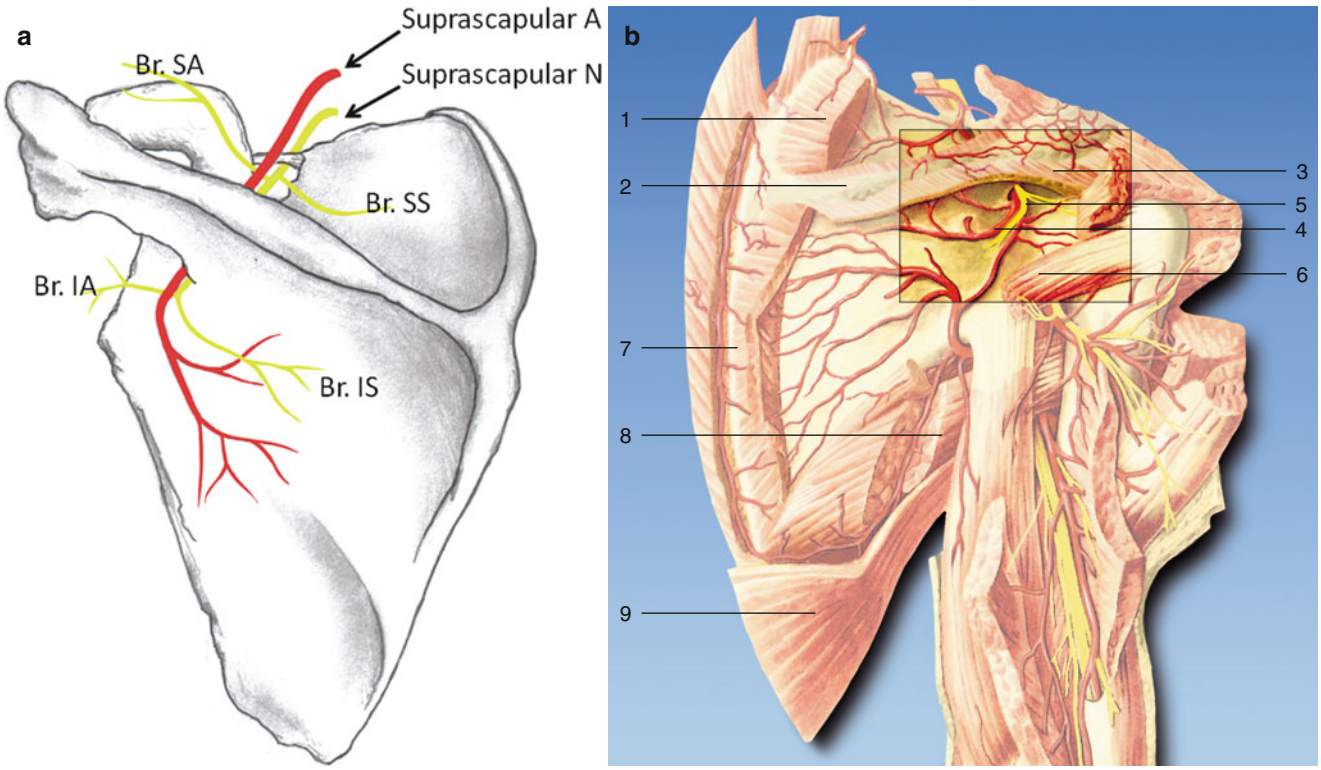


Fig. 20.1 (**a**) Suprascapular nerve and its branches of the left shoulder. Superior articular branch (*Br.SA*) supplies the coracohumeral ligament, subacromial bursa, and posterior aspect of the acromioclavicular joint capsule. Inferior articular branch (*Br.IA*) supplies the posterior joint capsule. Ac indicates acromion; *Br.IS* branch to the infraspinatus muscle, *Br.SS* branch to the supraspinatus muscle, *CP* coracoid process, *SS* scapula, spine, *TSL* transverse scapula ligament (Reproduced with permission from Philip Peng Educational Series). (**b**) Posterior view of the shoulder. (*1*) Supraspinatus muscle, (*2*) spine of the scapula, (*3*) deltoid muscle, (*4*) suprascapular artery, (*5*) suprascapular nerve, (*6*) teres minor muscle, (*7*) infraspinatus muscle, (*8*) teres major muscle, (*9*) latissimus dorsi muscle (with permission from Danilo Jankovic)

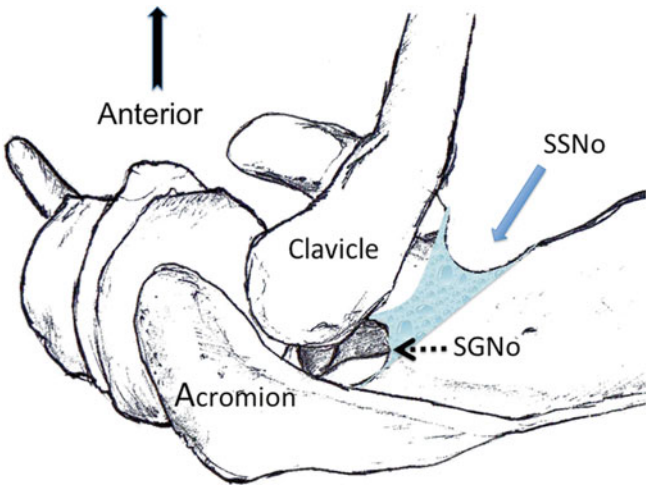


Fig. 20.2 Superior view of the left shoulder. The course of the suprascapular nerve (*shaded*) enters the suprascapular fossa through the suprascapular notch (*SSNo*) and then enters the infrascapular fossa through the spinoglenoid notch (*SGNo*). The clavicle (Cl) and coracoid process (Co P) are also displayed in the diagram (Reproduced with permission from Philip Peng Education Series)

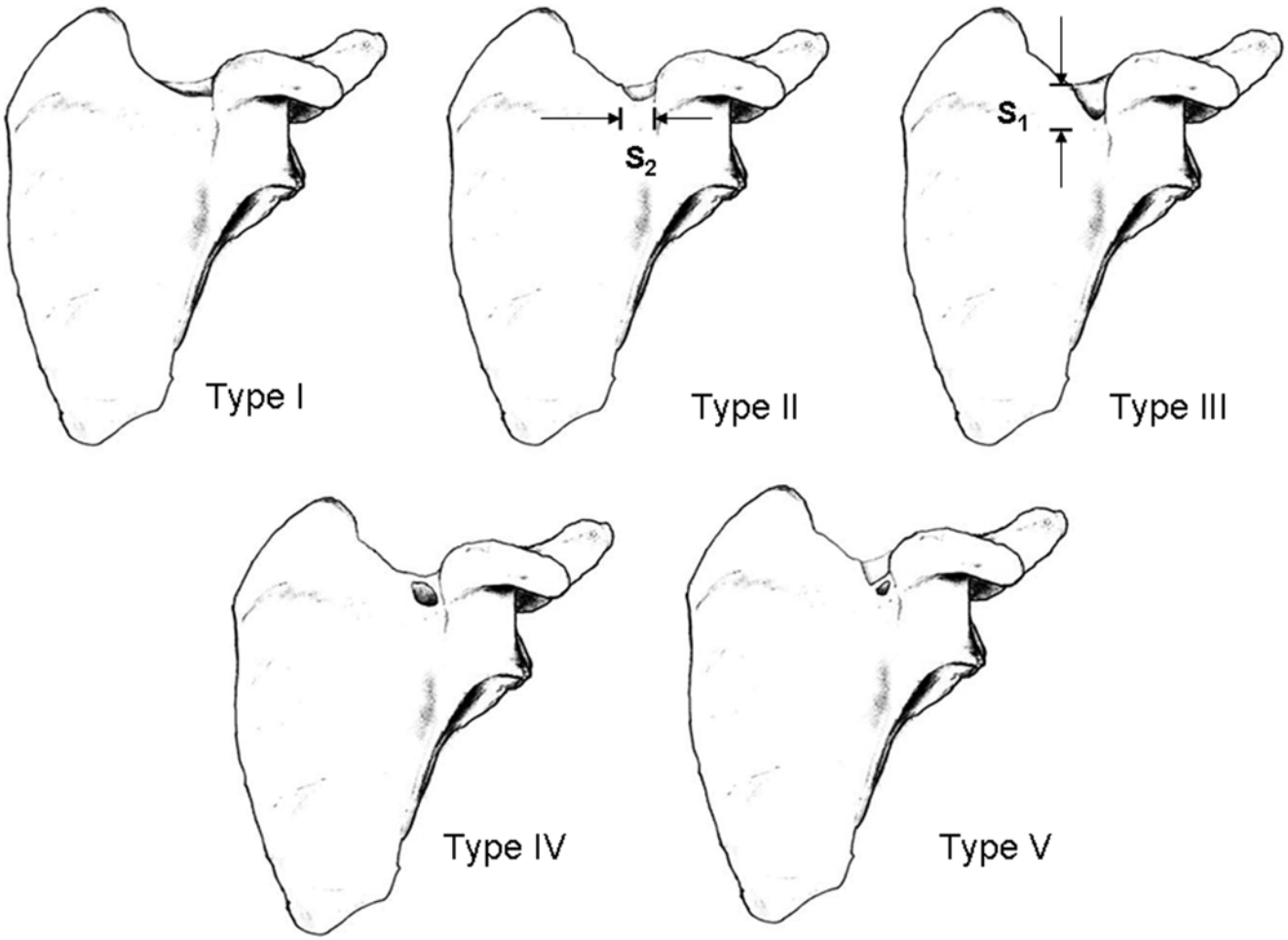


Fig. 20.3 Variation of morphology of the suprascapular notch. *Type I* indicates no notch (8.3 %); *type II*, notch with greater transverse diameter, S2 (41.85 %); *type III*, notch with greater vertical diameter, S1 (41.85 %); *type IV*, bony foramen (7.3 %); *type V*, notch with bony foramen (0.7 %) (Adapted from Natsis et al. [24]; Reproduced with permission from Philip Peng Education Series)

Technique of Localizing the Suprascapular Nerve

Traditionally, suprascapular nerve blockade has been performed via use of anatomical landmarks (blind techniques) [6]. More recently, the use of imaging guidance to more accurately guide needle placement has been described [6].

Blind Techniques

Various landmark approaches have been described and can be grouped into posterior, superior, and lateral approaches. The posterior approach attempts to block the SSN at the level of the suprascapular notch [25–29], while the superior approach aims to block the SSN by surrounding the nerve with local anesthetic on the floor of the supraspinous fossa [30, 31]. A lateral approach to localize the suprascapular nerve has also been described [32, 33]. To improve accuracy, the SSN has been localized using nerve stimulation [34, 35], paresthesia [36], and EMG [37]. The popularity of these techniques has reduced with the introduction of radiological guidance which will be discussed in the next section.

Posterior Approach

The posterior approach is generally performed while the patient sits on the operating table with the ipsilateral arm lying by his or her side [25, 27, 38]. To minimize the risk of pneumothorax, the scapula can be elevated from the posterior chest wall by repositioning the ipsilateral hand to the opposite shoulder, thereby increasing the potential distance the needle must travel from the skin to chest wall [36]. A 22-gauge, 3-in. needle is generally used.

The superficial landmarks described in the posterior approach techniques serve to guide the needle to slide into the notch. As discussed in the anatomy section (above), the notch is not a defined structure in 15 % of the population. Furthermore, the potential complication of this approach is pneumothorax as the trajectory of the needle is toward the thoracic cavity.

Superior Approach

The superior approach [30, 31] was initially described to permit SSNB performed in patients in the supine position but the sitting position is the preferred position in clinical practice.

The needle entry point for the superior approach has been described as cephalad and lateral to the midpoint of the spine of the scapula [30]. In general, the needle is directed to the lateral half of the floor of the suprascapular fossa because the supraspinatus muscle is attached to the medial half. A 1.5-in. needle is usually sufficient to reach the floor of the scapular fossa. Potential advantages of this approach include ease of access, no reference to the notch, and extremely low risk of pneumothorax [30, 31].

Lateral Approach

In this approach, the needle entry point is at a landmark known as the "Neviaser portal" [32, 33, 36]. The site can be found by palpating the "soft spot" 1 cm medial to the junction of the clavicle and the scapular spine (Fig. 20.4). The needle is inserted and angled toward the coracoid. When bone contact is made (scapula), the needle is then directed anteriorly until no bone is felt, then retracted marginally, and moved posteriorly until bone again is felt [32]. This positions the needle tip at the coracoid base of the supraspinous fossa [32]. As this places the needle tip in the supraspinous fossa, an adequate block is achieved by flooding the plane with local anesthetic. Initial reports advised using 25 mL of local anesthetic solution with this lateral approach [32]. Subsequently, a modified lateral SSNB approach was published which demonstrated sufficient spread of solution throughout the supraspinous fossa with a volume of 5 mL (see below) [33].

Comparison of Blind Approaches

Despite the many number of approaches and techniques published to date, few studies have actually compared them. An old study on pulsed radiofrequency lesioning of the SSN [39] compared 4 commonly used blind techniques [25, 26, 29, 40], in terms of the final position of the needle tip relative to the suprascapular notch with radiographical correlation. They found that the needle tip was usually a significant distance from the notch such that a heat lesion would not affect the SSN in all techniques. When comparing the blind methods, they found that the approach suggested by Granirer [29] offered the best approximation of "needle tip to notch" [39].

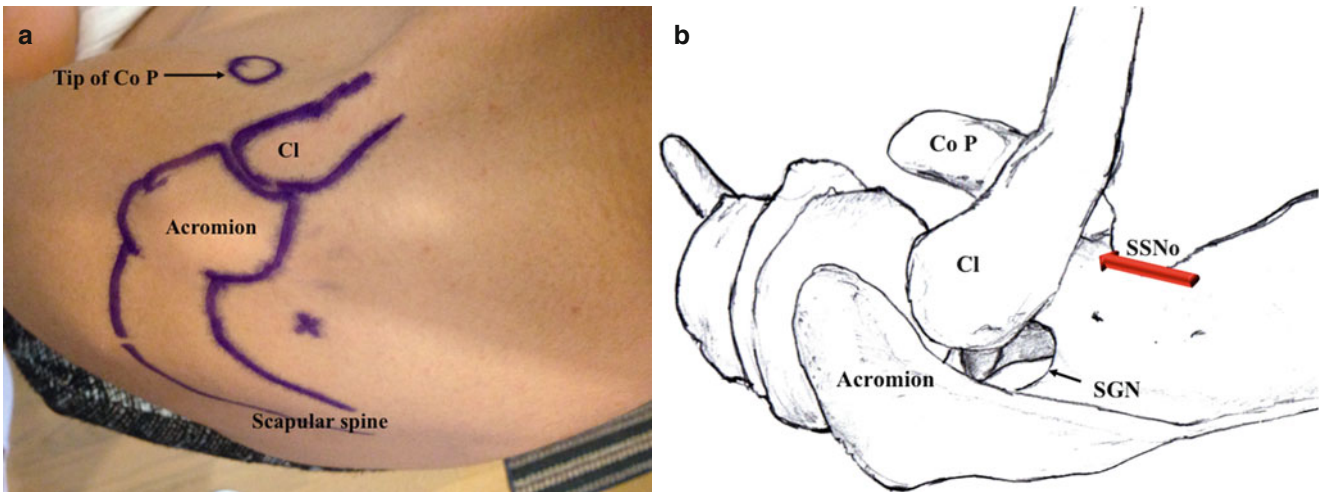


Fig. 20.4 (**a**) Superficial anatomy demonstrating landmarks for the lateral approach to SSNB. The x marks the site known as Neviaser portal. The o represents the coracoid process. (**b**) Final location of the needle at the base of the coracoid in the supraspinatus fossa where the SSN curves around the coracoid and heads to the glenohumeral joint (*red arrow*). Injection of high-volume local anesthetic floods the area where the SSN lies (Reproduced with permission from Philip Peng Education Series)

Image-Guided Techniques for SSNB

Techniques using imaging guidance such as fluoroscopy [41], computed tomography (CT) [34], and more recently ultrasound [7, 8, 35, 37, 42] have been described.

Conventional Imaging

Fluoroscopy and CT have been described to locate the suprascapular notch [34, 41]. For the fluoroscopic technique, the patient is placed in the prone position. A C-arm is then used to identify the notch (Fig. 20.5) [41]. The suprascapular notch will be seen superior to the spine of the scapula, medial to the coracoid process, and lateral to the rib margins (Fig. 20.5) [41]. To obtain an optimal image, the C-arm will often need to be obliquely angled away from the side of the proposed block and in the cephalocaudad orientation [41]. Visualization of the suprascapular notch is further improved when the ipsilateral arm is placed above the shoulder ("arm up" position) compared to when the affected arm is placed by the side ("arm down position") (91 % vs. 47 %; $p<0.0001$) [43].

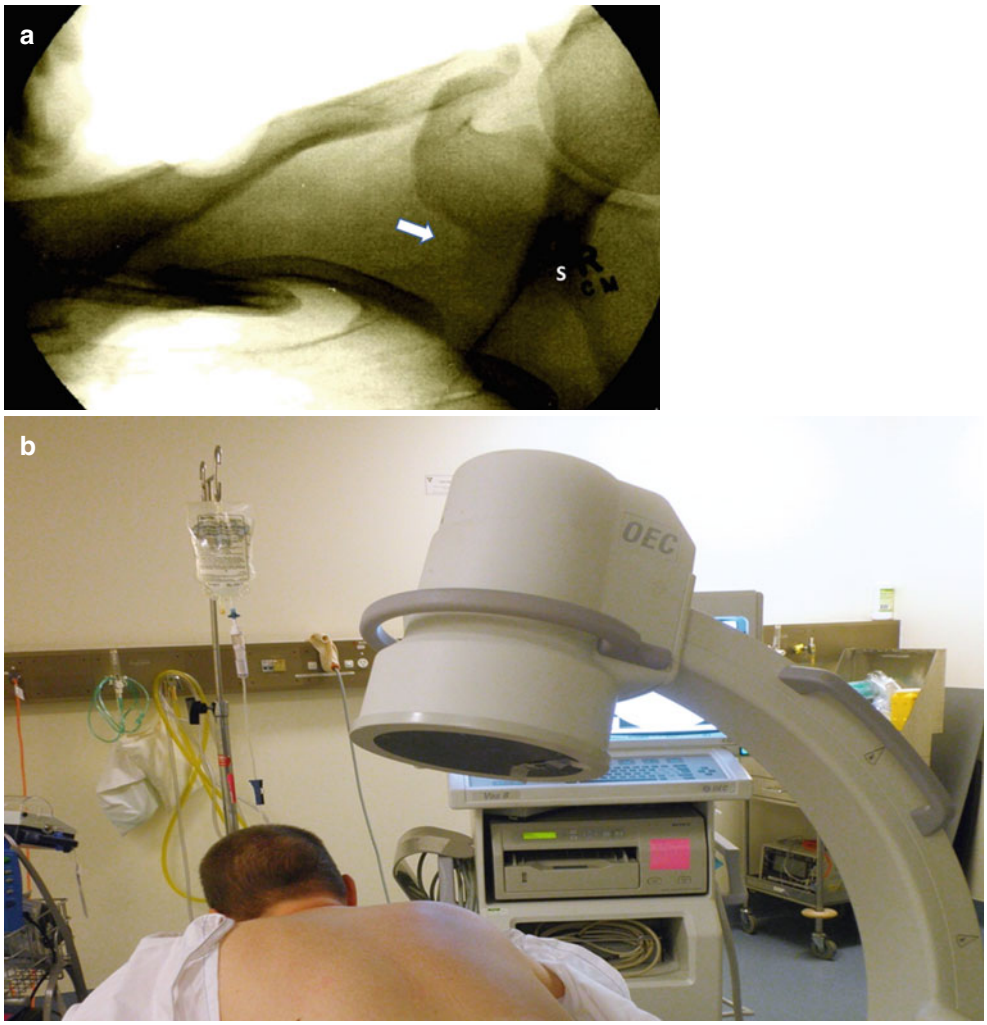


Fig. 20.5 (**a**) Radiograph of the right suprascapular notch. *S* indicates spine of scapula. *White arrow* points to the suprascapular notch. (**b**) C-arm positioning for imaging the suprascapular notch. The patient is placed in prone position. The C-arm is positioned over the shoulder. To image the suprascapular notch, the C-arm is rotated oblique to the treated side and angled cephalocaudal (Reproduced with permission from Philip Peng Education Series)

Ultrasound-Guided Suprascapular Nerve Blockade

Ultrasound (US) guidance is the most recent imaging technique that has been employed to assist SSNB. Several articles have been published describing this technique [7, 8, 35, 37, 42]. Ultrasound guidance is a relatively new technique, and various methods utilizing sonography have been published [7, 8, 35, 37, 42]. Both anterior and posterior approaches have been described. The anterior approach targets the suprascapular nerve shortly after it branches from the brachial plexus (in the supraclavicular area) underneath the omohyoid muscle [44]. The major limitation of this approach is the proximity to the brachial plexus (median 8 mm; range 4–15 mm). For the purpose of this chapter, the more widely practiced technique of US-guided SSNB will be described.

The ideal site to perform SSNB with ultrasound is at the floor of the suprascapular fossa, between the suprascapular notch and spinoglenoid notch [8] (Fig. 20.2). At this site, the SSN runs along the floor of the suprascapular fossa covered by the fascia of supraspinatus in a natural compartment, which will contain the spread of the local anesthetic or injectate [8]. Applying an ultrasound-guided injection technique approximated the needle tip to the nerve and has been shown to achieve a complete block with a reduced volume of local anesthetic [45]. A small volume (5 mL) of injectate will result in adequate flooding of the nerve [46] with minimal spread to the brachial plexus [33]. Furthermore, this target is independent of the suprascapular notch, which can be absent in some individuals. The risk of pneumothorax is substantially reduced because of the direction of the needle [47].

The ultrasound-guided SSNB may be performed with the patient either in the sitting or prone position. A high-frequency (7–13 Hz) linear ultrasound probe is used. The probe is placed in a coronal plane over the suprascapular fossa (Fig. 20.6). The probe should be placed such that the midpoint of the probe divides a line between the coracoid process and posterior aspect of the acromion (Fig. 20.6). A slight anterior tilt is placed on the probe to improve visualization of the target structures (Fig. 20.6). In this view, the identifiable structures (from superficial to deep) are the trapezius muscle, the supraspinatus muscle, and the suprascapular fossa floor (Fig. 20.6). The suprascapular nerve and artery are commonly seen on the floor running underneath the supraspinatus muscle layer (Fig. 20.6).

SSNB is performed by passing a 22-gauge, 3-in. needle in an in-plane approach from medial to lateral.

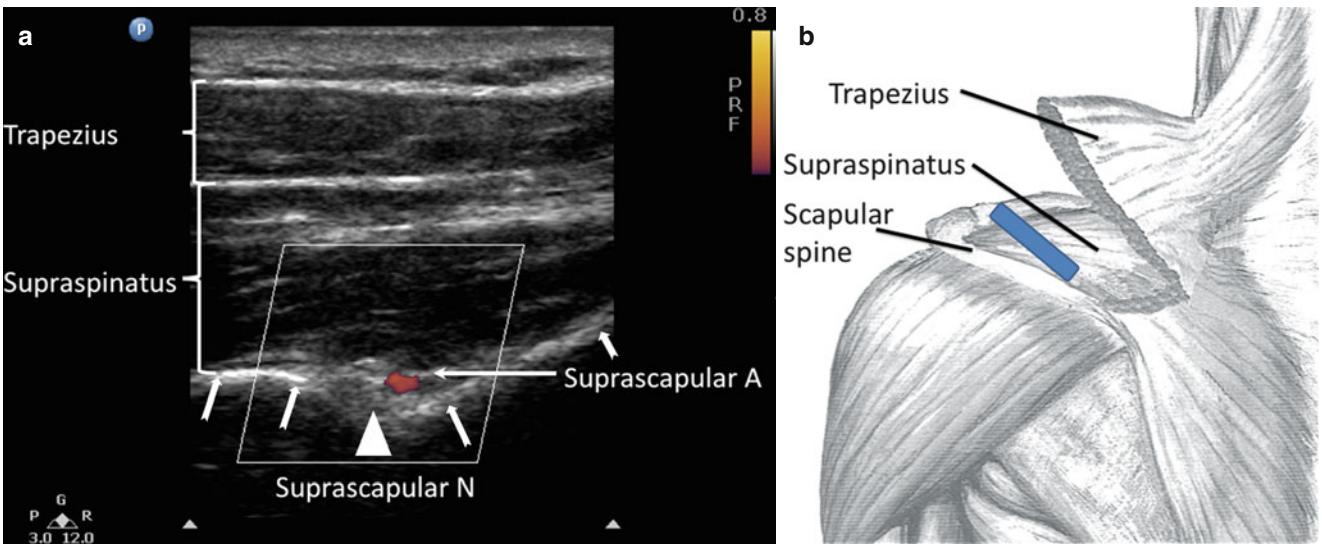


Fig. 20.6 (**a**) Ultrasonographic image of the suprascapular nerve on the floor of the scapular spine between suprascapular notch and spinoglenoid notch. Both suprascapular nerve and artery run underneath the fascia of supraspinatus muscle. Suprascapular A and N indicate suprascapular artery and nerve. *Bold arrows* outline the floor of the scapula fossa. (**b**) Approximate position of the ultrasound probe (*dark rectangle*). The patient can be in sitting or in prone position. Ultrasound scanning is performed with a linear ultrasound probe (7–13 MHz) placed in a coronal plane over the suprascapular fossa with a slight anterior tilt. The probe is placed in an orientation such that it is in the short axis to the line joining coracoid process and acromion (reflecting the position of the spinoglenoid notch). The trapezius muscle was removed to show the underlying supraspinatus muscle (Reproduced with permission from Philip Peng Education Series)

Imaging to Improve Needle Localization of the Suprascapular Nerve

One randomized single-blind trial compared the blind approach to SSNB with a CT-guided approach [48]. This study did not find any significant difference between the blind or CT-guided SSNB in terms of pain and disability scores [48]. Both groups showed significant improvement post SSNB [48]. There were no significant adverse effects in either group, and patient satisfaction scores were high [48].

The efficacy of ultrasound-guided SSNB was compared to the landmark-based technique [9]. In this study, patients with chronic nonspecific shoulder pain were randomized with 25 patients in each group. The investigators found that initially both groups improved in terms of pain relief. However, the analgesic effect was better sustained at 1 month in the ultrasound-guided group compared to the control group [9]. Furthermore, while there were no complications in the ultrasound group, the control group recorded 2 cases of arterial puncture and 3 cases of direct nerve injury with neurological deficit [9].

In summary, various approaches have been described for the blockade of SSN. Disadvantages of the approach using the notch as a landmark are the potential absence of the notch in some individuals and the potential risk of pneumothorax. The superior approach may negate these disadvantages. On limited evidence, these studies would suggest ultrasound is useful in approximating the block needle in the vicinity of the SSN and thereby increasing efficacy and reducing complications of SSNB. The majority of recent studies utilizing US-guided SSNB for acute and chronic shoulder pain management may reflect this.

Substances Used for Blockade of the Suprascapular Nerve

When the needle is placed in proximity to the SSN, several methods of nerve blockade have been published. The commonly used methods include local anesthetic, steroids, pulsed radiofrequency, and chemical neurolysis. These may be used alone or in combination. Bupivacaine (0.25 or 0.5 %) with epinephrine (1:200,000) is the popular local anesthetic agent [30, 31, 49–53]. Injectate volume is highly variable in the literature. However, some authors argue 5 mL is the optimal volume based on morphological evidence [46].

For treatment of chronic shoulder pain, injectable steroid (methylprednisolone) usually is added to the local anesthetic solution [5, 9, 54–56]. However, the value of this practice has been questioned by a double-blind study [52] demonstrating that the addition of methylprednisolone fails to confer any benefit.

SSNB achieved with radiofrequency or cryolesion provides a long-lasting effect that can endure for up to 18 months [11, 39, 57–59]. Furthermore one of these studies demonstrated a significant reduction in pain, improvement in function, and a reduction in analgesic medication (81 % of study patients) following pulsed RF of the SSN [11].

The use of chemical neurolysis for SSNB has mainly been in the form of case reports [39, 60]. Injection of phenol causes protein coagulation and necrosis when applied directly to the nerve, thereby alleviating pain. A larger study involved 16 patients with shoulder pain secondary to rheumatoid arthritis. These patients received SSNB with prilocaine (4 mL) and 6 % aqueous phenol (4 mL) with significant reduction in pain and improved shoulder range of motion at 13-week follow-up [61].

Complications

SSNB is a safe procedure with a generally low rate of complications (Table 20.1).

The largest study to date retrospectively analyzed 1,005 SSNBs performed by multiple clinicians in multiple centers over a 6-year period [62]. There were no major complications reported [62]. There were only 6 minor adverse events which included transient dizziness ($n = 3$), transient arm weakness ($n = 2$), and facial flushing ($n = 1$) [62].

The possible complications are pneumothorax (<1 %), intravascular injection, residual motor weakness, local trauma, and vasovagal response of the patient.

Table 20.1 Complications of suprascapular nerve block and rhizotomy

Study authors	Type of study	No. of patients	Clinical indication for SSNB or SSN rhizotomy	Complications reported (numbers)
Shanahan et al. (2012) [62]	O (multicenter study 2003–2009)	1,005	Chronic shoulder pain	6 side effects: Transient dizziness (3) Transient arm weakness (2) Facial flushing (1)
[a]Eyigor et al. (2010) [57]	R	50	Chronic shoulder pain	No complications reported
Gorthi et al. (2010) [9]	R, C	50	Chronic shoulder pain	US group—no complications; blind technique, arterial puncture (2), direct nerve injury (1)
Martinez-Barenys et al. (2010) [63]	R	74	Ipsilateral shoulder pain post-thoracotomy	No complications reported
Saha et al. (2010) [64]	O	178	Ipsilateral shoulder pain post-thoracotomy	No complications reported
Mitra et al. (2009) [65]	O	28	Adhesive capsulitis	No complications reported
[a]Liliang et al. (2009) [11]	O	11	Chronic shoulder pain	Puncture wound pain for 1 week (1)
[a]Kane et al. (2008) [10]	O	12	Painful cuff tear arthropathy in patients unfit for surgery	No complications reported
Checucci et al. (2008) [4]	O	20	Patients undergoing arthroscopic procedures for rotator cuff disease	No complications reported
Jerosch et al. (2008) [66]	R	260	Arthroscopic and shoulder surgery	No complications reported
Price (2007) [67]	O	40	Arthroscopic and open shoulder surgery	No complications reported from SSNB
Di Lorenzo et al. (2006) [47]	R	40	Rotator cuff tendinitis	No major complications
Taskaynata et al. (2005) [56]	R	60	Chronic shoulder pain	No complications reported
Singelyn et al. (2004) [68]	R, C	120	Arthroscopic shoulder surgery	No complications reported
Shanahan et al. (2004) [48]	R, SB	67	Degenerative joint or rotator cuff disease	No complications reported
Schneider-Kolsky et al. (2004) [34]	O	40	Chronic shoulder pain	No complications reported
Neal et al. (2003) [69]	R, DB, C	50	Acromioplasty, rotator cuff repair, or combination of both	No complications reported
Shanahan et al. (2003) [5]	R, DB, C	83	Shoulder pain from rheumatoid arthritis and/or degenerative disease of the shoulder	Minor bruising (1)
Tan et al. (2002) [70]	R, DB, C	44	Ipsilateral shoulder pain post-thoracotomy	No complications reported
Karatas and Meray (2002) [71]	R, SB	41	Adhesive capsulitis	No complications reported
Dahan et al. (2000) [72]	R, DB, C	34	Frozen shoulder	No major complications reported
Jones and Chattopadhyay (1999) [73]	R	30	Frozen shoulder	No major complications reported
Lewis (1999) [61]	O	16	Rheumatoid or osteoarthritis of shoulder	No complications reported
Ritchie et al. (1997) [3]	R, DB, C	50	Arthroscopic shoulder surgery	No complications
Dangoisse et al. (1994) [31]	O	12	Frozen shoulder (6 patients), others (6 patients)	Sensation of heaviness in the arm (1), numbness and aching shoulder (1)
Gado and Emery (1993) [52]	R, DB	26	Rheumatoid arthritis	No complications reported
Vecchio et al. (1993) [74]	R, C	28	Rotator cuff tendinitis	Mild aching in the injection area (16)
Wassef, (1992) [49]	O	9	Frozen shoulder	No complications reported
Emery et al. (1989) [51]	R, DB, C	17	Rheumatoid arthritis	No complications reported
[a]Brown et al. (1988) [39]	O	22	Rheumatoid arthritis	Impaired abduction (1)

C placebo-controlled, *DB* double-blind, *O* observational, *R* randomized, *No*. number, *SSNB* suprascapular nerve (SSN) block, *US* ultrasound
[a]Rhizotomy study

Suprascapular Nerve Blockade in Clinical Practice

Acute Pain

The studies investigating the efficacy of SSNB in acute pain states are summarized in Table 20.2.

SSNB has been used successfully for the control of postoperative pain following open and arthroscopic shoulder surgery (Table 20.2) [3, 32, 53, 66, 68]. Although shoulder arthroscopy recently has become popular as an outpatient procedure, it remains one of the most painful of the same-day surgical procedures [76]. At present, interscalene brachial plexus block (IBPB) is the usual regional technique employed for analgesia during and after shoulder surgery [77–79]. Blockade of the brachial plexus provides more complete analgesia of the shoulder joint. SSNB also allows good control of severe postoperative pain at rest and with movement in the early postoperative period, resulting in a significant reduction in analgesic dose and demand, discharge time, and the incidence of nausea [3].

Table 20.2 Suprascapular nerve block for acute pain control

Study authors	Type of acute pain	Study design	Number of participants	Results	Conclusions
SSNB as the only form of regional anesthesia					
Jerosch et al. (2008) [66]	Arthroscopic shoulder surgery: mixed	Prospective, non-randomized study. Comparison of 2 consecutive cohorts	260 patients Received SSNB = 130 No nerve block = 130	No difference in baseline VAS scores. Postoperatively, significant reduction in VAS scores at 24, 48, and 72 h in the SSNB group. No complications of SSNB	SSNB is effective in reducing postoperative shoulder pain in arthroscopic shoulder surgery. SSNB is associated with minimal complications
Singelyn et al. (2004) [68]	Arthroscopic shoulder acromioplasty	Prospective, randomized, blind study	120 patients randomized to 4 treatment groups: SSNB = 30 IALA = 30 ISBPB = 30 Control (no regional analgesia) = 30	No significant difference in pain scores between control and IALA groups. SSNB and ISBPB reported significantly less pain than the other 2 groups. The ISBPB group had significantly less pain on movement than the SSNB group. Only the ISBPB group recorded significantly less morphine consumption and higher satisfaction	ISBPB is the most efficient regional technique for arthroscopic shoulder acromioplasty. SSNB improves analgesia for arthroscopic acromioplasty but is less efficient than ISBPB. When ISBPB is contraindicated, SSNB is a clinically appropriate alternative
Ritchie et al. (1997) [3]	Arthroscopic shoulder surgery	Randomized, double-blind, placebo-controlled study	50 patients randomized to: Placebo = 25 SSNB = 25	VAS significantly lower in SSNB group at 120, 180 min. VPS score significantly lower in SSNB group at 120, 180, and 240 min. Significantly reduced morphine consumption (SSNB group) on the day of surgery. Significantly less nausea and vomiting in SSNB group. Reduced stay in ambulatory surgical unit	SSNB is an effective regional anesthetic technique for arthroscopic shoulder surgery in terms of improved analgesia, reduced opioid requirements, and less nausea and vomiting
Martinez-Barenys et al. (2010) [63]	Ipsilateral post-thoracotomy shoulder pain	Randomized, single-blind study	74 patients 1st group: phrenic group (PNI) received 10 mL 2 % lidocaine into perinephric fat pad prior to closure = 37 2nd group: SSNB with 10 mL 0.5 % bupivacaine at completion of surgery = 37	Shoulder pain intensity was significantly lower in the PNI group compared with the SSNB group	Shoulder pain post-thoracotomy does not appear to arise from the shoulder joint. This study suggests that pain arises from diaphragmatic irritation. Therefore, routine preemptive blockade of the suprascapular nerve is not recommended

(continued)

Table 20.2 (continued)

Study authors	Type of acute pain	Study design	Number of participants	Results	Conclusions
Saha et al. (2010) [64]	Ipsilateral post-thoracotomy shoulder pain	Retrospective case review of post-thoracotomy patients	178 patients post-thoracotomy. New-onset shoulder pain post-thoracotomy = 92 (51 %). 34 patients (27 %) with localizing signs suggestive of musculoskeletal origin underwent SSNB	29 of 34 patients reported satisfactory pain relief following SSNB	In patients with post-thoracotomy shoulder pain and who have localizing signs suggestive of musculoskeletal origin, SSNB is an effective treatment. However, SSNB is not the treatment per se for post-thoracotomy shoulder pain as the musculoskeletal system is responsible for less than one third of cases
Tan et al. (2002) [70]	Ipsilateral post-thoracotomy shoulder pain	Double-blind, randomized, placebo-controlled study	44 patients who had undergone thoracotomy under general anesthesia and mid-thoracic epidural. 30 patients experienced shoulder pain within 2 h postsurgery and were randomized to: SSNB with 10 mL 0.5 % bupivacaine = 15 Control: SSNB with 10 mL 0.9 % saline = 15	No significant decrease in VAS or VRS in patients receiving SSNB with bupivacaine	SSNB not effective for ipsilateral shoulder pain post-thoracotomy
SSNB in combination with another regional technique					
Lee et al. (2014) [75]	Ambulatory arthroscopic shoulder surgery	Prospective randomized controlled trial	42 patients 21 patients received US-guided SSNB and axillary nerve block 21 patients received US-guided SSNB alone	Addition of US-guided axillary nerve block to SSNB resulted in reduced pain scores (VAS) in the first 24 h postsurgery, compared to SSNB alone	Axillary nerve block improves analgesic efficacy of SSNB for ambulatory arthroscopic shoulder surgery
Checucci et al. (2008) [4]	Arthroscopic shoulder surgery	Case series	20 consecutive patients: each patient received a SSNB and an axillary nerve block as the sole anesthetic for the operation, with midazolam sedation	All patients were able to have surgery under the combination block. No patients required opioids, analgesics, or general anesthesia. Postoperative pain control was effective with negligible use of non-opiate analgesics. No opiate analgesic was required postoperatively	SSNB in combination with axillary nerve block is sufficient for arthroscopic shoulder surgery
Price (2007) [67]	Shoulder surgery: arthroscopic and open. Postoperative analgesia in patients who had ISBPB failure	Retrospective case series	40 patients with ISBPB failure received combined SSNB and axillary nerve block	57 % of cases required no morphine in PACU. 83 % of cases required no morphine overnight. Complications: radial nerve blockade which resolved (2/70 cases)	If ISBPB fails, combined SSNB and axillary nerve block is effective in providing postoperative analgesia for shoulder surgery
Neal et al. (2003) [69]	Ambulatory nonarthroscopic shoulder surgery	Prospective randomized study	50 patients SSNB and ISBPB: general anesthesia = 25 Sham injection and ISBPB: general anesthesia = 25	Addition of SSNB significantly delayed the time to first significant report of pain. However, addition of SSNB did not improve PACU measures, 24-h assessment of pain, supplemental analgesic use, or QOL measures	SSNB combined with ISBPB does not significantly improve outcomes in ambulatory nonarthroscopic shoulder surgery

IALA intra-articular local anesthetic, *ISBPB* interscalene brachial plexus block, *PACU* postanesthesia care unit, *PNI* phrenic nerve infiltration, *QOL* quality of life, *SSNB* suprascapular nerve block, *VAS* visual analog scale, *VPS* verbal pain scale, *VRS* verbal rating scale

Comparing SSNB with IBPB, however, Singelyn et al. found that IBPB provided superior analgesia in terms of pain scores at the 4-h follow-up and morphine consumption in patients receiving arthroscopic acromioplasty [68].

The superior analgesia with ISB compared to SSNB is no surprise, as at best SSNB can only anesthetize 70 % of the shoulder joint. The remaining sensory innervation is provided by the nerve to the subscapularis, axillary nerve, and lateral pectoral nerve [1]. To improve the success rate of SSNB, a combined technique with axillary nerve block has been used to provide increased coverage of the shoulder joint for arthroscopic rotator cuff procedures [4]. In a case series, this combined technique enabled all the patients to undergo the operation with sedation only without resorting to general anesthesia. A subsequent randomized controlled trial compared the effects of the combined US-guided axillary nerve and SSN blocks with SSNB alone for arthroscopic rotator cuff repairs [75]. This study found that the combination block (axillary and SSN block) resulted in superior analgesia in the immediate postoperative period [75].

The studies discussed above are limited to arthroscopic shoulder surgery. For nonarthroscopic/open shoulder surgery, the role of SSNB is limited [69]. Neal et al. conducted a randomized clinical trial comparing standard ISB with ISB plus SSNB for nonarthroscopic shoulder surgery [69]. They found that as an adjunct, SSNB provided more prolonged analgesia compared to ISB alone but did not affect other outcome measures such as supplemental analgesic use or quality of life outcomes [69]. They concluded that SSNB is less useful for nonarthroscopic shoulder surgery because these operations are usually anterior procedures which are outside the region of SSN sensory innervation compared to the posterior port stimulation of arthroscopic surgery [69].

While the placement of peripheral nerve catheters has been popular for interscalene brachial plexus block and femoral nerve block, this has been less so for the suprascapular nerve. Elsharkawy et al. described 2 cases in which SSN catheters placed via ultrasound guidance provided effective management of acute post shoulder arthroscopic surgery pain [80]. Further studies are required to further describe the insertion technique and efficacy of SSN catheters.

In summary, for pain associated with shoulder surgery, ISB is the most effective regional technique for analgesia and ambulatory outcome measures. SSNB will provide improved analgesia compared to a general anesthetic technique alone for arthroscopy but is inferior to ISB. SSNB combined with an axillary nerve block provides excellent operative and postoperative analgesia. For nonarthroscopic shoulder surgery, the role of SSNB as an adjunct to ISB is limited.

In addition to the management of acute pain associated with shoulder surgery, several studies have assessed SSNB for control of shoulder pain post-thoracotomy [63, 64, 70]. These studies provided conflicting results. Furthermore, the most recent RCT suggested that shoulder pain post-thoracotomy is not musculoskeletal in origin but referred pain from diaphragmatic irritation [63]. The difference in results may be due to the selection of patients. The study demonstrating that SSNB was beneficial screened patients post-thoracotomy and only those with shoulder pain and localizing signs suggestive of musculoskeletal pain improved with SSNB [64]. If selected in this manner then this study found that 85.3 % of selected patients obtained satisfactory pain relief post SSNB [64].

In summary, one may conclude SSNB is not effective in all patients who develop ipsilateral shoulder pain post-thoracotomy. However, in those patients who have localizing signs suggesting the shoulder pain is musculoskeletal in origin, SSNB is an appropriate intervention to relieve pain. This could be further investigated by a randomized clinical trial.

Chronic Pain

For patients with chronic pain, SSNB may be both a diagnostic and more commonly a therapeutic procedure. To achieve more prolonged analgesia for chronic pain, local anesthetic is combined with steroid, phenol, or pulsed radiofrequency when SSNB is performed (Table 20.3).

Diagnostic (Chronic Pain)

Suprascapular neuropathy is believed to be the cause in 1–2 % of patients with shoulder pain [84]. The possibility of suprascapular neuropathy is suggested by posterior shoulder pain, a history of trauma or traction to the suprascapular nerve, and weakness and atrophy of the muscles (supraspinatus, infraspinatus) supplied by the suprascapular nerve [85–88]. Neuropathy of the SSN can be caused by traction or compression of the nerve at the spinoglenoid region or the suprascapular notch. The causes of traction or compression include trauma, repetitive use, and space-occupying lesions [89, 90]. The differential diagnosis is broad. The diagnosis is often made based on clinical, investigative parameters (electrophysiologic and imaging studies) and exclusion of other pathologies, mainly rotator cuff pathology, cervical radiculopathy, and brachial plexopathy [91]. The optimal

Table 20.3 Suprascapular nerve block for chronic pain conditions

Study authors	Type of chronic pain	Study design	Number of participants	Results	Conclusions
Wu et al. (2014) [58]	Adhesive capsulitis	Prospective, randomized, controlled study	42 patients Control group: physical therapy alone = 21 Intervention group: PRF applied to SSN via US guidance and physical therapy =21	12-week follow-up The intervention group (PRF to SSN and physical therapy) demonstrated significantly shorter time to onset of significant pain relief, reduction in VAS, and improvement in SPADI compared to the control group (physical therapy alone)	PRF to the SSN combined with physical therapy is superior (reduction in pain and disability) to physical therapy alone in patients suffering from adhesive capsulitis
Jeon et al. (2014) [54]	Hemiplegic shoulder pain	Comparative clinical trial	30 patients SSNB = 10 Intra-articular injection of corticosteroid = 10 Combination (SSNB followed by intra-articular corticosteroid) = 10	Reduction in VAS and improvement in ROM over observed period in all groups No statistically significant difference between the groups	Both SSNB and intra-articular steroid injection alone and in combination improve pain and function over time for hemiplegic shoulder pain. However, neither type of injection was demonstrated to be superior to the other
Jang et al. (2013) [59]	Chronic intractable shoulder pain >6 months Heterogeneous etiology: adhesive capsulitis, rotator cuff tear, mixed etiology	Case series	11 patients underwent SSN PRF following favorable response (pain improvement >50 %) following diagnostic SSNB	Significant reduction in pain (VAS) and significant improvement in shoulder function (OSS) at 6- and 9-month follow-up	PRF SSN results in long-term improvement (greater than 9 months) in shoulder pain and function in patients with intractable shoulder pain
Adey-Wakeling et al. (2013) [55]	Hemiplegic shoulder pain	Randomized controlled trial	64 stroke patients randomized to SSNB or placebo injection	Patients who received SSNB recorded significantly superior pain reduction (VAS) compared to placebo at 1, 4, and 12 weeks post intervention No significant differences in function (Modified Rankin Scale, Croft Disability Index) or quality of life (EuroQol Health Questionnaire) were demonstrated	SSNB provides sustained pain reduction in patients with hemiplegic shoulder pain but does not result in improved quality of life

Table 20.3 (continued)

Study authors	Type of chronic pain	Study design	Number of participants	Results	Conclusions
Shabat et al. (2013) [81]	Chronic headache attributable to lower cervical nerve roots (C5, C6)	Case series	69 patients undergoing PRF SSN for shoulder pain with concomitant chronic headache attributed to lower cervical nerve roots (C5, C6)	At 12 months 45 % of patients reported greater than 30 % reduction in chronic headache pain	PRF SSN may provide analgesia for patients suffering from chronic headache secondary to lower cervical nerve root (C5, C6) irritation
Gofeld et al. (2013) [82]	Chronic shoulder pain >3 months Heterogeneous etiology: adhesive capsulitis, tendinosis, arthritis, rotator cuff, or capsular tears	Randomized double-blind active placebo-controlled study	22 patients SSNB with lidocaine alone = 10 PRF applied to SSN = 12	Reduction in pain (NRS); improvement in function (SPADI) over 6-month follow-up for PRF SSN No significant difference between lidocaine vs. PRF SSN groups High dropout rate in both treatment arms (50 % for lidocaine group; 33 % for PRF SSN group)	Study suggests efficacy of PRF SSN for chronic shoulder pain. Study limited by high dropout rate resulting in study being underpowered
Simopoulos et al. (2012) [83]	Chronic shoulder pain >12 months Heterogeneous etiology	Retrospective case series	6 patients who had failed conservative management, SSNB with steroid, and PRF SSN and are considered unfit for surgery These patients underwent continuous radiofrequency lesion of the SSN (80° for 60 s)	Significant reduction in pain (NRS); functional improvement as measured by ROM	This small study demonstrates that continuous radiofrequency lesioning of the SSN may be an effective treatment for chronic intractable shoulder pain
Eyigor et al. (2010) [57]	Chronic shoulder pain >3 months; heterogeneous etiology	Single-blind, randomized, comparative clinical trial Outcome measures: Pain scores using VAS at rest and movement Range of motion (ROM) of shoulder joint Shoulder pain and disability index (SPADI) Short Form 36 Beck Depression Inventory Medication requirements Complications	50 patients Intra-articular injection of corticosteroid = 25 PRF applied to the SSN = 25	Improvements in pain, ROM of shoulder joint, and quality of life in both groups In the SSN PRF group, improvement lasted for 12 weeks in VAS, ROM, and SPADI Pain reduction was superior in the intra-articular group compared to the SSN PRF group	Both intra-articular steroids and AAN PRF reduced pain and improved function. Intra-articular steroids showed a greater reduction in pain throughout the study period
Gorthi et al. (2010) [9]	Chronic shoulder pain	Prospective randomized comparative study	50 patients SSNB under US guidance (treatment group) = 25 SSNB blind technique (control group) = 25	Both groups recorded significantly reduced pain (VAS) and improved function (CSS) post-procedure The SSNB US group showed significantly superior VAS and CSS scores compared to the control group	Performing SSNB under US guidance results in greater efficacy of block in terms of pain and shoulder function measures. In addition, US reduces the risk of vascular and neurological complications

(continued)

Table 20.3 (continued)

Study authors	Type of chronic pain	Study design	Number of participants	Results	Conclusions
Mitra et al. (2009) [65]	Adhesive capsulitis	Retrospective chart review over 3 years	28 consecutive patients Received SSNB as part of a protocol for adhesive capsulitis management The protocol also included intra-articular steroid, volume dilation of the joint, and finally manipulation of the shoulder	After protocol, patients demonstrated significant improvements in ROM being flexion and abduction	SSNB as part of a multimodal therapy protocol improves shoulder function
Liliang et al. (2009) [11]	Chronic shoulder pain for 3 months	Prospective case series	11 patients, total of 13 shoulder joints Treatment: PRF of the SSN	Significant pain relief in 10/13 joints at 1 month. And 9/13 shoulders at 6 months. Decreased SPADI scores at 6 months and 9 out of 11 patients reduced their analgesic medication	SSN PRF reduces shoulder pain and disability in a range of shoulder pathologies. Furthermore, patient's analgesic consumption is reduced
Di Lorenzo et al. (2006) [47]	Rotator cuff tendinitis	Prospective, randomized, crossover investigation	40 patients Treatment: SSNB and standard rehabilitation treatment Control: standard rehabilitation treatment alone	The SSNB group reported significantly less pain at rest and activity and with rehabilitation exercises compared to the control group	SSNB and standard rehabilitation for rotator cuff tendinitis is superior to standard rehabilitation alone for pain control and functional improvement
Taskaynata MA et al. (2005) [56]	Chronic shoulder pain	Prospective randomized study	60 patients Intra-articular steroid injection = 30 SSNB = 30	Significant improvement in pain and ROM in both groups compared to baseline at 1 week and 1 month No significant difference between the two treatments Complications in the intra-articular steroid group No complications in the SSNB group	Both intra-articular steroids and SSNB are effective for managing shoulder pain and improving shoulder function. SSNB is safe with negligible risk of complications
Shanahan EM et al. (2004) [48]	Chronic shoulder pain due to degenerative joint/rotator cuff disease	Randomized, single blind	67 patients 77 shoulder randomized Group 1: SSNB via anatomical landmark approach Group 2: SSNB via CT guidance	Significant improvements in pain scores and disability in both groups No significant differences between the two groups No significant complications in either group No significant adverse events in either group	Clinically there is no significant difference between SSNB performed via anatomical landmarks or CT guidance in terms of efficacy and complication rate
Schneider-Kolsky et al. (2004) [34]	Chronic shoulder pain, range of pathology	Case series	40 consecutive patients. Treated with CT-guided SSNB	Significant reduction in pain and disability at both short-term and long-term follow-up At long term (greater than 3 weeks) 29 % of patients had sustained analgesia and reduced disability	CT-guided SSNB provides effective short-term pain relief in chronic shoulder pain

Table 20.3 (continued)

Study authors	Type of chronic pain	Study design	Number of participants	Results	Conclusions
Shanahan et al. (2003) [5]	Chronic shoulder pain due to rheumatoid arthritis and/or degenerative disease	Randomized, double-blind, placebo-controlled trial	83 patients, 108 shoulders studied in total Treatment group: SSNB: 56 Control/placebo group: 52	Treatment group compared to placebo: Significant reduction in pain in the treatment group at 12-week follow-up Modest but significant reduction in shoulder disability at 12 weeks in treatment group No difference in quality of life measures (SF-36) between the 2 groups	SSNB is more effective than placebo in reducing pain and disability at 3-month follow-up for chronic shoulder pain of degenerative causes. However, it does not significantly improve quality of life compared to placebo
Karatas and Meray (2002) [71]	Adhesive capsulitis (frozen shoulder)	Single-blind, randomized comparative clinical trial	41 patients randomized into two groups: Group A: SSNB via anatomical landmarks Group B: near-nerve EMG-guided technique	In both groups, improvements in pain scores and ROM scores from baseline were significant VAS scores were significantly lowered in the EMG group compared to the blind technique at 60 min.	EMG-guided SSNB provides more rapid analgesia than the blind approach in immediate post-block time
Dahan et al. (2000) [72]	Frozen shoulder (adhesive capsulitis)	Double-blind randomized controlled trial	34 patients randomized into two groups: Treatment: 3 SSNB at 7-day intervals with 10 cc bupivacaine 0.5 % each block Control: same as treatment group except 10 cc of normal saline used for SSNB	Significant reduction in pain in the treatment group (64 %) compared to the control group (13 %) at 1 month Nonsignificant improvement in shoulder function in treatment group. No improvement in shoulder ROM	Repeated SSNB with local anesthetic alone reduces pain compared to placebo but does not improve shoulder function or shoulder ROM
Jones and Chattopadhyay (1999) [73]	Frozen shoulder (adhesive capsulitis)	Randomized trial of 30 patients	30 patients randomized 1st group: single SSNB 2nd group: course of intra-articular injections	SSNB produced a faster and more complete reduction in pain and restoration of ROM than intra-articular steroid	SSNB is superior to intra-articular steroid injection for pain reduction and improvements in shoulder ROM
Lewis RN (1999) [61]	Chronic shoulder pain due to rheumatoid or osteoarthritis	Case series	16 patients Treated with combined SSNB and ACNb (4 mL 1 % prilocaine and 4 mL 6 % aqueous phenol)	Significant reduction in pain intensity (69 %) and improvement in ROM (36–67 %) over a mean follow-up of 13 weeks	The combined SSNB and ACNb with local anesthetic and phenol provides pain relief and improvement in shoulder ROM
Gado and Emery (1993) [52]	Chronic shoulder pain due to rheumatoid arthritis	Double-blind comparative study	29 patients (58 shoulders) 1st group: SSNB with local anesthetic (bupivacaine) alone 2nd group: SSNB with local anesthetic and steroid	Both groups recorded significant improvements in pain, stiffness, and ROM up to 3 months Steroid did not improve outcomes. In fact, the bupivacaine-alone group responded better	SSNB is effective for reducing shoulder pain and improving function. But the addition of steroid does not seem to confer added benefit

(continued)

Table 20.3 (continued)

Study authors	Type of chronic pain	Study design	Number of participants	Results	Conclusions
Vecchio et al. (1993) [74]	Chronic shoulder pain due to rotator cuff lesions tendinitis and tears	Randomized clinical controlled trial	28 patients Divided into tendinitis and tears Tendinitis 15 Active injection = 10 Placebo injection = 5 Tears Active injection = 5 Placebo injection = 8	Tendinitis group: Significant improvement in night pain up to 12 weeks; movement pain significantly improved at 1 week but no difference at later follow-up, no difference to placebo in rest pain. Improvement in ROM only till 4 weeks Tear group: Significant improvement in night pain up to 12 weeks, significant improvement in movement pain till 12 weeks, no difference to placebo in rest pain. Only active abduction improved till 4 weeks; other ROM parameters showed no difference to placebo	SSNB improves the pain of rotator cuff pathology for at least 3-month period. While there is an improvement in shoulder function, this is only short term
Emery et al. (1989) [51]	Chronic shoulder pain due to rheumatoid arthritis	Randomized study	17 patients with bilateral shoulder rheumatoid arthritis 34 shoulders in total In each patient: one shoulder, SSNB and sham intra-articular injection Second shoulder: Intra-articular steroid and sham SSNB	Compared with intra-articular steroids SSNB resulted in longer duration of pain relief, improvement in pain index and range of movement	
Brown et al. (1988) [39]	Chronic shoulder pain due to glenohumeral arthritis not suitable for and had received conservative medical management	Pilot study. Consecutive case series.	22 patients, 26 shoulders treated with RF heat lesion of the SSN	Analgesia: 7 produced no relief, 10 obtained good pain relief Duration: 9 produced good relief for 3 months, 14 produced relief for 6 months, 9 produced relief for more than 7 months. Three in last group had 18-month pain relief	SSNB PRF can provide variable duration pain relief in patients with advanced glenohumeral arthritis who are not suitable for surgery

ACNb articular branches of the circumflex nerve, *CSS* constant shoulder score, *CT* computer tomography, *EMG* electromyography, *OSS* Oxford shoulder score, *MPQ* McGill–Melzack pain questionnaire (MPQ), *NRS* numerical rating scale, *PRF* pulsed radiofrequency, *ROM* range of motion, *SF-36* Short Form 36 health survey, *SPADI* shoulder pain and disability index, *SSN* suprascapular nerve, *SSNB* suprascapular nerve block, *VAS* visual analog scale, *US* ultrasound

management of suprascapular neuropathy has not been determined. Studies have reported good to excellent results in either nonsurgical management [92, 93] or surgical management [84, 85, 88].

Due to the difficulty in differentiating suprascapular neuropathy from other shoulder pathologies, SSNB can be performed to aid in the diagnosis [2]. A diagnosis of SSNB is often based on clinical history and examination findings together with electrodiagnostic studies and MRI [2]. In cases where the diagnosis is uncertain after electrodiagnostic studies, SSNB may be helpful. The test is positive if the pain is completely relieved [89].

Therapeutic (Chronic Pain)

Chronic Shoulder Pain (General)

SSNB has been widely investigated in a variety of chronic pain conditions (Table 20.3). A number of trials have been performed which examined chronic shoulder pain in a heterogeneous group without studying individual pathologies [6]. Of these trials, 4 were randomized [9, 56, 57, 82]. Two of these randomized studies compared SSNB to intra-articular steroid for shoulder pain and function [56, 57]. Another study compared SSNB under US to SSNB via surface anatomy [9]. In the more recent trial by Eyigor et al., SSNB resulted in significant improvements in pain (as measured by the visual analog scale [VAS]), range of shoulder motion (active, passive), shoulder pain and disability index (SPADI), and SF 36 scores ($p<0.05$) (Table 20.3) [57]. Over the 12-week follow-up period, shoulder pain on movement (as measured by VAS) reduced from 6.3 to 1.6, while the total SPADI reduced from 120.3 to 26.7 [57].

The fourth randomized study assessed the effects of pulsed radiofrequency rhizotomy on the SSN versus an active placebo (combined SSNB with lidocaine and sham procedure) [82]. While there were high dropout rates (13 of 22 participants), the results suggested greater improvements in pain and function in the group receiving pulsed radiofrequency to the SSN [82].

There is only one randomized controlled trial which investigated chronic shoulder pain secondary to either degenerative disease or inflammatory in origin [5]. This investigation revealed a significant and sustained benefit in pain and disability scores as well as the range of movement at weeks 1, 4, and 12 [5]. The remaining trials consisted of case series which showed significant improvement in pain and disability in chronic nonspecific shoulder pain following SSNB [11, 34].

The most common pathologies individually studied are chronic pain from rheumatoid arthritis or osteoarthritis, adhesive capsulitis (frozen shoulder), and persistent rotator cuff lesions.

Shoulder Joint Arthritis: Rheumatoid Arthritis and Osteoarthritis

A number of studies have assessed the efficacy of SSNB for the pain and disability in patients with osteoarthritis and rheumatoid arthritis.

Local corticosteroid injection and gentle mobilization may improve rheumatoid shoulder in the early stages of disease [94]. However, when glenohumeral damage is advanced, this treatment option is not as effective [95]. Two randomized controlled trials have been published to suggest the efficacy of SSNB. One is a randomized controlled trial comparing the efficacy of intra-articular steroid injection with SSNB in patients with long-standing rheumatoid arthritis (average 17 years); SSNB provided prolonged pain relief (3 months) and superior improvement in shoulder movement [51]. Another one that is a double-blind placebo-controlled RCT including patients suffering from rheumatoid shoulder was performed recently [5]. A total of 108 shoulders were randomized, to receive an injection of 10 mL of bupivacaine 0.5 % and 40 mg methylprednisolone into the suprascapular fossa or a placebo injection of 5 mL of normal saline [5]. SSNB was performed using surface anatomical landmarks as described by Dangoisse et al.. [31]. At 3-month follow-up, the active injection (local anesthetic and steroid) group recorded significantly superior pain reduction (VAS scale) and functional improvement (shoulder pain and disability index [SPADI], SF-36 scales) compared to the placebo group. A notable finding was that 67 % of the patients receiving the active injection improved by at least 10 points on the SPADI which is a significant clinical improvement [96]. The only adverse effects were minor including chest wall tenderness in one subject which resolved and minor bruising in another [5].

Similarly, SSNB provided significantly better analgesia and superior movement in patients with long-standing rheumatoid arthritis who were unresponsive to intra-articular injection of steroid [39, 52]. Interestingly, supplementation of the local anesthetic solution with steroid conferred no additional benefit [52].

Adhesive Capsulitis (Frozen Shoulder)

Also known as adhesive capsulitis, frozen shoulder is characterized by significantly restricted shoulder movement in patients with shoulder pain [49]. This condition progresses from pain to pain accompanied by gradually worsening stiffness to reduced pain accompanied by profound stiffness. The last stage appears to be self-limiting and recovery is gradual and spontaneous, with an excellent chance of complete return of function within 1–2 years irrespective of therapy [97, 98]. The goal of treatment in the early stage is to alleviate pain so that physiotherapy can be effective in restoring normal shoulder movement and activity [98]. A comparative clinical trial performing SSNB on patients with adhesive capsulitis demonstrated a significant improvement in pain and ROM scores, but follow-up was limited to only 90 min post SSNB with local anesthetic alone [71]. Furthermore there was no placebo control [71]. A small randomized trial (30 patients) compared the effects of SSNB to intra-articular shoulder injections for adhesive capsulitis over a longer follow-up period [73]. The investigators found that SSNB produced faster onset and more effective analgesia compared to a series of intra-articular injections [73].

Furthermore, significantly improved shoulder function (measured by abduction and external rotation) was also observed [73]. These effects lasted up to 3 months [73].

A later placebo-controlled trial examined the response of SSNB with bupivacaine compared to placebo [72]. There was a significant reduction in pain in the patients receiving local anesthetic blockade up to 1-month follow-up [72]. However, no significant improvement in shoulder function or range of movement was found. This study did not inject steroid medication as part of their treatment [72].

The beneficial effects of pulsed radiofrequency treatment of the suprascapular nerve for adhesive capsulitis were demonstrated in a RCT [58]. One half of the studied population underwent physical therapy alone ($n=21$). The other half of the study population underwent physical therapy combined with SSNB via pulsed RF treatment ($n=21$). The group of patients undergoing the combination therapy (physical therapy and pulsed RF treatment) recorded significantly faster and greater analgesia, improved range of movement, and less disability than the group undergoing physical therapy alone [58]. This difference was evident to the 12-week follow-up period [58].

Persistent Rotator Cuff Lesions

Rotator cuff tendinitis is a common cause of shoulder pain in adults and may result in considerable morbidity [99, 100]. Many patients respond to conservative management, including avoiding activities likely to aggravate the lesion, use of nonsteroidal anti-inflammatory drugs, local injection of steroid, and physiotherapy [99, 100]. However, significant symptoms may persist: in one retrospective, long-term follow-up study, symptoms of severe shoulder pain persisted in approximately 26 % of patients after a mean duration of 12 months following the first presentation of pain [100].

In this subset of patients with persistent symptoms, SSNB has been demonstrated to provide effective pain relief and improved range of motion [74]. Although the therapeutic effect is temporary (4–12 weeks), it can be simply repeated in outpatient settings with minimal risk of complications. This block is also an effective way to control pain in patients awaiting surgery [74].

Recently, pulsed radiofrequency of the SSN has shown promise in providing prolonged analgesia for those patients responding to SSNB (rotator cuff lesion identified on clinical and radiological grounds) but where analgesia is not sustained [10]. After pulsed radiofrequency lesioning, a significant reduction in pain (VAS) and improvement in shoulder function (constant and Oxford shoulder scores) were reported, lasting till 3-month follow-up [10]. These results are similar to those of Liliang et al.. [11], who in addition to improvement in pain and function also demonstrated a reduction in medication requirements in their study group. However, both injection and RF trials did not include a placebo control group, and further investigation is required to confirm the efficacy of the neural blockade or ablation technique in the management of rotator cuff tendinitis.

Other Chronic Pain States Responsive to SSNB

SSNB has been found to reduce chronic hemiplegic shoulder pain [54, 55]. A retrospective study discovered that SSNB may be effective in reducing the severity of headaches attributable to lower cervical nerve root (C6, C7) irritation [81]. The further application of this finding awaits further confirmation by appropriately constructed trials.

In summary, SSNB is effective for short-term pain relief and improvement in shoulder function in a variety of painful shoulder conditions. The main causes of shoulder pain studied were arthritic conditions, rotator cuff lesions, and adhesive capsulitis. Unfortunately in many studies, the patient population was heterogeneous with regard to shoulder pathology. Therefore, interpreting specifically which pathology responds best to SSNB is difficult to determine. The results of recent studies demonstrate that pulsed radiofrequency to the SSN provides more sustained analgesia and functional improvement than earlier methods of SSNB.

Summary and Conclusion

On review of the literature discussed above, the uses of SSNB and pulsed radiofrequency lesioning of the SSN can be summarized in Table 20.4.

There have been many described techniques by which to perform SSNB. While the radiologically guided techniques (fluoroscopic, ultrasound guided) have grown in popularity, a knowledge of surface anatomical landmarks is still recommended. SSNB is a safe regional technique with a very low rate of complications.

While interscalene BPB provides the best form of regional anesthesia for shoulder surgery, SSNB is an appropriate alternative particularly for arthroscopic surgery. Combining axillary nerve block with SSNB improves the analgesic effect.

The studies mentioned in this chapter demonstrate efficacy of SSNB in chronic shoulder pain. While this review has concentrated on SSNB, the importance of physical therapy in patients with chronic shoulder pain cannot be ignored. The timing and place of SSNB as part of a multidisciplinary pain management program deserves further study.

Pulsed radiofrequency lesioning of the suprascapular nerve has emerged as an effective method by which to prolong the analgesic effects of SSNB with resultant functional improvement.

Perhaps the major limitation identified in the literature [6] is that many trials did not differentiate the efficacy of SSNB on different shoulder pathologies. Many trials were on heterogeneous populations suffering from chronic shoulder pain. This would include patients with osteo- or rheumatoid arthritis, rotator cuff lesions, and myofascial pain. By including a heterogeneous population, the external validity of these studies is reduced. Future research should attempt to identify which specific shoulder pathologies SSNB is effective for. This in turn would better assist the clinician to better select patients who should receive SSNB as part of their management.

Table 20.4 Summary of evidence on suprascapular nerve block and rhizotomy

Studied uses of SSNB	Level of evidence
SSNB is inferior to ISBPB for shoulder surgery	I
SSNB is not effective for reducing all cases of ipsilateral shoulder pain post-thoracotomy	I
SSNB does not improve outcomes in ambulatory nonarthroscopic shoulder surgery when added to ISBPB	I
SSNB reduces hemiplegic shoulder pain	I
SSNB is effective for postoperative pain control for shoulder arthroscopic surgery and reduces opioid requirements and nausea and vomiting	II-1
SSNB is effective for providing short-term (3 months) analgesia and improving function for chronic shoulder pain due to degenerative pathology or rotator cuff lesions	II-1
PRF of the SSN can provide longer-lasting analgesia and improved shoulder function than single SSNB	II-2
SSNB combined with axillary nerve block is sufficient for arthroscopic shoulder surgery	II-3

ISBPB interscalene brachial plexus block, *PRF* pulsed radiofrequency, *SSN* suprascapular nerve, *SSNB* suprascapular nerve block
Based on Quality of Evidence Gradings as recommended by the US Preventive Services Task Force (Appendix I) [101]

Appendix I

Level of evidence	Description
I	Evidence from at least one properly designed randomized controlled trial
II-1	Evidence obtained from well-designed controlled trials without randomization
II-2	Evidence obtained from well-designed cohort or case–control analytic studies, preferable from more than one center or research group
II-3	Evidence obtained from multiple time series with or without the intervention
III	Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

Quality of Evidence Grading as recommended by US Preventive Services Task Force [101]

References

1. Aszmann OC, Dellon AL, Birely BT, et al. Innervation of the human shoulder joint and its implications for surgery. Clin Orthop Relat Res. 1996;330:202–7.
2. Piasecki DP, Romeo AA, Bach BR, Nicholson GP. Suprascapular neuropathy. J Am Acad Orthop Surg. 2009;17:665–76.
3. Ritchie ED, Tong D, Chung F, Norris AM, Miniaci A, Vairavanathan SD. Suprascapular nerve block for postoperative pain relief in arthroscopic shoulder surgery: a new modality? Anesth Analg. 1997;84:1306–12.
4. Checcucci G, Allegra A, Bigazzi P, Gianesello L, Ceruso M, Gritti G. A new technique for regional anesthesia for arthroscopic shoulder surgery based on a suprascapular nerve block and an axillary nerve block: an evaluation of the first results. Arthroscopy. 2008;24:689–96.
5. Shanahan EM, Ahern M, Smith M, Wetherall M, Bresnihan B, Fitzgerald O. Suprascapular nerve block (using bupivacaine and methylprednisolone acetate) in chronic shoulder pain. Ann Rheum Dis. 2003;62:400–6.
6. Chan CW, Peng PP. Suprascapular nerve block: a narrative review. Reg Anesth Pain Med. 2011;36:358–73.
7. Harmon D, Hearty C. Ultrasound-guided suprascapular nerve block technique. Pain Physician. 2007;10:743–6.
8. Peng PWH, Wiley MJ, Liang J, Bellingham GA. Ultrasound-guided suprascapular nerve block: a correlation with fluoroscopic and cadaveric findings. Can J Anaesth. 2010;57:143–8.
9. Gorthi V, Moon YL, Kang JH. The effectiveness of ultrasonography-guided suprascapular nerve block for perishoulder pain. Orthopedics. 2010;16:238–41.
10. Kane TPC, Rogers P, Hazelgrove J, Wimsey S, Harper GD. Pulsed radiofrequency applied to the suprascapular nerve in painful tear arthroscopy. J Shoulder Elbow Surg. 2008;17:436–40.
11. Liliang PC, Lu K, Liang CL, Tsai YD, Hsieh CH, Chen HJ. Pulsed radiofrequency lesioning of the suprascapular nerve for chronic shoulder pain. Pain Med. 2009;10:70–5.
12. Ajmani ML. The cutaneous branch of the human suprascapular nerve. J Anat. 1994;185:439–42.
13. Voster W, Lange CPE, Briet RJP, et al. The sensory branch distribution of the suprascapular nerve: an anatomic study. J Shoulder Elbow Surg. 2008;17:500–2.
14. Lee HY, Chung IH, Sir WS, et al. Variations of the ventral rami of the brachial plexus. J Korean Med Sci. 1992;7:19–24.
15. Polguj M, Sibinski M, Grzegorzewski A, Grzelak P, Majos A, Topol M. Variation in morphology of suprascapular notch as a factor of suprascapular nerve entrapment. Int Orthop. 2013;37:2185–92.
16. Bigliani LU, Dalsey RM, McCann PD, April EW. An anatomical study of the suprascapular nerve. Arthroscopy. 1990;6:301–5.
17. Ozer Y, Grossman JA, Gilbert A. Anatomic observations on the suprascapular nerve. Hand Clin. 1995;11:539–44.
18. Warner JP, Krushell RJ, Masquelet A, Gerber C. Anatomy and relationships of the suprascapular nerve: anatomical constraints to mobilization of the supraspinatus and intraspinatus muscles in the management of massive rotator-cuff tears. J Bone Joint Surg. 1992;74-A:36–45.
19. Inouye Y. Conduction along the articular branch of the suprascapular nerve. Acta Neurol Scand. 1978;58:230–40.
20. Mestdagh H, Drizenko A, Ghestem P. Anatomical basis of suprascapular nerve syndrome. Anat Clin. 1981;3:67–71.
21. Alon M, Weiss S, Fishel B, Dekel S. Bilateral suprascapular nerve entrapment syndrome due to an anomalous transverse scapular ligament. Clin Orthop. 1988;234:31–3.
22. Cohen SB, Dines DM, Moorman CT. Familial calcification of the superior transverse scapular ligament causing neuropathy. Clin Orthop. 1997;334:131–5.
23. Rengacharry SS, Neff JP, Singer PA, Brackett CE. Suprascapular entrapment neuropathy: a clinical, anatomical and comparative study. Part 1: clinical study. Neurosurgery. 1979;5:441–6.
24. Natsis K, Totlis T, Tsikaras P, Appell HJ, Skandalakis P, Koebke J. Proposal for classification of the suprascapular notch: a study on 423 dried scapulas. Clin Anat. 2007;20:135–9.
25. Wertheim HM, Rovenstein EA. Suprascapular nerve block. Anesthesiology. 1941;2:541–5.
26. Moore DC. Block of the suprascapular nerve. In: Moore DC, editor. Regional block. 4th ed. Springfield: Charles C Thomas Co; 1979.
27. Gordh T. Suprascapular nerve block. In: Eriksson E, editor. Illustrated handbook in local anesthesia. 2nd ed. Copenhagen: Munksgaard; 1979.
28. Katz J. Atlas of regional anesthesia. 2nd ed. Norwalk: Appleton; 1994.
29. Granirer LW. A simple technique for suprascapular nerve block. N Y State J Med. 1951;51:1048.
30. Breen TW, Haigh JD. Continuous suprascapular nerve block for analgesia of scapular fracture. Can J Anaesth. 1990;37:786–8.
31. Dangoisse MJ, Wilson DJ, Glynn CJ. MRI and clinical study of an easy and safe technique of suprascapular nerve blockade. Acta Anaesthesiol Belg. 1994;45:49–54.
32. Barber FA. Suprascapular nerve block for shoulder arthroscopy. Arthroscopy. 2005;21:1015.
33. Fiegl GC, Anderhuber F, Dorn C, Pipam W, Rosmarin W, Likar R. Modified lateral block of the suprascapular nerve: a safe approach and how much to inject? A morphological study. Reg Anesth Pain Med. 2007;32:488–94.
34. Schneider-Kolsky ME, Pike J, Connell DA. CT-guided suprascapular nerve blocks: a pilot study. Skeletal Radiol. 2004;33:277–82.
35. Gofeld M. Ultrasonography in pain medicine: a critical review. Pain Pract. 2008;8:226–40.
36. Neviaser TJ. Arthroscopy of the shoulder. Orthop Clin North Am. 1987;18:361–72.
37. Yucesoy C, Akkaya T, Ozel O, et al. Ultrasonographic evaluation and morphometric measurements of the suprascapular notch. Surg Radiol Anat. 2009;31:409–14.
38. Meyer-Witting M, Foster JMG. Suprascapular nerve block in the management of cancer pain. Anaesthesia. 1992;47:626.

39. Brown DE, James DC, Roy S. Pain relief by suprascapular nerve block in glenohumeral arthritis. Scan J of Rheumatol. 1988;17:411–5.
40. Bonica JJ. Management of pain. Pittsburgh: Lean and Febiger; 1953.
41. Shah RV, Racz GB. Pulsed mode radiofrequency lesioning of the suprascapular nerve for the treatment of chronic shoulder pain. Pain Physician. 2003;6:503–6.
42. Hackworth RJ. A new and simplified approach to target the suprascapular nerve with ultrasound. J Clin Anesth. 2013;25:347–8.
43. Bennett DL, Cronin AM, Palmer WE, Kattapuram SV, Huang AJ. Optimization and standardization of technique for fluoroscopically guided suprascapular nerve blocks. AJR Am J Roentgenol. 2014;202:576–84.
44. Siegenthaler A, Morrigl B, Mlekusch S, Schliessbach J, Haug M, Curatolo M, Eichenberger U. Ultrasound-guided suprascapular nerve block, description of a novel supraclavicular approach. Reg Anesth Pain Med. 2012;37:325–8.
45. Riazi S, Carmichael N, Awad I, Holtby RM, McCartney CJ. 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.
46. Fiegl GC, Dorn C, Likar R. What local anesthetic volume should be used for suprascapular nerve block? Reg Anesth Pain Med. 2008;33:571–3.
47. Di Lorenzo L, Pappagallo M, Gimigliano R, Palmieri E, Saviano E, Bello A, Forte A, Deblasio E, Trombetti C. Pain relief in early rehabilitation of rotator cuff tendinitis: any role for indirect suprascapular nerve block? Eura Medicophys. 2006;42:195–204.
48. Shanahan EM, Smith MD, Wetherall M, Loft CW, Slavotinek J, Fitzgerald O, Ahern MJ. Suprascapular nerve block in chronic shoulder pain: are the radiologists better? Ann Rheum Dis. 2004;63:1035–40.
49. Wassef MR. Suprascapular nerve block. A new approach for the management of frozen shoulder. Anaesthesia. 1992;47:120–4.
50. Parris WC. Suprascapular nerve block: a safer technique. Anesthesiology. 1990;72:580–1.
51. Emery P, Bowman S, Wedderburn L, Grahame R. Suprascapular nerve block for chronic shoulder pain in rheumatoid arthritis. BMJ. 1989;299:1079–80.
52. Gado K, Emery P. Modified suprascapular nerve block with bupivacaine alone effectively controls chronic shoulder pain in patients with rheumatoid arthritis. Ann Rheum Dis. 1993;52:215–8.
53. Ridsall JE, Sharwood-Smith GH. Suprascapular nerve block. New indications and a safer technique. Anaesthesia. 1992;47:626.
54. Jeon WH, Park GW, Jeong HJ, Sim YJ. The comparison of effects of suprascapular nerve block, intra-articular steroid injection, and a combination therapy on hemiplegic shoulder pain: pilot study. Ann Rehabil Med. 2014;38(2):167–73.
55. Adey-Wakeling Z, Crotty M, Shanahan EM. Suprascapular nerve block for shoulder pain in the first year after stroke: a randomized controlled trial. Stroke. 2013;44:3136–41.
56. Taskaynata MA, Yilmaz B, Ozgul A, Yazicioglu K, Kalyon TA. Suprascapular nerve block versus steroid injection for nonspecific shoulder pain. Tohoku J Exp Med. 2005;205:19–25.
57. Eyigor C, Eyigor S, Korkmaz OK, Uyar M. Intra-articular corticosteroid injections versus pulsed radiofrequency in painful shoulder: a prospective, randomized, single-blinded study. Clin J Pain. 2010;26:386–92.
58. Wu YT, Ho CW, Chen YL, Li TY, Lee KC, Chen LC. Ultrasound-guided pulsed radiofrequency stimulation of the suprascapular nerve for adhesive capsulitis: a prospective, randomized, controlled trial. Anesth Analg. 2014;119(3):686–92.
59. Jang JS, Choi HJ, Kang SH, Yang JS, Lee JJ, Hwang SM. Effect of pulsed radiofrequency neuromodulation on clinical improvements in the patients of chronic intractable shoulder pain. J Korean Neurosurg Soc. 2013;54:507–10.
60. Rose DL, Kelly CR. Suprascapular nerve block in shoulder pain. J Kans Med Soc. 1969;70:135–6.
61. Lewis RN. The use of combined suprascapular and circumflex (articular branches) nerve blocks in the management of chronic arthritis of the shoulder joint. Eur J Anaesthesiol. 1999;16:37–41.
62. Shanahan EM, Shanahan KR, Hill CL, Ahern MJ, Smith MD. Safety and acceptability of suprascapular nerve block in rheumatology patients. Clin Rheumatol. 2012;31:145–9.
63. Martinez-Barenys C, Busquets J, de Castro PE, Garcia-Guasch R, Perez J, Fernandez E, Mesa A. Randomized double-blind comparison of phrenic nerve infiltration and suprascapular nerve block for ipsilateral shoulder pain after thoracic surgery. Eur J Cardiothorac Surg. 2010;40(1):106–12.
64. Saha S, Brish EL, Lowry AM, Boddu K. In select patients, ipsilateral post-thoracotomy shoulder pain relieved by suprascapular nerve block. Am J Ther. 2010;18(4):309–12.
65. Mitra R, Harris A, Umphrey C, Smuck M, Fredericson M. Adhesive capsulitis: a new management protocol to improve passive range of movement. PM R. 2009;1:1064–9.
66. Jerosch J, Saad M, Greig M, Filler T. Suprascapular nerve block as a method of preemptive pain control in shoulder surgery. Knee Surg Sports Traumatol Arthrosc. 2008;16:602–7.
67. 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.
68. 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.
69. Neal JM, McDonald SB, Larkin KL, Polissar NL. Suprascapular nerve block prolongs analgesia after nonarthroscopic shoulder surgery but does not improve outcome. Anesth Analg. 2003;96:982–6.
70. Tan N, Agnew NM, Scawn ND, Pennefather SH, Chester M, Russell GN. Suprascapular nerve block for ipsilateral shoulder pain after thoracic epidural analgesia: a double-blind comparison of 0.5% bupivacaine and 0.9% saline. Anesth Analg. 2002;94:199–202.
71. Karatas GK, Meray L. Suprascapular nerve block for pain relief in adhesive capsulitis: a comparison of two different techniques. Arch Phys Med Rehabil. 2002;83:593–7.
72. Dahan TH, Fortin L, Pelletier M, Petit M, Vadeboncoeur R, Suissa S. Double blind randomized clinical trial examining the efficacy of bupivacaine suprascapular nerve blocks in frozen shoulder. J Rheumatol. 2000;27:1464–9.
73. Jones DS, Chattopadhyay C. Suprascapular nerve block for the treatment of frozen shoulder in primary care: a randomized trial. Br J Gen Pract. 1999;49:39–41.
74. Vecchio PC, Adebajo AO, Hazleman BL. Suprascapular nerve block for persistent rotator cuff lesions. J Rheumatol. 1993;20:453–5.
75. Lee JJ, Kim DY, Jwang JT, Lee SS, Hwang SM, Kim GH, Jo YG. Effect of ultrasonographically guided axillary nerve block combined with suprascapular nerve block in arthroscopic rotator cuff repair: a randomized controlled trial. Arthroscopy. 2014;30(8):906–14.
76. McGrath B, Elgendy H, Chung F, Kamming D, Curti B, King S. Thirty percent of patients have moderate to severe pain 24 hr after ambulatory surgery: a survey of 5703 patients. Can J Anaesth. 2004;51:886–91.
77. Brown AR, Weiss R, Greenerg C, et al. Interscalene block for shoulder arthroscopy: a comparison with general anesthesia. Arthroscopy. 1993;9:295–300.

78. D'Alessio J, Rosenblum M, Shea K, Freitas D. A retrospective comparison of interscalene and general anesthesia for ambulatory surgery shoulder arthroscopy. Reg Anesth. 1995;20:62–8.
79. Borgeat A, Pershak H, Bird P, et al. Patient-controlled interscalene analgesia with ropivacaine 0.2% versus patient controlled intravenous analgesia after major shoulder surgery: effects on diaphragmatic and respiratory function. Anesthesiology. 2000;92:102–8.
80. Elsharkawy HA, Abd-Elsayed AA, Cummings 3rd KC, Soliman LM. Analgesic efficacy and technique of ultrasound-guided suprascapular nerve catheters after shoulder arthroscopy. Ochsner J. 2014;14:259–63.
81. Shabat S, Leitner J, Folman Y. Pulsed radiofrequency for the suprascapular nerve for patients with chronic headache. J Neurosurg Anesthesiol. 2013;25:340–3.
82. Gofeld M, Restrepo-Garces CE, Theodore BR, Faclier G. Pulsed radiofrequency of suprascapular nerve for chronic shoulder pain: a randomized double-blind active placebo-controlled study. Pain Pract. 2013;13:96–103.
83. Simopoulos TT, Nagda J, Aner MM. Percutaneous radiofrequency lesioning of the suprascapular nerve for the management of chronic shoulder pain: a case series. J Pain Res. 2012;5:91–7.
84. Vastamaki M, Gorannson H. Suprascapular nerve entrapment. Clin Orthop. 1993;297:135–43.
85. Antoniadis G, Richter HP, Rath S, Braun V, Moese G. Suprascapular nerve entrapment: experience with 28 cases. J Neurosurg. 1996;85:1020–5.
86. Callahan JD, Scully TB, Shapiro SA, Worth RM. Suprascapular nerve entrapment: a series of 27 cases. J Neurosurg. 1991;74:893–6.
87. Cummins CA, Messer TM, Nuber GW. Current concepts review suprascapular nerve entrapment. J Bone Joint Surg. 2000;82:415–24.
88. Post M. Diagnosis and treatment of suprascapular nerve entrapment. Clin Orthop Relat Res. 1999;223:92–100.
89. Aiello I, Serra G, Traina GC, Tugnoli V. Entrapment of the suprascapular nerve at the spinoglenoid notch. Ann Neurol. 1982;12:314–6.
90. Ogino T, Minami A, Kato H, Hara R, Suzuki K. Entrapment neuropathy of the suprascapular nerve by a ganglion: a report of three cases. J Bone Joint Surg Am. 1991;73:141–7.
91. Walsworth MK, Mills JT, Michener LA. Diagnosing suprascapular neuropathy in patients with shoulder dysfunction: a report of 5 cases. Phys Ther. 2004;84:359–72.
92. Martin SD, Warren RF, Martin TL, Kennedy K, O'Brien SJ, Wickiewicz TL. Suprascapular neuropathy: results of nonoperative treatment. J Bone Joint Surg Am. 1997;79:1159–65.
93. Drez Jr D. Suprascapular neuropathy in the differential diagnosis of rotator cuff injuries. Am J Sports Med. 1976;4:43–5.
94. Petersson CJ. Painful shoulders in patients with rheumatoid arthritis. Scand J Rheumatol. 1986;15:275–9.
95. Hayes JMW, Cats A. Rheumatoid arthritis-management: end stage and complication. In: Dieppe PA, Klippel JH, editors. Rheumatology. London: Mosby; 1994.
96. Green S, Buchbinder R, Forbes A, Bellamy N. A standardized protocol for measurement of range of movement of the shoulder using the Plurimeter-V inclinometer and assessment of its intrarater and interrater variability. Arthritis Care Res. 1998;11:43–51.
97. Grey RG. The natural history of 'idiopathic' frozen shoulder. J Bone Joint Surg. 1978;60:564–5.
98. Jayson MIV. Frozen shoulder: adhesive capsulitis. BMJ. 1981;283:1005–6.
99. Bigliani LU, Morrison DS. Miscellaneous degenerative disorders of the shoulder. In: Dee R, Mango E, Hurst LC, editors. Principles of orthopedic practice. New York: McGraw-Hill; 1989.
100. Chard MD, Satelle LM, Hazleman BL. The long term outcome of rotator cuff tendinitis-a review study. Br J Rheumatol. 1988;27:385–9.
101. US Preventive Services Task Force. Guide to clinical preventive services: report of the US Preventive Services Task force. Pennsylvania: Diane Publishing Co; 1989.

Chapter 21

Glenohumeral Joint

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_21,

Anatomy

The glenohumeral joint (GHJ) is a synovial "ball-and-socket" joint composed of a round humeral head and a relatively small, flat, pear-shaped glenoid fossa. The latter is deepened by a fibrocartilaginous rim, the glenoid labrum (Fig. 21.1). Because only one third of the humeral head is covered by the glenoid cavity, it confers the shoulder inherent instability, making it susceptible to subluxation and dislocation.

Inside the joint capsule, the synovial membrane overlies the long head of biceps (LHB) tendon and forms three synovial recesses: the biceps tendon recess anteriorly, the subscapularis recess medially, and the axillary pouch inferiorly (Fig. 21.2). The biceps tendon recess allows a portal of entry to the GHJ through the rotator cuff interval [1–2].

The rotator cuff interval (RCI) is a triangular space defined by the subscapularis (SSC) tendon, supraspinatus (SS) tendon, and the base of the coracoid process. It is a space where the GHJ synovial lining extends around the biceps tendon and where the surgeon inserts the arthroscope into the GHJ to avoid damaging the cuff tendons. Thus, this allows access for GHJ injection by the interventionist (Fig. 21.3). The contents of RCI are the LHB tendon and the superior glenohumeral ligament.

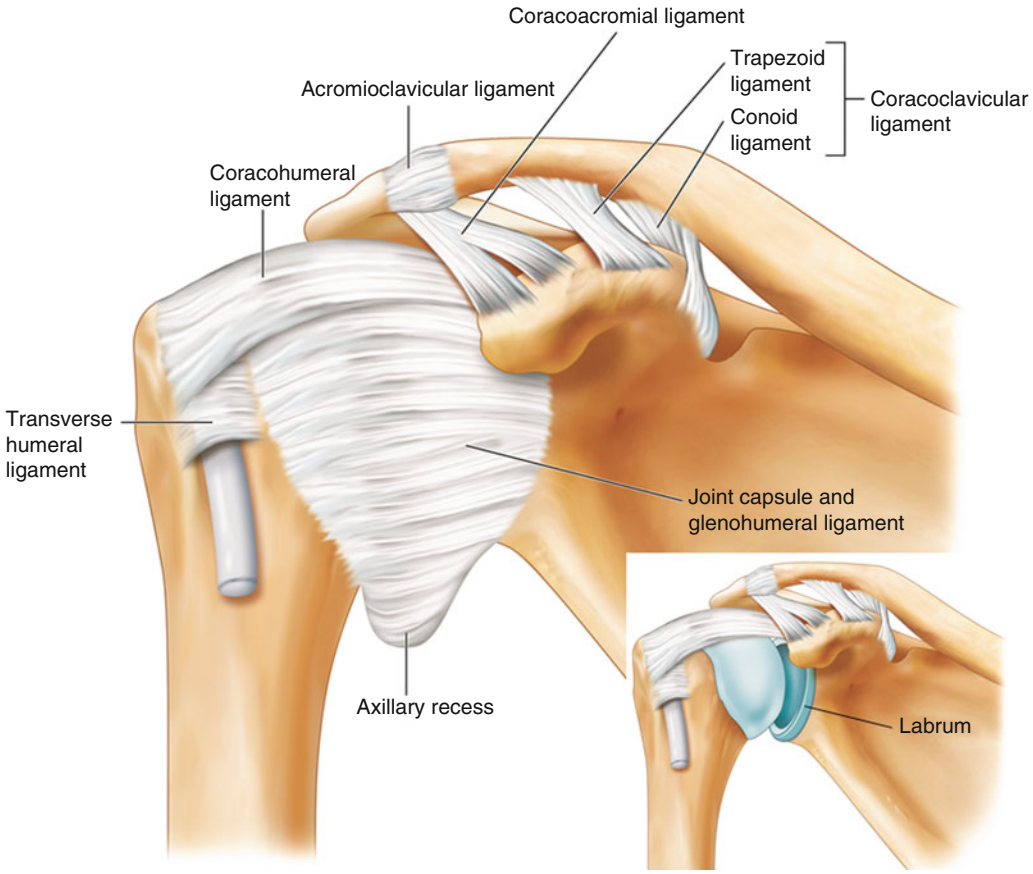


Fig. 21.1 Glenohumeral joint showing various ligaments and the joint capsule. The anterior capsule is reinforced by the superior, middle, and inferior glenohumeral ligament. The insert shows the articular surface, the glenoid process, and the labrum (Reproduced with permission from Philip Peng Educational Series)

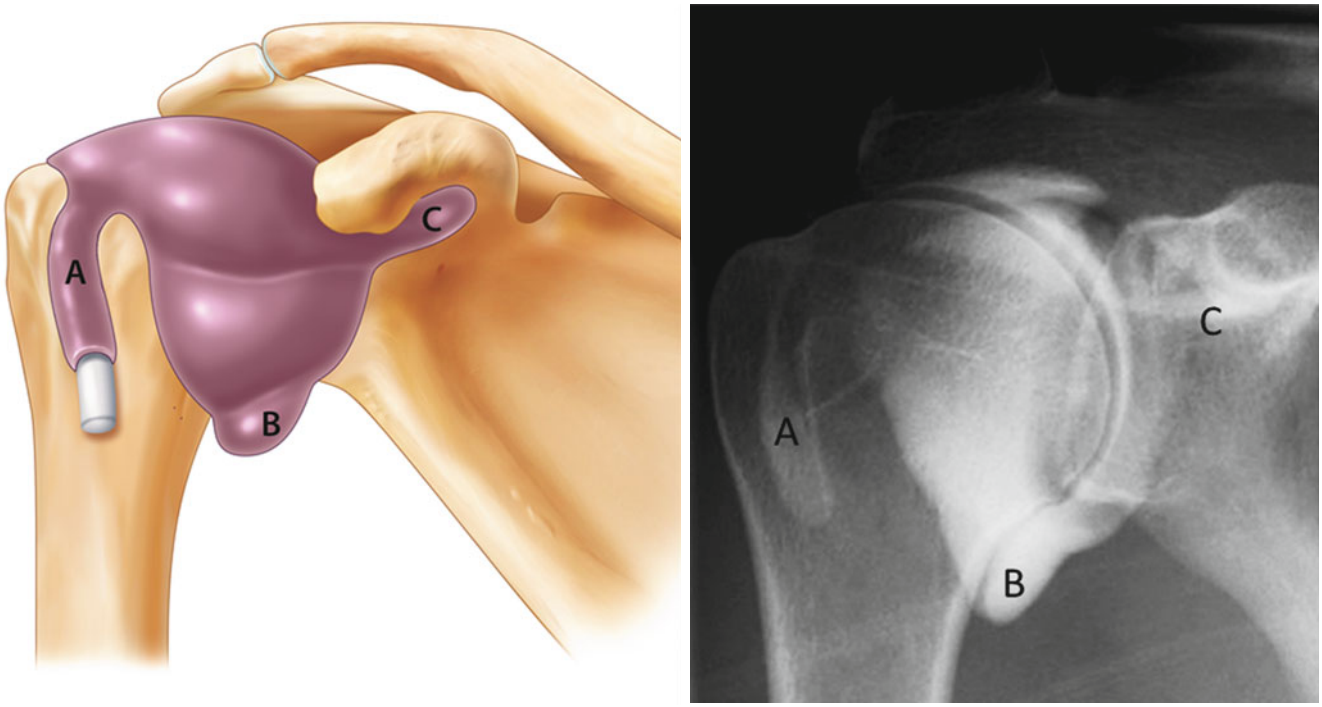


Fig. 21.2 The drawing of three main recesses of the joint (*left*)—A, the biceps tendon sheath; B, the axillary pouch; C, the subscapular recess—and the corresponding radiographic (arthrogram) appearance (*right*) (Reproduced with permission from Philip Peng Educational Series)

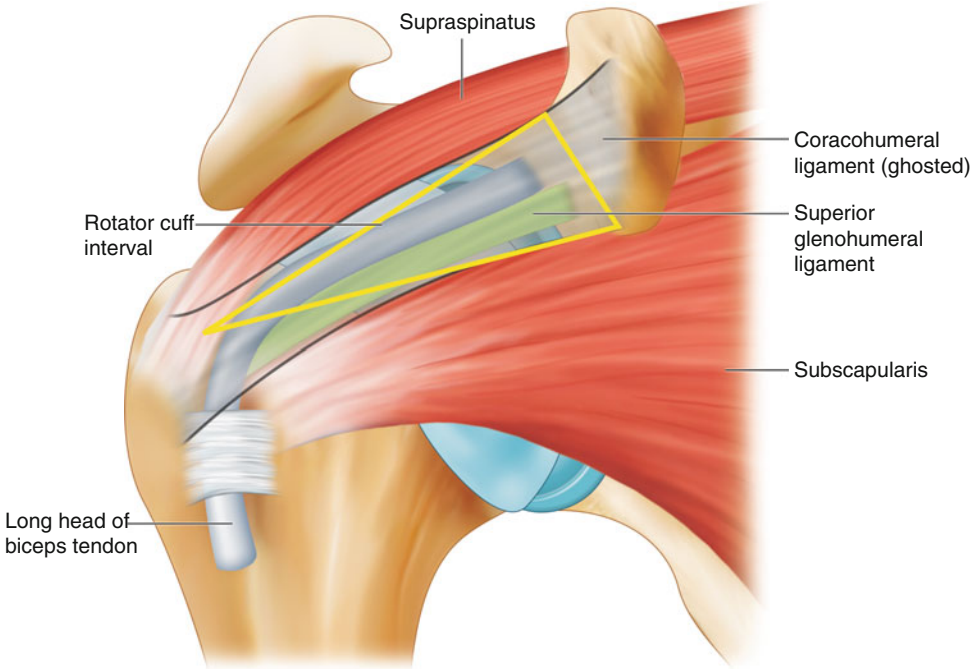


Fig. 21.3 The anterosuperior view showing the rotator cuff interval, which is a triangular space between the tendons of subscapularis (anterior) and supraspinatus (posterior) muscles, and the base of the coracoid process. The roof is the coracohumeral ligament (ghosted), and the contents are the long head of biceps tendon (*blue*) and superior glenohumeral ligament (*green*) (Reproduced with permission from Philip Peng Educational Series)

Sonoanatomy

In general, there are three approaches to access the GHJ: anterior, posterior, and rotator cuff interval [1–2]. Only the posterior approach and rotator cuff interval approach are described here.

For the posterior approach, the patient is placed in either sitting position or lateral decubitus position with the injection side as the nondependent side. In both cases, the patient's hand is placed on the contralateral shoulder to open up the GHJ space. A high-frequency linear probe (6–13 MHz) is used unless the patient is of very high BMI or very muscular. The lateral end of the probe is placed just caudal to the acromion over the infraspinatus tendon. This scan should reveal the glenoid process, labrum, capsule, humeral head, and infraspinatus muscle and tendon (Figs. 21.4 and 21.5).

To approach the rotator cuff interval, the patient is placed either in sitting or supine position. A high-frequency linear probe (6–13 MHz) is initially placed over the LHB tendon in the bicipital groove. The LHB is traced in cephalad and medial direction until it is seen in between the supraspinatus and subscapularis muscle underneath the coracohumeral ligament (CHL). At this interval, the LHB tendon is seen as a hyperechoic oval structure (Fig. 21.6).

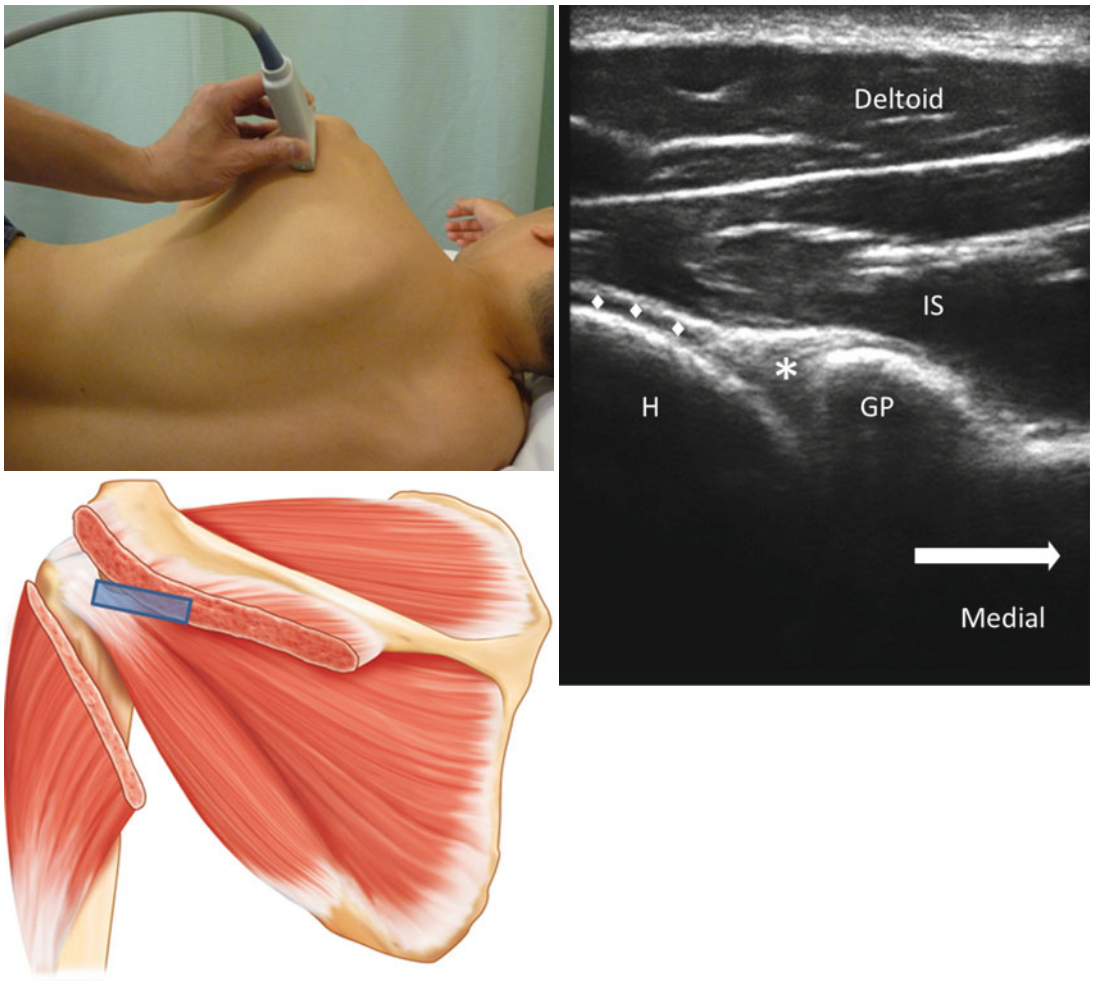


Fig. 21.4 Ultrasound image of the posterior glenohumeral joint. The glenoid process and humeral head both appear as hyperechoic structures with anechoic shadow. The insert on the top shows the position of the patient and the ultrasound probe while one below shows the probe position and the structures underneath. *IS* infraspinatus muscle, *H* humeral head, *GP* glenoid process, * glenoid labrum, ◆ the articular cartilage of the humeral head (Reproduced with permission from Philip Peng Educational Series)

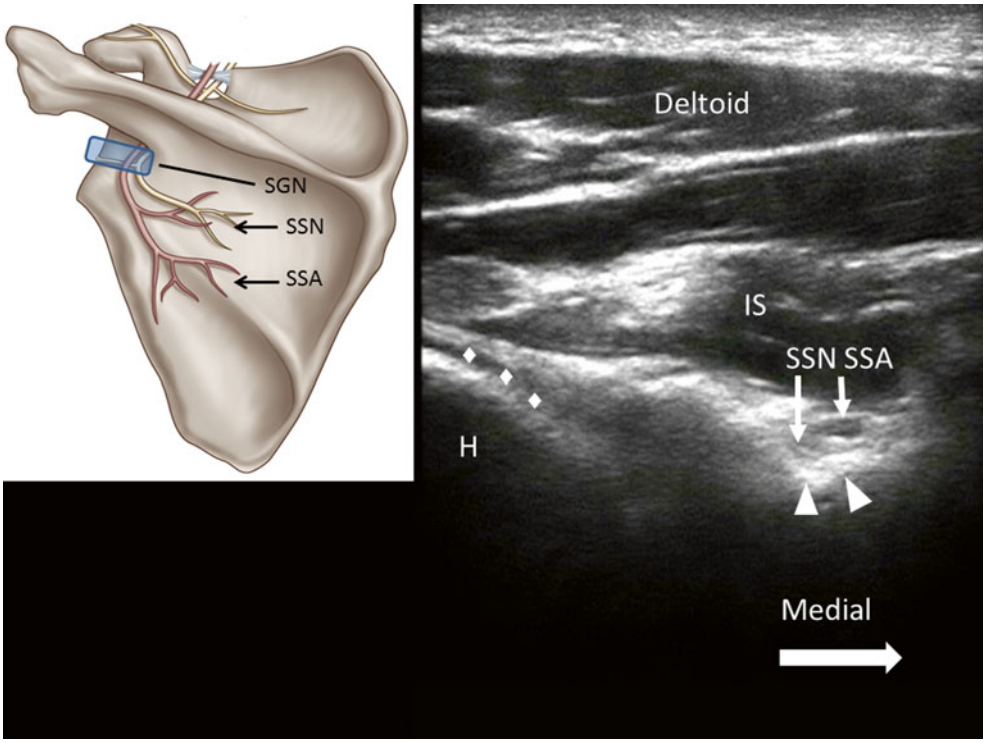


Fig. 21.5 Ultrasound image of the spinoglenoid notch by moving the ultrasound probe slightly medially compared with the probe position in Fig. 21.4. The insert shows the position of the probe and the spinoglenoid notch, as well as the suprascapular neurovascular bundle. SSN and SSA, suprascapular nerve and artery (*line arrows* in ultrasound image); SGN, spinoglenoid notch (*arrow heads*) (Reproduced with permission from Philip Peng Educational Series)

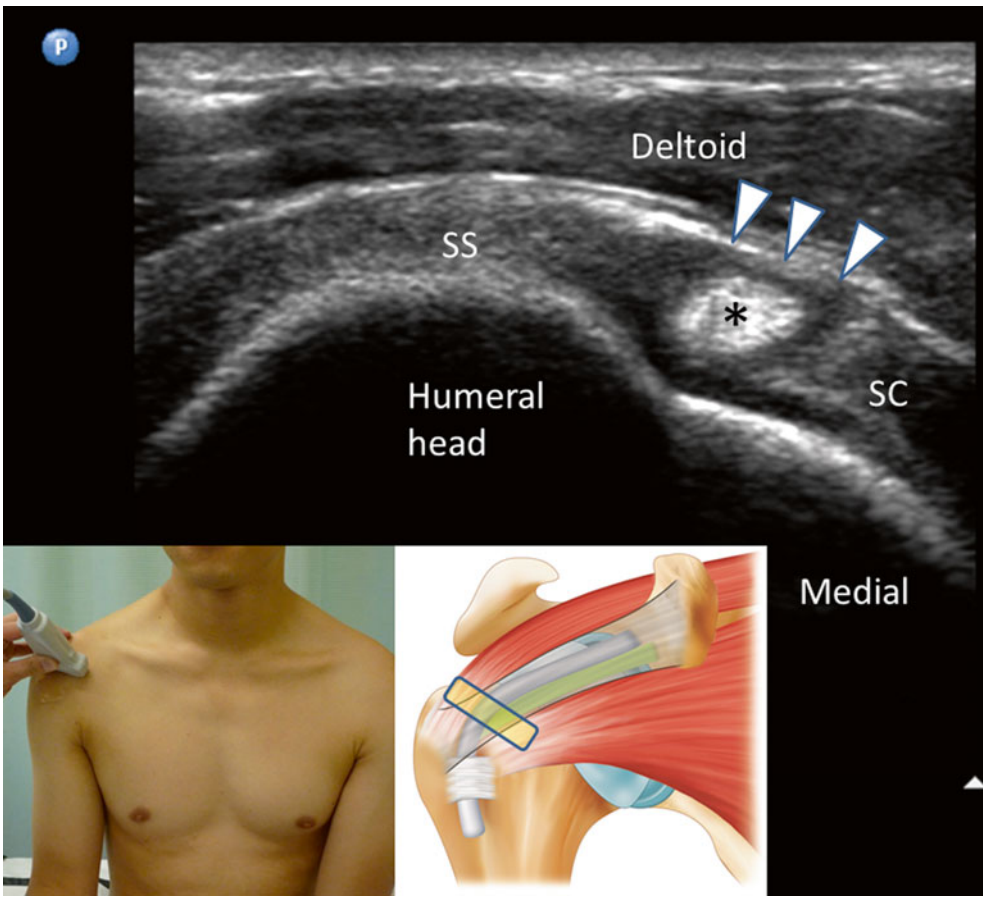


Fig. 21.6 Ultrasound image showing the presence of long head of bicep (LHB) tendon (*asterisk*) within the rotator cuff interval. This image is obtained by moving the ultrasound probe proximal to the bicipital groove along the orientation of the LGB tendon. The LHB tendon (*asterisk*) is always hyperechoic at this level and sandwiched between the supraspinatus tendon (SS) laterally and subscapularis tendon (SC) medially. The coracohumeral ligament (*arrow heads*) forms the roof of the interval. The insert on the left shows the orientation and position of the probe, and the insert on the right shows the probe position and the structures underneath it (Reproduced with permission from Philip Peng Educational Series)

Patient Selection

The main indications for GHJ injection are adhesive capsulitis and glenohumeral arthrosis. Adhesive capsulitis (frozen shoulder) is characterized by restricted active and passive range of motion (ROM) in all directions. There are three clinical phases: freezing, frozen, and thawing [3]. In the freezing phase, the patient experiences an insidious onset of pain with decreasing shoulder ROM at the end of this period. It is then followed by the frozen phase which is characterized by progressive stiffness and reducing pain. In the final resolution or thawing phase, the range of movement gradually returns.

Glenohumeral arthrosis is characterized by progressive and irreversible articular destruction and frequent involvement of the surrounding soft tissues. Primary osteoarthritis is uncommon and most of the causes of chondral damage are secondary to trauma, instability, postsurgical arthrosis, avascular necrosis, inflammatory arthropathy, osteochondritis dissecans, chondrolysis, and iatrogenic injury.

Ultrasound-Guided Procedure

The overall success rate of GHJ injection with ultrasound guidance is much higher than with landmark guidance. One interesting study compared the accuracy of the two approaches by two groups: one group performed only the ultrasound-guided approach by a rheumatology trainee (9 months in rheumatology program with 8 sessions of musculoskeletal ultrasound training), and the other group were represented by nine rheumatology consultants with a median of 9 years of experience and they all performed the conventional approach. The accuracy rates were 63 and 40 % for the ultrasound and conventional groups, respectively, despite the sharp contrast in experience [4].

For the posterior approach, the target is the joint space between the free edge of labrum and the cartilage. A 22-G 3.5-in. spinal needle is inserted in-plane in a lateral-to-medial direction (Fig. 21.7). If there is resistance upon injection, the needle is rotated 90°. If resistance persists, the needle is then retracted for a short distance. The injectate volume is 4 mL of mixture of local anesthetic with steroid (e.g., 2 % lidocaine and 40 mg Depo-Medrol).

Rotator cuff interval approach is performed with either out-of-plane or in-plane techniques. A 1.5-in. 25-G needle is inserted in a caudal-to-cephalad direction (out-of-plane) or in a lateral-to-medial direction (in-plane) on either side of the LHB tendon (Fig. 21.8). The volume of injectate is the same as that of posterior approach.

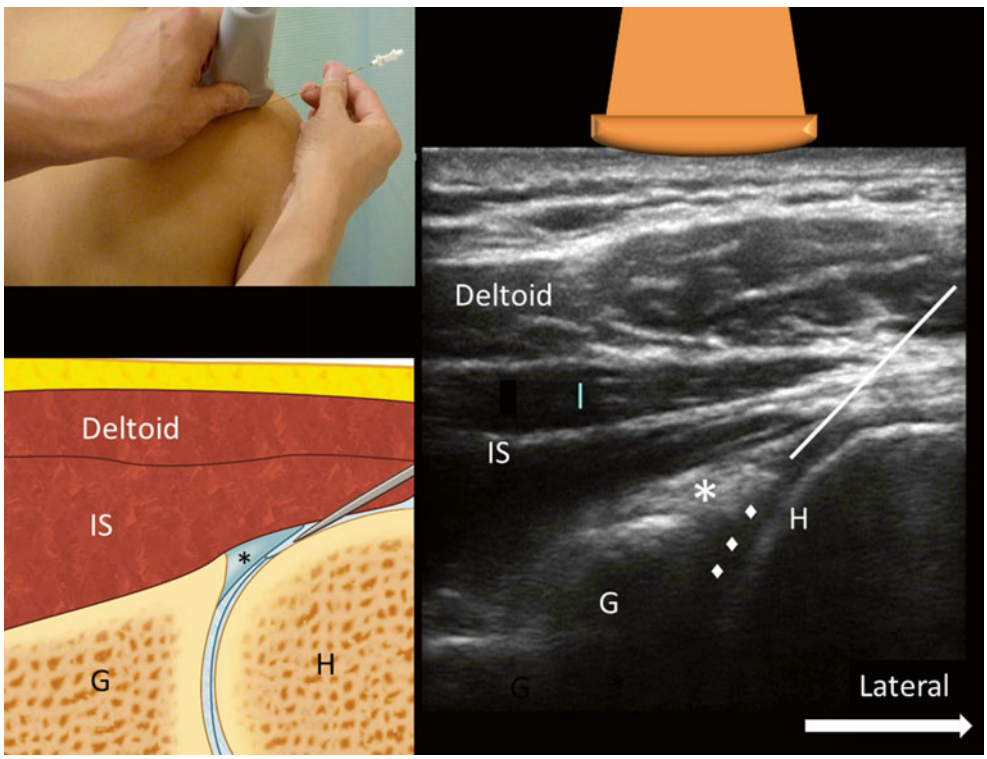


Fig. 21.7 Posterior approach to the glenohumeral joint: the insert (*left upper*) shows the position of the ultrasound probe and the needle with in-plane technique. The corresponding ultrasound image (*right*) is shown with the line representing the needle path, which was directed between the free edge of the labrum (*) and the hypoechoic articular cartilage (•) of the humeral head (H). G, glenoid; IS, infraspinatus. The insert in the lower left shows the anatomical drawing of the ultrasound image (Reproduced with permission from Philip Peng Educational Series)

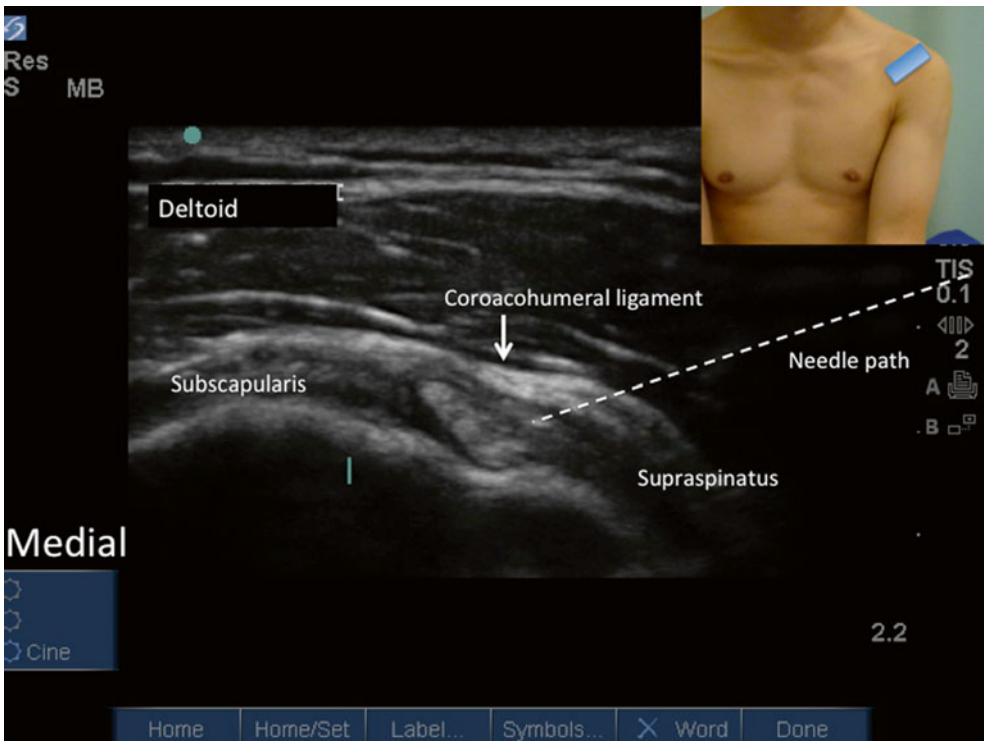


Fig. 21.8 Sonogram showing the needle path for the injection of rotator cuff interval (Reproduced with permission from Philip Peng Educational Series)

References

1. Peng PW. Shoulder. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, vol 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, California: Apple Inc.; 2014. p. 18–41.
2. Peng PWH, Cheng P. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy and procedures. Part III: shoulder. Reg Anesth Pain Med. 2011;36:592–605.
3. Tasto JP, Elias DW. Adhesive capsulitis. Sports Med Arthrosc Rev. 2007;15:216–21.
4. Cunnington J, Marshall N, Hide G, et al. A randomized, double-blind, controlled study of ultrasound-guided corticosteroid injection into the joint of patients with inflammatory arthritis. Arthritis Rheum. 2010;62:1862–9.

Chapter 22

Acromioclavicular Joint

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_22,

Anatomy

The acromioclavicular joint (ACJ) is a diarthrodial joint between the concave medial end of the acromion and the convex lateral end of the clavicle (Fig. 22.1). The articular surfaces are separated by a wedge-shaped fibrocartilaginous disc. The movement of this joint is limited, primarily rotation and translation in the anterior–posterior and the superior–inferior planes.

The ACJ is stabilized by both the static and dynamic stabilizers. The static stabilizers include the acromioclavicular ligaments (superior, inferior, anterior, and posterior), the coracoclavicular ligaments (trapezoid and conoid), and the coracoacromial ligament. The dynamic stabilizers include the deltoid and trapezius muscles (Fig. 22.2).

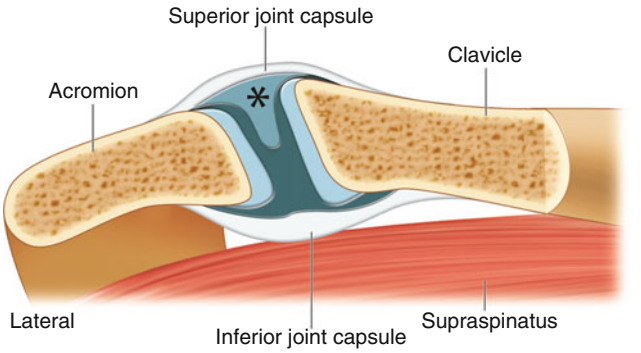


Fig. 22.1 Glenohumeral joint showing various ligaments and the joint capsule. The anterior capsule is reinforced by the superior, middle, and inferior glenohumeral ligament (Reproduced with permission from Philip Peng Educational Series)

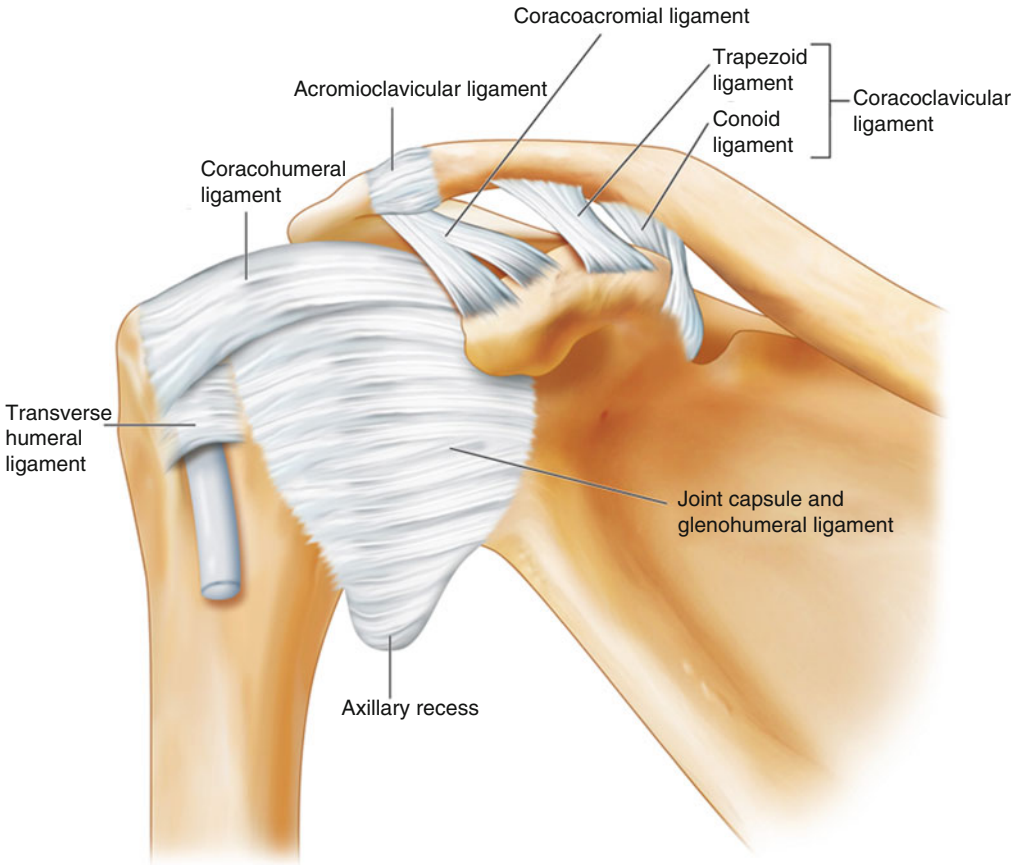


Fig. 22.2 The acromioclavicular joint is a synovial joint with the articular surfaces separated by a wedge-shaped fibrocartilaginous disc (*asterisk*). The inferior surface of the joint is in direct contact with the subacromial bursa and supraspinatus muscle and may play a role in the development of the impingement syndrome (Reproduced with permission from Philip Peng Educational Series)

Sonoanatomy

The patient is placed in sitting with arm in neutral position, in which the deep joint space of ACJ is the widest. Because of the superficial location of the joint, linear high-frequency (6–13 MHz) ultrasound probe is appropriate. The probe is initially placed over the joint in the coronal plane (medial part of probe in line with clavicle initially). The lateral end of the probe is then rotated in an anterior and posterior direction to obtain the best view of ACJ (Fig. 22.3). The sonogram should show the hyperechoic ends of acromion and distal clavicle, fibrocartilaginous disc, and superior joint capsule.

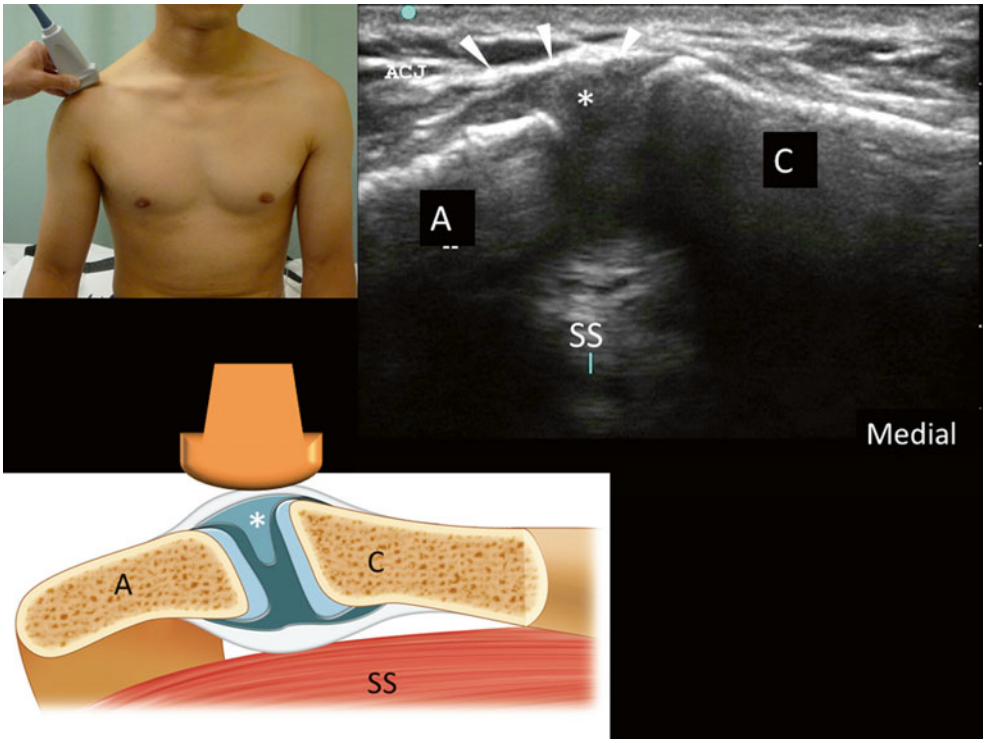


Fig. 22.3 Ultrasound image of the acromioclavicular joint. The upper insert shows the position of the probe and the patient, and the lower insert shows the position of the probe and the structures underneath. A, acromion process; C, clavicle; *, the wedge-shaped fibrocartilaginous disc; *arrowheads*, superior joint capsule; SS, supraspinatus muscle (Reproduced with permission from Philip Peng Educational Series)

Patient Selection

The ACJ injection can be used as a diagnostic or therapeutic block. It is commonly used as a diagnostic test to localize the source of pain when ACJ injury is suspected. The main therapeutic indication for ACJ injection is arthritis (post-traumatic and osteoarthritis) of this joint. Patient usually complains of anterior shoulder pain brought about by daily activities such as reaching the back pocket or unhooking a brassiere. Physical examination reveals point tenderness over ACJ and positive cross-arm adduction test.

Ultrasound-Guided Procedure

The accuracy of landmark-based or blind injection for ACJ ranges between 39 and 66 %. The accuracy did not differ significantly with different levels of experience (specialist, resident, or student). In contrast, the accuracy of ultrasound-guided injection ranged between 95 and 100 %.

Since the joint is superficial, the out-of-plane technique is preferred with a 25-G 1.5-in. needle (Fig. 22.4). The volume of injectate is 1 mL with a mixture of local anesthetic and steroid (2 % lidocaine and 20 mg Depo-Medrol). Since the joint space is narrow, one way to facilitate an easier entry is to put the needle shaft on the skin as a marker to the joint space. Once the joint space is located, the needle is then retracted and inserted into the joint. Because the joint space is very shallow, overzealous insertion of needle will deposit medication into the subacromial space.

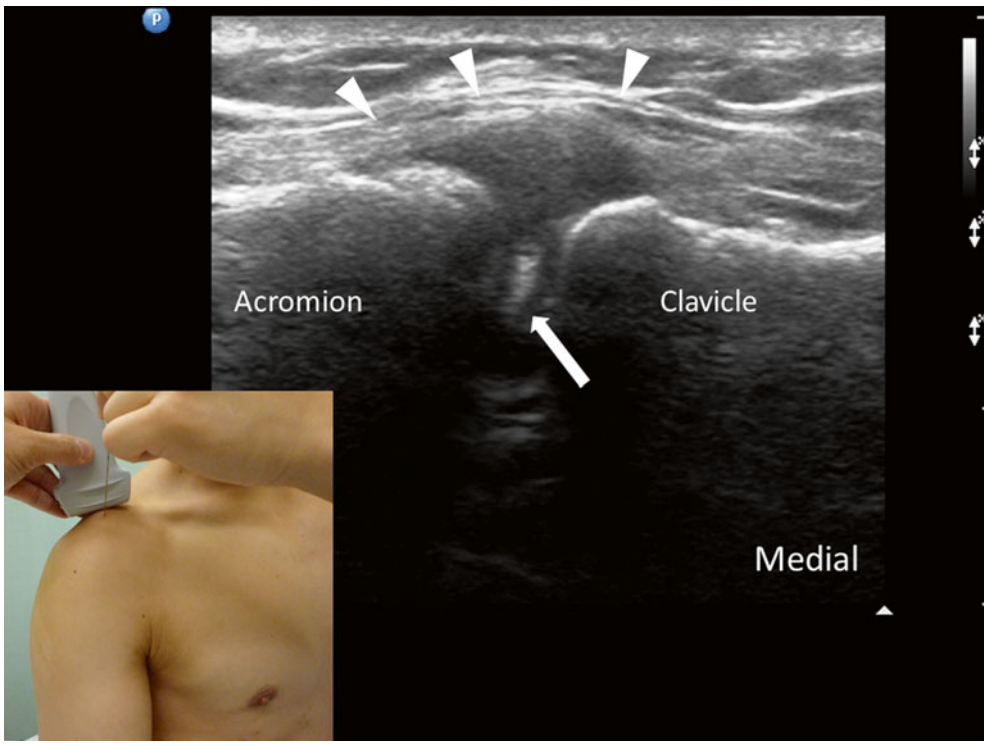


Fig. 22.4 The insert shows the position of the ultrasound probe and the needle with the out-of-plane technique. The corresponding ultrasound image shows the acromioclavicular joint with the image of the needle (*solid arrow*). The arrowheads outline the superior joint capsule (Reproduced with permission from Philip Peng Educational Series)

Suggested Reading

1. Shaffer BS. Painful conditions of the acromioclavicular joint. J Am Acad Orthop Surg. 1999;7:176–88.
2. Peng PWH, Cheng P. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy and procedures. Part III: shoulder. Reg Anesth Pain Med. 2011;36:592–605.
3. Peng PW. Shoulder. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, vol 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, California: Apple Inc.; 2014. p. 18–41.
4. Buttaci CJ, Stitik TP, Yonclas PP, Foye PM. Osteoarthritis of the acromioclavicular joint: a review of anatomy, biomechanics, diagnosis, and treatment. Am J Phys Med Rehabil. 2004;83:791–7.
5. Peck E, Lai JK, Pawlina W, Smith J. Accuracy of ultrasound-guided versus palpation-guided acromioclavicular joint injections: a cadaveric study. PM R. 2010;2:817–21.
6. Sabeti-Aschraf M, Lemmerhofer B, Lang S, et al. Ultrasound guidance improves the accuracy of the acromioclavicular joint infiltration: a prospective randomized study. Knee Surg Sports Traumatol Arthrosc. 2011;19:292–5.
7. Hossain S, Jacobs LGH, Hashmi R. The long-term effectiveness of steroid injections in primary acromioclavicular joint arthritis: a five-year prospective study. J Shoulder Elbow Surg. 2008;17:535–8.
8. van Riet RP, Goehre T, Bell SN. The long term effect of an intra-articular injection of corticosteroids in the acromioclavicular joint. J Shoulder Elbow Surg. 2012;21:376–9.

Chapter 23

Glenohumeral and Acromioclavicular Joint Injection Traditional Techniques

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_23,

Glenohumeral Joint Injection [1–4]

Indications

Synovial inflammatory conditions (capsulitis), severe resting pain, humeroscapular periarthritis, after shoulder bruising, and rheumatoid arthritis. The anatomy of the shoulder joint is shown in Fig. 23.1a, b.

> Strictly aseptic conditions are necessary when carrying out intra-articular injections.

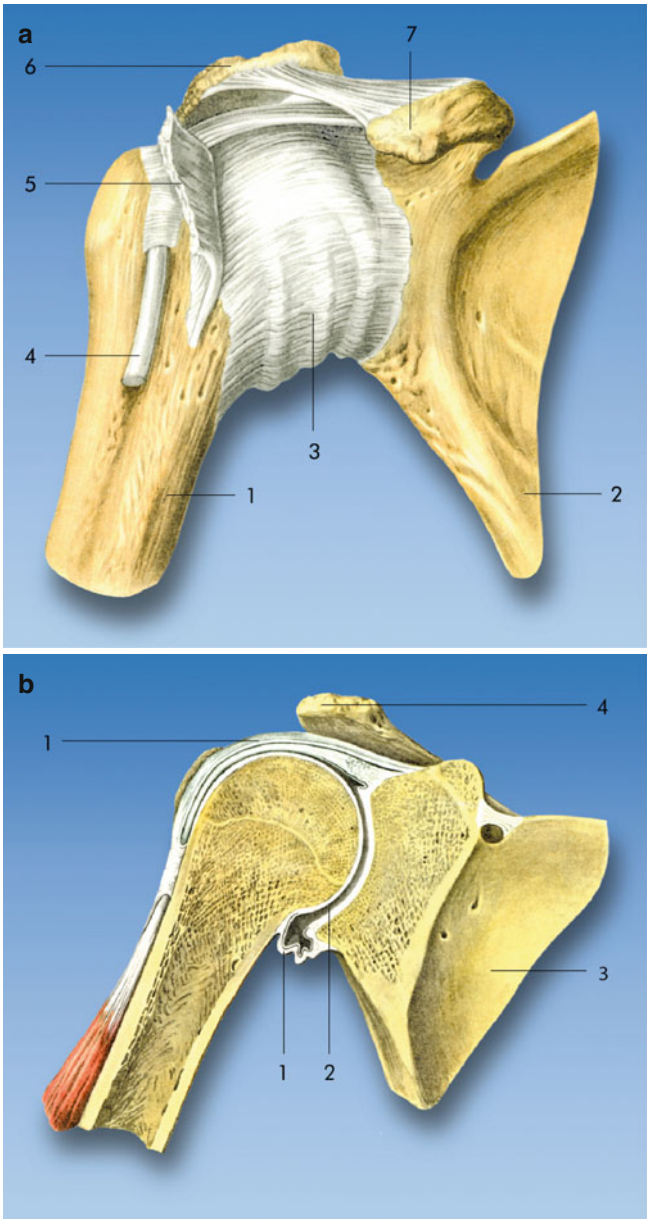


Fig. 23.1 (**a**) Anatomy of the shoulder joint. (*1*) Humerus, (*2*) scapula, (*3*) articular capsule, (*4*) tendon of the biceps brachii muscle, (*5*) subscapular muscle, (*6*) acromion, (*7*) coracoid process (With permission from Danilo Jankovic). (**b**) Shoulder joint. Articular cavity and articular capsule. (*1*) Articular capsule, (*2*) articular cavity, (*3*) scapula, (*4*) acromion (With permission from Danilo Jankovic)

Materials

25-G needle 30–40 mm long, 2- and 5-mL syringes, sterile swabs, disinfectant, sterile gloves, and sterile drape.

Ventral Access Route

Landmarks (Fig. 23.1a, b)

- Coracoid process
- Head of the humerus
- Clavicle

Technique

The patient is seated with the supinated arm hanging freely, and the articular cavity is palpated directly medial to the head of the humerus. The needle is introduced underneath the clavicle, directly lateral to the coracoid process toward the outside and back. The path to the joint is very short with this approach (Fig. 23.2).

Dosage

2 mL local anesthetic—e.g., 0.5–0.75 % ropivacaine or 0.25 % bupivacaine mixed with 40 mg methylprednisolone.

Dorsal Access Route

Landmarks (Fig. 23.1a, b)

- Spine of the scapula
- Lateral corner of the acromion
- Coracoid process

Technique

The patient is seated, with the upper arm slightly abducted and rotated inward, and the lateral corner of the acromion is palpated. The injection is made directly underneath this point and the needle is advanced between the posterior edge of the deltoid muscle and the tendon of the infraspinatus muscle (the muscle's dorsolateral tendon) in the direction of the coracoid process (Fig. 23.3). The articular cavity is reached after approximately 3–4 cm. After injection into the joint, a further 1 mL is distributed circumarticularly as the needle is withdrawn.

Dosage

2 mL local anesthetic—e.g., 0.5–0.75 % ropivacaine or 0.25 % bupivacaine mixed with 40 mg methylprednisolone.

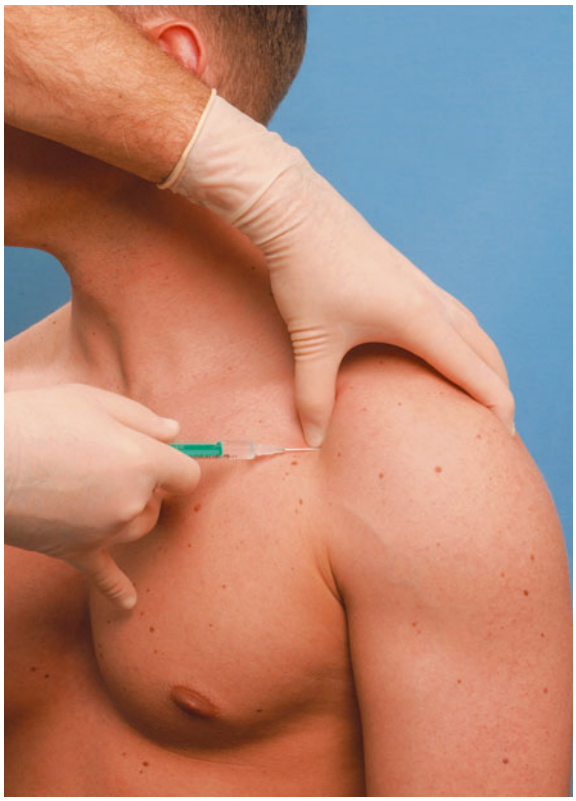


Fig. 23.2 Intra-articular injection into the shoulder joint from the ventral access route (With permission from Danilo Jankovic)

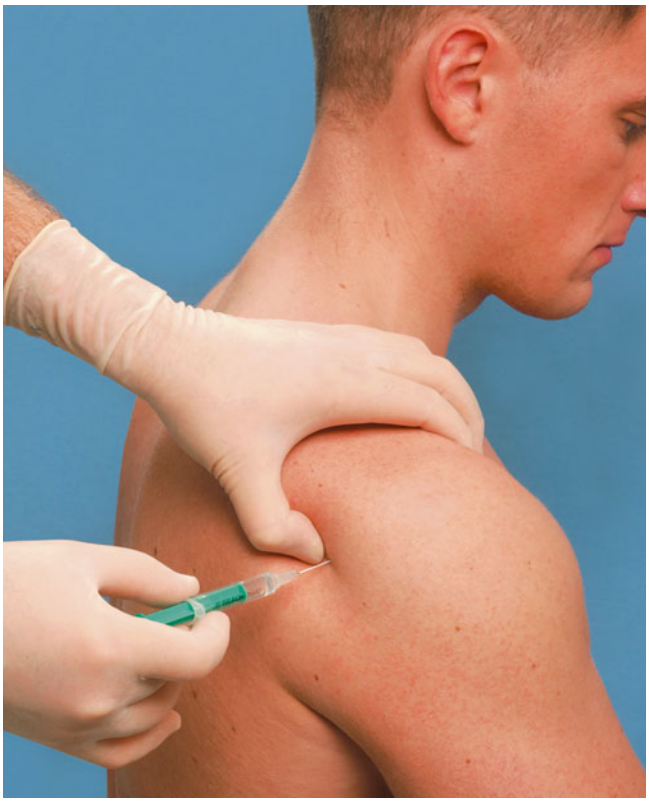


Fig. 23.3 Intra-articular injection into the shoulder joint from the dorsal access route (With permission from Danilo Jankovic)

Acromioclavicular Joint Injection [1–4]

Indications

Shoulder pain radiating to behind the ear and restricted mobility in the shoulder joint.

Landmarks

- Lateral edge of the clavicle
- Acromion
- Acromioclavicular ligament (Fig. 23.4)

The acromioclavicular joint has a very small volume, so that only a small amount of the injection solution is needed.

Injection Technique

The patient is seated, and the articular cavity between the lateral end of the clavicle and the acromion is palpated. The needle is advanced perpendicularly from above through the acromioclavicular ligament to a maximum depth of 1 cm. Provided there is no resistance, a small amount of the injection solution is injected (Fig. 23.5).

Dosage

0.5–1 mL local anesthetic—e.g., 0.5–0.75 % ropivacaine or 0.25 % bupivacaine mixed with 40 mg methylprednisolone or triamcinolone.

Intra-articular Shoulder Joint Injection

Side Effects

Some 25 % of patients report a transient increase in pain after an intra-articular injection in the shoulder joint. The patient should be advised of this potential side effect.

Complications

- Infection
- Hematoma (prophylactic compression should be carried out after the injection)

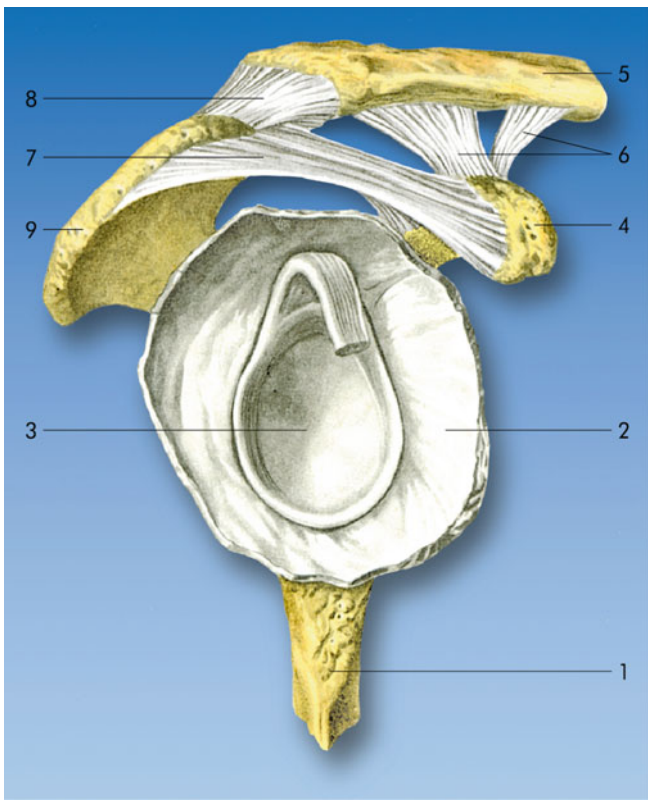


Fig. 23.4 Anatomy of the acromioclavicular joint and neighboring structures. (*1*) Scapula, (*2*) articular capsule, (*3*) glenoid cavity, (*4*) coracoid process, (*5*) clavicle, (*6*) coracoclavicular ligament, (*7*) coracoacromial ligament, (*8*) acromioclavicular joint, (*9*) acromion (With permission from Danilo Jankovic)

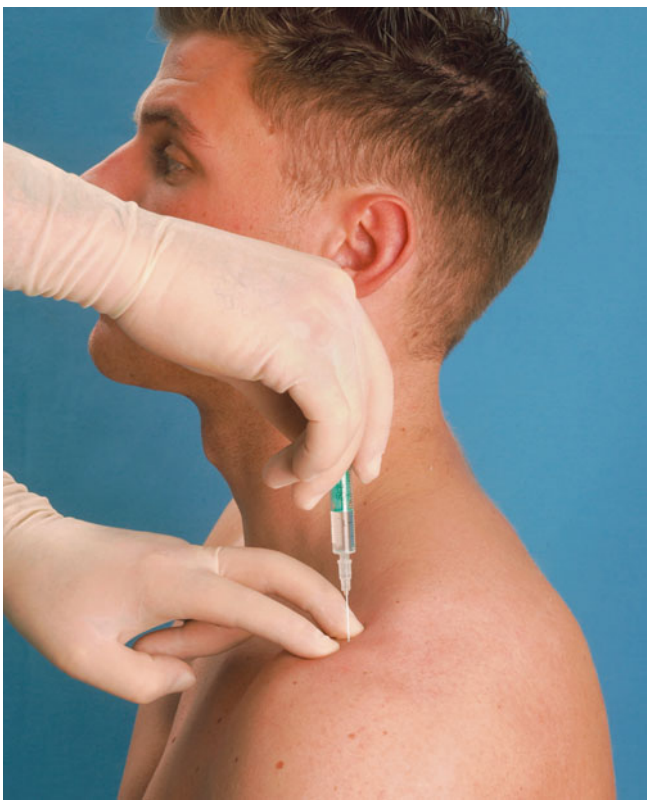


Fig. 23.5 Intra-articular injection into the acromioclavicular joint (With permission from Danilo Jankovic)

References

1. Jankovic D. Shoulder joint-intraarticular injections. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Text book and color atlas. Blackwell: Malden Massachusetts, Oxford, Carlton Victoria; 2004. p. 138–40.
2. Tilscher H, Eder M. Infiltrationstherapie. Therapeutische Lokal Anästhesie. 3rd ed. Tilscher/Eder:Hippocrates Verlag, Stuttgart; 1999. p. 84–5.
3. Waldmann S. Pain management injection techniques. Waldmann/ WB Saunders Company. Philadelphia, London, New York, St. Louis, Sydney, Toronto; 2000.
4. Hatz H. Lokale intraartikuläre Diagnostik und Therapie. (2 Auflage). Uni Med- Verlag AG. Bremen, London, Boston; 2005.114–122.

Chapter 24

Subacromial Subdeltoid Bursa

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_24,

Anatomy

The subacromial subdeltoid bursa (SASDB) lies beneath the acromion, the deltoid muscle, and the coracoacromial ligament (CAL), covering the superior aspect of the supraspinatus tendon, bicipital groove, and coracoid process (subcoracoid bursa) and reaching as lateral as 3 cm below the greater tuberosity. It is the largest bursa in the body. The main role is to minimize attrition of the supraspinatus tendon against the coracoacromial arch (acromion and CAL) and the deltoid muscle during movements of the arm.

Supraspinatus (SS) muscle originates from the suprascapular fossa of the scapula, passes beneath the acromion and CAL, and inserts on the upper facet of the greater tuberosity. It is one of the 4 rotator cuff muscles. Others are subscapularis (SSC), infraspinatus (IS), and teres minor (TMi) muscles. The rotator cuff is a tight layer of tendons around the glenohumeral joint on the anterior (SSC), superior (SS), and posterior (IS and TMi) aspects of the shoulder (Figs. 24.1, 24.2 and 24.3). They play an important role in stabilizing the humeral head in the shallow glenoid fossa during the movement of the arm.

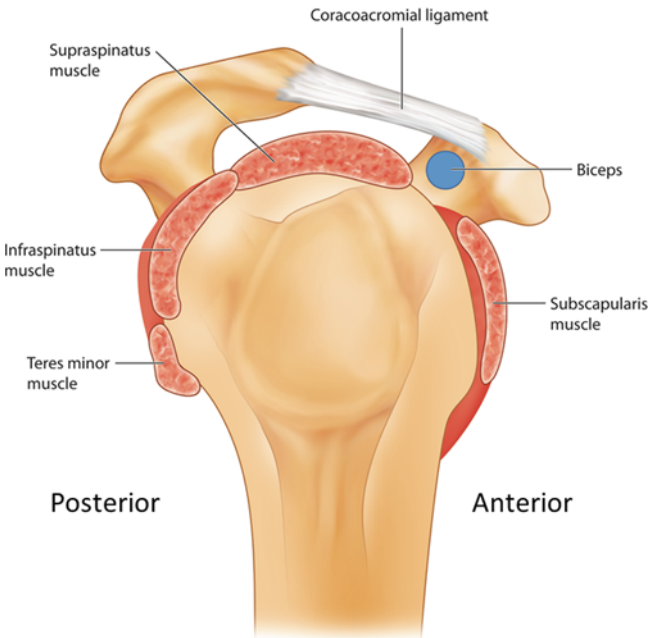


Fig. 24.1 A schematic diagram showing the arrangement of the four rotator cuff muscles: subscapularis, supraspinatus, infraspinatus, and teres minor (Reproduced with permission from Philip Peng Educational Series)

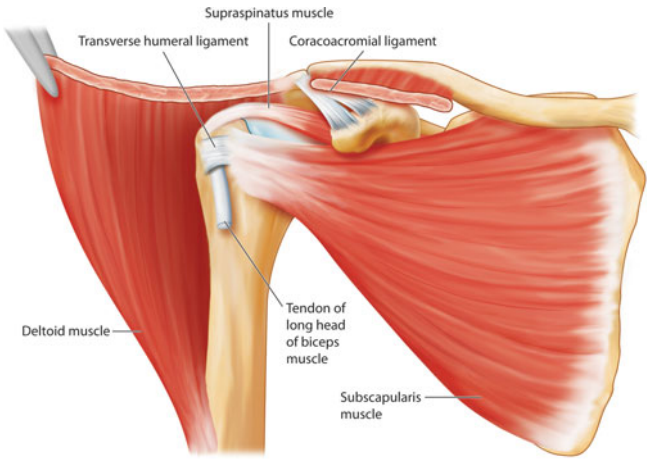


Fig. 24.2 Anterior view of the shoulder showing the subscapularis and supraspinatus muscles. The anterior portion of the deltoid muscle was reflected to show the underlying rotator cuff muscle (Reproduced with permission from Philip Peng Educational Series)

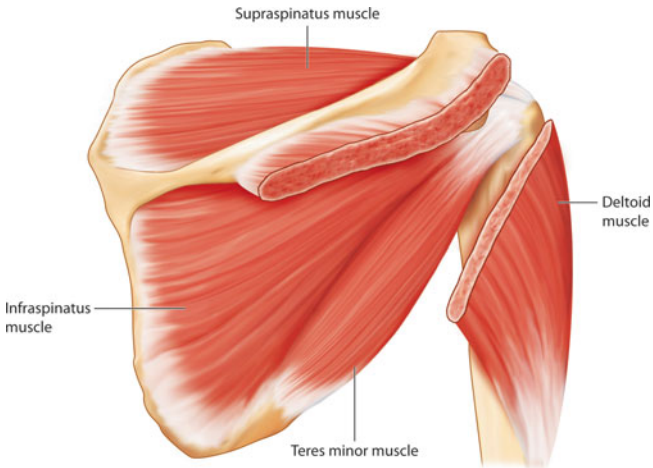


Fig. 24.3 Posterior view of the shoulder to show the infraspinatus and teres minor muscle. The posterior portion of the deltoid muscle was partially removed to show the underlying muscle (Reproduced with permission from Philip Peng Educational Series)

Sonoanatomy

The patient is examined in sitting position or semi-inclined position on an examination bed with the shoulder over the edge of the bed. The hand is put in modified Crass position (similar to the posture "putting the money back in the back pocket"). This maneuver allows a longer segment of supraspinatus tendon to be scanned. A high-frequency (6–13 MHz) linear ultrasound probe is placed over the SS tendon. A good reference point is the long head of biceps (LHB) tendon. The long-axis view of the SS tendon is almost parallel to the LHB tendon, and the SS tendon should be within 1.5 cm from the LHB tendon. Further away from the LHB tendon is the intercalated zone between the supraspinatus and infraspinatus tendon. To examine the presence of pathology of SS tendon, both the long- and short-axis scans are required. The SASDB appears as a hypoechoic space between the deltoid muscle and the SS tendon (Figs. 24.3, 24.4, and 24.5).

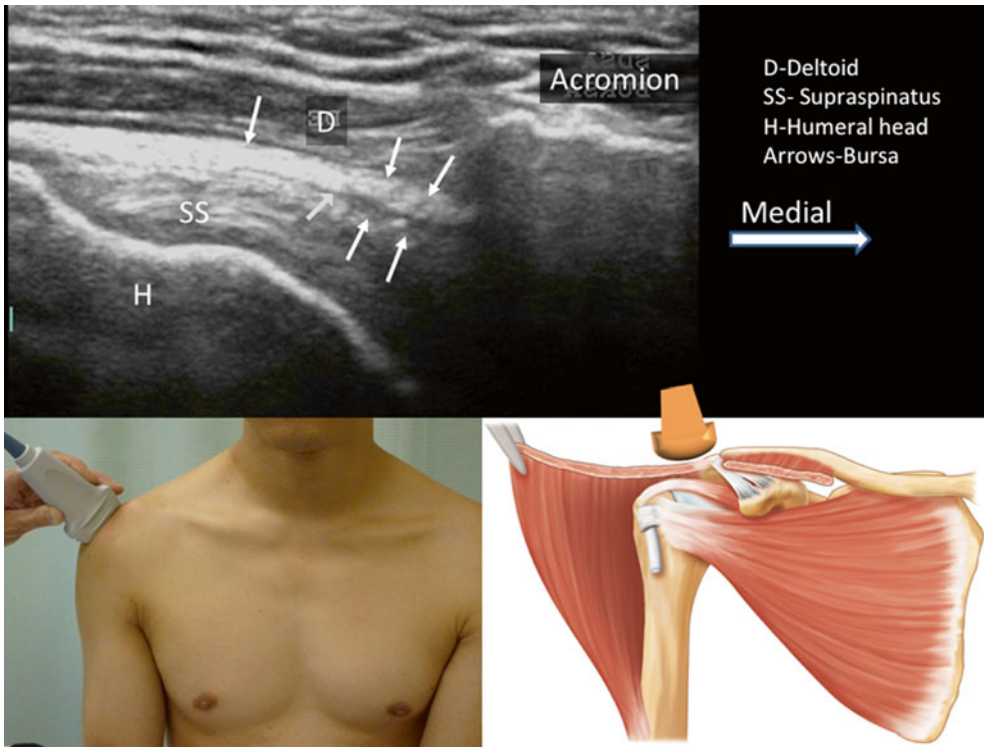


Fig. 24.4 Ultrasound image of the subacromial subdeltoid bursa (SASDB). The supraspinatus tendon (*SS*) was seen attached laterally onto the greater tuberosity of the humeral head (*H*). The insert on the left shows the position of the patient and the ultrasound probe and the one on the right shows the probe and the structures underneath. The deltoid muscle was reflected to show the underlying supraspinatus muscle (Reproduced with permission from Philip Peng Educational Series)

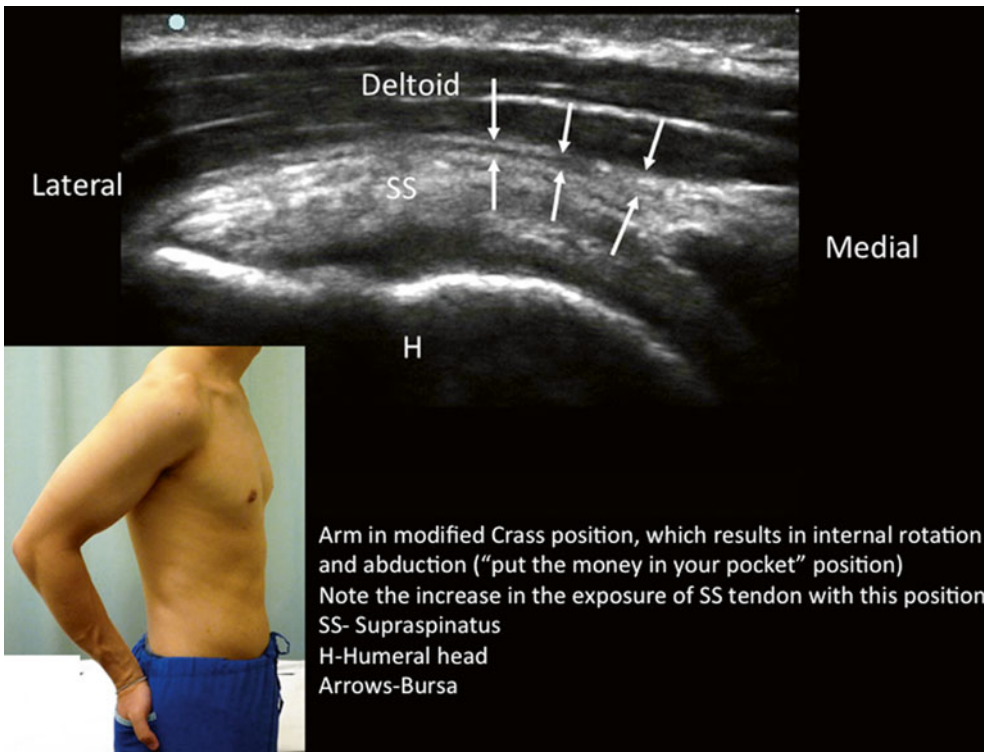


Fig. 24.5 Ultrasound image of the supraspinatus tendon when the arm is put in the modified Crass position. Note that the portion of the supraspinatus tendon lateral to the acromion process was significantly increased by this maneuver. The insert shows the position of the modified Crass position. *SS* supraspinatus tendon, *H* humeral head, *D* deltoid muscle. *Line arrows* outline the peribursal fat of the SASDB (Reproduced with permission from Philip Peng Educational Series)

Patient Selection

The indication for SASDB injection is subacromial impingement syndrome, which covers a constellation of conditions: partial-thickness and full-thickness rotator cuff tear and rotator cuff tendinopathy. The main presentation symptoms are pain and stiffness. Physical examination shows a painful arc and demonstrates positive signs in a few provocative tests: Hawkins' impingement test, Neer's test, and empty can supraspinatus test.

Ultrasound-Guided Procedure

The SASDB is usually a tight space. The literature showed that the success rates in most landmark-based injection clinical studies ranged from 29 to 70 % and were similar irrespective of different approaches and experience and confidence of the practitioners. In studies where the blind procedures were performed by very experienced orthopedic surgeons and shoulder specialists, the confidence correlation (the accuracy rate when the practitioners were very confident that they were accurate) ranged from 42 to 66 %.

In performing the SASDB injection, the target is the bursa outlined by peribursal fat. After obtaining a long-axis or short-axis view, either a 25-G 1.5-in. needle or 22-G 3.5-in. spinal needle is inserted into SASDB. If the needle tip is in the correct space, injection of a small volume (0.5 mL) of injectate will result in a rapid spread along the SASDB space (Fig. 24.6). The injectate volume is 4 mL of local anesthetic with steroid (0.25 % bupivacaine and 40 mg Depo-Medrol)

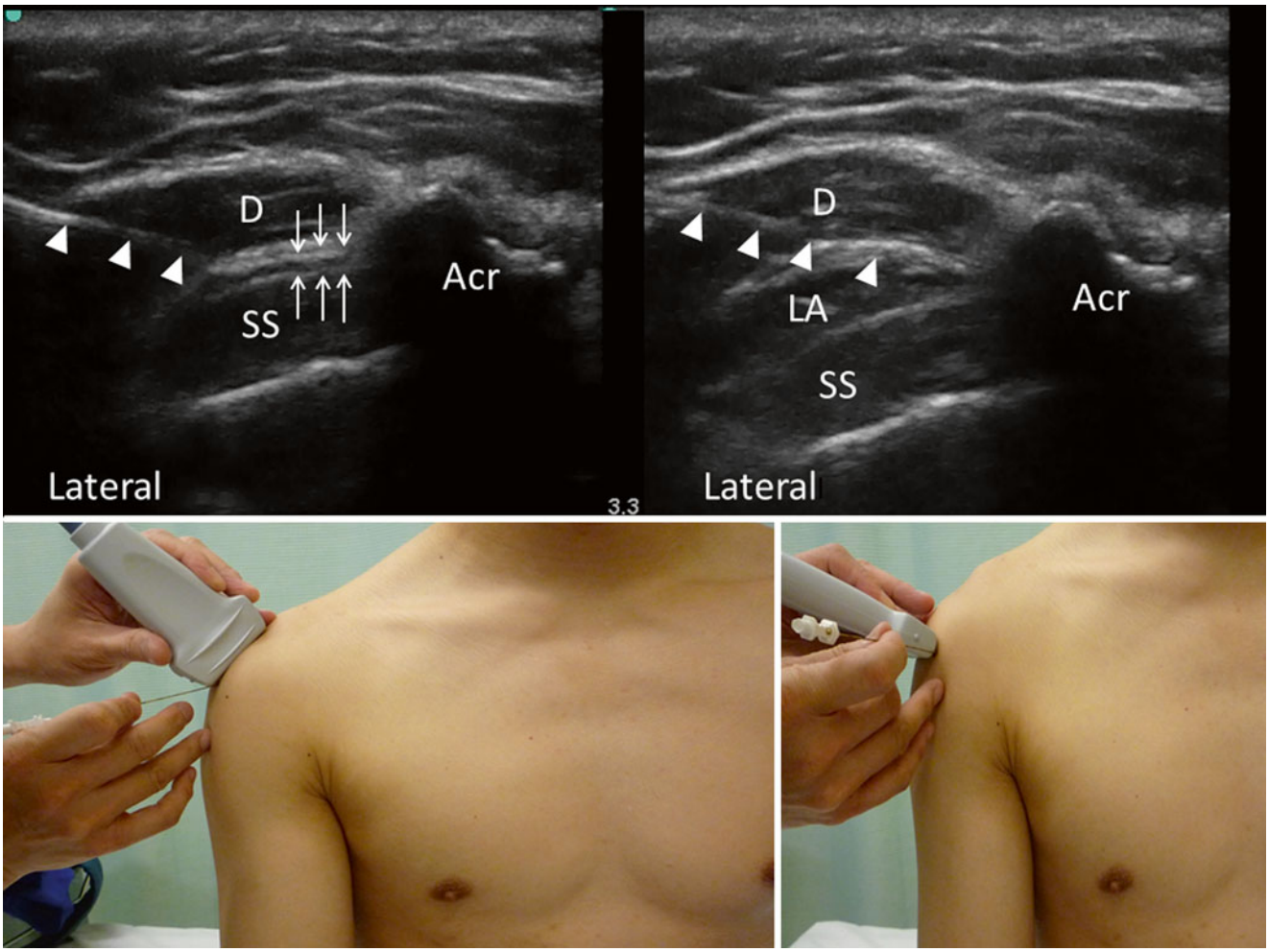


Fig. 24.6 Ultrasound images of subacromial subdeltoid bursa (SASDB) injection. The top-left figure shows the needle (*arrowheads*) inserted with the in-plane technique to the SASDB, as highlighted by the peribursal fat (*line arrows*). The image on the top right shows the presence of local anesthetic (*LA*) following the injection, with the separation of the deltoid muscle (*D*) and supraspinatus tendon (*SS*). The insert on the bottom left shows the position of the ultrasound probe and the needle with the in-plane technique. Note that the medial end of the ultrasound probe is placed over the acromion (*Acr*). However, in a subject with slim body built, the probe can be placed in another orientation as shown in the bottom-right insert (Reproduced with permission from Philip Peng Educational Series)

Suggested Reading

1. Peng PWH, Cheng P. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy and procedures. Part III: shoulder. Reg Anesth Pain Med. 2011;36:592–605.
2. Peng PW. Shoulder. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, vol 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, California: Apple Inc.; 2014. p. 18–41.
3. Gruson KI, Ruchelsman DE, Zuckerman JD. Subacromial corticosteroid injections. J Shoulder Elbow Surg. 2008;17:118S–30.
4. Burbank KM, Stevenson JH, Czarnecki GR, Dorfman J. Chronic shoulder pain: part I. Evaluation and diagnosis. Am Fam Physician. 2008;77:453–60.
5. Henkus HE, Cobben LP, Coerkamp EG, Nelissen RG, van Arkel ER. The accuracy of subacromial injections: a prospective randomized magnetic resonance. imaging study. Arthroscopy. 2006;22:277–82.
6. Iannotti JP, Ciccone J, Buss DD, et al. Accuracy of office-based ultrasonography of the shoulder for the diagnosis of rotator cuff tears. J Bone Joint Surg Am. 2005;87:1305–11.
7. Crawshaw DP, Helliwell PS, Hensor EMA, Hay EM, Aldous SJ, Conaghan PG. Exercise therapy after corticosteroid injection for moderate to severe shoulder pain: a large pragmatic randomised trial. BMJ. 2010;340:c3037.

Chapter 25

Long Head of Biceps Tendon

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_25,

Anatomy

The long head of biceps (LHB) tendon arises from the supraglenoid tubercle and the superior labrum. This part of LHB is intra-articular and extrasynovial. The tendon exits the joint within the bicipital groove, covered by the transverse humeral ligament and accompanied by the ascending branch of the anterior circumflex artery (Fig. 25.1).

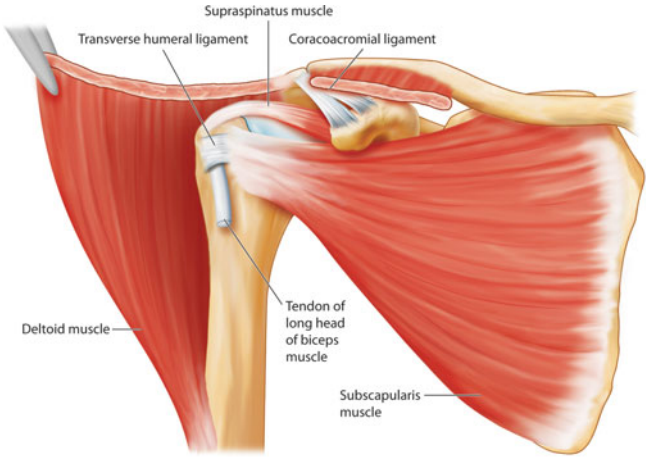


Fig. 25.1 Diagram showing the long head of biceps and its relationship with supraspinatus and subscapularis muscles (Reproduced with permission from Philip Peng Educational Series)

Sonoanatomy

The patient is examined in sitting position with hand supinate and arm in slight external rotation. A high-frequency (6–13 MHz) linear probe is placed over the bicipital groove halfway between the clavicle and anterior axillary fold (Fig. 25.2). The biceps tendon appears as a hyperechoic or hypoechoic structure depending on the tilting angle of the probe (anisotropic effects) (Fig. 25.3). With light pressure on the probe, Doppler scan may reveal the ascending branch of the anterior circumflex artery (Fig. 25.4).

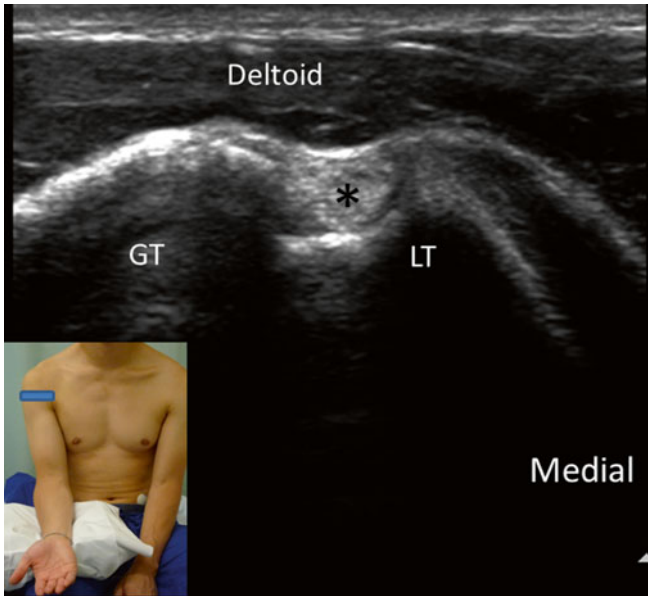


Fig. 25.2 Sonogram of the long head of biceps (*) in the bicipital groove. Note that the tendon appears hyperechoic. The insert shows the position of the patient and the ultrasound probe. *LT* lesser tubercle, *GT* greater tubercle (Reproduced with permission from Philip Peng Educational Series)

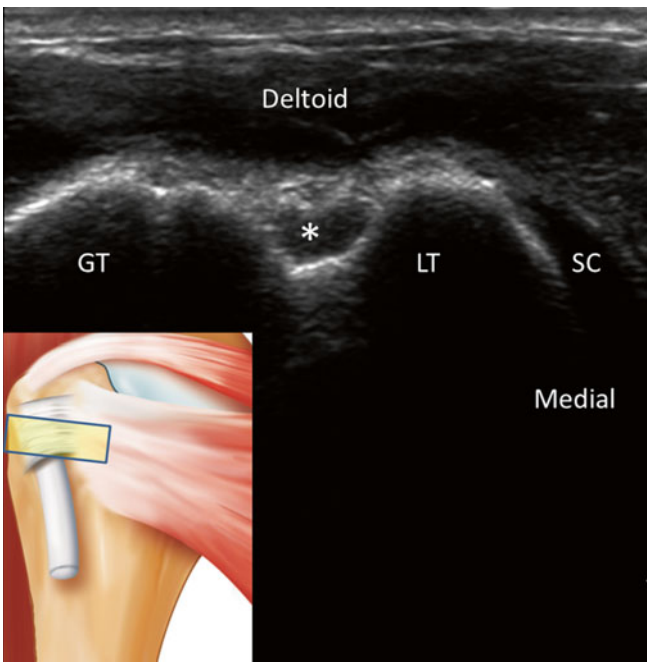


Fig. 25.3 Sonogram similar to Fig. 25.2 except a different tilt of the ultrasound probe. As a result, the long head of biceps (*) look hypoechoic. *SC* subscapularis (Reproduced with permission from Philip Peng Educational Series)

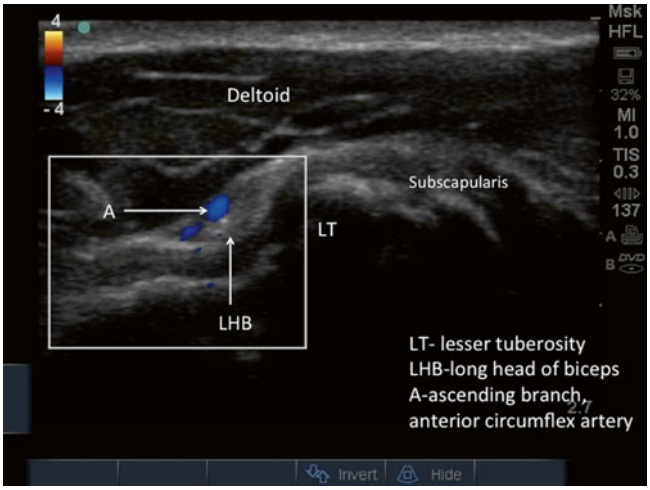


Fig. 25.4 Doppler scan of the long head of biceps which shows the ascending branch of anterior circumflex artery (Reproduced with permission from Philip Peng Educational Series)

Patient Selection

Primary biceps tendinitis is uncommon. Usually it is associated with other shoulder pathology. The main indication for the peritendon injection of the LHB tendon is biceps tendinopathy, which refers to a spectrum of pathology ranging from inflammatory tendinitis and tenosynovitis to degenerative tendinosis. Patient with biceps tendinitis presents with anterior shoulder pain. Tenderness in the bicipital grove is the most common finding. Different provocative tests (Speed test, Yergason test) have been described but are limited by the specificity and sensitivity.

Ultrasound-Guided Procedure

Validation study comparing ultrasound guided with blinded approach showed a definite superiority of the ultrasound-guided technique. All ultrasound-guided injections in this study reached the tendon sheath, while 33 % of blind injections did not reach tendon sheath at all. A randomized prospective trial showed that correct placement of the injectate results in better outcome.

In performing the procedure, it is important to reveal the artery with the Doppler scan. In some case, the Doppler scan reveals the increase vascularity of the tendon due to the inflammation. A 1.5-in. 25-G needle is inserted with out-of-plane technique with 2–3 mL of mixture of local anesthetic with steroid (0.25 % bupivacaine and 20 mg depoMedrol). Attention should be paid to make sure that there is a spread of injectate around the tendon (Fig. 25.5). Absence of those may suggest intratendinous or intravascular injection.

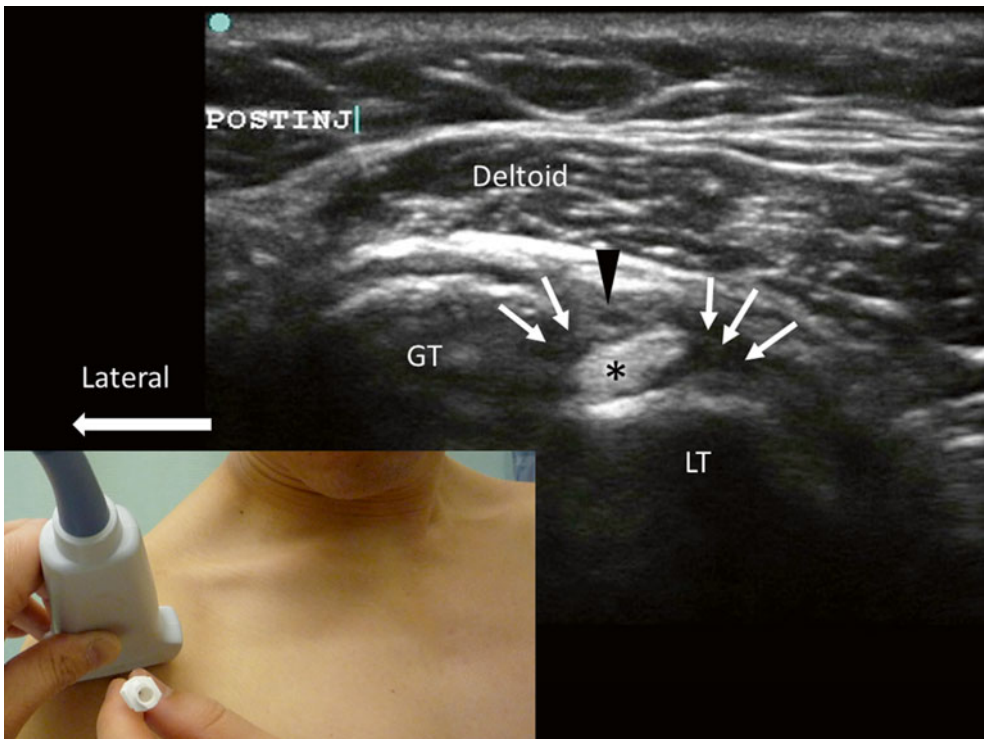


Fig. 25.5 Sonogram of the long head of biceps (*) following the injection. The arrows show the pooling of the injectate around the tendon. The insert shows the needle position for injection. *LT* lesser tubercle, *GT* greater tubercle (Reproduced with permission from Philip Peng Educational Series)

Suggested Reading

1. Peng PWH, Cheng P. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy and procedures. Part III: shoulder. Reg Anesth Pain Med. 2011;36: 592–605.
2. Nho SJ, Strauss EJ, Lenart BA, et al. Long head of the biceps tendinopathy: diagnosis and management. J Am Acad Orthop Surg. 2010;18:645–56.
3. Churgay CA. Diagnosis and treatment of biceps tendinitis and tendinosis. Am Fam Physician. 2009;80:470–6.
4. Peng PW. Shoulder. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, vol 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, California: Apple Inc.; 2014. p. 18–41.
5. Hashiuchi T, Sakurai G, Morimoto M, Komei T, Takakura Y, Tanaka Y. Accuracy of the biceps tendon sheath injection: ultrasound-guided or unguided injection? A randomized controlled trial. J Shoulder Elbow Surg. 2011;20:1069–73.
6. Zhang J, Ebraheim N, Lause GE. Ultrasound-guided injection for the biceps brachii tendonitis: results and experience. Ultrasound Med Biol. 2011;37:729–33.

Chapter 26

Calcific Tendinitis

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Peter Cheng, DO
Partner Emeritus, Southern California Permanente Medical Group, 21131 E. Lariat Court, Walnut, CA 91789, USA

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_26,

Calcific tendinitis, better termed as calcific tendinopathy in rotator cuff (as inflammatory cell is rarely encountered), is caused by the deposition of calcium mainly in the form of hydroxyapatite, in the critical zone of the tendon roughly 1 cm proximal to its insertion [1, 2]. Although this condition can occur in any tendon, supraspinatus tendon is the most commonly involved. The calcium deposits can remain in an asymptomatic stage. However, they can become symptomatic in 50 % of patients, causing acute or chronic pain. In patients with mild symptoms, simple conservative methods such as physical therapy and oral nonsteroidal anti-inflammatory agents can be quite effective. In patients with advanced symptoms, treatment options with iontophoresis, shockwave therapy, percutaneous fenestration, and surgery have been reported [3, 4]. Iontophoresis is no more effective than physiotherapy on long-term follow-up. Shockwave therapy is an effective treatment option, but the popularity is limited due to its cost and the requirement of at least three applications. Arthroscopic surgery is usually considered as the last option because of the invasiveness and the requirement of long rehabilitation. Therefore, minimal invasive treatment is a popular option for symptomatic calcific tendinopathy. This includes (1) fenestration (creating new opening in calcium deposit by repeated needling procedure to stimulate natural absorption), (2) barbotage (repeated alternating injection and aspiration of fluid with a syringe, lavage, and aspiration), and (3) steroid injection into the subacromial bursa [5, 6].

Pathophysiology and Clinical Presentation

The presenting symptoms are highly variable and depend on the stages of calcification (Fig. 26.1). Three stages of calcification have been described [7, 8].

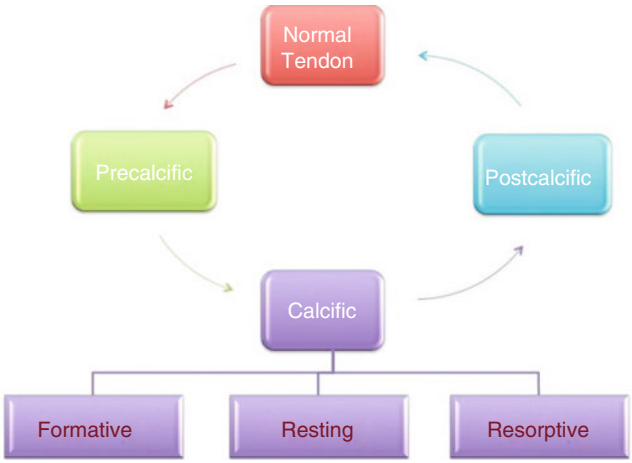


Fig. 26.1 The three stages of calcification (Reproduced with permission from Philip Peng Educational Series)

Precalcific Stage

During the precalcific stage, fibrocartilaginous metaplasia of the matrix takes place where the tenocytes becomes chondrocytes. The patient is essentially asymptomatic.

Calcific Stage

It is further divided into three phases. In the formative phase, calcium deposit appearing like chalk develops around the chondrocytes within the fibrocartilaginous matrix. After a variable period of inactivity (resting phase), the disease progresses to the resorptive phase where cell-mediated resorption takes place. At this phase, vascular channels develop at the periphery of the calcification. Migration of macrophages and multinucleated giant cells soon removes the calcium deposit by phagocytosis.

In the formative and resting phases, many patients are asymptomatic if the hard calcium deposit is not large enough to induce impingement syndrome. These calcifications tend to be well circumscribed and discrete when examined radiographically and often produce significant acoustic shadowing by ultrasound scan (Fig. 26.2). It is difficult to aspirate the calcifications in these two phases because the calcifications are quite hard and chalk-like. Most common indication of lavage at this stage is calcification over 1 cm in diameter, which commonly causes impingement syndrome.

In the resorptive phase, the calcium deposit is usually soft with toothpaste-like consistency but it is the most symptomatic phase. Shedding of calcium crystals into the adjacent subacromial bursa may result in severe acute pain and restricted range of motion. This phase typically lasts for 2 weeks or longer. These calcifications appear ill-defined on radiographs, producing little or no acoustic shadowing by ultrasonography (Fig. 26.3). This stage is the most common indication for ultrasound-guided intervention.

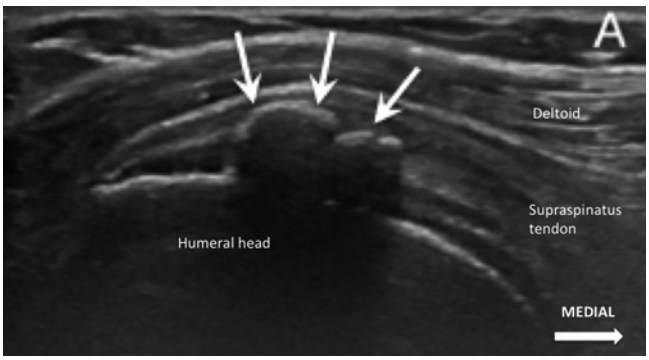


Fig. 26.2 Sonography of the calcium deposit in hard calcific phase. The *arrows* indicate the calcification. Note the anechoic shadow cast by the calcium (Reproduced with permission from Philip Peng Educational Series)

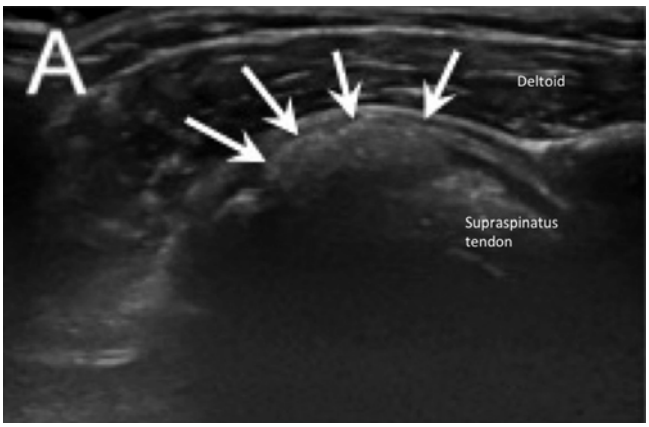


Fig. 26.3 Sonography of the calcium deposit in soft calcific phase. The *arrows* indicate the calcification. Note the minimal echogenic shadow cast by the calcium (Reproduced with permission from Philip Peng Educational Series)

Post-calcific Stages

The granulation tissue with fibroblasts and vascular channels grows into the location previously occupied by the calcification and replaces the calcification. The tendon undergoes fibrosis and eventual repair.

Dystrophic calcification is not included in the entity of calcific tendinopathy and is not a candidate for this procedure. This occurs in degenerative tissue and does not heal spontaneously. This is in contrast to the calcific tendinosis, which occurs in healthy tissue, is cell mediated, and is self-limiting.

Ultrasound-Guided Intervention for Calcific Tendinitis

Patient Preparation

The patient is placed in semi-reclined position in the modified Crass position (hand is placed on the ipsilateral hip with the palm against the body). A linear array ultrasound transducer (6–13 MHz) is positioned to show the long axis of the supraspinatus tendon and its insertion onto the greater tuberosity. To better appreciate the size, consistency, and shape of the calcium deposit in the supraspinatus tendon, at least two orthogonal planes (long and short axes) should be viewed [4]. With the ultrasound showing the long axis of supraspinatus tendon, a 25-gauge needle is inserted in-plane providing generous infiltration with lidocaine to the subcutaneous tissue, deltoid muscle, and subacromial bursa.

Fenestration

Single Needle Technique

An 18-gauge needle is advanced in-plane to fenestrate the calcium deposit (Fig. 26.4) [6]. Initially, the physician may encounter stiff resistance as the needle fractures the dense calcium. Rotating the needle will enhance fenestration (Fig. 26.5). With multiple "passes" throughout the deposit, the resistance will diminish as the deposit is fractured into smaller pieces. Care should be taken to avoid breaking the rim of the deposit.

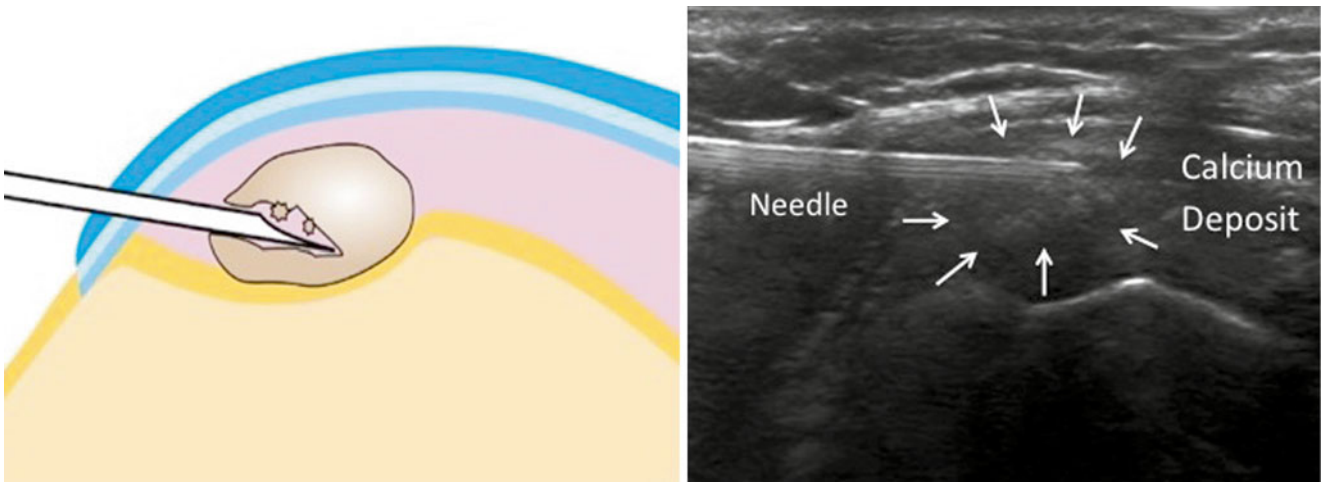


Fig. 26.4 Needle insertion into the center of calcium (outlined by *arrows*) with in-plane method (Reproduced with permission from Philip Peng Educational Series)

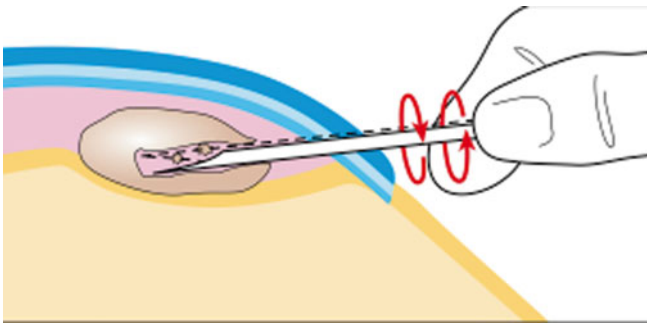


Fig. 26.5 Rotation of the needle within the calcium deposit (Reproduced with permission from Philip Peng Educational Series)

Fenestration: Double Needle Technique

The insertion of the first needle is similar to the single needle technique. However, the bevel of the needle is ensured to face upward following the insertion [3]. A second needle inserted superficial to the first needle with the bevel facing downward (Fig. 26.6). The two needles make an acute angle and should be in the same coronal plane as the transducer.

Barbotage

Single Needle Technique

With a single needle positioned within the calcific deposit, barbotage technique involves repeated injection and aspiration of saline into the fractured calcium particles. As aspirated particles may obstruct the lumen of the needle, the needle will be withdrawn and flushed with normal saline (Fig. 26.7). In the resorptive phase, the calcium deposit has a consistency consistent with soft toothpaste and is easily aspirated into the syringe.

Two Needle Technique

With the bevels of each needle facing each other, sterile saline is injected into superficial needle and the physician aspirates that fluid from the lower needle.

It is not clear whether there is a correlation between removal of a large amount of calcium and a good outcome, as some studies report satisfactory results, with minimal calcium retrieval [9–11].

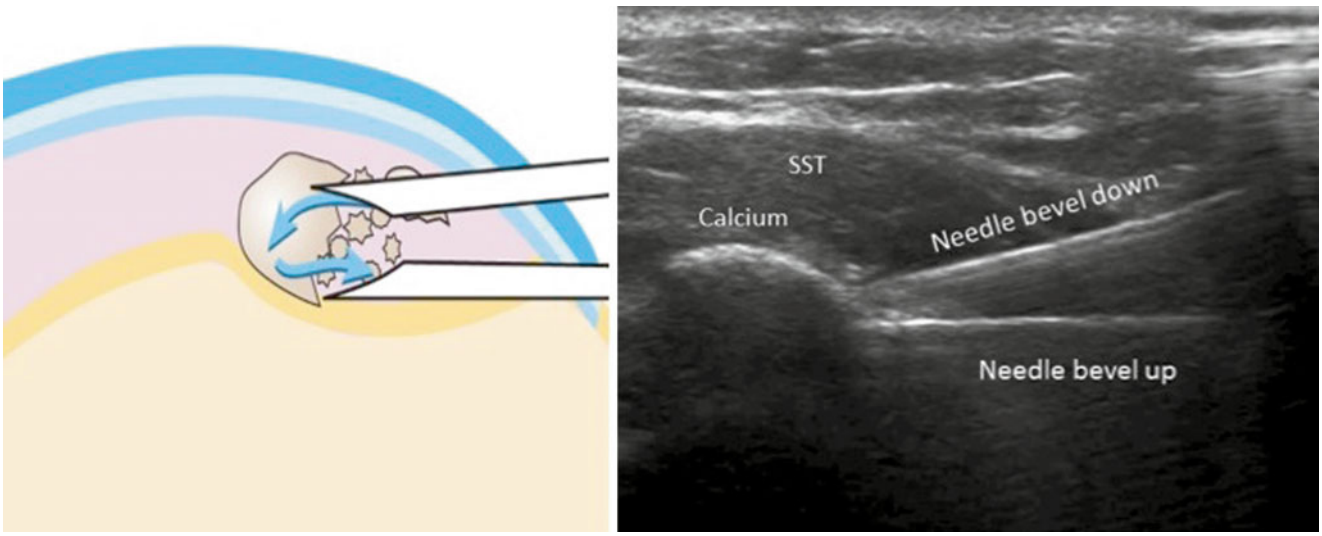


Fig. 26.6 Double needle method in which the second needle was inserted superficial to the first one. Note that the bevels of the needles were facing each other (Reproduced with permission from Philip Peng Educational Series)

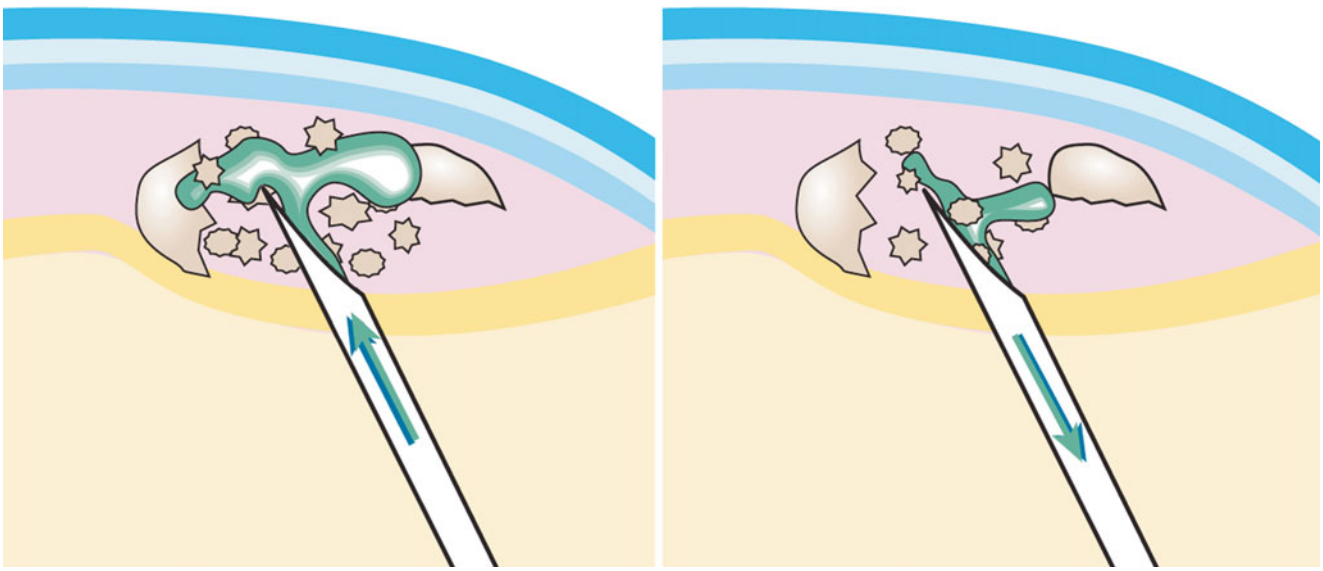


Fig. 26.7 Barbotage of fluid into and out of the calcium deposit to bring the broken calcium particles out of the needle (Reproduced with permission from Philip Peng Educational Series)

Subacromial (SA) Bursa Injection

After barbotage or aspiration, the needle is directed into the subacromial bursa, and steroid (triamcinolone acetonide 40 mg or methylprednisolone acetate 80 mg) is introduced to this space with 3–5 ml lidocaine 1 % (Fig. 26.7). After withdrawing the needle, an occlusive dressing and cold compress, as needed, are applied over the injection site. The shoulder is placed in a sling for support prior to discharge.

Post-procedure Care and Instructions

The patient is advised to avoid heavy lifting and overhead movement for about 2 weeks. For post-procedure pain, a cold compress over the shoulder that is supported by a sling can be prescribed [4]. Analgesics including a combination of acetaminophen and nonsteroid anti-inflammatory drugs should be considered. As post-procedure bursitis may occur in 15 % of patients, and within 2 months, additional steroid subacromial bursa injection may be needed.

Appendix: Equipment and Medications

1. Ultrasound machine and linear array transducer (4 cm footprint)
2. Syringes: one 5 ml, two 10 ml
3. Bowl to collect irrigation fluid
4. Cold compress
5. Shoulder sling
6. Needles:
   (a) One 22-gauge (8 cm) spinal needle
   (b) Two 16- or 18-gauge needles
7. Medication:
   (a) Local anesthetic: Lidocaine 1 %
   (b) Sterile saline solution: 100–200 ml
   (c) Steroid: Triamcinolone acetonide (Kenalog) 40 mg/ml or Methylprednisolone acetate (Depo-Medrol) 80 mg/ml.

References

1. Chiavaras MM, Jacobson JA. Ultrasound-guided tendon fenestration. Semin Musculoskelet Radiol. 2013;17:85–90.
2. Saboeiro GR. Sonography in the treatment of calcific tendinitis of the rotator cuff. J Ultrasound Med. 2012;31:1513–8.
3. Sconfienza LM, Viganò S, Martini C, et al. Double-needle ultrasound-guided percutaneous treatment of rotator cuff calcific tendinitis: tips & tricks. Skeletal Radiol. 2013;42:19–24.
4. Cheng P, Peng PW. Ultrasound guided intervention for calcific tendinosis-Part II. Newsletter, American Society of Regional Anesthesia; 2013. p. 14–17.
5. Cheng P. Ultrasound guided intervention for calcific tendinosis-Part I. Newsletter, American Society of Regional Anesthesia; 2013. p. 12–15.
6. Lee SH. Fenestration. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, vol 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, California: Apple Inc.; 2014. p. 67–76.
7. Uhthoff HK, Lapner P, Loehr JF. Calcific tendinitis. In: Rockwood CA, Matsen FA, Wirth MA, Lippitt SB, editors. The shoulder. 4th ed. Philadelphia: Saunders Elsevier; 2009. p. 1283–308.
8. Sarkar K, Uhthoff HK. Ultrastructural localization of calcium in calcifying tendinitis. Arch Pathol Lab Med. 1978;102:266–9.
9. Rizzello G, Franceschi F, Longo UG, Ruzzini L, Meloni MC, Spiezia F, Papalia R, Denaro V. Arthroscopic management of calcific tendinopathy of the shoulder–do we need to remove all the deposit? Bull NYU Hosp Jt Dis. 2009;67(4):330–3.
10. del Cura JL, Torre I, Zabala R, Legórburu A. Sonographically guided percutaneous needle lavage in calcific tendinitis of the shoulder: short- and long-term results. AJR Am J Roentgenol. 2007;189:W128–34.
11. Aina R, Cardinal E, Bureau NJ, Aubin B, Brassard P. Calcific shoulder tendinitis: treatment with modified US-guided fine-needle method. Radiology. 2001;221:455–61.

Chapter 27

Rotator Muscles and Subscapular Nerve Injection

Danilo Jankovic, MD
Director of Pain Management Center DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne-Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_27, © Springer International Publishing Switzerland 2015

Introduction

"Frozen Shoulder Syndrome"

A frozen shoulder may be a common stage of many disorders affecting the shoulder [1] or an independent, idiopathic condition [2]. The etiology of a frozen shoulder is complex [2, 3]. The numerous concepts are evident when the diagnostic labels applied to this condition are reviewed. It appears that many tissues, mainly sinovial, are involved in the ultimate frozen shoulder [1–4].

Current clinical nomenclature uses three categories of frozen shoulder [2]: idiopathic frozen shoulder [1, 3] adhesive capsulitis [1], and subacromial fibrosis [1, 3, 5]. Additional proposal etiologies of the frozen shoulder include acromioclavicular joint irritation [1], entrapment of the suprascapular nerve [5], prolonged immobilization of the upper extremity [5], cervical radiculopathy [4], hemiplegia [6–9], myocardial infarction [4], bicipital tendonitis [1], and muscle spasm [2]. There are several references in the literature that assume frozen shoulder to be an algoneurodystrophic process [6, 10]. Recent publications have pointed out similarities with Dupuytren's contracture [11]. Many other synonyms are used in the literature: periarthritis or periarticular arthritis [1, 3–5], pericapsulitis [4], scapulocostal syndrome [2], calcified tendinitis of the rotator cuff [1], degenerative tendinitis of the rotator cuff [1–4], and acromioclavicular arthritis [1]. Mc Laughlin stressed the importance of the subscapularis muscle in the etiology of the frozen shoulder [12].

Clinical Evaluation

Clinical Presentation

This scenario develops insidiously, beginning with pain and tenderness usually over the deltoid insertional area in the upper outer humerus. Motion aggravates the pain, and gradually a limitation of both active and passive motion develops. What at first hampers daily activities gradually interferes with sleep.

The usual stages of evolution are pain, gradual restriction of motion, and ultimately marked limitation without pain. The last stage is stiff, useless but painless shoulder which hurts only when forcefully moved to ensure or to determine limitation [2, 3]. The condition widely claimed to be a frozen shoulder still remains an enigma as to the exact origin, tissue involvement, causative mechanism, and the ideal forms of prevention and treatment.

Imaging Modalities

X-ray evaluation may reveal no specific diagnostic findings other than some osteopenia or cystic changes in the lateral aspect of the humerus [3]. An arthrogram depicts the presence and degree of adhesive capsulitis. An MRI may rule out other abnormalities but is not specifically diagnostic of adhesive capsulitis with regard to the exact tissues involved or the severity of involvement [1–4].

Management of Frozen Shoulder

Treatment for the frozen shoulder varies widely with experienced clinicians. Started early with a frequent intensity—before there is significant adhesion—active physical therapy is more applicable and is advocated by most clinicians who are studying and treating this conditions. Pain relief is achieved by local ice, heat, ultrasound application [3], TENS application [3], oral NSAIDs, electroacupuncture [13], oral and local corticosteroids, and oral analgesic medication. Mobilization and gentle manipulation may be also advisable in the early stage [14]. Rhythmic stabilization exercises have also proven to be of value [3]. Some of the nerve blocks, e.g., suprascapular [15] or subscapular nerve block [7, 16, 17], have been recently recommended.

Subscapular Nerve Block and Subscapularis Muscle Trigger Point Infiltration

Introduction

Irritation of subscapularis muscle trigger points and adjacent nerves produces pain both during movement and at rest, resulting in pain radiating to the scapula, posterior deltoid muscle, elbow, and/or dorsal wrist [2]. The pain usually worsens at night. The usual scenario restricts arm abduction and external rotation, reducing movement in all directions, ending in a stiff, frozen shoulder. A common finding is that the subscapularis muscle trigger point plays a key role in the development of a frozen shoulder [2, 6–9].

The subscapular nerve block and subscapularis trigger point infiltration could be very valuable in producing pain relief in the affected shoulder. Shortly after each block, intensive, physical therapy could be successfully performed [17]. We injected routinely 10–15 mL of a local anesthetic solution with or without the addition of a corticosteroid. The total volume guarantees adequate spread of the corticosteroids. The mechanism by which corticosteroids have been suggested to modify pain include reduced prostaglandin synthesis [18], a week of local anesthetic action [19], a change of activity in the dorsal horn cells [8], and reduced ectopic discharge from neuromatas [20]. Corticosteroids have also been shown to induce a reversible block of normal C fibers in an animal model [21].

Anatomy of the Fossa Subscapularis [16, 17, 22, 23]

The subscapularis muscle (the most important internal rotator muscle of the arm; it also acts as an adductor when the arm is raised or flexed and, together with the rotator cuff, keeps the head of the humerus pressed into the glenoid fossa) is located at the anterior surface of the subscapularis fossa. The anatomical insertions of the subscapular muscle are medial to the interior surface of the scapula (Figs. 27.1, 27.2, and 27.3) and lateral to the lesser tubercle on the anterior surface of the humerus.

The subscapular nerves consist of two or three nerves emerging from various parts of the brachial plexus for the subscapular, teres major, and latissimus dorsi muscles. The longest and most important of these is the thoracodorsal nerve, which runs along the axillary border of the scapula and supplies the latissimus dorsi muscle.

The superior subscapular nerve emerges from C5 and C6 (C7) and enters the subscapular muscle. The medial subscapular nerve (C5–6) arises from the posterior secondary trunks and supplies the lateral lower part of the subscapular muscle and teres major muscle.

The inferior subscapular nerve (thoracodorsal nerve) is the largest in this group. It arises from the posterior secondary branches or from the axillary nerve, or more rarely from the radial nerve, and passes along the lateral edge of the scapula to the latissimus dorsi muscle.

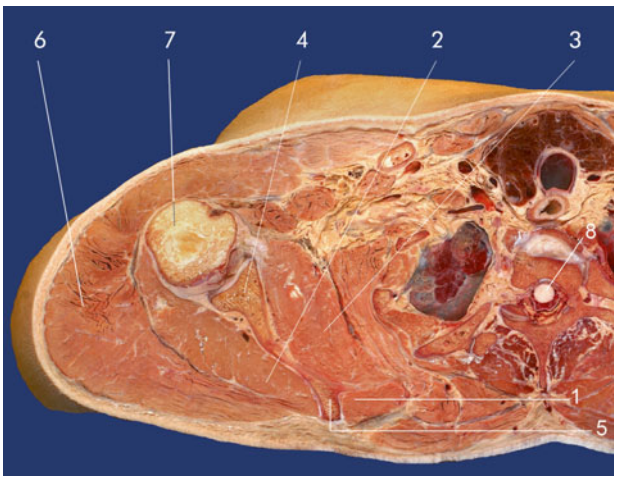


Fig. 27.1 Transverse section at the level of the T2-T3. Rotator cuff muscles and neighboring muscles. (*1*) Supraspinatus muscle, (*2*) infraspinatus muscle, (*3*) subscapularis muscle, (*4*, *5*) scapula, (*6*) deltoid muscle, (*7*) greater tubercle of the humerus, (*8*) spinal cord (Reproduced with permission from Danilo Jankovic)

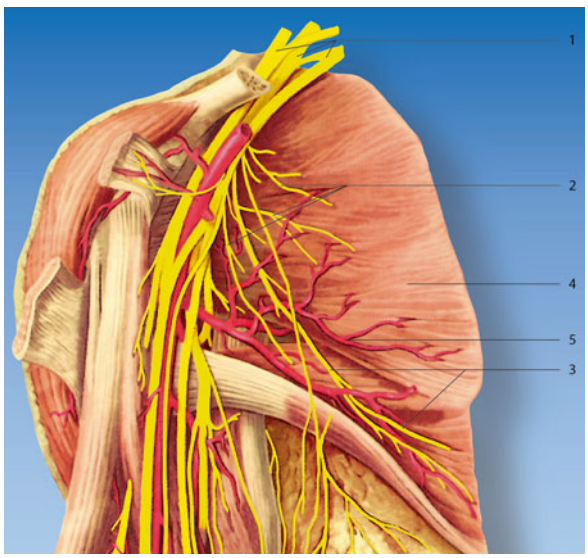


Fig. 27.2 Anatomy (anterior view): (*1*) cords of the brachial plexus, (*2*) subscapular nerve, (*3*) thoracodorsal nerve, (*4*) subscapular muscle, (*5*) circumflex scapular artery (Reproduced with permission from Danilo Jankovic)

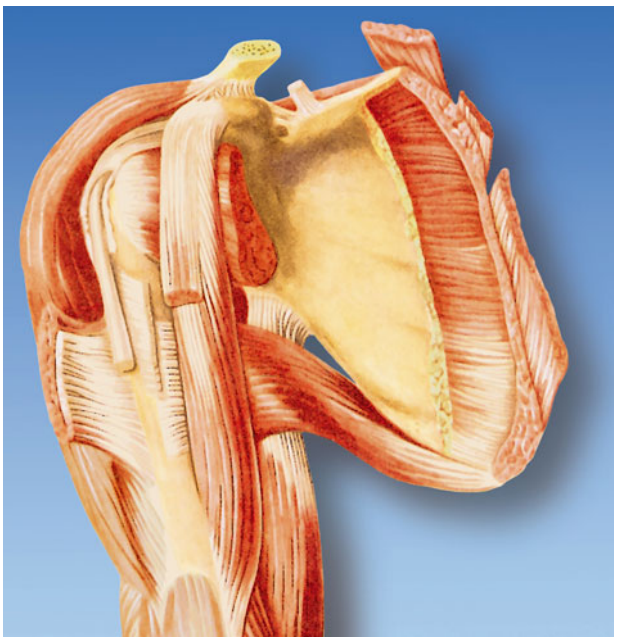


Fig. 27.3 Anatomy. Subscapular fossa and subscapular muscle (Reproduced with permission from Danilo Jankovic)

Innervation and Function

Table 27.1.

Indications

Indications for a subscapular nerve block and subscapularis trigger point infiltration are presented in Table 27.2.

Procedure

Preparations

Check that the emergency equipment is complete and in working order; sterile precautions, intravenous access. Prior information for the patient is an absolute necessity.

Materials

Fine 25-mm-long 26-G needle for local anesthesia, 70-mm-long 20-G needle (with the needle shaft angled by about 20°), local anesthetic, disinfectant, swabs, 2- and 10-mL syringes.

Technique [16, 17]

Position

Sitting, with the neck comfortably tilted and the shoulders relaxed.

Table 27.1 Rotator cuff muscle (dark blue) and neighboring muscles: innervation and function

Muscle	Innervation	Function
Supraspinatus	Supracapular nerve (C5**; superior trunk*)	Abducts the upper arm and pulls the head of the upper arm into the glenoid cavity
Infraspinatus	Supracapular nerve (C5, C6**; superior trunk*)	External rotation of the arm; stabilizes the head of the humerus in the glenoid cavity
Teres minor	Axillary nerve (C5, C6**; posterior fascicle*)	Almost identical to the infraspinatus muscle
Subscapularis	Subscapular nerve (C5, C6**; posterior fascicle*)	Internal rotation and adduction of the upper arm in the shoulder
Teres major	Inferior subscapular nerve (C5, C6**; posterior fascicle*)	Supports adduction, internal rotation and extension of the upper arm from a bent position
Deltoid	Axillary nerve (C5, C6**; posterior fascicle*)	Helps the supraspinatus muscle to abduct the upper arm in the shoulder
Latissimus dorsi	Thoracodoral nerve (C6–C8**; posterior fascicle*)	Adduction and internal rotation of the arm; strong downward movement of the scapula
Coracobrachialis	Musculocutaneous nerve (C5, C6**; lateral fascicle*)	

*Brachial plexus
**Spinal nerves

Table 27.2 Indications for subscapular nerve block and subscapularis trigger point

Infiltration
Diagnostic: in painful conditions of the shoulder girdle/joint
Therapeutic: in the following situations (in combination with physiotherapy):
Frozen shoulder syndrome
Rheuma
Diabetes
Post-trauma neuralgia
Postherpetic neuralgia
Post-hemiplegia neuralgia
Ankylosing spondylitis

Location (Fig. 27.4)

1. The patient's arm is pulled back, so that the contours of the scapula are easily recognized. The center of the medial border of the scapula is marked as the injection point.
2. Acromion

Skin prep, local anesthesia, drawing up the local anesthetic, and testing the injection needle for patency.

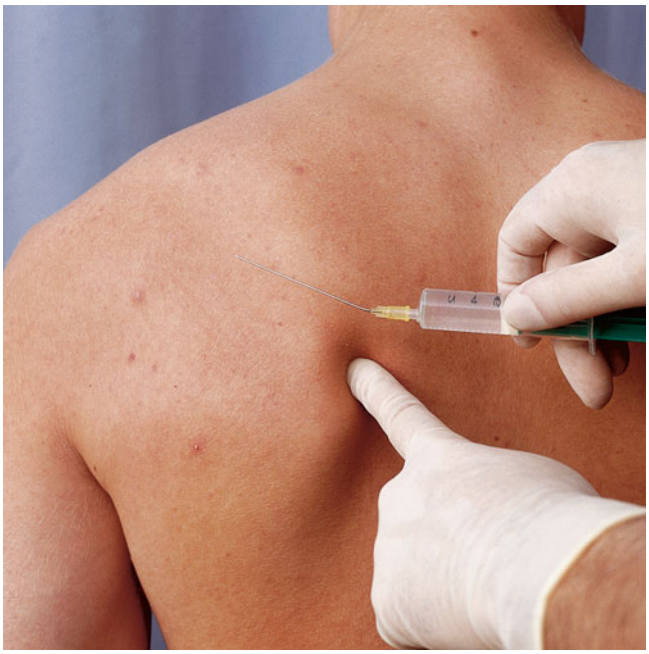


Fig. 27.4 Location. Marking the injection site (center of the medial border of the scapula) (Reproduced with permission from Danilo Jankovic)

1. Before the injection, the shaft of the injection needle should be bent by about 20°.
2. Targeted paresthesias are not elicited.
3. During the injection, observe the skin for possible subcutaneous spread of the local anesthetic.

Injection Technique

1. Introduce the 20°-angled needle into the center of the medial border of the scapula, in the direction of the acromion (Figs. 27.5 and 27.6a, b).
2. The needle is introduced subscapularly parallel to the skin surface between the anterior surface of the scapula (costal surface) and the posterior thoracic wall (ribs), into the subscapular fossa. If the needle meets the edge of the ribs, it is withdrawn as far as the subcutaneous tissue and reintroduced.
3. At a depth of 4 cm, then 5 cm, and finally 6 cm—depending on the anatomy—a total of 10–15 mL local anesthetic is then injected after prior aspiration (Figs. 27.5 and 27.6a).

The signs of a successful injection are spread extending into the shoulder joint, upper arm, and often as far as the wrist, corresponding to the radiation pattern of the trigger points of the subscapular muscle [2] (Fig. 27.7a, b).

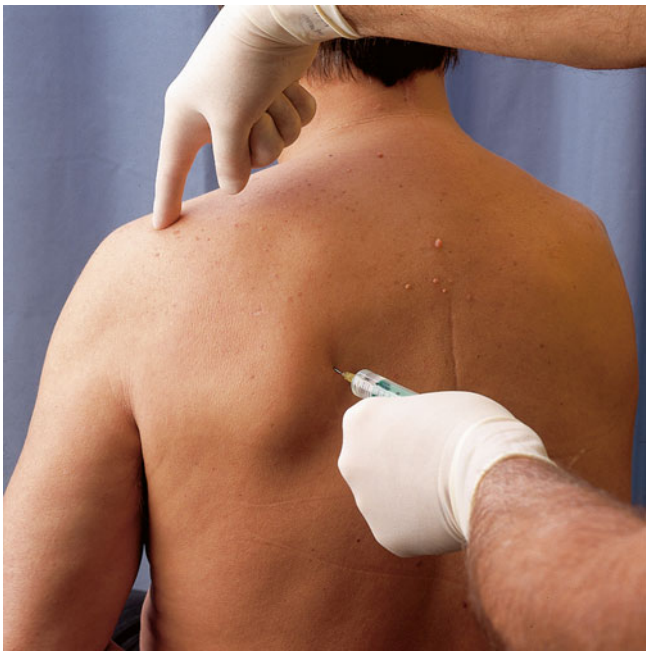


Fig. 27.5 Introducing the needle in the direction of the acromion (Reproduced with permission from Danilo Jankovic)

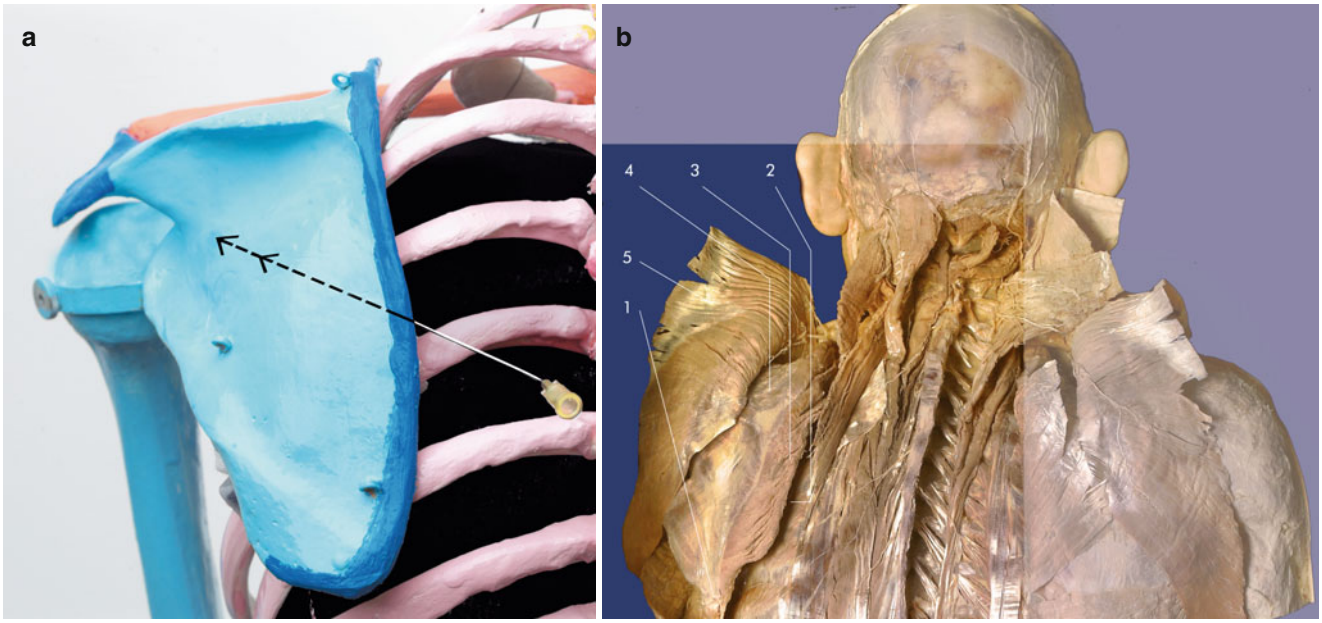


Fig. 27.6 (**a**) Introducing the needle in the direction of the acromion (skeletal model) (Reproduced with permission from Danilo Jankovic). (**b**) (*1*) Inferior angle of the scapula, (*2*) medial border of the scapula with subscapularis muscle inside, (*3*) rhomboideus major muscle, (*4*) infraspinatus muscle, (*5*) trapezius muscle (Reproduced with permission from Danilo Jankovic)

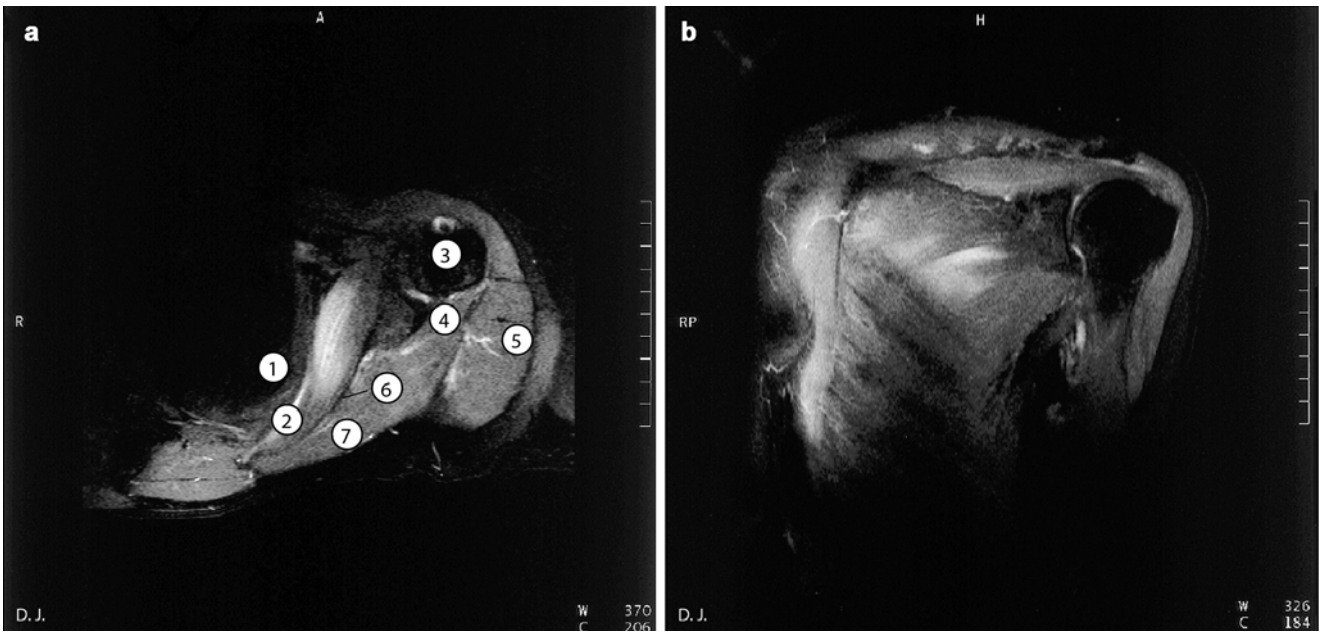


Fig. 27.7 Magnetic resonance images 10 min after injection of 10 mL ropivacaine, without radiographic contrast medium, into the subscapular fossa. (**a**) Axial (cross section). (**b**) Paracoronal. (*1*) Thorax wall, (*2*) subscapular muscle and subscapular fossa, (*3*) head of the humerus, (*4*) teres minor muscle, (*5*) deltoid muscle, (*6*) scapula, (*7*) infraspinatus muscle (Reproduced with permission from Danilo Jankovic)

Dosage

Diagnostic

Five millilitres local anesthetic—e.g., 1 % prilocaine or 1 % mepivacaine.

Therapeutic

Ten to fifteen millilitres local anesthetic– e.g., 0.5–0.75 % ropivacaine, 0.25–0.5 % bupivacaine (0.25–0.5 % levobupivacaine). In acute pain, the addition of 40 mg triamcinolone has proved useful.

A combination of the subscapular and suprascapular nerve blocks is possible and often desirable (Table 27.3).

Block Series

In all indications, a series of six to eight blocks is useful if an improvement trend is seen after the first and second treatments.

Side Effects

If the dosage is too high, transient weakness may occur in the shoulder and upper arm. Outpatients should be informed about this. A partial block of the intercostal nerves is possible due to spread of the local anesthetic and is often desirable.

Complications

- There is a potential risk of pneumothorax (unlikely if the correct technique is observed).
- Intravascular injection.

We could not observe any side effects or possible complications after injection of local anesthetic with corticosteroid [24, 25].

Table 27.3 Shoulder–arm region: blocking techniques in pain therapy

Indications				
		Acute and chronic pain conditions Target area		
Surgical	Postoperative pain therapy	Shoulder	Shoulder–arm	Mobilization of the shoulder
Interscalene[a] +++++ Dosage: 20–25 mL 0.75 % ropivacaine or 0.5 % bupivacaine (0.5 % levobupivacaine)	Interscalene[b] +++++ 20–25 mL 0.375–0.5 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)	Subscapular nerves +++++ 10–15 mL 0.5–0.75 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)	Interscalene +++++ 10–15 mL 0.375–0.5 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)	Interscalene +++++ 20–25 mL 0.375–0.5 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)
	Subscapular nerves[b] ++ 15 mL 0.5–0.75 % ropivacaine or 0.25–0.375 % bupivacaine (0.25–0.375 % levobupivacaine)	Interscalene +++ 10–15 mL 0.375–0.5 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)	Stellate ganglion ++ 10–15 mL 0.375 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)	Subscapular nerves[c] ++ 10–15 mL 0.5–0.75 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)

Comparison of interscalene block of the brachial plexus, blocks of the subscapular and suprascapular nerves, and blocks of the stellate ganglion
+++++ Best method
++++ Very suitable method
+++ Suitable method
++ Method suitable with some qualifications
+ Less suitable method
[a]Usually in combination with basic general anesthesia. This provides excellent pain relief
[b]In severe pain, a combination of the two techniques is possible
[c]Usually in combination with a suprascapular nerve block: 8–10 mL 0.5–0.75 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine)

Supraspinatus Muscle

Anatomical Insertions

The anatomical insertions are medial to the supraspinous fossa and lateral to the greater tubercle of the humerus (Fig. 27.8).

Innervation and Function

See Table 27.1

Myotatic Unit

This covers the middle part of the deltoid muscle and the upper part of the trapezius muscle, as synergists for abduction.

Trigger Points

The two trigger points (TrPs) in the supraspinatus muscle are located deep in the supraspinous fossa of the scapulae, underneath the relatively thick part of the trapezius muscle. The medial TrP lies directly above the spine of the scapula, lateral to the medial border of the scapula. The lateral TrP can be palpated medial to the acromion. A third TrP may be located in the tendon of the muscle at its lateral insertion on the joint capsule and the greater tuberosity [2] (Fig. 27.8).

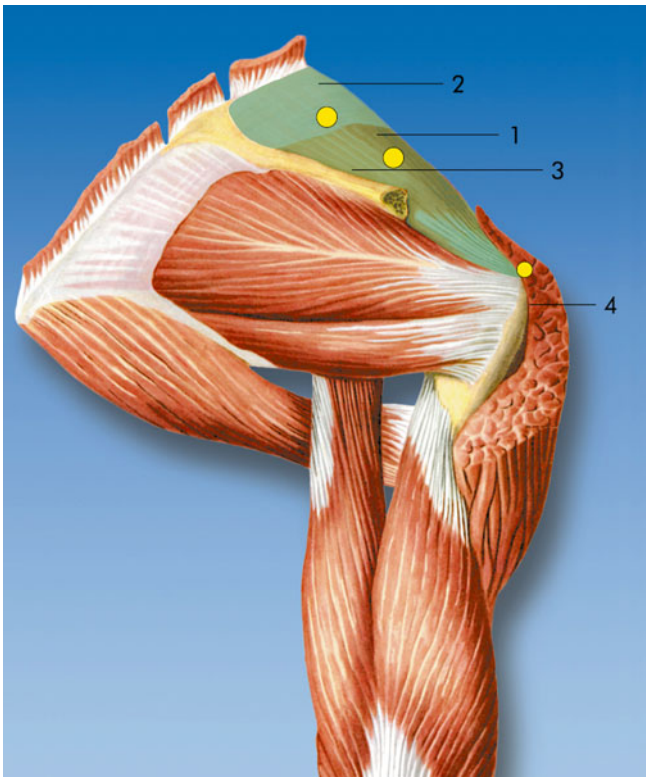


Fig. 27.8 Supraspinatus muscle. Anatomic insertions and myofascial trigger points (*yellow circles*) (Adapted from Travell and Simons [2]). (*1*) Infraspinatus muscle, (*2*) supraspinous fascia, (*3*) spine of the scapula, (*4*) greater tuberosity of the humerus (Reproduced with permission from Danilo Jankovic)

Symptoms

Pain in the middle deltoid region, sometimes radiating to the upper and lower arm, particularly in the area of the lateral epicondyle.

Procedure

Materials

Sterile precautions, 23-G needle 30 mm long, 2- and 5-mL syringes, local anesthetic.

Injection Technique

The lower arm of the seated patient is placed behind the back at waist level ("hand behind the back"; (Fig. 27.9)). After palpation, injection into the medial TrP is carried out in the direction of the suprascapular notch (Fig. 27.10). After careful aspiration, injection of the local anesthetic follows. The lateral TrP is sought directly medial to the acromion. The muscle's insertion point at the greater tubercle of the humerus requires perpendicular puncture until bone contact is made (Fig. 27.11).

Dosage

One to two millilitres local anesthetic—e.g., 0.2–0.375 % ropivacaine.

Complications

Pneumothorax must be regarded as a potential complication when injecting into the medial TrP of the supraspinatus muscle.

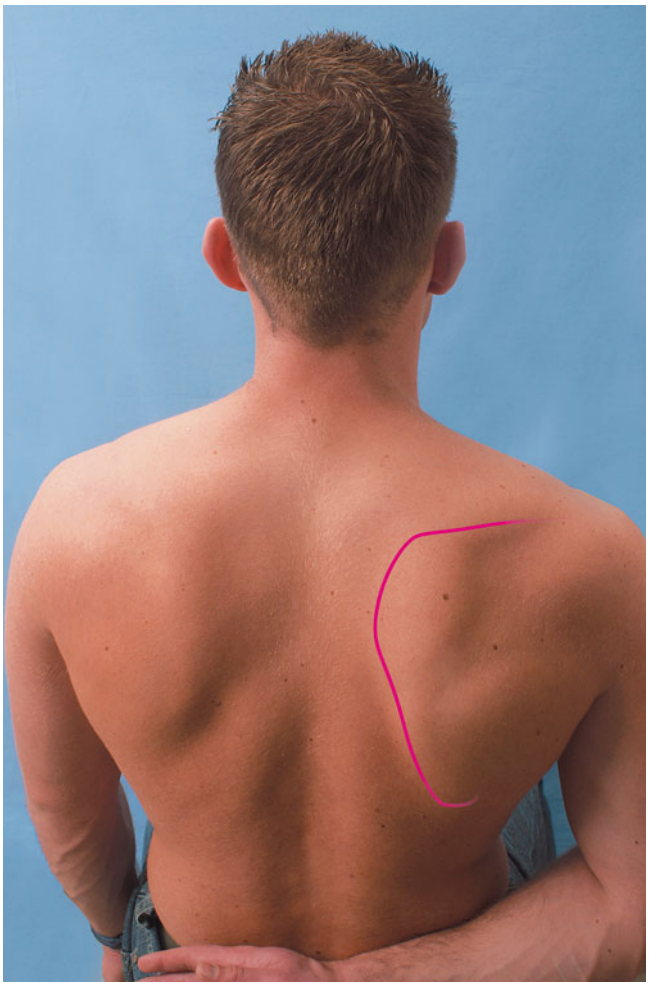


Fig. 27.9 Supraspinatus muscle. Positioning for trigger point injection ("hand behind the back" position) (Reproduced with permission from Danilo Jankovic)

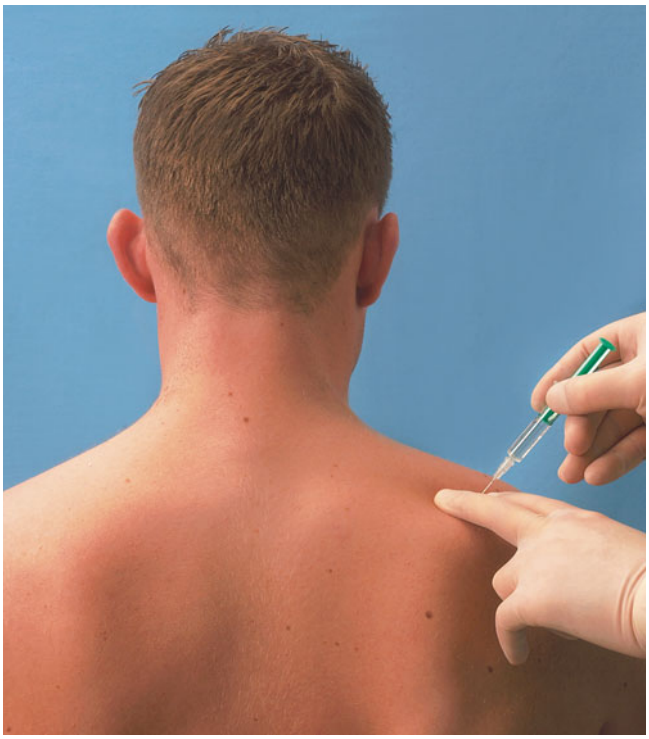


Fig. 27.10 Supraspinatus muscle. Injection into the medial trigger point in the direction of the suprascapular notch (Reproduced with permission from Danilo Jankovic)

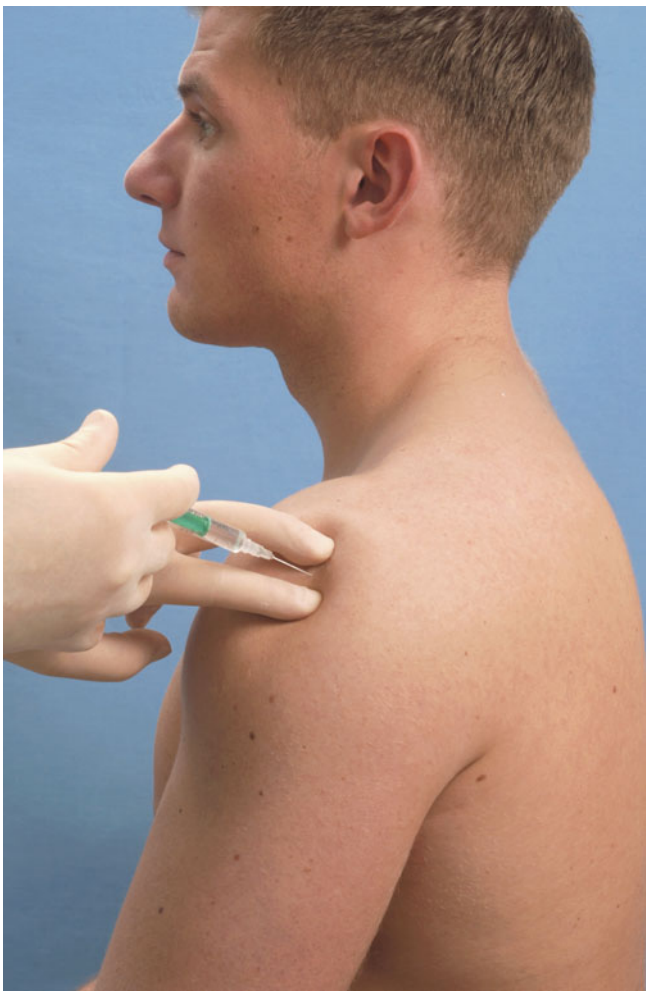


Fig. 27.11 Supraspinatus muscle. Infiltration of the insertion site at the greater tubercle of the humerus (Reproduced with permission from Danilo Jankovic)

Infraspinatus Muscle

Anatomic Insertions

The anatomic insertions are located medial to the infraspinous fossa of the scapula and lateral to the greater tuberosity of the humerus (Figs. 27.12 and 27.13).

Innervation and Function

See Table 27.1

Myotatic Unit

With the exception of external rotation of the arm, the infraspinatus muscle acts synergistically with the teres minor muscle (with almost identical function) and the posterior part of the deltoid muscle.

Trigger Points

Two active trigger points (medial and lateral) can be located approximately 2 cm below the spine of the scapula, and sometimes there is also another possible trigger point slightly caudally [2] (Fig. 27.12).

Symptoms

The symptoms consist of referred pain when sleeping in the lateral position and an inability to reach the rear trouser pockets or bra fastener or to comb the hair or brush the teeth.

Procedure

Materials

Sterile precautions, 23-G needle 30 mm long, 2- and 5-mL syringes, local anesthetic.

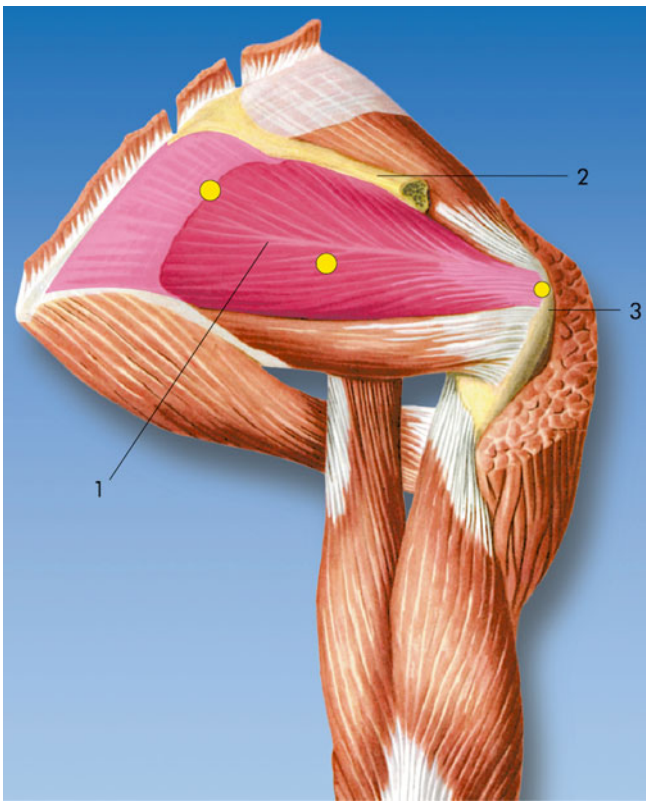


Fig. 27.12 Infraspinatus muscle. Anatomic insertions and myofascial trigger points (*yellow circles*) (Adapted from Travell and Simons [2]). (*1*) Infraspinatus muscle, (*2*) spine of the scapula, (*3*) greater tuberosity of the humerus (Reproduced with permission from Danilo Jankovic)

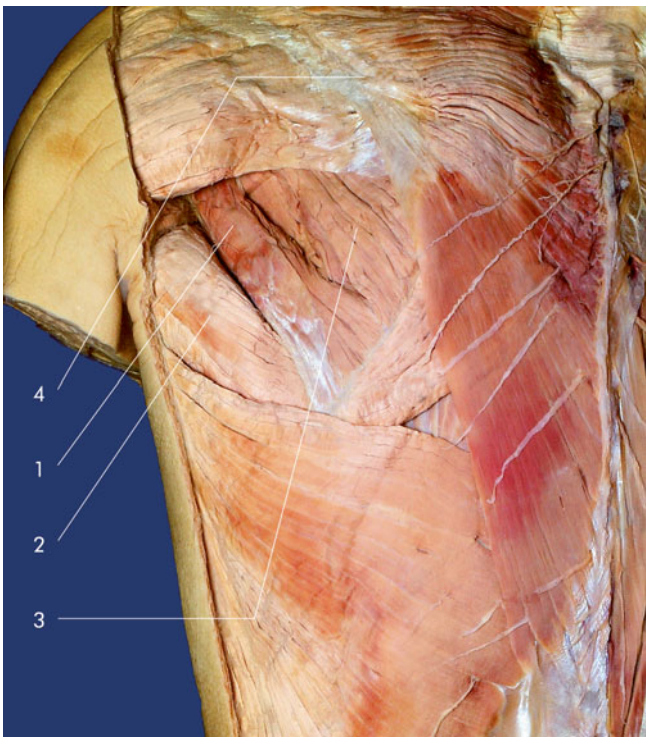


Fig. 27.13 (*1*) Teres minor muscle, (*2*) teres major muscle, (*3*) infraspinatus muscle, (*4*) spine of the scapula (Reproduced with permission from Danilo Jankovic)

Injection Technique

The patient lies on the side that is not being treated. The arm is bent to 90° and the elbow is laid on a cushion. The contour of the scapula has to be clearly defined.

After careful disinfection and palpation of the trigger point (TrP), the needle is slowly introduced in the direction of the TrP. During injection into the medial TrP, the left middle finger is pressed against the caudal edge of the spine of the scapula. During injection into the lateral TrP, the left ring finger presses against the caudal edge of the spine of the scapula (Figs. 27.14 and 27.15).

The puncture has to be carried out sensitively, as the scapula bones (part of the infraspinous fossa) sometimes offer very little resistance (resembling a fibrous membrane, so that there is a risk of pneumothorax). The insertion site of the muscle into the greater tuberosity of the humerus requires a perpendicular position to be maintained until bone contact is made (Fig. 27.16).

Dosage

One to two millilitres local anesthetic—e.g., 0.2–0.375 % ropivacaine.

Complications

- Pneumothorax is a potential complication [2].
- Infection.

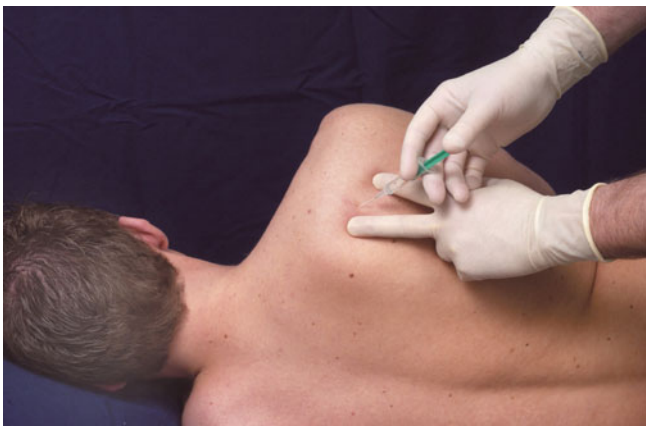


Fig. 27.14 Infraspinatus muscle. Injection into the medial trigger point in the direction of the caudal edge of the spine of the scapula (Reproduced with permission from Danilo Jankovic)

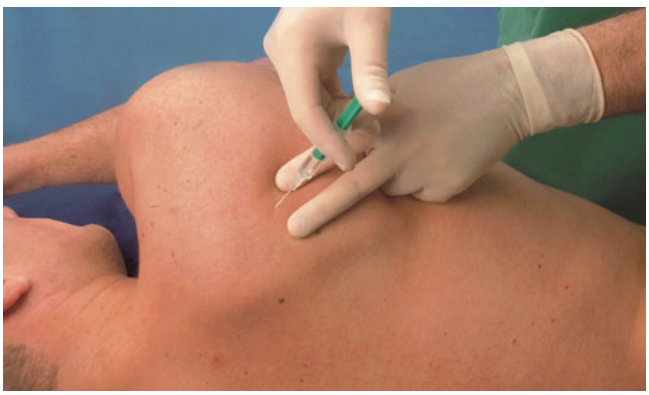


Fig. 27.15 Infraspinatus muscle. Injection into the caudal trigger point (Reproduced with permission from Danilo Jankovic)

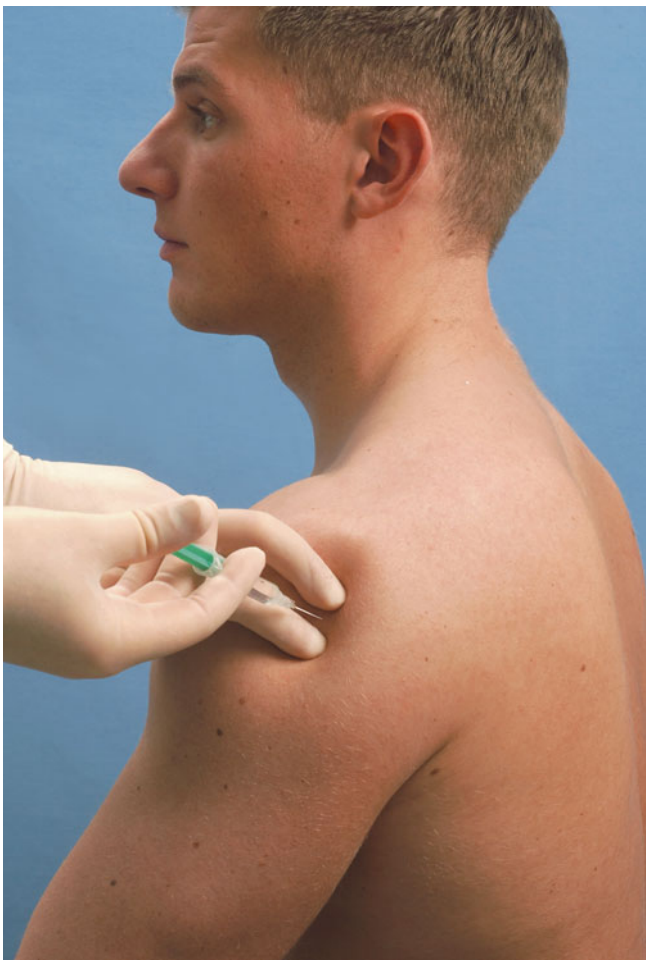


Fig. 27.16 Infraspinatus muscle. Infiltration of the insertion site on the greater tuberosity of the humerus (Reproduced with permission from Danilo Jankovic)

Teres Minor Muscle

Anatomic Insertions

The muscle's anatomic insertions are located directly alongside and caudal to those of the infraspinatus muscle (Figs. 27.13 and 27.17).

Innervation and Function

See Table 27.1

Myotatic Unit

The teres minor muscle acts synergistically with the infraspinatus muscle.

Trigger Points

The teres minor muscle is one of the most rarely affected muscles in the rotator cuff (only involved in 7 % of cases). The trigger point usually lies in the center of the muscle [2] (Figs. 27.13 and 27.17). The teres minor muscle is located above the teres major muscle.

Symptoms

Pain in the posterior deltoid area.

Procedure

Materials

Sterile precautions, 23-G needle 30 mm long, 2- and 5-mL syringes, local anesthetic.

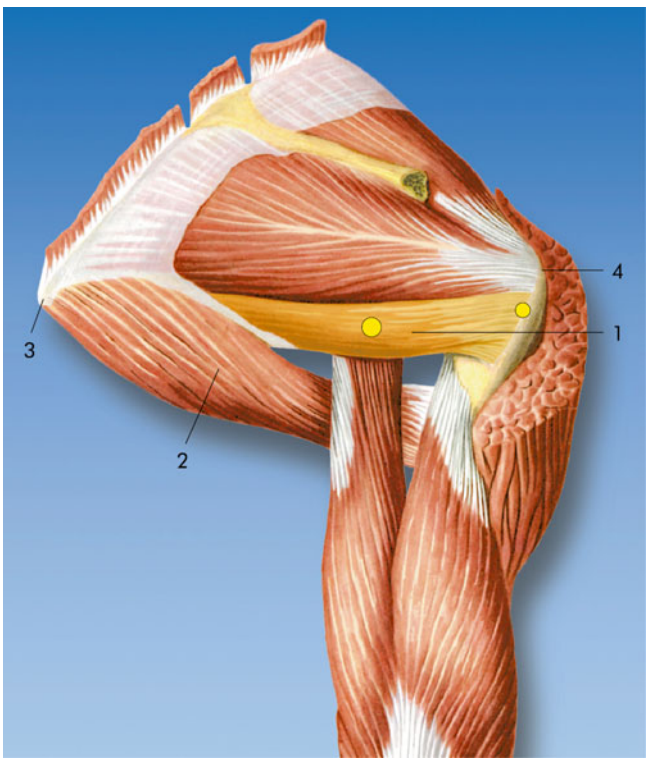


Fig. 27.17 Teres minor muscle. Anatomic insertions and myofascial trigger points (*yellow circles*) (Adapted from Travell and Simons [2]). (*1*) Teres minor muscle, (*2*) teres major muscle, (*3*) inferior angle of the scapula, (*4*) greater tuberosity of the humerus (Reproduced with permission from Danilo Jankovic)

Injection Technique

The arm is bent to 90°. The contour of the scapula has to be clearly defined (Fig. 27.18). The TrPs are sought between the teres major and infraspinatus muscles, near the lateral edge of the scapula. The index and middle finger fix the TrP. The 30-mm needle is directed toward the scapula (Fig. 27.19). The insertion site of the muscle on the greater tuberosity of the humerus requires a perpendicular needle direction until bone contact is made (Fig. 27.20).

Dosage

Two millilitres local anesthetic—e.g., 0.2–0.375 % ropivacaine.

Complications

Pneumothorax is a potential complication.

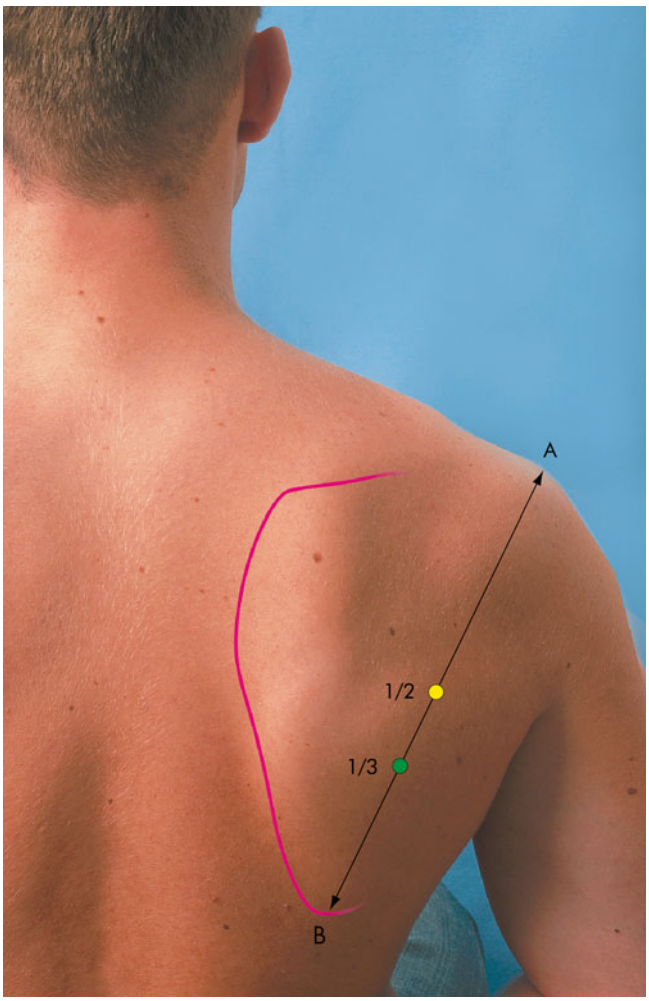


Fig. 27.18 Teres minor muscle (*yellow*) and teres major muscle (*green*). Landmarks for TrP injection. *A* Acromion, *B* inferior angle of the scapula (Reproduced with permission from Danilo Jankovic)

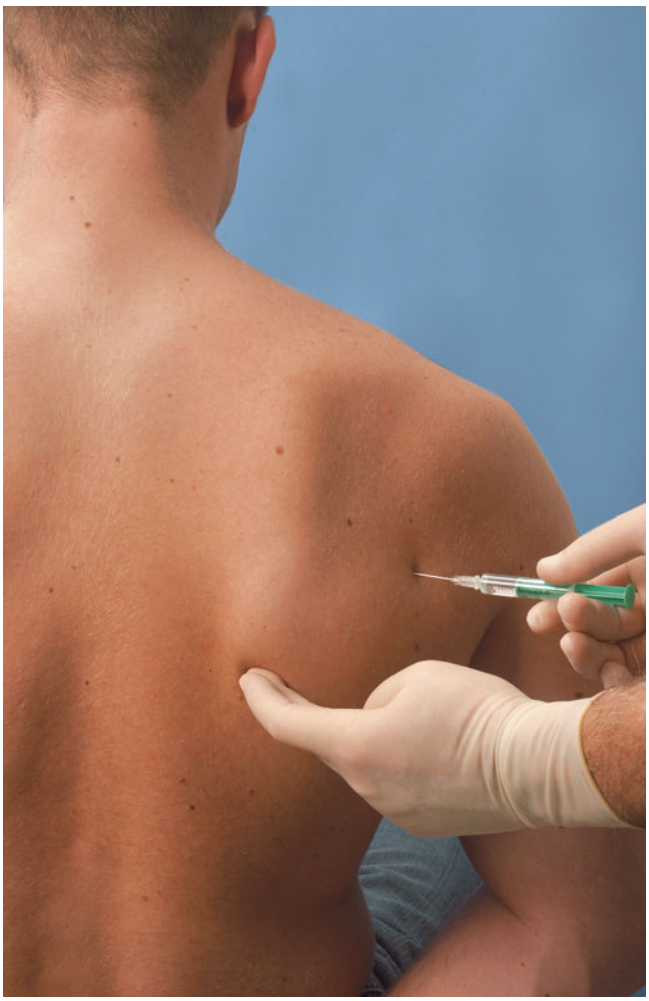


Fig. 27.19 Teres minor muscle. The needle is directed toward the scapula (Reproduced with permission from Danilo Jankovic)

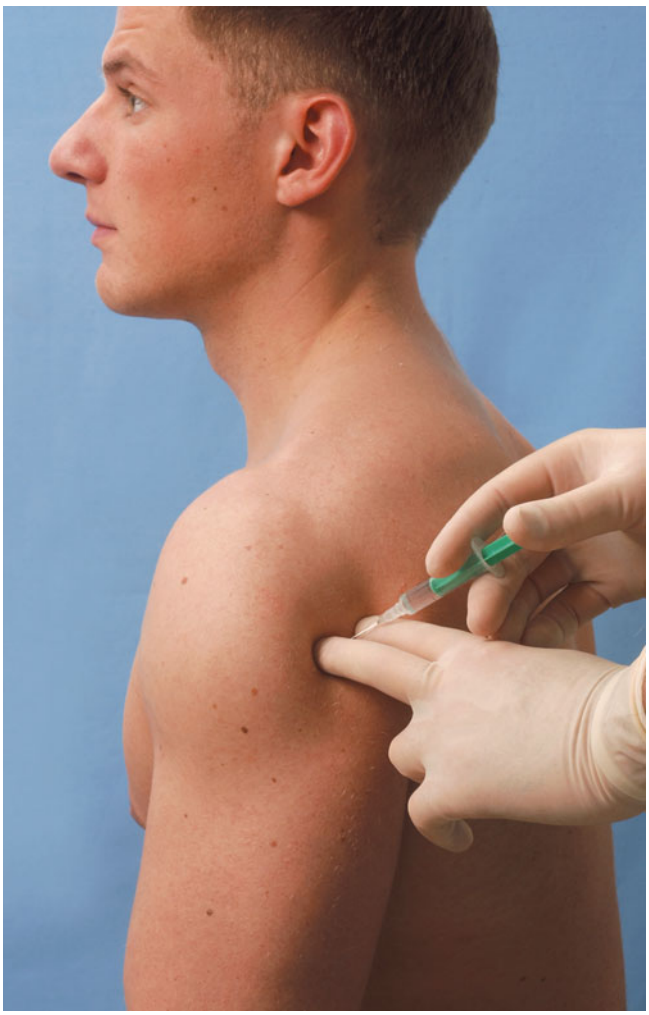


Fig. 27.20 Teres minor muscle. Injection at the insertion point of the greater tuberosity of the humerus (Reproduced with permission from Danilo Jankovic)

References

1. Batemann JE. The shoulder and neck. Philadelphia: W. B. Saunders; 1991. p. 134, 145–6, 149, 284–90.
2. Travell JG, Simons DG. Myofascial pain and dysfunction, The trigger point manual, vol. 1. Baltimore: Williams & Wilkins; 1983.
3. Cailliet R. Shoulder pain. 3rd ed. Philadelphia: Davis Company; 1991. p. 105–23, 193–226.
4. Cailliet R. Soft tissue pain and disability. Philadelphia: FA. Davis; 1977. p. 161–2.
5. Kopell HP, Thompson WL. Pain and the frozen shoulder. Surg Gynecol Obstet. 1959;109:92–6.
6. Dursun E, Dursun N, Ural CE, Cakci A. Glenohumeral joint subluxation and reflex sympathetic dystrophy in hemiplegic patients. Arch Phys Med Rehabil. 2000;81(7):944–6.
7. Hecht JS. Subscapular nerve block in the painful hemiplegic shoulder. Arch Phys Med Rehabil. 1992;73(11):1036–9.
8. Hall ED. Acute effects of intravenous glucocorticoids on cat spinal motor neuron electrical properties. Brain Res. 1982;240:186–90.
9. Pinedo S, de la Villa FM. Complications in the hemiplegic patients in the first year after the stroke. Rev Neurol. 2001;32:206–9.
10. Muller LP, Muller LA, Happ J, Kerschbaumer F. Frozen shoulder: a sympathetic dystrophy? Arch Orthop Trauma Surg. 2000; 120(1–2):84–7.
11. Ekelund A. New knowledge of the mysterious “frozen shoulder”. Surgical treatment can accelerate the recovery in more serious cases. Lakartidningen. 1998;95(48):5472–4.
12. Mc Laughlin HL. Lesions of the musculotendinous cuff of the shoulder. J Bone Joint Surg. 1944;26:31.
13. Chen CH, Chen TW, Weng MC, et al. The effect of electroacupuncture on shoulder subluxation for stroke patients. Kaohsiung J Med Sci. 2000;16:525–32.
14. Reichmister JP, Friedmann SL. Long-term functional results after manipulation of the frozen shoulder. Md Med J. 1999;48:7–11.
15. Dahan TH, Fortin L, Pelletier M, et al. Double blind randomized clinical trial examining the efficacy of bupivacaine suprascapular nerve blocks in frozen shoulder. J Rheumatol. 2000;27:1329–31.
16. Jankovic D. Subscapular nerve block & subscapularis trigger point infiltration. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. 3rd ed. Boston: Blackwell Scientists Oxford; 2004. p. 128–32.
17. Jankovic D, van Zundert A. The frozen shoulder syndrome. Description of a new technique and five case reports using the subscapular nerve block and subscapularis trigger point infiltration. Acta Anaesthesiol Belg. 2006;57:137–43.
18. Ontjes DA. Adrenal corticosteroids, corticotrophin releasing hormone, adrenocortico-tropin and anti-adrenal drugs. In: Munson PL, editor. Principles of pharmacology. New York: Chapmann & Hall; 1995. p. 764–5.
19. Seeman PM. Membrane stabilization by drugs: tranquilizers, steroids and anesthetics. Int Rev Neurobiol. 1996;9:145–221.
20. Devor M, Govin-Lippman R, Raber P. Corticosteroids suppress ectopic neural discharge originating in experimental neuromas. Pain. 1985;22:127–37.
21. Johansson A, Hao J, Sjölund B. Local corticosteroid application block transmission in normal nociceptive C-fibers. Acta Anaesthesiol Scand. 1990;34:335–8.
22. Gray H. Anatomy of the human body. In: Goss GM, editor. American edition 29. Philadelphia: Lea& Febiger; 1973. p. 455–7.
23. Netter FH. Nervengeflechte und periphere Nerven. In: Krämer G, editor. Farbatlanten der Medizin (Band 5). Nervensystem I: Neuroanatomie und Physiologie. Stuttgart/New York: Georg Thieme Verlag; 1987.
24. Mackinnon SE, Hudson AR, Gentili F, et al. Peripheral nerve injection injury with steroid agents. Plast Reconstr Surg. 1982;69: 482–9.
25. Abram S, O’Conor T. Complications associated with epidural steroid injections. Reg Anesth. 1996;21:149–61.

Part V
Upper Extremity

Chapter 28

Brachial Plexus (Introduction and Anatomy)

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_28,

Introduction

The classical blocks of the brachial plexus using Hirschel's [1] (axillary approach) and Kulenkampff's [2] (supraclavicular block) anesthesia have been continuously developed and supplemented with additional access routes. As representative techniques for a multitude of clinical procedures for plexus anesthesia, the axillary perivascular block [3–5], subclavian perivascular block using the Winnie and Collins technique [6], Winnie's interscalene block [5, 7], and Raj's infraclavicular approach [8] may be mentioned. All of the blocks of the brachial plexus are based on the concept that the nerve plexus lies within a perivascular and perineural space in its course from the transverse processes to the axilla. Like the epidural space, this space limits the spread of the local anesthetic and conducts it to the various trunks and roots. Within the connective-tissue sheath, the concentration and volume of the local anesthetic used determine the extent of the block's spread.

The presence of a well defined sheath varies along the course of the brachial plexus. For example, there is not evidence of a substantial sheath on cryomicrotome sections of the supraclavicular region [9].

The axillary sheath is a collection of connective tissue surrounding the neurovascular structures of the brachial plexus. It is a continuation of the prevertebral fascia separating the anterior and middle scalene muscle. Original descriptions of the sheath considered it to be a dense tubular structure extending from above the first rib to a point distal in the axilla. It was believed that the axillary vessels and nerves were all lying loose within its center, implying that conduction anesthesia of the upper extremity could be performed with a single injection at any site along the sheath, with local anesthetic volume being the primary determinant for successful block [5, 6, 10, 11].

However several investigators have since challenged the concept of a tubular axillary sheath proposing instead that the sheath is a multicompartmental structure formed by thin layers of fibrous tissue surrounding the plexus in firmly membranes and extending inward to create discrete fascial septae. Nerves are thus enmeshed in this tissue rather than lying separate and distinct. As a result, individual fascial compartments are created for each nerve and define the anatomic limits for that neural structure. These compartments could functionally limit the circumferential spread of injected solutions, thereby requiring separate injections into each compartment for maximal nerve blockade. However, proximal connections between compartments have been identified, which may account for the success of single injection techniques. Certain clinical observations may be interpreted as offering support or non-support for the existence of a functional tubular sheath [12–15].

Although the clinical significance of these septa remains controversial, it makes sense to inject local anesthetic in divided doses at several locations within the sheath.

Apart from technical aspects, the main differences between the various block procedures are that the injection is made into the interscalene space, the subclavian space, the infraclavicular space, or the axillary space – leading to different focuses for the block.

In this chapter, four techniques that are among the standard methods for plexus anesthesia will be described: the interscalene, supraclavicular, infraclavicular, and axillary blocks of the brachial plexus.

All of these procedures have well-known *advantages* in contrast with general anesthesia:

- They can be used on an outpatient basis.
- Use in patients with a full stomach, high-risk and emergency patients, and patients who are anxious about general anesthesia.
- Absence of side effects such as nausea and vomiting.
- Absence of postoperative pulmonary complications.
- Excellent postoperative pain control, particularly with the use of long-term local anesthetics (continuous procedures).
- Sympathetic block with vasodilation, better perfusion and faster recovery of traumatized extremities.

Certain points should always be observed when preparing for this procedure:

- Contraindications must be excluded.
- The anatomic relationships in each patient must be precisely studied and studied again for repeated blocks.
- Neurological abnormalities must be excluded.
- The procedure must be explained to the patient in detail in order to ensure cooperation.
- The patient must be placed in a comfortable position during the intervention.
- All patients should be informed of possible side effects and complications; outpatients in particular must also be advised of what they should and should not do after anesthesia or pain treatment.

Anatomy [12–14, 16, 17]

The brachial plexus arises from the union of the spinal nerve roots of C5, C6, C7, C8, and T1, and it often also contains fine fibers from the fourth cervical nerve and second thoracic nerve.

After they have left their intervertebral foramina, the roots of the plexus appear in the interscalene groove between the scalenus anterior and scalenus medius muscles and they join together there to form the primary cords or trunks (Fig. 28.1). The upper roots (C5, C6) form the superior trunk, the roots of C7 continue as the middle trunk, and the inferior trunk arises from the roots of C8 and T1. After passing through the interscalene groove, the primary cords of the plexus, lying close together, move toward the first rib (Fig. 28.2). The suprascapular nerve and subclavian nerve already branch off from the superior trunk here, in the posterior triangle of the neck above the clavicle. When crossing the first rib, the trunks of the plexus lie dorsolateral to the subclavian artery and are enclosed along with the artery by a connective-tissue sheath. The plexus runs through under the middle of the clavicle, following the course of the subclavian artery, into the tip of the axilla (Fig. 28.3). As it does so, each of the primary cords divides into the anterior (ventral) divisions and posterior (dorsal) divisions. These supply the ventral flexor muscles and the dorsal extensor muscles of the upper extremity.

In the axilla itself, the nerve cords regroup and separate into the individual nerves (Fig. 28.4).

The *ventral branches* of the superior and middle trunk combine to form the *lateral cord* (fasciculus lateralis, C5, C6, C7) (Figs. 28.5, 28.6, and 28.9).

The following nerves emerge from this:

- Musculocutaneous nerve (Fig. 28.6)
- Median nerve (lateral root) (Fig. 28.6)
- Lateral pectoral nerve

All of the *dorsal branches* of the three trunks form the *posterior cord* (fasciculus posterior, C5–8, T1). The end branches of this (Figs. 28.4, 28.5, and 28.9) are the:

- Radial nerve (Fig. 28.7)
- Axillary nerve (Fig. 28.7)
- Thoracodorsal nerve (Fig. 28.8)
- Inferior subscapular nerve
- Superior subscapular nerve

The *ventral branches of the inferior trunk* continue as the *medial cord* (fasciculus medialis, C8, T1). The following nerves (Figs. 28.4, 28.5, and 28.9) emerge from this:

- Ulnar nerve (Fig. 28.10)
- Median nerve (medial root) (Figs. 28.6 and 28.10)
- Medial pectoral nerve
- Medial antebrachial cutaneous nerve
- Medial brachial cutaneous nerve

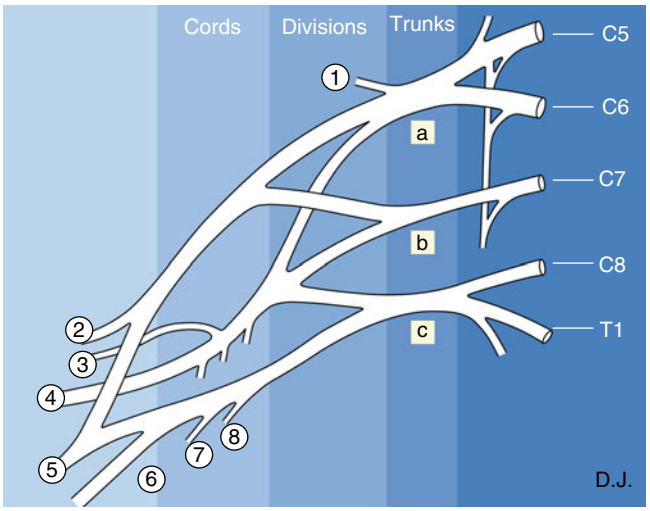


Fig. 28.1 Trunks: (**a**) superior trunk, (**b**) medial trunk, (**c**) inferior trunk, divisions and cords of the brachial plexus. (*1*) Suprascapular nerve, (*2*) musculocutaneous nerve, (*3*) axillary nerve, (*4*) radial nerve, (*5*) median nerve, (*6*) ulnar nerve, (*7*) medial antebrachial cutaneous nerve, (*8*) medial brachial cutaneous nerve (With permission from Danilo Jankovic)

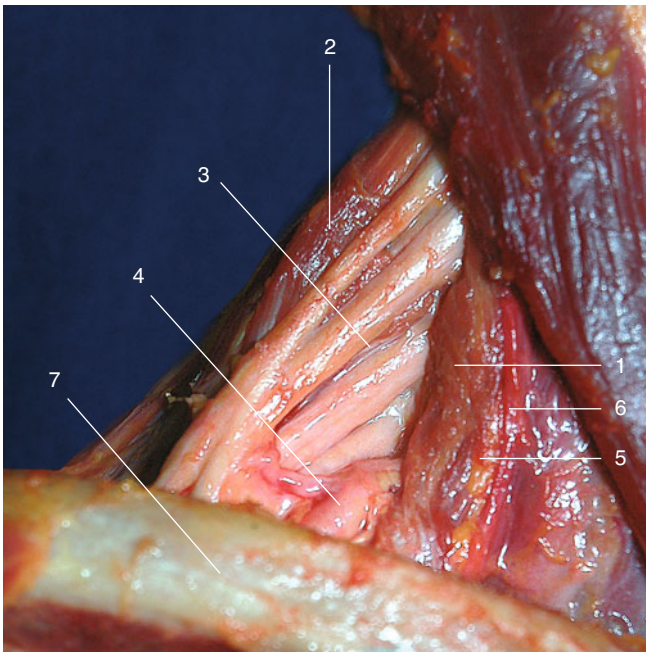


Fig. 28.2 Interscalene region. (*1*) Scalenus anterior muscle, (*2*) scalenus medius muscle, (*3*) trunks of the brachial plexus with subclavian artery, (*4*) proximal supraclavicular plexus sheath, (*5*) phrenic nerve, (*6*) ascendent cervical artery, (*7*) clavicle (With permission from Danilo Jankovic)

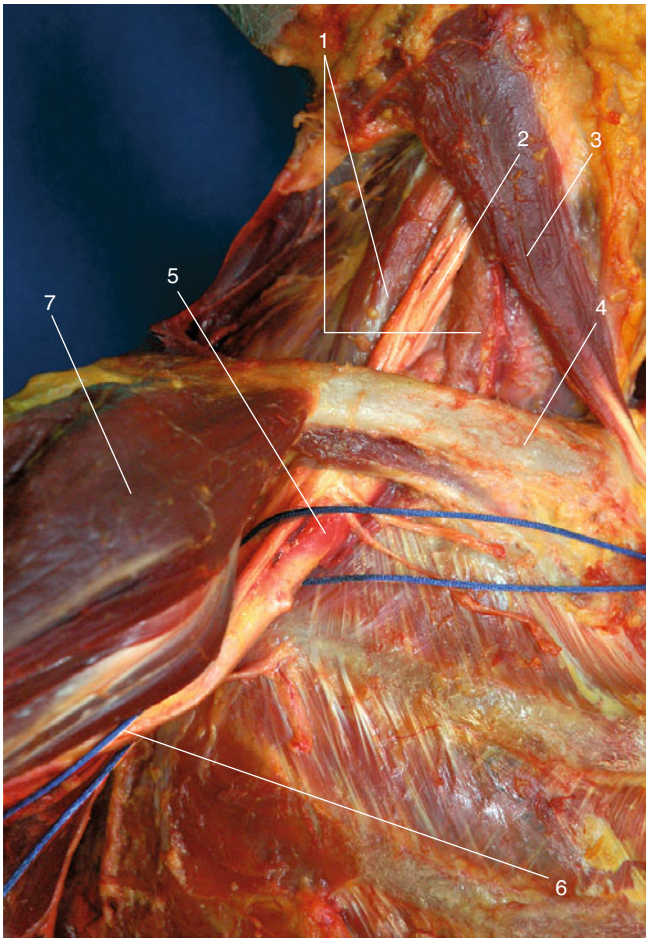


Fig. 28.3 Brachial plexus sheath extending from interscalene to subclavian and axillary region. Pectoralis muscle separated. (*1*) Middle and anterior scalene muscles, (*2*) trunks of the brachial plexus, (*3*) sternocleidomastoid muscle, (*4*) clavicle, (*5*) infraclavicular region, (*6*) axillary part of the brachial plexus, (*7*) deltoid muscle (With permission from Danilo Jankovic)

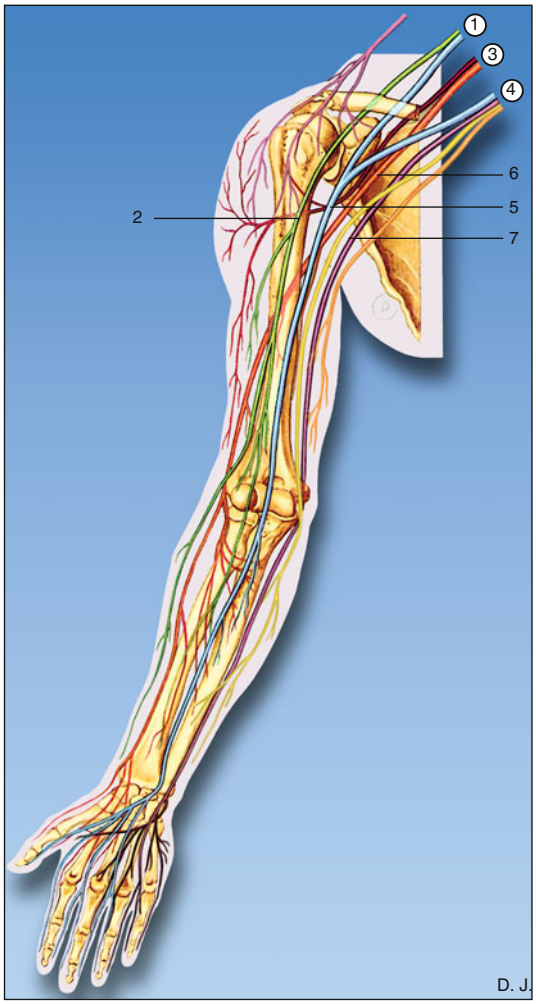


Fig. 28.4 Regrouping of the nerve cords in the area of the axilla and their distal distribution. (*1*) Lateral cord, (*2*) musculocutaneous nerve, (*3*) posterior cord, (*4*) medial cord, (*5*) median nerve, (*6*) radial nerve, (*7*) ulnar nerve (With permission from Danilo Jankovic)

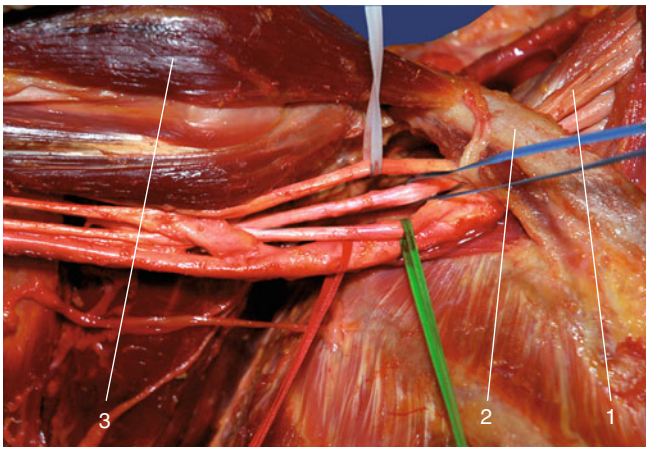


Fig. 28.5 Fascicles (Cords) of the brachial plexus. Lateral cord (*white*), posterior cord (*blue*), medial cord (*green*), axillary artery (*red*). (*1*) Trunks of the brachial plexus above the clavicle (*2*), deltoid muscle (*3*). Pectoralis major muscle separated (With permission from Danilo Jankovic)

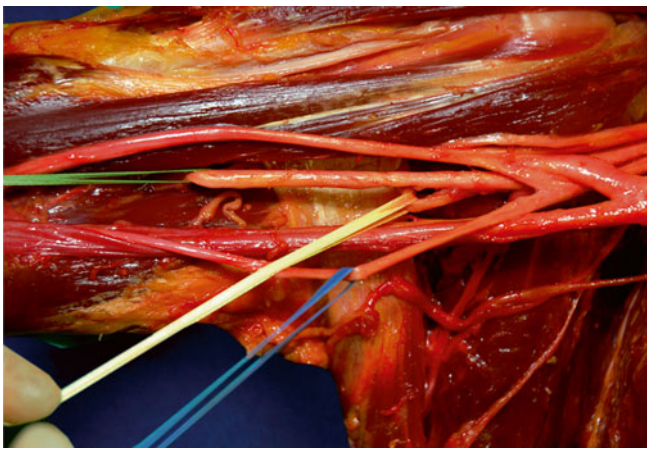


Fig. 28.7 Radial nerve (*green*) and axillary nerve (*yellow*) (With permission from Danilo Jankovic)

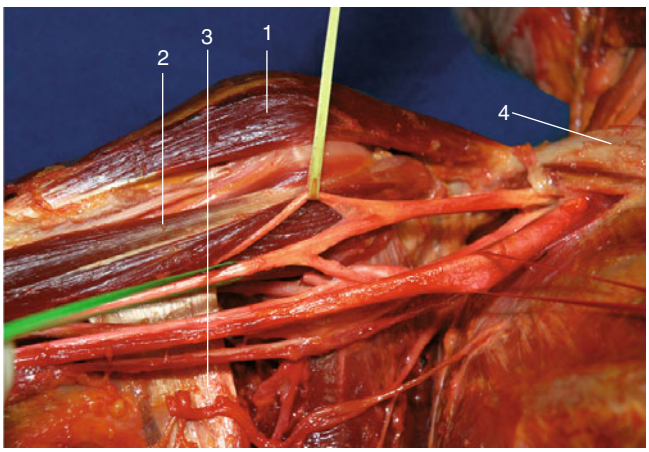


Fig. 28.6 Musculocutaneous nerve (*yellow*), and median nerve (*green*), axillary artery (*red*). (*1*) deltoid muscle, (*2*) biceps brachii muscle and coracobrachialis muscle, (*3*) latissimus dorsi muscle, (*4*) clavicle (With permission from Danilo Jankovic)

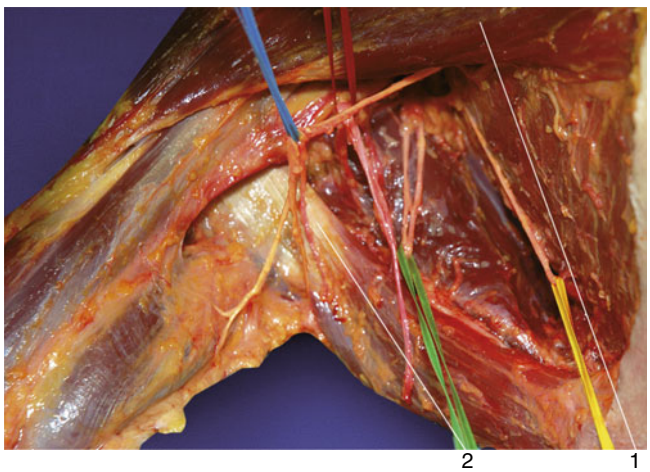


Fig. 28.8 Thoracodorsal nerve (*green*), thoracicus longus nerve (*yellow*), intercostobrachial nerve (*blue*), thoracodorsal artery (*red*). (*1*) pectoralis major muscle, (*2*) latissimus dorsi muscle (With permission from Danilo Jankovic)

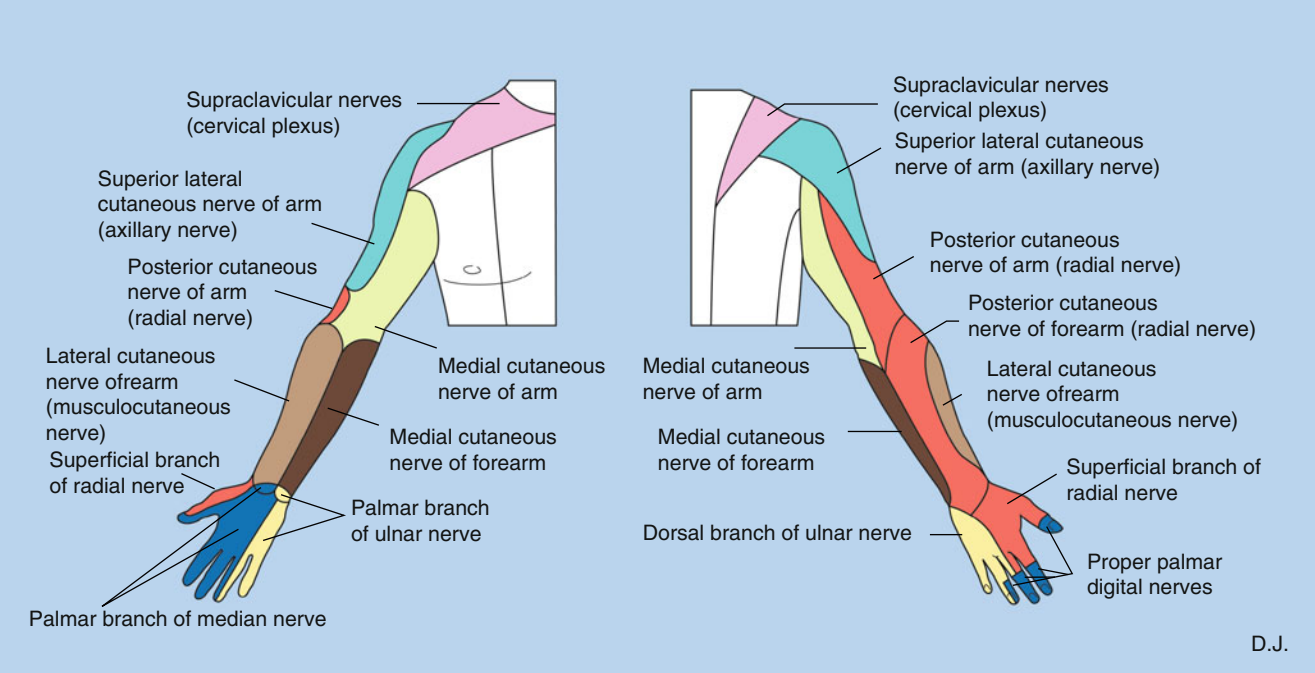


Fig. 28.9 Brachial plexus, cutaneous innervation (With permission from Danilo Jankovic)

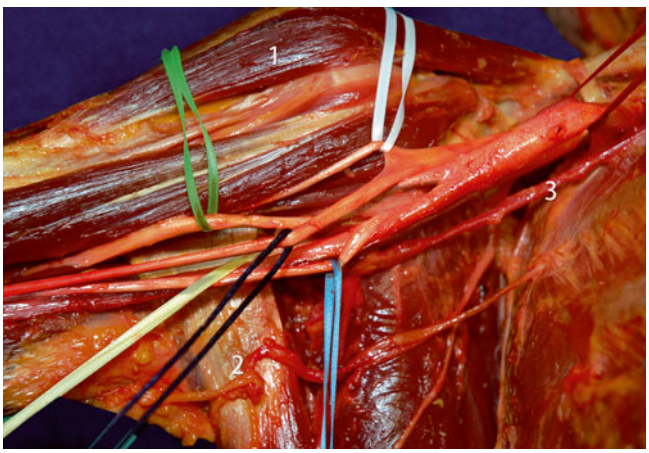


Fig. 28.10 Ulnar nerve (*light blue*), median nerve (*dark blue*), axillary nerve (*yellow*), radial nerve (*green*), musculocutaneous nerve (*white*), axillary artery (*red*). (*1*) deltoid muscle, (*2*) latissimus dorsi muscle, (*3*) axillary vein (With permission from Danilo Jankovic)

Suggested Reading

1. Hirschel G. Anästhesierung des Plexus brachialis bei Operationen an der oberen Extremität. Münchn Med Wschr. 1911;58:1555–6.
2. Kulenkampf D. Die Anästhesierung des Plexus brachialis. Dtsch med Wschr. 1912;38:1878–80.
3. Blanchard J, Ramamurthy S. Brachial plexus. In: Benumof LD, editor. Clinical procedures in anesthesia and intensive care. Philadelphia: Lippincott; 1992.
4. Hickey R, Rogers J, Hoffman J, Ramamurthy S. Comparison of the clinical efficacy of three perivascular techniques for axillary brachial plexus block. Reg Anesth. 1993;18:335–8.
5. Winnie AP, Radonjic R, Akkineni SR, Durrani Z. Factors influencing distribution of local anesthetics injected into the brachial plexus sheath. Anesth Analg. 1979;58:225–34.
6. Winnie AP, Collins VJ. The subclavian perivascular technique of brachial plexus anesthesia. Anesthesiology. 1964;25:353–63.
7. Winnie AP. Interscalene brachial plexus block. Anesth Analg. 1970;49:455–66.
8. Raj PP, Montgomery SJ, Nettles D, Jenkins MT. Infraclavicular brachial plexus block – a new approach. Anesth Analg. 1973;52:897–904.
9. Hogan QH, Ericson SJ. MR imaging of the stellate ganglion. Normal appearance. AJR Am J Roentgenol. 1992;158:655–9.
10. De Jong RH. Axillary block of the brachial plexus. Anesthesiology. 1961;22:215–25.
11. De Jong RH. Modified axillary block. Anesthesiology. 1965;26:615.
12. Neal J, Gerancher JC, Hebl J, Ilfeld B, et al. Upper extremity regional anesthesia. Essentials of our current understanding, 2008. Reg Anesth Pain Med. 2009;34(2):134–71.
13. Pertridge BL, Benirschke F. Functional anatomy of the brachial plexus sheath: implications for anesthesia. Anesthesiology. 1987;66:743–7.
14. Cornisch PB, Greenfield LJ. Brachial plexus anatomy. Reg Anesth. 1997;22:106–7.
15. Thompson GE, Rorie DK. Functional anatomy of the brachial plexus sheaths. Anesthesiology. 1983;59:117–22.
16. Netter FH. Nerve Plexuses and peripheral nerves. In: Netter FH, editor. Nervous system. (Volume 1): neuroanatomy and physiology. The Ciba Collection of Medical Illustrations, 14 Henderson Drive, West Caldwell, NJ 07006. Ciba-Geigy; 1991.
17. Kahle W. Taschenatlas der Anatomie. Band 3. Nervensystem und Sinnesorgane. Stuttgart/New York: Thieme; 2001.

Chapter 29

Brachial Plexus Blocks Above the Clavicle

Michael J. Barrington, MBBS, FANZCA, PhD (✉)
Department of Anesthesia and Acute Pain Medicine, St. Vincent's Hospital, Melbourne, Victoria Pde, PO Box 2900, Fitzroy, VIC 3065, Australia
Faculty of Medicine, Dentistry and Health Sciences, Melbourne Medical School, University of Melbourne, Melbourne, VIC, Australia
e-mail: michaelbarrington84@gmail.com

Anthony Hade
Department of Anesthesia and Acute Pain Medicine, St. Vincent's Hospital, Melbourne, VIC, Australia

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_29,

Interscalene Block

Definition

Interscalene brachial plexus block involves injecting local anesthetic around the trunks of the brachial plexus between the anterior and middle scalene muscles at the level of the cricoid cartilage.

Background

Interscalene brachial plexus block is widely used for postoperative analgesia following open and arthroscopic shoulder surgery [1]. Interscalene block is also effective for analgesia following surgery on the clavicle, although in this context, this is likely related to involvement of the cervical plexus. Interscalene block performed using landmark, paresthesia and nerve stimulator-guided techniques have traditionally used large volumes of local anesthetic. Recently, ultrasound-guided techniques have changed the practice of interscalene brachial plexus block. Ultrasound-guided techniques including interscalene block are associated with superior success rates, and both reduced side effects and local anesthetic requirements when compared to traditional techniques [2–4]. For these reasons, this chapter will largely focus on ultrasound technology as the localizing and monitoring technique used for interscalene block. Interscalene block is suitable for both single-injection and continuous techniques.

Anatomy

The brachial plexus is formed from the ventral rami of the C5–C8 and T1 spinal nerves. The roots located within or close to the intervertebral foramen form trunks located between the anterior and middle scalene muscles (the C5–C6 rami unite to form the superior trunk near the medial border of the middle scalene muscle). The anterior and middle scalene muscles run obliquely between the anterior and posterior tubercles (respectively) of the cervical transverse processes and the first rib. The space between the scalene muscles is often described as the interscalene groove and both the brachial plexus (roots, trunks and divisions) and the subclavian artery are located between the muscles. The interscalene approach to the brachial plexus targets the trunks of the brachial plexus between the anterior and middle scalene muscles. Landmark and nerve stimulator techniques use the cricoid cartilage to determine the C6 level. The main targets are the upper (C5, C6 origins) and middle trunks (C7 origin). The neural elements of brachial plexus at this level do not have an extensive connective tissue component compared to a distal peripheral nerve complex. The brachial plexus is deep to the prevertebral layer of the deep cervical fascia, which attaches to the anterior tubercles of the C3–C6 vertebrae. Anatomical variations of the brachial plexus include it being located in the anterior scalene muscle. Medial to the anterior scalene muscle is the carotid sheath and its contents and the entry of the vertebral artery into the transverse foramen of the C6 vertebrae. Deep to the carotid sheath is loose areolar tissue separating it from the prevertebral fascia and the longus colli muscle. Anatomical surface landmarks include the interscalene groove, sternocleidomastoid muscle, and cricoid cartilage. Vascular structures in this region include the superficial cervical artery and vein (usually superficial to the prevertebral fascia) and the external jugular vein [5].

Indications

Surgical

Interscalene brachial plexus block is commonly used for postoperative analgesia following open and arthroscopic shoulder surgery [1]. It is also effective for surgery on the

clavicle although the effectiveness of interscalene block for this surgical type is likely related to involvement of the cervical plexus. Interscalene block can be used for surgical anesthesia reducing the risk of side effects associated with general anesthesia [6].

Therapeutic

Interscalene brachial plexus block has also been used for chronic conditions involving the shoulder including frozen shoulder and other conditions where the block facilitates physiotherapy.

Evidence and Safety

The versatility of interscalene brachial plexus block is demonstrated by a study by Roxeborough and colleagues where regional anesthesia was the main component of the intraoperative anesthetic technique, potentially reducing the hemodynamic effects of the sitting position by avoiding general anesthesia and/or positive pressure ventilation [6]. Compared to patients having single-injection interscalene block or general anesthesia, patients receiving continuous interscalene block for outpatient rotator cuff repair surgery had improved pain scores at 7 days following surgery. This study demonstrated intermediate benefits of a continuous technique beyond the early postoperative period and 48 h of local anesthetic infusion [7]. Interscalene brachial plexus block has been subject to large-scale studies investigating quality and safety [1, 8–15].

Contraindications

General

Local infection and skin disease
Patient refusal

Relative

Coagulopathy
Reduced pulmonary reserve

Absolute

Contralateral interscalene block
Contralateral paresis of the recurrent laryngeal or phrenic nerve
Contralateral pneumonectomy or pneumothorax

Advantages/Disadvantages

Advantages

Relatively straightforward anatomy and superficial targets even in obese subjects.
Ultrasound-guided technique can be successful with low dosage techniques.
Suitable for continuous catheter techniques.

Disadvantages

Phrenic nerve blockade is a common side effect.
Being close to the neuraxis introduces specific risks.
The C8 and T1 ventral rami are incompletely anesthetized resulting in incomplete blockade of the medial border of the forearm and hand.

Procedure

Preparation

As with all regional anesthesia procedures, requirements include emergency equipment, monitoring, and assistance.

Materials and Disposables

A 25-mm high-frequency (e.g., 10–13 MHz) linear array transducer is suitable.

Sterile ultrasound probe cover and gel for all procedures.
Routine disposables including fenestrated drape and dressings.

22-gauge 50 mm short-bevel needle for single-injection and 18-gauge Tuohy needle for a continuous catheter technique.

Ten to fifteen milliliter of local anesthetic for block (see section on "Local anesthetic dosage, volume and spread")

Patient Positioning

For single-injection technique, supine with head turned to contralateral side.

For a continuous catheter technique, the patient is placed in the lateral position and a posterior approach is used.

Ergonomics and Pre-scanning

We suggest positioning the ultrasound machine on the opposite side of the bed/trolley at the level of the patient's mid-torso so that the operator looks over the patient's shoulder to directly face the screen. The operator can then position the probe with his/her nondominant hand and advance the needle in-plane using his/her dominant hand. The advantage of these ergonomics is that there is a direct line of sight from the operator's eye over the probe and the in-plane needle toward the ultrasound screen. The same configuration works for both left-sided and right-sided blocks and for both left-handed and right-handed operators.

A sonogram required for an interscalene block is relatively straightforward to obtain and this is in part related to the superficial location of key structures. Figure 29.1a, b demonstrate the approximate transducer positions in the posterior triangle of the neck for the interscalene and supraclavicular approaches respectively and their corresponding sonograms. There are two techniques for assisting in identification of the sonoanat-

omy of the interscalene brachial plexus block. We recommend identifying the sonoanatomy of the supraclavicular block in the supraclavicular fossa (Fig. 29.1b) and then tracing the plexus proximally (Fig. 29.1a). Alternatively, the sternocleidomastoid may be identified overlying the carotid artery and internal jugular vein and from this position the transducer may be slid laterally. The sternocleidomastoid tapers laterally, and the scalene muscles can be located just deep to its posterior border (Fig.29.1c). Dynamic scanning from the divisions of the brachial plexus proximally to the level of the trunks (and vice versa) is effective for training in imaging the brachial plexus [16]. Note that the transducer movement required to obtain these distinct sonograms is slight (Fig. 29.1a, b). As the scalene muscles course distally, progressively more muscle fibers join from the transverse processes of the lower cervical vertebrae. Consequently, the scalene muscles may be best visualized on ultrasound more caudal than the landmark technique at the level of the cricoid cartilage (C6). This explains why ultrasound-guided interscalene block is often performed with a more caudal needle insertion point compared to landmark techniques. The needle trajectory is commonly a lateral to medial in-plane approach (Fig. 29.1a). This is a significant departure from the landmark technique where the needle entry point for is more cephalad and medial.

The ultrasound appearance of the plexus at the interscalene level can be described as multiple [3, 4] hypoechoic round or oval structures. The neural structures at the interscalene level have reduced connective tissue compared to the divisions; therefore, the targets for interscalene blockade are relatively hypoechoic. Proximal to the interscalene level, the roots of the brachial plexus can be identified adjacent to the transverse processes. The morphology of the cervical transverse processes determines cervical level and therefore the neural origins. The C6 transverse process has a prominent anterior tubercle, and the C7 transverse process has no anterior tubercle. Color Doppler is recommended to scan for vessels in the planned needle trajectory. The vertebral artery may be imaged close to the C7 transverse process deep to the scalene muscles. Other vessels imaged in the neck include the inferior thyroid, transverse cervical (and its descending dorsal scapular branch) and suprascapular arteries. The C5 transverse process has tubercles of even size producing a symmetrical "U" shape on the sonogram. The phrenic nerve can be imaged as a small hypoechoic structure anterior to the anterior scalene muscle only a few mm away from the brachial plexus [17].

Injection Technique: General Comments

For a single shot technique, sterile gloves should be worn and skin asepsis adhered to. For continuous catheter technique, we recommend a full aseptic technique and barrier precautions (gown, gloves, and facemask). Interscalene block is suitable for either in-plane or out-of-plane techniques. Standard strategies to improve in-plane needle imaging are important and particular vigilance must be taken to keep the needle tip in view.

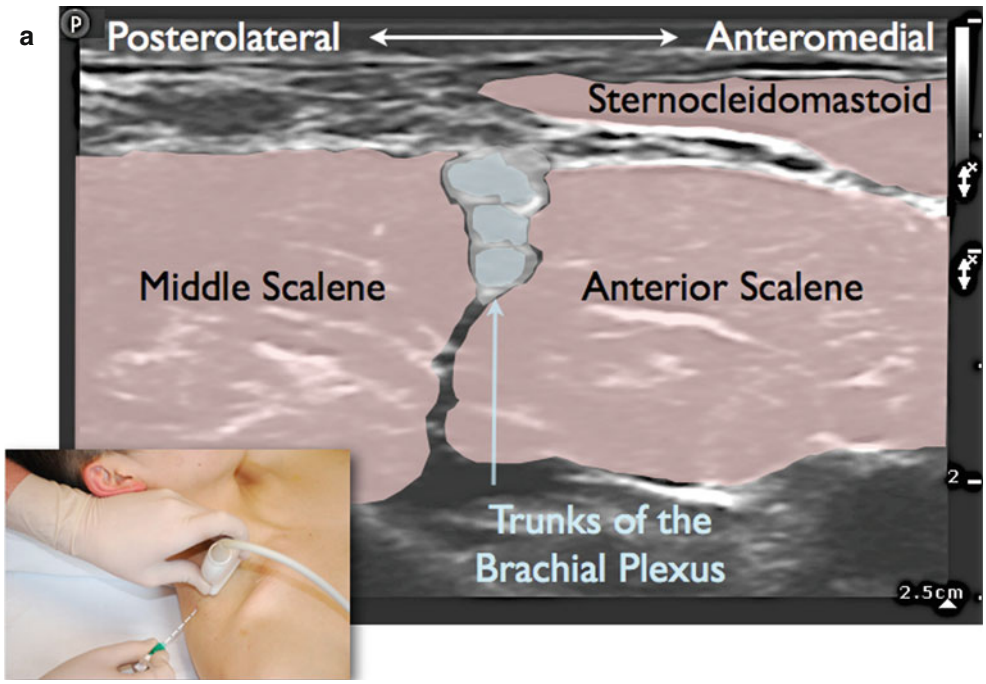


Fig. 29.1 (**a**) Transducer position and corresponding sonogram for the interscalene approach to the brachial plexus. (**b**) Transducer position and corresponding sonogram for the supraclavicular approach to the brachial plexus. (**c**) The great vessels of the neck can be used as a readily identifiable sonoanatomical landmark by which the sternocleidomastoid and scalene muscles (and therefore the brachial plexus) can be located (The inset figure showed the position of the ultrasound probe and the direction of the needle insertion)

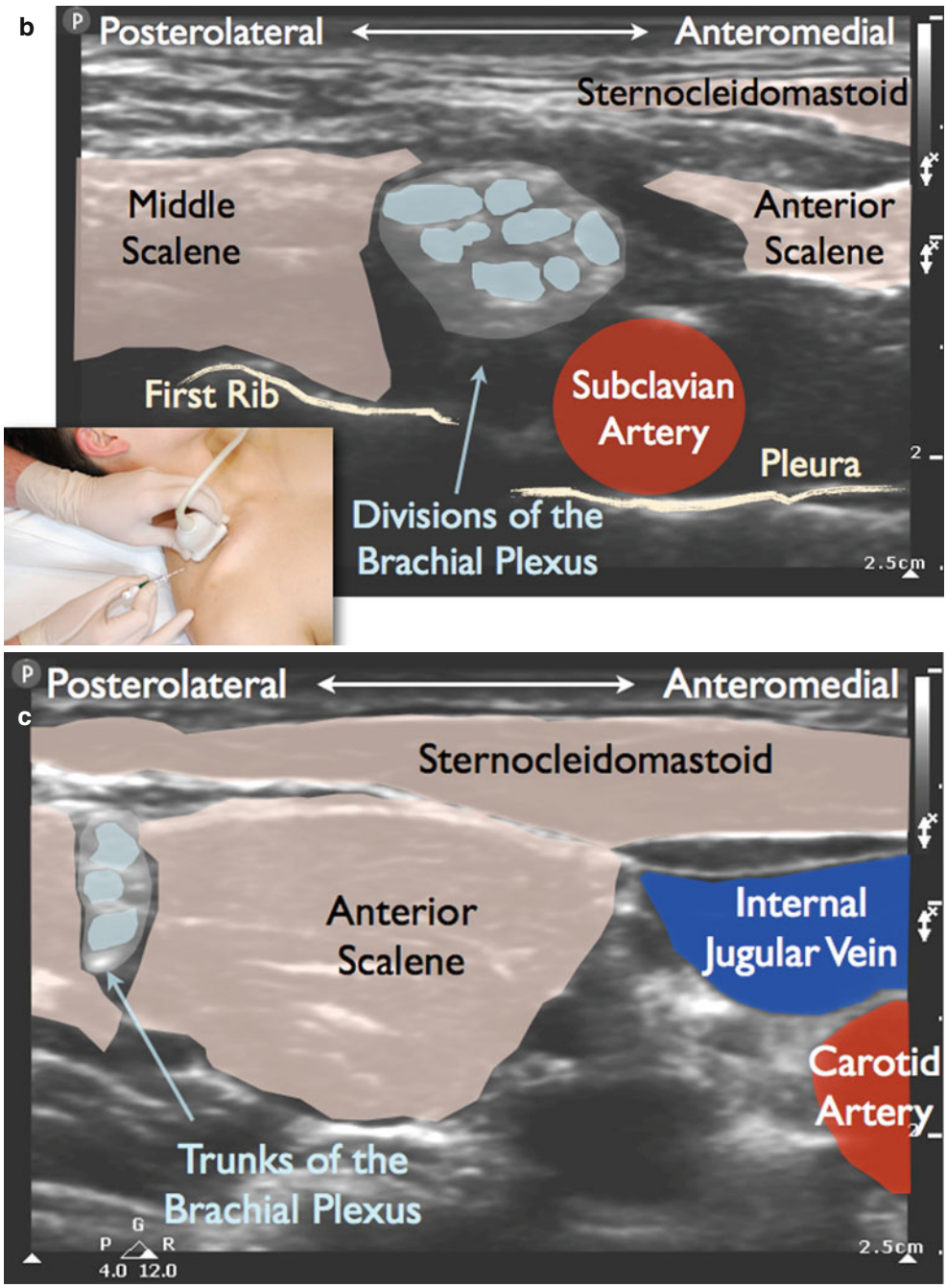


Fig. 29.1 (Continued)

Ultrasound-Guided Approaches

Single-Injection Techniques

The authors' preferred needle trajectory is from lateral to medial (through the middle scalene muscle) using an in-plane technique (Fig. 29.1a). It is sometimes necessary to adopt a brief steep initial needle trajectory in order to pierce the prevertebral layer of the deep cervical fascia that overlies the middle scalene muscle. Once this "pop" has been felt, the needle should be flattened out and advanced toward the plexus. In the authors' experience ultrasound guidance alone is adequate and nerve stimulator guidance is not usually required. The needle is advanced, and a second "pop" may be felt as the needle tip passes through the anterior border of the middle scalene muscle and the plexus. Incremental injection of 1–2 mL of local anesthetic can be used at this point to hydrodissect the plane between the muscles and plexus. Care should be taken to avoid local anesthetic being deposited inside the belly of the middle scalene muscle. Injection of local anesthetic on one side of the trunks at the C5–C7 level should be sufficient.

Continuous Catheter Techniques

The authors' preferred needle trajectory is similar to single-injection techniques except the ultrasound transducer is rotated slightly and translated so that the needle entry point is positioned more cephalad and posterior taking the catheter skin entry point away from the surgical site. The needle trajectory now has a slight caudad angulation. Separating the needle skin entry point increases ease of needle visualization and also creates a tunnel for the catheter, reducing risk of dislodgement. Figure 29.2 demonstrates a patient positioned on their side for an interscalene catheter placement using a posterior in-plane approach. Figure 29.3a demonstrates the needle trajectory with needle at muscle–plexus interface and Fig. 29.3b the post-injection displacement of the middle scalene muscle. The sonogram in Fig. 29.3c shows the catheter following removal of needle. Note that the rotation of the probe may distort the image such that the trunks of the brachial plexus are less well demarcated and the sternocleidomastoid and middle scalene muscles appear elongated. The catheter tip position should be determined before commencing a local anesthetic infusion.

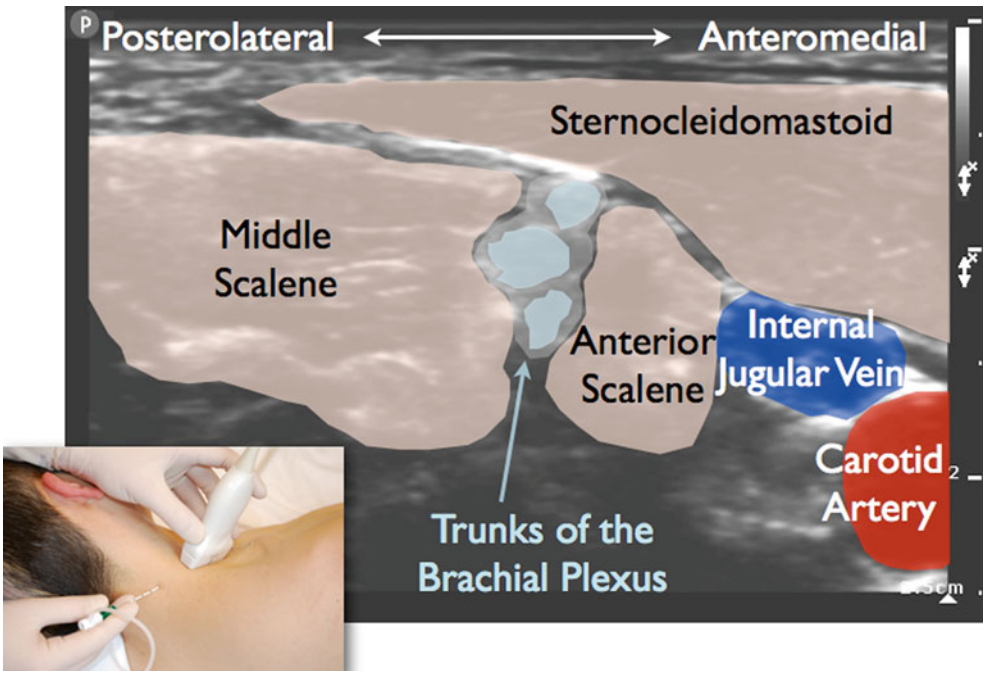


Fig. 29.2 When inserting a continuous interscalene catheter for shoulder surgery, it is desirable to maximize the distance between the needle insertion point and the surgical site (The inset figure showed the position of the ultrasound probe and the direction of the needle insertion)

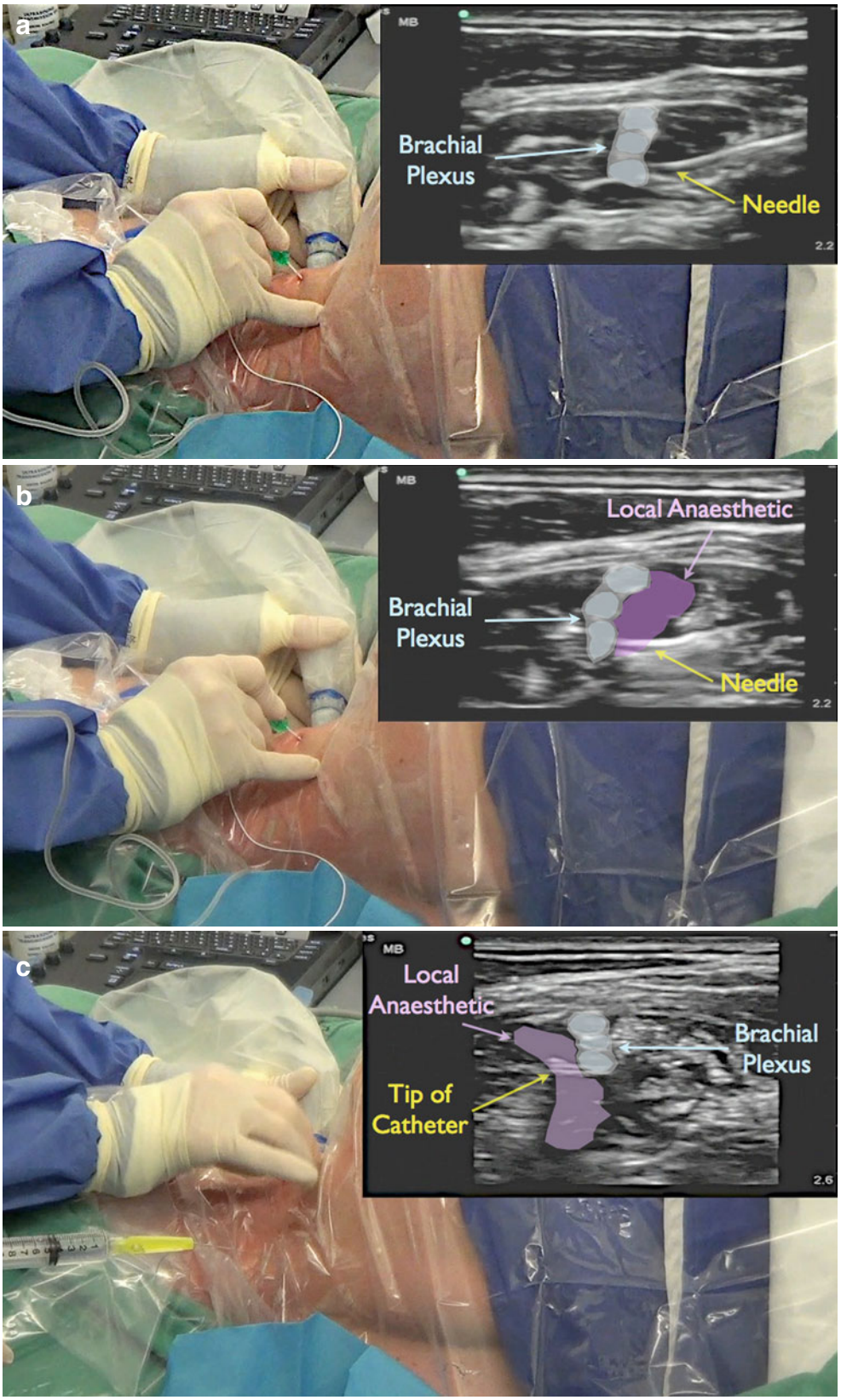


Fig. 29.3 (**a**) Continuous interscalene catheter technique with needle tip located at the anterior border of the middle scalene muscle just posterior to the trunks of the brachial plexus. (**b**) Local anesthetic has been injected separating the trunks of the brachial plexus from the middle scalene muscle. (**c**) A catheter has been threaded beyond the brachial plexus and withdrawn and its position confirmed by injecting through the catheter confirming spread of injectate around the trunks of the plexus (The inset figures showed the ultrasound images corresponded to the stages of the procedure)

Local Anesthetic Dosage, Volume, and Spread

Dosage

The ED_{95} required to achieve postoperative analgesia for C7 interscalene brachial plexus anesthesia has been calculated to be 3.6 mL of ropivacaine 0.75 % [18]. In routine practice, 10–15 mL of local anesthetic such as ropivacaine 0.2–0.75 % is appropriate. The exact concentration will depend on patient factors, the purpose of the block (analgesia or anesthesia), and if a catheter technique is used. If a catheter is placed, the initial dose can be reduced and additional local anesthetic can be injected through the catheter. An infusion of ropivacaine 0.2 % 4–10 mL/h is used for postoperative analgesia.

Distribution

The expected distribution of anesthesia following interscalene brachial plexus block is shown in Fig. 29.4 [19].

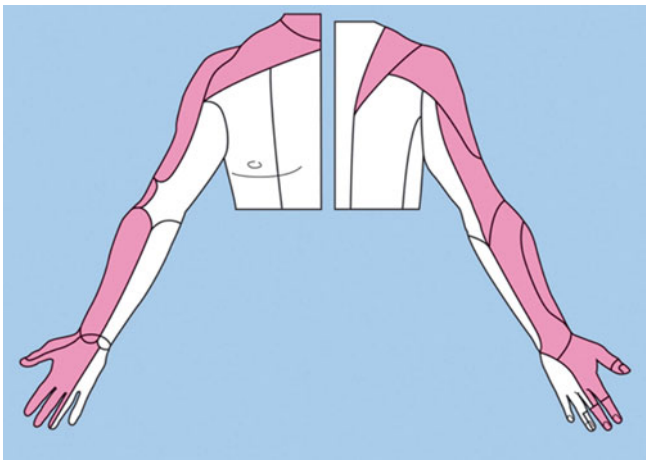


Fig. 29.4 Expected distribution of anesthesia following interscalene brachial plexus block (With permission from Danilo Jankovic)

Side Effects

Phrenic Nerve Block with Ipsilateral Hemidiaphragmatic Paralysis and Dyspnea

Phrenic nerve block occurs commonly following both interscalene and supraclavicular approaches to the brachial plexus. The proposed mechanism is that local anesthetic spreads across the anterior surface of the anterior scalene muscle and involves the phrenic nerve and/or there is cephalad spread toward C4. However, other mechanisms have been proposed including the existence of variations in the origin of the phrenic nerve from the brachial plexus itself [20, 21]. Usually phrenic nerve block is not of concern and management comprises patient reassurance. In patients with reduced pulmonary reserve, phrenic nerve block may be of concern; therefore strategies have been suggested to reduce the risk of phrenic nerve blockade. These strategies include dose reduction, using a more caudal target (C7), using ultrasound guidance, or avoiding interscalene block [22]. Following both interscalene and supraclavicular brachial plexus blockade, ultrasound guidance is associated with a reduced incidence of phrenic nerve paresis compared to nerve stimulator-guided techniques [23, 24].

Recurrent Laryngeal Nerve Block

This may present as hoarseness, occasionally stridor and a sensation of a lump in the throat. A case report has been published where it was thought that a deficient carotid sheath predisposed to recurrent laryngeal nerve block following continuous interscalene block [25].

Cervicothoracic Sympathetic Block

The cervicothoracic sympathetic trunk lies posterolaterally to the prevertebral fascia anterior to the longus colli muscle [26]. This is in close proximity to interscalene or supraclavicular brachial plexus block injection, hence involvement of the sympathetic trunk resulting in Horner's syndrome.

Complications

Pneumothorax

Pneumothorax should be rare with ultrasound-guided interscalene block. Strategies have been suggested to reduce the risk of pneumothorax following ultrasound-guided supraclavicular blockade [27].

Contralateral Spread of Local Anesthetic (Epidural or Intrathecal)

Contralateral spread is a rare complication of interscalene brachial plexus blockade [28, 29]. There may be more than one mechanism responsible for contralateral spread including spread in the tissue planes deep to the prevertebral fascia [30]. The risk of contralateral spread may be increased with increased exposure to the total dosage of local anesthetic (initial bolus and subsequent boluses or infusion). This complication should be taken into account when utilizing these techniques especially in the ambulatory setting.

Catheter Misplacement

Catheter location can be confirmed with a test dose and potentially ultrasound guidance.

Cervical Spinal Cord Injury

The risks associated with performing a procedure close to the neuraxis are clear with interscalene blockade. Permanent neurological injury has been reported following injection of local anesthetic into the cervical spinal cord when an interscalene block was performed under general anesthesia [31].

Vascular Injection

Injection of local anesthetic into the vertebral artery or branches of the thyrocervical trunk can lead rapidly to local anesthetic toxicity.

Nerve Injury

Postoperative nerve injury regardless of etiology is of concern to patients and health-care providers. The reader should be aware that the methods used to capture, define, and report neurologic outcomes vary considerably. A single-center study reported the incidence of postoperative neurologic symptoms including those following interscalene block greater than 6 months duration was 0.9 per 1,000 blocks (95 % confidence interval, 0.5–1.7) [13]. Observational studies consistently report that postoperative neurologic dysfunction may be related to patient and surgical factors and that the incidence of serious permanent neuropathy directly related to peripheral regional anesthesia is rare. Neurologic complications include brachial plexopathy or injury to the dorsal scapular or long thoracic nerve. These nerves pass through or close to the middle scalene muscle and therefore ultrasound-guided interscalene block with a needle trajectory through this muscle, potentially introduces a new risk. Permanent phrenic nerve palsy is also a recognized although likely rare complication of interscalene brachial plexus block [32]. Distal mononeuropathies (involving the median and ulnar nerve) can occur following shoulder surgery and interscalene block, and these complications should be carefully evaluated taking into account all possible causes. Neurologic deficits in the C5–C6 distribution are more likely to be block related, compared to deficits in the ulnar distribution [9].

Documentation

The following documentation is recommended following interscalene blockade: (1) needle type, length, and gauge; (2)

aseptic precautions, (3) anatomical, structures imaged; (4) monitoring techniques, verbal contact with a lightly sedated responsive patient, ultrasound guidance, nerve stimulation, injection pressure monitoring; (5) in-plane versus out-of-plane technique; (6) spread of local anesthetic; (7) local anesthetic dosage; and (8) complications.

Supraclavicular Block

Definition

Supraclavicular brachial plexus block involves injecting local anesthetic around the divisions of the brachial plexus deep to the prevertebral fascia posterolateral to the subclavian artery.

Background

Supraclavicular brachial plexus block was described in the early twentieth century by Kulenkampff and Persy [33] and was initially popular providing more effective anesthesia than other approaches to the brachial plexus. However, literature reviews and case series suggest an incidence of pneumothorax ranging from 0.5 to 6 % [34] and its popularity reduced [35]. Pneumothorax was the main deterrent to choosing this approach to the brachial plexus. More recently however, due to the popularity of ultrasound-guided regional anesthesia, with the potential to image the needle, brachial plexus, and pleura, the supraclavicular approach to the brachial plexus has increased in popularity as evidenced by case series [36], description of techniques [37], controlled clinical trials [38], and registries reports [11].

Anatomy

The anterior scalene muscle ends as a narrow tendon to insert on the scalene tubercle of the inner border of the first rib. Therefore, at this level, the anterior scalene muscle has a different morphology compared to at the interscalene level. In contrast, the middle scalene muscle inserts into a relatively larger area of the upper surface of the first rib (quadrangular area) and the muscle is relatively fleshy at the supraclavicular level. The prevertebral fascia over the brachial plexus at this level is well formed and distinct. The omohyoid muscle descends from the hyoid bone passes beneath the sternocleidomastoid over the carotid sheath to lie in the posterior triangle. The cylindrically shaped inferior belly of the omohyoid is often imaged and it passes almost horizontally above the level of the clavicle. The posterior triangle of the neck near the supraclavicular brachial plexus contains prominent vascular structures. These vessels include the dorsal scapular and suprascapular arteries (branches of the thyrocervical trunk or subclavian artery) often close to the planned needle trajectory or plexus and in some instances the vessels bisect the plexus [5].

Indications

Surgical

Supraclavicular brachial plexus block is indicated for surgery on the shoulder and entire upper extremity. Potentially supraclavicular block may miss the suprascapular nerve (C5 origin from the upper trunk) and supraclavicular nerve (C4 origin) from the cervical plexus, both important for innervation of the shoulder. Supraclavicular block may be "ulnar sparing" if local anesthetic fails to reach the anterior division of the inferior trunk due to its deep anteromedial location between the subclavian artery and the first rib. However a properly executed supraclavicular block has the potential to anesthetize the entire upper limb. It is suitable for surgical anesthesia or postoperative analgesia.

Evidence and Safety

Supraclavicular block is considered a reliable, fast-onset approach to the brachial plexus, and the term *spinal of the arm* reinforces this concept. However, randomized controlled trials have demonstrated that the supraclavicular approach has similar [38] or reduced [39, 40] efficacy compared to other approaches to the brachial plexus from below the clavicle. Controlled clinical trials also demonstrate that the rate of ulnar nerve sensory block is reduced following ultrasound-guided supraclavicular block compared to other approaches from below the clavicle [40, 41] and that the volume required for sensory blockade is not that different to the volume required using techniques not employing ultrasound guidance [42]. The supraclavicular block has been subject to large-scale studies investigating quality and safety [11, 36, 43].

Contraindications

General

Local infection and skin disease
Patient refusal

Relative

Coagulopathy
Reduced pulmonary reserve

Absolute

Contralateral supraclavicular block
Contralateral paresis of the recurrent laryngeal or phrenic nerve
Contralateral pneumonectomy or pneumothorax

Advantages/Disadvantages

Advantages
Relatively straightforward anatomy
Produces anesthesia/analgesia the entire upper extremity

Disadvantages
Phrenic nerve blockade is a common side effect.
Being close to the pleura introduces specific risk of pneumothorax.
The C8 and T1 ventral rami may have slow onset or incomplete blockade (medial border of forearm or hand).
The posterior triangle of the neck is a rich vascular area.
Not ideal for continuous catheter technique.

Procedure

Preparation
As with all regional anesthesia procedures, requirements include emergency equipment, monitoring, and assistance.

Materials and Disposables
A 25 mm width high-frequency (e.g., 10–13 MHz) linear array transducer is appropriate for most adult patients. Occasionally a morbidly obese patient has a deep target, and an intermediate frequency probe may be helpful for both tissue penetration and to increase the width of the field of view.

Patient Positioning
Supine with head turned to contralateral side

Ergonomics and Pre-scanning
Position the ultrasound machine so that the practitioner directly faces the screen. The exact location and arrangement of the bed/trolley, machine, regional trolley, and operator is influenced by personal preferences and handedness. The authors prefer to hold the needle for this approach with their dominant hand regardless of side blocked. Place the probe immediately cephalad to the clavicle along its lateral border. Slide the probe medially until the subclavian artery is identified as a large pulsatile hypoechoic structure. The brachial plexus between the anterior and middle scalene muscles can be traced from the interscalene to the supraclavicular level (Fig. 29.1a, b) and vice versa [16]. The change in morphology of the neural elements is distinct as the brachial plexus changes from trunks to divisions. The ultrasound appearance at the supraclavicular level can be described as a "bunch of grapes" or as having a "honeycomb" appearance. The plexus is immediately posterior and lateral to the artery. If the plexus is imaged immediately cephalad to the artery, the operator should tilt the transducer caudal so as to image the plexus so that it is posterolateral to the artery. This is the sonogram required for upper extremity anesthesia/analgesia. If the main surgical region is the proximal humerus, then a sonogram with the plexus immediately cephalad to the artery is appropriate.

Ultrasound-Guided Technique

Single-Injection Techniques
The authors' preferred needle trajectory is from lateral to medial using an in-plane approach. As with interscalene block, ultrasound guidance alone without nerve stimulation is adequate.

Continuous Catheter Techniques
In the author's experience, the supraclavicular block is not ideal for a continuous catheter technique. The catheter does not feed well and dislodgement is common at this location. However, a catheter at the more cephalad interscalene location can be directed caudal so that the catheter is located at the level of the divisions (refer to section "Continuous catheter technique," Interscalene brachial plexus block).

Local Anesthetic Dosage, Volume and Spread

Dosage
Surgical anesthesia: 25–30 mL ropivacaine 0.75 %; postoperative analgesia: 20–30 mL ropivacaine 0.2–0.5 %.

Distribution
The expected distribution of anesthesia following supraclavicular brachial plexus block is shown in Fig. 29.5 [19].

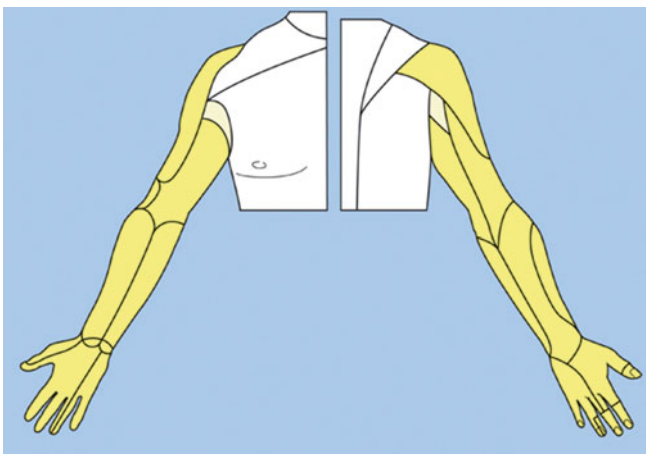


Fig. 29.5 Expected distribution of anesthesia following supraclavicular brachial plexus block (With permission from Danilo Jankovic)

Side Effects

Phrenic Nerve Block with Ipsilateral Hemidiaphragmatic Paralysis and Dyspnea

Phrenic nerve block occurs more commonly following interscalene than the supraclavicular approaches to the brachial plexus. There are variations in the origin of the phrenic nerve including from the brachial plexus itself [21]. Usually phrenic nerve block is not of concern and management comprises patient reassurance. Following both interscalene and supraclavicular brachial plexus blockade, ultrasound guidance is associated with a reduced incidence of phrenic nerve paresis compared to nerve stimulator-guided techniques [23, 24].

Recurrent Laryngeal Nerve Block

This may present as hoarseness, occasionally stridor and a sensation of a lump in the throat.

Cervicothoracic Sympathetic Block

The cervicothoracic sympathetic trunk lies posterolaterally to the prevertebral fascia anterior to the longus colli muscle [26]. This is in close proximity to interscalene or supraclavicular brachial plexus block injection, hence involvement of the sympathetic trunk resulting in Horner's syndrome.

Complications

Pneumothorax

Pneumothorax should be rare with ultrasound-guided techniques. Strategies have been suggested to reduce the risk of pneumothorax following ultrasound-guided supraclavicular blockade [27].

Vascular Injection

This risk relates to the vascular structures such as the dorsal scapular artery in the posterior triangle of the neck.

Nerve Injury

Postoperative nerve injury regardless of etiology is of concern to patients and health-care providers. The reader should be aware that the methods used to capture, define, and report neurologic outcomes vary considerably. A single-center study reported the incidence of postoperative neurologic symptoms including those following supraclavicular block greater than 6 months duration was 0.9 per 1,000 blocks (95 % confidence interval, 0.5–1.7) [13]. Observational studies consistently report that postoperative neurologic dysfunction may be related to patient and surgical factors and that the incidence of serious permanent neuropathy directly related to peripheral regional anesthesia is rare.

Documentation

The following documentation is recommended following supraclavicular blockade: (1) needle type, length, and gauge; (2) aseptic precautions; (3) anatomical, structures imaged; (4) monitoring techniques, verbal contact with a lightly sedated responsive patient, ultrasound guidance, nerve stimulation, injection pressure monitoring; (5) in-plane versus out-of-plane technique; (6) spread of local anesthetic; (7) local anesthetic dosage, and (8) complications.

References

1. Singh A, Kelly C, O'Brien T, Wilson J, Warner JJ. Ultrasound-guided interscalene block anesthesia for shoulder arthroscopy: a prospective study of 1319 patients. J Bone Joint Surg Am. 2012;94(22):2040–6.
2. 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.
3. Antonakakis JG, Ting PH, Sites B. Ultrasound-guided regional anesthesia for peripheral nerve blocks: an evidence-based outcome review. Anesthesiol Clin. 2011;29(2):179–91.
4. Neal JM, Brull R, Chan VW, Grant SA, Horn JL, Liu SS, 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.
5. Muhly WT, Orebaugh SL. Sonoanatomy of the vasculature at the supraclavicular and interscalene regions relevant for brachial plexus block. Acta Anaesthesiol Scand. 2011;55(10):1247–53.
6. Rohrbaugh M, Kentor ML, Orebaugh SL, Williams B. Outcomes of shoulder surgery in the sitting position with interscalene nerve block: a single-center series. Reg Anesth Pain Med. 2013;38(1):28–33.
7. Salviz EA, Xu D, Frulla A, Kwofie K, Shastri U, Chen J, et al. Continuous interscalene block in patients having outpatient rotator cuff repair surgery: a prospective randomized trial. Anesth Analg. 2013;117(6):1485–92.
8. Borgeat A, Ekatodramis G, Kalberer F, Benz C. Acute and nonacute complications associated with interscalene block and shoulder surgery: a prospective study. Anesthesiology. 2001;95(4):875–80.
9. Candido KD, Sukhani R, Doty Jr R, Nader A, Kendall MC, Yaghmour E, et al. Neurologic sequelae after interscalene brachial plexus block for shoulder/upper arm surgery: the association of patient, anesthetic, and surgical factors to the incidence and clinical course. Anesth Analg. 2005;100(5):1489–95, table of contents.
10. Christ S, Rindfleisch F, Friederich P. Superficial cervical plexus neuropathy after single-injection interscalene brachial plexus block. Anesth Analg. 2009;109(6):2008–11.
11. Liu SS, Gordon MA, Shaw PM, Wilfred S, Shetty T, Yadeau JT. A prospective clinical registry of ultrasound-guided regional anesthesia for ambulatory shoulder surgery. Anesth Analg. 2010;111(3):617–23.
12. Misamore G, Webb B, McMurray S, Sallay P. A prospective analysis of interscalene brachial plexus blocks performed under general anesthesia. J Shoulder Elbow Surg Am Shoulder Elbow Surgeons et al. 2011;20(2):308–14.
13. 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: an analysis from a prospective clinical registry. Reg Anesth Pain Med. 2012;37(5):478–82.

14. Sviggum HP, Jacob AK, Arendt KW, Mauermann ML, Horlocker TT, Hebl JR. Neurologic complications after chlorhexidine antisepsis for spinal anesthesia. Reg Anesth Pain Med. 2012;37(2):139–44.
15. Weber SC, Jain R. Scalene regional anesthesia for shoulder surgery in a community setting: an assessment of risk. J Bone Joint Surg Am. 2002;84-A(5):775–9.
16. Tsui BC, Lou L. Learning the 'traceback' approach for interscalene block. Anaesthesia. 2014;69(1):83–5.
17. Kessler J, Schafhalter-Zoppoth I, Gray AT. An ultrasound study of the phrenic nerve in the posterior cervical triangle: implications for the interscalene brachial plexus block. Reg Anesth Pain Med. 2008;33(6):545–50.
18. Renes SH, Bruhn J, Gielen MJ, Scheffer GJ, van Geffen GJ. In-plane ultrasound-guided thoracic paravertebral block: a preliminary report of 36 cases with radiologic confirmation of catheter position. Reg Anesth Pain Med. 2010;35(2):212–6.
19. Jankovic D. Interscalene block and supraclavicular block. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Textbook & color atlas. 4th ed. Blackwell / Malden Masachusetts, Oxford, Carlton Victoria, Australia; 2004. p. 82–99.
20. Renes SH, van Geffen GJ, Rettig HC, Gielen MJ, Scheffer GJ. Minimum effective volume of local anesthetic for shoulder analgesia by ultrasound-guided block at root C7 with assessment of pulmonary function. Reg Anesth Pain Med. 2010;35(6):529–34.
21. Bigeleisen PE. Anatomical variations of the phrenic nerve and its clinical implication for supraclavicular block. Br J Anaesth. 2003;91(6):916–7.
22. Verelst P, van Zundert A. Respiratory impact of analgesic strategies for shoulder surgery. Reg Anesth Pain Med. 2013;38(1):50–3.
23. Renes SH, Rettig HC, Gielen MJ, Wilder-Smith OH, van Geffen GJ. Ultrasound-guided low-dose interscalene brachial plexus block reduces the incidence of hemidiaphragmatic paresis. Reg Anesth Pain Med. 2009;34(5):498–502.
24. Renes SH, Spoormans HH, Gielen MJ, Rettig HC, van Geffen GJ. Hemidiaphragmatic paresis can be avoided in ultrasound-guided supraclavicular brachial plexus block. Reg Anesth Pain Med. 2009;34(6):595–9.
25. Shakespeare TJ, Tsui BC. Intermittent hoarseness with continuous interscalene brachial plexus catheter infusion due to deficient carotid sheath. Acta Anaesthesiol Scand. 2013;57(8):1085–6.
26. 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(5):475–9.
27. Abell DJ, Barrington MJ. Pneumothorax after ultrasound-guided supraclavicular block: presenting features, risk, and related training. Reg Anesth Pain Med. 2014;39(2):164–7.
28. Dooley J, Fingerman M, Melton S, Klein SM. Contralateral local anesthetic spread from an outpatient interscalene catheter. Can J Anaesth J Canadien d'anesthesie. 2010;57(10):936–9.
29. Whitaker EE, Edelman AL, Wilckens JH, Richman JM. Severe hypotension after interscalene block for outpatient shoulder surgery: a case report. J Clin Anesth. 2010;22(2):132–4.
30. Fritsch G, Hudelmaier M, Danninger T, Brummett C, Bock M, McCoy M. Bilateral loss of neural function after interscalene plexus blockade may be caused by epidural spread of local anesthetics: a cadaveric study. Reg Anesth Pain Med. 2013;38(1):64–8.
31. Benumof JL. Permanent loss of cervical spinal cord function associated with interscalene block performed under general anesthesia. Anesthesiology. 2000;93(6):1541–4.
32. Kaufman MR, Elkwood AI, Rose MI, Patel T, Ashinoff R, Fields R, et al. Surgical treatment of permanent diaphragm paralysis after interscalene nerve block for shoulder surgery. Anesthesiology. 2013;119(2):484–7.
33. Kulenkampff D, Persky MA. Brachial plexus anesthesia: its indications, techniques and dangers. Ann Surg. 1928;87:883–91.
34. Brown DL, Bridenbaugh LD. The upper extremity: somatic blockade. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 345–70.
35. Wishart HY. Pneumothorax complicating brachial plexus block anaesthesia. Br J Anaesth. 1954;26:120.
36. Perlas A, Lobo G, Lo N, Brull R, Chan VW, Karkhanis R. Ultrasound-guided supraclavicular block: outcome of 510 consecutive cases. Reg Anesth Pain Med. 2009;34(2):171–6.
37. Soares L, Brull R, Lai J, Chan V. Eight ball, corner pocket: the optimal needle position for ultrasound-guided supraclavicular block. Reg Anesth Pain Med. 2007;32(1):94–5.
38. de Tran QH, Russo G, 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(4):366–71.
39. Fredrickson MJ, Patel A, Young S, Chinchanwala S. Speed of onset of 'corner pocket supraclavicular' and infraclavicular ultrasound guided brachial plexus block: a randomised observer-blinded comparison. Anaesthesia. 2009;64(7):738–44.
40. Koscielniak-Nielsen ZJ, Frederiksen BS, Rasmussen H, Hesselbjerg L. A comparison of ultrasound-guided supraclavicular and infraclavicular blocks for upper extremity surgery. Acta Anaesthesiol Scand. 2009;53(5):620–6.
41. Subramanyam R, Vaishnav V, Chan VW, Brown-Shreves D, Brull R. Lateral versus medial needle approach for ultrasound-guided supraclavicular block: a randomized controlled trial. Reg Anesth Pain Med. 2011;36(4):387–92.
42. de Tran QH, Dugani S, Correa JA, Dyachenko A, Alsenosy N, Finlayson RJ. Minimum effective volume of lidocaine for ultrasound-guided supraclavicular block. Reg Anesth Pain Med. 2011;36(5):466–9.
43. Sites BD, Barrington MJ, Davis M. Using an international clinical registry of regional anesthesia to identify targets for quality improvement. Reg Anesth Pain Med. 2014;39(6):487–95.

Chapter 30

Brachial Plexus Blocks Above the Clavicle Traditional Techniques

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_30,

Interscalene Block [3, 4, 8, 9, 23]

Preparations

Check that the emergency equipment is present and in working order: sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, emergency medication, and ventilation facilities.

Materials

Nerve Stimulator

A 50-mm 22-G (15°) short- bevel insulated stimulating atraumatic needle with "immobile needle" injection lead.

Continuous Technique[1]

Anterior technique

- Catether kit: 50-mm 22-G (15°) stimulating atraumatic needle with catheter

or

- Tuohy continuous set: 38 (−52)-mm 18-G Tuohy needle with catheter

Posterior Technique

- Catether kit: 80 (−110)-mm 18-G (15°) stimulating atraumatic needle with catheter
- Tuohy continuous set: 102-mm 18-G Tuohy needle with catheter
- Syringes: 2, 10, and 20 mL.
- Local anesthetics, disinfectant, swabs, compresses, sterile gloves, and drape.

Skin Prep

In all blocks.

General Considerations

Patient Positioning

Supine, with the head turned to the opposite side.

Landmarks

Sternocleidomastoid muscle, interscalene groove between the scalenus anterior and scalenus medius muscles, transverse process (C6), and external jugular vein (Fig. 30.1a, b).

[1]If technical difficulties arise, the catheter and Tuohy puncture needle are always removed simultaneously. A catheter must never be withdrawn through a Tuohy puncture needle that remains in place (because of catheter shearing).

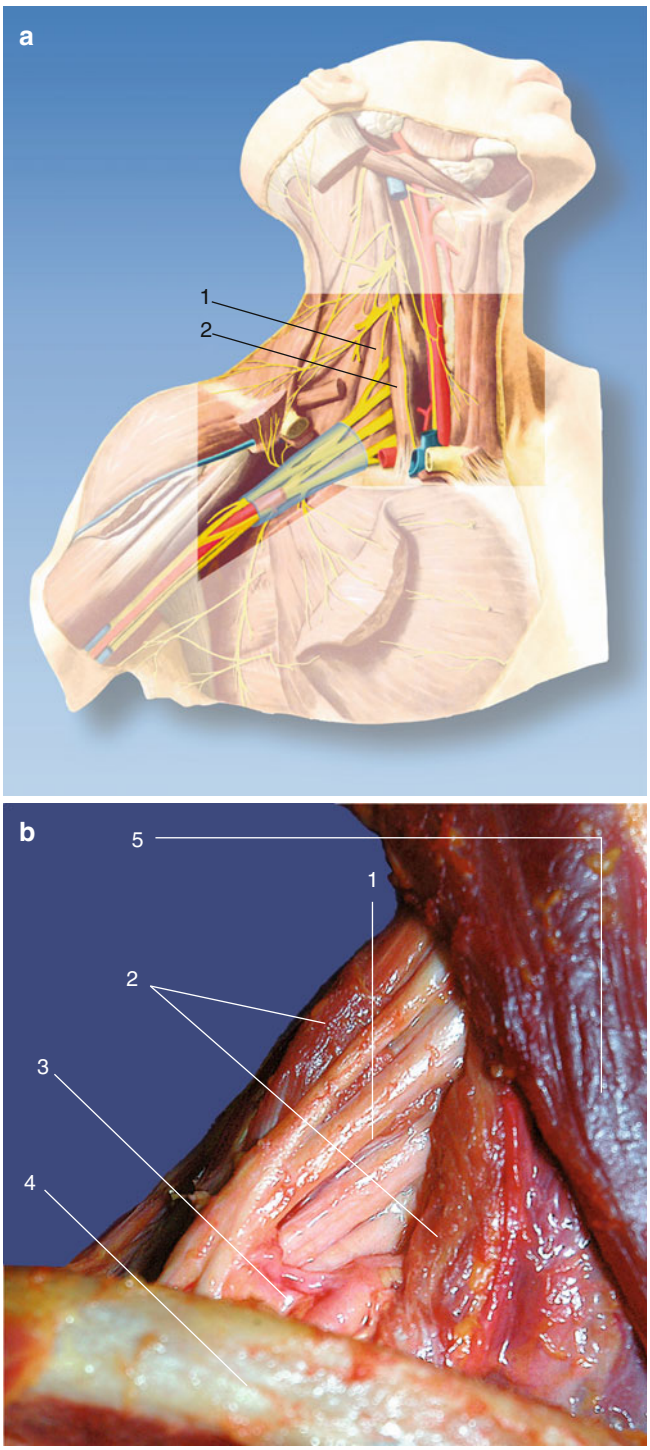


Fig. 30.1 (**a**) The interscalene groove. Scalenus medius muscle (*1*) and scalenus anterior muscle (*2*). Injection of the local anesthetic into the proximal neurovascular sheath of the brachial plexus. The plexus is located in a kind of "sandwich" between the scalenus anterior muscle and scalenus medius muscle (With permission from Danilo Jankovic). (**b**) (*1*) Trunks of the brachial plexus with subclavian artery, (*2*) middle and anterior scalene muscles, (*3*) proximal supraclavicular plexus sheath, (*4*) clavicle, (*5*) strnocleidomastoid muscle (With permission from Danilo Jankovic)

Location of the Puncture Site

To locate the injection site, the patient's arm is drawn in the direction of the knee (Fig. 30.2). The patient is asked to turn the head to the opposite side and to lift it slightly (ca. 20°), so that the posterior edge of the sternocleidomastoid muscle becomes evident (Fig. 30.3). The transverse process (C6) is palpated at the lateral edge of the sternocleidomastoid muscle. For confirmation (pleura) and guidance, the pulsation of the subclavian artery (at the lower end of the interscalene groove) and the upper edge of the clavicle can also be palpated and their distance from the injection site can be estimated (Fig. 30.4).

Posterior to the sternocleidomastoid muscle, the scalenus anterior muscle is palpated. The interscalene groove between the scalenus anterior and scalenus medius muscles is felt with "rolling fingers" and located (Fig. 30.5).

The injection site in the interscalene groove lies at the level of the cricoid, opposite the transverse process of C6 (Chassaignac's tubercle). The external jugular vein often crosses the level of the cricoid cartilage here (Fig. 30.6).

When there are anatomical difficulties, it is helpful for the patient to inhale deeply or to try and blow out the cheeks. The scalene muscles then tense up, and the interscalene groove becomes more easily palpable.

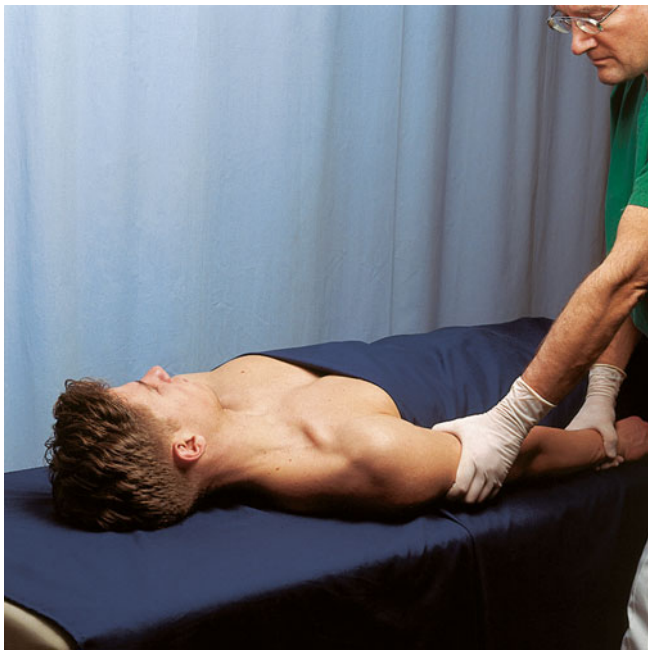


Fig. 30.2 Drawing the arm toward the knee (With permission from Danilo Jankovic)

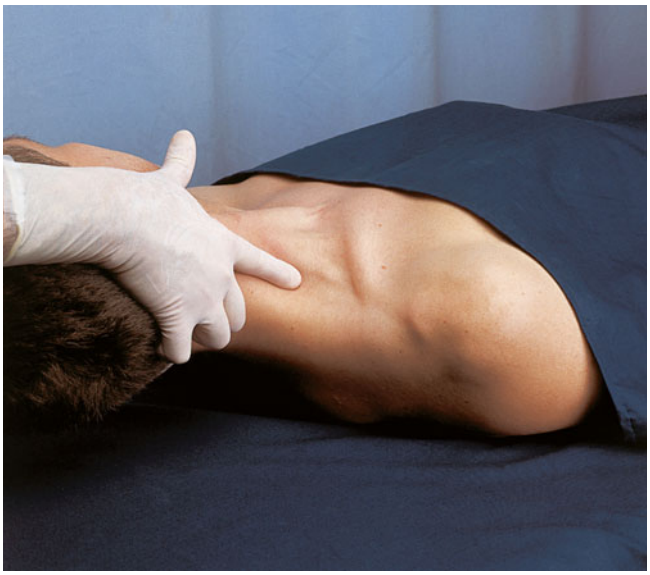


Fig. 30.3 Turning the head to the opposite side and raising it slightly (With permission from Danilo Jankovic)

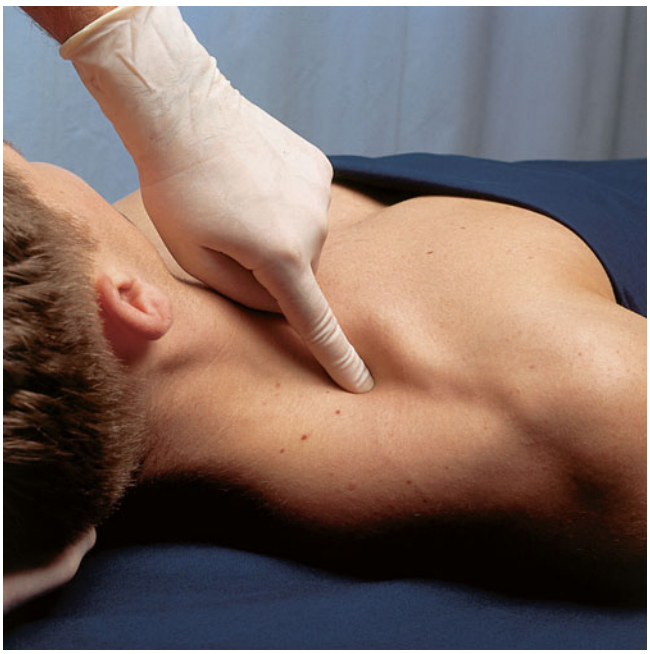


Fig. 30.4 Palpating the clavicle and subclavian artery (With permission from Danilo Jankovic)

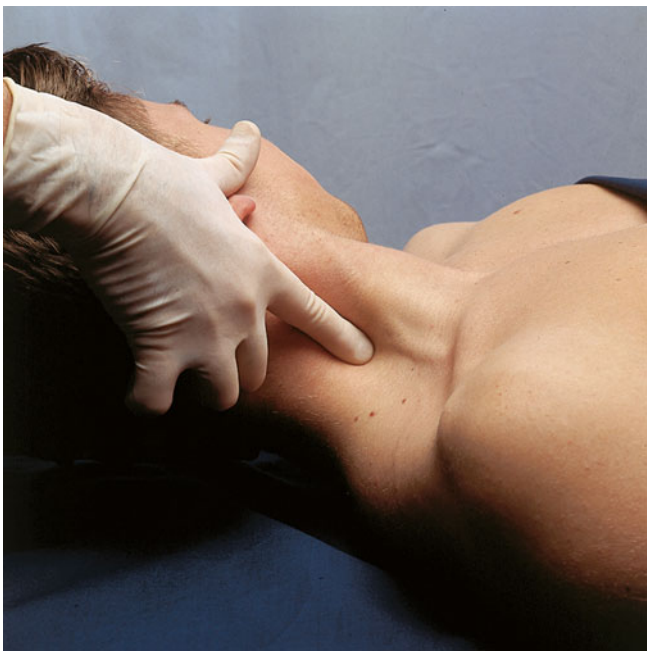


Fig. 30.5 Palpating the interscalene groove with "rolling" fingers (With permission from Danilo Jankovic)

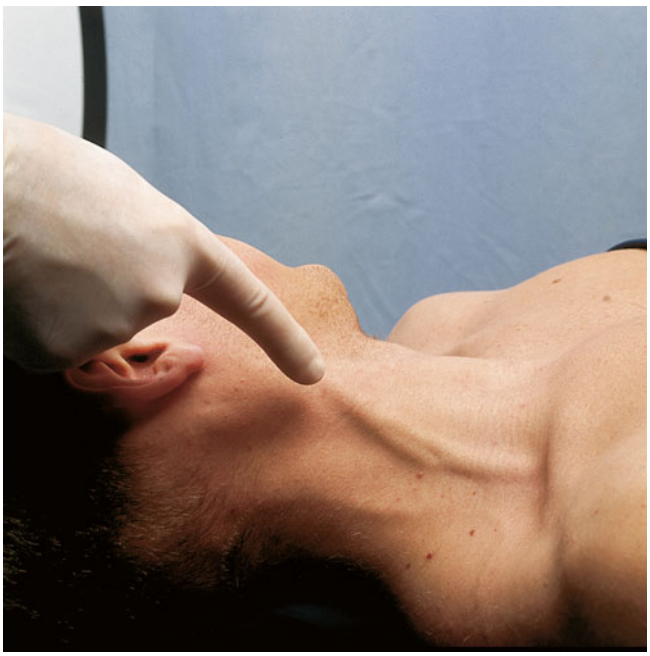


Fig. 30.6 Position of the external jugular vein (With permission from Danilo Jankovic)

Continuous Interscalene Block: Anterior Technique (Adapted from Meier)

Skin Prep

In all blocks.

Patient Positioning

See above the steps for locating the puncture site.

Landmarks (Fig. 30.7)

- Superior thyroid notch
- Posterior edge of the sternocleidomastoid muscle
- Posterior scalene groove
- External jugular vein
- Transition from the middle to lateral third of the clavicle

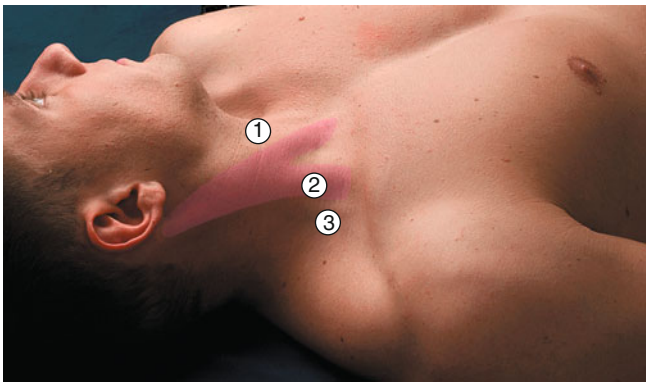


Fig. 30.7 Interscalene block (anterior access route). The posterior edge of the sternocleidomastoid muscle at the level of the superior thyroid notch. (*1*) Superior thyroid notch, (*2*) posterior edge of the sternocleidomastoid muscle, (*3*) external jugular vein and posterior scalene groove (With permission from Danilo Jankovic)

Technique [14]

After identification of the posterior edge of the sternocleidomastoid muscle at the level of the superior thyroid notch, the block needle (55-mm short bevel insulated atraumatic needle or Tuohy needle, 38 or 52 mm) is introduced at an angle of 30° caudally and slightly laterally, in the direction of the transition from the middle to the lateral third of the clavicle (Fig. 30.8). A stimulation current of 1–2 mA and 2 Hz is selected with a stimulus duration of 0.1 ms. After a motor response from the relevant musculature (twitching in the biceps brachii muscle – musculocutaneous nerve and/or deltoid muscle – axillary nerve is regarded to be as reliable as twitching of the distal muscles [23, 27–29], the stimulation current is reduced to 0.2–0.3 mA. Slight twitching suggests that the stimulation needle is in the immediate vicinity of the nerve. The catheter is advanced approximately 3 cm beyond the end of the cannula or needle (Fig. 30.9). After removal of the cannula or needle, fixation of the catheter and placement of a bacterial filter, and after careful aspiration and injection of a test dose, the bolus administration of the local anesthetic follows.

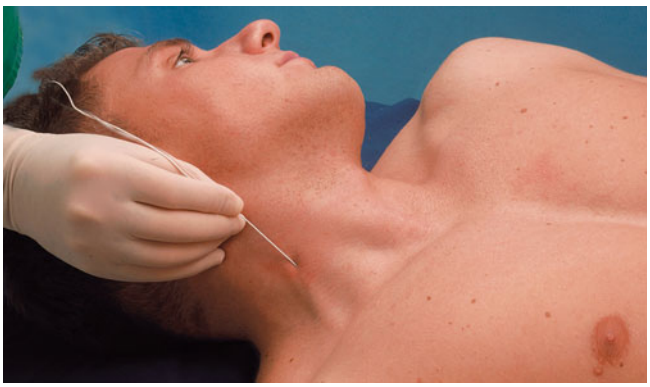


Fig. 30.8 Introducing the puncture needle at an angle of ca. 30° to the skin, caudally and laterally in the direction of the transition from the middle to the lateral third of the clavicle (With permission from Danilo Jankovic)

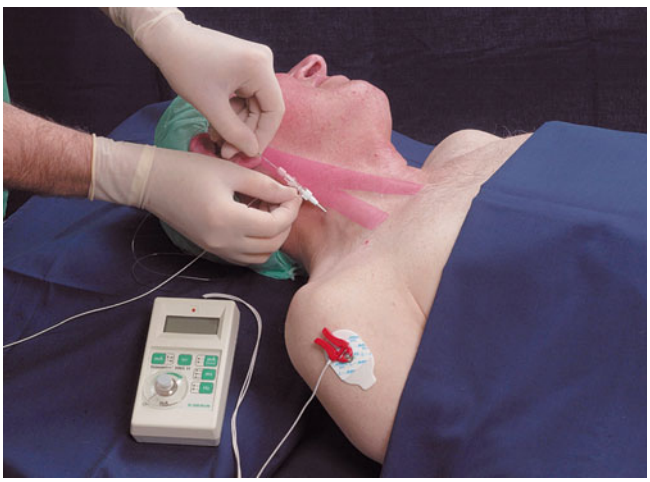


Fig. 30.9 Interscalene block (continuous technique). Introducing the catheter through a Tuohy needle (With permission from Danilo Jankovic)

Dosage

Surgical

"Single-shot" administration:

40 mL local anesthetic is sufficient for an adequate block of the brachial plexus and caudal part of the cervical plexus. In the literature [6, 11, 21, 24], the doses administered vary from 30 to 50 mL. A mixture of 20 mL 0.75 % ropivacaine or 0.5 % bupivacaine (0.5 % levobupivacaine) with 20 mL 1 % prilocaine (1 % mepivacaine) has proved its value very well in practice (in our own experience). This leads to a fast onset and long duration.

25 mL local anesthetic – e.g., 1 % prilocaine (1 % mepivacaine), in combination with 5–10 mg diazepam i.v. for reducing a dislocated shoulder.

20–25 mL local anesthetic – e.g., 0.75 % ropivacaine or 0.5 % bupivacaine (0.5 % levobupivacaine), in combination with basic general anesthesia for surgical interventions in the area of the shoulder and clavicle. This leads to very good postoperative pain control.

20 mL local anesthetic is sufficient to block the lower part of the cervical plexus and the upper part of the brachial plexus. The brachial plexus is only incompletely anesthetized with this amount and block of the ulnar nerve territory is often deficient.

Therapeutic

"Single-shot" administration (block series):

10 mL local anesthetic – e.g., 0.2 % ropivacaine or 0.125–0.25 % bupivacaine (0.125–0.25 % levobupivacaine) in shoulder and upper arm pain, shoulder arthritis, post-stroke pain, lymphedema after mastectomy.

10–20 mL local anesthetic – e.g., 0.2–0.375 % ropivacaine or 0.25 % bupivacaine (0.25 % levobupivacaine) in post-herpetic neuralgia, vascular diseases and injuries, complex regional pain syndrome (CRPS) types I and II, post-amputation pain.

25 mL local anesthetic – e.g., 1 % prilocaine or 1 % mepivacaine in combination with 5–10 mL diazepam i.v to mobilize the shoulder.

Continuous Interscalene Block: Posterior Technique (Pippa Technique)

The posterior cervical paravertebral block of the brachial plexus is an alternative to anterior route. This method was first described by Kappis in 1912 and was republished by Pippa in 1990 as a "loss of resistance" technique [15, 17, 20, 31]. The availability of electrical nerve stimulation has made this access route to the brachial plexus more important.

Indications and Contraindications

S. above.

Procedure

This block should only be carried out by experienced anesthetists or under their supervision. A detailed discussion with the patient is an absolute necessity.

Preparation

See anterior interscalene block.

Materials

See anterior interscalene block.

Skin Prep
In all blocks.

Patient Positioning

Sitting, with the neck flexed (to relax the cervical muscles) and supported by an assistant (the lateral recumbent position can be used as an alternative).

Landmarks

- Spinous processes of the sixth (C6) and seventh (C7 – vertebra prominens) cervical vertebrae (Fig. 30.10).
- The midpoint between the spinous processes of C6 and C7 is marked. The puncture site is located approximately 3 cm lateral to this point (Fig. 30.11).
- Level of the cricoid cartilage (target direction).

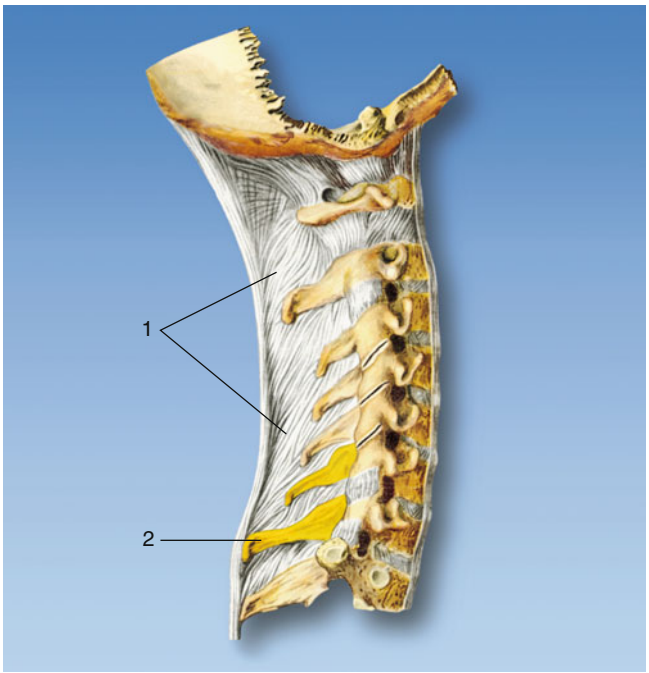


Fig. 30.10 Interscalene block, posterior access route. Landmarks: spinous processes of C6 and C7 (vertebra prominens). (*1*) nuchal ligament, (*2*) vertebra prominens (With permission from Danilo Jankovic)

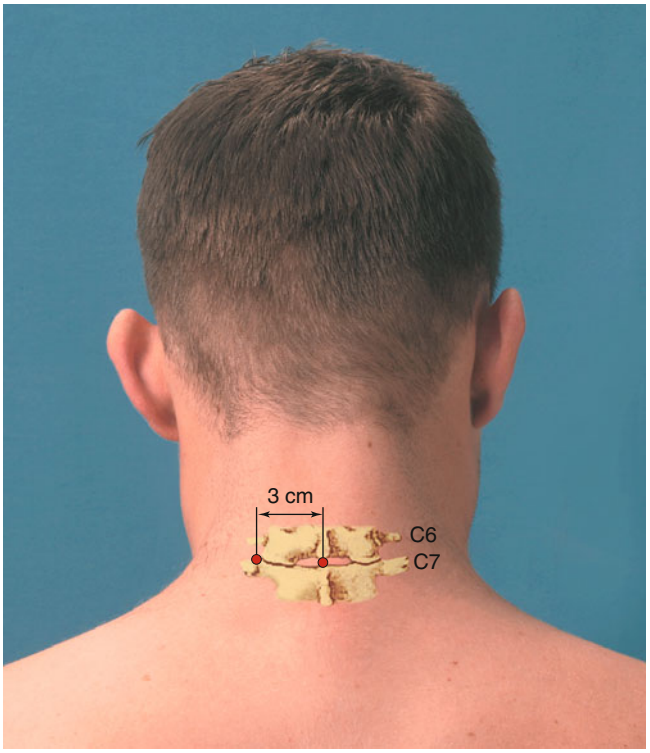


Fig. 30.11 Interscalene block, posterior access route. Puncture site: the midpoint between the spinous processes of C6 and C7 is marked. The puncture site is located ca. 3 cm lateral and paravertebral to this (With permission from Danilo Jankovic)

It is absolutely necessary to note the following points during this puncture procedure:

- Electrical nerve stimulation is the method of choice.
- After puncture, the needle should be passed toward the lateral edge of the cricoid cartilage.
- Contact with the transverse process (C7) is made after ca. 3.5–6 cm (depending on the anatomy).
- The transverse process is approximately 0.5–0.6 cm thick.
- There is a risk of perforating the dural sheath if the puncture is carried out too medially (epidural or subarachnoid injection).
- There is a risk of pneumothorax.
- Frequent aspiration should be carried out and the injection should be made in incremental doses (blood, CSF?).
- Note any contractions in the biceps brachii muscle (musculocutaneous nerve), deltoid muscle (axillary nerve), or index finger and thumb.

Technique

Disinfection of the puncture area, draping, and skin infiltration. After an incision with a stylet, the needle is introduced at the sagittal level and perpendicular to the skin, aiming approximately for the level of the ipsilateral cricoid cartilage (Fig. 30.12). The needle passes the major cervical muscles (trapezius, splenius cervicis, and levator scapulae (Fig. 30.13) on the way to the transverse process (C7). It is absolutely necessary to avoid any deviation in a medial direction from the sagittal level. At a depth of ca. 3.5–6 cm, contact is made with the transverse process of C7. The needle is withdrawn slightly, the injection direction is corrected slightly cranially, and one advances past the transverse process further 1.5–2 cm deeper. Stimulation current of 1–2 mA and 2 Hz is selected with a stimulus duration of 0.1 ms. After the motor response from the relevant musculature (biceps brachii muscle and/or deltoid, or muscles of the index finger and thumb), the current is reduced to 0.3–0.5 mA. The catheter is advanced approximately 3 cm beyond the end of the needle or cannula. After removal of the needle or cannula, fixation of the catheter and placement of a bacterial filter, and after careful aspiration and injection of a test dose, the bolus administration of the local anesthetic follows.

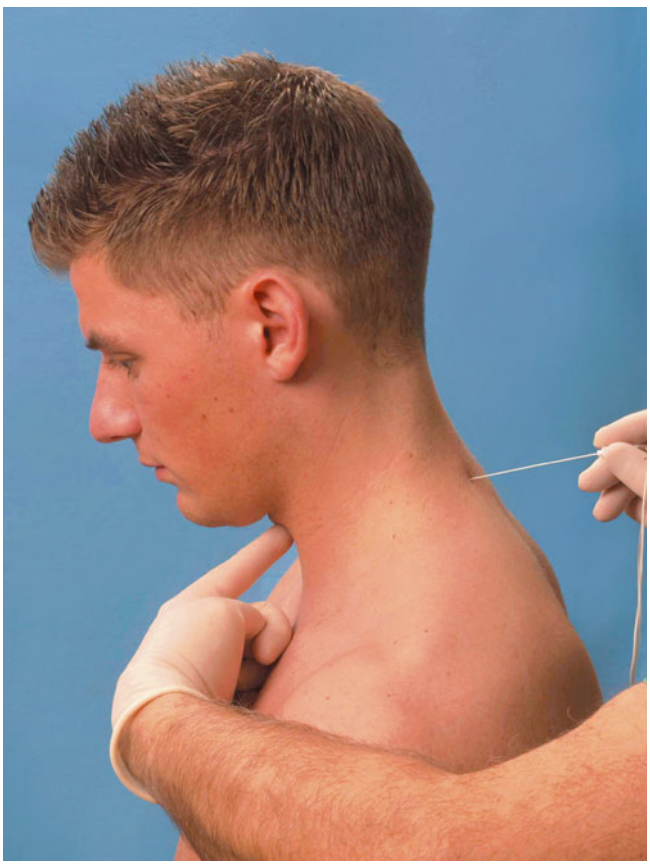


Fig. 30.12 Interscalene block, posterior access route. Puncture technique: the needle is introduced at the sagittal level and perpendicular to the skin in the direction of the ipsilateral cricoid cartilage (With permission from Danilo Jankovic)

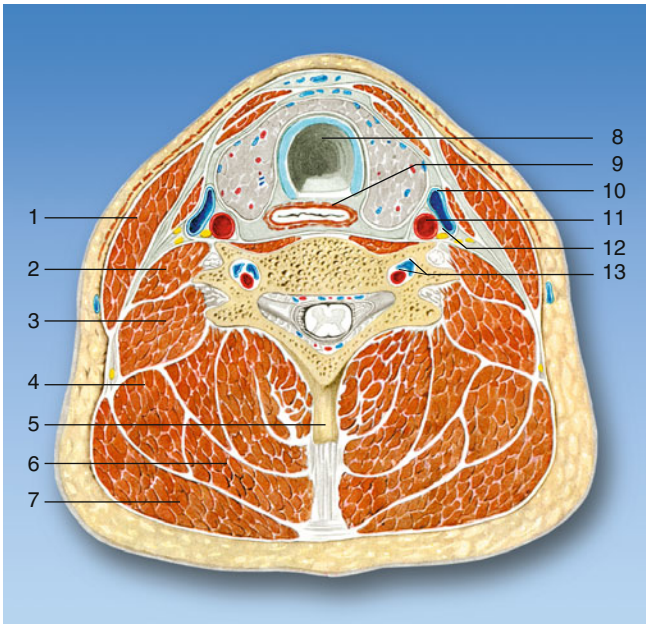


Fig. 30.13 Interscalene block, posterior access route. Puncture technique: the needle passes the strong cervical muscles (trapezius muscle, splenius cervicis muscle, and levator scapulae muscle). (*1*) Sternocleidomastoid muscle, (*2*) scalenus anterior muscle, (*3*) scalenus medius and scalenus posterior muscles, (*4*) levator scapulae muscle, (*5*) spine of the sphenoid bone, (*6*) splenius capitis and splenius cervicis muscles, (*7*) trapezius muscle, (*8*) trachea, (*9*) esophagus, (*10*) internal jugular vein, (*11*) common carotid artery, (*12*) vagus nerve, (*13*) vertebral artery and vein (With permission from Danilo Jankovic)

Dosage [1, 8, 9, 16, 30, 32]

40 mL local anesthetic (see above) is commonly used for the procedure. As a subsequent 24-h infusion for postoperative analgesia:

- 0.2 % ropivacaine, 6–14 mL/h (max. 37.5 mg/h)
- 0.25 % bupivacaine (0.25 % levobupivacaine), 0.25 mg/kg b.w./h
- 0.125 % bupivacaine (0.25 % levobupivacaine), 0.125 mg/kg b.w./h, combined with opioids if appropriate

Individual adjustment of the dosage and period of treatment is necessary. The following information is therefore only intended to provide guidance.

Distribution of the Blocks

The complete distribution of the anesthesia (S. above).

Side Effects

Simultaneous anesthesia of the following nerves and ganglia (Fig. 30.14):

- Vagus nerve, main symptoms: tachycardia, hypertension.
- Recurrent laryngeal nerve, main symptoms: hoarseness and foreign-body sensation in the throat.
- Phrenic nerve, main symptoms: unilateral paralysis of diaphragmatic movement and simulation of pneumothorax, particularly with continuous blocks [6, 16]. An ipsilateral block of the phrenic nerve has been observed as a side effect after an interscalene block in nearly 100 % of patients [27–29].
- Cervicothoracic (stellate) ganglion, with Horner's syndrome.
- Bronchospasm [26].
- Contralateral anesthesia [7].
- Bilateral distribution of the local anesthetic [13].
- Reversible "locked-in" syndrome [5].

The patient must be warned of these potential adverse events.

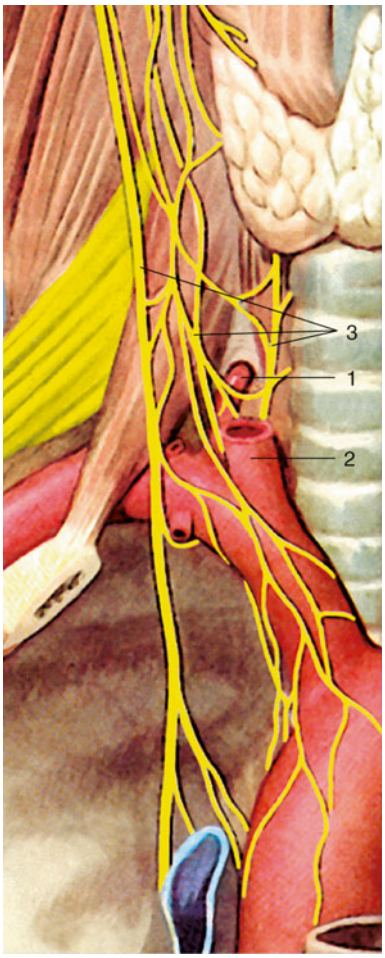


Fig. 30.14 The most important nerves and vessels in the injection area: (*1*) vertebral artery, (*2*) carotid artery, (*3*) laryngeal, vagus, and phrenic nerves (With permission from Danilo Jankovic)

Complications

Nerve Injuries

Traumatic nerve injuries are an extremely rare complication of this technique [2, 12, 18].

Prophylaxis: Only needles with short-beveled tips should be used. Intraneural positioning should be excluded. Vasopressor additives should be avoided. This procedure should not be performed in adult patients under general anesthesia. For details, see Chap. 5.

Intravascular Injection [5]

There is a particular risk of intravascular injection into the vertebral artery (Fig. 30.14) or other cervical vessels. This can very quickly lead to toxic reactions. For the symptoms and treatment, see Chap. 1.

Epidural or Subarachnoid Injection [10, 19, 25]

Epidural injection of the local anesthetic can lead to high epidural block, and subarachnoid administration can lead to a total spinal block. Both complications are significant and life threatening and require immediate treatment (see Chap. 1).

Prophylaxis: injection with short needles and introduction of the needle in a caudal direction.

CNS Toxicity

Overdose and/or intravascular diffusion of the local anesthetic can, in extremely rare cases, lead to CNS toxicity (see Chap. 1).

Pneumothorax

When the technique is carried out correctly, this complication is unlikely. The needle is advanced at a safe distance from the dome of the pleura.

Pressure on the Carotid Artery

Extremely rare and transient. Caused by the volume of the injection [22].

Interscalene block of the brachial plexus

Continuous technique ☐ Right ☐ Left

Purpose of block: ☐ *Surgical* ☐ *Therapeutic*
i.v. access: ☐ *Yes*
Monitoring: ☐ *ECG* ☐ *Pulse oxymetry*
Ventilation facilities: ☐ *Yes (equipment checked)*
Emergency equipment *(drugs):* ☐ *Checked*
Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Other*
Puncture technique: ☐ *Electrostimulation*
Access route: ☐ *Anterior* ☐ *Posterior*
Needle type: ☐ *Catheter kit 50 mm 22-G* ☐ *Tuohy ___ mm ___ G*
☐ *Stimulating needle*
☐ *Other* _____

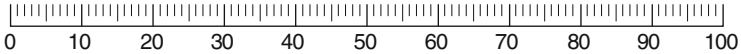
Puncture technique: ☐ *Interscalene groove located* ☐ *Level C6*
☐ *Ultrasound guided*
☐ *Electrostimulation*
☐ *Transducer* ☐ *Linear* ☐ *Curved*
Approach: ☐ *In plane* ☐ *Out of plane*
Nerve region _____ _____
Catheter: ☐ *Advanced* _____ *cm*
Aspiration test: ☐ *Carried out*
Bacterial filter: ☐ *Placed*
Bolus administration: _____ *mL* _____ *%* _____
(in incremental doses)
Addition to injection solution: ☐ *No* ☐ *Yes* _____ *µg/mg*
Patient's remarks during injection:
☐ *None* ☐ *Paresthesias* ☐ *Warmth*
Pain triggered (intraneural location?) _____
Nerve region _____

Objective block effect after 15 min:
☐ *Cold test* ☐ *Temperature measurement: right ___ °C left ___ °C*
☐ *Sensory* ☐ *Motor*
☐ *Continuous monitoring*

☐ Infusion for postoperative analgesia
Local anesthetic: _____ *%* _____ *ml/h*
☐ Addition to LA: _____ *mg* _____ *µg* _____
Patient-controlled anesthesia (PCA)
Local anesthetic: _____ *%*
Addition: _____
☐ Baseline rate _____ *ml/h* _____
☐ Bolus administration _____ *ml*
☐ Lockout interval _____ *min*

Complications:
☐ *None* ☐ *Intravascular* ☐ *Epidural/subarachnoid* ☐ *Pneumothorax*
Side effects:
☐ *None* ☐ *Hematoma* ☐ *Phrenic nerve*
☐ *Recurrent laryngeal nerve* ☐ *Horner's syndrome*

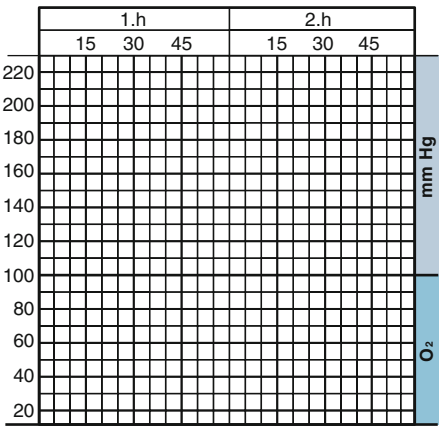
Subjective effects of the block: *Duration:* _____
☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*
VISUAL ANALOG SCALE

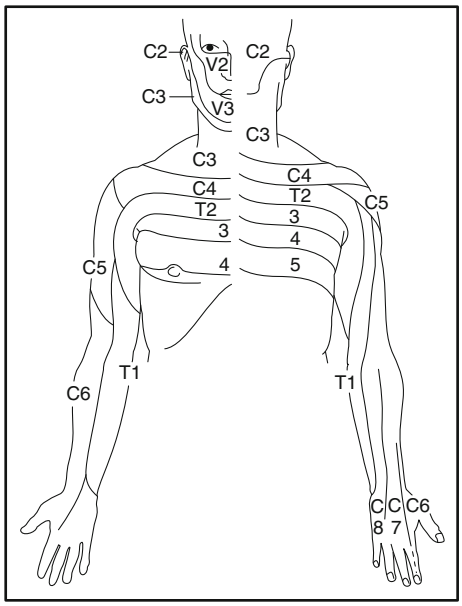


Record and checklist

Name: _____
Date: _____
Diagnosis: _____
Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No
☐ Yes (which?) _____

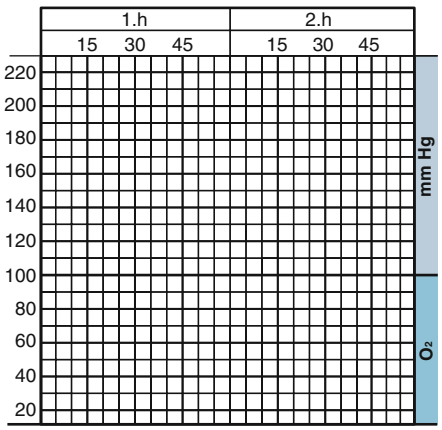


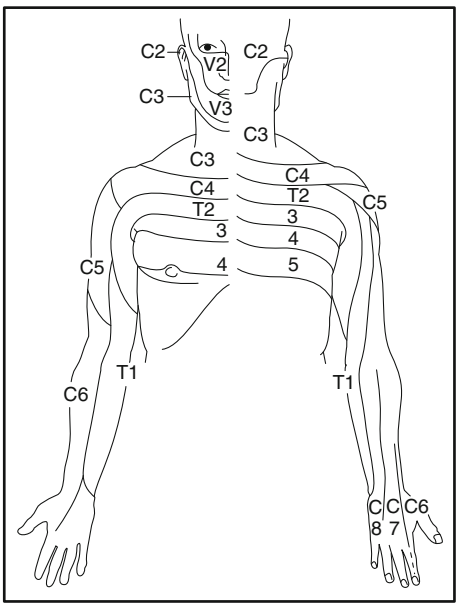


Special notes:
_____
_____
_____
_____

With permission from Danilo Jankovic

Record and checklist





Interscalene block of the brachial plexus

"Single-shot"-technique ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No

☐ Yes (which?) _____

Purpose of block: ☐ *Surgical* ☐ *Diagnostic* ☐ *Therapeutic*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oxymetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs)* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Head to contralateral side*

Needle type: ☐ *50mm* *22-G*

Puncture technique: ☐ *Interscalene groove located* ☐ *Level C6*

☐ *Ultrasound guided*

☐ *Electrostimulation*

☐ *Transducer* ☐ *Linear* ☐ *Curved*

Approach: ☐ *In plane* ☐ *Out of plane*

Nerve region _____ _____

Local anesthetic: _____ *ml* _____ *%* _____

(in incremental doses)

Addition: ☐ *Yes* _____ *µg/mg* ☐ *No*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth*

☐ *Pain triggered (intraneural location?)* _____

Nerve region _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* ____ *°C left* ____ *°C*

☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____

Complications:

☐ *None* ☐ *Intravascular* ☐ *Epidural/subarachnoid* ☐ *Pneumothorax*

Side effects::

☐ *None* ☐ *Hematoma* ☐ *Phrenic nerve* ☐ *Recurrent laryngeal nerve*

☐ *Horner's syndrome*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

_____

_____

With permission from Danilo Jankovic

References

1. Allesio JG, Rosenblum M, Shea K, Freitas D. A retrospective comparison of interscalene block and general anesthesia for ambulatory surgery and shoulder arthroscopy. Reg Anesth. 1995;20(1):62–8.
2. Barutell C, Vidal F, Raich M, Montero A. A neurological complication following interscalene brachial plexus block. Anesthesia. 1980;35:365–7.
3. Blanchard J, Ramamurthy S. Brachial plexus. In: Benumof LJ, editor. Clinical procedures in anesthesia and intensive care. Philadelphia: Lippincott; 1992.
4. Bridenbaugh DL. The upper extremity: somatic blockade. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988.
5. Durrani Z, Winnie AP. Brainstem toxicity with reversible locked-in syndrome after interscalene brachial plexus block. Anesth Analg. 1991;72:249–52.
6. Gentili ME, Le foulon-Gourves M, Mamelle JC. Acute respiratory failure following interscalene block: complications of combined general and regional anesthesia. Reg Anesth. 1994;19(4):292–3.
7. Gologorsky E, Leanza RF. Contralateral anesthesia following interscalene block. Anesth Analg. 1992;75:311–2.
8. Haasio J, Tuominen MK, Rosenberg PH. Continuous interscalene brachial plexus block during and after shoulder surgery. Ann Chir Gynaecol. 1990;79:103–7.
9. Kardash K, Schools A, Concepcion M. Effects of brachial plexus fentanyl on supraclavicular block. Reg Anesth. 1995;20(4):311–5.
10. Kumar A, Battit GE, Froese AB, Long MC. Bilateral cervical and thoracic epidural blockade complicating interscalene brachial plexus block: report of two cases. Anesthesiology. 1971;35:650–2.
11. Lanz E, Theiss D, Jankovic D. The extent of blockade following various techniques of brachial plexus block. Anesth Analg. 1983;62:55–8.
12. Löfström B, Wennberg A, Widen L. Late disturbances in nerve function after block with local anesthetic agents. Acta Anaesthiol Scand. 1966;10:111–22.
13. Lombard TP, Couper JL. Bilateral spread of analgesia following interscalene brachial plexus block. Anesthesiology. 1983;58: 472–3.
14. Meier G, Bauereis C, Heinrich C. Der interskalenäre Plexuskatheter zur Anästhesie und postoperativen Schmerztherapie. Anaesthesist. 1997;46:715–9.
15. Neil RS. Postoperative analgesia following brachial plexus block. Br J Anaesth. 1978;50:379–82.
16. Pere P. The effect of continuous interscalene brachial plexus block with 0,125 % bupivacaine plus fentanyl on diaphragmatic motility and ventilatory function. Reg Anesth. 1993;18:93–7.
17. Pippa P, Cominelli E, Marinelli C, Aito S. Brachial plexus block using the posterior approach. Eur J Anaesthiol. 1990;7:411–20.
18. Poeck K. Therapie der peripheren Nervenschädigungen. In: Poeck K, editor. Neurologie. Berlin/Heidelberg/New York: Springer; 1994.
19. Ross S, Scarborough CD. Total spinal anesthesia following brachial plexus block. Anesthesiology. 1973;39:458.
20. Rucci FS, Pippa P, Barbagli R, Doni L. How many interscalenic blocks are there? A comparison between the lateral and posterior approach. Eur J Anesthiol. 1993;10:303–7.
21. Selander D, Dhuner KG, Lundborg G. Peripheral nerve injury due to injection needles used for regional anesthesia. Acta Anesthiol Scand. 1977;21:182–8.
22. Siler JN, Liff PI, Davis JF. A new complication of interscalene brachial plexus block. Anesthesiology. 1973;38(6):590–1.
23. Silverstein WB, Moin U, Saiyed MD, Brown AR. Interscalene block with a nerve stimulator: a deltoid motor response is a satisfactory endpoint for successful block. Reg Anesthiol Pain Med. 2000;25(4):356–9.
24. Tetzlaff JE, Yoon HJ, Brems J. Interscalene brachial plexus block for shoulder surgery. Reg Anesth. 1994;19(5):339–43.
25. Tetzlaff JE, Yoon HJ, Dilger J, Brems J. Subdural anesthesia as a complication of an interscalene brachial plexus block. Reg Anesth. 1994;19(5):357–9.
26. Thiagarajah S, Lear E, Salzer J, Zeiligson E. Bronchospasm following interscalene brachial plexus block. Anesthesiology. 1984;61: 759–61.
27. Urmey WF. Interscalene block. The truth about twitches. Reg Anesth Pain Med. 2000;25(4):340–2.
28. Urmey WF, Talts KH, Sharrock NE. One hundred percent incidence of hemidiafragmatic paresis associated with interscalene brachial plexus anesthesia as diagnosed by ultrasonography. Anesth Analg. 1991;72:498–503.
29. 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.
30. Viel EJ, Eledjam JJ, de la Coussage JE, D'Athis F. Brachial plexus block with opioids for postoperative pain relief: comparison between buprenorphine and morphine. Reg Anesth. 1989;14: 274–8.
31. Vranken JH, van der Vegt MH, Zuurmond WA, Pij AJ, Dzoljic M. Continuous brachial plexus block at the cervical level using a posterior approach in the management of neuropathic cancer pain. Reg Anesth Pain Med. 2001;26(6):572–5.
32. Winchell SW, Wolf R. The incidence of neuropathy following upper extremity nerve blocks. Reg Anesth. 1985;10:12–5.

Chapter 31

Infraclavicular Brachial Plexus Block

Ki Jinn Chin, MBBS, MMed., FANZCA, FRCPC
Department of Anesthesia, Toronto Western Hospital, Toronto, Canada
e-mail: Kijinn.chin@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_31,

Introduction

The infraclavicular brachial plexus block was described as early as 1917 but was overshadowed in the early part of the twentieth century by the axillary and supraclavicular approaches. Refinements in the technique by authors such as Wilson et al. [1] and Kilka et al. [2] led to a deserved increase in popularity in the mid-1990s. The block is a highly effective and safe technique for providing anesthesia of the distal upper arm [3].

Indications

- Anesthesia or analgesia of the elbow, forearm, and hand. It may be performed as either a single-shot or continuous catheter technique.

Contraindications

- Absolute
  - There are no absolute contraindications specific to this block. Generic contraindications such as patient refusal, allergy to local anesthetics, and local infection at the site of injection apply as usual.
- Relative
  - Coagulopathy—the neurovascular bundle and associated vessels lie relatively deep, under the pectoralis muscles, and may therefore be less easy to compress in the event of vascular puncture. However, the likelihood of a clinically significant hematoma in most patients is low.
  - Distorted surface anatomy, e.g., from clavicular fracture, prior surgery, etc.
  - Foreign bodies in the infraclavicular area, e.g., subclavian central venous lines, pacemaker or other battery packs, etc.

Advantages Compared to Other Brachial Plexus Block Techniques

1. Both surface landmark-guided and ultrasound-guided techniques are associated with high block success rates.
2. The infraclavicular block is a safe block. It carries minimal risk of adverse effects associated with injections administered more proximally in the root of the neck, e.g., phrenic nerve palsy and Horner's syndrome. Pneumothorax has been described with more medial surface landmark-guided approaches (i.e., the vertical infraclavicular block), but we consider the risk to be minimal with other approaches where needle insertion occurs lateral to the thoracic cage.
3. The neurostimulation-guided technique utilizes simple surface anatomical landmarks and a straightforward needle approach.
4. The endpoint for injection in the ultrasound-guided technique (local anesthetic spread posterior to the artery) is simple and easily recognized.
5. There is evidence that tourniquet application may be tolerated better, which is attributed to local anesthetic spread to the intercostobrachial nerve [3].
6. The infraclavicular block may be performed with the arm in any position (adducted or abducted at the shoulder).
7. Nerve catheters are easily fixed in this location and do not interfere with patient movement.

Disadvantages Compared to Other Brachial Plexus Block Techniques

1. The brachial plexus in the ultrasound-guided infraclavicular block is usually least 3–4 cm deep in most adults, and a relatively steep needle trajectory is required to reach it.
2. Needle insertion through the pectoral muscles can be uncomfortable.
3. Nerve catheter placement is usually perpendicular to the plexus, rather than parallel to it. This may make advancement of the catheter more difficult and limit the length of catheter that can be advanced into the brachial plexus sheath, which in turn may increase the risk of dislodgement.

Functional Anatomy

The infraclavicular–axillary region can be visualized as a pyramidal space. The apex of the pyramid is formed medially by the confluence of the clavicle, scapula, and first rib; the base is the skin and subcutaneous tissue of the armpit. The posterior wall is formed by the scapula and its associated muscles and the anterior wall by the pectoralis major and minor. The humerus, together with the converging muscles and tendons of the anterior and posterior walls that insert into it, constitutes the lateral wall. The bony thoracic cage, and overlying serratus anterior muscle, forms the medial wall. The contents of this space are the axillary artery and vein, the brachial plexus, lymph nodes, and loose areolar tissue. Of note, the intercostobrachial nerve runs along the inferior aspect of the brachial plexus sheath in the infraclavicular–axillary fossa and is often blocked by anesthetic spread following infraclavicular injection (Fig. 31.1).

The coracoid process of the scapula and the clavicle are the chief bony landmarks. The coracoid process lies superior and lateral to the course of the brachial plexus.

The brachial plexus runs in a line between the base of the interscalene groove and the axilla, and knowing this is helpful when trying to mentally visualize the location of the brachial plexus [4]. Note that the direction in which this line runs will differ depending on whether the arm is adducted or abducted (Figs. 31.2 and 31.3).

It is important to appreciate that the orientation of the cords around the axillary artery varies with distance from the clavicle. As the brachial plexus emerges from under the clavicle, the cords are bunched together on the superior (cephalad) aspect of the artery. As the neurovascular bundle travels more distally, the cords gradually separate out and "spiral" around the axillary artery to eventually adopt the classically described position of lateral, posterior, and medial to the artery (Fig. 31.4). This arrangement is best appreciated when scanning the infraclavicular area with the arm abducted at the shoulder.

There is a fascial sheath around the neurovascular bundle that contains and directs the spread of local anesthetic. There is usually a fascial septum that separates the lateral cord from posterior and medial cord; piercing this septum is necessary to achieve the U-shaped local anesthetic spread posterior to the artery that is associated with block success [5] (Fig. 31.5). This also explains why obtaining a posterior cord motor response in the landmark-guided approach carries the highest success rates [6–9].

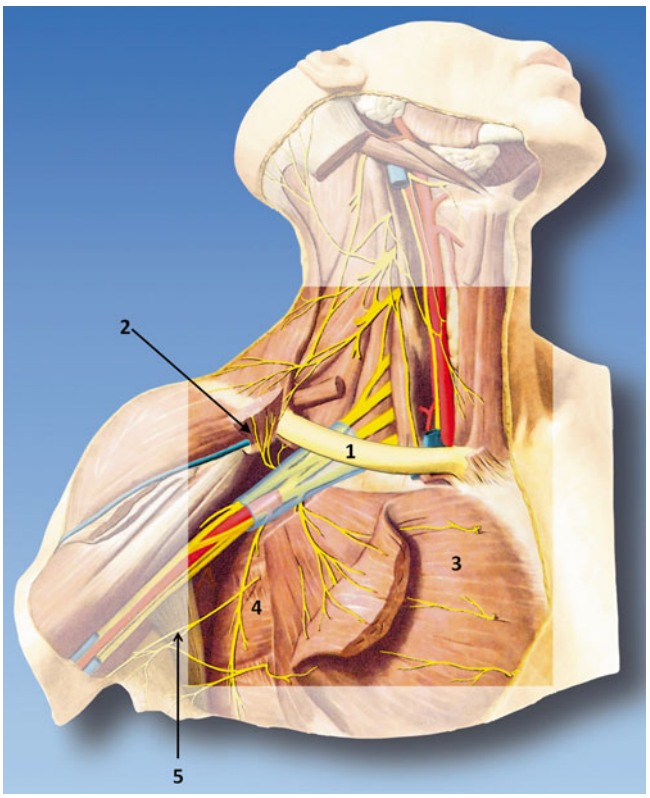


Fig. 31.1 Anatomy of the infraclavicular area. Note that the brachial plexus passes under the approximate midpoint of the clavicle and then deep to the pectoralis major and minor muscle, medial and inferior to the coracoid process. (*1*) clavicle, (*2*) coracoid process and pectoralis minor (cut and reflected), (*3*) pectoralis major (cut and reflected), (*4*) thoracic cage, (*5*) intercostobrachial nerve (With permission from Danilo Jankovic)

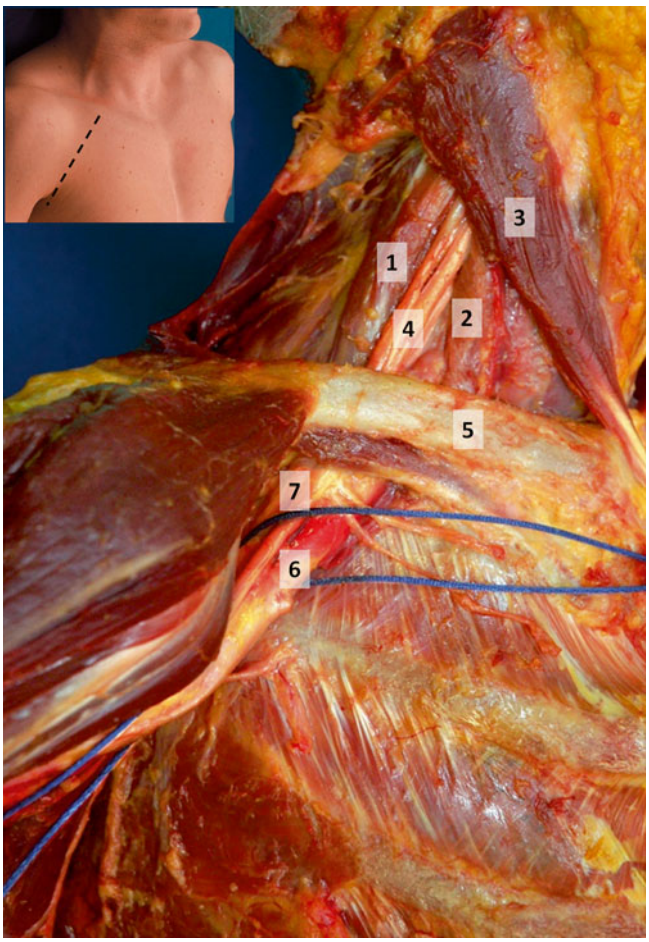


Fig. 31.2 The course of the infraclavicular brachial plexus with arm adducted at shoulder. The neurovascular bundle is encircled by the blue suture. Note that all the cords of the brachial plexus are clustered superolateral to the axillary artery in the medial portion of the infraclavicular fossa. (*1*) middle scalene muscle, (*2*) anterior scalene muscle, (*3*) sternocleidomastoid muscle, (*4*) trunks of the brachial plexus, (*5*) clavicle, (*6*) axillary artery, (*7*) cords of brachial plexus (With permission from Danilo Jankovic)

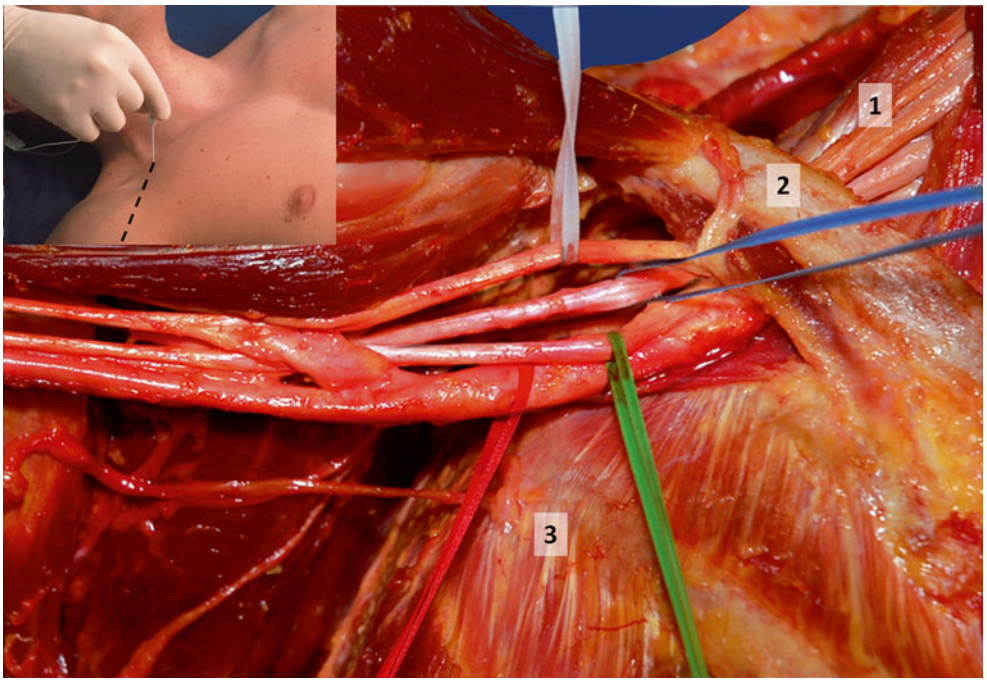


Fig. 31.3 Course of the infraclavicular brachial plexus with the arm abducted at the shoulder. Note that all the cords of the brachial plexus are clustered superolateral to the axillary artery in the medial portion of the infraclavicular fossa. Lateral cord (*white*), posterior cord (*blue*), medial cord (*green*), axillary artery (*red*). Note the relationship between the brachial plexus and the thoracic cage, which determines the relative risk of pneumothorax with more medial versus lateral approaches. (*1*) trunks of the brachial plexus, (*2*) clavicle, (*3*) thoracic cage of ribs and intercostal muscles (With permission from Danilo Jankovic)

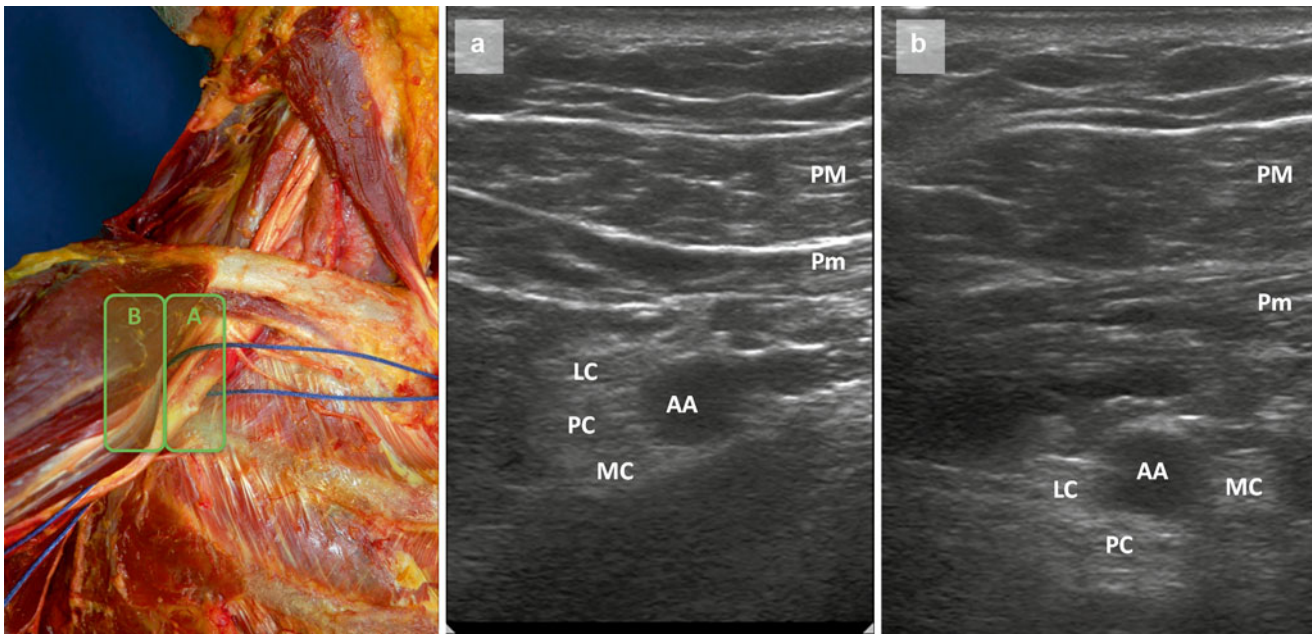


Fig. 31.4 Illustration of the variation in distribution of brachial plexus cords around the axillary artery (*AA*) depending on whether the probe is placed more medially (*position A*) or laterally (*position B*). In *position A*, the cords are clustered around the cephalad aspect of the artery and are often not distinguishable from each other. This view is usually only obtainable if the arm is abducted at the shoulder so that the clavicle moves up out of the way. In *position B*, the cords start to spread out around the artery. The lateral cord (*LC*) remains at the cephalad aspect of the artery, but the posterior cord (*PC*) and medial cord (*MC*) move into their classically described positions relative to the artery. *PM* pectoralis major, *Pm* pectoralis minor (With permission from Danilo Jankovic)

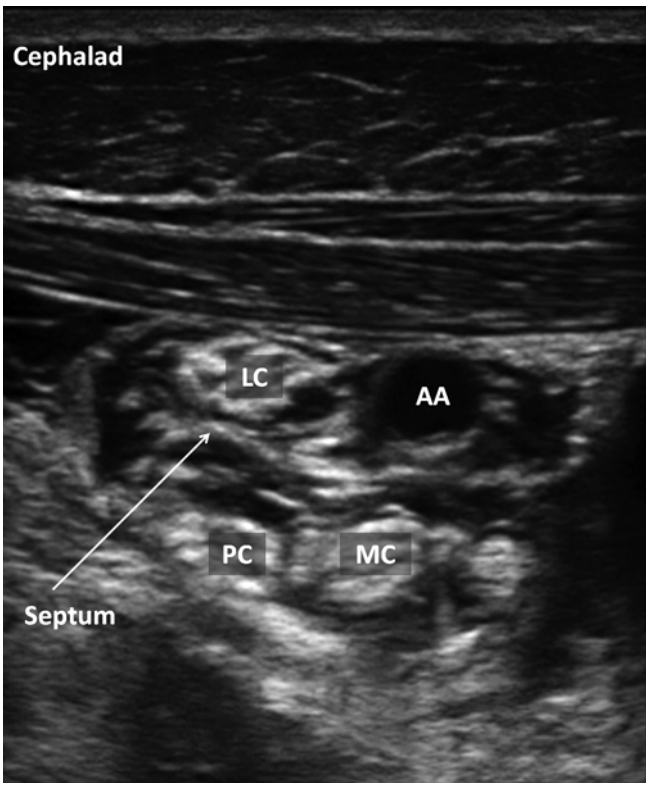


Fig. 31.5 Post-injection ultrasound image of the infraclavicular brachial plexus, illustrating the septum that separates the lateral cord (*LC*) from the posterior (*PC*) and medial (*MC*) cords. Our preference is therefore for a dual-injection technique, but there can be communication between the two compartments, and a single-injection posterior/deep to the artery (*AA*) may also suffice (Image courtesy of KJ Chin MPC)

Technique: Surface Landmark, Neurostimulation-Guided Approach, Single-Shot

Required Supplies and Equipment

- Disinfectant solution and swabs for skin preparation
- Sterile gloves and drapes
- Short-beveled 22-G block needle with extension tubing
  - A 50-mm needle is suitable in slimmer patients, but an 80-mm needle may be necessary in patients with a thick chest wall.
- Local anesthetic of choice in 10- or 20-ml syringes
- Peripheral nerve stimulator
- Lidocaine 1–2 % in 3-ml syringe with a 25–27-G hypodermic needle for skin infiltration (at operator's discretion)
- Equipment and supplies for managing life-threatening acute complications, including Intralipid for local anesthetic systemic toxicity
- Drugs for intravenous sedation during the block (at operator's discretion)

Preparation of Patient

- Obtain informed consent for the block.
- Explain expected clinical course including care of the insensate limb and managing the transition to systemic analgesia.
- Establish intravenous access, supplemental oxygen delivery, and standard monitors (ECG, noninvasive blood pressure, pulse oximetry).
- Perform a time-out to confirm patient identity and site and side of surgery.

Block Performance

Several slightly different surface landmark-guided techniques have been described, all of which use neurostimulation as the endpoint for nerve localization and injection. We recommend one of two techniques:

1. The coracoid (Wilson) approach [1]
2. The vertical infraclavicular plexus (VIP) approach [2]

Landmark-Guided Infraclavicular Block: Coracoid (Wilson) Approach

Patient Position

- The patient is placed supine with the operative arm adducted and if possible flexed at the elbow and placed on the patient's abdomen to facilitate observation of motor responses.
- It is recommended that the operator stands at the head of the patient, on the side to be blocked, as this makes it easier to observe and avoid inadvertent medial direction of the needle. However, the operator may also stand on the side of the patient if preferred.

Surface Landmarks

- The coracoid process is palpated and marked.
- A point 2 cm inferior and 2 cm medial to the coracoid process is marked—this is the site of needle insertion (Fig. 31.6).

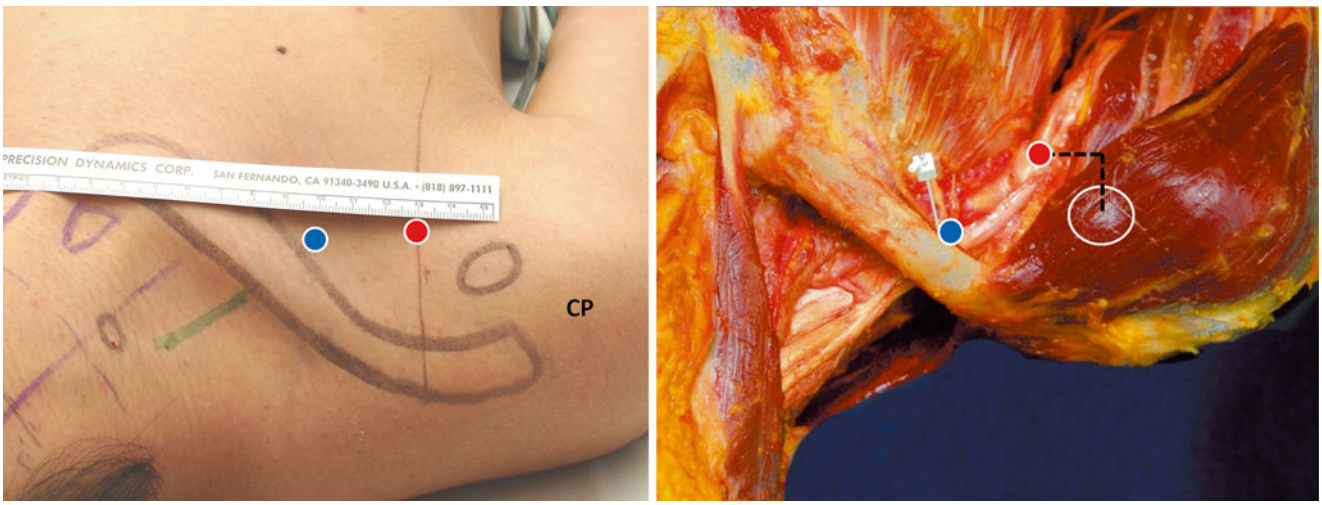


Fig. 31.6 Surface landmarks for the vertical infraclavicular plexus (VIP) approach and the coracoid (Wilson) approach to the infraclavicular block, illustrated in a volunteer and a cadaver. In the VIP approach, the needle insertion site (*blue circle*) is immediately inferior to the clavicle at its midpoint. In the coracoid approach, the needle insertion site (*red circle*) is located 2 cm medial and inferior to the coracoid process (*CP*). In both approaches, the needle is advanced perpendicular to the skin surface. Note that in the coracoid approach, the needle trajectory usually lies lateral to the thoracic cage. The green line indicates the interscalene groove and location of the brachial plexus (With permission from Danilo Jankovic)

Needle Insertion and Injection Technique

- Disinfect the skin, drape the area appropriately, prime the block needle and tubing with local anesthetic solution, and attach a nerve stimulator at an initial current setting of 0.5–0.8 mA, pulse duration 0.1–0.3 ms, and frequency of 1–2 Hz. This lower initial current setting is recommended to reduce patient discomfort during needle passage through the pectoral muscles.
- Infiltrate the needle insertion site with local anesthetic. Fixing the skin over the skin insertion point with the second and third fingers of the nondominant hand, insert a 50–80-mm block needle at a 90° angle to the skin in a strict parasagittal plane. Medial angulation must be avoided to prevent pleural puncture.
- The pectoral muscles will initially be observed to twitch due to local stimulation as the needle passes through it. Once this twitch disappears, the nerve stimulator current may be turned up to 1 mA or more for nerve localization.
- Advance the needle slowly and steadily until a distal motor response in the wrist or hand (1st– 3rd fingers) is obtained at a current threshold of 0.3–0.5 mA.
- The ideal motor response, associated with the highest block success rate, is a posterior cord response of wrist or finger extension [6–9]. A medial cord response may also be accepted [10]. If elbow, wrist, or finger flexion is obtained (lateral cord), the needle should be advanced a little deeper and may need to be redirected slightly more inferiorly or deeper [11].
- If no motor response is obtained despite inserting the needle to a depth of 4–5 cm in the average adult patient, or if bone is contacted (scapula), the needle should be withdrawn to the skin and redirected slightly more inferiorly or superiorly in the same parasagittal plane.
- Once an appropriate distal motor response has been obtained, incremental injection of local anesthetic is performed with intermittent aspiration to exclude intravascular injection

Landmark-Guided Infraclavicular Block: Vertical Infraclavicular Plexus (VIP) Approach

Patient Position

- The patient is placed supine with the operative arm adducted and if possible flexed at the elbow and placed on the patient's abdomen to facilitate observation of motor responses.
- It is recommended that the operator stand at the head of the patient, on the side to be blocked, as this makes it easier to observe and avoid inadvertent medial direction of the needle. However, the operator may also stand on the side of the patient if preferred.

Surface Landmarks

- The following landmarks are palpated and marked.
  - The suprasternal notch.
  - The anterior acromion. Precise location of the anterior acromion is very important, and it can be distinguished from the mobile humeral head by passive movement of the upper arm.
- The midpoint of the line between the suprasternal notch and anterior acromion is identified and marked—this is the needle insertion point, immediately inferior to the clavicle (Fig. 31.6).

Needle Insertion and Injection Technique

- Disinfect the skin, drape the area appropriately, prime the block needle and tubing with local anesthetic solution, and attach a nerve stimulator at an initial current setting of 1–2 mA, pulse duration 0.1–0.3 ms, and frequency of 1–2 Hz.

- Infiltrate the skin insertion point with local anesthetic and advance the block needle at a 90° angle to the skin in a strict parasagittal plane (Fig. 31.7).
- Advance the needle slowly and steadily until a distal motor response in the wrist or hand (1st–3rd fingers) is obtained at a current threshold of 0.3–0.5 mA.
- A posterior or medial cord response is preferred to a lateral cord response [10, 11].
- Medial angulation must be avoided to prevent puncture of the subclavian vessels. If blood is aspirated, it signifies that puncture has occurred and the needle should be redirected or reinserted more laterally.
- The plexus lies at a depth of 3–4 cm in the average adult patient. Caution should be exercised if this depth is reached without any motor response, as further insertion increases the risk of a pneumothorax. Contact with bone signals contact with the first rib, and this depth should not be exceeded on subsequent passes.
- Once an appropriate distal motor response has been obtained, incremental injection of local anesthetic is performed with intermittent aspiration to exclude intravascular injection

Landmark-Guided Continuous Nerve Block Technique

This is identical to the single-shot technique except that a peripheral nerve block catheter kit is used instead (Fig. 31.7). Either of the above approaches may be used, depending on operator preference and expertise.

The introducer needle is advanced as described above until the desired motor response is obtained. If a stimulating catheter is being used, 5–10 ml of 5 % dextrose solution can be injected through the introducer needle to distend the paraneural sheath. This should also result in augmentation of the motor response. Injection of other solutions will abolish the motor response.

The catheter is then advanced approximately 3 cm beyond the tip of the introducer needle, looking to maintain a motor response at current thresholds of at least 0.3 mA and up to 0.8–1 mA. As the angle of insertion is at right angles to the course of the plexus, some resistance can be expected when advancing the catheter. This may be improved by the injection of 5–10 ml of fluid as described above. The needle is then withdrawn and the catheter fixed in place in the usual manner.

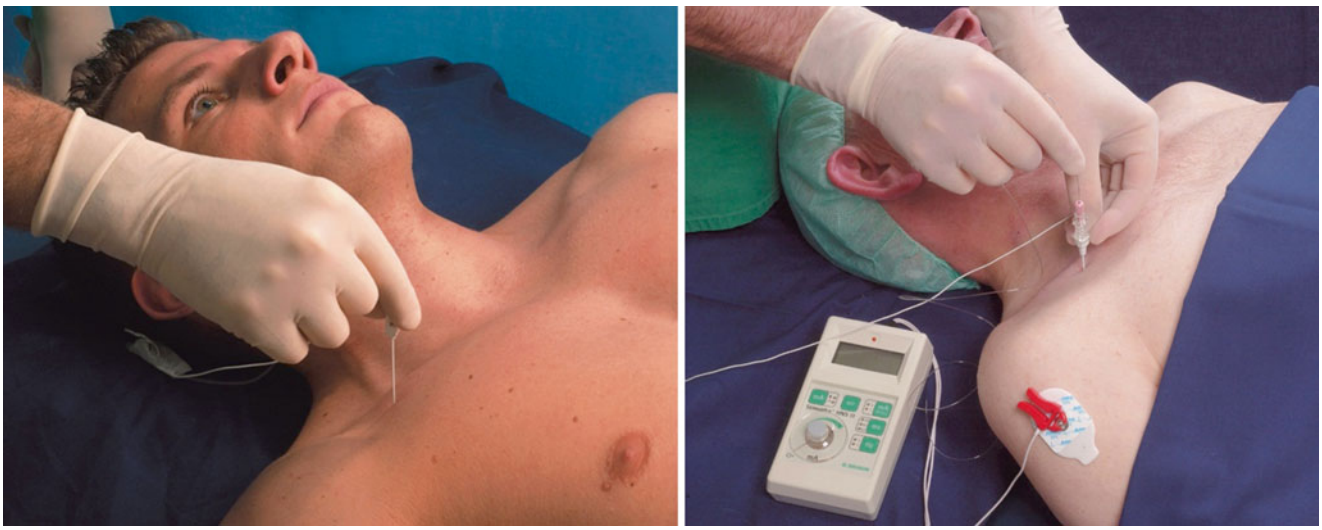


Fig. 31.7 The vertical infraclavicular plexus (VIP) approach to infraclavicular block, using a single-shot block needle (*left*) and a continuous peripheral nerve block catheter set (*right*). Note the needle trajectory perpendicular to the skin without any medial angulation (With permission from Danilo Jankovic)

Technique: Ultrasound-Guided Approach, Single-Shot

Required Supplies and Equipment

- Disinfectant solution and swabs for skin preparation.
- Sterile gloves and drapes.
- Short-beveled 22-G block needle with extension tubing.
  - An 80-mm needle is recommended in all adult patients.
  - An echogenic needle helps in needle localization.
- Local anesthetic of choice in 10- or 20-ml syringes.
- Lidocaine 1–2 % in 3-ml syringe with a 25–27-G hypodermic needle for skin infiltration (at operator's discretion).
- Ultrasound machine with high-frequency linear-array probe. A low-frequency curved-array probe may sometimes offer better image quality in patients with a thick chest wall.
- Equipment and supplies for managing life-threatening acute complications, including Intralipid for local anesthetic systemic toxicity.
- Drugs for intravenous sedation during the block (at operator's discretion).

Preparation of Patient

- Obtain informed consent for the block.
- Explain expected clinical course including care of the insensate limb and managing the transition to systemic analgesia.
- Establish intravenous access, supplemental oxygen delivery, and standard monitors (ECG, noninvasive blood pressure, pulse oximetry).
- Perform a time-out to confirm patient identity and site and side of surgery.

Block Performance

Patient and Operator Position

- The patient is placed supine. While it is also possible to perform the block with the arm adducted at the shoulder, it is recommended that the arm be abducted if possible. This moves the clavicle superiorly and out of the way of the ultrasound probe and block needle, which makes the block much easier to perform [12]. The brachial plexus also moves into a more superficial location and is easier to visualize as a result [13].
- It is recommended that the operator stand on the side of the patient to be blocked, either at the head of the patient or at the patient's side depending on operator preference. The ultrasound machine should be positioned in the operator's direct line of sight, i.e., toward the foot of the bed on the same side or on the opposite side of the patient, depending on whether the operator is standing at the head or at the side of the patient, respectively.

Pre-scanning and Identification of Anatomy

- Suggested initial ultrasound settings include a depth of 4 cm and a frequency in the middle of the probe's range to allow for the relatively greater penetration that is required. Gain, focus, and depth are subsequently adjusted as needed to optimize the image.
- The probe is placed inferior to the clavicle, in a parasagittal plane perpendicular to the line of the brachial plexus (Fig. 31.8).
- The chief landmark is the axillary artery, which is easily identified as a round hypoechoic pulsatile structure, deep to the pectoralis major and minor muscles.

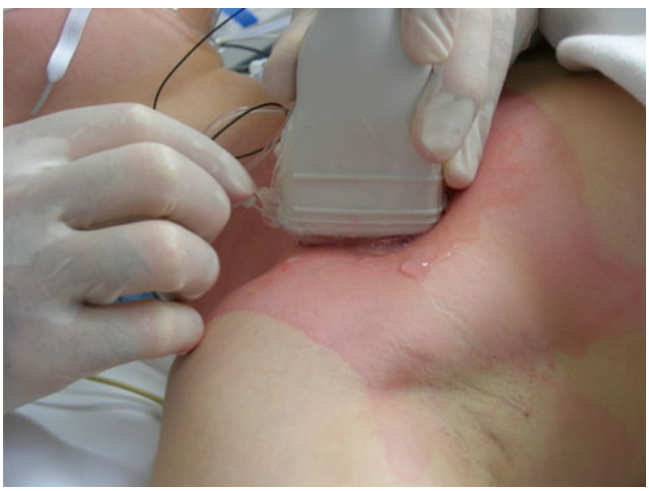


Fig. 31.8 Ultrasound-guided infraclavicular block. The probe is placed in a parasagittal plane, and the needle is advanced in a cephalad-to-caudad direction. Abduction of the shoulder moves the clavicle superiorly, which increases the available space for needle insertion cephalad to the probe (Image courtesy of KJ Chin MPC)

- The lateral cord is visible as a hyperechoic structure immediately superior to the artery and just deep to pectoralis minor. The posterior and medial cords are not usually distinct from each other but appear as a large hyperechoic complex structure posterior to the axillary artery (Fig. 31.4).
- The position of the cords in relation to the artery varies depending on where the neurovascular bundle is imaged along its course. More proximally (medially), the cords are grouped together along the superior aspect of the artery, and the plexus has a similar appearance to the first rib view that is used in the supraclavicular plexus block. More distally (laterally), the posterior and medial cords move into a location posterior to the artery, with the medial cord eventually taking up position between the axillary artery and vein (Fig. 31.4).
- The operator should scan along the course of the neurovascular bundle to identify the plane in which the plexus is most readily visualized and ideally where all the cords are bunched close together rather than spread out around the artery.
- The thoracic cage and pleura is visible in more medial probe positions but usually lies 1–2 cm deep to the neurovascular bundle and is therefore at little risk of inadvertent puncture.
- There are pectoral vessels running between the pectoralis major and minor muscles which should be identified to ensure that they do not lie in the path of the selected needle trajectory.

Needle Insertion and Injection Technique

- The needle is advanced using an in-plane approach toward the superior aspect of the lateral cord, aiming to pass at a tangent to its surface, rather than directly at it (Fig. 31.9a).
- The needle is advanced slowly and carefully through the pectoral muscles until the deep fascia of pectoralis minor has been pierced, which is usually signaled by a tactile pop. At this point, a test injection of 0.5 ml is made—if the needle tip is in the correct location, there will be visible fluid expansion next to the lateral cord, which has the added effect of pushing it out of the needle's path (Fig. 31.9b).
- The needle is advanced deeper, aiming to place the tip at the posterior (6 o'clock) aspect of the artery. A test injection at the correct location will create visible fluid spread immediately posterior or deep to the artery (the "double bubble" sign [14]).
- A total of 20–30 ml of local anesthetic is injected at this point, to create a U-shaped pattern of spread between the posterior aspect of the artery and the plexus (Fig. 31.9c).
- The needle is then slowly withdrawn until the tip once again lies adjacent to the lateral cord, at which point another 5–10 ml of local anesthetic is injected to produce spread in this compartment (Fig. 31.9d).
- Note that there is no statistically significant difference between the efficacy of single-injection techniques posterior to the artery and the dual-injection technique described above, or even a triple-injection technique [15, 16]. However the authors consider that a dual-injection technique does not add significant complexity and, in clinical practice, may further improve the already-high success rates.

Ultrasound-Guided Continuous Nerve Block Technique

This is identical to the single-shot technique except that a peripheral nerve block catheter kit is used instead.

The introducer needle is advanced as described above until the needle tip is located at the posterior aspect of the artery and injection produces the "double bubble" sign of fluid spread immediately adjacent to the artery. It is recommended that 5–10 ml of fluid be injected in this location to create a "pocket" into which the catheter can be advanced. If a stimulating catheter is being used to confirm tip location, 5 % dextrose solution should be used to preserve the motor response.

The catheter is advanced approximately not more than 3 cm beyond the tip of the introducer needle. If neurostimulation is used, an appropriate motor response should be sought at current thresholds of at least 0.3 mA and up to 0.8–1 mA. Catheter tip position may also be confirmed by (1) attempting to visualize the catheter tip (this is aided by making small jiggling movements of the catheter); (2) rapid injection of 3–5 ml of fluid which should produce visible expansion posterior to the artery; and (3) injection of agitated saline to produce a color Doppler signal adjacent to the artery. The needle is then withdrawn and the catheter fixed in place in the usual manner.

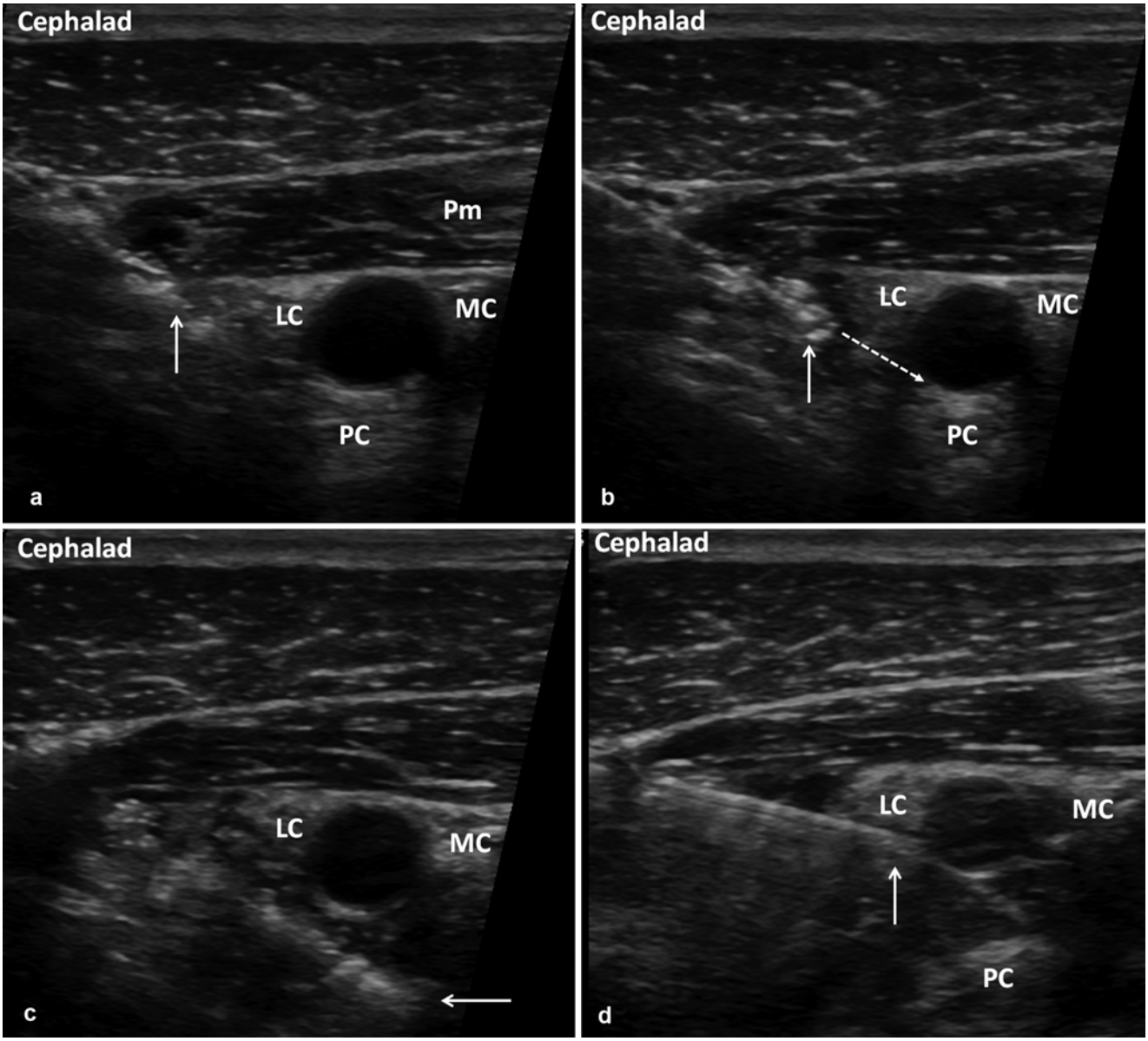


Fig. 31.9 Typical performance of an ultrasound-guided infraclavicular block. (**a**) The needle tip (*arrow*) pierces the deep fascia of pectoralis minor (*Pm*), just lateral to the lateral cord (*LC*). (**b**) 0.5–1 ml of fluid is injected to confirm that the needle tip is in the correct compartment and to push aside the lateral cord so that the needle can reach the posterior aspect of the artery (*dotted arrow* indicates trajectory). (**c**) Injection at the posterior aspect of the artery creates a pocket of local anesthetic that spreads between the artery and the posterior cord (*PC*) and medial cord (*MC*)—the "double bubble" sign. (**d**) In the dual-injection technique, after injection of 20–30 ml posterior to the artery, the needle tip is withdrawn back into the compartment of the lateral cord where a further 5–10 ml is injected (Image courtesy of KJ Chin MPC)

Local Anesthetic Dosages

Single-Shot Block

- A volume of 30–40 ml (0.4–0.5 ml/kg) is generally recommended.
- The choice of local anesthetic and concentration depends on the desired speed of onset and duration. Commonly used local anesthetics include:
  - A 1:1 mixture of an intermediate-acting local anesthetic (e.g., 2 % mepivacaine or lidocaine) and a long-acting local anesthetic (e.g., 0.5 % bupivacaine), which will have a faster onset than bupivacaine alone and a longer duration than lidocaine alone.
  - 0.25–0.5 % bupivacaine
  - 0.5–0.75 % ropivacaine
- Epinephrine may be added in a concentration of 25 mcg/ml as a marker of intravascular injection and to reduce systemic vascular absorption.

Continuous Infraclavicular Plexus Block

- 0.2–0.4 % ropivacaine at 4–8 ml/h [17, 18].
- 0.125–0.25 % bupivacaine at 4–8 ml/h.
- PCA boluses of 2–5 ml may be added if this function is available on the infusion pump.
- Both infusion rates and bolus volumes should be titrated to achieve the optimal balance between adequate analgesia and an excessively dense sensory and motor block.

Complications and Adverse Effects

In general, the infraclavicular block is an extremely safe technique. The following adverse events have been reported but all are uncommon.

Vascular Puncture

- This is the most commonly reported complication in the literature [3].
- Axillary vein puncture is more likely than arterial puncture in the neurostimulation-guided approach as the brachial plexus cords envelop and protect the axillary artery.
- Arterial puncture should be avoidable with careful needle handling in the ultrasound-guided approach as it is difficult to puncture the thick elastic arterial wall with a short-beveled block needle.
- The main concern is that this area can be difficult to compress, and coagulopathy is therefore a relative contraindication to the infraclavicular block. However, no significant complications arising as a result of vascular puncture have been reported to date.

Pneumothorax

- The only published report of pneumothorax following a surface landmark-guided approach is with the vertical infraclavicular plexus approach [19], which is more medial than the coracoid approach.
- Although pneumothorax has been reported following ultrasound-guided infraclavicular block [20], it is extremely unlikely if proper technique is observed [21], given the distance between the plexus and the pleura in most patients. An out-of-plane approach and advancement of the needle in a medial direction may increase risk.

Phrenic Nerve Palsy and Horner's Syndrome

- Both of these complications are extremely unlikely, as they require spread of local anesthetic cephalad into the neck.
- They have however been reported with the vertical infraclavicular plexus block, which, as previously mentioned, targets the plexus in a more proximal/medial location.

Infraclavicular block of the brachial plexus

Continuous technique ☐ Right ☐ Left

Technique: ☐ *Vertical infraclavicular* ☐ *Other*

Purpose of block: ☐ *Surgical* ☐ *Therapeutic*
i.v. access: ☐ *Yes*
Monitoring: ☐ *ECG* ☐ *Pulse oximetry*
Ventilation facilities: ☐ *Yes (equipment checked)*
Emergency equipment *(drugs):* ☐ *Checked*
Patient: ☐ *Informed*

Position: ☐ *Supine*
Puncture technique: ☐ *Electrostimulation*
☐ Ultrasound - guided
☐ *Transducer* ☐ *Linear* ☐ *Curved*
☐ *Approach* ☐ *In Plane* ☐ *Out of plane*
Needle type ☐ *Contiplex® D* ___ *mm* ___ *G* ☐ *Tuohy* ___ *mm* ___ *G*
☐ *Other* _____
Catheter: ☐ *Advanced* _____ *cm*
Aspiration test: ☐ *Carried out*
Bacterial filter: ☐
Test dose: _____ *mL* _____ *%* _____
Bolus administration: _____ *mL* _____ *%* _____
(in incremental doses)
Addition to injection solution: ☐ *No* ☐ *Yes* _____ *µg / mg*
Patient's remarks during injection:
☐ *None* ☐ *Paresthesias* ☐ *Warmth*
☐ *Pain triggered (intraneural location?)* _____
Nerve region _____

Objective block effect after 15 min:
☐ *Cold test* ☐ *Temperature measurement: right* ___ *°C left* ___ *°C*
☐ *Sensory* ☐ *Motor*
☐ *Continuous monitoring*

☐ Infusion for postoperative analgesia
Local anesthetic: _____ *%* _____ *ml/h*
Addition to LA: _____ *mg* _____ *µg* _____
Patient-controlled anesthesia (PCA)
Local anesthetic: _____ *%*
Addition: _____
☐ Baseline rate _____ *mL/h* _____
☐ Bolus administration _____ *ml*
☐ Lockout interval _____ *min*

Complications:
☐ *None* ☐ *Signs of intoxication* ☐ *Pneumothorax*
☐ *Hematoma* ☐ *Neurological injury (median nerve, ulnar nerve, radial nerve)*

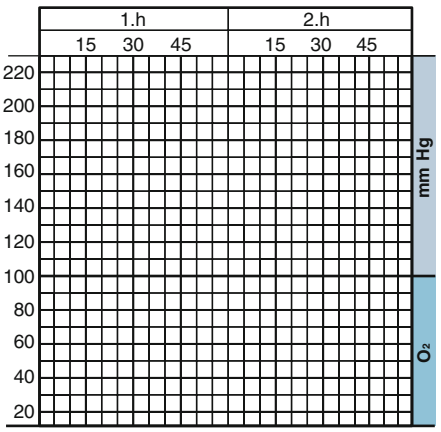
Subjective effects of the block: *Duration:* _____
☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*
VISUAL ANALOG SCALE

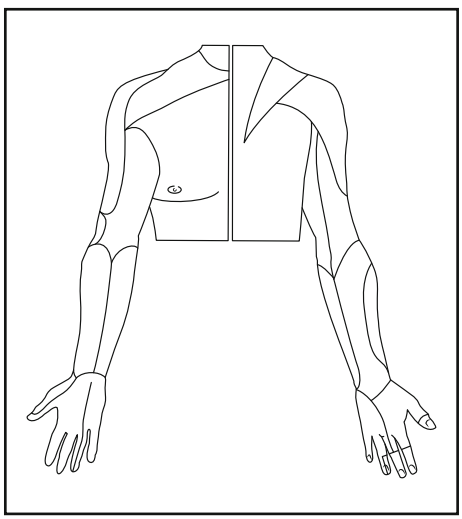
0 10 20 30 40 50 60 70 80 90 100

Record and checklist

Name: _____
Date: _____
Diagnosis: _____
Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No
☐ Yes (which?) _____

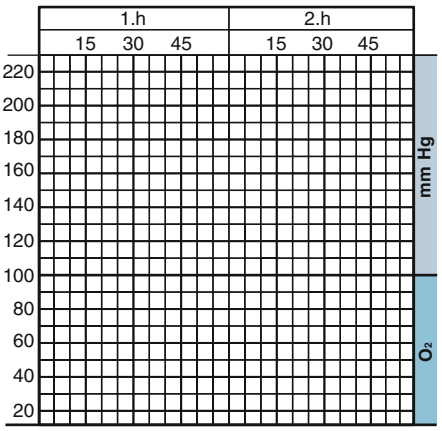


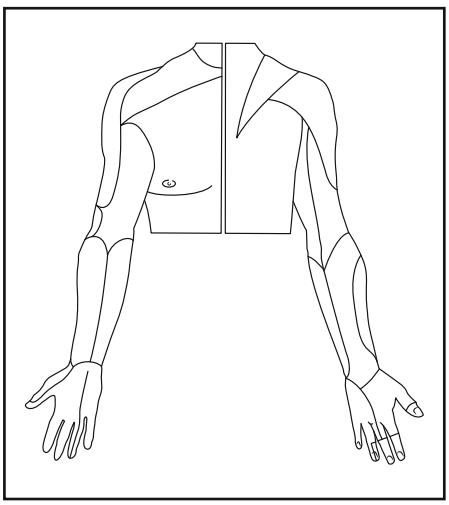


Special notes:
_____
_____
_____

With permission from Danilo Jankovic

Record and checklist





With permission from Danilo Jankovic

Infraclavicular block of the brachial plexus

"Single–shot"–technique ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No

☐ Yes (which?) _____

Technique: ☐ *Vertical infraclavicular* ☐ *Other*

Purpose of block: ☐ *Surgical* ☐ *Therapeutic*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine*

Needle type: ☐ *Stimuplex®* *D* ____ *mm (15°)* ☐ *Other* _____

Puncture technique: ☐ *Electrostimulation* ☐ *Ultrasound guided* ☐ *Transducer*
☐ *Linear* ☐ *Curved* ☐ *Approach* ☐ *In Plane* ☐ *Out of Plane*

Local anesthetic: _____ *ml* _____ *%* _____
(in incremental doses)

Addition to injection solution: ☐ *No* ☐ *Yes* _____ *µg / mg*

Patient's remarks during injection:
☐ *None* ☐ *Paresthesias* ☐ *Warmth*

Pain triggered (intraneural location?) _____

Nerve region _____

Objective block effect after 15 min:
☐ *Cold test* ☐ *Temperature measurement: right* ___*°C* *left* ___*°C*
☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge _____

Complications: ☐ *None* ☐ *Signs of intoxication* ☐ *Pneumothorax*
☐ *Hematoma* ☐ *Neurological injury (median nerve, ulnar nerve, radial nerve)*

Subjective effects of the block: *Duration:* _____
☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

References

1. 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.
2. Kilka HG, Geiger P, Mehrkens HH. Infraclavicular vertical brachial plexus blockade. A new method for anesthesia of the upper extremity. An anatomical and clinical study. Anaesthesist. 1995;44: 339–44.
3. Chin KJ, Alakkad H, Adhikary SD, Singh M. Infraclavicular brachial plexus block for regional anaesthesia of the lower arm. Cochrane Database Syst Rev. 2013;(8):CD005487.
4. Grossi P. Brachial plexus block. The anaesthetic line is a guide for new approaches. Minerva Anestesiol. 2001;67:45–9.
5. Dingemans E, Williams SR, Arcand G, Chouinard P, Harris P, Ruel M, Girard F. Neurostimulation in ultrasound-guided infraclavicular block: a prospective randomized trial. Anesth Analg. 2007;104: 1275–80, tables of contents.
6. Sharma D, Srivastava N, Pawar S, Garg R, Nagpal VK. Infraclavicular brachial plexus block: comparison of posterior cord stimulation with lateral or medial cord stimulation, a prospective double blinded study. Saudi J Anaesth. 2013;7:134–7.
7. Rodríguez J, Taboada M, Oliveira J, Ulloa B, Bárcena M, Alvarez J. Single stimulation of the posterior cord is superior to dual nerve stimulation in a coracoid block. Acta Anaesthesiol Scand. 2010;54:241–5.
8. Lecamwasam H, Mayfield J, Rosow L, Chang Y, Carter C, Rosow C. Stimulation of the posterior cord predicts successful infraclavicular block. Anesth Analg. 2006;102:1564–8.
9. Minville V, Fourcade O, Bourdet B, Doherty M, Chassery C, Pourrut J-C, Gris C, Eychennes B, Colombani A, Samii K, Bouaziz H. The optimal motor response for infraclavicular brachial plexus block. Anesth Analg. 2007;104:448–51.
10. Yang CW, Jung SM, Kwon HU, Kang PS, Cho CK, Oh JY, Lee Y, Choi J. A comparison of posterior and medial cord stimulation for neurostimulation-guided vertical infraclavicular block: a randomized noninferiority clinical trial. Anesth Analg. 2014;118:874–8.
11. Moayeri N, Renes S, van Geffen GJ, Groen GJ. Vertical infraclavicular brachial plexus block: needle redirection after elicitation of elbow flexion. Reg Anesth Pain Med. 2009;34:236–41.
12. Auyong DB, Gonzales J, Benonis JG. The Houdini clavicle: arm abduction and needle insertion site adjustment improves needle visibility for the infraclavicular nerve block. Reg Anesth Pain Med. 2010;35:403–4.
13. Bigeleisen P, Wilson M. A comparison of two techniques for ultrasound guided infraclavicular block. Br J Anaesth. 2006;96:502–7.
14. Tran DQH, Charghi R, Finlayson RJ. The “double bubble” sign for successful infraclavicular brachial plexus blockade. Anesth Analg. 2006;103:1048–9.
15. De Tran QH, Bertini P, Zaouter C, Muñoz 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.
16. Fredrickson MJ, Wolstencroft P, Kejriwal R, Yoon A, Boland MR, Chinchanwala S. Single versus triple injection ultrasound-guided infraclavicular block: confirmation of the effectiveness of the single injection technique. Anesth Analg. 2010;111:1325–7.
17. Ilfeld BM, Le LT, Ramjohn J, Loland VJ, Wadhwa AN, Gerancher JC, Renehan EM, Sessler DI, Shuster JJ, Theriaque DW, Maldonado RC, Mariano ER, PAINfRETM Investigators. The effects of local anesthetic concentration and dose on continuous infraclavicular nerve blocks: a multicenter, randomized, observer-masked, controlled study. Anesth Analg. 2009;108:345–50.
18. Ilfeld BM, Morey TE, Enneking FK. Infraclavicular perineural local anesthetic infusion: a comparison of three dosing regimens for postoperative analgesia. Anesthesiology. 2004;100:395–402.
19. Mehrkens HH, Geiger PK. Continuous brachial plexus blockade via the vertical infraclavicular approach. Anaesthesia. 1998;53 Suppl 2:19–20.
20. Koscielniak-Nielsen ZJ, Rasmussen H, Hesselbjerg L. Pneumothorax after an ultrasound-guided lateral sagittal infraclavicular block. Acta Anaesthesiol Scand. 2008;52:1176–7.
21. Gauss A, Tugtekin I, Georgieff M, Dinse-Lambracht A, Keipke D, Gorsewski G. Incidence of clinically symptomatic pneumothorax in ultrasound-guided infraclavicular and supraclavicular brachial plexus block. Anaesthesia. 2014;69:327–36.
22. Lanz E, Theiss D, Jankovic D. The extent of blockade following various techniques of brachial plexus block. Anesth Analg. 1983;62:55–8.

Chapter 32

Axillary Brachial Plexus Block

Ki Jinn Chin, MBBS, MMed., FANZCA, FRCPC
Department of Anesthesia, Toronto Western Hospital, Toronto, Canada
e-mail: Kijinn.chin@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_32,

Introduction

The axillary brachial plexus block has a long history as a popular technique for providing regional anesthesia at and below the elbow. The surface landmarks (chiefly the axillary artery pulsation) are easily ascertained, and there is no risk of pneumothorax. A variety of landmark-guided approaches have been described, including elicitation of paresthesia, transarterial injection, and single- and multiple-injection neurostimulation techniques. The main issues with the landmark-guided approaches were variable efficacy [1] and the risk of intravascular injection of local anesthetic. The advent of ultrasound guidance has effectively addressed these concerns. It is at least as effective as the supraclavicular and infraclavicular blocks, if not more so because of the ability to individually target the four main terminal nerves of the arm. The shallow depth of the brachial plexus in this location allows excellent visualization of both nerves and needle. Inadvertent intraneural and intravascular injections remain the only significant risks, but both are easily avoided with ultrasonographic visualization.

Indications

- Anesthesia or analgesia of the elbow, forearm, and hand (Fig. 32.1).
- It may be performed as either a single-shot or continuous catheter technique; however it can be more difficult to maintain hygiene of the catheter insertion site compared to other brachial plexus approaches.

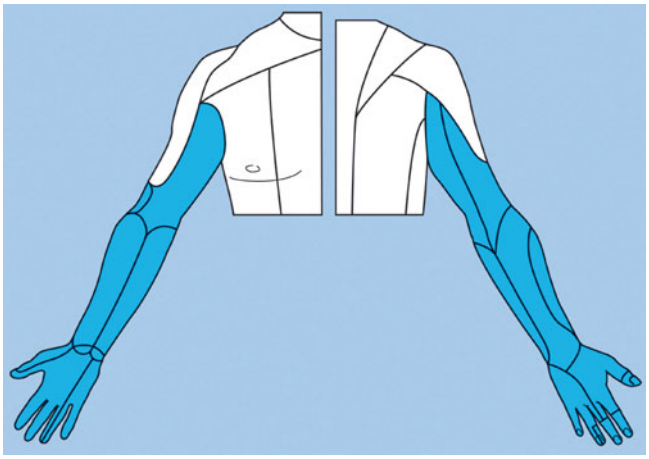


Fig. 32.1 Typical distribution of anesthesia and analgesia following successful axillary plexus blockade [13] (With permission from Danilo Jankovic)

Contraindications

- Absolute
  - Inability to abduct the arm at the shoulder and access the injection site
  - Generic contraindications such as patient refusal, allergy to local anesthetics, and local infection at the site of injection apply as usual
- Relative
  - Previous axillary node clearance
  - Congenital or acquired vascular malformations in the upper arm or axilla (e.g., arteriovenous fistula), although the ultrasound-guided approach may still be feasible in skilled hands

Advantages Compared to Other Brachial Plexus Block Techniques

1. The ultrasound-guided technique is a highly efficacious technique, with block success rates as high as 97.5 % reported [2].
2. It is a superficial site that is easily compressible and carries a low risk of compressive injury to surrounding structures by hematoma formation. This makes it a viable option in coagulopathic patients, particularly if ultrasound guidance is used.
3. There is no risk of pneumothorax or of adverse effects associated with injections administered more proximally in the root of the neck, e.g., phrenic nerve palsy and Horner's syndrome.
4. Adequate ultrasound images can be obtained even in very obese patients.

Disadvantages Compared to Other Brachial Plexus Block Techniques

1. The axillary block can only be performed with the arm abducted (and ideally externally rotated) at the shoulder.
2. The landmark-guided techniques of axillary block have relatively poor block success rates, especially if single-injection techniques are used. Multiple-injection techniques are complex and require good understanding of anatomy as well as dexterity with needle and nerve stimulator.

Functional Anatomy

At the level of the axillary block, the cords of the brachial plexus have divided into the major terminal nerves: the median nerve, ulnar nerve, radial nerve, musculocutaneous nerve, and medial brachial and antebrachial cutaneous nerves (Fig. 32.2). These last two nerves are not usually sought out separately as they lie close to the ulnar nerve and are readily anesthetized with it.

The nerves are distributed around the axillary artery, together with one or more accompanying axillary veins, and together they constitute the neurovascular bundle (Fig. 32.3). The median nerve is most commonly located in the anterolateral quadrant, the ulnar nerve in the anteromedial quadrant, and the radial nerve in the posteromedial quadrant around the artery (Fig. 32.4). However, these nerves are highly mobile, and on ultrasound, their position can be observed to vary significantly with the pressure exerted through the probe. The musculocutaneous nerve starts in a location adjacent to the median nerve in the posterolateral quadrant around the artery but then courses out laterally in the fascial plane between biceps and coracobrachialis muscle. Its exact location in any given subject is highly variable and depends on how proximally or distally it takes off from the lateral cord; this is the main reason for the high failure rate with single-injection landmark-guided approaches [1].

A fascial sheath surrounds the brachial plexus in this location but is subdivided by thinner septa into compartments that contain the median, ulnar, and radial nerves. These are most evident during ultrasound-guided perineural block. While large-volume injection in a single location close to the artery may traverse these septa, they are nevertheless likely to be another reason for the poor efficacy of the single-injection landmark-guided techniques [1].

The most important landmark in the ultrasound-guided approach, apart from the axillary artery, is the conjoint tendon of teres major and latissimus dorsi (Figs. 32.3 and 32.4). This inserts onto the proximal humerus and forms a "floor" on which the artery and all the associated nerves lie [3]. In particular the radial nerve can always be visualized lying superficial to the conjoint tendon on ultrasound. Distal to the lateral edge of the conjoint tendon, the radial nerve rapidly courses posteriorly and away from the axillary artery, descending between the heads of the triceps muscle to lie in the spiral groove of the humerus. Ultrasound-guided axillary nerve blocks performed where the conjoint tendon cannot be visualized therefore carry a significant risk of failure to block the radial nerve.

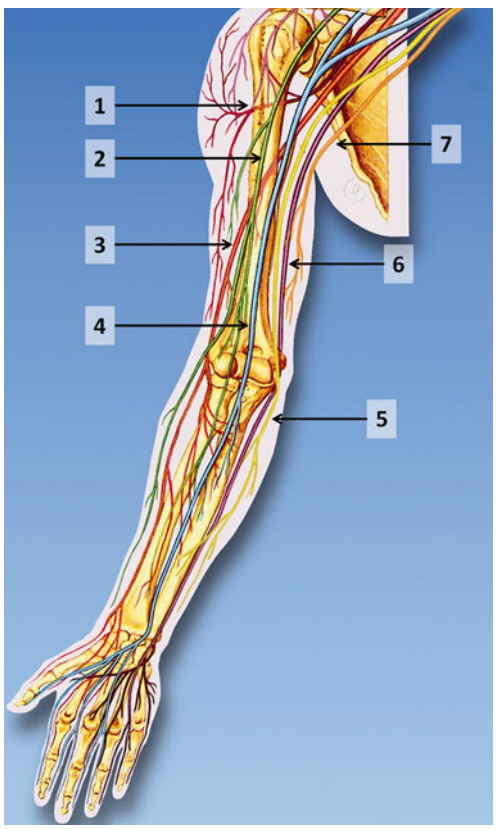


Fig. 32.2 Major terminal branches of the brachial plexus in the axillary region. (*1*) Axillary nerve, (*2*) musculocutaneous nerve, (*3*) radial nerve, (*4*) median nerve, (*5*) medial antebrachial cutaneous nerve, (*6*) ulnar nerve, (*7*) medial brachial cutaneous nerve (With permission from Danilo Jankovic)

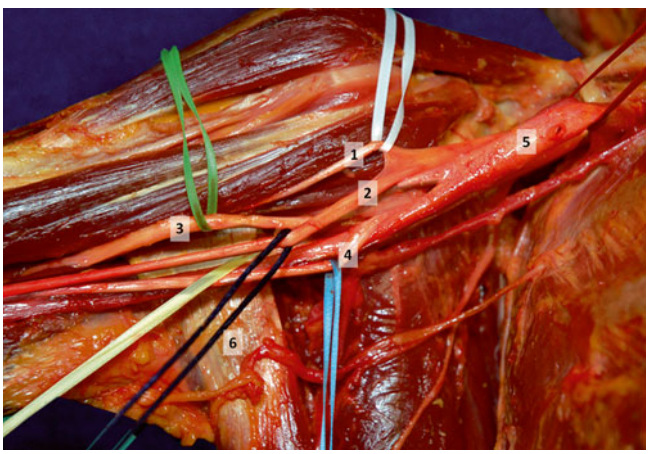


Fig. 32.3 Cadaveric dissection of the axillary brachial plexus. The musculocutaneous nerve (*1*) (*white*) and median nerve (*2*) (*dark blue*) originate from the lateral cord. The radial nerve (*3*) (*green*) and ulnar nerve (*4*) (*light blue*) run posterior and medial to the axillary artery (*5*) but have been displaced here for visibility. Note the conjoint tendon (*6*) of teres major and latissimus dorsi which inserts onto the humerus posterior to the axillary neurovascular bundle (With permission from Danilo Jankovic)

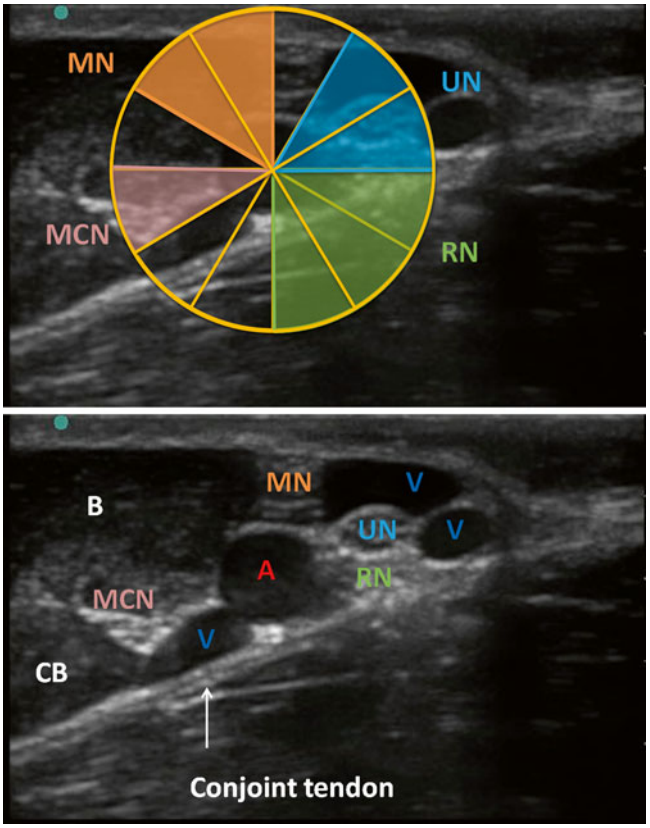


Fig. 32.4 Typical ultrasound image of the axillary brachial plexus. The pulsatile axillary artery (*A*) is usually accompanied by more than one vein (*V*). All the vessels and nerves lie on the hyperechoic conjoint tendon. The most common location of the median nerve (*MN*), ulnar nerve (*UN*), radial nerve (*RN*), and musculocutaneous nerve (*MCN*) in relation to the artery is illustrated in the *top image*. The musculocutaneous nerve often lies in the fascial plane between biceps (*B*) and coracobrachialis (*CB*) (Image courtesy of KJ Chin MPC)

Technique: Surface Landmark-Guided Approach, Single-Shot

Required Supplies and Equipment

- Disinfectant solution and swabs for skin preparation
- Sterile gloves and drapes
- Short-beveled 22-G, 50-mm block needle with extension tubing
  - Note that a sharp hypodermic needle may be preferred in the transarterial approach.
- Local anesthetic of choice in 10- or 20-ml syringes
- Peripheral nerve stimulator (optional, depending on approach)
- Lidocaine 1–2 % in 3-ml syringe with a 25–27-G hypodermic needle for skin infiltration (at operator's discretion)
- Equipment and supplies for managing life-threatening acute complications, including Intralipid for local anesthetic systemic toxicity
- Drugs for intravenous sedation during the block (at operator's discretion)

Preparation of Patient

- Obtain informed consent for the block.
- Explain expected clinical course including care of the insensate limb and managing the transition to systemic analgesia.
- Establish intravenous access, supplemental oxygen delivery, and standard monitors (ECG, noninvasive blood pressure, pulse oximetry)
- Perform a time-out to confirm patient identity and site and side of surgery.

Block Performance

Patient Position

- The patient is placed supine with the arm externally rotated and abducted to 90° at the shoulder, with the elbow flexed (Fig. 32.5). Abduction beyond 90° should be avoided, as it makes arterial palpation more difficult and may impair distribution of the local anesthetic.
- The operator stands on the side of the patient to be blocked.

Surface Landmarks

- The axillary artery is palpated as proximally as possible under the lateral edge of the pectoralis major muscle (Fig. 32.6). This increases the efficacy of the block for two reasons: (1) there is a higher likelihood that the musculocutaneous nerve will be close enough to the plexus for local anesthetic spread to reach it; (2) the plexus is targeted where it lies on the conjoint tendon, which is an important boundary for containing and directing local anesthetic spread, especially around the radial nerve.
- The median nerve is expected to lie superior and anterior (superficial) to the artery, the ulnar nerve lies inferior and posterior, and the radial nerve lies posterior (deep) to the artery.

Needle Insertion and Injection Technique

A variety of methods of nerve localization have been described, including the elicitation of paresthesia and seeking a fascial click to indicate entry into the fascial sheath [4, 5]. These are primarily of historical interest and will not be described further here. The two methods that will be described are:

1. Transarterial injection technique [6]
2. Neurostimulation-guided technique

1. Transarterial double-injection technique

   - Disinfect the skin, drape the area appropriately, and prime the block needle and tubing with local anesthetic solution.
   - Place the 2nd and 3rd fingers of the nondominant hand on either side of the artery to anchor it in position.
   - Advance the block needle at a 90° angle to the skin to puncture the artery, as evidenced by pulsatile backflow of bright-red blood into the tubing (Fig. 32.7).
   - Advance the needle further until blood can no longer be aspirated, signaling that the needle tip is now posterior (deep) to the artery. Fractionated injection of half of the total dose of local anesthetic is performed at this location.
   - Withdraw the needle slowly until the needle tip is lying just superficial to the artery, signaled again first by resumption and then cessation of pulsatile blood flow. The remaining volume of local anesthetic is injected in this location.
   - The primary concerns with the transarterial approach are (1) an increased risk of intravascular injection and local anesthetic systemic toxicity and (2) hematoma formation. Nevertheless it is an effective technique that does not require additional equipment (nerve stimulator or ultrasound machine).

2. Neurostimulation-guided technique

   - Disinfect the skin, drape the area appropriately, prime the block needle and tubing with local anesthetic solution, and attach a nerve stimulator at an initial current setting of 1–1.5 mA, pulse duration 0.1–0.3 ms, and frequency of 1–2 Hz.
   - Place the 2nd and 3rd fingers of the nondominant hand on either side of the artery to anchor it in position (Fig. 32.8). Infiltrate the skin insertion site with local anesthetic if desired.
   - Insert the block needle at a 45° angle to the skin in a proximal direction, aiming for the anterolateral aspect of the artery where the median nerve is expected to lie. Once a median nerve response is located at a current threshold of 0.3–0.5 mA, 5–8 ml of local anesthetic is injected. The needle is withdrawn to the skin and redirected to the anteromedial aspect of the artery, where the ulnar nerve is expected to lie. The radial nerve lies deeper on the posteromedial aspect of the artery. Finally, the musculocutaneous nerve is sought by redirecting the needle lateral to the arterial pulsation, in the groove between biceps and coracobrachialis. Once an appropriate motor response is obtained at a current threshold of 0.3–0.5 mA, 5–8 ml of local anesthetic is injected at each location.
   - Variations of the multiple-injection technique have been described, ranging from a single-injection on a motor response from median, ulnar, or radial nerves to a double injection [1]. It is probably most effective to perform the single injection posterior to the artery, i.e., on a radial nerve response. Double injections should be performed anterior and posterior to the artery, i.e., median or ulnar + radial. Larger volumes (40–50 ml) of local anesthetic are recommended to try and ensure adequate spread. The musculocutaneous nerve is usually sought out separately.
   - It is recommended that the 2nd and 3rd fingers of the nondominant hand which are palpating the artery also be used to exert pressure distal to the site of injection, which promotes cephalad spread and increases the effectiveness of the block, particularly if the musculocutaneous nerve is not separately sought and injected. Compression and massage of the axilla for 3–5 min after completion of injection has also been recommended for the same reason.
   - If paresthesias are obtained, the needle should be withdrawn very slightly to avoid intraneural injection.

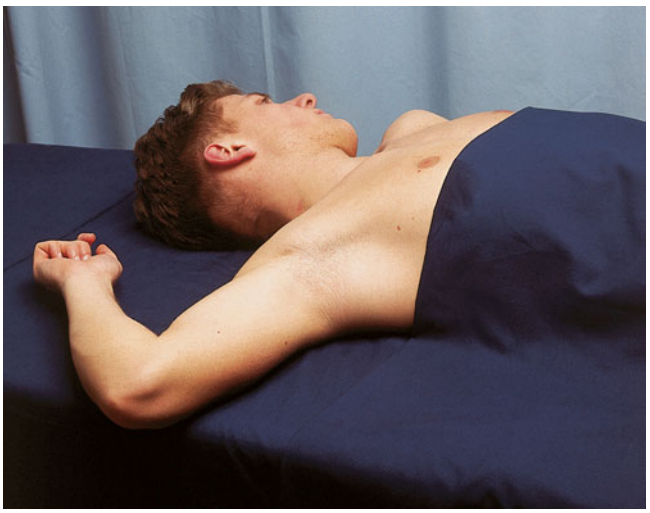


Fig. 32.5 Patient positioning for the axillary block. The patient is supine with the arm externally rotated and abducted to 90° at the shoulder. The shoulder should not be abducted more than 90°, as it makes arterial palpation more difficult, distorts the ultrasonographic view, and may impair distribution of the local anesthetic [8] (With permission from Danilo Jankovic)

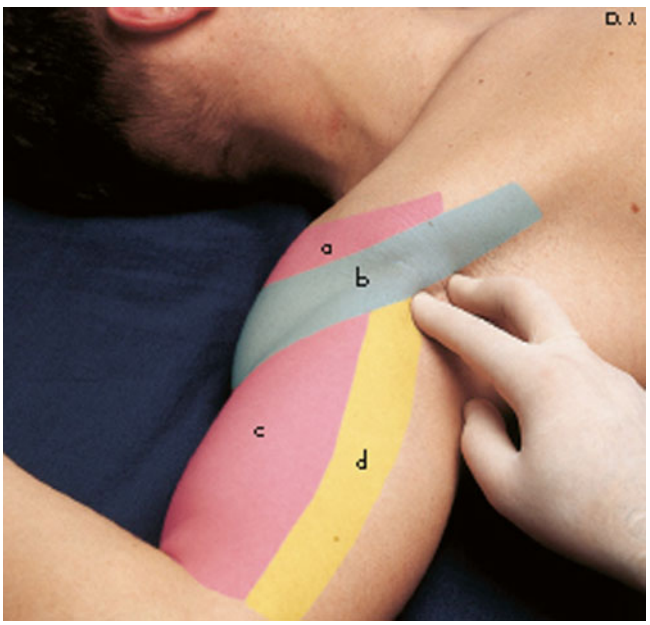


Fig. 32.6 Palpation of the axillary artery. This is done with two fingers (usually the 2nd and 3rd fingers), so that the skin and artery may be fixed in position between them. Palpation should be performed as proximal as possible (*a* deltoid muscle, *b* pectoralis major muscle, *c* biceps muscle, *d* coracobrachialis muscle) (With permission from Danilo Jankovic)

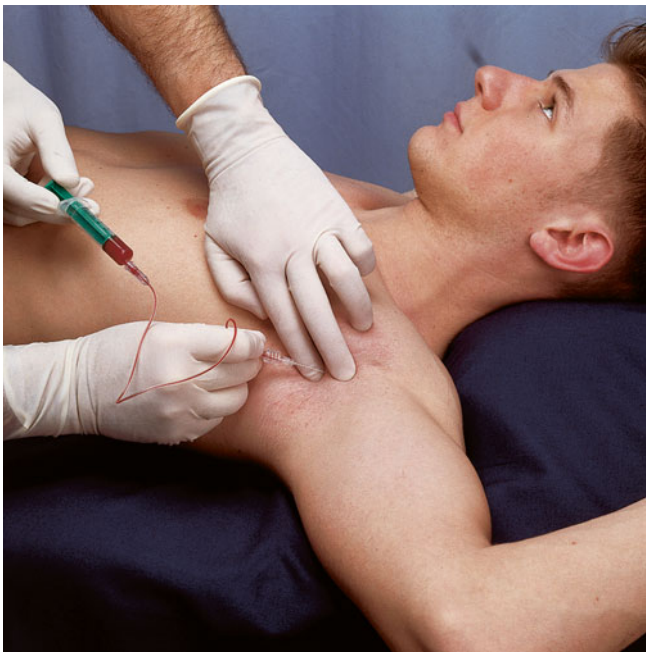


Fig. 32.7 Performance of the transarterial axillary block. The artery is fixed firmly between the fingers of the nondominant hand. The operator advances the block needle, while an assistant aspirates to confirm that the artery has been pierced. The needle is advanced further until blood can no longer be aspirated, signaling that the needle tip is now posterior to the artery. Half the volume of local anesthetic is injected in this location (in fractionated doses). The needle tip is withdrawn; blood is again aspirated as the needle tip reenters the arterial lumen and ceases as the tip exits the lumen to lie anterior to the artery, at which time the remaining half of local anesthetic is injected (again in fractionated doses) (With permission from Danilo Jankovic)

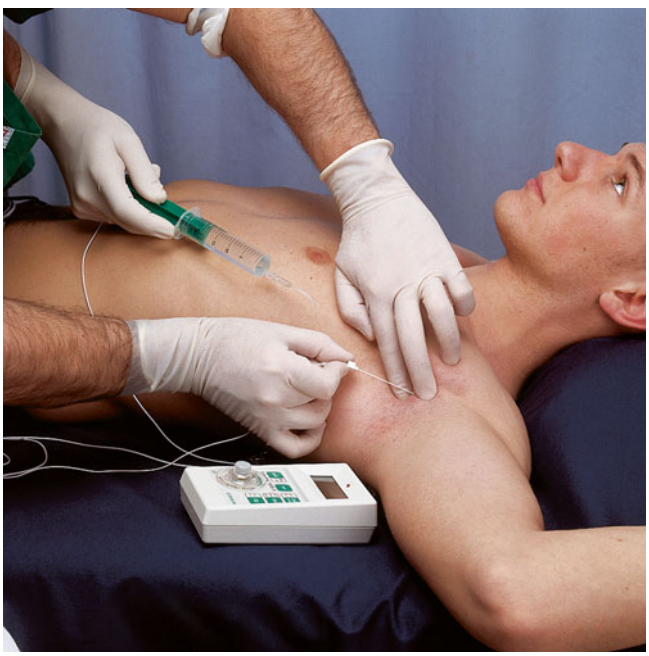


Fig. 32.8 Performance of the neurostimulation-guided axillary brachial plexus block. The artery is fixed firmly between the fingers of the nondominant hand. The operator advances the block needle, while an assistant adjusts the nerve stimulator as required. The operator redirects the needle around the artery to locate one or all of the median, ulnar, radial, and musculocutaneous nerves, depending on whether a single- or multiple-injection technique is employed (With permission from Danilo Jankovic)

Landmark-Guided Continuous Nerve Block Technique

This is similar to the neurostimulation-guided single-shot technique except that a peripheral nerve block catheter kit is used instead.

The patient is positioned as described above, and the introducer needle is advanced at a 45° angle to the skin in a proximal direction, aiming to pass just medial to the artery (Fig. 32.9a–c). Ideally, a radial nerve response is sought as this indicates that the needle tip is posterior to the artery. If a stimulating catheter is being used, 5–10 ml of 5 % dextrose solution can be injected through the introducer needle to distend the paraneural sheath. This should also result in augmentation of the motor response. Injection of other solutions will abolish the motor response.

The catheter is then advanced 3–5 cm beyond the tip of the introducer needle, looking to maintain a brachial plexus motor response at current thresholds of at least 0.3 mA and up to 0.8–1 mA. The needle is then withdrawn and the catheter fixed in place in the usual manner.

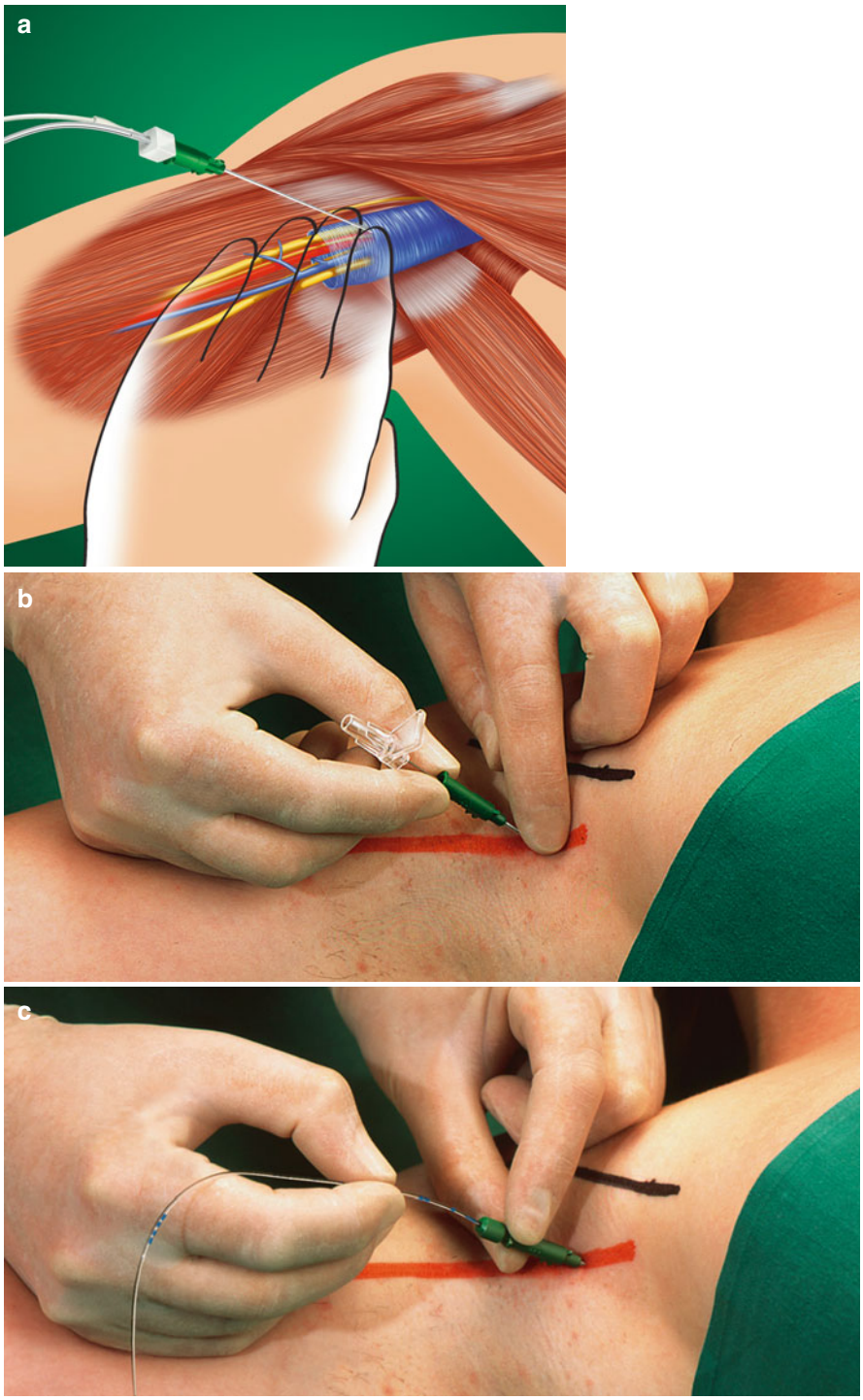


Fig. 32.9 Continuous axillary brachial plexus blockade. (**a**) Schematic illustration of the needle approach. The needle tip is placed within the brachial plexus sheath. (**b**) In this particular model of continuous peripheral nerve catheter (Contiplex® D or Contiplex® Tuohy continuous set, e.g., B. Braun Melsungen), an introducer cannula is first inserted into the brachial plexus sheath over the needle. (**c**) The nerve catheter is then threaded through the introducer cannula (With permission from Danilo Jankovic)

Technique: Ultrasound-Guided Approach, Single-Shot

Required Supplies and Equipment

- Disinfectant solution and swabs for skin preparation
- Sterile gloves and drapes
- Short-beveled 22-G block needle with extension tubing
  - An 80-mm needle is recommended in all adult patients.
  - An echogenic needle helps in needle localization.
- Local anesthetic of choice in 10- or 20-ml syringes
- Lidocaine 1–2 % in 3-ml syringe with a 25–27-G hypodermic needle for skin infiltration (at operator's discretion)
- Ultrasound machine with high-frequency linear array probe
- Peripheral nerve stimulator (optional)
- Equipment and supplies for managing life-threatening acute complications, including Intralipid for local anesthetic systemic toxicity
- Drugs for intravenous sedation during the block (at operator's discretion)

Preparation of Patient

- Obtain informed consent for the block.
- Explain expected clinical course including care of the insensate limb and managing the transition to systemic analgesia.
- Establish intravenous access, supplemental oxygen delivery, and standard monitors (ECG, noninvasive blood pressure, pulse oximetry).
- Perform a time-out to confirm patient identity and site and side of surgery.

Block Performance

Patient and Operator Position

- The patient is placed supine with the arm externally rotated and abducted to 90° at the shoulder, with the elbow flexed. Abduction beyond 90° should be avoided, as it alters the sonoanatomy and can make image recognition more difficult.
- It is recommended that the operator stand on the side of the patient to be blocked, either at the head of the patient or at the patient's side depending on operator preference. The ultrasound machine should be positioned in the operator's direct line of sight, i.e., toward the foot of the bed on the same side or on the opposite side of the patient, depending on whether the operator is standing at the head or at the side of the patient, respectively.

Pre-scanning and Identification of Anatomy

- The transducer is oriented perpendicular to the axis of the brachial plexus and axillary artery to image the neurovascular bundle in cross section. It is essential that the transducer is placed as high up (proximal) in the axilla as possible. The conjoint tendon of the latissimus dorsi and teres major is identified as a sloping hyperechoic line on ultrasound (Fig. 32.4). The entire neurovascular bundle will be visible superficial (anterior) to it.
- The veins in the axillary neurovascular bundle are revealed by varying the downward pressure of the transducer in a "bouncing" motion to alternately expand and compress the veins. This helps to minimize the risk of inadvertent puncture and often helps delineate the nerves, which might otherwise appear as a single hyperechoic mass.
- The median, ulnar, and radial nerves lie in characteristic locations around the artery (Fig. 32.4) and are readily identified with experience. Their identity can further be confirmed by using a "traceback" approach of scanning distally along the arm and observing the characteristic course that each nerve takes.
- The median and ulnar nerves both lie very close to the axillary artery in the axilla. More distally, the ulnar nerve diverges in a medial direction away from the artery, while the median nerve remains adjacent to the artery. Because of its subcutaneous location, the ulnar nerve can be difficult to distinguish from pockets of adipose tissue on a static image but is easily recognizable on dynamic scanning.
- The radial nerve is always found sandwiched between the axillary artery and the conjoint tendon. Its appearance in this location, as a hyperechoic structure with indistinct margins, resembles that of a "cotton-wool ball." Its identity may be further confirmed by scanning distally and observing the nerve descend in a fascial plane between the long and medial heads of the triceps muscle toward the posteromedial aspect of the humerus (Fig. 32.10).
- The location of the musculocutaneous nerve is highly variable. In general, the nerve lies lateral to the axillary artery and, by scanning in a proximal–distal direction, can be observed to "slide" in a lateral–medial direction in the fascial plane between the biceps and coracobrachialis muscles. Its cross-sectional shape varies from triangular to elliptical, depending on its location.

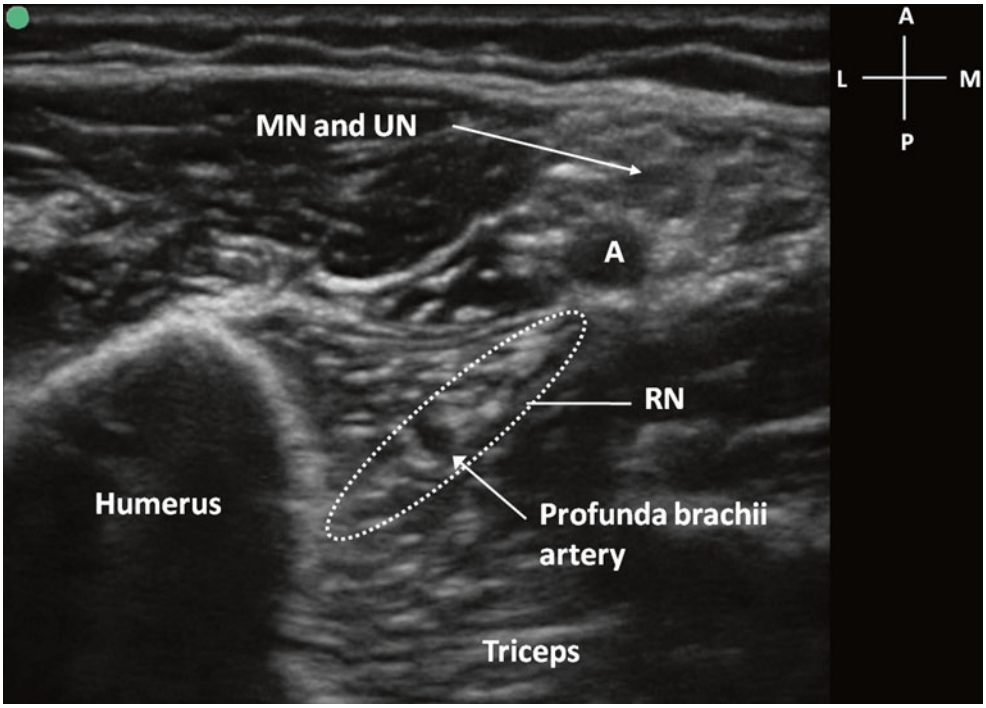


Fig. 32.10 Distal to the conjoint tendon zone, the radial nerve runs posterolaterally in a fascial plane between the long and medial heads of the triceps toward the humerus. It is accompanied by the profunda brachii artery (*A* artery, *MN* median nerve, *RN* radial nerve, *UN* ulnar nerve) (Image courtesy of KJ Chin MPC)

Needle Insertion and Injection Technique

Different methods of performing the axillary block have been described, including using an out-of-plane (OOP) needle approach [7] instead of an in-plane (IP) approach, approaching from both the lateral and medial sides of the probe, and a perivascular technique [8, 9] in which the aim is to deposit local anesthetic in a circumferential pattern around the artery rather than targeting individual nerves (perineural technique). The in-plane perineural approach is described here as we believe it affords greater precision and therefore greater efficacy and safety.

- Having identified the individual nerves as described above, place the probe such that the conjoint tendon is visible and the axillary artery is approximately in the center of the screen.
- The block needle is inserted close to the lateral edge of the probe—this should not be too far away from the plexus or else it creates a longer needle track and hinders needle redirection.
- The block needle is advanced to place the tip onto the conjoint tendon just where it meets the axillary artery (Fig. 32.11). A 0.5-ml bolus of local anesthetic is injected which will hydrodissect the artery and radial nerve off the conjoint tendon. The needle may be advanced a little further under the artery, but does not need to be advanced beyond its 6 o'clock aspect. Injection of 5–8 ml of local anesthetic here will be seen to spread under and around the radial nerve.
- The needle is now withdrawn until the tip lies in the subcutaneous layer. It is then re-advanced in a shallow trajectory toward the median nerve, aiming to pierce its investing fascia at a tangent to the surface of the nerve so as to avoid accidental transfixion of the nerve. Penetration of the fascia is signaled by a tactile "pop," whereupon local anesthetic is injected in 0.5–1-ml boluses to surround the median nerve and to hydrodissect a safe passage toward the ulnar nerve (Fig. 32.12). A total of 5–8 ml of local anesthetic is injected around the median nerve—note that this can be done before advancing toward the ulnar nerve, or it can be done after injection around the ulnar nerve as the needle is being withdrawn (the author's preference).
- The needle is advanced further into the anteromedial quadrant of the neurovascular bundle and adjacent to the ulnar nerve (Fig. 32.13). Injection of 0.5–1-ml boluses of local anesthetic, up to a total of 5–8 ml, in this area will surround the ulnar nerve and usually delineate it clearly.
- Depending on how far lateral the musculocutaneous nerve lies from the axillary artery, the needle may need to be withdrawn and reinserted through a second skin puncture. The nerve lies in the fascial plane between the biceps and coracobrachialis muscles. Once again the needle should be advanced to contact the nerve at a tangent to its surface and pierce this fascial plane (Fig. 32.14a). Three to five milliliters of local anesthetic injected in this plane will spread to encircle the nerve (Fig. 32.14b).
- The endpoint for injection is adequate spread of local anesthetic around the individual nerves.
- Excessive needle repositioning and contact with the nerves is not recommended. Each nerve has its own fascial compartment which directs and contains local anesthetic spread around the nerve.
- Although neurostimulation can be used as an additional confirmation of nerve identification, the operator must be aware that needle–nerve contact can occur even in the absence of a motor response at commonly accepted current thresholds [10, 11]. Aggressive needling merely to provoke a motor response at low current thresholds should be avoided.

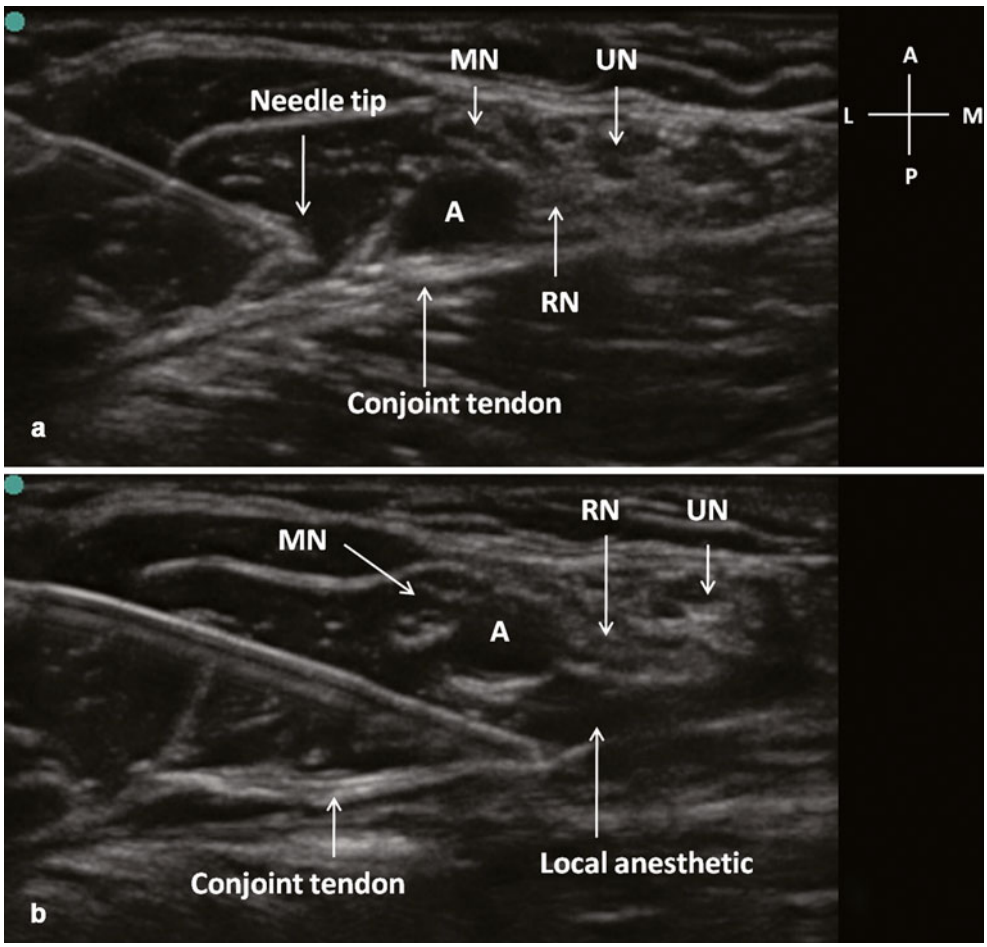


Fig. 32.11 (**a**) The needle tip is advanced toward the posterolateral junction of the axillary artery (*A*) and the conjoint tendon. (**b**) Injection at this point causes local anesthetic to spread anterior to the conjoint tendon and under (posterior to) the radial nerve, lifting it up (*A* artery, *MN* median nerve, *RN* radial nerve, *UN* ulnar nerve) (Image courtesy of KJ Chin MPC)

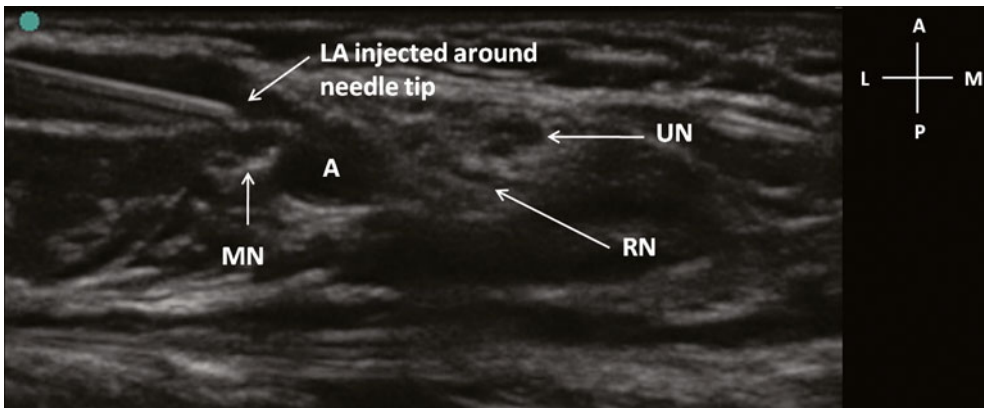


Fig. 32.12 The needle is advanced at a tangent to the median nerve in order to pierce its enveloping fascia without transfixing the nerve. Local anesthetic is injected here to surround the nerve and to hydrodissect a safe passage toward to the ulnar nerve (*A* artery, *MN* median nerve, *RN* radial nerve, *UN* ulnar nerve) (Image courtesy of KJ Chin MPC)

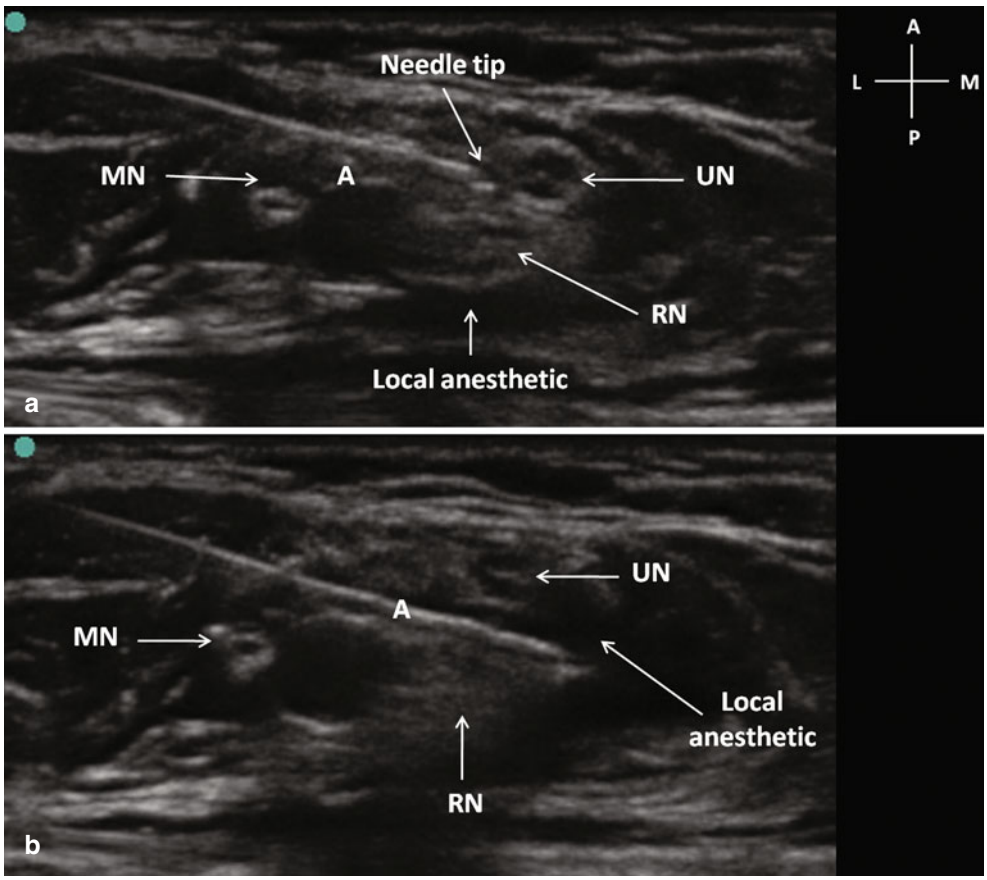


Fig. 32.13 (**a**) The needle is advanced over the axillary artery (*A*) toward the ulnar nerve (*UN*) at a tangent to its surface. (**b**) Injection here surrounds the ulnar nerve (*UN*) and also often spreads superficial to the radial nerve (*RN*) (*MN* median nerve) (Image courtesy of KJ Chin MPC)

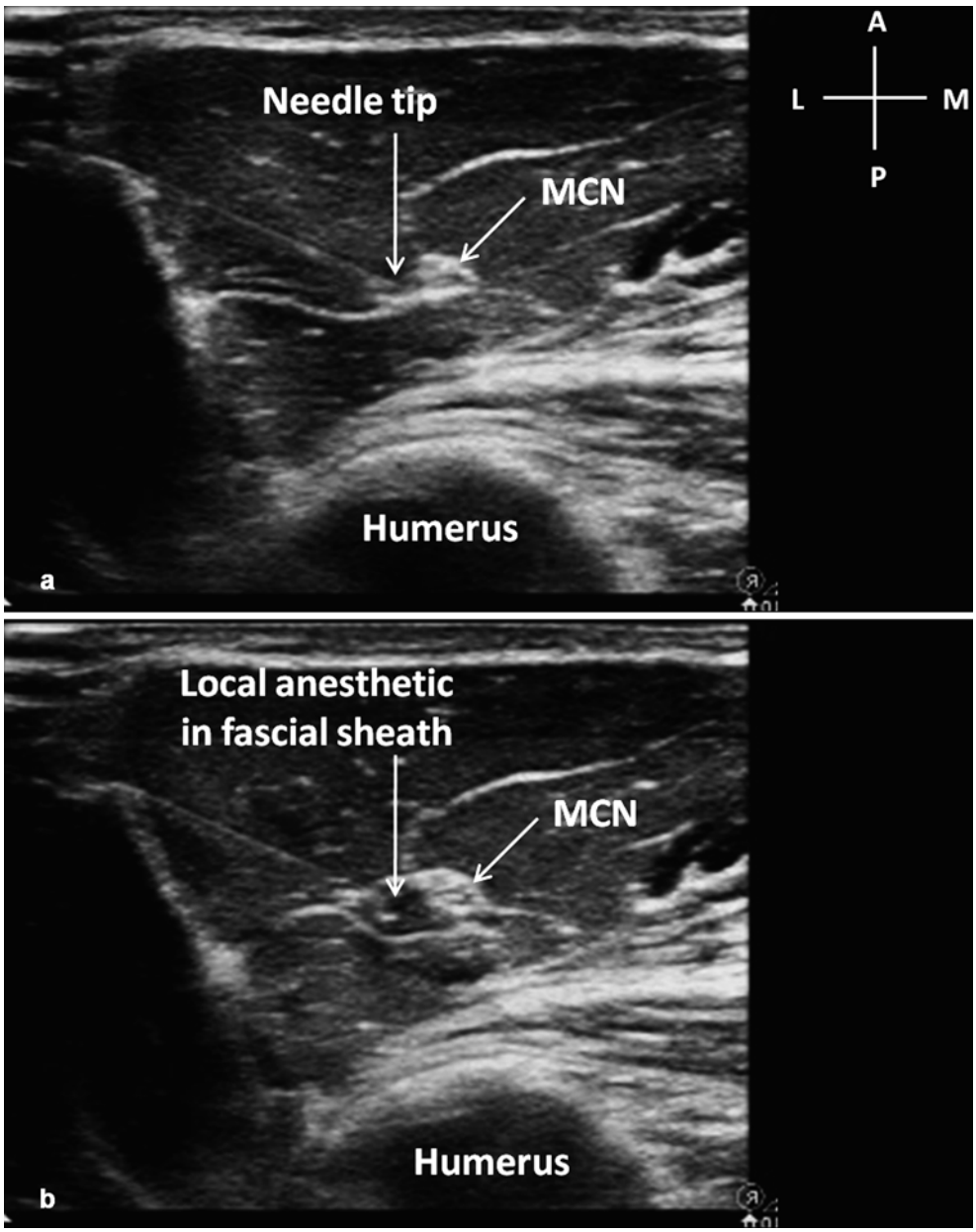


Fig. 32.14 (**a**) The musculocutaneous nerve (*MCN*) is approached at a tangent, the aim being to enter the enveloping fascia without piercing the nerve. (**b**) Injection with the needle tip within the fascial sheath will surround the nerve with local anesthetic

Ultrasound-Guided Continuous Nerve Block Technique

- This is not a commonly used site for long-term catheter placement as it is difficult to maintain adequate hygiene of the insertion site.
- The nerves are spread out around the artery which can limit the effectiveness of continuous local anesthetic infusion without intermittent boluses.
- Patient positioning and pre-scanning are performed in a similar manner to the single-shot technique.
- If an in-plane approach is selected, the introducer needle should be advanced from the lateral side of the probe, aiming to place the tip immediately posterior to the axillary artery. Five to ten milliliters of fluid should be injected here to produce a perivascular pocket of fluid into which the catheter should be advanced, usually not more than 3 cm beyond the tip of the needle.
- If an out-of-plane approach is selected, the introducer needle is advanced at a 45–60° angle to the skin in a proximal direction, aiming to reach the posteromedial aspect of the artery, i.e., placing the needle tip between the artery and radial nerve. Injection of 0.5–1-ml boluses of fluid should be used to confirm needle tip position during advancement as well as to hydrodissect a safe path and avoid needle–nerve trauma. Five to ten milliliters of fluid is injected in the final needle position to create a fluid pocket; the catheter is advanced 3–5 cm beyond the needle tip to travel in a proximal direction parallel to the plexus.

Local Anesthetic Dosages

Single-Shot Block

- A volume of 40–50 ml (0.5–0.6 ml/kg) is generally recommended for the landmark-guided techniques of axillary nerve blockade, but lower volumes of 25–40 ml can be used in the ultrasound-guided approach as long as adequate local anesthetic spread is observed [12].
- The choice of local anesthetic and concentration depends on the desired speed of onset and duration. Commonly used local anesthetics include:
  - A 1:1 mixture of an intermediate-acting local anesthetic (e.g., 2 % mepivacaine or lidocaine) and a long-acting local anesthetic (e.g., 0.5 % bupivacaine), which will have a faster onset than bupivacaine alone and a longer duration than lidocaine alone.
  - 0.25–0.5 % bupivacaine.
  - 0.5–0.75 % ropivacaine.
- Epinephrine in a concentration of 25–50 mcg/ml should always be added when performing an axillary block as a marker of intravascular injection and to reduce systemic vascular absorption.

Continuous Axillary Brachial Plexus Block

- 0.2–0.4 % ropivacaine at 4–8 ml/h.
- 0.125–0.25 % bupivacaine at 4–8 ml/h.
- PCA boluses of 2–5 ml may be added if this function is available on the infusion pump.
- Both infusion rates and bolus volumes should be titrated to achieve the optimal balance between adequate analgesia and an excessively dense sensory and motor block.

Complications and Adverse Effects

- The axillary plexus block is a safe technique compared to other brachial plexus blocks as there are no vital structures in the vicinity.
- The main complications relate (1) to inadvertent intravascular injection and local anesthetic systemic toxicity and (2) nerve injury.
- The risk of local anesthetic systemic toxicity can be minimized by taking appropriate precautions, including (1) the addition of epinephrine to local anesthetic and (2) fractionated injections with intermittent *gentle* negative aspiration. If ultrasound guidance is being used, fluid spread must always be observed with injection. If this is not seen, the needle tip must be assumed to be intravascular until proven otherwise, and repositioning must be undertaken before further injection.
- The risk of needle–nerve injury can be minimized by avoiding paresthesia and respecting patient's complaints of pain or discomfort on injection during a surface landmark-guided technique. If ultrasound guidance is used, individual nerves should be clearly visualized and identified, and needle–nerve contact kept to a minimum.

Axillary block of the brachial plexus

"Single–shot"–technique ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No

☐ Yes (which?) _____

Purpose of block: ☐ *Surgical* ☐ *Diagnostic* ☐ *Therapeutic*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs)*: ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Abducted upper arm (90-100°)*

Needle type: ☐ *Plexufix®* 24 G (45°) ☐ *25 mm* ☐ *50 mm*

☐ *Stimuplex® D* ____*mm*

☐ *Other* _____

☐ Ultrasound - guided

☐ *Transducer* ☐ *Linear* ☐ *Curved*

☐ *Approach* ☐ *In plane* ☐ *Out of plane*

Puncture technique: ☐ *Perivascular* ☐ *Paresthesias*

☐ *Transarterial* ☐ *Electrostimulation*

Local anesthetic: _____ *mL* _____ % _____

(in incremental doses)

Addition to injection solution: ☐ *No* ☐ *Yes* _____ *µg/mg*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth*

Pain triggered (intraneural location?) _____

Nerve region _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement:* *right* ___ °C *left* ___ °C

☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge _____

Complications: ☐ *None* ☐ *Signs of intoxication*

☐ *Hematoma* ☐ *Neurological injuries (median nerve, ulnar nerve, radial nerve)*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

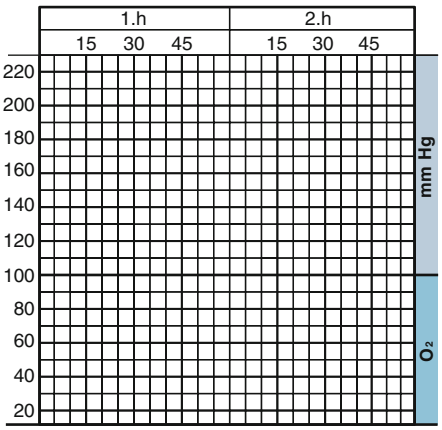
0 10 20 30 40 50 60 70 80 90 100

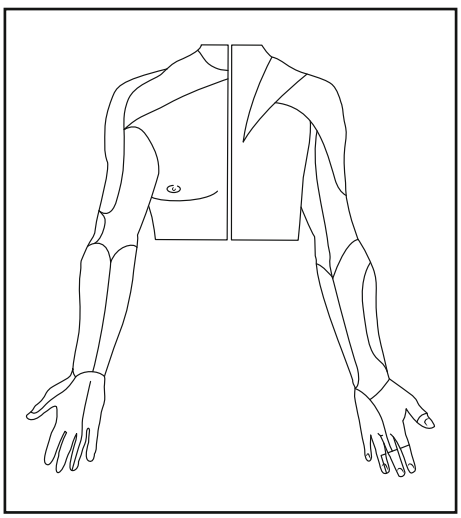
Special notes:

_____

_____

Record and checklist





With permission from Danilo Jankovic

Axillary block of the brachial plexus

Continuous technique ☐ Right ☐ Left

Purpose of block: ☐ *Surgical* ☐ *Therapeutic*
i. v. access: ☐ *Yes*
Monitoring: ☐ *ECG* ☐ *Pulse oximetry*
Ventilation facilities: ☐ *Yes (equipment checked)*
Emergency equipment *(drugs)*: ☐ *Checked*
Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Abducted upper arm (90-100°)*
Puncture technique: ☐ *Electrostimulation*
☐ Ultrasound - guided
☐ *Transducer* ☐ *Linear* ☐ *Curved*
☐ *Approach* ☐ *In plane* ☐ *Out of plane*
Needle type ☐ *Contiplex®* D ___ *mm* ___ *G* ☐ *Tuohy* ___ *mm* ___ *G*
☐ *Other* _____
Catheter: ☐ *Advanced* _____ *cm*
Aspiration test: ☐ *Carried out*
Bacterial filter: ☐
Test dose: _____ *mL* _____ *%* _____
Bolus administration: _____ *mL* _____ *%* _____
(in incremental doses)
Addition to injection solution: ☐ *No* ☐ *Yes* _____ *µg / mg*
Patient's remarks during injection:
☐ *None* ☐ *Paresthesias* ☐ *Warmth*
Pain triggered (intraneural location?) _____
Nerve region _____

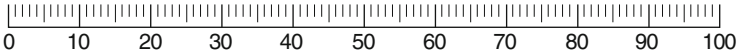
Objective block effect after 15 min:
☐ *Cold test* ☐ *Temperature measurement: right* ___ *°C left* ___ *°C*
☐ *Sensory* ☐ *Motor*
☐ *Continuous monitoring*

☐ Infusion for postoperative analgesia
Local anesthetic: _____ *%* _____ *ml/h*
Addition to LA: _____ *mg* _____ *µg* _____
Patient-controlled anesthesia (PCA)
☐ Local anesthetic _____ *%*
☐ Addition: _____
☐ Baseline rate _____ *m/Lh* _____
☐ Bolus administration: _____ *mL*
☐ Lockout interval _____ *min*

Complications: ☐ *None* ☐ *Signs of intoxication*
☐ *Hematoma* ☐ *Neurological injuries (median nerve, ulnar nerve, radial nerve)*

Subjective effects of the block: *Duration:* _____
☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

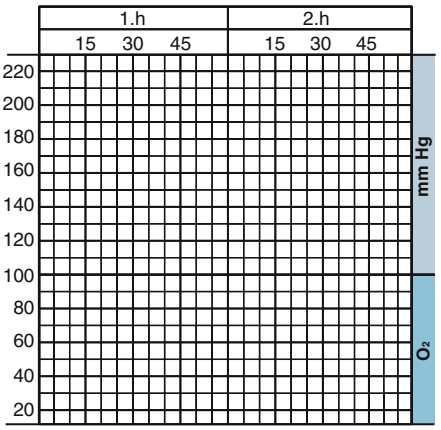


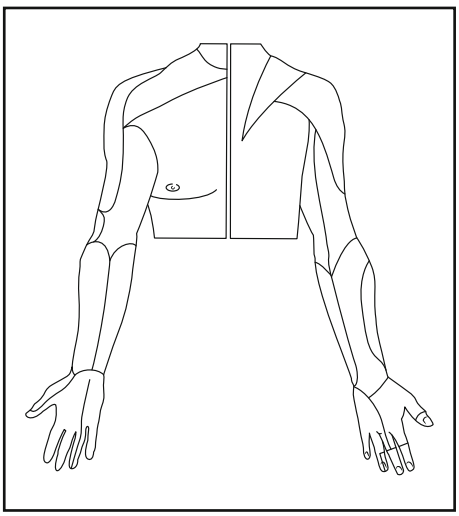
Special notes:

Record and checklist

Name: _____
Date: _____
Diagnosis: _____
Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No
☐ Yes (which?) _____





With permission from Danilo Jankovic

References

1. Tran DQH, Russo G, Muñoz 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.
2. Chin KJ, Alakkad H, Cubillos JE. Single, double or multiple-injection techniques for non-ultrasound guided axillary brachial plexus block in adults undergoing surgery of the lower arm. Cochrane Database Syst Rev. 2013;(8):CD003842.
3. Berthier F, Lepage D, Henry Y, Vuillier F, Christophe J-L, Boillot A, Samain E, Tatu L. Anatomical basis for ultrasound-guided regional anaesthesia at the junction of the axilla and the upper arm. Surg Radiol Anat: SRA. 2010;32:299–304.
4. Selander D, Edshage S, Wolff T. Paresthesiae or no paresthesiae? Nerve lesions after axillary blocks. Acta Anaesthesiol Scand. 1979;23:27–33.
5. Baranowski AP, Pither CE. A comparison of three methods of axillary brachial plexus anaesthesia. Anaesthesia. 1990;45:362–5.
6. Stan TC, Krantz MA, Solomon DL, Poulos JG, Chaouki K. The incidence of neurovascular complications following axillary brachial plexus block using a transarterial approach. A prospective study of 1,000 consecutive patients. Reg Anesth. 1995;20:486–92.
7. Bloc S, Mercadal L, Dessieux T, Garnier T, Estebe J-P, Le Naoures A, Komly B, Leclerc P, Morel B, Ecoffey C, Dhonneur G. The learning process of the hydrolocalization technique performed during ultrasound-guided regional anesthesia. Acta Anaesthesiol Scand. 2010;54:421–5.
8. Bernucci F, Gonzalez AP, 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.
9. Cho S, Kim YJ, Kim J-H, Baik H-J. Double-injection perivascular ultrasound-guided axillary brachial plexus block according to needle positioning: 12 versus 6 o'clock position of the axillary artery. Korean J Anesthesiol. 2014;66:112–9.
10. 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.
11. 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.
12. Casati A, Danelli G, Baciarello M, Corradi M, Leone S, Di Cianni S, Fanelli G. A prospective, randomized comparison between ultrasound and nerve stimulation guidance for multiple injection axillary brachial plexus block. Anesthesiology. 2007;106:992–6.
13. Lanz E, Theiss D, Jankovic D. The extent of blockade following various techniques of brachial plexus block. Anesth Analg. 1983;62:55–8.

Chapter 33

Intravenous Regional Anesthesia (IVRA)

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323–325, 50354, Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

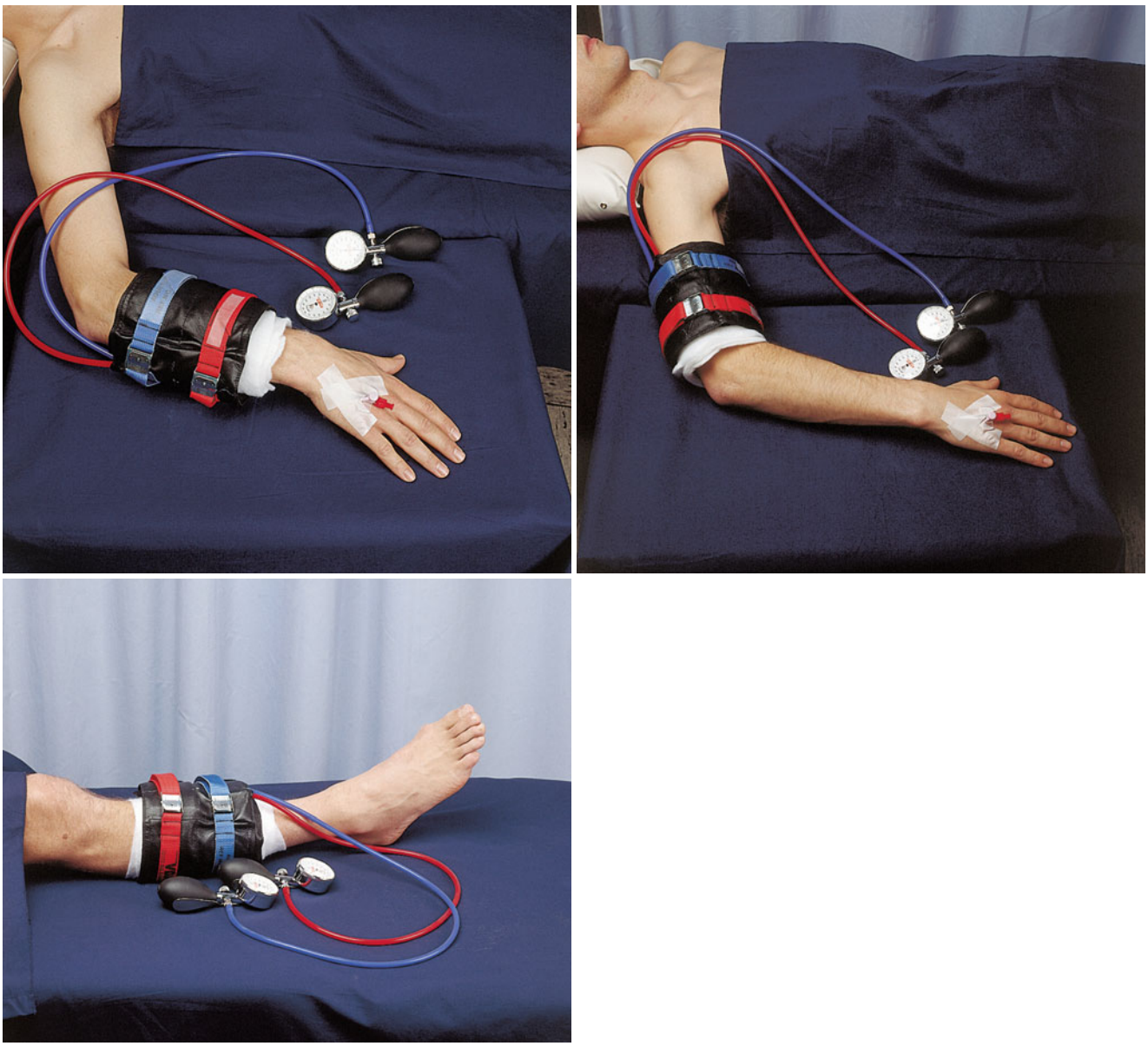
Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_33,

The injection of local anesthetics into a vein in an exsanguinated extremity was first described by August Bier in 1908. Originally, anesthesia was obtained by the intravenous injection of procaine in a previously exsanguinated vascular space, isolated from the rest of the circulation by two Esmarch bandages used as tourniquet. In 1963 Holmes reintroduced the Bier block with the novel use of lidocaine. It caused anesthesia and a motor block.

Indications

Outpatient surgical procedures with a maximum length of 1 h in the forearm or hand (standard application) and in the lower leg and foot (more rarely; Figs. 33.1, 33.2, and 33.3)



Figs. 33.1, 33.2, and 33.3 Areas of application of intravenous regional anesthesia

Specific Contraindications

- Patient refusal
- Local infection in the area to be anesthetized
- Local nerve damage
- Peripheral vascular diseases
- Severe decompensated hypovolemia, shock
- Certain cardiovascular diseases
- Hypertonia, bradycardia, second-degree AV block, any history of a tendency to syncope
- Musculoskeletal diseases

Procedure

This block should only be carried out when full anesthetic facilities are available. Full prior information for the patient is mandatory.

Preparations

Check that the appropriate emergency equipment is present and in working order. Sterile precautions. Two intravenous access points (in the healthy extremity as well as the one being operated on), BP and ECG monitoring, pulse oximetry, anesthesia machine. Patient preparation is the same as for general anesthesia.

Materials (Fig. 33.4)

A 20- and 50-mL syringes, saline, cotton-wool padding, pneumatic tourniquet (double-lumen), Esmarch bandage, local anesthetic, disinfectant, pneumatic tourniquet device (e.g., VBM Medizintechnik Ltd., Sulz am Neckar, Germany; Fig. 33.5).

Fig. 33.4 Materials

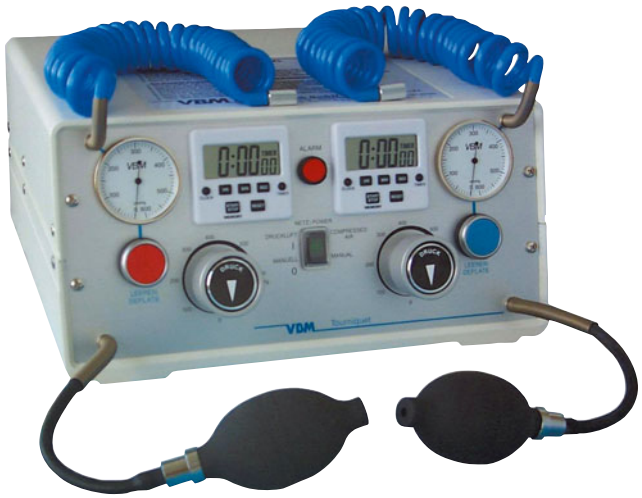


Fig. 33.5 Pneumatic tourniquet device (VBM Medizintechnik Ltd., Sulz am Neckar, Germany)

Patient Positioning

Supine, with the extremity free.

Technical Procedure

1. Insert two intravenous catheters—one in a healthy extremity and the other as distally as possible in the extremity being operated on.
2. Place the extremity being operated on in a free position and put soft padding under the tourniquet to help prevent nerve injury (Fig. 33.6).
3. Position the double-lumen tourniquet.
4. Elevate and massage the limb for a few minutes and then wrap it completely with an Esmarch bandage (Fig. 33.7).
5. Inflate the proximal cuff: the pressure in the cuff has to be ca. 80–100 mmHg higher than the patient's systolic blood pressure. The pressure that should be used depends on the thickness of the muscles being compressed. A pulse oximeter is used to document changes in, and cessation of, the pulse ("pulse occlusion pressure") and the disappearance of the pulse in the radial artery. The "pulse occlusion pressure" can be used to determine the optimal pressure in the proximal cuff (Fig. 33.8).
6. Remove the bandage and place the extremity in a horizontal position.
7. Slowly inject the local anesthetic (20 mL/min; Fig. 33.9).
8. Perform stroking massage of the extremity (this improves the spread of the local anesthetic) and remove the catheters.
9. Good analgesia and muscle relaxation develop after ca. 5–10 min.
10. Inflation of the distal cuff, which is now in the analgesic area, so that the cuff is better tolerated. Deflate the proximal cuff. After the anesthetic effect has been tested, the operation can begin.

- Minimum tourniquet time is 15–20 min after injection of the local anesthetic. The tourniquet must not be released during this period (risk of toxic reactions!).
- Tourniquet pressure must be monitored continuously.
- After completion of the procedure: intermittent deflation over a period of 10 min, with complete inflation in between (Fig. 33.10).

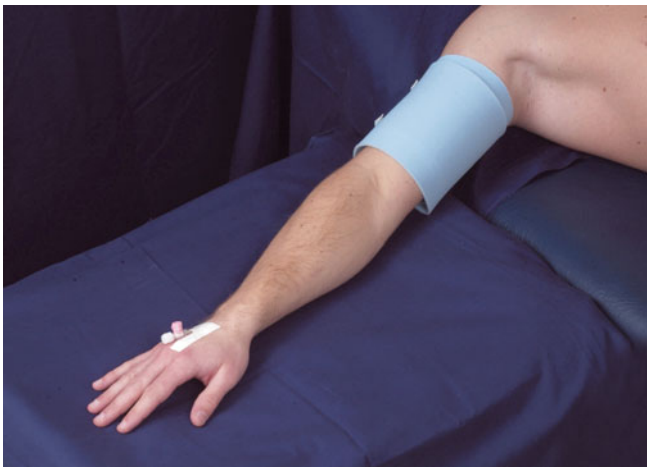


Fig. 33.6 Padding of the tourniquet area, intravenous access

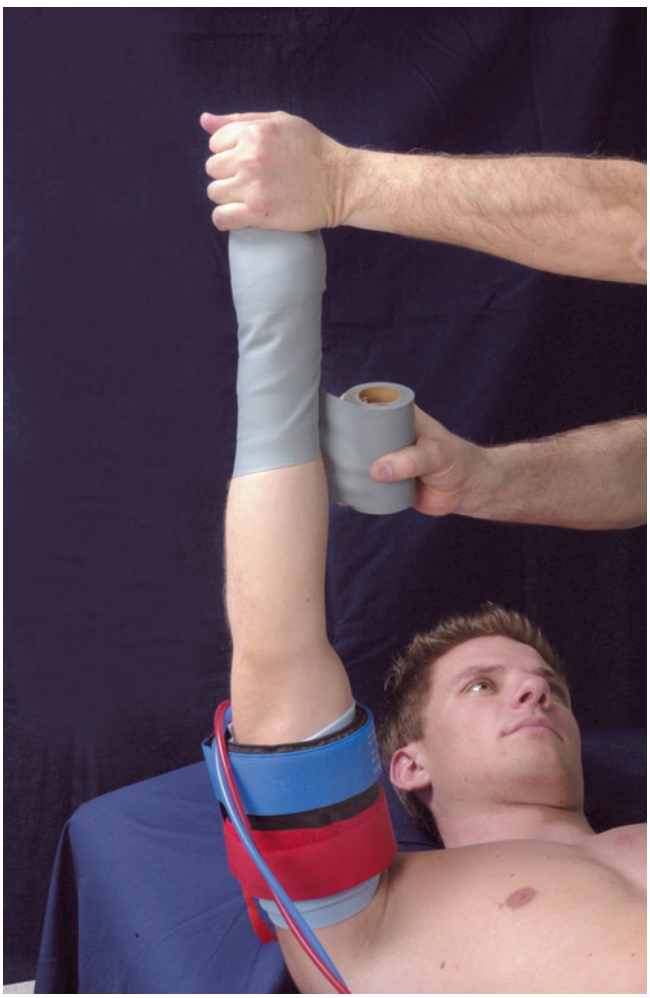


Fig. 33.7 Wrapping with an Esmarch bandage

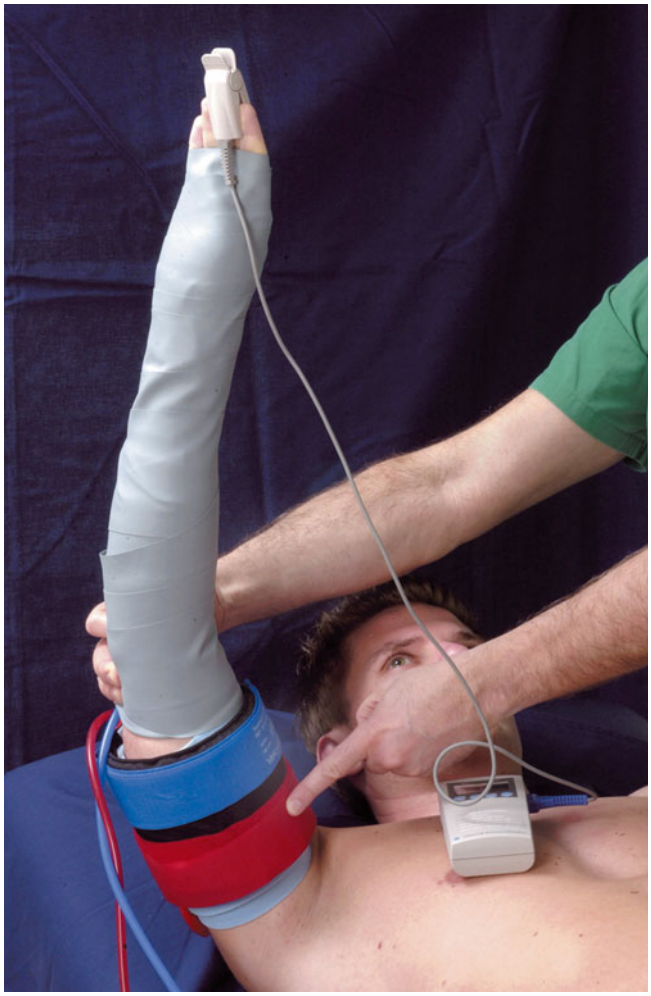


Fig. 33.8 Inflation of the tourniquet

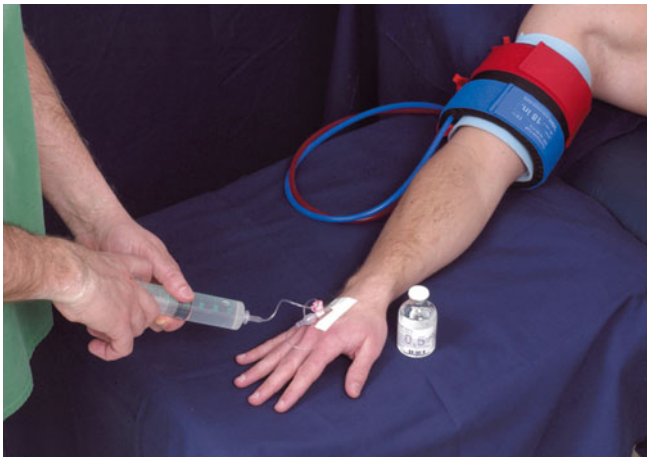


Fig. 33.9 Injection of the local anesthetic

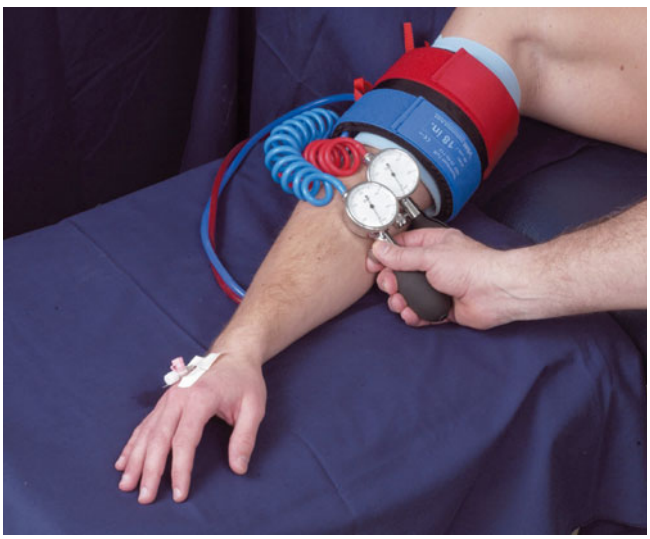


Fig. 33.10 Intermittent deflation of the cuff over a period of 10 min

Dosage

Only local anesthetics that contain no vasoconstrictors may be used!

A 40–50 mL local anesthetic, e.g.:

- Prilocaine 0.5 %, 3–4 (5) mg/kg body weight
- Among the amide local anesthetics, prilocaine provides the best ratio between anesthetic potency and toxicity and should be regarded as the agent of choice for intravenous regional anesthesia (see Chap. 1).
- Mepivacaine 0.5 % or lidocaine 0.5 %, 1.5–3 mg/kg b.w.

*Do not use long-acting, more toxic local anesthetics (e.g. bupivacaine)

Additions to local anesthetic agents

- Clonidine 1 µg/kg or ketamine 0.1 mg/kg b.w.
- Fentanyl (0.1–0.2 mg)
- Morphine (1–6 mg)

Complications

1. Systemic toxic reactions can occur if the local anesthetic enters the circulation due to release of the tourniquet cuff (see Chap. 1). Prophylaxis: intermittent opening of the tourniquet, maintaining verbal contact with the patient, and avoiding strong premedication.

Have intralipid readily available.

2. Toxic effects on the cardiovascular system only occur after very high doses of local anesthetic and become apparent as a drop in blood pressure, bradycardia, circulatory collapse, and cardiac arrest. This type of complication rarely occurs in intravenous regional anesthesia.
3. Nerve damage due to the cuff pressure.

Advantages

1. Simple technique.
2. No specific anatomical expertise is needed.
3. Wide safety margins and very high success rate (>98 %) [7].
4. Fast onset of effect (5–10 min).
5. Good muscle relaxation.
6. Controllable spread of the anesthesia (below the tourniquet cuff).
7. Fast return of sensation.
8. No risk of infection.

Disadvantages

1. Tourniquet cuff is needed.
2. Limited operating time (<1 h).
3. Procedures in the upper arm are not possible.
4. Tourniquet pain during the procedure.
5. Nerve damage due to the tourniquet cuff.
6. Does not provide a blood-free operating area.
7. Insufficient postoperative analgesia due to fast recovery from the anesthesia.

Intravenous regional anesthesia

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical*

i.v. access: ☐ *No. 1* ☐ *No. 2*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Supine* ☐ *Other*

Location of the tourniquet cuff: ☐ *Right* ☐ *Left*

☐ *Forearm* ☐ *Upper arm* ☐ *Lower leg* ☐ *Thigh*

BP: _____ *mmHg* Pulse _____ *min*

Ischemia: _____ *mmHg* *At* _____

Local anesthetic: _____ *mL* _____ *%* _____

☐ Addition to LA: _____ *mL* _____ µg

Ischemia: *From* _____ *To* _____

Cuff release over: ☐ *5 min* ☐ *10 min intermittently*

Patient's remarks during injection: ☐ *None*

☐ *Pain* ☐ *Paresthesias* ☐ *Warmth* ☐ *Cold*

Objective block effect after: ☐ *5 min* ☐ *10 min*

☐ *Temperature measurement right* __°C *left* ____°C

☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge _____

Complications and side effects: ☐ *None*

☐ *BP reduction* ☐ *Bradycardia* ☐ *Cardiac arrhythmia*

☐ *Fatigue* ☐ *Tingling lips* ☐ *Tongue paresthesia*

☐ *Perioral numbness* ☐ *Metallic taste* ☐ *Anxiety*

☐ *Restlessness* ☐ *Trembling* ☐ *Other*

Special notes:

_____

_____

_____

_____

_____

_____

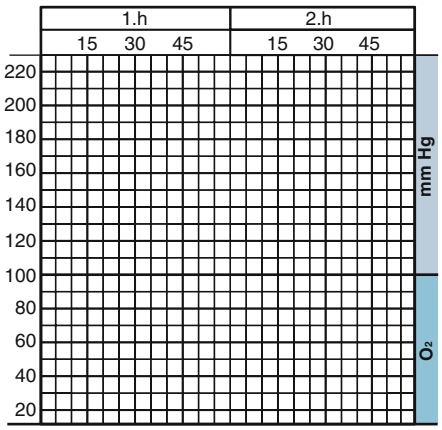
_____

_____

_____

_____

Record and checklist



With permission from Danilo Jankovic

Suggested Reading

1. Armstrong P, Power I, Wildsmith JA. Addition of fentanyl to prilocaine for+intravenous regional anesthesia. Anaesthesia. 1991;46:278–80.
2. de May JC. Bier's block. Anesthesia. 1997;52:713.
3. Erciyes N, Akturk G, Solak M. Morphine/prilocaine combination for intravenous regional anesthesia. Acta Anaesth Scand. 1995;39:845–6.
4. Gorgias N, Maidatsi P, Kyriakidis AM, et al. Clonidine versus Ketamine to prevent tourniquet pain during intravenous anesthesia with lidocaine. Reg Anesth Pain Med. 2001;26(6):512–7.
5. Hannington-Kiff J. Intravenous regional sympathetic block with guanethidine. Lancet. 1974;I:1010–20.
6. Hannington-Kiff J. Antisympathetic drugs in limbs. In: Wall PD, Melzack R, editors. Textbook of pain. London: Churchill Livingstone; 1984.
7. Hadzic A, Vloka JD, Kuroda MM, Koorn R, Birnbach DJ. The practice of peripheral nerve blocks in the United States: a national survey. Reg Anesth Pain Med. 1998;23:241–6.
8. Hilgenhurst G. The Bier block after 80 years: a historical review. Reg Anesth. 1990;15:2.
9. Holmes C. Intravenous regional nerve blockade. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade. 3rd ed. Philadelphia/New York: Lippincott-Raven; 1998. p. 395–409.
10. Jankovic D. Intravenous regional anesthesia. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Oxford/Berlin: Blackwell Scientists; 2004. p. 164–8.
11. Pitkänen MT. Intravenous regional anesthesia. In: Rosenberg P, editor. Local and regional anesthesia. London: BMJ Books; 2000. p. 55–6.
12. Simgen WLA. Intravenöse Regionalanästhesie. In: Hörster W, Zenz M, Niesel HC, Kreuscher H, editors. Regionalanästhesie. Stuttgart/New York: Gustav Fischer; 1989. p. 82–5.
13. Wahren KL, Gordh T, Torebjörk E. Effects of regional intravenous guanethidine in patients with neuralgia in the hand, a follow up study over a decade. Pain. 1995;62:379–85.

Part VI
Elbow and Wrist

Chapter 34

Peripheral Nerve Blocks in the Elbow Region

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354, Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, University of Toronto, McL 2-405 Toronto Western Hospital,
399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_34,

Anatomy and Sonoanatomy
(Figs. 34.1, 34.2, 34.3, and 34.9)

Ulnar Nerve

The ulnar nerve originates from the medial cord of the brachial plexus (C8–T1, occasionally C7). Having pierced the medial fascial septum halfway down the upper arm to join the posterior fascia compartment, the ulnar nerve runs anterior to the medial head of triceps (Fig. 34.4). It then descends into the cubital tunnel bound by the medial epicondyle, the Osborne reticulum between the two heads of the flexor carpi ulnaris, the medial collateral ligament, and the olecranon. The nerve is easily palpated at this location. In the forearm, it runs between the humeral and ulnar head of the flexor carpi ulnaris muscle on the medial side of the forearm, sandwiched between the flexor digitorum profundus and superficialis (Fig. 34.5a, b).

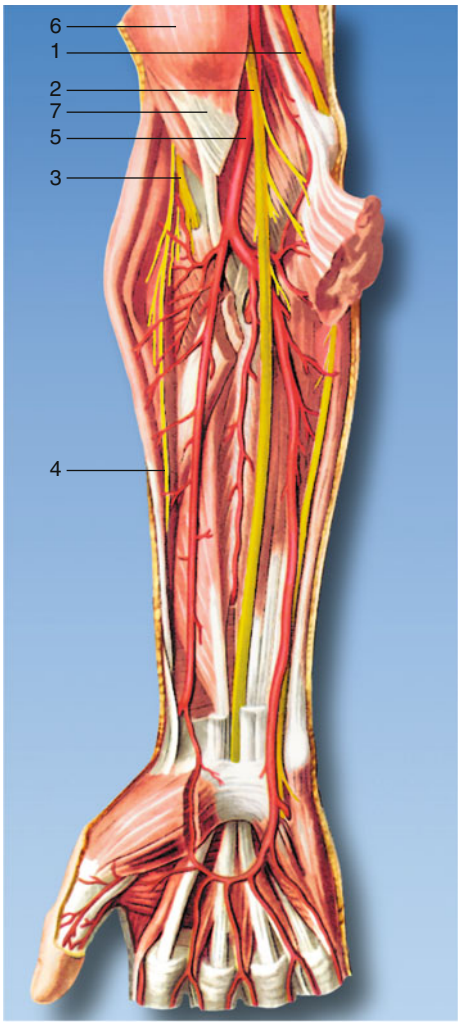


Fig. 34.1 Anatomy. (*1*) Ulnar nerve, (*2*) median nerve, (*3*) deep branch of the radial nerve (anterior interosseous nerve), (*4*) superficial branch of the radial nerve, (*5*) brachial artery, (*6*) biceps brachii muscle, (*7*) bicipital aponeurosis (Reproduced with permission from Danilo Jankovic)

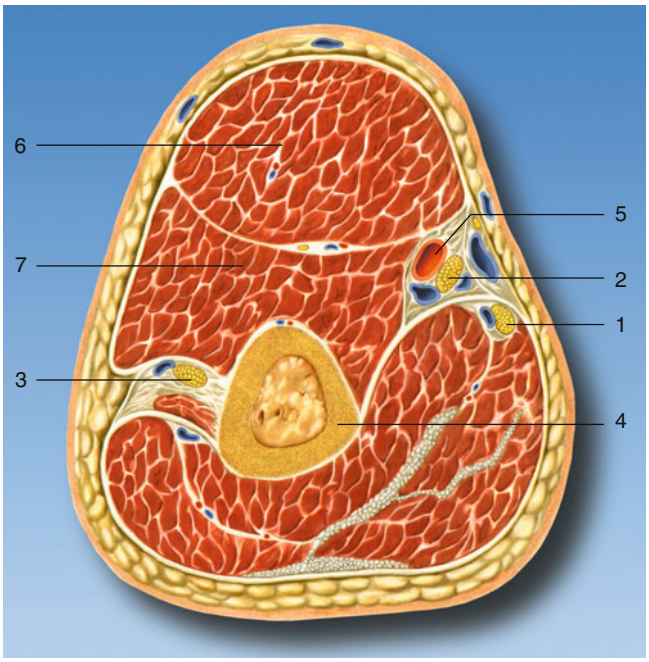


Fig. 34.2 Distal third of the humerus. (*1*) Ulnar nerve, (*2*) median nerve, (*3*) radial nerve, (*4*) humerus, (*5*) brachial artery, (*6*) biceps brachii muscle, (*7*) brachialis muscle (Reproduced with permission from Danilo Jankovic)

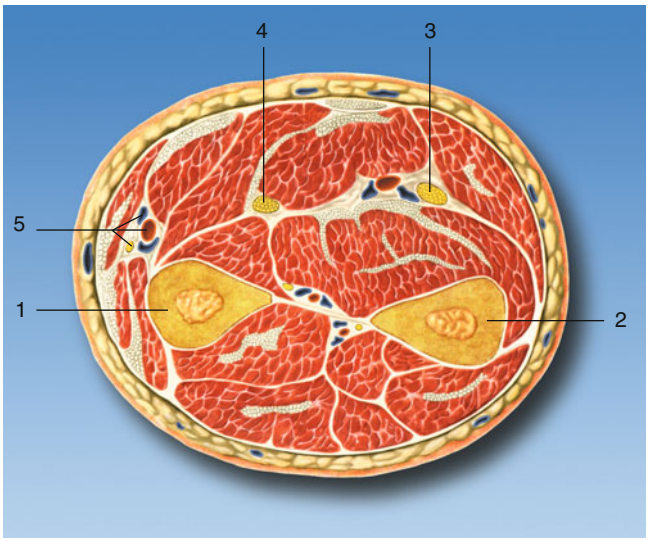


Fig. 34.3 Mid-forearm. (*1*) Radius, (*2*) ulna, (*3*) ulnar nerve, (*4*) median nerve, (*5*) radial artery, radial vein, and radial nerve (Reproduced with permission from Danilo Jankovic)

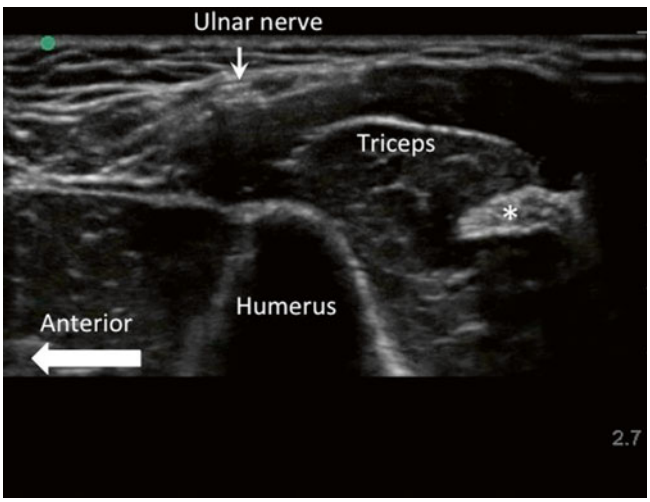


Fig. 34.4 Sonogram of the ulnar nerve proximal to the cubital tunnel. The ultrasound probe is placed on the medial aspect of the arm. * Indicates the tendon of the medial head of triceps (Reproduced with permission from Philip Peng Educational Series)

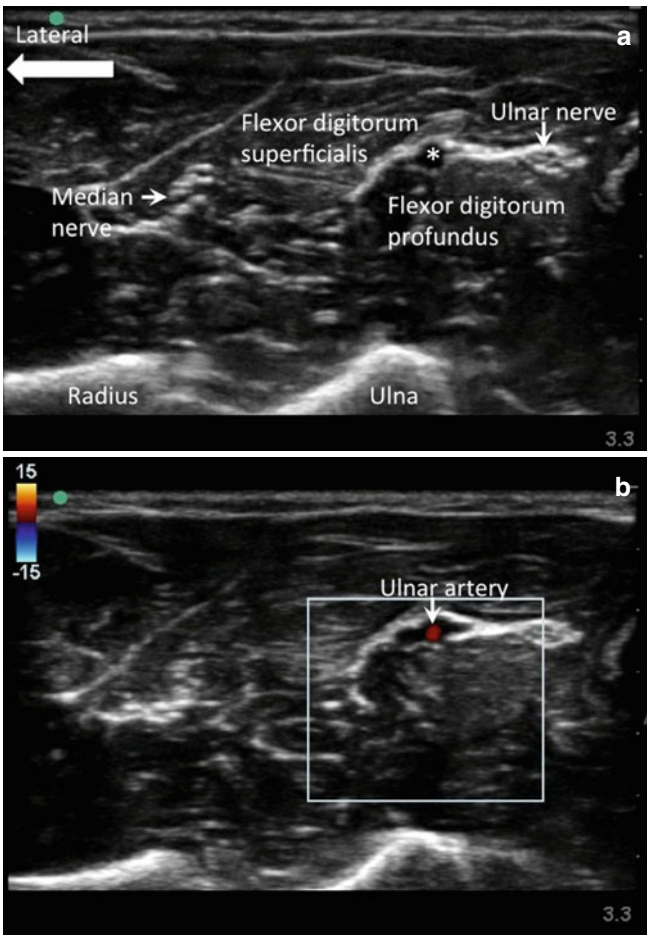


Fig. 34.5 (**a**) Sonogram of ulnar nerve distal to the cubital tunnel. The ultrasound probe is placed on the ventromedial aspect of the forearm three fingerbreadths distal to the elbow. Please note that the ulnar artery (*) moves from medial nerve toward the ulnar nerve as the neurovascular bundles travel distally in the forearm. At this level, this is ideal for injection as the ulnar nerve is sandwiched between the flexor digitorum superficialis and profundus muscles and the ulnar artery is further from the ulnar nerve. (**b**) Doppler scan to show the ulnar artery (Reproduced with permission from Philip Peng Educational Series)

Median Nerve

The median nerve originates from the medial and lateral cords of the brachial plexus (C5–T1). At the elbow, it lies medial to the brachial artery, which in turn lies on the medial side of the biceps brachii tendon on the medial surface of the brachialis muscle (Figs. 34.6a, b and 34.9). Passing the elbow, the median nerve goes between the two heads of the pronator teres where it gives the anterior interosseous nerve. The median nerve then continues between the flexor digitorum profundus and superficialis. The anterior interosseous nerve descends to the anterior surface of interosseous membrane.

Radial Nerve

The radial nerve (C5–T1) is the longest branch of the brachial plexus and represents a direct continuation of the posterior cord. It runs in the middle of the upper arm in the groove of the radial nerve along the dorsal side of the humerus. At the level of lateral epicondyle of the humerus and the elbow joint capsule, it runs between the brachioradialis muscle and the brachialis muscle (Figs. 34.7 and 34.9), dividing into the deep branch (posterior interosseous nerve, motor supply to most of the extensor muscles) and the superficial branch (sensory). The former descends between the two layers of the supinator muscle while the latter follows the course of the radial artery up to the distal forearm.

Lateral Cutaneous Nerve of the Forearm

The musculocutaneous nerve (C5–C7) arises from the lateral cord of the brachial plexus. At the level of middle and distal forearm, it runs between the biceps muscle and the brachialis muscle to the brachial fascia, which it penetrates, becoming the lateral antebrachial cutaneous nerve (Figs. 34.8 and 34.9).

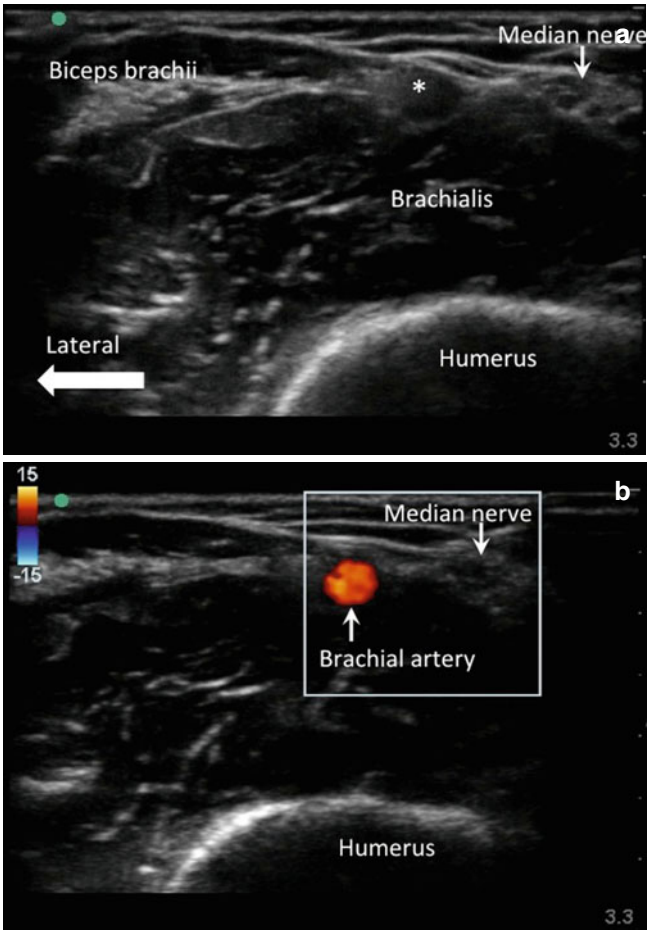


Fig. 34.6 (**a**) Sonogram of the median nerve at the elbow level. The median nerve is seen medial to the biceps brachii tendons and lies on the surface of the brachialis muscle. * Indicates brachial artery. (**b**) Doppler scan of the brachial artery (Reproduced with permission from Philip Peng Educational Series)

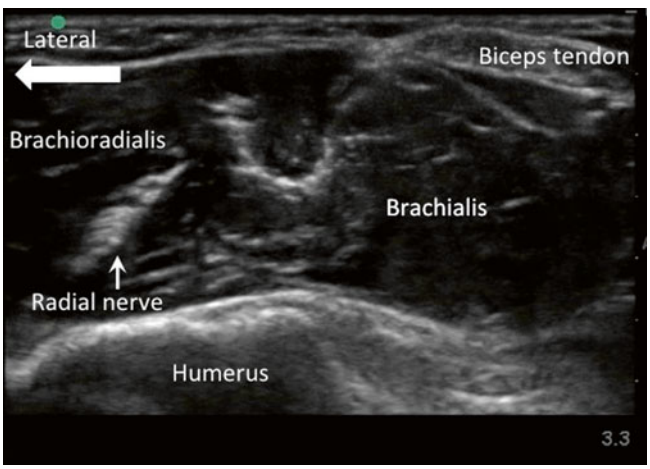


Fig. 34.7 Sonogram of the radial nerve. At the level of lateral epicondyle, the radial nerve is consistently located between the brachioradialis and brachialis muscles (Reproduced with permission from Philip Peng Educational Series)

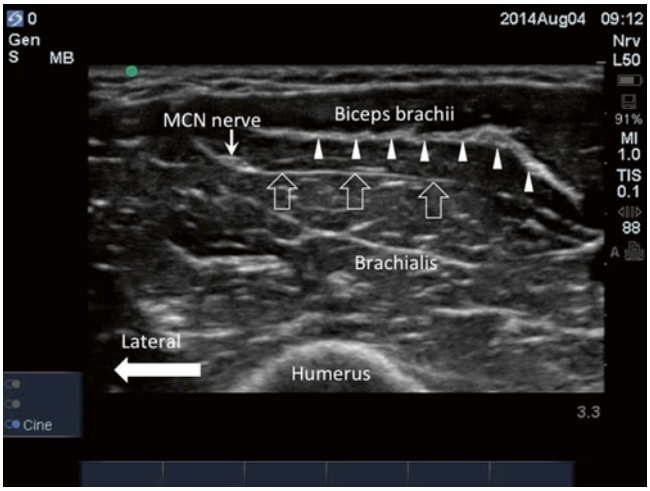


Fig. 34.8 Sonogram of the cutaneous branch of musculoskeletal nerve. At distal one third of the arm, the nerve is consistently in the fascia plane between the biceps brachii and brachialis muscles (*void arrows*). The fascia plane between these two muscles can be difficult to be visualized because of anisotropy, and tilting the ultrasound until the plane is clearly seen is crucial. Some practitioners may mistake the tendon (*arrowheads*) of the biceps brachii as the fascia plane (Reproduced with permission from Philip Peng Educational Series)

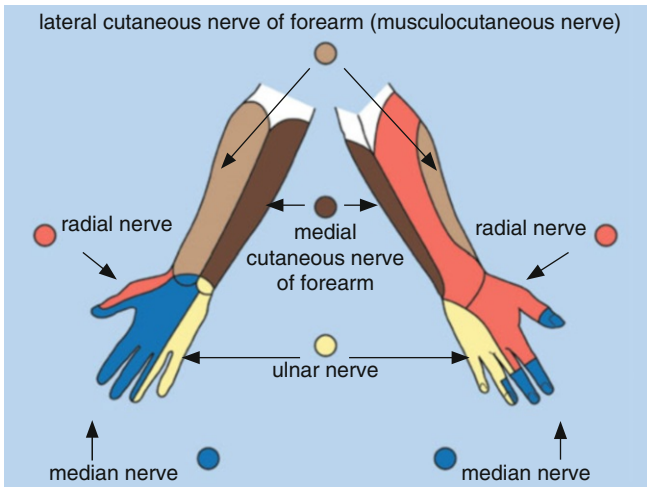


Fig. 34.9 Skin innervations (With permission from Danilo Jankovic)

Indications

1. Minor interventions in the innervated area
2. Supplementation of incomplete anesthesia of the brachial plexus

Procedure

*S*ince all the nerves are superficial nerve, high-frequency (6–15 MHz) linear ultrasound probe is used. During injection, the practitioner should pay attention to the spread of the injectate and the size of the nerve. Absence of spread suggests either intravascular injection or needle tip is not visualized. Expansion of the nerve suggests intraneural injection.

Ulnar Nerve

To avoid injecting around the ulnar nerve at the cubital tunnel, the target can be either above or below the cubital tunnel. To target the ulnar nerve above the cubital tunnel, the patient is in supine position with elbow flexed and arm rotated outward. The ultrasound probe is place just proximal to the medial epicondyle. The ulnar nerve appears in the subcutaneous plane (Fig. 34.4). The needle is inserted out-of-plane because of the superficial position of the nerve. Alternatively, the patient is put in supine position with the arm rotated outward in extension position. The target is the ulnar nerve 3–4 fingerbreadths distal to the elbow joint. At this level, the nerve is medial to the flexor carpi ulnaris and between the flexor digitorum profundus and superficialis (Fig. 34.5). The ultrasound probe is placed on the ventromedial aspect of the forearm and the needle is inserted in-plane. If the ultrasound prone is placed too distally, the ulnar artery will join the ulnar nerve and the risk of artery puncture is higher. A total of 5 mL of local anesthetic is sufficient.

Median Nerve

The patient is in supine position and the arm is in extension position. The injection site is at the elbow joint where the nerve can be visualized medial to the brachial artery (Fig. 34.6). Blocking the nerve distal to the elbow joint may miss the anterior interosseous nerve. The ultrasound probe is placed just proximal to the elbow. The needle is inserted in-plane from medial to lateral or out-of-plane. A total volume of 5 mL of local anesthetic is sufficient.

Radial Nerve

The target is at the lateral epicondyle where the needle is between the brachioradialis and brachialis muscle. The patient is in supine position with the arm extended. Ultrasound probe is placed over the lateral aspect of the arm over the lateral epicondyle (Fig. 34.7). A needle is inserted in-plane from medial to lateral direction. A total volume of 5 mL of local anesthetic is sufficient.

Lateral Cutaneous Nerve of the Forearm

At distal third of the arm, the lateral cutaneous nerve is small but can be seen between the brachialis and biceps brachii muscles. Attention should be paid to the anisotropic nature of the nerve and the fascia plane between the brachialis and biceps brachii muscles (Fig. 34.8). A total volume of 3 mL of local anesthetic is sufficient.

Suggested Reading

1. Bianchi S, Martinoli C. Elbow. In: Bianchi S, Martinoli C, editors. Ultrasound of the musculoskeletal system. Berlin/Heidelberg: Springer; 2007.
2. Bridenbaugh DL. The upper extremity: somatic blockade. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988.
3. Brown D. Distal upper extremity blocks. In: Atlas of regional anesthesia. 3rd ed. Philadelphia: Saunders & Elsevier; 2006.
4. Chan VWS. Peripheral nerve block. In: Chan VWS, editor. Ultrasound imaging for regional anesthesia: a practical guide booklet, Upper limb, vol. 2. Toronto Center for Ultrasound Education. 1st ed. iBook, California CA: Apple Inc.; 2013. p. 116–36.
5. Covic D. Blockaden peripherer Nerven im Ellenbogenbereich. Blockade peripherer Nerven im Handwurzelbereich. In: Hörster W, Kreuscher H, Niesel H, et al., editors. Regionalanästhesie. Stuttgart: Fischer Verlag; 1989. p. 86–101.
6. Hahn MB, McQuillan PM, Sheplock GJ. Regional anesthesia. St. Louis: Mosby; 1996.
7. Löfström B. Blockade der peripheren Nerven des Armes in der Handwurzelgegend. In: Eriksonn E, editor. Atlas der Lokalanästhesie (2. Auflage). Berlin/New York: Springer; 1980. p. 86–92.
8. Plaikner M, Gruber H, Judmaier W, Brenner E. Upper extremity nerves. In: Peer S, Gruber H, editors. Atlas of peripheral nerve ultrasound with anatomic and MRI correlation. Berlin/Heidelberg: Springer; 2013.

Chapter 35

Peripheral Nerve Blocks in the Wrist Region

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_35, © Springer International Publishing Switzerland 2015

Anatomy

Ulnar Nerve (Fig. 35.1)

In the medial distal third of the forearm, about three fingerbreadths proximal to the wrist, the ulnar nerve divides into a dorsal branch (sensory to the dorsal surface of the hand and fingers), a small palmar cutaneous branch to the hypothenar eminence, and a mixed branch—the palmar branch. The latter runs superficially between the tendons of the flexor carpi ulnaris and flexor digitorum superficialis muscle. The ulnar artery lies directly radially, alongside the nerve.

Median Nerve (Fig. 35.1)

The median nerve lies between the tendons of the palmaris longus muscle and the flexor carpi radialis muscle. It runs in the direction of the long axis of the radius. Three fingerbreadths proximal to the wrist, the medial nerve gives rise to a palmar cutaneous branch to the thenar eminence.

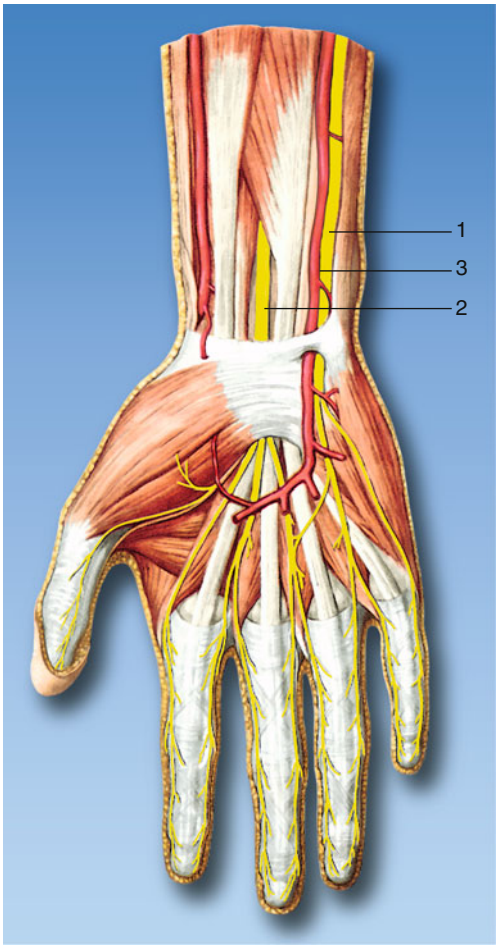


Fig. 35.1 Anatomy. (*1*) Ulnar nerve, (*2*) median nerve, (*3*) ulnar artery (Reproduced with permission from Danilo Jankovic)

Radial Nerve (Fig. 35.2)

The superficial branch of the radial nerve runs—together with the radial artery, initially—in the forearm along the medial side of the brachioradialis muscle in the direction of the wrist. About 7–8 cm proximal to the wrist, it crosses under the tendon of the brachioradialis muscle and reaches the extensor side of the forearm. At the level of the wrist, the radial nerve divides into several peripheral branches.

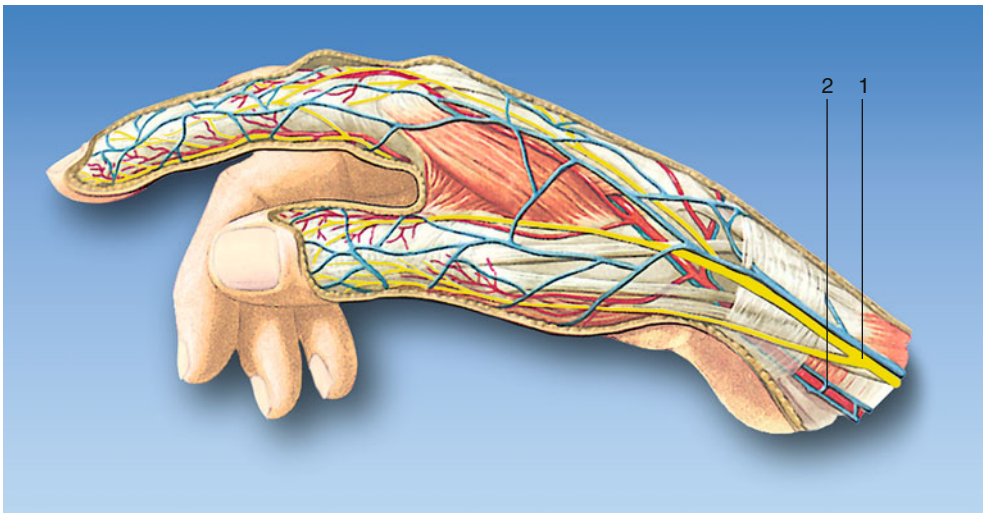


Fig. 35.2 Anatomy. (*1*) Radial nerve (superficial branch), (*2*) radial artery (Reproduced with permission from Danilo Jankovic)

Indications

Surgical

1. Minor surgical interventions in the innervated area
2. Supplementation of an incomplete block of the brachial plexus

Diagnostic

Differential diagnosis of painful conditions in the hand

Therapeutic

Carpal tunnel syndrome

Ultrasound-Guided Technique

Ulnar Nerve (Fig. 35.3)

The patient is in supine position with the forearm supinated. The target is just proximal to the proximal palmar crease. As the injection at the wrist level misses both the palmar cutaneous branch to hypothenar eminence and the dorsal cutaneous branch, this injection will require supplementation of the two cutaneous branches with subcutaneous infiltration. The authors prefer the injection at the elbow level.

For the injection at the wrist level, an ultrasound probe is placed on the ventromedial aspect of the distal forearm. The ulnar nerve is seen on the ulnar side of the ulnar artery. The needle is inserted out-of-plane to the ulnar side of the ulnar artery, which is usually accompanied by one or two veins. Gently releasing the pressure of the probe will reveal the vein, and this maneuver helps to avoid inadvertent venous puncture (Fig. 35.4). A volume of 3 mL of local anesthetic is sufficient. This is followed by subcutaneous infiltration along the ulnar aspect of the wrist (volar and dorsal sides) with 5 mL of local anesthetic.

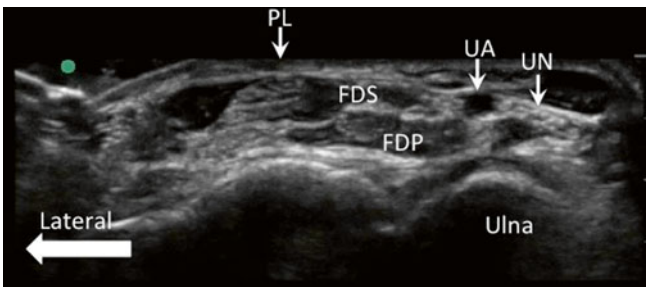


Fig. 35.3 Sonogram of the ulnar nerve just proximal to the carpal tunnel. At this level, the ulnar nerve (*UN*) is on the ulnar side of the ulnar artery (*UA*). *FDS* flexor digitorum superficialis, *FDP* flexor digitorum profundus, *PL* palmaris longus (Reproduced with permission from Philip Peng Educational Series)

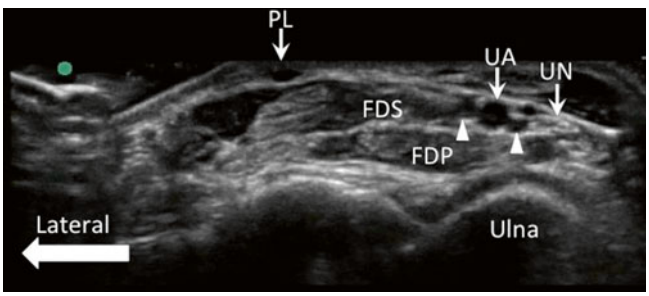


Fig. 35.4 Same scan as Fig. 35.3. Be careful of the veins (*arrowheads*) accompanying the ulnar artery. Always release the pressure to reveal the vein to avoid inadvertent intravascular injection (Reproduced with permission from Philip Peng Educational Series)

Median Nerve (Fig. 35.5)

Positioning is similar to that of the ulnar nerve.

The target at the wrist is just proximal to the proximal palmar crease. At this level, the palmar cutaneous branch of the thenar eminence has already left the median nerve. Thus, for surgical supplementation, a subcutaneous infiltration along the radial volar aspect of the forearm is required. The ultrasound probe is placed just proximal to the proximal palmar crease. The median nerve is located along with various tendons which resemble the median nerve in ultrasonography. There are two methods to identify the median nerve at this level. One is to flex the thumb to help identify the flexor pollicis longus (FPL). The median nerve is always on the 2 o'clock position of the left hand and 10 o'clock position of the right hand (Fig. 35.5). The other is the tilting the ultrasound probe. The tendons are much more anisotropic than the median nerve (Fig. 35.6). By this maneuver, the median nerve will stand out. The needle is usually inserted out-of-plane on the side of the nerve. A volume of 3–5 mL of local anesthetic is sufficient.

Radial Nerve: Superficial Branch

The forearm is in mid-prone position. The superficial branch of radial nerve is a small nerve that may already divide into a few branches and is not easily found at this level. The nerve is usually on the dorsal side of the radial artery in the "snuff" box (Figs. 35.7 and 35.8). If the nerve cannot be seen, the local anesthetic can be deposited dorsal to the radial artery with out-of-plane technique. A volume of 3 mL is sufficient.

Alternatively, the superficial branch of the radial nerve can be visualized in the mid-forearm as it crosses under the tendon of the brachioradialis muscle and tendon and reaches the extensor side of the forearm (Figs. 35.9 and 35.10). The needle is inserted with out-of-plane technique. A volume of 3 mL is sufficient.

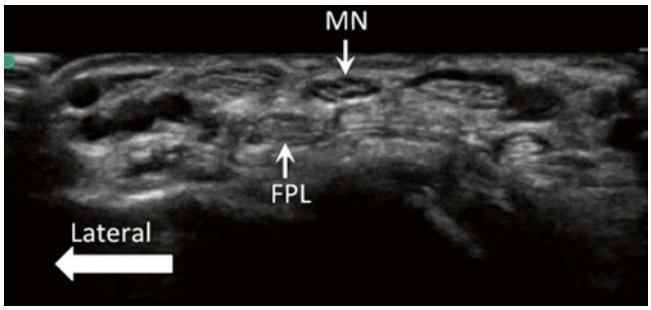


Fig. 35.5 Sonogram of the median nerve (*MN*) at the proximal palmar crease. At this level, the median nerve is usually superficial and on the ulnar side of the flexor pollicis longus (*FPL*) (Reproduced with permission from Philip Peng Educational Series)

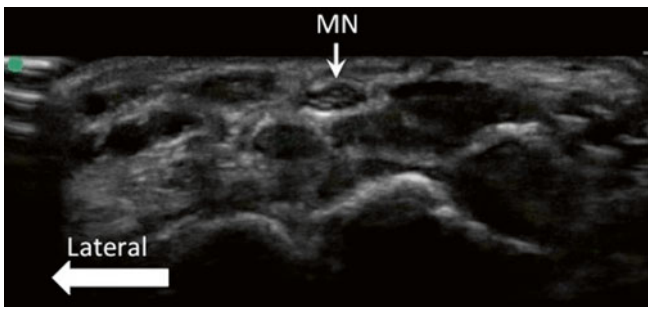


Fig. 35.6 Same scan as Fig. 35.5. The ultrasound probe is tilted. With this maneuver, the tendons "disappear" while the median nerve (*MN*) remains because tendons in general are more anisotropic than the nerves (Reproduced with permission from Philip Peng Educational Series)

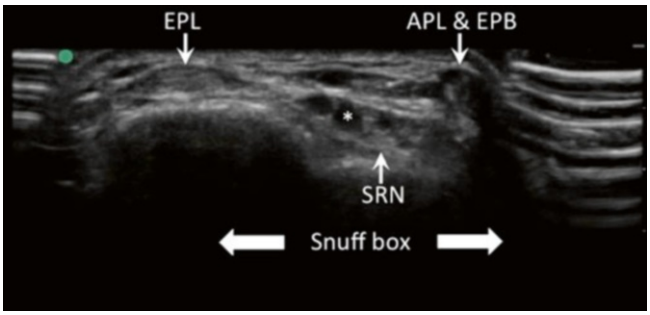


Fig. 35.7 Sonogram of the superficial branch of the radial nerve (*SRN*) in the snuff box. This anatomical region is bound by the abductor pollicis longus (*APL*) and extensor pollicis brevis (*EPB*) on the radial side and the extensor pollicis longus (*EPL*) on the ulnar side. The radial artery is indicated with *asterisk* (*) (Reproduced with permission from Philip Peng Educational Series)

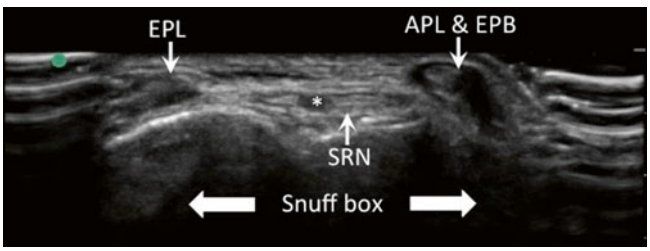


Fig. 35.8 Same scan as Fig. 35.7 except that the veins are deliberately compressed to enhance the visualization of the superficial branch of the radial nerve. The radial artery is indicated with *asterisk* (*) (Reproduced with permission from Philip Peng Educational Series)

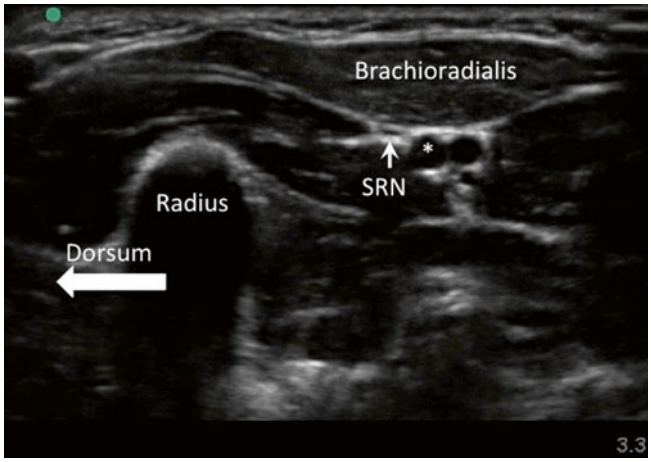


Fig. 35.9 Sonogram of the superficial branch of the radial nerve (*SRN*) at approximately four fingerbreadths distal to the elbow. The forearm is put in mid-prone position and the probe is placed over the radius. At this level, the SRN is seen in location close to the radial artery (*) beneath the brachioradialis muscle (Reproduced with permission from Philip Peng Educational Series)

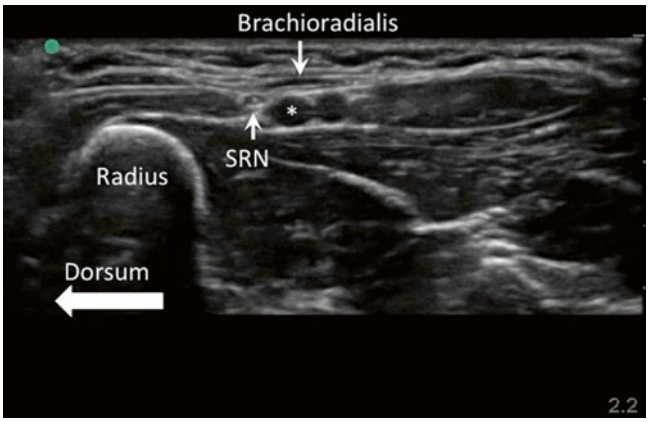


Fig. 35.10 Moving the ultrasound probe distally compared to the position in Fig. 35.9. The brachioradialis muscle become tendon the superficial branch of the radial nerve (*SRN*) is seen coming out to the dorsal surface of the forearm. The radial artery is indicated with *asterisk* (*) (Reproduced with permission from Philip Peng Educational Series)

Suggested Reading

1. Bianchi S, Martinoli C. Elbow. In: Bianchi S, Martinoli C, editors. Ultrasound of the musculoskeletal system. Berlin/Heidelberg: Springer; 2007.
2. Covic D. Blockaden peripherer Nerven im Ellenbogenbereich. Blockade peripherer Nerven im Handwurzelbereich. In: Hörster W, Kreuscher H, Niesel H, et al., editors. Regionalanästhesie. Stuttgart: Fischer Verlag; 1989. p. 86–101.
3. Dealunay L, Chelly JE. Blocks at the wrist provide effective anesthesia for carpal tunnel release. Can J Anesth. 2001;48:656–60.
4. Gebhard RE, Al-Samsam T, Greger J, et al. Distal nerve blocks at the wrist for outpatient carpal tunnel surgery offer cardiovascular stability and reduce discharge time. Anesth Analg. 2002;95:351–5.
5. Hahn MB, McQuillan PM, Sheplock GJ. Regional anesthesia. St. Louis: Mosby; 1996.
6. Löfström B. Blockade der peripheren Nerven des Armes in der Handwurzelgegend. In: Eriksonn E, editor. Atlas der Lokalanästhesie (2. Auflage). Berlin/New York: Springer; 1980. p. 86–92.
7. Plaikner M, Gruber H, Judmaier W, Brenner E. Upper extremity nerves. In: Peer S, Gruber H, editors. Atlas of peripheral nerve ultrasound with anatomic and MRI correlation. Berlin/Heidelberg: Springer ; 2013.

Chapter 36

Elbow Joint and Tennis Elbow Injection

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_36, © Springer International Publishing Switzerland 2015

Tennis Elbow or Lateral Epicondylitis

Lateral epicondylitis, commonly termed "tennis elbow," presents with pain and tenderness of the affected lateral elbow region. Current evidence suggests that the pathology is related to enthesopathy, tendinosis, or insertional tendon tear of the common extensor tendon (CET), which includes the extensor carpi radialis brevis (ECRB) and the extensor digitorum communis (EDC), or radial collateral ligament (Fig. 36.1). Percutaneous interventions include steroid injection or tendon fenestration. Although these injections are commonly practiced with landmark-based technique, ultrasound enables direct visualization of the target tissues, the needle, and the spread of injectate. Thus, sonographic guidance can potentially improve the efficacy of percutaneous procedures.

Symptoms

The main presenting symptom is stabbing elbow pain (lateral epicondyle), radiating as far as the thumb (Fig. 36.2). Physical examination reveals point tenderness in the region of the lateral epicondyle and pain and weakness exacerbated by resisted wrist extension and supination (Cozen test and third-digit tennis elbow test).

Procedure

The patient is usually in sitting position with the elbow 90° in flexion rest comfortably on a pillow over a table. With sterile precaution, a linear high-frequency probe (6–13 MHz) is placed over the lateral elbow region, revealing the lateral epicondyle, capitellum, and head of radius (Fig. 36.3). The target structure CET can be visualized in long-axis view. A 23-G or 25-G needle is inserted in-plane from inferior to superior direction all the way to the epicondylar insertion of CET (Fig. 36.3). Applying light pressure to the plunger of the syringe while withdrawing the needle, the steroid solution is injected in the target point. The practitioner should avoid overpressurizing the plunger which may cause artificial tissue injury. The total volume is approximately 1 mL with a mixture of local anesthetic and 10 mg of triamcinolone.

When tendon fenestration is performed, the patient position, scanning procedure, and needle used are the same. The needle repeatedly fenestrate the tendinotic tendon until the tissue is softened, and crepitation is diminished.

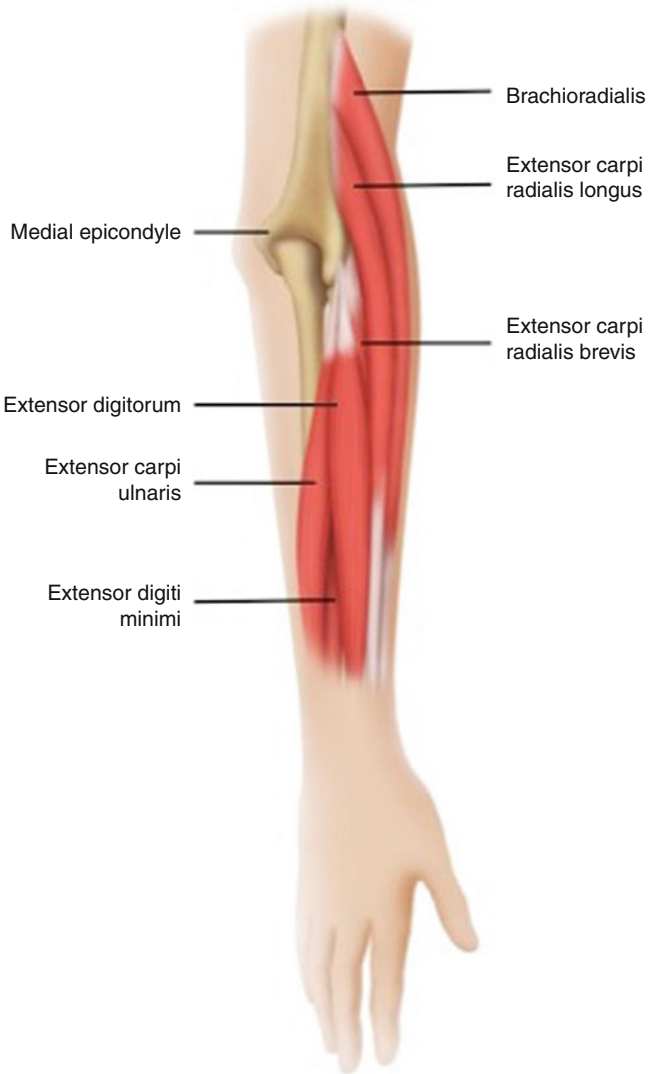


Fig. 36.1 Illustration demonstrating the musculotendinous anatomy of the lateral aspect of the elbow, near the site of the tendon origin on the lateral epicondyle (Reproduced with permission from Philip Peng Educational Series)

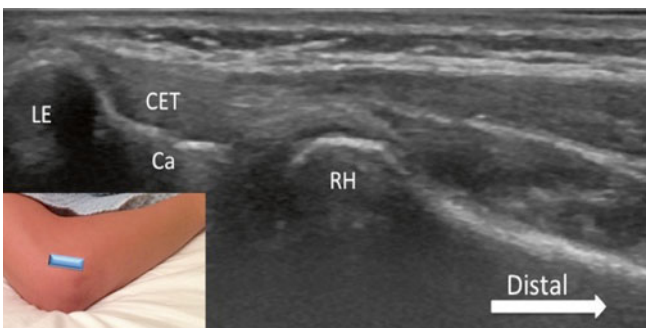


Fig. 36.3 Sonogram of the common extensor tendon (*CET*) on its long axis. The position of the ultrasound probe is shown in the *inset*, with the cranial end of the probe placed on the lateral epicondyle (*LE*). The CET origin is seen as a continuous band of longitudinally oriented fibers. *Ca* capitellum, *RH* radial head (Reproduced with permission from Philip Peng Educational Series)

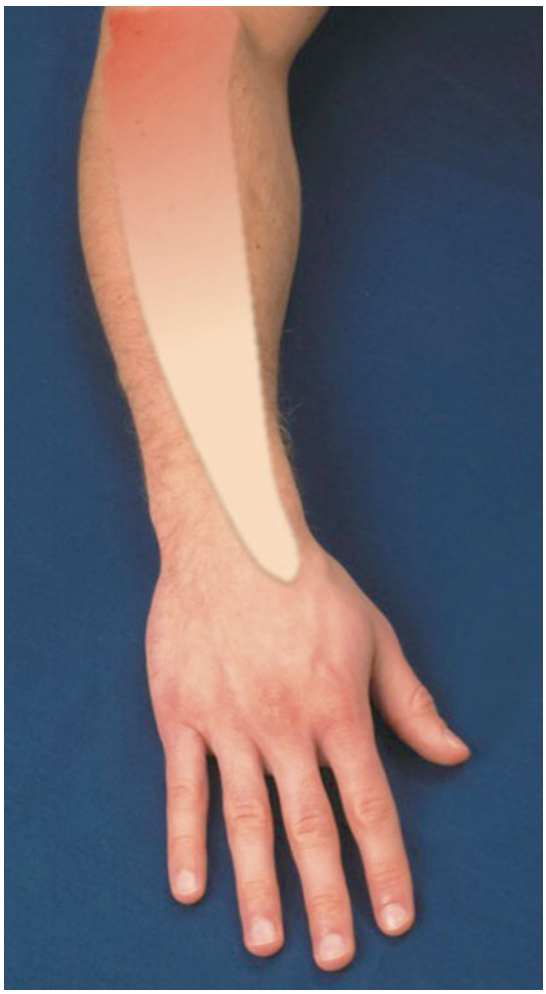


Fig. 36.2 Diagram showing the pain area in a patient with tennis elbow (Reproduced with permission from Philip Peng Educational Series)

Intra-articular Injection of the Elbow Joint

The elbow joint is a compound joint formed by the proximal ends of the ulna and radius and the distal end of the humerus. The ulna (olecranon) and humeral capitellum form a hinge joint (Fig. 36.4). The radial head articulates with the capitellum of the humerus, allowing swivel movement, while it also revolves within the radial notch of the ulna. Both movements from radial head articulations allow axial rotation of the forearm (Fig. 36.5). The joint capsule encloses the entire elbow joint including three fat pads in different fossae: radial, coronoid, and olecranon fossae (Fig. 36.5). The elbow joint is transversed by three nerves, and understanding the anatomy is important to avoid nerve injury to these nerves (Fig. 36.6). At the level just proximal to the elbow joint, the radial nerve is sandwiched between the brachioradialis and brachialis muscles, the ulna lies subcutaneously lateral to the medial head of the triceps muscle in the posterior compartment of the arm, and the median nerve runs besides the brachial artery lateral to the biceps brachii and brachialis muscles.

Indications

Intra-articular injections of the elbow are indicated for the management of pain from osteoarthritis or rheumatoid arthritis, as well as performance of arthrogram for diagnostic purpose. Intra-articular aspiration can be diagnostic and therapeutic when infection is suspected.

Ultrasound-Guided Posterior Approach

The patient is placed in supine position with the elbow in 90° flexion and the arm resting on a pillow over the chest. With sterile precautions, a linear ultrasound probe is placed in-plane over the muscle and tendon of the triceps (Fig. 36.7). The cephalad end of the probe is then rotated 30° in lateral direction until the triceps tendon is out of sight while the hyaline cartilage (hypoechoic shadow) of the humerus is still in sight (Fig. 36.8). A 1.5-inch, 22-G needle is inserted in-plane from cephalad to caudal, deep to the joint capsule, and 3 mL of medication (2% lidocaine and 40mg Depo-Medrol) is injected.

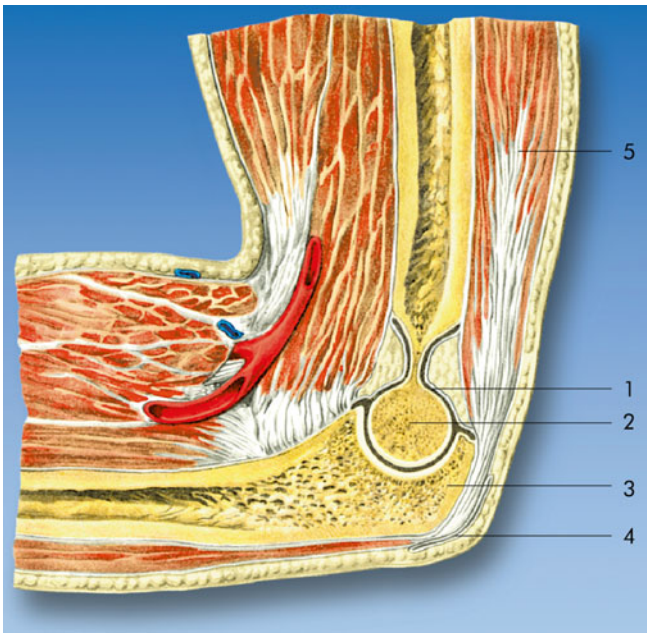


Fig. 36.4 Elbow joint. (*1*) Articular cavity, (*2*) trochlea of the humerus, (*3*) olecranon, (*4*) subcutaneous olecranon bursa, (*5*) triceps brachii muscle (With permission from Danilo Jankovic)

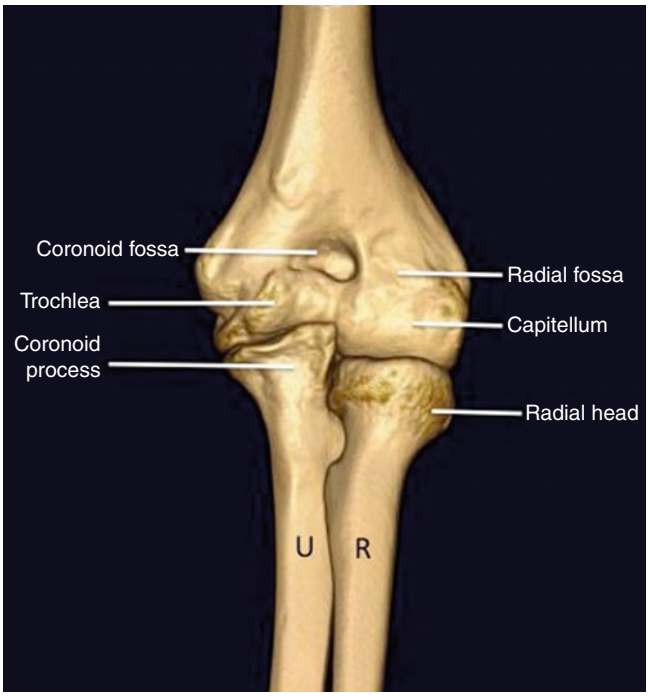


Fig. 36.5 Three-dimensional picture of the elbow—front view. *U* ulna, *R* radius (Reproduced with permission from Philip Peng Educational Series)

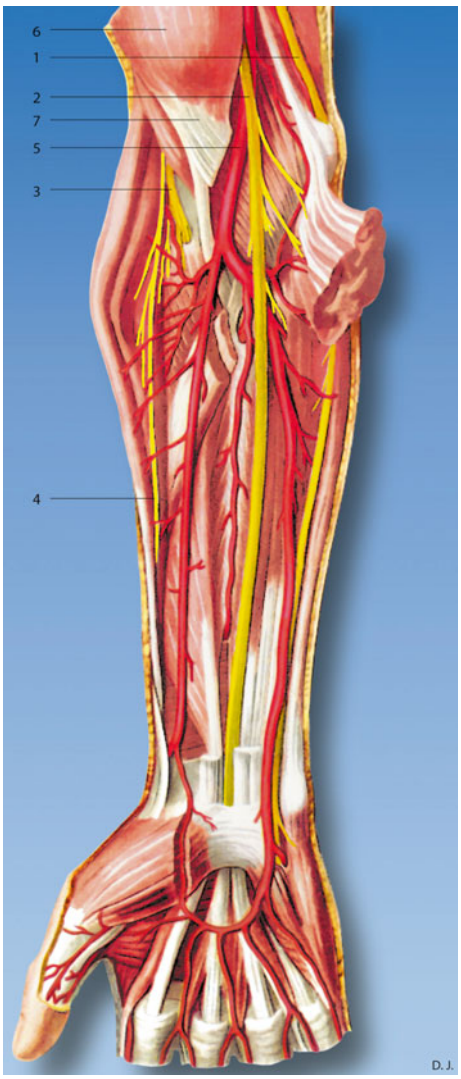


Fig. 36.6 Anatomy. (*1*) Ulnar nerve, (*2*) median nerve, (*3*) deep branch of the radial nerve (anterior interosseous nerve), (*4*) superficial branch of the radial nerve, (*5*) brachial artery, (*6*) biceps brachii muscle, (*7*) bicipital aponeurosis (With permission from Danilo Jankovic)

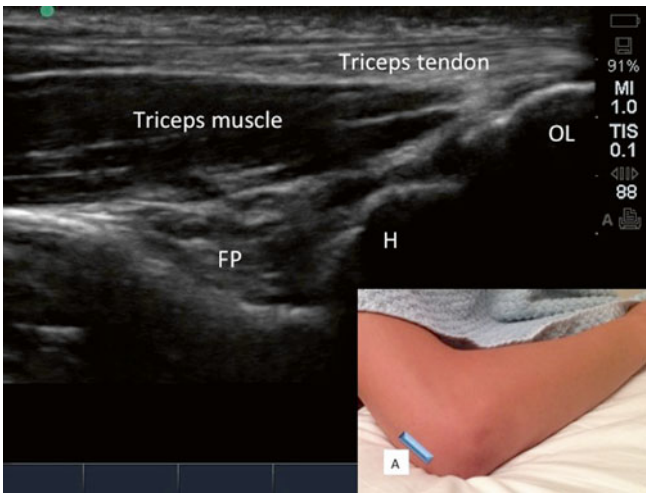


Fig. 36.7 Sonogram of the elbow joint viewing from the posterior aspect. The position of the elbow and the ultrasound probe is shown in the *inset*. *OL* olecranon, *FP* fat pad in the olecranon fossa, *H* humerus (Reproduced with permission from Philip Peng Educational Series)

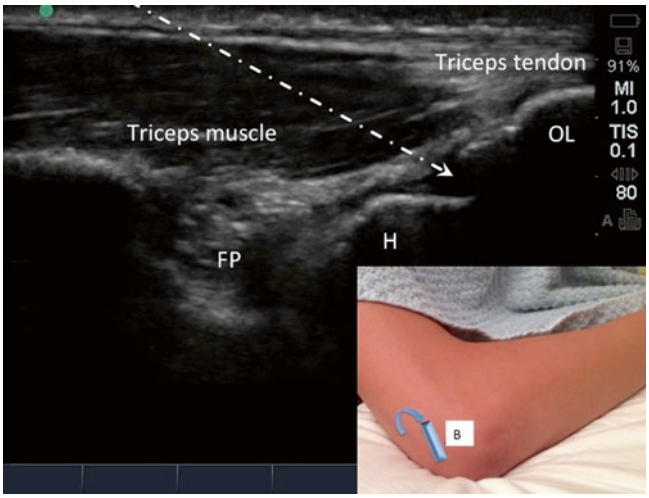


Fig. 36.8 Sonogram of the elbow joint as in Fig. 36.7 except that the ultrasound probe is rotated as shown in the *inset*. The *dotted arrow* indicates the needle insertion into the elbow joint. *FP* fat pad; *H* humerus; *OL* olecranon (Reproduced with permission from Philip Peng Educational Series)

Suggested Reading

1. Bodor M, Lescher JM, Colio S. Ultrasound-guided hand, wrist, and elbow injection. In: Narouze S, editor. Atlas of ultrasound guided procedures in interventional pain management. New York: Springer; 2010. p. 307–24.
2. Jankovic D. Elbow and wrist. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. 3rd ed. Malden: Blackwell-Malden Masachussets, Oxford, Carlton Victoria; 2004. p. 149–58.
3. Lee SH. Lateral epicondylitis. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, Musculoskeletal pain, vol. 3. Philip Peng Educational Series. 1st ed. iBook, California CA: Apple Inc.; 2013. p. 42–50.
4. Louis LJ. Musculoskeletal ultrasound intervention: principles and advances. Radiol Clin North Am. 2008;46:515–33.

Chapter 37

Carpal Tunnel Injection

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_37, © Springer International Publishing Switzerland 2015

The most frequent entrapment neuropathy is carpal tunnel syndrome, which affects approximately 3–6 % of adults in the general population. Predisposing factors include obesity, chronic polyarthritis, diabetes mellitus, gout, and dysproteinemia. The feature common to all of these conditions is that a discrepancy exists between the size of the carpal tunnel and the volume of its contents. This compresses the median nerve. Typically, carpal tunnel syndrome is associated with numbness, with or without pain, in the distribution of the median nerve (Fig. 37.1). Motor weakness and atrophy of the intrinsic thenar muscles may follow. Patients wake in the morning or at night with a feeling that their hands have "gone to sleep." The symptoms can be provoked by pressure on the flexor retinaculum, such as holding their wrist in complete and forced flexion (pushing the dorsal surfaces of both hands together) for 30–60 s (Phalen's sign).

The carpal tunnel is a fibro-osseous tunnel, which contains the flexor tendons of the fingers (tendons of the flexor digitorum profundus, flexor digitorum superficialis, and flexor pollicis longus) and the median nerve (Fig. 37.2). The volar border is covered by the flexor retinaculum, a broad ligament that extends from the tuberosities of the scaphoid and the trapezium to the pisiform and the hook of the hamate.

The median nerve lies superficial to the tendons of the flexor pollicis longus and medial to the flexor carpi radialis just before entering the carpal tunnel. However, the location of the median nerve can be more medial, and therefore blind injection may result in nerve injury.

Ultrasound-Guided Carpal Tunnel Injection

The patient is in sitting position with the hand supinated resting on a pillow. The procedure is performed with sterile technique. A high-frequency linear ultrasound probe (6–13 MHz) is placed at the level of the pisiform and scaphoid tubercle. With the probe in this position, the ulnar artery, ulnar and median nerves should be visualized. The median is usually superficial and medial to the flexor pollicis longus (Fig. 37.3). A 1.5-inch, 25-G needle is inserted in-plane superficial and lateral to the ulnar artery toward the space between the median nerve and flexor retinaculum. Advancement of needle may be facilitated by hydrodissection with normal saline to release adhesion. A total volume of 2–3 mL of 1 % lidocaine mixed with 20–40 mg triamcinolone acetonide is sufficient. The patient is instructed to rest the hand over the next 24 h.

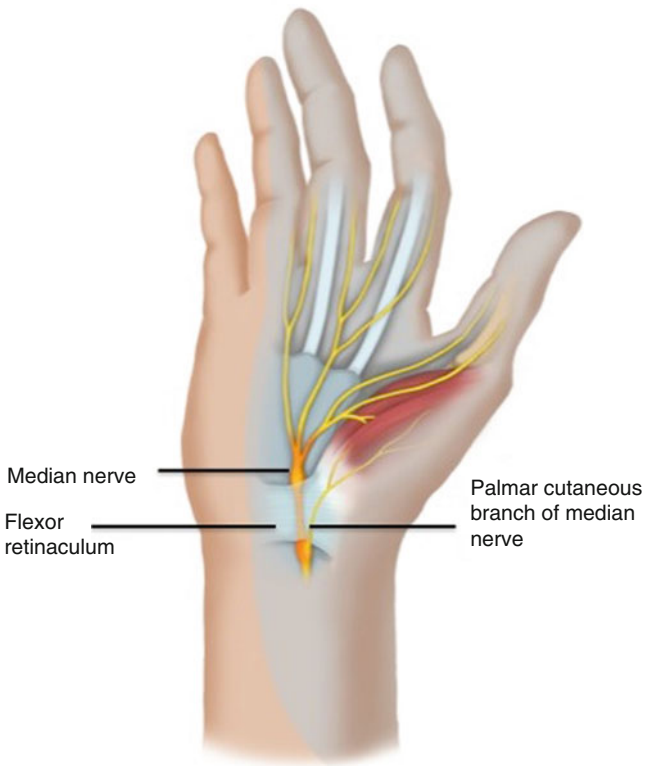


Fig. 37.1 Sensory distribution of the median nerve

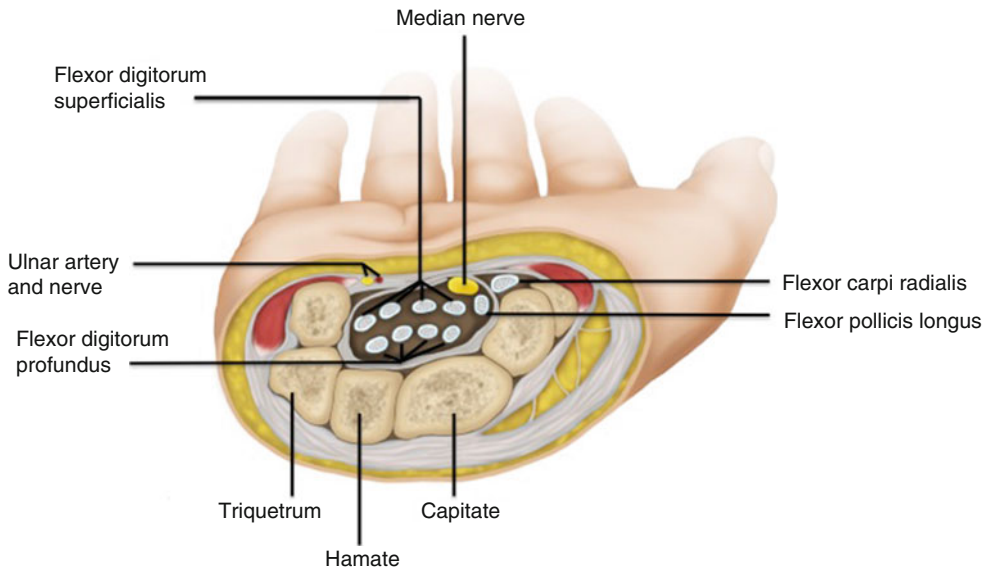


Fig. 37.2 Illustration of cross section across wrist

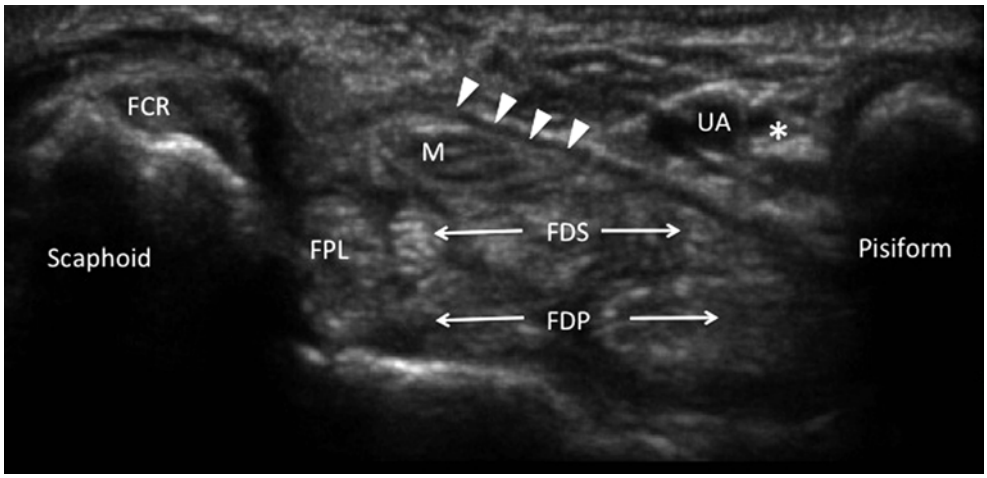


Fig. 37.3 Short-axis view of the carpal tunnel (proximal) in a healthy volunteer shows honeycomb appearance of the normal median nerve (*M*) with multiple rounded hypoechoic fascicles surrounded by hyperechoic perineurium and epineurium. *UA* ulnar artery, *arrowheads* flexor retinaculum (hypoechoic), *M* median nerve, *FCR* flexor carpi radialis, *FPL* flexor pollicis longus, * ulnar nerve, *FDS* flexor digitorum superficialis, *FDP* flexor digitorum profundus

Suggested Reading

1. Bodor M, Lescher JM, Colio S. Ultrasound-guided hand, wrist, and elbow injection. In: Narouze S, editor. Atlas of ultrasound guided procedures in interventional pain management. New York: Springer; 2010. p. 307–24.
2. Demircay E, Civelek E, Cansever T, Kabatas S, Yilmaz C. Anatomic variations of the median nerve in the carpal tunnel: a brief review of the literature. Turk Neurosurg. 2011;21:388–96.
3. Graham B. The value added by electrodiagnostic testing in the diagnosis of carpal tunnel syndrome. J Bone Joint Surg Am. 2008;90:2587–93.
4. Katz JN, Simmons BP. Clinical practice. Carpal tunnel syndrome. N Engl J Med. 2002;346:1807–12.
5. Lee SH. Carpal tunnel syndrome. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide, Musculoskeletal pain, vol. 3. Philip Peng Educational Series. 1st ed. iBook, California CA: Apple Inc.; 2013. p. 51–8.
6. Linskey ME, Segal R. Median nerve injury from local steroid injection in carpal tunnel syndrome. Neurosurgery. 1990;26:512–5.
7. Singer G, Ashworth CR. Anatomic variations and carpal tunnel syndrome: 10-year clinical experience. Clin Orthop Relat Res. 2001;392:330–40.
8. Smith J, Wisniewski SJ, Finnoff JT, Payne JM. Sonographically guided carpal tunnel injections: the ulnar approach. J Ultrasound Med. 2008;27(10):1485–90.

Part VII
Thoracic Region

Chapter 38

Thoracic Paravertebral Block

Steven Renes, MD, PhD, FIPP (✉) • Santine A.S. van den Heuvel • Geert-Jan van Geffen
Radboud University Nijmegen Medical Center, Department of Anesthesiology, Pain and Palliative Medicine, Huispost 717, Route 717, Postbus 9101, 6500 HB Nijmegen, The Netherlands
e-mail: Steven.Renes@radboudumc.nl

Danilo Jankovic, MD (Classic Landmark-Based Thoracic Paravertebral Block)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_38,

Introduction

In thoracic paravertebral block (TPVB), local anesthetic is injected in the vicinity of the thoracic spinal nerves, in order to obtain an ipsilateral somatic, segmental, and sympathetic nerve block.

Indications

Thoracic paravertebral block can be used for unilateral surgical procedures such as thoracotomy, major breast surgery, cholecystectomy, renal surgery, inguinal herniorrhaphy, and laparotomy. A bilateral thoracic paravertebral block can be performed to obtain bilateral analgesia.

In pain medicine, thoracic paravertebral block is useful in management of acute and chronic pain such as acute herpes zoster, cancer pain, post-thoracotomy pain, rib fractures, and postherpetic neuralgia.

Contraindications

Contraindications to thoracic paravertebral blockade include allergy to local anesthetics, coagulation disorders, patient refusal, infection at the insertion opening, tumors in the thoracic paravertebral space, and pleural-thoracic empyema.

Anatomy

The thoracic paravertebral space is a wedge-shaped area located on both sides of the vertebral column that contains the thoracic nerves and the sympathetic trunk. The posterior wall is formed by the superior costotransverse ligament. The medial wall is formed by the vertebral body and intervertebral disc; through the intervertebral foramen, the thoracic paravertebral space is continuous with the epidural space. The anterolateral wall is formed by the parietal pleura, and laterally, it is continuous with the intercostal space (Fig. 38.1a, b; see also Chap. 39, Figs. 39.1 and 39.2). The cranial border of the thoracic paravertebral space is mid- to low cervical. Caudally the thoracic paravertebral space is continuous with the retroperitoneal space via the medial and lateral arcuate ligament of the diaphragm.

The thoracic paravertebral space contains fatty tissue, the spinal nerves, the dorsal rami, the intercostal vessels, the rami communicantes, and the sympathetic chain. The thoracic paravertebral space is divided in two communicating compartments by a layer of loose connective tissue, the endothoracic fascia which is the deep fascia of the thorax; ventral the "extrapleural" paravertebral space between the parietal pleura and the endothoracic fascia and dorsal to the endothoracic fascia the "subendothoracic" paravertebral space. The sympathetic chain and a fine loose areolar tissue (the fascia subserosa) are located in the "extrapleural" paravertebral space, while the intercostal nerves and vessels are located in the "subendothoracic" paravertebral space.

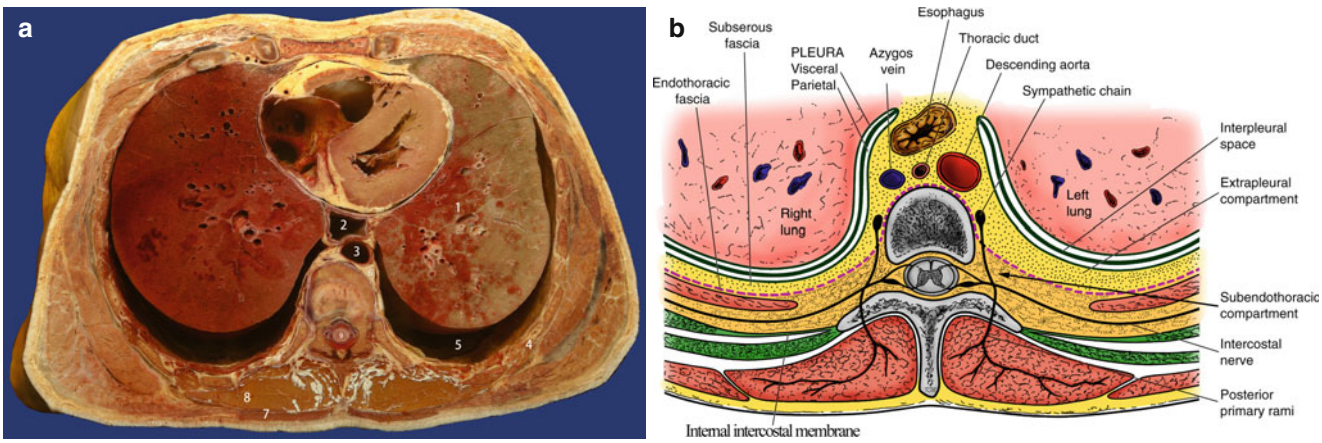


Fig. 38.1 (**a**) Transversal dissection at the level of the T9. (*1*) Lung, (*2*) esophagus, (*3*) aorta, (*4*) intercostal space, (*5*) interpleural space, (*6*) sympathetic trunk, (*7*) trapezius muscle, (*8*) longissimus muscle, (*9*) spinal cord (Reproduced with permission from Danilo Jankovic). (**b**) Anatomy of the thoracic paravertebral space (Reproduced with permission from www.aic.cuhk.edu.hk/usgraweb [1])

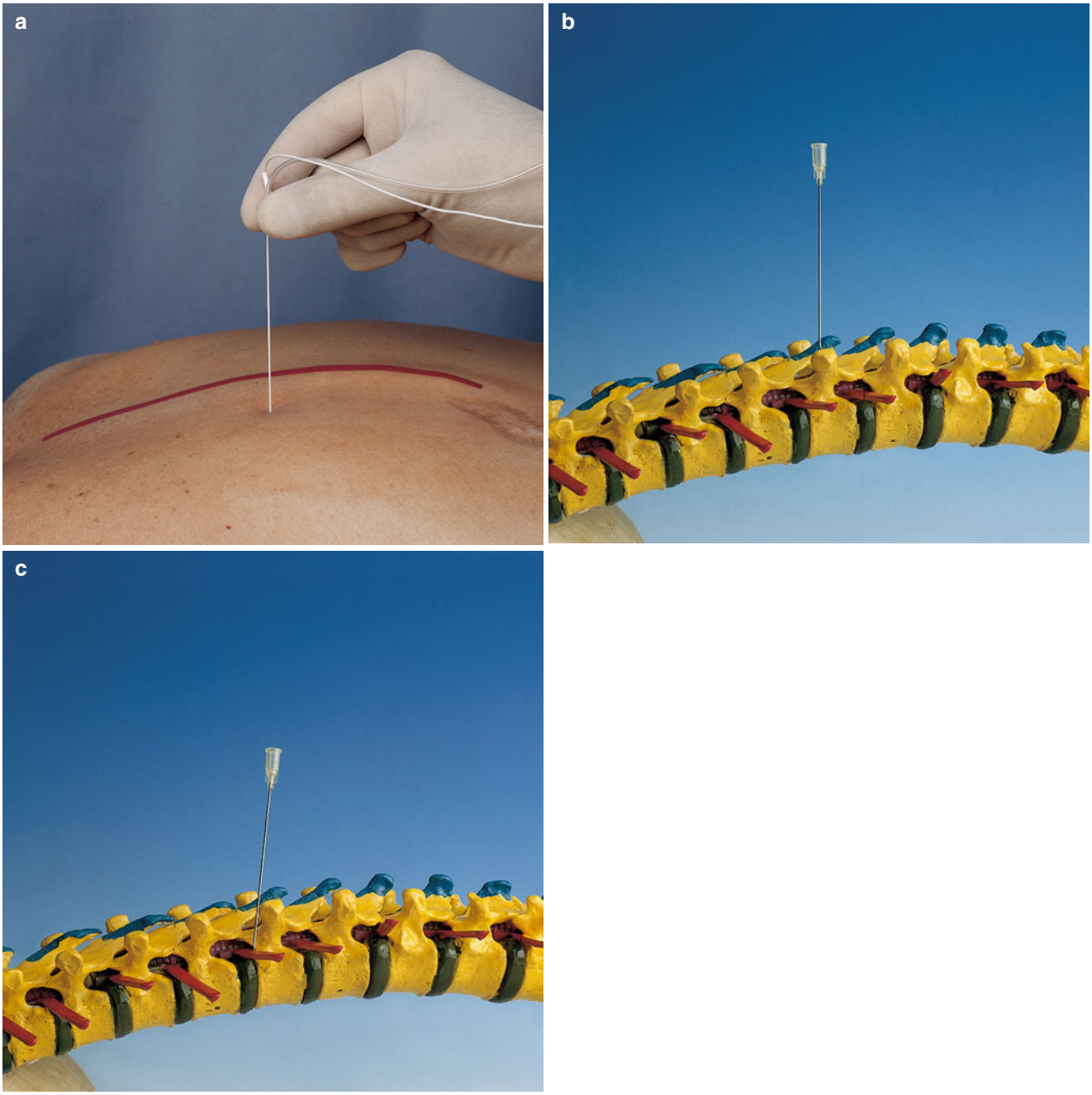


Fig. 38.2 (**a**) Introducing the needle perpendicular to the skin surface (Reproduced with permission from Danilo Jankovic). (**b**) Bone contact (transverse process) (Reproduced with permission from D. Jankovic). (**c**) The needle is withdrawn to lie subcutaneously and then introduced 2 cm deeper past the transverse process (Reproduced with permission from D. Jankovic)

Technique

Thoracic paravertebral block can be performed with the patient in the sitting, lateral decubitus, or prone position. The thoracic paravertebral block can be performed in an awake patient, under sedation or general anesthesia.

A 18–20-gauge blunt-tipped block needle or a Tuohy needle can be used for block performance.

There are different techniques of performing the thoracic paravertebral blockade: classic landmark-based with or without a "loss of resistance," pressure monitoring, nerve stimulation, intercostally placed paravertebral catheterization, and ultrasound-guided thoracic paravertebral block. In this chapter, the classic landmark-based technique and the ultrasound-guided thoracic paravertebral block will be described.

To obtain anesthesia or analgesia in more than one dermatome, either a multi-injection technique, where in each adjoining dermatome 4–5 mL local anesthetic is injected, or a single-shot injection technique, where a single 20 mL bolus of local anesthetic is injected at the appropriate level, can be used. It is the opinion of the authors that the latter technique is preferable in order to avoid accidental pleural puncture. In the single-injection technique, 20 mL of local anesthetic consistently blocks five adjoining dermatome levels. It should be noted however that the distribution is preferentially four dermatomes caudal and only one dermatome level cranial with respect to the puncture site. Therefore, the appropriate level to perform a single-injection multilevel thoracic paravertebral block for mastectomy, thoracotomy/VATS, major renal surgery, and unilateral laparotomy is at vertebral body interspace T2–T3, T3–T4, T8–T9, and T9–T10, respectively.

Classic Landmark-Based Thoracic Paravertebral Block

At the level of the appropriate vertebral body, a needle is inserted 2.5–4 cm lateral to the most cranial part of the spinous process (Fig. 38.2a). Subsequently, the needle is advanced perpendicular to the skin in all directions until it contacts the transverse process, which is usually located at a depth of 2–4 cm (Fig. 38.2b).

If no contact is made with the transverse process at this depth, the needle can be located in between two transverse processes; the needle has to be withdrawn and redirected in a more cranial or caudal direction. For safety, it is important that the transverse process is identified in order to minimize the risk of an accidental pleural puncture. After bony contact is made with the transverse process, the needle is withdrawn slightly and the transverse process is slowly walked off in either a cranial or caudal direction until a "loss of resistance" is observed, usually at a depth of 1.5–2 cm from the transverse process (Fig. 38.2c). The observed "loss of resistance" is the needle traversing through the superior costotransverse ligament and is more subtle compared to the definite give in epidural block when the needle traverses the flavum ligament.

If a catheter technique is indicated, the catheter can be threaded 1–2 cm into the thoracic paravertebral space (more than 2 cm will increase the incidence of inadvertent epidural placement).

An alternative to the "loss-of-resistance" technique is the "blind" classic landmark-based technique: after identification of the transverse process as described above, the needle is withdrawn slightly and the transverse process is slowly walked off in either a cranial or caudal direction for 1–2 cm. This "blind" technique is not associated with a higher risk of pneumothorax with respect to the "loss-of-resistance" technique.

During injection, the following points must be observed without fail:

1. The person performing the injection must stand on the side being blocked.
2. The injection point must be located within 4 cm lateral to the midline (rib contact, risk of pneumothorax!).
3. There is a risk of perforating the dural cuff if the injection is made too far medially (epidural or subarachnoid injection).
4. The transverse process is ca. 0.6–0.7 cm thick.
5. If there is no bone contact after 2.5–5 cm (depending on the habitus of the patient), the direction of the needle must be corrected (the transverse process has been missed; risk of pleural puncture).
6. If the patients coughs, it can indicate pleural irritation. The procedure should be halted.
7. Eliciting paresthesias is not obligatory.
8. The injection should be carried out on a incremental basis, with frequent aspiration (blood, CSF?).

Ultrasound-Guided Thoracic Paravertebral Block

The two most commonly used ultrasound-guided thoracic paravertebral block techniques are the paramedian sagittal thoracic paravertebral block and the transverse thoracic paravertebral block. Before describing these techniques, first the basic sonoanatomy of the relevant structures of the thoracic paravertebral space is described.

Basic Sonoanatomy of the Relevant Structures of the Thoracic Paravertebral Space

A 5–12-MHz linear array ultrasound transducer or a 2–5-MHz curved array ultrasound transducer can be used for performing the paramedian sagittal thoracic paravertebral block and the transverse thoracic paravertebral block. In the opinion of the authors, the curved array ultrasound transducer is often more useful in performing the transverse thoracic paravertebral block.

Key anatomical structures for the ultrasound-guided thoracic paravertebral block techniques that must be identified and visualized are the transverse process and the parietal pleura.

Paramedian Sagittal Thoracic Paravertebral Block

The paramedian sagittal thoracic paravertebral block may be performed using an in-plane and out-of-plane needle insertion.

The ultrasound transducer is placed vertical 2–3 cm lateral to the midline at the appropriate thoracic level with the orientation marker directed cranial. The transverse processes are visible on the ultrasound image as a rounded hyperechoic structure with acoustic shadowing anteriorly (ultrasound waves do not penetrate bone; thus, all the ultrasound waves are reflected backwards resulting in a black shadow beneath the hyperechoic line). Located more ventrally is the parietal pleura which is visible as a bright hyperechoic line between the transverse processes (Fig. 38.3a). Between the transverse processes, the superior costotransverse ligament can be often visualized. Color Doppler can be used to identify presence of the intercostal vessels.

For the in-plane technique, the needle is inserted from caudal in the plane of the ultrasound beam and advanced until the needle traverses the superior costotransverse ligament; if the superior costotransverse ligament cannot be visualized, the needle is advanced until it is just positioned posterior of the parietal pleura. After aspiration for blood and air, local anesthetic can be injected in increments of 5 mL with concomitant anterior movement of the parietal pleura indicating correct needle placement (Fig. 38.3b). Catheter insertion in this approach may be difficult due to the steep entry angle of the needle with respect to the thoracic paravertebral space.

It should be noted that although this is an in-plane technique, the needle is often difficult to visualize due to the steep angle with the skin; injection of small aliquots of normal saline to observe "tissue hydrosection" can be helpful in identifying the tip of the needle.

For the out-of-plane technique, the needle is inserted just lateral to the probe. Tissue hydrosection with normal saline can be used to evaluate needle-tip position. The needle is advanced until it traverses the superior costotransverse ligament or is located just posterior to the parietal pleura. After aspiration for blood and air, local anesthetic can be injected in increments of 5 mL with concomitant anterior movement of the parietal pleura indicating correct needle placement. Again, catheter insertion in this approach may be difficult due to the steep entry angle of the needle with respect to the thoracic paravertebral space.

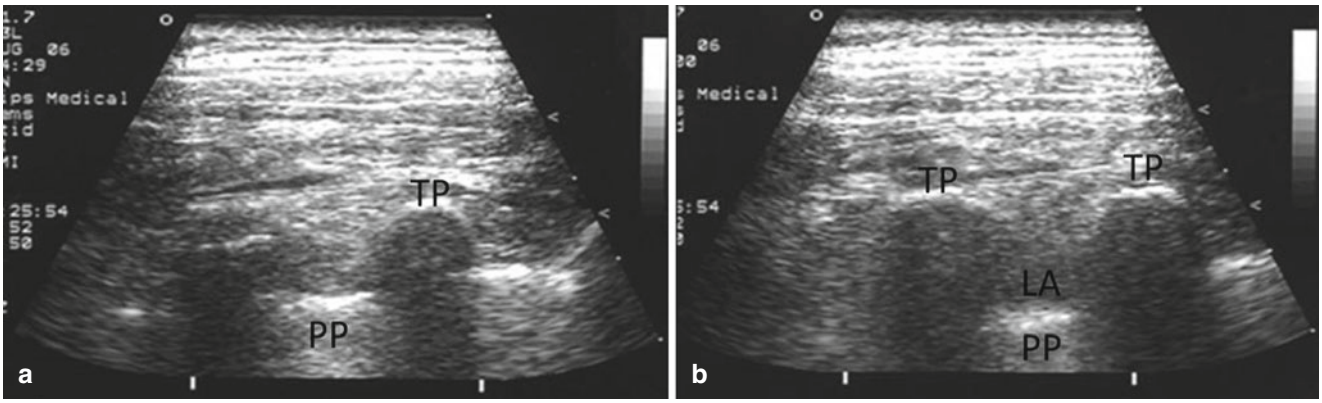


Fig. 38.3 Paramedian sagittal thoracic paravertebral block. Visualization of local anesthetic and the downward shift of parietal pleura on ultrasound longitudinal image. (**a**) Before injection, (**b**) after injection. *TP* transverse process, *PP* parietal pleura, *LA* local anesthetic (Reproduced with permission from Hara et al. [2])

Transverse Thoracic Paravertebral Block

The transverse thoracic paravertebral block may be performed using an in-plane and out-of-plane needle insertion. The authors recommend the use of a 2–5-MHz curved array ultrasound transducer for this technique.

The ultrasound transducer is first positioned in a transverse and partial oblique position to the vertebral column, parallel to the rib with the orientation marker directed medially. On the ultrasound image, the transverse process is visualized medially on the ultrasound image as a hyperechoic structure with acoustic shadowing anteriorly. On the lateral side of the ultrasound image, the parietal pleura is visible as a hyperechoic line that disappears under the transverse process due to the acoustic shadowing. Subsequently the ultrasound transducer is moved either cranially or caudally (approximately 5 mm) until the transverse process is only visible as an ultrasound shadow and thus an intercostal ultrasound image is obtained. In the thoracic paravertebral region, the innermost intercostal muscle becomes the internal intercostal membrane, which is continuous medially with the superior costotransverse ligament, and can often be visualized as a very thin hyperechoic line.

For the in-plane technique, the needle is inserted from lateral (approximately 40 mm from the midline) to medial in the plane of the ultrasound beam and advanced until the needle traverses the internal intercostal membrane *or* until the tip of the needle is positioned just posterior from the parietal pleura in the region where the innermost intercostal muscle is absent (Fig. 38.4a). After aspiration for blood and air, local anesthetic can be injected in increments of 5 mL with concomitant anterior movement of the parietal pleura indicating correct needle placement (Fig. 38.4b). Lack of anterior movement of the parietal pleura with injection of local anesthetics indicates incorrect needle-tip placement. Catheter insertion in this approach is usually very easy. In contrast with the in-plane paramedian sagittal thoracic paravertebral block, needle visualization is easy in this approach.

For the out-of-plane technique, the ultrasound image should only depict the transverse process medially and the parietal pleura lateral (the probe is not moved cranial or caudal). The needle is inserted in the middle of the ultrasound transducer and directed cephalad. The needle is advanced until the tip of the needle is lateral to the transverse process and just posterior of the parietal pleura. Injection of small aliquots of normal saline to observe "tissue hydrosection" can be helpful in identifying the tip of the needle.

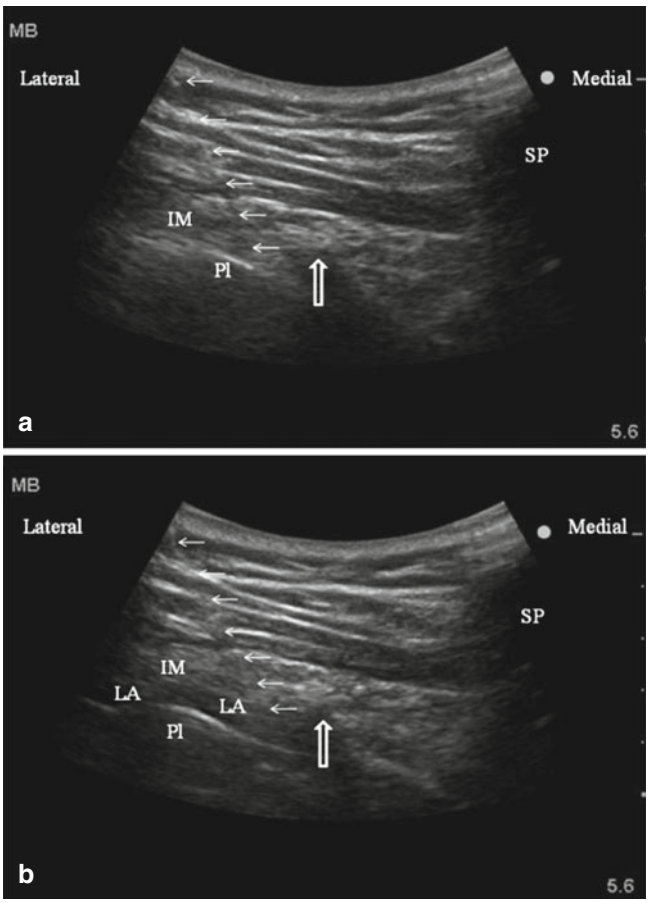


Fig. 38.4 Transverse thoracic paravertebral block, in-plane technique. Sonogram at T3–T4 vertebral level, before (**a**) and after (**b**) injecting 5 mL *LA*. The needle (*small white arrows*) is positioned in the thoracic paravertebral space. The needle approach is from lateral to medial through the intercostal muscles (*IM*). *Large arrow* indicates the shadow of the transverse process, *SP* spinous process, *Pl* pleura. The innermost intercostal muscle is absent in the thoracic paravertebral space (Reproduced with permission from Renes et al. [3])

Continuous Infusion of Local Anesthetic

Bupivacaine 0.25 or 0.5 % is not recommended for continuous infusion, due to its tendency for rising bupivacaine plasma levels as a result of stacking, although there is no literature that this leads to clinical signs of local anesthetic toxicity. Lidocaine 1 % and ropivacaine 0.2 % are considered to be safe. A typical dose for continuous infusion of ropivacaine 0.2 % is 0.1–0.2 ml/kg/h up to a maximum of 14 mL/h.

Success Rate and Quality of Analgesia

Using the conventional method, the thoracic paravertebral blockade has a success rate of 90–93 %, which is similar to the success rate of epidural blockade. The use of ultrasound for thoracic paravertebral blockade placement seems to result in a higher success percentage.

A unique feature of the thoracic paravertebral blockade is the very strong inhibition of somatosensory-evoked potentials (SSEPs). This inhibition of SSEPs is much more marked as compared to epidural and spinal anesthesia, because thoracic paravertebral blockade also blocks the sympathetic trunk in contrast to central neuraxial blocks.

Complications and Side Effects

The incidence of complications and side effects of the thoracic paravertebral blockade is low. The most important are hypotension (3.9 %), pleural puncture (1.1 %), and pneumothorax (0.5 %). Inadvertent pleural puncture only sporadically results in a pneumothorax; if pleural puncture occurs, conversion to interpleural analgesia can be considered. The calculated risk of pneumothorax in a bilateral thoracic paravertebral blockade is 1:40,000. Application of ultrasound for thoracic paravertebral block may result in reduction in the incidence of pneumothorax.

Bilateral Thoracic Paravertebral Blockade

A bilateral thoracic paravertebral block has been described for several indications, including bilateral thoracotomy and abdominal surgery. A bilateral thoracic paravertebral blockade is associated with good efficacy and low incidence of complications and side effects [4]. To date, no serious adverse events have been reported. Bilateral thoracic paravertebral blockade may be considered as an alternative for surgical procedures in which epidural analgesia is strongly contraindicated, for example, in patients with coagulation disorders.

Chest Radiography

A chest radiograph can be considered in patients with a thoracic paravertebral catheter to evaluate spread of radiopaque dye. The thoracic paravertebral space is separated by the endothoracic fascia into an anterior and posterior compartment. Spread of radiopaque dye in the posterior compartment results in a cloud-like radiologic pattern, whereas in the anterior compartment, spread results in a more longitudinal pattern (Fig. 38.5). Both spread patterns are consistent with adequate block.

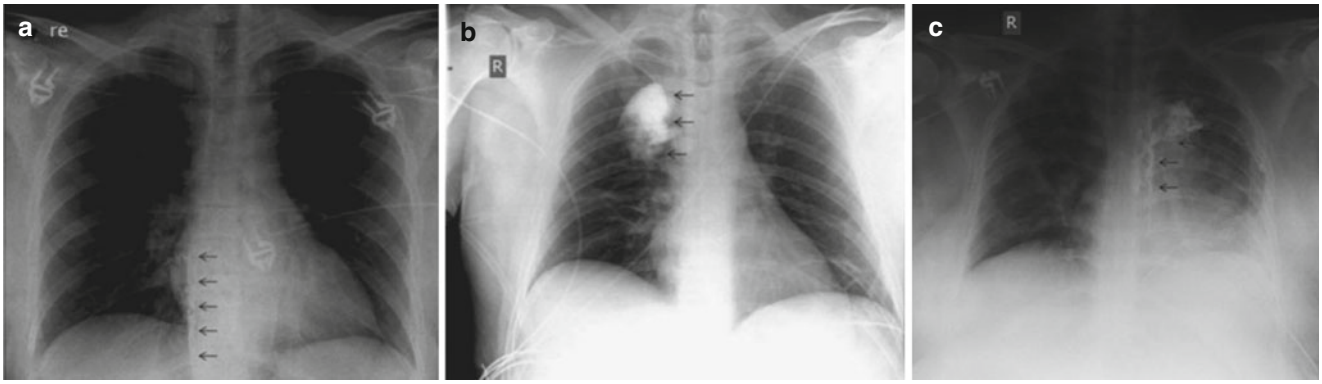
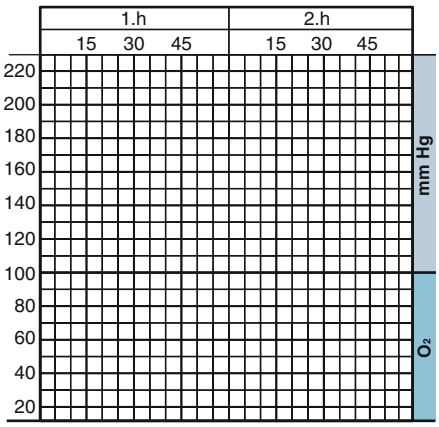
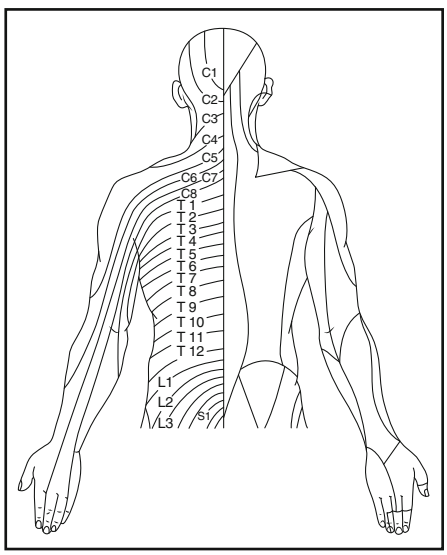


Fig. 38.5 Longitudinal (**a**), cloudlike (**b**), and combined longitudinal and cloudlike (**c**) spreading patterns confirm correct catheter placement in the thoracic paravertebral space. *Black arrows* indicate spread of radiopaque dye (Reproduced with permission from Renes et al. [3])

Record and checklist





With permission from Danilo Jankovic

Thoracic paravertebral nerve block

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic* ☐ *Surgical*

Needle: *G* _____ *Length* _____ *cm*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs)*: ☐ *Checked*

Patient: ☐ *Informed*

Technique: ☐ *Landmark–based* ☐ *In plane*
☐ *Ultrasound–guided* ☐ *Out of plane*
☐ *Transducer*
☐ *Linear*

Position: ☐ *Prone* ☐ *Sitting*

Injection level: ☐ *T* _____ _____ _____

Injection:

Local anesthetic: _____ *mL* _____ *%*
(in incremental doses)

Addition to LA: ☐ *Yes* _____ *µg/mg* ☐ *No*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* _____ *°C left* _____ *°C*

Segments affected: *T* _____

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge _____

Complications:

☐ *None* ☐ *Intravascular injection* ☐ *Subarachnoid/epidural*
☐ *Pneumothorax* ☐ *Other*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*
☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

_____

_____

_____

_____

References

1. Karmakar MK. Thoracic paravertebral block. Anesthesiology. 2001;95:771–80.
2. Hara K, Sakura S, Nomura T, Saito Y. Ultrasound guided thoracic paravertebral block in breast surgery. Anaesthesia. 2009;64:223–5.
3. Renes SH, Bruhn J, Gielen MJ, Scheffer GJ, van Geffen GJ. In-plane ultrasound-guided thoracic paravertebral block: a preliminary report of 36 cases with radiologic confirmation of catheter position. Reg Anesth Pain Med. 2010;35:212–6.
4. Richardson J, Lonnqvist PA, Naja Z. Bilateral thoracic paravertebral block: potential and practice. Br J Anaesth. 2011;106:164–71.

Chapter 39

Intercostal Nerve Block

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, M5T 2S8 ON, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_39,

Anatomy

There are 12 pairs of thoracic spinal nerves. With the exception of the first two, these spinal nerves are smaller in size compared with the lower half of the cervical nerves. These spinal nerves emerge from the spinal cord with two roots—the sensory dorsal root (posterior) and the motor ventral root (anterior) (Fig. 39.1). After leaving the dural sac, the two roots are surrounded by a dural sheath. The sensory root expands to accommodate the sensory neurons and forms the dorsal root ganglion. Beyond the ganglion, the roots form a common mixed spinal nerve trunk, which divides into four branches after exiting through the intervertebral foramen: the dorsal primary rami, the ventral primary rami, the meningeal branches which supply the spinal canal and the meninges, and the white and gray communicating branches, which anastomose with each neighboring ganglion of the sympathetic trunk and thus extend to the viscera and vessels, mediating and involving the sympathetic nervous system. They also carry sympathetic fibers to the spine.

The dorsal rami of the thoracic nerves pass between the two transverse processes to their area of distribution and divide into the two typical branches, the medial and lateral branches; they give off muscular branches (back muscles) and cutaneous branches (spinous processes, posterior wall of the thorax, and lumbar region). The ventral rami of the thoracic nerves are also termed intercostal nerves, and they are distributed segmentally (Fig. 39.2).

The 11 upper nerves are (relative to the thoracic ribs) genuinely intercostal because the nerves at least partially run in the intercostal space. The twelfth, however, lies caudal to the twelfth rib and is known as the subcostal nerve. The six upper intercostal nerves run entirely in the intercostal spaces, as far as the edge of the sternum; the six lower ones reach the area of the linea alba. All of the intercostal nerves, with the exception of the twelfth, run in the relevant intercostal space in front of the superior costotransverse ligament and on the inner surface of the external intercostal muscles.

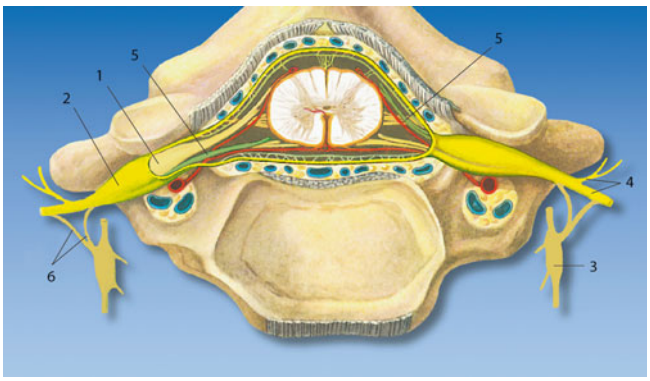


Fig. 39.1 Anatomy of the thoracic spinal nerves. (*1*) Spinal ganglion, (*2*) spinal nerve, (*3*) ganglion of the sympathetic trunk, (*4*) dorsal and ventral branch, (*5*) dorsal and ventral root, (*6*) white and gray communicating branches (With permission from Danilo Jankovic)

The internal intercostal muscles are absent from the spine far as the costal angle, replaced by internal intercostal membrane (Fig. 39.3). Over this area, the intercostal nerves lie over the endothoracic fascia and costal or parietal pleura. Approaching the angle of rib, the nerves lie between the internal intercostal muscles and the innermost intercostal muscles, and they are accompanied by the intercostal vessels (the intercostal artery and vein). They lie caudal to the vessels. Special care needs to be taken during procedures, as due to the proximity of blood vessels to the nerves, toxic concentrations of local anesthetic can easily be reached.

The intercostal nerves give out various branches: muscular branches for various chest wall muscles such as serratus posterior and rectus abdominis muscles; lateral cutaneous branches supplying the skin of lateral sides of the thorax and abdomen, as well as the skin of the axilla (first intercostal nerve); anterior cutaneous branches supplying the anterior side of the thorax; and pleural and peritoneal branches supplying the pleura and thoracic wall and the peritoneum of the lateral and anterior abdominal wall, as well as the pleural and peritoneal covering at the origin of the diaphragm.

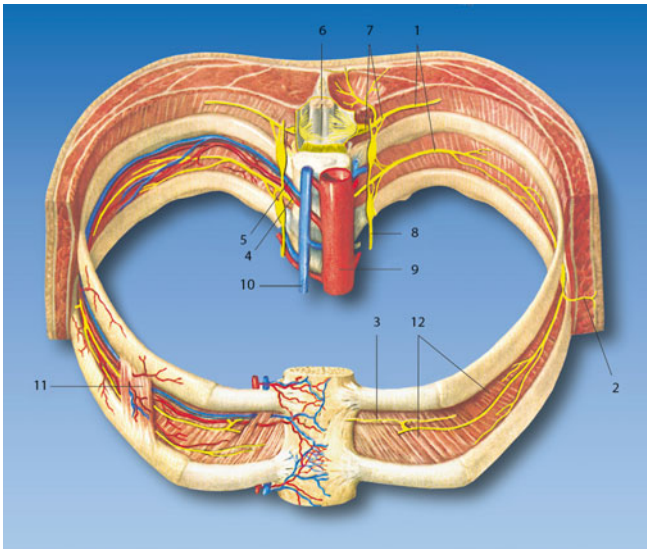


Fig. 39.2 Intercostal nerves. (*1*) Ventral branches (intercostal nerves), (*2*) lateral cutaneous branch, (*3*) anterior cutaneous branch, (*4*) posterior intercostal artery, (*5*) posterior intercostal vein, (*6*) spinal cord, (*7*) spinal nerve, (*8*) sympathetic trunk, (*9*) thoracic aorta, (*10*) azygos vein, (*11*) external intercostal muscles, (*12*) internal intercostal muscles (With permission from Danilo Jankovic)

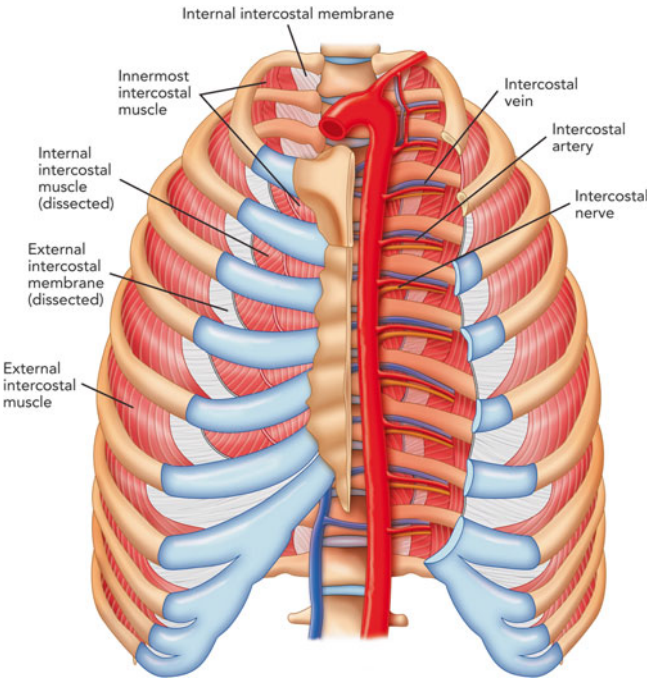


Fig. 39.3 Intercostal muscles in the chest wall (Reproduced with permission from Philip Peng Educational Series)

Sonoanatomy

The ideal location for ultrasound-guided injection is at the level of angle of rib because the internal intercostal muscle is formed from this point onward and the lateral cutaneous branch is still incorporated in the intercostal nerve.

The patient lies on the contralateral side or in prone position with arm abducted to bring the scapula away from midline (Fig. 39.4). A high-frequency ultrasound probe is used. The key landmarks are the upper and lower ribs of each intercostal space, the intercostal muscles, and the pleura. The probe is placed 90° to the course of the ribs so that upper and lower ribs, intercostal muscles, and pleura can be visualized. The target is the intercostal nerve deep to the internal intercostal muscle just caudal to the cranial rib and is usually less than 5 mm from the pleura (Fig. 39.5). To enhance the visualization of the internal intercostal muscle, the cranial end of the ultrasound probe is tilted laterally to align with the muscle (Fig. 39.6). If an in-plane approach is used, the transducer is positioned on the intercostal space, parallel to the upper and lower ribs. It is advisable to measure the distance between the skin and the pleura, and the needle should never be introduced more that this distance.

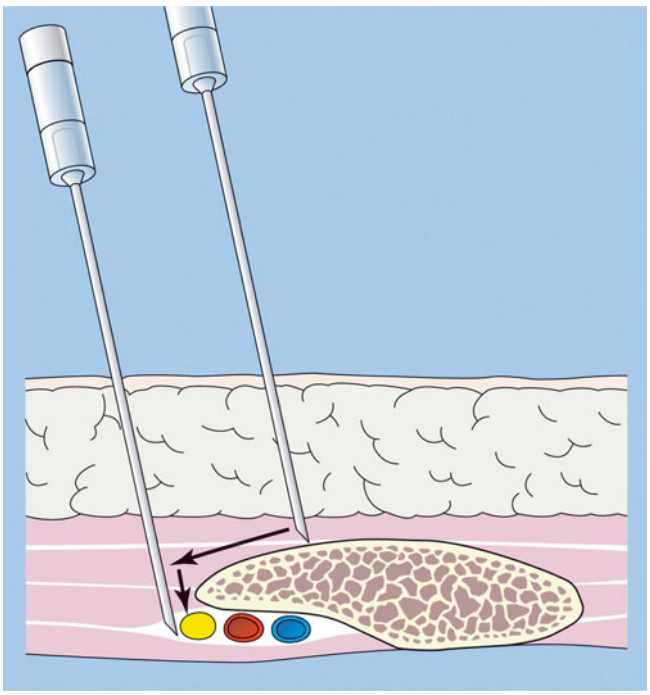


Fig. 39.5 The needle position with landmark technique (With permission from Danilo Jankovic)

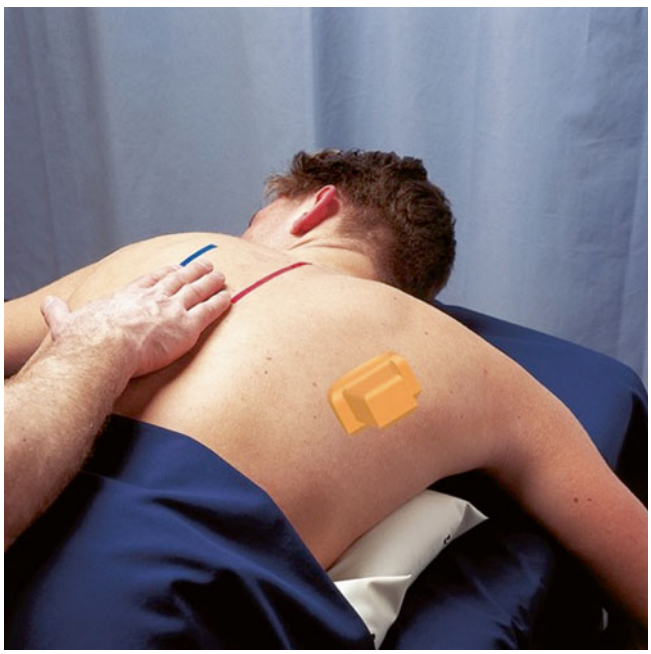


Fig. 39.4 Prone position. The *red line* indicates the midline and *blue* indicates line joining the angle of ribs. The ultrasound probe position is indicated on the *right side*

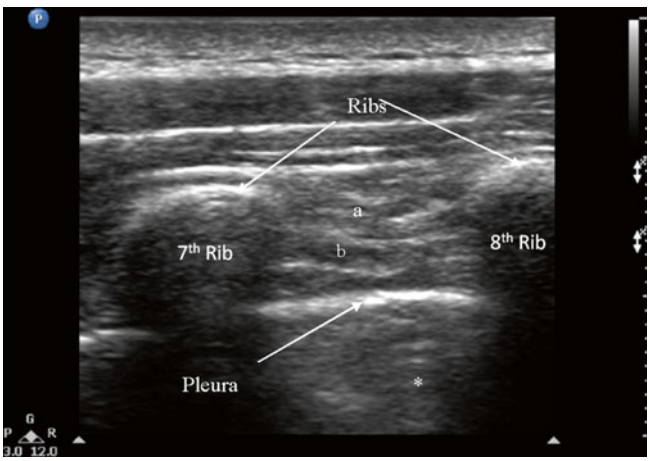


Fig. 39.6 Ultrasonographic image showing the intercostal muscles and pleura at the angle of rib. *a* external intercostal muscle, *b* internal intercostal muscle, * reverberation artifact. The pleura appears as a hyperechoic line (Reproduced with permission from Philip Peng Educational Series)

Indications

Surgical
1. Superficial surgery in the innervation area
2. Insertion of a chest drain

Diagnostic
1. Differential diagnosis of somatic and autonomic pain conditions

Therapeutic
1. Pain in the intercostal area (neuralgia and causalgia)
2. Pain after rib fractures or contusions of the thoracic wall and pleuritic pain
3. Acute phase of herpes zoster (in combination with a somatic paravertebral block)
4. Postoperative pain therapy after upper abdominal and thoracic surgery, to relieve muscular spasms and wound pain (to allow coughing and deep breathing)

In most of the indications mentioned, an epidural catheter procedure is preferable (see Chap. 41).

Contraindications

Specific
1. Anticoagulant treatment
2. Infections and skin diseases in the injection area

Relative
1. Slim patients
2. Chronic obstructive lung disease with poor lung reserve

Procedure

Preparations

Check that the emergency equipment is complete and in working order; sterile precautions, intravenous access, ECG monitoring, pulse oximetry, endotracheal anesthesia set, ventilation facilities, and emergency medication.

Procedure

Landmark Based

The first four intercostal nerves are blocked paravertebrally, 3.5–4 cm or two fingerbreadths lateral to the spinous processes (see Chap. 38).For the other level, the patient is placed in prone (preferred position for bilateral block) or lateral recumbent position (Fig. 39.7). When the procedure is performed in prone position, a pillow is placed under the mid-abdomen, between the arch of the ribs and the iliac crest line, with the patient's arms hanging (Fig. 39.4). The rib level is counted from the 12th rib onward cranially. The target is the caudal boundary of the rib at costal angle (7–8 cm or four fingerbreadths lateral to the midline and lateral to the musculature of the erector muscle of the spine).

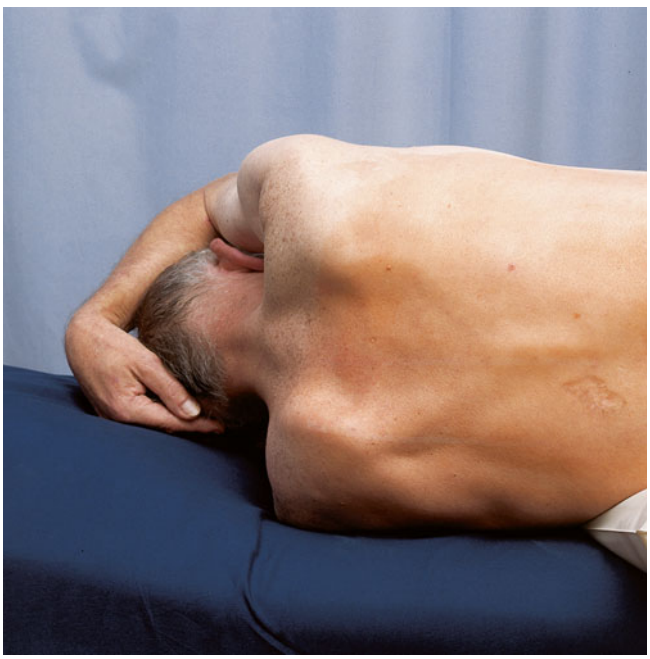


Fig. 39.7 Lateral position (With permission from Danilo Jankovic)

During the injection, the following points must be observed:

1. The person carrying out the injection must stand on the side being blocked.
2. The intercostal nerve runs dorsocaudal to the vessels in the inferior costal groove.
3. The rib is ca. 0.6–0.7 cm thick.
4. Start with the lowest rib.
5. The injection must only be carried out after definite identification of the rib being blocked.
6. Targeted paresthesias are not elicited.
7. If the patient coughs, it indicates pleural irritation. The procedure should be halted.
8. Injection should be carried out on an incremental basis, with frequent aspiration.

Using the index and middle fingers of the left hand, palpate the rib being blocked, and press the skin around the contours of the ribs. The index finger locates the lower edge of the rib. A 25-G 3.5-cm-long needle is advanced at an angle of 80° to the skin surface until bone contact (costal periosteum) is made. The needle is withdrawn slightly, and the skin and needle are then simultaneously pushed caudally until the needle slides under the lower edge of the rib (Fig. 39.8). After loss of bone contact, the needle must only be introduced 2–3 mm deeper. The hub of the needle is fixed between the thumb and index finger as this is done; the middle finger fixes the shaft and directs the needle. The side of the left hand (left hypothenar eminence) rests on the patient's back; initially it serves as a brake, and then during the injection it serves as fixation. After aspiration at various levels, the local anesthetic is injected on an incremental basis.

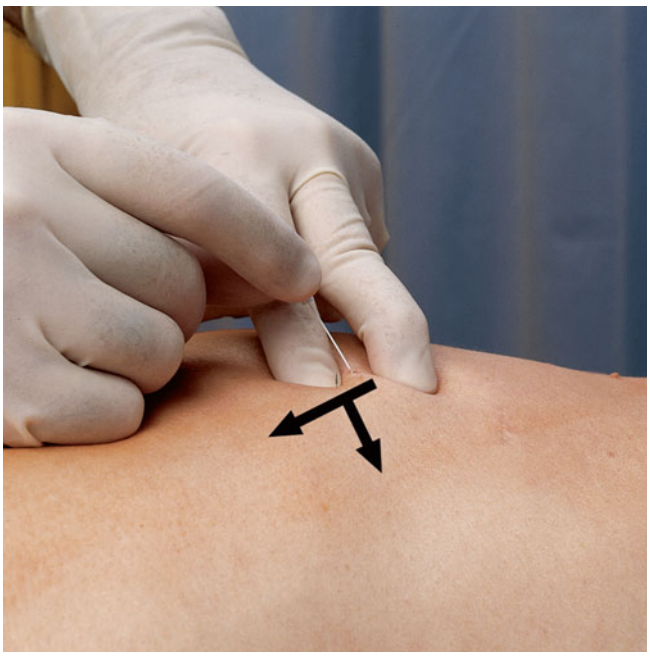


Fig. 39.8 The skin and the needle are simultaneously pushed caudally, and the needle is then introduced a further 2–3 mm (With permission from Danilo Jankovic)

Due to overlap, at least three nerves have to be blocked in order to achieve a complete segmental block. A volume of 3–5 mL local anesthetic is injected per segment, e.g., 0.5–0.75 % ropivacaine and 0.25–0.5 % bupivacaine (0.25–0.5 % levobupivacaine). As intercostal block is the procedure in which the highest blood levels of local anesthetic per milligram injected are achieved (due to very fast absorption), there is a risk of overdose. The individual maximum dose must be carefully calculated and must never be exceeded.

Ultrasound-Guided Injection

The preferred position for ultrasound-guided technique is prone position for two reasons: it is easier to count the level of ribs in this position, and it is the preferred position to diagnose the presence of pneumothorax as air tends to stay in the nondependent position. These two are also the advantages of the use of ultrasound over the landmark-guided technique. In addition, ultrasound guidance allows the target and the needle to be visualized, as well as the confirmation of the spread of injectate, avoiding inadvertent vascular injection.

With out-of-plane technique, the ultrasound probe is placed in short axis to the two ribs of the intercostal space. The target is the intercostal nerve deep to the internal intercostal muscle just caudal to the cranial rib and is usually less than 5 mm from the pleura. The ultrasound probe should be positioned so that this target is in the center of view in the ultrasound screen. A 25-G 3.5 cm needle is inserted out-of-plane in an angle almost parallel to the midpoint of the ultrasound probe. The hand holding the ultrasound probe should adjust the tilting of the probe constantly to follow the needle tip. When the needle is in the external intercostal muscle (Fig. 39.9a), the position of the needle tip should be confirmed with hydrolocation technique by injecting a small amount of local anesthetic (0.2–0.4 mL). The needle is then carefully advanced to the target. If the tip is in the right location, the pleura is seen pushed down by the spread of injectate (Fig. 39.9b). A volume of 2 mL is sufficient, in contrast to the landmark-guided technique.

Following the injection of various levels, it is advisable to perform a scan of the pleura to document the absence of pneumothorax. The feature to look for is the pleura which is a thin but densely hyperechoic structure gliding freely with reverberation and comet-tail artifacts (Fig. 39.6). Combining the signs of absent lung sliding and the loss of comet-tail artifact, ultrasound has a reported sensitivity of 100 %, specificity of 96.5 %, and negative predictive value of 100 %. The other sign to look for is the seashore sign in the M mode (Fig. 39.10). In the presence of pneumothorax, the differentiation of "sea" and "shore" disappear and the picture will look like barcode (barcode sign).

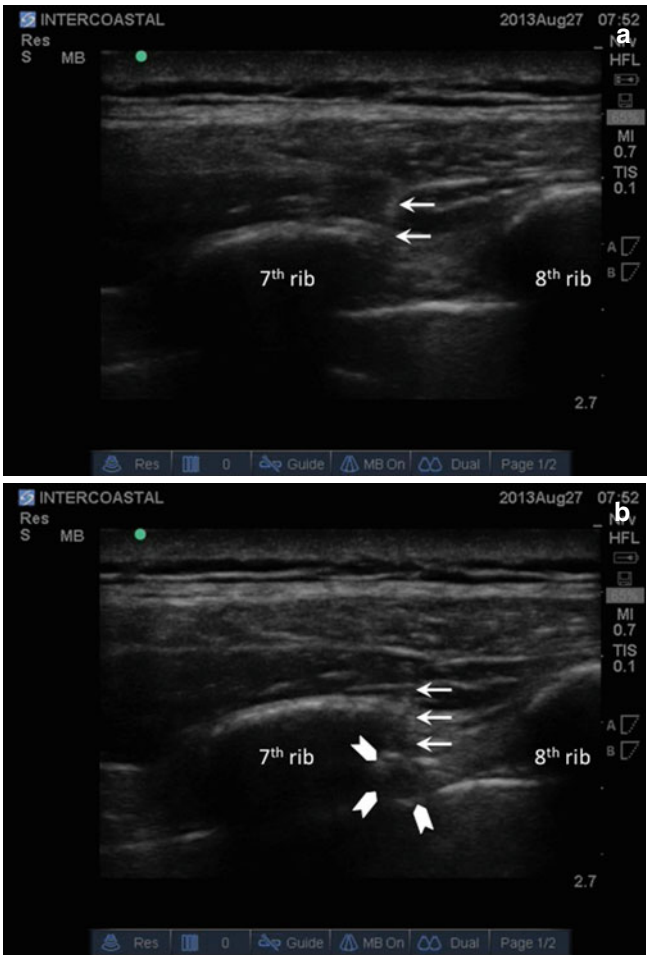


Fig. 39.9 (**a**) The *arrows* indicate the needle position inserted with out-of-plane technique. It indicates the needle tip is in external intercostal muscle. (**b**) Further insertion of needle result in the tip just deep to the internal intercostal muscle. Injection of local anesthetic will result in the appearance of pooling of local anesthetic, which pushes the pleura deeper (Reproduced with permission from Philip Peng Educational Series)

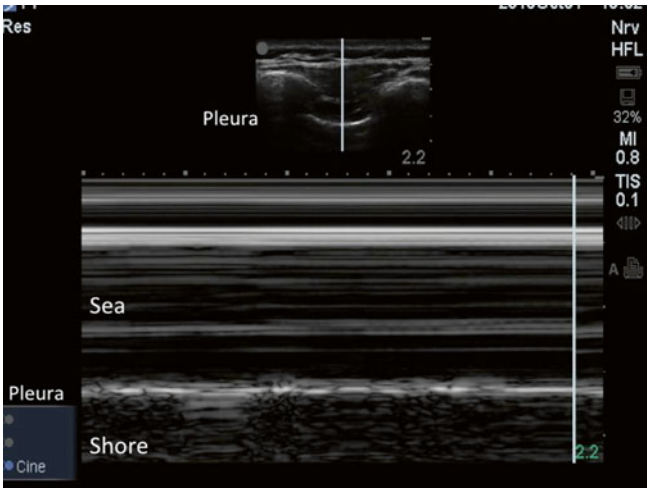


Fig. 39.10 The M mode of the pleura. It shows seashore sign. Sea is a result of the parallel lines depicting the layers of tissue above the pleura (Reproduced with permission from Philip Peng Educational Series)

Effects of the Block

The anesthesia effect of the block should cover the skin, lateral and anterior thoracic wall, motor block of the intercostal muscles, as well as of the pleura and thoracic wall. The six lower intercostal nerves reach the area of the linea alba, leading to motor block of the abdominal musculature and sensory block of the skin and abdomen, as well as of the peritoneum and lateral and anterior thoracic walls.

Complications

1. Pneumothorax
2. Toxic reactions due to overdosage (see Chap. 1)
3. Intravascular injection (see Chap. 1)

Suggested Reading

1. Brown DL. Intercostal block. In: Brown DL, editor. Atlas of regional anesthesia. Philadelphia: W.B. Saunders Company; 1992. p. 211–7.
2. Vlassakov KV, Narang S, Kissin I. Local anesthetic blockade of peripheral nerves for treatment of neuralgias: systematic analysis. Anesth Analg. 2011;112:1487–93.
3. Curatolo M, Bogduk N. Diagnostic and therapeutic nerve blocks. In: Fishman SM, Ballantyne JC, Rathmell JP, editors. Bonica's management of pain. Philadelphia: Lippincott William & Wilkins; 2010. p. 1401–23.
4. Curatolo M, Bogduk N. Diagnostic blocks for chronic pain. Scand J Pain. 2010;1:186–92.
5. Shankar H, Eastwood D. Retrospective comparison of ultrasound and fluoroscopic image guidance for intercostal steroid injections. Pain Pract. 2010;10:312–7.
6. Peng P, Narouze S. Ultrasound-guided interventional procedures in pain Medicine: A review of anatomy, sonoanatomy and procedures. Part I: non-axial structures. Reg Anesth Pain Med. 2009; 34:458–74.
7. Curatolo M. Intercostal nerve block. In: Peng PW, editor. Ultrasound for pain medicine intervention: a practical guide, Peripheral structures. Philip Peng educational series, vol. 1. 1st ed. Cupertino: iBook, Apple Inc; 2013. p. 65–8.
8. Katz J, Renck H. Thorakale paravertebrale Blockade; Interkostale Nervenblockade. In: Katz J, Renck H, editors. Thorakoabdominale Nervenblockaden. Weinheim: Edition Medizin; 1988.
9. Kirvelä O, Antila H. Thoracic paravertebral block in chronic postoperative pain. Reg Anesth. 1992;17:348–50.
10. Klein SM, Greengrass RA, Weltz C, Warner DS. Paravertebral somatic nerve block for outpatient inguinal herniorrhaphy: an expanded case report of 22 patients. Reg Anesth Pain Med. 1998;23(3):306–10.
11. Netter FH. Nervengeflechte und periphere Nerven. In: Krämer G, editor. Farbatlanten der Medizin (Band 5). Nervensystem I: Neuroanatomie und Physiologie. Stuttgart/New York: Georg Thieme Verlag; 1987.
12. Jankovic D. Thoracic spinal nerve blocks. In: Jankovic D, editor. Regional nerve blocks &infiltration therapy. 3rd ed. Malden Masachussets, Oxford, Carlton Victoria: Blackwell; 2004. p. 171–85.

Part VIII
Lumbosacral Spine

Chapter 40

Neuraxial Anatomy and Sonoanatomy

Danilo Jankovic, MD (✉) (Anatomy)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Ki Jinn Chin, MBBS, MMed., FANZCA, FRCPC (✉) (Sonoanatomy)
Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, Canada
e-mail: kijinn.chin@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_40,

Spine and Sacrum Spine

The spinal column consists of 33 vertebrae—seven cervical vertebrae; 12 thoracic vertebrae; five lumbar vertebrae; the sacrum, consisting of five fused sacral vertebrae; and the coccyx, consisting of four fused coccygeal segments (Fig. 40.1a–c).

According to research findings, the mean length of the spinal column, from the foramen magnum to the tip of the coccyx, is 73.6 cm (range 67.4–78.8 cm), while in women, it is 7–10 cm shorter [9].

All of the vertebrae have the same basic shape, which is subject to certain variations in the individual sections of the spine. The basic shape consists of an anterior body (the body of the vertebra) and a dorsal arch (the vertebral arch), which consists of pedicles and laminae (Fig. 40.2).

The laminae of the vertebral arch join dorsally to form the spinous process. A transverse process branches off on each side of the vertebral arch, as well as a superior and an inferior articular process. The vertebrae in the cervical region are smaller, but their size increases from cranial to caudal. The angle of inclination of the spinous processes—important topographic signposts for neuraxial injections—varies at different levels of the spine.

The cervical spinous processes, the first two thoracic spinous processes, and the lumbar spinous processes lie at the same level as their vertebrae. From T3 to L1, the spinous processes are angled caudally (particularly in the T4–T9 area) (Fig. 40.3a–c).

The vertebral canal (which provides excellent protection for the spinal cord) and the spinal cord, with its meningeal covering, extend throughout the whole length of the spine terminating in the cauda equina. The spinal vessels and nerves emerge laterally through openings at the upper and lower margins of the roots of the arches of the adjoining vertebrae (the intervertebral foramina).

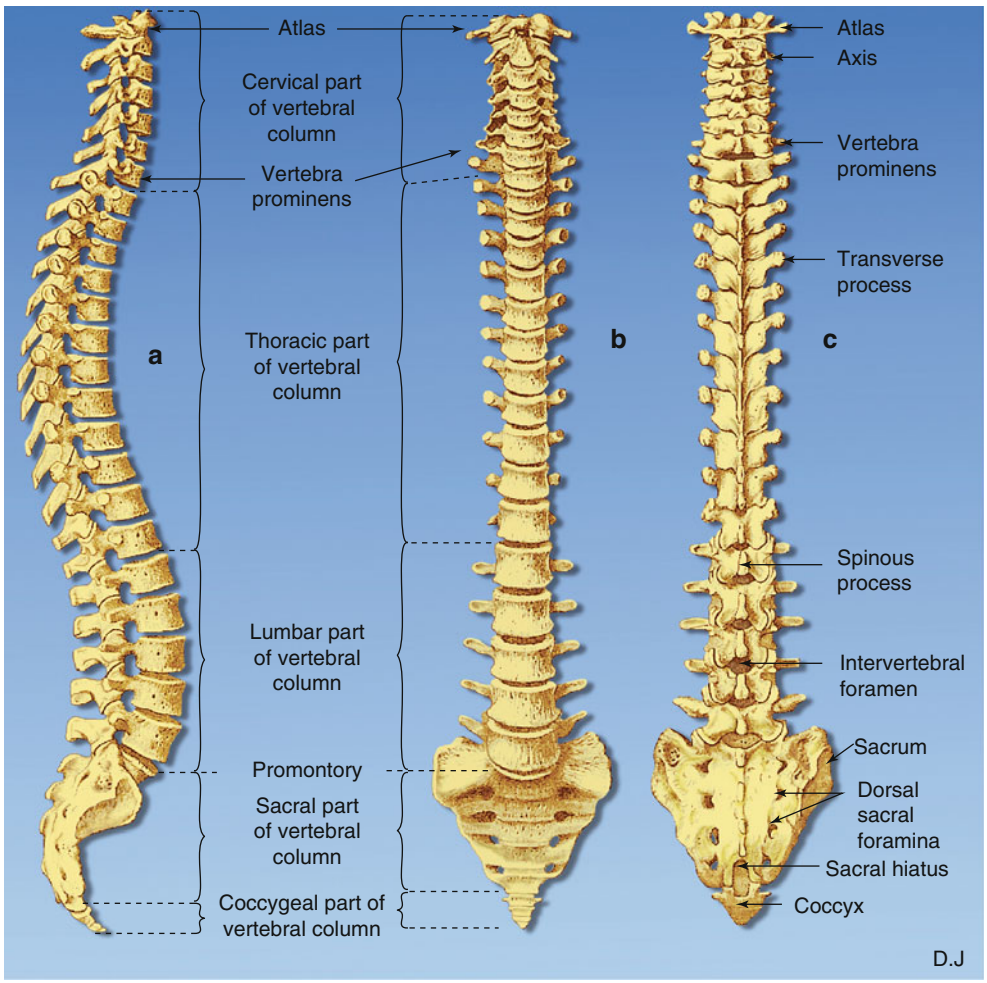


Fig. 40.1 (**a–c**) Spine. (**a**) Lateral, (**b**) **v**entral, (**c**) dorsal (Reproduced with permission from Danilo Jankovic)

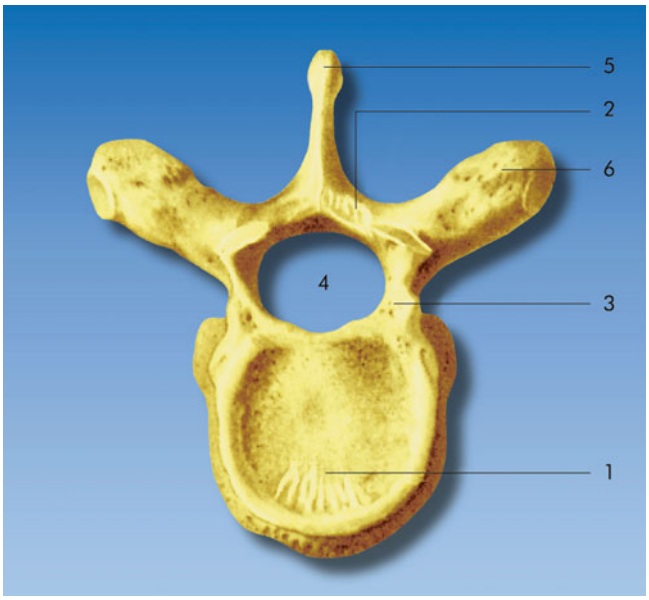


Fig. 40.2 Basic shape of a vertebra. (*1*) Vertebral body, (*2*) vertebral arch, (*3*) pedicle of the vertebral arch, (*4*) vertebral foramen, (*5*) spinous process, (*6*) transverse process (Reproduced with permission from Danilo Jankovic)

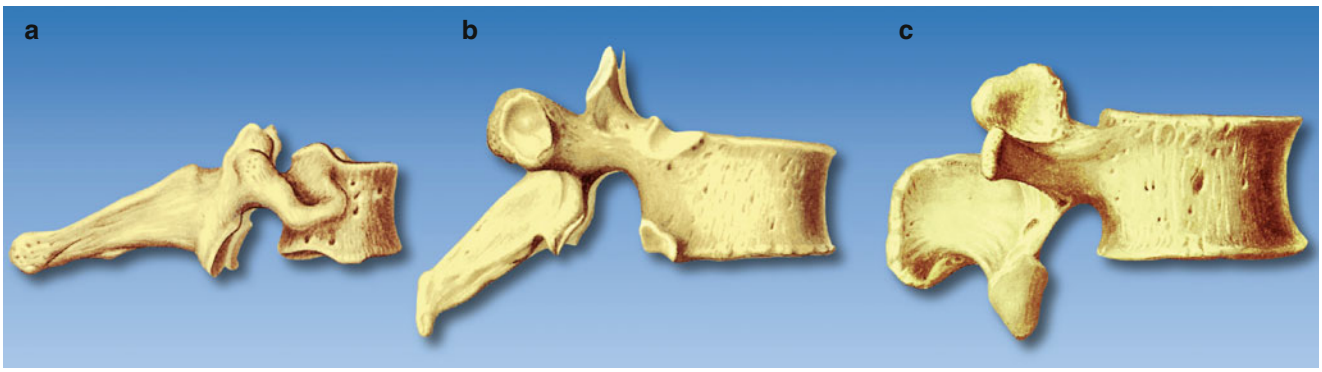


Fig. 40.3 (**a**–**c**) Cervical, thoracic, and lumbar spinous processes. (**a**) C7 cervical vertebra (vertebra prominens, nuchal tubercle). (**b**) T8 thoracic vertebra. (**c**) L3 lumbar vertebra (Reproduced with permission from Danilo Jankovic)

Sacrum

The sacrum is wedge shaped and consists of five vertebrae fused together. It lies distal to the fifth lumbar vertebra and is connected distally, at its apex, to the coccyx. Dorsally, the sacrum has a convex surface, in the middle of which the median sacral crest stands out (Fig. 40.4a).

The crest is produced by the fusion of the rudimentary spinous processes of the upper third or fourth sacral vertebrae. Normally, the arch of the fifth and occasionally also of the fourth sacral vertebra is absent, so that there is a sacral hiatus at this point. The hiatus is bounded by the sacral horn as a remnant of the caudal articular process, and it is used as a passage by the five small sacral nerves and by the coccygeal nerves. Between the median sacral crest and the lateral sacral crest lie the four sacral openings (the posterior sacral foramina), through which the dorsal branches of the sacral spinal nerves emerge. The anterior view shows a concave aspect. Alongside the transverse lines (fused vertebrae), there are large anterior openings (the anterior pelvic sacral foramina), through which the primary anterior parts of the sacral nerves emerge (Fig. 40.4b).

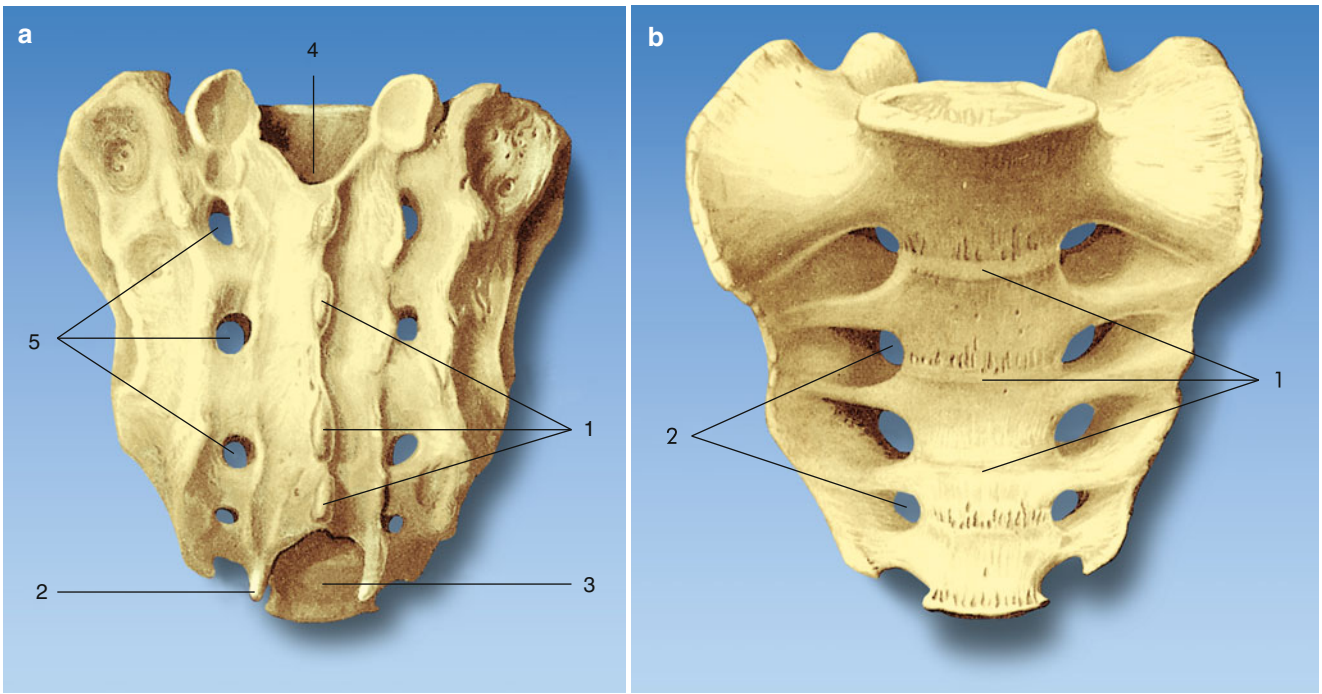


Fig. 40.4 (**a**) Sacrum (dorsal view). (*1*) Median sacral crest, (*2*) sacral horn, (*3*) sacral hiatus, (*4*) sacral canal, (*5*) posterior sacral foramina (Reproduced with permission from Danilo Jankovic). (**b**) Sacrum (ventral view). (*1*) Transverse lines, (*2*) anterior pelvic sacral foramina (With permission from Danilo Jankovic)

Spinal Ligaments

The vertebrae are supported from the axis to the cranial sacrum by intervertebral disks and by various ligaments (Fig. 40.5a, b). The intervertebral disks lie between neighboring vertebrae and function as fixed connecting elements and pressure-absorbing buffers. The disks are at their thinnest in the area of T3–T7 and thickest in the lumbar area.

The anterior longitudinal ligament (Figs. 40.6 and 40.7) is attached at the anterior edge of the vertebral bodies and intervertebral disks and is at its thickest in the thoracic area. The posterior longitudinal ligament (Figs. 40.6 and 40.7) is wider cranially than it is caudally, and it lies behind the vertebral bodies in the medullary canal. The supraspinous ligaments extend as far as the sacrum along the tips of the spinous processes, with which they are connected, and continue cranially in the nuchal ligament and caudally in the interspinous ligament. They become thicker from cranial to caudal. The interspinous ligaments connect the roots and tips of the spinous processes.

The intertransverse ligaments serve to connect the transverse processes (Fig. 40.5b).

The ligamentum flavum largely consists of yellow, elastic fibers and it connects the neighboring laminae (Figs. 40.6 and 40.7). It is at its thinnest in the midline (small fissure spaces exist for the veins running from the internal vertebral venous plexus to the external vertebral venous plexus), and its thickness increases laterally. The size and shape of the ligamentum flavum vary at the various levels of the spine. Caudally, for example, it is thicker than in the cranial direction.

There are usually only 22 genuine ligamenta flava, as they do not develop between the atlas and occiput or between the atlas and the axis. The mean length of the ligamentum flavum is 0.84 cm in the cervical spine, 1.03 cm in the thoracic spine, and 1.61 cm in the lumbar spine. The mean width is 2.30 cm in the cervical region, 2.0 cm in the thoracic region, and 3.22 cm in the lumbar region. The ligamenta flava have a mean thickness of 1.37 mm in the cervical spine, 1.75 mm in the thoracic spine, and 2.55 mm in the lumbar spine. According to Yong-Hing et al. (1976), the human ligamentum flavum consists of 50–80 % elastin and 20–50 % collagen [9].

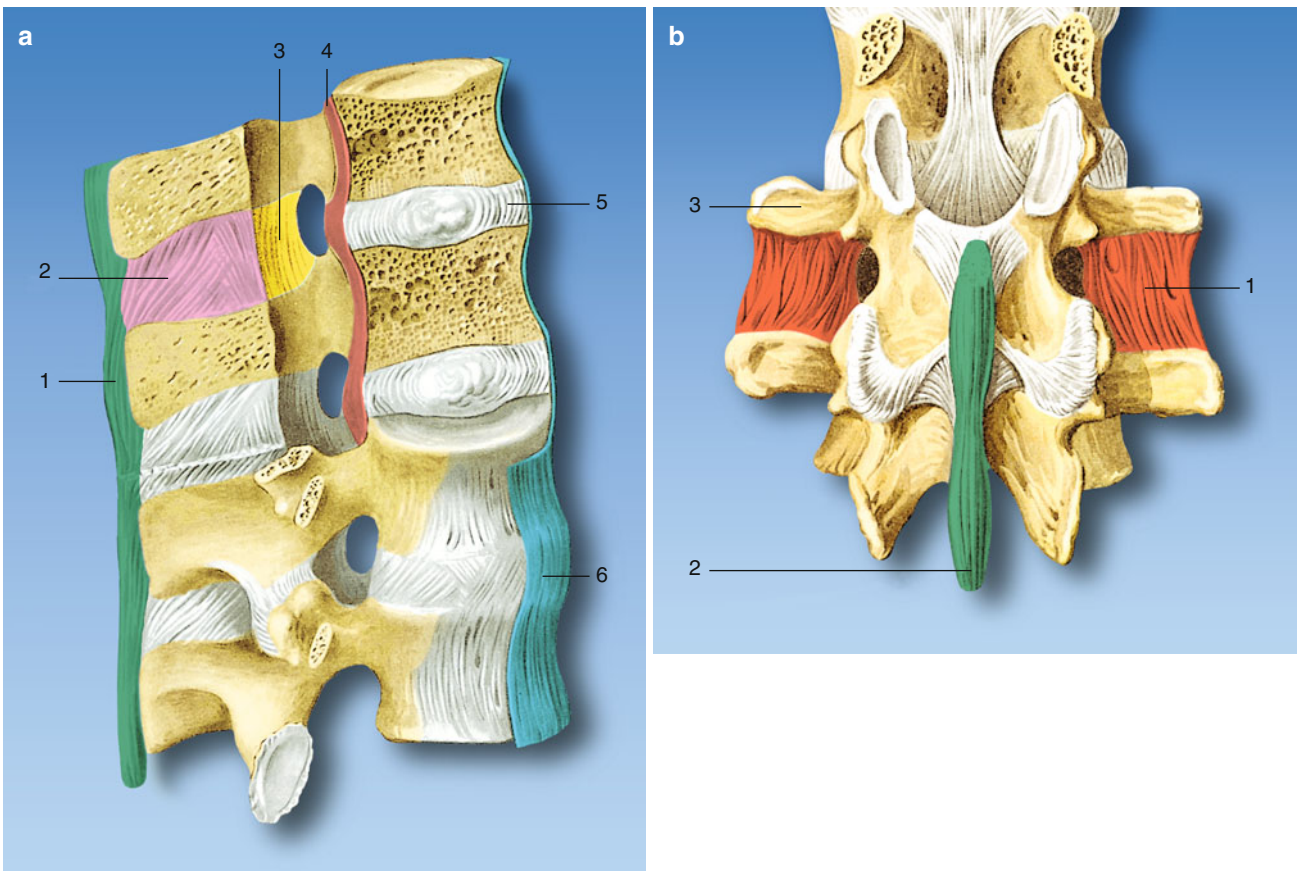


Fig. 40.5 (**a**) Ligaments of the spinal cord. (*1*) Supraspinous ligament, (*2*) interspinous ligament, (*3*) ligamentum flavum, (*4*) posterior longitudinal ligament, (*5*) intervertebral disk, (*6*) anterior longitudinal ligament (with permission from Danilo Jankovic). (**b**) Ligaments of the spinal cord. (*1*) Intertransverse ligament, (*2*) supraspinous ligament, (*3*) transverse process (With permission from Danilo Jankovic)

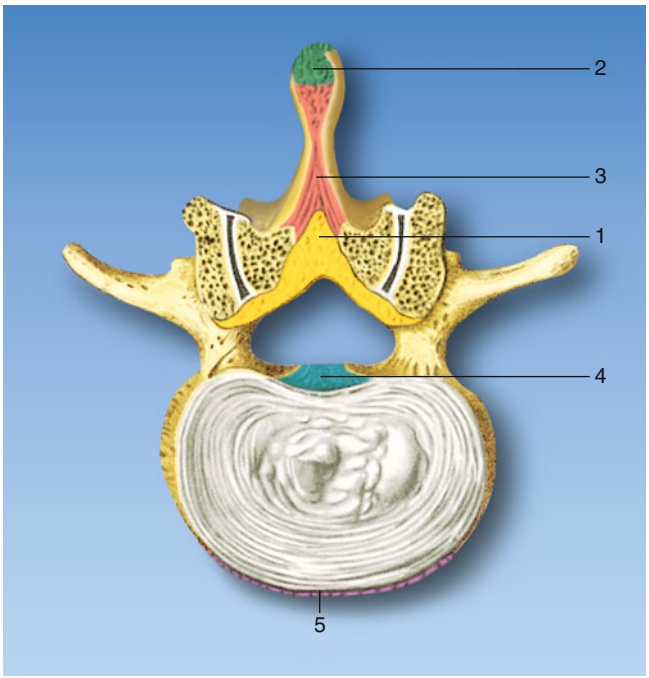


Fig. 40.6 Ligaments of the spinal cord. (*1*) Ligamentum flavum, (*2*) supraspinous ligament, (*3*) interspinous ligament, (*4*) posterior longitudinal ligament, (*5*) anterior longitudinal ligament (With permission from Danilo Jankovic)

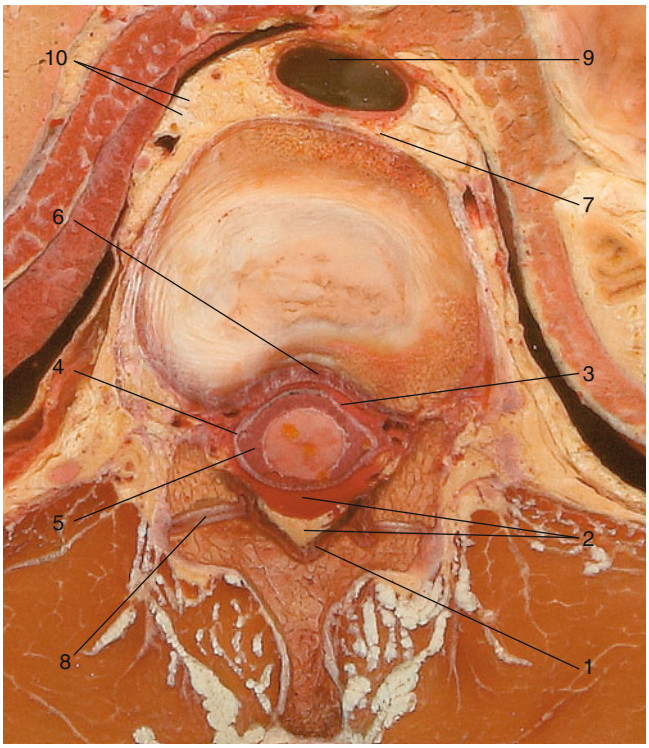


Fig. 40.7 Transverse section through the thorax at the level of T9. (*1*) Ligamentum flavum, (*2*) posterior epidural space, (*3*) anterior epidural space, (*4*) spinal dura mater, (*5*) subarachnoid space and spinal cord, (*6*) posterior longitudinal ligament, (*7*) anterior longitudinal ligament, (*8*) zygapophyseal joint, (*9*) aorta, (*10*) sympathetic trunk ganglion (With permission from Danilo Jankovic)

Iliolumbosacral Ligaments (Sacroiliac Joints)

The stability of the iliolumbosacral region is ensured by lumbosacral and sacroiliac connections that transfer the entire weight of the trunk via the hip bones to the lower extremities. These ligamentous connections serve to connect the vertebrae with one another and to stabilize the sacrum.

Clinically important ligaments: interspinous, supraspinous, iliolumbar, interosseous sacroiliac, sacrospinous, and sacrotuberous ligaments (Fig. 40.8a, b).

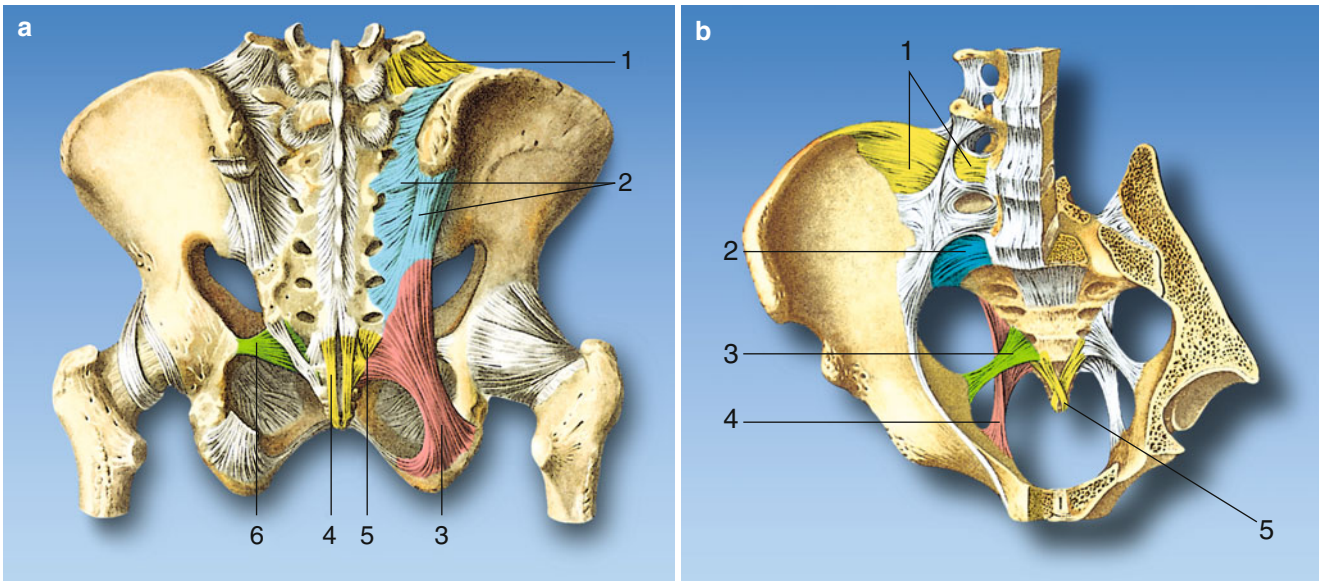


Fig. 40.8 (**a**) Iliolumbosacral ligaments (dorsal view). (*1*) Iliolumbar ligament, (*2*) dorsal sacroiliac ligament, (*3*) sacrotuberous ligament, (*4*) superficial dorsal and deep dorsal sacrococcygeal ligaments, (*5*) lateral sacrococcygeal ligament, (*6*) sacrospinous ligament (with permission from Danilo Jankovic). (**b**) Iliolumbosacral ligaments (ventral view). (*1*) Iliolumbar ligament, (*2*) ventral sacroiliac ligament, (*3*) sacrospinous ligament, (*4*) sacrotuberous ligament, (*5*) ventral sacrococcygeal ligament (With permission from Danilo Jankovic)

Spinal Cord

The mean length of the spinal cord from the foramen magnum downward is 45.9 cm in men and 41.5 cm in women [8, 9]. The conus medullaris continues in the threadlike median filum terminale as far as the posterior side of the coccyx (Figs. 40.7, 40.9a–c, and 40.13).

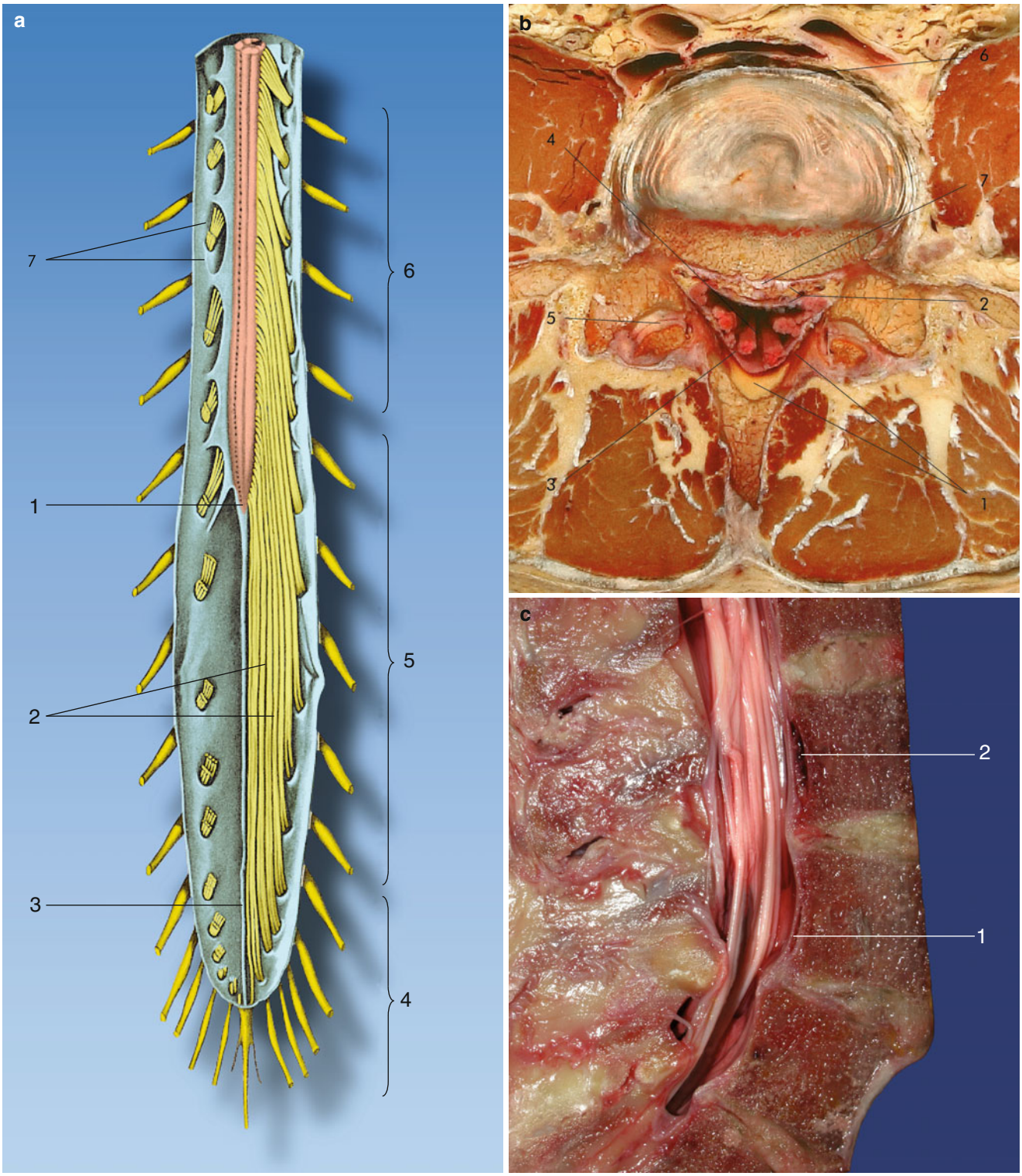


Fig. 40.9 (**a**) Spinal cord (lower half). (*1*) Conus medullaris, (*2*) cauda equina, (*3*) filum of spinal dura mater (filum terminale), (*4*) sacral nerves, (*5*) lumbar nerves, (*6*) thoracic nerves, (*7*) dura mater (With permission from Danilo Jankovic). (**b**) Transverse section at the level of L4/5. (*1*) Posterior epidural space with fat, (*2*) anterior epidural space with veins, (*3*) spinal dura mater, (*4*) subarachnoid space and cauda equina, (*5*) zygapophyseal joint, (*6*) anterior longitudinal ligament, (*7*) posterior longitudinal ligament (With permission from Danilo Jankovic). (**c**) The cauda equina at the level of L2–5. Lateral view. (*1*) Spinal dura mater, (*2*), epidural space (With permission from Danilo Jankovic)

Rootlets: Cauda Equina [8, 9]

The cauda equina is generally regarded as consisting of the thick, horsetail-shaped fascicle of nerve fibers in the lower part of the dural sac, containing the paired rootlets of the lowest thoracic and entire lumbar and coccygeal medulla. All of the motor and sensory fibers of the lumbosacral plexus, pudendal nerve, and coccygeal nerve course inside the lumbosacral subarachnoid space. A constant increase in the thickness of the root fascicle is seen between the L1 and L3 segments. The terminal filum of the spinal cord is approximately 153 mm long (range 123–178 mm) (Figs. 40.9b, 40.11, 40.13, and 40.14).

The dura mater and arachnoid, and consequently the subarachnoid space as well, extend downward as far as the level of the second sacral vertebra.

Meninges

The spinal cord is surrounded and protected by the meninges (the dura mater, arachnoid mater, and pia mater) and by cerebrospinal fluid, epidural fatty tissue, and veins (Figs. 40.10 and 40.11).

The meninges form a connected, unified organ with important protective functions for the brain and spinal cord; they provide mechanical, immunological, and thermal protection and are also important for metabolism in the CNS. The mechanical function of the meninges consists of providing fixation for the brain in the cranium and for the spinal cord in the vertebral canal, as well as in forming a highly adaptable fluid mantle that functions like a water bed. The fixation is provided by the dura mater.

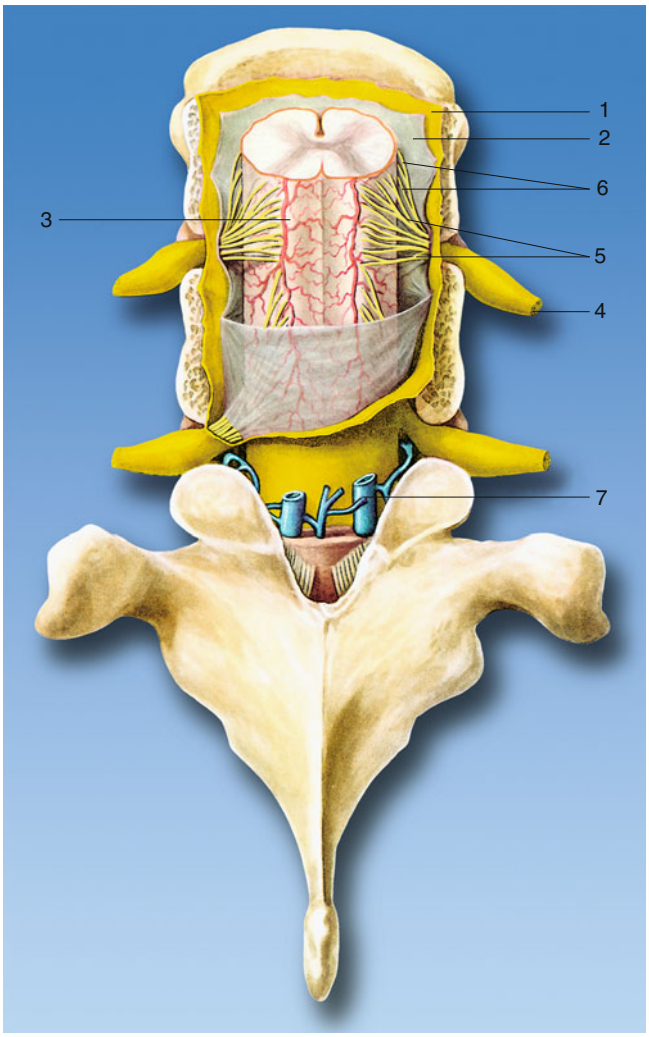


Fig. 40.10 Meninges. (*1*) Dura mater, (*2*) arachnoid mater, (*3*) pia mater, (*4*) spinal nerve, (*5*) dorsal (posterior) root, (*6*) ventral (anterior) root, (*7*) internal vertebral venous plexus (With permission from Danilo Jankovic)

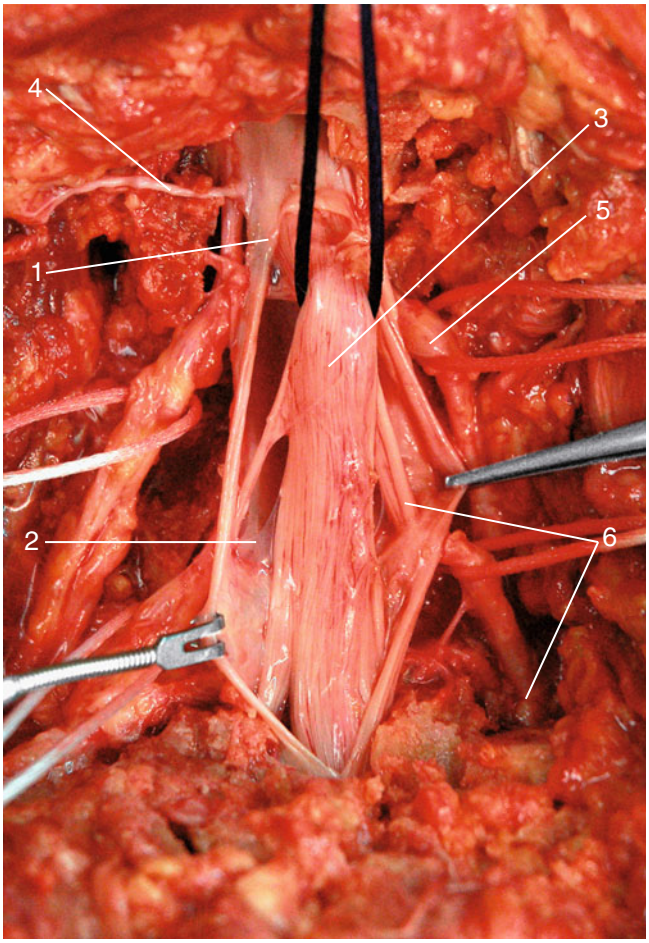


Fig. 40.11 A lumbar vertebral canal opened from posterior in the area of L3 and L4. The cauda equina has been exposed by partial resection of the spinal dura mater and arachnoid mater. (*1*) Spinal dura mater, (*2*) arachnoid mater, (*3*) with posterior and anterior roots, (*4*) dorsal root, (*5*) spinal ganglion, (*6*) posterior and anterior spinal branches (With permission from Danilo Jankovic)

Dura Mater of the Spinal Cord (Figs. 40.7, 40.10, 40.11, 40.12, 40.13, and 40.14)

The dura mater of the spinal cord, a fibroelastic membrane, extends as far as the second sacral vertebra, where it ends in a blind sac. It encloses the anterior and posterior spinal nerve roots.

It surrounds the anterior and posterior spinal nerve roots. It consists of collagenous fibers and a few elastic fibers; it is 0.1–0.5 mm thick and is usually thinner anteriorly than it is posteriorly. The dural sac is often enlarged in the area between L4 and S1 and also between T12 and L1 (Spischarny's terminal cystoma). The thickness of the spinal dura mater declines from medial to lateral. The dura mater is often thinner ventrally than dorsally, and it sends off leaves and fibers into the epidural space. It shows notable foliation near the intervertebral foramina, where it passes on the one hand into the epineurium of the peripheral nerves and into the capsule of the spinal ganglia and on the other hand radiates into the cavernous body of the vascular plexus characterizing this area. One of the major characteristics of the dura mater is its extremely marked vascularization. Particularly in the boundary zone with the arachnoid, numerous capillaries and venules are fenestrated, promoting the exchange of various metabolites and cells between the blood and dura. The vascularization increases strongly in the lateral areas, particularly in the area of the dural infundibulum. The dural vessels are connected with the epidural vascular plexus and thus with the internal and external vertebral vein plexus as well. There are numerous nerve fibers in close proximity to the dural vessels [6, 12]. In terms of size, C fibers predominate. In terms of function, the dural nerves consist of sympathetic, parasympathetic, and sensory fibers. A whole range of neurotransmitters and neuropeptides have been identified in the dural nerves, such as acetylcholine, serotonin, substance P, calcitonin gene-related peptide (CGRP), neuropeptide Y, vasoactive intestinal polypeptide (VIP), etc. [3, 7]. In addition to vasoactive nerves, local factors also play a role in the regulation of dural perfusion, such as adenosine, which has been found abundantly in the dura [4]. Via a multilayered cell cluster known as the subdural nerve papilla (neurothele) [1, 5], the arachnoid membrane is directly connected with the innermost lamellae of the dura. A "subdural fissure"—often present in postmortem and only arising during life in specific circumstances—must be regarded as an artifact.

Between the dura mater and the arachnoid, there is a space, the subdural space, in which a small amount of lymph-like fluid is located.

The arachnoid mater (Figs. 40.10 and 40.11), a nonvascularized membrane, also ends at the level of the second sacral vertebra. Between the arachnoid mater and the pia mater lies the subarachnoid space, which is filled with cerebrospinal fluid (see the section "Cerebrospinal fluid", below). The expansion forces present here reduce the weight of the brain "floating" in the cerebrospinal fluid to just under 50 g.

This protects the brain from harmful acceleration forces.

The same applies to the spinal cord. There is a substantial amount of exchange between the cranial and spinal cerebrospinal fluid. Magnetic resonance studies have confirmed Du Boulay's older view that there are fluid waves synchronous with cardiac systole that move the cerebrospinal fluid from the basal cisterns into the cervical subarachnoid space [10]; respiration-synchronous fluid movements are superimposed on these [11]. The spinal subarachnoid space, with the extremely elastic spinal dura and adaptable arachnoid, makes it possible to absorb these rhythmic fluid waves (compliance). Artificial losses of cerebrospinal fluid undoubtedly have severe effects on the system's natural mechanism and reduce the water-bed function of the exterior subarachnoid space. The subarachnoid space is important for the thermoregulation of the CNS, as it acts as a temperature buffer [13] both against excessive external temperatures and also against functional or pathological increases in temperature in the CNS. The normal basic temperature of the cerebrospinal fluid is approximately 37 °C. It is important to note that the cerebrospinal fluid does not represent "standing water" but is instead constantly in movement and continually being freshly produced, mainly in the choroid plexus. It is reabsorbed over a wide surface, not only via the arachnoid granulations or villi in the area of the nerve exit sites but also diffusely through the arachnoid into the dural vascular system and via the pia into the vessels of the pial vascular network [12]. The choroid plexus is not only a production organ but also a site for reabsorption of cerebrospinal fluid.

The spinal pia mater (Figs. 40.10 and 40.13) is a thin, very well-vascularized membrane that tightly encloses the spinal cord.

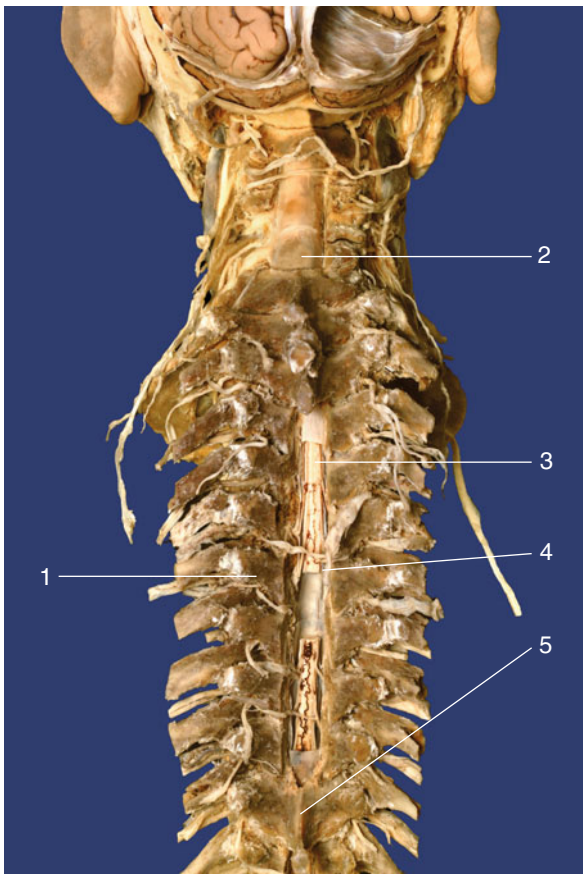


Fig. 40.12 Thoracic region of the spine. (*1*) Transverse process, (*2*) spinal dura mater (note the covering of the spinal ganglion), (*3*) spinal cord with vessels lying above it—the denticulate ligament is located to the left and right, and (*4*) the spinal dura mater has been removed—posterior longitudinal ligament, (*5*) thoracic spinous process; note the epidural space above (With permission from Danilo Jankovic)

Caudal to the medullary cone, it develops into the thin filum terminale, which descends medial to the cauda equina, penetrates the final part of the dural sac and arachnoid, and fuses with the connective tissue posterior to the first coccygeal segment.

The pia mater sends off 22 denticulate ligaments on each side, which attach to the dura mater and thus stabilize the spinal cord (Figs. 40.10, 40.12, and 40.13).

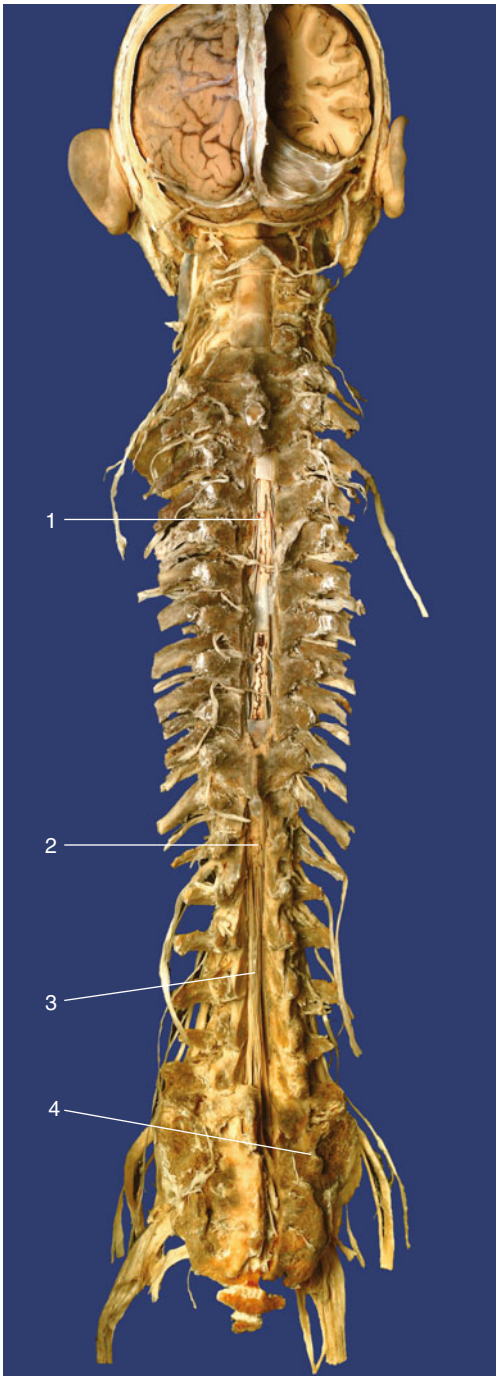


Fig. 40.13 The spinal cord. (*1*) Spinal cord with vessels on it—the denticulate ligament is located to the right and left, (*2*) conus medullaris, (*3*) terminal filum with cauda equina, (*4*) sacrum (With permission from Danilo Jankovic)

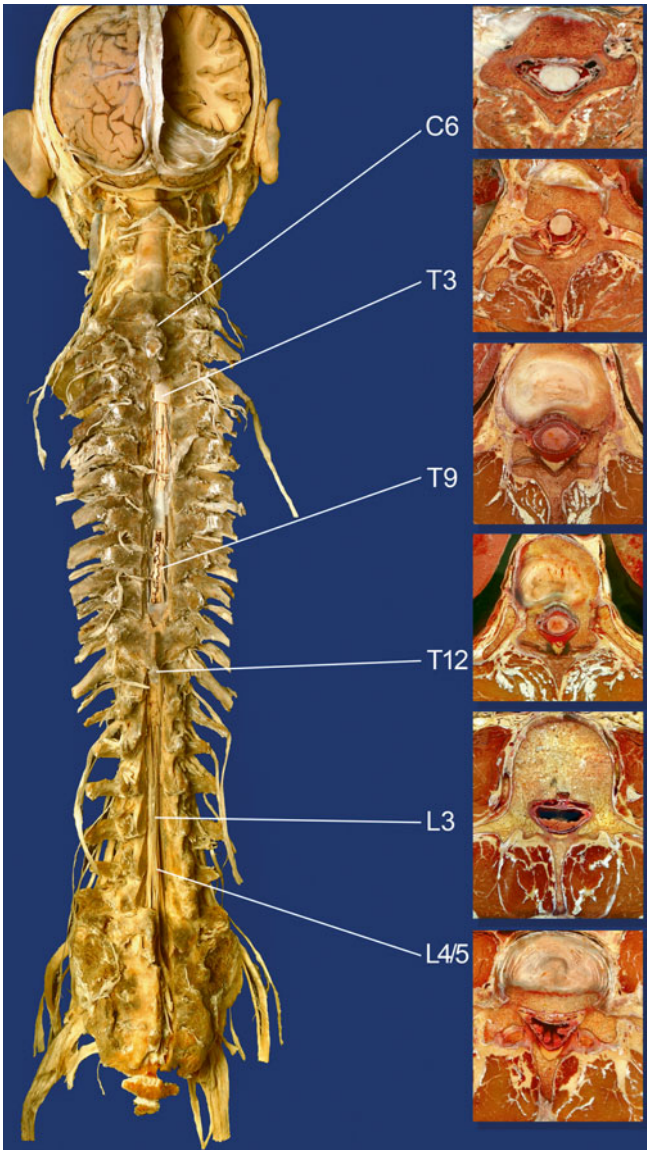


Fig. 40.14 The spinal cord. Cervical, thoracic, and lumbar. Serial transverse sections at the levels of C6, T3, T9, T12, L3, and L4/5. Note the conus medullaris (*T12*) and cauda equina (*L3*, *L4/5*) (With permission from Danilo Jankovic)

Spinal Nerves

There are 31 pairs of spinal nerves in the human: eight cervical pairs, 12 thoracic pairs, five lumbar pairs, five sacral pairs, and one coccygeal pair. These are connected to the spinal cord by a series of ventral and dorsal radicular filaments, which combine to form the nerve roots (Figs. 40.7, 40.10, 40.11, 40.12, 40.15, and 40.16).

The thicker dorsal (posterior) root is responsible for conducting afferent impulses (pain, temperature, touch, position). Each of the dorsal spinal nerve roots has a sensory spinal ganglion incorporated in it.

The ventral (anterior) root is responsible for conducting efferent impulses (muscles, glands). The nerve roots in the lower segments of the spinal cord descend in the horsetail-like cauda equina to their exit openings.

After exiting from the subarachnoid space, the ventral and dorsal roots cross the epidural space.

In spinal anesthesia, the nerve roots are the principal targets for local anesthesia (Figs. 40.7, 40.14, and 40.15).

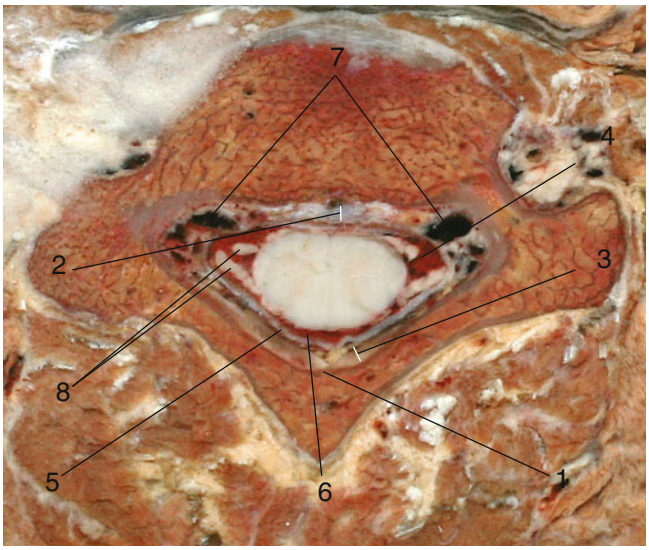


Fig. 40.15 Transverse section at the level of the C6 segment. (*1*) Ligamentum flavum, (*2*) anterior epidural space, (*3*) posterior epidural space, (*4*) subarachnoid space with the spinal cord, (*5*) spinal dura mater, (*6*) spinal pia mater, (*7*) epidural veins, (*8*) anterior and posterior spinal nerve roots (With permission from Danilo Jankovic)

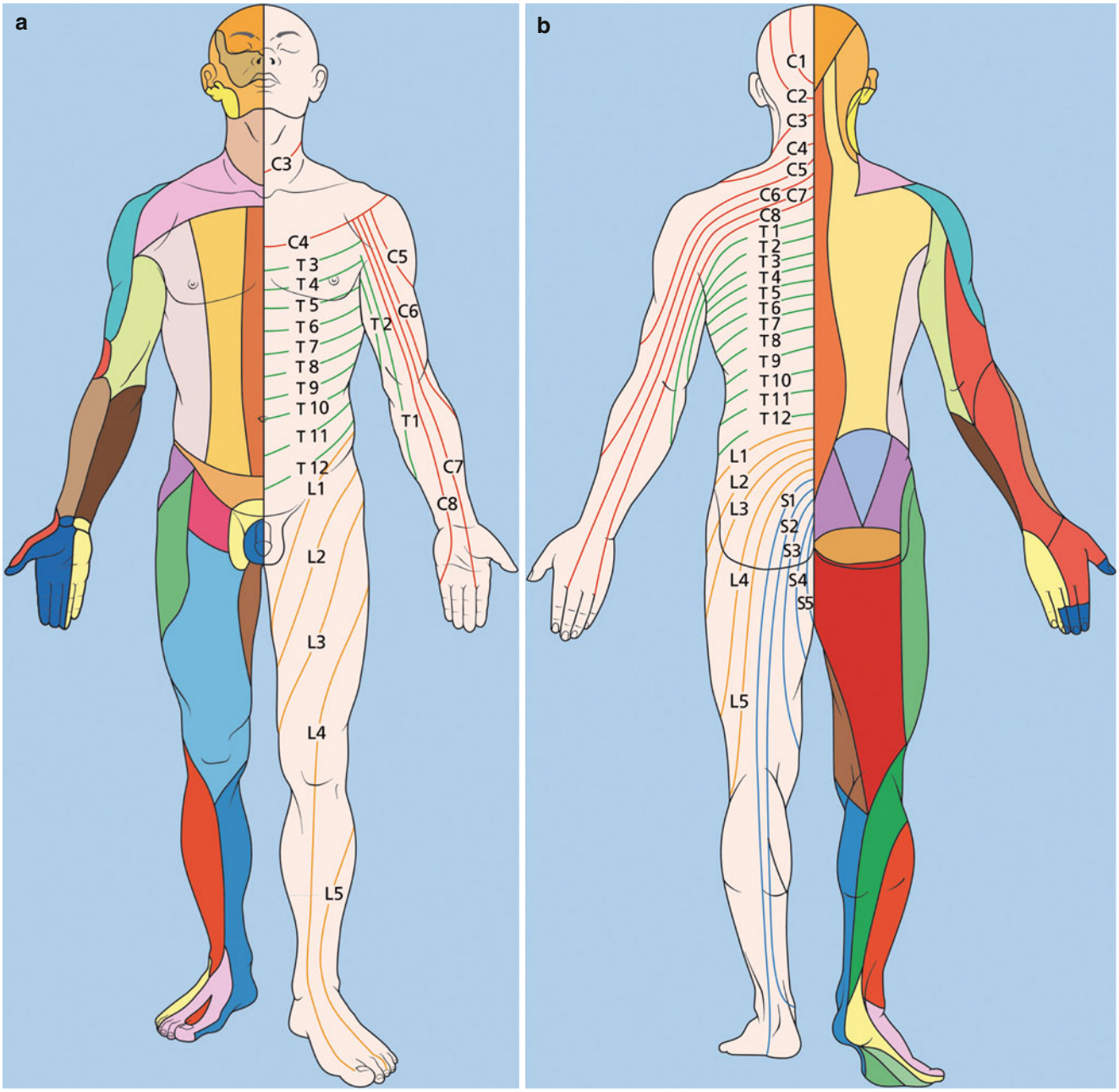


Fig. 40.16 (**a**) Spinal dermatomes and the corresponding spinal cord segments (With permission from Danilo Jankovic). (**b**) Cutaneous innervation areas (detailed descriptions are given in the relevant chapters) (With permission from Danilo Jankovic)

Spinal Dermatomes

Via its branching spinal nerves, each segment of the spinal cord provides the sensory supply for a specific area of the skin, known as the dermatome. These areas of the skin, which often overlap, are very important for checking and verifying the spread of anesthesia (Fig. 40.16a, b).

Arteries of the Spinal Cord

The spinal cord is supplied by numerous radicular arteries, which form the anterior spinal artery and twin posterior spinal arteries.

The radicular arteries branch off from the cervical vertebral artery, the thoracic intercostal arteries, and the abdominal lumbar arteries (Figs. 40.17 and 40.18).

The anterior spinal artery, which arises from the fourth segment of the vertebral arteries, accompanies the spinal cord in the midline (anterior median fissure) along its entire course.

Via the central branches and small branches of the arterial pial network, the anterior spinal artery supplies the anterior two-thirds of the spinal cord.

The cervical and first two thoracic spinal cord segments receive blood from the radicular branches of subclavian artery branches.

In the mediothoracic spinal cord region (T3–T7), there is a radicular branch at the level of T4 or T5.

The thoracolumbar segment of the spinal cord (T8 to the medullary cone) draws its arterial supply mainly from the large-caliber arteria arteria radicularis magna (the artery of Adamkiewicz), which arises from an intercostal artery on the left side.

The cauda equina is supplied by branches of the lumbar, iliolumbar, and lateral or median sacral arteries.

These also supply the medullary cone.

The paired *posterior spinal arteries* arise from the fourth segment of the vertebral artery, receiving flow from 10 to 23 posterior radicular branches, and supply the dorsal third of the spinal cord.

Thin pial branches run from the spinal arteries, forming a network on the surface of the spinal cord known as the arterial pial network.

Veins of the Spinal Cord and Vertebrae

The entire spinal canal is traversed by two venous plexuses, the internal and external vertebral venous plexuses (Fig. 40.19 a, b).

Together, these form a ring around each vertebra, freely anastomosing with one another and receiving flow from the vertebrae, ligaments, and spinal cord. They are largely avalvular. Pressure changes in the thoracic or cerebrospinal fluid (CSF) spaces consequently affect the blood volume in the venous plexuses. The plexuses are most strongly developed in the anterolateral area of the epidural space. They drain not only the spinal cord and its canal but also part of the CSF.

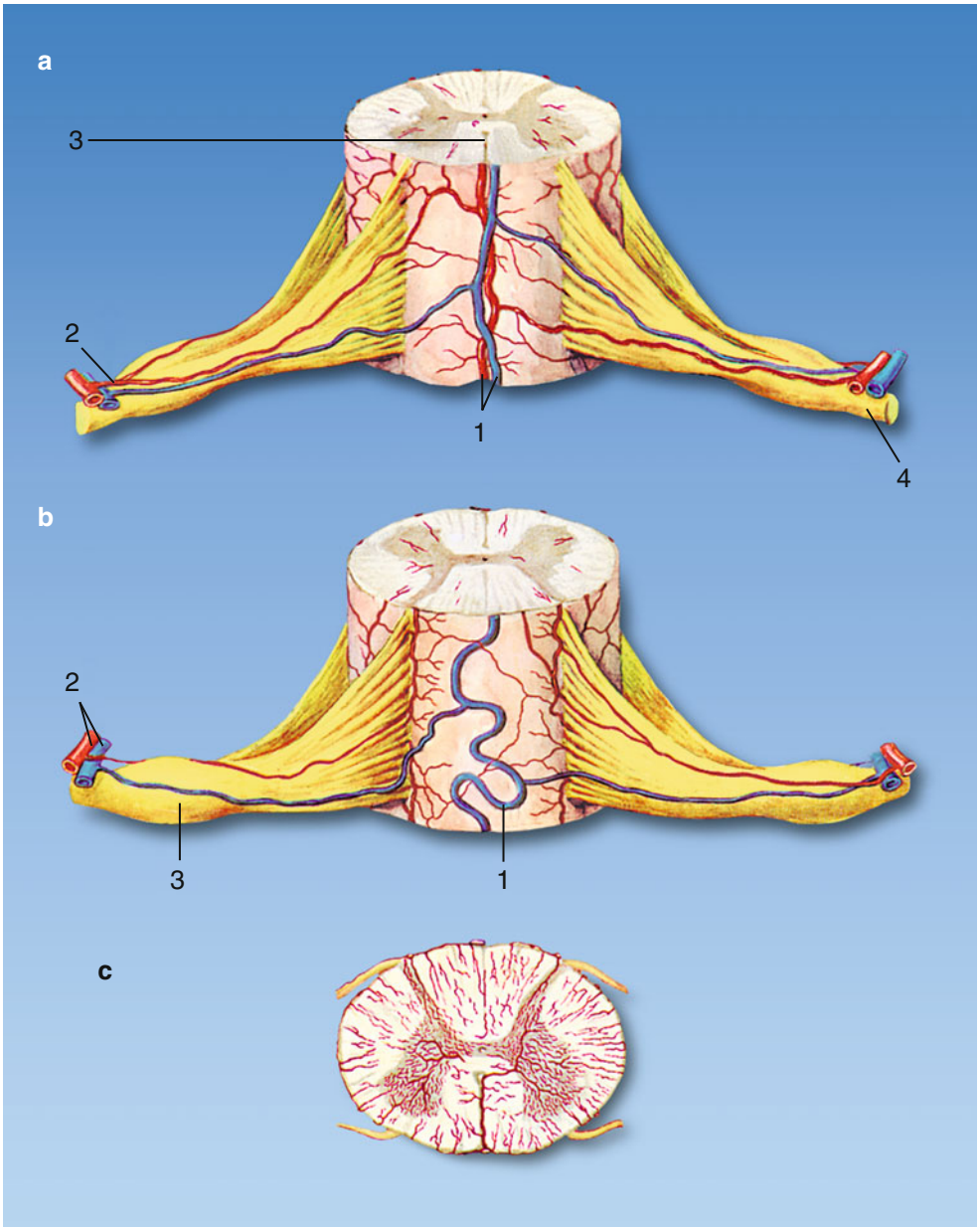


Fig. 40.17 (**a**–**c**) Spinal cord. (**a**) Ventral view: (*1*) anterior spinal artery and vein, (*2*) spinal branch, (*3*) anterior median fissure, (*4*) spinal nerve. (**b**) Dorsal view: (*1*) posterior spinal vein, (*2*) dorsal branch of the posterior intercostal artery, (*3*) spinal ganglion. (**c**) Cross section (With permission from Danilo Jankovic)

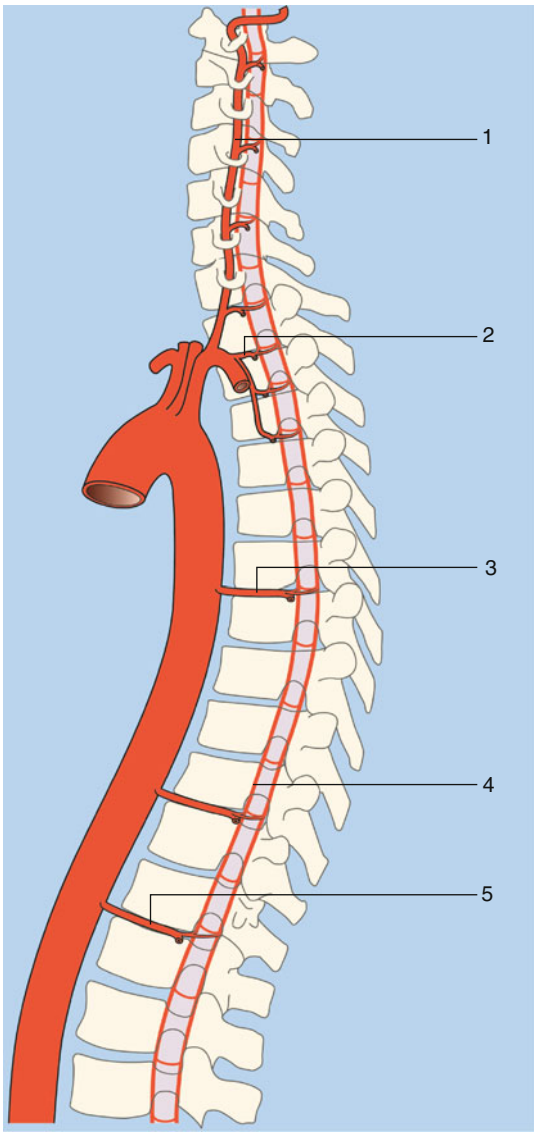


Fig. 40.18 Arteries of the spinal cord (*side view*). (*1*) Vertebral artery, (*2*) deep cervical artery, (*3*) intercostal artery, (*4*) anterior and posterior spinal artery, (*5*) arteria radicularis magnus (artery of Adamkiewicz) (With permission from Danilo Jankovic)

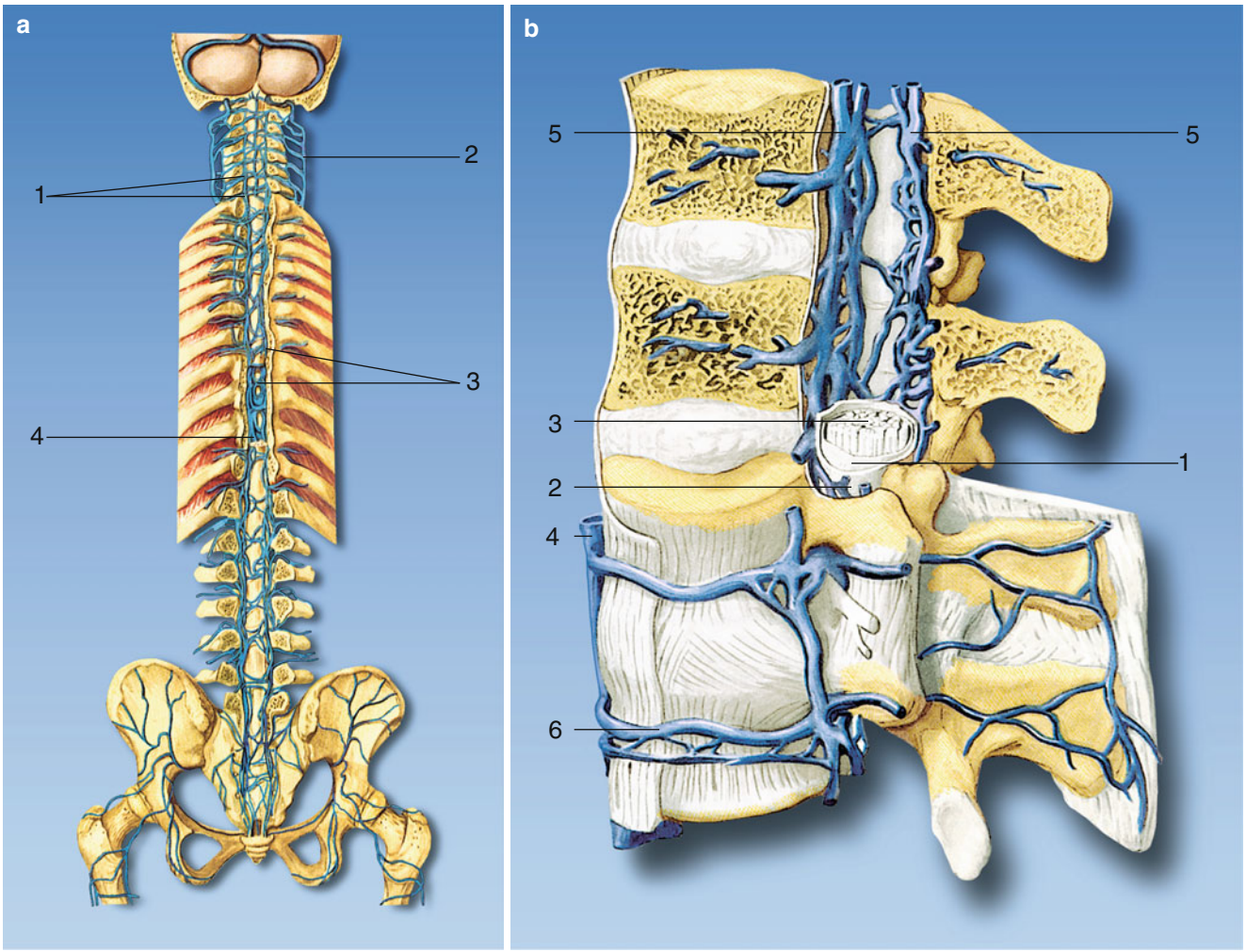


Fig. 40.19 (**a**) Veins of the spinal cord. (*1*) Vertebral veins, (*2*) deep cervical vein, (*3*) internal vertebral venous plexus, (*4*) spinal veins (with permission from Danilo Jankovic). (**b**) Veins of the spinal cord (lumbar region). (*1*) Arachnoid, (*2*) dura mater, (*3*) cauda equina, (*4*) inferior vena cava, (*5*) internal vertebral venous plexus, (*6*) lumbar vein (With permission from Danilo Jankovic)

Cerebrospinal Fluid [2]

The production of CSF is mainly achieved by active secretion and diffusion through the epithelial cells of the *choroid plexus* but also to a small extent in the *subarachnoid space* and *perivascularly.*

The main tasks of the cerebrospinal fluid are:

- To function as a hemodynamic buffer and physical protection against forces affecting the spinal cord and brain
- To substitute for the function of the lymphatic vessels, which are absent in the central nervous system
- To allow metabolic exchange between blood and neural tissue

There is a selective barrier between the blood and the CSF, the *blood–brain barrier*, which is formed by capillary endothelial cells and the choroid plexus. This barrier is clinically significant, as it is impermeable to many drugs.

The total quantity of the CSF in the adult is about 120–150 mL (with about 20–35 mL below the foramen ovale and about 15 mL below T5).

Approximately 400–450 mL of CSF is produced every day, and complete exchange of the fluid takes place every 10–12 h.

Lumbar *CSF pressure* in a supine position is about 6–10 cmH_2O, while in a seated position it is about 20–25 cmH_2O.

The *specific gravity* of the CSF is 1.007 (1.003–1.009), and this must be taken into account in relation to the local anesthetic being used.

The *osmolarity* of CSF is comparable with that of the blood plasma (300 osmol/L), and the pH value is approximately the same as the physiological value. Injected drugs mainly spread by diffusion, since CSF in the spinal canal circulates very little, if at all. *Resorption* of CSF into the blood takes place via the *arachnoid granulations* and through the walls of the capillary vessels in the central nervous system and pia mater.

The liquid in the CSF sheaths of the cranial nerves and in the root pockets of the spinal nerves is an exception to the above rule. This liquid can enter the extradural lymphatic vessels directly.

Ultrasound Imaging for Central Neuraxial Blockade

Introduction

The main technical challenge in central neuraxial blockade is to identify a soft tissue window into the vertebral canal. This is usually inferred from palpation of the spinous processes and tactile feedback from the needle tip as it is inserted. However, this can be difficult if surface landmarks are obscured, altered, or absent or if the interspinous and interlaminar spaces are narrowed by age-related changes. Technical difficulty and multiple needle insertion attempts are associated with patient discomfort and, more importantly, with serious complications including spinal hematoma and neurologic injury [14–16].

Pre-procedural ultrasound imaging of the spine is extremely useful in delineating the spinal anatomy more clearly, which permits more accurate planning of needle insertion, trajectory, and depth and in turn improves ease of performance.

Anatomy and Sonoanatomy of the Lumbar Spine

- Knowledge of the gross bony anatomy of the spine is essential for the interpretation of neuraxial ultrasound. It is particularly important to appreciate the contours of the posterior bony surfaces of the spine (Fig. 40.20) as these are responsible for the characteristic shapes of the acoustic dropout shadows seen on ultrasound.
- A curvilinear low-frequency ultrasound probe with its wider field of view and better beam penetration is recommended for use in all adult patients.
- Two planes and ultrasound probe orientations are commonly used to image the spine (Fig. 40.21):
  1. Transverse (or axial)
  2. Parasagittal (or longitudinal)
- The transverse plane is most useful in determining an appropriate needle insertion point for a midline approach. The parasagittal plane on the other hand provides valuable additional information on the location and identity of interlaminar spaces, particularly in the patient with more difficult anatomy. The ultrasound examination should therefore always utilize both probe orientations.
- There are five typical ultrasonographic views of the lumbar spine that may be obtained: three in the parasagittal plane and two in the transverse plane.
  1. Parasagittal oblique view
  2. Parasagittal transverse process view
  3. Parasagittal articular process view
  4. Transverse midline interlaminar view
  5. Transverse midline spinous process view
- The *parasagittal oblique (PSO)* and *transverse midline (TM) interlaminar* views are the two most important views to obtain.

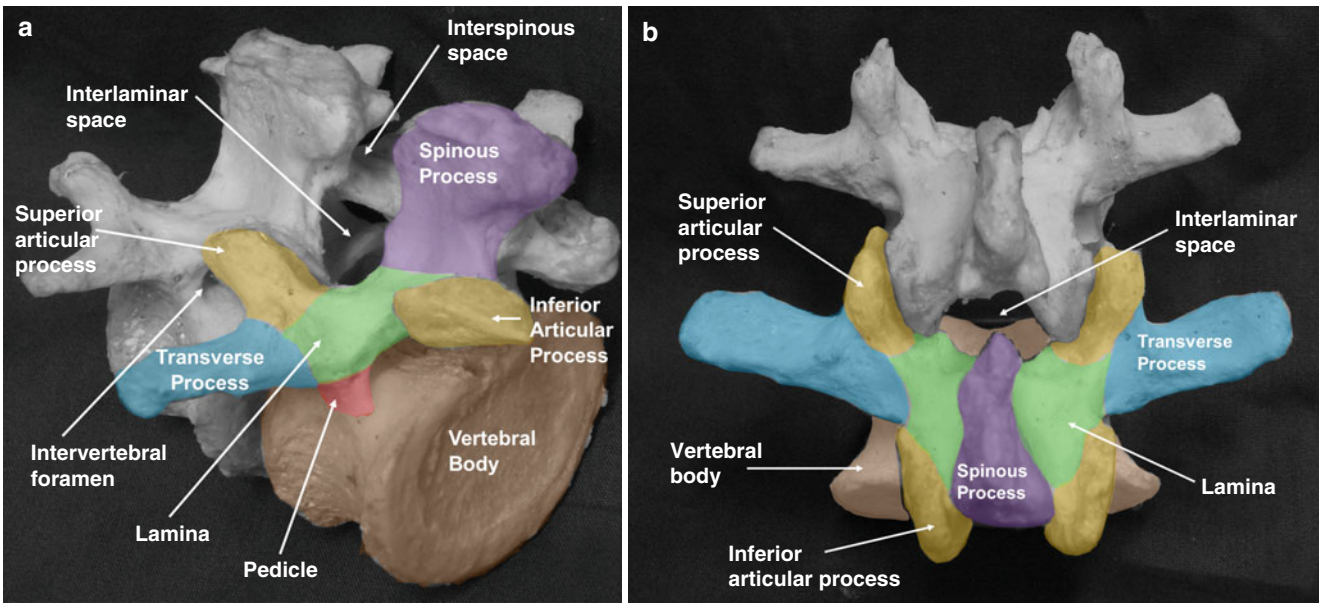


Fig. 40.20 Three-quarter oblique view (**a**) and posterior view (**b**) of adjacent lumbar vertebrae. The interlaminar space is located posteriorly and is bounded by the bases of the spinous processes, the laminae, and the inferior articular processes. It is roofed over by the ligamentum flavum. The interspinous space lies in the midline and is filled by the supraspinous and interspinous ligaments. The intervertebral foramina are located laterally and are bounded by the pedicles, the vertebral body, the laminae, and the superior and inferior articular processes and contain the spinal nerve roots and their accompanying blood vessels (Courtesy of www.usra.ca)

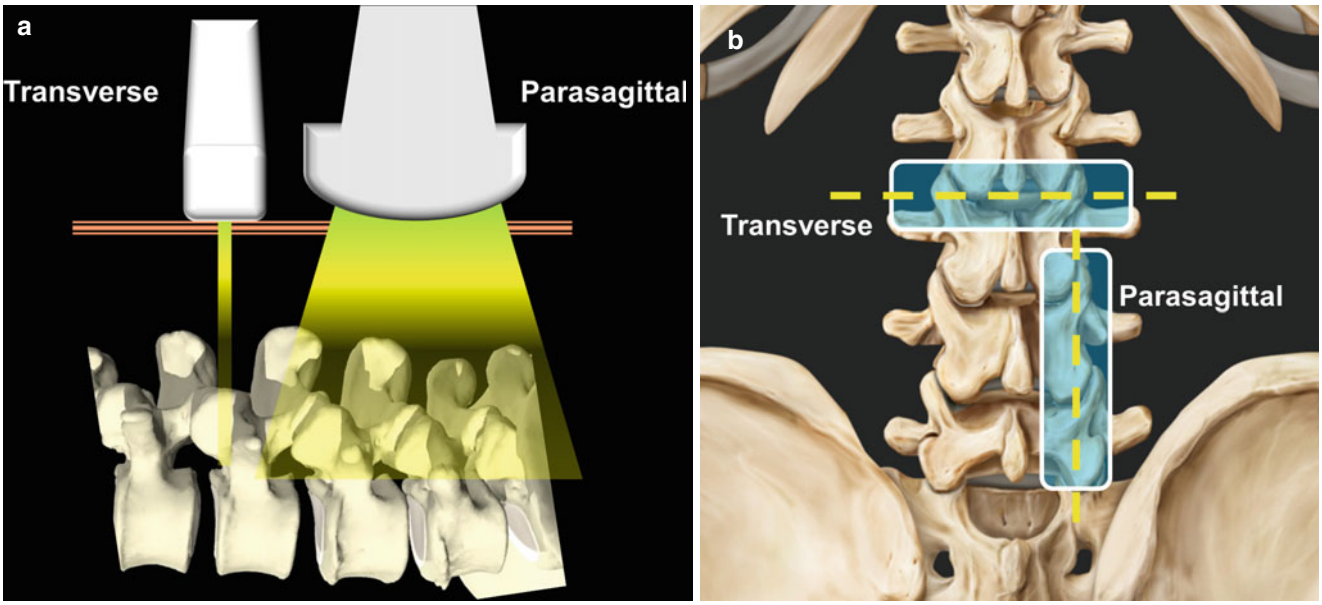


Fig. 40.21 (**a**) is a lateral view, and (**b**) is a posterior view, of the surface placement of the ultrasound probe over the lumbar spine. The ultrasound probe may be placed in two basic orientations: transverse or parasagittal, which determines the "cut" of the spine that will be visualized (Courtesy of www.usra.ca)

Parasagittal Oblique (PSO) View

- The probe is placed in a longitudinal parasagittal orientation and angled toward the midline to direct the beam through the paramedian interlaminar spaces.
- The beam transects the sloping laminae of the lumbar vertebrae, producing a characteristic "sawtooth" pattern of acoustic shadows (Fig. 40.22).
- Two hyperechoic linear structures are seen in the interlaminar gap between the "sawteeth." The more superficial one that adjoins the bases of adjacent "sawteeth" is the *posterior complex* and represents the ligamentum flavum, epidural space, and posterior dura. The deeper hyperechoic structure is the *anterior complex* and represents the anterior dura, posterior longitudinal ligament, and posterior aspect of the vertebral body and intervertebral disk, which together constitute the anterior wall of the vertebral canal.
- The intervening hypoechoic area is the intrathecal space. The conus medullaris has the same echogenicity as cerebrospinal fluid and cannot be distinguished in the adult patient. Pulsatile hyperechoic streaks may occasionally be seen within the intrathecal space and most likely represent elements of the cauda equina.

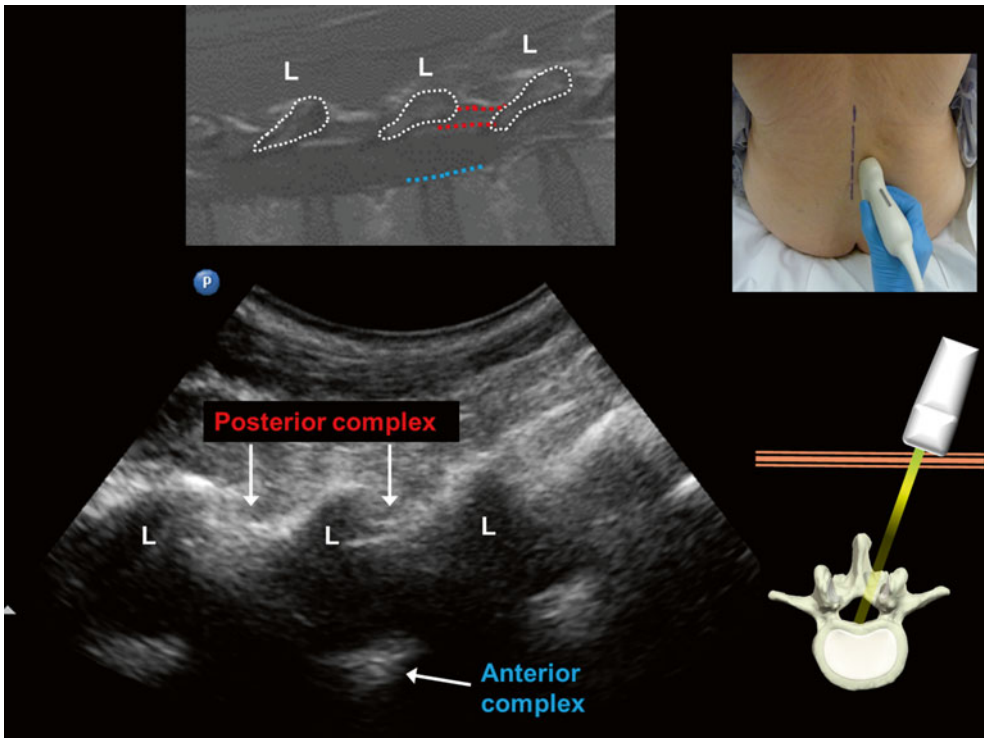


Fig. 40.22 Parasagittal oblique (PSO) view of the lumbar spine with corresponding magnetic resonance image *above*. The laminae (*L*) are visible in cross section as sloping hyperechoic lines with acoustic shadowing beneath and form a "sawtooth" pattern. The hyperechoic posterior complex, consisting of ligamentum flavum, posterior epidural space, and posterior dura, is visible between laminae; these structures cannot always be distinguished from one another. The deeper hyperechoic linear structure is the anterior complex, consisting of the anterior dura, anterior epidural space, posterior longitudinal ligament, and posterior aspect of the vertebral body and intervertebral disk (Courtesy of www.usra.ca)

Parasagittal Views of the Articular Processes and Transverse Processes

- If the probe is positioned more laterally in the parasagittal plane or is insufficiently angled toward the midline, an image of the articular processes or transverse processes may be obtained instead. Recognition of these two views will indicate the appropriate manipulation of the probe required to obtain the PSO view.
- The overlapping superior and inferior articular processes can be recognized as a continuous, wavy hyperechoic line with a dense acoustic shadow beneath (Fig. 40.23).
- The transverse processes, on the other hand, cast characteristic "fingerlike" acoustic shadows with the psoas major muscle visible in between (Fig. 40.24).

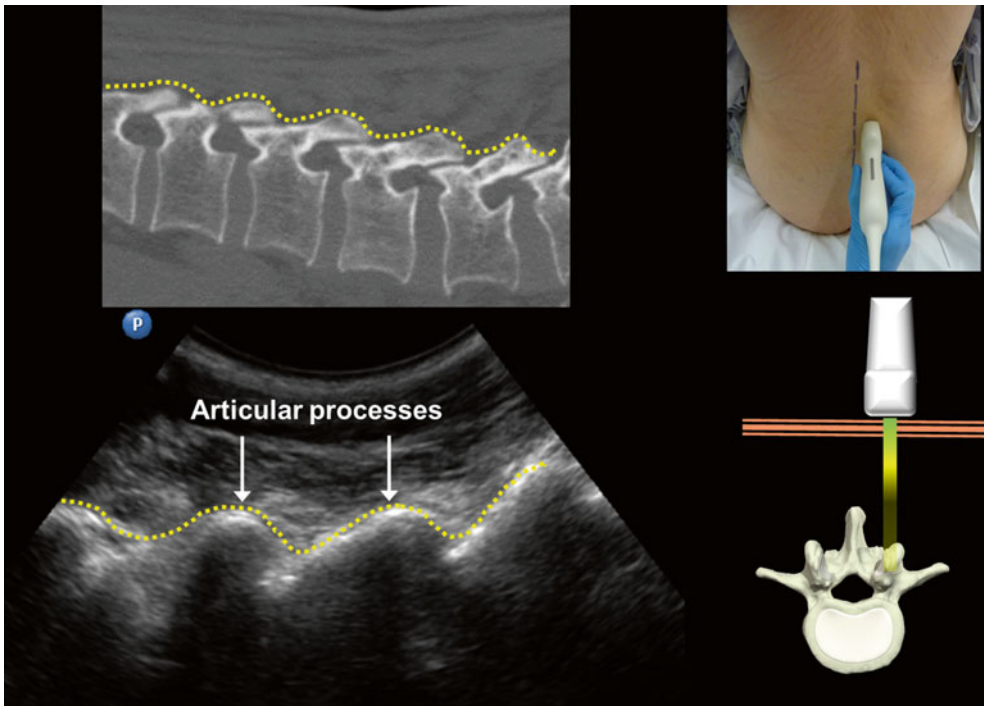


Fig. 40.23 Parasagittal articular process view of the lumbar spine and corresponding computed tomography image (*above*). The overlapping bony superior and inferior articular processes are seen as a continuous hyperechoic line of "humps" with acoustic shadowing beneath (Courtesy of www.usra.ca)

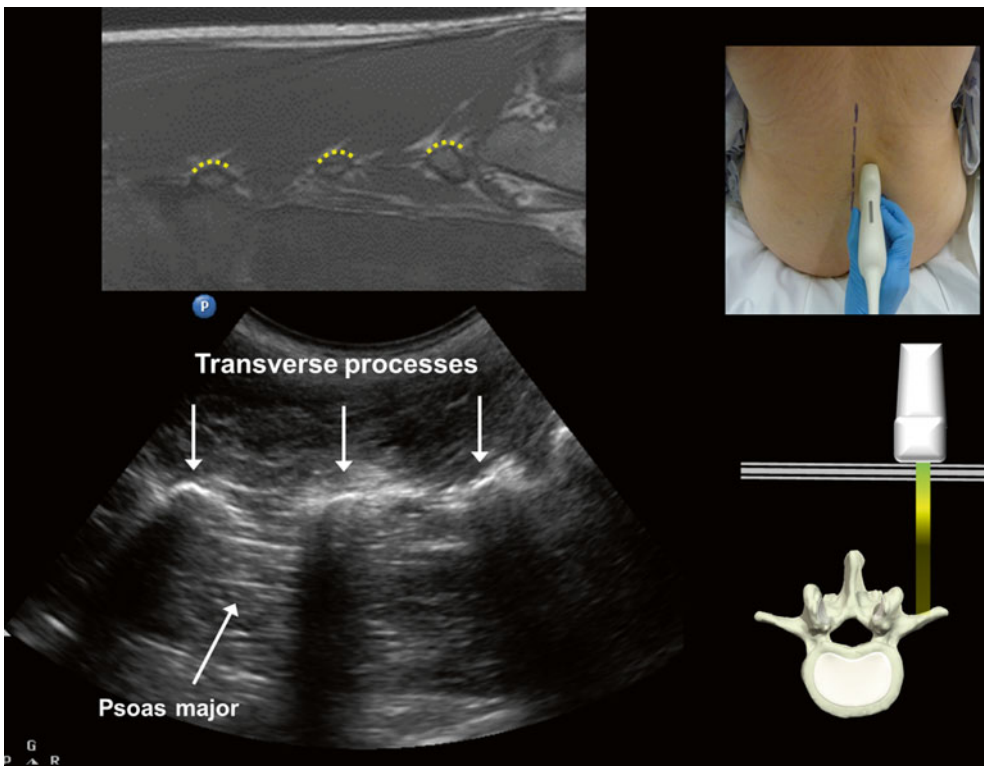


Fig. 40.24 Parasagittal transverse process view of the lumbar spine and corresponding magnetic resonance image (*above*). The probe is placed over the tips of the transverse processes (TP), which appear as hyperechoic curvilinear structures with "fingerlike" acoustic shadowing beneath. The erector spinae muscle and the psoas muscle lie superficial and deep to the transverse processes, respectively (Courtesy of www.usra.ca)

Transverse Midline (TM) Interlaminar View

- The most important characteristic of the TM interlaminar view is visualization of the hyperechoic *anterior complex*, which signifies that the ultrasound beam has penetrated the vertebral canal through the interspinous and interlaminar spaces (Fig. 40.25).
- The *posterior complex* is usually visible as well but is generally less distinct compared to the PSO view.
- The articular processes and transverse processes lie in the same transverse plane and their bony contours are usually visible lateral to the posterior and anterior complexes. In the obese patient, these are important surrogate markers of the interlaminar space as the posterior and anterior complexes may be poorly visible due to soft tissue attenuation of the ultrasound beam.

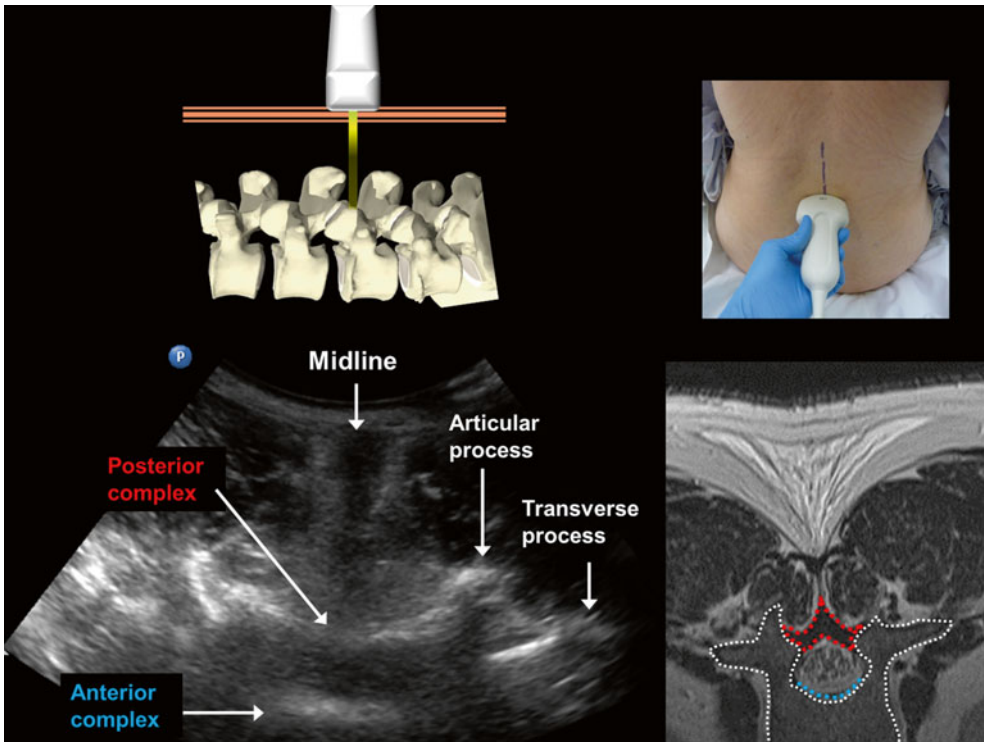


Fig. 40.25 Transverse midline interlaminar view of the lumbar spine and corresponding magnetic resonance imaging scan (*right*). The intrathecal space is a dark hypoechoic band sandwiched between the hyperechoic posterior and anterior complex. The transverse processes and articular processes lie in the same transverse plane and are usually visible. The ligamentum flavum, posterior epidural space, and dura usually cannot be distinguished from one another in the transverse view. The midline is indicated by the dark vertical stripe of the interspinous ligament (Courtesy of www.usra.ca)

TM Spinous Process View

- The TM spinous process view is easily recognized by the hyperechoic appearance of the tip of the spinous process and adjacent lamina and the dense acoustic dropout shadow that they cast (Fig. 40.26).
- This view is a cue to manipulate the ultrasound probe cephalad or caudad to obtain the TM interlaminar view.
- It may also be used to definitively establish and mark the position of the spinous process and midline, which in itself can be helpful in performing neuraxial block if an adequate TM interlaminar view cannot be obtained.

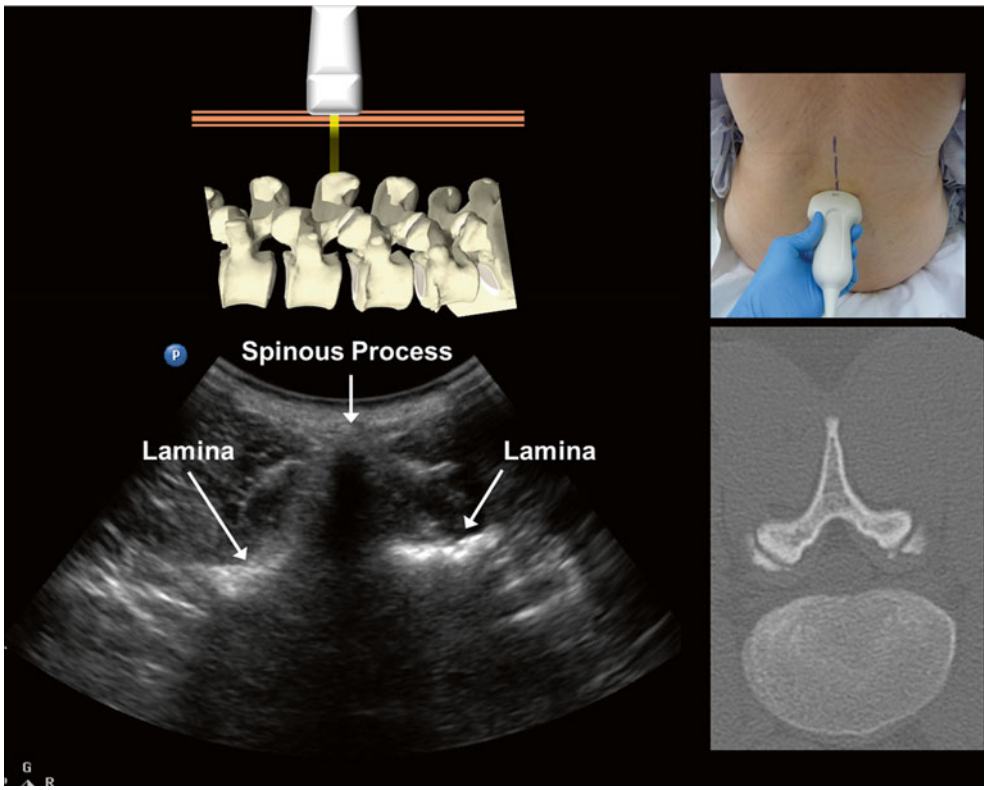


Fig. 40.26 Transverse midline spinous process view of the lumbar spine and corresponding computed tomography image (*right*). The tip of the spinous process and the lamina are brightly hyperechoic on ultrasound with pronounced acoustic shadowing that obscures all deeper structures (Courtesy of www.usra.ca)

Recommended Pre-procedural Scanning Technique in the Lumbar Spine

Patient, Machine, and Operator Positioning

- The operator stands or sits behind the patient, who may be placed in the sitting or lateral decubitus position.
- The ultrasound machine is ideally placed on the opposite side of the bed from the operator.

Parasagittal and Parasagittal Oblique Scan

- The probe is first placed in a parasagittal orientation over the sacrum of the patient. The sacrum is easily recognizable as a continuous hyperechoic line with acoustic shadowing beneath (Fig. 40.27).
- The probe is slid in a cephalad direction and angled toward the midline to obtain a PSO view of the L5–S1 interlaminar space. Successive interlaminar spaces are identified by continuing to slide the probe in a cephalad direction.
- Each interlaminar space should be centered on the ultrasound screen and a corresponding mark made on the patient's skin in the middle of the long edge of the probe (Fig. 40.28). These marks serve to confirm the identity of the intervertebral level being imaged when scanning in the transverse plane. In the event an adequate TM interlaminar view cannot be obtained, these marks also indicate the approximate location of the interlaminar space and can thus guide needle insertion.

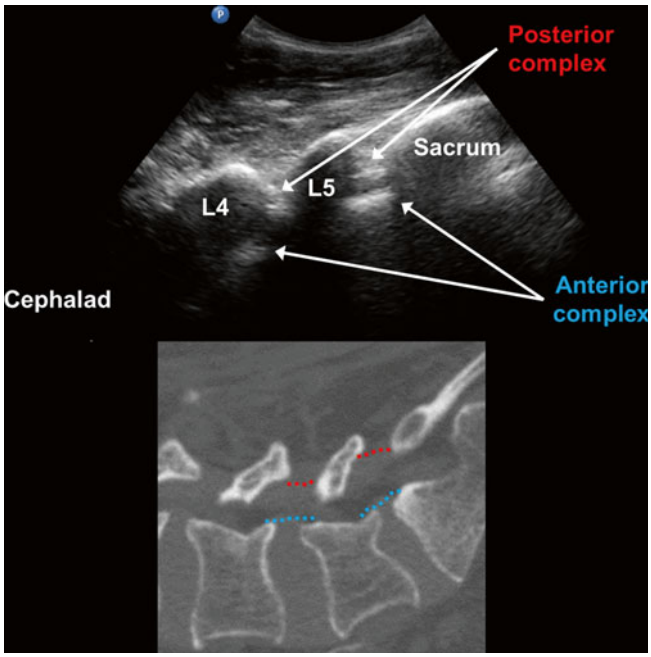


Fig. 40.27 Parasagittal oblique view of the L5–S1 junction and corresponding computed tomography image (*below*). The sacrum is recognizable as a horizontal hyperechoic linear structure, and the L5 lamina has the typical "sawtooth" appearance. The posterior and anterior complexes are visible through the intervening gap (Courtesy of www.usra.ca)

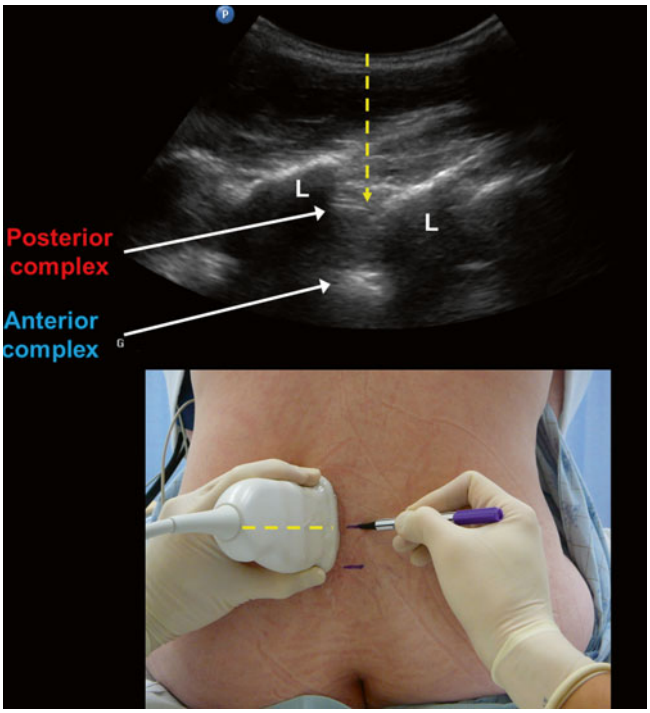


Fig. 40.28 In the parasagittal oblique view, each interlaminar space is centered in turn on the ultrasound screen. A corresponding skin mark is made at the midpoint of the probe's long edge to mark its position (Courtesy of www.usra.ca)

Transverse Scan

- The probe is then rotated into the transverse plane and manipulated to obtain the TM interlaminar views at the relevant interspaces. Slight cephalad tilting of the probe may be required to optimize image quality in patients with limited spine flexion.
- When the appropriate view is obtained, skin marks made at the midpoints of the long and short edges of the probe will indicate the locations of the neuraxial midline and interlaminar space respectively (Fig. 40.29). The intersection of these two marks is an appropriate needle insertion point for a midline approach to the vertebral canal.
- The depth to the deep aspect of the posterior complex may be measured in either the PSO or TM interlaminar view and correlates well with actual needle depth to the epidural and intrathecal space.

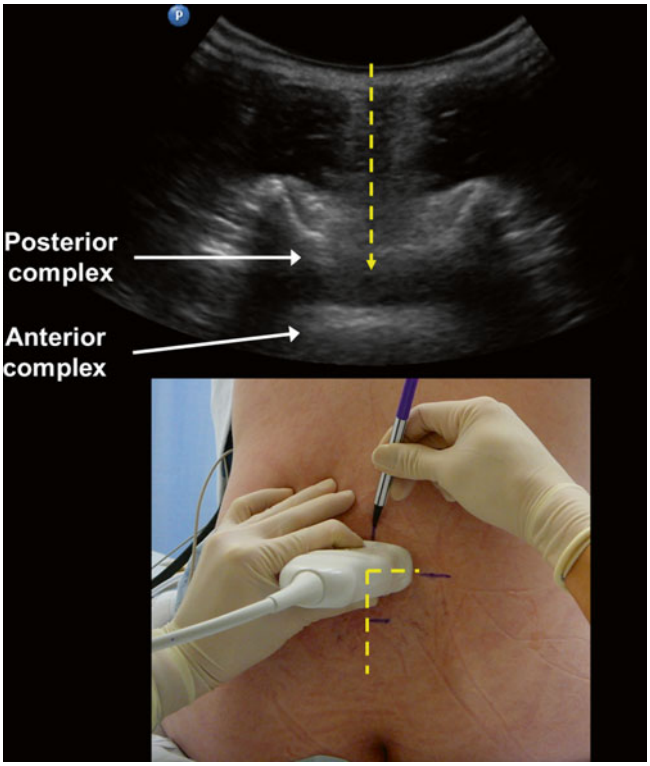


Fig. 40.29 In the transverse midline interlaminar view, the midline is centered on the ultrasound screen, and skin marks are made at the midpoint of the probe's long and short edges. The intersection of these two marks provides an appropriate needle insertion point for a midline approach to the epidural or intrathecal space at that level (Courtesy of www.usra.ca)

Strategies in Subjects with Poor-Quality Views

The Obese Patient

- Obese patients are primarily a challenge because attenuation of the ultrasound by the increased depth reduces the echogenicity of the relevant anatomical structures.
- In the obese patient, image quality may be improved by adjustments in gain and focus and reducing frequency to enhance penetration.
- Firm probe pressure helps by enhancing skin-probe contact and reducing the effective depth to the structures of interest.
- Experience in scanning and image interpretation will also facilitate recognition of more subtle appearances of the characteristic patterns described above (Fig. 40.30).

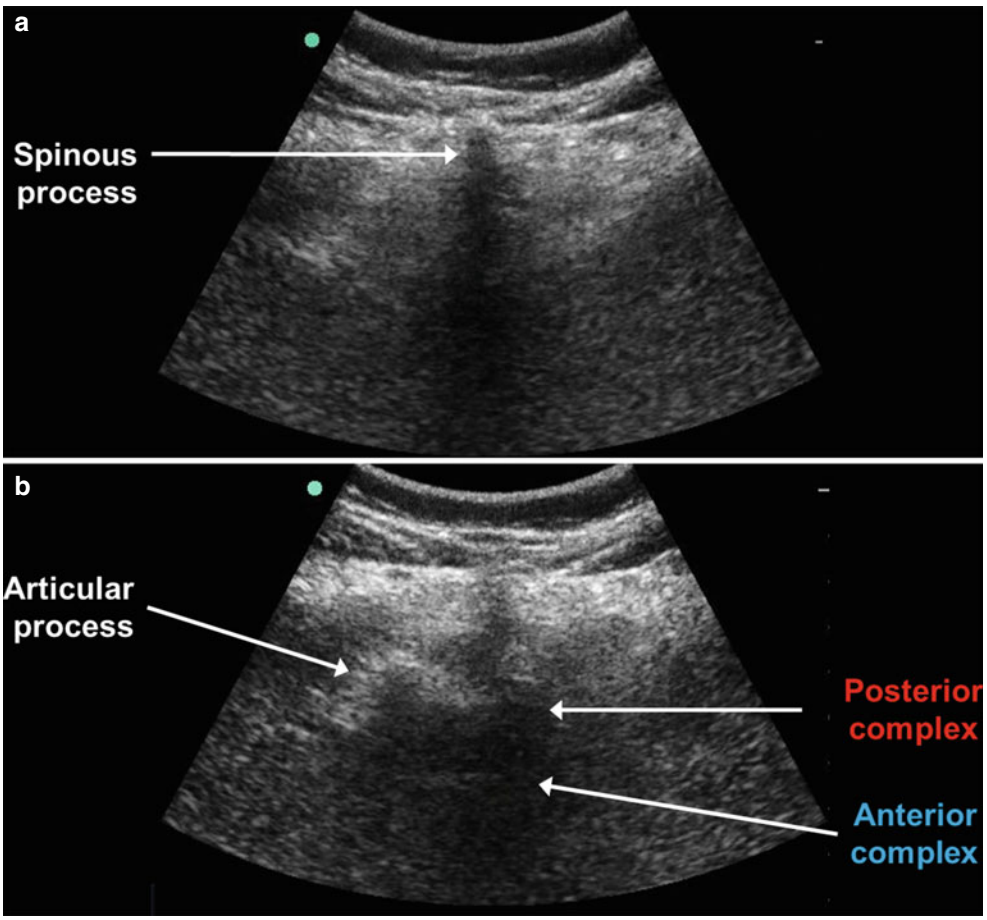


Fig. 40.30 An example of poor-quality transverse midline spinous process (**a**) and interlaminar (**b**) views in one individual. In the interlaminar view (**b**), the posterior and anterior complexes are only faintly hyperechoic, but nevertheless are visible, especially when contrasted with the dense acoustic shadow cast by the spinous process and lamina in (**a**). The other visual cue that (**b**) is an interlaminar view as opposed to a spinous process view is the contour of the tip of the articular process which lies in the same transverse plane as the interlaminar space (Courtesy of www.usra.ca)

The Elderly Patient

- Elderly patients may present a challenge due to narrowing of the interspinous and interlaminar spaces associated with age-related degenerative changes. The probe must be manipulated in a careful and controlled manner to try and direct the beam through these narrowed spaces.
- Prominent spinous processes in a thinner elderly patient may hinder adequate skin-probe contact and contribute to poor visualization of neuraxial structures. In such patients, a TM interlaminar view may be physically difficult or impossible to obtain, and the PSO view may be a better alternative. Contact may also be improved by using a probe with a smaller footprint.

Anatomy and Sonoanatomy of the Thoracic Spine

- There are significant differences in the morphology of the thoracic spine relative to the lumbar spine (Fig. 40.31).
- In the mid-thoracic spine (T5–8), the spinous processes angle steeply in a caudad direction such that the inferior border of the spinous process overlies the midpoint of the lamina below. The thoracic laminae also lie closer together, and the interlaminar spaces are therefore much narrower.
- The lower four thoracic vertebrae (T9–12) are similar to lumbar vertebrae in that their spinous processes are broad, flat, and only slightly angled.
- A curvilinear low-frequency probe is generally recommended; however, in slimmer individuals, a linear high-frequency probe may also produce adequate images.
- As in the lumbar spine, the two most important views are the PSO and TM interlaminar views.
- In the lower thoracic spine, these views are similar to those of the lumbar spine except that the interlaminar spaces tend to be narrower. The mid-thoracic views are quite different however and are described below.

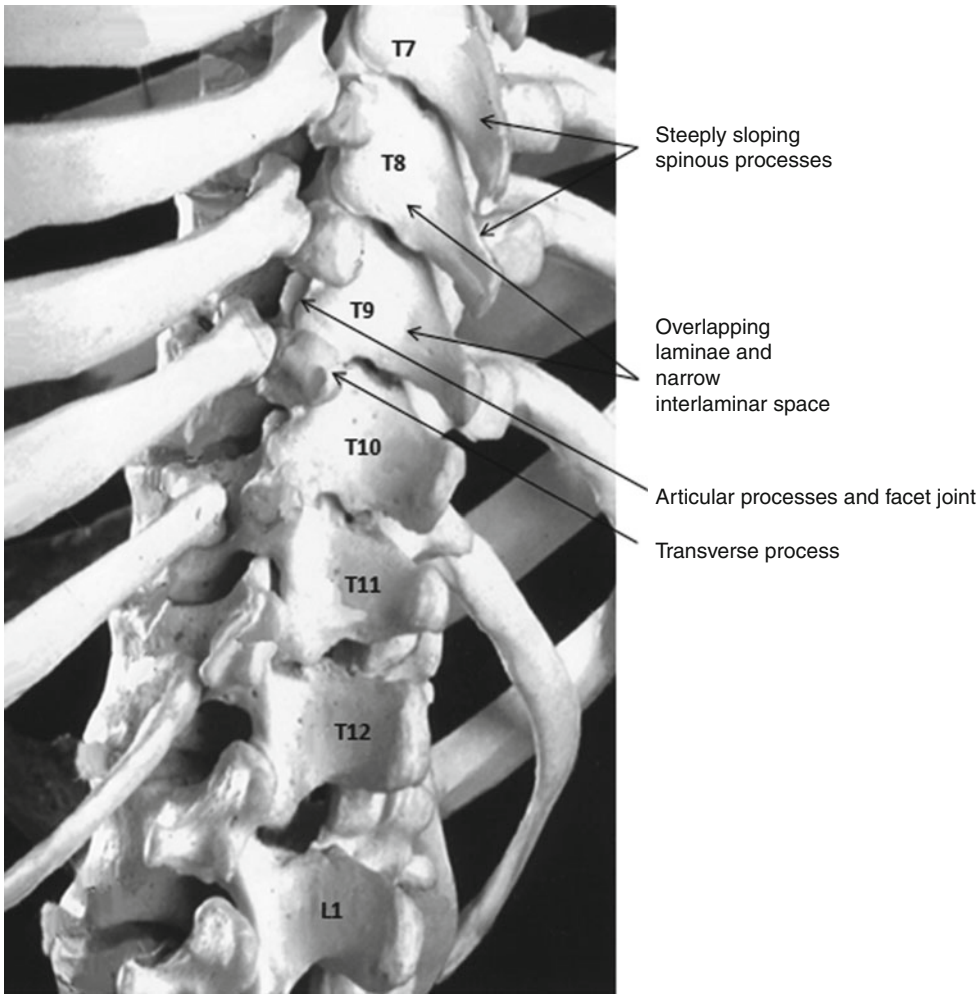


Fig. 40.31 The lower thoracic vertebrae (*T10–T12*) are similar in morphology and ultrasonographic appearance to lumbar vertebrae. The middle thoracic vertebrae have steeply sloping spinous processes that make it impossible to obtain a transverse interlaminar view. The interlaminar spaces are also small, and the paramedian sagittal oblique view into the vertebral canal is limited as a result (Courtesy of www.usra.ca)

Recommended Pre-procedural Scanning Technique in the Mid-thoracic Spine

Patient, Machine, and Operator Positioning

- The operator stands or sits behind the patient, who may be placed in the sitting or lateral decubitus position.
- The ultrasound machine is ideally placed on the opposite side of the bed from the operator.

Parasagittal Oblique Scan

- The mid-thoracic laminae appear as almost horizontal hyperechoic linear structures, separated by narrow gaps which represent the paramedian interlaminar spaces (Fig. 40.32).
- Due to the narrow interlaminar spaces, the posterior complex and anterior complex are rarely visible and are usually smaller and fainter than in the lumbar spine [17].
- The interlaminar spaces are identified and marked using the PSO view in a process similar to that described for the lumbar spine.
- The depth to the lamina or posterior complex is measured to estimate needle depth to the epidural space.
- Identification of intervertebral level can be achieved by counting upward from the L5–S1 junction. Alternatively the spaces can also be counted down from T1 or up from T12, having first identified the articulation of the first rib with T1 or the 12th rib with T12, respectively.

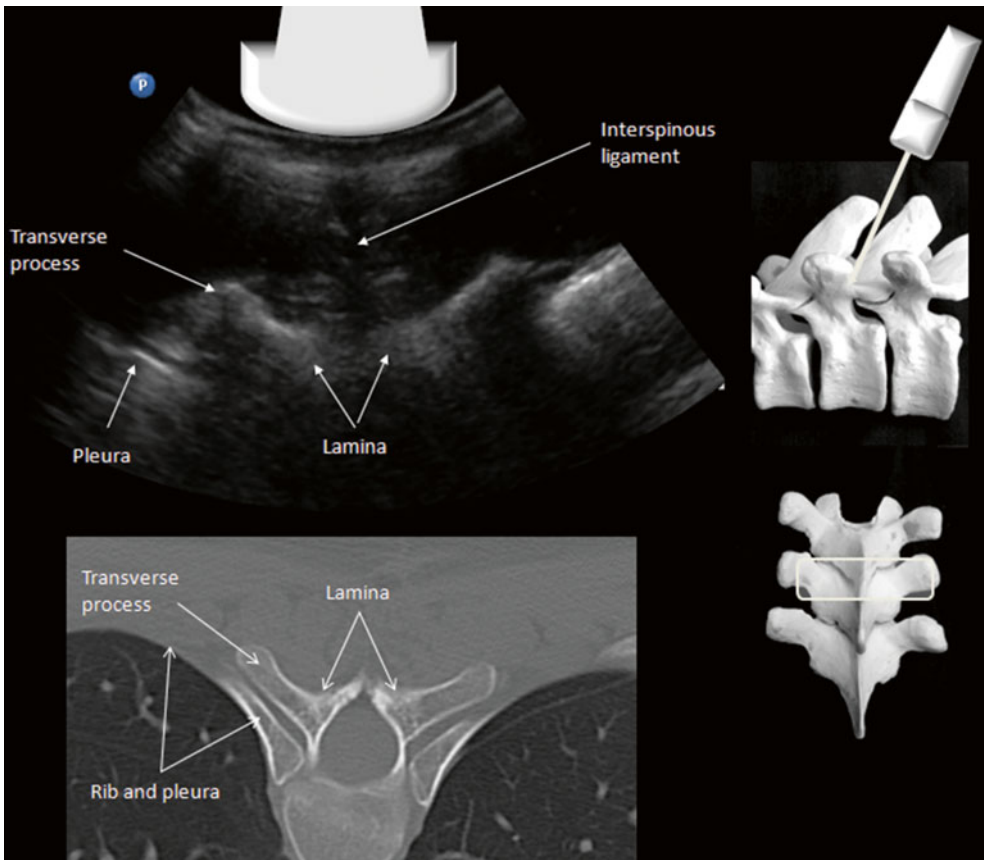


Fig. 40.32 Transverse view of the mid-thoracic spine and corresponding computed tomography image. An *interlaminar* view into the vertebral canal cannot be obtained because of the steeply sloping spinous processes and overlapping laminae (Courtesy of www.usra.ca)

Transverse Midline Scan

- The overlapping spinous processes in the mid-thoracic spine make it impossible to obtain a TM interlaminar view of posterior and anterior complexes.
- The visible structures are the hyperechoic bony contours of the spinous process, lamina, and transverse processes and, more laterally, the ribs and pleura (Fig. 40.33).
- The TM view is therefore used mainly to map and mark out the location of the neuraxial midline particularly if it cannot be palpated or if it is distorted, e.g., by scoliosis. With this additional information, the needle approach to the thoracic epidural space can be triangulated more accurately.

Learning Strategies in Neuraxial Ultrasound

Familiarization with the gross anatomy and sonoanatomy of the spine is essential prior to performing the ultrasound-guided neuraxial blockade in clinical practice.

- This can be done by:
  - Reviewing didactic material [17–19]
  - Attending educational workshops
  - Repetitive practice on a water-based spine phantom or human volunteers
- An additional educational resource is an interactive 3-dimensional digital spine model available online at http://www.usra.ca/vspine.php.
- Competency in the technique of pre-procedural scanning and marking should be acquired in normal patients before attempting it in patients with more challenging anatomy.

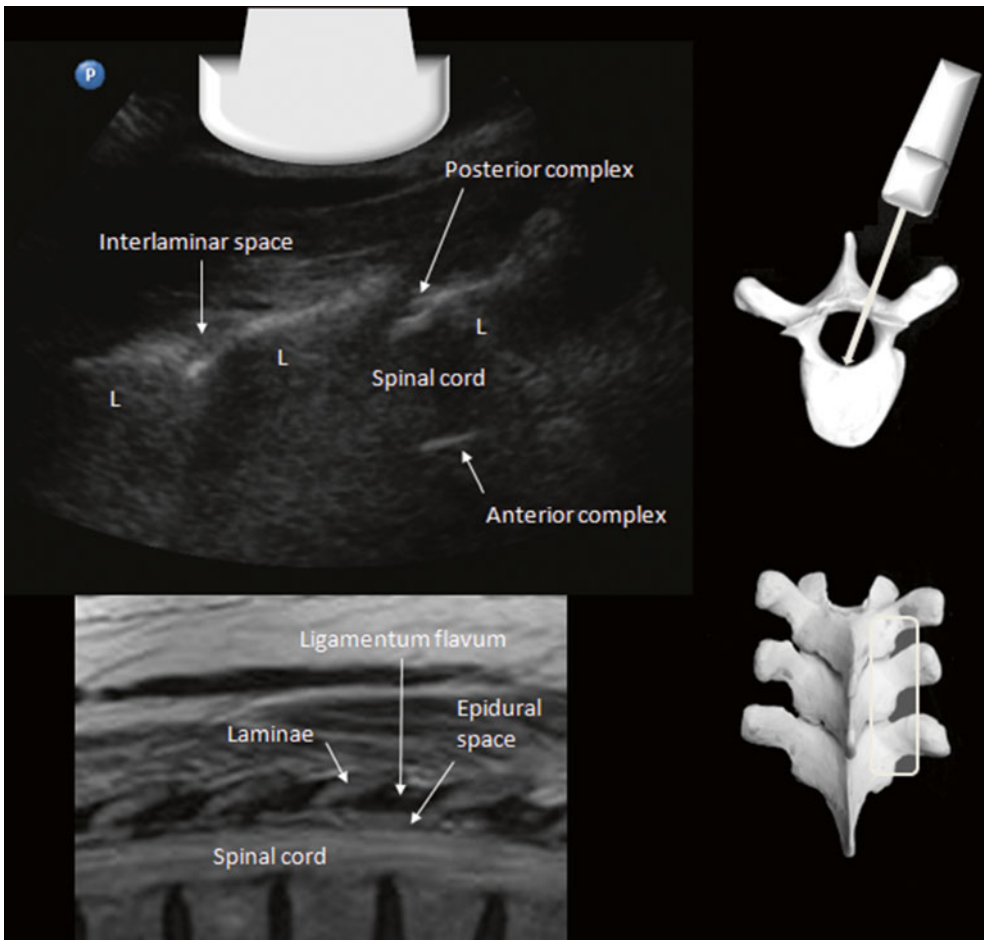


Fig. 40.33 Paramedian sagittal oblique view of the mid-thoracic spine and corresponding magnetic resonance image. Despite the narrow interlaminar space, it is possible to visualize the posterior and anterior complex at one or more levels. At a minimum, the location of the interlaminar space can be determined by the dip or gap between successive laminae (*L*). Note that the spinal cord is hypoechoic and cannot be distinguished from the surrounding cerebrospinal fluid (Courtesy of www.usra.ca)

References

1. Andres KH. Über die Feinstruktur der Arachnoidea und Dura mater von Mammalia. Z Zellforsch Mikrosk Anat. 1967;79:272–92.
2. Du Boulay GH. Pulsatile movements in the CNS pathways. Br J Radiol. 1966;39:255–62.
3. Düring MV, Bauersachs M, Böhmer B, et al. Neuropeptide Y- and substance P-like immunoreactive nerve fibers in the rat dura mater encephali. Anat Embryol (Berl). 1990;182:363–73.
4. Faraci FM, Kadel KA, Haistad DD. Vascular responses of dura mater. Am J Physiol. 1989;257(1 Pt 2):H157–61.
5. Frederickson RG. The subdural space interpreted as a cellular layer of meninges. Anat Rec. 1991;230:38–51.
6. Groen GJ, Baljet J, Drukker J. The innervation of the spinal dura mater: anatomy and clinical applications. Acta Neurochir (Wien). 1988;92:39–46.
7. Keller JT, Marfurt CF. Peptidergic and serotoninergic innervation of the rat dura mater. J Comp Neurol. 1991;309:515–34.
8. Lang J. Funktionelle Anatomie der Halswirbelsäule und des benachbarten Nervensystems. In: Hohman D, Kügelgen B, Liebig K, Schirmer M, editors. Neuroorthopädie, vol. 1. Berlin/Heidelberg: Springer; 1983.
9. Lang J. Klinische Anatomie der Halswirbelsäule. Stuttgart/New York: Thieme Verlag; 1991.
10. Schroth G, Klose U. Cerebrospinal fluid flow. I. Physiology of cardiac-related pulsations. Neuroradiology. 1992;35:1–9.
11. Schroth G, Klose V. Cerebrospinal fluid flow. II. Physiology of respiration-related pulsations. Neuroradiology. 1992;35:10–5.
12. Zenker W. Einige neue Aspekte zu Feinbau und Funktion der Dura mater und des Subarachnoidalraumes. Symposium über Spinalanästhesietechnik. Bad Säckingen;1994.
13. Zenker W. Anatomisches Institut der Universität zu Zürich. Personal communication. 2005.
14. Drug Safety Communications: updated recommendations to decrease risk of spinal column bleeding and paralysis in patients on low molecular weight heparins (Safety Announcement) [Internet]. c2013 [cited 2014 May 1]. Available from: http://www.fda.gov/downloads/Drugs/DrugSafety/UCM373735.pdf.
15. Vandermeulen EP, Van Aken H, Vermylen J. Anticoagulants and spinal-epidural anesthesia. Anesth Analg. 1994;79:1165–77.
16. de Sèze M, Sztark F, Janvier G, Joseph P. Severe and long-lasting complications of the nerve root and spinal cord after central neuraxial blockade. Anesth Analg. 2007;104:975–9.
17. Chin KJ, Karmakar MK, Peng P. Ultrasonography of the adult thoracic and lumbar spine for central neuraxial blockade. Anesthesiology. 2011;114:1459–85.
18. Balki M. Locating the epidural space in obstetric patients-ultrasound a useful tool: continuing professional development. Can J Anaesth. 2011;57:1111–26.
19. Karmakar MK, Li X, Kwok WH, Ho AM, Ngan Kee WD. Sonoanatomy relevant for ultrasound-guided central neuraxial blocks via the paramedian approach in the lumbar region. Br J Radiol. 2012;85:e262–9.

Chapter 41

Neuraxial Blocks: Spinal and Epidural Anesthesia

Herman Sehmbi, MBBS, MD, EDAIC, EDRA • Ki Jinn Chin, MBBS, MMed., FANZCA, FRCPC (✉)
Department of Anesthesia, Toronto Western Hospital, Toronto, Canada
e-mail: Kijinn.chin@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_41,

Introduction

Central neuraxial techniques are among the most reliable regional anesthesia techniques at the disposal of the anesthesiologist. Although they are relatively simple to perform, a thorough knowledge of underlying neuraxial anatomy and factors determining the spread and duration of anesthesia is critical to their success. Also, an understanding of the physiological effects and potential complications of these neuraxial techniques is paramount to ensure safe application of these methods.

Subarachnoid block or *spinal anesthesia* is the administration of an appropriate dose of local anesthetic into the cerebrospinal fluid and the subarachnoid space, which results in rapid onset of dense sensory and motor block that is suitable for surgical anesthesia. *Epidural anesthesia*, on the other hand, involves instillation of local anesthetic into the fat-filled epidural space and results in slower onset and a less-dense sensory and motor block, which is most often used to provide analgesia rather than surgical anesthesia. Spinal anesthesia is generally only performed at the lower lumbar intervertebral levels in order to avoid spinal cord injury; epidural anesthesia may however be performed at the lumbar, thoracic, or cervical levels depending upon the desired area of sensory blockade.

Applied Anatomy

A thorough, three-dimensional knowledge of spinal anatomy, including the vertebral column, the vertebral curves, the spinal ligaments, the meninges, and the spinal cord, is essential for performing spinal and epidural anesthesia and is particularly helpful when technical difficulty is encountered. The concept of spinal cord segments, the segmental spinal nerves, and respective dermatomes is also important in understanding the physiological effects of neuraxial techniques.

Surface Anatomy

Surface landmarks are generally used to locate a particular vertebral level. These include the vertebra prominens which is the spinous process of the C7 vertebra, the root of the spine of the scapula corresponding to the T3 vertebra, and the inferior angle of the scapula corresponding to the T7 vertebra. The inferior margin of the 12th rib lies at the level of the L1 vertebra, *Tuffier's line* (the line connecting the iliac crests) crosses the vertebral column at the level of the L4 vertebra, while the posterior superior iliac spine lies at level with S2 (Fig. 41.1a, b) [1, 2]. *However, it is important to note that these are only estimations and various factors including normal anatomic variation, subcutaneous fat, and patient position can render these surface landmarks inaccurate in any given individual* [3–5]. This may be overcome to some extent by the use of ultrasound which can improve the correct identification of intervertebral space levels [5–7].

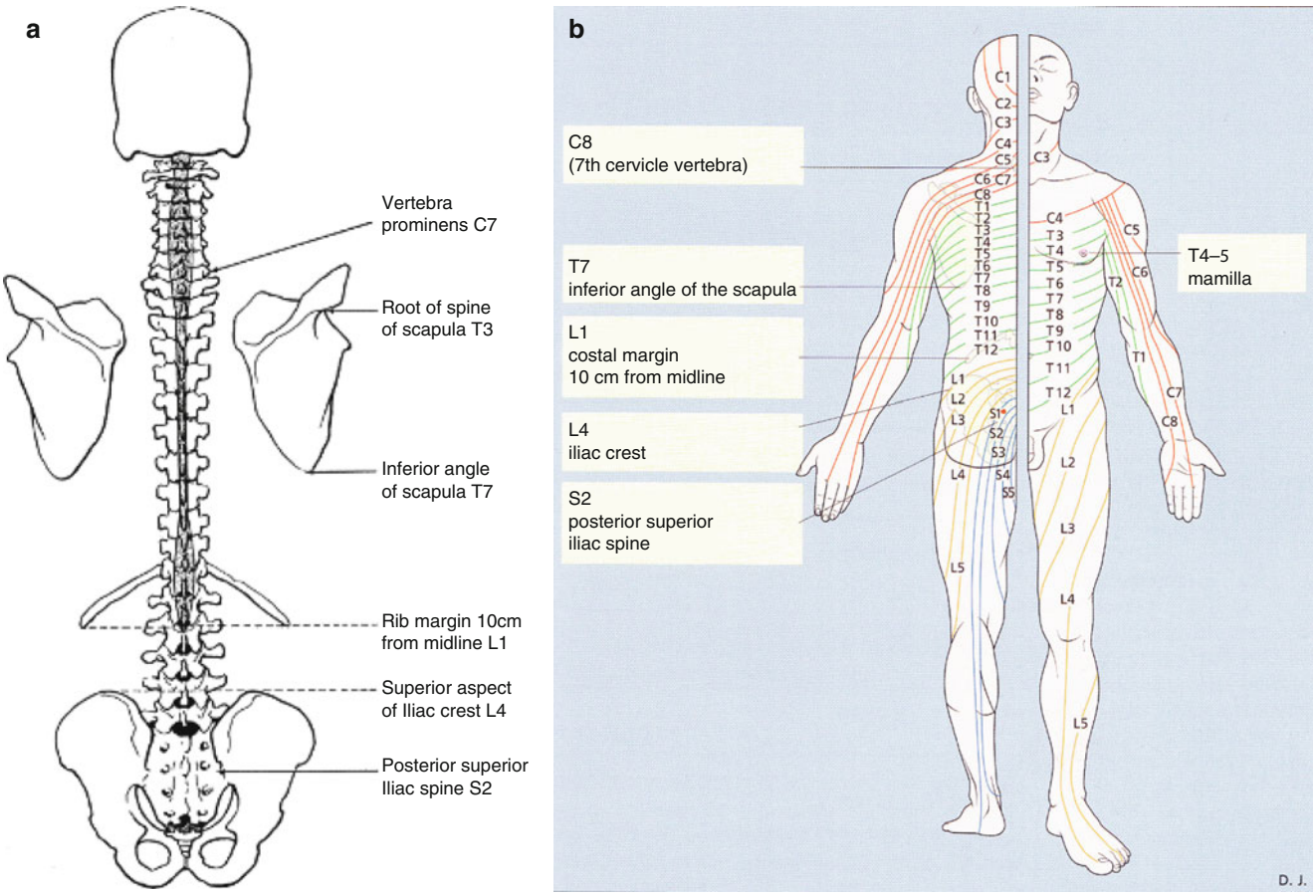


Fig. 41.1 (**a**) The common surface landmarks of the vertebral column. (**b**) The common surface landmarks of the vertebral column (With permission from Danilo Jankovic)

The spinous processes are angled caudally at the thoracic levels. However, they are nearly horizontal at cervical and lumbar levels (Fig. 41.2a–c) [8–10]. *As a result, cranial angulation of the needle is required when performing a thoracic epidural, while needle insertion perpendicular to the skin usually suffices in the lumbar spine.*

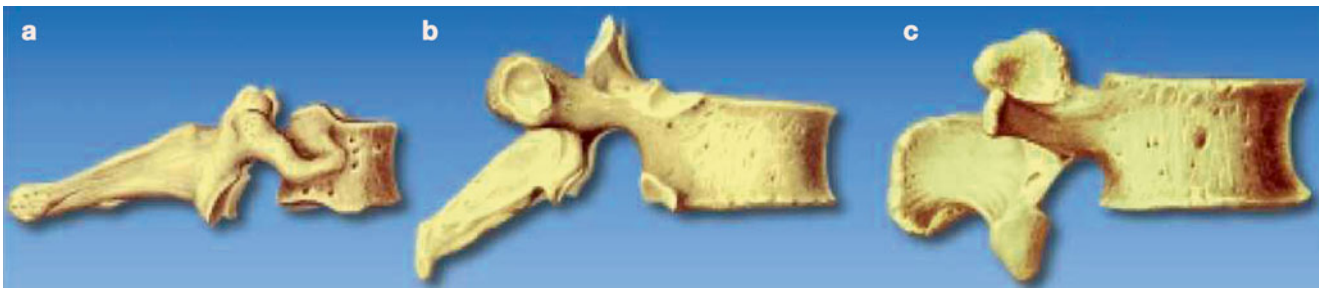


Fig. 41.2 Cervical, thoracic, and lumbar spinous processes (With permission from Danilo Jankovic). (**a**) C7 cervical vertebra (vertebra prominens, nuchal tubercle). (**b**) T8 thoracic vertebra. (**c**) L3 lumbar vertebra

The kyphotic thoracic curve and the lordotic lumbar spine determine the spread of subarachnoid local anesthetic in a supine adult. An injection of hyperbaric local anesthetic placed at the height of the lumbar lordosis will spread both caudad and cephalad to a variable extent (Fig. 41.3a, b). The cephalad extent of this spread is limited by the pooling of the solution at the mid-thoracic concavity [11].

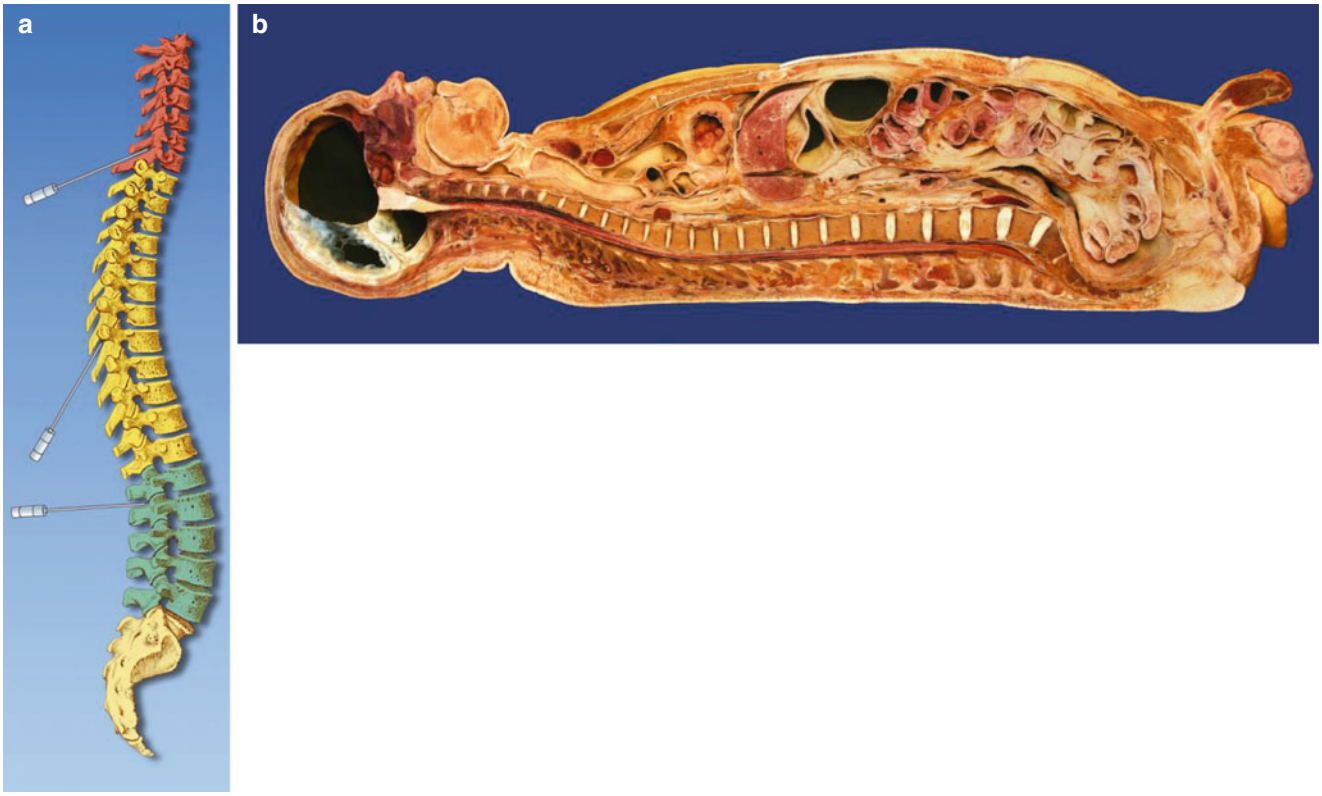


Fig. 41.3 (**a**) Anatomy: cervical, thoracic, and lumbar spine (With permission from Danilo Jankovic). (**b**) Paramedian sagittal section. Cervical, thoracic, and lumbar spine (With permission from Danilo Jankovic)

The three meningeal layers cover the spinal cord and protect it. The *dura mater* (Fig. 41.4) is the outermost meningeal layer and extends from the base of the skull to the second sacral vertebra. It is thickest in the posterior midline and thinner in the lumbar area than the thoracic or cervical levels [12, 13]. The *epidural space* is superficial to this layer and is the target for epidural anesthesia. The *subdural space* lies deep to this layer but superficial to the arachnoid mater.

The *arachnoid mater* lies deep to the dura mater and encloses the *subarachnoid space* and the cerebrospinal fluid (Fig. 41.4). Both spinal cord and spinal nerve roots are exposed to cerebrospinal fluid and thus are the sites of action of local anesthetic deposited in the subarachnoid space during a spinal anesthetic. Cysts in the subarachnoid space have been reported and may be a potential cause of inadequate spinal anesthesia [14, 15].

The *pia mater* is the innermost meningeal layer and covers the spinal cord (Fig. 41.4). The pia mater extends to the tip of the spinal cord and continues as the *filum terminale*, anchoring the spinal cord to the sacrum.

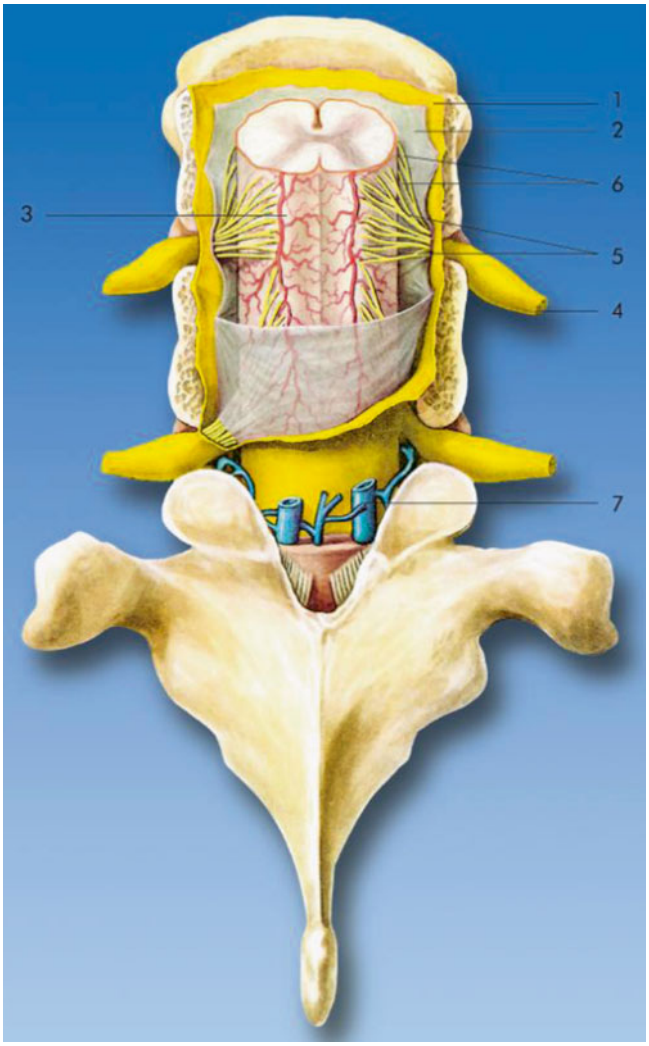


Fig. 41.4 Meninges. (*1*) Spinal dura mater, (*2*) arachnoid mater, (*3*) pia mater, (*4*) spinal nerve, (*5*) dorsal (posterior) nerve root, (*6*) ventral (anterior) nerve root, and (*7*) internal vertebral venous plexus (With permission from Danilo Jankovic)

A needle inserted in the *midline* will traverse the skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, dura mater, subdural space, and arachnoid mater to access the subarachnoid space (Fig. 41.5a, b). A *paramedian* insertion of the needle, on the other hand, bypasses the supraspinous and interspinous ligaments and can be useful in the older patient with degenerative spine disease and narrowed interspinous spaces.

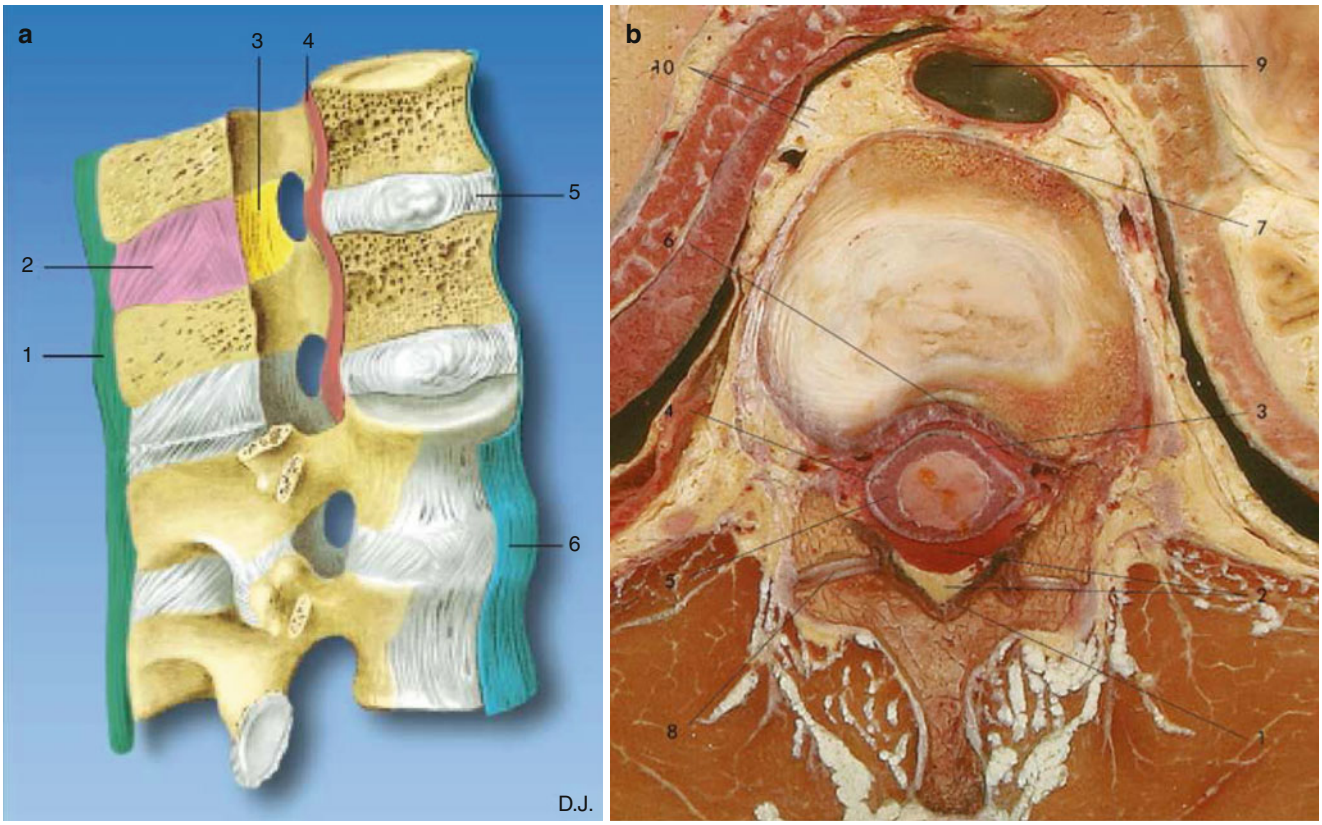


Fig. 41.5 (**a**) Sagittal section of the vertebral column illustrating the ligaments of the spinal cord. (*1*) Supraspinous ligament, (*2*) interspinous ligament, (*3*) ligamentum flavum, (*4*) posterior longitudinal ligament, (*5*) intervertebral disc, and (*6*) anterior longitudinal ligament (With permission from Danilo Jankovic). (**b**) Transverse dissection at the level of T9. (*1*) Ligamentum flavum, (*2*) posterior epidural space with fat, (*3*) anterior epidural space with veins, (*4*) spinal dura mater, (*5*) subarachnoid space with spinal cord, (*6*) posterior longitudinal ligament, (*7*) anterior longitudinal ligament, (*8*) zygapophysial joint, (*9*) aorta, and (*10*) sympathetic ganglion (With permission from Danilo Jankovic)

The length of the spinal cord varies with age. In the fetus, the spinal cord extends to the end of the vertebral column. However, by birth, the terminal end of the cord, the conus medullaris, extends only until L3 due to a faster growth of the vertebrae than the spinal cord. In the majority of adults, the conus ends at L1. *Spinal anesthetics must therefore be performed at the L2–L3 intervertebral space or lower to avoid the risk of spinal cord injury* [16]. The portion of the spinal cord that gives rise to the dorsal nerve root, ventral nerve root, and paired spinal nerves is called the *spinal cord segment*. The area of the skin supplied by a given cord segment and its spinal nerve is termed a *dermatome*. Qualitative assessment of sensory changes within the dermatomes allows rapid estimation of the spread of the local anesthetic within the subarachnoid or epidural space and is used to assess and document the extent of spinal or epidural anesthesia. Table 41.1 lists the most clinically relevant dermatomal levels and their corresponding surface landmarks.

Table 41.1 Surface landmarks and their relevant dermatomal levels (see Chap. 40 for figures)

Dermatomal level	Surface landmark
C8	Little finger
T1–T2	Inner arm
T4	Nipples
T6	Xiphoid
T7	Inferior border of scapula
T10	Umbilicus
T12	Inguinal ligament
L2–L3	Anterior thigh
L3	Patella
L5	Ankle
S1	Heel
S2	Back of the leg, back of the thigh, gluteal region
S3–S5	Perianal region

The ligamentum flavum and interspinous ligaments are *not continuous* (Fig. 41.5a). While the interspinous ligament extends from the spinous process above to the spinous process below, the ligamentum flavum extends from the lamina superior to a given intervertebral space to the lamina inferior to it. Failure of the left- and the right-sided ligamentum flavum to fuse in the midline has been described and may result in difficulty in localizing epidural space using a midline "loss-of-resistance" technique [17].

The epidural space is not a continuous space, but *segmented* along its length (Fig. 41.6) [18]. Cryomicrotome sectioning and imaging has shown that the various compartments of the epidural space are segmented by areas where the dura is directly fused with the bone. The lateral epidural compartment is divided by intervening pedicles which are in contact with the dura, while the posterior epidural compartment is divided by dural contact with bone beneath the cephalad half of each lamina. The anterior epidural compartment is divided by the attachment of the posterior longitudinal ligament to the intervertebral disc at each level. These *posterior*, *lateral*, *and anterior compartments* of the epidural space may impede the movement of injectate and may explain the unpredictable epidural drug spread that is occasionally seen [19].

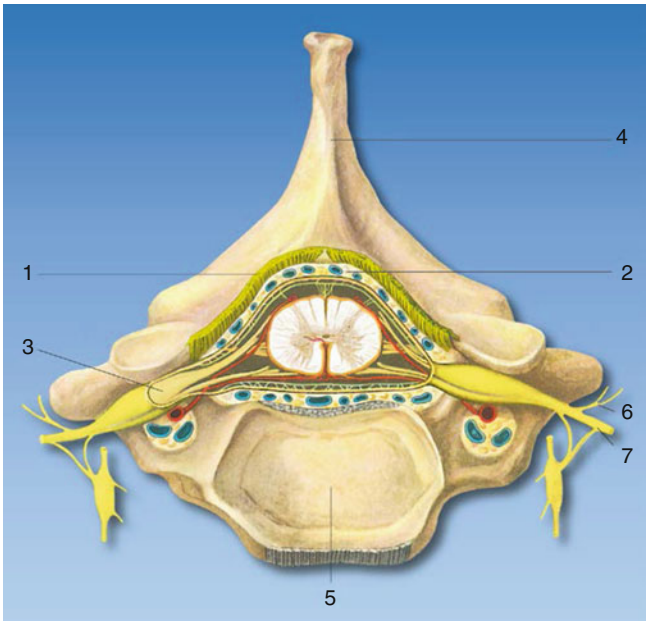


Fig. 41.6 Cross section of the epidural space. (*1*) Ligamentum flavum, (*2*) epidural space, (*3*) spinal ganglion, (*4*) spinous process, (*5*) body of vertebra, (*6*) dorsal branch of spinal nerve, and (*7*) ventral branch of spinal nerve (With permission from Danilo Jankovic)

Epidural fat in the epidural space (see also Fig. 41.5b) may play an important role in the pharmacokinetics of epidurally administered lipophilic drugs by acting as a *reservoir*. This may result in a delayed onset and a longer duration of action [20, 21]. A reduction in the epidural fat with age may partly explain the age-related changes in epidural dose requirements [22].

The depth of the epidural space from the skin varies with *body habitus*, being less in a thin individual and more in an obese or pregnant individual [23, 24]. Ultrasound is a useful tool for measuring this depth and has been shown to correlate well with the actual depth [25].

Physiological Effects of Neuraxial Block

The physiological effects of both subarachnoid and an epidural block are quite similar. However, the effects of an epidural block have a slower onset and are usually segmental in nature, due to the segmental spread of local anesthetic in the epidural space. These effects are summarized below.

Neurological Blockade

The injection of local anesthetic within the intrathecal or epidural space produces nerve blockade. This blockade first affects the smaller-diameter sympathetic fibers, before the larger myelinated sensory–motor fibers [26, 27]. As a result, *autonomic block* manifests before *sensory block*, which in turn precedes the *motor block*. Among the sensory modalities, the sequence of blockade is *temperature*, *pain*, *touch*, *pressure*, and finally *proprioception*.

Block dissipation occurs in the reverse manner with autonomic fibers being the last to recover. In general, more dilute solutions affect the sensory fibers preferentially, while higher concentration is needed to block the motor fibers. Sensory block extends two to four segments higher than motor block, and the sympathetic block extends two to four segments higher than the sensory block. This is termed *differential block* [28].

Cardiovascular Effects

Blockade of the thoracolumbar sympathetic nerves manifests as the cardiovascular effects that follow a neuraxial block, including *hypotension* and *bradycardia*. These effects are in proportion to the extent of the block produced and may be exaggerated in a hypovolemic patient. Hypotension is a result of arteriolar vasodilatation and venous pooling which diminishes preload. Bradycardia is partially due to blockade of cardiac accelerator fibers (T1–T5) [29] but is more often a result of decreased preload which activates cardiac reflexes involving intracardiac stretch receptors [30]. The Bezold–Jarisch reflex in particular is often invoked as an explanation for severe bradycardia and asystole following spinal anesthesia in young healthy adults [31].

In general, the sympathetic block following a spinal anesthetic is more rapid and greater in extent compared to an epidural. Thus, a gradually dosed epidural may be useful in providing hemodynamic stability. Additionally, a high thoracic epidural block (T1–T4) provides coronary vasodilatation and reduces work of the myocardium by reducing afterload and reducing the heart rate [32]. However, this unopposed vagal tone has been thought to contribute to bradycardia, asystole, or laryngospasm [33, 34].

Respiratory Effects

In a patient with normal lung function, neuraxial blocks produce a clinically insignificant impact on respiratory function [35]. However, in high thoracic blockade or in a patient with preexisting respiratory compromise, this effect may be significant. The paralysis of abdominal and intercostal muscles negatively impacts expiratory function causing a reduction in peak expiratory flows, expiratory reserve volume, and maximal minute ventilation. This may manifest as dyspnea. However, these effects are negligible in comparison to the pain relief obtained following thoracic or abdominal surgery, and overall improvement in postoperative outcome and a reduction in postoperative pulmonary complications are observed [36].

Spread of the local anesthetics to cervical segments can affect inspiration by blocking the phrenic nerve and diaphragmatic function. Respiratory arrest following a total spinal may be due to medullary hypoperfusion rather than an effect of local anesthetics per se [35].

Gastrointestinal Function

The splanchnic blockade (T6–L2) produced following a neuraxial block leads to unopposed parasympathetic activity. This results in increased gastrointestinal secretions, increased intestinal motility, and relaxation of sphincters. This contracts the bowel and allows better access during abdominal surgery. Also, improved visceral perfusion due to vasodilation may improve healing and contribute to an earlier return of bowel function following surgery [37, 38].

Nausea and vomiting, if observed, are usually secondary to increased vagal tone and hypotension. A higher block (above T5), accompanying hypotension, use of opioids, and a history of motion sickness are risk factors for developing nausea and vomiting [39].

Genitourinary

Neuraxial anesthesia does not affect renal blood flow since it is autoregulated. Sacral blockade produces an atonic bladder and an increased bladder sphincter tone. This may result in urinary retention until the resolution of the block.

Thermoregulation

Redistribution of heat following sympathetic block results in mild hypothermia. This induces thermoregulatory vasoconstriction and shivering above the level of the neuraxial block [40]. Epidural fentanyl may be used to abolish shivering during anesthesia [41].

Neuroendocrinal Effects

Neuraxial block effectively blocks the afferent innervation from the surgical site and is responsible for the inhibition of the surgical stress response that involves release of a variety of mediators (including catecholamines, vasopressin, growth hormone, renin, angiotensin, glucose, antidiuretic hormone, and thyroid-stimulating hormone). This effect is greater for a lumbar epidural than a thoracic epidural [42]. The magnitude of the stress response correlates with postoperative morbidity, and thus, its attenuation should be of benefit [43].

Indications

Spinal Versus Epidural Anesthetic

Neuraxial techniques (spinal and epidural anesthesia) at the lumbar levels allow for a temporary blockade of nerve conduction in the autonomic, sensory, and the motor fibers. However, it is important to recognize differences between the two. These are summarized in Table 41.2.

Table 41.2 Important differences between a spinal and an epidural anesthetic

Variables	Spinal anesthetic	Epidural anesthetic
Space accessed	Deposition of local anesthetic in subarachnoid space	Deposition of local anesthetic in epidural space
Time needed to perform	Takes less time (since it is usually a single-shot technique)	Takes more time (since it is usually a catheter technique)
Onset	Faster	Slower
Nature of blockade	Denser, therefore mainly employed as an anesthetic technique	Less dense, therefore mainly employed as an analgesic technique. However, with the use of sufficient volume and concentration, an anesthetic block can be produced
Duration	Dependent on dose and agent; fixed duration unless a catheter is inserted	Duration is flexible as a catheter is usually inserted
Cardiovascular effect	Rapid drop in blood pressure	A slower drop in blood pressure

Comparison to General Anesthesia

When compared to a general anesthetic, the central neuraxial techniques offer the following advantages:

- Avoidance of airway manipulation
- Avoidance of side effects of a general anesthetic (sore throat, nausea and vomiting, dental damage, malignant hyperthermia, aspiration of gastric contents, etc.)
- Simple and reliable block
- Predictable physiologic changes
- Minimal metabolic disturbances (in hepatic or renal disease)
- Preservation of consciousness
- Extension into postoperative analgesia

When compared to general anesthesia, neuraxial techniques have been associated with a reduction in cardiovascular adverse events, perioperative pulmonary complications, thromboembolic events, preserved gastrointestinal motility, development of chronic pain, surgical stress response, immune dysfunction, and morbidity and mortality [44–48].

However, general anesthesia is advantageous over neuraxial block when better control over hemodynamics is desired (such as in severe or critical aortic stenosis). It offers definitive control of the airway and allows for immediate postoperative pain assessment.

Indications

The application of neuraxial anesthesia depends on the following factors:

- The area of surgery
- The type and expected duration of the procedure
- The degree of muscle relaxation required
- The presence of concomitant disease
- The expected blood loss

The relevant indications for spinal and epidural anesthetic are considered in Table 41.3.

Table 41.3 Indications for spinal and epidural anesthesia (also see Fig. 41.7)

Spinal anesthetic	Epidural anesthetic
Surgical	*Surgical indications*
Spinal anesthesia is particularly advantageous for all types of surgical procedures below the level of the umbilicus	Procedures in the area of the lower extremities, hip joints, and inguinal region
Surgical procedures in the area of the lower extremities, hip joint, and inguinal region	Vascular surgery
Vascular surgery	Upper abdominal and thoracic procedures, in combination with general anesthesia
Prostate and bladder surgery	Urological procedures (prostate, bladder)
Gynecological and obstetric procedures	Gynecological and obstetric procedures
Surgery in the perineal and perianal region	Procedures in the perineal and perianal region
Pain therapy	Interventional radiology
Chemical intraspinal neurolysis with phenol in glycerol or alcohol (in advanced stages of malignant disease)	*Postoperative and post-traumatic pain therapy*
	Usually in combination with local anesthetics and opioids
	Therapeutic block with injection of depot corticosteroids at caudal, lumbar, or cervical levels
	Epidural injection of homologous blood or dextran or fibrin glue patch in post-dural puncture headache

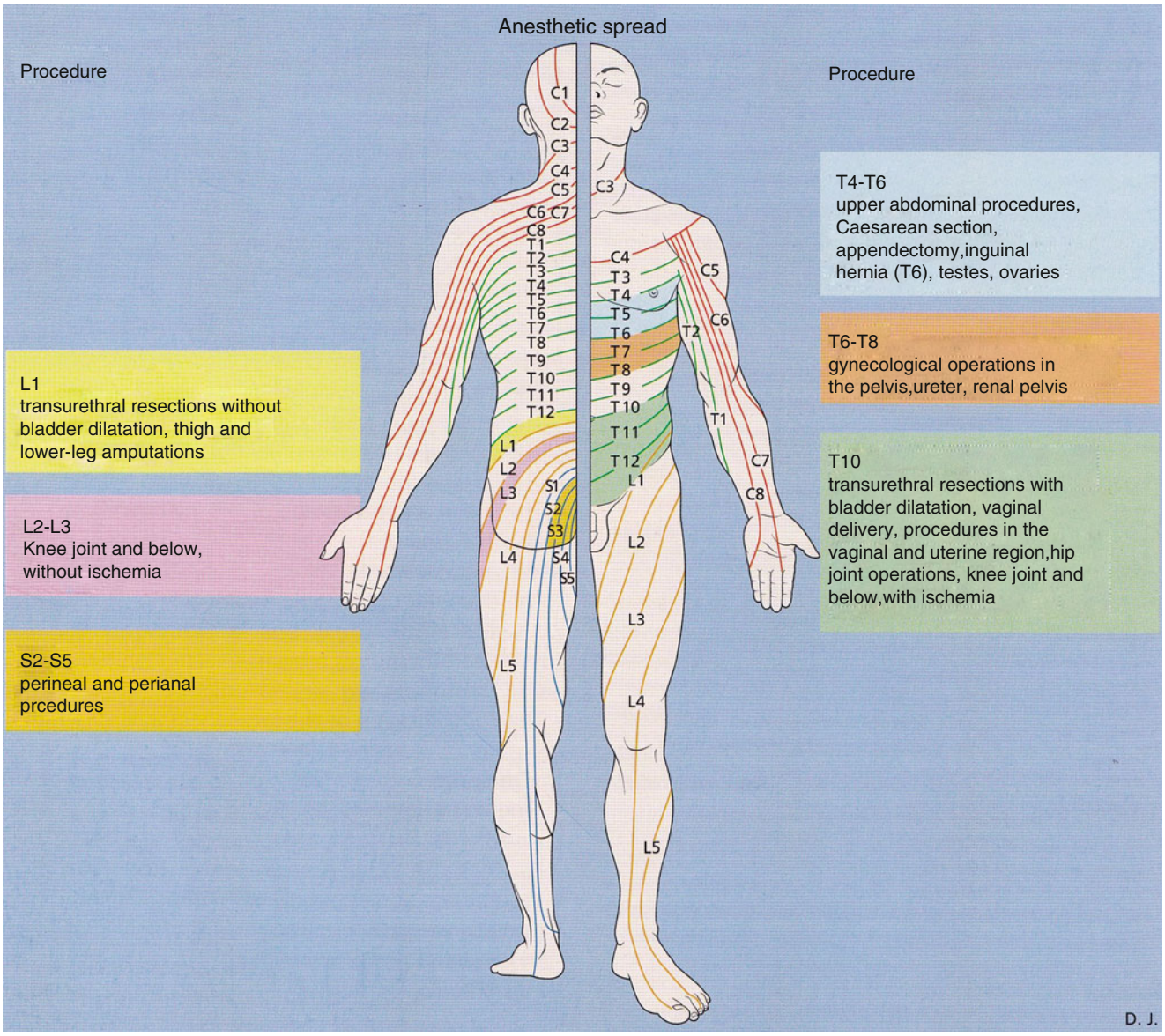


Fig. 41.7 Location of the procedure and required sensory spread of local anesthetic (With permission from Danilo Jankovic)

Contraindications

These are summarized in Table 41.4.

Coagulopathy, whether iatrogenic or idiopathic, is now considered a relative contraindication. According to the consensus statements by the American Society of Regional Anesthesia and Pain Medicine (2010), epidural block can be placed 4 h after the last dose of heparin. NSAIDs including aspirin are not a contraindication to epidural placement provided that catheter placement is uncomplicated (one or two attempts). LMWH should be held at least 12 h before placement of catheter and 2 h after its removal. Epidural placement is relatively safe with international normalized ratio (INR) <1.5. If an epidural vein is punctured, subcutaneous heparin administration should be held at least 2 h and LMWH held at least 24 h. GIIa/IIIb inhibitors should be withheld for at least 4 weeks after epidural placement. Epidural placement is best avoided for 7 days after clopidogrel and 14 days after ticlopidine. For a further review on the use of neuraxial techniques in anticoagulated patients, patients on antiplatelet drugs, or patients with coagulopathy, please refer to the "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)" [49].

Table 41.4 Contraindications to central neuraxial block

Absolute	Relative
Patient refusal	Coagulopathy: coagulation disorders or anticoagulant therapy
Sepsis, local infections at the injection site, or immune deficiency	Spinal pathology including severe spinal deformities, arthritis, osteoporosis, intervertebral disc prolapse, spinal canal stenosis, post-spinal surgery, and spinal metastases
Severe decompensated hypovolemia, shock	Unknown duration of surgery
Severe specific cardiovascular diseases of myocardial, ischemic, or valvular origin	Mild valvular disease (aortic stenosis)
Acute cerebral or spinal cord diseases	Preexisting neurological deficits (radiculopathies, peripheral neuropathies, multiple sclerosis)
Increased intracranial pressure	
Indeterminate neurological disease	
Hypersensitivity to local anesthetic agents	

Performing a Spinal Anesthetic

Before performing a spinal anesthetic, a full discussion of the risk–benefit and informed consent should take place.

Preparation and Materials

- Check that the emergency equipment is complete and in working order (intubation kit, emergency drugs); sterile precautions, intravenous access, and anesthetic machine.
- Start an intravenous infusion and ensure adequate volume loading (250–500 ml of a balanced electrolyte solution).
- Vasopressors such as ephedrine, phenylephrine, or metaraminol should be readily available.
- ECG, noninvasive blood pressure, and pulse oximetry monitoring are essential.
- Skin prep using an alcohol, iodine, or chlorhexidine. Note that if chlorhexidine is used, extreme care must be taken to prevent its inadvertent introduction into the neuraxis, as it has been implicated in chemical arachnoiditis [50, 51].
- Local anesthetic for skin infiltration and intrathecal injection.
- *Spinal needles* (Fig. 41.8a–c) are commonly of the following two types:

– *25–27-G spinal needles with conical tip* (pencil-point)—e.g., Sprotte, Pencan, and Whitacre—in current standard use. When the dura is penetrated with these needles, the dural fibers are separated and then closed together again. This reduces the risk of post-dural puncture headache [52, 53].
– *25–27-G spinal needles with Quincke tip*. The needle bevel should be directed laterally during the puncture in order to pass through the dura in a longitudinal direction [54]. A 22-G needle may be used where increased stiffness is desired to facilitate needle redirection—e.g., in older patients with narrowed interlaminar spaces or when there are difficulties with positioning.

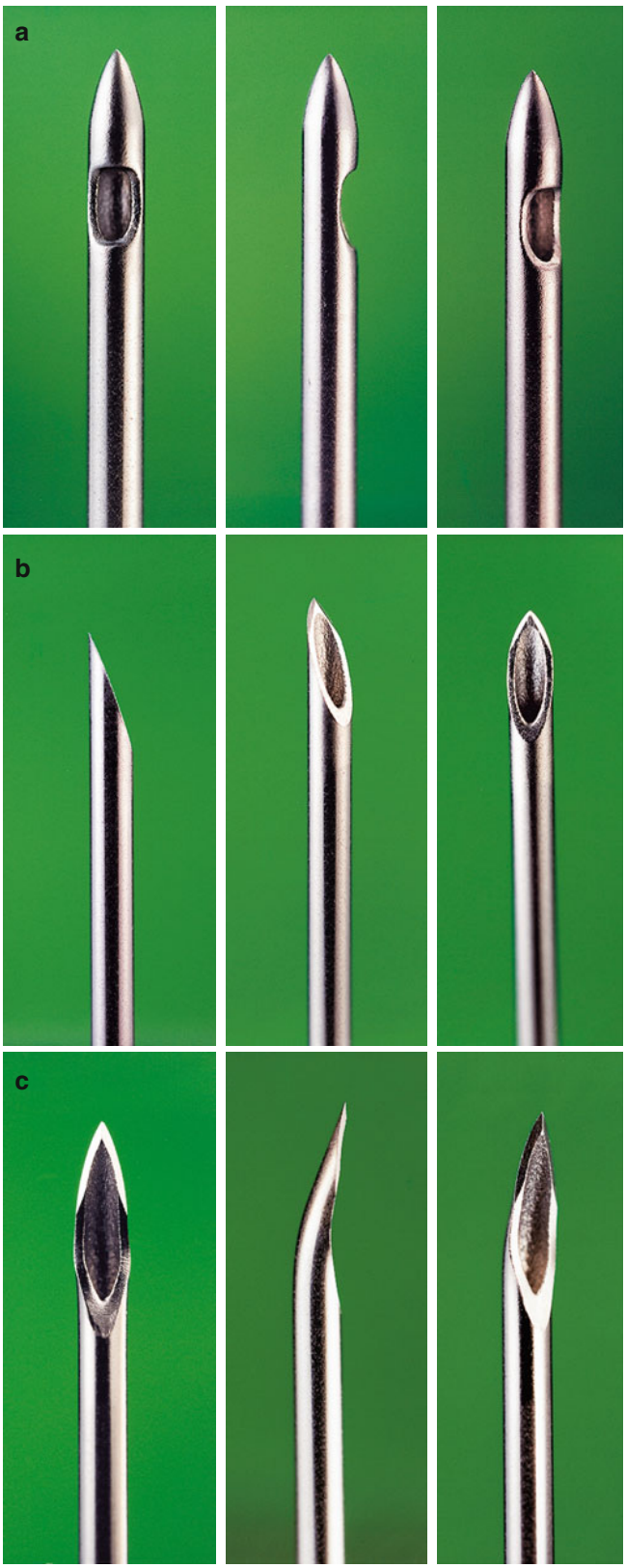


Fig. 41.8 (**a**–**c**) Spinal needles. (**a**) Pencil-point, 25 G. (**b**) Quincke tip, 27 G. (**c**) Atraucan Special Cut, 26 G (With permission from Danilo Jankovic)

Patient Positioning

Optimal patient positioning during puncture and during the fixation phase of the local anesthetic is a prerequisite for successful spinal anesthesia. The following positions may be used:

- Lateral decubitus position
- Sitting
- Prone

In all three positions, it is important to locate the *midline* and to follow it during the entire injection procedure. Lumbar lordosis must be minimized.

Lateral Decubitus Position

The assistant stands in front of the patient. If the anesthetist is right-handed, the patient is placed in the left lateral position. The patient is asked to adopt a *hunchback* position (legs flexed up against the abdomen and chin flexed down onto the chest) in order to flex the lumbar spine and allow optimal expansion of the intervertebral spaces. It is important here for the spine to be parallel and for the intercristal line and the line connecting the two scapular tips to be perpendicular to the operating table (Fig. 41.9).

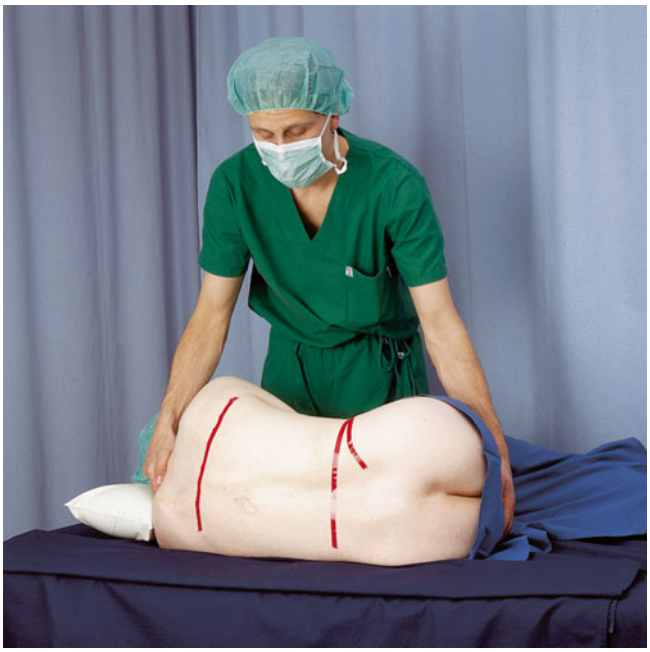


Fig. 41.9 Position: lateral decubitus (With permission from Danilo Jankovic)

Advantages

- More comfortable for the patient and thus particularly suitable for frail patients (risk of collapse).
- The reduction in blood pressure is less marked [55].
- When hyperbaric solutions are used, unilateral anesthesia is more easily obtained.
- Can be used in pregnant patients (since the lateral tilt or lateral decubitus position reduces the extent of aortocaval compression) [56].

Disadvantages

- Difficulty may arise in the correct identification of the midline, especially if the pelvis is tilted too anteriorly.

Sitting Position

The patient is seated on the edge of the operating table and is supported by an assistant standing in front of him or her (Fig. 41.10). It may also be helpful to provide a padded Mayo stand, an adjustable table, or a similar device for the patient to lean on for additional support.

Advantages

- When palpation of the spinous processes is difficult (e.g., in obese patients or those with spinal deformities), it is easier to locate the midline in the sitting position.
- Facilitates the performance of a saddle block with hyperbaric local anesthetic when anesthesia in only the perineal or perianal region is required.

Disadvantages

- This position may lead to orthostatic hypotension (and risk of collapse) and should be avoided in frail and heavily sedated patients [55].
- More support and assistance may be required to keep patient upright.

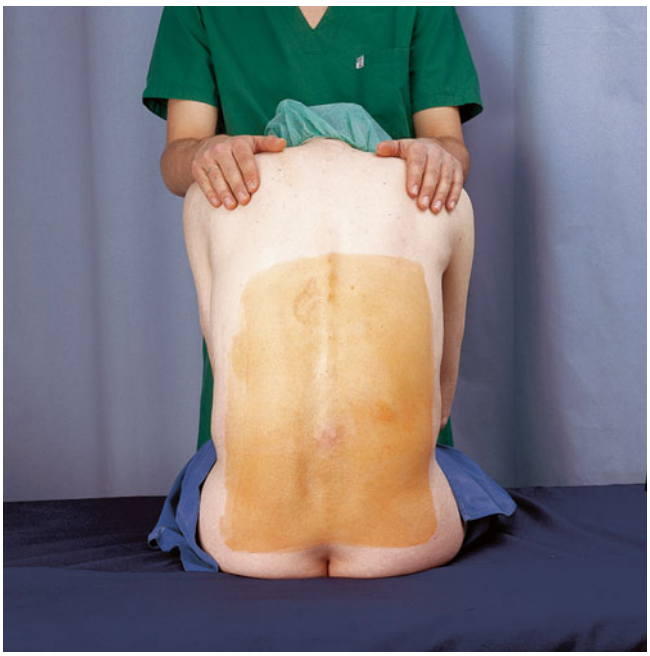


Fig. 41.10 Position: sitting (With permission from Danilo Jankovic)

Prone Jackknife

This position is only used in the very rarely practiced hypobaric technique for spinal anesthesia (procedures in the rectum, perineum, sacrum, lower spine) [57]. An assistant and subsequent repositioning of the patient are not required (Fig. 41.11).

Fig. 41.11 Position: prone ("jackknife" position) (With permission from Danilo Jankovic)

Injection Technique

The subarachnoid space may be accessed using a midline approach, a paramedian approach, or a Taylor's approach. These are discussed below.

Median Approach (Midline)

Landmarks

The injection is carried out in the midline below the L2 segment (conus medullaris), usually between the spinous processes of L2/L3 or L3/L4 (depending on the desired level of anesthesia). The patient is asked to draw the legs tightly up to the abdomen and to place the chin on the chest. A line is drawn from one iliac crest to the other. This connection (Tuffier's line) crosses either the spinous process of L4 (50 %) or the intervertebral space of segments L4/L5. However, this palpatory method alone may be unreliable, and ultrasound may help to correctly identify the vertebral levels [7].

The spinous processes and intervertebral spaces are palpated to identify the *midline*, which is the important landmark. It is recommended that the index and middle fingers of the nondominant hand be used to palpate and identify the chosen interspace, as they can be subsequently used to fix the overlying skin, which is important for precision and accuracy in needle insertion (Fig. 41.12).

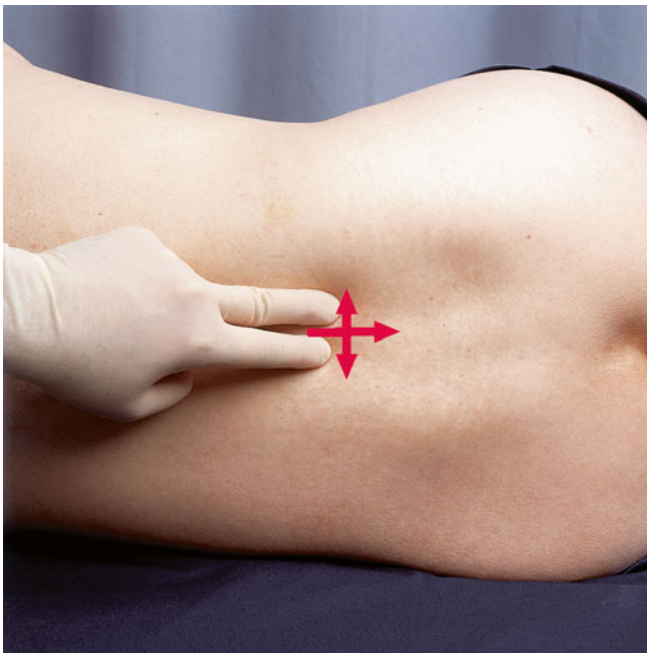


Fig. 41.12 Palpation of the intervertebral space (With permission from Danilo Jankovic)

Strict Asepsis

Thorough, repeated, and wide skin prep and drying and covering of the injection site with a drape.

Local Anesthesia

The skin and supraspinous and interspinous ligaments are anesthetized with 1–1.5 ml of a local anesthetic (e.g., 1 % lignocaine). The injection is carried out between the spread index and middle fingers of the left hand (Fig. 41.13). In the patient with less-distinct surface landmarks, the local anesthetic needle may be used to explore the underlying anatomy. Bony contact at a relatively shallow depth indicates contact with the tip of the spinous process and the location of the midline. Firm resistance to further injection of local anesthetic indicates that the needle tip lies within the interspinous ligament in the midline and not in the paraspinous muscles on either side.

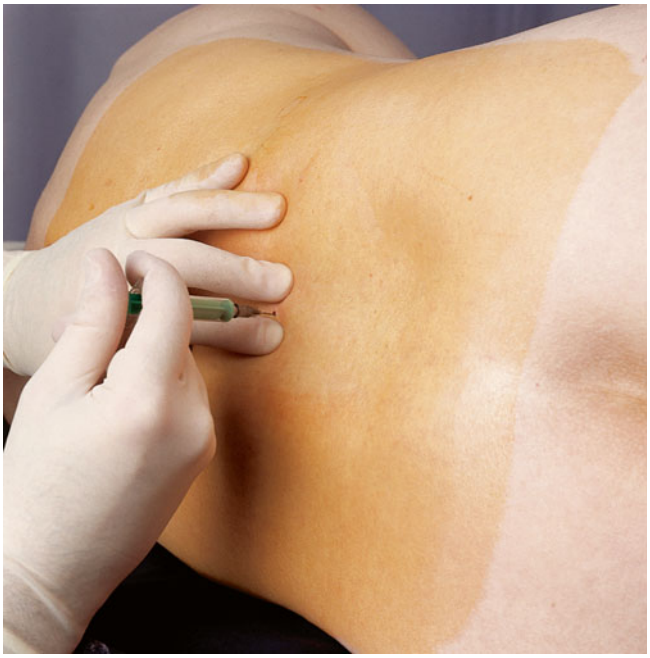


Fig. 41.13 Local anesthesia (With permission from Danilo Jankovic)

Injection

Advancing the Introducer

Without moving the spread index and middle finger of the *left* hand away from the intervertebral space (which prevents inadvertent movement of the skin overlying the chosen interspace), the introducer is grasped between the thumb and index finger of the *right* hand and advanced parallel to the operating table and slightly cranially (10°) deep enough for it to sit firmly in the interspinous ligament (Fig. 41.14). It should be ensured that the *midline position* is maintained. After this, the introducer is fixed with the thumb and index finger of the left hand, with the dorsum of the hand lying firmly on the patient's back.

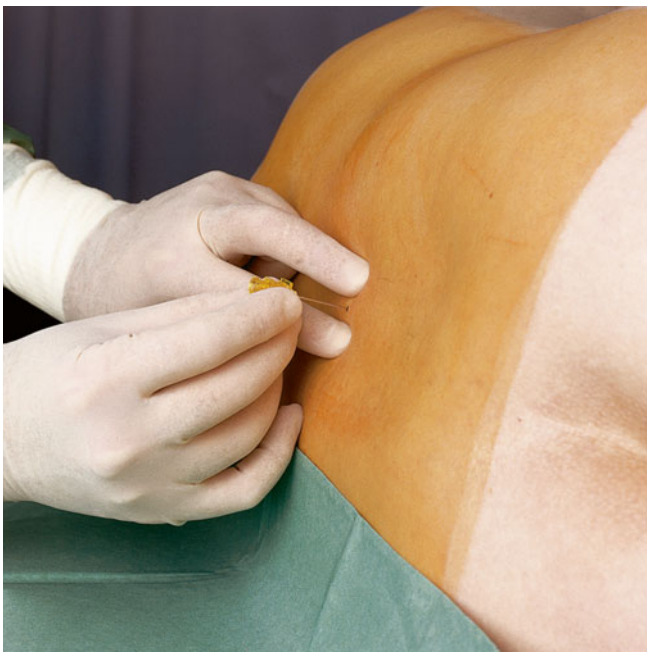


Fig. 41.14 Advancing the guiding cannula (With permission from Danilo Jankovic)

Introducing the Spinal Needle, Puncture of the Subarachnoid Space

The spinal needle, held between the thumb and index finger (or middle finger) of the right hand, is introduced through the interspinous ligament, ligamentum flavum, epidural space, and dura/arachnoid as far as the subarachnoid space. Penetration of the ligamentum flavum is usually evident by the "rubbery" resistance to needle advancement. A characteristic "dural click" may be felt when the subarachnoid space is reached; however, this does not always occur (Fig. 41.15). When a Quincke needle is used, it should be ensured that the needle bevel is directed laterally, so that the dura is punctured in a longitudinal direction.

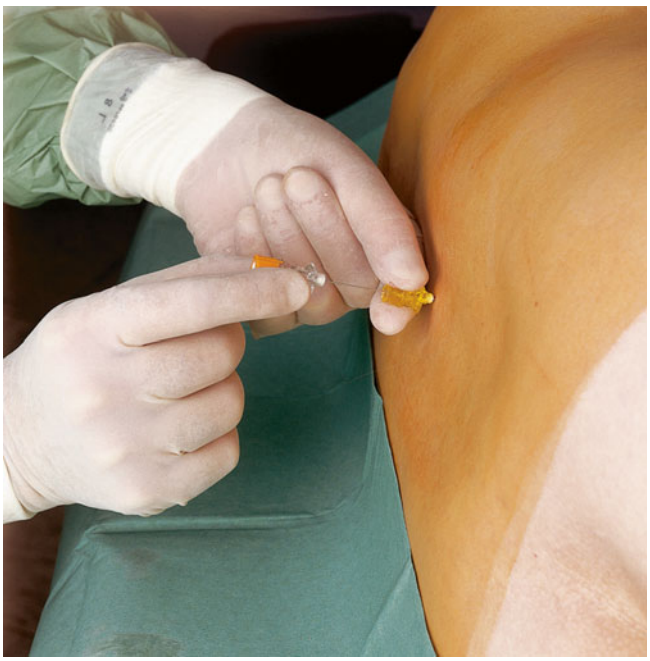


Fig. 41.15 Introducing the spinal needle. Puncture of subarachnoid space (With permission from Danilo Jankovic)

Removing the Stylet

The following may occur here:

- *CSF flows freely*
- The injection needle is advanced 1 mm and fixed between the thumb and index finger of the left hand, which is supported on the patient's back. The desired amount of local anesthetic can now be injected (Figs. 41.16 and 41.17a, b).
- With the single-injection technique, aspiration of CSF (0.1 ml) should be attempted immediately before and after injection of local anesthetic. The subarachnoid injection is made at the rate of 1 ml per 5 s.
- *Blood in the CSF*
- Slightly bloody CSF which clears quickly (spontaneously or after aspiration) usually occurs after penetration of an epidural vein on the way into the subarachnoid space. The local anesthetic can be injected.
- However, backflow of frank blood indicates that the injection needle is likely positioned within a vein. A new attempt at puncture must be made and possibly in a different intervertebral space.
- *No CSF flow*
- Rotation of the needle to all four quadrants and careful aspiration may help. The needle may also be advanced slightly with the stylet in place.
- If no CSF flows in spite of all these measures, the needle should be removed and the procedure repeated with a different needle direction or at a different interspace.

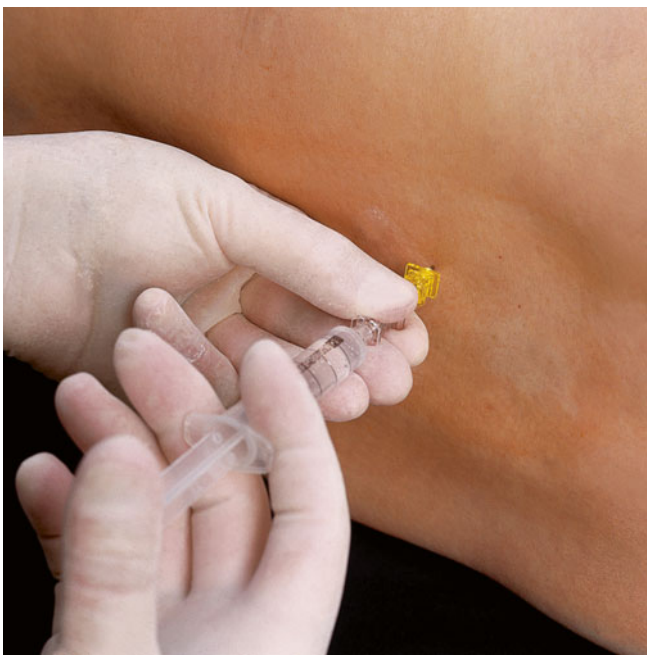


Fig. 41.16 Removing the stylet. Subarachnoid injection (With permission from Danilo Jankovic)

- Unexpectedly deep bone contact suggests that the posterior side of the vertebra or an intervertebral disc has been reached. CSF appears in most cases after the needle has been slightly withdrawn and aspiration has been repeated.
- *Pain or paresthesias during puncture*
- Pain or paresthesia prior to entry into the epidural or intrathecal space is most often due to needle contact with the facet joint, which forms the lateral border of the interlaminar space. The needle should be directed slightly more medially, away from the side on which the pain or paresthesia occurred. It is not uncommon for a transient paresthesia radiating down one leg to occur upon needle entry into the intrathecal space; this signals contact with the cauda equina. If it persists, the needle must be withdrawn slightly or repositioned. When paresthesias occur during the injection, the needle must be repositioned before any further drug is injected.

  The local anesthetic must never be injected without evidence of CSF! The location and distribution of paresthesias arising during the puncture procedure must be recorded.
- Experience shows that failure is usually due to deviation of the needle from the midline or excessive cranial angulation of the needle.

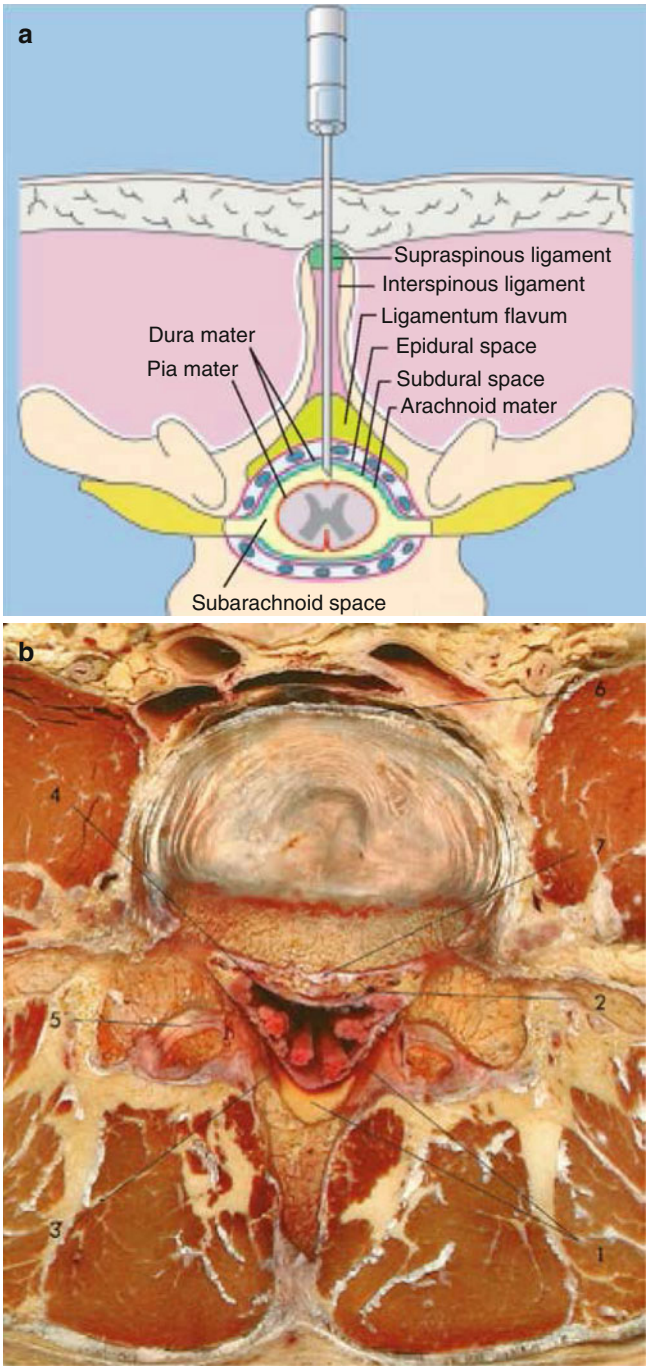


Fig. 41.17 (**a**) Subarachnoid position of the needle (With permission from Danilo Jankovic). (**b**) Transverse dissection at the level of L4/L5. (*1*) Posterior epidural space with fat, (*2*) anterior epidural space with veins, (*3*) spinal dura mater, (*4*) subarachnoid space and cauda equina, (*5*) zygapophysial joint, (*6*) anterior longitudinal ligament, and (*7*) posterior longitudinal ligament (With permission from Danilo Jankovic)

Paramedian Approach (Lateral, Paraspinal)

In this technique (Figs. 41.18 and 41.20), the supraspinous and interspinous ligaments are avoided, so that the ligamentum flavum is the primary target on the way to the subarachnoid space.

Procedure

This technique can be used in all the patient positions mentioned above. Flexion of the spine is not essential. The caudal edge of the spinous process is marked. The injection site is located 1–1.5 cm lateral and caudal to this. The puncture is carried out in a cranio-medial direction, at an angle of about 10–15°. The dura is reached after about 4–6 cm. Most mistakes arise when the needle is angled too cranially or when the needle entry point is too lateral.

This technique can be used in:

- Degenerative changes in the spine
- Older patients with marked calcification of the supraspinous and interspinous ligaments [58]
- Obesity
- Fractures or other pathological conditions in which pain makes it impossible to flex the spine

Taylor's Approach

This lumbosacral approach (Figs. 41.19 and 41.20) is a paramedian injection via the intervertebral space of L5 and S1, the largest interlaminar space in the spinal region.

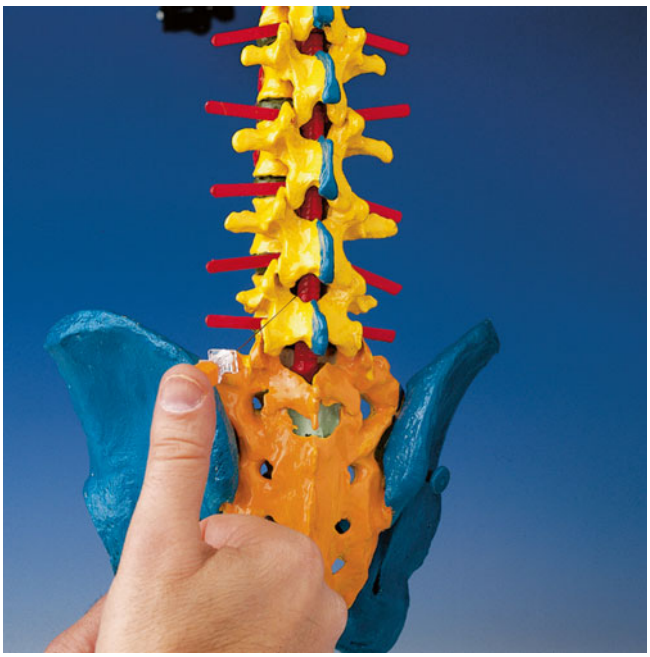


Fig. 41.18 Paramedian approach (With permission from Danilo Jankovic)

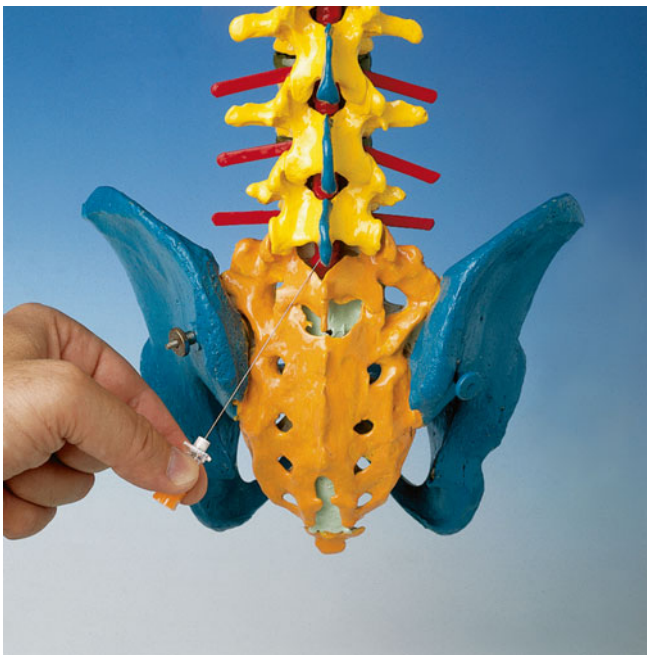


Fig. 41.19 Taylor's approach (With permission from Danilo Jankovic)

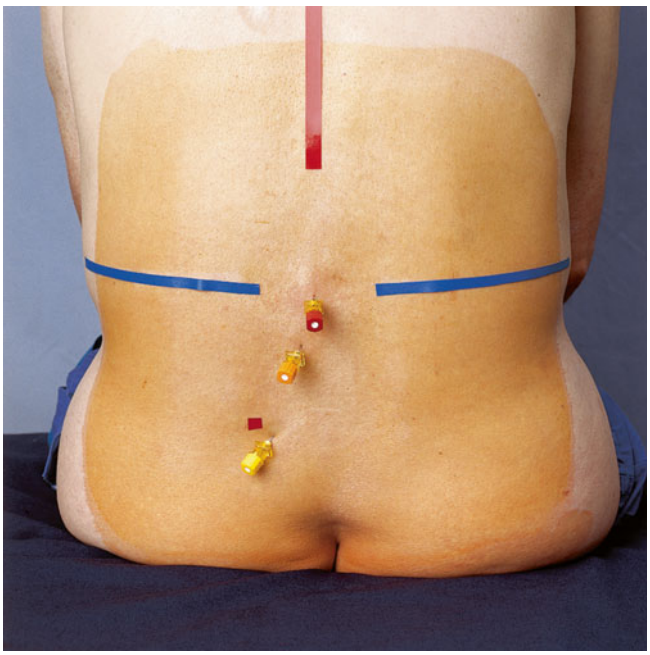


Fig. 41.20 Puncture of the subarachnoid space: (*1*) median, (*2*) paramedian, (*3*) Taylor (With permission from Danilo Jankovic)

Procedure

The injection site is located about 1 cm medial and about 1 cm caudal to the posterior superior iliac crest. The injection needle is advanced in a cranio-medial direction and at an angle of about 55°. If it touches the periosteum (sacrum), the needle must be withdrawn and its direction must be corrected.

The indications for this access route include procedures in the perineal and perianal region, as spread to the higher spinal levels may not always reliably occur.

Unilateral Spinal Anesthesia

Unilateral spinal anesthesia is intended to block only the anterior and posterior spinal nerve roots on the side being operated on, while the contralateral side—and particularly its sympathetic fibers—remains unblocked. This leads to a reduced incidence of hypotension.

Indications

- Surgical and orthopedic procedures on the lower extremities

Procedure

- Patient positioning: lateral decubitus position, lying on the side that is to be operated on.
- Injection technique: this is the same as for conventional spinal anesthesia. The opening of the pencil-point needle is rotated to the operating side, and the desired amount of local anesthetic is slowly injected [59]. A hyperbaric solution of local anesthetic such as bupivacaine should always be used for maximum effectiveness.
- Patient position after the injection: the patient remains lying on the side that is to be operated on for about 20 min.

Advantages

- Reduction in the extent of the sympathetic block (by about 70 %), since smaller volumes and slower injection of the local anesthetic mean that fewer spinal segments are involved [60]
- Hemodynamic stability (hypotension is only observed in 5 % of patients)
- Faster recovery from anesthesia
- Suitable for outpatient procedures [61]
- Greater acceptance by patients

Disadvantage

- Strict unilateral anesthesia is only achieved if adequate time is allowed for the local anesthetic to settle on the operative side. However, a differential block of some degree can almost always be achieved.

Continuous Spinal Anesthesia

The insertion of a catheter into the subarachnoid space allows a continuous or repeated intermittent dosing of local anesthetic.

Indications

- Lower abdominal and lower limb surgery in elderly patients and high-risk patients [62]
- Postoperative pain relief
- Chronic pain relief (in cancer patients)

Procedure

- The procedure is similar to a standard spinal anesthetic. However, usually a 20–22-G spinal needle is used to gain access to the subarachnoid space, and a 25–27-G catheter is threaded (usually 2–3 cm) into the subarachnoid space (Fig. 41.21).

Advantages

- A smaller initial dose of local anesthetic can be injected without concern about inadequate block height or duration. This helps prevent hypotension due to excessive sympathetic block caused by large doses or greater spread of local anesthetic [62].
- Ability to prolong spinal anesthetic as needed.

Disadvantages

- Higher risk of post-dural puncture headache in younger population [63].
- Use of small micro-catheters (less than 24 G) may predispose to sacral pooling of local anesthetic leading to *cauda equina syndrome* and is therefore not recommended [64].

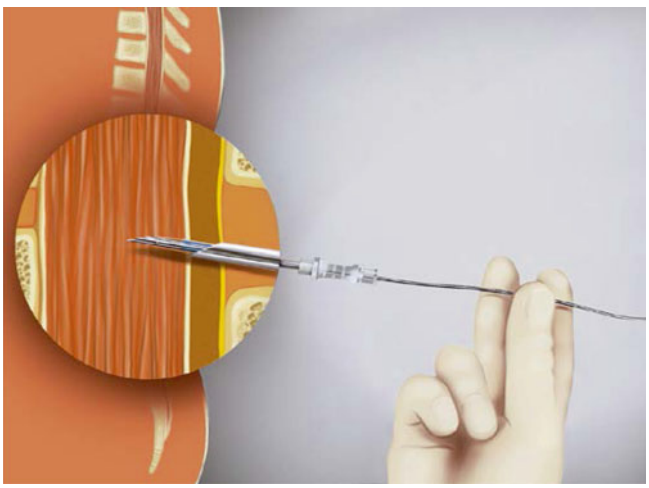


Fig. 41.21 Introducing the catheter in the subarachnoid space (With permission from Danilo Jankovic)

Management of the Patient After Intrathecal Injection

Patient Positioning

The level of the anesthetic spread is controlled by patient positioning measures and checked with cold tests at intervals of 2–5 min.

Hyperbaric Spinal Anesthesia

- Lateral decubitus position: the patient remains on the side of surgery for 10–15 min if unilateral anesthesia is desired. The patient is laid supine if bilateral anesthesia is required.
- Sitting position: the patient is immediately laid supine to allow the anesthetic to spread cephalad. The patient remains sitting if sacral spread is desired.

Hypobaric Technique

- Hypobaric spinal anesthesia is ideal for perineal and perirectal surgeries requiring a prone "jackknife" position, so that the patient does not need to be repositioned (Fig. 41.11).
- The hypobaric technique has also gained popularity in major hip surgery, which is performed in the lateral position. Thus, repositioning of the patient is often not needed when the spinal is placed in the lateral decubitus position, with the operative side nondependent. Compared to isobaric bupivacaine, a hypobaric solution demonstrated a significant delayed block recession and time until analgesia, for total hip arthroplasty [65].

Isobaric Spinal Anesthesia

- Horizontal positioning is adequate; other positions have no significant influence on the spread of the anesthesia.

Fixation Phase

- The phase immediately after injection of the local anesthetic is particularly critical and requires precise monitoring. The fixation phase lasts about 10–15 min.

Patient Monitoring

- Sympathetic block following a spinal anesthetic results in a drop in the blood pressure and heart rate. This may be profound in elderly and volume-depleted patients. Bradycardia may occur after the peak of sympathetic block is achieved (30–60 min after spinal injection) due to the blockade of cardiac accelerator fibers. Unexpected bradycardia and cardiac arrest may occur in young healthy patients [66]. Thus, frequent monitoring of the vitals (heart rate, blood pressure, and oxygen saturation) is advocated during and after the conduct of a spinal anesthetic.
- Supplemental oxygen administration is recommended after a spinal anesthetic, especially if sedation is used for patient comfort.
- End-tidal carbon dioxide monitoring is also used to monitor the respiratory rate.
- Postoperatively, the patient should be monitored until the effects of spinal anesthetic have receded.

Block Assessment

- Injecting a local anesthetic into the subarachnoid space blocks *autonomic*, *sensory*, and *motor* function. The main targets of local anesthesia are the posterior roots with the ganglia, the anterior roots of the spinal nerves, the autonomic nerve fibers, and mixed neural trunks.
- The spread of the anesthesia should be checked at short intervals (2–5 min) and confirmed (with a cold spray or pinprick) shortly before the start of the operation [67, 68]. The first sign of an effect on the spinal nerve roots is a subjective sensation of *warmth* in the feet. The further development of the block encompasses touch, deep pressure, motor function, vibration sensitivity, and positional sense.
- Motor function is completely blocked at the site of the greatest concentration of the local anesthetic. Sensory block extends two to four segments higher than motor block, and the sympathetic block extends for a further two to four segments higher than the sensory block.
- Resolution of the block is marked by a return of motor function, followed by the sensory modalities. The autonomic function is the last to recover [28].

Performing an Epidural Anesthetic

Preparation and Materials

Preparation for an epidural anesthetic is similar to that for a spinal block. This has already been discussed in Sect. *Patient Positioning*.

Epidural needles: The epidural needles required to perform an epidural block are generally larger in gauge than spinal needles. Additionally, they may have distinct tip design to facilitate entry into the epidural space and introduction of the catheter. Some of these are already discussed in Sect. *Patient Positioning*. Of these, Tuohy-tip epidural needles are most commonly used.

Positioning of the Patient

An epidural block can be performed with the patient in lateral decubitus or sitting position. While the former is commonly used in obstetric patients, the latter is preferred in other patient groups. Prone position is rarely used. The pros and cons of these positions have been already discussed on p. 514 (Patient Positioning).

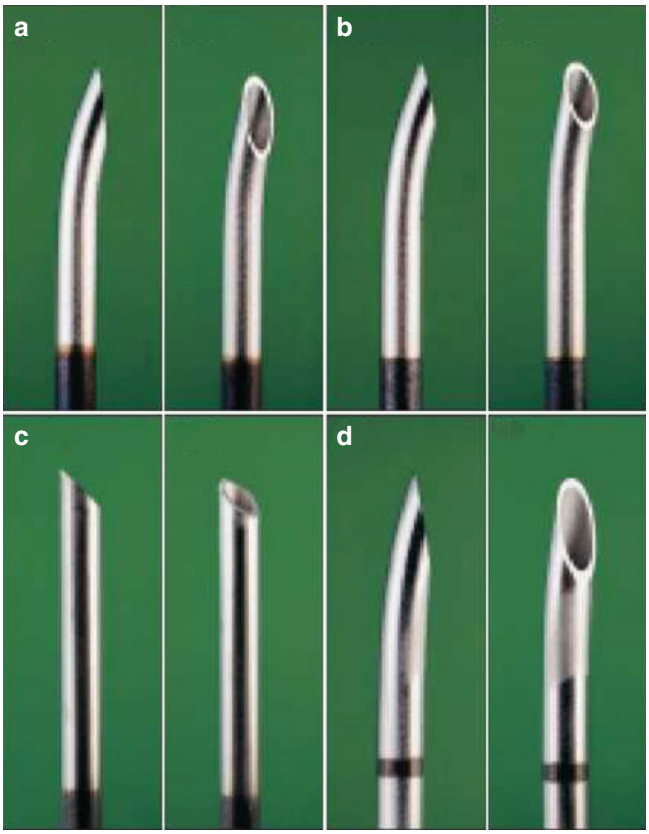


Fig. 41.22 (**a**–**d**) Epidural needles. (**a**) Tuohy, (**b**) Hustead, (**c**) Crawford, (**d**) Weiss (With permission from Danilo Jankovic)

Performing a Lumbar Epidural

Lumbar epidural space may be accessed using a midline approach which is most commonly used or using a paramedian approach. These are discussed below.

Midline Approach

Landmarks

The injection is carried out in the midline below the L2 segment (conus medullaris) (Fig. 41.23a, b), usually between the spinous processes of L2/L3 or L3/L4. The intervertebral space is palpated, and the midline is located to serve as the most important signpost. In the midline, the ligamentum flavum is at its thickest, the epidural space is widest, and the blood vessels are at their smallest [69].

Strict Asepsis

Thorough, repeated, and wide skin prep is done. After drying, the injection site is covered using a sterile drape.

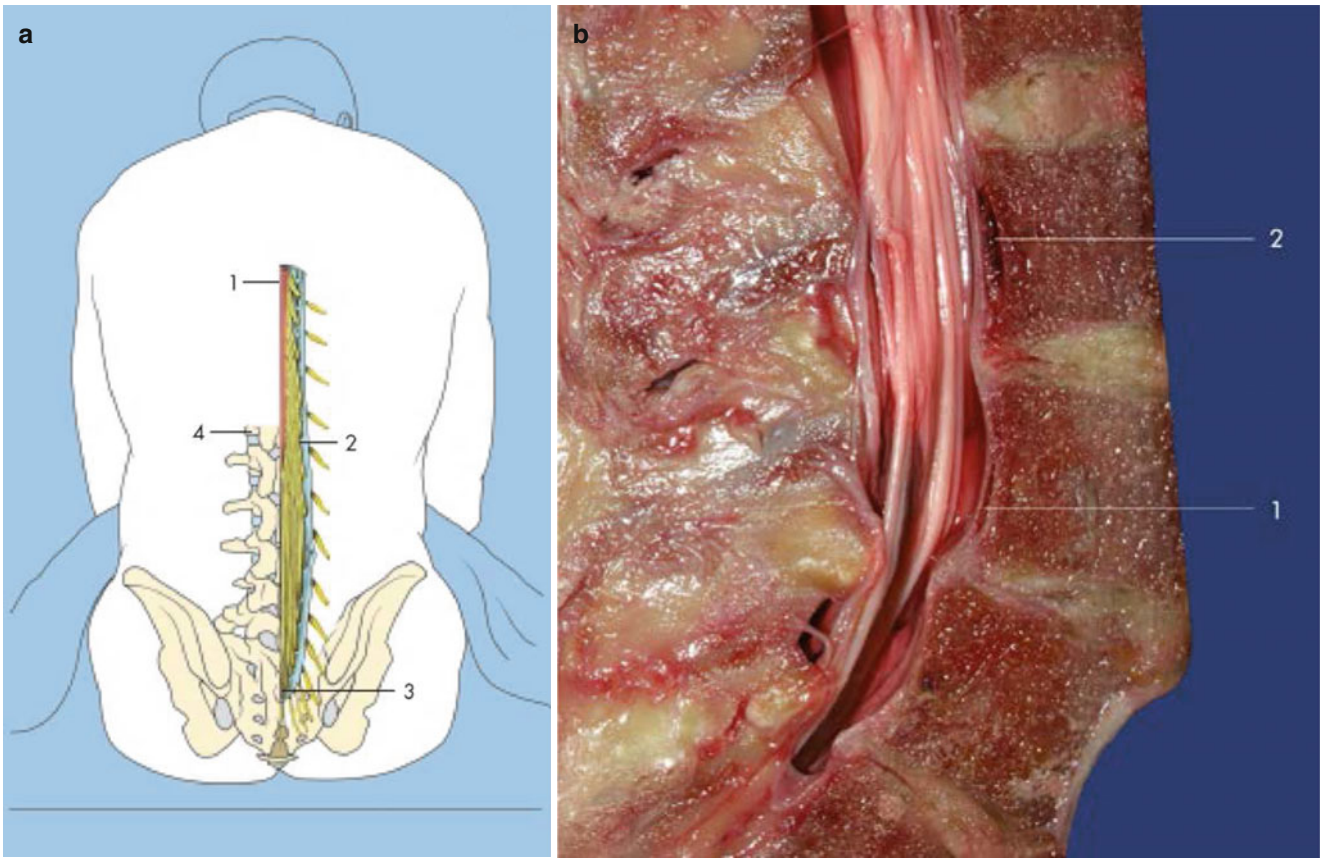


Fig. 41.23 (**a**) Conus medullaris (lower edge of the first lumbar vertebra). (*1*) Conus medullaris, (*2*) cauda equina, (*3*) dural sac, (*4*) L1 segment (With permission from Danilo Jankovic). (**b**) Cauda equina at the level of L2–L5. Paramedian sagittal section. (*1*) Spinal dura mater, (*2*) epidural space (With permission from Danilo Jankovic)

Local Anesthesia

The skin and supraspinous and interspinous ligaments are anesthetized with 1–1.5 ml of a local anesthetic (e.g., 1 % lignocaine). The injection is carried out between the spread index and middle fingers of the left hand (Fig. 41.24).

Needle Insertion

Without moving the spread index and middle fingers of the left hand from the intervertebral space, an epidural needle is fixed between the thumb of the right hand (hub) and the index and middle finger (shaft) and advanced through the skin (Fig. 41.25). After passing the supraspinous ligament, which is about 1-cm thick, the needle, with its bevel directed cephalad or caudad, is slowly advanced a further 2–3 cm (depending on the anatomy), until it rests firmly in the interspinous ligament. This often results in a "gritty" sensation. The trocar is removed and a low-friction (loss of resistance) syringe is attached (Fig. 41.26).

Care should be taken to ensure that the needle is kept in the midline. Inadvertent deviation from the midline leads to the needle passing the supraspinous ligament, with an angled entry into the interspinous ligament with only brief resistance and a subsequent false loss of resistance. This type of puncture ends in the paravertebral musculature and is accompanied by local pain.

After passing the interspinous ligament, the needle must be advanced carefully, millimeter by millimeter, in the direction of the ligamentum flavum.

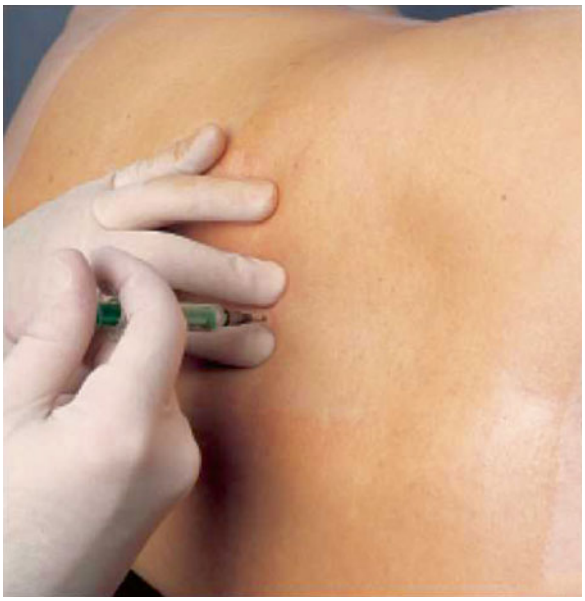


Fig. 41.24 Local anesthesia (With permission from Danilo Jankovic)

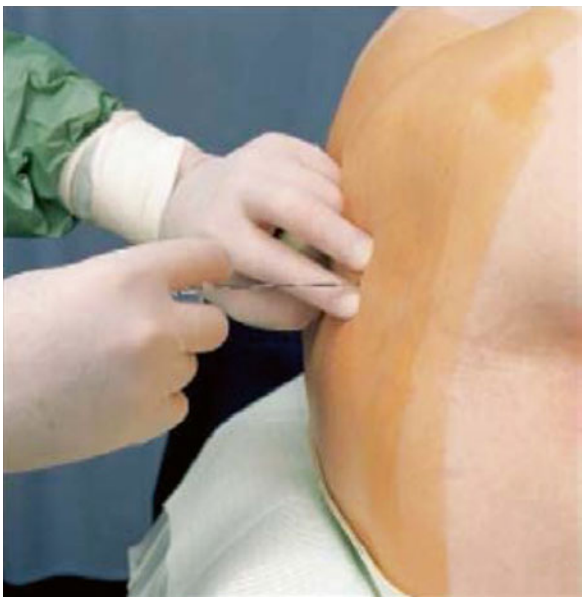


Fig. 41.25 Introducing the epidural needle (With permission from Danilo Jankovic)

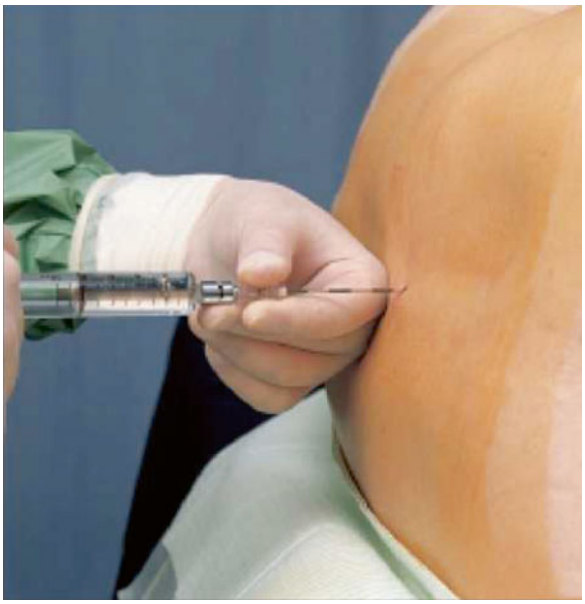


Fig. 41.26 Removing the stylet and attaching a low-friction syringe (With permission from Danilo Jankovic)

Puncturing the Epidural Space

The thumb and index finger of the left hand, which is resting with the back of the hand firmly against the patient's back, secure the needle, advance it millimeter by millimeter, and at the same time serve as a "brake" to prevent inadvertent forward movement. The thumb of the right hand applies pressure on the syringe plunger. *Loss of resistance* indicates that the epidural space has been reached. The contents of the syringe are easily injected.

Identification of the epidural space is carried out using the *loss-of-resistance technique* (Fig. 41.27). The following variations on this technique can be applied:

(a) *Saline with an air bubble*: after the interspinous ligament has been reached, the stylet is removed, and a low-friction syringe filled with a saline solution and with a small air bubble in it, serving as a visual indicator, is attached. When the ligamentum flavum is encountered, the air bubble is compressed by pressure on the syringe plunger (Fig. 41.28a); when the epidural space is reached, the bubble returns to its normal, larger shape (Fig. 41.28b). This technique is the most common choice as it provides a good identification of the epidural space and an idea of the force being applied (by the compression of the air bubble).

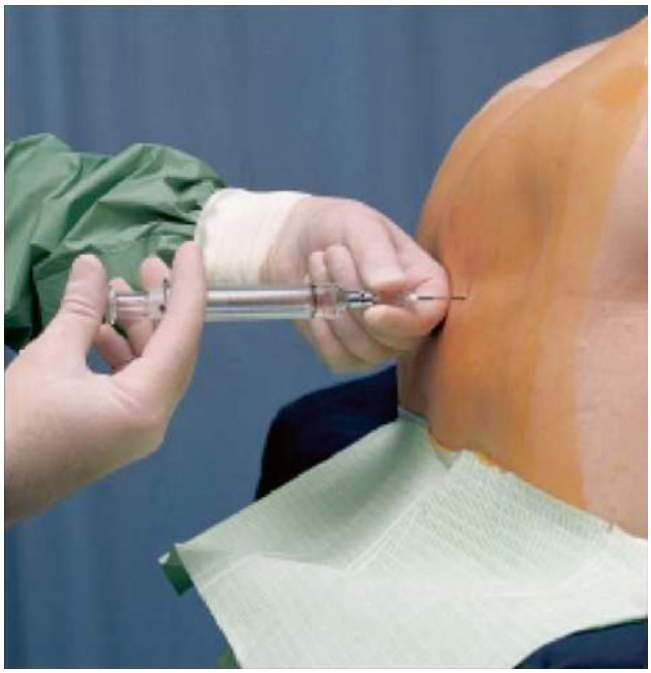


Fig. 41.27 Identifying the epidural space (loss of resistance) (With permission from Danilo Jankovic)

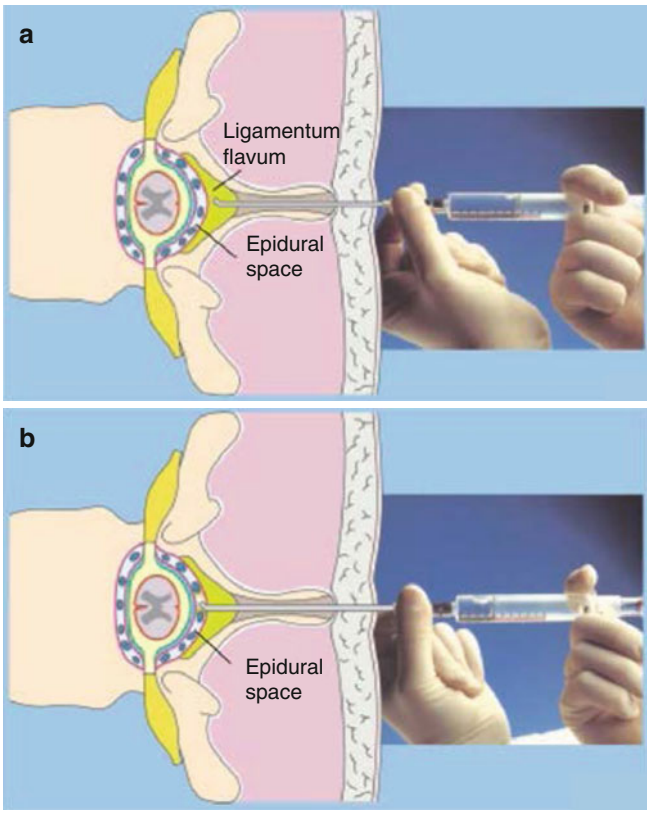


Fig. 41.28 (**a**) Loss-of-resistance technique with saline. The air bubble is compressed by pressure on the syringe plunger (With permission from Danilo Jankovic). (**b**) Loss-of-resistance technique with saline. The epidural space has been reached. The air bubble has returned to its normal, loose shape (With permission from Danilo Jankovic)

(b) *Saline-only technique*: since the introduction of air in the epidural space may lead to "patchy" block, some clinicians prefer to use only saline. The disadvantage of doing so is the inability to objectively judge the amount of force being applied on the plunger.

(c) *Air-only technique*: this technique allows for a more subjective "feel" while identifying the epidural space. It also allows clearer identification of an accidental dural puncture as any fluid that emerges from the hub of the needle must be CSF. However, using air to locate epidural space has been associated with patchy blocks, venous air embolism, and pneumocephalus [70].

(d) *Hanging drop technique*: in this technique, after the interspinous ligament has been reached, a drop of saline is placed within the hub of the needle (Fig. 41.29a). After the ligamentum flavum has been passed and the epidural space has been reached, the drop is "sucked in" due to the negative pressure in the epidural space (Fig. 41.29b) [71].

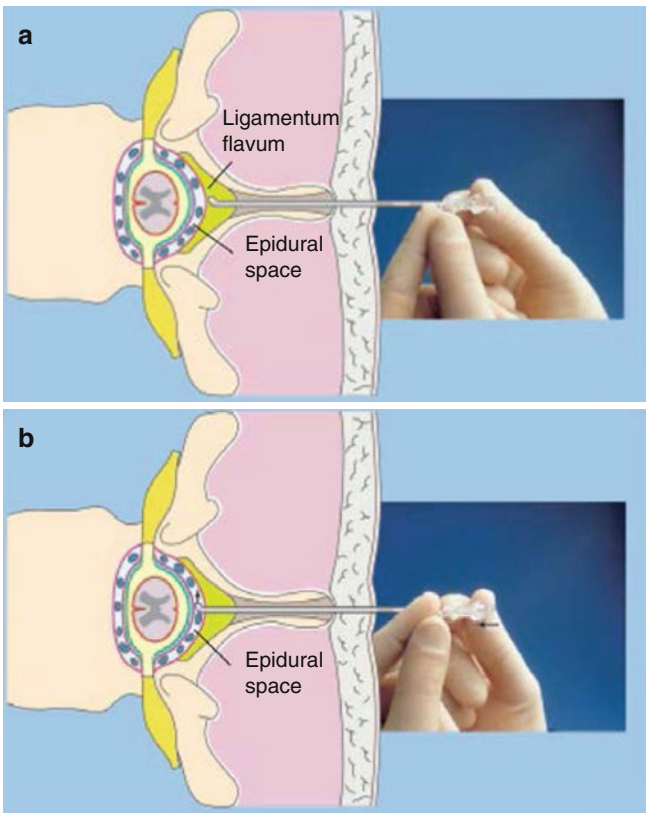


Fig. 41.29 (**a**) "Hanging drop" technique. The epidural needle is positioned in the ligamentum flavum (With permission from Danilo Jankovic). (**b**) "Hanging drop" technique. The epidural space has been reached. The drop is sucked back in (With permission from Danilo Jankovic)

Aspiration and Injection of a Test Dose

Having reached the epidural space, a careful aspiration is carried out to detect CSF or blood while the needle continues to be secured by the thumb and index finger of the left hand, resting with the back of the hand firmly against the patient's back (Fig. 41.30).

After a negative aspiration, a test dose of 3–4 ml of local anesthetic (usually 1.5 % lignocaine) and 15 mcg epinephrine can be injected. This allows detection of intrathecal injection (rapid development of spinal block) or intravascular injection (tachycardia).

The addition of epinephrine can lead to unreliable results in patients taking beta-blockers, patients under general anesthesia, and pregnant patients. Due caution must be exercised while adding epinephrine in pregnant patients (may cause transient fetal bradycardia due to reduced uterine blood flow), older patients with coronary artery disease, and hypertensive patients. The use of epinephrine is best avoided in patients with closed-angle glaucoma and tachyarrhythmia [72].

Maintaining constant verbal contact with the patient and careful cardiovascular monitoring after a test dose is recommended. If all is well, a further dose of the local anesthetic can be injected.

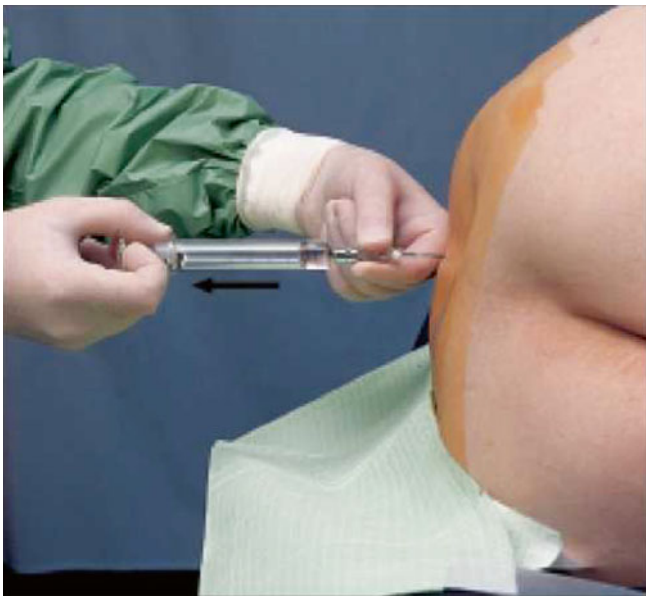


Fig. 41.30 Aspiration test (With permission from Danilo Jankovic)

Drug Administration

The epidural injection may be performed as a single-shot injection or as a catheter technique (allowing further dosing).

(a) *Single-injection technique*: incremental administration of a local anesthetic is carried out in aliquots of 3–5 ml, waiting 15–20 s between each dose, with intermittent aspiration (Fig. 41.31). After aspiration has been repeated shortly before the end of the injection, the needle is withdrawn and the patient is placed in the desired position.

(b) *Catheter technique*: upon confirmation of the epidural space and negative aspiration, a catheter may be introduced. For this, the thumb and index finger of the left hand secure the epidural needle, with the back of the hand lying firmly on the patient's back. The catheter is advanced cranially, using the thumb and index finger of the right hand, to a maximum of 3–4 cm beyond the tip of the needle (Fig. 41.32). Advancing it further than this can lead to lateral deviation of the catheter, with accompanying paresthesias. After placement of the catheter in the desired position, the needle is slowly withdrawn (Fig. 41.33), while at the same time the thumb and index finger of the left hand secure the catheter at the injection site (Fig. 41.34).

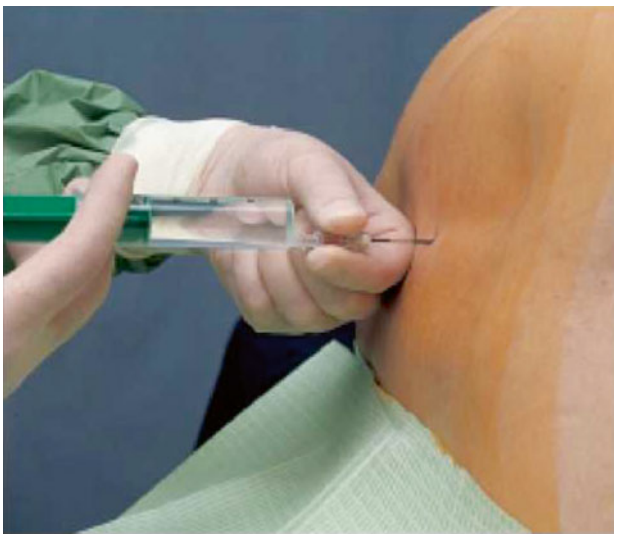


Fig. 41.31 Incremental injection of local anesthetic (With permission from Danilo Jankovic)

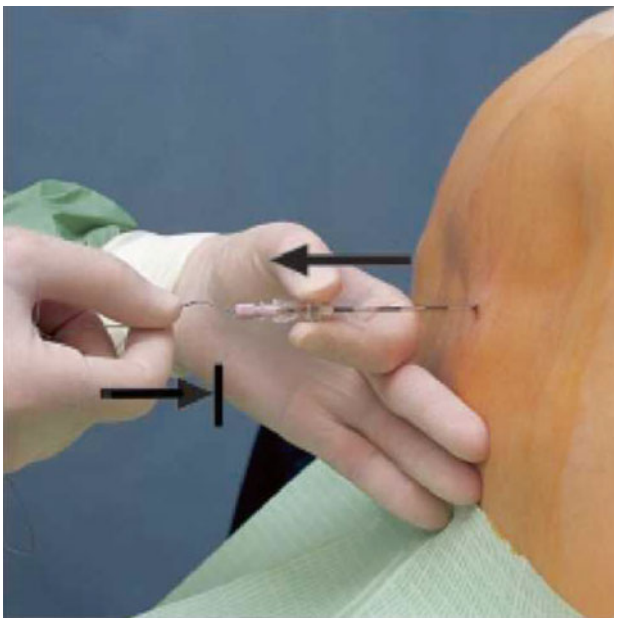


Fig. 41.33 Withdrawing the injection needle (With permission from Danilo Jankovic)

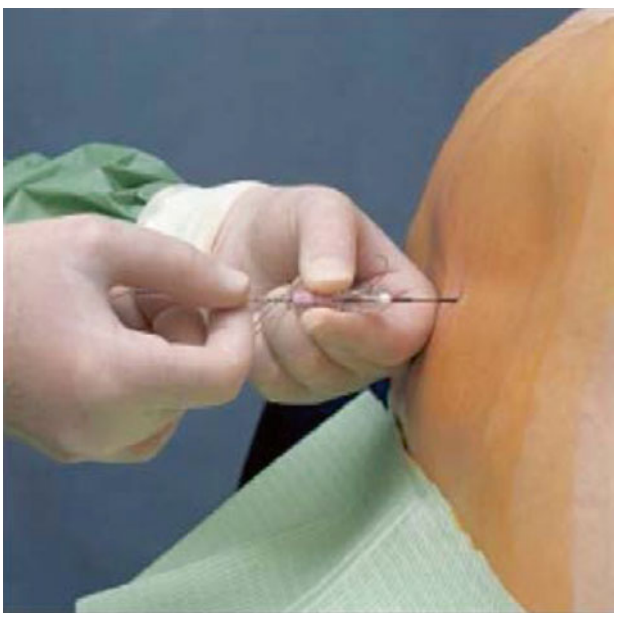


Fig. 41.32 Introducing the catheter (With permission from Danilo Jankovic)

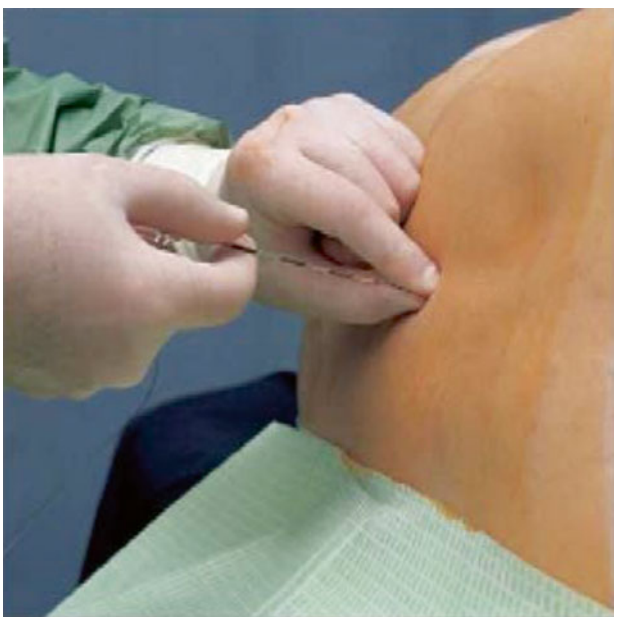


Fig. 41.34 The catheter is secured at the injection site with the thumb and index finger (With permission from Danilo Jankovic)

An adapter is attached to the end of the catheter. The patency of the catheter is tested by injecting 1–2-ml saline (Fig. 41.35). After aspiration, the syringe is disconnected, and the open end of the catheter is placed on a sterile drape below the puncture site. Attention must be given to any escaping fluid (CSF or blood) (Fig. 41.36). A bacterial filter is then attached (Fig. 41.37), and the catheter is secured and dressed to prevent dislodgement and ensure sterility (Fig. 41.38). Fixation of the catheter can be accomplished in a variety of ways, including skin suture or proprietary dressings.

The patient is placed in the desired position and a test dose is administered, as with the single-shot injection. During the waiting period, it is important to maintain verbal contact with the patient and to check the spread of the anesthesia, to exclude the ever-present risk of inadvertent intrathecal injection. After 5 min, the remainder of the dose, adjusted for the individual patient, can be administered on an incremental basis (max. 5 ml each injection) until the desired level of anesthesia is reached.

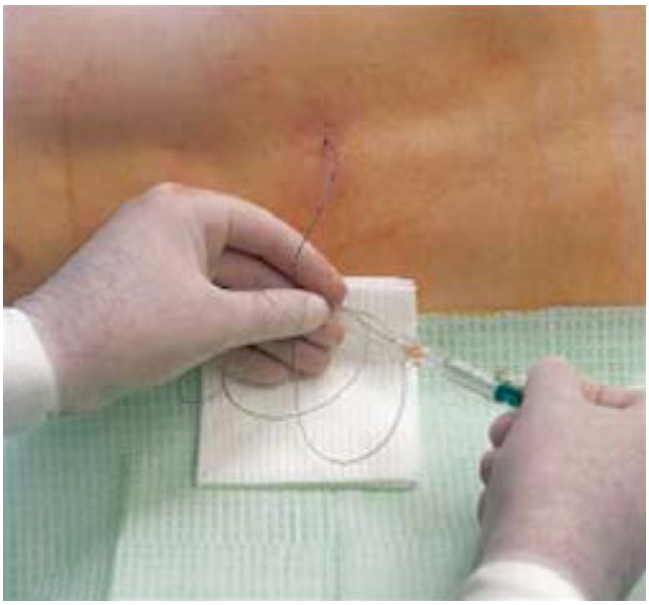


Fig. 41.35 Injection of 1 ml saline (With permission from Danilo Jankovic)

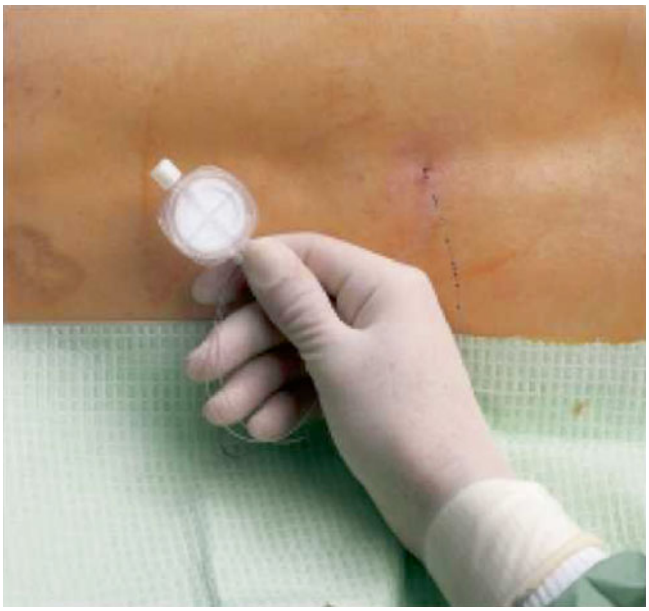


Fig. 41.37 Placing a bacterial filter (With permission from Danilo Jankovic)

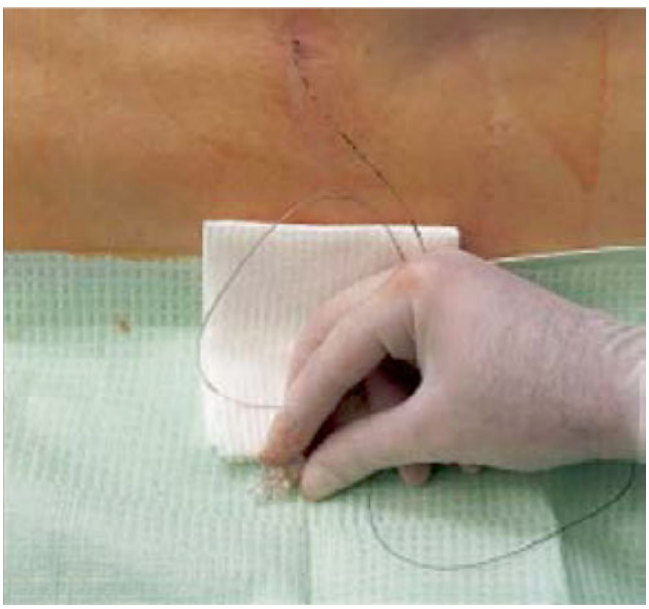


Fig. 41.36 The end of the catheter is placed below the injection site (With permission from Danilo Jankovic)

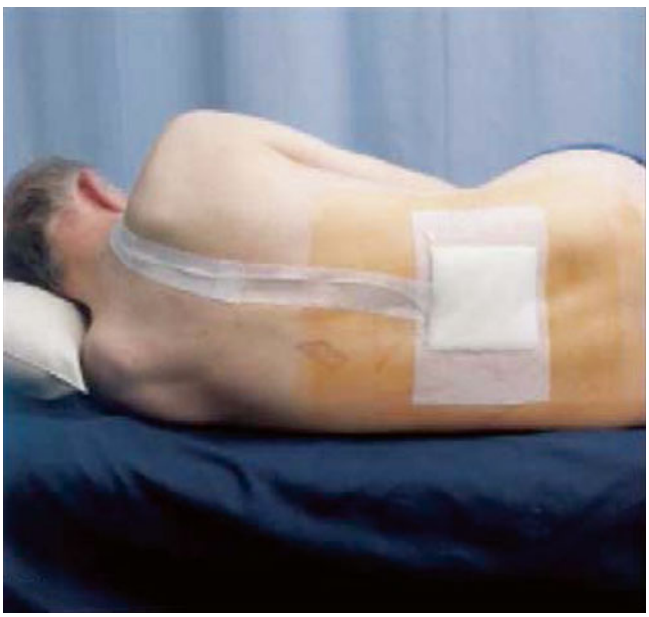


Fig. 41.38 Securing the catheter and dressing (With permission from Danilo Jankovic)

Troubleshooting

(a) *Escaping fluid*: after the epidural space has been identified or after administration of the test dose, a few drops of fluid may still drip from the positioned needle. This may be saline from the syringe or CSF if intrathecal space has been punctured. A higher viscosity, higher temperature (Fig. 41.39), near-neutral pH, higher glucose content, and turbidity with thiopentone help identify the fluid as CSF [73]. If the fluid is not CSF, one can proceed with the procedure.

(b) *Escaping blood*: in this case (Fig. 41.40), it is best to withdraw the needle or the catheter. One may attempt another insertion at a segment higher or lower or abandon the procedure and choose to administer general anesthesia.

(c) *Escaping CSF*: the options in the event of accidental dural puncture include conversion to a spinal anesthetic by injecting an appropriately reduced dose of local anesthetic or inserting a continuous spinal catheter (this must clearly be labeled as such, to avoid inadvertent overdosing with subsequent top-ups). Another attempt at insertion can also be made keeping in mind the possibility of another dural tap and that an epidural dose of local anesthetic may spread intrathecally through the first dural puncture site and lead to total spinal anesthesia. Finally, one may choose to abandon the procedure and administer general anesthesia. In any case, the patients must be informed about the possibility of post-dural puncture headache.

(d) *Inability to thread the catheter*: this may happen when a *false* loss of resistance has been encountered in a superficial tissue plane. Injection of local anesthetic at this point will not be effective. Reattempting the injection is the best course, aiming to obtain a convincing loss of resistance.

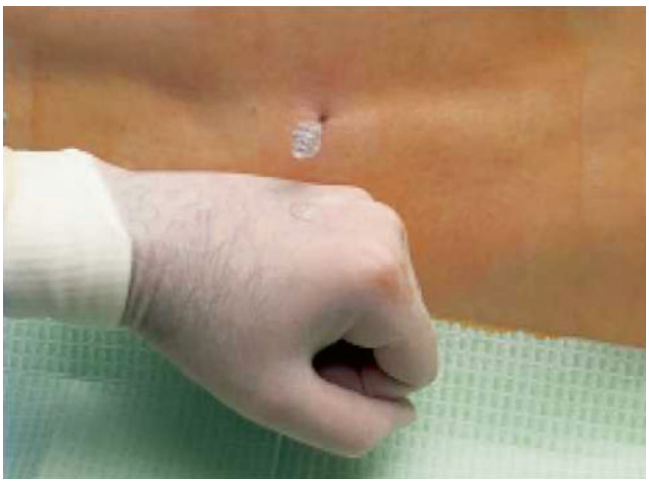


Fig. 41.39 Escaping fluid: is it cold or warm? (With permission from Danilo Jankovic)

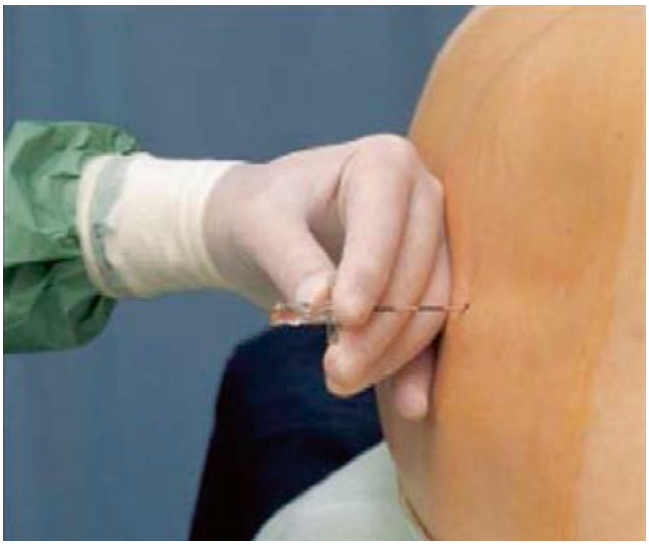


Fig. 41.40 Blood-tinged fluid (With permission from Danilo Jankovic)

Paramedian Approach

This technique, which is independent of lumbar lordosis or the ability of the spine to flex, avoids puncture of the supraspinous ligament and the frequently ossified interspinous ligament. This approach offers a much larger opening into the epidural space than the midline approach.

Indications

- Patients who cannot be positioned easily or cannot flex the spine (trauma/ arthritic)
- Calcified ligament (interspinous)
- Kyphoscoliosis or prior lumbar surgery

Procedure

The puncture site (Fig. 41.41) is located in the selected intervertebral space, about 1.5–2 cm lateral from the upper edge of the lower spinous process. Fan-shaped local anesthesia identifies the depth of the vertebral arches (laminae), which are then marked (4–6 cm). The epidural needle is introduced in a cranio-medial direction at an angle of about 15° to the sagittal level and about 35° to the skin surface, so that it passes the laminae and slides into the interlaminar fissure. The only ligament that needs to be penetrated on the way to the epidural space is the ligamentum flavum. Reaching this is characterized by a *rubbery* resistance. The most important step in this technique is to identify the depth of the ligamentum flavum. The trocar is then removed from the puncture needle, and identification of the epidural space is carried out in the same way as described for the single-shot technique.

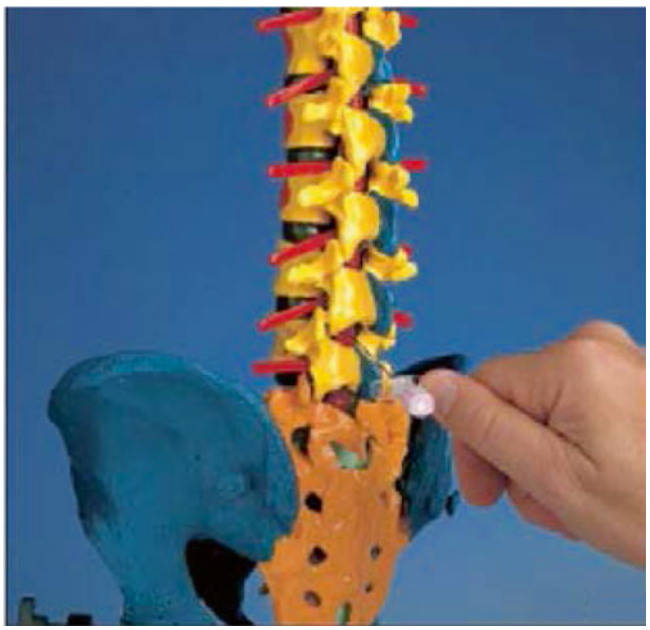


Fig. 41.41 Paramedian puncture (With permission from Danilo Jankovic)

Performance of a Thoracic Epidural

The spinous process of lower thoracic vertebrae is directed horizontally; however, those of the upper to mid-thoracic vertebrae are angulated steeply downward (Fig. 41.42a). Thus, while a midline approach suffices at the lower thoracic levels, a paramedian approach is recommended at the upper to mid-thoracic levels. A cross section of structures at T3 is depicted in Fig. 41.42b.

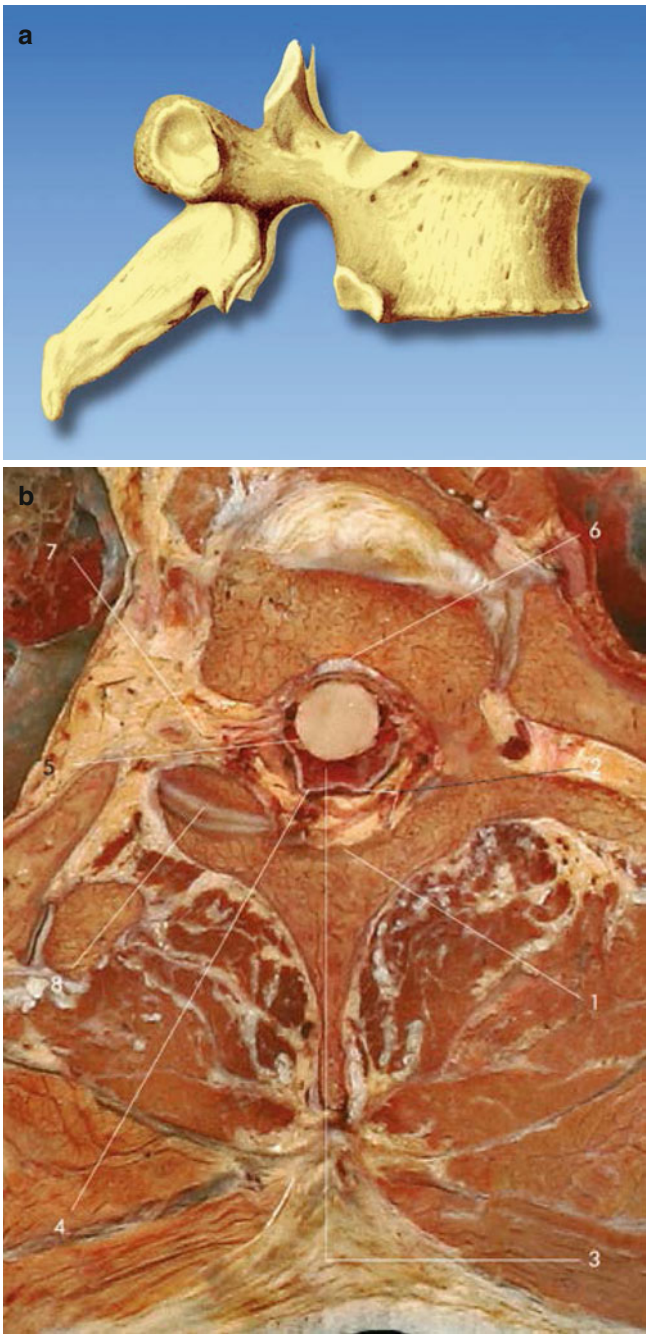


Fig. 41.42 (**a**) Angulation of the spinous processes of upper to mid-thoracic vertebrae (With permission from Danilo Jankovic). (**b**) Transverse dissection at the level of T3. (*1*) Ligamentum flavum, (*2*) epidural space, (*3*) subarachnoid space with spinal cord, (*4*) spinal dura mater, (*5*) spinal pia mater, (*6*) posterior longitudinal ligament, (*7*) neural foramen with spinal nerves, and (*8*) zygapophysial joint (With permission from Danilo Jankovic)

Midline Approach for Lower Thoracic Levels

This is similar to the technique used at the lumbar levels. The patient is usually placed in a sitting position, and monitoring is applied. Intravenous access is established before starting. Landmarks are identified by palpating the spinous processes of the lower thoracic vertebrae. Thorough, repeated, and wide skin prep is applied. After drying, the injection site is covered using a sterile drape. The skin and supraspinous and interspinous ligaments are anesthetized with 1–1.5 ml of a local anesthetic as described earlier. A 16-G Tuohy-tip epidural needle is fixed between the thumb of the right hand (hub) and the index and middle finger (shaft) and advanced through the skin, at an angle of 10–15° cephalad (Fig. 41.43). After passing the supraspinous ligament, the needle, with its bevel directed cephalad, is slowly advanced a further 2 cm until it rests firmly in the interspinous ligament. The trocar is removed and a low-friction (loss of resistance) syringe is attached. After passing the interspinous ligament, the needle must be advanced carefully, millimeter by millimeter, in the direction of the ligamentum flavum. The thumb of the right hand applies pressure on the syringe plunger. *Loss of resistance* indicates that the epidural space has been reached. Extreme care should be exercised at the thoracic level since the spinal cord lies beneath the dura and can be damaged if the needle tip is advanced beyond the epidural space.

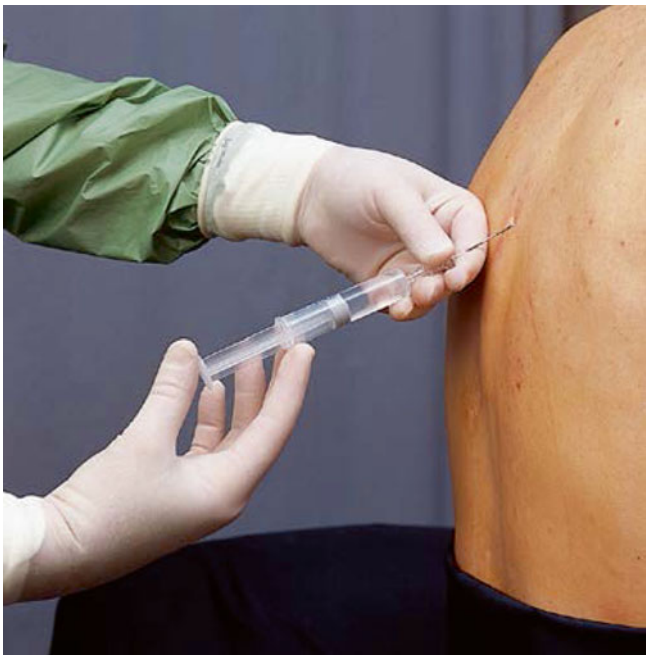


Fig. 41.43 Midline approach for thoracic epidural (With permission from Danilo Jankovic)

Paramedian Approach for Upper to Mid-thoracic Levels

At this level, the puncture site is located about 1–1.5 cm lateral from the caudal edge of the lower spinous process. A fan-shaped local anesthesia infiltration is used to anesthetize the skin and also to identify the depth of the vertebral arches (laminae). The epidural needle is introduced in a *cranio-medial direction* at an angle of about 10–15° to the sagittal level and about 35–45° to the skin surface (Fig. 41.44). The only ligament that needs to be penetrated on the way to the epidural space is the ligamentum flavum. The trocar is then removed from the puncture needle and identification of the epidural space is carried out using a loss-of-resistance syringe. If a bone is contacted, the needle is withdrawn by 0.5 cm, walked off the bone in a medial/cephalad direction until the ligamentum flavum is pierced.

After accessing the epidural space, a catheter is threaded 3–4 cm in the space akin to that for lumbar levels. However, one should not inject more than 3–4 ml of test dose at a time at thoracic levels as they tend to spread more. Using a larger volume may lead to profound hypotension due to a widespread sympathetic block.

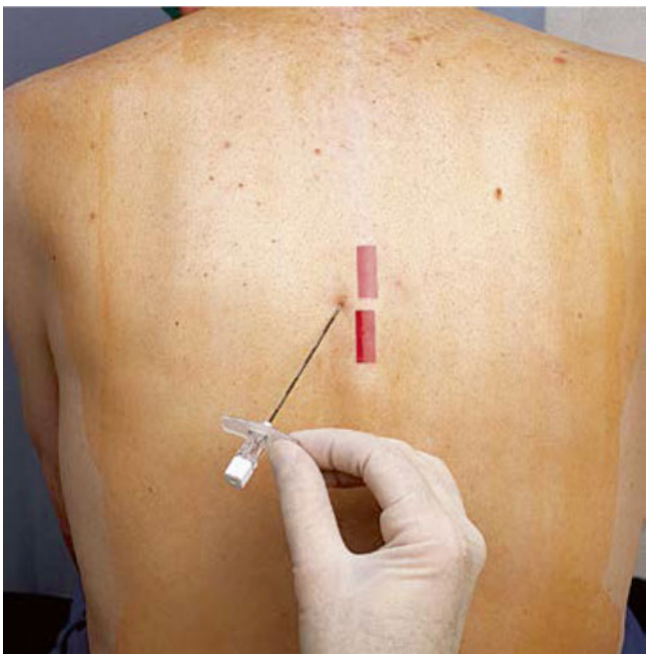


Fig. 41.44 The needle angulation for a mid-thoracic paramedian approach (With permission from Danilo Jankovic)

Management of the Patient After an Epidural Block

Patient Positioning

Gravity does not play a clinically significant role in determining the spread of local anesthetic in the epidural space. Thus, the patient is generally placed supine after the block.

Patient Monitoring

(a) The patient should be monitored during and after the placement of epidural block, just like a spinal block. However, the cardiovascular changes seen with an epidural block are in general slower and less profound when compared to a spinal anesthetic. Fluids and vasopressors are used to treat hypotension.
(b) Supplemental oxygen administration is recommended, especially if sedation is used for patient comfort.
(c) End-tidal carbon dioxide monitoring is also used to monitor the respiratory rate.
(d) Postoperatively, the patient should be monitored until the effects of epidural anesthetic have receded or until the epidural catheter is removed.

Troubleshooting an Inadequate or Ineffective Epidural Block

(a) *Inadequate sensory block*: this may require repeat dosing if the block fails to reach adequate sensory height after 30 min of the initial dose. Addition of opioids (such as fentanyl) to local anesthetic solution may speed the onset and extend the number of segments blocked [74]. An inadequate block in lower segments (L5–S1) may be difficult to troubleshoot in particular.
(b) *Missed segments*: this may be due to an inadequate volume of initial dose used. A repeat dose often improves this. If the missed segment is unilateral, turning the patient on the spared side before dosing can help [75]. A 2 % lignocaine solution with epinephrine is most effective in dealing with missed segments or an inadequate block.

Assessment of the Block

Similar to the subarachnoid block, an epidural block leads to sensory, autonomic, and motor blockade. These can be assessed as follows:

(a) *Sensory block*: this can be assessed by testing for loss of touch, temperature, or pinprick. A differential block is noted between the complete loss of cold sensation (being two dermatomes cephalad) and the complete loss of both pinprick and light touch sensation (being caudal) [76].
(b) *Autonomic block*: this can be subjectively judged by the skin temperature in the involved areas and the degree of blood pressure drop (sympathetic block). Digital plethysmogram and skin conductance are used in research settings to test this objectively.
(c) *Motor block*: this can be assessed by using the Bromage scale for a lumbar epidural and a RAM (rectus abdominis muscle) test for thoracic epidural [77, 78]. These are summarized in Tables 41.5 and 41.6.

Table 41.5 Bromage scale for assessment of motor block

Grade	Criteria	Degree of block
I	Free movement of legs and feet	Nil (0 %)
II	Just able to flex knees with free movement of feet	Partial (33 %)
III	Unable to flex knees but with free movement of feet	Almost complete (66 %)
IV	Unable to move legs or feet	Complete (100 %)

Table 41.6 RAM test of abdominal muscles

Power (%)	Criteria
100	Able to rise from supine to sitting position with hands behind the head
80	Can sit only with arms extended
60	Can lift only head and scapulae off the bed
40	Can lift only shoulders off the bed
20	An increase in abdominal muscle tension can be felt during effort; no other response seen

Considerations in Patients with Challenging Anatomy

Performing a neuraxial block can be technically challenging in certain patient groups. This includes patients with high body mass index, patients with scoliosis, and those having undergone a spinal surgery. Altered anatomy makes performing the block technically challenging, while spinal stenosis or postoperative epidural fibrosis can impair the spread of spinal anesthetics in the subarachnoid or epidural space, resulting in failed blocks.

Patients with High Body Mass Index

The anthropometric changes associated with obesity make performing a neuraxial block particularly difficult. Difficulty in proper positioning, obscured anatomical landmarks, increased depth of the ligamentum flavum, and the occasional inadequacy of usual equipment (e.g., needle too short) contribute to making neuraxial blocks in obese technically challenging [79]. This can result in a higher incidence of dural punctures while performing epidural anesthesia in morbidly obese patients [80]. Ultrasound helps in the correct identification of midline, intervertebral spaces, and estimates of depth of the ligamentum flavum. It has been successfully used to improve the success rate of epidural placement in obese parturients [81].

Patients with Scoliosis

Lateral deviation of the spine is accompanied by the rotation of the vertebral bodies toward the convex side of the curvature, while the spinous processes are rotated toward the concave side [82]. This makes performing a neuraxial block quite difficult. Ultrasound assists in identifying the lateral curvature and rotation and in its quantifications. The usual approach to this situation is to perform the block using a paramedian injection on the convex side, which provides a more direct access to the neuraxis [83]. Alternatively, if a midline insertion is used, the needle should be directed in a transverse plane, toward the convex side [84].

Patients with Previous Spine Surgery

Epidural fibrosis following spine surgery, altered spine anatomy, and the possibility of worsening neurological symptoms make this patient subset a challenge. Despite this, there have been several reports and reviews of successful neuraxial blocks in patients with previous spine surgery [85–87].

While the absence of spinous process makes it hard to locate intervertebral spaces, laminectomy may actually increase the chances of obtaining a successful dural puncture by increasing the size of the interlaminar gap. However, performing an epidural at the level of previous spinal surgery poses additional challenges due to spinal stenosis immediately above the fusion or decompression and tethering of the dura to the ligamentum flavum by scar formation [88, 89]. The epidural space may also be scarred which reduces the reliability of the loss-of-resistance technique. Thus, performing the epidural injection one or two spaces above or below the level of the surgery is advocated to reduce the chances of accidental dural puncture and ineffective block. Note that scarring of the epidural space may also lead to patchy block.

Ultrasound can help immensely to locate the intervertebral space, identify the interlaminar window, and visualize and estimate the depth of the ligamentum flavum [90].

Pharmacology of Neuraxial Drugs

Many local anesthetics and other adjuvant drugs are used for the conduct of a spinal anesthetic. A reliably rapid onset, adequate distribution and duration, and timely return of the neurological function make spinal anesthesia a good choice for many procedures. However, the successful application of a spinal anesthetic requires a good understanding of the effects of intrathecal drugs and factors that determine them.

Pharmacokinetics of Intrathecal Local Anesthetics

Determinants of Intrathecal Distribution

The local anesthetic deposited within the subarachnoid space spreads to the nerve via bulk flow. Many factors have been proposed to influence the spread of local anesthetics within the subarachnoid space [91, 92]. These may be classified as:

Characteristics of Injected Drug

(a) *Baricity*: it is defined as the ratio of the density of the local anesthetic solution relative to the patient CSF at 37 ° C. *Isobaric*, hyperbaric, and *hypobaric* solutions have same, greater, and less density than the CSF, respectively. While hyperbaric solutions will sink to the most dependent areas within the subarachnoid space, the hypobaric solutions rise upward toward the nondependent areas. In general, hyperbaric solutions will produce a greater spread than isobaric or hypobaric solutions [93].

(b) *Mass of the drug*: mass, volume, and concentration have been investigated for their effects on the spread of the local anesthetics in the intrathecal compartment. For plain solutions, the effect of drug mass injected is more important than the volume or the concentration [94]. For

hyperbaric solutions, this may not always be true, with some studies finding no effect [95].

(c) *Effect of additives*: while the addition of vasoconstrictors prolong the block duration, the addition of opioids may increase spread and delay block recession [96, 97].

Technical Considerations

(a) *Patient position*: immediately after, a spinal anesthetic determines the extent of spread under the influence of gravity. This is further influenced by the curvatures of the spine [95, 98].

(b) *Level of injection*: for an isobaric solution, a higher level of injection results in a greater cephalad spread of the block [99]. This is not consistently observed with hyperbaric solutions where the effect of gravity may be more profound [100].

(c) *Needle direction*: turning the needle aperture cephalad may result in a higher spread of the drug, with a shorter duration of action and faster resolution of the block [101]. Cephalad angulation of insertion has shown similar results [102].

(d) *Speed of injection*: has clinically minimal effects.

(e) *Barbotage*: may shorten the time of onset with hyperbaric solutions [103].

Patient Factors

Individual patient factors such as age, height, body mass index, and sex do not help to predict the spread of intrathecal local anesthetics. However, excessive lordosis in pregnancy may promote cephalad spread, and lowering doses is recommended [104, 105].

Uptake of Local Anesthetics

The local anesthetic deposited in the subarachnoid space spreads within the CSF. It is then taken up by the nerve roots in the cauda equina, resulting in neuronal block. This uptake is affected by the following factors:

(a) Concentration of the local anesthetic in the CSF

(b) The surface area of the nerve root exposed to the CSF [106]

(c) The lipid content of the nerve root (since local anesthetics are lipid soluble)

(d) The blood flow within the nerve

Elimination of Local Anesthetics

The elimination of local anesthetics from the subarachnoid space is determined by the following factors:

(a) Vascular absorption of the local anesthetic (this is the most important route) [107]

(b) Escape of the drug to the epidural space, with subsequent vascular absorption [108]

(c) Lipophilicity of the local anesthetic (highly lipophilic drugs such as bupivacaine have a slower elimination due to greater binding with the neuronal tissue) [109]

Determinants of Duration

Elimination of local anesthetics from the intrathecal space determines the duration of their neural block. The factors influencing this are:

(a) *Physicochemical properties of the local anesthetic chosen*: while prilocaine and 2-chloroprocaine are short-acting agents, lignocaine and mepivacaine are short- to intermediate-acting local anesthetics. Bupivacaine, levobupivacaine, and ropivacaine are longer-acting agents. However, it must be noted that there is wide inter-patient variability.

(b) *Dose injected*: in general, the duration of the block is increased with an increase in the dose (or mass) of the local anesthetic injected.

(c) *Block spread*: for a given dose of the local anesthetic, a block with a greater spread (i.e., higher peak sensory block) will regress faster, thereby shortening the duration of action [110].

(d) *Addition of adjuvants*: as mentioned above, the addition of vasoconstrictors and opioids may delay the regression of the block [111, 112].

Intrathecal Local Anesthetics

The physicochemical properties of local anesthetics such as lipid solubility and protein binding impact the duration of block. Depending upon this, these local anesthetics may be classified as follows:

Short-Acting Agents

(a) *Procaine* is an amino ester with a rapid onset (3–5 min) but short duration (50–60 min) of block. This is due to its poor lipid solubility and protein binding. Compared to lignocaine, it has a higher rate of block failure but a lower rate of transient neurologic syndromes (TNS) [113, 114].

(b) *2-Chloroprocaine* is an amino ester with a rapid onset and short duration (60 min) of spinal block comparable to lignocaine. It has a lower incidence of TNS [115].

(c) *Prilocaine* is a short-acting amino-amide with a short duration of action (60–120 min). It has been recently used for ambulatory surgery, with shorter recovery times when compared to lignocaine [116, 117].

Short- to Intermediate-Acting Agents

(a) *Lignocaine* is an amino-amide with a rapid onset and short to intermediate duration of action (60 min, depending upon dose). Its use has dramatically declined due to the higher frequency of TNS observed with its use (15–33 %) [118].

(b) *Mepivacaine* is an amino-amide with similar profile to lignocaine but lower incidence of TNS (3–6 %).

Table 41.7 Dosages of hyperbaric local anesthetics

	0.5 % Bupivacaine (5–8 % glucose)		5 % Lignocaine (7.5 % glucose)		4 % Mepivacaine (9.5 % glucose)		1 % Tetracaine (5 % glucose)	
Local anesthetic	**ml**	**mg**	**ml**	**mg**	**ml**	**mg**	**ml**	**mg**
T6—high	2.5–4.0	12.0–20.0	1.5–2.0	75–100	1.5–2.0	60–80	1.5–2.0	7.5–10.0
T10—medium	2.0–2.5	10.0–12.5	1.0–1.5	50–75	1.0–1.5	40–60	1.0–1.5	5.0–7.5
L1—deep	1.5	7.5	1.0–1.2	50–60	1.0–1.2	40–48	1.0–1.2	5.0–6.0
S1–S5 saddle block	1.0	5.0	0.6–1.0	30–50	0.6–1.0	24–40	0.5–1.0	2.5–5.0
Onset of effect (min)	10–20		5–10		5–10		10–20	
Duration of effect (min)	Up to 160		Up to 60		Up to 60		Up to 150	
Prolongation with vasopressors	No clinically significant prolongation						Up to 180–240 min	

Long-Acting Agents

(a) *Tetracaine* is a long-acting (3 h) amino ester with a high lipid solubility. It has been almost entirely replaced by bupivacaine due to poor reliability.

(b) *Bupivacaine* is a prototypical amino-amide with a long duration of action due to high lipid solubility. It is the most widely used intrathecal local anesthetic. It has an onset time of 10 min and a block duration of 3–4 h. Reducing the doses for unilateral spinal or ambulatory surgery shortens the duration of action but may also increase the incidence of block failures [119].

(c) *Levobupivacaine*: this less cardiotoxic stereoisomer of bupivacaine is almost identical to bupivacaine in its spinal anesthetic profile [120].

(d) *Ropivacaine* is a long-acting amino-amide which has gained popularity by virtue of its less cardiotoxic potential. When compared to bupivacaine, it is less potent and produces a block of shorter duration [121].

Dosages and duration of commonly used local anesthetics are summarized in Tables 41.7 and 41.8.

Table 41.8 Dosage with isobaric local anesthetic

Local anesthetic	Dose	Duration of effect (min)
Prilocaine 2 %	3–4 ml (60–80 mg)	60–120
Mepivacaine 2 %	3–5 ml (60–100 mg)	30–90
Lidocaine 2 %	3–5 ml (60–100 mg)	30–90
Bupivacaine 0.5 %	3–4 ml (15–20 mg)	160
Ropivacaine 0.5 %	3–5 ml (15–25 mg)	60–120

Intrathecal Adjuvants

Additives are often used along with intrathecal local anesthetics to prolong or intensify their block. Although many drugs have been used and evaluated for this, the commonly used intrathecal additives include opioids, vasoconstrictors, and the α2-adrenergic agonists.

Opioids

Opioids act *synergistically* with local anesthetics by blocking the opioid receptors at the spinal level.

(a) *Morphine* is the most commonly used hydrophilic opioid. Because of its slow distribution within the CSF and a slow plasma clearance, it has a long duration of action when given intrathecally. Used in the doses of 100–400 mcg, it provides good postoperative analgesia up to 24 h. However, the intrathecal use of morphine has been implicated in delayed respiratory depression due to rostral migration within the CSF [122]. Higher doses of intrathecal morphine are associated with higher incidence of side effects such as nausea, vomiting, pruritus, urinary retention, and respiratory depression [123, 124].

(b) *Fentanyl* is the most commonly used lipophilic opioid having a rapid onset (5–10 min) and an intermediate duration (1–2 h) of action. In the dose range of 10–25 mcg, it increases the intensity of the block without prolonging it. This makes it a suitable option for ambulatory surgery. It should be noted however that there is a risk of nausea and vomiting and pruritus, especially at higher doses.

(c) *Sufentanil* is a lipophilic opioid used in the dose range of 2.5–7.5 mcg. It has been used in orthopedic surgery and labor analgesia [125, 126].

Vasoconstrictors

These drugs reduce the vascular uptake of intrathecal local anesthetics, thereby prolonging their duration. However, the significant prolongation is observed when vasoconstrictors are added to tetracaine, when compared with their use along with lignocaine or bupivacaine [127]. The recommended dose range of intrathecal *epinephrine* is 0.2–0.3 mg and that of *phenylephrine* is 2–5 mg. Due to delay in return of sacral autonomic function causing delay in the ability to void and increased risk of urinary retention, the use of vasoconstrictors is not recommended in ambulatory surgery [111].

α2-Adrenergic Agonists

α2-Adrenergic agonists such as *clonidine* and *dexmedetomidine* act on α2-adrenergic receptors in *substantia gelatinosa* in the spinal cord, intensifying and prolonging both sensory and motor block produced by intrathecal local anesthetics. Clonidine also prolongs spinal block when given orally or intravenously. This, however, is accompanied by a higher incidence of undesirable side effects such as bradycardia, hypotension, and sedation, when compared with the intrathecal route [128]. The recommended doses of clonidine is 15–150 mcg (but lower doses are advocated), while that of dexmedetomidine is 3 mcg [129, 130].

Pharmacology of Epidurally Administered Drugs

The precise site of action of epidural local anesthetic is not known. However, they are distributed by resorption into the circulation (via the epidural venous plexus), diffuse through the dura into the CSF, and spread laterally through the intervertebral foramina (associated paravertebral block of the spinal nerves) [131].

The local anesthetic spreads both cephalad and caudad in the epidural space. Since the band of anesthesia produced thus cannot be predicted accurately in a given patient, clinicians must be aware of major factors determining this spread. The factors affecting the spread of the local anesthetic within the epidural space are discussed below:

(a) *Drug mass*, *concentration*, *and volume*: both total drug dose and volume are independent determinants of the spread of epidural block. However, they are *not linearly* related. A higher concentration produces a profound block of both motor and sensory nerves, as opposed to a more selective sensory block produced by using lower concentrations [132].

(b) *Site of injection*: this is a *major determinant* of the epidural spread. For example, the same volume of drug in the caudal space covers less dermatomes when compared to the thoracic level [133].

(c) *Other technical factors*: patient position, needle angulation, direction of needle opening, and the speed of injection are not clinically significant.

(d) *Length of catheter in space*: threading an epidural catheter more than 5 cm may cause lateral location of the catheter tip, resulting in missed segments or unilateral block.

(e) *Patient factors*: increasing age, shorter height, and increased body mass index are associated with increased spread, but this is highly variable and cannot be predicted. The epidural spread is not affected by differences in gender.

Epidurally Used Local Anesthetics

Nearly all local anesthetics have been used for epidural block. They are commonly classified by their duration of action. *Time for two segment regression* is the time taken for the block to recede by two dermatomes from its maximal

Table 41.9 Commonly used local anesthetic agents for a surgical epidural block

Drug and concentration	Usual dose (ml)	Onset time (min)	Time for two segment resolution (min)	Recommended "top-up" time (min)	Time for complete resolution (min)
Short-acting agents					
Chloroprocaine 2–3 %	15–25	6–12	45–60	45	100–160 min
Intermediate-acting agents					
Lignocaine 1–2 %	10–20	10–20	60–100	60	160–200
Mepivacaine 1.5–2.0 %	15–30	10–30	60–100	60	160–200
Long-acting agents					
Ropivacaine 0.2–1.0 %	10–20		90–180	120	180–360
Bupivacaine 0.25–0.5 %	10–20	5–20	120–240	120	300–480
Levobupivacaine 0.25–0.5 %	10–20	5–20	120–240	120	300–480
Etidocaine 1–1.5 %	15–30	10–15	120–240	120	300–480

extent, while the *time for complete resolution* is the time taken for the recovery from sensory block. While the former helps to time the repeating of an epidural dose intraoperatively, the latter is used to estimate the time for discharge for outpatients. Commonly used agents with doses are mentioned in Table 41.9.

In general, more dilute concentrations suffice for analgesia, while higher concentrations are used for a surgical block. The total dose and volume needed depends upon the surgery and other factors already discussed in section "Management of the patient after an epidural block".

Epidurally Administered Adjuvants

Similar to spinal anesthetic, many adjuvants have been used to improve the quality of an epidural block. These are summarized below.

Epinephrine

In a concentration of 2.5–5 mcg/ml (1:400,000–1:200,000), epinephrine prolongs both the sensory and motor block when added to short- to intermediate-acting local anesthetics [134]. It reduces the vascular uptake of local anesthetics into the systemic circulation. It may also exert an analgesic effect through α2-adrenergic receptors, reducing pain transmission within the spinal cord [135]. The addition of epinephrine in an epidural block produces a vasodilatation secondary to β2-adrenergic effects in the periphery. This decreases the mean arterial pressure but also increases the cardiac output [136].

Clonidine

Clonidine prolongs the sensory block only, when added to epidural local anesthetics. It is used in doses of 150–300 mcg. Hypotension and sedation are common secondary to systemic uptake.

Opioids

Commonly used epidural opioids include fentanyl (0.5–0.15 mcg/kg), sufentanil (0.3–0.7 mcg/kg), hydromorphone (0.8–1.5 mg), and morphine (4–6 mg) [137].

Bicarbonate

Addition of sodium bicarbonate (0.1 mEq/ml) to local anesthetics has been proposed as a means to hasten the onset of the blocks. While some studies have shown a faster onset, others have found no difference [138].

Complications of Neuraxial Blocks

Complications arising from a spinal or an epidural anesthetic are mostly cardiovascular and neurological. However, practically, they can be classified as early or late.

Early Complications

These include complications seen immediately following a neuraxial block and in the early perioperative period.

Cardiovascular

These are the most prominent complications of a central neuraxial anesthetic and occur as a consequence of profound sympathetic block caused by the intrathecal local anesthetic.

(a) Hypotension: arteriolar dilation and venous pooling lead to hypotension, which is the most frequent complications (16–33 %) observed [139]. A block above T5, emergency surgery, age greater than 40, chronic hypertension, a combined spinal–general anesthetic, and an intrathecal injection above L2–L3 are risk factors for developing hypotension [39]. Although hypotension mostly follows immediately after the neuraxial block, it may be delayed as well. Epidural anesthetic in general is associated with a more gradual fall of blood pressure, making it a better choice in certain circumstances. Prophylactic fluid administration (20 ml/kg) and use of vasopressors such as ephedrine (in 5–10-mg increments) or phenylephrine (50–100-mcg increments) can be used to effectively counteract hypotension.

(b) Bradycardia: results usually from a high block, as a consequence of blockade of the cardiac accelerator fibers (T1–T4) or more commonly from a vagal reflex associated with intracardiac stretch receptors in the presence of decreased cardiac filling. Apart from a high block, a younger age, ASA class 1 status, preoperative use of beta-blockers, and male gender are risk factors for bradycardia [140]. Anticholinergics such as atropine and adrenergics such as ephedrine are commonly used to treat this.

(c) Cardiac collapse: asystole and cardiac arrest following a spinal is a known complication [34]. It is usually preceded by bradycardia resulting from the mechanisms described above.

Nausea and Vomiting

It is associated with hypotension (hypoperfusion of the chemoreceptor trigger zone) and the use of opioids (direct stimulation of chemoreceptor trigger zone). A sympathetic–parasympathetic imbalance may also have a role to play in the causation of nausea. It can be effectively treated by dexamethasone and 5-HT_3 antagonists (ondansetron) [141].

Tissue Trauma and Back Pain

Multiple attempts at a neuraxial block lead to trauma to soft tissue and ligaments. This can contribute to persistent backache [142]. However, other factors may have a role to play as well.

Total Spinal Anesthetic

This may result due to inappropriate dosing during a spinal anesthetic, positioning error, or unintended passage of local anesthetic from epidural space to subarachnoid space (secondary to an unrecognized dural tap or catheter migration) [143, 144]. It manifests as a rapidly ascending motor–sensory block, hypotension, bradycardia, and respiratory compromise. Medullary paralysis may result in a respiratory arrest and loss of consciousness. It is life-threatening and requires immediate treatment.

Management: this involves prompt recognition, securing the airway, ventilation, and supporting the hemodynamics till spontaneous recovery of the patient.

- Immediate tracheal intubation (thiopental 1–2 mg/kg b.w. routinely ca. 150 mg i.v.); succinylcholine if appropriate, since the muscles of mastication are not affected
- Ventilation with 100 % oxygen
- Raising the legs
- Rapid volume administration
- Atropine
- Vasopressor
- Dopamine infusion
- Careful cardiovascular monitoring

Subdural Anesthetic

This presents as widespread but ineffective patchy anesthesia (Fig. 41.45) [145]. Subdural injection can never be excluded with certainty. It may occur slightly more frequently after spinal anesthesia or myelography than after epidural anesthesia. The subdural space is at its widest in the cervical region, particularly dorsolaterally. It does not end at the great foramen as does the epidural space but continues cranially.

(a) *Warning signs*: an unusually high sensory block, which develops very slowly (even after 20 min) and a much less marked motor block. The clinical picture resembles that of total spinal anesthesia and is characterized by moderate hypotonia, breathing difficulties with retained consciousness, and often involvement of the cranial and cervical nerves.

Trigeminal nerve: trigeminal nerve palsy, with accompanying paresthesias in the area supplied by the nerve and transient weakness of the muscles of mastication with simultaneous Horner's syndrome, has been observed after high epidural anesthesia or subdural spread [146–148].

Horner's syndrome: Horner's syndrome is produced after neuraxial anesthesia with a high spread of the injected local anesthetic and block of very sensitive sympathetic nerve fibers in the areas of segments T4–C8 [148, 149]. Most often, Horner's syndrome is seen after high spinal or epidural anesthesia in obstetrics and when there is subdural spread of the local anesthetic [150]. Relatively rapid resolution of the symptoms is characteristic.

(b) *Differential diagnosis*:

Total spinal anesthesia: this has a dramatic course, unrecordable blood pressure and respiratory arrest, and takes a longer time required for symptoms to resolve.

(c) *Prophylaxis*:

- Individual dosage and dose reduction in older patients, pregnant patients, obese patients, and those with diabetes mellitus or arteriosclerosis
- Incremental injection in epidural anesthesia

Dural Tap

It presents as a gush of CSF through the epidural needle or upon aspiration through the catheter (Fig. 41.45) [151]. Management options include passing a catheter through the puncture made or reattempting the epidural in another space. In over 75 % of the cases, use of an epidural needle is associated with development of post-dural puncture headache.

Local Anesthetic Toxicity

Inadvertent deposition of drug into a vein or systemic absorption of local anesthetic following infusions may lead to local anesthetic toxicity.

Delayed Complications

These present usually after a few days or weeks of having performed a neuraxial block.

Nerve Injury

These include the following:

(a) Direct needle trauma: this commonly occurs at the time of performance of the block and is associated with sever paresthesia in the dermatomal distribution of the nerve root injured (Fig. 41.45) [152].

(b) Neurotoxicity: all local anesthetics are potentially neurotoxic [153]. Despite this, local anesthetic-induced neural damage is rarely seen clinically.

(c) Transient neurological syndrome: this presents as low back pain that radiates to the lower extremities after a spinal anesthetic. The symptoms last for a week and are treated supportively using simple analgesics. Risk factors such as outpatient procedures, the lithotomy position, obesity, and the use of lignocaine have been implicated in their development [154].

(d) Cauda equina syndrome: this presents as motor weakness and loss of bladder and bowel function, following the use of continuous spinal anesthesia. The pooling of local anesthetic around sacral nerves is thought to be the causative mechanism in its development [155].

Infections

Infections such as localized skin infections, a spinal abscess, an epidural abscess, or meningitis can rarely occur following a neuraxial anesthetic (Fig. 41.45). A spinal or epidural abscess presents a localized back pain and tenderness upon palpation, with sensory or motor deficits and fever. A magnetic resonance imaging of the vertebral canal is considered best for establishing a diagnosis. Intravenous antibiotics and surgical drainage or decompression may be needed.

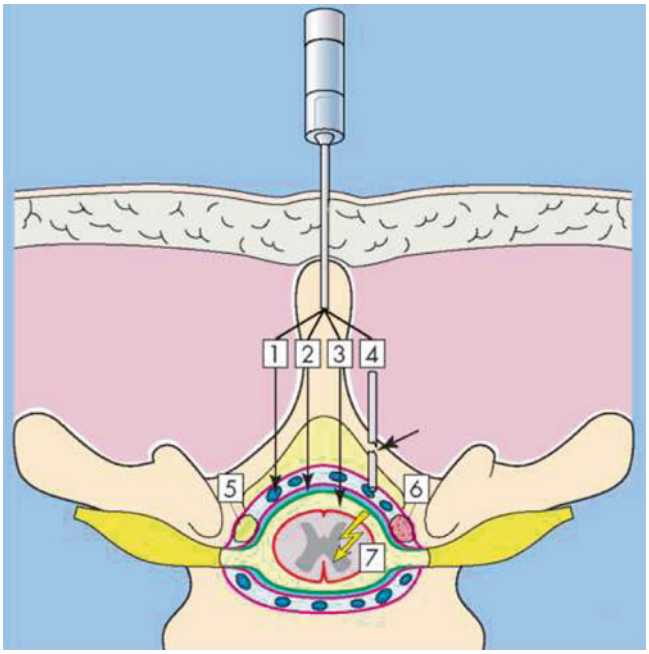


Fig. 41.45 Complications. (*1*) Intravascular injection, (*2*) subdural injection, (*3*) subarachnoid injection, (*4*) catheter shearing, (*5*) epidural abscess, (*6*) epidural hematoma, and (*7*) injury to the spinal cord and nerve roots (With permission from Danilo Jankovic)

Post-dural Puncture Headache (PDPH)

It is a relatively common complication of a spinal or an epidural anesthetic. The incidence of PDPH with the use of smaller-gauge spinal needle (25–26G) is 0.5–1 %, while puncture with an epidural needle (17–18G) results in PDPH in over 75 % of cases [44].

(a) *Mechanism*: the proposed mechanism of the headache is the loss of CSF causing decrease in CSF pressure. This causes sagging of intracranial structure, with consequent traction on pain-sensitive structures (such as meninges, cranial nerves, and veins) [156]. The second hypothesis suggests that decreased CSF pressure leads to intrathecal hypotension and painful vasodilatation of the intracranial blood vessels (known as the "intracranial vascular response") [157]. Mainly when the patient is in a standing position, painful areas dilate (meninges, tentorium, vessels), and there is further pain transmission via the cerebral nerves and upper cervical nerves.

(b) *Clinical presentation*: PDPH presents as a positional headache, worse on sitting up, 12–48 h after a dural puncture. PDPH is bilateral, predominantly frontal–temporal, and dull or throbbing in nature. Its severity varies, and it may be accompanied by associated symptoms such as nausea, vomiting, photophobia, diplopia, and hearing impairment. While younger females and parturients are at a higher risk of PDPH, older males are at a lower risk [158].

(c) *CSF hypotension syndrome and involvement of the cranial and cervical nerves*: all of the cranial nerves, with the exception of the olfactory nerve, glossopharyngeal nerve, and vagus nerve, can be affected by low CSF pressure. The abducent nerve and vestibulocochlear nerve are most frequently affected (Fig. 41.46). The long intracranial course of the abducent nerve leads to traction and consequent irritation of the nerve when there are changes in intracranial pressure. The patient complains of double vision, with parallel horizontal images and difficulties in focusing on objects [150]. When precise audiometric examinations are carried out, unilateral or bilateral hypoacusis (vestibulocochlear nerve involvement) can be observed in 0.4–40 % of patients with CSF hypotension syndrome [150]. The prognosis is good.

(d) *Differential diagnosis*: this includes migraine, tension headache, cervical myofascial pain (particularly in the sternocleidomastoid muscle, with what is known as "pseudospinal headache"), CNS infections (bacterial meningitis), sinus thrombosis (in the second half of pregnancy or in the puerperium, frequently in pre-eclampsia), and pneumocephalus (after accidental dural perforation in attempted epidural anesthesia when using the loss-of-resistance technique with air) [159].

(e) *Initial management* is conservative and includes simple analgesics and oral hydration. The majority of patients experience marked improvement in the symptoms or complete recovery after 5–7 days with this form of treatment. Other drugs such as caffeine, sumatriptan, and ACTH have been found useful [160–162]. Caffeine produces cerebral vasoconstriction and consequent decrease in cerebral blood flow providing a transient relief from headache. ACTH may stimulate the adrenal gland to increase cerebrospinal fluid production and possibly also increase [beta]-endorphin output [163]. This may thus relieve low CSF pressure and also provide analgesia. Sumatriptan, a serotonin-type 1D receptor agonist, may relieve headache by producing cerebral vasoconstriction [164].

(f) *Invasive management*: the gold standard for the treatment of PDPH remains an autologous blood patch (Figs. 41.47 and 41.48). Its mechanism appears to be the tamponade of dural leak, with subsequent improvement of CSF pressures [165]. It often produces a dramatic relief of symptoms but may need to be repeated occasionally.

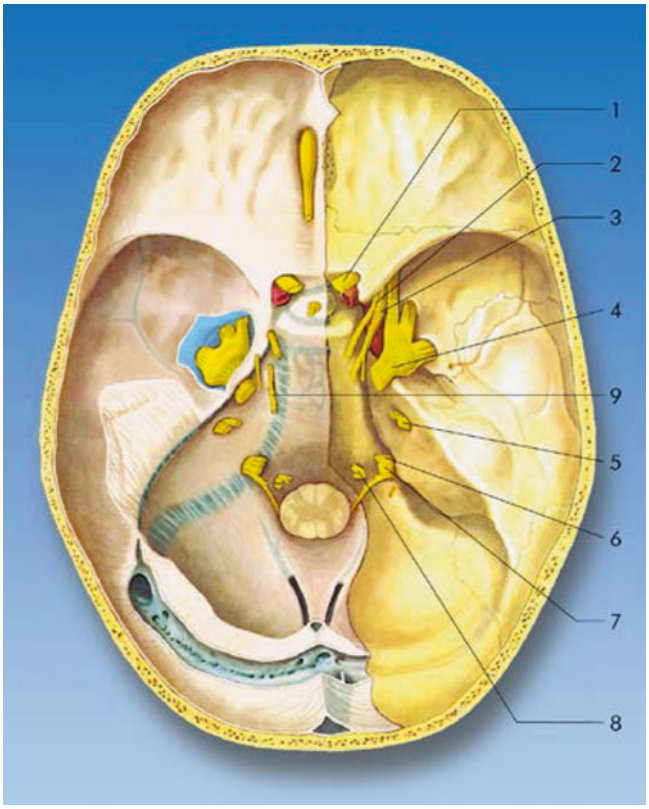


Fig. 41.46 Cranial nerves. (*1*) Optic nerve, (*2*) trochlear nerve, (*3*) trigeminal nerve, (*4*) vestibulocochlear nerve, (*5*) glossopharyngeal nerve, (*7*) vagus nerve, (*8*) hypoglossal nerve, and (*9*) abducent nerve (With permission from Danilo Jankovic)

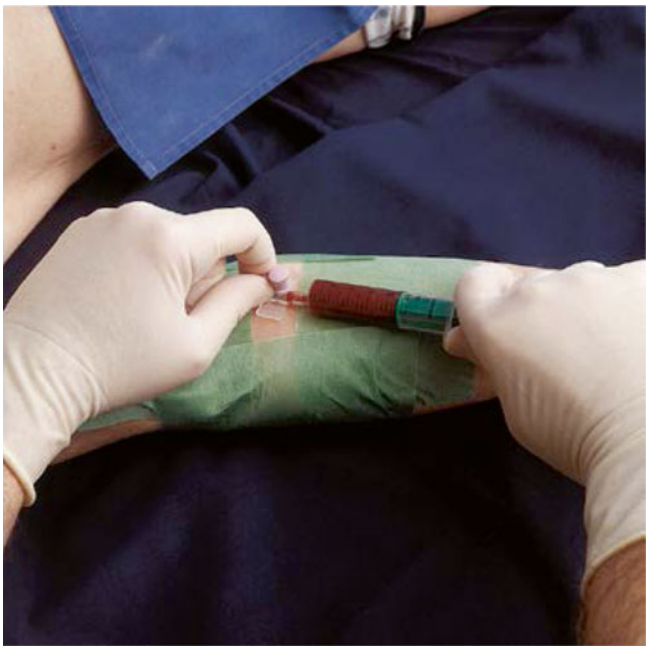


Fig. 41.47 Sterile withdrawal of blood (With permission from Danilo Jankovic)

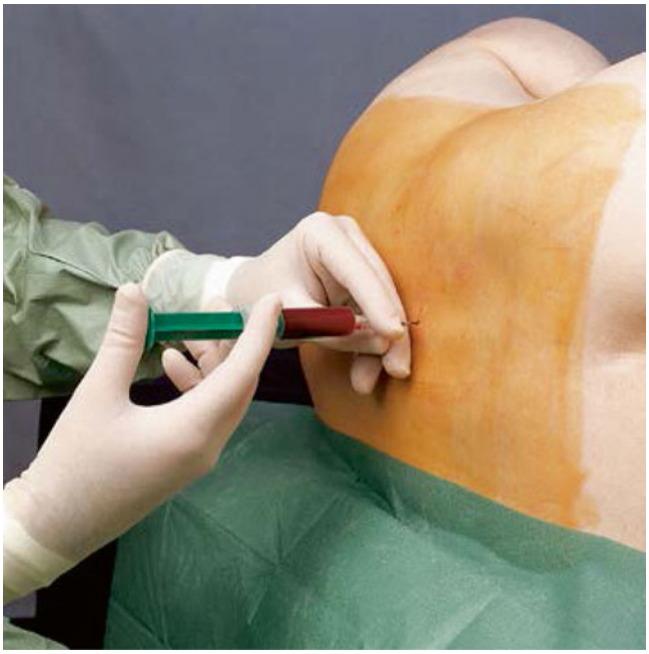
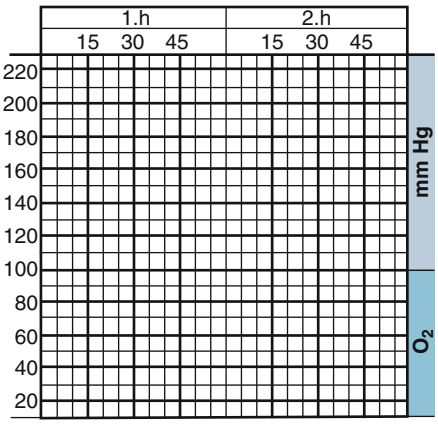


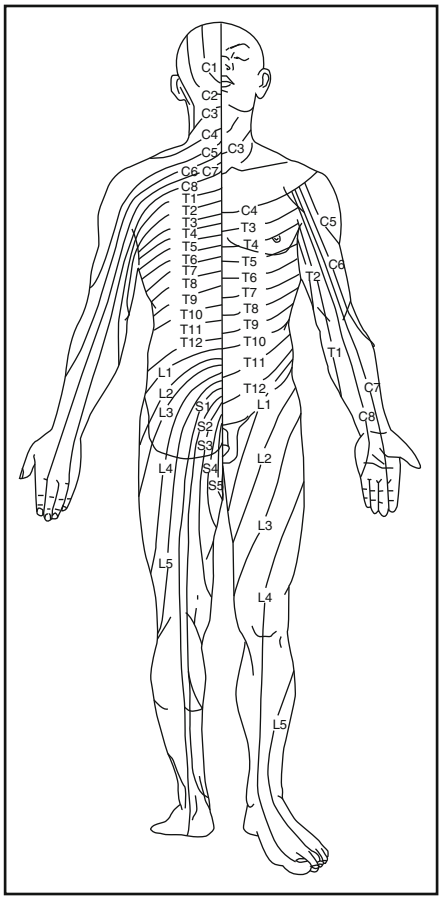
Fig. 41.48 Epidural injection of homologous blood (With permission from Danilo Jankovic)

Spinal or Epidural Hematoma

Although rare, this is the most feared complication of a central neuraxial block (Fig. 41.45). Because the vertebral canal is a confined space, any bleeding may result in compression of the spinal cord resulting in a sensory–motor loss below the level of the compression. Patient with altered hemostasis and ongoing antiplatelet or anticoagulant therapy is at increased risk of developing a spinal or epidural hematoma. This usually presents as a sensory–motor impairment of unusually prolonged duration. Confusing this with an ongoing effect of local anesthetic often delays the correct diagnosis and management. Back pain and bladder or bowel dysfunction may also point toward the possibility of neural damage. Prompt imaging and neurosurgical consultation should be arranged, as the prognosis is poor if the delay between the onset of paralysis and surgical decompression is more than 6–8 h [166]. Guidelines concerning neuraxial anesthesia in anticoagulated patents should be followed to minimize the risks of this devastating complication [167].

Record and checklist





Lumbar epidural anesthesia

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Therapeutic* ☐ *Diagnostic*

Needle: ☐ *Tuohy G*___ ☐ *Other* ___

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Lateral decubitus* ☐ *Sitting*

Access: ☐ *Median* ☐ *Paramedian*

Injection level: ☐ *L3/L4* ☐ *Other* _____

Injection technique: ☐ *Loss of resistance* ☐ *Other* _____

☐ *Ultrasound guided* ☐ *Transducer* ☐ *Curved* ☐ *Linear*

Approach: ☐ *In plane* ☐ *Out of plane*

Epidural space: ☐ *Identified*

Aspiration test: ☐ *Carried out*

Test dose: _____ Epinephrine added: ☐ *Yes* ☐ *No*

Check on sensorimotor function after 5 min: ☐ *Carried out*

Abnormalities: ☐ *No* ☐ *Yes*_____

Injection:

☐ Local anesthetic: _____ *mL* _____ *%*
(incremental)

☐ Addition: _____ *µg/mg*

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 20 min:

☐ *Cold test* ☐ *Temperature measurement before*_____ *°C after* _____ *°C*

☐ *Sensory: L* _____ *T* _____

☐ *Motor*

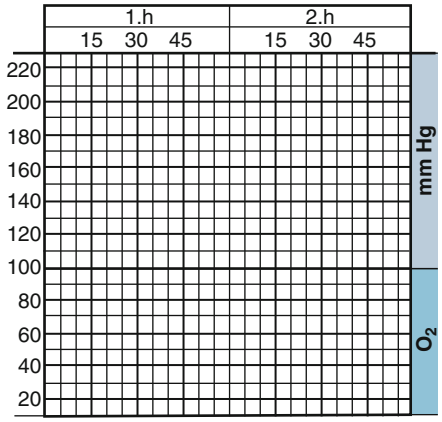
Complications:

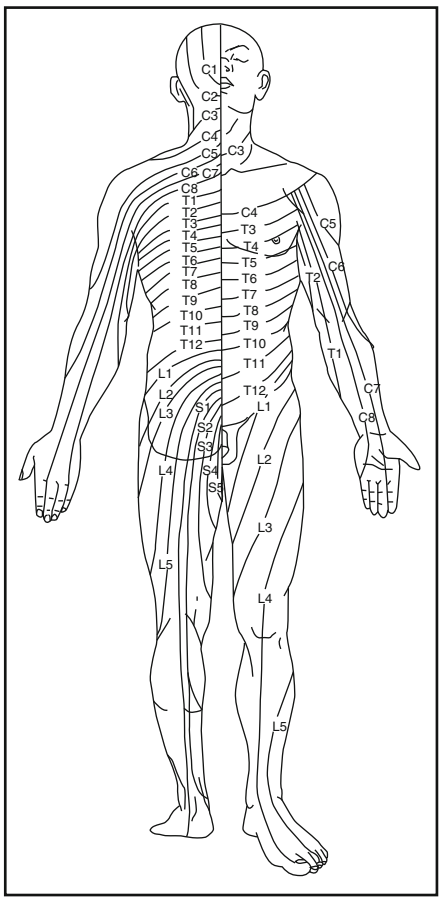
☐ *None*
☐ *Radicular symptoms*
☐ *BP drop*
☐ *Vascular puncture*
☐ *Massive epidural anesthesia*
☐ *Subdural spread*
☐ *Drop in body temperature*
☐ *Bladder emptying disturbances*
☐ *Back pain*
☐ *Pain*
☐ *Vasovagal reactions*
☐ *Dural puncture*
☐ *Intravascular injection*
☐ *Total spinal anesthesia*
☐ *Respiratory disturbance*
☐ *Muscle tremor*
☐ *Postdural puncture headache*
☐ *Neurological complications*

Special notes:

With permission from Danilo Jankovic

Record and checklist





With permission from Danilo Jankovic

Lumbar catheter epidural anesthesia

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Therapeutic*

Needle: ☐ *Tuohy G*___ ☐ *Other* ___

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Lateral decubitus* ☐ *Sitting*

Access: ☐ *Median* ☐ *Paramedian*

Injection level: ☐ *L3/L4* ☐ *Other* _____

Injection technique: ☐ *Loss of resistance* ☐ *Other* _____

☐ *Ultra sound guided* ☐ *Transducer* ☐ *Curved* ☐ *Linear*

Approach: ☐ *In plane* ☐ *Out of plane*

Epidural space: ☐ *Identified*

Catheter: ☐ *Advanced 3–4 em cranially*

Aspiration test: ☐ *Carried out*

Catheter end: ☐ *Placed deeper than the puncture site*

Bacterial filter: ☐

Test dose: _____ Epinephrine added: ☐ *Yes* ☐ *No*

Check on sensorimotor function after 5 min: ☐ *Carried out*

Abnormalities: ☐ *No* ☐ *Yes*_____

Injection:

☐ Local anesthetic: _____ *mL* _____ *%*
(incremental)

☐ Addition: _____ *µg/mg*

☐ Subsequent injection (incremental): _____ *mL* _____ *%*

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 20 min:

☐ *Cold test* ☐ *Temperature measurement before*_____ *°C* *after* _____ *°C*

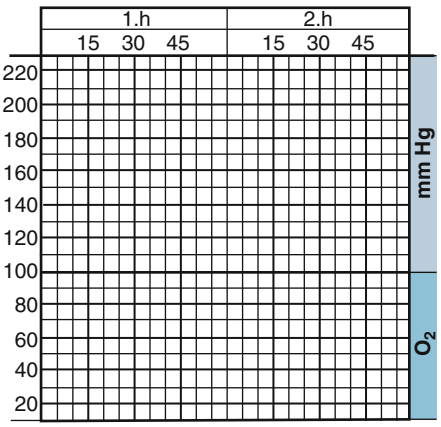
☐ *Sensory: L* _____ *T*_____ ☐ *Motor*

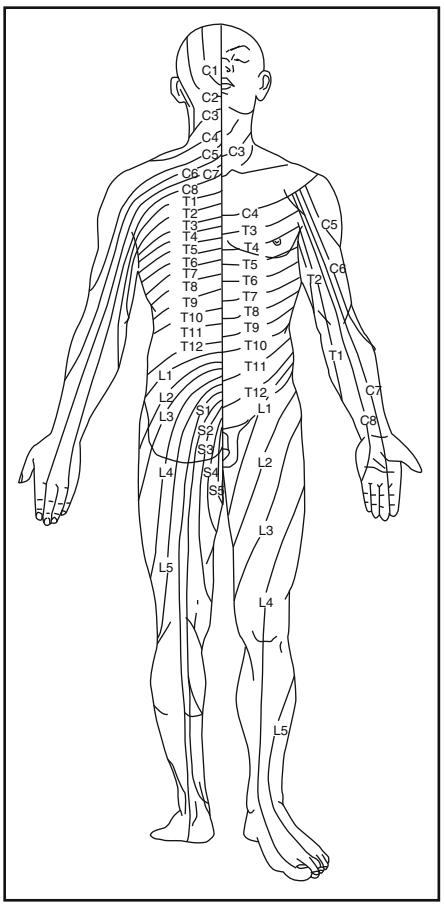
Complications:

☐ *None* ☐ *Radicular symptoms* ☐ *BP drop* ☐ *Vascular puncture*
☐ *Massive epidural anesthesia* ☐ *Subdural spread* ☐ *Drop in body temperature*
☐ *Bladder emptying disturbances* ☐ *Back pain* ☐ *Pain* ☐ *Vasovagal reactions*
☐ *Dural puncture* ☐ *Intravascular injection* ☐ *Total spinal anesthesia*
☐ *Respiratory disturbance* ☐ *Muscle tremor* ☐ *Postdural puncture headache*
☐ *Neurological complications*

Special notes:

Record and checklist





With permission from Danilo Jankovic

Spinal anesthesia

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical*

Needle: ☐ *Pencil-Point G* _____ ☐ *Quincke* ☐ *Other*

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed (behavior after block)*

Position: ☐ *Lateral recumbent* ☐ *Sitting*

Access: ☐ *Median* ☐ *Paramedian* ☐ *Taylor*

Injection level: ☐ *L3/4* ☐ *Other*

CSF: ☐ *Clear* ☐ *Slightly bloody* ☐ *Bloody*

Abnormalities: ☐ *No* ☐ *Yes* _____

Injection technique: ☐ *Traditional* ☐ *Ultrasound guided*

Transducer: ☐ *Curved* ☐ *Linear*

Approach: ☐ *In plane* ☐ *Out of plane*

Local anesthetic: _____ % _____ *mg*

☐ *Hyperbaric* ☐ *Isobaric*

Addition to LA: _____ % _____ *µg/mg*

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement before* ____ *°C after* ____ *°C*

☐ *Sensory: L* _____ *T* _____

☐ *Motor*

Complications:

☐ *None* ☐ *Pain*

☐ *Radicular symptoms* ☐ *Vasovagal reactions*

☐ *BP drop* ☐ *Total spinal anesthesia*

☐ *Subdural spread* ☐ *Respiratory disturbance*

☐ *Drop in body temperature* ☐ *Muscle tremor*

☐ *Bladder emptying disturbances* ☐ *Back pain*

☐ *Postdural puncture headache* ☐ *Neurological complications*

Special notes:

_____

_____

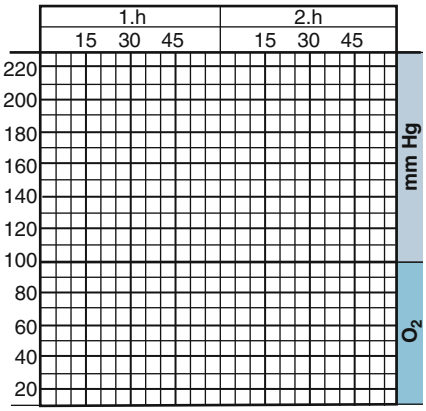
_____

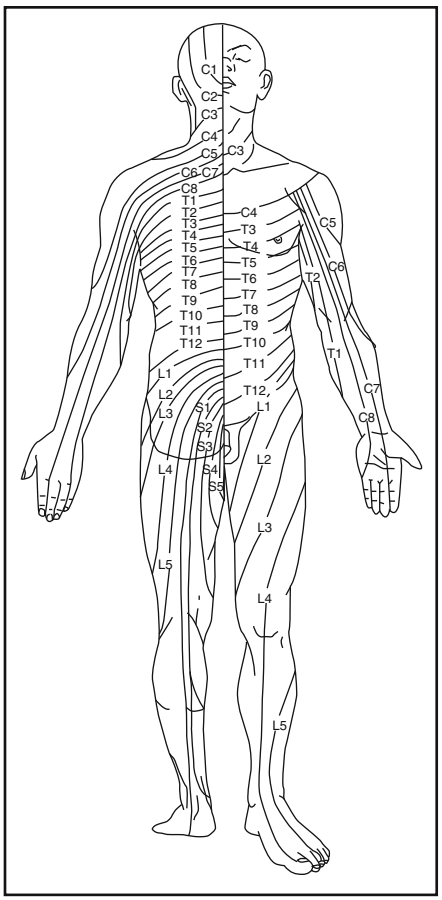
_____

_____

_____

Record and checklist





Continuous spinal anesthesia

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Therapeutic (postoperative)*

Needle: ☐ *G* ____ *Tip:* ____ ☐ *Spinal catheter G* _____ *(micro, macro)* _____

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Lateral recumbent* ☐ *Sitting*

Access: ☐ *Median* ☐ *Paramedian*

Injection level: ☐ *L3/4* ☐ *Other* _____

Technique: ☐ *Through-the-needle* ☐ *Over-the-needle*

Subarachnoid space: ☐ *Identified*

Catheter: ☐ *Advanced 2–3 em*

Aspiration test: ☐ *No* ☐ *Yes*

Bacterial filter: ☐ *No* ☐ *Yes*

Abnormalities: ☐ *No* ☐ *Yes* _____

Injection:

Local anesthetic (isobaric, hyperbaric): _____ *mg* _____ *%*
(incremental)

☐ *Opioid* _____ *(µg)* ☐ *Other* _____ *µg/mg/mL*

☐ *Subsequent injection of* _____ *mL* _____ *%*

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement before* ____ *°C after* ____ *°C*

☐ *Sensory: L* _____ *T* _____

☐ *Motor*

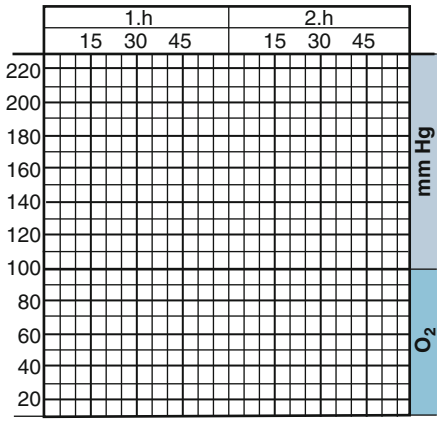
Complications:

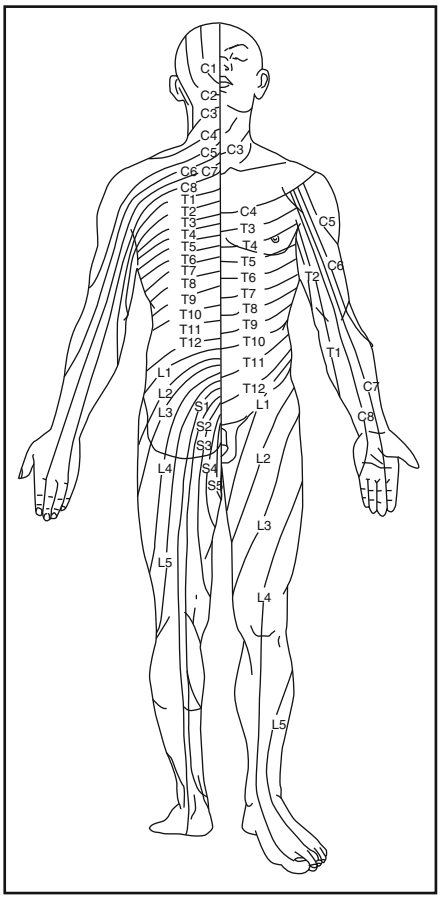
☐ *None*
☐ *Radicular symptoms*
☐ *BP drop*
☐ *Subdural spread*
☐ *Drop in body temperature*
☐ *Bladder emptying disturbances*
☐ *Postdural puncture headache*
☐ *Pain*
☐ *Vasovagal reactions*
☐ *Total spinal anesthesia*
☐ *Respiratory disturbance*
☐ *Muscle tremor*
☐ *Back pain*
☐ *Neurological complications*

Special notes:

With permission from Danilo Jankovic

Record and checklist





Thoracic catheter epidural anesthesia

Name: _____ Date: _____
Diagnosis: _____
Premedication: ☐ No ☐ Yes _____
Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Treatment (postoperative)*
Needle: ☐ *Tuohy G*___ ☐ *Other* ___
i.v. access, infusion: ☐ *Yes*
Monitoring: ☐ *ECG* ☐ *Pulse oximetry*
Ventilation facilities: ☐ *Yes (equipment checked)*
Emergency equipment (drugs): ☐ *Checked*
Patient: ☐ *Informed*

Position: ☐ *Lateral decubitus* ☐ *Sitting*
Access: ☐ *Median* ☐ *Paramedian*
Injection level: ☐ *T* _____
Injection technique: ☐ *Loss of resistance* ☐ *Other* _____
☐ *Ultra sound guided* ☐ *Transducer* ☐ *Curved* ☐ *Linear*
Approach: ☐ *In plane* ☐ *Out of plane*
Epidural space: ☐ *Identified*
Catheter: ☐ *Advanced 3–4 em cranially*
Aspiration test: ☐ *Carried out*
Catheter end positioned lower than the injection site ☐
Bacterial filter: ☐
Test dose: _____ Epinephrine added: ☐ *Yes* ☐ *No*
Check on sensorimotor function after 5 min: ☐ *Carried out*
Abnormalities: ☐ *No* ☐ *Yes*_____
Injection:
☐ Local anesthetic: _____ *mL* _____ *%*
(incremental)
☐ Addition: _____ *µg/mg*
☐ Additional injection *(incremental):* _____ *mL* _____ *%*

Patient's remarks during injection:
☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*
Duration and area: _____
Objective block effect after 20 min:
☐ *Cold test* ☐ *Temperature measurement before*_____ *°C after* _____ *°C*
☐ *Sensory: L* _____ *T* _____ ☐ *Motor*

Complications:
☐ *None* ☐ *Radicular symptoms* ☐ *BP drop* ☐ *Vascular puncture*
☐ *Massive epidural anesthesia* ☐ *Subdural spread* ☐ *Drop in body temperature*
☐ *Bladder emptying disturbances* ☐ *Back pain* ☐ *Pain* ☐ *Vasovagal reactions*
☐ *Dural puncture* ☐ *Intravascular injection* ☐ *Total spinal anesthesia*
☐ *Respiratory disturbance* ☐ *Muscle tremor* ☐ *Postdural puncture headache*
☐ *Neurological complications*

Special notes:

With permission from Danilo Jankovic

References

1. 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.
2. Tamakawa S. Comparing the confidence of the 7th cervical spinous process, the inferior angle of the scapula, and Tuffier's line. Reg Anesth Pain Med. 1999;24(6):585–6.
3. Kim J-T, Bahk J-H, Sung J. Influence of age and sex on the position of the conus medullaris and Tuffier's line in adults. Anesthesiology. 2003;99(6):1359–63.
4. Margarido CB, Mikhael R, Arzola C, Balki M, Carvalho JCA. The intercristal line determined by palpation is not a reliable anatomical landmark for neuraxial anesthesia. Can J Anaesth. 2011;58(3):262–6.
5. Snider KT, Kribs JW, Snider EJ, Degenhardt BF, Bukowski A, Johnson JC. Reliability of Tuffier's line as an anatomic landmark. Spine (Phila Pa 1976). 2008;33(6):E161–5.
6. Furness G, Reilly MP, Kuchi S. An evaluation of ultrasound imaging for identification of lumbar intervertebral level. Anaesthesia. 2002;57(3):277–80.
7. Pysyk CL, Persaud D, Bryson GL, Lui A. Ultrasound assessment of the vertebral level of the palpated intercristal (Tuffier's) line. Can J Anaesth. 2010;57(1):46–9.
8. Panjabi MM, Duranceau J, Goel V, Oxland T, Takata K. Cervical human vertebrae. Quantitative three-dimensional anatomy of the middle and lower regions. Spine (Phila Pa 1976). 1991;16(8):861–9.
9. Panjabi MM, Takata K, Goel V, Federico D, Oxland T, Duranceau J, et al. Thoracic human vertebrae. Quantitative three-dimensional anatomy. Spine (Phila Pa 1976). 1991;16(8):888–901.
10. Panjabi MM, Goel V, Oxland T, Takata K, Duranceau J, Krag M, et al. Human lumbar vertebrae. Quantitative three-dimensional anatomy. Spine (Phila Pa 1976). 1992;17(3):299–306.
11. Barker AE. A report on clinical experiences with spinal analgesia in 100 cases, and some reflections on the procedure. Br Med J. 1907;1(2412):665–74.
12. Cheng PA. The anatomical and clinical aspects of epidural anesthesia. II. Anesth Analg. 1963;42(4):407–15.
13. Cheng PA. The anatomical and clinical aspects of epidural anesthesia. I. Anesth Analg. 1963;42(3):398–406.
14. Hughes G, Ugokwe K, Benzel EC. A review of spinal arachnoid cysts. Cleve Clin J Med. 2008;75(4):311–5.
15. Stace JD, Gaylard DG. Failed spinal anaesthesia. Anaesthesia. 1996;51(9):892–3.
16. Bromage PR. Neurological complications of subarachnoid and epidural anaesthesia. Acta Anaesthesiol Scand. 1997;41(4):439–44.
17. Lirk P, Kolbitsch C, Putz G, Colvin J, Colvin HP, Lorenz I, et al. Cervical and high thoracic ligamentum flavum frequently fails to fuse in the midline. Anesthesiology. 2003;99(6):1387–90.
18. Hogan QH. Lumbar epidural anatomy. A new look by cryomicrotome section. Anesthesiology. 1991;75(5):767–75.
19. Hogan Q. Distribution of solution in the epidural space: examination by cryomicrotome section. Reg Anesth Pain Med. 2002;27(2):150–6.
20. Reina MA, Franco CD, López A, Dé Andrés JA, van Zundert A. Clinical implications of epidural fat in the spinal canal. A scanning electron microscopic study. Acta Anaesthesiol Belg. 2009; 60(1):7–17.
21. Bernards CM, Shen DD, Sterling ES, Adkins JE, Risler L, Phillips B, et al. Epidural, cerebrospinal fluid, and plasma pharmacokinetics of epidural opioids (part 1): differences among opioids. Anesthesiology. 2003;99(2):455–65.
22. Igarashi T, Hirabayashi Y, Shimizu R, Saitoh K, Fukuda H, Mitsuhata H. The lumbar extradural structure changes with increasing age. Br J Anaesth. 1997;78(2):149–52.
23. Harrison GR, Clowes NW. The depth of the lumbar epidural space from the skin. Anaesthesia. 1985;40(7):685–7.
24. Ravi KK, Kaul TK, Kathuria S, Gupta S, Khurana S. Distance from skin to epidural space: correlation with body mass index (BMI). J Anaesthesiol Clin Pharmacol. 2011;27(1):39–42.
25. Balki M, Lee Y, Halpern S, Carvalho JCA. Ultrasound imaging of the lumbar spine in the transverse plane: the correlation between estimated and actual depth to the epidural space in obese parturients. Anesth Analg. 2009;108(6):1876–81.
26. Fink BR, Cairns AM. Differential slowing and block of conduction by lidocaine in individual afferent myelinated and unmyelinated axons. Anesthesiology. 1984;60(2):111–20.
27. Jaffe RA, Rowe MA. Differential nerve block. Direct measurements on individual myelinated and unmyelinated dorsal root axons. Anesthesiology. 1996;84(6):1455–64.
28. Liu S, Kopacz DJ, Carpenter RL. Quantitative assessment of differential sensory nerve block after lidocaine spinal anesthesia. Anesthesiology. 1995;82(1):60–3.
29. Neal JM. Hypotension and bradycardia during spinal anesthesia: significance, prevention, and treatment. Tech Reg Anesth Pain Manag. 2000;4(4):148–54.
30. Crystal GJ, Salem MR. The Bainbridge and the "reverse" Bainbridge reflexes: history, physiology, and clinical relevance. Anesth Analg. 2012;114(3):520–32.
31. Campagna JA, Carter C. Clinical relevance of the Bezold-Jarisch reflex. Anesthesiology. 2003;98(5):1250–60.
32. Otton PE, Wilson EJ. The cardiocirculatory effects of upper thoracic epidural analgesia. Can Anaesth Soc J. 1966;13(6):541–9.
33. Subramani K, Paul A. Laryngospasm during subarachnoid block. Br J Anaesth. 2005;94(5):668–70.
34. Pollard JB. Cardiac arrest during spinal anesthesia: common mechanisms and strategies for prevention. Anesth Analg. 2001; 92(1):252–6.
35. Steinbrook RA, Concepcion M. Respiratory effects of spinal anesthesia: resting ventilation and single-breath CO_2 response. Anesth Analg. 1991;72(2):182–6.
36. Groeben H. Epidural anesthesia and pulmonary function. J Anesth. 2006;20(4):290–9.
37. Leslie JB, Viscusi ER, Pergolizzi JV, Panchal SJ. Anesthetic routines: the anesthesiologist's role in GI recovery and postoperative ileus. Adv Prev Med. 2011;2011:976904.
38. Shi W-Z, Miao Y-L, Yakoob MY, Cao J-B, Zhang H, Jiang Y-G, et al. Recovery of gastrointestinal function with thoracic epidural vs. systemic analgesia following gastrointestinal surgery. Acta Anaesthesiol Scand. 2014;58(8):923–32.
39. Carpenter RL, Caplan RA, Brown DL, Stephenson C, Wu R. Incidence and risk factors for side effects of spinal anesthesia. Anesthesiology. 1992;76(6):906–16.
40. Sessler DI, Ponte J. Shivering during epidural anesthesia. Anesthesiology. 1990;72(5):816–21.
41. Shehabi Y, Gatt S, Buckman T, Isert P. Effect of adrenaline, fentanyl and warming of injectate on shivering following extradural analgesia in labour. Anaesth Intensive Care. 1990;18(1):31–7.
42. Hjortsø NC, Neumann P, Frøsig F, Andersen T, Lindhard A, Rogon E, et al. A controlled study on the effect of epidural analgesia with local anaesthetics and morphine on morbidity after abdominal surgery. Acta Anaesthesiol Scand. 1985;29(8): 790–6.
43. Hosoda R, Hattori M, Shimada Y. Favorable effects of epidural analgesia on hemodynamics, oxygenation and metabolic variables in the immediate post-anesthetic period. Acta Anaesthesiol Scand. 1993;37(5):469–74.
44. Liu S, Carpenter RL, Neal JM. Epidural anesthesia and analgesia. Their role in postoperative outcome. Anesthesiology. 1995;82(6): 1474–506.
45. Ballantyne J. Does regional anesthesia improve outcome after surgery? Curr Opin Anaesthesiol. 1999;12(5):545–9.
46. Breivik H, Bang U, Jalonen J, Vigfússon G, Alahuhta S, Lagerkranser M. Nordic guidelines for neuraxial blocks in dis-

turbed haemostasis from the Scandinavian Society of Anaesthesiology and Intensive Care Medicine. Acta Anaesthesiol Scand. 2010;54(1):16–41.
47. Kettner SC, Willschke H, Marhofer P. Does regional anaesthesia really improve outcome? Br J Anaesth. 2011;107 Suppl 1: i90–5.
48. Memtsoudis SG, Sun X, Chiu Y-L, Stundner O, Liu SS, Banerjee S, et al. Perioperative comparative effectiveness of anesthetic technique in orthopedic patients. Anesthesiology. 2013;118(5):1046–58.
49. 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(1):102–5.
50. Hurst E. Adhesive arachnoiditis and vascular blockage caused by detergents and other chemical irritants: an experimental study. J Pathol Bacteriol. 1955;70(1):167–78.
51. Cook TM, Counsell D, Wildsmith JAW. 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.
52. Lambert DH, Hurley RJ, Hertwig L, Datta S. Role of needle gauge and tip configuration in the production of lumbar puncture headache. Reg Anesth. 1997;22(1):66–72.
53. Parker RK, White PF. A microscopic analysis of cut-bevel versus pencil-point spinal needles. Anesth Analg. 1997;85(5): 1101–4.
54. Richman JM, Joe EM, Cohen SR, Rowlingson AJ, Michaels RK, Jeffries MA, et al. Bevel direction and postdural puncture headache: a meta-analysis. Neurologist. 2006;12(4):224–8.
55. Obasuyi BI, Fyneface-Ogan S, Mato CN. A comparison of the haemodynamic effects of lateral and sitting positions during induction of spinal anaesthesia for caesarean section. Int J Obstet Anesth. 2013;22(2):124–8.
56. Kinsella SM, Lohmann G. Supine hypotensive syndrome. Obstet Gynecol. 1994;83(5 Pt 1):774–88.
57. Edgcombe H, Carter K, Yarrow S. Anaesthesia in the prone position. Br J Anaesth. 2008;100(2):165–83.
58. Rabinowitz A, Bourdet B, Minville V, Chassery C, Pianezza A, Colombani A, et al. The paramedian technique: a superior initial approach to continuous spinal anesthesia in the elderly. Anesth Analg. 2007;105(6):1855–7.
59. Enk D, Prien T, Van Aken H, Mertes N, Meyer J, Brüssel T. Success rate of unilateral spinal anesthesia is dependent on injection flow. Reg Anesth Pain Med. 2001;26(5):420–7.
60. Casati A, Fanelli G. Unilateral spinal anesthesia. State of the art. Minerva Anestesiol. 2001;67(12):855–62.
61. Kuusniemi KS, Pihlajamäki KK, Pitkänen MT, Korkeila JE. A low-dose hypobaric bupivacaine spinal anesthesia for knee arthroscopies. Reg Anesth. 1997;22(6):534–8.
62. Denny NM, Selander DE. Continuous spinal anaesthesia. Br J Anaesth. 1998;81(4):590–7.
63. Gosch UW, Hueppe M, Hallschmid M, Born J, Schmucker P, Meier T. Post-dural puncture headache in young adults: comparison of two small-gauge spinal catheters with different needle design. Br J Anaesth. 2005;94(5):657–61.
64. Horlocker TT, McGregor DG, Matsushige DK, Chantigian RC, Schroeder DR, Besse JA. Neurologic complications of 603 consecutive continuous spinal anesthetics using macrocatheter and microcatheter techniques. Perioperative Outcomes Group. Anesth Analg. 1997;84(5):1063–70.
65. Faust A, Fournier R, Van Gessel E, Weber A, Hoffmeyer P, Gamulin Z. Isobaric versus hypobaric spinal bupivacaine for total hip arthroplasty in the lateral position. Anesth Analg. 2003;97(2):589–94, table of contents.
66. Dyamanna DN, Bs SK, Zacharia BT. Unexpected bradycardia and cardiac arrest under spinal anesthesia: case reports and review of literature. Middle East J Anesthesiol. 2013;22(1):121–5.
67. Russell IF. A comparison of cold, pinprick and touch for assessing the level of spinal block at caesarean section. Int J Obstet Anesth. 2004;13(3):146–52.
68. Ousley R, Egan C, Dowling K, Cyna AM. Assessment of block height for satisfactory spinal anaesthesia for caesarean section. Anaesthesia. 2012;67(12):1356–63.
69. Zarzur E. Anatomic studies of the human ligamentum flavum. Anesth Analg. 1984;63(5):499–502.
70. Saberski LR, Kondamuri S, Osinubi OY. Identification of the epidural space: is loss of resistance to air a safe technique? A review of the complications related to the use of air. Reg Anesth. 1997; 22(1):3–15.
71. Todorov L, VadeBoncouer T. Etiology and use of the "hanging drop" technique: a review. Pain Res Treat. 2014;2014:Article ID 146750, 10 p.
72. Guay J. The epidural test dose: a review. Anesth Analg. 2006;102(3):921–9.
73. Walker DS, Brock-Utne JG. A comparison of simple tests to distinguish cerebrospinal fluid from saline. Can J Anaesth. 1997;44(5 Pt 1):494–7.
74. Hermanides J, Hollmann MW, Stevens MF, Lirk P. Failed epidural: causes and management. Br J Anaesth. 2012;109(2):144–54.
75. Arendt K, Segal S. Why epidurals do not always work. Rev Obstet Gynecol. 2008;1(2):49–55.
76. Camorcia M, Capogna G. Sensory assessment of epidural block for Caesarean section: a systematic comparison of pinprick, cold and touch sensation. Eur J Anaesthesiol. 2006;23(7):611–7.
77. Graham AC, McClure JH. Quantitative assessment of motor block in labouring women receiving epidural analgesia. Anaesthesia. 2001;56(5):470–6.
78. Van Zundert A, Vaes L, Van der Aa P, Van der Donck A, Meeuwis H. Motor blockade during epidural anesthesia. Anesth Analg. 1986;65(4):333–6.
79. Ellinas EH, Eastwood DC, Patel SN, Maitra-D'Cruze AM, Ebert TJ. The effect of obesity on neuraxial technique difficulty in pregnant patients: a prospective, observational study. Anesth Analg. 2009;109(4):1225–31.
80. Hood DD, Dewan DM. Anesthetic and obstetric outcome in morbidly obese parturients. Anesthesiology. 1993;79(6): 1210–8.
81. Grau T, Leipold RW, Conradi R, Martin E, Motsch J. Efficacy of ultrasound imaging in obstetric epidural anesthesia. J Clin Anesth. 2002;14(3):169–75.
82. White AA, Panjabi MM. The clinical biomechanics of scoliosis. Clin Orthop Relat Res. 1976;118:100–12.
83. Boon JM, Prinsloo E, Raath RP. A paramedian approach for epidural block: an anatomic and radiologic description. Reg Anesth Pain Med. 2003;28(3):221–7.
84. Huang J. Paramedian approach for neuroaxial anesthesia in parturients with scoliosis. Anesth Analg. 2010;111(3):821–2; author reply 822.
85. Berkowitz S, Gold MI. Spinal anesthesia for surgery in patients with previous lumbar laminectomy. Anesth Analg. 1980;59(11):881–2.
86. Kim SH, Jeon D-H, Chang CH, Lee S-J, Shin Y-S. Spinal anesthesia with isobaric tetracaine in patients with previous lumbar spinal surgery. Yonsei Med J. 2009;50(2):252–6.
87. Sharrock NE, Urquhart B, Mineo R. Extradural anaesthesia in patients with previous lumbar spine surgery. Br J Anaesth. 1990;65(2):237–9.
88. Brodsky AE. Post-laminectomy and post-fusion stenosis of the lumbar spine. Clin Orthop Relat Res. 1976;115:130–9.

89. LaRocca H, Macnab I. The laminectomy membrane. Studies in its evolution, characteristics, effects and prophylaxis in dogs. J Bone Joint Surg Br. 1974;56B(3):545–50.
90. Yamauchi M, Honma E, Mimura M, Yamamoto H, Takahashi E, Namiki A. Identification of the lumbar intervertebral level using ultrasound imaging in a post-laminectomy patient. J Anesth. 2006;20(3):231–3.
91. Greene NM. Distribution of local anesthetic solutions within the subarachnoid space. Anesth Analg. 1985;64(7):715–30.
92. Hocking G, Wildsmith JAW. Intrathecal drug spread. Br J Anaesth. 2004;93(4):568–78.
93. Stienstra R, van Poorten JF. Plain or hyperbaric bupivacaine for spinal anesthesia. Anesth Analg. 1987;66(2):171–6.
94. Van Zundert AA, Grouls RJ, Korsten HH, Lambert DH. Spinal anesthesia. Volume or concentration–what matters? Reg Anesth. 1996;21(2):112–8.
95. Wildsmith JA, McClure JH, Brown DT, Scott DB. Effects of posture on the spread of isobaric and hyperbaric amethocaine. Br J Anaesth. 1981;53(3):273–8.
96. Kito K, Kato H, Shibata M, Adachi T, Nakao S, Mori K. The effect of varied doses of epinephrine on duration of lidocaine spinal anesthesia in the thoracic and lumbosacral dermatomes. Anesth Analg. 1998;86(5):1018–22.
97. Singh H, Yang J, Thornton K, Giesecke AH. Intrathecal fentanyl prolongs sensory bupivacaine spinal block. Can J Anaesth. 1995; 42(11):987–91.
98. Hirabayashi Y, Shimizu R, Saitoh K, Fukuda H, Furuse M. Anatomical configuration of the spinal column in the supine position. I. A study using magnetic resonance imaging. Br J Anaesth. 1995;75(1):3–5.
99. Tuominen M, Taivainen T, Rosenberg PH. Spread of spinal anaesthesia with plain 0.5 % bupivacaine: influence of the vertebral interspace used for injection. Br J Anaesth. 1989;62(4): 358–61.
100. Sakura S, Sumi M, Morimoto N, Yamamori Y, Saito Y. Spinal anesthesia with tetracaine in 0.75 % glucose: influence of the vertebral interspace used for injection. Reg Anesth Pain Med. 1998;23(2):170–5.
101. Urmey WF, Stanton J, Bassin P, Sharrock NE. The direction of the Whitacre needle aperture affects the extent and duration of isobaric spinal anesthesia. Anesth Analg. 1997;84(2):337–41.
102. Stienstra R, van Poorten F, Kroon JW. Needle direction affects the sensory level of spinal anesthesia. Anesth Analg. 1989;68(4):497–500.
103. Janik R, Dick W, Stanton-Hicks MD. Influence of barbotage on block characteristics during spinal anesthesia with hyperbaric tetracaine and bupivacaine. Reg Anesth. 1989;14(1):26–30.
104. Van Bogaert LJ. Lumbar lordosis and the spread of subarachnoid hyperbaric 0.5 % bupivacaine at cesarean section. Int J Gynaecol Obstet. 2000;71(1):65–6.
105. Roofthooft E, Van de Velde M. Low-dose spinal anaesthesia for Caesarean section to prevent spinal-induced hypotension. Curr Opin Anaesthesiol. 2008;21(3):259–62.
106. Cohen EN. Distribution of local anesthetic agents in the neuraxis of the dog. Anesthesiology. 1968;29(5):1002–5.
107. Burm AG, van Kleef JW, Gladines MP, Spierdijk J, Breimer DD. Plasma concentrations of lidocaine and bupivacaine after subarachnoid administration. Anesthesiology. 1983;59(3):191–5.
108. Greene NM. Uptake and elimination of local anesthetics during spinal anesthesia. Anesth Analg. 1983;62(11):1013–24.
109. Burm AG, Van Kleef JW, Vermeulen NP, Olthof G, Breimer DD, Spierdijk J. Pharmacokinetics of lidocaine and bupivacaine following subarachnoid administration in surgical patients: simultaneous investigation of absorption and disposition kinetics using stable isotopes. Anesthesiology. 1988;69(4):584–92.
110. Kooger Infante NE, Van Gessel E, Forster A, Gamulin Z. Extent of hyperbaric spinal anesthesia influences the duration of spinal block. Anesthesiology. 2000;92(5):1319–23.
111. Moore JM, Liu SS, Pollock JE, Neal JM, Knab JH. The effect of epinephrine on small-dose hyperbaric bupivacaine spinal anesthesia: clinical implications for ambulatory surgery. Anesth Analg. 1998;86(5):973–7.
112. Rathmell JP, Lair TR, Nauman B. The role of intrathecal drugs in the treatment of acute pain. Anesth Analg. 2005;101(5 Suppl):S30–43.
113. Axelrod EH, Alexander GD, Brown M, Schork MA. Procaine spinal anesthesia: a pilot study of the incidence of transient neurologic symptoms. J Clin Anesth. 1998;10(5):404–9.
114. 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(5):470–3.
115. Casati A, Fanelli G, Danelli G, Berti M, Ghisi D, Brivio M, et al. Spinal anesthesia with lidocaine or preservative-free 2-chloroprocaine for outpatient knee arthroscopy: a prospective, randomized, double-blind comparison. Anesth Analg. 2007;104(4):959–64.
116. Black AS, Newcombe GN, Plummer JL, McLeod DH, Martin DK. Spinal anaesthesia for ambulatory arthroscopic surgery of the knee: a comparison of low-dose prilocaine and fentanyl with bupivacaine and fentanyl. Br J Anaesth. 2011;106(2):183–8.
117. Gebhardt V, Herold A, Weiss C, Samakas A, Schmittner MD. Dosage finding for low-dose spinal anaesthesia using hyperbaric prilocaine in patients undergoing perianal outpatient surgery. Acta Anaesthesiol Scand. 2013;57(2):249–56.
118. Zaric D, Pace NL. Transient neurologic symptoms (TNS) following spinal anaesthesia with lidocaine versus other local anaesthetics. Cochrane Database Syst Rev. 2009;2:CD003006.
119. Nair GS, Abrishami A, Lermitte J, Chung F. Systematic review of spinal anaesthesia using bupivacaine for ambulatory knee arthroscopy. Br J Anaesth. 2009;102(3):307–15.
120. Alley EA, Kopacz DJ, McDonald SB, Liu SS. Hyperbaric spinal levobupivacaine: a comparison to racemic bupivacaine in volunteers. Anesth Analg. 2002;94(1):188–93, table of contents.
121. McDonald SB, Liu SS, Kopacz DJ, Stephenson CA. Hyperbaric spinal ropivacaine: a comparison to bupivacaine in volunteers. Anesthesiology. 1999;90(4):971–7.
122. Gehling M, Tryba M. Risks and side-effects of intrathecal morphine combined with spinal anaesthesia: a meta-analysis. Anaesthesia. 2009;64(6):643–51.
123. 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.
124. Sultan P, Gutierrez MC, Carvalho B. Neuraxial morphine and respiratory depression: finding the right balance. Drugs. 2011;71(14):1807–19.
125. Motiani P, Chaudhary S, Bahl N, Sethi AK. Intrathecal sufentanil versus fentanyl for lower limb surgeries – a randomized controlled trial. J Anaesthesiol Clin Pharmacol. 2011;27(1):67–73.
126. Nelson KE, Rauch T, Terebuh V, D'Angelo R. A comparison of intrathecal fentanyl and sufentanil for labor analgesia. Anesthesiology. 2002;96(5):1070–3.
127. Concepcion M, Maddi R, Francis D, Rocco AG, Murray E, Covino BG. Vasoconstrictors in spinal anesthesia with tetracaine–a comparison of epinephrine and phenylephrine. Anesth Analg. 1984;63(2):134–8.
128. Dobrydnjov I, Axelsson K, Samarütel J, Holmström B. Postoperative pain relief following intrathecal bupivacaine combined with intrathecal or oral clonidine. Acta Anaesthesiol Scand. 2002;46(7):806–14.
129. Elia N, Culebras X, Mazza C, Schiffer E, Tramèr MR. Clonidine as an adjuvant to intrathecal local anesthetics for surgery:

systematic review of randomized trials. Reg Anesth Pain Med. 2008;33(2):159–67.
130. Kim JE, Kim NY, Lee HS, Kil HK. Effects of intrathecal dexmedetomidine on low-dose bupivacaine spinal anesthesia in elderly patients undergoing transurethral prostatectomy. Biol Pharm Bull. 2013;36(6):959–65.
131. Bromage PR. Mechanism of action of extradural analgesia. Br J Anaesth. 1975;47(suppl):199–211.
132. Duggan J, Bowler GM, McClure JH, Wildsmith JA. Extradural block with bupivacaine: influence of dose, volume, concentration and patient characteristics. Br J Anaesth. 1988;61(3):324–31.
133. Visser WA, Lee RA, Gielen MJM. Factors affecting the distribution of neural blockade by local anesthetics in epidural anesthesia and a comparison of lumbar versus thoracic epidural anesthesia. Anesth Analg. 2008;107(2):708–21.
134. Bromage P, Burfoot M, Crowell D, Pettigrew R. Quality of epidural blockade. I. Influence of physical factors. Br J Anaesth. 1964;36:342–52.
135. Reddy SV, Yaksh TL. Spinal noradrenergic terminal system mediates antinociception. Brain Res. 1980;189(2):391–401.
136. Bonica JJ, Kennedy WF, Ward RJ, Tolas AG. A comparison of the effects of high subarachnoid and epidural anesthesia. Acta Anaesthesiol Scand Suppl. 1966;23:429–37.
137. De Leon-Casasola OA, Lema MJ. Postoperative epidural opioid analgesia: what are the choices? Anesth Analg. 1996;83(4):867–75.
138. Curatolo M, Petersen-Felix S, Arendt-Nielsen L, Lauber R, Högström H, Scaramozzino P, et al. Adding sodium bicarbonate to lidocaine enhances the depth of epidural blockade. Anesth Analg. 1998;86(2):341–7.
139. 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.
140. Lesser JB, Sanborn KV, Valskys R, Kuroda M. Severe bradycardia during spinal and epidural anesthesia recorded by an anesthesia information management system. Anesthesiology. 2003;99(4):859–66.
141. Nortcliffe S-A, Shah J, Buggy DJ. Prevention of postoperative nausea and vomiting after spinal morphine for Caesarean section: comparison of cyclizine, dexamethasone and placebo. Br J Anaesth. 2003;90(5):665–70.
142. Butler R, Fuller J. Back pain following epidural anaesthesia in labour. Can J Anaesth. 1998;45(8):724–8.
143. Chan YK, Gopinathan R, Rajendram R. Loss of consciousness following spinal anaesthesia for Caesarean section. Br J Anaesth. 2000;85(3):474–6.
144. Palkar NV, Boudreaux RC, Mankad AV. Accidental total spinal block: a complication of an epidural test dose. Can J Anaesth. 1992;39(10):1058–60.
145. Singh B, Sharma P. Subdural block complicating spinal anesthesia? Anesth Analg. 2002;94(4):1007–9, table of contents.
146. Dahlgren N, Törnebrandt K. Neurological complications after anaesthesia. A follow-up of 18,000 spinal and epidural anaesthetics performed over three years. Acta Anaesthesiol Scand. 1995;39(7):872–80.
147. Gazmuri RR, Ricke CA, Dagnino JA. Trigeminal nerve block as a complication of epidural anesthesia. Reg Anesth. 1992;17(1):50–1.
148. Sprung J, Haddox JD, Maitra-D'Cruze AM. Horner's syndrome and trigeminal nerve palsy following epidural anaesthesia for obstetrics. Can J Anaesth. 1991;38(6):767–71.
149. Evans JM, Gauci CA, Watkins G. Horner's syndrome as a complication of lumbar epidural block. Anaesthesia. 1975;30(6):774–7.
150. Day CJ, Shutt LE. Auditory, ocular, and facial complications of central neural block. A review of possible mechanisms. Reg Anesth. 1996;21(3):197–201.
151. Okell RW, Sprigge JS. Unintentional dural puncture. A survey of recognition and management. Anaesthesia. 1987;42(10):1110–3.
152. Reynolds F. Damage to the conus medullaris following spinal anaesthesia. Anaesthesia. 2001;56(3):238–47.
153. Radwan IAM, Saito S, Goto F. The neurotoxicity of local anesthetics on growing neurons: a comparative study of lidocaine, bupivacaine, mepivacaine, and ropivacaine. Anesth Analg. 2002;94(2):319–24, table of contents.
154. Pollock JE. Neurotoxicity of intrathecal local anaesthetics and transient neurological symptoms. Best Pract Res Clin Anaesthesiol. 2003;17(3):471–84.
155. Rigler ML, Drasner K, Krejcie TC, Yelich SJ, Scholnick FT, DeFontes J, et al. Cauda equina syndrome after continuous spinal anesthesia. Anesth Analg. 1991;72(3):275–81.
156. Turnbull DK, Shepherd DB. Post-dural puncture headache: pathogenesis, prevention and treatment. Br J Anaesth. 2003;91(5):718–29.
157. Camann WR, Murray RS, Mushlin PS, Lambert DH. Effects of oral caffeine on postdural puncture headache. A double-blind, placebo-controlled trial. Anesth Analg. 1990;70(2):181–4.
158. Evans RW. Complications of lumbar puncture. Neurol Clin. 1998;16(1):83–105.
159. González-Carrasco FJ, Aguilar JL, Llubiá C, Nogués S, Vidal-López F. Pneumocephalus after accidental dural puncture during epidural anesthesia. Reg Anesth. 1993;18(3):193–5.
160. Halker RB, Demaerschalk BM, Wellik KE, Wingerchuk DM, Rubin DI, Crum BA, et al. Caffeine for the prevention and treatment of postdural puncture headache: debunking the myth. Neurologist. 2007;13(5):323–7.
161. Basurto Ona X, Martínez García L, Solà I, Bonfill Cosp X. Drug therapy for treating post-dural puncture headache. Cochrane Database Syst Rev. 2011;8:CD007887.
162. Ghaleb A, Khorasani A, Mangar D. Post-dural puncture headache. Int J Gen Med. 2012;5:45–51.
163. Carter BL, Pasupuleti R. Use of intravenous cosyntropin in the treatment of postdural puncture headache. Anesthesiology. 2000;92(1):272–4.
164. Carp H, Singh PJ, Vadhera R, Jayaram A. Effects of the serotonin-receptor agonist sumatriptan on postdural puncture headache: report of six cases. Anesth Analg. 1994;79(1):180–2.
165. Gielen M. Post dural puncture headache (PDPH): a review. Reg Anesth. 1989;14(3):101–6.
166. 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(1):143–52.
167. 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(1):64–101.

Chapter 42

Neuraxial Analgesia in Obstetrics

Stephen Halpern, MD, MSc, FRCPC
Department of Anesthesia, Sunnybrook Health Sciences Centre, 2075 Bayview Ave Room M5-318, Toronto, ON M4N 3M5, Canada
e-mail: Stephen.halpern@sunnybrook.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_42,

Introduction

Since earliest recorded history, childbirth has been recognized as one of the most painful human experiences. Various pharmacologic and non-pharmacologic analgesic remedies have been described in ancient writings [1]. In an era when a "successful birth" was defined as survival of both mother and baby, early practitioners recognized that systemic drugs given to the parturient could depress the newborn. In 1900, the first spinal anesthetic was given for labor. This was followed in 1902 by spinal anesthesia for Cesarean section. In the early twentieth century, spinal anesthesia resulted in a maternal mortality rate of almost 0.1 % and led to severe side effects such as post-dural puncture headache, nausea, and vomiting. As a result, the technique was almost abandoned because of the dangers involved [2].

In the 1940s and 1950s, safety improved as skilled personnel became available, better equipment was developed, and an understanding of the maternal physiology was acquired. Over the intervening 60 years, neuraxial analgesia has become the most popular form of labor analgesia. By 2008, 61 % of American women who had singleton vaginal deliveries received neuraxial analgesia [3]. In this chapter, we will discuss the physiologic changes in pregnancy that directly impact conduct of neuraxial analgesia for the parturient. We will then discuss the mechanism of the pain of parturition. Finally, we will discuss the conduct of analgesia, including the goals of therapy, pharmacology, and side effects.

Anatomy of the Lumbar Epidural Space

A description of the normal anatomy of the lumbar spine can be found in Chap. 40. Pregnancy changes the anatomy of lumbar vertebral column and the contents of the epidural space. As pregnancy progresses, there is an increase in the lumbar lordosis. This is most marked in parturients with multiple gestation or polyhydramnios. The bulk of the abdominal contents may change maternal posture, narrowing the lumbar interspaces and making needle introduction difficult. In most parturients, this can be overcome with careful positioning.

Relaxin is a polypeptide, produced in the corpus luteum and the placenta. It has a positive role in remodeling collagen in the cervix (allowing for labor to take place) and ligaments (increasing the space in the pelvis) [4]. It may also soften the ligaments of the lumbar spine leading to back pain. When introducing an epidural needle, the ligamentum flavum may seem softer in the parturient than in the nonpregnant patient and may put her at risk for accidental dural puncture.

While fat takes up the majority of the epidural space in the lumbar region, other elements such as blood vessels and nerves are also present [5]. Increased blood volume in the anterior and lateral extradural venous plexus reduces the volume of other elements in the epidural space. This may account for increased spread of local anesthetic compared to nonpregnant patients [6]. In addition, cerebrospinal fluid volume is reduced, accentuating the spread of local anesthetic in the subarachnoid space [7].

Landmark identification may be difficult in the parturient. Midline structures may be obscured by edema and an increase in adipose tissue. Because of rotation of the pelvis, the line joining the iliac crests (Tuffier's line) is more cephalic leading to misinterpretation of level. This is particularly true of patients with a high body mass index [8]. It may be useful to use ultrasound to identify the interspace in obese patients, particularly when subarachnoid techniques are used [9]. For example, combined spinal–epidural techniques may place the spinal cord at risk of damage if the needle placement is above L2 [10].

Local Anesthetic Pharmacology

As described above, neuraxial local anesthetics are more potent in pregnant than in nonpregnant patients. In 1962, Bromage showed a reduction in local anesthetic requirements for epidural anesthesia (Fig. 42.1) [11]. Recently, Camorcia et al. compared the amount of intrathecal local anesthetic required to obtain lower limb motor block in male, female, and pregnant female patients undergoing surgical procedures [12]. Compared to male patients, pregnancy reduced the amount of local anesthetic by almost 50 %. Female patients required somewhat less local anesthetic, but this difference between sexes was not clinically significant.

Several factors may cause pregnant patients to be more sensitive to local anesthetics. As mentioned above, reduction in epidural and CSF volume may be factors. Other factors such as hormonal milieu may act to make individual neural elements more sensitive to local anesthetics [13].

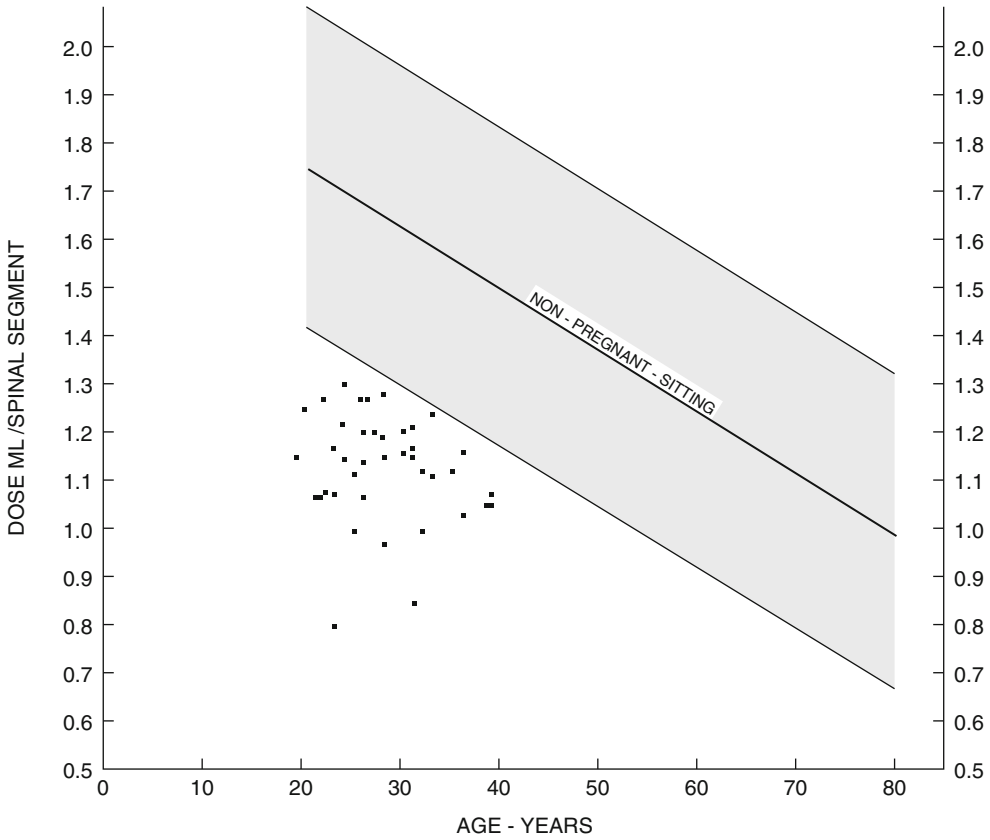


Fig. 42.1 Local anesthetic requirements for pregnant vs. nonpregnant patients (With permission from Oxford University Press [11])

Pain Pathways

Labor pain has both visceral and somatic components that evolve as labor progresses. During the first stage of labor, the visceral component dominates. Uterine contractions result in myometrial ischemia and the release of nociceptive mediators such as bradykinin, potassium, and serotonin. In addition, mechanoreceptors are stretched as the cervix dilates. Pain impulses are transmitted via the sympathetic pathways in the paracervical region, through the pelvis to the hypogastric plexus, entering the lumbar sympathetic chain that enters the dorsal horn of the spinal cord at the level of T10 to L1. During the second stage, a somatic component caused by a stretch of the perineum is added. These enter the spinal cord at the level of S1–S4 (Fig. 42.2).

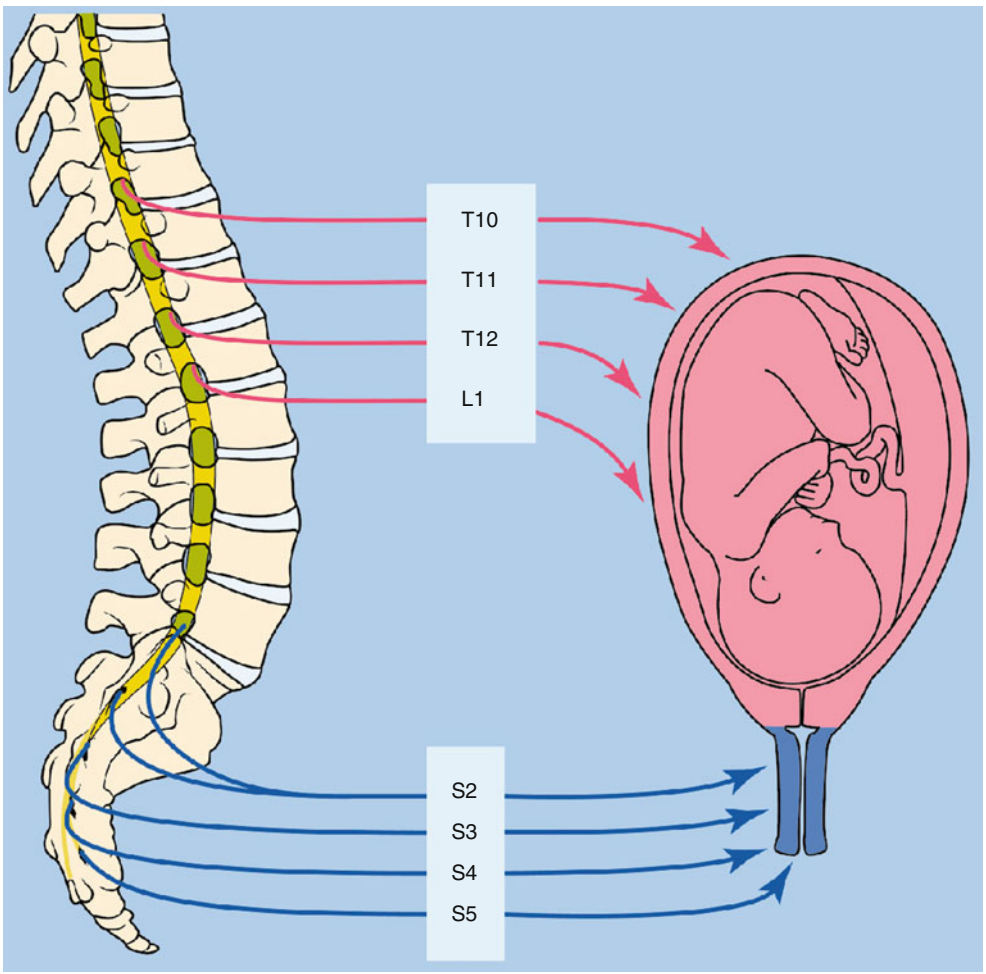


Fig. 42.2 Pain pathways for parturition (With permission from Danilo Jankovic)

Goals of Therapy

Childbirth is a multidimensional experience, and when considering treatment, one must strike a balance between pain relief and other aspects, such as physical, emotional, psychological, sociological, and other needs. For example, some women may consider analgesia unsatisfactory if it results in reduced ability to participate in the second stage of labor. A list of the characteristics of an ideal labor analgesic can be found in Table 42.1. Neuraxial analgesia can come very close to this ideal when properly conducted.

Table 42.1 Characteristics of an ideal labor analgesic

1. Effective pain relief
2. Maternal safety
3. Fetal and neonatal safety
a. Minimal effect on the fetal physiology
b. Minimal effect on the newborn
4. No adverse effects on the progress of labor including
a. Duration of the first or second stage of labor
b. Incidence of operative vaginal delivery
c. Incidence of Cesarean section
5. Few maternal side effects

Effective Pain Relief

Neuraxial analgesia is more effective than other methods. A recent meta-analysis of 17 randomized controlled trials comprised of 6,019 patients reported that 1 % of women required additional means of analgesia when neuraxial analgesia was used compared to 22 % for other methods [14]. Further, in an earlier meta-analysis of seven randomized trials comprised of 2,000 patients reported significantly reduced visual analog pain scores in patients who received neuraxial analgesia compared to those who received parenteral opioids. In the first stage, the difference was 40 mm (on a 100-mm scale), and in the second stage, it was 30 mm. Both were highly statistically and clinically significant [15].

Maternal Safety

When properly conducted, neuraxial analgesia has an excellent maternal safety record. The incidence of severe neurological complications is lower in parturients that in nonpregnant patients receiving neuraxial anesthesia in the perioperative setting [16]. When placing a neuraxial block, the anesthesiologist must be aware of the lumbar interspace and ensure that it is below the termination of the spinal cord. Other precautions, such as meticulous sterile technique, a system to ensure the correct drug is injected, etc., should be observed in the same fashion as in the operating room. There are a number of special precautions that should be taken to minimize the possibility of maternal injury. These include:

1. Avoid hypotension. This can occur because of pressure from the uterus on the inferior vena cava causing a reduction in preload. Hypotension may result in maternal lightheadedness or fainting and can be avoided by displacing the uterus (usually to the left side) off the vena cava. Caval compression becomes clinically significant when the fundus of the uterus can be palpated at the level of the umbilicus, usually after the 20th week of gestation. A vasopressor such as ephedrine or phenylephrine should be immediately available to treat hypotension. A large bolus of fluid prehydration is likely unnecessary before initiation of low-dose neuraxial analgesia [17, 18].
2. Post-dural puncture headache. This can occur in up to 1.6 % of patients who receive epidural analgesia. The incidence of accidental dural puncture with a large-bore epidural needle may be reduced by ensuring a controlled entry into the epidural space and advancing the needle when the patient is not having a contraction. A recent systematic review of randomized controlled trials did not find any maneuvers that were effective in reducing accidental dural puncture [19]. When deliberately puncturing the dura with a spinal needle during a combined spinal–epidural technique (CSE), dural puncture headache can be minimized by using the smallest gauge needle possible and a non-cutting needle [20].
3. Avoid accidental intravenous or intrathecal injection of local anesthetic. Fortunately, the doses of local anesthetic used for neuraxial analgesia for labor are very low. Some clinicians prefer to perform a "test dose" before the initial loading dose. This may include a combination of epinephrine that may be detected as maternal tachycardia when injected intravenously and a small dose of lidocaine that would immediately cause profound analgesia and motor block if injected intrathecally. Unfortunately, this combination does not achieve its goals. The baseline heart rate during labor is variable, and there may be confusion between the tachycardia caused by the test dose and the tachycardia caused by contraction pain. The lidocaine component may be effective in demonstrating an intrathecal injection, but it also causes unnecessary motor block when injected into the epidural space [21]. It may be preferable to fractionate the loading dose so that each fraction becomes a "test dose."

Fetal and Neonatal Safety

While providing analgesia, it is important to cause as little disturbance to the fetal physiology as possible. In addition, any drug given to the mother will be transferred to the maternal circulation and ultimately to the fetal circulation. These drugs may have a direct or indirect effect on the fetus and newborn. Drugs that remain in the maternal circulation will be present in breast milk, providing additional exposure to the newborn.

Local anesthetics, in the doses given for labor analgesia, do not cause any important direct physiologic or pharmacologic effects on the fetus [22]. Indirect effects may be more important and may be beneficial or harmful. Neuraxial analgesia that results in hypotension causes abnormal fetal heart rate patterns that are reversed when hypotension is treated. Severe labor pain increases maternal ventilation and hypocarbia resulting in acute respiratory alkalosis. This may result in a shift in the oxygen dissociation curve and reduced oxygen delivery to the fetus. Severe pain may also cause an increase in circulating catecholamines that may result in discoordinated labor and reduced uteroplacental perfusion [23]. Pain relief reverses these changes. However, if pain relief is too rapid, some authors suggest that uterine tetany may result in an acute, short-lived increase in uterine tone, restricting blood flow. Thus, fetal bradycardia is observed more often when a CSE is given for labor analgesia compared to epidural alone. It should be noted that the effect is transient and does not increase the incidence of Cesarean section for fetal distress or affect neonatal outcome [23].

Opioids, such as fentanyl and sufentanil, are often given combined with local anesthetics either in the epidural or subarachnoid space. These have few direct effects on the fetus because the dose is low. If there is maternal respiratory depression, this may indirectly lead to fetal acidosis and

hypoxia. Whether or not opioids, when given in the epidural space, can cause breastfeeding problems is controversial. Unfortunately, measurement of breastfeeding success is very difficult because there are no standard measures and there are multiple factors that may impact on the outcome. These include maternal health, other pharmacologic interventions, surgical interventions, sociologic considerations, and hospital policies [24]. The effect of neuraxial analgesia on breastfeeding is discussed in detail below.

Progress of Labor

Whether or not neuraxial analgesia causes maternal harm by impeding progress of labor has been controversial. Uncontrolled cohort studies consistently show a strong association between epidural analgesia and an increased incidence of Cesarean section, even when known confounding demographics are considered [25]. Women who have a prolonged and more painful and latent phase of labor are more likely to require obstetric intervention compared to those that do not [26], likely because of an increased incidence of dystocia in this group [27]. Therefore, increased pain may be a marker for poor obstetric outcome, and these patients are more likely to request epidural analgesia.

Over the last 20 years, there have been numerous randomized controlled trials comparing neuraxial to non-neuraxial analgesia for labor. Patients in the study group received either epidural or combined spinal–epidural analgesia. Patients in the control group received parenteral opioids or non-pharmacologic interventions. A recent meta-analysis comprised of 38 studies, and almost 10,000 patients concluded that neuraxial analgesia did not increase the risk of Cesarean section (relative risk = 1.10, 0.97–1.25) but may be associated with an increased incidence of instrumental vaginal delivery (relative risk = 1.42, 1.28–1.57). They also found that the duration of the second stage of labor was prolonged by about 13 min.

It is not clear whether or not mode of analgesia was the direct cause of the increased incidence of instrumented vaginal delivery. In particular, the presence of neuraxial blockade may induce behavioral changes in the obstetrical team. For example, epidural analgesia facilitates forceps delivery. At least one author described an excess of instrumented vaginal delivery in their parturients with epidurals for the purpose of resident training [28]. As discussed in detail below, the rate of instrumental vaginal delivery can be minimized by using low concentrations of local anesthetic to maintain analgesia [29].

Maternal Side Effects

The main focus of research into neuraxial analgesia for labor concentrates on reduction of maternal side effects. Effective maneuvers for prevention and treatment will be discussed below.

Pharmacology

The main drugs used in the epidural and subarachnoid space for labor include local anesthetics, opioids, and epinephrine. Clinical trials using other adjuvants such as clonidine, magnesium, and neostigmine have been reported, but at this time, none of these are used outside of the research setting and will not be considered further.

Local Anesthetics

Long-acting, amide local anesthetics are most commonly used for labor. Bupivacaine and ropivacaine are the only anesthetic agents available in North America. Levobupivacaine is available in most other parts of the world.

Lidocaine was the first local anesthetic used for labor analgesia, but its use was limited because of the high incidence of motor block and tachyphylaxis as labor progresses. Bupivacaine, first manufactured in 1957, was superior and became the most common local anesthetic used for labor epidural analgesia. In 1979, Albright reported a case series of six patients who suffered cardiac arrest because of accidental intravenous injection of a large dose of bupivacaine or etidocaine, calling the safety of these long-acting local anesthetics into question [30]. Ropivacaine and levobupivacaine were developed in an attempt to increase safety. It should be noted that cardiac toxicity has not been reported with any local anesthetics in the low doses currently recommended for initiation and maintenance of labor analgesia.

There have been numerous studies comparing ropivacaine to bupivacaine for labor in terms of efficacy, side effects, and labor outcome. It should be noted that ropivacaine is about 60–75 % as potent as bupivacaine when given as a bolus for initiation of labor [31]. When potency is considered, there is very little difference between the two drugs. In particular, both provide excellent analgesia and do not interfere with the progress of labor. In patients who received epidural analgesia for more than 6 h, there is a statistically significant increase in the incidence of motor block in the lower extremities [32]. However, the effect is small. At this time, ropivacaine tends to be somewhat more expensive than bupivacaine and economic factors, rather than clinical factors may decide which drug to use.

There is less information about levobupivacaine than either bupivacaine or ropivacaine. The potency of levobupivacaine seems to be about the same as bupivacaine. Block characteristics including the incidence of motor block and analgesic efficacy seem to be similar to bupivacaine [33].

Opioids

Lipid soluble opioids such as fentanyl and sufentanil bind to receptors in the spinal cord and work at that level, rather than

through systemic mechanisms [34, 35]. These are often combined with local anesthetics for both initiation and maintenance of labor analgesia. While other opioids have been studied, none are commonly used in clinical practice and will therefore not be considered further (see reference [36] for a full review).

When fentanyl is used for initiation of analgesia with 20 ml of bupivacaine, the median local anesthetic concentration (MLAC) for 50 % effectiveness was reduced from 0.064 to 0.034 %, demonstrating significant local anesthetic-sparing effect [34]. When used for maintenance [35], a similar local anesthetic-sparing effect was demonstrated. When used for initiation of analgesia, the addition of sufentanil increases the speed of onset (10.3 vs. 8.7 min) [37]. Clinically, the lowest, clinically effective concentration of local anesthetic should be used to reduce the intensity of motor block and possibly reduce the incidence of operative vaginal delivery [29].

Of interest, epidural fentanyl alone will provide moderate analgesia early in labor, but it is inadequate for the late first stage and second stage of labor [38]. Large doses of fentanyl cause severe pruritus, nausea, and vomiting. Significant systemic uptake also causes maternal sedation and possibly neonatal depression. For these reasons, lipid soluble opioids should be used with local anesthetic to take advantage of the synergy between the two classes of drug.

In summary, numerous studies have shown the benefit of combining low concentrations of long-acting local anesthetics such as bupivacaine or ropivacaine with lipid soluble opioids. Low doses of opioids should be used to reduce the incidence and severity of common side effects such as pruritus, nausea, and sedation. Low doses of opioids do not appear to affect neonatal outcomes.

Epinephrine

Epinephrine is sometimes used as an intravenous test for accidental intravenous injection through the epidural catheter. For this purpose, 15 mcg is often combined with 45–60 mg of lidocaine. The test is based on detecting maternal tachycardia. Unfortunately, maternal heart rate is extremely variable during labor and detection of tachycardia is often "false positive." This would lead to removal and reinsertion of the epidural catheter unnecessarily. While this test has been recommended by some investigators, most obstetric anesthesiologists have abandoned this practice [39].

Some clinicians add a small amount of epinephrine to local anesthetics to prolong duration of action and to reduce local anesthetic absorption. These advantages are not relevant for labor analgesia. In most cases, initiation of labor analgesia is immediately followed by an infusion eliminating the need for prolonged duration analgesia. Typically, a very low cumulative dose of local anesthetic is used, even in prolonged labor so that local anesthetic absorption is not an issue. Epinephrine may have some harmful effects such as transiently increasing maternal heart rate and reducing uterine activity. Finally, there may be an increased incidence of lower limb motor block [40].

Initiation of Analgesia

Initiation of analgesia can be accomplished either by using a standard epidural technique or a combined spinal–epidural (CSE) technique (see below).

Epidural analgesia is usually initiated with a low concentration of local anesthetic with or without fentanyl or sufentanil. Typically, 15–20 ml of 0.080–0.125 % bupivacaine (or the equivalent concentration of ropivacaine) can be given with or without up to 25 mcg of fentanyl in divided doses. Low concentrations of local anesthetic result in a reduced incidence of operative vaginal delivery compared to 0.25 % bupivacaine [41].

Initiation of labor analgesia using a CSE technique has become a popular method. This is usually done using a needle-through-needle technique. The epidural space is found with an epidural needle using a loss-of-resistance technique. Then, a long spinal needle is advanced through the epidural needle into the cerebrospinal fluid (CSF). The spinal needle is removed and an epidural catheter is placed. No additional medication is required at this time, but a syringe should be attached to the epidural catheter to aspirate for CSF. This is done in order to ensure the epidural catheter has not accidentally been passed into the intrathecal space. The epidural catheter is secured and maintenance of analgesia can begin.

Proponents of the technique note the very rapid onset of analgesia without the attendant motor block. This technique takes advantage of the synergistic analgesia obtained using a low dose of intrathecal local anesthetic with a lipophilic opioid such as fentanyl [42]. Some clinicians are concerned that rapid pain relief may lead to an increase in uterine tone and result in an excess incidence of fetal bradycardia.

A recent meta-analysis comprised of 27 randomized controlled trials and 3,274 women compared the effectiveness and side effects of epidural analgesia and CSE for labor [43]. CSEs had a faster onset of analgesia (5.4 min, 95 % confidence interval 3.6–7.2). One small study noted that all patients ($n=50$) reported good analgesia at 10 min after a CSE while only 50 % had good analgesia after a low dose epidural. However, the incidence of successful analgesia was similar in both groups at later time points [44]. Low-dose epidural analgesia did not impair mobility, and there was no difference between groups. The incidence of pruritus was significantly higher in the CSE group. Whether labor analgesia is initiated

with a CSE or epidural, maternal satisfaction rates are high and there is no difference between groups. There were no differences between groups for any other maternal side effects such as dural puncture headache, nausea, vomiting, or hypotension. Importantly, there is no difference in the incidence of spontaneous vaginal delivery or Cesarean section between the two groups, nor was there any difference in any measured neonatal outcomes. There was a statistically significant increase in the rate of operative vaginal deliveries, but the difference between groups was small, except when high concentrations of local anesthetic were used to initiate analgesia [41]. There was no difference between groups when only low-dose epidurals were compared to CSEs. The authors of the meta-analysis noted that there were insufficient data to comment on the maternal safety of CSEs since the incidence of such complications a meningitis and nerve damage are very low [43].

Fetal bradycardia has been observed in relation to CSEs. An early systematic review [45] and a large randomized controlled trial [23] concluded that the incidence of fetal bradycardia may be increased after CSE compared to epidural. A recent randomized controlled trial compared the incidence of abnormal fetal heart rate patterns in nulliparous patients who received epidural analgesia or CSE. The investigators noted that there was an increased incidence of abnormal fetal heart rate patterns in both groups after the initiation of analgesia. There was no difference between groups in the rate of Cesarean section or neonatal outcomes [46].

Maintenance of Analgesia

The use of a continuous epidural catheter allows provision of analgesia for the duration of labor. Low concentrations of long-acting local anesthetics such as bupivacaine or ropivacaine combined with lipophilic opioid provide analgesia throughout labor. Intermittent boluses of local anesthetic by clinicians can be an effective strategy for maintenance of analgesia, but it is time-consuming. A continuous infusion of local anesthetic provides a more constant level of analgesia throughout labor. More recently, patient-controlled epidural analgesia (PCEA), in addition to a low-volume continuous infusion, has become the preferred technique. A meta-analysis of nine studies comprised of 640 patients compared to continuous epidural infusion alone to PCEA. Patients in the PCEA group had a reduction in the number of interventions required by clinicians, a reduction in the amount of drug given, and a reduction in the incidence of motor block of the lower extremities. There was no difference between groups in other analgesic outcomes, maternal satisfaction, progress of labor, or neonatal outcome [47].

When using PCEA, the clinician must determine the concentration of local anesthetic, the size of the allowed bolus dose, the lockout interval between doses, and the presence of a background continuous infusion. While there is no single "best" setting, there are some principles that can guide dosing.

Dilute solutions of local anesthetic should be used. Concentrations of bupivacaine between 0.0625 and 0.125 % are commonly used. Low concentrations require the addition of fentanyl or sufentanil to be effective. Higher concentrations of local anesthetic do not increase the analgesic efficacy but do lead to an increase in motor block of the lower extremities [48]. In addition, higher concentrations make operative vaginal delivery and prolong second stage of labor more likely [29].

There is a wide range of patient-initiated bolus doses that is safe and effective. It is important that the bolus should not be too large in case of the unlikely event that the epidural catheter migrates intravenously or intrathecally. The usual dose range, using the concentrations outlined above, is between 4 and 10 ml, although up to 20 ml has been reported [48].

The interval between allowed bolus doses should be short enough to allow the patient a sufficient dose of drug, but should not allow the patient a second dose before the first has a chance to work. The usual setting is 10 min, but up to 25 min has been reported in combination with larger bolus doses [48].

Most clinicians use a continuous infusion as a background with patient-controlled boluses because the analgesia is better [49]. The rate can vary between 2 and 14 ml per hour in the concentrations discussed above. A background continuous infusion reduces the need for clinician intervention. While the cumulative dose of drug is higher when continuous infusions are used, they do not approach toxicity, nor are they associated with an increased in lower limb motor block [48]. The infusion can be maintained at a very low rate early in labor and increased as labor progresses. Some clinicians prefer to reduce the continuous infusion rate during the second stage to allow the patient to push effectively. This strategy has not been studied in detail.

There is some research to suggest that mandatory bolus dosing, instead of continuous infusion, in combination with PCEA may be superior to continuous infusion. This is a relatively new technique that has recently become available for clinical use. The purported advantages include a reduction in motor block and possibly a reduction in the need for instrumental vaginal delivery. A recent systematic review and meta-analysis was hampered by the small amount of data available. There was a trend toward the need for less local anesthetic and possibly a reduction in the incidence of instrumental vaginal delivery. The authors caution that more experience is necessary before recommending this technique over continuous infusion with PCEA [50].

Side Effects and Treatment

While neuraxial analgesia is usually safe, complications can occur. Some of these are caused directly by the drugs or technique. Others are often attributed to the technique but are coincidental. The cause of some complications is unclear and may or may not be caused by neuraxial analgesia.

Direct Complications

There are major and minor complications. The major complications arising from either epidural or CSE analgesia are shown in Table 42.2. Fortunately, these rates are much lower than those in the non-obstetric population. This may be due to differences in anatomy and the relatively short duration of the block.

Table 42.2 Major complications of neuraxial analgesia

Complication or side effect	Epidural	CSE	References
Nerve damage caused by needle trauma	0.6 per 100,000	3.9 per 100,000	[16]
Meningitis	0–3.5 per 100,000	0–3.5 per 100,000	[16]
Epidural hematoma	1:168,000	–	[51]
Dural puncture headache	0.21–1.6 %	0.21–1.6 %	[52, 53]

Minor Complications

Inadequate Analgesia

The reported incidence of inadequate analgesia varies widely among studies and depends on the study design, method of follow-up, and the definition used by the investigators. In a retrospective study in a teaching hospital, Pan et al. reported a 12 % incidence of failure to achieve analgesia. The incidence was significantly lower in parturients who received a CSE (10 %) compared to those that received an epidural (14 %). In this study, an additional 5.6 % of epidural catheters were replaced at some time during labor because they stopped functioning. Heeson et al. recently published a meta-analysis of randomized controlled trials comprised of 10 studies and 1,450 patients of mixed parity. This review did not confirm that there was a significant difference in the rate of failure or need for catheter replacement [54] between the two techniques. There was, however, an increased incidence of unilateral block in the epidural group. The authors concluded that there is insufficient evidence, at this time to recommend CSE technique based on reliability.

Pruritus

Pruritus is one of the most common side effects of neuraxial opioids. The frequency is dose dependent and may be more common with intrathecal opioids compared to epidural administration [55]. The exact cause is unknown, but it is unlikely to be related to histamine release. Antihistamines, when used for treatment, are usually ineffective.

When used for prevention of pruritus, the serotonin receptor antagonist ondansetron did not reduce the incidence but did reduce severity as measured by need for additional treatment. In addition, the reported incidence of severe pruritus was reduced. Ondansetron is an effective treatment of established pruritus [56].

There is evidence to suggest that central mu receptors play an important role in causing neuraxial opioid-induced pruritus. Oral naltrexone has been used to prevent pruritus in parturients who received epidural morphine after Cesarean section. Given orally, 6 mg of naltrexone is effective and does not reduce the analgesic duration [57]. Intravenous naloxone is effective in treating pruritus secondary to neuraxial opioid administration. Nalbuphine, in doses of 2.5–5 mg, is more effective than naloxone because of its longer duration of action [58].

Post-dural Puncture Headache

Accidental dural puncture is an uncommon complication of epidural analgesia, occurring in up to 1.6 % of the population. Recognized puncture in the obstetric population results in severe postural headache in over 50 % of affected patients [59]. While dural puncture is usually recognized immediately because of the outflow of cerebrospinal fluid, there is a small number of patients that have unrecognized dural punctures and develop typical headaches. The pain of these headaches usually begins 6–24 h after the puncture. The pain is typically described as severe and affecting the occiput and/or neck. It is worse in the upright position and relieved by lying down. The headache can severely limit a new mother's ability to care for her newborn and should therefore be treated promptly after diagnosis. An epidural blood patch is the most effective treatment for this complication [60].

Indirect Complications

Neuraxial analgesia can indirectly have a negative impact on the fetus. If the mother becomes hypoxic because of respiratory depression secondary to neuraxial opioids, the fetus will also become hypoxemic. Similarly, hypotension will reduce uterine blood flow and adversely affect the fetus. Finally, as noted above, fetal bradycardia may occur. In some instances, this is due to uterine hypertonus, secondary to sudden reduction in circulating maternal catecholamines associated with rapid analgesia [61].

Associated Complications

A rise in maternal temperature has been associated with neuraxial analgesia, but the cause is unclear. Several retrospective studies have noted the association, but these do not control for important variables such as duration of labor and maternal temperature before initiation of analgesia. Importantly, neuraxial analgesia may lead to an increase in internal examinations by the obstetrician since these no longer cause pain. Examinations may cause chorioamnionitis and therefore be associated with fever. Randomized controlled trials that compare early to late neuraxial analgesia do not show an increase in maternal fever in patients with prolonged exposure [62]. Finally, the mechanism for increased body temperature is unknown since in most other circumstances where epidural analgesia is used, hypothermia results.

Some clinicians believe that epidural analgesia may interfere with breastfeeding. In particular, there is a fear that drug transfer will result in a change in newborn neurobehavior, a change in fluid metabolism resulting in breast engorgement or increased fetal weight, or an increase in obstetric interventions.

Breastfeeding is extremely difficult to study. This is because the outcomes have not been standardized and success is multifactorial. There have been several scales developed to measure breastfeeding, but unfortunately, the inter-rater reliability is poor and normal values have not been defined. There is some concern that large doses of fentanyl, if given in the epidural space, may result in a reduction in breastfeeding success. Beilin et al. performed a randomized controlled trial in 180 multiparous patients who had previous experience breastfeeding into three different doses of

epidural fentanyl in a continuous epidural analgesia for labor. The incidence of breastfeeding difficulties, as reported by both the lactation consultant and the mother, was the same in all groups in the first 24 h after birth. However, there was a reduced incidence of breastfeeding 6 weeks postpartum in patients who received more than 150 mcg of fentanyl during labor [63]. While the authors postulated that high dose fentanyl may have been the cause, other data does not support this conclusion. A secondary analysis of the COMET study noted no difference in breastfeeding regardless of the fentanyl dose [64]. In addition, a more recent cohort did not confirm the reduction in breastfeeding [65]. While it is important to use as little medication to prevent side effects (e.g., pruritus), it is unlikely that the dose of opioid, given as neuraxial analgesia for labor, causes reduced breastfeeding success.

Summary

Regional analgesia is the most efficacious form of pain relief for labor. When performing neuraxial blocks in this population, the practitioner must be aware of the physiologic changes of pregnancy and altered pharmacology. In particular, low concentrations of local anesthetic, combined with lipophilic opioids, are highly effective for most laboring women. Consideration must also be given to the direct and indirect effects of therapy on the fetus.

References

1. Mander R. Analgesia and anaesthesia in childbirth: obscurantism and obfuscation. J Adv Nurs. 1998;28:86–93.
2. Gogarten W, Van Aken H. A century of regional analgesia in obstetrics. Anesth Analg. 2000;91:773–5.
3. Osterman MJK, Martin JA. Epidural and spinal anesthesia use during labor: 27 state reporting area 2008. Natl Vital Stat Rep. 2011;59:1–16.
4. Weiss G. Relaxin. Annu Rev Physiol. 1984;46:43–52.
5. Hogan Q, Toth J. Anatomy of soft tissues of the spinal canal. Reg Anesth Pain Med. 1999;24:303–10.
6. Hirabayashi Y, Shimizu R, Fukuda H, Saitoh K, Igarashi T. Soft tissue anatomy within the vertebral canal in pregnant women. Br J Anaesth. 1996;77:153–6.
7. Onuki E, Higuchi H, Takagi S, Nishijima K, Fujita N, Matsuura T, et al. Gestation-related reduction in lumbar cerebrospinal fluid volume and dural sac surface area. Anesth Analg. 2010;110:148–53.
8. Kettani A, Tachinante R, Tazi A. Evaluation of the iliac crest as anatomic landmark for spinal anaesthesia in pregnant women. Ann Fr Anesth Reanim. 2006;25:501–4.
9. Halpern SH, Banerjee A, Stocche R, Glanc P. The use of ultrasound for lumbar spinous process identification: a pilot study. Can J Anaesth. 2010;57:817–22.
10. Reynolds F. Damage to the conus medullaris following spinal anaesthesia. Anaesthesia. 2001;56:238–47.
11. Bromage PR. Spread of analgesic solutions in the epidural space and their site of action: a statistical study. Br J Anaesth. 1962;34:161–78.
12. Camorcia M, Capogna G, Columb MO. Effect of sex and pregnancy on the potency of intrathecal bupivacaine: determination of ED50 for motor block with the up-down sequential allocation method. Eur J Anaesthesiol. 2011;28:240–4.
13. Flanagan HL, Datta S, Lambert DH, Gissen AJ, Covino BG. Effect of pregnancy on bupivacaine-induced conduction blockade in the isolated rabbit vagus nerve. Anesth Analg. 1987;66:123–6.
14. Anim-Somuah M, Smyth RM, Jones L. Epidural versus non-epidural or no analgesia in labour. Cochrane Database Syst Rev. 2011;12:000331.
15. Leighton BL, Halpern SH. The effects of epidural analgesia on labor, maternal, and neonatal outcomes: a systematic review. Am J Obstet Gynecol. 2002;186(5 Suppl):S69–77.
16. 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;102:179–90.
17. Kinsella SM, Pirlet M, Mills MS, Tuckey JP, Thomas TA. Randomized study of intravenous fluid preload before epidural analgesia during labour. Br J Anaesth. 2000;85:311–3.
18. Kubli M, Shennan AH, Seed PT, O'Sullivan G. A randomised controlled trial of fluid pre-loading before low dose epidural analgesia for labour. Int J Obstet Anesth. 2003;12:256–60.
19. Bradbury CL, Singh SI, Badder SR, Wakely LJ, Jones PM. Prevention of post-dural puncture headache in parturients: a systematic review and meta-analysis. Acta Anaesthesiol Scand. 2013;57:417–30.
20. Halpern S, Preston R. Postdural puncture headache and spinal needle design. Meta analyses. Anesthesiology. 1994;81:1376–83.
21. Breen TW, Giesinger CM, Halpern SH. Comparison of epidural lidocaine and fentanyl to intrathecal sufentanil for analgesia in early labour. Int J Obstet Anesth. 1999;8:226–30.
22. Reynolds F. Labour analgesia and the baby: good news is no news. Int J Obstet Anesth. 2011;20:38–50.
23. Gambling D, Berkowitz J, Farrell TR, Pue A, Shay D. A randomized controlled comparison of epidural analgesia and combined spinal-epidural analgesia in a private practice setting: pain scores during first and second stages of labor and at delivery. Anesth Analg. 2013;116:636–43.
24. Szabo AL. Intrapartum neuraxial analgesia and breastfeeding outcomes: limitations of current knowledge. Anesth Analg. 2013;116:399–405.
25. Lieberman E, Lang JM, Cohen A, D'Agostino Jr R, Datta S, Frigoletto Jr FD. Association of epidural analgesia with cesarean delivery in nulliparas. Obstet Gynecol. 1996;88:993–1000.
26. Wuitchik M, Bakal D, Lipshitz J. The clinical significance of pain and cognitive activity in latent labor. Obstet Gynecol. 1989;73:35–42.
27. Panni MK, Segal S. Local anesthetic requirements are greater in dystocia than in normal labor. Anesthesiology. 2003;98:957–63.
28. Bofill JA, Vincent RD, Ross EL, Martin RW, Norman PF, Werhan CF, et al. Nulliparous active labor, epidural analgesia, and cesarean delivery for dystocia. Am J Obstet Gynecol. 1997;177:1465–70.
29. Sultan P, Murphy C, Halpern S, Carvalho B. The effect of low concentrations *versus* high concentrations of local anesthetics for labour analgesia on obstetric and anesthetic outcomes: a meta-analysis. Can J Anaesth. 2013;80:840–53.
30. Albright GA. Cardiac arrest following regional anesthesia with etidocaine or bupivacaine. Anesthesiology. 1979;51:285–7.
31. Beilin Y, Halpern S. Focused review: ropivacaine versus bupivacaine for epidural labor analgesia. Anesth Analg. 2010;111:482–7.

32. Halpern SH, Breen TW, Campbell DC, Muir HA, Kronberg J, Nunn R, et al. A multicenter, randomized, controlled trial comparing bupivacaine with ropivacaine for labor analgesia. Anesthesiology. 2003;98:1431–5.
33. Halpern SH. Choice of local anesthetic for labor and delivery–bupivacaine, ropivacaine and levobupivacaine. In: Halpern SH, Douglas MJ, editors. Evidence-based obstetric anesthesia. Malden: Blackwell Publishing; 2005. p. 56–67.
34. Polley LS, Columb MO, Naughton NN, Wagner DS, Dorantes DM, van de Ven CJ. Effect of intravenous versus epidural fentanyl on the minimum local analgesic concentration of epidural bupivacaine in labor. Anesthesiology. 2000;93:122–8.
35. Ginosar Y, Columb MO, Cohen SE, Mirikatani E, Tingle MS, Ratner EF, et al. The site of action of epidural fentanyl infusions in the presence of local anesthetics: a minimum local analgesic concentration infusion study in nulliparous labor. Anesth Analg. 2003;97:1439–45.
36. Wong CA. Epidural and spinal analgesia/anesthesia for vaginal delivery. In: Chestnut DH, Wong CA, Tsen LC, Ngan Kee WD, Beilin Y, Mhyre JM, editors. Obstetric anesthesia: principles and practice. Philadelphia: Elsevier/Saunders; 2014. p. 457–517.
37. Van Steenberge A, Debroux HC, Noorduin H. Extradural bupivacaine with sufentanil for vaginal delivery. A double-blind trial. Br J Anaesth. 1987;59:1518–22.
38. Justins DM, Francis D, Houlton PG, Reynolds F. A controlled trial of extradural fentanyl in labour. Br J Anaesth. 1982;54:409–14.
39. Camorcia M. Testing the epidural catheter. Curr Opin Anaesthesiol. 2009;22:336–40.
40. Soetens FM, Soetens MA, Vercauteren MP. Levobupivacaine-sufentanil with or without epinephrine during epidural labor analgesia. Anesth Analg. 2006;103:182–6.
41. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK. Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomised controlled trial. Lancet. 2001;358:19–23.
42. Ngan Kee WD, Khaw KS, Ng FF, Ng KK, So R, Lee A. Synergistic interaction between fentanyl and bupivacaine given intrathecally for labor analgesia. Anesthesiology. 2014;120:1126–36.
43. Simmons SW, Taghizadeh N, Dennis AT, Hughes D, Cyna AM. Combined spinal-epidural versus epidural analgesia in labour. Cochrane Database Syst Rev. 2012;10:003401.
44. Zeidan AZ. Combined spinal-epidural compared with low dose epidural during ambulatory labour analgesia in nulliparous women. Egypt J Anaesthesia. 2004;20:273–81.
45. Mardirosoff C, Tramer MR. Intrathecal opioids in labor–do they increase the risk of fetal bradycardia? In: Halpern SH, Douglas MJ, editors. Evidence-based obstetric anesthesia. Malden: Blackwell Publishing; 2005. p. 68–76.
46. Patel NP, El-Wahab N, Fernando R, Wilson S, Robson SC, Columb MO, et al. Fetal effects of combined spinal-epidural vs epidural labour analgesia: a prospective, randomised double-blind study. Anaesthesia. 2014;69:458–67.
47. van der Vyver M, Halpern S, Joseph G. Patient-controlled epidural analgesia versus continuous infusion for labour analgesia: a meta-analysis. Br J Anaesth. 2002;89:459–65.
48. Halpern SH, Carvalho B. Patient-controlled epidural analgesia for labor. Anesth Analg. 2009;108:921–8.
49. American Society of Anesthesiologists Task Force on Obstetric, Anesthesia. Practice guidelines for obstetric anesthesia: an updated report by the American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Anesthesiology. 2007;106:843–63.
50. George RB, Allen TK, Habib AS. Intermittent epidural bolus compared with continuous epidural infusions for labor analgesia: a systematic review and meta-analysis. Anesth Analg. 2013;116:133–44.
51. Ruppen W, Derry S, McQuay HJ, Moore RA. Incidence of epidural haematoma and neurological injury in cardiovascular patients with epidural analgesia/anaesthesia: systematic review and meta-analysis. BMC Anesthesiol. 2006;12:6.
52. Norris MC, Fogel ST, Conway-Long C. Combined spinal-epidural versus epidural labor analgesia. Anesthesiology. 2001;95:913–20.
53. Van de Velde M, Schepers R, Berends N, Vandermeersch E, De Buck F. Ten years of experience with accidental dural puncture and post-dural puncture headache in a tertiary obstetric anaesthesia department. Int J Obstet Anesth. 2008;17:329–35.
54. Heesen M, Van de Velde M, Klohr S, Lehberger J, Rossaint R, Straube S. Meta-analysis of the success of block following combined spinal-epidural vs epidural analgesia during labour. Anaesthesia. 2014;69:64–71.
55. Norris MC, Grieco WM, Borkowski M, Leighton BL, Arkoosh VA, Huffnagle HJ, et al. Complications of labor analgesia: epidural versus combined spinal epidural techniques. Anesth Analg. 1994;79:529–37.
56. George RB, Allen TK, Habib AS. Serotonin receptor antagonists for the prevention and treatment of pruritus, nausea, and vomiting in women undergoing cesarean delivery with intrathecal morphine: a systematic review and meta-analysis. Anesth Analg. 2009;109:174–82.
57. Abboud TK, Afrasiabi A, Davidson J, Zhu J, Reyes A, Khoo N, et al. Prophylactic oral naltrexone with epidural morphine: effect on adverse reactions and ventilatory responses to carbon dioxide. Anesthesiology. 1990;72:233–7.
58. Ganesh A, Maxwell LG. Pathophysiology and management of opioid-induced pruritus. Drugs. 2007;67:2323–33.
59. Choi PT, Galinski SE, Takeuchi L, Lucas S, Tamayo C, Jadad AR. PDPH is a common complication of neuraxial blockade in parturients: a meta-analysis of obstetrical studies. Can J Anaesth. 2003;50:460–9.
60. Macarthur A. Postpartum headache. In: Chestnut DH, Wong CA, Tsen LC, Ngan Kee WD, Beilin Y, Mhyre JM, editors. Chestnut's obstetric anesthesia: principles and practice. Philadelphia: Elsevier/Saunders; 2014. p. 713–38.
61. Clarke VT, Smiley RM, Finster M. Uterine hyperactivity after intrathecal injection of fentanyl for analgesia during labor: a cause of fetal bradycardia? Anesthesiology. 1994;81:1083.
62. Arendt KW, Segal BS. The association between epidural labor analgesia and maternal fever. Clin Perinatol. 2013;40:385–98.
63. Beilin Y, Bodian CA, Weiser J, Hossain S, Arnold I, Feierman DE, et al. Effect of labor epidural analgesia with and without fentanyl on infant breast-feeding: a prospective, randomized, double-blind study. Anesthesiology. 2005;103:1211–7.
64. Wilson MJ, MacArthur C, Cooper GM, Bick D, Moore PA, Shennan A, et al. Epidural analgesia and breastfeeding: a randomised controlled trial of epidural techniques with and without fentanyl and a non epidural comparison group. Anaesthesia. 2010;65:145–53.
65. Wieczorek PM, Guest S, Balki M, Shah V, Carvalho JCA. Breastfeeding success rate after vaginal delivery can be high despite the use of epidural fentanyl: an observational cohort study. Int J Obstet Anesth. 2010;19:273–7.

Chapter 43

Combined Spinal and Epidural Anesthesia (CSE)

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_43, © Springer International Publishing Switzerland 2015

Introduction

This combined technique was introduced in neuraxial regional anesthesia in order to exploit as many advantages of both procedures as possible and to minimize their disadvantages.

In CSE, the reliability, fast onset of effect, high success rate, excellent muscle relaxation, and low toxicity of spinal anesthesia are combined with the advantages of epidural anesthesia: flexibility, good controllability, ability to prolong the anesthesia as required, and potential transition to postoperative pain treatment.

CSE allows better titration and a substantial reduction in the dose of local anesthetic, opioid, or combination of the two. The advantages of this technique can be used particularly effectively in obstetrics, with results showing a substantial reduction in maternal hypotension during birth.

Brief History

In 1937, the New York surgeon Soresi [1] reported that it was possible to inject procaine first epidurally and then intrathecally through the same needle.

In 1979, Curelaru [2] described the use of CSE in abdominal surgery, urology, and orthopedics. After placement of an epidural catheter, a subarachnoid injection was carried out one or two segments below the puncture site.

In 1981, Brownridge [3] reported the use of CSE for Cesarean section. He used two different segments for puncture.

A modification of this technique with one-segment puncture ("needle through needle") was used in 1982 by Coates [4] and Mumtaz et al. [5] in orthopedic surgery and in 1984 by Carrie [6] in obstetric surgery.

In 1986, Rawal [7–11] described the sequential (two-stage) CSE technique for Cesarean section.

Indications

Surgical Procedures

1. General surgery
2. Outpatient surgery [12]
3. Vascular surgery [13]
4. Orthopedics [4, 5, 15, 16]
5. Gynecology [7, 9, 10]
6. Obstetrics [6–10]
7. Urology [2]
8. Pediatric surgery [14]
9. Postoperative pain therapy

Contraindications

The contraindications are the same as those for spinal anesthesia (see Chap. 41 spinal and epidural anesthesia).

Procedure

Full prior information for the patient is mandatory.

Preparation and Materials

1. Check that the emergency equipment is complete and in working order (intubation kit, emergency drugs); anesthetic machine.
2. Set up an intravenous infusion and give a volume load (500–1,000 mL of a balanced electrolyte solution).
3. Careful monitoring: ECG, BP, pulse oximeter.
4. Maintain strict asepsis.

The use of purpose-designed kit is recommended.

This procedure must only be carried out by an experienced anesthetist.

Patient Positioning

The puncture is carried out below the L2 segment, with the patient either in the lateral decubitus position (preferable) or sitting.

Injection Technique

"Needle Through Needle"

After locating and marking the puncture site (L2/3 or L3/4), thorough skin prep is carried out, followed by local anesthesia and a skin incision using a stylet. Insertion is carried out in the midline using an 18-G epidural Tuohy needle, with the bevel directed cranially. Identification of the epidural space is carried out using the loss-of-resistance technique (Fig. 43.1).

After identification of the epidural space and injection of a test dose (Fig. 43.2), a thin 27-G pencil-point spinal needle is carefully advanced through the epidural needle in the direction of the subarachnoid space, until dural perforation is confirmed by a click (Fig. 43.3a).

The adapted form of the Tuohy needle tip, which has a central opening positioned in the needle axis ("back eye"), allows the needle to take a direct path, so that the spinal needle does not need to bend. The plastic coating of the spinal needle expands its outer diameter, so that it fits the epidural needle precisely, maintains its central position as it is advanced, and easily passes through the axial opening ("back eye") (Fig. 43.3b). After careful aspiration of CSF, subarachnoid injection of a local anesthetic, opioid, or a combination of the two is carried out (Fig. 43.4). As this is done, the hub of the spinal needle should be secured with the thumb and index finger of the left hand, which rests on the patient's back. This is the critical phase of the puncture procedure.

The spinal needle is then withdrawn, and the epidural catheter is introduced up to a maximum of 3–4 cm (Fig. 43.5a, b).

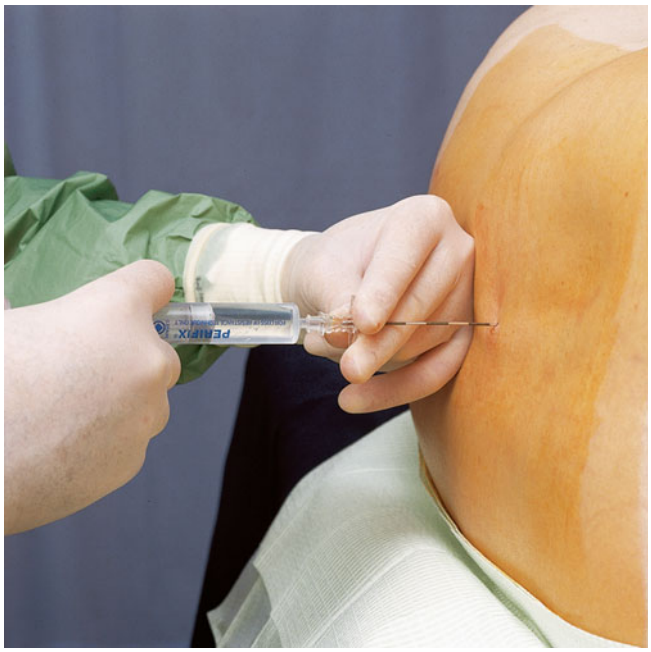


Fig. 43.1 Identifying the epidural space using the loss-of-resistance technique (With permission from Danilo Jankovic)

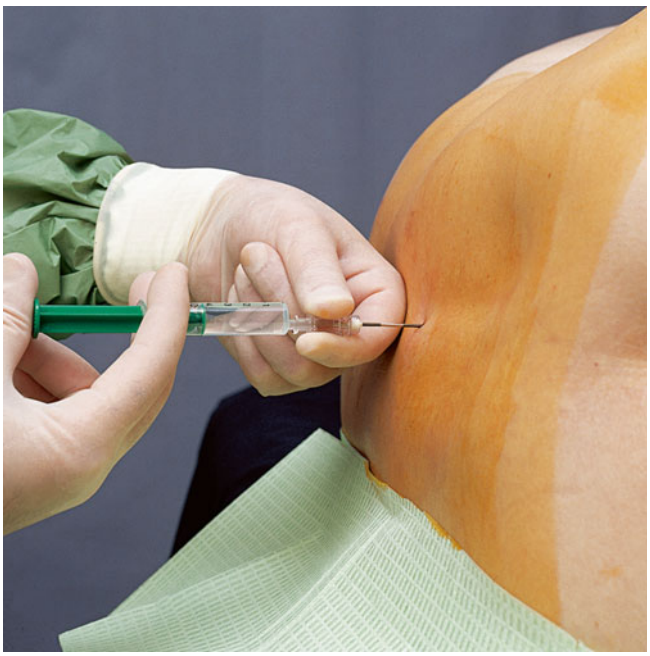


Fig. 43.2 Injecting a test dose (With permission from Danilo Jankovic)

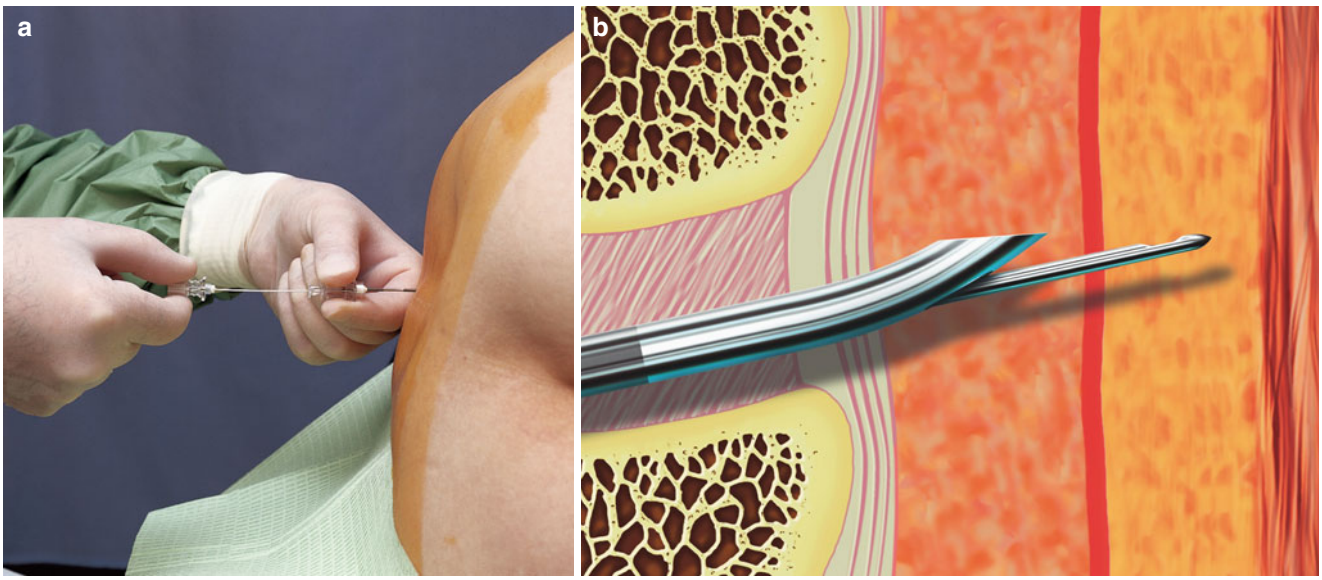


Fig. 43.3 (**a**) 27-G pencil-point spinal needle is introduced through the positioned epidural needle. (**b**) Identification of the subarachnoid space with the dural click (With permission from Danilo Jankovic)

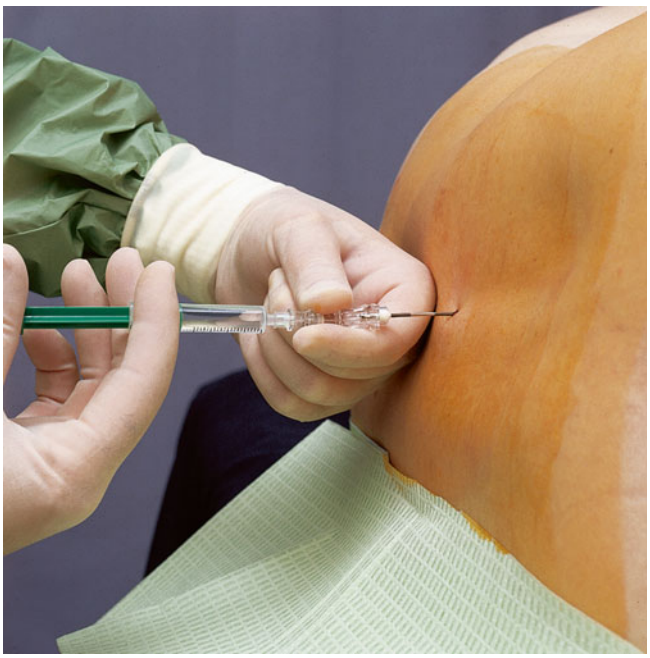


Fig. 43.4 Subarachnoid injection. The spinal needle is then withdrawn (With permission from Danilo Jankovic)

- Repeated aspiration.
- As low a dose as possible.
- Always use incremental injections (several test doses).
- Maintain verbal contact.
- Check the spread of anesthesia carefully.
- Careful monitoring.

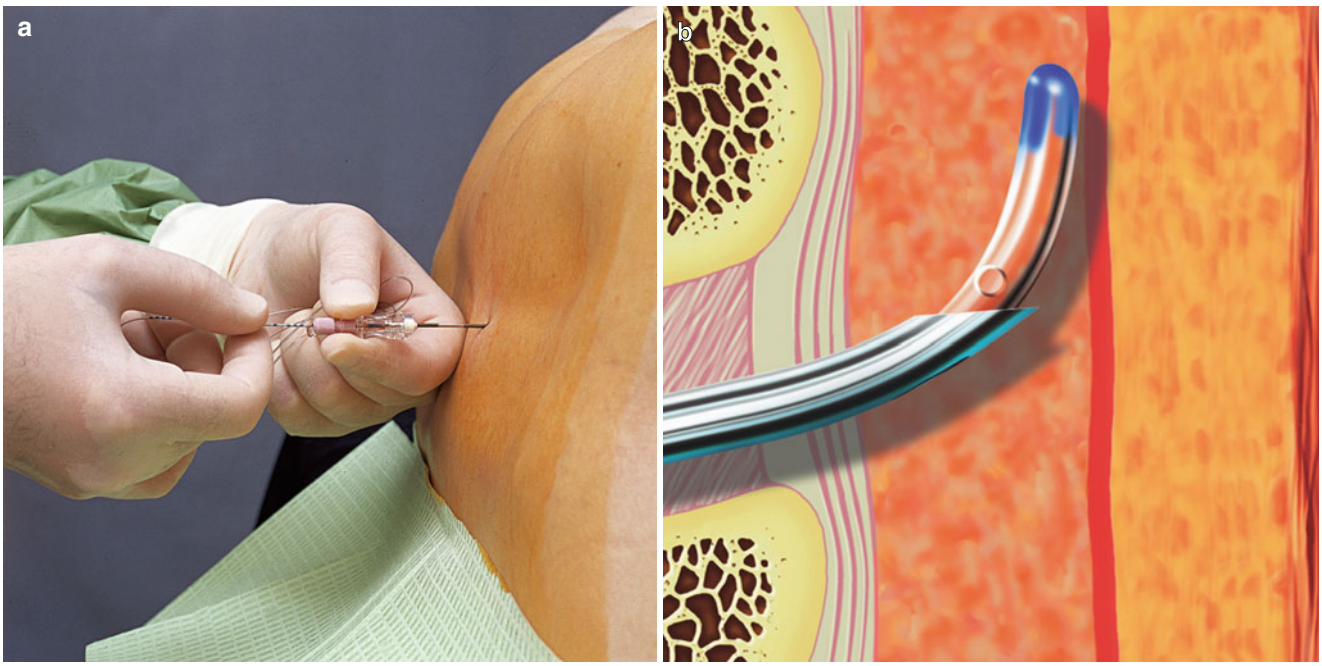


Fig. 43.5 (**a**) Introducing the epidural catheter. (**b**) The epidural catheter is advanced by a maximum of 3–4 cm cranially (With permission from Danilo Jankovic)

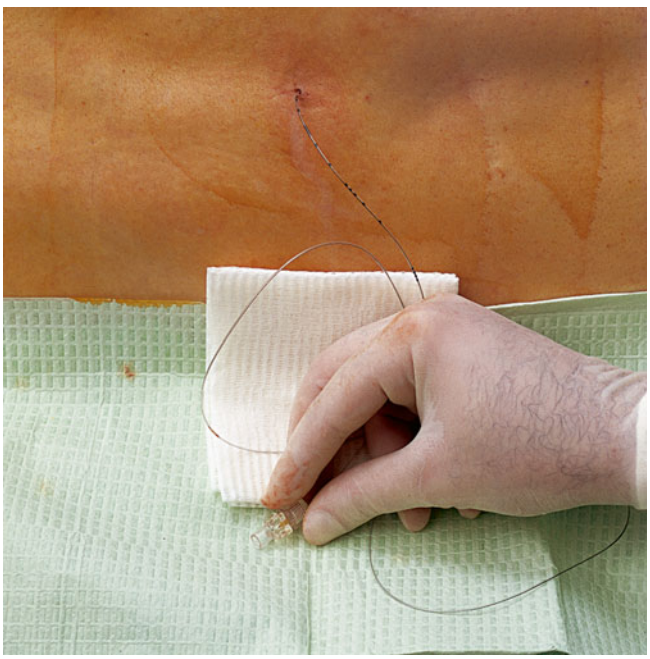


Fig. 43.6 The open end of the catheter is placed below the puncture site (With permission from Danilo Jankovic))

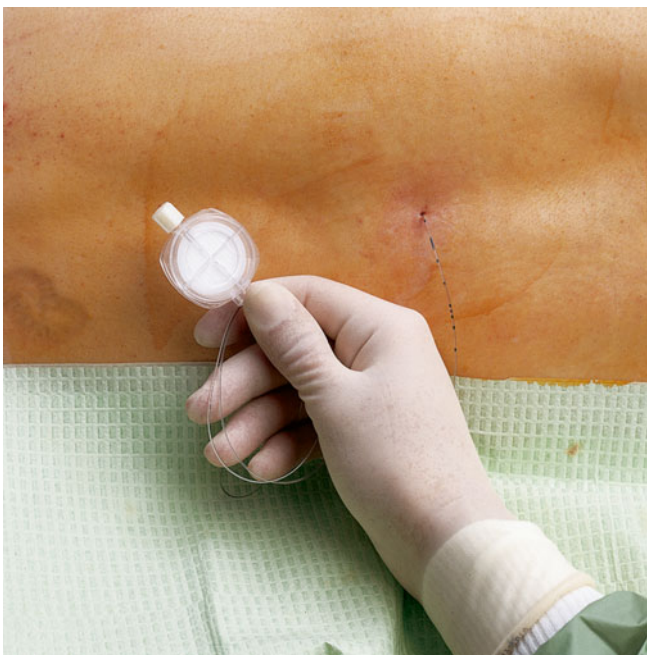


Fig. 43.7 The catheter is secured and a bacterial filter is attached (With permission from Danilo Jankovic)

After aspiration, the open end of the catheter is laid on a sterile surface below the puncture site and any escaping fluid (CSF or blood) is noted (Fig. 43.6).

To test the patency of the catheter, 1–2 mL saline is then injected. The catheter is secured and a bacterial filter is attached (Fig. 43.7).

Problem Situations

Specific problems during CSE occur in connection with the administration of a test dose to exclude subarachnoid positioning of the catheter.

As spinal anesthesia is being given, it is not possible to test for incorrect intrathecal positioning of the catheter, and incorrect positioning usually becomes evident through high or total spinal anesthesia.

This technique should therefore only be carried out by experienced anesthetists.

Local anesthetics must only be injected in small incremental amounts (test doses), the spread of the anesthesia must be carefully checked and verbal contact with the patient must be maintained.

Two-Segment Technique

Puncture is carried out in the midline at the level of L2/3 or L3/4. An 18-G Tuohy needle is used. After identification of the epidural space, the epidural catheter is advanced to a maximum of 3–4 cm. A test dose is then administered. One or two segments lower, conventional spinal anesthesia is then carried out using a 27-G pencil-point needle. The remainder of the procedure is the same as in the "needle-through-needle" technique.

Dosages in the "Needle-Through-Needle" and Two-Segment Techniques [7–11, 17]

Subarachnoid

- *Opioid*: 10 |jg sufentanil + 1 mL 0.9 % saline
- *Local anesthetic*:

  0.5 % ropivacaine 1–1.5 mL (5–7.5 mg) ± 0.2 mL
  0.5 % hyperbaric bupivacaine 1–1.5 mL (5–7.5 mg) ± 0.2 mL

- *Local anesthetic + opioid:*

  0.5 % ropivacaine + sufentanil 7.5–10 pg or
  0.5 % ropivacaine + fentanyl 25 pg
  0.5 % hyperbaric bupivacaine 1–2.5 mg + sufentanil 7.5–10 pg or
  0.5 % hyperbaric bupivacaine 1–2.5 mg + fentanyl 25 pg

Epidural

- Top-up dose

  After the fixation period for the local anesthetic injected intrathecally (ca. 15 min):
  0.5 % ropivacaine, 1.5–2 mL per unblocked segment or
  0.25–0.5 % bupivacaine, 1.5–2 mL per unblocked segment

- Epidural infusion after bolus administration of

  10–15 mL 0.1 % ropivacaine + 1–2 pg/mL sufentanil (10–20 pg) or 2 pg/mL fentanyl (30 pg) or
  10 mL 0.0625–0.125 % bupivacaine + 10–20 pg sufentanil or
  10 mL 0.125–025 % bupivacaine + 50 pg fentanyl
  Continuous infusion of:
  0.1 % ropivacaine + 0.2–0.3 pg/mL sufentanil (2 pg/mL fentanyl) at 10–12 mL/h or
  0.031–0.0625 % bupivacaine + 0.2–0.3 pg/mL sufentanil at 6–10 mL/h or
  0.0625 % bupivacaine + 1–2 pg/mL fentanyl at 10 mL/h

Sequential (Two-Stage) CSE in Cesarean Section

This technique has proved particularly useful for Cesarean section, reducing the frequency and severity of maternal hypotension [7–11].

First stage: procedure in sitting position

1. Identification of the epidural space (18-G Tuohy needle).
2. Advancing the spinal needle (27-G pencil-point) until dural perforation is achieved (dural click and free CSF flow).
3. Intrathecal administration of a local anesthetic and/ or opioid. The aim is to reach the segmental level of S5–T9 with as low a concentration as possible (e.g., hyperbaric bupivacaine 1.5 mL ± 0.2 mL).
4. Introduction of the epidural catheter.

Second stage: procedure in the supine position (left lateral decubitus)

1. 5–20 min wait until full spread of the subarachnoid local anesthetic is achieved (fixation period).
2. After subarachnoid spread of the local anesthetic, incremental epidural injection in small doses (top-up) is carried out through the epidural catheter. Ca. 1.5–2 mL 0.5 % bupivacaine is administered for each unblocked segment.

Advantages

The slow, incremental administration of the local anesthetic and/or opioid markedly reduces the risk of severe circulatory reactions during Cesarean section. The sympathetic block is less marked (lowest possible subarachnoid dosage and slow onset of epidural anesthesia). The body has time to activate compensatory mechanisms.

This procedure is particularly suitable for high-risk patients.

Disadvantage

More time-consuming

Dosage in Cesarean Section [7–11, 18]

Subarachnoid

0.5 % hyperbaric bupivacaine 1.5 ± 0.2 mL. Block target: S5–T8/9

Epidural

Top-up dose in left lateral decubitus position after the fixation period (ca. 15 min) of the local anesthetic injected subarachnoidally: 1.5–2 mL 0.5 % bupivacaine per unblocked segment

Dosage in Outpatient Obstetrics [7–11]

Subarachnoid (Single-Shot)

0.5 % hyperbaric bupivacaine 1–2.5 mg + 7.5–10 pg sufentanil or
0.5 % hyperbaric bupivacaine 1–2.5 mg + 25 pg fentanyl

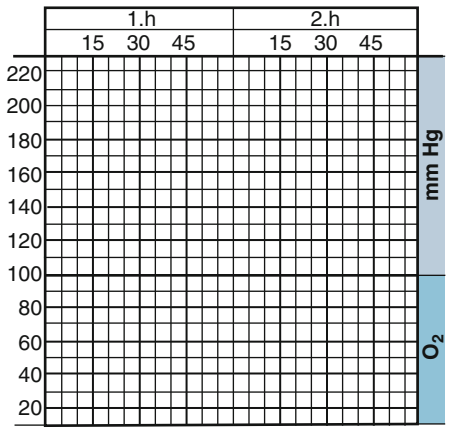
Epidural Top-up Dose (Continuous Infusion 10 mL/h)

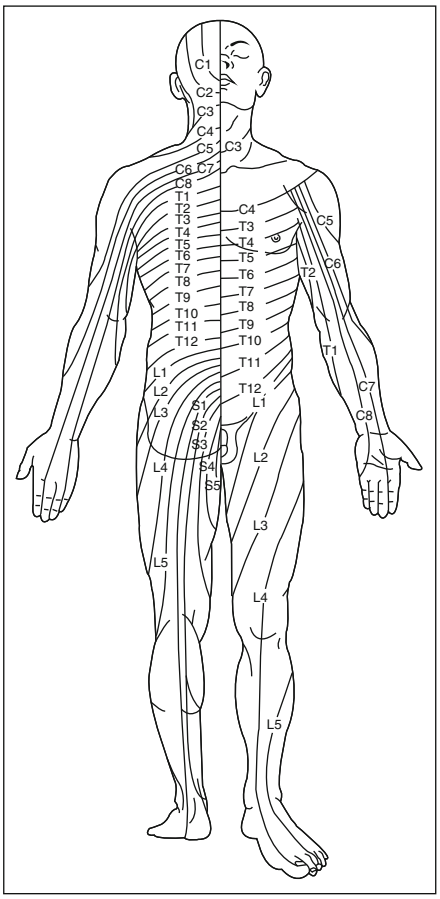
Bupivacaine 1 mg + 0.075–1.0 pg/mL sufentanil or
Bupivacaine 1 mg + 2 pg fentanyl

Complications

See Chap. 41 spinal and epidural anesthesia.

Record and checklist





With permission from Danilo Jankovic

Combined spinal and epidural anesthesia (CSE)

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Obstetric* ☐ *Postoperative*

Needle: Spinal: *G* _____ *Tip* _____

Epidural: *Tuohy* _____ *G* ☐ *Other*

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs)*: ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Lateral decubitus* ☐ *Sitting*

Access: ☐ *Median* ☐ *Paramedian*

Injection level: ☐ *L3 / L4* ☐ *L4 / L5* ☐ *Other* _____

☐ *Ultra sound guided* ☐ *Transducer* ☐ *Curved* ☐ *Linear*

Approach ☐ *In plane* ☐ *Out of plane*

Injection technique: ☐ *Needle-through-needle* ☐ *Two-segment*

Epidural space: ☐ *Identified*

Test dose: _____ Epinephrine added: ☐ *Yes* ☐ *No*

Subarachnoid:

Injection: ☐ *Carried out*

CSF aspiration: ☐ *Possible* ☐ *Not possible*

Local anesthetic: _____ *mL* _____ *%*

☐ Addition: _____ *µg/mg*

Epidural:

Epidural catheter: ☐ *Advanced 3–4 cm cranially*

Aspiration test: ☐ *Carried out*

Catheter end: ☐ *Positioned lower than the puncture site*

Bacterial filter: ☐

Local anesthetic: _____ *mL* _____ *%*
(incremental)

Abnormalities: ☐ *No* ☐ *Yes* _____

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 20 min:

☐ *Cold test* ☐ *Temperature measurement before* _____ *°C after* _____ *°C*

☐ *Sensory L* _____ *T* _____ ☐ *Motor*

Complications:

☐ *None* ☐ *Radicular symptoms* ☐ *BP drop* ☐ *Vascular puncture*
☐ *Massive epidural anesthesia* ☐ *Subdural spread* ☐ *Drop in body temperature*
☐ *Bladder emptying disturbances* ☐ *Back pain* ☐ *Aortocaval compression syndrome* ☐ *Pain* ☐ *Vasovagal reactions* ☐ *Dural puncture (epidural needle) or inadvertent dural puncture* ☐ *Intravascular injection* ☐ *Total spinal anesthesia* ☐ *Respiratory disturbance* ☐ *Muscle tremor* ☐ *Postdural puncture headache* ☐ *Neurological complecations*

Special notes:

References

1. Soresi A. Episubdural anesthesia. Anesth Analg. 1937;16:306–10.
2. Curelaru I. Long duration subarachnoid anaesthesia with continuous epidural block. Prakt Anaesth. 1979;14:71–8.
3. Brownridge P. Epidural and subarachnoid analgesia for elective caesarean section. Anaesthesia. 1981;36:70.
4. Coates M. Combined subarachnoid and epidural techniques. A single space technique for surgery of the hip and lower limb. Anaesthesia. 1982;37:89.
5. Mumtaz MH, Daz M, Kuz M. Combined subarachnoid and epidural techniques. Anaesthesia. 1982;37:30.
6. Carrie LES, O'Sullivan G. Subarachnoid bupivacaine 0.5 % for caesarean section. Eur J Anaesthesiol. 1984;1:275–83.
7. Rawal N. Single segment combined spinal-epidural block for caesarean section. Can Anaesth Soc J. 1986;33:254–5.
8. Rawal N. The combined spinal-epidural technique. Permanyer: S. L. Publications; 1997.
9. Rawal N. Combined spinal-epidural anesthesia. In: Van Zundert A, Ostheimer GW, editors. Pain relief and anaesthesia in obstetrics. New York: Churchill Livingstone; 1996. p. 413–26.
10. Rawal N, Schollin J, Wesström G. Epidural versus combined spinal-epidural block for caesarean section. Acta Anaesthesiol Scand. 1998;32:61–6.
11. Rawal N, Van Zundert A, Holmström B, Crowhurst J. Combined spinal-epidural technique. Reg Anesth. 1997;22(5):406–23.
12. Urmey WF, Stanton J, Peterson M, Sharrock NE. Combined spinal epidural anesthesia for outpatient surgery. Dose-response characteristic of intrathecal isobaric lidocaine using a 27-gauge Whitacre needle. Anesthesiology. 1995;83:528–34.
13. Vercauteren MP, Geernaert K, Vandeput DM, Adriansen H. Combined continuous spinal epidural anaesthesia with a single interspace, double catheter technique. Anaesthesia. 1993;48: 1002–4.
14. Peutrell J, Hughes DG. Combined spinal and epidural anaesthesia for inguinal hernia repair in babies. Pediatr Anaesth. 1994;4: 221–7.
15. Holmström B, Laugaland K, Rawal N, Hallberg S. Combined spinal epidural block versus spinal and epidural block for orthopedic surgery. Can J Anaesth. 1993;40:601–6.
16. Wilhelm S, Standl T. CSA vs. CSE bei Patienten in der Unfallchirurgie. Anaesthesist. 1997;46:938–42.
17. Vassiliev D, Nystrom E, Leicht G. Combined spinal and epidural anesthesia for labor and cesarean delivery in patient with Guillain-Barre syndrome. Reg Anesth Pain Med. 2001;26(2):174–6.
18. Kumar C. Combined subarachnoid and epidural block for caesarean section. Can J Anaesth. 1987;34:329–30.

Chapter 44

Caudal Epidural Injections in Adult Patients

John G. Hanlon
Department of Anesthesia, St. Michael's Hospital, University of Toronto, Toronto, Ontario, Canada

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, M5T 2S8 ON, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_44,

History and Background

The historical development of caudal epidural injections started with a flurry of reports in 1901. Sicard, a radiologist [27], was the first to describe the injection of cocaine through the sacral hiatus to treat a patient suffering from intractable sciatica. Independently, a week later, Cathelin, a urologist [8], reported on the successful caudal administration of local anesthetic for surgical procedures and relief of cancer pain due to an inoperable carcinoma of the rectum. Pasquier and Leri also reported on the use of caudal epidural injection for sciatica that same year [24]. Approximately 50 years passed before the first publication of caudal epidural steroids was published [7].

Despite the continued widespread use of caudal epidural injections for over a 100 years, debate continues regarding their effectiveness. This is due to the lack of well-designed, randomized controlled studies of epidural injections in general and caudal epidural injections in particular. The first systematic review of the effectiveness of caudal epidural steroid injections was published in 1985 and stated a lack of evidence of efficacy [16]. Since that first review, multiple systematic reviews and guidelines have been published by a variety of groups and societies both supporting and refuting the benefits of caudal epidural steroid injections [5, 12, 13, 23, 29]

Most recently, in 2012 Parr et al. [23] undertook a systematic review of caudal epidural injections with or without steroids in managing chronic pain secondary to lumbar disc herniation or radiculitis, post-lumbar laminectomy syndrome, spinal stenosis, and discogenic pain without disc herniation or radiculitis. Their primary outcome measures were short-term [less than six months] and long-term [greater than six months] pain relief. Secondary outcome measures included reduction in opioids, functional and psychological status, as well as return to work. Out of 73 identified studies, only 11 RCT and five non-RCT studies met their inclusion criteria. They concluded that despite the paucity of literature [specifically for chronic pain without disc herniation], there was good evidence for short- and long-term relief of chronic pain secondary to disc herniation or radiculitis with local anesthetics and steroids and fair relief with local anesthetics only. Their review also found fair evidence for caudal epidural injections and managing chronic axial or discogenic pain, spinal stenosis, and postsurgery syndrome.

Unfortunately, heterogeneous methodology, small size of study populations, and other problems including limited follow-up and outcome parameters of the original studies all contribute to the often conflicting results of systematic evaluation of the evidence by different individuals and societies.

Currently, the use of epidural steroids is generally recommended by the ASA and ASRA guidelines [2] which utilized a combined approach of physician consensus and systematic review. Conversely, the American College of Occupational and Environmental Medicine [ACOEM] guidelines as well as the American Academy of Neurology [AAN] [4] guidelines are not generally supportive of the use of epidural steroids. Interestingly, a reassessment of evidence synthesis of the ACOEM guidelines by Manchikanti et al. produced vastly different conclusions that support the use of epidural steroids [18, 19].

Anatomy

The sacrum is a triangular bone, dorsally convex, formed by the fusion of five sacral vertebrae. In most people, there is a natural defect resulting from incomplete fusion of the S5 vertebrae in the posterior midline where the spinous process (called median sacral crest) would otherwise exist. In many patients, there is also incomplete fusion of the lower portion of the S4 posterior midline, and in some patients, this midline defect can extend as high as S2 or S3 [15]. This bony defect [called the sacral hiatus] can have a variety of heights and shapes dependent on the mode and extent of fusion and ossification. Despite this variability, it is predictably bounded laterally at its most caudal part by two bony prominences called the sacral cornua (remnants of the S5 inferior articular processes) and is covered by the superficial dorsal, deep dorsal, and lateral sacrococcygeal ligaments which connect between the two cornua, the sacrum and coccyx. Sacral cornua may be impalpable in up to 21 % of cases [1]. The space deep to be sacrococcygeal ligament represents the most caudad component of the epidural space and is termed the sacral epidural canal. It encloses and protects the dura, arachnoid, and subarachnoid space, which, in most cases, ends at the level of the second sacral vertebra, as well as the sacral and coccygeal roots of the cauda equina, the sacral epidural venous plexus, lymphatic vessels, and epidural fat. Sample anatomic dissections can be seen in Fig. 44.1a, b. Studies have shown the mean diameter of the sacral canal at the sacral hiatus to be 5.3 ± 2 mm and the distance between the sacral cornua [bilateral] to be 9.7 ± 1.9 mm [10]. The sacral canal itself is shown in Figs. 44.2 and 44.3. It can have a diameter of 2–10 mm in an anteroposterior direction, and a volume capacity varies from 12 to 65 mL (average 30–34 mL) [26, 32]

Of note, the dura normally ends at the level of the second sacral foramina. However, due to anatomic variants of dura termination and sacral hiatus size, older studies have suggested the distance from the dura to the hiatus can range from 1.6 to 7.5 cm (average 4.5 cm) [26]. It has now been shown that the minimum distance between S2 and hiatus apex is as little as 0.75 cm [1]. The epidural venous plexus which is concentrated in the anterior space of the caudal canal generally terminates at the level of S4, although it can terminate even lower in elderly patients [15].

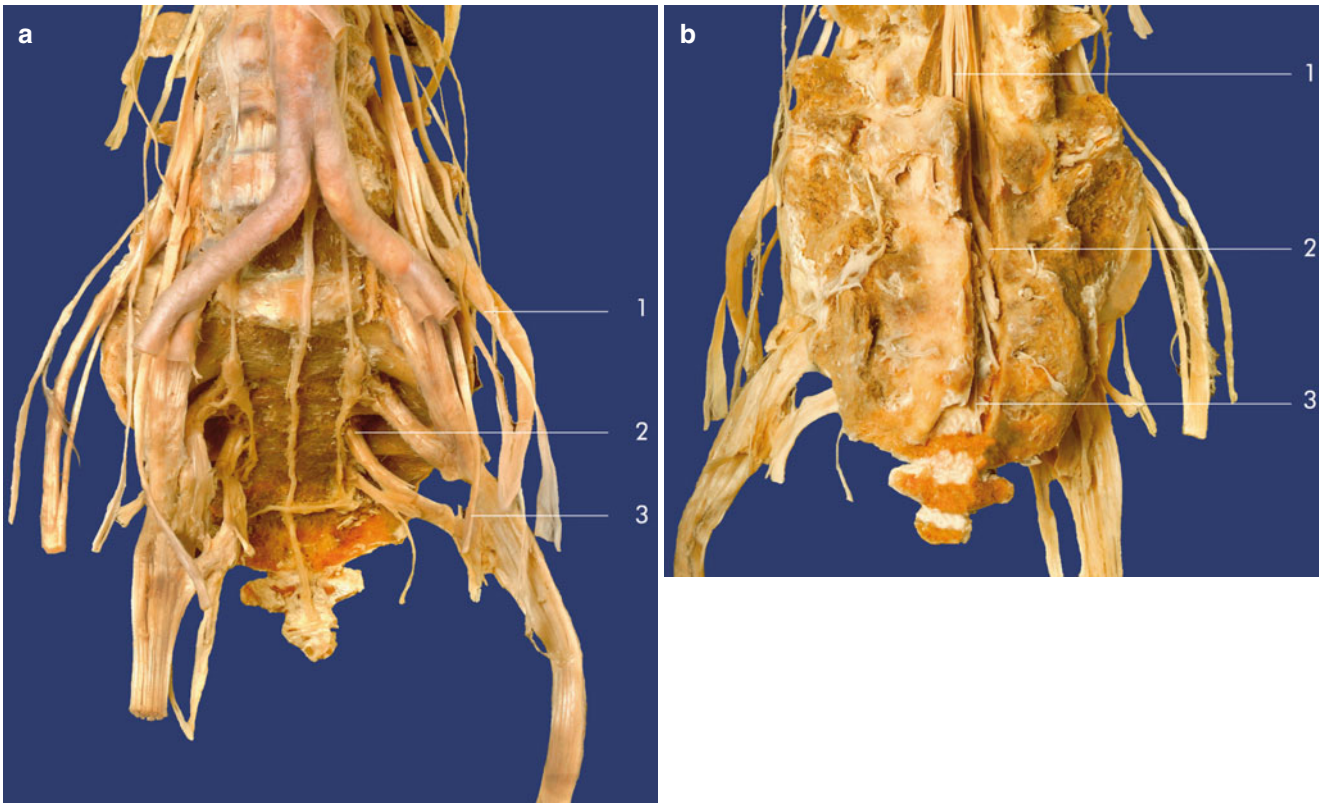


Fig. 44.1 Sacrum and lumbosacral nerves. (**a**) Posterior view displaying the terminal filum in the cauda equina (*1*), the spinal ganglia in the hemilaterally opened sacral canal (*2*), and the sacral hiatus (*3*) (Images kindly supplied by Danilo Jankovic). (**b**) Anterior view displaying the lumbosacral trunk (*1*), the anterior pelvic sacral foramina (*2*), and the sacral plexus (*3*) (Images kindly supplied by Danilo Jankovic)

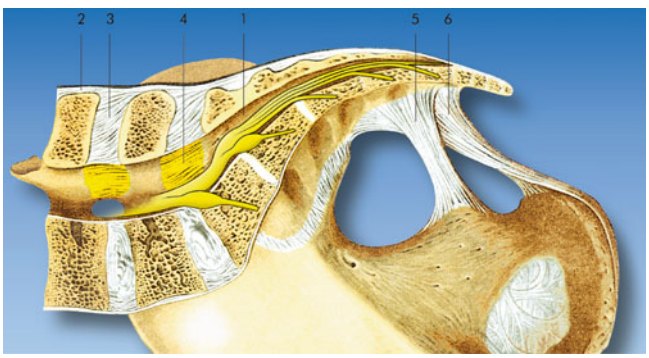


Fig. 44.2 Sacral canal cross section. (*1*) Sacral canal, (*2*) supraspinous ligament, (*3*) interspinous ligament, (*4*) ligamentum flavum, (*5*) sacrospinous ligament, (*6*) sacrotuberous ligament (Images kindly supplied by Danilo Jankovic)

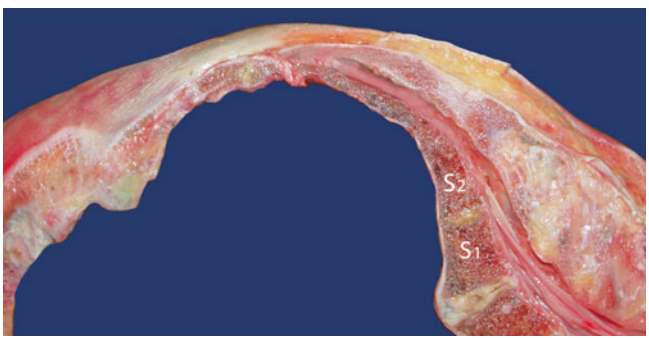


Fig. 44.3 Sacral canal cross section. The sacral canal has a diameter of 2–10 mm in the anteroposterior direction, with a variable capacity of 12–65 mL. The dura normally ends at the level of the second sacral foramina. *S1* and *S2* referred to the first and second sacral vertebra (Images kindly supplied by Danilo Jankovic)

Indications and Contraindications

Caudal epidural steroids are most commonly considered for patients with chronic low back pain with radicular pain secondary to disc herniation or radiculitis who have not responded to conservative modalities of treatment. The caudal approach is especially appealing in postlaminectomy patients with post-lumbar laminectomy syndrome or sacral involvement or those who have bilateral and multilevel pathology. These injections are also performed for patients with chronic axial or discogenic pain and spinal stenosis [13, 23].

Contraindications for caudal epidural injections can be divided into absolute and relative. Accordingly, the known risks of epidural steroids must be weighed against any potential benefit. Ideally, patients should be actively involved in the decision-making process.

Absolute

- Patient refusal
- Coagulation disorders, anticoagulant therapy
- Sepsis
- Local infections (skin diseases) at the puncture site
- Arachnoiditis
- Immune deficiency
- First trimester pregnancy
- Severe decompensated hypovolemia, shock
- Specific cardiovascular diseases of myocardial, ischemic, or valvular origin, if the planned procedure requires higher sensory spread
- Acute diseases of the brain and spinal cord
- Increased intracranial pressure
- A history of hypersensitivity to local anesthetics, without a prior intradermal test dose

Relative

- Pilonidal cyst
- Congenital anomalies of the dural sac and its contents

Technique: General Considerations

A written medical note with detailed neurologic history, physical examination, and radiographic reports including a working diagnosis should be in the chart and reviewed immediately prior to the procedure.

Written informed consent should be completed documenting a discussion of the options, potential benefits, and potential risks.

Intravenous access should be established and appropriate monitoring should be applied and maintained throughout. Accordingly the presence of all necessary equipment including resuscitation equipment should be confirmed.

The patient is then placed in a prone position, with one pillow under their head and one under their lower abdomen to reduce spinal lordosis. They are instructed to abduct their heels while keeping their toes together; this internal rotation ensures relaxation of the gluteal musculature. In obese patients, the gluteal cleft may be separated by attaching a broad band of sticky plaster between the skin of the buttocks and the operating table. Strict sterile technique must be employed and a large area should be prepped with an antiseptic solution that is approved for neuraxial procedures. It is also important to keep accurate records of the procedure and any unexpected events or complications.

For the procedure, some clinicians recommend a 22-g spinal needle to minimize procedural pain, whereas the authors suggest a Tuohy needle and catheter in order to deliver the injectate more cephalad in the sacral canal. Care should be taken not to introduce free skin particles with epidermal cells into the spinal canal, due to the potential risk of epidermoid tumors [6]. There are wide variations in the volume of the sacral canal; we recommend a preparation of corticosteroids with local anesthetic agents and/or normal saline in volumes ranging from 10 to 15 mL.

Caudal epidural injections can be performed using fluoroscopy, ultrasonography, or a landmark-based technique. These techniques are discussed below. It should be noted that in adults, the rate of inaccurate placement with landmark-based technique can be as high as 35 % in experienced hands [14, 30]. In addition, intravascular placement rates of 14 % and extra-epidural placement rates of 9 % have been reported. There are controlled studies that suggest "blind" caudal injections are not more effective than epidural injections of normal saline either for short-term outcomes or in the long term [5].

X-Ray-Guided Technique

Fluoroscopically guided caudal epidural anesthesia is considered the gold standard by most clinicians. Despite the lack of strong evidence that fluoroscopically guided injections are more effective, they are undoubtedly safer than a blind injection, and as such the use of C-Arm fluoroscopy makes rational sense [5].

It is recommended to start with the C-arm in an AP (anterior–posterior) orientation to ensure that the patient's pelvis is not laterally tilted. This is especially true in obese patients. Subsequently, in the lateral view, the sacral hiatus will usually be visible as a translucent opening toward the base of the caudal canal. Identification of the coccyx immediately caudad can also help with localization of the hiatus. Similarly, the clinician may palpate the coccyx and sacral cornua to help identify the point of needle insertion.

After adequate local anesthesia infiltration, an 18-gauge needle may be used to perforate the skin. At this point, the desired needle (spinal or Tuohy) is pushed through this perforation and advanced toward the hiatus. AP and lateral fluoroscopic images are taken to confirm the depth and direction of needle advancement. Once the needle is visualized just inside the canal, the Tuohy needle can be rotated 180° as necessary to enable smooth advancement. It should not be advanced beyond the S3 foramen to avoid damaging the sacral nerve roots and to minimize the risk of subarachnoid or subdural placement.

After negative aspiration for blood and CSF, appropriate myelographic quality radiocontrast material should be injected under live fluoroscopy. If venous runoff is noted, the needle tip can be withdrawn and moved until the venous runoff is no longer seen. If CSF is aspirated or a subdural or subarachnoid pattern is seen, the procedure should be terminated and repeated at a later date. Under normal circumstances, when contrast media is injected into the epidural space, a classic "Christmas-tree" pattern will be visualized within the bony canal. It should be noted that adhesions will limit dye spread. Subarachnoid dye spread will be noted centrally and extend significantly cephalad with only a small volume. Subdural injections will tend to be more patchy, and the contrast spread will not be as wide or predictable as subarachnoid pattern [5, 25]. Images of stages of a fluoroscopically guided caudal injection are seen below in Fig. 44.4. Examples of a wide variety of contrast spread patterns have been published elsewhere [28].

After final placement of the needle or catheter and repeat negative aspiration, local anesthetic should be injected as a test dose prior to injection of the full dose of medication in aliquots. The patient may report pressure upon injection which should be halted until this pain has dissipated. Upon completion of the procedure, the stylet should be reinserted prior to needle removal. A sterile adhesive bandage should also be applied over the puncture site.

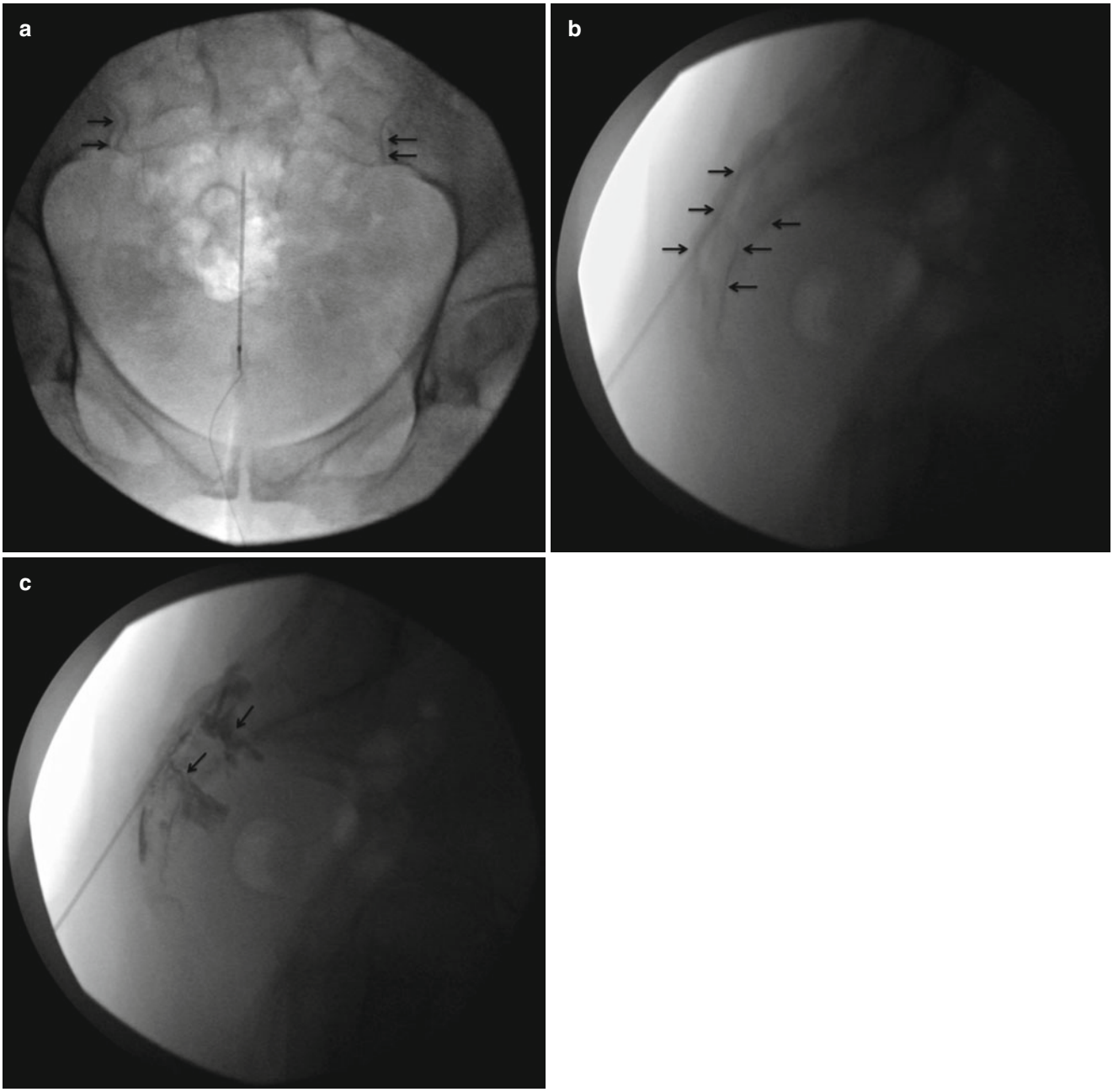


Fig. 44.4 Images of fluoroscopically guided caudal injection of steroids (Reproduced with permission from Philip Peng Educational Series). (**a**) Anteroposterior view of needle in the sacral canal. With the presence of bowel gas, it is a challenge to visualize the details of the sacrum and caudal canal. The sacroiliac joint is indicated by the *arrows*. (**b**) Lateral view showing the needle in the sacral canal which is indicated by the *arrows*. (**c**) Lateral view after injection of contrast medium. The contrast (indicated by *arrows*) is seen leaking out from the sacral foramen

Ultrasound-Guided Technique

Ultrasonography in the field of pain medicine continues to grow as the technology advances. It is noninvasive, safe, simple to use, does not involve exposure to radiation, and has the potential to provide real-time images.

The potential benefit of ultrasound imaging for caudal epidural steroids was first described in 2003 [17]. The investigators found it to be especially helpful in moderately obese patients who were unable to assume the prone position. It is also useful when faced with decreased bone density, gas or feces in the rectum, or where fluoroscopy is not available or contraindicated [15, 20, 22].

Consistently it has been demonstrated that ultrasonography can be an excellent aid for needle placement. However, it is limited by the fact that the needle tip is no longer visualized after advancing into the sacral epidural space secondary to bony artifacts [3, 9, 31], and as such, dural puncture and intravascular placement cannot be readily identified. Investigators have looked at the use of color Doppler ultrasonography to help detect intravascular injection. Although Doppler is possibly a helpful adjunct, it is not sufficiently specific or sensitive to rule out intravascular injection or rule in epidural placement of the injectate [22, 33] Thus, the role of ultrasonography is to facilitate the placement of needle into the sacral canal, especially in patients with abnormal or variant pelvic/spinal anatomy, decreased bone density, gas or feces in the rectum obscuring visualization of sacrum, or where fluoroscopy is not available or contraindicated [15, 20, 22].

A number of general considerations must be addressed before one considers performing caudal ultrasonography. The sacrum and sacrococcygeal ligament is very superficial, even in obese patients. As such, unlike sonoanatomy of the lumbar spine, a high-frequency linear transducer can and should be used for most caudal epidural injections. Accordingly, since ultrasonography depends on the presence of acoustic windows and cannot penetrate bone, one must be cognizant that they can only appreciate the most superficial bony outline of the sacrum, which is the most variable and unpredictable component of the adult spine. The last and perhaps the most important consideration is the fact that most ultrasonography coupling mediums [such as sterile gel] have not been demonstrated to be safe for neuraxial procedures. As such individuals must wipe off the gel completely at the site of needle insertion and/or may consider saline or another local anesthetic as the coupling medium.

Following the strict aseptic technique and the use of sterile ultrasound cover, the scanning starts with the probe in a transverse orientation at the level of S5, the sacral hiatus, which can be determined initially by palpation. On the screen, the sacral cornua will be seen as two hyperechoic upside-down "U"-shaped structures ("frog's eye sign" because of its resemblance to the eyes and head of a frog) (Fig. 44.5a). This view also shows two hyperechoic structures: the more superficial sacrococcygeal ligament and the deeper bony posterior surface of the sacrum. By rotating the probe 90°, a midline sagittal sonogram demonstrates the posterior bony outline of the sacrum and coccyx, the hyperechoic band of the sacrococcygeal ligament, and the sacral canal (Fig. 44.5b). Clinicians should pay attention to sonographic features such as sacral hiatus diameter of <1.5 mm or closed sacral hiatus (on sonography) as these suggest greater probability for technical failure [11].

With the long-axis view of the sacral canal, local anesthetic infiltration of the skin is performed approximately 2 cm away from the caudal end of the probe. The Tuohy needle is advanced cephalad within the acoustic window created by the sacrococcygeal ligament (Fig. 44.5c). Following the puncture of the sacrococcygeal ligament, the needle is advanced, when possible, in the posterior part of the canal for a short distance (1 cm) as there is no way to determine needle-tip position once it has moved beyond this acoustic window and because the sacral venous plexus lays on the anterior (deep) wall of the sacral canal terminating at S4 [15]. If one plans to insert the catheter, it is suggested to rotate so that the bevel is facing posteriorly. Two groups have described using fluoroscopy during contrast injection to confirm the position of needles that were placed into the caudal canal using ultrasound guidance and reported 100 % [10] and 95.6 % success rates [21], respectively. Others have prospectively compared ultrasound-guided vs. fluoroscopy-guided caudal epidural steroid injection for the treatment of unilateral lower lumbar radicular pain.

Recently, a single-blind randomized study of patients with unilateral lower limb radicular pain showed similar improvements in short-term pain relief, function, and patient satisfaction with both ultrasound and fluoroscopic guidance [22]. Despite this encouraging study, it is the opinion of the authors that ultrasonography, albeit, better than a landmark technique, is not equivalent to fluoroscopically guide approach, due to its limitations in recognizing intravascular and subdural or subarachnoid needle/catheter placement. Instead, it is a useful adjunct for the advancement of needle that may improve success rates and patient comfort while decreasing exposure to radiation.

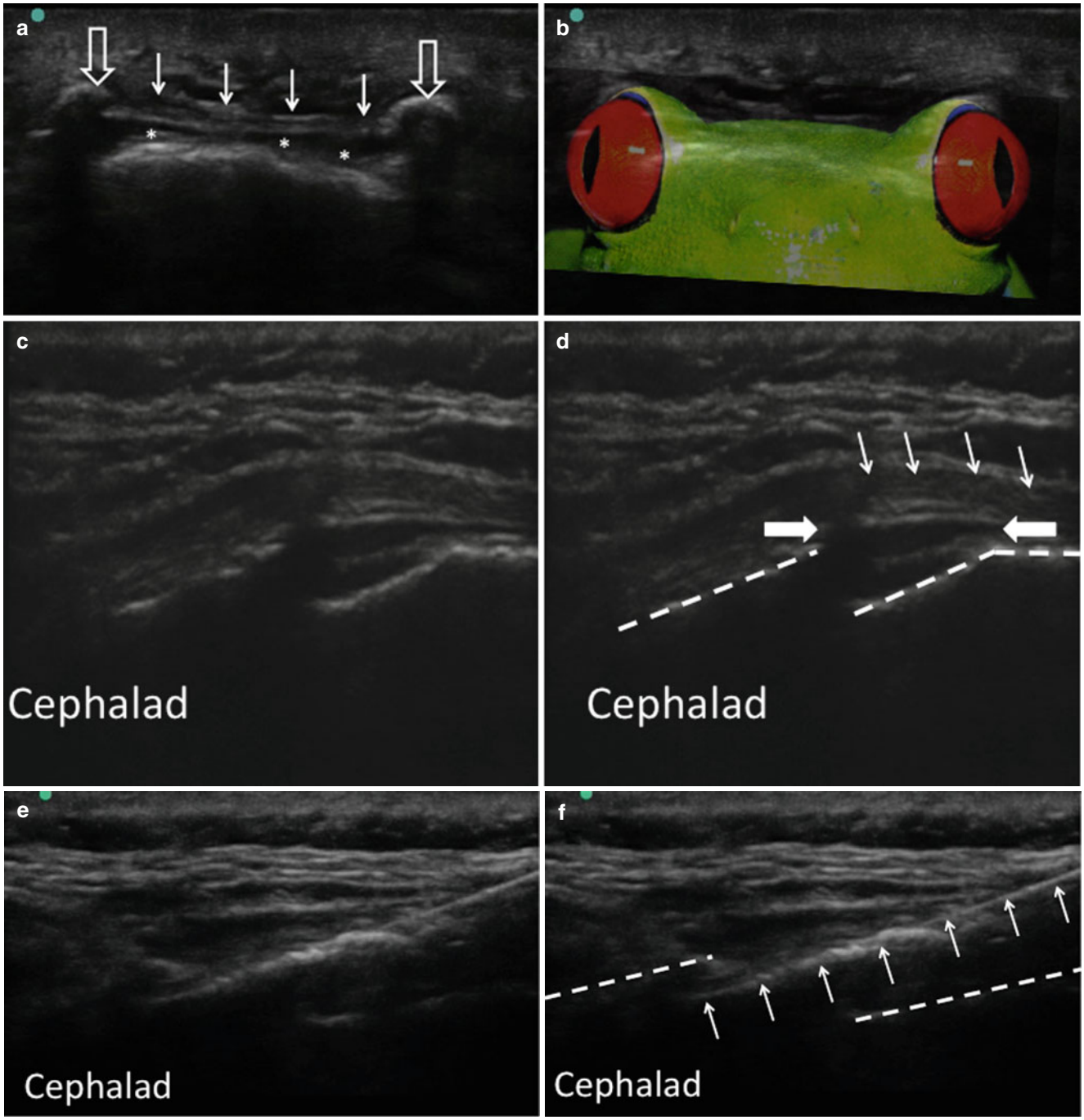


Fig. 44.5 Ultrasound images of sacral canal (Reproduced with permission from Philip Peng Educational Series). (**a**) Short-axis scan of the sacrum at the level of the sacral hiatus, displaying the two sacral cornua (*open arrows*), and the hyperechoic sacrococcygeal ligament (*line arrows*) that extends between the two cornua and the sacral hiatus (*asterisk*) which is the hypoechoic space between the sacrococcygeal ligament and the posterior surface of the sacrum. (**b**) Figure same as **a** with frog eyes superimposed on the ultrasound image. (**c**) Long-axis view of the sacrum at the level of the sacral hiatus. (**d**) Same as **c** with the sacral hiatus outlined by *bold arrows*. The sacrococcygeal ligament (*line arrows*) is seen extending from the sacrum to the coccyx. More cephalad, the acoustic shadow of the dorsal sacrum bone completely obscures the ventral sacrum bone of the sacral canal (*dashed lines*). (**e**) Long-axis view of sacrum showing the needle insertion. (**f**) Same as **e** with *line arrows* to outline the needle which cannot be visualized deep in the sacral canal (*dashed lines*). The needle was inserted with in-plane technique

Post-procedure Care

Patients should be monitored for a minimum of 30 min after the procedure to ensure the absence of motor block or other complications. Even in the absence of obvious motor block, patients should be advised of the possibility of mild motor block affecting their balance and walking for hours following the procedure. They may be discharged home when ambulatory with written discharge instructions to contact their treating physician or an emergency department as needed for any problems [25].

Complications

A variety of side effects and complications are possible after caudal epidural steroid injections. Many of the rare and common complications are outlined below:

- Worsened pain
- Nausea and vomiting
- Dizziness and vasovagal reactions
- Infection, epidural abscess
- Aseptic meningitis
- Allergic or anaphylactoid reactions
- Hypotension and bradycardia
- Sacral or lumber nerve root injury, cauda equina syndrome
- Spinal cord injury
- Bladder or bowel dysfunction
- Intravascular puncture: bleeding, epidural hematoma, venous air embolism
- Steroid effects: metabolic and fluid retention effects, Cushing's syndrome, epidural lipomatosis, avascular necrosis
- Dural puncture: CSF leak, intracranial hypotension, and intracranial hematomas
- Subarachnoid or subdural injection: profound motor block, post-dural puncture headache, arachnoiditis
- Injury to surrounding structures: rectal perforation (Fig. 44.6)
- Needle breakage or catheter sheering

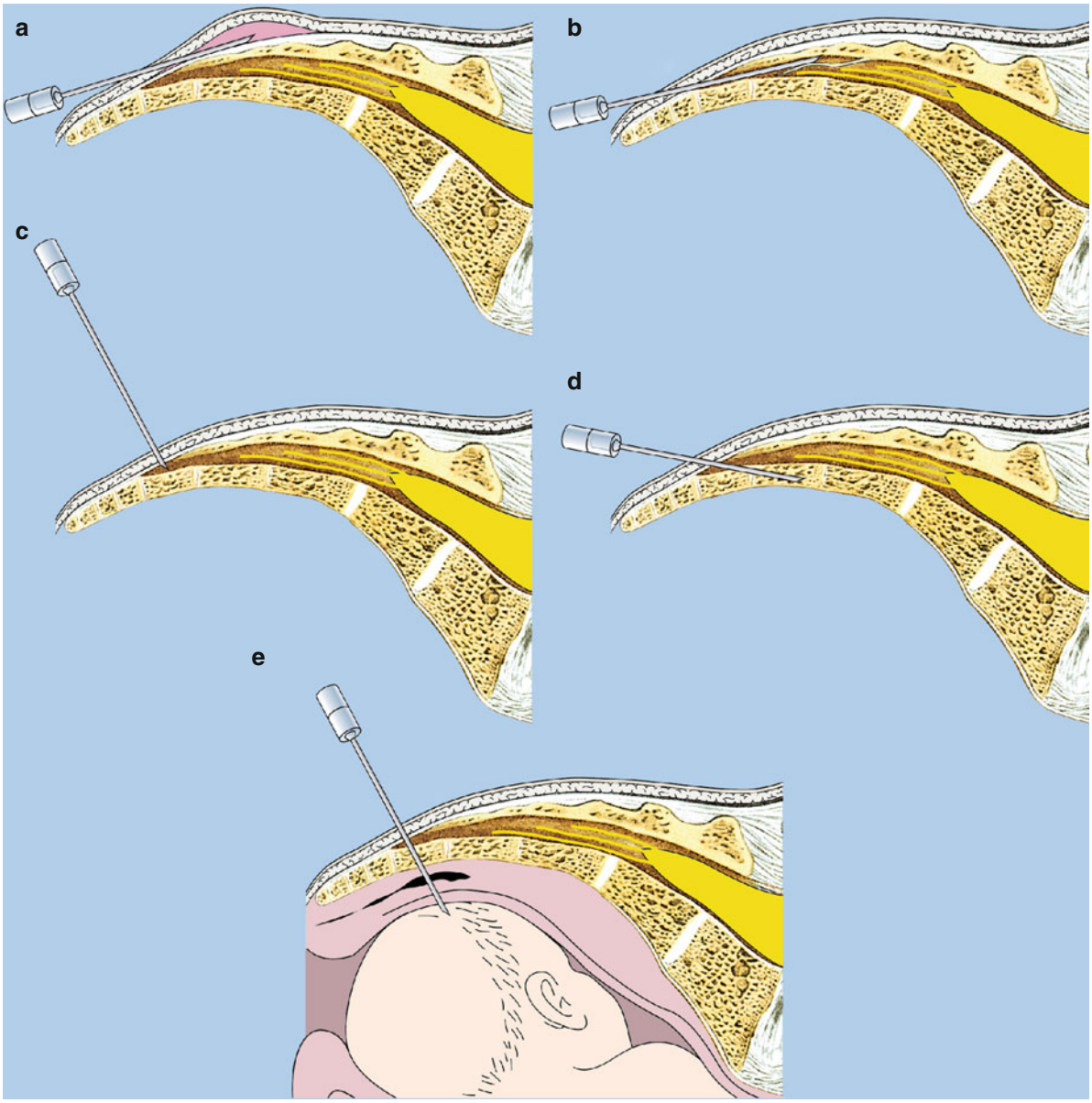


Fig. 44.6 Complications due to incorrect technique. (**a**) Outside the sacral canal, (**b**) subperiosteal, (**c**) into the sacrococcygeal ligament, (**d**) spongiosa, (**e**) through the sacrum and rectum (Reproduced with permission from Danilo Jankovic)

Caudal anesthesia

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Therapeutic* ☐ *Diagnostic*

Needle: ☐ *G* _____ ☐ *With stylet* ☐ *Without stylet*

i.v. access, infusion: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Position: ☐ *Prone* ☐ *Lateral decubitus*

Epidural space: ☐ *Identified*

Checking position of needle tip relative to dural sac (2nd sacral foramen):
☐ *Carried out*

Aspiration test: ☐ *Carried out*

Injection: *(5 cm³ air or 0.9% Nacl)* ☐ *Carried out*

Test dose: _____ Epinephrine added: ☐ *Yes* ☐ *No*

Motor and sensory function check after 5 min: ☐ *Carried out*

Abnormalities: ☐ *No* ☐ *Yes* _____

Injection: ☐ *Ultrasound guided* ☐ *Transducer* ☐ *Curved* ☐ *Linear*

Approach: ☐ *In plane* ☐ *Out of plane*

Local anesthetic: _____ *mL* _____ *%*
(incremental)

☐ Addition: _____ *µg/mg*

Patient's remarks during injection:

☐ *None* ☐ *Pain* ☐ *Paresthesias* ☐ *Warmth*

Duration and area: _____

Objective block effect after 20 min:

☐ *Cold test* ☐ *Temperature measurement before* _____*°C* *after* _____*°C*

☐ *Sensory: L* _____ *T* _____

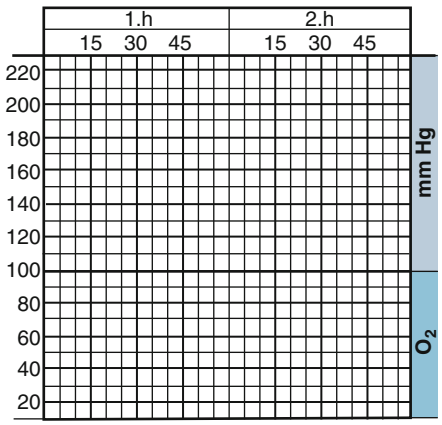
☐ *Motor*

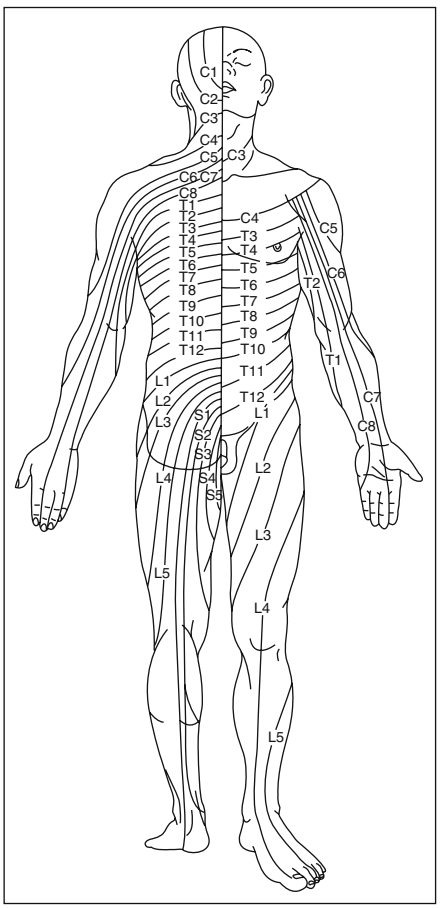
Complications:

☐ *None*
☐ *Radicular symptoms*
☐ *BP drop*
☐ *Vascular puncture*
☐ *Massive epidural anesthesia*
☐ *Bladder emptying disturbances*
☐ *Coccygeal pain*
☐ *Neurological complications*
☐ *Pain*
☐ *Vasovagal reactions*
☐ *Dural puncture*
☐ *Intravascular injection*
☐ *Total spinal anesthesia*
☐ *Respiratory disturbance*
☐ *Postdural puncture headache*

Special notes:

Record and checklist





With permission from Danilo Jankovic

References

1. Aggarwal A, Aggarwal A, Harjeet SD. Morphometry of sacral hiatus and its clinical relevance in caudal epidural block. Surg Radiol Anat. 2009;31:793–800.
2. American Society of Anesthesiologists Task Force on Chronic Pain Management, American Society of Regional Anesthesia and Pain Medicine. Practice guidelines for chronic pain management: an updated report by the American Society of Anesthesiologists Task Force on Chronic Pain Management and the American Society of Regional Anesthesia and Pain Medicine. Anesthesiology. 2010;112:810–33.
3. Apan A, Cuvas O. Caudal block in adults: new horizons with ultrasound. Minerva Anestesiol. 2013;79:1332–3.
4. Armon C, Argoff CE, Samuels J, Backonja M-M. Assessment: use of epidural steroid injections to treat radicular lumbosacral pain: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2007;68:723–9.
5. Bogduk N. Epidural steroid injections. In: Fishman SM, Ballantyne JC, Rathmell JP, editors. Bonica's management of pain. 4th ed. Baltimore: Lippincott Williams & Wilkins; 2010. p. 1424–58.
6. Boyd H. Iatrogenic intraspinal epidermoid. J Neurosurg. 1966;24:105–7.
7. Cappio M. Il trattamento idrocortisonico per via epidurale sacrale delle lombosciatalgie. Reumatismo. 1957;9:60–70.
8. Cathelin F. Mode d'action de a cocaineinjete daus l'escapte epidural par le procede du canal sacre. C R Soc Biol. 1901;53:478–9.
9. Chen CPC, Lew HL, Tang SFT. Ultrasound-guided caudal epidural injection technique. Am J Phys Med Rehabil. 2014.
10. Chen CPC, Tang SFT, Hsu T-C, et al. Ultrasound guidance in caudal epidural needle placement. Anesthesiology. 2004;101:181–4.
11. Chen CP, Wong AM, Hsu C-C, et al. Ultrasound as a screening tool for proceeding with caudal epidural injections. Arch Phys Med Rehabil. 2010;91:358–63.
12. Chou R, Loeser JD, Owens DK, et al. Interventional therapies, surgery, and interdisciplinary rehabilitation for low back pain: an evidence-based clinical practice guideline from the American Pain Society. Spine (Phila Pa 1976). 2009;34:1066–77.
13. Conn A, Buenaventura RM, Datta S, et al. Systematic review of caudal epidural injections in the management of chronic low back pain. Pain Physician. 2009;12:109–35.
14. El-Khoury G, Ehara S, Weinstein J. Epidural steroid injection: a procedure ideally performed with fluoroscopic control. Radiology. 1988;168:161–78.
15. Karmakar M. Ultrasound-guided central neuraxial blocks. In: Narouze S, editor. Atlas of ultrasound-guided procedures in interventional pain management. 1st ed. New York: Springer; 2011. p. 1–372.
16. Kepes E, Duncalf D. Treatment of backache with spinal injections of local anesthetics, spinal and systemic steroids. Pain. 1895;22:33–47.
17. Klocke R, Jenkinson T, Glew D. Sonographically guided caudal epidural steroid injections. J Ultrasound Med. 2003;22:1229–32.
18. Manchikanti L, Cash K, Pampati V, et al. Evaluation of fluoroscopically guided caudal epidural injections. Pain Physician. 2004;7:81–92.
19. Manchikanti L, Singh V, Derby R, et al. Reassessment of evidence synthesis of occupational medicine practice guidelines for interventional pain management. Pain Physician. 2008;11:393–482.
20. Narouze S, Peng PWH. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures. Reg Anesth Pain Med. 2010;35:386–96.
21. Nikooseresht M, Hashemi M, Mohajerani SA, et al. Ultrasound as a screening tool for performing caudal epidural injections. Iran J Radiol. 2014;11:e13262.
22. Park Y, Lee J-H, Park KD, et al. Ultrasound-guided vs. fluoroscopy-guided caudal epidural steroid injection for the treatment of unilateral lower lumbar radicular pain: a prospective, randomized, single-blind clinical study. Am J Phys Med Rehabil. 2013;92:575–86.
23. Parr AT, Manchikanti L, Hameed H, et al. Caudal epidural injections in the management of chronic low back pain: a systematic appraisal of the literature. Pain Physician. 2012;15:E159–98.
24. Pasquier N, Leri D. Injection intra-et extradurales de cocaine a dose minime daus le traitment de la sciatique. Bull Gen Ther. 1901;142:196.
25. Racz GB, Noe CE. Pelvic spinal neuroaxial procedures. In: Raj PP, editor. Interventional pain management. Image-guided procedures, 2nd ed. Philadelphia: Saunders, an imprint of Elsevier Inc.; p. 405–28.
26. Reisner L, Ellis J. Epidural and caudal puncture. In: Benumof LJ, editor. Clinical procedures in anesthesia and intensive care. Philadelphia: Lippincott; 1992. p. 681.
27. Sicard J. Les injections medicamenteuse extraduraqles per voie saracoccygiene. C R Seances Soc Biol Fil. 1901;53:396–8.
28. Singh V, Manchikanti L. Caudal epidural injections. Interv. Tech. Chronic Spinal Pain; 2007. p. 331–354.
29. Staal J, de Bie R, de Vet H, et al. Injection therapy for subacute and chronic low back pain: an updated cochrane review. Spine (Phila Pa 1976). 2009;34:49–59.
30. Stitz MY, Sommer HM. Accuracy of blind versus fluoroscopically guided caudal epidural injection. Spine (Phila Pa 1976). 1999;24:1371–6.
31. Tsuchiya M, Kyoh Y, Mizutani K, et al. Ultrasound-guided single shot caudal block anesthesia reduces postoperative urinary catheter-induced discomfort. Minerva Anestesiol. 2013;79:1381–8.
32. Wills R. Caudal epidural blockade. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 2nd ed. 1988; Philadelphia, PA: JB Lippincott; p. 361–83
33. Yoon JS, Sim KH, Kim SJ, et al. The feasibility of color Doppler ultrasonography for caudal epidural steroid injection. Pain. 2005;118:210–4.

Chapter 45

Lumbar Sympathetic Block

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_45, © Springer International Publishing Switzerland 2015

Definition

Injection of a local anesthetic or neurolytic in the sympathetic ganglia of the lumbar sympathetic trunk.

Anatomy (Figs. 45.1, 45.2 and 45.3a–d) [1–5]

From the T12 ganglion, the sympathetic trunk passes into the abdominal cavity. The abdominal part of the trunk reaches the anterolateral surface of the lumbar vertebrae, lying directly medial to the origin of the psoas, to the right behind the inferior vena cava and cisterna chyli, and to the left beside the aorta. The lumbar part of the sympathetic trunk usually contains only four lumbar ganglia (due to fusion of the twelfth thoracic and first lumbar ganglion), with a spindle shape or oval shape. The final ganglion is usually the largest.

The average length of the ganglia is ca. 3–5 mm (more rarely, up to 10–15 mm). The psoas muscle and its fascia separate the sympathetic nerve trunk from the lumbar somatic spinal nerves.

The sympathetic trunks give off and receive communicating and visceral branches, as well as vascular, muscular, osseous, and articular branches.

White communicating branches only reach the lumbar sympathetic ganglia from the two cranial spinal nerves, as well as from the three lumbar spinal nerves. They carry preganglionic fibers and visceral afferents. Gray communicating branches are given off from the corresponding ganglia to all the lumbar spinal nerves. They contain vasomotor, sudomotor, and pilomotor fibers, which are distributed with the lumbar spinal nerves. From the lumbar part of the sympathetic chain, some branches run to the renal plexus, but most pass to the abdominal aortic plexus and the hypogastric plexus. Most of the sympathetic nerve fibers responsible for the lower extremity pass through the L2 (dominant) and L3 ganglia.

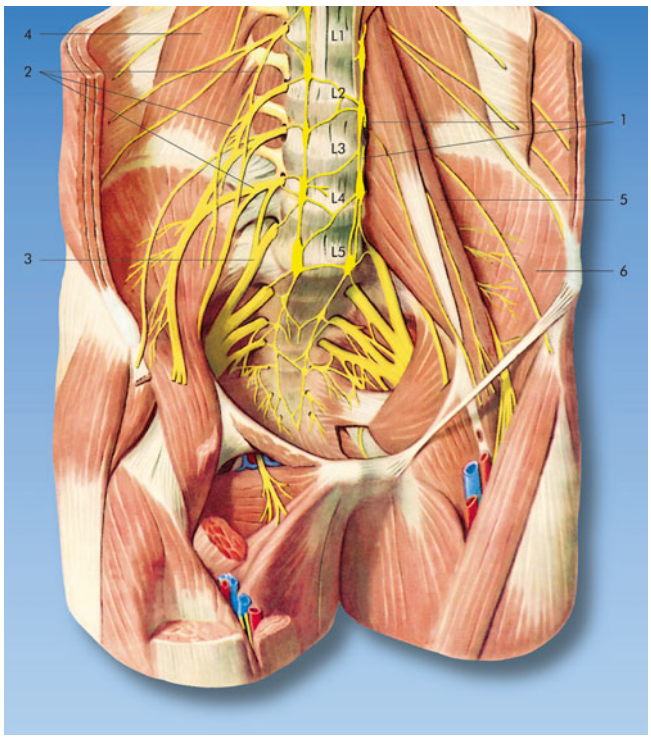


Fig. 45.1 Anatomy (anterior view): (*1*) sympathetic trunk with communicating branches, (*2*) lumbar plexus, (*3*) lumbosacral trunk, (*4*) quadratus lumborum muscle, (*5*) psoas major muscle, (*6*) iliac muscle (Reproduced with permission from Danilo Jankovic)

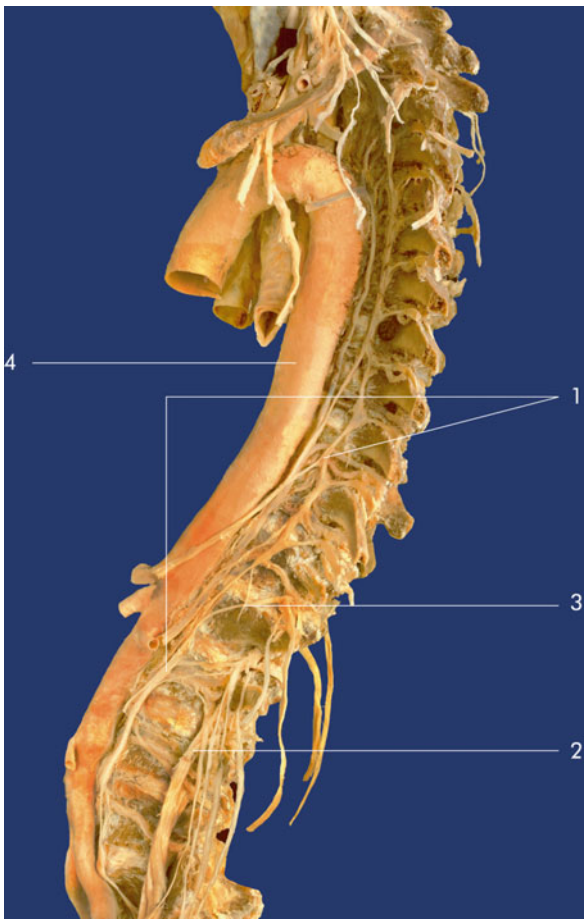


Fig. 45.2 Anatomy.(*1*) Lumbar sympathetic chain, with its accompanying ganglia, (*2*)somatic paravertebral nerves, (*3*) rami communicantes, (*4*) aorta (Reproduced with permission from Danilo Jankovic)

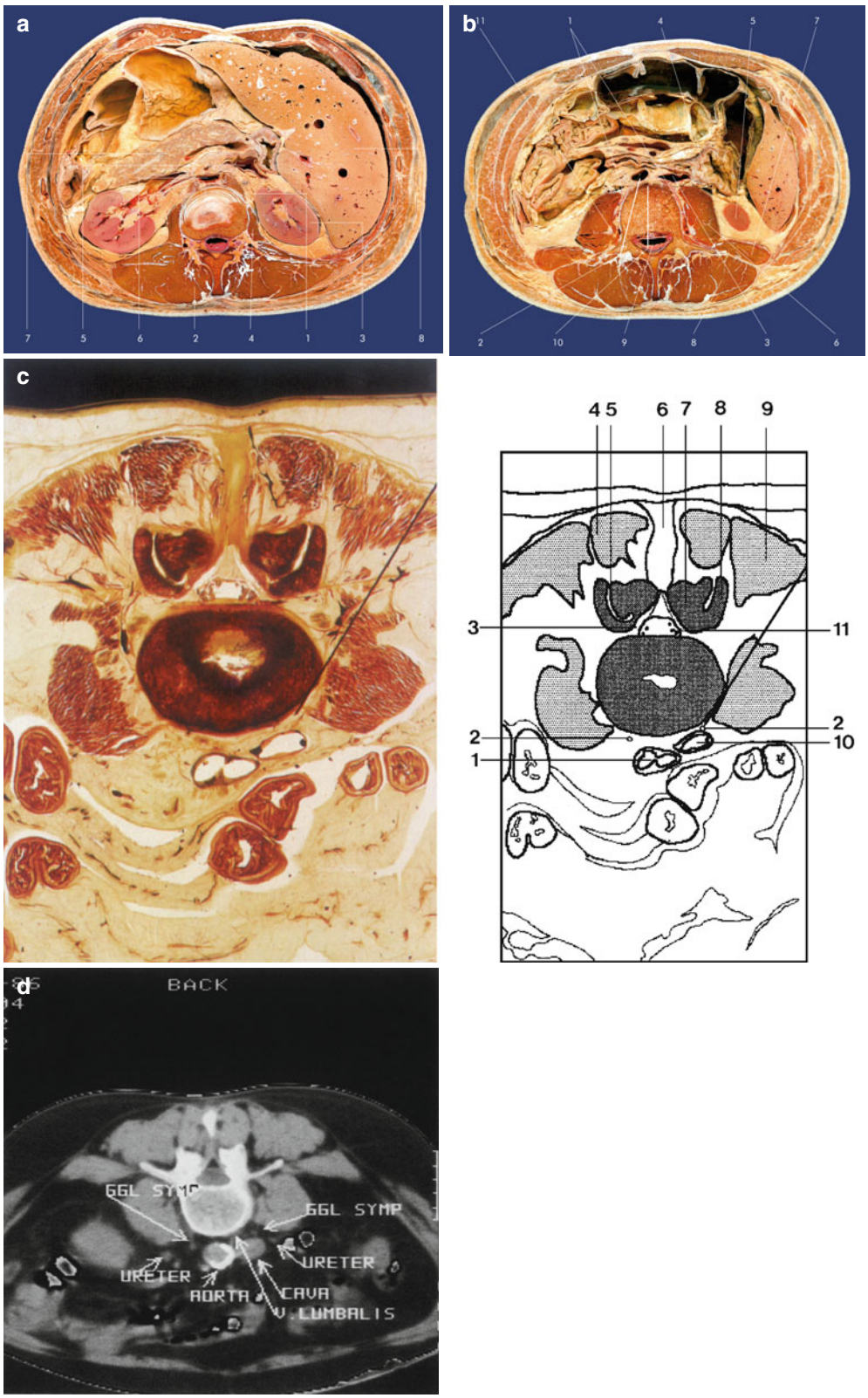


Fig 45.3 (**a**) Lumbar sympathetic block. Cross -sectional anatomy at the level of L1, L2. (*1*) Sympathetic ganglion, (*2*) erector spinae muscle, (*3*) medulla renalis, (*4*) psoas major muscle, (*5*) inferior vena cava, (*6*) aorta, (*7*) pancreas, (*8*) liver (Reproduced with permission from Danilo Jankovic). (**b**) Lumbar sympathetic block. Cross-sectional anatomy at the level of L3. (*1*) Sympathetic trunk, (*2*) aorta, (*3*) inferior vena cava, (*4*) ureter, (*5*) kidney, (*6*) psoas major muscle, (*7*) quadratus lumborum muscle, (*8*) erector spinae muscle, (*9*) cauda equina, (*10*) anterior longitudinal ligament, (*11*) lumbar plexus (Reproduced with permission from Danilo Jankovic). (**c**) Lumbar sympathetic block (anatomy and diagram). (*1*) Aorta, (*2*) sympathetic ganglion, (*3*) epidural space, (*4*) medial tract of back muscles, (*5*) intervertebral joint, (*6*) interspinal ligament, (*7*) superior articular process of L3, (*8*) inferior articular process of L4, (*9*) lateral tract of back muscles, (*10*) vena cava, (*11*) filum terminale (Reproduced with permission from Danilo Jankovic). (**d**) CT section in the center of the L4 vertebra. The sympathetic ganglia are well delineated in the fatty tissue on each side. Neighboring structures, such as the vessels and ureters, can also be precisely differentiated

Indications [6–9]

Diagnostic and Prognostic

- Differentiation between various forms of vasospastic disease in the area of the lower extremities
- Prognostic block to establish an indication for surgical sympathectomy or a neurolytic block
- Checking (confirmation) of a surgical sympathectomy

Therapeutic

- Pain caused by perfusion disturbances in vasospastic diseases in the region of the lower extremities, in the form of arterial or venous dysfunction or a combination of the two
- Intermittent claudication
- Embolism and thrombosis
- Thrombophlebitis and post-phlebitic edema
- Post-reconstructive vascular procedures
- After frostbite or trauma
- Complex regional pain syndrome (CRPS) types I and II
- Phantom limb pain
- Erythromelalgia
- Acrocyanosis
- Phlegmasia alba dolens (milk leg)
- Persistent infection of the leg
- Poorly healing ulcers
- Neuropathy after radiotherapy
- Hyperhidrosis of the lower body
- Acute phase of herpes zoster
- Visceral pain (e.g., renal colic)

Block Series

A series of six to eight blocks is recommended. When there is evidence of improvement in the symptoms, additional blocks can also be carried out.

Contraindications

1. Anticoagulant treatment
2. Infections and skin diseases in the injection area
3. Addition of vasopressors in patients with peripheral circulatory disturbances

Procedure [1–5]

This block should only be carried out by experienced anesthetists or under their supervision. Full information should be given to the patient.

Preparations

Check that the emergency equipment is complete and in working order; sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, emergency medication.

Materials

Fine 26-G needle, 25 mm long, for local anesthesia. Atraumatic 22-G needle, 0.7 × 120 mm (15°) with injection lead or spinal needle, 0.7 (0.9) × 120 mm (150 mm), 20–22 G Syringes: 2, 5 and 10 mL.

Disinfectant, swabs, compresses, sterile gloves and drape, flat, firm pillow.

Patient Positioning

1. Prone position: support with a pillow in the mid-abdomen (to eliminate lumbar lordosis). The patient's arms should be dangling.

   The patient should breathe with the mouth open, to reduce tension in the back muscles. This position is preferable, particularly when the block is carried out under radiographic control using an image intensifier.
2. Lateral decubitus: the flank is supported with a pillow. The side being blocked should be uppermost.

Landmarks (Fig. 45.4a, b)

1. L4 iliac crest line.
2. L2: a parallel line is drawn ca. four fingerbreadths (7 cm) from the midline. The intersection point between this line and the twelfth rib is at the level of L2.
3. Midpoint of each spinous process of the relevant lumbar vertebra.

Disinfection, generous local anesthetic infiltration of the injection channel, covering with a sterile drape, drawing up the local anesthetic, testing the patency of the injection needle.

During the injection, the following points must be observed:

- In clinical routine, a sympathetic block is started at the L2 segment; block of segment L4 follows.
- The person carrying out the injection must stand on the side being blocked.
- Usually, the injection point should not be located more than 8 cm lateral to the midline (risk of renal puncture) and no more than 5 cm medial to it (the lateral side of the vertebra is more difficult to reach).
- Paresthesias occur relatively frequently during introduction of the needle and indicate irritation of the lumbar somatic nerves.
- Inadvertent block of the lumbar somatic nerves can lead to motor weakness of the contralateral leg. The patient should be warned of this.
- There is a risk of perforating the dural cuff if the injection is made too far medially (epidural or subarachnoid injection).
- Aspirate frequently and inject incrementally.
- No neurolytics should be administered without precise confirmation of the needle position using radiographic control with an image intensifier.

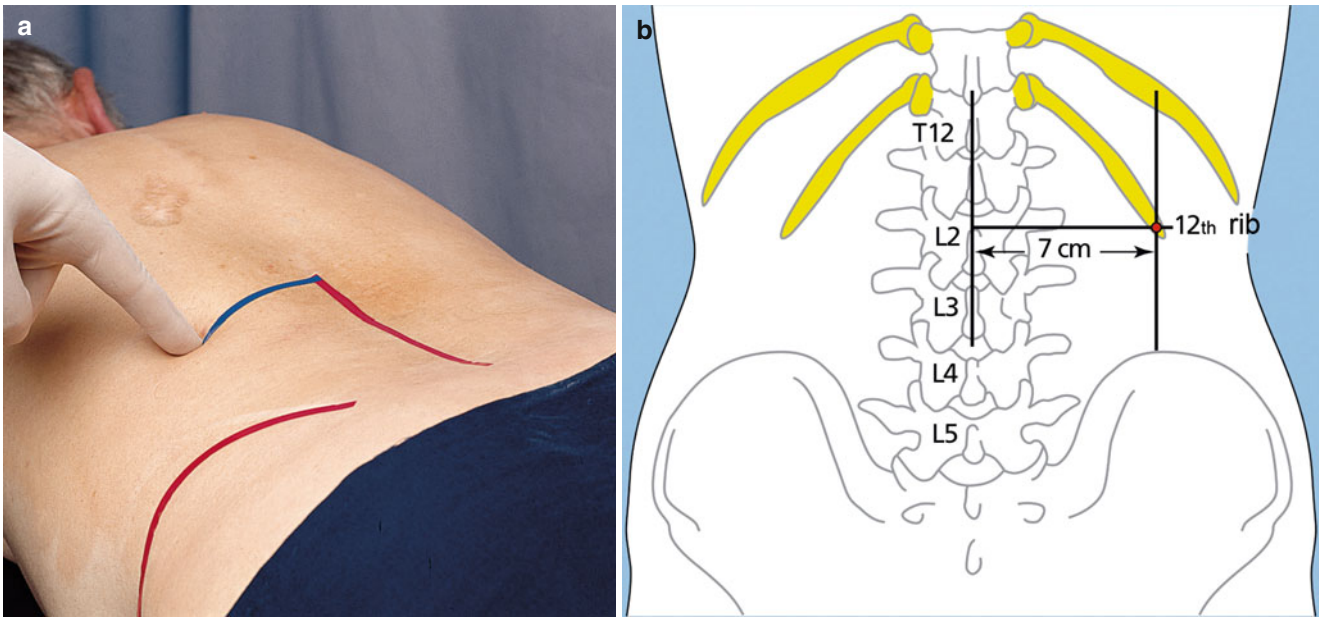


Fig. 45.4 (**a**, **b**) Localization

Injection Technique

1. The injection needle is introduced in the direction of the intended vertebra, ca. four fingerbreadths (7 cm) lateral to the midline and at an angle of ca. 30–40° to the skin surface and slightly cranially (Fig. 45.5).
2. Locating the transverse process at a depth of ca. 3–5 cm is helpful. The depth of the transverse process is marked (Fig. 45.6a, b).
3. The needle is withdrawn to lie subcutaneously.
4. The needle is then reintroduced slightly more steeply, with stepwise correction of the direction cranially or caudally (to avoid the transverse process) and medially (to obtain contact with the vertebral periosteum).
5. After contact with the periosteum, the needle is rotated 180° so that the bevel is directed toward the vertebra and can slide off the bony edge. After sliding off, the needle is further advanced by 1–2 cm (Fig. 45.7a–c).

The lumbar sympathetic trunk lies about twice as deep as the distance between the skin and the transverse process (Fig. 45.8).

The distance from the transverse process and the ganglia of the lumbar sympathetic trunk is ca. 3.8–5 cm and is relatively constant.

The distance from the skin to the transverse process depends on the anatomy and is rather more variable.

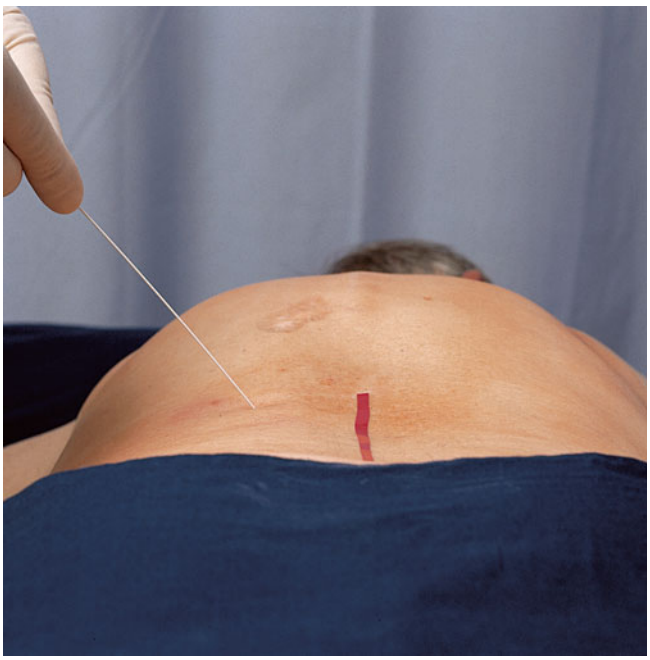


Fig. 45.5 Introducing the injection needle (Reproduced with permission from Danilo Jankovic)

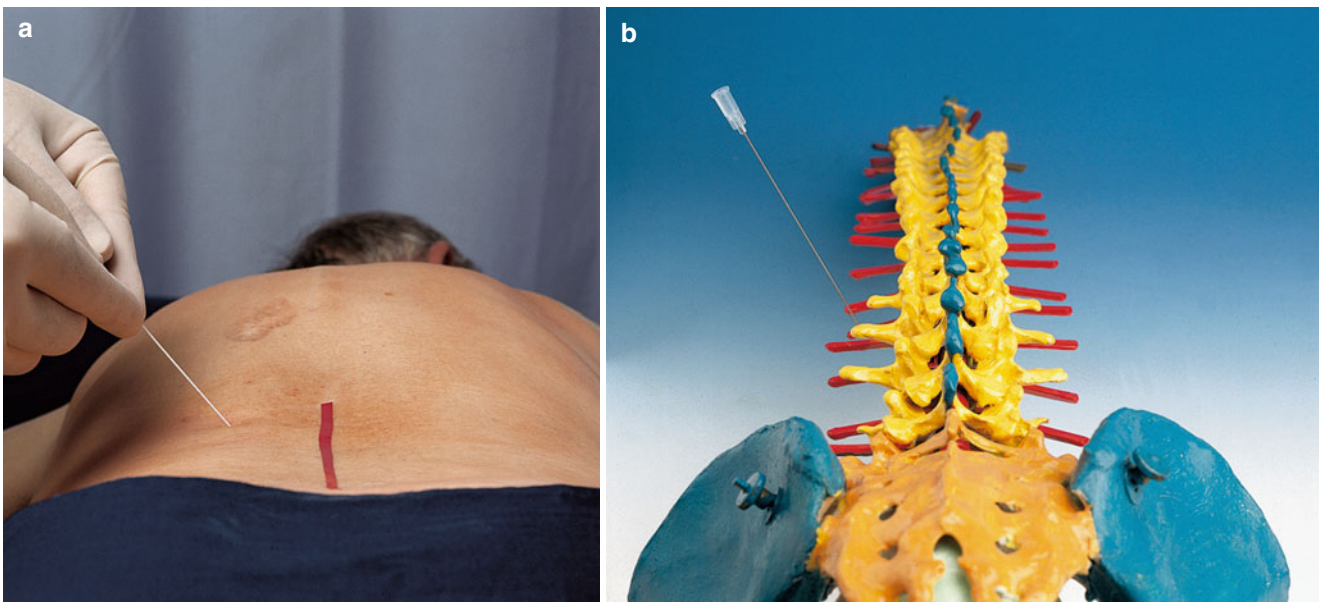


Fig. 45.6 (**a**, **b**) Bone contact with the transverse process: in a patient (**a**) and in the skeleton (**b**) (Reproduced with permission from Danilo Jankovic)

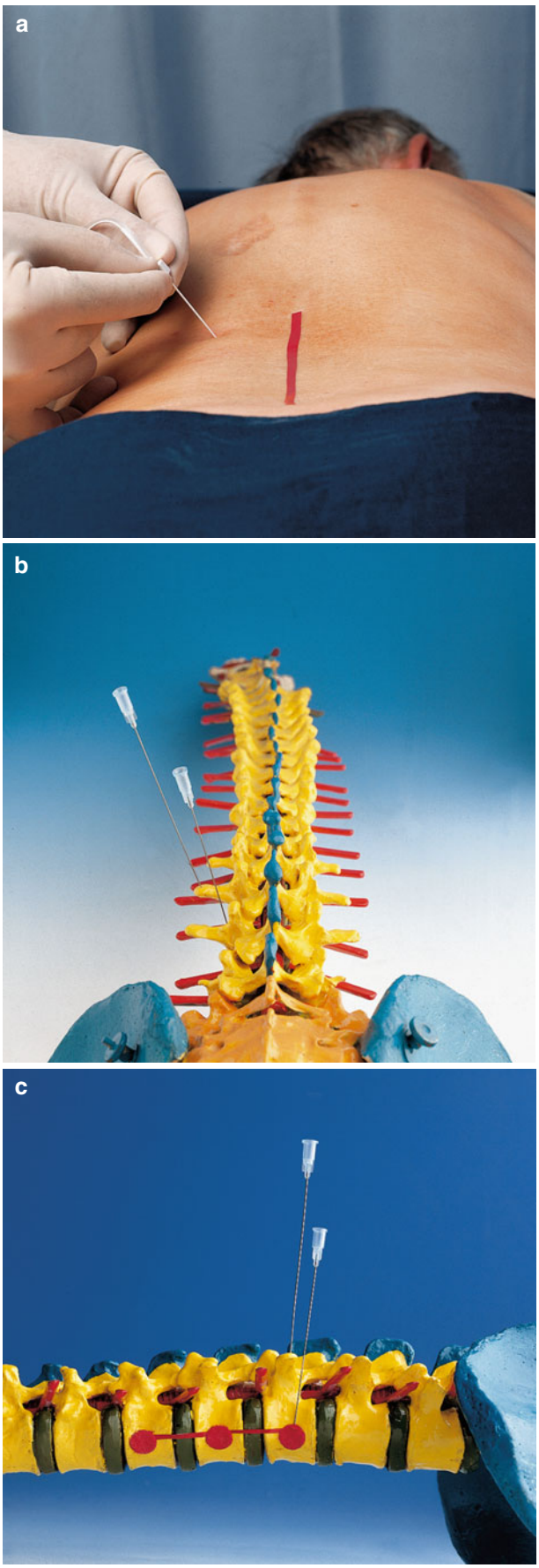


Fig. 45.7 (**a**–**c**) In a skeleton (anterior and lateral views) (Reproduced with permission from Danilo Jankovic)

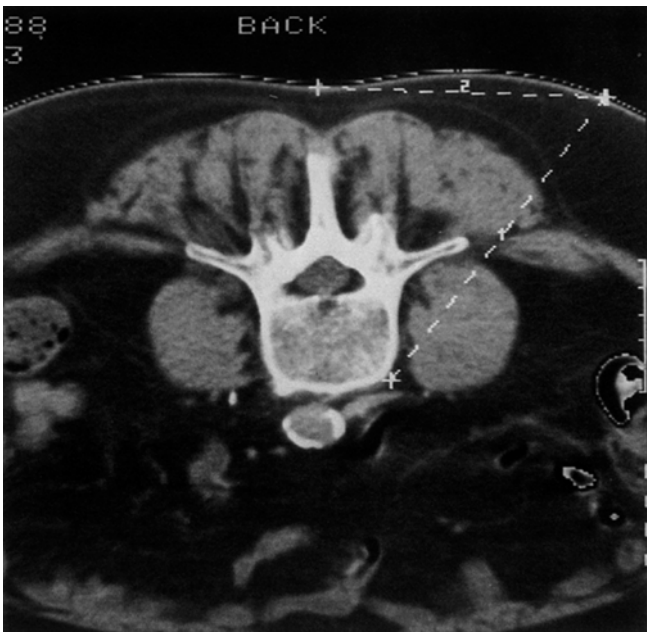


Fig. 45.8 Position of the lumbar sympathetic trunk (Reproduced with permission from Danilo Jankovic)

Confirming the Correct Needle Position

1. Radiographic control.
2. Loss-of-resistance technique with 0.9 % saline or air.

Perforation of the psoas fascia is similar to the sensation experienced when carrying out an epidural.

A false loss of resistance can occur when the needle is positioned superficially between the psoas muscle and the quadratus lumborum muscle, leading to inadvertent block of the lumbar somatic nerves, with consequent numbness of the lower extremity during the period of effect of the local anesthetic.

Injection of a neurolytic into this area without prior radiographic control of the position can have fatal consequences.

After careful aspiration testing at all levels, resistance-free incremental injection of the local anesthetic is carried out.

Neurolytic Block [9, 10]

Injection of neurolytics—45–95 % ethanol, 7 % phenol in water, or 7–10 % phenol in Conray (iothalamate meglumine)—at the lumbar sympathetic ganglia.

Prerequisite: the procedure must be carried out under radiographic guidance.

Usually, three needles are introduced at the level of L2, L3, and L4, and the neurolytic is only injected after definite confirmation of the correct needle position. After this, 1 mL of air is injected per needle, to clear any residual neurolytic from the needle.

Effects of the Block

Signs of vascular dilation in the area of the ipsilateral leg are:

- Increase in skin temperature
- Hyperthermia and anhidrosis
- Loss of the sympathogalvanic reflex
- Reduced pain or absence of pain
- No signs of sensory or motor block (assuming that the lumbar somatic nerves have not been concomitantly anesthetized)

Dosage

Diagnostic

Five milliliters local anesthetic with contrast medium—e.g., 0.5 prilocaine, 0.5 % mepivacaine, 0.5 % lidocaine.

Therapeutic

Twenty milliliters local anesthetic (single-needle technique).

Ten milliliters local anesthetic per needle (in the two-needle or three-needle technique)—e.g., 0.2–0.375 % ropivacaine, 0.25 % bupivacaine (0.25 % levobupivacaine).

Neurolytics

Three milliliters per segment.

Side Effects

1. Transient motor weakness due to block of the lumbar somatic nerves. Paresthesias during the injection are a warning signal. This undesired effect is always liable to occur and is caused by superficial injection in the area of the lumbar somatic nerves or by spread after the administration of large volumes of a local anesthetic. It is therefore necessary to monitor the patient for at least 1 h after the block
2. Fall in blood pressure due to sympathetic block.

Complications [1–5]

Severe

1. Intravascular injection (aorta, vena cava) with toxic reactions (see Chap. 1)
2. Epidural or subarachnoid injection (see Chap. 41)

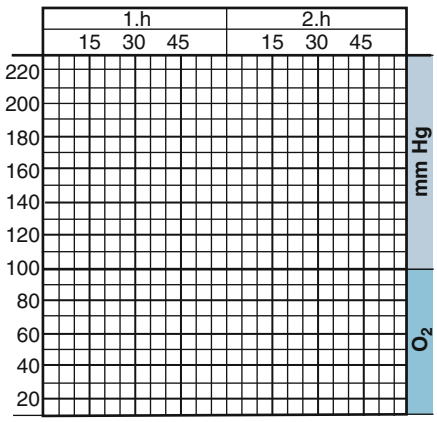
Potential

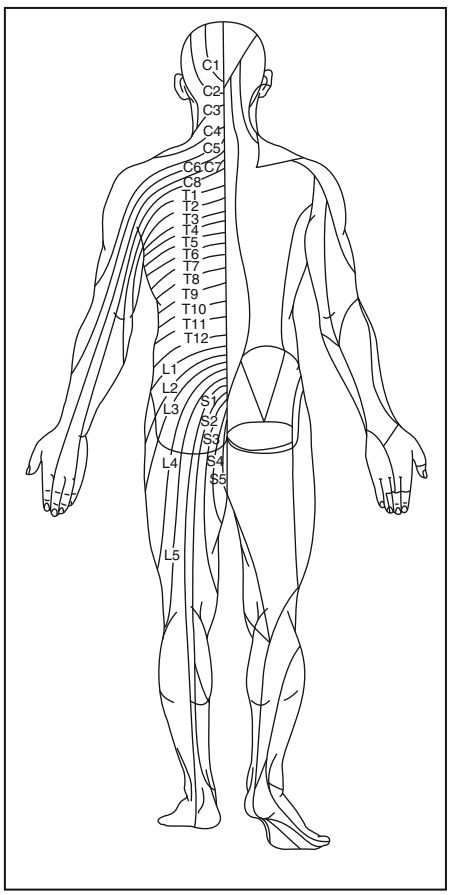
1. Retroperitoneal hemorrhage
2. Hemorrhage in the psoas area (with subsequent pain in the thigh and transient weakness in the quadriceps muscle)
3. Renal injury accompanied by hematuria
4. Back pain
5. Perforation of an intervertebral disc
6. Ejaculation disturbances (in younger patients with bilateral block) [11]

Complications of Neurolytic Block

1. Injury to the lumbar somatic nerves (neuritis, 1 %)
2. Neuralgia in the genitofemoral nerve (5–10 %)
3. Ureteral stricture

Record and checklist





Lumbar sympathetic block

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: *G* _____ *Length* _____ *cm*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed (What to do after block)*

Position: ☐ *Prone* ☐ *Lateral recumbent*

Injection level ☐ *L* _____ _____ _____

Injection technique: ☐ *X-ray image intensifier* ☐ *Loss of resistance* ☐ *CT-guided*

Injction:

Local anesthetic: _____ *mL* _____ *%*

(in incremental doses)

Addition to LA: ☐ *Yes* _____ *µg/mg* ☐ *No*

Neurolytic: _____ *mL* _____ *%*

Addition: ☐ *Yes* ☐ *No*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* _____ *°C* *left* _____ *°C*

Segments affected: *L* _____

Monitoring after block: ☐ *<1h* ☐ *>1h*

Time of discharge: _____ *Motor / sensory status checked*

Complications:

☐ *None* ☐ *Intravascular injection* ☐ *Subarachnoidlepidural*

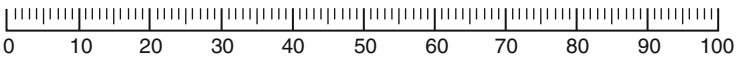
☐ *Drop in BP* ☐ *Other*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE



Special notes:

With permission from Danilo Jankovic

References

1. Breivik H, Cousins M, Löfström B. Lumbar sympathetic block. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988. p. 431–40.
2. Jankovic D. Lumbar sympathetic block. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Malden Masachusetts, Oxford, Carlton Victoria; 2004. p. 191–8.
3. Brown DL. Lumbar sympathetic block. In: Brown DL, editor. Atlas of regional anesthesia. 3rd ed. Philadelphia: Saunders/Elsevier; 2006. p. 305–9.
4. Bryce-Smith R. Injection of the lumbar sympathetic chain. Anaesthesia. 1951;6:150.
5. Hastangdi VS, Boas RV. Lumbar sympathectomy: a single needle technique. Br J Anaesth. 1985;57:285.
6. Strand L. Lumbar sympathectomy in the treatment of peripheral obliterative disease. An analysis of 167 patients. Acta Chir Scand. 1969;135:597.
7. Bergan JJ, Conn J. Sympathectomy for pain relief. Med Clin North Am. 1968;52:147.
8. Blain A, Zadeh AT, Teves ML, Bing RJ. Lumbar sympathectomy for arteriosclerosis obliterans. Surgery. 1963;53:164.
9. Cross FW, Cotton LT. Chemical lumbar sympathectomy for ischemic pain. A randomized, prospective controlled clinical trial. Am J Surg. 1985;150:341.
10. Cousins MJ, Reeve TS, Glynn CJ, Walsh JA. Neurolytic lumbar sympathetic blockade: duration of denervation and relief of rest pain. Anaesth Intensive Care. 1979;7(2):121.
11. Baxter AD, O'Kafo BA. Ejaculatory failure after chemical sympathectomy. Anesth Analg. 1984;63:770.

Chapter 46

Lumbar Facet Joint and Nerve Injection

Philip W.H. Peng , MBBS, FRCPC
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8 Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_46,

Persistent low back pain is common globally. Epidemiological surveys in the United States reported a prevalence rate of 19 % for chronic low back pain during a 12-month period. Other industrialized nations reported similar findings, with prevalence rates ranging 13–28 %. Over 13 million Americans are permanently disabled by back pain, which results in a significant economic burden to the society [1]. However, it is often difficult to reach a definitive diagnosis and provide appropriate treatment. Potential sources of low back pain include intervertebral discs, nerve roots, facet joints, and sacroiliac joints [2, 3]. The lumbar facet joint was first considered as a source for low back pain in 1911 by Goldthwaite. Based on systematic reviews and diagnostic accuracy studies, the prevalence of lumbar facet pain ranges between 25 and 45 % [4]. Facet joint pain may be managed by facet joint nerves block, intra-articular injections, and radiofrequency denervation of facet joint nerves. In the chapter, only the injection techniques will be discussed.

Anatomy

The facet joint of the lumbar spine is a diarthrodial or synovial joint containing the hyaline cartilaginous surface, synovial membrane, and a fibrous capsule. The joint space is small, with a volume of about 1 and 1.5 mL. Each facet joint is innervated by the medial branches of the dorsal rami from the same vertebral level and from the vertebral level above [5, 6]. For example, L4–5 facet joint receives dual innervations from L3 and L4 medial branches, which are typically blocked on the transverse processes of L4 and L5, respectively. The medial branches of the L1–L4 dorsal rami have a similar course (Fig. 46.1). Each medial branch crosses in a groove formed by the junction of the transverse process and SAP of the vertebral level below (e.g., L3 crosses the transverse process of L4). Here, it runs beneath the mamilloaccessory ligament before it innervates the multifidus muscle, interspinous ligament, and the periosteum of the neural arch. The L5 dorsal ramus differs from the other lumbar dorsal rami in that it itself runs along the junction of the sacral ala and superior articular process of the sacrum and gives off the medial branch only as it reaches the caudal aspect of the L5–S1 zygapophyseal joint. Histologic studies supported the lumbar facet joints as a source of pain generator as they are richly innervated with encapsulated (Ruffini-type endings, pacinian corpuscles), unencapsulated, and free nerve endings [5, 6].

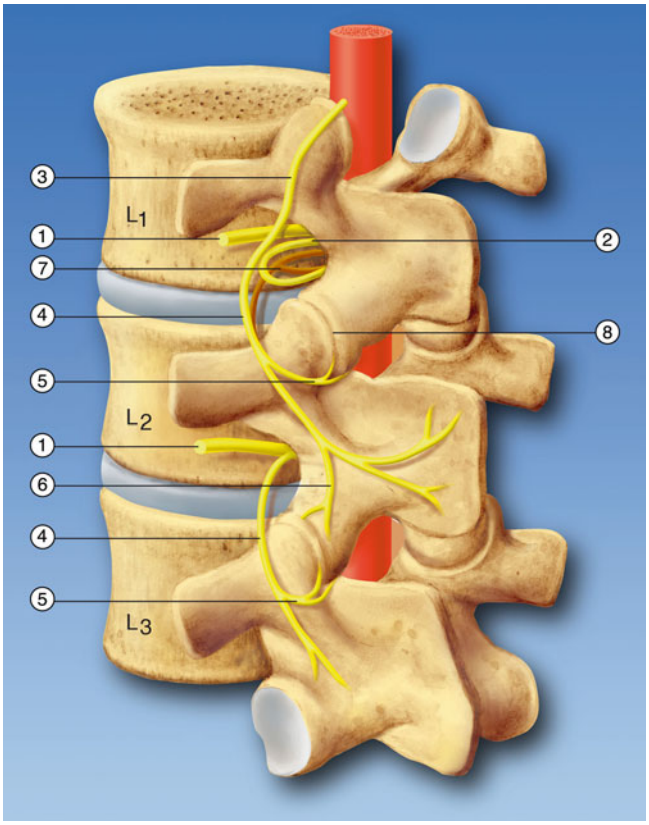


Fig. 46.1 Dual neural supply of the facet joint. Oblique parasagittal view of overlapping segmental innervation of the facet joint. (*1*) Ventral branch of spinal nerve, (*2*) dorsal branch of spinal nerve, (*3*) ascendant branch of dorsal ramus, (*4*) medial branch of dorsal ramus, (*5*) distal branch of medial ramus to facet joint, (*6*) proximal branch of medial ramus to facet joint, (*7*) posterior ramus (sinuvertebral nerve of Luschka), (*8*) facet joint (Reproduced with permission of Danilo Jankovic)

Technique

The popular technique for both facet nerve and joint block is by fluoroscopic guidance, but ultrasound-guided techniques have been described and validated [7–10].

Fluoroscopic Guided Facet Nerve Block

The patient is placed in prone position with a pillow supporting the pelvis for comfort and to obliterate the lumbar lordosis. The techniques for the blockade of L1–L4 medial branches are the same [11]. The first step is to count the appropriate level. Once the level is counted, the target vertebral level is squared, that is, the anterior and posterior silhouettes of the lower border of vertebra are at the same level. The C-arm is then rotated ipsilaterally to the side of injection to obtain an oblique view. In this view, the outline of the "scotty dog" is clearly evident (Fig. 46.2). The target is the junction of the superior border of transverse process with the superior articular process. Alternatively, another end point has been described, which is the midpoint between the mamilloaccessory notch and the target point just described in Fig. 46.1 (Fig. 46.3). This target point has been shown to minimize the inadvertent spread of injectate into the intervertebral foramen or epidural space. For the L5 dorsal rami block, the target point is the ala of the sacrum at the base of the superior articular process of sacrum in an anteroposterior view (Fig. 46.4).

A 22-gauge spinal needle is inserted following local anesthetic infiltration of the skin. Once the bone is contacted at the target point, a small amount of contrast (0.2 mL) is injected to detect possible venous uptake. If there is no venous uptake, 0.5 mL of local anesthetic is injected.

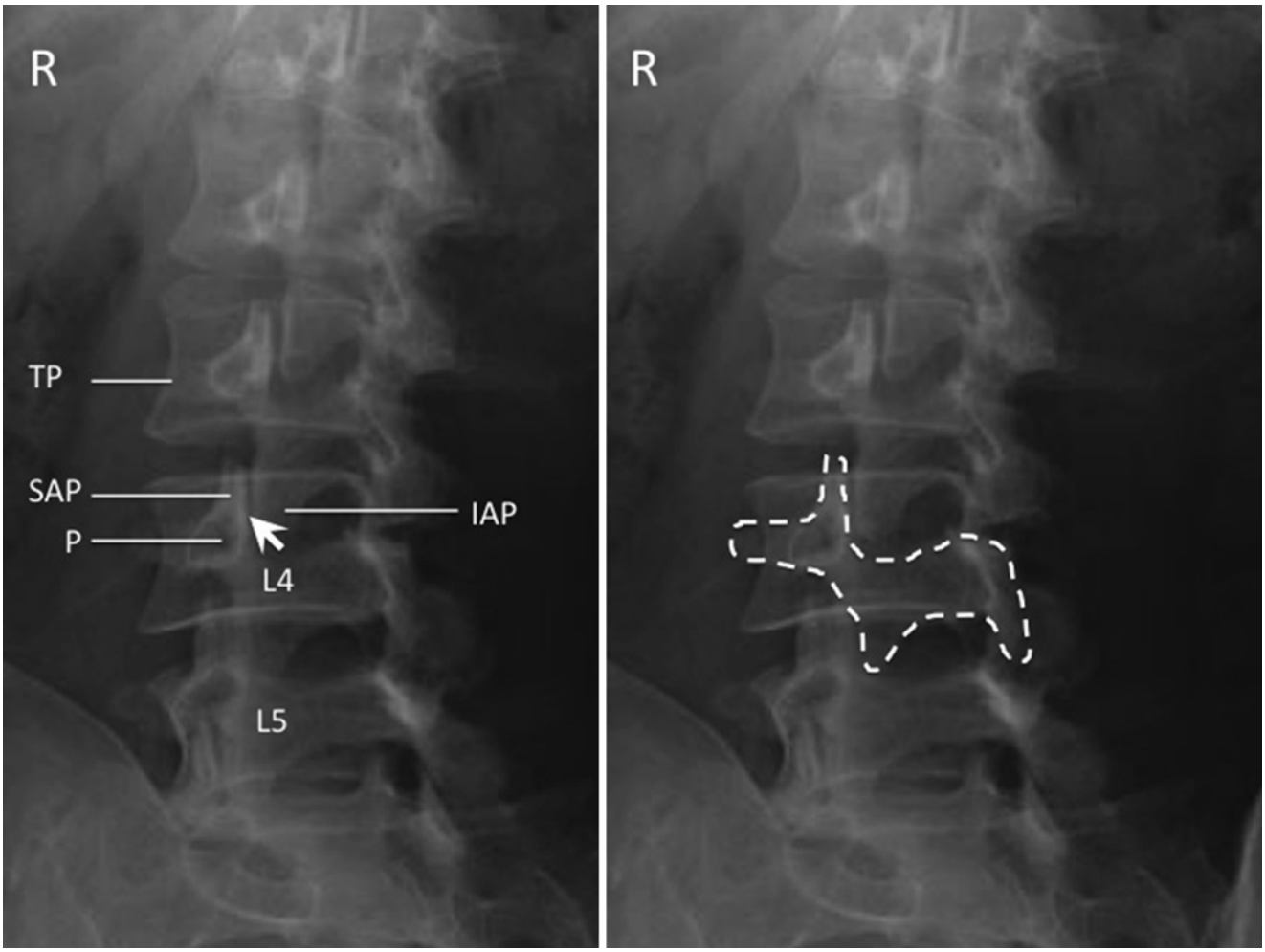


Fig. 46.2 Oblique radiograph of the lumbar spine. The *left figure* shows the individual structures: *SAP* superior articular process, *IAP* inferior articular process, *P* pedicle, *TP* transverse process; facet joint is indicated by the *arrow*. The *right figure* shows the "scotty dog" appearance of the same structure (Reproduced with permission from Philip Peng Educational Series)

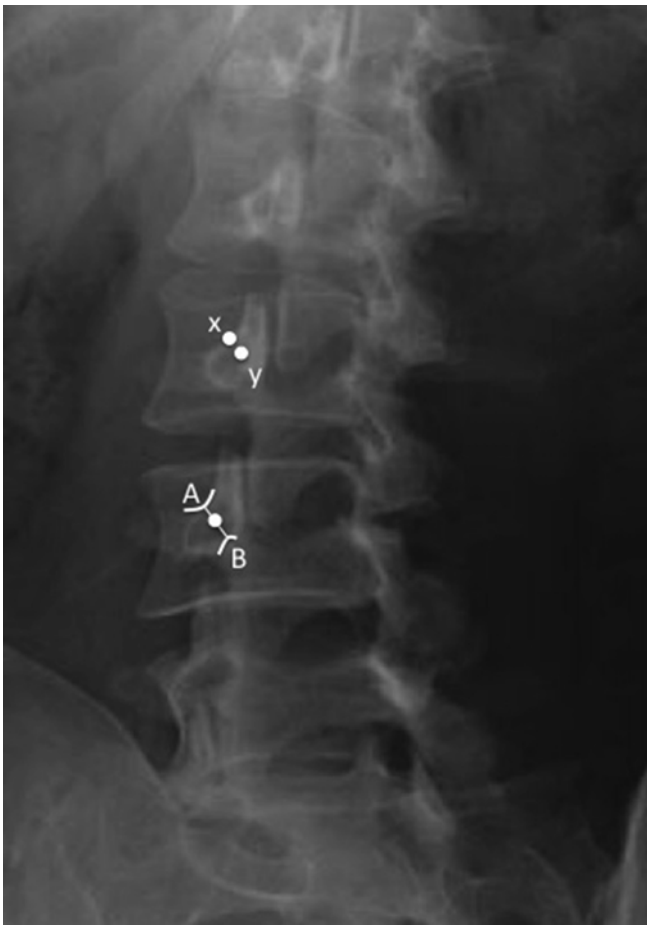


Fig. 46.3 The radiograph shows the target points (*white dots*) for facet medial branch injection. *A* The junction of the superior border of transverse process with the superior articular process. *B* The mamilloaccessory notch. *x* The conventional target which is the junction of the superior border of transverse process with the superior articular process. *Y* The midpoint between A and B (Reproduced with permission from Philip Peng Educational Series)

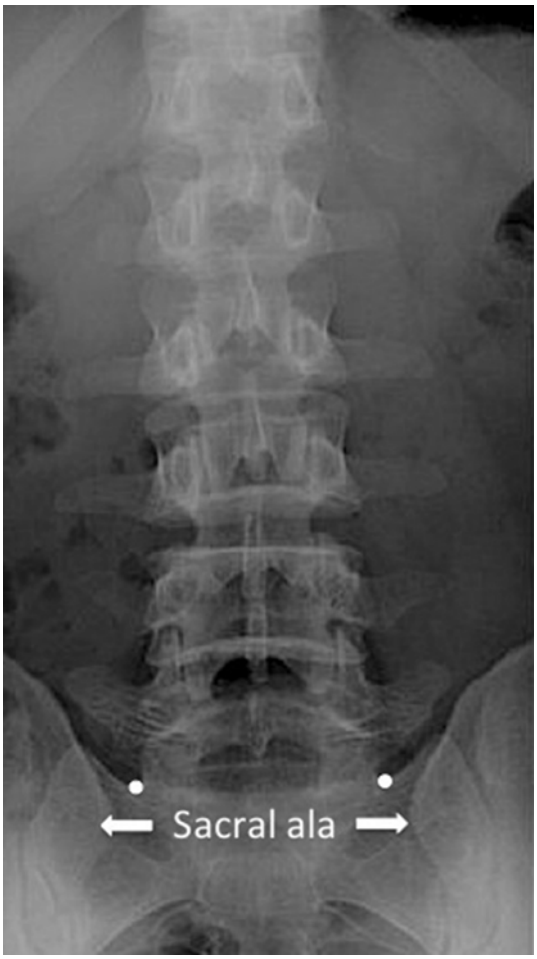


Fig. 46.4 The target points for L5 dorsal rami was indicated by the white dots, which are the ala of the sacrum at the base of the superior articular process of sacrum (Reproduced with permission from Philip Peng Educational Series)

Fluoroscopic Guided Facet Joint Injection

The patient is positioned as for the facet medial branch block. Oblique view is obtained until the joint space formed between the articular processes is optimally seen (Fig. 46.2). A 22-gauge spinal needle is inserted with coaxial technique. A small amount of contrast (0.2 mL) is injected to confirm the intra-articular placement. A volume of 0.5–1 mL injectate (20 mg methylprednisolone or triamcinolone) is sufficient. In patient with advanced disease, the joint space may be further obliterated. In that case, periarticular injection may be performed.

Ultrasound-Guided Facet Medial Branch Injection

The position of the patient is the same as the fluoroscopic guided technique. The target point is the junction between the superior articular process and transverse process. A curvilinear ultrasound probe (2–5MHz) is used to obtain a parasagittal oblique view. In this scan, both the sacrum and lamina can be seen and used for counting (Fig. 46.5). The probe is then moved laterally to visualize the transverse process (Fig. 46.6). Once an appropriate level is identified, the probe is rotated 90° to obtain a short axis view of the spine showing the transverse process and the corresponding superior articular process. The probe is then moved in cephalad direction until the cephalad aspect of the transverse process disappears. When this part of the transverse process reappears on sliding back the probe in caudal direction, the target is defined (Fig. 46.7). A 22-gauge spinal needle is inserted in-plane from lateral to medial to hit the bony target. Once this is achieved, the probe is turned 90° to demonstrate the needle is at the cephalic part of the transverse process (Fig. 46.8). The L5 dorsal ramus is usually difficult to assess because of the ilium.

Ultrasound-Guided Facet Joint Block

The initial scanning technique is similar to that of facet medial branch. Once the short axis scan of the spine shows the transverse process and the superior articular process, the final position of the probe is tilted in cephalad and caudal direction until a joint space opening is clearly seen between the superior and inferior articular process (Fig. 46.9). A 22-gauge spinal needle is inserted in-plane from lateral to medial to hit the bony target. In patient with limited joint space, advancing the needle into the joint space can be difficult, and a periarticular injection is reasonable.

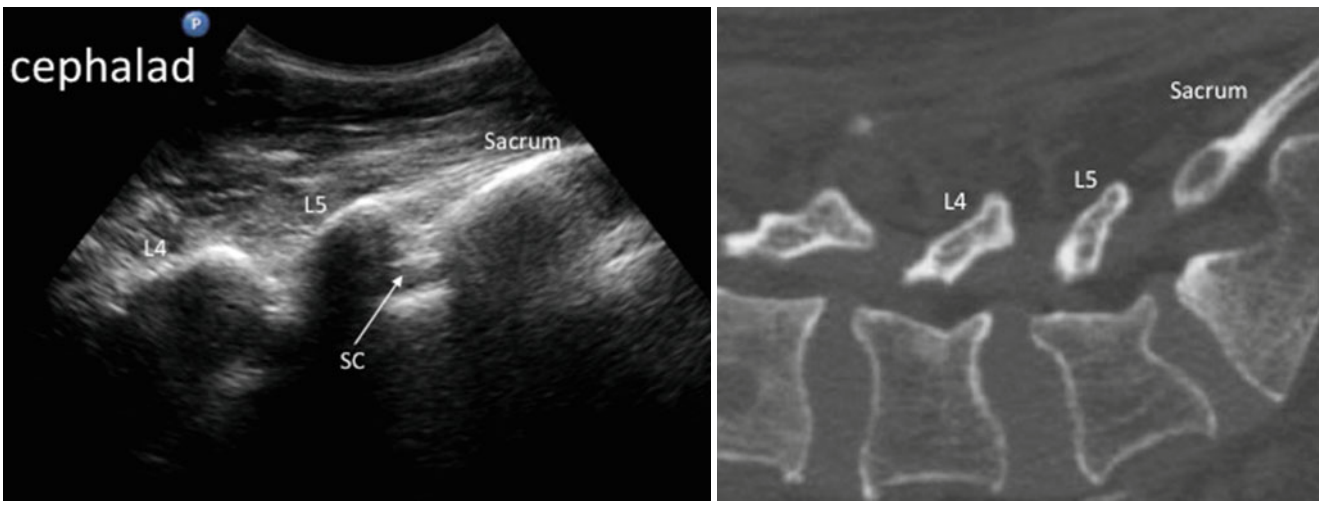


Fig. 46.5 Parasagittal oblique ultrasound view of the spine at the lumbosacral junction on the left and the corresponding magnetic resonance image on the right. *SC* spinal canal (Reproduced with permission from Philip Peng Educational Series)

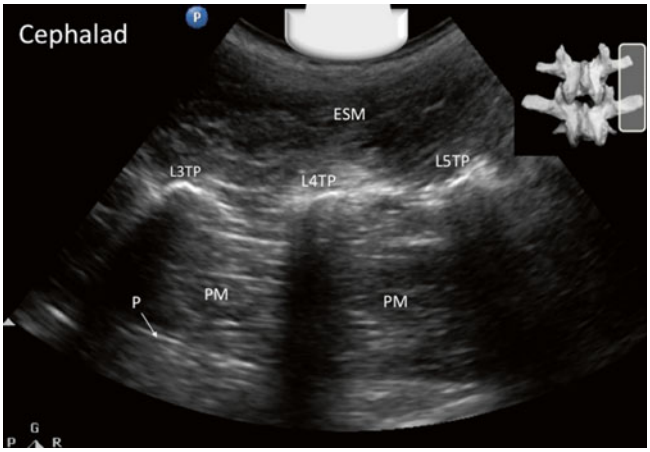


Fig. 46.6 Parasagittal ultrasound scan of the transverse process. The *insert* showed the position of the ultrasound probe. *ESM* erector spinae muscle, *PM* psoas muscle, *L3TP* transverse process of L3 (Reproduced with permission from Philip Peng Educational Series)

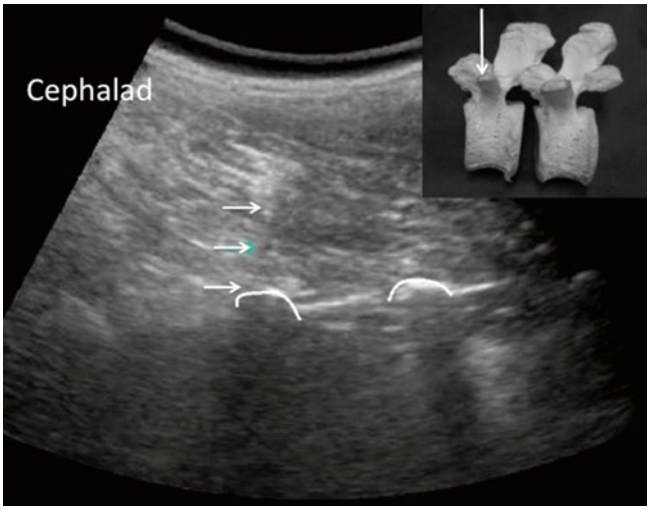


Fig. 46.8 Parasagittal of the transverse process showed the needle (indicated by the *arrows*), which was out-of-plane to this diagram. The transverse process was indicated by the line, and the *insert* showed the position of the needle (Reproduced with permission from Philip Peng Educational Series)

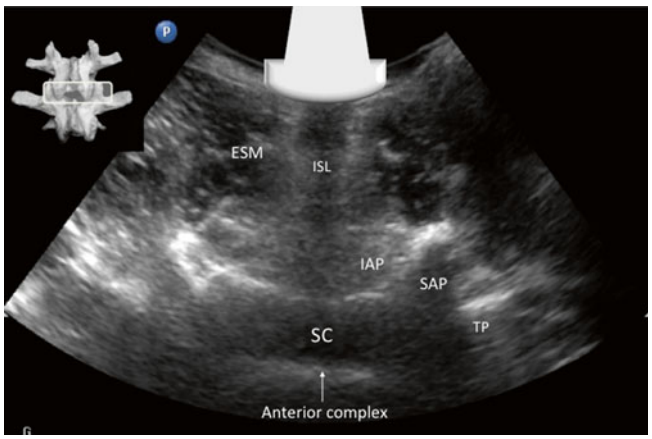


Fig. 46.7 Transverse scan of the spine at the interlaminar space. The insert showed position of the ultrasound probe. *ISL* interspinous ligament, *ESM* erector spinae muscle, *SAP* superior articular process, *IAP* inferior articular process, *TP* transverse process, *SC* spinal canal (Reproduced with permission from Philip Peng Educational Series)

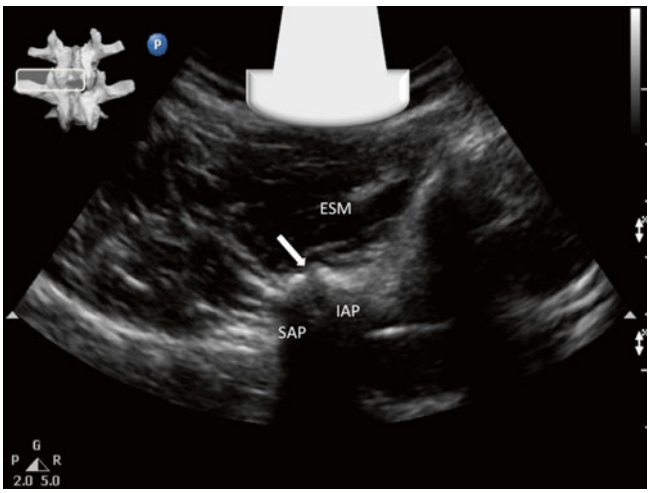


Fig. 46.9 Sonograph showed the facet joint indicated by the *bold arrow*. *ESM* erector spinae muscle, *SAP* superior articular process, *IAP* inferior articular process. The *insert* showed the position of the probe (Reproduced with permission from Philip Peng Educational Series)

Use of Facet Block in Clinical Practice

Both fluoroscopy and computer tomography (CT) scan are the gold standard for the guidance of facet nerve or joint block. Although the application of ultrasound has been validated both in clinical and preclinical setting, the accuracy is only approximately 90 %. In addition, those validated studies were performed on low body mass index (BMI) patient [8–10]. The accuracy dropped to 60 % when the BMI is above 30 [12]. In addition, the utility of this in L5 dorsal rami is not well described. The author reserves the use of ultrasound-guided facet block as an alternative to patients with low BMI only.

In clinical practice, it is generally accepted that diagnostic facet blocks are the most reliable means for diagnosing facet joints as pain generators, as historic, physical, and radiologic examination findings can reliably predict response to diagnostic facet nerve or joint blocks [4, 5]. Facet nerve and joint blocks are often described as "equivalent," as neither of these approaches have been shown to be superior [13]. In addition, both medial branch and intra-articular blocks are associated with significant false-positive and false-negative rates [13]. A positive response refers to 50 % or greater pain reduction lasting for the duration of action of the local anesthetic (e.g., >30 min with lidocaine and 3 h with bupivacaine). Using a pain relief cutoff over 50 % did not result in any significant improvement in RF outcomes [14]. Double, comparative blocks are associated with a significant false-negative rate and have not been shown to be cost-effective [13]. Thus, a single block is usually adopted for the diagnosis of facet joint pain.

References

1. Turk DC, Theodore BR. Epidemiology and economics of chronic and recurrent pain. In: Lynch ME, Craig K, Peng PWH, editors. Clinical pain management: a practical guide. Oxford: Wiley-Blackwell; 2011. p. 6–13.
2. Hancock MJ, Maher CG, Latimer J, et al. Systematic review of tests to identify the disc, SIJ or facet joint as the source of low back pain. Eur Spine J. 2007;16:1539–50.
3. Schwarzer AC, Aprill CN, Derby R, Fortin J, Kine G, Bogduk N. Clinical features of patients with pain stemming from the lumbar zygapophysial joints. Is the lumbar facet syndrome a clinical entity? Spine. 1994;19:1132–7.
4. Falco FJE, Manchikanti L, Datta S, et al. An update of the effectiveness of therapeutic lumbar facet joint interventions. Pain Physician. 2012;15:E909–53.
5. Cohen SP, Raja SN. Pathogenesis, diagnosis, and treatment of lumbar zygapophysial (facet) joint pain. Anesthesiology. 2007;106:591–614.
6. Narouze S, Peng PWH. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures. Part II: axial structures. Reg Anesth Pain Med. 2010;35:386–96.
7. Gofeld M, Bristow SJ, Chiu S. Ultrasound-guided injection of lumbar zygapophyseal joints. An anatomic study with fluoroscopy validation. Reg Anesth Pain Med. 2012;37:228–31.
8. Greher M, Scharbert G, Kamolz LP, Beck H, Gustorff B, Kirchmair L, et al. Ultrasound-guided lumbar facet nerve block: a sonoanatomic study of a new methodologic approach. Anesthesiology. 2004;100:1242–8.
9. Shim JK, Moon JC, Yoon KB, Kim WO, Yoon DM. Ultrasound-guided lumbar medial-branch block: a clinical study with fluoroscopy control. Reg Anesth Pain Med. 2006;31:451–4.
10. Greher M, Kirchmair L, Enna B, Kovacs P, Gustorff B, Kapral S, et al. Ultrasound-guided lumbar facet nerve block: accuracy of a new technique confirmed by computed tomography. Anesthesiology. 2004;101:1195–200.
11. Bogduk N. International Spinal Injection Society guidelines for the performance of spinal injection procedures. Part 1: zygapophysial joint blocks. Clin J Pain. 1997;13:285–302.
12. Rauch S, Kasuya Y, Turan A, Neamtu A, Vinayakan A, Sessler DI. Ultrasound-guided lumbar medial branch block in obese patients. A fluoroscopically confirmed clinical feasibility study. Reg Anesth Pain Med. 2009;34:340–2.
13. van Kleef M, Vanelderen P, Cohen SP, Lataster A, Van Zundert J, Mekhail N. Evidence-based interventional pain medicine according to clinical diagnoses. Pain originating from the lumbar facet joints. Pain Pract. 2010;10:459–69.
14. Cohen SP, Strassels SA, Kurihara C, et al. Establishing an optimal "cutoff" threshold for diagnostic lumbar facet blocks. A prospective correlational study. Clin J Pain. 2013;29:382–91.

Chapter 47

Lumbar Percutaneous Facet Denervation

Robert Jan Stolker, MD, PhD (✉)
Department of Anesthesiology, Erasmus MC, P.O. Box 2040, Rotterdam 3000 CA, The Netherlands
e-mail: r.stolker@erasmusmc.nl

Hans van Suijlekom • André van Zundert, MD, PhD, FRCA, EDRA, FANZA
Department of Anesthesiology, ICU & Pain Therapy, Catharina-ziekenhuis, P.O. Box 1350, Eindhoven 5602 ZA, The Netherlands

Gerbrand Groen
Division of Perioperative Medicine, Department of Anesthesiology and Emergency Care, University Medical Centre Utrecht, P.O Box 85500, Utrecht 3508 TA, The Netherlands

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_47, © Springer International Publishing Switzerland 2015

Anatomy

The goal of a lumbar percutaneous facet denervation procedure is to block the medial branch of the dorsal ramus of the spinal nerve (Fig. 47.1). When the dorsal and ventral roots come together, the spinal nerve is formed, which splits up into a ventral and a dorsal ramus [1]. This dorsal ramus divides into a medial and a lateral ramus (=branch). The dorsal or posterior ramus supplies the so-called dorsal compartment of the back, which consists of structures of the back situated behind the intervertebral foramen. This dorsal compartment contains muscles, ligaments, blood vessels, and the facet joints. The facet or zygapophyseal joints are joints between the inferior articular process of a vertebra and the superior articular process of the vertebra below. Lumbar facet joints are small joints located in pairs on the back of the spine, providing stability and guide motion in the lower back. These joints at the dorsal side of the spine form together with the intervertebral disc at the ventral side, the three moving parts of a motion segment.

The facet joints are innervated by medial branches of the dorsal ramus from the corresponding level and by the medial branch of the dorsal ramus one level above. So the facet joint L5–S1 is innervated by the dorsal rami L4 and L5.

The medial branch innervates skin ligaments and muscles, whereas at level L5 the medial branch mostly consists of sensory fibers. The lateral branch innervates muscles. The medial branches have to be blocked in the gutter where the superior articular process and the transverse process come together (Fig. 47.1).

The anatomy of the back and the innervation of the posterior compartment are described elsewhere more extensively (see Chaps. 49 and 59).

If the joints become painful, they may cause pain in the low back, abdomen, buttocks, groins, or legs. The lumbar zygapophyseal joints are a potential source of low back pain and referred leg pain.

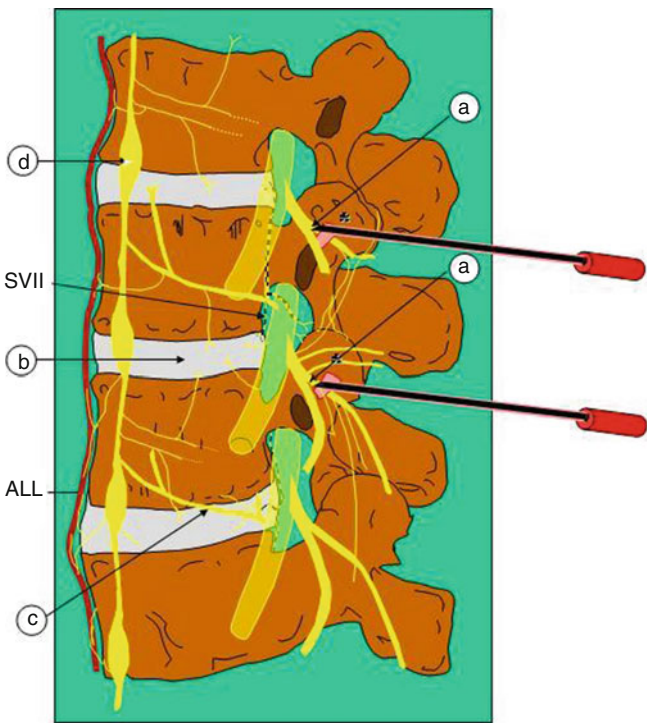


Fig. 47.1 Anatomic overview of lumbar spine – position of needles at the medial branch of the dorsal ramus. (*a*) medial branch dorsal ramus. (*b*) intervertebral disc. (*c*) communicating ramus. (d) sympathetic chain. *svn* sinuvertebral nerve, *ALL* anterior longitudinal ligament. * mamillo-accessory ligament

Introduction: Technique

The technique of lumbar facet denervation was first described by Shealy in 1974. Later several authors modified this technique. Anatomical studies revealed more exactly the place where the block had to be performed. In this chapter, the most common technique will be described using fluoroscopy and bony landmarks. The procedure can be performed in an outpatient setting with the patient awake. Most patients are sufficiently cooperative. Administration of a sedative is rarely necessary. As the innervation of the joint is always from two levels, the procedure must always be done at two levels, in case of pain from two adjacent joints at three levels. The procedure can be performed uni- or bilaterally.

Indications

In the literature there is doubt about the existence of a specific facet syndrome, but in Table 47.1 some signs and symptoms are listed, suggesting pain originating from facet joints.

Uni- or bilateral pain lasting longer than 3–6 months not reacting on physical therapy and other conservative management could be an indication for percutaneous facet denervation (PFD). In the absence of a specific facet syndrome, most authors advocate to perform a test block prior to a PFD. This test block is a medial branch block or an intra-articular block with local anesthetics. Some authors adjust steroids in order to achieve a longer lasting effect of the test procedure. They believe that the addition of steroids will further reduce any inflammation that may exist within the joint.

Radiological findings per se, such as facet arthritis, are no indication for a PFD (Tables 47.2 and 47.3).

This procedure should be performed by an experienced anesthesiologist or under his/her supervision.

Table 47.1 Possible signs and symptoms of facet pain in the lumbar region

Back pain with or without irradiation to the groin, buttock, leg, sometimes abdomen
Pain aggravated by rest in any posture (standing, sitting, laying in bed)
Pain relieved by movement
Radiculopathy is absent (no neurological deficit)
Pain should not radiate below the knee
Morning stiffness
Awakening by turning in bed
Pain on anteflexion and/or rotation of the spine
Paravertebral tenderness

Table 47.2 Contraindications

Sensory loss
Lack of cooperativeness
Bleeding disorders or use of anticoagulants
Signs of local infection
Signs of local malignancy
Presence of osteosynthesis material
Allergy to local anesthetics

Table 47.3 Procedure

Material: two to four 10 cm 22G Radiofrequency needles with 5 mm blank tip (e.g., Radionics)
Local infiltration (e.g., lidocaine 2 %)
Thermocouple 10 cm (e.g., Radionics)
Radiofrequency lesion generator
Ground plate
Connecting wires

The patient lays in the prone position on a special table allowing the use of a fluoroscope. The fluoroscope is directed in anteroposterior view. Sometimes a 5–10° oblique view is needed to obtain more reliable pictures. The insertion place is marked at the skin by using a plastic calliper with an iron ring, which has to be placed at the site of the projection of the junction of the transverse and superior articular process. After disinfection of the skin, 1 ml lidocaine 1–2 % is (sub) cutaneously injected, the needle is inserted, and its position is controlled by fluoroscopic viewing. The direction of the needle is, if necessary, corrected and the needle is advanced until bone contact is made. After correcting the needle position slightly, the tip of the needle is advanced just a little more, keeping bone contact. The depth of the needle is controlled by a fluoroscopic lateral view. The projection of the needle may not be ventral to the dorsal rim of the intervertebral foramen (Figs. 47.2, 47.3, and 47.4).

After obtaining the correct anatomical position, the stylet of the needle is removed, and a thermocouple electrode inserted. The electrode has to be connected to the lesion generator. The ground plate is connected. Stimulation with a 50 Hz current is performed and the patient is asked to warn if a tingling feeling laterocaudal of the site of the needle and/or at the buttock will be felt. Stimulation threshold must be less than 1 V. The lower the threshold, the better the outcome, as the value of the threshold reflects the distance to the nerve. A tingling feeling in the leg refers to a too anterior position of the needle (too close to the spinal nerve), which has to be withdrawn. The same stimulation procedure can be done with a 2 Hz current. As the medial branch is a mixed nerve (possible exception L5), a response at the same threshold (or 0.1 V higher) is expected. Local contractions will occur. Contractions of the leg are a sign of a too anterior position, close to the spinal nerve. A threshold for motor response of the leg at 2 V or less must never be accepted.

If these thresholds are met, ½ml lidocaine must be injected after careful aspiration in each needle in order to anesthetize the site of the thermolesion.

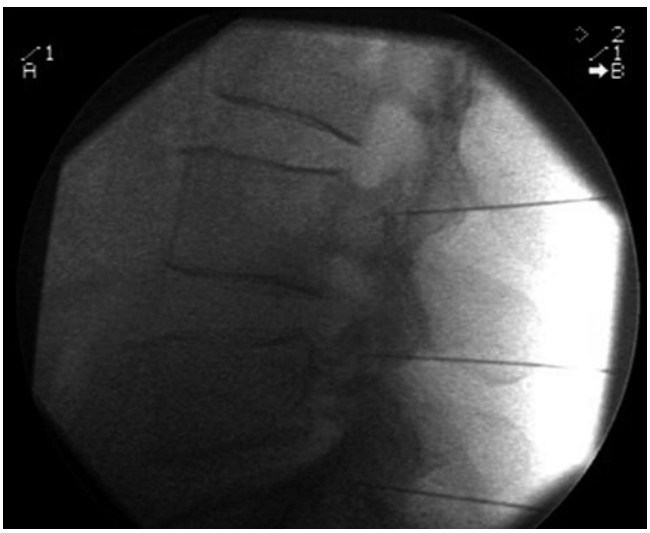


Fig. 47.2 Lumbar facet denervation – X-ray – lateral view

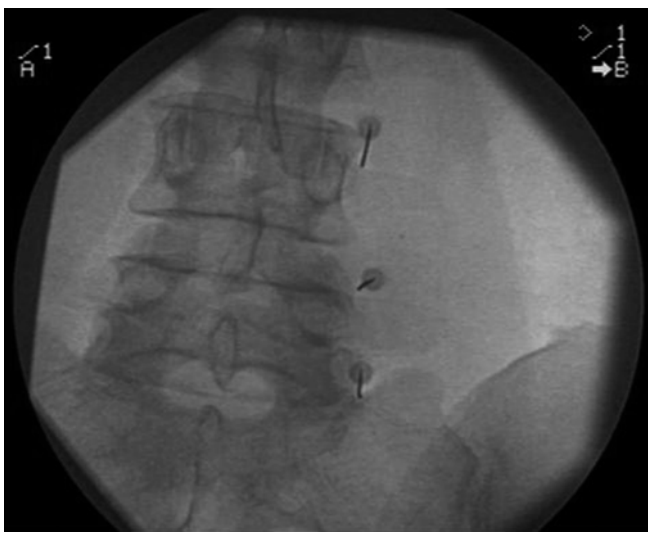


Fig. 47.3 Lumbar facet denervation – X-ray – axial view

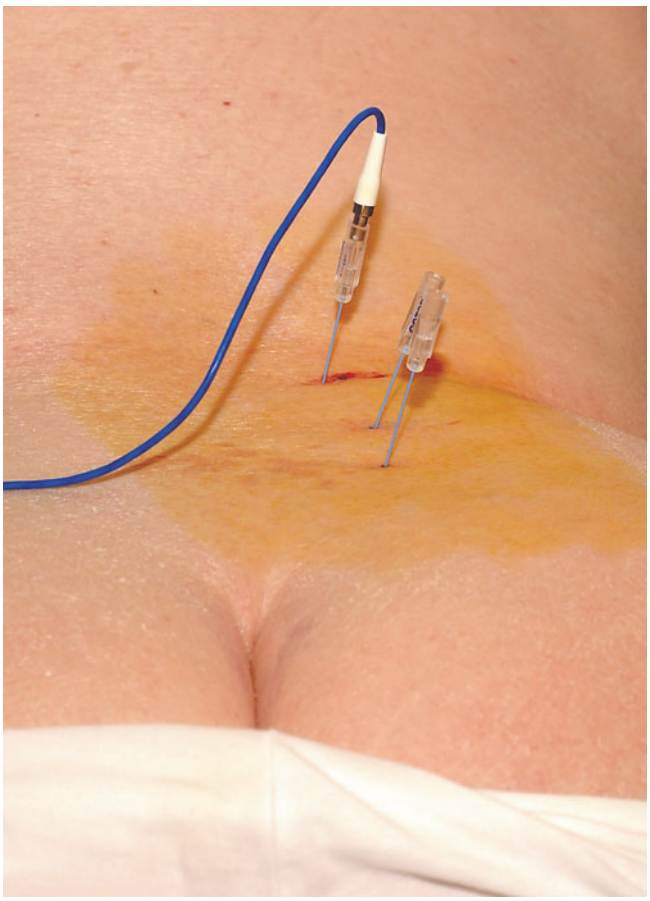


Fig. 47.4 Lumbar facet denervation – positioning needles L4–L5–S1

After waiting for 1–2 min the radiofrequency lesion at 80 °C for 60 s can be made.

In the thoracic spine, a similar technique can be used. The position of the needle is somewhat more lateral compared to the lumbar procedure [2].

Results

A few prospective placebo-controlled studies [3–5] have been published. Their long-term results vary from 30 to 50 %. Inclusion criteria, the use of medial branch, or intra-articular blocks with local anesthetics as selection for the procedure are still subject of discussion. Some authors use single test blocks, others double blocks, whereas others perform no blocks at all as selection for the PFD. Their patient selection is fully based on clinical signs and symptoms.

Furthermore, technical differences, such as exact positioning, size of the electrodes [6], used temperature and exposure time, may be the cause of a different outcome. Finally the criteria for success are not equal in the published studies (VAS-score, use of medication, inability scales, global perceived effect, or combinations of these).

Complications and Side Effects

The main complication is postoperative pain. Not only pain of the needle lasting for a few days but also serious burning pain may occur in 20 % of the patients. Patients should be informed that they can experience pain 7–9 days after the procedure, which can last for a couple of weeks. This pain will always subside within 4–6 weeks. Bruising and hematomas at the back occur in a low percentage and are rarely serious. Neurological damage is very rare if the positioning and stimulation have been performed carefully. The procedure has three preventive moments avoiding neurological deficits: (1) the lateral view (although not completely reliable); (2) electrostimulation; (3) the injection of ½ ml lidocaine is too small to anesthetize a spinal nerve when the electrode is accidentally placed too close. Sometimes a leg is numb and/or weak after the procedure due to overflow of the local anesthetic. This will subside spontaneously within a few hours.

Instructions to Patients

Patients have to give their informed consent. If patients are using acetylsalicyl acid or clopidogrel, they are asked to stop 5 days before. In case of the use of coumarin derivates, the intake of these drugs should be interrupted and the INR controlled before the procedure. An INR of 1.8 or less is appropriate.

It is recommended to let the patient eat a light meal before the procedure. Diabetic patients should not change their habits. Intake of food is free after the procedure. Because of possible temporary leg numbness and/or weakness, it is recommended that the patients stay at least 1 h following the procedure. Sometimes discharge must be postponed if the patient is unable to walk properly. In all cases the patient has to be driven home accompanied by a competent adult and they are not allowed to participate in the traffic (e.g. drive a car) during the day of operation for legal and liability reasons.

Conclusions

A selected group of patients with disabling low back pain, not relieved by conservative measures, attributed to the facet joints can be successfully treated by facet joint denervation. Adequate patient selection and the use of an accurate lesioning technique under fluoroscopy are essential elements to get good long-term results following radiofrequency denervation. This minimally invasive technique however is appealing given the rather easy accessibility of the medial branch of the dorsal ramus and the reassurance of virtually nonexistent complications.

References

1. Bogduk N, Wilson AS, Tynan W. The human dorsal rami. J Anat. 1982;134:383–97.
2. Stolker RJ, Vervest ACM, Groen GJ. Percutaneous facet denervation in chronic thoracic spinal pain. Acta Neurochir (Wien). 1993;122:82–90.
3. van Kleef M, Barendse GA, Kessels A, Voets HM, Weber WE, de Lange S. Randomized trial of radiofrequency lumbar facet denervation for chronic low back pain. Spine. 1999;24:1937–42.
4. van Wijk RMAW, Geurts JWM, Wynne HJ, Hammink E, Buskens E, Lousberg R, Knape JTA, Groen GJ. Radiofrequency facet denervation in the treatment of chronic low back pain: a randomized, double-blind, placebo-controlled trial. Clin J Pain. 2005;21:335–44.
5. Leclaire R, Fortin L, Lambert R, Bergeron YM, Rossignol M. Radiofrequency facet joint denervation in the treatment of low back pain: a placebo-controlled clinical trial to assess efficacy. Spine. 2001;26:1411–6.
6. Buijs EJ, van Wijk RMAW, Geurts JWM, Weeseman RR, Stolker RJ, Groen GJ. Radiofrequency lumbar facet denervation: a comparative study to the reproducibility of lesion size following two current radiofrequency techniques. Reg Anesth Pain Med. 2004;29:400–7.

Chapter 48

Sacroiliac Joint

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_48,

Pain from sacroiliac joint (SIJ) is an underappreciated source of chronic mechanical low back pain. Depending on the sample population and diagnostic criteria, SIJ accounts for an estimated 10–27 % of chronic mechanical low back pain [1].

Immunohistological studies showed that the nociceptors in SIJ could be found throughout the joint capsule, ligaments, and to a lesser extent subchondral bone. Injury to any of these intra-articular or extra-articular structures can lead to pain [2, 3]. Among intra-articular etiologies, arthritis and spondyloarthropathies are the two most common causes. Spondyloarthropathies may also associate with extra-articular source of pain such as ligamentous, muscular injuries, and enthesopathy.

Anatomy

The SIJ is a diarthrodial joint, consisting of two surfaces held together by fibrous capsule. The anterior third of the joint surface between the sacrum and ilium is a true synovial joint while the rest of the joint is comprised of an intricate set of ligamentous connections (Figs. 48.1 and 48.2). The stability of the SIJ is firmly supported with ligaments that limit the mobility of the joint. These include the anterior sacroiliac ligament, dorsal sacroiliac ligament, sacrospinous ligament, sacrotuberous ligament, and interosseous ligaments (Fig. 48.3) [4, 5].

The innervation of the SIJ is complex. The posterior joint is better understood and more relevant for treatment purposes. A recent detailed anatomical study revealed that the SIJ was innervated by the posterior sacral network, which is 100 % contributed by S1–S2, 88 % by S3, 8 % by L5, and 4 % by S4 [6].

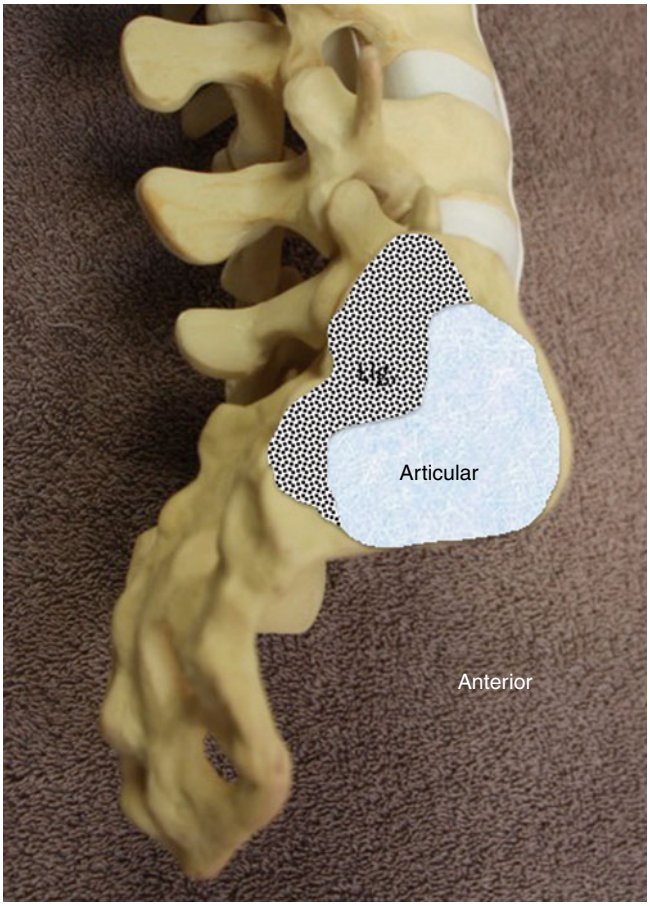


Fig. 48.1 Lateral view of the sacroiliac joint with the synovial surface (*blue*) and the ligamentous (lig) area. In the ligamentous area, the joint surfaces are connected with an intricate set of ligamentous connection (Reproduced with permission of Philip Peng Educational Series)

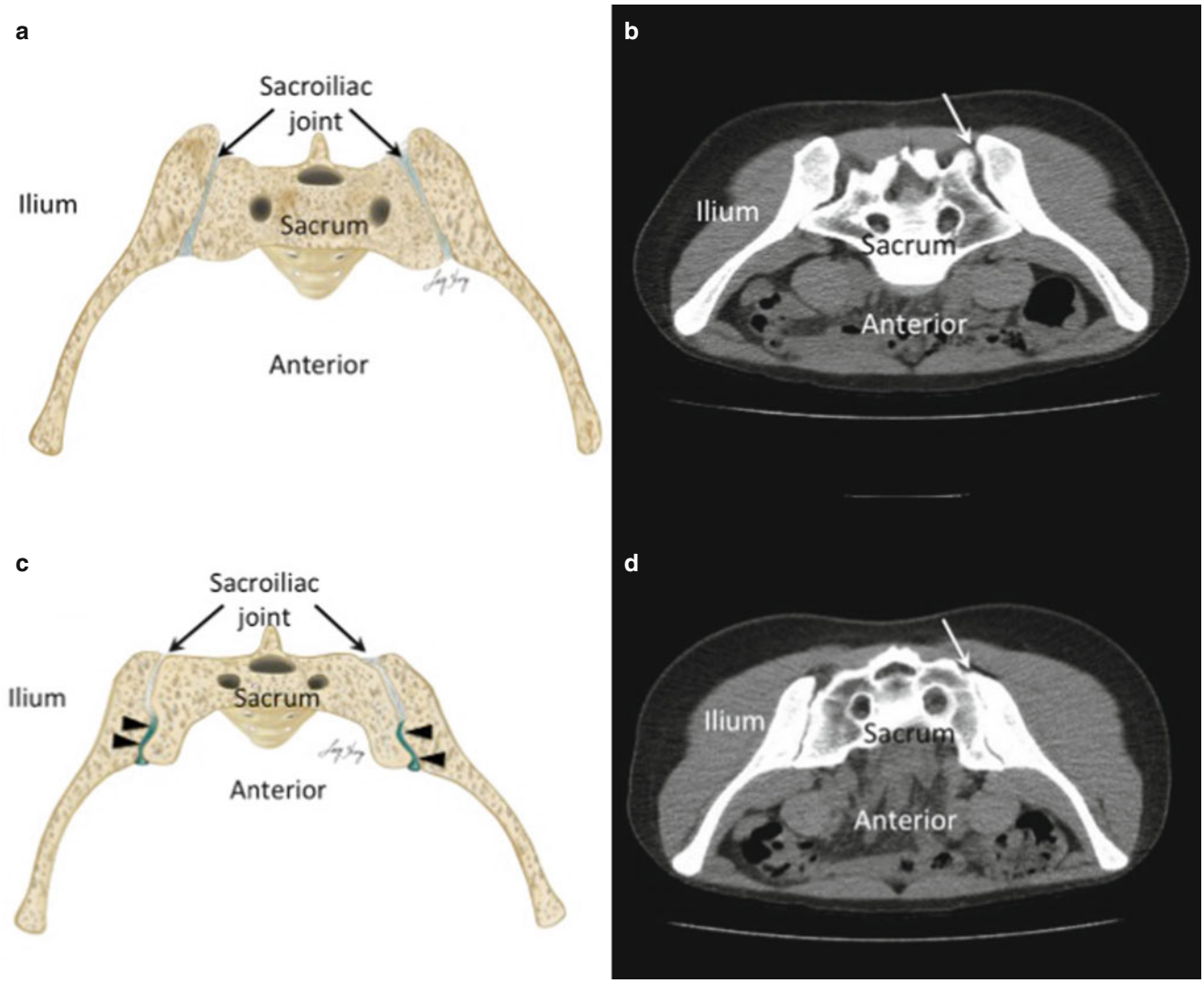


Fig. 48.2 Schematic and CT scan pictures of the upper and lower sacroiliac joint (SIJ). (**a**) In the upper half of the SIJ, the ilium is seen as a prominent bony structure from the posterior SIJ surface. (**b**) Corresponding CT scan. (**c**) In the lower half of the SIJ, the ilium is seen flat from the posterior joint surface. The synovial portion is indicated with *black arrow heads*. (**d**) Corresponding CT scan (Reproduced with permission of Philip Peng Educational Series)

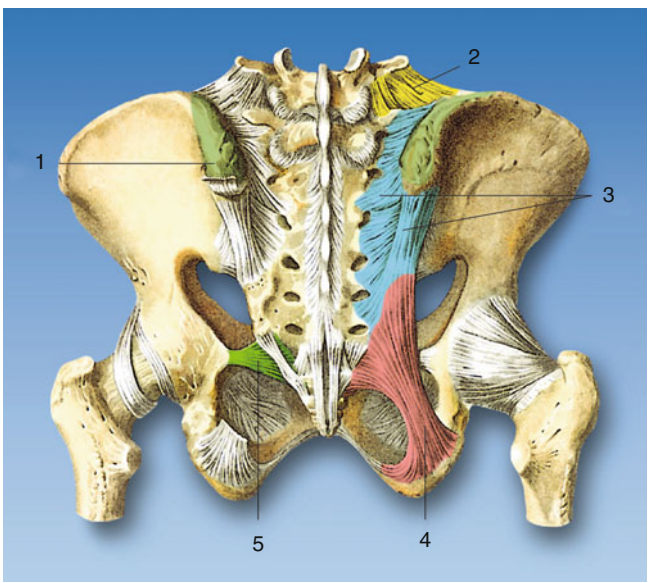


Fig. 48.3 Anatomy. (*1*) Posterior superior iliac spine, (*2*) iliolumbar ligament, (*3*) dorsal sacroiliac ligament, (*4*) sacrotuberous ligament, (*5*) sacrospinous ligament (Reproduced with permission from Dr. Danilo Jankovic)

Clinical Features

Fortin et al. performed contrast injection and provocative test in volunteers and patients in an attempt to identify an SI pain pattern. He found that patients with SIJ pain present with buttock pain extending into the posterolateral thigh [7, 8]. Compared with other source of back pain (e.g., facetogenic and discogenic), patients with SIJ pain are more likely to report lateral pain rather than central pain [9, 10], pain radiation into the groin [11], unilateral pain, pain arising from sitting, and absence of lumbar pain [12].

Controversy exists whether medical history or physical examination maneuvers were reliable in the diagnosis of SIJ pain (Slipman, Dreyfuss, Laslett) [13–15]. Szadek et al. performed a systematic review and concluded that three positive provocation tests had significant discriminative power (diagnostic odds ratio: 17.16) for diagnosing SIJ pain using the reference standard of two positive blocks [16].

Thus, the presence of three or more positive provocative tests appears to have reasonable sensitivity and specificity in identifying those individuals who will positively respond to diagnostic SIJ injections [4].

Intervention

Interventional procedures to SIJ include injection to SIJ (intra-articular or extra-articular) and radiofrequency lesioning of the lateral branches of sacral nerves. The last one will be out of the scope of this chapter.

In determining the types of articular block (intra-articular or extra-articular), several factors need to be considered: the clinical evidence supporting a putative diagnosis, the evidence supporting the treatment and any anatomical considerations that may affect the decision-making process (e.g., spondyloarthropathy or multiple previously failed interventions) [17]. In elderly, the source of pain is usually related to arthritis (intra-articular pathology), while in young, active people, the SIJ pain is more related to the soft tissue structures (i.e., ligaments and muscles) that comprise the SI articulation (extra-articular pathology). As discussed previously, immunohistological studies suggest that the source of nociception can come from the SIJ capsule, surrounding ligaments and subchondral bone. Depending on the patient, both intra-articular and extra-articular injections may provide benefit.

Literature on periarticular injection with local anesthetic and steroid includes two controlled trials [18, 19], one on patients with seronegative spondyloarthropathy and the other on patients with nonspondyloarthropathic SIJ pain. Both supported the analgesic efficacy of extra-articular injection up to 2 months. For intra-articular injections, there is only one controlled trial supporting analgesic efficacy for spondyloarthropathy and anecdotal evidence for a beneficial effect in nonspondyloarthropathy SIJ pain [4].

Technique

With landmark guidance, the success rate ranged from 12 to 22 % [20, 21]. Fluoroscopic (FL) guidance is commonly used to improve accuracy of this procedure, and more recently, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound have also been utilized. This chapter will discuss the technique with fluoroscopy and ultrasound guidance.

Fluoroscopy-Guided Technique

The patient is placed in prone position. The C-arm is placed initially in neutral position. The target point is the inferior, posterior aspect of the joint, approximately 1–2 cm cephalad of the most inferior end [22]. In this view, both anterior and posterior joint lines are seen and the posterior joint line is usually the more medial one (Fig. 48.4). The C-arm is rotated and adjusted until the medial cortical line of the medial silhouette is maximally crisp, which coincides with the beam directed into the posterior opening of the inferior joint space (Fig. 48.5). Usually, this view is obtained with 5–20° of contralateral rotation. A 22- or 25-gauge spinal needle is used and directed toward initially to the sacrum first to appreciate the depth required. Once the bone is contacted, the needle is redirected toward the joint line. The operator should note the depth of the initial contact so that the needle should not penetrate a few more millimeters deeper than this depth (Fig. 48.6). Once the needle has entered the joint space, 0.3–0.5 mL of contrast is injected. In posteroanterior view, the contrast material should be seen traveling rostral along the joint (Fig. 48.7). A lateral X-ray should be used to confirm the needle position (Fig. 48.8). The joint volume in asymptomatic individual is approximately 1.6 mL but is about 1.08 mL in patients requiring SIJ injection. Thus, the injectate is usually 1 mL.

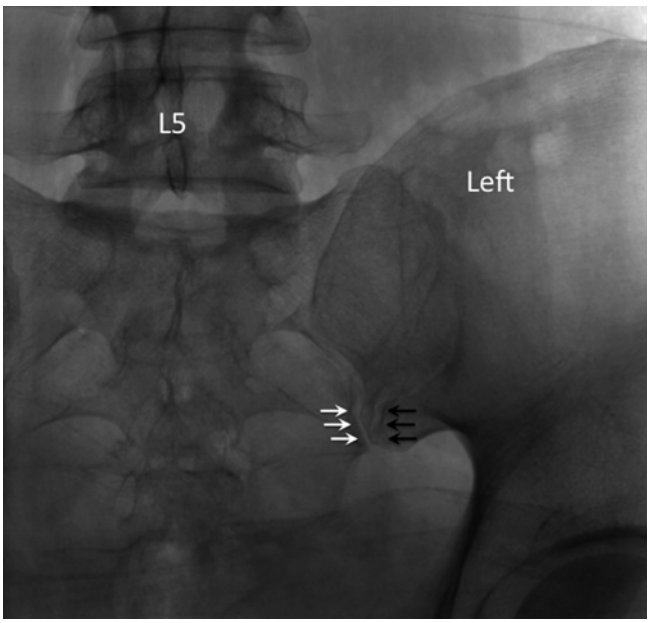


Fig. 48.4 Straight posteroanterior X-ray of the sacroiliac joint. In this view, both anterior (*black arrows*) and posterior joint (*white arrows*) lines are seen. The posterior joint line (*white arrows*) is usually the more medial one (Reproduced with permission of Philip Peng Educational Series)

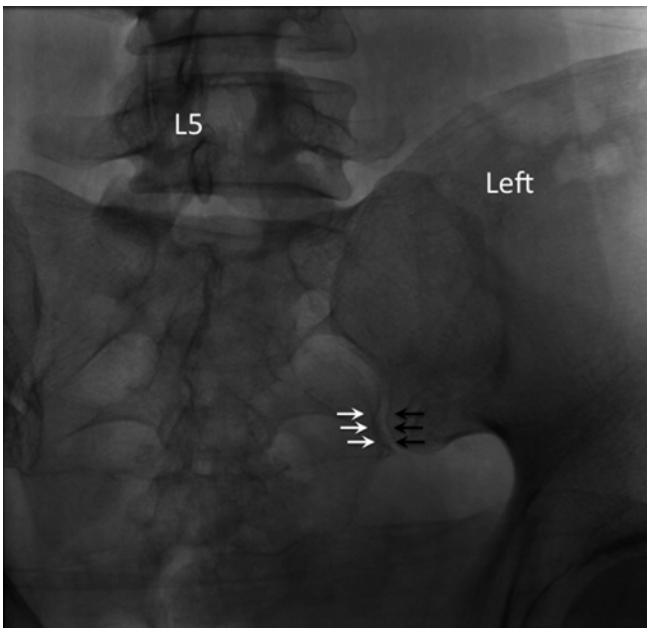


Fig. 48.5 Contralateral oblique X-ray of the sacroiliac joint. The anterior and posterior joint lines aligned to form a crisp silhouette of the joint (Reproduced with permission of Philip Peng Educational Series)

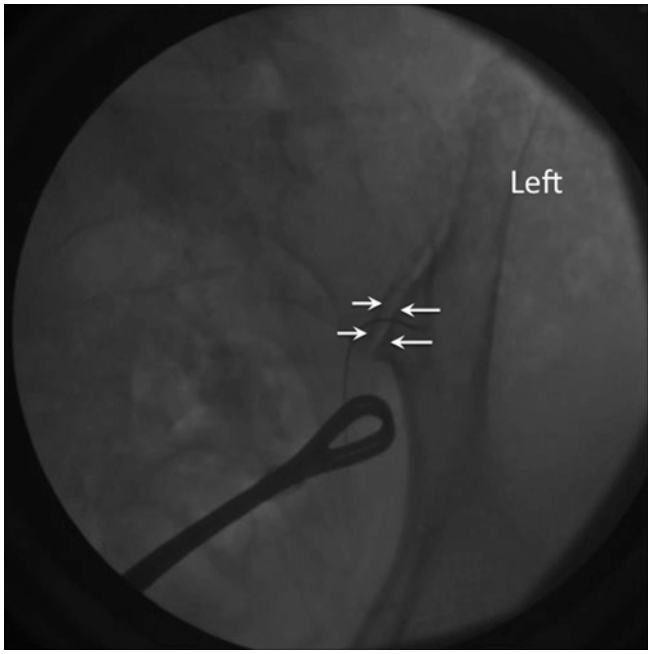


Fig. 48.6 Insertion of needle into the sacroiliac joint (SIJ). The needle is slightly bended by a swap forceps and the site of angulation was at the entrance of SIJ and the needle inside the SIJ was only a few millimeters in length (Reproduced with permission of Philip Peng Educational Series)

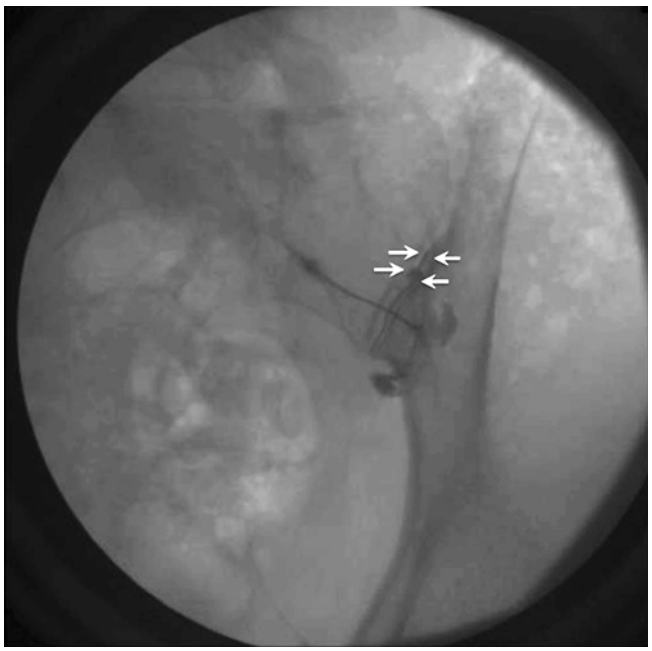


Fig. 48.7 Injection of contrast showed the contrast traveling in the rostral direction (*white arrow heads*) (Reproduced with permission of Philip Peng Educational Series)

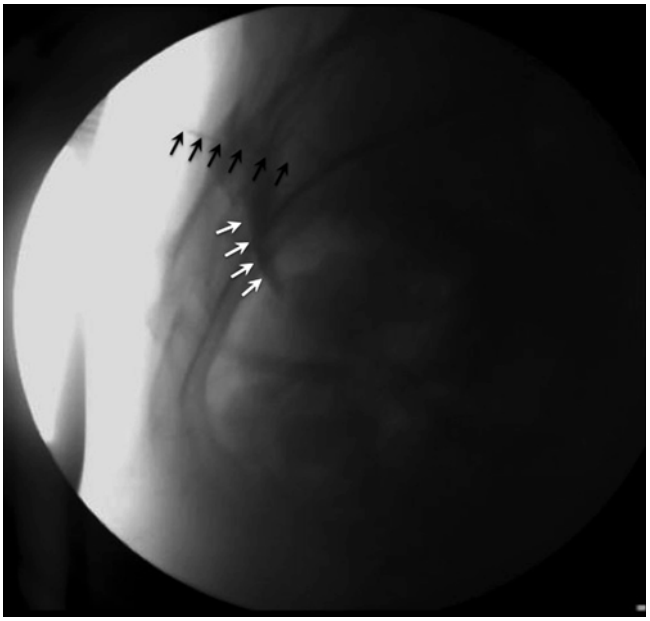


Fig. 48.8 Lateral view of the sacroiliac joint. The needle was marked by *black arrows*. The lower perimeter of the joint space was marked by the *white arrows* (Reproduced with permission of Philip Peng Educational Series)

Ultrasound-Guided Technique

The target is the lowest 1 cm of the SIJ. The typical profile of the upper SIJ reveals a prominent ilium and dorsal sacroiliac ligament (Fig. 48.9). When the ultrasound probe is at the lowest portion of the SIJ, the ilium will become "flat" and the SIJ appear lateral to the lateral crest (Fig. 48.10). The needle can be inserted out-of-plane or in-plane. If in-plane technique is chosen, the author suggests a few steps to facilitate the needle insertion. First, the needle is made curve at the end. Second, the needle is inserted from medial to lateral. Third, the ultrasound probe is tilted away from midline (Fig. 48.11). In this case, more gel is added to the medial aspect of the probe to improve the contact with the sacrum. With these preparatory steps, the needle is inserted from medial to lateral aiming at the tip of the lateral crest (Fig. 48.12). Once the needle passes the lateral crest, the needle is rotated so that the tip is curved toward the SIJ. If the needle is inside the joint, real-time imaging will not show any movement of injectate.

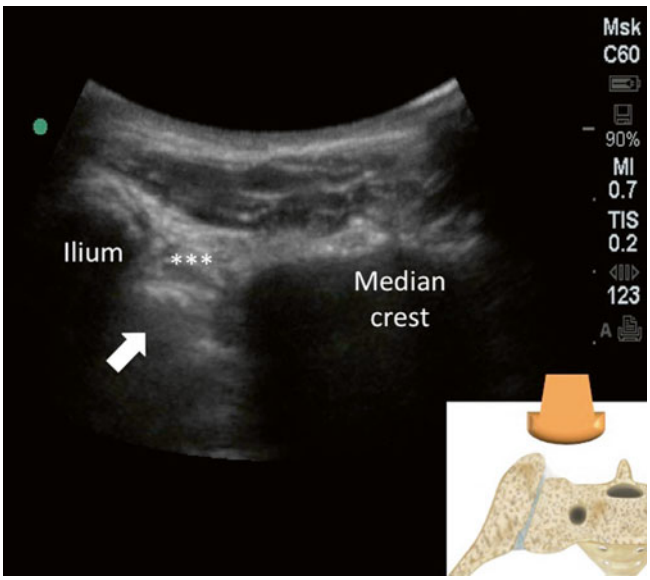


Fig. 48.9 Sonography of the upper sacroiliac joint (SIJ). The probe position was indicated in the insertion in the lower right corner. The SIJ was indicated by the *bold arrow*. Note the prominence of the ilium. *** dorsal sacroiliac ligament (Reproduced with permission of Philip Peng Educational Series)

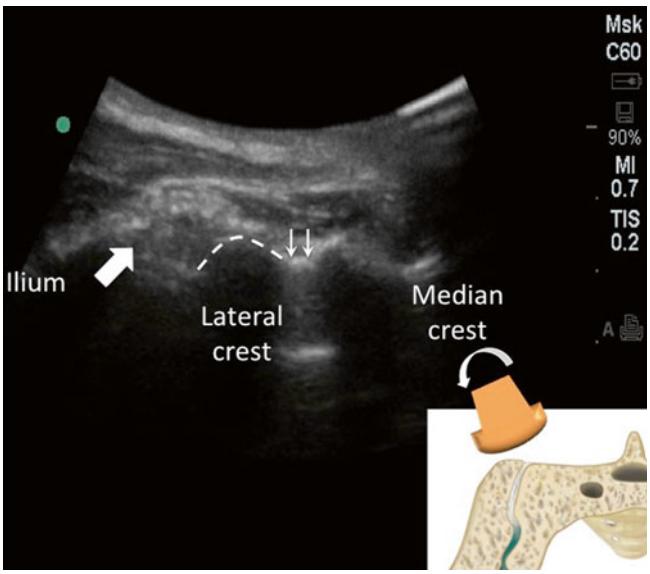


Fig. 48.11 Sonography of the lower sacroiliac joint (SIJ) similar to Fig. 48.10 but the ultrasound probe was tilted toward the midline to allow a steeper angle of the needle. The probe position was indicated in the insert in the lower right corner. The SIJ was indicated by the *bold arrow* (Reproduced with permission of Philip Peng Educational Series)

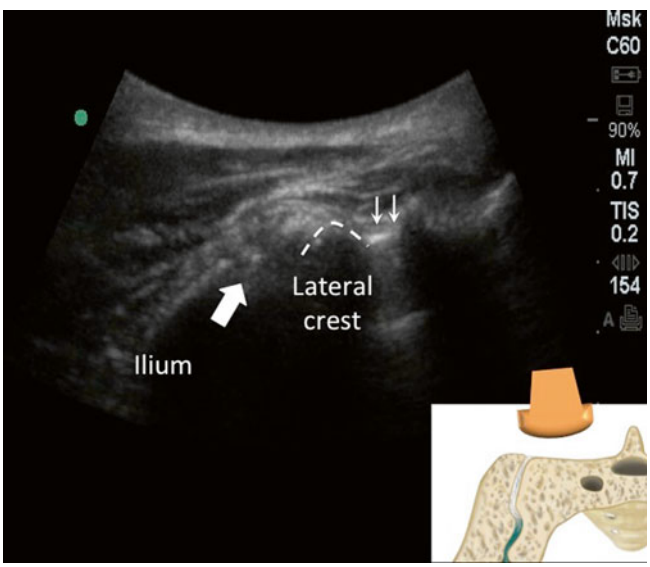


Fig. 48.10 Sonography of the lower sacroiliac joint (SIJ). The probe position was indicated in the *insert* in the *lower right corner*. The SIJ was indicated by the *bold arrow*, which is typically lateral to the lateral crest. Note the flat appearance of the ilium. The S2 foramen was indicated by the *line arrows* (Reproduced with permission of Philip Peng Educational Series)

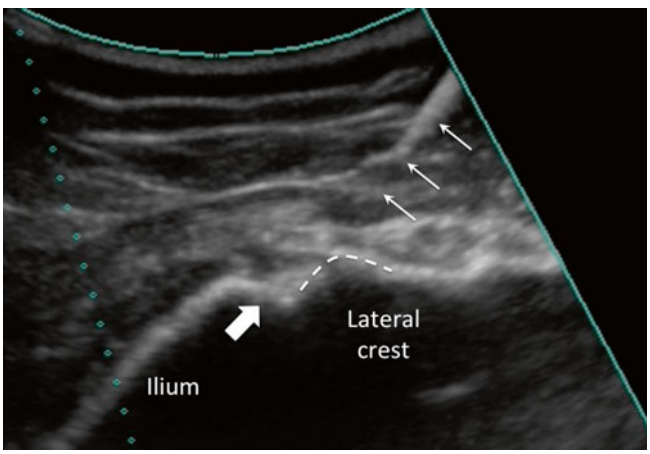


Fig. 48.12 Sonography of the lower sacroiliac joint (SIJ) with the needle (*line arrows*) inserting in-plane in a medial to lateral direction. The SIJ was indicated by the *bold arrow* (Reproduced with permission of Philip Peng Educational Series)

References

1. Hansen HC, McKenzie-Brown AM, Cohen SP, Swicegood JR, Colson JD, Manchikanti L. Sacroiliac joint interventions: a systematic review. Pain Physician. 2007;10:165–84.
2. Szadek KM, Hoogland PV, Zuurmond WW, de Lange JJ, Perez RS. Nociceptive nerve fibers in the sacroiliac joint in humans. Reg Anesth Pain Med. 2008;33:36–43.
3. Szadek KM, Hoogland PV, Zuurmond WW, De Lange JJ, Perez RS. Possible nociceptive structures in the sacroiliac joint cartilage: an immunohistochemical study. Clin Anat. 2010;23: 192–8.
4. Cohen SP, Chen Y, Neufeld NJ. Sacroiliac joint pain: a comprehensive review of epidemiology, diagnosis and treatment. Expert Rev Neurother. 2013;13:99–116.
5. Foley BS, Buschbacher RM. Sacroiliac joint pain: anatomy, biomechanics, diagnosis, and treatment. Am J Phys Med Rehabil. 2006;85:997–1006.
6. Roberts S, Burnham R, Ravichandiran K, Agur A, Loh E. Cadaveric study of sacroiliac joint innervation: implications for diagnostic blocks and radiofrequency ablation. Reg Anesth Pain Med. 2014;39:456–64.
7. Fortin JD, Dwyer AP, West S, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part I: asymptomatic volunteers. Spine. 1994;19:1475–82.
8. Fortin JD, Aprill CN, Ponthieux B, Pier J. Sacroiliac joint: pain referral maps upon applying a new injection/arthrography technique. Part II: Clinical evaluation. Spine. 1994;19: 1483–9.
9. Depalma MJ, Ketchum JM, Trussell BS, Saullo TR, Slipman CW. Does the location of low back pain predict its source? PM R. 2011;3:33–9.
10. Laslett M. Evidence-based diagnosis and treatment of the painful sacroiliac joint. J Man Manip Ther. 2008;16:142–52.
11. Schwarzer AC, Aprill CN, Bogduk N. The sacroiliac joint in chronic low back pain. Spine. 1995;20:31–7.
12. Young S, Aprill C, Laslett M. Correlation of clinical examination characteristics with three sources of chronic low back pain. Spine J. 2003;3:460–5.
13. Dreyfuss P, Michaelsen M, Pauza K, McLarty J, Bogduk N. The value of medical history and physical examination in diagnosing sacroiliac joint pain. Spine. 1996;21:2594–602.
14. Slipman CW, Sterenfeld EB, Chou LH, Herzog R, Vresilovic E. The predictive value of provocative sacroiliac joint stress maneuvers in the diagnosis of sacroiliac joint syndrome. Arch Phys Med Rehabil. 1998;79:288–92.
15. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. Man Ther. 2005;10:207–18.
16. Szadek KM, van der Wurff P, van Tulder MW, Zuurmond WW, Perez RS. Diagnostic validity of criteria for sacroiliac joint pain: a systematic review. J Pain. 2009;10:354–68.
17. Boswell MV, Trescot AM, Datta S et al.; American Society of Interventional Pain Physicians. Interventional techniques: evidence-based practice guidelines in the management of chronic spinal pain. Pain Physician. 2007;10:7–111.
18. Luukkainen R, Nissilä M, Asikainen E, et al. Periarticular corticosteroid treatment of the sacroiliac joint in patients with seronegative spondyloarthropathy. Clin Exp Rheumatol. 1999;17:88–90.
19. Luukkainen RK, Wennerstrand PV, Kautiainen HH, Sanila MT, Asikainen EL. Efficacy of periarticular corticosteroid treatment of the sacroiliac joint in non-spondyloarthropathic patients with chronic low back pain in the region of the sacroiliac joint. Clin Exp Rheumatol. 2002;20:52–4.
20. Rosenberg JM, Quint TJ, de Rosayro AM. Computerized tomographic localization of clinically-guided sacroiliac joint injections. Clin J Pain. 2000;16:18–21.
21. Hansen HC. Is fluoroscopy necessary for sacroiliac joint injections? Pain Physician. 2003;6:155–8.
22. ISIS. Sacroiliac joint access. In: Bogduk N, editor. Practice guidelines for spinal diagnostic and treatment procedures. 2nd ed. San Francisco: ISIS; 2013.

Chapter 49

Sacral Nerve Root Block

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_49,

Definition

A paravertebral blockade of the sacral nerves in the region of the nerve exit points in the area of the sacral foramina.

Anatomy

The sacrum is wedge-shaped and consists of five fused vertebrae. Dorsally, it has a convex surface, with the median sacral crest projecting in the middle of it. Between the median and lateral sacral crests, there are four sacral foramina (the *posterior sacral foramina*), through which the dorsal branches of the sacral spinal nerves emerge. The anterior view shows a concave surface. In addition to the transverse lines, the large anterior foramina are seen (the *anterior pelvic sacral foramina*), through which the primary anterior sacral nerves emerge (cf. Chap. 40).

The *lumbosacral trunk* (part of L4 and L5) and the *S1–S3 ventral branches* combine on the anterior surface of the piriformis muscle to form the *sacral plexus* (Figs. 49.1 and 49.2). From the plexus, direct branches are given off to the muscles in the pelvic region: to the piriformis, the gemelli, the obturator internus, and the quadratus femoris.

The *sacral canal* contains the five paired sacral nerves. They course caudally and exit through the sacral foramina.

- The *sciatic nerve* arises from the ventral branches of spinal nerves L4–S3 (cf. Chaps. 60, 61).
- The *superior gluteal nerve* (L4–S1) supplies the gluteus medius and gluteus minimus muscles.
- The *inferior gluteal nerve* (L5–S2) supplies the gluteus maximus.
- The *posterior cutaneous nerve of the thigh* (S1–S3) is a purely sensory nerve and gives off branches to the lower edge of the buttocks (the inferior clunial nerves) and to the perineal region (perineal branches).
- The *pudendal nerve* (S2–S5) leaves the pelvis through the infrapiriform foramen, courses dorsally round the ischial spine, and passes through the lesser sciatic foramen into the ischiorectal fossa. In the fossa, it courses along the lateral wall in the pudendal canal to below the symphysis and with its terminal branch to the dorsal side of the penis or clitoris. Numerous branches are given off in the pudendal canal (cf. Chap. 56):
  - The *inferior rectal nerves*, which may also arise directly from sacral nerves S2–S4, provide the motor supply to the external anal sphincter and sensory supply to the perianal skin and the lower two thirds of the anal canal.
  - The *perineal nerves* are involved in the innervation of the external anal sphincter muscles and bulbospongiosus, ischiocavernosus, and superficial transverse perineal muscles.
  - The *muscular branches* (S3, S4) supply the levator ani and coccygeal muscles.
  - The urinary bladder, urethra, and external genitalia are mainly innervated by nerves from S2 to S4.

The external genitalia, bladder, and rectal orifice are border areas between the autonomic smooth muscles and voluntary striated muscles. Autonomic and somatomotor fibers are therefore intertwined in them.

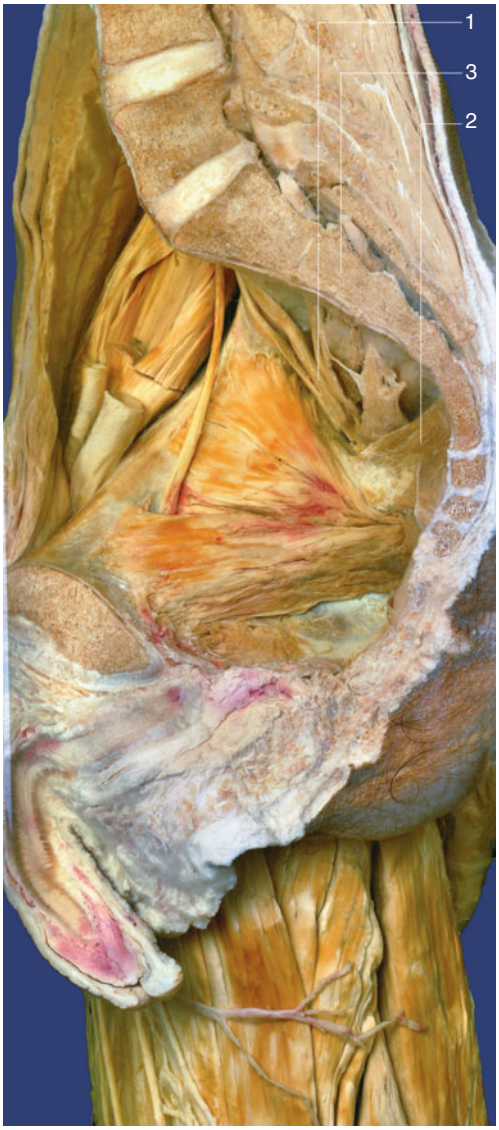


Fig. 49.1 Anatomic specimen. Lateral view of the lumbosacral plexus. (*1*) Lumbosacral plexus from segments L5 to S3, (*2*) sacrospinal ligament, (*3*) sacrum (Reproduced with permission from D. Jankovic)

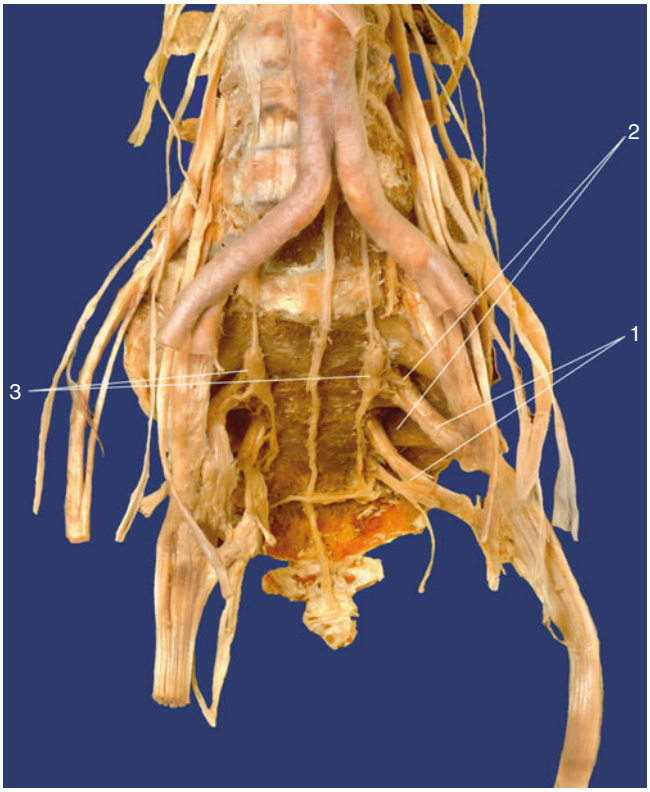


Fig. 49.2 Anatomic specimen. Sacrum, ventral view. (*1*) Sacral plexus, (*2*) anterior sacral foramina, (*3*) sympathetic trunk (Reproduced with permission from D. Jankovic)

Indications

Surgical

None.

Diagnostic

1. For differentiation between pain states in the lower extremities (localization of the affected segment in cases of lumbosacral radiculopathy), suprapubic region, and around the perineum.
2. Bladder dysfunction (bladder sphincter spasm in spinal cord injuries—provided that cystometry has confirmed there is good tone in the bladder muscles).
3. The S1 nerve root block is commonly performed for diagnostic block in patients with radicular pain of uncertain contribution from lumbosacral nerve root (selective spinal nerve root block).

Therapeutic

1. Treatment of bladder sphincter spasm (see above).
2. Sciatic pain with transforaminal S1 nerve root injection.
3. Neurolytic blockade: to support pain treatment in malignant processes (pelvis, perineum, or suprapubic pain) as an alternative to intrathecal neurolysis (with less severe incontinence effects or motor dysfunction).
4. Bladder dysfunction. The musculature of the inferior urinary tract has parasympathetic, sympathetic, and somatic innervation. The motor innervation of the detrusor muscle is via parasympathetic efferents, the motor neurons of which are closely adjacent to the (somatic) anterior horn cells of the pudendal nerve and originate from the second to fourth sacral segments. The preganglionic parasympathetic neurons course as the pelvic nerve to the pelvic plexus, from which they reach the bladder wall. Preganglionic efferents arise from segments T11–L2, pass through the sympathetic trunk, and then form the hypogastric nerve. From the latter, branches course to the pelvic nerve and together with it form the pelvic plexus. Postganglionic sympathetic fibers mainly innervate the bladder neck region. The striated external urethral sphincter and the pelvic floor muscles are supplied by the somatic pudendal nerve (S2–S4). Sensory impulses are derived from stretching and pain stimuli in the bladder and are conducted both via the pelvic nerve and also the hypogastric nerve. The pudendal nerve also has afferent functions and conducts signals from the urethra that convey the sensation of urinary flow, as well as proprioceptive impulses from the pelvic floor muscles. The sacral micturition center ensures urination without residual urine (muscular urination control, S2–S4).
5. Bladder emptying after paraplegia. When the spinal cord is severed above the sacral medulla, reflex emptying is no longer observed after bladder filling in either animals or humans (spinal shock), and the urinary bladder becomes flaccid and atonic for a period of days to weeks. In the chronic state, this phase gradually shifts to the reflex bladder phase, in which minor bladder filling leads to reflex contraction of the bladder detrusor and frequent urination. The reflex arc has a spinal course. The motor neurons to the external urethral sphincter no longer have reflex inhibition, but instead reflex stimulation. This leads to detrusor–sphincter dyssynergia. Paraplegic patients can learn bladder emptying control. If cystometric examinations show good bladder tone in paraplegic patients, a transsacral blockade of the second and third sacral nerves bilaterally can trigger sphincter spasm.

Contraindications

Absolute

1. When declined by the patient
2. Coagulation disturbances and anticoagulant treatment
3. Local infections (skin diseases) at the injection site

Relative

Obesity.

Procedure

Preparations

Check that the emergency equipment is complete and in working order; sterile precautions; and intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, and emergency drugs.

An information discussion with the patient is absolutely necessary.

Transformal S1 Nerve Root Block

Materials

- Fluoroscopy
- Needles: fine 26-G needle, 25 mm long for local anesthesia An 80-mm-long 22-G spinal needle curved at the tip
- Syringes: 2, 5, and 10 mL
- Local anesthetics, steroid (methylprednisolone or triamcinolone 40 mg or dexamethasone 10 mg)
- Contrast for spinal injection (Omnipaque® or Isovue300/370)
- Disinfectant, swabs, compresses, sterile gloves, and fenestrated drapes

Patient Position

The puncture procedure is carried out with the patient in the prone position, with a cushion under the pelvis (Fig. 49.3).

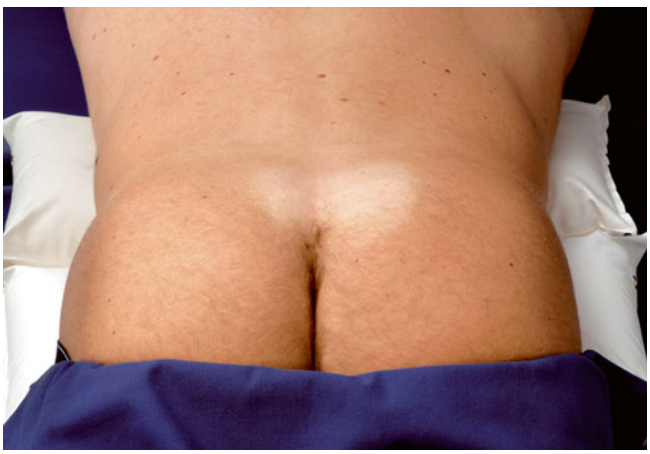


Fig. 49.3 Abdominal positioning with a cushion under the pelvis (Reproduced with permission from D. Jankovic)

Technique

The target point is the superior lateral aspect of S1 neural foramen on the caudal border of the S1 pedicle (Fig. 49.4). To locate this target, the first step is to locate the S1 foramen and superimpose the anterior and posterior S1 foramen. This can be achieved by rotating an image intensifier in cephalocaudad and occasionally ipsilateral oblique direction.

The needle punctures the skin directly over the target with the needle curve facing lateral and superior border of the foramen. The needle therefore passes in an inferior to superior and lateral to medial trajectory, with the attempt to contact the bone of the superolateral border of the S1 foramen. This maneuver is to ensure the practitioner to appreciate the depth of the foramen. Upon bony contact, the needle is rotated medially to "walk off" the bone. Once the needle is through the dorsal aspect of the foramen, the needle is then rotated with curve toward the superolateral aspect of the foramen and advanced no more than a few millimeters. A lateral view is obtained to ensure the needle tip well within the S1 foramen but not ventral to the floor of sacral canal. Confirmation of needle placement is made by injecting contrast (0.25–0.5 mL) during lateral and AP fluoroscopic visualization. Presence of vascular uptake is detected by either real-time fluoroscopy or digital subtraction angiography. Once the needle position is satisfactory, a mixture of 2 mL of local anesthetic and 1 mL of steroid is administered.

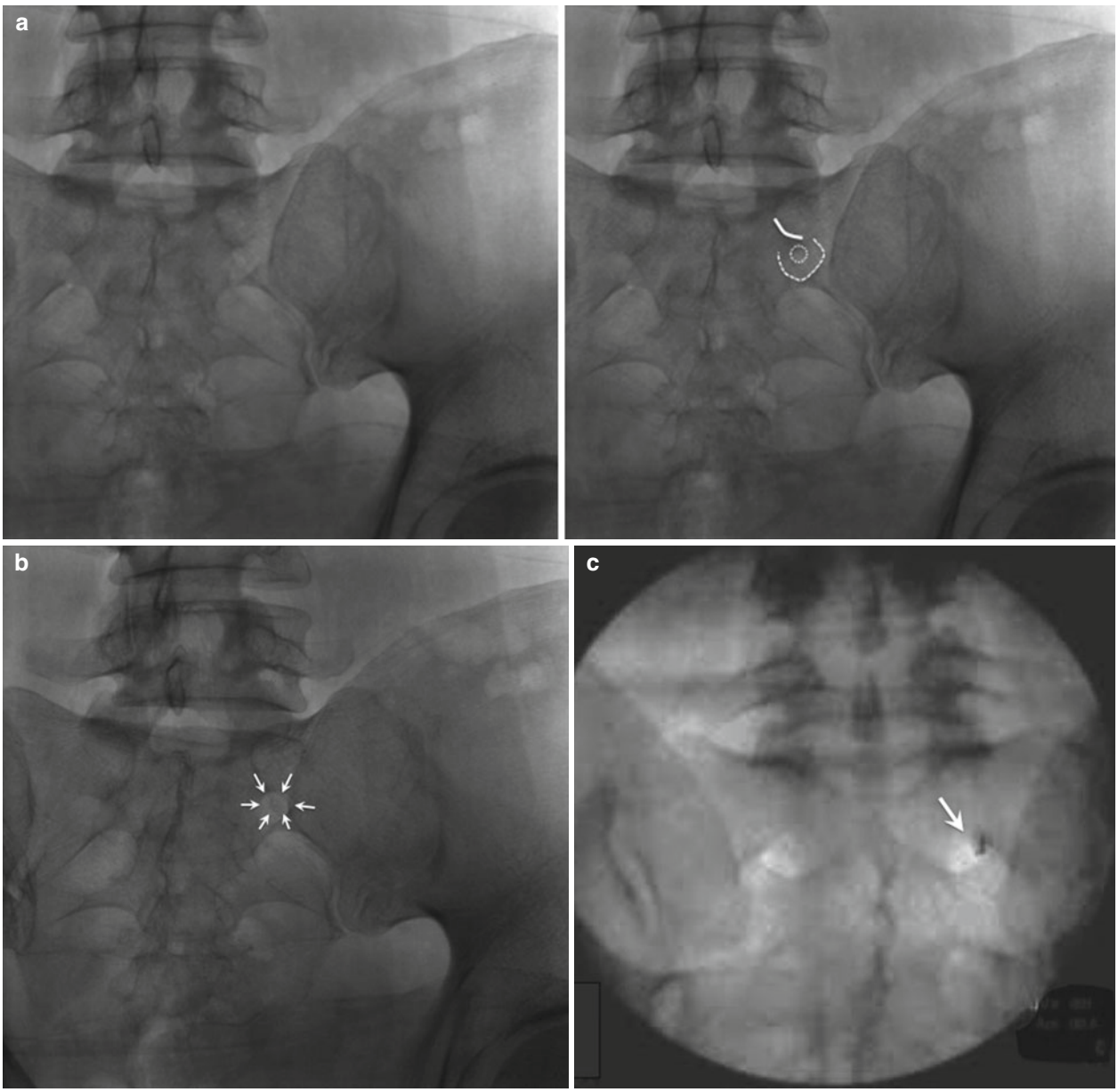


Fig. 49.4 (**a**) Radiograph of lumbosacral spine without label. (**b**) The pedicle of S1 was indicated by a solid line on the radiograph on the *right side*. Caudal to this, the dorsal silhouette of the S1 foramen was indicated by the complete smaller circle and the ventral silhouette by the larger incomplete circle. (**c**) The C-arm was rotated to give ipsilateral oblique view. In this view, the dorsal silhouette of the S1 foramen was much better appreciated. (**d**) A 22-gauge spinal needle was inserted to the lateral and cephalad aspect of the S1 foramen as indicated by the *arrow* (Reproduced with permission from Philip Peng Education Series)

S2 to S4 Nerve Root Block

This blockade should be carried out by experienced anesthetists or under their supervision.

Materials

- Needles: fine 26-G needle, 25 mm long for local anesthesia
  An 80-mm-long 22-G spinal needle
- Electrostimulation: nerve stimulator
- 22-G (15°) 80-mm stimulating needle with injection tubing syringes: 2, 5, and 10 mL

Local anesthetics, disinfectant, swabs, compresses, sterile gloves, and fenestrated drapes

Patient Positioning (Fig. 49.3)

The puncture procedure is usually carried out with the patient in the abdominal position, with a cushion under the pelvis and legs spread so that the heels are rotated outward with the toes pointing inward.

A swab is placed in the gluteal sulcus to protect the anal and perineal area from disinfectants (cf. Chap. 44).

Puncture-Relevant Anatomy

The sacral bone is deformed in 20–25 % of the patients, with the sacral foramina not running parallel to the sacral crest.

The distance to the *skin* and *sacral periosteum* in the region of S2 depends on the anatomy and ranges from 1.3 cm in slim patients to up to 5 cm in obese patients [4] (Fig. 49.5).

The diameter of the sacrum declines from cranial to caudal. It needs to be borne in mind that due to the sacrum's concave surface, its distance from the skin surface declines from cranial (S1, S2) to caudal (S3, S4). The length of needle required varies correspondingly (8–10 cm in the area of S1 to 5 cm in the area of S4).

The distance between the anterior and posterior sacral foramina (transforaminal canal) declines from S1 (approx. 2.5 cm at S1, 1.3–2.0 cm at S2) to caudal as far as S4 by approximately 0.5 cm for each additional foramen (Fig. 49.6).

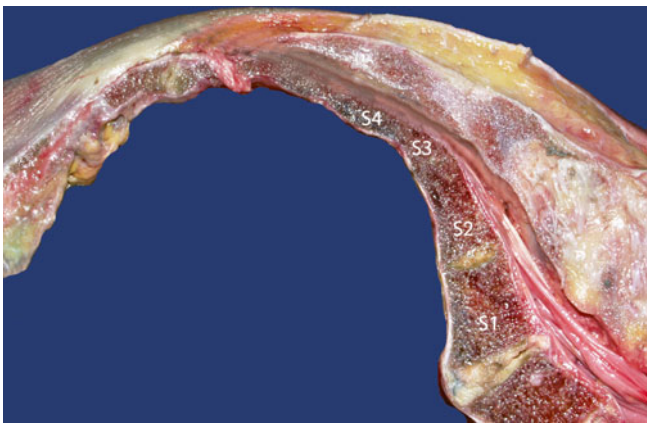


Fig. 49.5 Due to the sacrum's concave surface, the distance to the skin surface declines from cranial (S1, S2) to caudal (S3, S4) (Reproduced with permission from D. Jankovic)

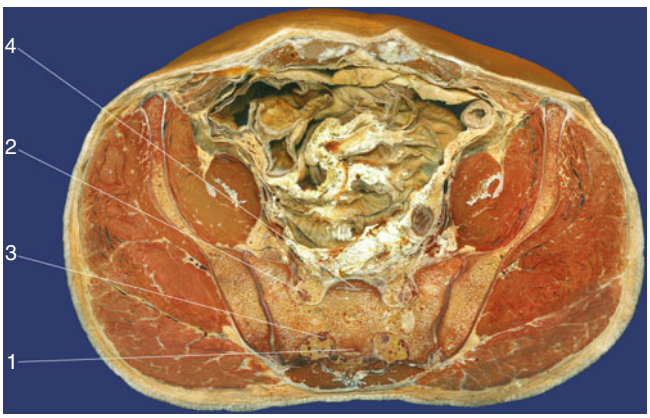


Fig. 49.6 Anatomic specimen. Sagittal section. (*1*) Sacral canal, (*2*) posterior sacral foramen, (*3*) anterior sacral foramen, (*4*) intervertebral disk The distance between the anterior and posterior sacral foramina declines from S1 caudally to S4 by approximately 0.5 cm for each additional foramen (Reproduced with permission from D. Jankovic)

Localization

1. Localization and marking of the sacral cornua
2. Palpation of the posterior superior iliac spines. The second sacral foramina are located approximately 1.0–1.5 cm (depending on anatomy) caudal and medial to the posterior superior iliac spines (Fig. 49.7).
3. The fourth sacral foramina are located approximately 1 cm lateral and 1 cm caudal to the sacral cornua.
4. The third sacral foramen is located in the middle between sacral foramina 2 and 4.
5. The fifth sacral nerve does not enter through a foramen, but below the sacral cornu on the corresponding side, approximately 2 cm caudal to the fourth foramen.
6. The connecting line between sacral foramina 2 and 4 courses almost parallel to the median sacral crest. The sacral foramina are located at a distance of approximately 2 cm from one another.

During the puncture procedure, it is absolutely necessary to observe the following:

- Strict asepsis.
- The operator should stand on the side being blocked.
- The line connecting the two second sacral foramina marks the level of the dural sac in most patients (epidural/subarachnoid injection!) (Fig. 49.8).
- The S2 sacral foramen is easier to locate, so that the blockade can be started there.
- Using electrical nerve stimulation increases the success rate with the blockade.
- Due to the variability of sacral anatomy, carrying out the procedure with fluoroscopic guidance is recommended.
- No neurolytics should be administered before the needle position has been checked fluorographically with an image converter.
- Frequent aspiration and fractionated injections.
- Cardiovascular monitoring.
- Verbal monitoring.
- Motor weakness in the leg must always be anticipated. This should be explained to patients.

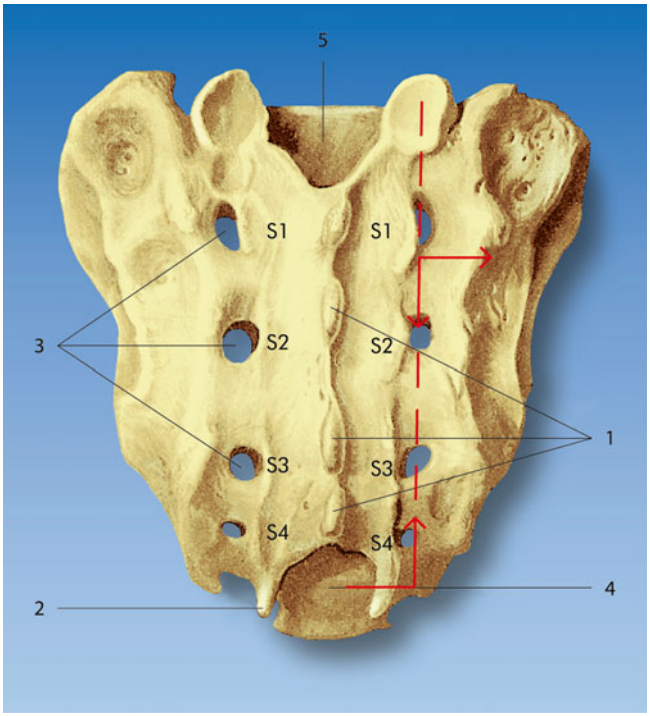


Fig. 49.7 Localization. (*1*) Median sacral crest, (*2*) sacral cornu, (*3*) posterior sacral foramina S1–S4, (*4*) sacral hiatus, (*5*) sacral canal. The second sacral foramina are located approximately 1.0–1.5 cm caudal and medial to the superior posterior iliac spines. The fourth sacral foramina are located approximately 1 cm lateral and 1 cm caudal to the sacral cornua (Reproduced with permission from D. Jankovic)

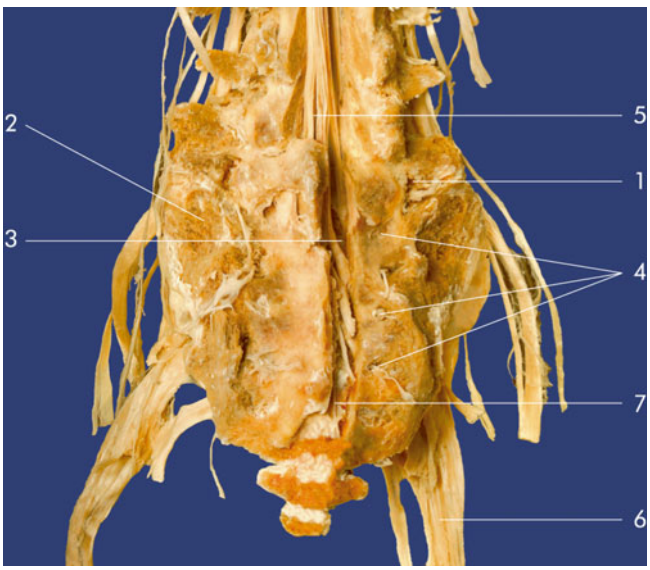


Fig. 49.8 Clinically relevant anatomy, dorsal view. (*1*) Posterior superior iliac spine, (*2*) sacrum; (*3*) spinal ganglia in the sacral canal, opened on one side; (*4*) sacral foramina; (*5*) terminal filum of the cauda equina; (*6*) sacral plexus; (*7*) sacral hiatus (Reproduced with permission from D. Jankovic)

Injection

The puncture procedure is started at the second sacral foramen. After this, sacral foramina 3 and 4 are punctured.

Following local anesthesia at the injection site, a 6–10-cm-long 22-G spinal needle is introduced vertically to the skin surface until there is contact with the periosteum (Fig. 49.9). The needle is withdrawn slightly, and the needle length is corrected until the corresponding foramen is located ("hunt and peck"). The needle is then advanced through the dorsal foramen up to the ventral foramen by approximately 2 cm (S2) to 0.5–1.0 cm (S4).

As soon as paresthesia is triggered (depending on the foramen, in the leg, penis/vagina, perineum, buttocks, or rectum), the needle is minimally withdrawn. Following aspiration (blood, cerebrospinal fluid), fractionated injection of the local anesthetic is carried out.

Electrostimulation

The skin is incised at the puncture site using a hemostylet, to make it easier to introduce the needle. The electrostimulation needle is advanced vertical to the examination table. Stimulation current of 1–2 mA and 2 Hz is selected with a stimulation period of 0.1 ms. When the relevant sacral nerve has been localized, the stimulation current is reduced to 0.3–0.5 mA. After aspiration, fractionated administration of a local anesthetic is carried out (Fig. 49.9).

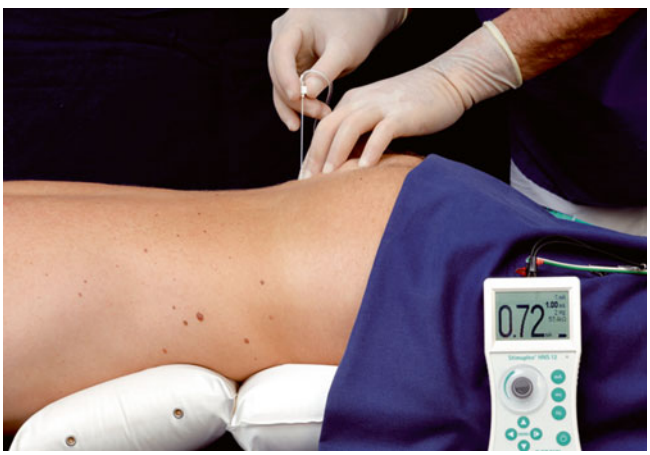


Fig. 49.9 Puncture in the area of the second sacral foramen (Reproduced with permission from D. Jankovic)

Dosage

Therapeutic

1–2 mL of local anesthetic per sacral foramen—e.g., ropivacaine 0.2–0.375 %, mixed with glucocorticoid if appropriate.

Important Notes for Outpatients

Long-lasting block can occur (even after administration of low-dose local anesthetics, e.g., 0.125 % bupivacaine or 0.2 % ropivacaine). The blocked leg can give way even 10–18 h after the injection. The patient must therefore use walking aids during this period.

Complications

1. Subarachnoid or epidural injection
2. Intravascular injection, with toxic reactions (cf. Chap. 1)
3. Hematoma formation
4. Infection
5. Bowel and urinary incontinence is theoretically possible
6. Breakage of puncture needles

Suggested Reading

1. Blank JW, Kahn CH, Warfield CA. Transsacral nerve root. In: Hahn MB, McQuillan PM, Sheplock GJ, editors. Regional anesthesia. St Louis: Mosby; 1996.
2. Bridenbaugh PO, Wedel D. Sacral plexus nerve block. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade. 3rd ed. Philadelphia: Lippincott-Raven; 1998.
3. Frotcher M, Kahle W. Nervensystem und Sinnesorgane. In: Taschenatlas der Anatomie. Stuttgart/New York: Thieme; 2001.
4. Grehl H, Reinhardt F, Neuberger J. Checkliste Neurologie. 2nd ed. Stuttgart/New York: Thieme; 2002.
5. Jenkner FL. Nervenblockaden auf pharmakologischem und auf elektrischem Weg. Wien: Springer; 1980.
6. Moore DC. Regional block. 4th ed. Springfield: Charles Thomas; 1981.
7. Poeck K. Störungen der Balsenentleerung bei neurologischen Krankheiten. In: Poeck K, editor. Neurologie. 9th ed. Berlin/Heidelberg/New York: Springer; 1994.

Chapter 50

Lumbosacral Epiduroscopy

James E. Heavner, DVM, PhD (✉) • Hemmo Bosscher, MD
Department of Anesthesiology, Texas Tech University Health Sciences Center, Lubbock, TX, USA
e-mail: james.heavner@ttuhsc.edu

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_50,

Percutaneous insertion of a flexible fiberscope into the epidural cavity for diagnostic and therapeutic purposes began in the late 1980s. The procedure is known as epiduroscopy and is defined for purposes of this communication as direct visualization of the lumbosacral epidural cavity with a percutaneously inserted fiberoptic device that has a channel through which instruments can be inserted and fluids plus medication can be injected. The target population is patients with lumbar or sacral nerve radicular pain and/or low back pain who are not candidates for surgery, who have failed back surgery syndrome, or who have not obtained sustained benefit from more conservative therapy such as single epidural steroid injection. Patients who present to us for epiduroscopy generally have failed back surgery syndrome, spinal canal stenosis, neuroforamen stenosis, degenerative disc disease, or nonremarkable CT/MRI.

The objectives of epiduroscopy are (1) to gain information by direct visual inspection that contributes to documentation of pathological processes responsible for low back pain and/or radicular pain and (2) to do therapeutic interventions under direct visual control to relieve pain. Our goals in doing epiduroscopy are to better localize and define pathology causing pain, narrow therapeutic options, focus treatment, and assist in establishing a long-term prognosis [2, 9, 24]. General indications for epiduroscopy (spinal canal epiduroscopy) presented in a consensus paper [22] by an international group of experts are:

1. Observation of pathology and anatomy
2. Direct drug application
3. Direct lysis of scarring (with medication, blunt dissection, laser, and other instruments)
4. Placement of catheter and electrode systems (epidural, subarachnoid)
5. An adjunct to minimally invasive surgery

Reviews by Helm et al. [11] and Kallewaard et al. [14] summarize findings of recent literature surveys. Helm et al. focused their systematic review on literature related to the use of endoscopic adhesiolysis in post-lumbar surgery syndrome. Of 21 studies identified, 1 randomized controlled trial and 5 observational studies met inclusion criteria for evaluation. The authors concluded that the evidence is fair that spinal endoscopy is effective in the treatment of post-lumbar surgery syndrome.

Kallewaard et al. reviewed literature dealing with epiduroscopy for patient with lumbosacral radicular pain. They identified one prospective double-blind randomized controlled trial, nine prospective studies, and three retrospective studies that yielded positive results after epiduroscopy in terms of pain scores and functional status. Most studies included patients with failed back surgery syndrome, one study included patients with degenerative lumbar spinal canal stenosis, and one study included patients with sciatica. The authors concluded that there is reasonable evidence for short-term and long-term effects in patients with chronic radicular pain due to failed back surgery syndrome.

Literature reports claim that visualization of pathological tissue and observation of targeted drug administration via epiduroscopy result in substantial and prolonged pain relief [6]. The authors of some studies reported that adhesions unreported by MRI can be identified by epiduroscopy. Manchikanti et al. [15] concluded that endoscopic adhesiolysis with the administration of corticosteroids is also a safe and possibly cost-effective technique for relief of chronic intractable pain failing to respond to other modalities of treatments. There are many reports showing that epiduroscopy is a safe procedure if precautions are taken, e.g., to limit the rate and volume of fluid injected during the procedure [7, 11, 12, 15, 16, 18–21, 23].

Epiduroscopy equipment continues to be improved. Increased clarity of visualization and experience enhances the ability to recognize structures. According to Helm et al., changing the focal length of the scope from infinity to 6 mm markedly improved visualization [11]. Nevertheless, the objective of finding a bulging disc or a diseased nerve root, as is often the case, may seldom be a realistic expectation. On the other hand, information may be obtained by evaluating more readily identifiable tissues such as dura, periosteum, fat and scar tissue, and blood vessels. By assessing these structures relative to other clinical findings and treatment outcomes, we believe advancement has been made in the interpretation of epiduroscopy findings [4].

Like many therapeutic approaches, epiduroscopy does not meet high evidence-based standards demonstrating efficacy. What became clear as we began prospective investigation of epiduroscopy outcome is that there is considerable variability in pathology observed in patients who are candidates for epidural neurolysis and epiduroscopy. We hypothesize that the significance of this variability with respect to treatment outcome must be determined to aid in the design of adequately powered outcome studies.

Results of a literature search conducted in November 2005 retrieved 729 references to the word “epiduroscopy.” A similar search done in January 2015 retrieved 17,700 listings, reflecting an increasing awareness and interest in epiduroscopy. The references to epiduroscopy can be classified as advertisements, published articles, and third-party payer policies. There is significant redundancy.

Fiberoptic Images of the Epidural Space

Interpreting images obtained through spinal endoscopy is not easy. Most of the difficulties can be understood if one realizes that the study of structures in the epidural cavity is done through a small tube. Images of only a fraction of the epidural cavity can be obtained at a time. A larger spatial context depends on a sequence of images obtained by maneuvering the scope in the area of interest. Since anatomical landmarks are sparse, fluoroscopic orientation, using anterior/posterior or lateral views, is needed to place these images in a larger anatomical context.

Landmarks such as the filum terminale, dura, and pedicles are useful in the orientation of the scope. Another limiting factor is cross-sectional size of the epidural cavity. The spinal canal is largely occupied by the dural sac and epidural fat. The remaining space is limited. Expected structures such as discs, disc herniations, and compressed nerve roots are difficult to see. In addition, an intervertebral disc is partially covered by the posterior longitudinal ligament, which in turn is covered by epidural fat obscuring the disc almost completely. A large disc herniation may be present but shows itself as an irregular fibrotic mass that is difficult to penetrate, rather than a smooth disc bulge. Nerve roots are visible (just next to the pedicle); however, they are easily missed if not actively sought. Once a nerve root is identified, it may supply only limited information. The question arises: what does one look for? In order to answer this question, images of the tissues and structures as seen, not as expected, through epiduroscopy will be discussed. Abnormalities of the epidural cavity are most easily understood by the absence of expected tissues and by the presence of abnormal tissues at these sites.

When the scope enters the normal epidural space, one usually observes epidural fat. On advancement of the scope, multiple small blood vessels can be seen traversing the epidural space.

The filum terminale can be seen in the midline. The scope advances most easily in the dorsal epidural cavity, which consists of lamina and ligamentum flavum. Ligaments (plica mediana dorsalis) may connect the posterior wall of the spinal canal to the dura. The scope can be easily passed across the midline most of the time. Most laterally, the pedicle is relatively easy to identify by its smooth rounded shape. So is the medial aspect of the pars superior to the facet joint. The nerve roots are variable in size and are recognized by a characteristic blood vessel on their surface. The roots can be followed medial than inferior to the pedicle. The scope can often be placed through the neuroforamen into the paraspinous space. In fact, our best treatment results are obtained when we are able to pass the scope tip along the cephalid border of the pedicle through the neuroforamen. The nerve root is small relative to the large neuroforamen, and abundant epidural fat in the neuroforamen, continuous with extra spinal fat, makes the nerve root sometimes hard to find. The disc should be found superior to the pedicle but is hard to identify. The indentations of the dura and posterior longitudinal ligaments, between nerve root sleeves, are large and allow inspection of the anterior epidural cavity or at least the lateral part of it. If no pathology obstructs the advancement of the scope and it is long enough, the entire lumbosacral epidural cavity can be investigated in this fashion.

Fat Tissue

Fat tissue is derived from mesenchyme and may serve as a buffer between the hard spinal wall and the more delicate neural structures in the spine. It differs from subcutaneous fat by the relative absence of connective tissue. The presence of fat may be a marker for adequate regional perfusion and lack of pathological (inflammatory) processes. It is most easily recognized by its color. It is white yellow to yellow brown and globular in appearance and can be easily penetrated with the scope (Fig. 50.1). It seems most abundantly present in the lateral recesses and the neural foramina and less so in the dorsal epidural space except for the sacral epidural space which is often completely filled with fat. Absence of fat may be the results of fat atrophy associated with the normal aging process or, more likely, as the result of an inflammatory process and local ischemia. Its absence may play an important role in the pathophysiology of back pain, since epidural fat plays a role in the mechanical buffering of the thecal sac with flexion and extension.

Whether fat tissue differentiates in an alternative direction (i.e., fibrous tissue) to noxious stimuli or is replaced by abnormal pathological tissue is not known.

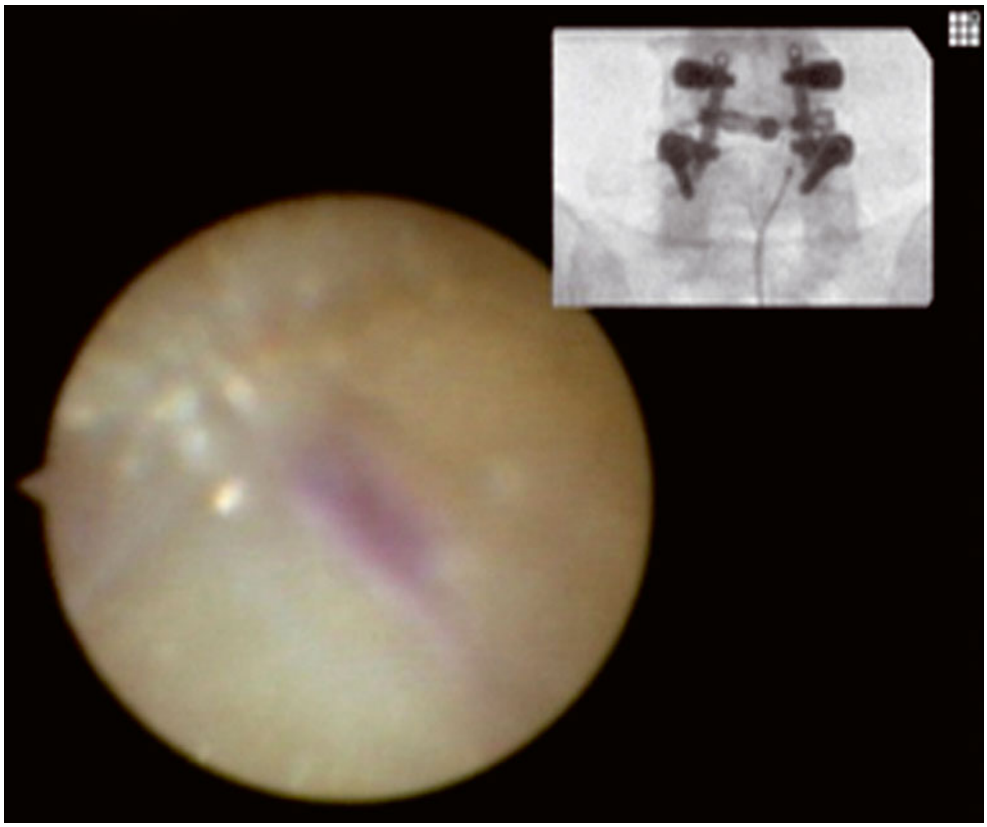


Fig. 50.1 Rt L5—normal peridural fat (With permission from James Heavner)

Vasculature

Arteries and veins traverse the epidural space. Arteries usually are small and clear. Veins usually are transparent and may be difficult to differentiate from adhesions. Arteries are easily recognizable by their pulsating behavior. Obviously, in the absence of fat, the number of visible vessels will increase. However, this may also be part of an inflammatory response. Abnormal vasculature is recognized by its increased density and chaotic orientation in areas of pathology (Fig. 50.2). Since these vessels are dilated, they contain more blood and make a strikingly purple-red appearance. Obstruction to venous blood flow leads to dilated, tortuous vessels, not only on an affected nerve root but at any site that is involved in the pathology. During epiduroscopy, some bleeding from ruptured vessels may occur; however, these vessels are often small and bleeding seems relatively insignificant.

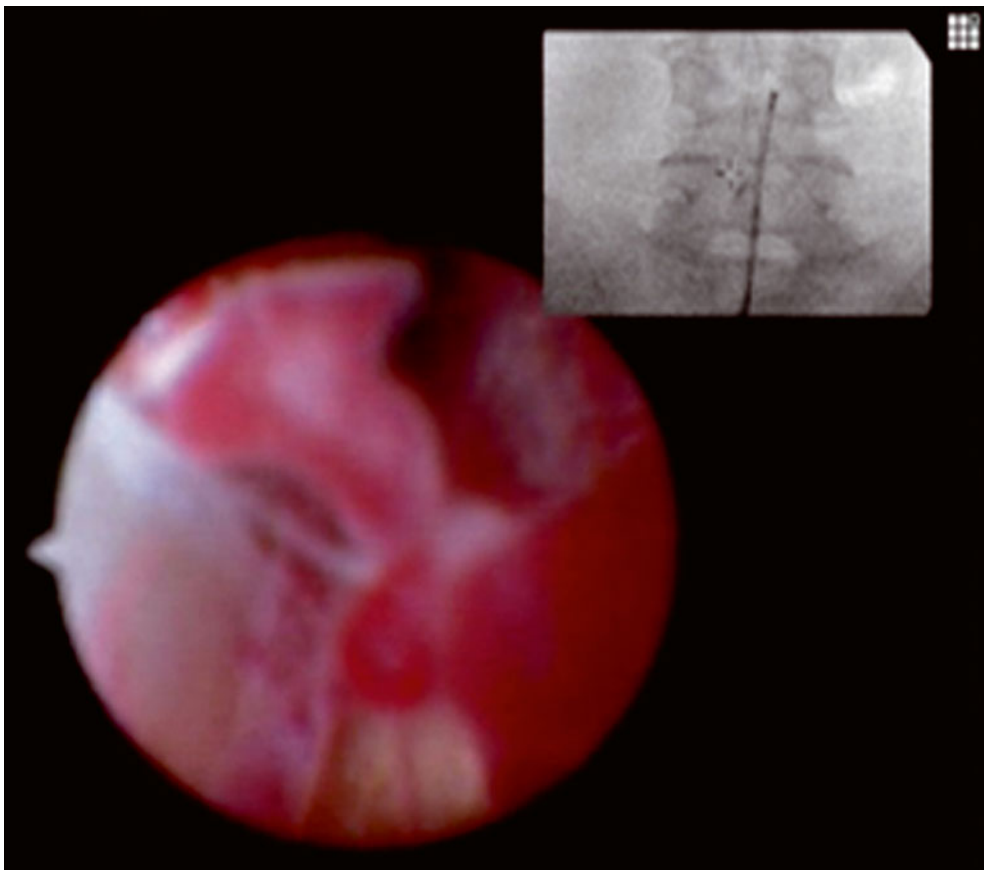


Fig. 50.2 Rt L4—hypervascularity, engorged vessels with Grade 1 fibrosis (With permission from James Heavner)

Inflammation

Inflammatory changes are common and may be part of the normal aging process. They can be found diffusely throughout the epidural space and do not seem to lead to clinical symptoms. Inflamed tissue is recognized by an ink/purple color (Fig. 50.3). It is diffuse and heterogeneous. It is the result of hyperemia and is associated with increased vasculature, exudates, and fibrosis (Fig. 50.4). If the area of inflammation is extensive or is located near the nerve root, maneuvering the scope is painful. Fragility of blood vessels in inflamed areas causes the tissues to bleed on the slightest touch with the scope. In some patients, inflammation is diffusely present in the entire lumbosacral epidural space. Total epidural fat is diminished or absent. The periosteum and dura are hyperemic, and maneuvering of the scope is painful and causes diffuse bleeding.

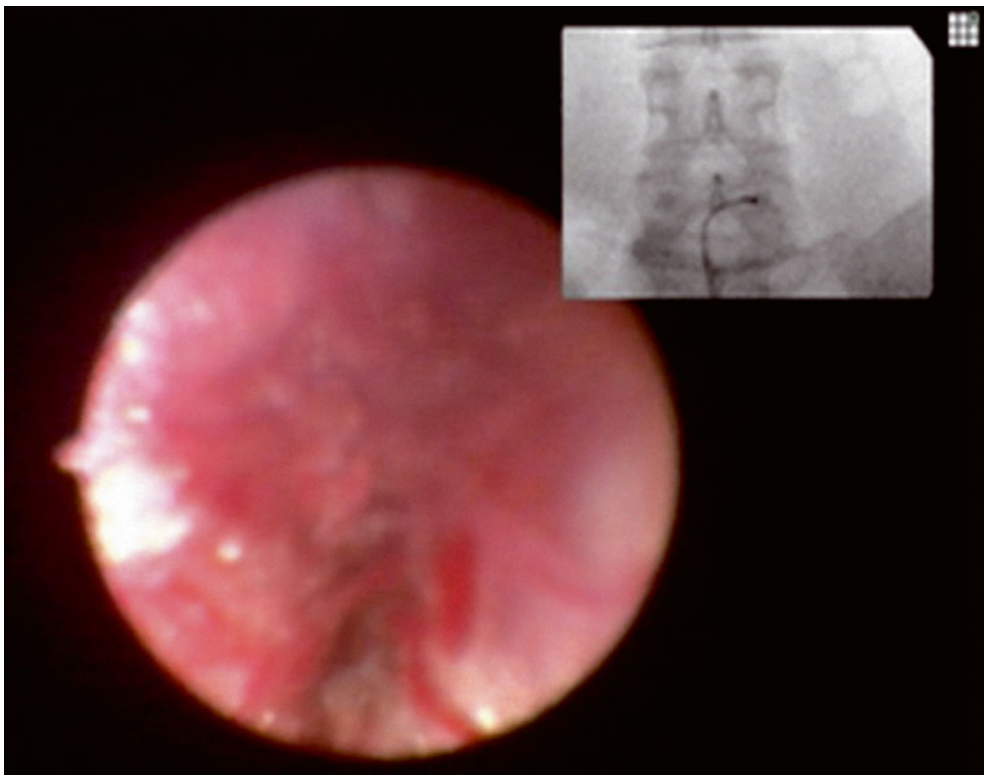


Fig. 50.3 Rt L5—active inflammation (With permission from James Heavner)

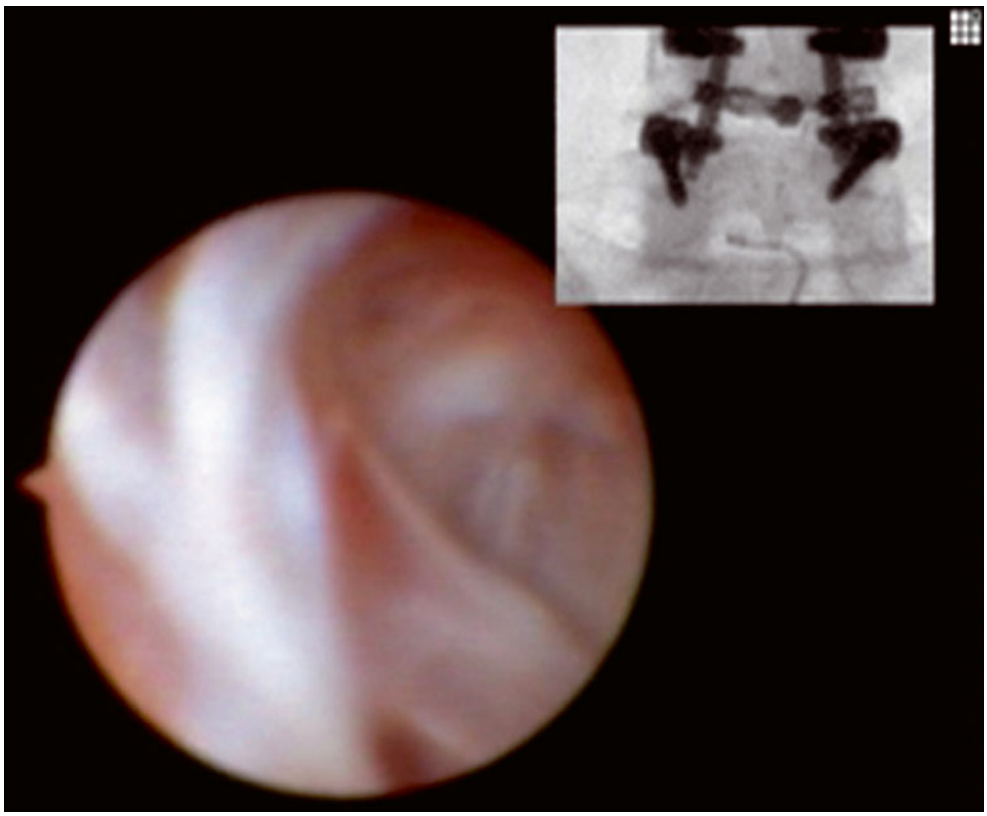


Fig. 50.4 L5 midline—active inflammation with fibrosis (With permission from James Heavner)

Fibrosis

One of the most striking images obtained through epiduroscopy is the one of white scar. Fibrosis is most likely the result of direct trauma (i.e., surgery), chronic inflammation, or local ischemia.

Independent of its cause, it is characterized by the abnormal presence of homogenous white tissue. It is typically located in the lateral recess and the neuroforamen [16]. Since epidural fibrosis may play an important role in patients with chronic back pain and radicular pain, it is useful to grade the extensiveness of the epidural fibrosis [3].

Grade I: The fibrosis consists of strings and sheets traversing the epidural space and attaching to bone or dura. It is often mixed with fat tissue and may be part of the normal aging process. It offers no resistance to advancement of the scope. Grade I fibrosis is unlikely to cause clinical symptoms.

Grade II: Strings and sheets of fibrosis increase in size and number and form a more continuous network. Inflammatory changes are often present as well. The fibrosis is starting to replace the epidural fat. There is some resistance to the scope, but the scope can still be advanced. Maneuvering the scope may be painful. It is clearly abnormal and is likely part of a pathological process that leads to clinical symptoms.

Grade III: Fibrosis is continuous and occupies discrete areas of the epidural space. There is no epidural fat and inflammation may be less pronounced. The scope cannot be advanced in these areas and attempts to do so are painful.

Grade IV: Fibrosis is now one large dense mass that is continuous and occupies an anatomical region of the epidural space (Fig. 50.5). The scope cannot be advanced at all. It is important to recognize that epidural fibrosis is associated with multiple different pathological processes and should be placed in the appropriate clinical context. For example, after back surgery, the dura may adhere to the walls of the spinal canal and be covered by strings and sheets of fibrosis. In this case, the scope cannot be advanced due to dural adhesions. This may look like a dense mass of fibrosis, but it is not. Large disc herniations may also appear as a white irregular fibrotic (grade IV) mass that are impossible to penetrate with the scope. This may be extruded disc material or a fibrotic response to chronic inflammation.

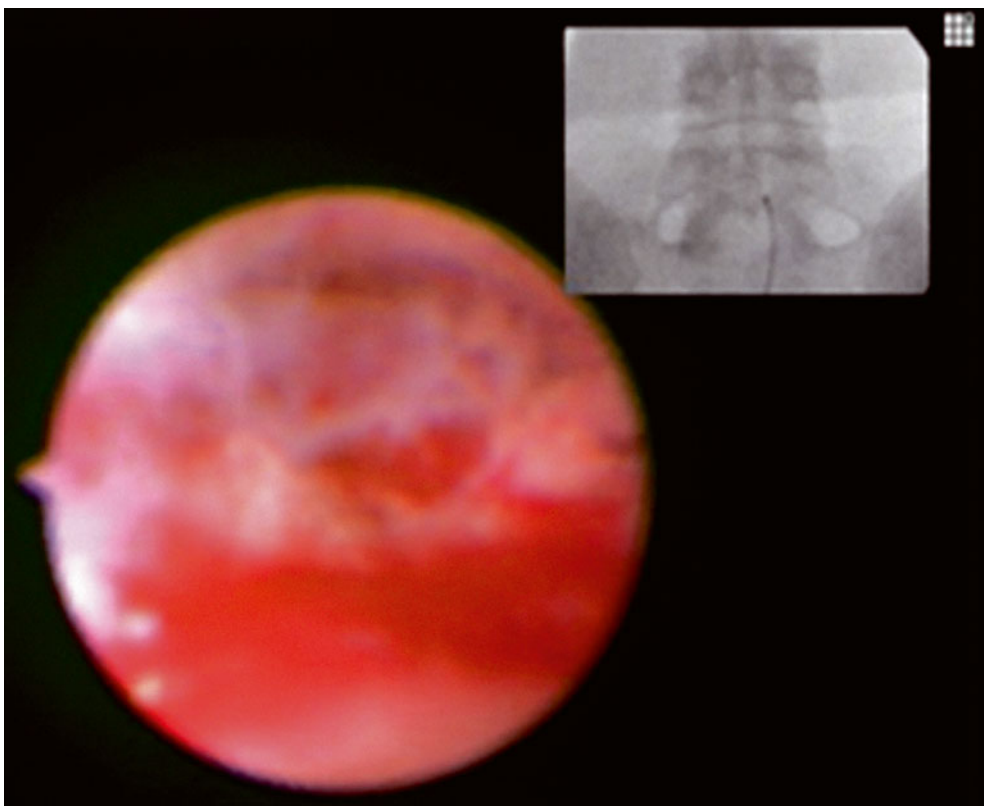


Fig. 50.5 Lt L–S junction—dense (Grade 4) avascular fibrosis (With permission from James Heavner)

Nerve Roots

Nerve roots are surprisingly hard to find for structures that occupy 30 % of the neuroforamen. The S3, S4, and S5 nerve roots are usually missed completely. This may explain why damage to these structures is rare. The S2 nerve root is easier to find, as it is larger in size. The S1 nerve root can be found in the lateral recess of the sacral epidural space on its way to the sacral neuroforamen. It passes medial and inferior to a bony structure equivalent to the pedicle of the lumbar vertebra. This is commonly the site of fibrosis and may explain persistent radicular pain in the S1 distribution after a L5/S1 fusion of the lumbar spine. The lumbar nerve roots can be found below and medial to the pedicle, the perpendicular space. A characteristic blood vessel accompanies the nerve root and can be seen through the root sleeve. Under normal conditions, this vessel is straight; with pathology it becomes dilated and gives an effaced appearance. A tortuous vessel may be a sign of circulatory obstruction. The nerve root sleeve itself can make a large dilated appearance. Touching a healthy nerve root is not painful although a paresthesia may be felt. However, in the case of pathology, touching the nerve with a scope is often very painful.

Discs, Dura, and Ligaments

As mentioned earlier, disc and disc material is difficult to identify. On gross examination, i.e., during surgery, the disc is easily identified by its white almost yellow appearance in contrast to the darker brown vertebral bone. It is only partially covered by the longitudinal ligament. The disc can probably be seen during epiduroscopy by placing the scope in the neuroforamen just superior to the pedicle; however, color, texture as seen through the scope, and, in addition, the presence of obscuring epidural fat make it difficult to obtain useful information with respect to disc pathology. A known disc herniation is characterized by the absence of epidural fat and the inability to place the scope in the neuroforamen. A dense white mass obstructs the advancement of the scope. Areas of inflammation and fibrosis may be seen as well. One does not see a nice smooth, possible red disc protrusion (compressing a nerve root) as shown in textbook pictures.

The dura is recognized by its white smooth appearance. It may pulsate with changes in CSF pressure. Blood vessels can be seen on the dura as may be fibrotic structures, possibly ligaments. In pathology, numerous areas of increased vasculature, hyperemia, and fibrosis cover the dura. These areas of inflammation may be continuous with inflammation that accompanies the nerve root. Manipulation of the scope is usually painful.

Peridural Membrane

The peridural membrane is a generally overlooked membrane between the dura and bony vertebrae [1, 5]. We became aware of the membrane when doing epiduroscopy. We are pursuing investigation of possible significant role of the membrane as a focus of pathology causing common low back pain.

Technical Aspects of Epiduroscopy

The Scope

We use an epiduroscope marketed by Karl Storz (Figs. 50.6 and 50.7). The usual equipment for endoscopic procedures and any flexible fiberoptic endoscope may be used as long as the images are of reasonable quality.

Fig. 50.6 Epiduroscope flexible, 2.8 mm, 40/70 cm long marketed by Karl Storz Endoskope, Tüttlingen (With permission from Danilo Jankovic)

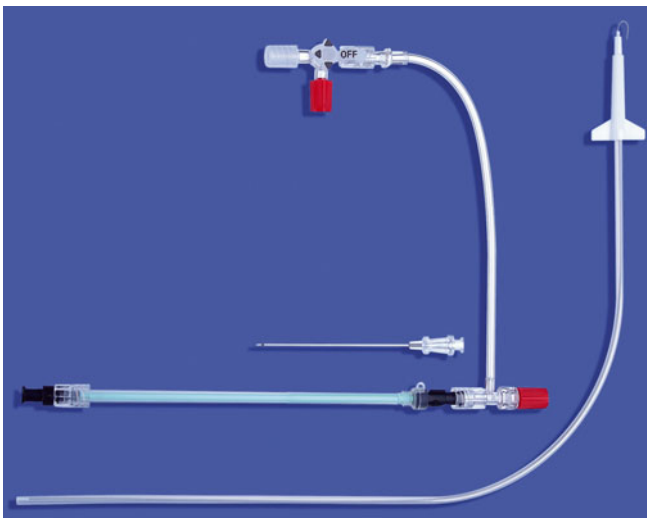


Fig. 50.7 Materials (With permission from Danilo Jankovic)

The Procedure

At this time only one acceptable route of insertion of the scope, placement through the sacral hiatus is recommended (Figs. 50.8 and 50.9). With appropriate application of local anesthetics, the procedure can be performed without the help of an anesthesiologist. However, because of the nature of the procedure, the requirement of endoscopy, and fluoroscopy equipment, we perform the procedure in the operating room under monitored anesthesia care. Prophylactic treatment with an antibiotic may be considered. The patient is put in the prone position in such a fashion that the lumbar lordosis is minimized. The sacral area is cleansed and the skin anesthetized; an 18-G needle is advanced through the sacral hiatus in the sacral epidural space. With the assistance of fluoroscopy, a Seldinger technique is used to place a nine- or ten-French introducer in the epidural cavity.

Guidewire, dilator, and introducer sheet should all be kept below the level of S3, so as to avoid penetration of the dura and damage to the neural structures. To reduce procedure related pain, we may inject 5 ml of 0.2 % ropivacaine through the sheath before inserting the epiduroscope. The scope is then advanced into the sacral epidural cavity. In general the procedure can be performed with minimal trauma. Occasionally, the scope cannot be passed through the distal sacral spinal canal. Attempts to pass the scope with force will almost certainly result in damage of the scope and possible complications to the patient. Once the scope is placed in the sacral epidural cavity, fluoroscopy is used to obtain the proper orientation. In patients with unilateral pathology, it is advisable to inspect the contralateral epidural cavity first. This will provide the endoscopist with information about what the normal epidural space looks like or asymptomatic pathology may be identified. The scope is then advanced to the region of pathology as predicted by the clinical exam and imaging studies. During the procedure, fluoroscopy will be needed to confirm the orientation of the scope. Judicious amount of isotonic fluids is infused through the working channel. These fluids will dilate the epidural space and make visualization of the epidural tissues possible. Limits to the amount and rate of fluids injected are not known. Increase in pressure will be dependent on the rate of injection and the compliance of the system. Since the neuroforamen provide a continuous exit route for injected fluids, thus significant amounts of fluids can be injected without significant consequences. In patients with pathology, such as extensive epidural fibrosis, rapid injections may lead to pressure waves in the epidural and subarachnoid space that may lead to severe complications [7]. We plan to inject no more than 60 ml although much larger amounts have been injected. Pain between the scapulae or in the neck and headache are warning symptoms that signal pressure is increasing to dangerous level.

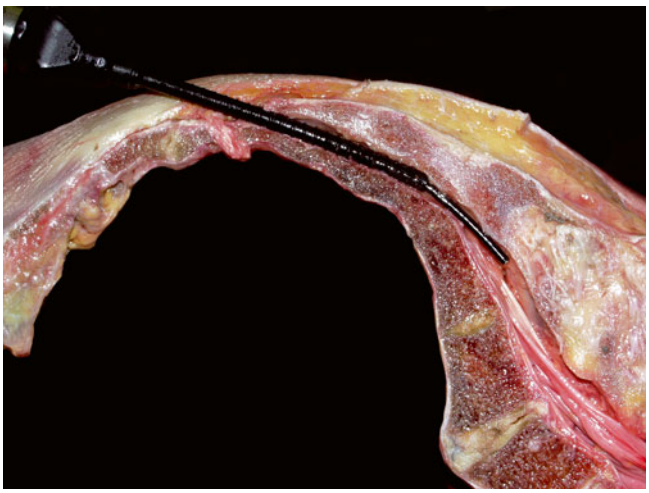


Fig. 50.8 Puncture of the caudal epidural space (With permission from Danilo Jankovic)

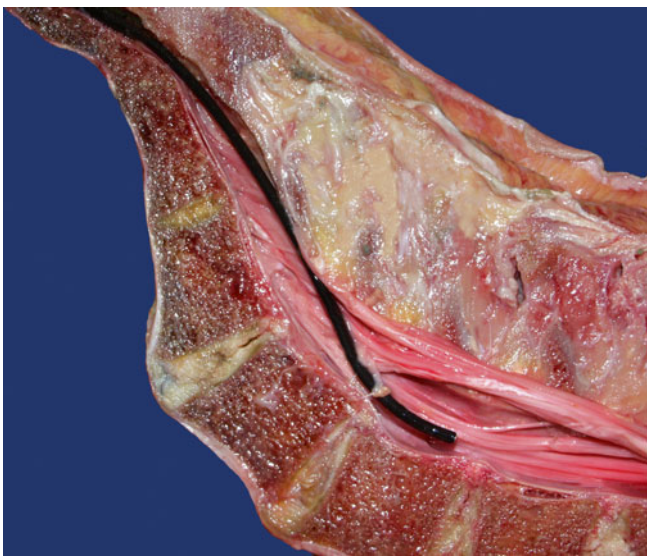


Fig. 50.9 Inspection of the epidural space (With permission from Danilo Jankovic)

Treatment of Radicular Pain Using Epiduroscopy

Targeted Drug Delivery

When an area of pathology is identified, treating the abnormality by local drug injection through the working channel of the scope may be attempted. The objectives of such an injection in the treatment of radiating pain include the reduction or prevention of inflammatory lesions adjacent to the nerve root and reduction or prevention of epidural fibrosis through chemical adhesiolysis. Placement of glucocorticosteroids at a site of inflammation may have an advantage over injection in the epidural cavity through more standard methods. In addition, the nerve root is small relative to the size of the neuroforamen, and the epidural cavity is largely patent near the nerve root.

Mechanical Adhesiolysis

One of the most impressive images through epiduroscopy is that one of epidural fibrosis. Indeed, epidural scarring has been implicated in failed back surgery and chronic radicular pain. The exact mechanism by which fibrosis may cause neuropathic pain is unclear, but local ischemia may play a role. Removal of perineural fibrosis may lead to improved local circulation and decrease of neuropathic symptoms. Multiple attempts to remove fibrosis or methods to prevent the buildup of epidural scar after surgery have been attempted with variable success. Several investigators have reported on attempts to decompress nerve roots using epiduroscopy. Multiple mechanisms by which this may be achieved may play a role. Advancement of the scope in areas of fibrosis may have a mechanical lytic effect on fibrosis. Obviously, the effect will be more pronounced in areas of light fibrosis (grade I or II) than in dense epidural scarring (grade III and IV). Placement of the scope in the space between the nerve root and pedicle may be an area of interest as inflammatory material seems to accumulate here and fibrosis is not too extensive. Significant amounts of normal saline are used to dilate the epidural space in order to obtain images. These normal saline injections in relatively closed compartments may lead to build up of pressure and result in effective adhesiolysis. Forceful injections with saline, contrast material, hyaluronidase, and other drugs have been studied. Outcomes of these injections have been variable. The effectiveness of these forceful injections on radicular pain is most likely highly dependent on the precise placement of the scope in the neuroforamen at the site of the pathology. Experience suggests that with precise placement of the scope, improvement of the epidurogram, i.e., improved patency of the nerve root in the neuroforamen and lateral recess, is associated with clinical improvement. Several studies support this notion. Of course these kind of observations need to be substantiated by more rigorously controlled studies. More involved techniques such as the use of fiberoptic LASER in the ablation of fibrous material have been studied [13]. This might be helpful but we have reservations about doing it.

Combined Adhesiolysis and Targeted Drug Delivery

There are an increasing number of studies that report on the effectiveness of epiduroscopy in the treatment of chronic radicular pain. In general, these studies use a combination of drug delivery and mechanical adhesiolysis. Corticosteroids are injection with local anesthetics. A mixture of clonidine, hyaluronidase, and hypertonic saline may be injected in addition to variable amounts of saline to dilate the epidural space. The emphasis is on fluoroscopic guidance of the scope to the area of the pathology. Very little description of epidural pathology as observed through the scope is provided, i.e., in these studies the scope is mainly used as a tool for targeted drug delivery, and the exact target is not always clear.

Indications and Contraindications

Precise guidelines with respect to indications and contraindications for epiduroscopy are becoming better defined as our experience with the procedure increases.

Epiduroscopy is indicated in patients with radicular symptoms, which do not respond to epidural steroid injections or surgical intervention. For example, this may be in symptomatic patients with minimal changes on lumbar MRI who have a temporary response to epidural steroid injections only. Or this may be a patient who has had a surgical nerve root decompression with or without fusion with persistent radicular pain. In addition, patients with low back pain without radicular symptoms, who have responded to epidural steroid injections in the past, may benefit from further investigation of the epidural cavity.

As any other invasive procedure, epiduroscopy is contraindicated in patients with systemic infections, in patients with regional infection at the site of insertion, and in patients with bleeding disorders. The caudal area is prone to contamination, and the tract to the sacral hiatus may be a port of entry to the spinal canal. A prophylactic dose with antibiotics (e.g., ceftriaxone, ciprofloxacin) is recommended. Neurological deficit is a relative contraindication since most of the patients will have neurological abnormalities. Since decompression of the sacral nerve roots may lead to improved bladder and bowel function, neurogenic incontinence should

not be an absolute contraindication to the procedure. The nerve roots of S2–S5 are small and difficult to find in the epidural space; damage is unlikely but not impossible. Prior urodynamic evaluation in patients with symptoms of neurogenic bladder will provide a baseline status.

Anatomic abnormalities of the sacrum may preclude placement of the scope but is often feasible with fluoroscopic guidance. However, resistance to scope placement may lead to scope damage and complications. Interestingly, the distorted anatomy seen with Tarlov's cysts does not seem to interfere with epiduroscopy. Complication may arise from increased epidural and intracranial pressure with the infusion of dilating fluids in the epidural space. Therefore epiduroscopy in patients with increased intracranial pressure is contraindicated. Significant resistance to injection of fluids may indicate low compliance of the epidural space and forms a relative contraindication. Locally increased pressure may lead to circulatory abnormalities associated with compartment syndrome, with potentially devastating neurological consequences. Since significant amounts of contrast material may be used during epiduroscopy, impaired kidney function, particularly in the elderly, and caution is advised in its use.

Complications

Complications of epiduroscopy are similar to other spinal procedures and include infection, bleeding with hematoma formation, and drug reactions [8, 10, 17]. Some complications are specific to epiduroscopy. Trauma to the sacral hiatus and canal may occur but can be minimized with proper technique. Such trauma may result in persistent pain after the procedure. Infection may lead to a fistula or abscess. Any sign of infection at the puncture site should therefore be treated aggressively. Manipulation of the scope in the epidural spinal canal may lead to direct damage to neural structures or to circulatory compromise of these structures. In particular, this may occur in a region of pathology when injected drugs loculate and the extramural pressure increases sufficiently to jeopardize local blood flow. In addition the dura may be punctured by the scope, especially in areas of pathology. Gentle manipulation of the scope and use of direct vision may prevent this from happening. The dura is surprisingly resilient to puncture by the scope. On occasion, the dural sac extends far into the sacral epidural space. Aspiration of CSF and the typical appearance of the subarachnoid space on first inspection will show that entry occurred and will prevent the epiduroscopist from causing damage to neural structures.

Persistent CSF leak has been reported; however, complications from this may be limited since the sacral epidural space is not a closed space.

One of the more feared complications of epiduroscopy is visual disturbance [8]. Rapid epidural infusion generating a pressure gradient may explain disruption of retinal circulation and result retinal hemorrhages. Avoiding infusion pressures and significant pressure changes may prevent these complications. Advancement of a scope through a too narrow sacral epidural canal at the hiatus may damage the scope to the point of breakage of the scope requiring surgical removal. If significant resistance to the scope is encountered, the procedure should be aborted.

References

1. Ansari S, Heavner JE, McConnell DJ, Azari H, Bosscher H. The peridural membrane of the spinal canal: a critical review. Pain Pract. 2012;12:315–25.
2. Bosscher HA, Heavner JE. Diagnosis of the vertebral level from which low back or leg pain originates. A comparison of clinical evaluation, MRI and epiduroscopy. Pain Pract. 2012;12:506–12.
3. Bosscher HA, Heavner JE. Incidence and severity of epidural fibrosis after back surgery: an endoscopic study. Pain Pract. 2010;10:18–24.
4. Bosscher HA, Heavner JE. Lumbosacral epiduroscopy finding predict treatment outcomes. Pain Pract. 2014;14:506–14.
5. Bosscher HA, Heavner JE. Treatment of common low back pain: a new approach to an old problem. Pain Pract. 2014;14. doi:10.1111/papr.12224. Article first published online 24 Jun 2014.
6. Geurts JW, Kallewaard JW, Richardson J, Groen GJ. Targeted methylprednisolone acetate/hyaluronidase/clonidine injection after diagnostic epiduroscopy for chronic sciatica: a prospective, 1-year follow-up study. Reg Anesth Pain Med. 2002;27:343–52.
7. Gill B, Heavner JE. Visual impairment following fluid injection and epiduroscopy: a review. Pain Med. 2005;6:367–74.
8. Heavner JE, Bosscher HA. Complications of lumbosacral epiduroscopy. Pain Clin. 2007;19:178–84.
9. Heavner JE, Bosscher HA, Wachtel M. Cell types obtained from the epidural space of patients with low back pain/radiculopathy. Pain Pract. 2009;9:167–72.
10. Heavner JE, Wyatt DE, Bosscher HA. Lumbosacral epiduroscopy complicated by intravascular injection: a report of two cases. Anesthesiology. 2007;107:347–50.
11. Helm S, Hayek SM, Colson J, et al. Spinal endoscopic adhesiolysis in post lumbar surgery syndrome: an update of the assessment of the evidence. Pain Phys. 2013;16:SE125–50, ISSN 2150–1149.
12. Igarashi T, Hirabayashi Y, Seo N, Saitoah K, Fukuda H, Suzuki H. Lysis of adhesions and epidural injection of steroid/local anaesthetic during epiduroscopy potentially alleviate low back and leg pain in elderly patients with lumbar spinal stenosis. Br J Anaesth. 2004;93:181–7.
13. Jo DH, Kim ED, Oh HJ. The comparison of the result of epiduroscopic laser neural decompression between FBSS or not. Korean J Pain. 2014;27:63–7.
14. Kallewaard JW, Vanelderen P, Richardson J, et al. Epiduroscopy for patients with lumbosacral radicular pain. Pain Pract. 2014;14:365–77.
15. Manchikanti L. The value and safety of epidural endoscopic adhesiolysis. Am J Anesthesiol. 2000;27:275–9.
16. Manchikanti L, Pampati V, Bakhit C, Pakanati R. Non-endoscopic and endoscopic adhesiolysis in post lumbar laminectomy syndrome. A one-year outcome study and cost effectiveness analysis. Pain Physician. 1999;2:52–8.

17. Racz GB, Heavner JE. Complications associated with lysis of epidural adhesions and epiduroscopy. In: Rathmell JP, Neal JM, co-editors. Complications in regional anesthesia and pain medicine. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2012. p. 373–84.
18. Richardson J, McGurgan P, Cheema S, Gupta S. Spinal endoscopy in chronic low back pain with radiculopathy: a prospective case series. Anaesthesia. 2001;56:454–60.
19. Ruetten S, Meyer O, Godolias G. Endoscopic surgery of the lumbar epidural space (epiduroscopy): results of therapeutic intervention in 93 patients. Min Invasive Neurosurg. 2003;46:1–4.
20. Saberski L. A retrospective analysis of spinal canal endoscopy and laminectomy outcome data. Pain Physician. 2000;3:193–6.
21. Saberski LR, Kitahata LM. Direct visualization of the lumbosacral epidural space through the sacral hiatus. Anesth Analg. 1995;80:839–40.
22. Saberski L. Spinal Endoscopy: Current Concepts. In: Waldman S, editor. Interventional Pain Management. 2nd ed. Philadelphia: WB Saunders 2001. p 143–61.
23. Schütze G, Kurtse G, Grol O, Enns E. Endoscopic method for the diagnosis and treatment of spinal pain syndromes. Anesteziol Reanimatol. 1996;4:62–4.
24. Vanelderen P, Van Boxem K, Van Zundert J. Epiduroscopy: the missing link connecting diagnosis and treatment? Pain Pract. 2012;12:499–501.

Chapter 51

Percutaneous Epidural Neuroplasty

James E. Heavner, DVM, PhD (✉) • Gabor B. Racz, MD • Miles Day, MD
Department of Anesthesiology, Texas Tech University Health Sciences Center, Lubbock, TX, USA
e-mail: james.heavner@ttuhsc.edu

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_51,

Introduction

Percutaneous epidural neuroplasty (epidural neurolysis, epidural adhesiolysis) is a form of interventional pain treatment that was first described in 1989 [17]. The method is used at all levels of the spine to treat neuraxial pain conditions or radiculopathies, or both, as well as certain forms of cervicogenic headache.

Over the years since the technique was introduced, it has been used in many parts of the world on thousands of people. When performed by properly trained personnel using appropriate equipment, epidural neuroplasty is widely recognized to be safe, effective, and cost-effective [2, 5, 8, 9]. Various modifications of the original technique have been tried (equipment and drugs, including various doses and volumes). However, all modifications have been based on the original ideas that lead to the introduction of percutaneous epidural neuroplasty. Perhaps the biggest gains have been in understanding the scope of potential complications and how to avoid or mitigate them [16].

Originally introduced as a 3-day procedure with one infusion given on each of 3 sequential days, we now give the three infusions in 2 days. The efficacy of a 1-day treatment has been documented [11]. The focus of this chapter is on the 2-day caudal neuroplasty procedure most commonly performed at our institution.

The development of this procedure and its acceptance were promoted by the following factors: (a) information regarding the importance of epidural and intervertebral structural changes and their role in the development of back pain and radicular pain, (b) better understanding of the structures involved in the origin of pain in the epidural space and its surroundings, (c) data on the type and location of pain arising due to stimulation of certain pathological structures in the epidural space and its vicinity, (d) the development of reliable percutaneous puncture techniques to access the epidural space, (e) recognition of epidurography as a valuable method of diagnosis and treatment, (f) clear guidelines and theoretical justifications for the procedure and the drugs used, (g) evidence of the effectiveness of the treatment, and (h) USCPT code provided for the procedure.

The aims in percutaneous epidural neuroplasty are:

1. To diagnose pathological changes in the epidural space (e.g., epidural fibrosis) that may prevent administered drugs from reaching areas where there is pathology. Radiographic contrast media is used to identify the filling defects.
2. To remove all pathological obstructions and scar tissue. For this purpose, physiological saline mixed with hyaluronidase is applied to the scar tissue.
3. To determine whether the pathological obstructions causing pain have been removed after a procedure. Radiographic contrast media is again injected for this purpose.
4. To administer drugs directly to the area of pathology to relieve or reduce pain (local anesthetics, steroids, and hypertonic saline).

The Origins of Back Pain and Sciatica

When conducting surgery under local anesthesia in the lumbar spine, Kuslich et al. [6] found that sciatica could be triggered by irritation of swollen, overextended, or compressed nerve roots. By contrast, back pain could be triggered by stimulation of various tissues in the lumbar region—most frequently in the outer layer of the anulus fibrosus and posterior longitudinal ligament. Roffe [19] showed that both of these structures are richly supplied with nerves connected to the CNS via meningeal branches (sinuvertebral nerves) (Fig. 51.1). By contrast, stimulation of the capsule of the facet joints rarely caused back pain and never caused sensitivity in the synovial bursa or cartilaginous surfaces. In patients who had previously undergone a laminectomy, there were always one or more areas of marked perineural fibrosis. It was never the scar tissue itself that was sensitive; instead, there was often marked irritability in the nerve root. It is suspected that the scar tissue immobilizes the nerve root and thereby favors the development of pain when the nerve root is subject to traction or pressure.

Kusslich et al. concluded that "Sciatica can only be caused by direct pressure or traction on an inflamed, stretched, or compressed nerve root. No other tissue in the spine is able to trigger pain in the leg."

However, nerve roots are exposed not only to mechanical effects but also to proinflammatory material from degenerated intervertebral disks or facet joints [14].

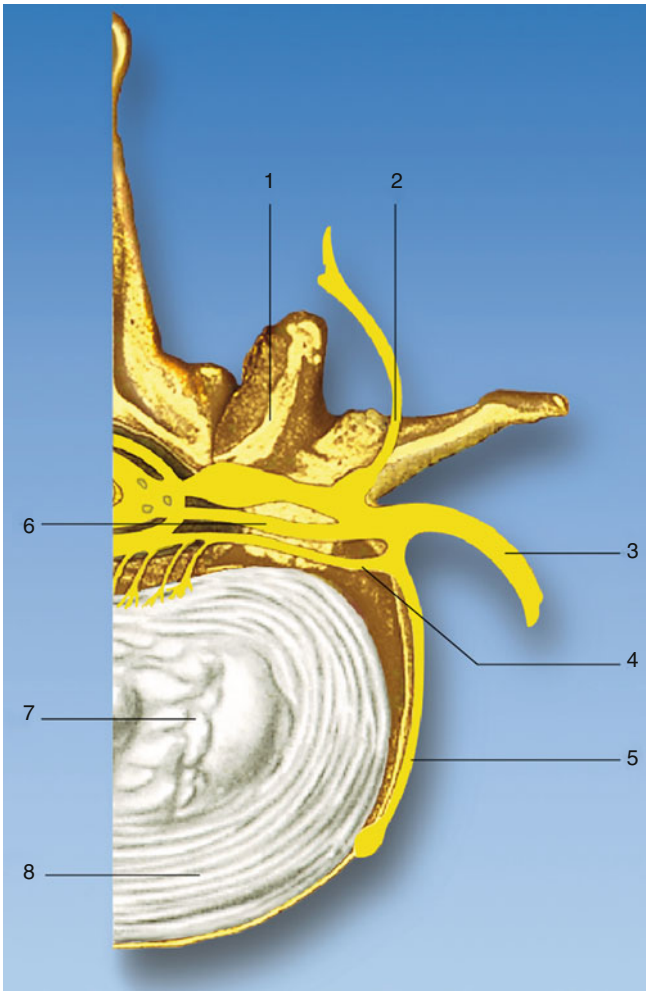


Fig. 51.1 One half of a cross-section through the spinal cord, showing the innervation. As described in the text, the sinuvertebral nerve supplies the anulus fibrosis and the posterior longitudinal ligament—two structures that play an important role in the development of back pain. (*1*) Facet, (*2*) posterior primary ramus, (*3*) anterior ramus, (*4*) sinuvertebral nerve, (*5*) sympathetic, (*6*) root, (*7*) nucleus, (*8*) anulus (With permission from Danilo Jankovic)

Lower Level for Pressure (Fig. 51.2)

The effects of pressure exerted on the nerves depend on whether the pressure is high or low. High pressure can produce direct mechanical effects on the nerve tissue and can distort the nerve fibers, shift the nodes of Ranvier, or press in the paranodal myelin sheath. Lower pressures lead to tissue changes caused by a reduction in the blood supply to the nerve tissue. In animal experiments, it has been found that when inflation of a balloon attached to the spinal cord makes the pressure on the cauda equina equivalent to arterial pressure, blood flow in the cauda equina is interrupted [14]. Even a pressure of 5–10 mmHg interrupts venous blood flow in some small veins, while a pressure of 10 mmHg reduces the transport of nutrients to the nerve roots by 20–30 %. Compression can also cause changes in the permeability or transmural pressure conditions in the endoneurial capillaries in the nerve roots and can lead to edema formation (in the animal experiment, e.g., this occurred after 2 min of compression at 50 mmHg).

Intraneural edema due to chronic nerve injury is associated with the development of neural fibrosis, which may contribute to the very slow rate of symptomatic improvement observed in some patients with nerve compression.

Compression of spinal nerves at 10 mmHg for 2 h by two adjacent balloons (to simulate the clinical conditions occurring in multiple nerve compression) reduced neural conduction and led to a reduction in the recorded amplitudes of action potentials by ca. 65 %. By contrast, compression by a single balloon at 50 mmHg for 2 h did not alter the amplitude of the action potentials. A pressure of 10 mmHg (with incomplete blockage of small veins) only appears to be capable of causing changes in nerve function if the spinal nerve roots are compressed into two segments [14]. Intervertebral disk prolapses or protrusions can cause higher levels of compression pressure than central spinal stenosis.

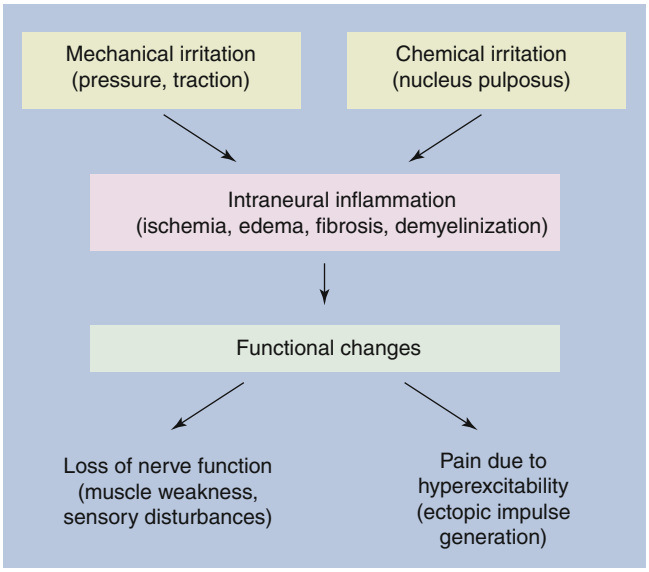


Fig. 51.2 Mechanical and chemical stimulants trigger the development of neuraxial pain and radiculopathy, as well as cervicogenic headache [12]

Chemical Irritation (Fig. 51.2)

Some chemical substances have been identified in the nucleus pulposus that can lead to irritation of neighboring structures if tears in the anulus fibrosus lead to them being released into the vertebral canal. These substances, which can cause inflammation of nerve roots and meninges, include lactic acid, glycoprotein, cytokines, and histamine. In addition, it is considered theoretically possible that components of the nucleus pulposus can act as foreign proteins and trigger an autoimmune reaction. This type of chemically caused irritation can arise without any compression by an intervertebral disk.

Structural Changes

Intervertebral disk anomalies can manifest as degenerative changes, protrusion, or herniation. Constriction of the intervertebral space due to disk injury is often associated with osteophyte formation and arthrosis of the facet joints, which can lead to increased pressure on the spinal nerves. Stretching of the posterior longitudinal ligament by protruding intervertebral disks initially leads to localized back pain, while more severe protrusions also cause pressure on neighboring nerve roots and can lead to radicular pain.

Theoretical Considerations

In patients with chronic neuraxial pain and/or radiculopathy or cervicogenic headache, one or more of the following pathological changes may be present (Fig 51.2):

- Inflammation
- Edema
- Fibrosis
- Venous engorgement
- Mechanical pressure on:
  - Posterior longitudinal ligament
  - Anulus fibrosus
  - Spinal nerve
- Reduced or absent nutrient supply to the spinal nerves or nerve roots
- Central sensitization

The inflammatory tissue changes can activate nociceptors or axons that conduct nociceptive information to the CNS. Equally, owing to inflammation, nociceptors or nociceptive axons may react more sensitively to mechanical stimuli. This type of mechanical irritation may be triggered either by pressure stress, as described above, or may be caused by movement-dependent stretching due to entrapment of spinal nerves or nerve roots by fibrous tissue.

Most experience with neuroplasty has been gathered in the treatment of chronic pain conditions. The patients probably have both peripheral and central changes (e.g., central sensitization) contributing jointly to the chronic pain condition.

It is therefore theoretically justifiable to treat back pain with or without radiculopathy by local administration of drugs. Possible forms of treatment include:

- Anti-inflammatory drugs (e.g., corticosteroids).
- Drugs that reduce edema formation (e.g., hypertonic saline 10 %, hyaluronidase, and corticosteroids).
- Local anesthetics to block the nerve fibers that conduct pain information to the CNS (hypertonic saline also has a local anesthetic effect).
- Addition of hyaluronidase to remove scar tissue. This makes it possible for the drug being used to reach the target tissue.

Figure 51.3 shows the selection criteria for patients who are candidates for percutaneous epidural neuroplasty; noninvasive, conservative treatment methods should be attempted first. Our technique is described in Table 51.1. Table 51.2 lists the most frequently used injection solutions.

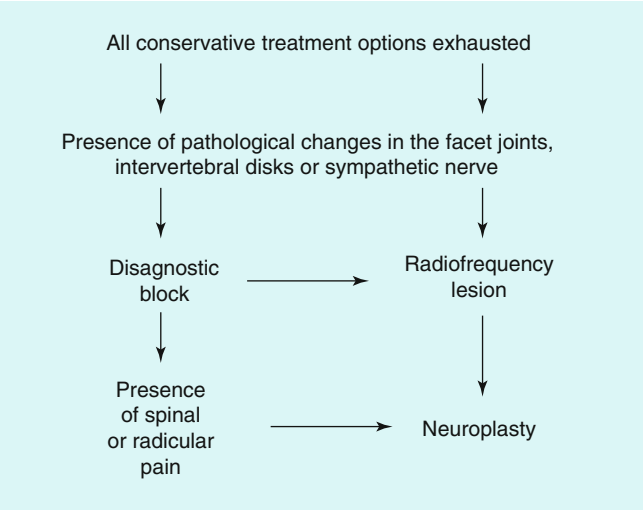


Fig. 51.3 Algorithm for the treatment of neuraxial pain and radiculopathy, as well as cervicogenic headache. Neuroplasty is considered when conservative forms of treatment have proved to be ineffective and the appropriate diagnosis is confirmed

Table 51.1 Overview of neuroplasty

In the operating room:
1. Placement of the epidural needle
2. Injection of contrast and imaging of its spread (epidurogram)
3. If there is a filling defect in the area in which the pain is located, introduction of a Racz catheter into the filling defect (scar)
4. Repeat contrast injection to ensure that the contrast is now also spreading in the area of the filling defect
5. Injection of preservative-free saline, with or without hyaluronidase
6. Injection of local anesthetic and corticosteroids
7. Secure the catheter
In the recovery room:
8. 30 min after the injection of a mixture of steroids and local anesthetic, infusion of 10 mL hypertonic saline (10 %) over 30 min
On the ward:
9. On the following day, inject local anesthetic followed 30 min later by hypertonic saline (10 %) twice with a 6–8 h interval between injections
10. The epidural catheter is removed after the final treatment

Table 51.2 Injected solutions according to the spinal cord section (volume in mL) in the injection sequence

Solution	Cervical	Thoracic	Lumbar	Caudal
Iohexol	2–3	4–6	10	10
Iohexol	1–2	2–3	3	3
Saline 0.9 % + 1,500 IU hyaluronidase	4–6	6–8	10	10
Ropivacaine 0.2 % + corticosteroid[a] (test dose)	2	2	3	3
Ropivacaine 0.2 % + corticosteroid[a]	2–4	4–6	7	7
Hypertonic saline	4–6	6–8	10	10
Saline 0.9 %	2	2	2	2
Then, on each of the 2 following days:				
Ropivacaine 0.2 % + corticosteroid (test dose)	2	2	3	3
Ropivacaine 0.2 %	4	6	7	7
Hypertonic saline	4–6	6–8	10	10
Saline 0.9 %	2	2	2	2

[a]4 mg dexamethasone or 40 mg methylprednisolone or triamcinolone

Techniques of Cervical Epidural Neuroplasty

Caudal Access Route

Procedure

Careful Information Discussion with the Patient Before the Block

Full prior information for the patient is mandatory. The patient should be informed about all of the potential complications that can occur during and after the procedure (e.g., epidural hematoma, epidural abscess, numbness in the extremities, rectal or bladder emptying disturbances, paralyses, infection, sexual dysfunction, shearing of the epidural catheter, etc.).

Materials

See Chap. 47 on epidural caudal anesthesia.

16-G or 15-G RX Coudé needles—fluoropolymer-coated epidural catheter made of stainless steel with a spiral tip TunL Kath or TunL-XL (stiffer); Epimed International, Inc., Irving, Texas, USA).

Preparations

See chapter on epidural caudal anesthesia.

Intravenous access is required in order to treat potential adverse events (e.g., total spinal anesthesia, subdural injection, intravascular injection, etc.), as well as to administer analgesia and sedation during the procedure and antibiotics postoperatively.

Analgesia and sedation are recommended before the procedure (e.g., 1–2 mg midazolam + 25–50 µg fentanyl), as the injection is usually painful in patients with epidural adhesions. The injection pain is probably caused by stretching of the nerve roots affected, and it spreads in the corresponding cutaneous innervation area. The patient should not receive deep sedation. The patient needs to be capable of cooperating during the procedure to ensure that any signs of spinal cord compression are not overlooked. (The patient has to be able to move the extremity affected and report any weakness or paralysis during the procedure.)

All procedures are conducted under fluoroscopic guidance, using a C-arm with a storage function in pulse mode (reduced radiation exposure). Fluoroscopic guidance optimizes the results of the procedure (correct needle positioning, easier identification of the defect, ability to check the spread of the contrast, and correct positioning of the catheter). The usual protective measures for staff are obligatory.

Selection of Drugs

Radiographic Contrast Media

To exclude inadvertent subarachnoid injection, a water-soluble, nonionic contrast medium, i.e., iohexol 240, is used. In our experience, the presence of epidural adhesions increases the risk of subarachnoid injections. Subarachnoid injection of a contrast medium that is not water soluble can lead to serious complications (spinal cord irritation, spinal cramp or clonus, arachnoiditis, paralysis, and death).

Local Anesthetic

For example, 0.2 % ropivacaine.

Corticosteroids

The choice of the corticosteroid to be used (Table 51.2) mainly depends on which agents are available. Long-acting steroid emulsions have a particle size of ca. 20 µm and it is therefore not possible to inject them through bacterial filters.

Hypertonic Saline (10 %)

The local anesthetic effect of hypertonic saline is used to prolong the intended pain relief so that the patient can receive physiotherapy twice a day. The osmotic action of hypertonic saline reduces edema.

Antibiotics

Thirty minute before the start of the procedure, 1 g ceftriaxone (Rocephin) is administered intravenously. During the hospital stay, the same dose is administered every 24 h. Patients who are allergic to penicillin receive 500 mg ciprofloxacin (Ciprobay) or levofloxacin orally 1 h before the procedure, as well as over the following 5 days (500 mg cephalexin or ciprofloxacin every 12 h or 500 mg levofloxacin every 12 h).

Technique

Patient Position

The patient is placed in the prone position on the fluoroscopy table.

Landmarks

The sacral region is prepared and covered with sterile drapes, and the sacral cornua and sacral hiatus are palpated. The puncture site is located in the gluteal cleft *opposite the affected side*, approximately 1 cm lateral to and 2.5 cm below the sacral hiatus. From this point, it is easier to guide the needle and catheter toward the affected side. The lateral access reduces the risk of the needle or catheter penetrating the dural sac or subdural space.

Local Anesthesia

The puncture site is anesthetized by injecting locally 1 % lidocaine.

Puncture

After skin puncture with an 18-gauge A-bevel needle, an epidural needle (preferably a 16-G or 15-G RX-Coudé needle; Fig. 51.4) is introduced into the sacral hiatus.

Tuohy needles should not be used.

The needle is advanced until the tip is caudal to the S3 foramen. Lateral fluoroscopy is used to check that the needle is positioned inside the osseous canal. This radiographic check is particularly important when there are unusual anatomical features in the sacral bone. Anteroposterior radiography is used to check that the needle tip is directed toward the affected side. A check is then made for escaping fluid (CSF or blood), and an aspiration test is performed. After a negative aspiration test, 10 mL iohexol (Omnipaque 240) is injected under fluoroscopic guidance. When injected into the epidural space, the contrast forms a *Christmas-tree-shaped distribution pattern*. The presence of epidural adhesions prevents the contrast from spreading in this characteristic pattern, with the affected nerve roots being omitted.

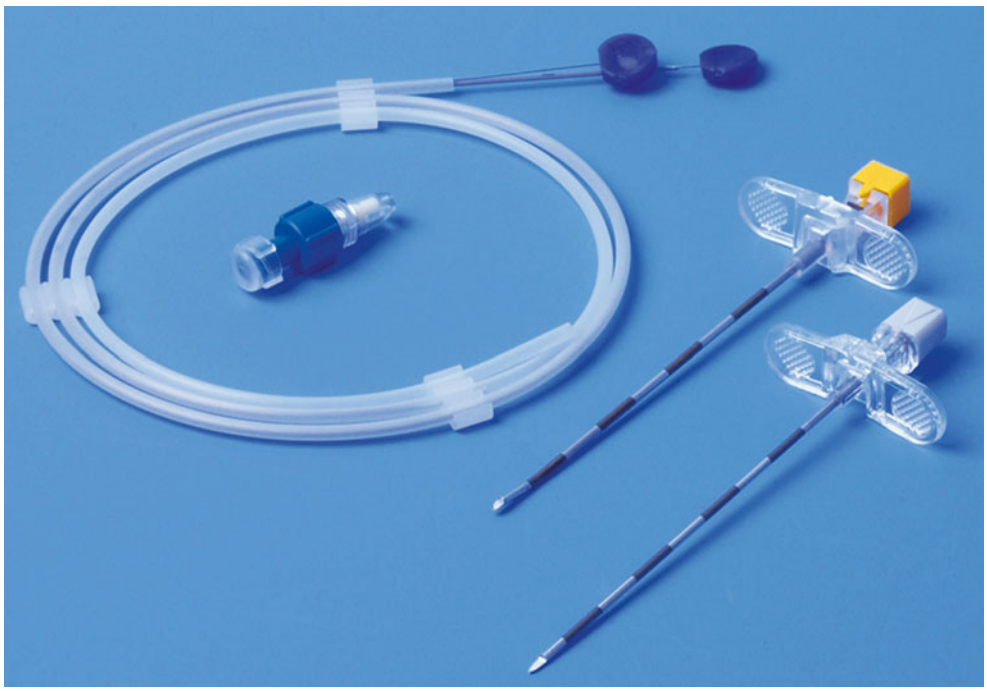


Fig. 51.4 Materials. 16-G or 15-G RX Coudé needles; fluoropolymer-coated epidural catheters made of stainless steel with spiral tip (TunL Kath, Epimed International, Inc., Irving, Texas, USA)

Problem Situations

Subarachnoid Needle Location

If the puncture needle is in a subarachnoid location, the contrast medium spreads in a central and cranial direction. If it is in a subdural position, the contrast spreads in a similar fashion but not as far as with a subarachnoid injection. Despite this, the contours of the nerve roots and dura are visible, since the contrast spreads into the less-resistant subdural space.

Injection of a local anesthetic into the subarachnoid or subdural space leads to a motor block that is much stronger and has a much faster onset of effect than injection into the epidural space.

Aspiration of Blood

When blood is aspirated, the needle position should be carefully corrected until no more blood is aspirated.

Aspiration of CSF

If CSF is aspirated, it is best to halt the procedure and repeat it on the following day.

Allergic Reactions

If a patient has an allergic reaction following iodine-containing contrast agent injection, treat as needed with corticosteroid and antihistamine.

Introducing the Epidural Catheter (Fig. 51.5)

Radiopaque catheters with soft tips are used to inject local anesthetics, corticosteroids, and hypertonic saline as described below. A fluoropolymer-coated epidural catheter made of stainless steel, with a spiral tip (Tun L Kath) or a less-flexible TunL-XL Kath (Fig. 51.4), is introduced into the adhesions through the needle. The beveled side of the needle should be directed toward the ventrolateral side of the caudal canal, since this position—together with a 15–30° bend about 2.5 cm below the catheter tip—makes it easier for the catheter tip to reach the desired anterolateral position and reduces the risk of catheter shearing. As the epidural adhesions are usually irregularly distributed, several corrections of the catheter position may be needed to achieve the correct position of the catheter in the desired area. For this reason, it is recommended to use a 16-G or 15-G RX Coudé epidural needle to facilitate making necessary corrections of the position of the catheter tip. Tuohy epidural needles should not be used.

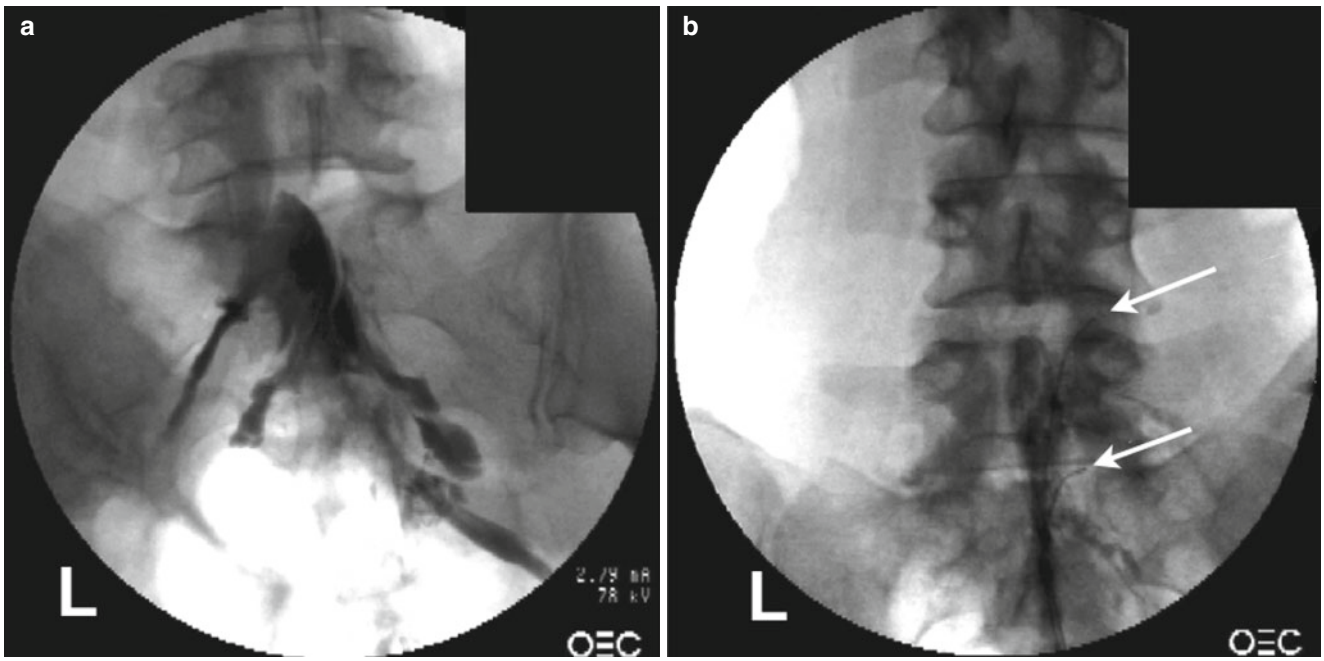


Fig. 51.5 (**a**, **b**) Radiographs of a patient with "failed back surgery syndrome" and bilateral sciatica in the region of L2–L5. The pain was more severe on the right than on the left. (**a**) Epidurogram at the start of the procedure. The contrast has not spread beyond the sacroiliac joint, and a filling defect is seen on the right. (**b**) After catheter placement and injection of the solutions (see text). The tips of two catheters were placed in the intervertebral spaces in L4–L5 and L5–S1

Contrast Injection

After the catheter tip has been placed in the correct position and after a negative aspiration test, contrast is injected again (Table 51.2). Previously recognizable filling defects along the targeted spinal nerves or nerve roots should now fill.

Injection of Hyaluronidase

After negative aspiration, slowly inject 1,500 units of animal-derived hyaluronidase or 200 units of human recombinant hyaluronidase dissolved in 10 mL of preservative-free 0.9 % saline [15]. This will further open previous filling defects.

Injection of the Local Anesthetic and Corticosteroid

Following a repeated aspiration test, 0.2 % ropivacaine and 40 mg triamcinolone acetate is now injected through the catheter (Table 51.2). The areas in which epidural adhesions had developed and were dissolved should be documented.

Injection of Hypertonic Saline

30 min later, after a negative aspiration test, the patient is placed in the lateral decubitus position on the painful side for 20–30 min. Next, 10 mL of a hypertonic saline solution (10 %) is injected epidurally via an infusion pump over 15–30 min. The indwelling catheter is then rinsed with 0.9 % saline (Table 51.2). Hypertonic saline has a reversible weakly anesthetic effect and reduces edema formation in previously scarred or inflamed nerve roots. However, the injection of hypertonic solutions into the epidural space is extremely painful if no local anesthetic has been administered beforehand. Consequently, if the hypertonic saline spreads beyond the segment in which local anesthesia was previously applied, it is possible that the patient may experience extreme pain requiring intravenous administration of sedatives or an additional epidural dose of local anesthetic. However, the pain rarely lasts for more than 5 min.

If iodine-containing radiographic contrast is not used when the patient has a known history of allergy, the procedure is carried out in the same way without it. To exclude a subarachnoid or subdural position of the needle or catheter, a test dose of local anesthetic is administered. In this case, the patient experiences pain in the skin area corresponding to the scarred epidural region. As the catheter is advanced, resistance is felt when contact is made with adhesions. It is necessary to advance the catheter slowly to avoid entering the subarachnoid or subdural space.

After the Procedure

Securing the Catheter

When the procedure has been completed, the catheter is firmly secured with a skin suture. The exit point is generously covered with antibiotic ointment (triple combination) and covered with two slit compresses (5×5 cm). Benzoin tincture is spread on the surrounding skin. A Tegaderm transparent dressing (10×12 cm) is placed over the site. Finally, four strips of a porous elastic Hypafix plaster are applied, so the patient cannot "sweat off" the plaster.

An injection syringe adapter and a bacterial filter are attached to the catheter. The free end of the catheter is attached to the patient's side. During the hospital stay, prophylactic antibiotic treatment continues to be administered to prevent bacterial colonization (which is favored by steroid administration). After discharge, antibiotics are prescribed for a further 5 days.

Technique for Subsequent Injections

The indwelling catheter remains in place through the next post-op day. A second and third hypertonic saline infusion are done on post-op day one with 6–8 h between infusions. After each negative aspiration test and administration of a test dose, as described above, a local anesthetic is injected and then after ca. 30 min hypertonic 10 % saline is slowly infused. About 10 min after the third injection, the catheter is removed and sterile dressing applied. The patient is discharged home with 5 days of oral cephalexin at 500 mg twice a day or oral levofloxacin (Levaquin) at 500 mg once a day for penicillin-allergic patients. Clinic follow-up is in 30 days. The patient should keep the insertion site as dry as possible for as long as the catheter is in place. We also recommend our patients keep the area dry for a further 48 h after removal of the catheter to reduce the risk of infection.

Epidural adhesiolysis usually leads to a significant improvement in pain symptoms and motor function. After this, it is important to start intensive physiotherapy in order to improve muscle strength and muscle tone. We recommend neural flossing exercises that were designed to mobilize nerve roots by "sliding" them in and out of the neuroforamen. These exercises are done three to four times per day for a few months after the procedure [3].

Due to their size, existing epidural adhesions cannot always be fully dissolved. The procedure can be repeated if necessary. A 3-month pause is recommended between each treatment (due to the steroids used). During this period, intense physiotherapy must be administered with targeted muscle training.

Percutaneous Epidural Neuroplasty in the Cervical, Thoracic, and Lumbar Regions

The technique and volumes of fluid used have to be modified for percutaneous epidural neuroplasty in the cervical, thoracic, and lumbar regions. These modifications are necessary to ensure that the needle is located in the epidural space and to avoid compression of the spinal cord during subsequent injections.

Technique of Cervical Epidural Neuroplasty

The patient is placed in the left lateral position on the fluoroscopy table. The "3D" technique (direction, depth, direction) is used.

Cervical Placement of the Epidural Catheter Using the 3D Technique

Preoperative

- Examine the patient and identify the puncture area.
- Order laboratory tests to assess the usual parameters that are important when doing neuraxial blocks.

Intraoperative

- Place the patient in the left lateral position.
- Prepare and drape the needle insertion site.
- Puncture site: T1–T2 or T2–T3.
- Access: paramedian, 1 cm or less lateral to the midline, one intervertebral space below the planned epidural access.
- Epidural puncture with a 16-G or 15-G RX Coudé needle.
- Anteroposterior fluoroscopy to assess the puncture *d*irection.
- Lateral fluoroscopy to assess the injection *d*epth.
- Anteroposterior fluoroscopy to assess again and if necessary correct the puncture *d*irection.
- Advance the needle to the base of the spinous process. The injection depth corresponds to the position of the lamina of the vertebral arch in the vicinity of the posterior epidural space. This technique has been improved with the advent of the RX Coudé 2 needle, which has a second interlocking stylet that protrudes slightly beyond the tip of the needle and functions to push the dura away from the needle tip as it is turned 180° cephalad.
- Remove the stylet.
- Attach a pulsator syringe (low friction) filled with 4 mL 0.9 % NaCl and 2 mL air.
- Identify the epidural space using the loss-of-resistance technique.
- Position the needle in the midline.
- Inject 1–2 mL radiographic contrast (for the cervical epidurogram).
- Introduce the catheter through the epidural needle in the direction of the targeted nerve root in the lateral epidural space. Anterolateral positioning of the catheter is important because the nociceptors are concentrated in the anterolateral space. Repeated aspiration is important.
- Inject 1,500 IU hyaluronidase mixed with 4–6 mL NaCl 0.9 %.
- Inject local anesthetic and corticosteroid (incremental volume of 2–3 mL).
- Remove the needle under fluoroscopic guidance.
- Attach a bacterial filter.
- Secure the catheter with a skin suture; an antibacterial and antimycotic ointment is applied, followed by a dressing.

Postoperative (Recovery Room)

- Carefully monitor the patient for at least 30 min after the procedure, checking the spread of the anesthesia to exclude the ever-present risk of inadvertent subarachnoid or subdural injection.
- Administer 6 mL of hypertonic 10 % saline through the epidural catheter using an infusion pump.
- Rinse the catheter with 2 mL of preservative-free 0.9 % saline.

On the Following Postoperative Day (Pain Treatment Unit)

- The second and third infusions are performed at the same interval as for caudal infusion.
- Do an aspiration test before injection.
- Inject a test dose of 2 mL 0.2 % ropivacaine through the catheter. After 5 min—after excluding a subarachnoid or subdural injection—a further 4 mL 0.2 % ropivacaine is administered.
- Wait 20 min.
- Infuse 6 mL hypertonic 10 % saline using an infusion pump, over 30 min.
- Rinse the catheter with 2 mL 0.9 % saline.
- Remove the catheter.

The patient must be informed about the risk of infection (e.g., meningitis that may develop 2–4 weeks after the procedure, as the injected corticosteroids have a long-lasting effect).

Larkin et al. [7] described a technique of epidural steroid injection in which the catheter is used as a monopolar stimulation electrode for better localization of the cause of the pain.

Percutaneous Thoracic Epidural Neuroplasty

A paramedian access route is also used for catheterization of the thoracic epidural space (percutaneous thoracic neuroplasty). The procedure is carried out in the same sequence of steps as that described for lumbosacral and cervical neuroplasty. The dosages for the drugs injected are listed in Table 51.2.

Placement of the Catheter in the Anterior Epidural Space or in an Intervertebral Foramen

Drugs that are injected into the posterior or posterolateral epidural space do not reach possible pathological changes in the intervertebral foramina or anterior epidural space. It may therefore be necessary to place the catheter in these areas. The catheter's direction is checked (a) by introducing the R-K epidural needle or SCA catheter introducer in the target direction and (b) by bending the catheter tip to make it easier to guide the catheter in the desired direction. The transforaminal approach, which is not discussed in this chapter, may be necessary to reach target sites.

Complications

The potential side effects and complications of percutaneous epidural neuroplasty include:

- Inadvertent subarachnoid or subdural injection of local anesthetics or hypertonic 10 % saline. A subarachnoid injection of hypertonic saline can lead to cardiac arrhythmia, paralyses, or loss of sphincter function.
- Epidural abscess (see Chap. 41).
- Epidural hematoma (see Chap. 41).
- Paralyses.
- Disturbances of bladder or rectal function.
- Infections (corticosteroid administration leads to immune suppression, with the resulting risk of infection; strictly aseptic conditions should therefore be observed).
- Catheter shearing.

It is possible in some circumstances for loculation to occur if injections are made into a confined space within the epidural space. This is easily recognized by patients reporting pain when injection is made and by confined distribution of radiopaque contrast. Loculation is associated with a substantial increase in epidural pressure, which can lead to local damage or even—when the pressure is transferred via the subarachnoid space—to injury to the central nervous system [18].

Undiagnosed Neurogenic Bladder and Rectal Disturbances

The problem of preoperatively unrecognized neurogenic bladder disturbances in patients with "failed back surgery syndrome" or spinal cord injuries led to distrust both of practicing physicians and of the technique. It is therefore important to perform and document the necessary urological examinations before neuroplasty. This prevents previously existent micturition disturbances or rectal disturbances from being incorrectly attributed to injury after a neuroplasty procedure has been done. This type of examination is particularly important in patients with constrictive arachnoiditis, as this not infrequently leads to disturbances of rectal and bladder function and of sexual function as well in men.

Spinal Cord Compression

As discussed above, all injections should be slow. Fast injections into the epidural space can in some circumstances create a strong increase in CSF pressure, with the risk of cerebral hemorrhage, visual disturbances, headache, and disturbances of the blood supply to the spinal cord.

Infection

Patients are informed that epidural infections can occur in the first 2–6 weeks after the procedure—both due to the procedure itself and due to the steroid-related immunosuppression it involves. Until proved otherwise, any occurrence of nausea, vomiting, stiffness in the neck, severe pain, weakness, numbness, or paralysis must therefore be regarded as due to the procedure and correspondingly treated.

Patients should be advised to contact the physician who did the procedure, or their general practitioner, immediately if any of these symptoms arise. Appropriate inpatient treatment must be immediately instituted (see Chap. 41). No cases of epidural abscess have been observed so far in the patients we have treated. However, due to the potentially extremely serious sequelae of unrecognized and untreated epidural abscesses, extreme attentiveness and careful patient information are indispensable.

Hyaluronidase Hypersensitivity

In a follow-up study by Moore [13] including 1,520 patients who underwent epidural hyaluronidase administration, hyaluronidase hypersensitivity was observed in 3 % of the patients. The fact that this 3 % incidence was not observed with the technique described here may possibly be due to the injection of a corticosteroid in the same site at which the hyaluronidase was injected. The steroid remains in the epidural space for longer than the hyaluronidase and may provide protection against allergic reactions.

The reduction in the incidence of treatment-resistant pain conditions obtained with this procedure entirely justifies the use of hyaluronidase; however, very careful attention must be given to any signs of hypersensitivity.

Experience at the Texas Tech University Health Sciences Center (TTUHSC)

At our center, the epidural adhesiolysis technique described here has been performed on thousands of patients. Only a few complications were observed. Subarachnoid or subdural injection of the local anesthetic is extremely rare. Two of the patients developed meningitis after epidural adhesiolysis and were quickly and effectively treated with antibiotics. No cases of paralysis were observed in any of the patients; one patient developed transient motor weakness (caudal access). There were no significant, longer-lasting cases of rectal dysfunction, although a few patients developed mild micturition or defecation disturbances during the first 2 weeks after the procedure. There was a report of transient numbness in the perineal area, which resolved again after 1–2 months. There was 1 case of bladder dysfunction (urinary retention) lasting 3 years after the hypertonic saline spread subdural during the first infusion. The urinary retention resolved on its own after 3 years of intermittent in and out catheterization.

Additional Aspects

This chapter describes a procedure involving a 3-day course of injection treatment based on the results of a randomized, prospective double-blind study [4]. However, Manchikanti et al. [11] have shown that 1-day treatments are also effective. The pain reduction is more persistent, however, when more frequent and repeated injections are done [10]. There is evidence that the epidural neuroplasty procedure is also suitable for pain treatment in cases of spinal canal stenosis. Previously disturbed motor function also improves after treatment in some patients [12].

Based on findings of a systematic literature review, Chopra et al. [1] concluded that the evidence of effectiveness of percutaneous adhesiolysis is moderate to strong.

Summary

Percutaneous epidural neuroplasty is an interventional procedure in the treatment of pain caused by structures in the epidural space or its vicinity and in the intervertebral foramina in all segments of the spine. Adequate scientific justification for the procedure appears to be provided by the current state of knowledge regarding the pathogenesis of back pain and radiculopathy. To ensure the safety of the procedure and achieve the best possible results, it is recommended that the details of the procedure described here (regarding technique and patient selection) should be followed precisely.

References

1. Chopra P, Smith HS, Deer TR, Bowman RC. Role of adhesiolysis in the management of chronic spinal pain: a systematic review of effectiveness and complications. Pain Physician. 2005;8:87–100.
2. Gerdesmeyer L, Wagenpfeil S, Birkenmaier C, et al. Percutaneous epidural lysis of adhesions in chronic lumbar radicular pain: a randomized, double-blind, placebo-controlled trial. Pain Physician. 2013;16:185–96.
3. Gilbert K, Brismee J, Collins D, et al. Lumbosacral nerve roots and strain: part 1. A novel measurement technique during straight leg raise unembalmed cadavers. Spine. 2007;32:1513–20.
4. Heavner JE, Racz GB, Raj P. Percutaneous epidural neuroplasty: prospective evaluation of 0.9 % NaCl versus 10 % NaCl with or without hyaluronidase. Reg Anesth Pain Med. 1999;24:202–7.
5. Helm II S, Benyamin R, Chopra P, Deer T, Justiz R. Percutaneous adhesiolysis in the management of chronic low back pain in post lumbar surgery syndrome and spinal stenosis: a systematic review. Pain Physician. 2012;15:E435–62.
6. Kuslich SD, Ulstrom CL, Michael CJ. The tissue origin of low back pain and sciatica. Orthop Clin N Am. 1991;22:181–7.
7. Larkin TM, Carragee E, Cohen S. A novel technique for delivery of epidural steroids and diagnosing the level of nerve root pathology. J Spinal Disord Tech. 2003;16:186–92.
8. Manchikanti L, Cash K, McManus C, et al. The preliminary results of a comparative effectiveness of adhesiolysis and caudal epidural injections in managing chronic low back pain secondary to spinal stenosis. Pain Physician. 2009;12:E341–54.
9. Manchikanti L, Helm II S, Pampati V, Racz GB. Cost utility analysis of percutaneous adhesiolysis in managing pain of post-lumbar surgery syndrome and lumbar central spinal stenosis. Pain Pract. 2014. doi:10.1111/papr.12195.
10. Manchikati L, Pakanati RR, Bakhit CE, Pampati V. Role of adhesiolysis and hypertonic saline neurolysis in management of low back pain: evaluation of modification of the Racz protocol. Pain Dig. 1999;9:91–6.
11. Manchikanti L, Pampati V, Fellows B, Rivera J, Beyer CD, Damron KS. Role of one day epidural adhesiolysis in management of chronic low back pain: a randomized clinical trial. Pain Physician. 2001;4:153–66.
12. Manchikanti L, Pampati V, Fellows B, Rivera J, Beyer CD, Damron KS, Cash KA. Effectiveness of percutaneous adhesiolysis with hypertonic saline neurolysis in refractory spinal stenosis. Pain Physician. 2001;4:366–73.
13. Moore DC. The use of hyaluronidase in local and nerve block analgesia other than the spinal block. 1520 cases. Anesthesiology. 1951;12:611–26.
14. Olmarker K, Rydevik B. Pathophysiology of sciatica. Orthop Clin N Am. 1991;22:223–33.
15. Racz G, Day M, Heavner J, et al. Hyaluronidase: a review of approved formulations, indications and off-label use in chronic pain management. Expert Opin Biol Ther. 2010;10:127–31.
16. Racz, GB, Heavner, JE, et al. Epidural lysis of adhesions and percutaneous neuroplasty. 2014; http://dx.doi.org/10.5772/58753.
17. Racz GB, Holubec JT. Lysis of adhesions in the epidural space. In: Racz GB, editor. Techniques of neurolysis. Boston: Kluwer Academic; 1989. p. 57–72.
18. Rocco AG, Philip JH, Boas RA, Scott D. Epidural space as a Starling resistor and elevation of inflow resistance in a diseased epidural space. Reg Anesth. 1997;22:167–77.
19. Roffe PG. Innervation of the annulus fibrosis and posterior longitudinal ligament. Arch Neurol Psychiatry. 1940;44:100.

Part IX

Abdominal and Pelvic Region

Chapter 52

Celiac Plexus Block

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Hariharan Shankar, MD
Pain Clinic, Clement Zablocki VA Medical Center, Department of Anesthesiology, Medical College of Wisconsin, Milwaukee, WI, 53295, USA
e-mail: hshankar@mcw.edu

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_52, © Springer International Publishing Switzerland 2015

Definition

Injection of a local anesthetic or neurolytic in the region of the celiac plexus.

Anatomy (Figs. 52.1, 52.2, and 52.3)

The celiac plexus is the largest of the three large sympathetic plexuses (cardiac plexus, thorax; celiac plexus, abdomen; hypogastric plexus, pelvis).

It receives its primary innervation from the preganglionic splanchnic nerves (greater splanchnic nerve T5–10, lesser splanchnic nerve T10–11, and least splanchnic nerve T11–12), the postganglionic fibers of which, after synapsing in the celiac ganglion, radiate to the associated plexus and innervate most of the abdominal organs. This large network, with a diameter of about 50 mm, surrounds the origins of the celiac artery and superior mesenteric artery and extends laterally as far as the adrenal glands, upward as far as the aortic hiatus, and downward as far as the root of the renal artery. It lies on the initial part of the abdominal aorta, at the level of the first lumbar vertebra, anterior to the medial crus of the diaphragm.

The most important roots of the celiac plexus are the splanchnic nerves, the abdominal branches of the vagus nerves, and several branches of the last thoracic ganglion and two highest lumbar ganglia. Cranially, the celiac plexus is connected to the thoracic aortic plexus, and caudally it continues into the abdominal aortic plexus. A paired celiac ganglion forms the basis for the celiac plexus although up to five ganglia may be involved.

The left ganglion lies closer to the midline and partly on the aorta, while the right one (ventrolateral to the vena cava) lies slightly more to the side in the area of the fissure between the medial and lateral crura of the diaphragm. The two ganglia are connected to one another. With closer approximation and fusion, the double ganglion takes on a ring shape, which is also known as the solar ganglion (solar plexus).

The smaller superior mesenteric ganglion and aorticorenal ganglion are associated with the celiac ganglion.

The lesser splanchnic nerve usually enters the latter ganglion, while the greater splanchnic nerve passes to the posterior surface of the lateral part of the celiac ganglion.

The phrenic ganglion is a third, unpaired, ganglion. The following, sometimes paired and sometimes unpaired, secondary (associated) plexuses emerge from the celiac plexus:

- Paired (phrenic plexus, suprarenal plexus, renal plexus, and spermatic plexus)
- Unpaired (superior gastric plexus, hepatic plexus, splenic plexus, and superior mesenteric plexus)

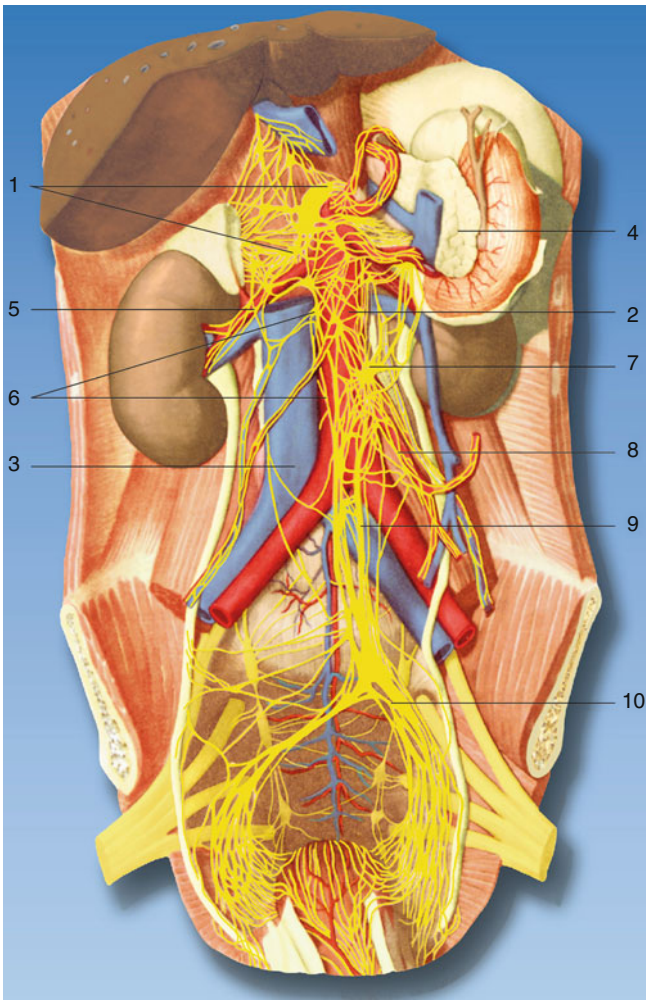


Fig. 52.1 Anatomy. (*1*) Celiac plexus, (*2*) aorta, (*3*) inferior vena cava, (*4*) pancreas, (*5*) renal plexus, (*6*) abdominal aortic plexus, (*7*) inferior mesenteric ganglion, (*8*) inferior mesenteric plexus, (*9*) superior hypogastric plexus, (*10*) inferior hypogastric plexus (Reproduced with permission from Danilo Jankovic)

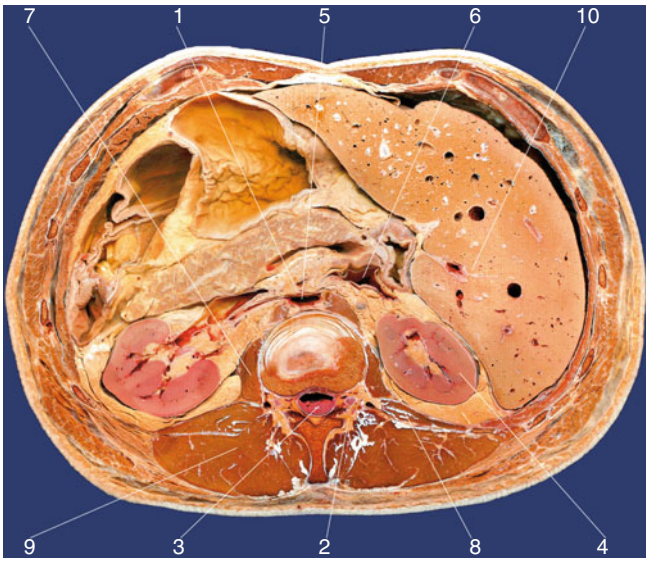


Fig 52.2 Transversal section through abdomen at the level of the first lumbar vertebra. (*1*) Celiac plexus with celiac ganglion, (*2*) spinal ganglion, (*3*) cauda equina, (*4*) medulla renalis, (*5*) aorta, (*6*) inferior vena cava, (*7*) psoas muscle, (*8*) quadratus lumborum muscle, (*9*) erector spinae muscle, (*10*) liver (Reproduced with permission from Danilo Jankovic)

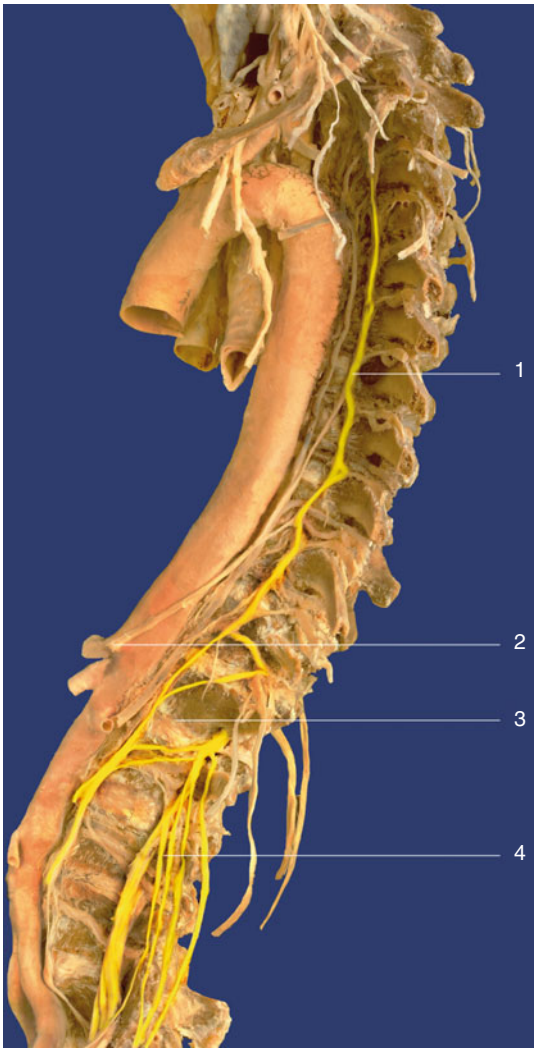


Fig. 52.3 (*1*) Sympathetic trunk (underneath: greater and minor splanchnic nerves), (*2*) celiac plexus (underneath: superior mesenteric artery and renal artery), (*3*) vertebral body of the first lumbar vertebra with rami communicantes albi, (*4*) lumbar plexus (Reproduced with permission from Danilo Jankovic)

Indications

Diagnostic

Differential diagnosis of pain in the abdominal region (visceral, epigastric, or cardiac pain).

Prognostic

In upper abdominal cancer pain, before a neurolytic block.

Therapeutic

1. Relief of upper abdominal pain (pancreas, stomach)
2. Treatment of the dumping syndrome
3. Injection of neurolytics as palliation measure in malignant intra-abdominal disease-related pain (pancreas, stomach)

Contraindications

1. Anticoagulant treatment
2. Infections and skin diseases in the injection area
3. Hypovolemia
4. General debility
5. Injection of a neurolytic without precise identification of the needle position (with guidance by CT or image intensifier)

Fluoroscopic Guided Procedure

This block should be carried out by very experienced anesthetists. Full information for the patient is mandatory.

Preparations

Check that the emergency equipment is complete and in working order: sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, and emergency medication.

Materials

Fine 26-G needle for local anesthesia. 20–22-G spinal needle, 0.7 (0.9) 120 mm (150 mm). Syringes: 2, 10 and 10 mL. Disinfectant, swabs, compresses, sterile gloves and drape, and flat, firm pillow.

Patient Positioning

1. Prone position: supported with a pillow in the mid-abdomen (to relieve lumbar lordosis). The patient's arms are dangling, with the head lying to the side. The patient breathes with the mouth open, to reduce tension in the back muscles. This position is preferable.
2. Lateral decubitus position, with support under the flank. The side being blocked lies upward.

Location

1. L4 iliac crest line (count the spinous processes cranially).
2. L2: a parallel line is drawn about 7–8 cm lateral to the midline. The intersection between this line and the lower edge of the twelfth rib determines the level of the upper edge of L2.
3. T12–L1 is located in the midline and joined with dots to the lower edge of the twelfth rib. This produces a triangle, the equal sides of which provide basic guidance for the needle direction (Fig. 52.4).

Skin prep, generous local anesthetic infiltration of the injection channel, covering with a sterile drape, drawing up the local anesthetic, checking the patency of the injection needle.

During the injection, the following points must be observed:

1. Bilateral injection is usually unnecessary depending on the spread of the contrast agent.
2. The operator performing the injection must stand on the side being blocked.
3. In most patients, the distance between the skin and the celiac plexus is about 9–11 cm.
4. Superficial bone contact (after 3–5 cm) indicates contact with the transverse process and requires cephalocaudad redirection.
5. Paresthesias during the introduction of the needle arise due to stimulation of the lumbar somatic nerves.
6. There is a risk of perforating the dural cuff (epidural or subarachnoid injection).
7. If the patient coughs, it indicates pleural irritation or injury. The procedure should be halted.
8. Aspirate frequently and inject on an incremental basis.
9. The method of choice when administering neurolytics is CT-guided injection.

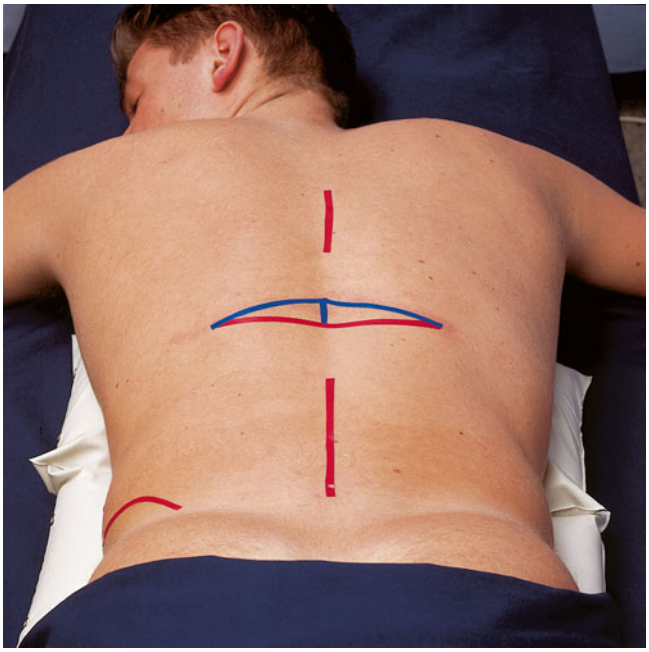


Fig. 52.4 Location (Reproduced with permission from Danilo Jankovic)

Injection Technique (Dorsal, Retrocrural)

1. About 7–8 cm lateral to the midline (lower edge of the twelfth rib), at an angle of 45° to the skin surface and directed slightly cranially, the injection needle is advanced toward the L1 vertebra (Fig. 52.5).

   Bone contact is usually made at a depth of about 7–9 cm (Figs. 52.6 and 52.8).
2. The depth of the needle is marked visually.
3. The needle is withdrawn to lie subcutaneously.
4. The needle is then redirected at a steeper angle of 60° to the skin surface, so that it can just slide past the lateral edge of the L1 vertebra (Figs. 52.7 and 52.8).
5. The needle introduced on the left (the side of the aorta) can then be carefully advanced a further 1.5–2 cm deeper.
6. After the needle is positioned in the periaortic space, pulsations are transmitted via the needle shaft to the fingertips. The needle introduced on the right side can be advanced in a similar fashion or slightly deeper (2–3 cm) (Figs. 52.8 and 52.10).
7. The end of the needle must be observed for spontaneous backflow of liquid (blood, CSF, urine?).
8. Aspiration test at all four levels.
9. Test dose of the local anesthetic.
10. Resistance-free incremental injection of a local anesthetic.

It is now the standard procedure to carry out this injection with guidance using a radiographic image intensifier or computed tomography (CT).

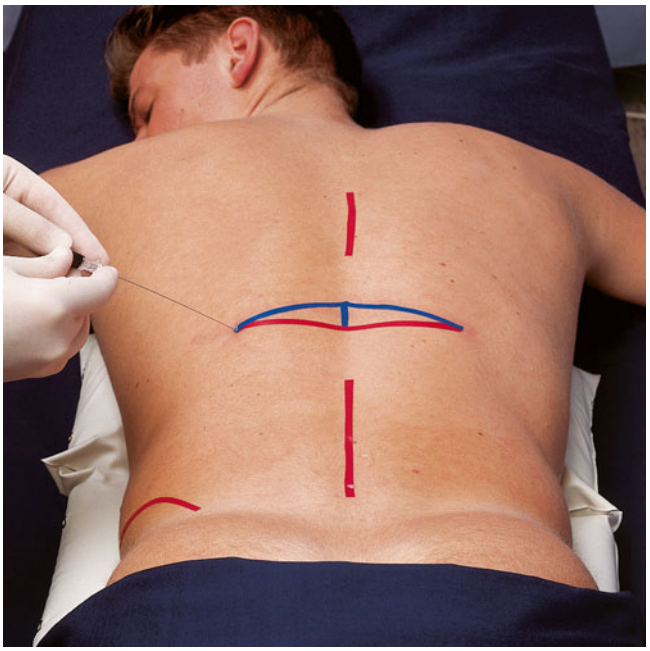


Fig. 52.5 Introducing the injection needle at an angle of about 45° to the skin surface (Reproduced with permission from Danilo Jankovic)

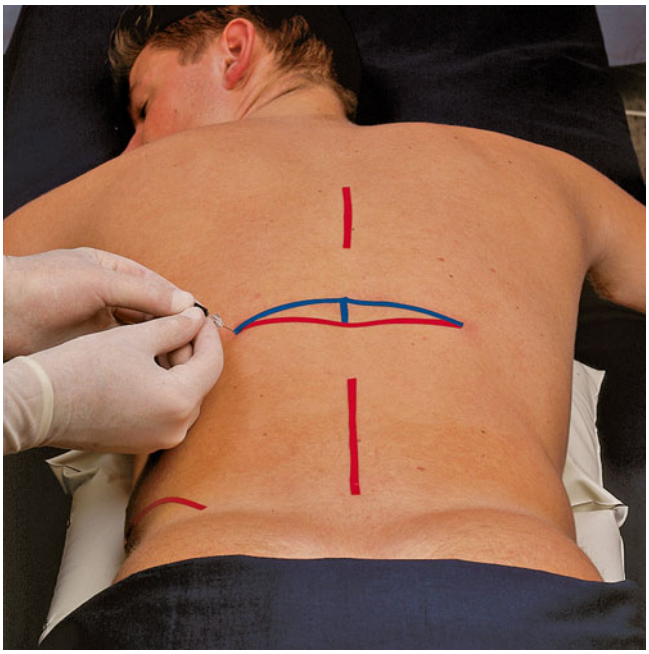


Fig. 52.7 Redirection of the needle at an angle of 60° to the skin surface (Reproduced with permission from Danilo Jankovic)

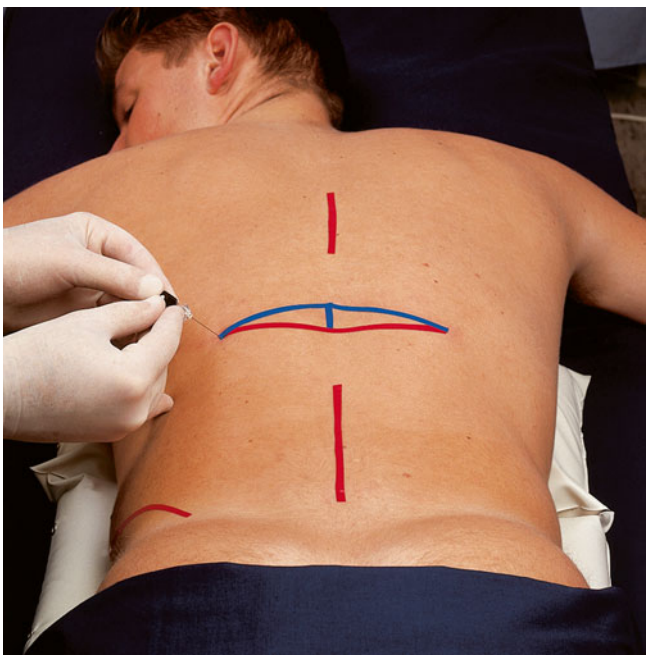


Fig. 52.6 Bone contact with the L1 vertebra. After visual marking of the depth, the needle is withdrawn back to the subcutis (Reproduced with permission from Danilo Jankovic)

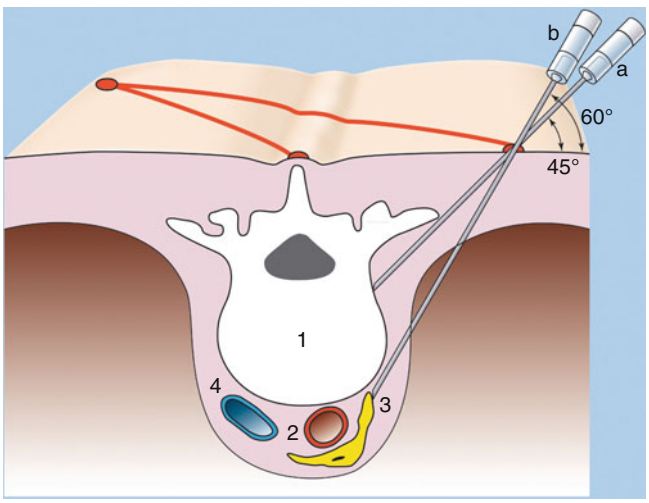


Fig. 52.8 Path of the needle to the celiac plexus. *Diagram*: (*a*) contact with the L1 vertebra, (*b*) introduction of the needle at a steeper angle of 60° to the skin surface. (*1*) L1 vertebra, (*2*) aorta, (*3*) celiac plexus, (*4*) inferior vena cava (Reproduced with permission from Danilo Jankovic)

Ultrasound-Guided Procedure

Only anesthetists well experienced in ultrasound-guided pain interventions should carry out this block. Ultrasound-guided anterior approach is ideal in persons who are unable to lay prone.

Preparations

See above.

Materials

Fine 26-G needle for local anesthesia. 20–22-G Chiba needle, 0.7 (0.9) 120 mm (150 mm). Syringes: 2, 5 and 10 mL. Chloraprep, sterile ultrasound gel, sterile gloves, and drape.

The needle length may be chosen after scout scanning to determine the location and depth to the target.

Patient Positioning

Patient is positioned supine, standard monitors are applied and an intravenous line started. Intravenous antibiotic is administered 30 min prior to start usually Ancef 1 g. Skin prep, generous local anesthetic infiltration of the injection channel, covering with a sterile drape. Drawing up the local anesthetic, checking the patency of the injection needle.

Scout Scan

1. The scanning sequence starts with an axial view just beneath the xiphoid to identify the aorta, inferior vena cava, and vertebral body (Fig. 52.9a).
2. Subsequently the transducer is rotated sagittal to obtain a longitudinal view of the aorta (Fig. 52.9b).
3. By scanning further caudad, the celiac trunk and its bifurcation into hepatic and splenic arteries and then the superior mesenteric artery may be identified using color flow Doppler (Fig. 52.9c).The target is just above the celiac trunk and between the trunk and the superior mesenteric artery.
4. Prior to local anesthetic/neurolytic injection, it is always a good practice to inject saline and watch the spread real time (Fig. 52.9d).

During the injection, the following points must be observed:

1. In-plane or out-of-plane approach may be used depending on operator preference.
2. For an out-of-plane approach, the needle is advanced from the middle of the broad side of the transducer. Hydrolocalization is used to facilitate needle tip identification.
3. For an in-plane approach, the needle is advanced from the short end of the transducer approximately 1–2 cm away from the transducer. Both approaches are possible with either the short-axis view or the long-axis view of the target.
4. The safest trajectory should be planned during the scout scan.
5. It is usually safe to pass through the liver without any major complications.

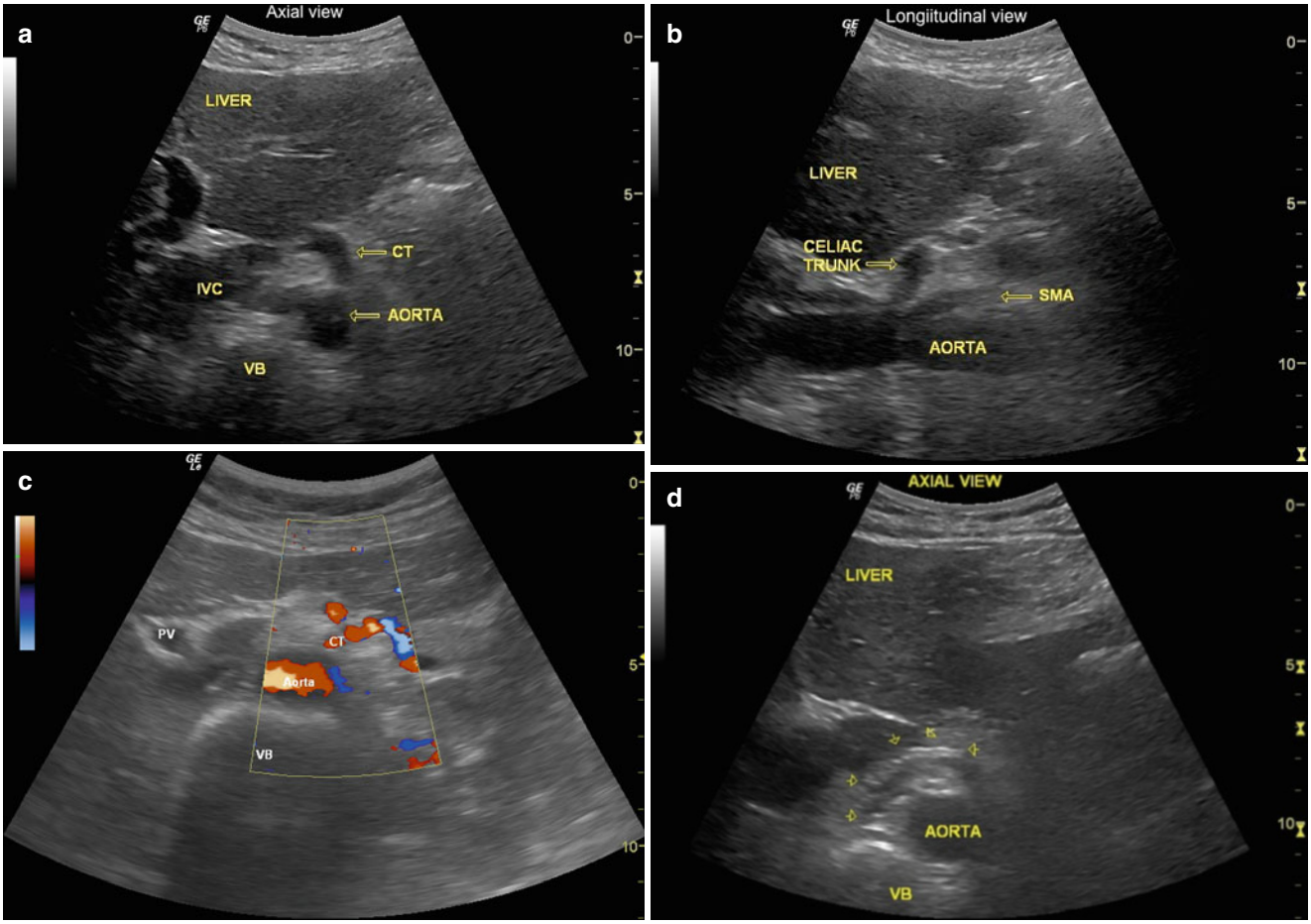


Fig. 52.9 (**a**) Axial ultrasound image of the abdomen at the level of the celiac trunk (*CT*) showing the aorta, vertebral body (*VB*), liver, and inferior vena cava (*IVC*) (Reproduced with permission from Hariharan Shankar). (**b**) Longitudinal ultrasound image of the abdomen at the level of the celiac trunk showing the aorta, liver, and the superior mesenteric artery (SMA) (Reproduced with permission from Hariharan Shankar). (**c**) Color flow Doppler axial image of the abdomen at the level of the celiac trunk (*CT*) showing the aorta, vertebral body (*VB*), and portal vein (*PV*) (Reproduced with permission from Hariharan Shankar). (**d**) Post-injection axial ultrasound image of the abdomen showing the injectate spread (*arrowheads*), liver, aorta, and the vertebral body. The celiac trunk is not well visualized as it has been distorted by the injectate spread (Reproduced with permission from Hariharan Shankar)

Neurolytic Block

Unilateral or bilateral injection of neurolytics (50 % ethanol) in the region of the celiac plexus.

The block should be carried out with CT guidance if possible, in order to reduce potential complications to a minimum and increase accuracy. Several access routes are possible (Figs. 52.10 and 52.11).

A diagnostic block with local anesthetics is a prerequisite. This measure can only achieve the desired result if the disease is not too far advanced and is not producing additional neuropathic pain (e.g., extension to the epigastric nerves, intercostal nerves, or lumbar plexus).

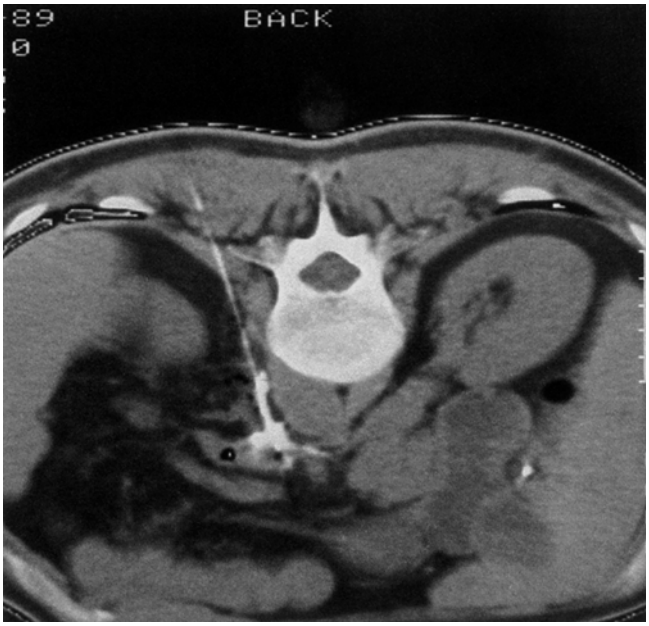


Fig. 52.10 Dorsal CT-guided celiac plexus injection. After injection of contrast medium, a good spread in the area of the celiac plexus can be seen (Reproduced with permission from Danilo Jankovic)

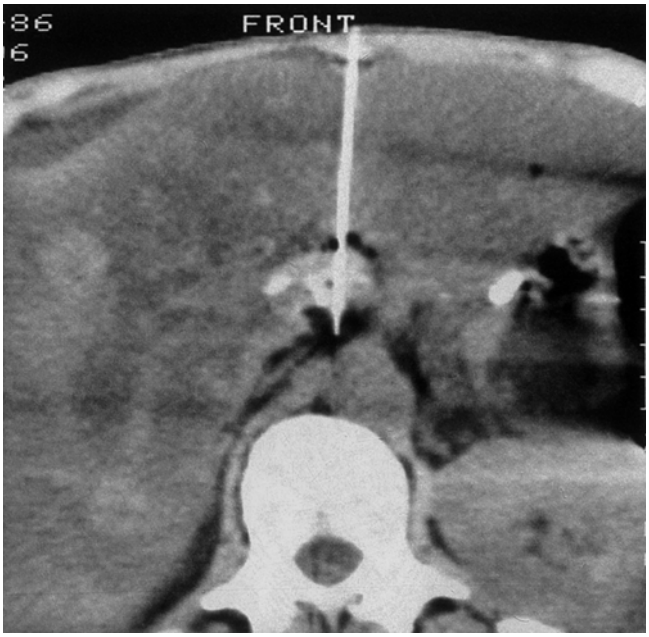


Fig. 52.11 Ventral transhepatic CT-guided injection. Several access routes are possible in celiac plexus injection. In this case, the injection was carried out using a ventral transhepatic approach. The tip of the needle lies directly alongside the celiac trunk. The alcohol spreads precisely within the celiac plexus (Reproduced with permission from Danilo Jankovic)

Effects of the Block

1. Hyperthermia in the upper abdominal region (vascular dilation in the splanchnic region)
2. Increased intestinal motility
3. Pain reduction

Dosage

Diagnostic

20–30 mL local anesthetic—e.g., 0.5–1 % prilocaine, 0.5–1 % mepivacaine, and 0.5–1 % lidocaine.

Therapeutic

1. Local anesthetics

   20–30 mL local anesthetic—e.g. 0.375–0.5 % ropivacaine and 0.25–0.375 % bupivacaine. A mixture with methylprednisolone is recommended in acute conditions.
2. Neurolytics

   25–50 mL 50 % ethanol in combination with 0.2 % ropivacaine or 0.125 % bupivacaine. Some authors recommend prior administration of 5 mL 2 % lidocaine to relieve the pain of the ethanol injection.

Side Effects

Hypotension due to sympathetic block (caution in older patients).

Complications

Severe

1. Intravascular injections (aorta, inferior vena cava, celiac artery, renal artery) with toxic reactions (see Chap. 1)
2. Epidural or subarachnoid injection (see Chap. 41 anesthesia)
3. Pneumothorax

Potential

1. Vascular injury (hemorrhage, retroperitoneal hematoma formation)
2. Injection into kidneys and other intra-abdominal organs
3. Aortal pseudoaneurysm
4. Abscess or cyst formation
5. Intraosseous or psoas injection

Complications of Neurolytic Block

1. Paraplegia
2. Monoparesis, with loss of sphincter function in the rectum and bladder
3. Sexual dysfunction
4. Diarrhea
5. Retroperitoneal fibrosis
6. Renal necrosis
7. Chemical peritonitis
8. Chemical pericarditis

Celiac plexus block

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: *G* _____ *Length* _____ *cm*

i. v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs):* ☐ *Checked*

Patient: ☐ *Informed*
Contraindications excluded:

Position: ☐ *Prone* ☐ *Lateral recumbent* ☐ *Supine*

Injection level: ☐ *L1*

Injection technique: ☐ *Dorsal* ☐ *Ventral*

☐ *X-ray image intensifier guidance* ☐ *CT-guided*

☐ *Ultra sound guided:* ☐ *Transducer* ☐ *Curved* ☐ *Linear*

Approach: ☐ *In plane* ☐ *Out of plane*

Injection:

Local anesthetic: _____ *mL* _____ *%*
(in incremental doses)

Addition to LA: Yes _____ No _____

Neurolytic: _____ *mL* _____ *%*

Addition: ☐ Yes ☐ No

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement? before* _____ *°C after* _____ *°C*

Monitoring after block: < 1 h > 1 h

Time of discharge: _____

Complications:

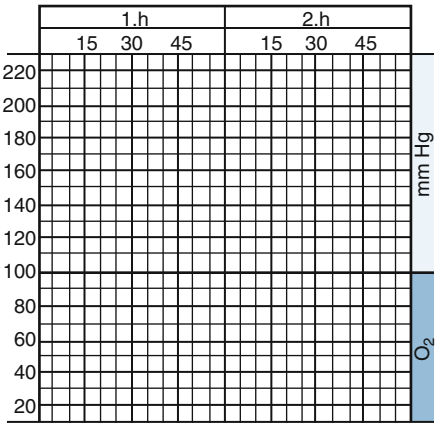
☐ *None* ☐ *Intravascular injection*
☐ *Subarachnoid/epidural* ☐ *Pneumothorax*
☐ *Drop in BP* ☐ *Nerve injury*
☐ *Other*

Subjective effect after of the block : Duration: _____

☐ *None* ☐ *Increased pain*
☐ *Reduced pain* ☐ *Relief of pain*
☐ *Visual analog scale*

Special notes:

Record and checklist



With permission from Danilo Jankovic

Suggested Reading

1. Bhatnagar S, Gupta D, Mishra S, et al. Bedside ultrasound-guided celiac plexus neurolysis with bilateral paramedian needle entry technique can be an effective pain control technique in advanced upper abdominal cancer pain. J Palliat Med. 2008;11:1195–9.
2. Brown D. Atlas of regional anesthesia. 3rd ed. Philadelphia: Saunders & Elsevier; 2006.
3. Buy JN, Moss AA, Singler RC. CT guided celiac plexus and splanchnic nerve neurolysis. J Comput Assist Tomogr. 1982;6:315.
4. Galizia EJ, Lahiri SK. Paraplegia following coeliac plexus block with phenol. Br J Anaesth. 1974;46:539.
5. Hahn MB, McQuillan PM, Sheplock GJ. Regional anesthesia. St. Louis: Mosby; 1996.
6. Hegedues V. Relief of pancreatic pain by radiography-guided block. Am J Roentgenol. 1979;133:1101.
7. Jankovic D. Celiac plexus block. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Berlin: Blackwell Scientists; 2004. p. 199–205.
8. Sayed I, Elias M. Acute chemical pericarditis following celiac plexus block. Middle East J Anesthesiol. 1997;14(3):201–5.
9. Thompson GE, Moore DC. Celiac plexus, intercostal and minor peripheral blockade. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade. 2nd ed. Philadelphia: Lippincott; 1988.
10. Wong GY, Brown DL. Transient paraplegia following alcohol celiac plexus block. Reg Anesth. 1995;20(4):352–5.

Chapter 53

Nerve Blocks of the Abdominal Wall

Gloria J. Seah (✉) • Anthony Hade
Department of Anaesthesia and Acute Pain Medicine, St. Vincent's Hospital, Melbourne, VIC, Australia
e-mail: gloria.seah@svhm.org.au

Michael J. Barrington, MB, BCh, FRCA
Department of Anaesthesia and Acute Pain Medicine, St. Vincent's Hospital, Melbourne, VIC, Australia
Faculty of Medicine, Dentistry and Health Sciences, Melbourne Medical School, University of Melbourne, Melbourne, VIC, Australia

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_53,

Transversus Abdominis Plane Block

Definition

Transversus abdominis plane (TAP) blocks involve injecting local anesthetic into the neurovascular plane between the internal oblique and transversus abdominis muscles. The neurovascular plane between the internal oblique and transversus abdominis muscles is known as the TAP plane, hence the term TAP block.

Background

The TAP block was first described by Rafi [1] and subjected to randomized controlled trials by McDonnell [2, 3]. Their landmark approach utilized the lumbar triangle of Petit to access the neurovascular plane (Fig. 53.1). Ultrasound techniques (lateral/posterior and subcostal approaches) were then described and popularized by Hebbard [4, 5]. Based on our results from the International Registry of Regional Anesthesia (IRORA), over 99 % of TAP blocks are performed using ultrasound guidance [6]. Blind TAP blocks lead to an unacceptable number of inadvertent needle placements [7]. Knowledge of the location of the thoracolumbar nerves in the TAP is important for performing this procedure.

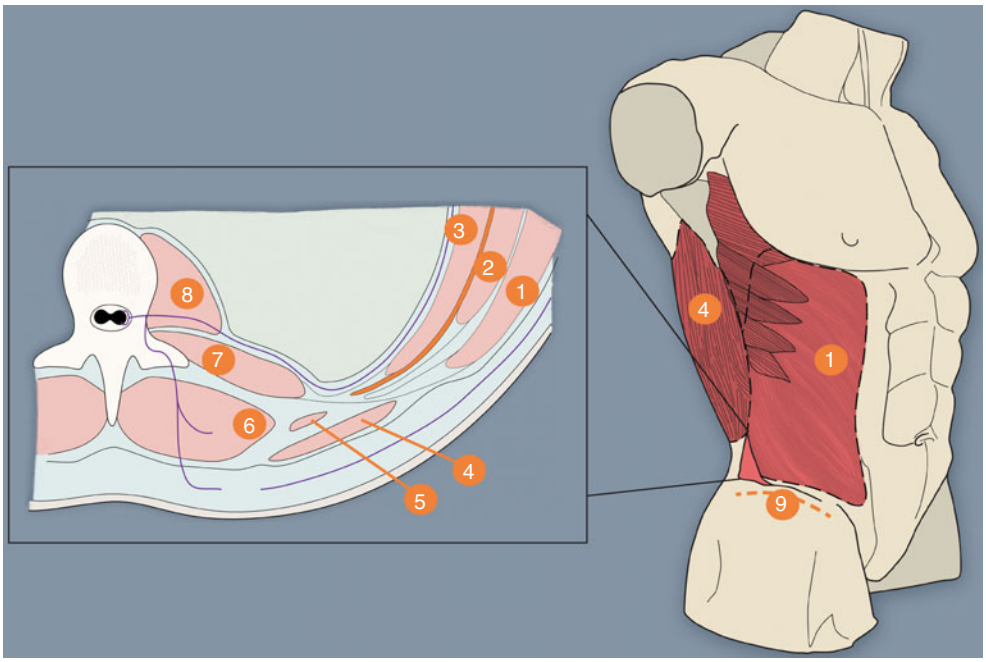


Fig. 53.1 Lumbar triangle (triangle of Petit) and the neurovascular plane targeted for landmark approach to TAP block. (*1*) External oblique, (*2*) internal oblique, (*3*) transversus abdominis, (*4*) latissimus dorsi, (*5*) serratus posterior inferior muscle, (*6*) erector spinae, (*7*) quadratus lumborum (*8*) psoas major, (*9*) iliac crest. Transversus abdominis plane is shaded orange (Original illustration by Michael Ee with permission)

Anatomy

The anterolateral abdominal wall muscles (external oblique, internal oblique, and transversus abdominis) are replaced by a well-defined aponeurosis, the linea semilunaris, at the lateral border of the rectus sheath (Fig. 53.2). This is an important sonoanatomical landmark for performing TAP and rectus sheath blocks [8, 9]. The rectus sheath contains the rectus abdominis muscles, the anterior rami of the lower sixth thoracic nerves (T7–T12), the superior and inferior epigastric vessels, and lymph vessels. It is formed by the fusion of the aponeuroses of the three anterolateral abdominal wall muscles. The abdominal wall is innervated by the T6–T11 intercostal and T12 subcostal nerves (Fig. 53.3). The intercostal nerves exit the intervertebral foramina and enter the paravertebral region related to the intercostal muscles posteriorly. Between the midline and the anterior axillary line, segmental nerves T6–T9 emerge from the costal margin to enter the TAP. The T10 segmental nerve is located caudal to the costal margin (rib 10) and T11, T12, and L1 are located in a caudal direction, toward the iliac crest. The TAP contains intercostal, subcostal, and first lumbar (L1) nerves and blood vessels (deep circumflex iliac, inferior epigastric, superior epigastric arteries) (Fig. 53.3). The lateral cutaneous branch of the intercostal nerves divides into anterior and posterior cutaneous branches. The origins of the lateral cutaneous nerves are proximal close to the costal angle. However, the point at which lateral cutaneous nerves pierces muscle layers is more anterior at the angle of rib or midaxillary line [10]. The lateral cutaneous branches are significant because they innervate much of the abdominal wall (T6–T12) and thorax (T1–T5). The anterior cutaneous nerve sends twigs to the external oblique muscle as well as skin to the lateral margin of the rectus abdominis. The posterior branch runs backward supplying the paravertebral region as well as the erector spinae.

The iliohypogastric nerve (IHN) and ilioinguinal nerve (IHN) are branches of L1 and these pass laterally through the psoas muscle, course anterior to the quadratus lumborum and travel caudally toward the iliac crest on the inner surface of the transversus abdominis muscle. The nerves pierce the transversus abdominis muscle to enter the TAP plane at variable locations (Fig. 53.4). They are located in the TAP for a short distance only. Medial to the anterior superior iliac spine, the IIN passes through internal oblique close to the inguinal ligament [11, 12]. It is at this location that landmark techniques usually aim to locate the IIN. The IHN is usually above and medial to it [13]. The IIN and IHN supply the skin and muscles of the pubic and inguinal region and genitalia. In summary, the IHN and IIN are located in the TAP close to the iliac crest and anterior superior iliac spine [14]. Medial and inferior to the anterior superior iliac spine, the nerves are located between the internal and external oblique muscles [15].

The lower five intercostal nerves pierce the lateral margin of the linea semilunaris to enter the rectus sheath posterolaterally. The intercostal (T6–T9), subcostal, and L1 nerves terminate in the rectus abdominis muscle with three patterns: (1) terminate simply within the muscle, (2) supply the muscle and then terminate as a cutaneous branch, or (3) pass through the muscle and terminate as a cutaneous branch [10]. Rozen et al. [16] noted that as the nerves approach the posterior surface of the rectus abdominis, a longitudinal branch of fibers run craniocaudally with the deep inferior epigastric artery. In addition, the cutaneous branches were closely related to the perforating musculocutaneous vessels. Together T7–L1 supply the skin from the xiphoid sternum (T7 nerve root) to the pubic symphysis (L1 nerve root), with T10 nerve supplying the umbilical segment. The IHN nerve supplies the lowermost segment of the rectus abdominis muscle and overlying skin.

The posterior abdominal wall is composed of muscles bound by the thoracolumbar fascia (Fig. 53.5). The thoracolumbar fascia is an extensive tough membranous sheet that envelops the muscles of the posterior abdominal wall, dividing these into anterior, middle, and posterior layers. Key paired muscles of the posterior abdominal wall are psoas major, iliacus, and quadratus lumborum [17].

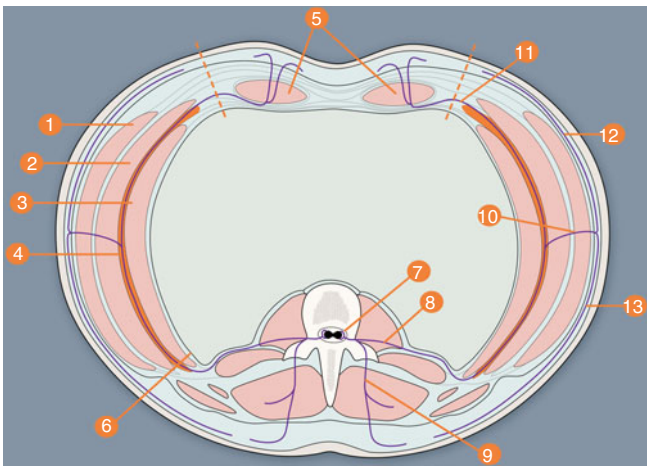


Fig. 53.2 Cross section of abdominal wall. (*1*) External oblique, (*2*) internal oblique, (*3*) transversus abdominis, (*4*) transversus abdominis plane (shaded *orange*), (*5*) rectus abdominis, (*6*) parietal peritoneum, (*7*) spinal cord, (*8*) ventral ramus, (*9*) dorsal ramus, (*10*) lateral cutaneous nerve, (*11*) anterior cutaneous nerve, (*12*) anterior branch of lateral cutaneous nerve, (*13*) posterior branch of lateral cutaneous nerve; linea semilunaris (*orange-dotted lines*) (Original illustration by Michael Ee with permission)

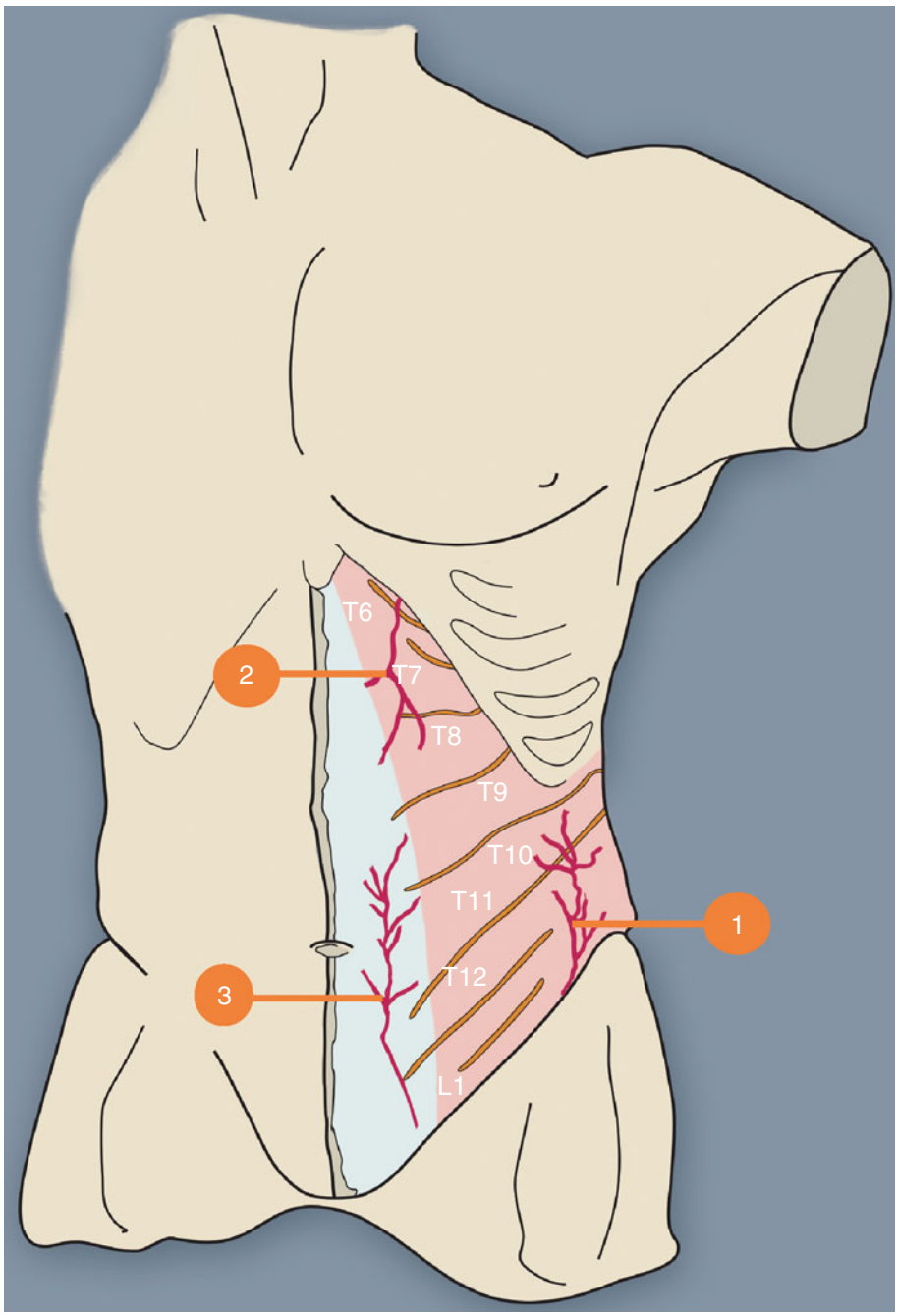


Fig. 53.3 Nerves and vessels within transversus abdominis plane. (*1*) Deep circumflex iliac artery, (*2*) superior epigastric artery, (*3*) inferior epigastric artery (Original illustration by Michael Ee with permission)

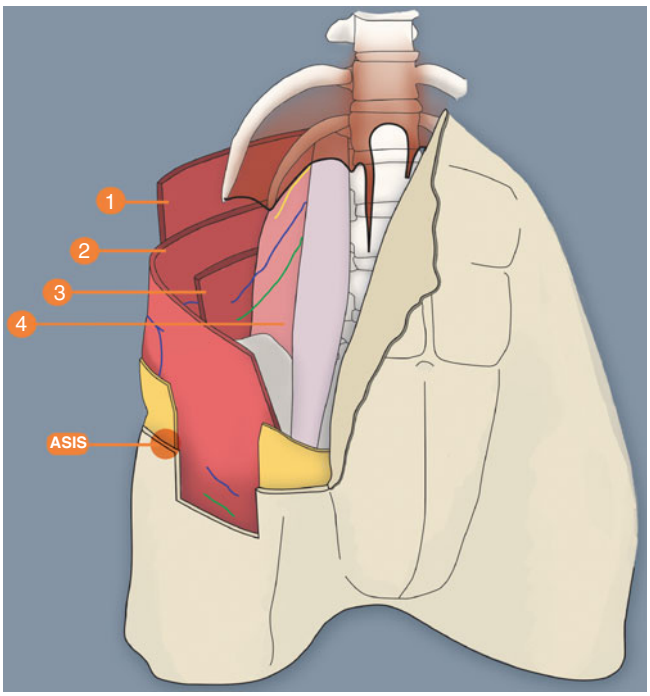


Fig. 53.4 Course of subcostal, iliohypogastric, and ilioinguinal nerves. (*1*) External oblique, (*2*) internal oblique, (*3*) transversus abdominis, (*4*) quadratus lumborum; anterior superior iliac spine (ASIS); subcostal nerve (*yellow*); iliohypogastric nerve (*blue*); ilioinguinal nerve (*green*); diaphragmatic crura (*black outline*) (Original illustration by Michael Ee with permission)

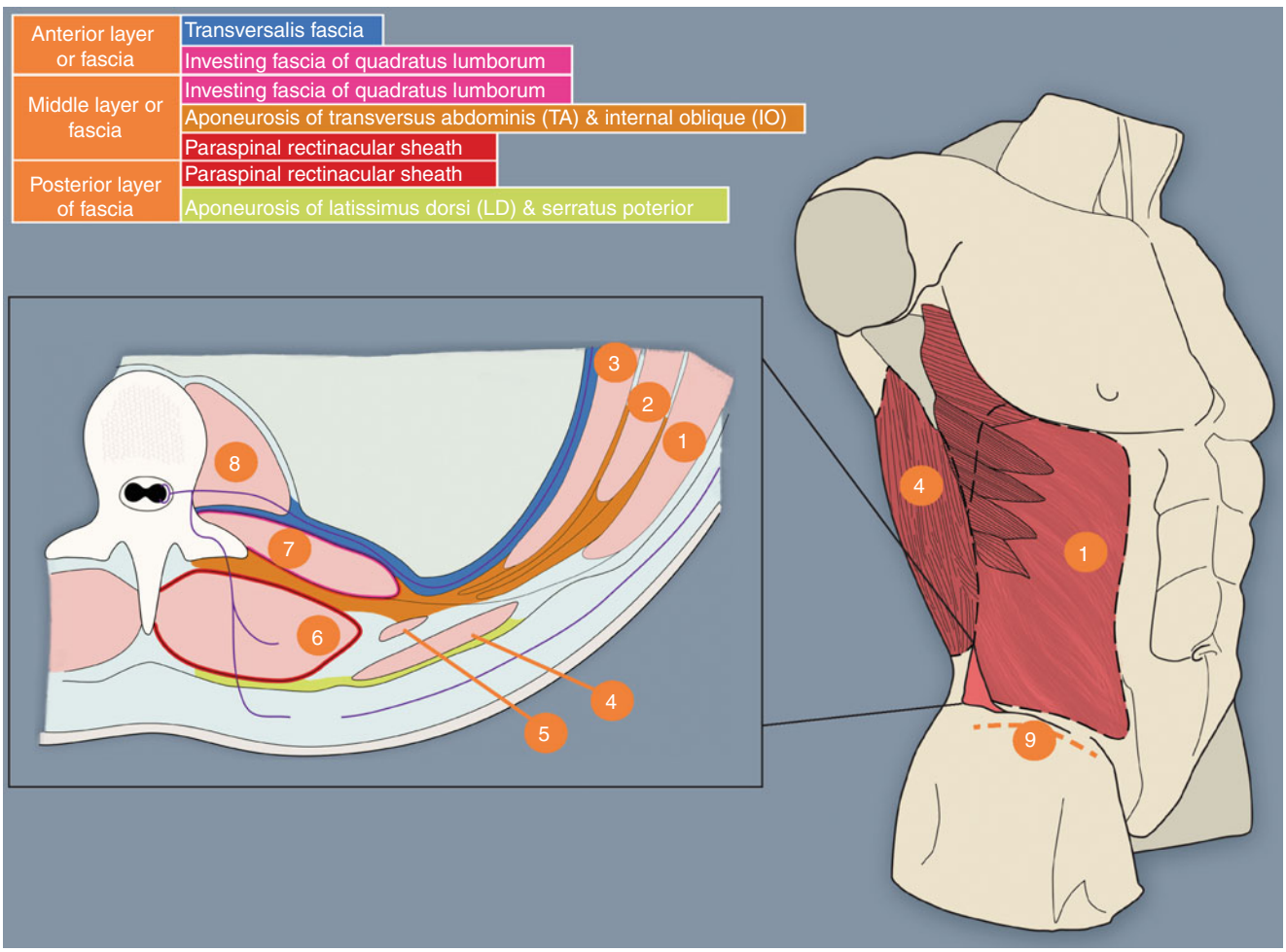


Fig 53.5 Thoracolumbar fascia and relationship to posterior and lateral abdominal wall muscles. (*1*) External oblique, (*2*) internal oblique, (*3*) transversus abdominis, (*4*) latissimus dorsi, (*5*) serratus posterior inferior muscle, (*6*) erector spinae, (*7*) quadratus lumborum, (*8*) psoas major, (*9*) iliac crest (Original illustration by Michael Ee with permission)

Indications

Surgical

TAP blocks have provided surgical anesthesia in high-risk patients for abdominal wall hernia repair, emergency laparotomy [18, 19], and elective Cesarean delivery [20]. The site of block insertion can be modified according to the anticipated site of surgical incision [21]. Despite these case reports, TAP blocks are rarely used for surgical anesthesia.

Therapeutic

Most commonly, TAP blocks are used as one component of multimodal postoperative analgesic technique. They have been effective following colorectal surgery [3], appendicectomy [22], cholecystectomy, prostatectomy, Cesarean section [23], gynecological surgery [24], hernia repair, and renal transplantation [25]. The TAP block is well described following pediatric surgery [26, 27] providing analgesia following ambulatory surgery including hernia repair [28]. A systematic review of studies has demonstrated clinically significant reductions of postoperative opioid requirements and pain [29]. In addition to reduced opioid-related side effects [30], TAP blocks have reduced postoperative opioid consumption in laparoscopic colorectal surgery [31]. A summary of current evidence is provided in Table 53.1. Continuous TAP blockade has been used for treatment of chronic pain [32], for complex traumatic pelvic fractures [33] and for rescue analgesia [34].

Table 53.1 Efficacy of TAP blocks reported in randomized controlled trials

Publication	Surgical procedure	N	Local anesthetic dosage	Comparator	Effect of TAP block on primary outcome
El-Dawlatly (2009) [35]	Laparoscopic cholecystectomy	42	15 ml 0.5 % bupivacaine bilaterally ($n=21$)	No TAP ($n=21$)	Decrease in PCA morphine use in PACU and ward 1st 24 h
Niraj (2009) [36]	Open appendicectomy	52	20 ml 0.5 % bupivacaine unilaterally ($n=26$)	No TAP ($n=26$)	Reduced morphine use and reduced VAS score (at rest and cough) in 1st 24 h. Decreased PONV at 30 min but not at 24 h
Jankovic (2009*) [37]	Renal transplant recipients	40	20 ml 0.375 % levobupivacaine then 0.15 % bupivacaine 10 ml/h ($n=7$)	No TAP ($n=33$)	TAP block reduced morphine requirement and duration of PCA use. No difference in pain scores
Griffiths (2010) [24]	Gynecological cancer surgery	65	20 ml 0.5 % ropivacaine bilaterally ($n=32$)	Normal saline placebo ($n=33$)	No significant difference in opioid use
Mukhtar (2010) [38]	Renal transplant recipients	20	20 ml 0.5 % bupivacaine unilaterally ($n=10$)	No TAP ($n=10$)	Decrease in morphine use. Decrease in pain up to 12 h. Less sedation up to 6 h. Lower PONV scores up to 6 h
Ra (2010) [39]	Laparoscopic cholecystectomy	54	30 ml 0.25 % compared with 30 ml 0.5 % levobupivacaine bilaterally ($n=18$, 18)	Normal saline placebo ($n=18$)	In TAP groups: decrease in i.v. opioid use and pain up to 24 h. No difference between 0.25 % and 0.5 % levobupivacaine
Conaghan (2010) [40]	Laparoscopic colorectal surgery	74	20 ml of 0.25 % levobupivacaine bilaterally ($n=40$)	No TAP ($n=34$)	Decrease in i.v. opioid use. Decreased length of hospital stay
Milan (2011) [41]	Liver transplant	34	20 ml 0.5 % levobupivacaine bilaterally ($n=17$)	No TAP ($n=17$)	Reduced morphine consumption and median pain scores
Aniskevich (2011)[a] [42]	Pancreas transplant	1	20 ml 0.5 % ropivacaine bilaterally	n/a	Successful use of TAP block for postoperative rescue analgesia in chronic opioid user
Bharti (2011) [43]	Colorectal surgery	40	20 ml 0.25 % bupivacaine bilaterally ($n=20$)	Normal saline placebo ($n=20$)	Decreased morphine use and rest and dynamic pain. Reduced sedation in TAP group (0 to 6 h). Improved patient satisfaction
Atim (2011) [44]	Hysterectomy	55	20 ml 0.25 % bupivacaine bilaterally ($n=18$)	Normal saline placebo ($n=18$) and local wound infiltration 0.25 % bupivacaine ($n=19$)	Reduced pain compared to both placebo and local infiltration
Sandeman (2011) [26]	Pediatric Laparoscopic appendicectomy	87	0.5 ml/kg 0.2 % ropivacaine bilaterally ($n=42$)	Local infiltration to port sites only ($n=45$)	No clinically important benefit
Niraj (2011) [45]	Open hepatectomy and renal surgery	62	Bilateral TAP catheter ($n=29$), 1 mg/kg 0.375 % bupivacaine	Epidural ($n=33$)	No difference in pain scores and PONV

(continued)

Table 53.1 (continued)

Publication	Surgical procedure	N	Local anesthetic dosage	Comparator	Effect of TAP block on primary outcome
Melnikov (2011) [46]	Major gynecological surgery for cancer	58	0.375 ml/kg 0.25 % bupivacaine with 5 mcg/ml adrenaline bilaterally ($n=19$)	Bilateral thoracic paravertebral block ($n=19$)	No difference in pain scores and PONV
Kadam (2011) [47]	Major abdominal surgery	20	15 ml 0.5 % ropivacaine with continuous infusion 0.2 % ropivacaine bilaterally ($n=10$)	No TAP ($n=10$)	Reduced pain scores and fentanyl consumption
Petersen (2012) [48]	Laparoscopic cholecystectomy	74	20 ml 0.5 % ropivacaine bilaterally ($n=37$)	Normal saline placebo ($n=37$)	Some reduction in VAS scores for pain on coughing and slightly less opioid requirement
Tan (2012) [49]	Cesarean section	40	20 ml 0.25 % levobupivacaine bilaterally ($n=20$)	No block ($n=20$)	Reduction morphine consumption, increased patient satisfaction
Tolchard (2012) [50]	Laparoscopic cholecystectomy	43	1 mg/kg bupivacaine unilaterally ($n=21$)	Local infiltration to port sites only ($n=22$)	Reduced VAS scores and fentanyl requirement in PACU
Walter (2013) [31]	Laparoscopic colorectal surgery	68	20 ml 0.2 % levobupivacaine bilaterally ($n=33$)	No TAP ($n=35$)	Reduced opioid use 1st 24 h
Albrecht (2013) [51]	Laparoscopic gastric-bypass surgery	70	30 ml 0.25 % bupivacaine with adrenaline bilaterally ($n=35$)	Local infiltration to port sites only ($n=35$)	No difference in opioid consumption during 1st 24 h. Rates of PONV equivocal
Sinha (2013) [52]	Laparoscopic bariatric surgery	100	20 ml 0.375 % ropivacaine bilaterally ($n=50$)	No TAP ($n=50$)	Reduced opioid requirements and VAS scores
Sahin (2013) [53]	Pediatric inguinal hernia repair	57	0.25 % levobupivacaine 0.5 ml/kg unilaterally ($n=29$)	Wound infiltration ($n=28$)	Reduced pain, prolonged effect
Wu (2013) [54]	Radical gastrectomy	82	20 ml 0.375 % ropivacaine bilaterally ($n=27$)	Thoracic epidural ($n=29$), GA only ($n=26$)	TAP reduced opioid consumption than GA alone; equivocal pain scores. Thoracic epidural less opioid consumption than TAP block; equivocal pain scores
Parikh (2013) [55]	Donor nephrectomy	60	25 ml 0.375 % bupivacaine unilaterally ($n=30$)	Placebo control with saline ($n=30$)	Reduced tramadol consumption in the 1st 24 h
Gasanova (2013) [56]	Total abdominal hysterectomy	74	Group 1, 20 ml 0.5 % bupivacaine bilaterally ($n=25$) with multimodal analgesia; Group 2, TAP block only ($n=24$)	Group 3, multimodal analgesia only, no TAP block ($n=25$)	Pain on coughing less variable where TAP block was combined with multimodal analgesia. No difference in pain at rest
Sivapurapu (2013) [57]	Lower abdominal gynecological surgery	52	0.3 ml/kg 0.25 % bupivacaine bilaterally ($n=26$)	Direct wound infiltration ($n=26$)	Reduced VAS and increased time to request first rescue analgesia
Gomez-Rios (2014)[b] [58]	Major gynecological and obstetric surgery	6	Continuous infusion of 0.125 % levobupivacaine 2 ml/h bilaterally for 50 h	n/a	Reduced opioid requirements and improved postoperative mobility
Niraj (2014) [59]	Laparoscopic cholecystectomy	61	Four-quadrant TAP block 2.5 mg/kg 0.375 % levobupivacaine plus continuous infusion 0.25 % levobupivacaine ($n=30$)	Epidural ($n=31$)	No difference between groups in VAS scores at rest or with coughing
Aniskevich (2014) [60]	Laparoscopic assisted nephrectomy	21	20 ml 0.5 % ropivacaine, lateral approach bilaterally ($n=10$)	Placebo saline control ($n=11$)	Reduced opioid requirements and lower pain scores at 24 h
Mckeen (2014) [61]	Post-Cesarean delivery	74	Spinal anesthetic and post-op US-guided TAP, low-dose ropivacaine ($n=35$)	Control ($n=39$)	No statistically significant difference in pain scores, sedation, or opioid consumption
Soltani (2014) [25]	Renal transplant recipients	44	Post-induction under US guidance 15 ml 0.25 % bupivacaine and adrenaline ($n=22$)	Placebo control with saline ($n=22$)	Decreased morphine consumption and lower pain scores in 1st 24 h. Reduced intraoperative fentanyl consumption in TAP group

(continued)

Table 53.1 (continued)

Publication	Surgical procedure	N	Local anesthetic dosage	Comparator	Effect of TAP block on primary outcome
Calle (2014) [62]	Laparoscopic hysterectomy	197	Bilateral with 0.25 % bupivacaine	Placebo control with saline	No difference in opioid requirements at 24, 48, 72 h postoperatively
Marais (2014) [63]	Open total abdominal hysterectomy	30	20 ml 0.25 % bupivacaine bilaterally ($n=15$)	Placebo control with saline ($n=15$)	Reduced PCA morphine use
Chandon (2014) [64]	Post-Cesarean analgesia	65	20 ml 0.375 % levobupivacaine bilaterally ($n=36$)	Continuous wound infusion ($n=29$)	No difference in rest and dynamic pain scores between groups. Study terminated early due to generalized seizure in 1 patient in TAP group
Bhatacharjee (2014) [65]	Total abdominal hysterectomy	90	1 ml/kg 0.25 % bupivacaine bilaterally ($n=45$)	Placebo control with saline ($n=45$)	Lower rest and dynamic VAS scores in immediate postoperative period
Heil (2014) [66]	Hernia surgery	20	20 ml 0.5 % ropivacaine bolus plus continuous infusion 0.2 % ropivacaine ($n=10$)	Placebo control with saline infusion ($n=10$)	No significant difference in dynamic pain scores on postoperative day 1
De Oliveira Jr (2014) [67]	Laparoscopic gastric banding	19	20 ml 0.5 % ropivacaine bilaterally ($n=10$)	Placebo control with saline ($n=9$)	Improved quality of postoperative recovery and reduced opioid use

*Retrospective audit, [a]case report, [b]case series. *PACU* post anesthetic care unit, *VAS* visual analog scale, *PCA* patient-controlled analgesia, *PONV* postoperative nausea and vomiting, *n/a* not applicable, *i.v.* intravenous

Contraindications

Infections and skin diseases in the injection area

Surgical dressings obstructing access

Patient refusal

Advantages/Disadvantages

TAP blocks reduce side effects associated with epidural or opioid analgesia failure rate and need for re-siting of block or catheter.

Inadequate analgesic coverage for visceral pain.

Procedure

Preparation

As with all regional anesthesia procedures, requirements include emergency equipment, monitoring, and assistance.

Materials and Disposables

We recommend a 38–50-mm intermediate frequency probe for adult patients.

Sterile ultrasound probe cover and gel for all procedures.

Routine disposables including fenestrated drape and dressings.

21- or 18-gauge short-bevel needles for single-injection or continuous catheter techniques, respectively, 100–150-mm needle required for in-plane technique

20–30 ml of local anesthetic for block (see section on *Local Anesthetic Dosage*, *Volume, and Spread*)

Patient Positioning

Supine however consider lateral or lateral tilt for more posterior approach

Ergonomics

We suggest positioning the ultrasound machine on the opposite side of the patient to the proceduralist, so that he/she directly faces the screen. We suggest first scanning by identifying the rectus abdominis muscles, then the linea semilunaris aponeurosis (separating the rectus abdominis from the three anterolateral abdominal wall muscles), and then the anterolateral muscles (Fig. 53.6). Dynamic scanning from medial to lateral or vice versa helps with correct identification of muscle layers (Fig. 53.7). In many patients, the rectus abdominis muscle is displaced further away from the midline than expected. To identify the TAP, it is useful to appreciate: (1) the transversus abdominis muscle may appear hypoechoic, and (2) the transverse abdominis muscle passes under the rectus abdominis close to the xiphisternum. Therefore, scanning in the upper part of the abdomen toward the midline is very helpful in locating the transverse abdominis muscle. Injecting between the rectus abdominis and transverse abdominis muscles close to the xiphisternum may increase the likelihood of anesthetizing T6–8 segmental nerves.

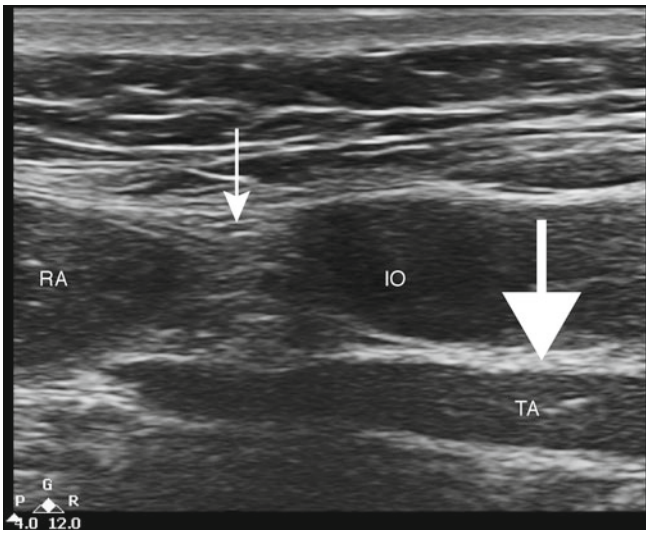


Fig. 53.6 Linea semilunaris under ultrasound. Linea semilunaris (*white arrow*), rectus abdominis (*RA*), internal oblique (*IO*), transversus abdominis (*TA*), transversus abdominis plane (*bold white arrow*)

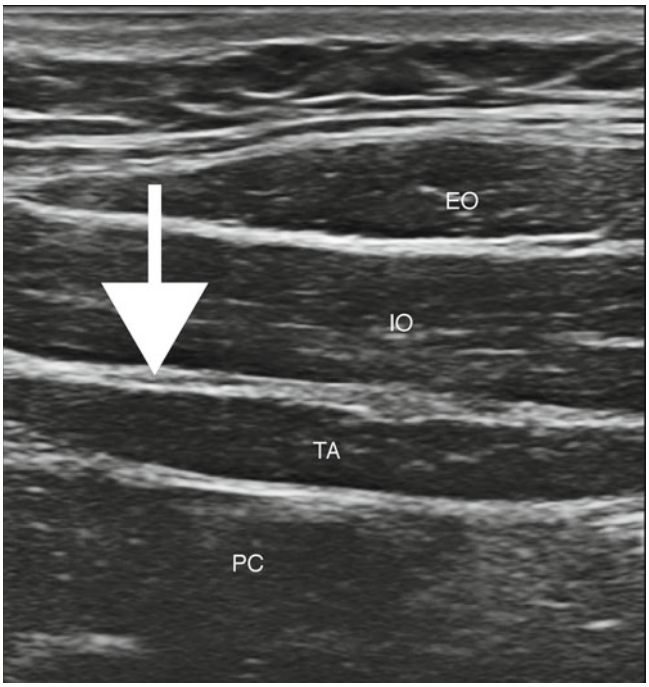


Fig. 53.7 Identification of muscle layers under ultrasound. External oblique (*EO*), internal oblique (*IO*), transversus abdominis (*TA*), peritoneal cavity (*PC*), transversus abdominis plane (*bold white arrow*)

Injection Technique: General Comments

For a single-shot technique, sterile gloves should be worn and skin asepsis adhered to. For continuous catheter technique, we recommend a full aseptic technique (gown, gloves, and facemask). Needle imaging can be improved by separating the initial needle entry point through the skin from the probe by about 5 cm and heel–toe the probe (rocking probe in its long axis). Particular vigilance must be taken to always keep the needle tip in view, especially with the use of longer needles.

Specific Approaches

Subcostal TAP Block

The transversus abdominis muscle is located posterior to the rectus abdominis muscle close to the xiphoid process. The segmental nerves are located in this plane, but T6–T8 may only have a short course between the transversus abdominis and the rectus abdominis muscles. The nerves may, after a short distance beyond the costal margin, penetrate the posterior rectus sheath and rectus muscle; therefore, the injection close to the costal margin may improve anesthesia [68]. For a subcostal approach, the authors' preferred needle trajectory is from anteromedial to posterolateral using an in-plane technique with the needle trajectory parallel to the costal margin (Fig. 53.8). Beginning at the transversus–rectus abdominis interface medially near the xiphoid process, an incremental injection of 3–5 ml of local anesthetic can then extend the hydrodissection posterolaterally [69] (Fig. 53.9).

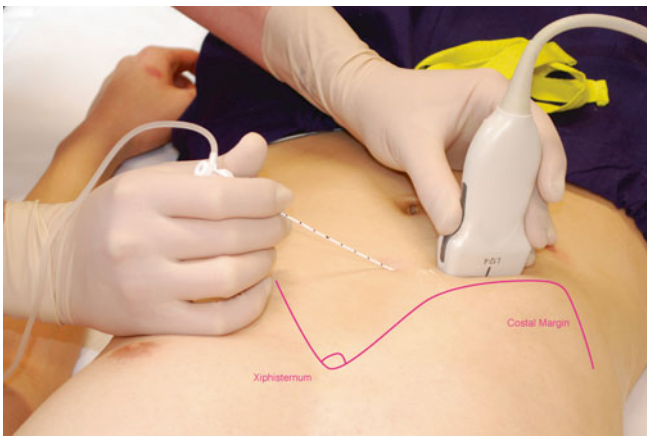


Fig. 53.8 Injection technique for subcostal TAP block

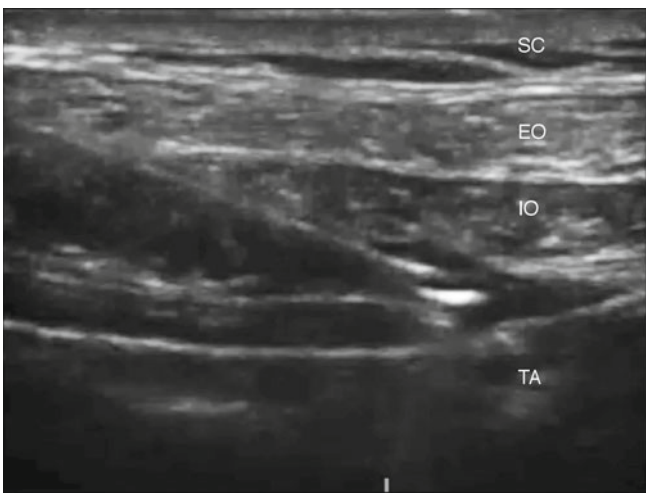


Fig. 53.9 Endpoint for TAP block under ultrasound guidance. Subcutaneous tissue (*SC*), external oblique (*EO*), internal oblique (*IO*), transversus abdominis (*TA*). Note hydrodissection around needle tip

Lateral TAP Block

The ultrasound probe is positioned in the mid-to-anterior axillary line between the iliac crest and the costal margin (Fig. 53.10). In many patients, the distance between these two landmarks is small, so the exact position (between the iliac crest and costal margin) probably does not matter. An in-plane technique with the needle directed anterior to posterior (toward the midaxillary line) is straightforward. Incremental injections, hydrodissection, and then needle advancement are similar to the subcostal technique. Placing the probe closer to the iliac crest may improve coverage of L1.

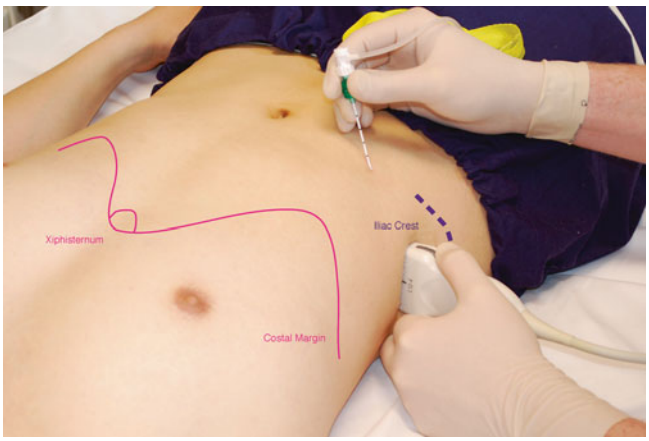


Fig. 53.10 Injection technique for lateral TAP block

Local Anesthetic Dosage, Volume, and Spread

Dosage

Optimal volumes and concentrations of local anesthetics for use in TAP block have yet to be determined. The area of spread is related to the total volume of injectate used, rather than concentration. A balance must be found between achieving optimal spread of sensory anesthesia and avoiding toxicity. It is the author's practice to use approximately ropivacaine 3–3.5 mg/kg as the total dose received by the patient diluted to 20–30 ml for each side. Caution should be applied in pregnant patients, where smaller doses should be considered. If using a catheter technique, a starting infusion rate of 5 ml/h of ropivacaine 0.2 % may be used, with subsequent increase to 7 ml/h following a 10-ml bolus [5].

Distribution

Descriptions of the extent of sensory blockade achieved with TAP have been inconsistent. Landmark techniques appear to produce wider sensory blockade than ultrasound techniques. The reason for this is unclear; however, Carney has hypothesized that this may be due to posterior spread of local anesthetic cephalad and caudad within the paravertebral space with landmark technique [68]. In contrast, visualization of the needle tip under ultrasound guidance allows precise deposition of local anesthetic within the TAP plane, resulting in anterior spread of anesthesia [68]. Boerglum et al. [70] compared the spread of blockade using low (15 ml) and high volumes (30 ml) of local anesthetic for the lateral and subcostal approaches and reported that each approach has separate and distinct boundaries as seen on magnetic resonance imaging (Fig. 53.11). Lee et al. demonstrated the upper limit of sensory blockade to be T8 (lower limit T11) with the subcostal approach compared to an upper limit of T10 (lower limit T12) with the lateral approach [21]. Therefore, it would appear that specific approaches may be targeted to the type of abdominal surgery—subcostal for upper abdominal surgery, lateral for lower abdominal surgery (Fig. 53.11) [21]. Sensory blockade of the entire anterior abdomen (T6-T12) may be obtained with dual subcostal and lateral injections performed bilaterally [70].

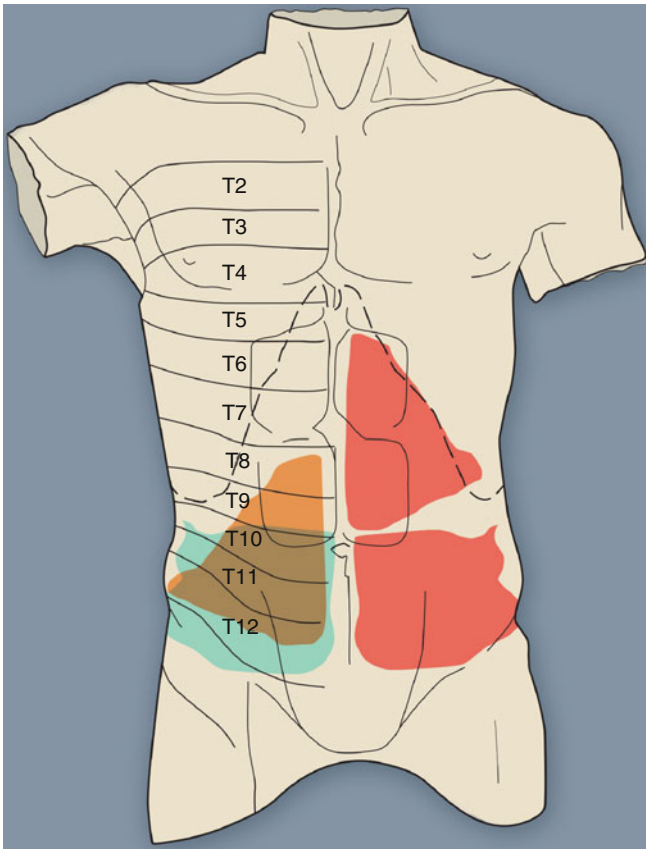


Fig. 53.11 Spread of sensory blockade with TAP block. In *orange*, sensory blockade obtained with subcostal TAP demonstrated by Lee et al. (2010); in *blue*, sensory blockade obtained with lateral TAP demonstrated by Lee et al. (2010); in *red*, sensory blockade obtained with combined dual lateral and subcostal injections by Boerglum et al. (2012). Note greater spread of sensory blockade with dual versus single injections in the study by Boerglum et al. (Original illustration by Michael Ee with permission)

Side Effects and Complications

Accidental puncture of abdominal viscera has been reported using the landmark technique and with ultrasound guidance. Intraperitoneal injection, bowel and flank hematoma [71], local anesthetic toxicity manifesting as generalized convulsions [64, 72], intravascular injection, and block failure have been reported. With the use of continuous catheter techniques, the risk of leakage, filter disconnection, and dislodgement of the catheter are possible [47].

Rectus Sheath Block

The rectus sheath block has been used effectively following incisional and umbilical hernia repair in children, Cesarean section (when midline incision is used), and laparoscopy. The rectus sheath block aims to block the terminal branches of segmental nerves T6–L1. Anteriorly, the rectus sheath is tough and fibrous from the xiphisternum to the pubis. Posteriorly, it is well-defined and identifiable cephalad to the level of the umbilicus and inferiorly fades into the transversalis fascia. The posterior rectus sheath has been described as a "backboard" for injecting local anesthetics [73].

Materials and Disposables

Similar as for TAP block

Patient Positioning

Supine

Ultrasound-Guided Technique

Identifying the linea semilunaris on the lateral border of the rectus abdominis muscle as for the subcostal approach is useful. The transducer is then slid medially to locate the rectus abdominis. The layers of the rectus sheath are readily identified with ultrasound with transverse scanning of the rectus abdominis and either out-of-plane or in-plane needle imaging techniques. The target for deposition of a local anesthetic bolus is between the posterior rectus sheath and posterior rectus muscle. In a training study, ultrasound techniques significantly improved the accuracy of needle placement compared with landmark technique [73]. Ultrasound-guided rectus sheath catheters have been described using an in-plane technique [74].

Side Effects and Complications

Local anesthetic toxicity, block failure, and localized hemorrhage are possible as in TAP blocks. Higher risk of abdominal viscera puncture may be associated with difficulty visualizing the posterior rectus sheath especially below the umbilicus—a problem that appears to be alleviated with ultrasound guidance.

Ilioinguinal–Iliohypogastric Nerve Block

IIN and IHN blocks are commonly performed blocks suitable for anesthesia/analgesia for infraumbilical procedures such as hernia repair. More recently, they have also been found to be effective in the treatment of persistent postherniorrhaphy pain [75]. They can and are typically performed "blind"; however, previously reported failure rates may be between 10 and 25 % [76]. This may be improved with ultrasound guidance. A study comparing the use of landmark-based IIN/IHN nerve blocks with ultrasound-guided TAP blocks in ambulatory open inguinal hernia repair found the latter to provide superior analgesia with reduction in pain scores and opioid use [28].

Materials and Disposables

Similar as for TAP and rectus sheath blocks

Patient Positioning

Supine

Ultrasound-Guided Technique

The TAP and IIN and IHN nerves within it can be identified using sonography close to the anterior superior iliac spine. The nerves may be visualized as flat, ovoid-shaped structures, and the deep circumflex artery can serve as a useful landmark [13, 77]. Compared to the TAP and rectus sheath blocks, smaller volumes of local anesthetic may be used here, e.g., 10–12 ml. Even with ultrasound guidance, the IIN and IHN nerves are often blocked together due to spread of local anesthetic [78].

Side Effects and Complications

As with TAP and rectus sheath blocks, side effects and complications are related to local anesthetic toxicity, block failure, vessel puncture, and visceral organ injury. Cases of bowel perforation have been reported in children [79].

Quadratus Lumborum Block

Definition

Local anesthetic is deposited within close proximity to the quadratus lumborum muscle in the lower lumbar region of the trunk [80]. The local anesthetic potentially spreads cranially along the muscle, posterior to the arcuate ligaments of the diaphragm, and reaches the thoracoabdominal nerves (T6–T12) as they traverse the lower thoracic paravertebral space [68].

Background

The quadratus lumborum block (QLB) is a novel technique. Reports of its clinical use in the peer-reviewed literature are limited to abstracts from scientific meetings [81] and case reports [82, 83]. As such, the safety profile and effectiveness of the QLB is unknown. QLB was first described by Blanco in 2007 [80]. It can be thought of as a posterior extension of an ultrasound-guided TAP block. The aim is to position the needle tip at the lateral border of quadratus lumborum, where it intersects with the aponeurosis of transversus abdominis. Local anesthetic may be placed along the anterolateral border or the posterior border of quadratus lumborum. Care must be taken to stay superficial to the transversalis fascia. The transmuscular approach was described by Boerglum et al. in 2013 [84]. It differs from the original description in terms of patient positioning, sonoanatomy, needle trajectory, and target site for local anesthetic deposition. Nevertheless, the aim is still to deposit local anesthetic along the anterior border of quadratus lumborum to achieve cranial spread to the paravertebral space.

Anatomy

Quadratus lumborum is a posterior abdominal wall muscle. It originates from the iliac crest and inserts onto the transverse processes of the first four lumbar vertebrae (L1–L4) and the inferior border of the twelfth rib (T12) (Fig. 53.4). It is thick at the lower lumbar level and tapers off cranially as its medial fibers insert into the vertebral column, which is why it is best imaged at the level of L4. The characteristic sonoanatomy for the transmuscular approach is that of a shamrock. The stem of the shamrock is the transverse process of L4, and the leaves are the paraspinal muscles, quadratus lumborum, and psoas major (Fig. 53.12). The "Shamrock sign" was originally described with reference to performing an ultrasound-guided psoas compartment block (lumbar plexus block) [85]; however, it has also been adopted for the transmuscular approach to quadratus lumborum [84].

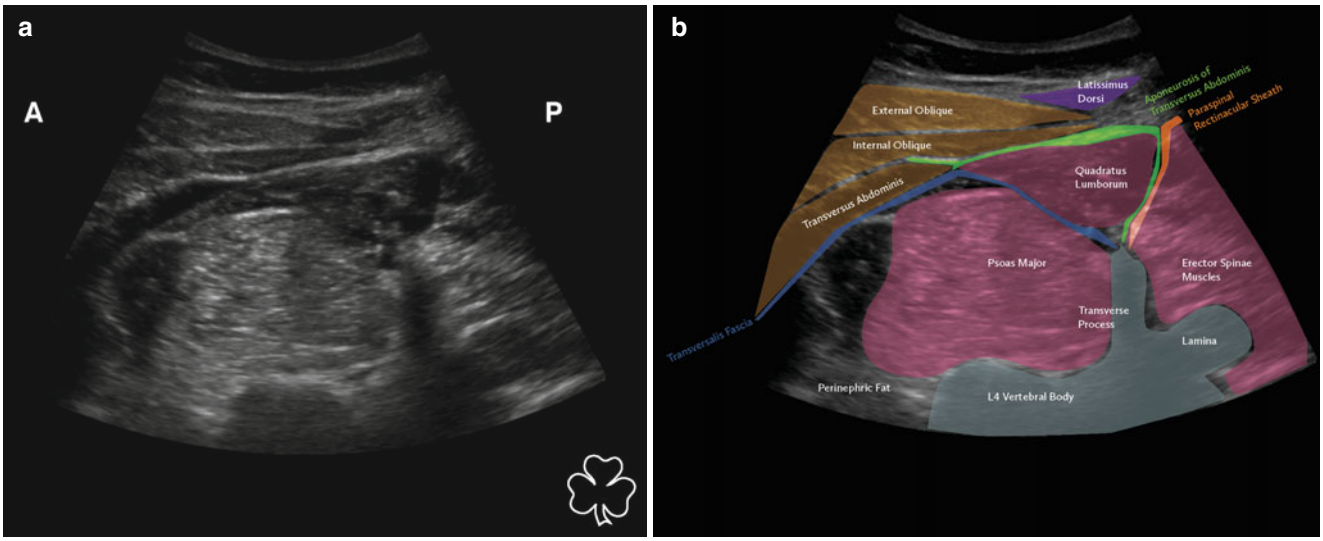


Fig. 53.12 Sonoanatomy of "Shamrock sign." (**a**) Raw sonogram, (**b**) overlying annotations outlining relevant structures. *A* anterior, *P* posterior

Indications

Surgical specific indications for QLB await results of randomized controlled trials.

Contraindications

Infection and skin diseases in the injecting area

Poor sonoanatomical localization of structure

Advantages/Disadvantages

The QLB has the potential to reduce opioid requirements and therefore opioid-related side effects. It has a relatively long onset time (30 min).

Procedure

Preparation

As with all regional anesthesia procedures, requirements include emergency equipment, monitoring, and assistance.

Materials and Disposables

Ultrasound machine with curvilinear probe (an intermediate frequency probe may suffice in very thin patients)

Sterile ultrasound probe cover and sterile ultrasound gel

100–150-mm short-bevel needle for single-injection block

20–30 ml of local anesthetic (e.g., 20–30 ml of 0.5 % ropivacaine)

Patient Positioning

For the transmuscular approach, the patient lies in the lateral position with the side that is to be blocked uppermost.

The proceduralist sits or stands behind the patient such that his/her nondominant hand will be scanning the lateral abdominal wall.

Technique: Transmuscular Approach

Ultrasound machine is placed on the opposite side of the patient to the proceduralist.

Place the curvilinear probe in the midaxillary line just cephalad to the iliac crest (Fig. 53.13).

Scan anteriorly to identify the three muscles of the anterior abdominal wall (transversus abdominis, internal oblique, and external oblique). The transversus abdominis typically tapers to form a hyperechoic aponeurosis that passes posterior to quadratus lumborum. Caudad angulation of the probe may help accentuate quadratus lumborum.

Continue to scan posteriorly until the "Shamrock sign" is imaged.

Position the needle such that it enters the skin posterior to the ultrasound probe and passes in-plane lateral to the paraspinal muscles, lateral to the transverse process of L4, and through quadratus lumborum. The target site for injection is the plane between quadratus lumborum and psoas major (Fig. 53.14).

Inject a 1–2-ml test dose to confirm correct needle-tip position, followed by 20–30 ml of 0.5 % ropivacaine.

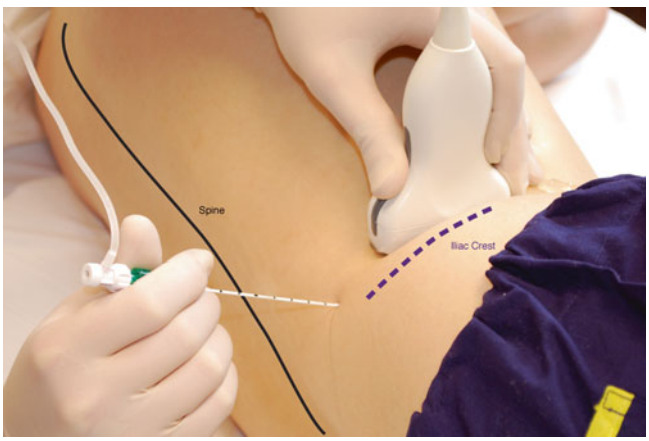


Fig. 53.13 Injection technique for quadratus lumborum block. Spine (*black line*), iliac crest (*purple-dotted line*)

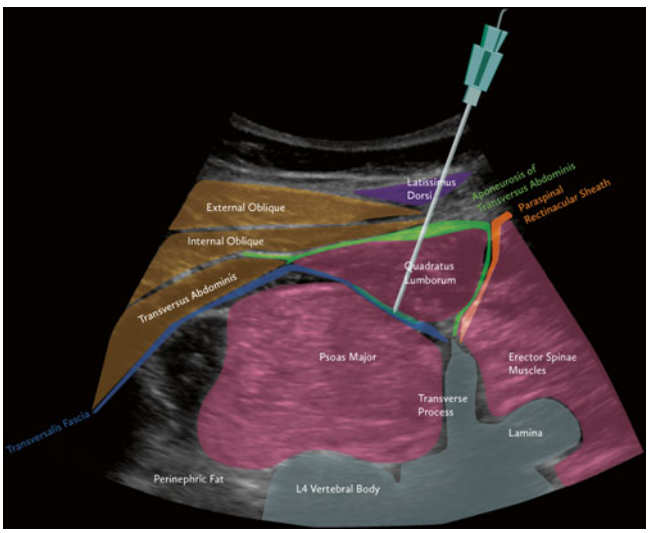


Fig. 53.14 Target site for deposition of local anesthetic under ultrasound guidance for transmuscular approach to quadratus lumborum block. Note the deposition of local anesthetic between quadratus lumborum and psoas major, above the transversalis fascia

Side Effects

Advancement of the needle through psoas major into the retroperitoneum could potentially cause hemorrhage or renal injury. It is therefore important to only embark on the transmuscular approach when sonoanatomy is favorable such that psoas major is imaged anterior to quadratus lumborum.

Transversalis Fascia Block

Definition

Local anesthetic is deposited between the transversus abdominis muscle and underlying transversalis fascia adjacent to the quadratus lumborum muscle. Target nerves are the T12 subcostal nerve and the iliohypogastric and ilioinguinal nerves (L1).

Background

The transversalis fascia plane block was first described by Hebbard in 2009 [86]. It shares similarities with the standard ultrasound-guided TAP block and the QLB with regards to sonoanatomy and needle trajectory. It is a novel technique which may have a role for postoperative analgesia following iliac crest bone graft harvest [87].

Anatomy

The transversalis fascia lies deep to transversus abdominis and continues posteromedially to cover the anterior surface of quadratus lumborum. The subcostal nerve, iliohypogastric nerve, and ilioinguinal nerves originate within the body of psoas major as part of the lumbar plexus (Fig. 53.4). They emerge from the lateral border of psoas major and travel across the anterior surface of quadratus lumborum before piercing transversus abdominis to course within the TAP plane. Because the iliohypogastric and ilioinguinal nerves course within the TAP plane for such a short and variable length, the L1 dermatomes are not reliably or consistently blocked as part of a standard ultrasound-guided TAP block. Sonoanatomy for the transversalis fascia plane block overlaps with the lateral ultrasound-guided TAP block and the QLB. The key difference is that the target site for needle-tip position and local anesthetic deposition is immediately beneath the transversus abdominis muscle but more anterior to quadratus lumborum muscle.

Indications

Regional analgesia for surgeries that involve primarily L1 dermatome (e.g., iliac crest bone graft harvest, inguinal herniorrhaphy)

Contraindications

Infection and skin diseases in the injecting area
Poor sonoanatomical localization of structure

Advantages/Disadvantages

The transversalis fascia plane block may reduce opioid requirements and therefore opioid-related side effects. The main disadvantage is the relatively small area anesthetized (limited to the T12/L1 dermatome); however, it may be combined with a TAP block technique to extend anesthesia more cephalad than T12.

Procedure

Preparation

As with all regional anesthesia procedures, requirements include emergency equipment, monitoring, and assistance.

Materials and Disposables

Ultrasound machine with curvilinear probe (an intermediate frequency probe may sufficient in very thin patients)

Sterile ultrasound probe cover

Sterile ultrasound gel

100–150-mm short-bevel needle for single-injection block

20 ml of local anesthetic (e.g., 20 ml of 0.5 % ropivacaine)

Patient Positioning

The patient lies in the lateral position with the side that is to be blocked uppermost.

The proceduralist sits or stands in front of the patient such that his/her nondominant hand will be scanning the lateral abdominal wall.

Technique

Ultrasound machine is placed on the opposite side of the patient to the proceduralist.

Place the curvilinear probe in the midaxillary line just cephalad to the iliac crest (Fig. 53.15).

Scan anteriorly to identify the three muscles of the anterior abdominal wall (transversus abdominis, internal oblique, and external oblique). The transversus abdominis typically tapers to form a hyperechoic aponeurosis that passes posterior to quadratus lumborum.

Continue to scan posteriorly such that there are no solid organs (e.g., kidney, liver) or viscera deep to the transversus abdominis that may be injured with the needle.

Position the needle such that it enters the skin anterior to the ultrasound probe and passes in-plane posterolateral through the three lateral abdominal muscles.

The target site for injection should be in the plane between the transversus abdominis muscle and the transversalis fascia anterolateral to quadratus lumborum (Fig. 53.16).

Inject a 1–2-ml test dose to confirm correct needle-tip position, followed by 20 ml of 0.5 % ropivacaine.

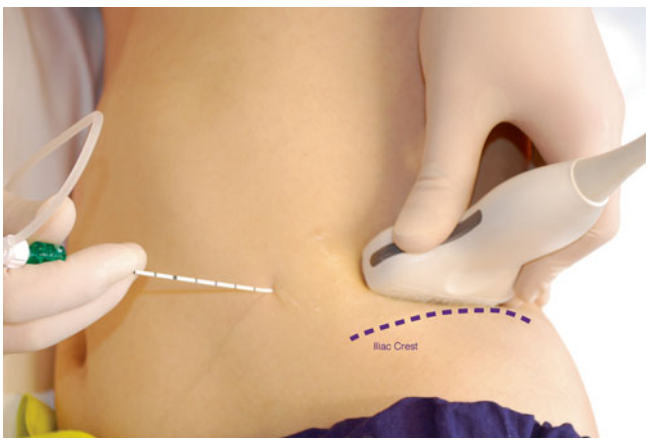


Fig. 53.15 Injection technique for transversalis fascia block. Iliac crest (*purple-dotted line*)

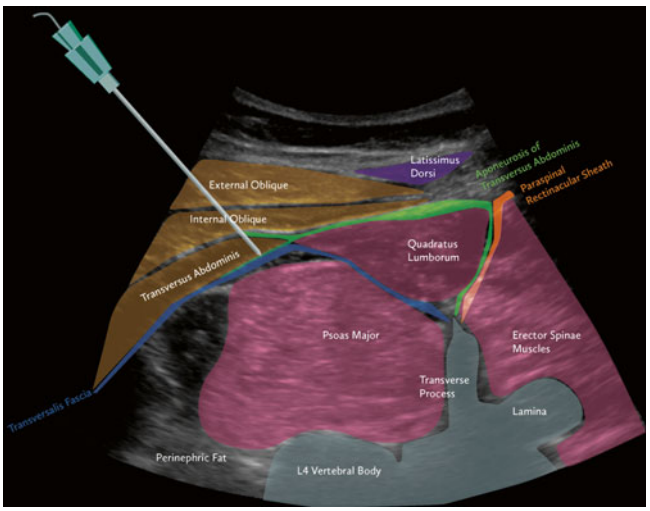


Fig. 53.16 Target site for deposition of local anesthetic under ultrasound guidance for transversalis fascia block. Note the deposition of local anesthetic between transversus abdominis and transversalis fascia

Side Effects

There is a theoretical risk of perforating solid organs (e.g., kidney, liver) or viscera if the needle passes beyond the transversalis fascia. This risk is mitigated against by positioning the patient lateral (rather than supine) and performing the block as posteriorly and as caudad as possible.

References

1. Rafi AN. Abdominal field block: a new approach via the lumbar triangle. Anaesthesia. 2001;56(10):1024–6.
2. McDonnell JG, Curley G, Carney J, Benton A, Costello J, Maharaj CH, et al. The analgesic efficacy of transversus abdominis plane block after cesarean delivery: a randomized controlled trial. Anesth Analg. 2008;106(1):186–91.
3. McDonnell JG, O'Donnell B, Curley G, Heffernan A, Power C, Laffey JG. The analgesic efficacy of transversus abdominis plane block after abdominal surgery: a prospective randomized controlled trial. Anesth Analg. 2007;104(1):193–7.
4. Hebbard P. Subcostal transversus abdominis plane block under ultrasound guidance. Anesth Analg. 2008;106(2):674–5.
5. Hebbard PD, Barrington MJ, Vasey C. Ultrasound-guided continuous oblique subcostal transversus abdominis plane blockade: description of anatomy and clinical technique. Reg Anesth Pain Med. 2010;35(5):436–41.
6. Barrington M. The International Registry of Regional Anaesthesia [Internet]. Available from: http://regionalanaesthesia.wordpress.com.
7. McDermott G, Korba E, Mata U, Jaigirdar M, Narayanan N, Boylan J, et al. Should we stop doing blind transversus abdominis plane blocks? Br J Anaesth. 2012;108(3):499–502.
8. McMinn RM. Last's Anatomy – Regional and Applied. 8th ed. Edinburgh/New York: Churchill Livingstone; 1990.
9. Snell R. Clinical anatomy by regions. 8th ed. Baltimore: Lippincott Williams and Wilkins; 2008.
10. Sakamoto H, Akita K, Sato T. An anatomical analysis of the relationships between the intercostal nerves and the thoracic and abdominal muscles in man. II. Detailed analysis of innervation of the three lateral abdominal muscles. Acta Anat Basel. 1996;156:143–50.
11. Rab M, Ebmer J, Dellon A. Anatomical variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
12. Ndiaye A, Diop M, Ndoye JM, Ndiaye A, Mané L, Nazarian S, et al. Emergence and distribution of the ilioinguinal nerve in the inguinal region: applications to the ilioinguinal anaesthetic block (about 100 dissections). Surg Radiol Anat. 2010;32(1):55–62.
13. Gofeld M, Christakis M. Sonographically guided ilioinguinal nerve block. J Ultrasound Med Off J Am Inst Ultrasound Med. 2006;25(12):1571–5.
14. Eichenberger U, Greher M, Kirchmair L, Curatolo M, Moriggl B. Ultrasound-guided blocks of the ilioinguinal and iliohypogastric nerve: accuracy of a selective new technique confirmed by anatomical dissection. Br J Anaesth. 2006;97:238–43.
15. Klaassen Z, Marshall E, Tubbs R, Louis RJ, Wartmann C, Loukas M. Anatomy of the ilioinguinal and iliohypogastric nerves with observations of their spinal nerve contributions. Clin Anat. 2011;24:1618–23.
16. Rozen W, Tran T, Barrington M, Ashton M. Avoiding denervation of the rectus abdominis muscle in DIEP flap harvest III:a functional study of the nerves to the rectus using anaesthetic blockade. Plast Reconstr Surg. 2009;124:519–22.
17. Moore KL. Clinically oriented anatomy. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 1999. 1164 p.
18. Jensen K, Baek N, Jensen JT, Brglum J. Bilateral dual transversus abdominis plane block providing surgical anaesthesia for abdominal wall surgery. Anaesthesia. 2013;68(1):106–8.
19. Mishra L, Pani N, Mishra D, Patel N. Bilateral transversus abdominis plane block as a sole anesthetic technique in emergency surgery for perforative peritonitis in a high risk patient. J Anaesthesiol Clin Pharmacol. 2013;29(4):540.
20. Mei W, Jin C, Feng L, Zhang Y, Luo A, Zhang C. Bilateral ultrasound-guided transversus abdominis plane block combined with ilioinguinal-iliohypogastric nerve block for cesarean delivery anesthesia. Anesth Analg. 2011;113(1):134–7.
21. Lee THW, Barrington MJ, Tran TMN, Wong D, Hebbard PD. Comparison of extent of sensory block following posterior and subcostal approaches to ultrasound-guided transversus abdominis plane block. Anaesth Intensive Care. 2010;38(3):452–60.
22. Carney J, Finnerty O, Rauf J, Curley G, McDonnell JG, Laffey JG. Ipsilateral transversus abdominis plane block provides effective analgesia after appendectomy in children: a randomized controlled trial. Anesth Analg. 2010;111(4):998–1003.
23. Belavy D, Cowlishaw PJ, Howes M, Phillips F. Ultrasound-guided transversus abdominis plane block for analgesia after Caesarean delivery. Br J Anaesth. 2009;103(5):726–30.
24. Griffiths JD, Middle JV, Barron FA, Grant SJ, Popham PA, Royse CF. Transversus abdominis plane block does not provide additional benefit to multimodal analgesia in gynecological cancer surgery. Anesth Analg. 2010;111(3):797–801.
25. Soltani Mohammadi S, Dabir A, Shoeibi G. Efficacy of transversus abdominis plane block for acute postoperative pain relief in kidney recipients: a double-blinded clinical trial: efficacy of TAP block on postrenal transplantation pain. Pain Med. 2014;15(3):460–4.
26. Sandeman DJ, Bennett M, Dilley AV, Perczuk A, Lim S, Kelly KJ. Ultrasound-guided transversus abdominis plane blocks for laparoscopic appendicectomy in children: a prospective randomized trial. Br J Anaesth. 2011;106(6):882–6.
27. Suresh S, Chan VWS. 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.
28. Aveline C, Le Hetet H, Le Roux A, Vautier P, Cognet F, Vinet E, et al. Comparison between ultrasound-guided transversus abdominis plane and conventional ilioinguinal/iliohypogastric nerve blocks for day-case open inguinal hernia repair. Br J Anaesth. 2011;106(3):380–6.
29. Siddiqui MRS, Sajid MS, Uncles DR, Cheek L, Baig MK. A meta-analysis on the clinical effectiveness of transversus abdominis plane block. J Clin Anesth. 2011;23(1):7–14.
30. Petersen PL, Mathiesen O, Torup H, Dahl JB. The transversus abdominis plane block: a valuable option for postoperative analgesia? A topical review. Acta Anaesthesiol Scand. 2010;54(5):529–35.
31. Walter C, Maxwell-Armstrong C, Pinkney T, Conaghan P, Bedforth N, Gornall C, et al. A randomised controlled trial of the efficacy of ultrasound-guided transversus abdominis plane (TAP) block in laparoscopic colorectal surgery. Surg Endosc. 2013;27(7):2366–72.
32. Guirguis MN, Abd-Elsayed AA, Girgis G, Soliman LM. Ultrasound-guided transversus abdominis plane catheter for chronic abdominal pain. Pain Pract Off J World Inst Pain. 2013;13(3):235–8.
33. Schaeffer E, Millot I, Landy C, Nadaud J, Favier J-C, Plancade D. Another use of continuous transversus abdominis plane (TAP) block in trauma patient: pelvic ring fractures: letter to the editor. Pain Med. 2014;15(1):166–7.
34. Mirza F, Carvalho F. Transversus abdominis plane blocks for rescue analgesia following Cesarean delivery: a case series. Can J Anaesth. 2013;60(3):299–303.
35. El-Dawlatly AA, Turkistani A, Kettner SC, Machata A-M, Delvi MB, Thallaj A, et al. Ultrasound-guided transversus abdominis plane block: description of a new technique and comparison with conventional systemic analgesia during laparoscopic cholecystectomy. Br J Anaesth. 2009;102(6):763–7.
36. Niraj G, Searle A, Mathews M, Misra V, Baban M, Kiani S, et al. Analgesic efficacy of ultrasound-guided transversus abdominis plane block in patients undergoing open appendicectomy. Br J Anaesth. 2009;103(4):601–5.
37. Jankovic ZB, Pollard SG, Nachiappan MM. Continuous transversus abdominis plane block for renal transplant recipients. Anesth Analg. 2009;109(5):1710–1.

38. Mukhtar K, Khattak I. Transversus abdominis plane block for renal transplant recipients. Br J Anaesth. 2010;104(5):663–4.
39. Ra YS, Kim CH, Lee GY, Han JI. The analgesic effect of the ultrasound-guided transverse abdominis plane block after laparoscopic cholecystectomy. Korean J Anesthesiol. 2010;58(4):362.
40. Conaghan P, Maxwell-Armstrong C, Bedforth N, Gornall C, Baxendale B, Hong L, et al. Efficacy of transversus abdominis plane blocks in laparoscopic colorectal resections. Surg Endosc. 2010;24(10):2480–4.
41. Milan ZB, Duncan B, Rewari V, Kocarev M, Collin R. Subcostal transversus abdominis plane block for postoperative analgesia in liver transplant recipients. Transplant Proc. 2011;43(7):2687–90.
42. Aniskevich S, Clendenen SR, Torp KD. Bilateral transversus abdominis plane block for managing pain after a pancreas transplant. Exp Clin Transplant Off J Middle East Soc Organ Transplant. 2011;9(4):277–8.
43. Bharti N, Kumar P, Bala I, Gupta V. The efficacy of a novel approach to transversus abdominis plane block for postoperative analgesia after colorectal surgery. Anesth Analg. 2011;112(6):1504–8.
44. Atim A, Bilgin F, Kilickaya O, Purtuloglu T, Alanbay I, Orhan ME, et al. The efficacy of ultrasound-guided transversus abdominis plane block in patients undergoing hysterectomy. Anaesth Intensive Care. 2011;39(4):630–4.
45. Niraj G, Kelkar A, Jeyapalan I, Graff-Baker P, Williams O, Darbar A, et al. Comparison of analgesic efficacy of subcostal transversus abdominis plane blocks with epidural analgesia following upper abdominal surgery: Subcostal transversus abdominis plane blocks following upper abdominal surgery. Anaesthesia. 2011;66(6):465–71.
46. Melnikov A, Bjoergo S, Kongsgaard U. Thoracic paravertebral block versus transversus abdominis plane block in major gynecological surgery: a prospective, randomized, controlled, observer-blinded study. Local Reg Anesth. 2012;5:55–61.
47. Kadam R, Field J. Ultrasound-guided continuous transverse abdominis plane block for abdominal surgery. J Anaesthesiol Clin Pharmacol. 2011;27(3):333.
48. Petersen PL, Stjernholm P, Kristiansen VB, Torup H, Hansen EG, Mitchell AU, et al. The beneficial effect of transversus abdominis plane block after laparoscopic cholecystectomy in day-case surgery: a randomized clinical trial. Anesth Analg. 2012;115(3):527–33.
49. Tan TT, Teoh WHL, Woo DCM, Ocampo CE, Shah MK, Sia ATH. A randomised trial of the analgesic efficacy of ultrasound-guided transversus abdominis plane block after caesarean delivery under general anaesthesia. Eur J Anaesthesiol. 2012;29(2):88–94.
50. Tolchard S, Martindale S, Davies R. Efficacy of the subcostal transversus abdominis plane block in laparoscopic cholecystectomy: comparison with conventional port-site infiltration. J Anaesthesiol Clin Pharmacol. 2012;28(3):339.
51. Albrecht E, Kirkham KR, Endersby RVW, Chan VWS, Jackson T, Okrainec A, et al. Ultrasound-guided transversus abdominis plane (TAP) block for laparoscopic gastric-bypass surgery: a prospective randomized controlled double-blinded trial. Obes Surg. 2013;23(8):1309–14.
52. Sinha A, Jayaraman L, Punhani D. Efficacy of ultrasound-guided transversus abdominis plane block after laparoscopic bariatric surgery: a double blind, randomized controlled study. Obes Surg. 2013;23(4):548–53.
53. Sahin L, Sahin M, Gul R, Saricicek V, Isikay N. Ultrasound-guided transversus abdominis plane block in children: a randomised comparison with wound infiltration. Eur J Anaesthesiol. 2013;30(7):409–14.
54. Wu Y, Liu F, Tang H, Wang Q, Chen L, Wu H. The analgesic efficacy of subcostal transversus abdominis plane block compared with thoracic epidural analgesia and intravenous opioid analgesia after radical gastrectomy. Anesth Analg. 2013;117(2):507–13.
55. Parikh B, Waghmare V, Shah V, Mehta T, Butala B, Parikh G, et al. The analgesic efficacy of ultrasound-guided transversus abdominis plane block for retroperitoneoscopic donor nephrectomy: a randomized controlled study. Saudi J Anaesth. 2013;7(1):43.
56. Gasanova I, Grant E, Way M, Rosero EB, Joshi GP. Ultrasound-guided transversus abdominal plane block with multimodal analgesia for pain management after total abdominal hysterectomy. Arch Gynecol Obstet. 2013;288(1):105–11.
57. Sivapurapu V, Vasudevan A, Gupta S, Badhe A. Comparison of analgesic efficacy of transversus abdominis plane block with direct infiltration of local anesthetic into surgical incision in lower abdominal gynecological surgeries. J Anaesthesiol Clin Pharmacol. 2013;29(1):71.
58. Gómez-Ríos MA, Paech MJ. Postoperative analgesia with transversus abdominis plane catheter infusions of levobupivacaine after major gynecological and obstetrical surgery. A case series. Rev Esp Anestesiol Reanim [Internet]. 2014 Apr [cited 2014 Sep 11]; Available from: http://linkinghub.elsevier.com/retrieve/pii/S0034935614001157.
59. Niraj G, Kelkar A, Hart E, Horst C, Malik D, Yeow C, et al. Comparison of analgesic efficacy of four-quadrant transversus abdominis plane (TAP) block and continuous posterior TAP analgesia with epidural analgesia in patients undergoing laparoscopic colorectal surgery: an open-label, randomised, non-inferiority tri. Anaesthesia. 2014;69(4):348–55.
60. Aniskevich S, Taner B, Perry D, Robards C, Porter S, Thomas C, et al. Ultrasound-guided transversus abdominis plane blocks for patients undergoing laparoscopic hand-assisted nephrectomy: a randomized, placebo-controlled trial. Local Reg Anesth. 2014;7:11–6.
61. McKeen DM, George RB, Boyd JC, Allen VM, Pink A. Transversus abdominis plane block does not improve early or late pain outcomes after Cesarean delivery: a randomized controlled trial. Can J Anesth. 2014;61(7):631–40.
62. Calle GA, López CC, Sánchez E, De Los Ríos JF, Vásquez EM, Serna E, et al. Transversus abdominis plane block after ambulatory total laparoscopic hysterectomy: randomized controlled trial. Acta Obstet Gynecol Scand. 2014;93(4):345–50.
63. Marais A, Porrill O, James M, Dyer R. The use of ultrasound-guided transversus abdominis plane blocks for total abdominal hysterectomy: a double-blind, controlled trial. South Afr J Anaesth Analg. 2014;20(2):117–21.
64. Chandon M, Bonnet A, Burg Y, Barnichon C, DesMesnards-Smaja V, Sitbon B, et al. Ultrasound-guided transversus abdominis plane block versus continuous wound infusion for post-caesarean analgesia: a randomized trial. Gemzell-Danielsson K, editor. PLoS One. 2014;9(8):e103971.
65. Bhattacharjee S, Ray M, Ghose T, Maitra S, Layek A. Analgesic efficacy of transversus abdominis plane block in providing effective perioperative analgesia in patients undergoing total abdominal hysterectomy: a randomized controlled trial. J Anaesthesiol Clin Pharmacol. 2014;30(3):391.
66. Heil JW, Nakanote KA, Madison SJ, Loland VJ, Mariano ER, Sandhu NS, et al. Continuous Transversus Abdominis Plane (TAP) blocks for postoperative pain control after Hernia Surgery: a randomized, triple-masked, Placebo-Controlled Study: continuous TAP blocks. Pain Med. 2014;15(11):1957–64.
67. De Oliveira GS Jr. Transversus abdominis plane infiltration for laparoscopic gastric banding: a pilot study. World J Gastrointest Surg. 2014;6(2):27.
68. 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*: transversus abdominis plane block. Anaesthesia. 2011;66(11):1023–30.
69. 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.

70. Børglum J, Jensen K, Christensen AF, Hoegberg LCG, Johansen SS, Lönnqvist P-A, et al. Distribution patterns, dermatomal anesthesia, and ropivacaine serum concentrations after bilateral dual transversus abdominis plane block. Reg Anesth Pain Med. 2012;37(3):294–301.
71. Murthy A, Garcia C, Shulman M. Transversus Abdominis Plane Block Associated Flank Hematoma. Reg Anesth Pain Med. 2012. Available from: http://www.asra.com/display_fall_2012.php?id=197
72. Weiss E, Jolly C, Dumoulin J-L, Meftah RB, Blanié P, Laloë P-A, et al. Convulsions in 2 patients after bilateral ultrasound-guided transversus abdominis plane blocks for cesarean analgesia. Reg Anesth Pain Med. 2014;39(3):248–51.
73. Dolan J, Lucie P, Geary T, Smith M, Kenny GNC. 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.
74. Shido A, Imamachi N, Doi K, Sakura S, Saito Y. Continuous local anesthetic infusion through ultrasound-guided rectus sheath catheters. Can J Anaesth. 2010;57(11):1046–7.
75. Thomassen I, van Suijlekom JA, van de Gaag A, Ponten JEH, Nienhuijs SW. Ultrasound-guided ilioinguinal/iliohypogastric nerve blocks for chronic pain after inguinal hernia repair. Hernia. 2013;17(3):329–32.
76. Van Schoor AN, Boon JM, Bosenberg AT, Abrahams PH, Meiring JH. Anatomical considerations of the pediatric ilioinguinal/iliohypogastric nerve block. Paediatr Anaesth. 2005;15(5):371–7.
77. Sviggum HP, Niesen AD, Sites BD, Dilger JA. Trunk Blocks 101: Transversus Abdominis Plane, Ilioinguinal-Iliohypogastric, and Rectus Sheath Blocks. Int Anesthesiol Clin. 2012;50(1):74–92.
78. Schmutz M, Schumacher PM, Luyet C, Curatolo M, Eichenberger U. Ilioinguinal and iliohypogastric nerves cannot be selectively blocked by using ultrasound guidance: a volunteer study. Br J Anaesth. 2013;111(2):264–70.
79. Amory C, Mariscal A, Guyot E, Chauvet P, Leon A, Poli-Merol ML. Is ilioinguinal/iliohypogastric nerve block always totally safe in children? Paediatr Anaesth. 2003;13(2):164–6.
80. Blanco R, McDonnell JG. Optimal point of injection: The quadratus lumborum type I and II blocks. [Internet]. 2014. Available from: http://www.respond2articles.com/ANA/forums/post/1550.aspx.
81. Binzer J, Jensen K, Jørgensen L, Lenz K, Vazin M, Tanggaard, et al. Ultrasound-guided (USG) transmuscular quadratus lumborum (TQL) block for elective laparoscopic cholecystectomy. A prospective, randomised double-blind study. Reg Anesth Pain Med. 2013;38(5 Suppl. 1):E138.
82. Kadam V. Ultrasound-guided quadratus lumborum block as a postoperative analgesic technique for laparotomy. J Anaesthesiol Clin Pharmacol. 2013;29(4):550.
83. Visoiu M, Yakovleva N. Continuous postoperative analgesia via quadratus lumborum block – an alternative to transversus abdominis plane block. Paediatr Anaesth. 2013;23(10):959–61.
84. Boerglum J, Moriggl B, Jensen K, Lönnqvist P. Ultrasound-guided transmuscular quadratus lumborum blockade [Internet]. 2013. Available from: http://bja.oxfordjournals.org/forum/topic/brjana_el%3b9919.
85. Sauter A, Ullensvang K, Bendtsen T, Boerglum J. The "Shamrock Method" a new and promising technique for ultrasound guided lumbar plexus block [Internet]. Br J Anaesth. 2014. [cited 2014 Jun 14]. Available from: http://bja.oxfordjournals.org/forum/topic/brjana_el%3B9814.
86. Hebbard PD. Transversalis fascia plane block, a novel ultrasound-guided abdominal wall nerve block. Can J Anaesth. 2009;56(8):618–20.
87. Chin KJ, Chan V, Hebbard P, Tan JS, Harris M, Factor D. Ultrasound-guided transversalis fascia plane block provides analgesia for anterior iliac crest bone graft harvesting. Can J Anesth. 2012;59(1):122–3.

Chapter 54

Ilioinguinal, Iliohypogastric, and Genitofemoral Nerve Blocks

Dario Bugada, MD
Department of Anesthesia and Intensive Care – Pain Therapy Service, IRCCS Foundation Policlinico S. Matteo, University of Pavia, Pavia (PV) 27100, Italy
e-mail: dariobugada@gmail.com

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: Philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_54, © Springer International Publishing Switzerland 2015

The ilioinguinal (II), iliohypogastric (IH), and genitofemoral (GF) nerves are the primary nerves providing sensory innervation to the skin bordering between the thigh and abdomen. Therefore, they have been collectively called "border nerves" [1, 2].

Anatomy

The II and IH nerves originate from the anterior rami of T12 and L1 nerve roots, emerging near the lateral border of the psoas major muscle. These two nerves extend diagonally toward the crest of the ilium (Fig. 54.1).

The IH nerve pierces the transversus abdominis muscle above the iliac crest, midway between the iliac crest and the twelfth rib. The II nerve runs caudally and parallel to the IH nerve. Here, both nerves can be found consistently (90 %) between the transversus abdominis and internal oblique muscles [2, 3]. Terminal branches of the IH nerve perforate the external oblique muscle aponeurosis approximately 4 cm lateral to the midline to supply the skin over the lower portion of the rectus abdominis [2, 3]. The IH nerve also provides sensory innervation to the skin above the tensor fasciae latae through a lateral cutaneous branch. Terminal branches of the II nerve enter the inguinal canal through the deep inguinal ring; it may lie upon the cremaster muscle and fascial layer of the spermatic cord in men or round ligament in women [4, 5]. Here, the II nerve is often accompanied by the genital branch of the GF nerve, and wide variations in the course of these nerves within the inguinal canal have been documented [6–9]. The terminal sensory branches may innervate the skin of the mons pubis, inner thigh, inguinal crease, and anterior surface of the scrotum or anterior one third of the labia (Fig. 54.2).

The GF nerve originates from the first and second lumbar nerve roots (Fig. 54.1). It emerges on the anterior surface of the psoas muscle either as a single trunk or separate genital and femoral branches [6]. The femoral branch passes laterally over the external iliac artery and then penetrates the fascia lata to enter the femoral sheath. Terminal branches provide cutaneous innervation to the femoral triangle [7]. The genital branch will also cross in front of the external iliac artery but then passes through the ventral aspect of the internal ring of the inguinal canal (Fig. 54.1). Anatomical studies describe this branch as running between the cremasteric and internal spermatic fascia, incorporating with the cremasteric fascia, or lying outside of the spermatic cord [5, 7, 8]. Terminal sensory branches may innervate the scrotum and possibly the upper, inner, and medial thigh [5].

However, the main concept to remember when dealing with the "border nerves" is the high rate of variability they are associated with. Anatomical studies highlight this variability, which has been reported with respect to their origin and spinal contribution [10], communication between nerves, penetration of fascial layers, branching patterns, and dominance patterns [2]; in some cases, one of the nerves may be entirely absent [9]. Although the general anatomy of the IH and II nerves has been well documented in standard anatomy textbooks, recent studies related to nerve injury during surgery or based on cadaver dissections were able to identify multiple and different spinal nerves contributing to the formation of these nerves. The II and IH nerves mainly originate from the T12 and L1 nerve roots, but a contribution from T11 to L3 exists in a minority of patients [10].

The most consistent anatomical location for the II and IH nerves to perforate the abdominal muscular layers is lateral and superior to the anterior superior iliac spine, where they run between the transversus abdominis and internal oblique muscular layers, even though the distance from the ASIS to the point the nerves enter the fascial layer can widely vary [11, 12].

As well, communication between the GF, IH, and II nerves (as well as the lateral femoral cutaneous nerve) is common and results in sensory overlap. Finally, the sites at which the II and IH nerves pierce the abdominal wall muscle layers are significantly variable [13].

Special considerations are needed for pediatric patients. Anatomically, pediatric patients have varying II and IH nerves compared with adults; anatomical results from adults cannot be downscaled to infants and children, in whom the iliohypogastric and ilioinguinal nerves lie closer to the ASIS than originally thought [14], and differences have been noted according to different age groups [15].

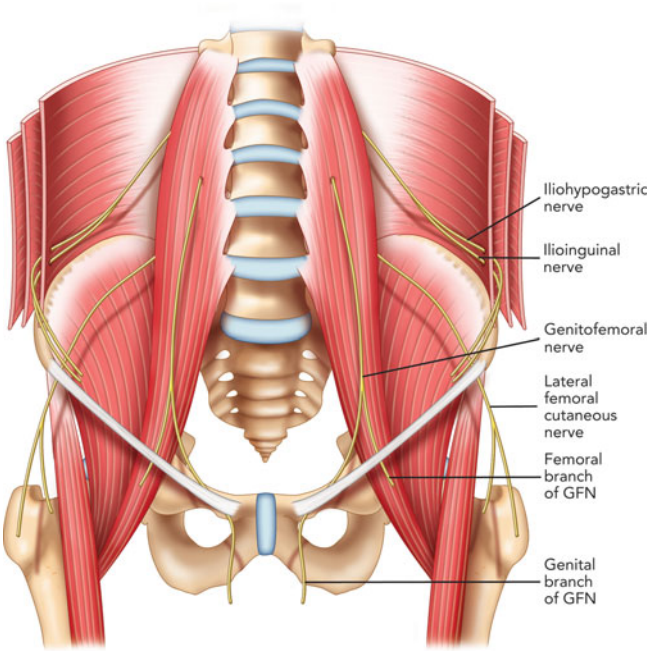


Fig. 54.1 Schematic diagram showing the pathway of the ilioinguinal, iliohypogastric, and genitofemoral nerve. *GFN* genitofemoral nerve (Reproduced with permission from Philip Peng Educational Series)

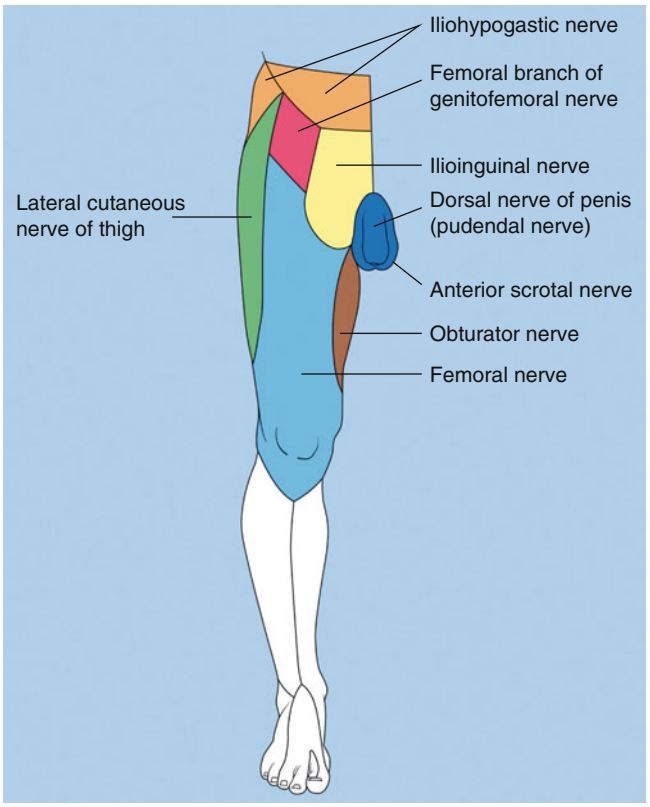


Fig. 54.2 Cutaneous innervation of the ilioinguinal region (Reproduced with permission from Dr. Danilo Jankovic)

Existing Technique for Neural Blockade

As discussed above, there is a high degree of anatomic variability in not only the course of the nerves but also their branching patterns, areas of penetration of the fascial layers, and dominance patterns [2]. By far, the most consistent location of the II and IH nerves is lateral and superior to the ASIS, where the nerves are found between the transversus abdominis and internal oblique muscular layers in nearly 90 % of the cases [16, 17]. While most of the anatomic variations occur medial to the ASIS, virtually all landmark-guided injection techniques described in the literature are performed in the anterior abdominal wall [2, 3, 18].

Because the existing landmark-based techniques rely on blind infiltration of local anesthetic through different layers, the risk of complications are well understandable: these include inadvertent femoral nerve block [14, 19], colonic puncture [20, 21], and vascular injury [22]. Nevertheless, block failure is still an important matter of concern, and failure rate of the blind technique lies in the range of 10–40 % [14, 23]. This is attributed to the potential for injecting local anesthetics into the wrong abdominal plane [23], and in some situation, it leads to injection inside the peritoneum [24].

As well, the conventional description of the blockade of the genital branch of the GF nerve is mainly a "blind" technique [25, 26] and relies on the pubic tubercle, inguinal ligament, inguinal crease, and femoral artery as landmarks. One involves infiltration of local anesthetic immediately lateral to the pubic tubercle caudal to the inguinal ligament [25]. In another method, a needle is inserted into the inguinal canal to block the genital branch [26]. The blind techniques described are essentially infiltration techniques and rely on high volumes of local anesthetic for consistent results. Although the basis of this landmark is not clear, the needle is likely directed toward the spermatic cord, and important structures of the spermatic cord (testicular artery and vas deferens) or the peritoneum are at risk.

Ultrasound-Guided Injection Technique

Advantages of ultrasound guidance include the identification of muscular and fascial layers (including structures lying deeper to the peritoneum), the visualization of needle's trajectory and spread of injectate, and the detection of vascular structures. Ultrasound helps physicians to overcome the wide anatomic variability associated with the structures in this area, helping to identify the nerves themselves, or the plane they lie within. Ultrasound guidance has demonstrated to improve accuracy in the injection of local anesthetics, with less risk of inadvertent injections in the wrong fascial plane [24], reduced amount of local anesthetics used for anesthesia, and lower consumption of systemic pain medications compared to landmark techniques [23, 27].

The IH and II nerves are superficial nerves, best viewed with a linear probe of high frequency (6–13 MHz). In very obese patient, a linear medium-frequency probe may be useful. Most of clinical studies or reports used ASIS as landmark, and the position of the probe was above ASIS and moved along the line joining the ASIS and umbilicus. This approach carries some limitation: when the probe is placed too near to ASIS, the external oblique muscle layer may be missed; when the probe is too far away from the ASIS (the "bone shadow of iliac crest" not in the scan), the nerve seen in between the transversus abdominis and internal oblique muscular layers is likely the 12th intercostal nerve or subcostal nerve instead.

The recommended area for ultrasound scanning of II and IH nerves is lateral and superior to the anterior superior iliac spine (Fig. 54.3), because the two nerves are detectable in this area in 90 % of the cases. The probe is placed cranial and three fingerbreadths lateral to the ASIS with the transducer perpendicular to the inguinal ligament and its lateral end in contact with the iliac crest (Fig. 54.3). At this position, the iliac crest will appear as a hyperechoic structure adjacent to which will appear the three muscular layers of the abdominal wall (Figs. 54.4 and 54.5). Below the transversus abdominis, peristaltic movements of the bowel may be detected. Once the muscular layers are identified, the II and IH nerves will be found in the split fascial plane between the internal oblique and transversus abdominis muscle layers. Both nerves should be within 1.5 cm of the iliac crest at this site, with the II nerve closer to the iliac crest [28]. The nerves are usually in close proximity to each other [9] and located on the "upsloping" split fascia close to the iliac crest. In some cases, the nerves may run approximately 1 cm apart. The deep circumflex iliac artery which is close to the two nerves in the same fascial layer can be revealed with the use of color Doppler (Fig. 54.6). A neural structure within the fascial split may also be seen medial and on the flat part of the internal oblique and transversus abdominis muscle junction: this is the subcostal nerve, and if mistaken for the II and IH nerves, the nerve blockade will result in aberrant distribution of anesthesia.

In some patients (those with previous surgery, multiple childbirth) or when the procedure is performed by inexperienced practitioners, either the II and IH or the fascia split is difficult to visualize; in these cases, the target will be the fascial plane between the internal oblique and transverse abdominal muscles [29].

Both out-of-plane and in-plane techniques can be used and have been described [28, 30]. As the two nerves run very closely within the fascial plane (sometimes as a common trunk) and frequently overlap in sensory innervation, a selective block of one of the two nerves has recently been demonstrated to be very difficult to realize, even in experienced hands and injecting minimal volumes of local anesthetics [31].

Ultrasound-guided blockade of the genital branch of the GF nerve has been described in several review articles [2, 3, 18, 32]. The genital branch is difficult to visualize directly unless a high-frequency probe (up to 18 MHz) is used. Furthermore, the GF nerve can be found within or outside the spermatic cord. Thus, the most reliable method is to infiltrate the inguinal canal and deposit the injectate both inside and outside the spermatic cord [2, 3]. The patient is placed in a supine position, and surface anatomy of the ASIS, inguinal ligament, and femoral artery is established. A linear ultrasound transducer of high frequency (6–13 MHz) is placed over the femoral artery along its long axis (Fig. 54.2). The transducer is moved cephalad over the artery, which starts to descend at a steep angle toward the inguinal ligament. At this point, the femoral artery transitions to become the external iliac artery and runs in a retroperitoneal plane (Fig. 54.7). Once the external iliac artery is identified, the inguinal canal can be viewed superficial to the vessel as an oval, soft tissue structure (Fig. 54.7). This will contain the spermatic cord in men and the round ligament in women. The canal is then scanned medially, so that the final ultrasound probe position is approximately one fingerbreadth away from the pubic tubercle. In males, testicular arteries may be identified in the spermatic cord as pulsatile structures and confirmed with the use of color Doppler (Fig. 54.8). Vessels may be accentuated by asking the patient to cough or perform a Valsalva maneuver, which increases blood flow through the pampiniformis plexus. The vas deferens may also be identified as a thick, tubular structure.

An in-plane or out-of-plane technique may be used. With an out-of-plane technique, the needle approaches the inguinal canal from the lateral aspect of the probe. Hydrodissection with saline should be utilized to confirm adequate spread within the inguinal canal or within the cord. The absence of any observed spread is indicative of intravascular injection or loss of needle tip visualization. Given the anatomic variability of the genital branch of the GF nerve, local anesthetic is injected within and outside of the spermatic cord in men; in women, local anesthetic is injected around the round ligament only. In males, the local anesthetic should not contain epinephrine so as to avoid vasoconstriction of the testicular arteries. Steroid can be added for the management of chronic pain syndromes.

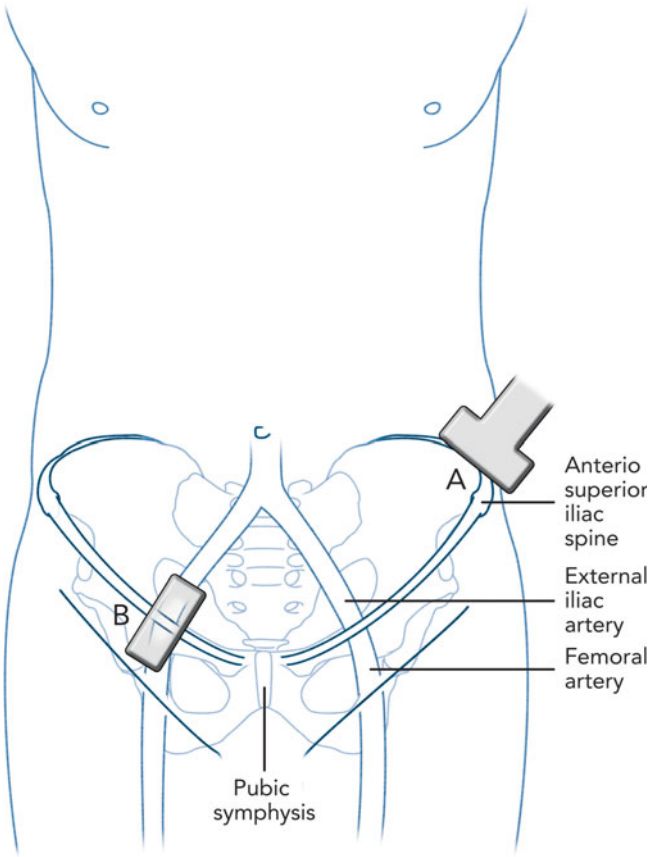


Fig. 54.3 Similar view as in Fig. 54.3. In addition to the ilioinguinal (*solid arrowhead*) and iliohypogastric (*) nerves, a third nerve appears and is usually mistaken as the ilioinguinal nerve but is much further from the iliac crest. It is the subcostal nerve (12th intercostal nerve) indicated by *line arrow*. *EO* external oblique muscle, *IO* internal oblique muscle, *TA* transversus abdominis muscle, *PE* peritoneum, (Reproduced with permission from Philip Peng Educational Series)

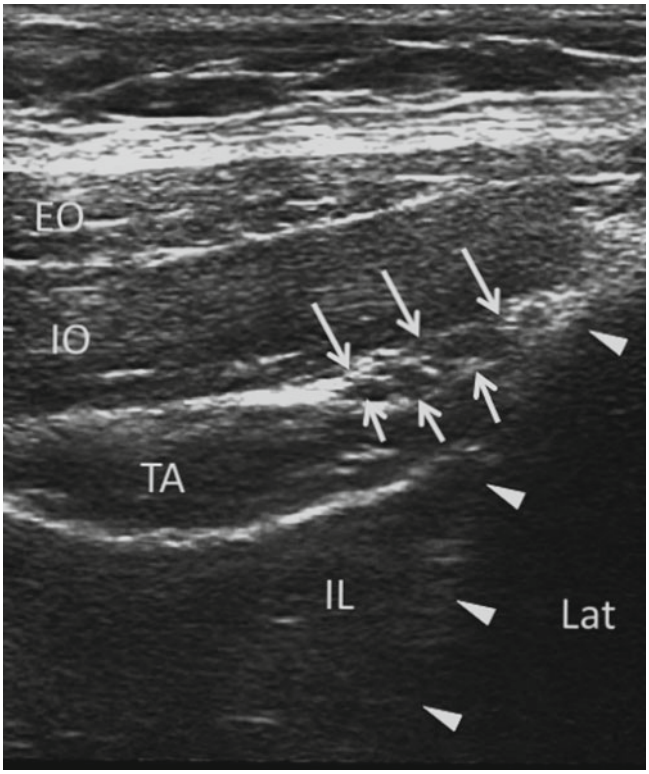


Fig. 54.4 Figure showing the three layers of the muscles and the fascial split (*white line arrows*) with the ilioinguinal and iliohypogastric nerves inside. *Solid triangles* outline the iliac crest. *EO* external oblique muscle, *IO* internal oblique muscle, *TA* transversus abdominis muscle, *IL* iliacus (Reproduced with permission from Philip Peng Educational Series), *Lat* lateral side

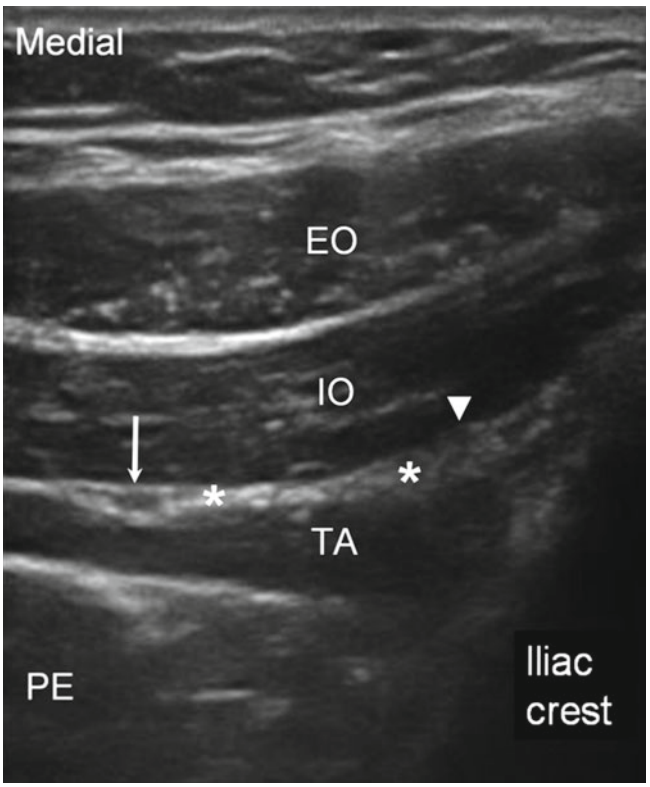


Fig. 54.5 Similar view as in Fig. 54.3. In addition to the ilioinguinal (*solid arrowhead*) iliohypogastric (*) nerves, a third nerve appears and is usually mistaken as the ilioinguinal nerve but is much further from the iliac crest. It is the subcostal nerve (12th intercostal nerve) indicated by *line arrow*. *EO* external oblique muscle, *IO* internal oblique muscle, *TA* transversus abdominis muscle, *PE* peritoneum (Reproduced with permission from Philip Peng Educational Series)

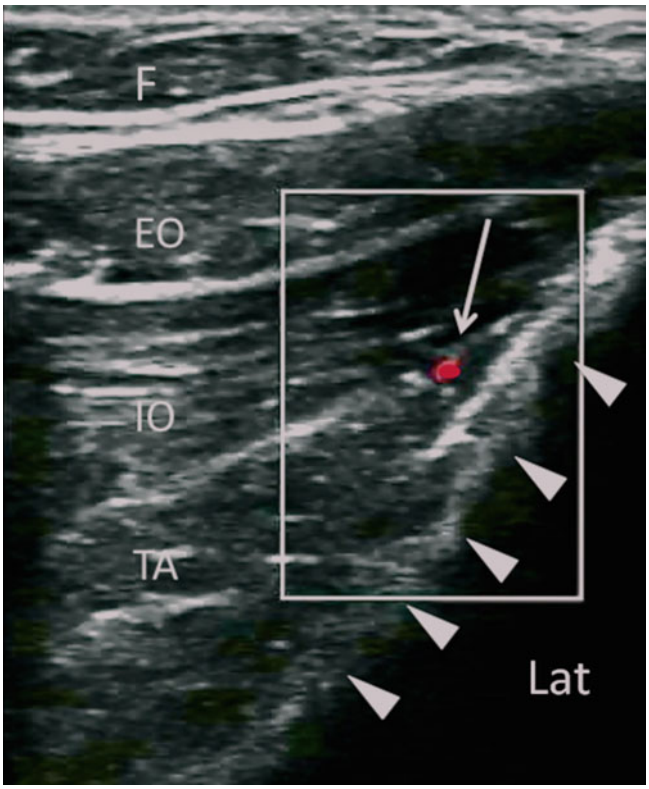


Fig. 54.6 Similar view as in Fig. 54.3 with color Doppler. A branch of the deep circumflex iliac artery is shown in *red color*. *Solid arrows* outline the iliac crest. *F* subcutaneous fat, *EO* external oblique muscle, *IO* internal oblique muscle, *TA* transversus abdominis muscle, *Lat* lateral (Reproduced with permission from Philip Peng Educational Series)

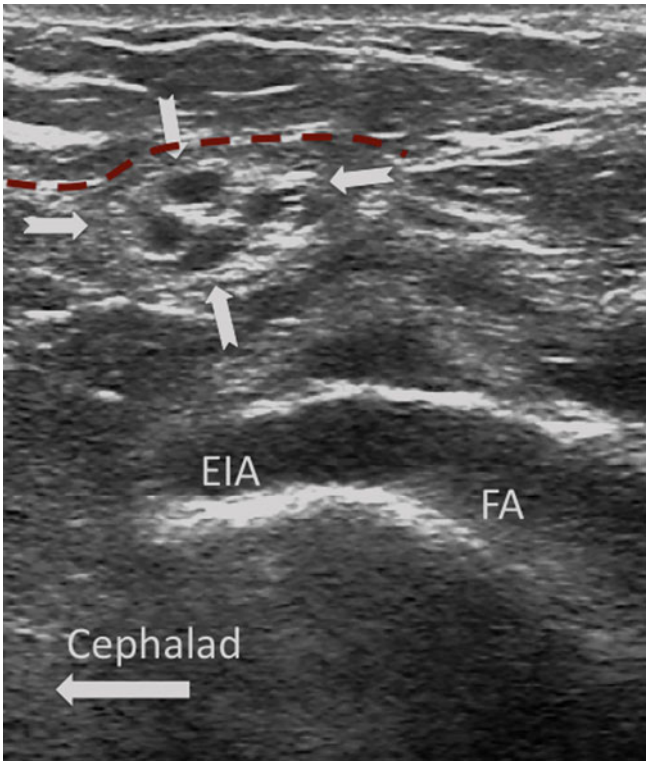


Fig. 54.7 Long axis view of the femoral and external iliac artery showing the cross section of the spermatic cord (outlined by *solid arrows*) in a male patient. The *red dashed line* outlines the deep abdominal fascia. *EIA* external iliac artery, *FA* femoral artery (Reproduced with permission from Philip Peng Educational Series)

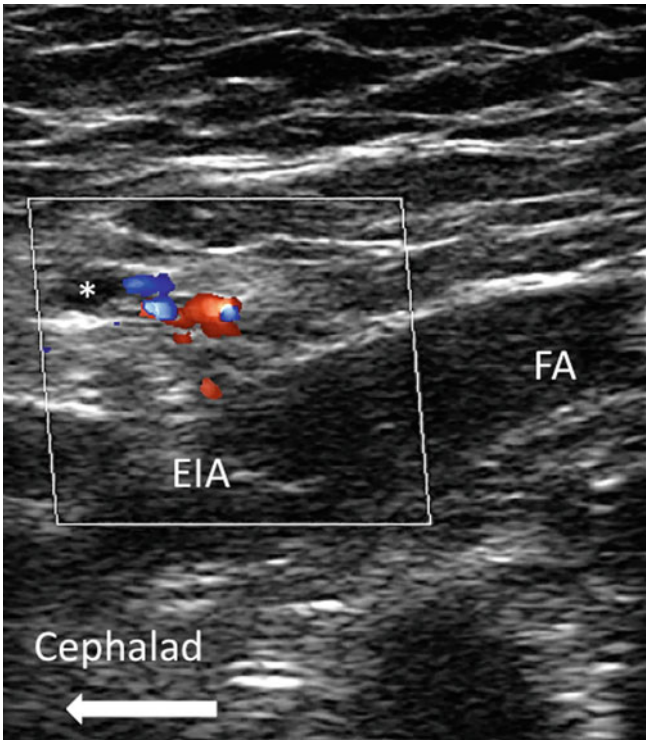


Fig. 54.8 Similar view as in Fig. 54.6 with color Doppler showing the vessels inside the spermatic cord (Reproduced with permission from Philip Peng Educational Series)

Clinical Application

Blocks of the border nerves have been used both in the surgical and nonsurgical settings.

II and IH local anesthetic nerve blocks provide analgesia for operations involving the region between the thigh and abdomen. Ultrasound-guided block of II and IH nerves can be used as part of a multimodal analgesic regimen after Cesarean section or surgeries including a lower abdominal transverse incision (in all cases in which the surgical incision crosses the midline, a bilateral block is needed) [33, 34]. Furthermore, its clinical usefulness has been proven also for inguinal hernia repair in both adult [35] and pediatric patients [27, 36–38]. Only a little clinical benefit has been described with the addition of a GF nerve block to II and IH nerve block for hernia repair [39].

These nerves are also prone to injury during open appendectomy incisions, inguinal herniorraphies, low transverse incisions (e.g., Pfannenstiel incision), and trocar insertion for laparoscopic surgery of the abdomen and pelvis [13, 40–43]. When II or IH nerves are involved, the pain is in the lower abdominal wall and sometimes radiates to the genital area. The clinician has to differentiate this from the visceral pain, which is deep sited and poorly localized. A sensory change with objective signs is sometimes present. Other physical signs include a discrete tender spot, abnormal pain sensitivity (allodynia and hyperalgesia), and Tinel's sign. When the GF nerve is involved, the patient usually presents with groin pain that may extend to the scrotum or the testicle in men, the labia majora in women, and the medial aspect of the thigh. Cremasteric reflex may be absent. Most of the patient has a previous history of surgery, and only a small number of them have no previous history of trauma.

Accurate neural block of the "border nerves" can be used for pain relief in these patients or assist in understanding the etiology of chronic inguinal pain, even if the specific contribution of each of the nerves is often difficult to determine because of the abovementioned anatomic variability and the frequent overlapping in sensory innervation [31, 44].

II and IH nerve block is useful for the treatment of groin pain, like chronic pain syndromes after hernia repair [45]. Ultrasound-guided block of the genital branch of the GF nerve has been described for chronic pain relief in the groin area after surgery [46], and ultrasound-guided pulsed radiofrequency has been used for effective treatment of severe orchialgia [47]. However, one should be cautious about the application of radiofrequency technique as the genital branch of the GF nerve is not usually visualized. Ultrasound-guided blockade of the femoral branch of the GF nerve has also been described, and cryoanalgesia of the femoral branch of the GF nerve was performed [48].

References

1. Peng PW Peripheral applications of ultrasound for chronic pain. In: Benzon H, Huntoon M, Narouze S, editors. Spinal injections and peripheral nerve blocks: interventional and neuromodulatory techniques for pain management (Deer T: Series Editor), vol 2, 1st ed. Philadelphia: Elsevier. p 101–20.
2. Peng PW, Narouze S. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures: part I: nonaxial structures. Reg Anesth Pain Med. 2009;34(5):458–74. doi:10.1097/AAP.0b013e3181aea16f.
3. Soneji N, Peng PW. Ultrasound-guided pain interventions – a review of techniques for peripheral nerves. Korean J Pain. 2013;26(2):111–24. doi:10.3344/kjp.2013.26.2.111.
4. Oelrich TM, Moosman DA. The aberrant course of the cutaneous component of the ilioinguinal nerve. Anat Rec. 1977;189(2):233–6. doi:10.1002/ar.1091890209.
5. Ducic I, Dellon AL. Testicular pain after inguinal hernia repair: an approach to resection of the genital branch of genitofemoral nerve. J Am Coll Surg. 2004;198(2):181–4. doi:10.1016/j.jamcollsurg.2003.09.025.
6. Rab M, Ebmer J, Dellon AL. Anatomic variability of the ilioinguinal and genitofemoral nerve: implications for the treatment of groin pain. Plast Reconstr Surg. 2001;108(6):1618–23.
7. Liu WC, Chen TH, Shyu JF, Chen CH, Shih C, Wang JJ, Kung SP, Lui WY, Wu CW, Liu JC. Applied anatomy of the genital branch of the genitofemoral nerve in open inguinal herniorrhaphy. Eur J Surg Acta Chir. 2002;168(3):145–9. doi:10.1080/110241502320127748.
8. Lichtenstein IL, Shulman AG, Amid PK, Montllor MM. Cause and prevention of postherniorrhaphy neuralgia: a proposed protocol for treatment. Am J Surg. 1988;155(6):786–90.
9. al-dabbagh AK. Anatomical variations of the inguinal nerves and risks of injury in 110 hernia repairs. Surg Radiol Anat. 2002;24(2):102–7.
10. Klaassen Z, Marshall E, Tubbs RS, Louis Jr RG, Wartmann CT, Loukas M. Anatomy of the ilioinguinal and iliohypogastric nerves with observations of their spinal nerve contributions. Clin Anat. 2011;24(4):454–61. doi:10.1002/ca.21098.
11. Ndiaye A, Diop M, Ndoye JM, Ndiaye A, Mane L, Nazarian S, Dia A. Emergence and distribution of the ilioinguinal nerve in the inguinal region: applications to the ilioinguinal anaesthetic block (about 100 dissections). Surg Radiol Anat. 2010;32(1):55–62. doi:10.1007/s00276-009-0549-0.
12. Avsar FM, Sahin M, Arikan BU, Avsar AF, Demirci S, Elhan A. The possibility of nervus ilioinguinalis and nervus iliohypogastricus injury in lower abdominal incisions and effects on hernia formation. J Surg Res. 2002;107(2):179–85.
13. Whiteside JL, Barber MD, Walters MD, Falcone T. Anatomy of ilioinguinal and iliohypogastric nerves in relation to trocar placement and low transverse incisions. Am J Obstet Gynecol. 2003;189(6):1574–8. discussion 1578.
14. van Schoor AN, Boon JM, Bosenberg AT, Abrahams PH, Meiring JH. Anatomical considerations of the pediatric ilioinguinal/iliohypogastric nerve block. Paediatr Anaesth. 2005;15(5):371–7.
15. Hong JY, Kim WO, Koo BN, Kim YA, Jo YY, Kil HK. The relative position of ilioinguinal and iliohypogastric nerves in different age groups of pediatric patients. Acta Anaesthesiol Scand. 2010;54(5):566–70. doi:10.1111/j.1399-6576.2010.02226.x.
16. Jamieson RW, Swigart LL, Anson BJ. Points of parietal perforation of the ilioinguinal and iliohypogastric nerves in relation to optimal sites for local anaesthesia. Q Bull Northwest Univ Med Sch. 1952;26(1):22–6.
17. Mandelkow H, Loeweneck H. The iliohypogastric and ilioinguinal nerves. Distribution in the abdominal wall, danger areas in surgical

incisions in the inguinal and pubic regions and reflected visceral pain in their dermatomes. Surg Radiol Anat. 1988;10(2):145–9.
18. 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.
19. Rosario DJ, Jacob S, Luntley J, Skinner PP, Raftery AT. Mechanism of femoral nerve palsy complicating percutaneous ilioinguinal field block. Br J Anaesth. 1997;78(3):314–6.
20. Johr M, Sossai R. Colonic puncture during ilioinguinal nerve block in a child. Anesth Analg. 1999;88(5):1051–2.
21. Amory C, Mariscal A, Guyot E, Chauvet P, Leon A, Poli-Merol ML. Is ilioinguinal/iliohypogastric nerve block always totally safe in children? Paediatr Anaesth. 2003;13(2):164–6.
22. Vaisman J. Pelvic hematoma after an ilioinguinal nerve block for orchialgia. Anesth Analg. 2001;92(4):1048–9.
23. Weintraud M, Marhofer P, Bosenberg A, Kapral S, Willschke H, Felfernig M, Kettner S. Ilioinguinal/iliohypogastric blocks in children: where do we administer the local anesthetic without direct visualization? Anesth Analg. 2008;106(1):89–93, table of contents. doi:10.1213/01.ane.0000287679.48530.5f.
24. McDermott G, Korba E, Mata U, Jaigirdar M, Narayanan N, Boylan J, Conlon N. Should we stop doing blind transversus abdominis plane blocks? Br J Anaesth. 2012;108(3):499–502. doi:10.1093/bja/aer422.
25. Conn D, Nicholls B. Regional anesthesia. In: Wilson IH, Allman KG, editors. Oxford Handbook of Anaesthesia. 2nd ed. New York: Oxford University Press; 2006. p. 1055–104.
26. Broadman LM. Complications of pediatric regional anesthesia. Reg Anesth. 1996;21(6 Suppl):64–70.
27. Willschke H, Bosenberg A, Marhofer P, Johnston S, Kettner S, Eichenberger U, Wanzel O, Kapral S. Ultrasonographic-guided ilioinguinal/iliohypogastric nerve block in pediatric anesthesia: what is the optimal volume? Anesth Analg. 2006;102(6):1680–4. doi:10.1213/01.ane.0000217196.34354.5a.
28. Eichenberger U, Greher M, Kirchmair L, Curatolo M, Moriggl B. Ultrasound-guided blocks of the ilioinguinal and iliohypogastric nerve: accuracy of a selective new technique confirmed by anatomical dissection. Br J Anaesth. 2006;97(2):238–43. doi:10.1093/bja/ael103.
29. Ford S, Dosani M, Robinson AJ, Campbell GC, Ansermino JM, Lim J, Lauder GR. Defining the reliability of sonoanatomy identification by novices in ultrasound-guided pediatric ilioinguinal and iliohypogastric nerve blockade. Anesth Analg. 2009;109(6):1793–8. doi:10.1213/ANE.0b013e3181bce5a5.
30. Peng PW. Ilioinguinal, iliohypogastric and genitofemoral nerve. In: Peng PW, editor. Ultrasound for pain medicine intervention: a practical guide, vol 2. Pelvic pain. Philip Peng educational series. 1st ed. iBook, California, CA: Apple Inc.; 2013. p. 16–23
31. Schmutz M, Schumacher PM, Luyet C, Curatolo M, Eichenberger U. Ilioinguinal and iliohypogastric nerves cannot be selectively blocked by using ultrasound guidance: a volunteer study. Br J Anaesth. 2013;111(2):264–70. doi:10.1093/bja/aet028.
32. Bellingham G, Peng P. Ultrasound-guided interventional procedures for chronic pelvic pain. Tech Reg Anesth Pain Manag. 2009;13:171–8.
33. Vallejo MC, Steen TL, Cobb BT, Phelps AL, Pomerantz JM, Orebaugh SL, Chelly JE. Efficacy of the bilateral ilioinguinal-iliohypogastric block with intrathecal morphine for postoperative cesarean delivery analgesia. Sci World J. 2012;2012:107316. doi:10.1100/2012/107316.
34. 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):1220–22, table of contents. doi:10.1213/ane.0b013e3181683821.
35. Baerentzen F, Maschmann C, Jensen K, Belhage B, Hensler M, Borglum J. Ultrasound-guided nerve block for inguinal hernia repair: a randomized, controlled, double-blind study. Reg Anesth PainMed.2012;37(5):502–7.doi:10.1097/AAP.0b013e31825a3c8a.
36. Willschke H, Marhofer P, Bosenberg A, Johnston S, Wanzel O, Cox SG, Sitzwohl C, Kapral S. Ultrasonography for ilioinguinal/iliohypogastric nerve blocks in children. Br J Anaesth. 2005;95(2):226–30. doi:10.1093/bja/aei157.
37. Weintraud M, Lundblad M, Kettner SC, Willschke H, Kapral S, Lonnqvist PA, Koppatz K, Turnheim K, Bsenberg A, Marhofer P. Ultrasound versus landmark-based technique for ilioinguinal-iliohypogastric nerve blockade in children: the implications on plasma levels of ropivacaine. Anesth Analg. 2009;108(5):1488–92. doi:10.1213/ane.0b013e31819cb1f3.
38. 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. doi:10.1016/j.jclinane.2008.10.012.
39. Sasaoka N, Kawaguchi M, Yoshitani K, Kato H, Suzuki A, Furuya H. Evaluation of genitofemoral nerve block, in addition to ilioinguinal and iliohypogastric nerve block, during inguinal hernia repair in children. Br J Anaesth. 2005;94(2):243–6. doi:10.1093/bja/aei031.
40. Sippo WC, Burghardt A, Gomez AC. Nerve entrapment after Pfannenstiel incision. Am J Obstet Gynecol. 1987;157(2):420–1.
41. Luijendijk RW, Jeekel J, Storm RK, Schutte PJ, Hop WC, Drogendijk AC, Huikeshoven FJ. The low transverse Pfannenstiel incision and the prevalence of incisional hernia and nerve entrapment. Ann Surg. 1997;225(4):365–9.
42. Choi PD, Nath R, Mackinnon SE. Iatrogenic injury to the ilioinguinal and iliohypogastric nerves in the groin: a case report, diagnosis, and management. Ann Plast Surg. 1996;37(1):60–5.
43. Cardosi RJ, Cox CS, Hoffman MS. Postoperative neuropathies after major pelvic surgery. Obstet Gynecol. 2002;100(2):240–4.
44. Bischoff JM, Koscielniak-Nielsen ZJ, Kehlet H, Werner MU. Ultrasound-guided ilioinguinal/iliohypogastric nerve blocks for persistent inguinal postherniorrhaphy pain: a randomized, double-blind, placebo-controlled, crossover trial. Anesth Analg. 2012;114(6):1323–9. doi:10.1213/ANE.0b013e31824d6168.
45. Thomassen I, van Suijlekom JA, van de Gaag A, Ponten JE, Nienhuijs SW. Ultrasound-guided ilioinguinal/iliohypogastric nerve blocks for chronic pain after inguinal hernia repair. Hernia J Hernias Abdom Wall Surg. 2013;17(3):329–32. doi:10.1007/s10029-012-0998-y.
46. Shanthanna H. Successful treatment of genitofemoral neuralgia using ultrasound guided injection: a case report and short review of literature. Case Rep Anesthesiol. 2014;2014:371703. doi:10.1155/2014/371703.
47. Terkawi AS, Romdhane K. Ultrasound-guided pulsed radiofrequency ablation of the genital branch of the genitofemoral nerve for treatment of intractable orchalgia. Saudi J Anaesth. 2014;8(2):294–8. doi:10.4103/1658-354X.130755.
48. Campos NA, Chiles JH, Plunkett AR. Ultrasound-guided cryoablation of genitofemoral nerve for chronic inguinal pain. Pain Physician. 2009;12(6):997–1000.

Chapter 55

Injection for Piriformis Syndrome

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_55, © Springer International Publishing Switzerland 2015

Introduction

Piriformis syndrome (PS) is caused by prolonged or excessive contraction of the piriformis muscle (PM). Because of the close proximity to the sciatic nerve, PS is associated with pain in the buttocks, hips, and lower limbs [1–10].

Pain due to PS can have a significant impact on a patient's functional activities and quality of life. Anesthesiologists are commonly involved in the management of this group of patients, due to their expertise in interventional procedures and pain medicine. This chapter is intended to provide a narrative review of the history, epidemiology, etiology, anatomy, clinical presentation, and management of this pain syndrome, with an emphasis on the roles and techniques of piriformis muscle injection.

Background and Epidemiology

Yeoman (1928) was the first to ascribe sciatic pain to PS [11]. Beginning with Mixter and Barr's classic article (1934) [12], the cause of sciatica and buttock pain was increasingly attributed to the lumbar spine. Although PS is still regarded as an interesting cause of sciatica in the literature, it is given a low priority in the differential diagnosis of back–buttocks–leg pain in clinical practice [6, 13, 14]. With a few exceptions, the literature only includes isolated case reports [14–16]. There are many structures in this region that can produce the classic symptoms of PS, and many synonyms for the condition are used in the literature, such as "deep gluteal syndrome" and "pelvic outlet syndrome" [17]. By analogy with other entrapment neuropathies (such as carpal tunnel syndrome), this clinical picture could also be correctly termed "infrapiriform foramen syndrome" [18].

Recent developments in diagnosis and successful treatment based on newer diagnostic methods suggest that PS is not rare [19]. Although there is no reliable measure of the prevalence of PS, it has been suggested that PS is responsible for 5–6 % of cases of sciatica [6, 18, 20]. Taking a conservative estimate of new cases of low back pain and sciatica at 40 million annually, the incidence of PS would be 2.4 million per year [21, 22]. In the majority of cases, PS occurs in middle-aged patients (mean age 38 years) [23]. The ratio of female to male patients with PS has been reported as 6:1 [4].

Anatomy

The piriformis muscle (Latin: *pirum,* pear; *forma,* shape) was named by the Flemish anatomist Adriaan van der Spieghel (Spigelius) (1578–1625). Originating from the anterior surface of the sacrum between the first and fourth sacral foramina, the PM passes through the greater sciatic foramen and inserts on the upper edge of the greater trochanter. The greater sciatic foramen is a rigid opening formed anteriorly and superiorly by the ilium, posteriorly by the sacrotuberous ligament, and inferiorly by the sacrospinous ligament (Fig. 55.1). The PM is the only muscle that courses transversely through the greater sciatic notch, and it is the key landmark to all the important nerves and vessels that pass from the pelvis to the gluteal region, such as the sciatic nerve, the pudendal nerve and artery, and the superior and inferior gluteal arteries and nerves (Figs. 55.2 and 55.3).

The function of the PM in the non-weight-bearing limb is lateral rotation of the thigh with the hip extended and abduction when the hip is fixed at 90°. In weight-bearing activities, the piriformis restrains vigorous or excessive medial rotation of the thigh [7, 9, 14, 18]. The other short lateral rotators of the thigh at the hip (the superior gemellus, obturator internus, inferior gemellus, and quadratus femoris muscle), lying distal to the PM, may cause symptoms in PS (especially the obturator internus muscle, which is partly an intrapelvic muscle and partly a hip muscle) [7, 24]. The hamstring muscles may also cause symptoms through activation and perpetuation of trigger points (anatomic attachments of the three hamstring muscles to the ischial tuberosity) [7, 25]. The innervations of PM are usually derived from the first and second sacral nerves. The sciatic nerve arises from the ventral branches of the spinal nerves, from L4 to S3. Exiting from the pelvic cavity at the lower edge of the PM, its trunk measures 16–20 mm in breadth at its origin; runs between the ischial tuberosity and the greater trochanter; turns downward over the gemelli, the obturator internus tendon, and the quadratus femoris muscle, which separate it from the hip joint; and leaves the buttock to enter the thigh beneath the lower border of the gluteus maximus [26]. This is the region where the course of the sciatic nerve is intimately related to the PM and short rotators of the hip. There are six routes by which portions of the sciatic nerve may exit the pelvis, and these are illustrated in Fig. 55.4 [7, 13, 27–30].

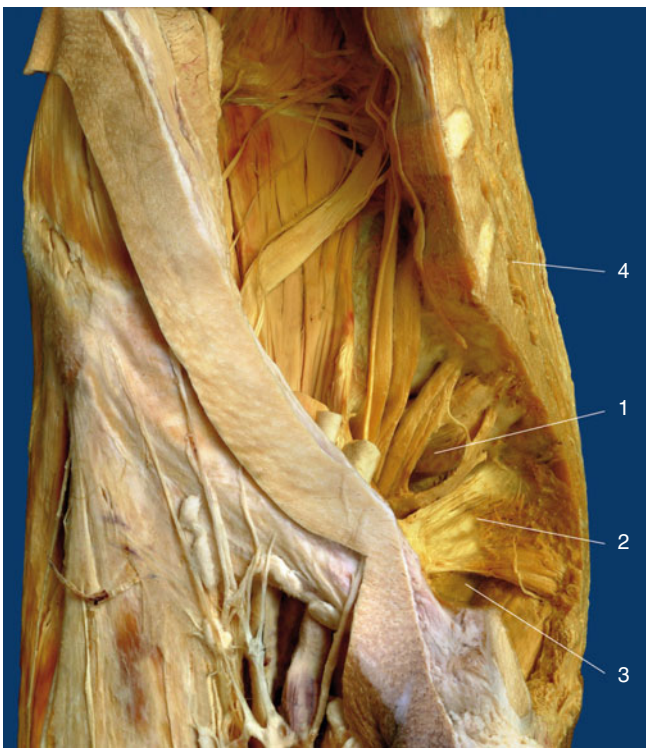


Fig. 55.1 An anatomic preparation (*anterior view*) seen from the inside of the pelvis in midsagittal view, showing the muscle attachment inside the sacrum, usually located between the first and fourth anterior sacral foramina. (*1*) Piriformis muscle, (*2*) sacrospinous ligament, (*3*) sacrotuberous ligament, (*4*) sacrum (Reproduced with permission from Danilo Jankovic)

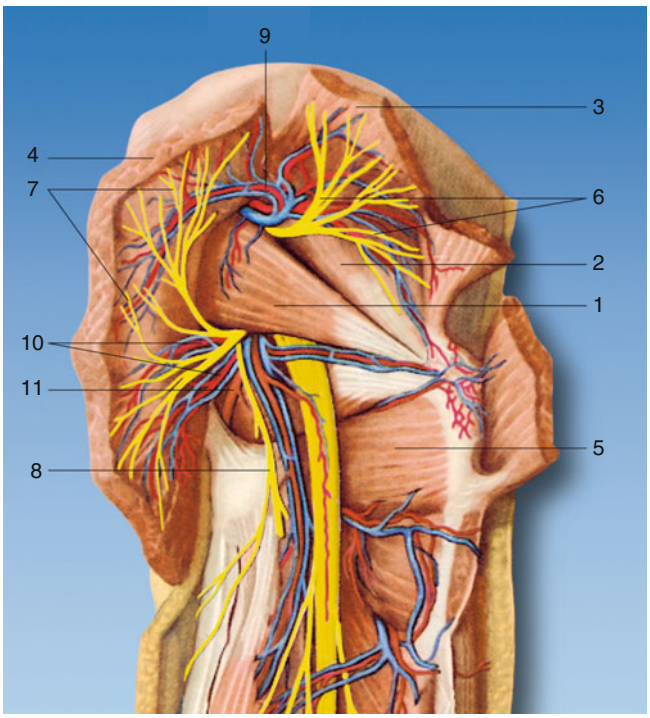


Fig. 55.2 The piriformis muscle (*1*) and neighboring muscles, nerves, and vessels: (*2*) gluteus minimus, (*3*) gluteus medius, (*4*) gluteus maximus, (*5*) quadratus femoris, (*6*) superior gluteal nerve, (*7*) inferior gluteal nerve, (*8*) posterior cutaneous femoral nerve, (*9*) superior gluteal artery, (*10*) inferior gluteal artery and vein, (*11*) internal pudendal artery (Reproduced with permission from Danilo Jankovic)

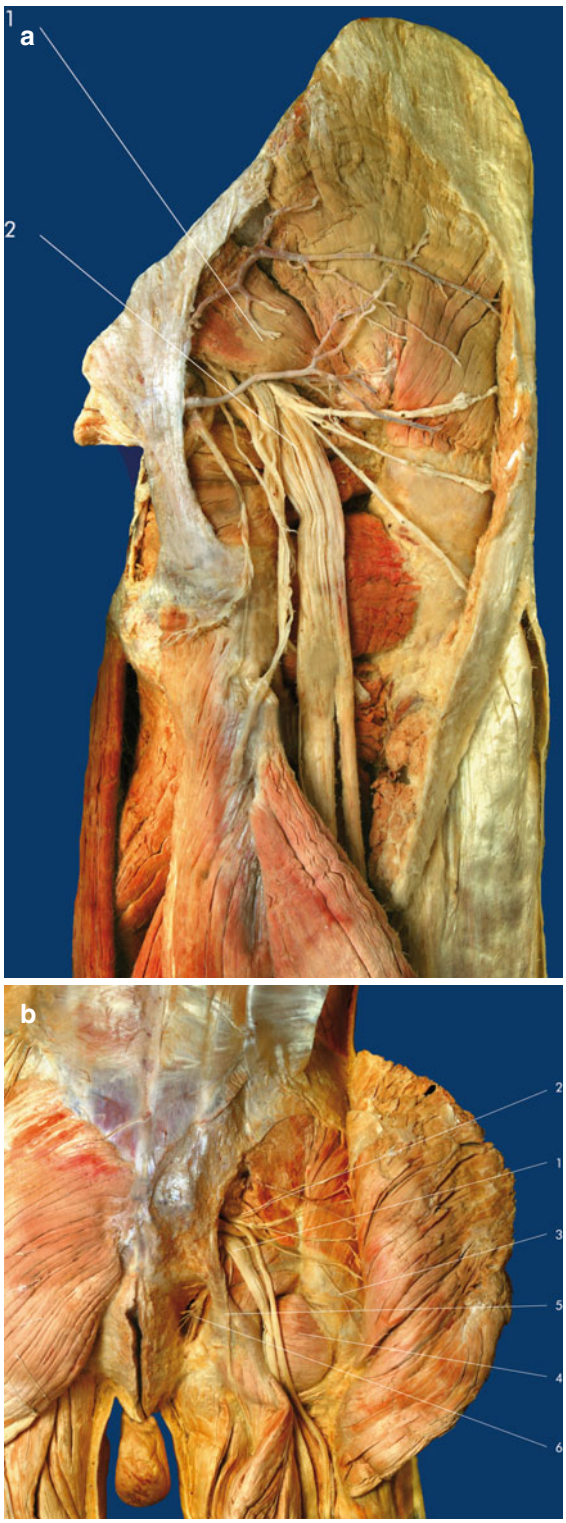


Fig. 55.3 (**a**) Posterior view of the piriformis muscle (*1*) and sciatic nerve (*2*). (**b**) High separation of the sciatic nerve (*1*): the common peroneal nerve courses through the piriformis and the tibial nerve through the infrapiriform foramen, (*2*) the piriformis muscle with the gluteal inferior nerve, (*3*) the greater trochanter, (*4*) quadratus femoris, (*5*) sacrotuberous ligament, (*6*) the pudendal nerve in the ischiorectal fossa (Reproduced with permission from Danilo Jankovic)

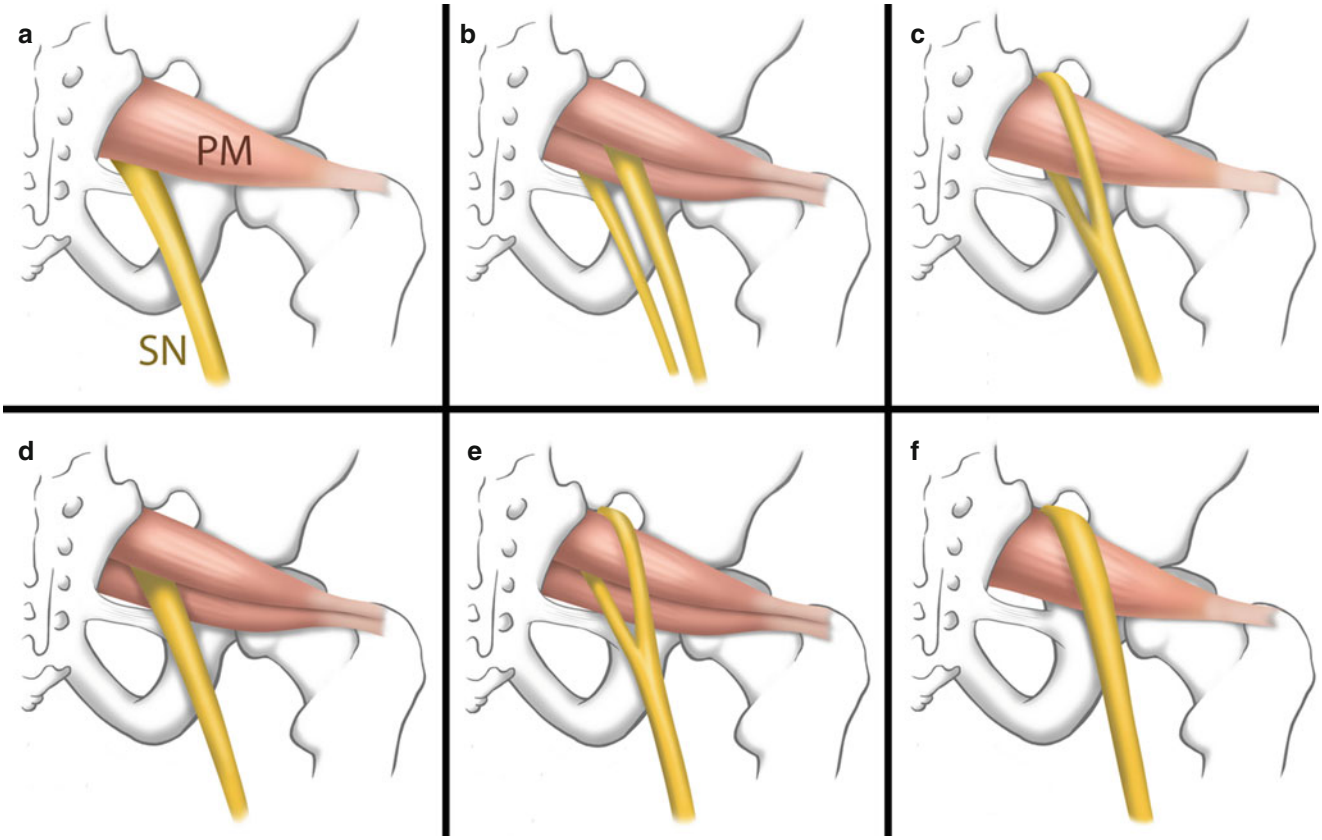


Fig. 55.4 The *six routes* by which portions of the sciatic nerve may exit the pelvis (Reproduced with permission from Philip Peng Educational Series). *SN* sciatic nerve; *PM* piriformis muscle

Pathophysiology and Etiology

There are two components that contribute to the clinical presentation: somatic and neuropathic. The *somatic component* underlying PS is a myofascial pain syndrome caused by contraction of the PM [2, 7, 14, 31, 32]. Piriformis syndrome rarely presents as a single-muscle pain syndrome [7]. Trigger points in the PM are most likely to be associated with trigger points in adjacent synergists, such as the posterior part of the gluteus minimus muscle, the gluteus maximus muscle, three of the lateral rotator group muscles (two gemelli and the obturator internus muscle), the levator ani, and the coccygeus muscles [7, 33, 34]. The *neuropathic component* refers to the compression or irritation of the sciatic nerve as it courses through the infrapiriform foramen [5, 9, 14, 20, 35–39]. In addition, irritation and compression of the neighboring nerves and vessels (superior gluteal artery, inferior gluteal artery, internal pudendal artery; Figs. 55.2 and 55.5) can give rise to pain, with a classic distribution pattern [7].

A number of etiological factors that may account for the presence of PS have been described (Table 55.1) [3–5, 7, 13–15, 18, 20, 23, 33–35, 40–59]. In most patients, there is no identifiable cause.

Previous gluteal trauma can cause sciatica-like pain [23, 35]. This is probably the most common cause of PS [13, 23, 35]. The trauma may be direct (low back or buttocks) [35] or indirect (unusual stretching of the lumbosacral and/or hip muscles through athletic or other strenuous activities), leading to inflammation, spasm, hypertrophy, and eventual contraction of the muscle [13, 23, 35]. Certain anatomic variants, such as double piriformis, and course variants of the sciatic nerve, posterior cutaneous femoral, inferior gluteal nerve, and superior gluteal nerve [4, 5, 7, 14, 15, 30, 40, 41, 60] can predispose to PS through two kinds of entrapment: entrapment between the piriformis muscle and the rim of the greater sciatic foramen and entrapment within the muscle. The first kind of entrapment has been well documented during surgery for the sciatic nerve and other surrounding neurovascular structures (superior gluteal, inferior gluteal, and pudendal nerves and vessels). The second kind of entrapment is depicted in Figs. 55.3b, 55.4, and 55.5, which depends on variations in the way in which the sciatic nerve passes through the piriformis muscle [7, 27, 30, 36, 44, 61].

Some authors have considered that *hypertrophy and spasm of the PM* [14, 18, 43, 44] is one of the most frequent myotonic reflexes in lumbar osteochondrosis [7, 62]. Previous spine surgery may cause scarring or arachnoiditis around the nerve roots, decrease the excursion of the sacral plexus, and predispose the nerve to a piriformis contraction effect.

Table 55.1 Etiology of piriformis syndrome

Gluteal trauma in the sacroiliac or gluteal areas (possibly several years previously) [13, 23, 35]
Predisposing anatomic variants [4, 5, 7, 14, 15, 40, 41]
Myofascial trigger points [7, 20, 33, 34, 42]
Hypertrophy and spasm of the piriformis muscle [14, 18, 43–45]
Secondary to laminectomy [13, 33, 34, 43, 46–50]
Abscess [51], hematoma [52, 53], myositis [54], bursitis of the piriformis muscle [55], neoplasms in the area of the infrapiriform foramen [56], colorectal carcinoma [57], neurinoma of the sciatic nerve [18], episacroiliac lipoma [50]
Intragluteal injection [58]
Femoral nailing [18]
Myositis ossificans of the piriformis muscle [3, 59]
Klippel–Trénaunay syndrome [18]

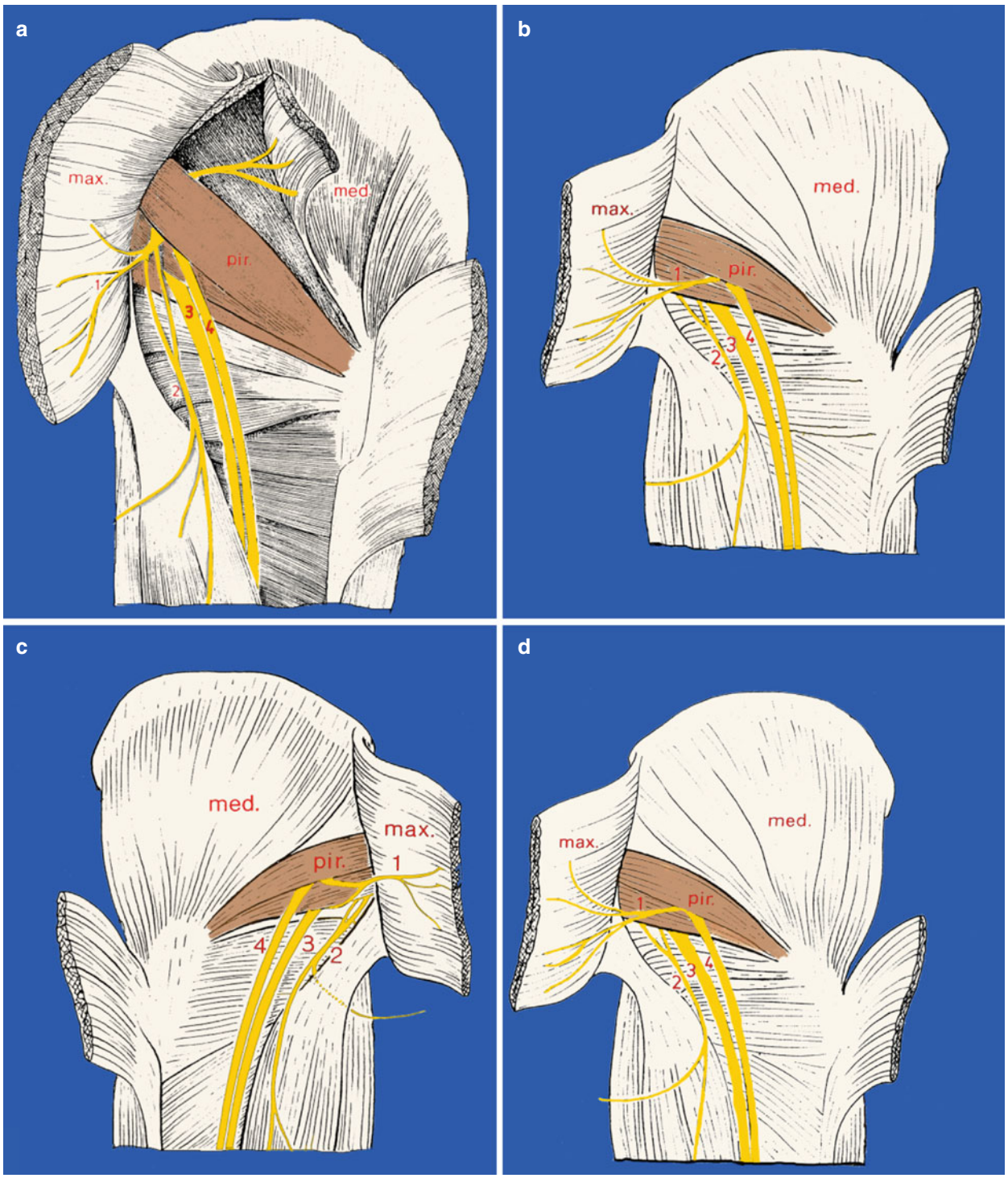


Fig. 55.5 (**a**) The right gluteal area. The gluteal inferior nerve, with the dorsal branches of the posterior femoral cutaneous nerve (*1*) and the common peroneal nerve (*2*), courses through the piriformis muscle (*4*) (intrapiriform foramen), (*3*) tibial nerve; pir, piriformis muscle; med, gluteus medius; max, gluteus maximus. (**b**) The right gluteal area. The gluteal inferior nerve (*1*) courses through the piriformis muscle. The common peroneal nerve (*4*) runs partly through the intrapiriform foramen, with a small portion passing through the infrapiriform foramen. (**c**) The left gluteal area. The inferior gluteal nerve (*1*) courses through the piriform muscle and the infrapiriform foramen. (**d**) The right gluteal area. The inferior gluteal nerve (*1*) and common peroneal nerve (*4*) exit the pelvis through the intrapiriform foramen [61] (Reproduced with permission from Danilo Jankovic)

Differential Diagnosis

The presence of PS is frequently overlooked; the differential diagnosis is presented in Table 55.2 [3, 4, 7, 9, 14, 18, 41, 45, 51, 62–67].

Table 55.2 Differential diagnosis of the piriformis syndrome

Dysfunction, lesion, and inflammation of sacroiliac joint [3, 7, 9, 63, 64]
Pseudoaneurysm in the inferior gluteal artery following gynecologic surgery [4, 65]
Thrombosis of the iliac vein [18, 51, 65]
Painful vascular compression syndrome of the sciatic nerve, caused by gluteal varicosities [6]
Herniated intervertebral disc [67]
Post-laminectomy syndrome or coccygodynia [18, 41]
Pseudoradicular S1 syndrome [45]
Posterior facet syndrome at L4–5 or L5–S1 [63]
Unrecognized pelvic fractures [14]
Lumbar osteochondrosis [7, 62]
Undiagnosed renal stones [14]

Clinical Evaluation

Clinical Presentation

Three specific conditions may contribute to PS: myofascial pain referred from trigger points in the PM, nerve and vascular entrapment by the PM at the greater sciatic foramen, and dysfunction of the sacroiliac joint.

Myofascial pain syndrome in the PM is well recognized [7, 20, 33, 34, 50, 64, 68, 69]. Additional pain referred from trigger points in the adjacent members of the lateral rotator group or gluteal muscles may be difficult to distinguish from pain originating in the piriformis trigger points. PS is often characterized by bizarre and initially apparently unconnected symptoms [7, 70]. Gluteal pain is reported to be observed in 97.9 % of cases [71]. Patients report pain (and paresthesias) in the small of the back, groin, perineum, buttocks, hip, back of the thigh (81.9 %) [71], calf (59 %) [71], foot, and also in the rectum (during defecation) and in the area of the coccyx. Low back pain is reported to be observed in 18.1 % of cases [43, 71]. Some authors have suspected that contraction of the PM is an often overlooked cause of coccygodynia [13, 28, 41]. Edwards [72] and Retzlaff et al. [9] have described the syndrome as "neuritis of the branches of the sciatic nerve," while TePoorten suspected involvement of the peroneal nerve [73]. Swelling in the affected leg and disturbances of sexual function are observed (dyspareunia in women, 13–100 % [74], and disturbances of potency in men are very often present as accompanying symptoms) [4, 7, 74]. When the common peroneal nerve is involved, there may be paresthesia of the posterior surface of the upper leg and some portions of the lower leg supplied by this nerve [23, 45, 75–77]. Dysfunction of the sacroiliac joint has been considered a common and important component of PS [9, 63, 64]. Displacement of the sacroiliac joint may interact with myofascial trigger points in the PM to establish a self-sustaining condition. Sustained tension in the muscle caused by the trigger points may maintain displacement of the joint [63], and the dysfunction induced by the joint displacement apparently perpetuates piriformis trigger points. In this situation, both conditions must be corrected [7].

However, true neurologic findings are not usually present in PS, and sensory deficits may be completely absent [3, 5, 14, 33, 34, 64]. There is no gold standard in diagnosing PS. The physical examination may reveal several of the following well-described signs (Table 55.3) [13, 71]. External palpation of the *piriformis line* can be used to elicit trigger-point tenderness through a relaxed gluteus maximus muscle. The patient is placed in the Sims position. The line overlies the superior border of the PM and extends from immediately above the greater trochanter to the cephalic border of the greater sciatic foramen at the sacrum [7, 13]. The line is divided into equal thirds. The fully rendered thumb presses on the point of maximum trigger-point tenderness, which is usually found just lateral to the junction of the middle and last thirds of the line. A positive test is reported to be observed in 59–92 % [13, 71, 74] of the patients. The *piriformis sign,* which presents as tonic external rotation of the affected lower extremity, is reported to be observed in 38.5 % of the patients [13]. The medial end of the PM should be palpated within the pelvis by *rectal or vaginal examination* (this test is positive in almost 100 % of the patients) [7, 13, 41, 73, 78, 79]. Rectal or pelvic examination may reveal a tender, palpable, sausage-shaped mass along the lateral pelvic wall. *Freiberg's sign* involves pain on passive, forced internal rotation of the hip in the supine position, thought to result from passive stretching of the PM and pressure on the sciatic nerve at the sacrospinous ligament (Fig. 55.1) [13, 14, 19, 67]. This test is positive in 56.2 % of the patients (32–63 %) [71, 74]. *Pace's sign* [33, 34] consists of pain and weakness on resisted abduction and external rotation of the thigh in a sitting position [13, 33]. A positive test is reported to occur in 46.5 % of the patients (30–74 %) [71, 74]. *Lasègue's sign* involves pain on the affected side on voluntary adduction, flexion, and internal rotation [80]. *Beatty's maneuver* [1, 37] is an active test that involves elevation of the flexed leg on the painful side while the patient lies on the asymptomatic side. Abducting the thigh to raise the knee off the table elicits deep buttock pain in patients with PS but back and leg pain in those with lumbar disc disease. The *Hughes test* [81] may be also positive in PS (external isometric rotation of the affected lower extremity following maximal internal rotation). *Gluteal atrophy* may be present, depending on the duration of the condition. When there is abnormality of the PM, gluteal atrophy may be observed because of the close proximity and involvement of the first and internal rotation second sacral nerves [13, 33, 35, 45]. Examining the patient in the supine position sometimes reveals apparent *shortening of the limb on the affected side* (this is due to contracture of the piriformis muscle) [7, 45, 73]. In the most severe cases, the patient will be unable to lie or stand comfortably, and changes in position will not relieve the pain. Intense pain will occur when the patient sits or squats (39–95 %) [74]. *Sacroiliac tenderness* is reported to be observed in 38.5 % of the patients [13]. The buttock on the same side as the PM lesion is sensitive to touch or palpation [9]. Criticism of the use of these signs has focused on the fact that they lack critical evaluation [68].

Table 55.3 Diagnostic maneuvers useful in the evaluation of piriformis syndrome

Maneuver	Sign	Cause of pain	Frequency of positive physical sign		
			n	%	Ref.
Freiberg	Buttock pain	Stretching of the PM	81	56.2	[71]
Piriformis line	Buttock pain	Contraction of the PM	118	81.9	[71]
			24	92.3	[13]
Pace	Buttock pain	Contraction of the PM	67	46.5	[71]
Rectal or vaginal examination		Reproduction of sciatic pain	26	100	[13]

Electrophysiological Tests

The role of unprovoked electrophysiological tests (in an anatomical position) is minimal. However, the diagnostic value of such tests can be improved by stressing the muscle in *flexion, adduction,* and *internal rotation* (the FAIR test). The test compares posterior tibial and peroneal H reflexes elicited in the anatomic position with H reflexes obtained in flexion, adduction, and internal rotation (normal mean prolongation: 0.01 ± 0.62 ms). A prolongation in the FAIR test of 1.86 ms is an electrophysiological criterion for diagnosing PS [14, 82]. The test correlates well with visual analog scale estimates of pain [14, 15, 19, 71, 83, 84]. Somatosensory evoked cortical potentials are also reported to objectivize sensory abnormalities of innervation [14, 85].

Imaging Modalities

Plain pelvic radiography can only identify calcification of the PM or its tendon in exceptional circumstances [14, 42]. Involvement of the PM in sciatic neuropathy has been supported by evidence from computed tomography (CT), magnetic resonance imaging (MRI) (Fig. 55.6) [62, 68, 86–90], scintigraphy [75], and ultrasound [38, 91]. However, if PS is suspected, a CT examination of the pelvis should certainly be conducted in order to detect side-to-side differences in the PM or other causes of the narrowing of the infrapiriform foramen [30, 92, 93]. If uncertainties remain, an MRI examination of the sciatic nerve and its vicinity—particularly with regard to structural changes in the PM—is indicated [94]. The newly introduced neuroradiological technique of magnetic resonance neurography, alongside established imaging methods such as MRI for evaluating unexplained chronic sciatica, has led to the identification of various changes relating to the piriformis muscle and sciatic nerve which have been further demonstrated with surgical exploration [30].

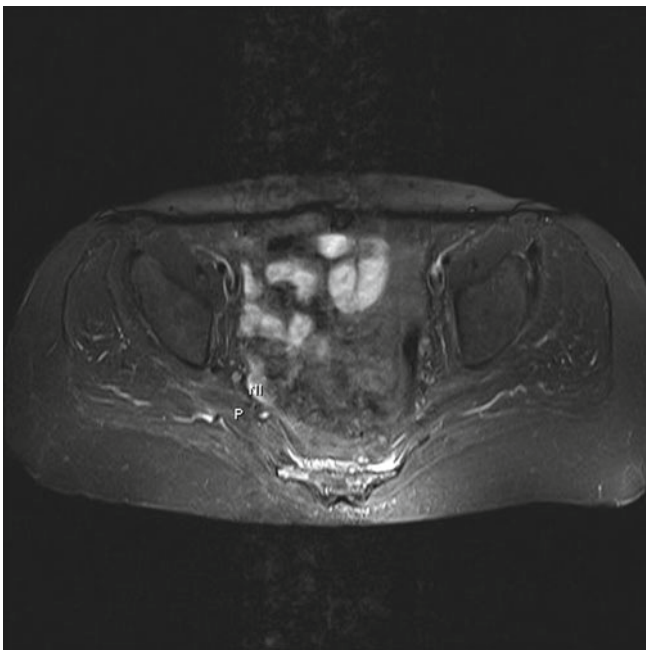


Fig. 55.6 A normal magnetic resonance image of the piriformis muscle (*P*) and sciatic nerve (*NI*) (Reproduced with permission from Danilo Jankovic)

Diagnostic Injection with Local Anesthetics and Steroids

Although piriformis muscle injection has not been compared with other diagnostic tests, it is a widely used method of establishing the diagnosis after initial evaluation [13, 33, 34]. Anesthesiologists are often involved in managing this group of patients, due to their expertise in administering diagnostic and therapeutic blocks. The utility of diagnostic injection is based on anecdotal and expert experience.

Management of Piriformis Syndrome

General

Piriformis syndrome causing sciatica usually responds to conservative treatments, including physical therapy; lifestyle modification; symptomatic relief of muscle and nerve pain with nonsteroidal anti-inflammatory agents, tricyclic antidepressants, muscle relaxants, and neuropathic pain agents such as gabapentin, pregabalin, and carbamazepine [95]; and psychotherapy. When patients fail to respond to simple conservative therapy, interventional modalities are considered. In rare circumstances surgical release of the piriformis muscle has been described for difficult cases of PS, but this is occasionally accompanied by morbidity.

Noninvasive Techniques

There is a paucity of controlled trials examining the effectiveness of the management modalities.

Physical Therapy

If etiologically treatable causes of PS have been ruled out, behavioral and physical therapy should be the first step. With regard to physical therapy, relaxation exercises for the pelvic floor muscles, stretching exercises for the gluteal muscles, massage of the PM, and exercises to mobilize the sacroiliac articulation and lumbar spine should be performed [14]. The primary consideration in treating PS with osteopathic manipulation is that the method used must relieve the contracture of the affected musculature and reduce connective-tissue adhesions in the area [9]. Osteopathic physicians use numerous methods of treatment [23, 72], and physical therapy methods are well documented [4, 5, 7, 19–21, 40, 41, 43, 64, 79, 96]. Other recommended techniques include intermittent cold with stretching [7, 34, 79], internal transrectal or transvaginal massage of the muscle [41, 68], ischemic compression with a tennis ball [7, 41], ultrasound application [7, 20], shortwave diathermy in conjunction with a full course of physical therapy [97], self-stretching of the muscle [7], corrective actions (e.g., for lower limb length differences) [7, 20], postural and activity stress reduction, and avoidance of prolonged immobilization of the affected lower limb when driving vehicles for long distances. If a Morton foot structure (mediolateral rocking foot) is present, it should be identified and corrected [7]. In general, physical therapy is performed only as part of multimodal therapy. Criticism of these methods has focused on the lack of critical evaluation.

Treatment with Focused Shock Waves

Focused shock waves have been successfully used for some 15 years now for common orthopedic indications [91, 98–102]. Although the biological mechanism of action is not yet fully understood, shock-wave therapy is being used successfully to improve the blood supply and metabolic processes [103–107]. Focused shock waves, as a unique method, make it possible to locate deep trigger points in gluteal muscles and to provoke referred trigger-point pain. The treatment of trigger points in the piriformis muscle with focused shock waves is a new and promising noninvasive method [108] as a part of multimodal therapy.

Piriformis Muscle Injection

Piriformis muscle injection with local anesthetics and corticosteroids is usually offered to patients as part of multimodal therapy. Although the optimal frequency and dosage have not been well examined, the procedure may be repeated up to three to five times if required. Optimal results are achieved by injecting botulinum toxin into the PM. The muscle can be targeted by landmark-based or image-guided techniques.

Various techniques have been described to help localize the piriformis muscle. These include imaging techniques such as ultrasound, CT, and fluoroscopy to guide the needle to the vicinity of the muscle and electrophysiological techniques such as electromyography and nerve stimulation to confirm activation of the piriformis muscle or sciatic nerve. Physicians often use combinations of one component from each technique to improve the accuracy of needle placement [43, 80, 109]. With the increasing popularity of ultrasound for intervention, the ultrasound-guided technique will be described in details.

Transgluteal Techniques

- *Fan-shaped "blind infiltration"* of the trigger points in the piriformis muscle, located by transrectal (transvaginal) palpation [33], or injection while externally palpating the "sausage-shaped" muscle when palpation is possible [13]. With all these blind approaches, the depth of needle insertion is determined subjectively and relies on judgment and expertise.
- *Transgluteal technique using a nerve stimulator* [47, 110]. One can take advantage of the close anatomic relationship of the piriformis muscle to the sciatic nerve and use a nerve stimulator to position the needle just posterior to the nerve at the level of the piriformis muscle [47]. The patient is placed in a lateral decubitus position. A line is drawn between the posterior superior iliac spine (PSIS) and the ischial tuberosity. The needle insertion point lies approximately 6 cm caudad to the PSIS on this line (parasacral approach). The insulated needle is inserted at this point and advanced in a sagittal plane until sciatic nerve stimulation is observed with a current of less than 2 mA. The needle is then withdrawn slightly until the twitching completely disappears. Occasionally, it is necessary to inject laterally in a blind fashion as well, to treat a trigger point in the lateral portion of the muscle.
- *Injection of the piriformis muscle under fluoroscopic guidance.* CT scanners and electromyography machines are not widely available to interventional physicians, and a fluoroscopy-guided technique is commonly used. Fluoroscopy displays bony landmarks such as the sacrum or sacroiliac joint but not the piriformis muscle itself. The target is the belly of the piriformis muscle in the sciatic notch, which can be approximated by measuring 2 cm lateral and 1 cm caudal to the sacroiliac joint [43] or 1 cm lateral to the S2–S3 sacral border [111]. The depth of the needle can be estimated using two methods. Firstly, the needle can be directed toward the lateral border of the sacrum at the S2–S3 level. Once bony contact has been made, the needle is redirected 1 cm away from the sacrum and advanced 1 cm deep. Radiographic contrast is then injected to show a characteristic intrapiriformis contrast pattern, which suggests needle placement within the piriformis muscle (Fig. 55.7) [111, 112]. Secondly, electromyography (with gluteus maximus and piriformis muscle contraction) and nerve stimulation (with a motor response to sciatic nerve stimulation) have been used to position the needle in the piriformis muscle [80].

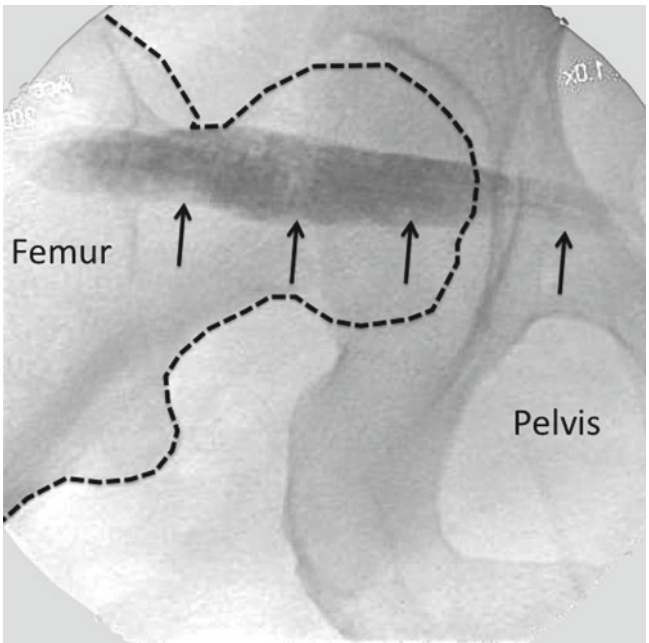


Fig. 55.7 Radiographic contrast image outlining the piriformis muscle (*arrows*) (Reproduced with permission from Philip Peng Educational Series)

Ultrasound-Guided Injection

The target for injection is the piriformis muscle, which is a soft tissue. Although both CT and ultrasound allow direct visualization of the piriformis muscle, ultrasound is regarded as an attractive and affordable imaging technique, as it allows real-time imaging of needle insertion toward the target without exposing the patient to radiation [109]. There have been many reports of ultrasound-guided piriformis muscle injection that describe similar techniques with minor variations [27, 38, 86, 111, 113, 114]. The accuracy of needle placement with ultrasound was recently validated in a cadaver study, suggesting an accuracy of 95 % [111].

Sonoanatomy [109, 114]

The key for locating the PM is the greater sciatic notch (Fig. 55.8a). The patient is placed in the prone position. The ultrasound probe is placed just lateral to the posterior superior iliac spine (PSIS), revealing a hyperechoic bone shadow from the ilium (Fig. 55.8b). The ultrasound probe is then moved in the caudal direction toward the sciatic notch. At this level, the hyperechoic shadow of the bone will disappear from the medial aspect and two muscle layers will be visible—the gluteus maximus and the piriformis. The piriformis muscle can be better visualized by rotating the hip externally and internally with the knee flexed. This movement allows gliding of the piriformis muscle in real time and helps the practitioner distinguish the PM from the gluteus muscle (Fig. 55.8c). The ultrasound scan should also demonstrate the presence of the sciatic nerve, inferior gluteal artery, and pelvic cavity, which are deep to the piriformis muscle (Fig. 55.8c, d).

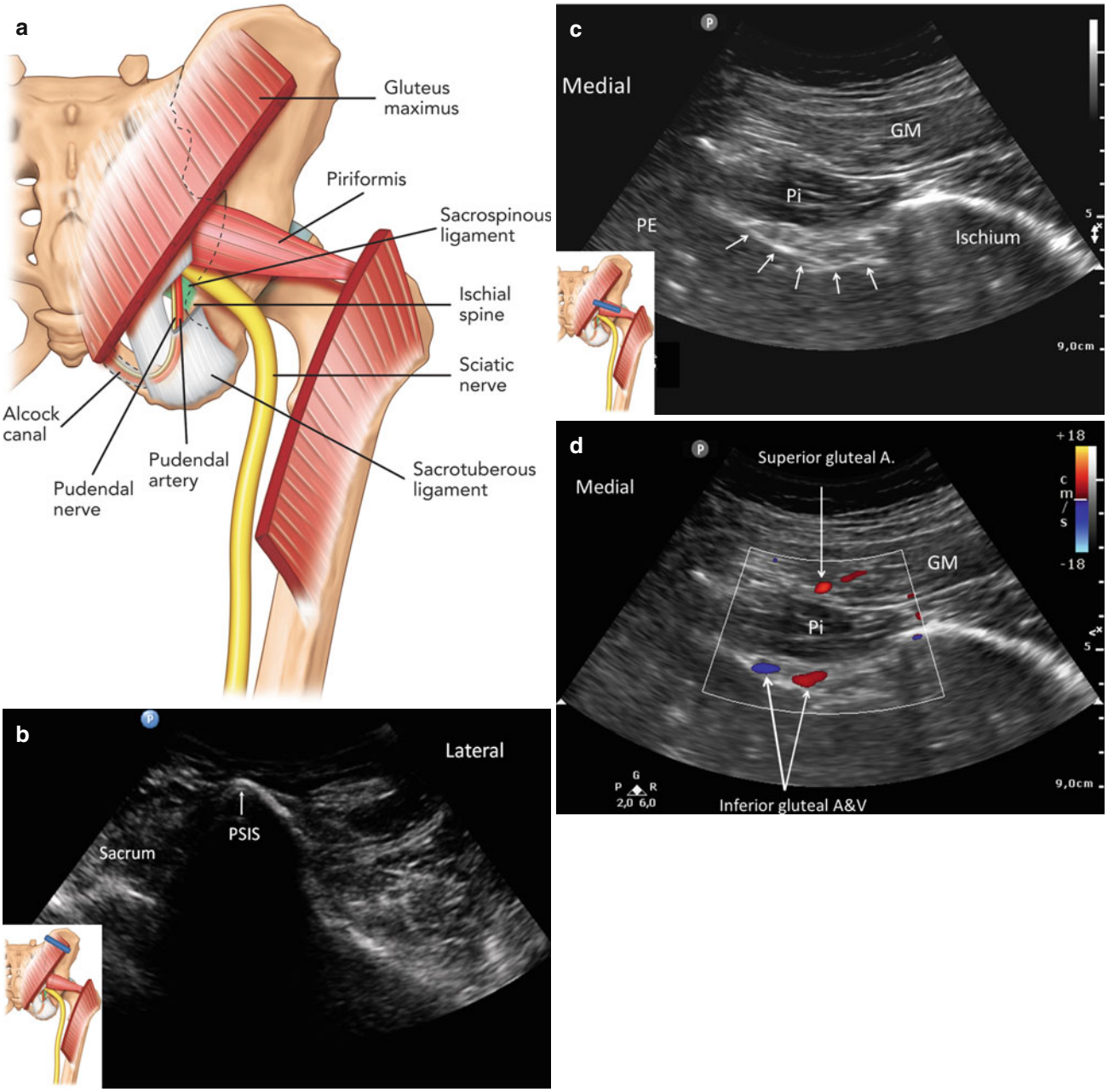


Fig. 55.8 (**a**) Posterior view of the pelvis, showing the piriformis muscle and surrounding structures. The gluteus maximus muscle has been transected to show the deeper structures. It should be noted that the sciatic nerve typically emerges caudal to the piriformis muscle in the greater sciatic notch. (**b**) Ultrasonography of the ilium cephalad to the greater sciatic notch. The position of the ultrasound probe (*dark rectangle*) is indicated in the insert. The ilium appears as a hyperechoic line. PSIS, posterior superior iliac spine. (**c**) Ultrasound of the greater sciatic notch, with the position of the ultrasound probe indicated in the insert (*dark rectangle*). The sciatic nerve is seen as a structure deep to the piriformis muscle, indicated by the arrows. *GM* gluteus maximus muscle, *PE* peritoneum, *Pi* piriformis muscle. (**d**) Ultrasonography of the sciatic notch as in **c**, with Doppler imaging. The inferior gluteal artery is seen adjacent to the sciatic nerve, and the superior gluteal artery is located between the gluteus maximus (*GM*) and piriformis muscle (*Pi*). *A* artery, *V* vein (Reproduced with permission from Philip Peng Educational Series)

Injection Technique

The needle is inserted from medial to lateral using an in-plane technique. Due to anatomic anomalies of the sciatic nerve within and below the piriformis muscle, a practitioner with limited experience with ultrasound-guided injection is advised to perform the needle insertion with the nerve stimulator, to prevent unintentional injection in the vicinity of the sciatic nerve. The stimulating current is usually set at 1 mA. Either a 3.5-in 22-G spinal needle or an 80-mm insulated needle is usually sufficient, but a longer needle is required for patients with a high body mass index. A very small amount of normal saline (<0.5 mL) is injected to confirm the intramuscular location of the needle (hydrolocation). The author (P.P) usually chooses a small volume (1–1.5 mL) of injectate, whether it is botulinum toxin or a mixture of local anesthetic with steroid. It is not uncommon for sciatic nerve stimulation to be observed when the needle is advanced through the muscle.

Limitations of the Various Injection Techniques

Given the proximity of the piriformis muscle to the pelvic cavity, sciatic nerve, and inferior gluteal artery (Figs. 55.2 and 55.8c, d), landmark-based infiltration is not recommended.

Frequently, the *landmark-based* technique is accompanied with an electrophysiological stimulation method, such as the use of a nerve stimulator [47, 48, 110] or electromyography [80, 82, 96]; however, there are limitations with localization method that use electrophysiological techniques. The premise in these techniques is that the close proximity of the needle to the muscle or nerve will reliably produce a brisk motor unit action potential or muscle contraction. Although this concept has *not* been validated for the electromyography-guided technique, the needle-to-nerve proximity relationship in nerve stimulation has been examined [115]. Several studies using in vivo models have shown that the minimum stimulating current may not reliably reflect the distance of the needle tip from the nerve [116–119]. Furthermore, the nerve stimulation technique cannot reliably differentiate whether the needle tip is within the muscle or lying in plane between muscles (an important consideration when botulinum toxin is being injected). Both electrophysiological approaches neither allow direct visualization of the muscle nor ensure accurate positioning of the needle within the PM [109].

Localization of the piriformis muscle using the *fluoroscopy-assisted* contrast injection technique has also been examined. A cadaver study showed that the accuracy of this method was only 30 %, with most of the needle tip being positioned in the gluteus maximus muscle [111]. This is not surprising given the fact that the fluoroscopy technique does not allow direct visualization of the soft tissue.

Ultrasound and *computer tomography (CT)* have the advantage of allowing direct visualization of the piriformis muscle. The reliability of the ultrasound-guided method has been confirmed in a cadaver study [111]. Compared with a CT-guided technique, ultrasound is much more affordable and accessible. The ultrasound-guided technique also offers the additional advantages of avoiding radiation exposure and real-time injection [109]. In the experience of one of the present authors (P.P.), it is not uncommon for the patient to react when the practitioner injects the medication into the muscle. The pressure sensation on injection may elicit gluteus muscle contraction, which can displace the needle tip from the piriformis muscle. This is particularly the case if the patient has developed piriformis atrophy with repeated injections of botulinum toxin. Real-time surveillance of the spread of the injectate can ensure that the needle is positioned within the muscle throughout the injection procedure.

Injection Solution

Mixing the local anesthetic solution with 20–40 mg of a long-acting corticosteroid (e.g., long-acting methylprednisolone) is also recommended. Experience shows that long-acting local anesthetics do not provide any substantial advantages over short-acting agents [7, 13, 110]. In addition to the anti-inflammatory properties, steroids also have membrane-stabilizing properties, by suppressing transmission in unmyelinated C fibers and inhibiting ectopic discharge from the experimentally created neuroma [120, 121]. It should be pointed out to the patient that spreading of the local anesthetic (particularly with a long-acting agent) in the region of the sciatic nerve can lead to the leg suddenly giving away later (written information should be provided for outpatients).

Response to Injections

The response to injections can be immediate but may be of short duration. Benzon et al. [43] reported that 16 of 19 patients responded to piriformis muscle injections over a short-term follow-up period of 3 months. However, the majority of these patients had associated diagnoses, including herniated discs, spinal stenosis, failed back surgery syndrome, and complex regional pain syndrome. Filler et al. [46] reported on 46 patients who received local anesthetic injections. Sixteen percent had sustained pain relief for periods ranging from 8 months to 6 years; 8 % had 2–4 months of relief but required a second injection; 37 % had 2–4

months of relief but experienced recurrence after a second injection; 24 % of the patients benefited for only 2 weeks; and 16 % experienced no benefit. Recent reports have focused on botulinum toxin injections.

Botulinum Toxin Injections in PS [14, 21, 31, 37, 48, 77, 90, 93, 96, 112, 122]

Botulinum toxin type A is one of the seven immunologically distinct serotypes (A–G) of neurotoxin produced by *Clostridium botulinum.* Botulinum toxin type A inhibits the release of acetylcholine from peripheral cholinergic, motor, and autonomic nerve endings. After it reaches the nerve terminals at a neuromuscular junction, botulinum toxin type A functionally denervates muscle fibers and causes flaccid paralysis [77]. Initial clinical use of botulinum toxin type A was limited to the treatment of strabismus and blepharospasm associated with dystonia. However, botulinum toxin type A is now regarded as the treatment of choice for hemifacial spasms, spasmodic torticollis (cervical dystonia), oromandibular dystonia, laryngeal dystonia, and pediatric cerebral palsy. It has also been beneficial in many other conditions, including myofascial pain syndrome, focal hyperhidrosis, tremors, achalasia, tension-type headaches, and PS. Botulinum toxin type A can be administered with fluoroscopic, electromyelographic, CT, or MRI guidance. The recommended dose of botulinum toxin type A in PS is usually 100–200 units [2, 14, 21, 37, 77].

In summary, the indications, techniques, dosages, monitoring, and location vary significantly. This variability limits any comparison of studies and treatment groups. There is a lack of double-blind, randomized, controlled trials. More controlled studies are needed in order to determine the number of nerve blocks required in chronic pain therapy and to establish selection criteria for patients who are suitable for nerve blocks in pain therapy. The efficacy of nerve blocks depends on the stage of development of chronic pain.

Surgical Treatment

Surgical intervention should be considered when nonsurgical treatment has failed and the symptoms are becoming intractable and disabling. There is a lack of literature on surgical treatment for PS.

Division of the piriformis muscle is intended only to relieve intractable sciatic pain in well-selected patients and cannot cure or alleviate any other neurological deficit such as reflex changes, muscular weakness, or sensory losses [49]. Classic indications for surgical treatment include abscess, neoplasms, hematoma [5, 23, 35, 45, 46], and painful vascular compression of the sciatic nerve caused by gluteal varicosities, etc. [66]. Since the introduction of botulinum toxin therapy, however, surgical interventions have rarely been necessary in patients with PS. Surgical severance of the piriformis muscle or its tendon and neurolysis of the constricting tissue around the nerve may be considered [23, 35, 44, 49, 70]. Severance of the tendon is not always successful [84]. Indrevekam and Sudmann reported on a series of 19 patients with PS who were treated using tenotomy [123], and Filler et al. reported the results of surgical treatment for PS in 64 patients (82 % with good results) [46]. In 78 documented operations, the treatment was described as successful in two thirds of the cases but showed no benefit in one third [71]. The technical details of surgical treatment are beyond the scope of this review.

Conclusion

PS continues to be a controversial diagnosis for sciatic pain. In view of the distribution of the nerves and blood vessels that accompany the piriformis muscle through the greater sciatic foramen, it is understandable why contracture of a single muscle, the piriformis, can have such widespread effects. The diagnosis is mainly made on clinical grounds. Piriformis syndrome is associated with pain (and paresthesias) in the buttocks, hips, and lower limbs. Electrophysiological testing and nerve blocks play important roles when the diagnosis is uncertain. Clinicians should be aware that there are many etiological factors involved, which it may be possible to modify or treat. Most patients respond to conservative measures, including nerve blocks. Anesthesiologists are commonly involved in the management of PS, due to their expertise in pain management and in carrying out nerve blocks. Injections of local anesthetics, steroids, and botulinum toxin into the piriformis muscle can serve both diagnostic and therapeutic purposes. The practitioner should be familiar with variations in the anatomy and the limitations of landmark-based techniques. An ultrasound-guided injection technique has recently been described, which offers improved accuracy in the nerve blockade. This technique has been shown to have both diagnostic and therapeutic value in the treatment of PS. The results of surgical treatment for PS are disappointing. Optimizing the therapeutic approach requires an interdisciplinary evaluation of treatment.

References

1. Beatty RA. The piriformis muscle syndrome: a simple diagnostic maneuver. Neurosurgery. 1994;34:512–4.
2. Fanucci EA, Masala SA, Sodani GA, et al. CT-guided injection of botulinic toxin for percutaneous therapy of piriformis muscle syndrome with preliminary MRI results about denervative process. Eur Radiol. 2001;11:2543–8.
3. Foster MR. Piriformis syndrome. Orthopedics. 2002;25:821–5.
4. Papadopoulos EC, Khan SN. Piriformis syndrome and low back pain: a new classification and review of the literature. Orthop Clin North Am. 2004;35:65–71.
5. Parziale JR, Hudgins TH, Fischman LM. The piriformis syndrome. Am J Orthop (Belle Mead NJ). 1996;25:819–23.
6. Silver JK, Leadbetter WB. Piriformis syndrome: assessment of current practice and literature review. Orthopedics. 1998;21:1133–5.
7. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual, The lower extremities, vol. 2. Baltimore: Williams & Wilkins; 1992. p. 186–214.
8. Hollinshead WH. Buttock, hip joint and thigh. In: Hollinshead WH, editor. Anatomy for surgeons, The back and limbs, vol. 3. 3rd ed. New York: Harper and Row; 1982. p. 666–8, 702.
9. Retzlaff EW, Berry AH, Haight AS, et al. The piriformis muscle syndrome. J Am Osteopath Assoc. 1974;73:799–807.
10. McCrory P, Bell S. Nerve entrapment syndromes as a cause of pain in the hip, groin and buttock. Sports Med. 1999;27:261–74.
11. Yeoman W. The relation of arthritis of the sacroiliac joint to sciatica: with an analysis of 100 cases. Lancet. 1928;2:1119–22.
12. Mixter WJ, Barr JS. Ruptures of the intervertebral disc with involvement of the spinal canal. N Engl J Med. 1934;211:210.
13. Durrani Z, Winnie AP. Piriformis muscle syndrome: an underdiagnosed cause of sciatica. J Pain Symptom Manage. 1991;6:374–9.
14. Reichel G. Treatment of piriformis syndrome with botulinum toxin. Pain Headache. 2003;14:140–58.
15. Huber HM. The piriformis syndrome — a possible cause of sciatica. Schweiz Rundsch Med Prax. 1990;79:235–6. German.
16. Pfeifer T, Fitz WF. The piriformis syndrome. Z Orthop Ihre Grenzgeb. 1989;127:691–4. German.
17. Hopayian K. Sciatica in the community — not always disc herniation. Int J Clin Pract. 1999;53:197–8.
18. Reichel G, Gaerisch Jr F. Piriformis syndrome. A contribution to the differential diagnosis of lumbago and coccygodynia. Zentralbl Neurochir. 1988;49:178–84. German.
19. Fishman LM, Dombi GW, Michaelsen C, et al. Piriformis syndrome: diagnosis, treatment, and outcome — a 10-year study. Arch Phys Med Rehabil. 2002;83:295–301.
20. Hallin RP. Sciatic pain and the piriformis muscle. Postgrad Med. 1983;74:69–72.
21. Fishman LM, Anderson C, Rosner B. Botox and physical therapy in the treatment of piriformis syndrome. Am J Phys Med Rehabil. 2002;81:936–42.
22. Bigos S, Bowyer O, Braen G, et al. Acute low back problems in adults. Rockville: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services; 1994 (Clinical practice guideline no. 14. AHCPR Publication No. 95–0642).
23. Benson ER, Schutzer SH. Posttraumatic piriformis syndrome: diagnosis and results of operative treatment. J Bone Joint Surg Am. 1999;81:941–9.
24. Meknas K, Christensen A, Johansen O. The internal obturator muscle may cause sciatic pain. Pain. 2003;104:375–80.
25. Puranen J, Orava S. The hamstring syndrome — a new gluteal sciatica. Ann Chir Gynaecol. 1991;80:212–4.
26. Netter FH. Anatomie. In: Firbas W, editor. Farbatlanten der Medizin, Bewegungsapparat I: Anatomie, Embryologie, Physiologie und Stoffwechselkrankheiten, vol. 7. Stuttgart: Thieme; 1992. p. 79–84.
27. Peng PH. Piriformis syndrome. In: Peng PH, editor. Ultrasound for pain medicine intervention: a practical guide, Pelvic pain, vol. 2. Philip peng educational series. 1st ed. iBook, California CA: Apple Inc; 2013.
28. Beaton LE, Anson BJ. The relation of the sciatic nerve and of its subdivisions to the piriformis muscle. Anat Rec. 1937;70:1–5.
29. Uluutku MH, Kurtoğlu Z. Variations of nerves located in deep gluteal region. Okajimas Folia Anat Jpn. 1999;76:273–6.
30. Cassidy L, Walters A, Bubb K, Shoja M, Tubbs SR, Loukas M. Piriformis syndrome: implications of anatomical variations, diagnostic techniques, and treatment. Surg Radiol Anat. 2012;34:479–86.
31. Childers MK. Use of botulinum toxin type A in pain management. Columbia/Austin: Academic Information Systems/Greenleaf Book Group; 2002.
32. Porta M. A comparative trial of botulinum toxin type A and methylprednisolone for the treatment of myofascial pain syndrome and pain from chronic muscle spasm. Pain. 2000;85:101–5.
33. Pace JB. Commonly overlooked pain syndromes responsive to simple therapy. Postgrad Med. 1975;58:107–13.
34. Pace JB, Nagle D. Piriform syndrome. West J Med. 1976;124:435–9.
35. Robinson DR. Piriformis syndrome in relation to sciatic pain. Am J Surg. 1947;73:355–8.
36. Pećina M. Contribution to the etiological explanation of the piriformis syndrome. Acta Anat (Basel). 1979;105:181–7.
37. Kirschner JS, Foye PM, Cole JL. Piriformis syndrome, diagnosis and treatment. Muscle Nerve. 2009;40:10–8.
38. Smith J, Hurdle MF, Locketz AJ, Wisniewski SJ. Ultrasound-guided piriformis injection: technique description and verification. Arch Phys Med Rehabil. 2006;87:1664–7.
39. Goldner JL. Piriformis compression causing low back and lower extremity pain. Am J Orthop (Belle Mead NJ). 1997;26:316–8.
40. Douglas S. Sciatic pain and piriformis syndrome. Nurse Pract. 1997;22:166–8.
41. Thiele GH. Coccygodynia and pain in the superior gluteal region. JAMA. 1937;109:1271–5.
42. Stark P, Hildebrandt-Stark HE. Calcific tendinitis of the piriform muscle. Rofo. 1983;138:111–2.
43. Benzon HT, Katz JA, Benzon HA, Iqbal MS. Piriformis syndrome: anatomic considerations, a new injection technique, and a review of the literature. Anesthesiology. 2003;98:1442–8.
44. Chen WS, Wan YL. Sciatica caused by piriformis muscle syndrome: report of two cases. J Formos Med Assoc. 1992;91:647–50.
45. Rodrigue T, Hardy RW. Diagnosis and treatment of piriformis syndrome. Neurosurg Clin N Am. 2001;12:311–9.
46. Filler AG, Haynes BA, Jordan SE, et al. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. J Neurosurg Spine. 2005;2:99–115.
47. Hanania M. New technique for piriformis muscle injection using a nerve stimulator. Reg Anesth. 1997;22:200–2.
48. Hanania M, Kitain E. Perisciatic injection of steroid for the treatment of sciatica due to piriformis syndrome. Reg Anesth Pain Med. 1998;23:223–8.
49. Mizugushi T. Division of the piriformis muscle for the treatment of sciatica. Postlaminectomy syndrome and osteoarthritis of the spine. Arch Surg. 1976;111:719–22.
50. Pace JB, Henning C. Episacroiliac lipoma. Am Fam Physician. 1972;6:70–3.
51. Arai Y, Kawakami T, Soga H, Okada Y. Psoas abscess associated with iliac vein thrombosis and piriformis and gluteal abscesses. Int J Urol. 1999;6:257–9.

52. Katati M, Vilchez R, Piñar L, et al. Haematoma of the piriformis muscle simulating a giant presacral tumour: unusual case of lumbosacral radiculopathy. Acta Neurochir (Wien). 1998;140: 403–4.
53. Ku A, Kern H, Lachman E, Nagler W. Sciatic nerve impingement from piriformis hematoma due to prolonged labor. Muscle Nerve. 1995;18:789–90.
54. Chusid MJ, Hill WC, Bevan JA, Sty JR. Proteus pyomyositis of the piriformis muscle in a swimmer. Clin Infect Dis. 1998;26:194–5.
55. Peh WC, Reinus WR. Piriformis bursitis causing sciatic neuropathy. Skeletal Radiol. 1995;244:474–6.
56. Höckel M. Laterally extended endopelvic resection: surgical treatment of infrailiac pelvic wall recurrences of gynecologic malignancies. Am J Obstet Gynecol. 1999;180:306–12.
57. LaBan MM, Meerschaert JR, Taylor RS. Electromyographic evidence of inferior gluteal nerve compromise: an early representation of recurrent colorectal carcinoma. Arch Phys Med Rehabil. 1982;63:33–5.
58. Obach J, Aragones JM, Ruano D. The infrapiriformis foramen syndrome resulting from intragluteal injection. J Neurol Sci. 1983;58:135–42.
59. Beauchesne RP, Schutzer SF. Myositis ossificans of the piriformis muscle: an unusual cause of piriformis syndrome. A case report. J Bone Joint Surg Am. 1997;79:906–10.
60. Rask MR. Superior gluteal nerve entrapment syndrome. Muscle Nerve. 1980;3:304–7.
61. Tillmann B. Variations in the pathway of the inferior gluteal nerve (author's transl). Anat Anz. 1979;145:293–302. German.
62. Kipervas IP, Ivanov LA, Urikh EA, Pakhomov SK. Clinico-electromyographic characteristics of piriform muscle syndromes. Zh Nevropatol Psikhiatr Im S S Korsakova. 1976;76:1289–92. Russian.
63. Kirkaldy-Willis WH, Hill RJ. A more precise diagnosis for low-back pain. Spine (Phila Pa 1976). 1979;4:102–9.
64. Steiner C, Staubs C, Ganon M, Buhlinger C. Piriformis syndrome: pathogenesis, diagnosis, and treatment. J Am Osteopath Assoc. 1987;87:318–23.
65. Papadopoulos SM, McGillicuddy JE, Albers JW. Unusual cause of "piriformis muscle syndrome". Arch Neurol. 1990;47:1144–6.
66. Bendszus M, Rieckmann P, Perez J, Koltzenburg M, Reiners K, Solymosi L. Painful vascular compression syndrome of the sciatic nerve caused by gluteal varicosities. Neurology. 2003;61:985–7.
67. Freiberg H. Sciatic pain and its relief by operations on muscle and fascia. Arch Surg. 1937;34:337–50.
68. Stewart JD. The piriformis syndrome is overdiagnosed. Muscle Nerve. 2003;28:644–6.
69. Wynant GM. Chronic pain syndromes and treatment III. The piriformis syndrome. Can Anaesth Soc J. 1979;26:305–8.
70. Solheim LF, Siewers P, Paus B. The piriformis muscle syndrome. Sciatic nerve entrapment treated with section of the piriformis muscle. Acta Orthop Scand. 1981;52:73–5.
71. Blaser-Sziede R. Piriformissyndrom — kritische Beurteilung der Literatur und Diskussion der klinischen Zusammenhänge. Man Ther. 2006;10:159–69.
72. Edwards FO. Piriformis syndrome. In: Academy of Applied Osteopathy Yearbook. Carmel, CA: Academy of Applied Osteopathy; 1962:39–41.
73. TePoorten BA. The piriformis muscle. J Am Osteopath Assoc. 1969;69:150–60.
74. Hopayian K, Song F, Riera R, Sambandan S. The clinical features of the piriformis syndrome: a systematic review. Eur Spine J. 2010;19:2095–109.
75. Karl Jr RD, Yedinak MA, Hartshorne MF, et al. Scintigraphic appearance of the piriformis muscle syndrome. Clin Nucl Med. 1985;10:361–3.
76. Sayson SC, Ducey JP, Maybrey JB, Wesley RL, Vermilion D. Sciatic entrapment neuropathy associated with an anomalous piriformis muscle. Pain. 1994;59:149–52.
77. Yoon SJ, Ho J, Kang HY, et al. Low-dose botulinum toxin type A for the treatment of refractory piriformis syndrome. Pharmacotherapy. 2007;27:657–65.
78. Barton PM, Grainger RW, Nicholson RL, et al. Toward a rational management of piriformis syndrome. Arch Phys Med Rehabil. 1988;69:784–9.
79. Barton PM. Piriformis syndrome: a rational approach to management. Pain. 1991;47:345–52.
80. Fishman SM, Caneris OA, Bandmann TB, Audette JF, Borsook D. Injection of the piriformis muscle by fluoroscopic and electromyographic guidance. Reg Anesth Pain Med. 1998;23:554–9.
81. Hughes SS, Goldstein MN, Hicks DG, Pellegrini Jr VD. Extrapelvic compression of the sciatic nerve. An unusual cause of pain about the hip: report of five cases. J Bone Joint Surg Am. 1992;74:1553–9.
82. Fishman LM, Zybert PA. Electrophysiologic evidence of piriformis syndrome. Arch Phys Med Rehabil. 1992;73:359–64.
83. Dumitru D, Nelson MR. Posterior femoral cutaneous nerve conduction. Arch Phys Med Rehabil. 1990;71:979–82.
84. Spinner RJ, Thomas NM, Kline DG. Failure of surgical decompression for a presumed case of piriformis syndrome. Case report. J Neurosurg. 2001;94:652–4.
85. Nainzadeh N, Lane ME. Somatosensory evoked potentials following pudendal nerve stimulation as indicators of low sacral root involvement in a postlaminectomy patient. Arch Phys Med Rehabil. 1987;68:170–2.
86. Broadhurst NA, Simmons DN, Bond MJ. Piriformis syndrome: correlation of muscle morphology with symptoms and signs. Arch Phys Med Rehabil. 2004;85:2036–9.
87. Jankiewicz JJ, Hennrikus WL, Houkom JA. The appearance of the piriformis muscle syndrome in computed tomography and magnetic resonance imaging. A case report and review of the literature. Clin Orthop Relat Res. 1991;262:205–9.
88. Lee EY, Margherita AJ, Gierada DS, Narra VR. MRI of piriformis syndrome. AJR Am J Roentgenol. 2004;183:63–4.
89. Rossi P, Cardinali P, Serrao M, Parisi L, Bianco F, De Bac S. Magnetic resonance imaging findings in piriformis syndrome: a case report. Arch Phys Med Rehabil. 2001;82:519–21.
90. Yue SK. Morphological findings of asymmetrical and dystrophic psoas and piriformis muscles in chronic lower back pain during CT guided botulinum toxin injections [abstract]. Reg Anesth Pain Med. 1998;23(3 Suppl):104.
91. Jankovic D. Case study: shock waves treatment of diabetic gangrene. Int Wound J. 2011;8:206–9.
92. Ueno K, Matsuzawa H, Inoue A. ^{67}Ga imaging of gluteal muscle inflammation secondary to pyogenic sacroiliitis (PSI). Rinsho Hoshasen. 1985;30:319–22. Japanese.
93. Fanucci E, Masala S, Squillaci E, et al. Piriformis muscle syndrome: CT/MR findings in the percutaneous therapy with botulinic toxin. Radiol Med. 2003;105:69–75.
94. Almanza MY, Poon-Chue A, Terk MR. Dual oblique MR method for imaging the sciatic nerve. J Comput Assist Tomogr. 1999;23:138–40.
95. Dworkin RH, O'Connor AB, Bakonja M, et al. Pharmacologic management of neuropathic pain: evidence-based recommendations. Pain. 2007;132:237–51.
96. Fishman LM, Konnoth C, Rozner B. Botulinum neurotoxin type B and physical therapy in the treatment of piriformis syndrome: a dose-finding study. Am J Phys Med Rehabil. 2004;83:42–50.
97. Jan MH, Lin YF. Clinical experience of applying shortwave diathermy over the piriformis in sciatic patients. Taiwan Yi Xue Hui Za Zhi. 1983;82:1065–70. Chinese.

98. Dreisilker U, Wess O, Novak P. Extrakorporal erzeugte Stoss- und Druckwellen — eine wirksame Therapieform für die Geweberegeneration. Orthop Rep Spez. 2007;147–51.
99. Valchanou VD, Michailov P. High energy shock waves in the treatment of delayed and nonunion of fractures. Int Orthop. 1991;15:181–4.
100. Piontkowski U, Sommer S. Combined EPAT/focussed shock wave therapy and trigger points in sports medicine. [Paper presented at the Eleventh International Society of Medical Shockwave Treatment (ISMST) Congress, Juan-les-Pins, 5–7 June 2008]. Abstract available at: http://www.ismst.com/pdf/abstracts11congress.pdf.
101. Moretti B, Notarnicola A, Maggio G, et al. ESWT-induced healing of diabetic foot ulcers. [Paper presented at the Eleventh International Society of Medical Shockwave Treatment (ISMST) Congress, Juan-les-Pins, 5–7 June 2008]. Abstract available at: http://www.ismst.com/pdf/abstracts11congress.pdf.
102. Schaden W, Thiele R, Kölpl C, et al. Shock wave therapy for acute and chronic soft tissue wounds: a feasibility study. J Surg Res. 2007;143:1–12.
103. Cleveland R, Chitnis P, McClure SR. Acoustic field of ballistic shock wave therapy device. Ultrasound Med Biol. 2007;33:1327–35.
104. Ueberle F, Delius M, Guo L. Using shock waves for transfer of molecules in cells. Biomed Tech (Berl). 2002;47(Suppl 1 Pt 1):382–5. German.
105. Gollwitzer H, Diehl P, von Korff A, Rahlfs VW, Gerdesmeyer L. Extracorporeal shock wave therapy for chronic painful heel syndrome: a prospective, double blind, randomized trial assessing the efficacy of a new electromagnetic shock wave device. J Foot Ankle Surg. 2007;46:348–57.
106. Mariotto SW, Cavalieri E, Amelio E, et al. Extracorporeal shock waves: from lithotripsy to anti-inflammatory action by NO production. Nitric Oxide. 2005;12:89–96.
107. Wess O. Physik and Technik der Stoss- und Druckwellentherapie. Med Orthop Techn. 2005;5:7–32.
108. Jankovic D. Mit der Stosswellen gegen das Piriformis Syndrom. Schmerztherapie. 2011;1:11–3.
109. Peng P, Narouze S. Ultrasound-guided interventional procedures in pain medicine: a review of anatomy, sonoanatomy, and procedures: part I: nonaxial structures. Reg Anesth Pain Med. 2009;34:458–74.
110. Jankovic D. Infiltration der Triggerpunkte des M. piriformis ("Piriformis Syndrom"). In: Jankovic D, editor. Regionalblockaden & Infiltrattionstherapie. Lehrbuch und Atlas. 4th ed. Berlin: ABW-Verlag; 2008. p. 306–9.
111. Finoff JT, Hurdle MFB, Smith J. Accuracy of ultrasound-guided versus fluoroscopically guided contrast controlled piriformis injections. A cadaveric study. J Ultrasound Med. 2008;27:1157–63.
112. Childers MK, Wilson DJ, Gnatz SM, Conway RR, Sherman AK. Botulinum toxin type A use in piriformis muscle syndrome: a pilot study. Am J Phys Med Rehabil. 2002;81:751–9.
113. Huerto AP, Yeo SN, Ho KY. Piriformis muscle injection using ultrasonography and motor stimulation — report of a technique. Pain Physician. 2007;10:687–90.
114. Peng PWH, Tumber PS. Ultrasound-guided interventional procedures for patients with chronic pelvic pain — a description of techniques and review of the literature. Pain Physician. 2008;11:215–24.
115. Macfarlane AJR, Bhatia A, Brull R. Needle to nerve proximity: what do the animal studies tell us? Reg Anesth Pain Med. 2011;36:290–302.
116. Rigaud M, Filip P, Lirk P, Fuchs A, Gemes G, Hogan Q. Guidance of block needle insertion by electrical nerve stimulation: a pilot study of the resulting distribution of injected solution in dogs. Anesthesiology. 2008;109:473–8.
117. Chan VW, Brull R, McCartney CJ, 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.
118. Tsai TP, Vuckovic I, Dilberovic F, 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.
119. 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.
120. Abram SE. Treatment of lumbosacral radiculopathy with epidural steroids. Anesthesiology. 1999;91:1937–41.
121. Johansson A, Hao J, Sjölund B. Local corticosteroid application blocks transmission in normal nociceptive C-fibres. Acta Anaesthesiol Scand. 1990;34:335–8.
122. Lang AM. Botulinum toxin type B in piriformis syndrome. Am J Phys Med Rehabil. 2004;83:198–202.
123. Indrevekam K, Sudmann E. Piriformis muscle syndrome in 19 patients treated by tenotomy — a 1- to 16-year follow-up study. Int Orthop. 2002;26:101–3.

Chapter 56

Pudendal Nerve Blockade

Geoff Bellingham
Department of Anesthesia and Perioperative Medicine, St. Joseph's Health Care London, University of Western Ontario, London, ON, Canada
e-mail: geoff.Bellingham@sjhc.london.on.ca

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_56, © Springer International Publishing Switzerland 2015

Introduction

The pudendal nerve is a branch of the sacral plexus, originating from the ventral rami of S2, 3, and 4 nerve roots. It provides sensory innervation to the skin of the perineum and mucosa of the anal canal. It also provides motor control of the external anal sphincter, urethral sphincter, and perineal musculature [1–6].

Blockade of the pudendal nerve has been used to facilitate anesthesia and analgesia for surgical procedures involving the perineum. Blockade can also aid in the diagnosis and therapy of pelvic pain syndromes, specifically pudendal neuralgia.

A range of nerve block techniques have been described from landmark-guided approaches to the use of magnetic resonance neurography (MRN).

Pudendal Nerve Anatomy

Once the pudendal nerve is formed from its nerve roots, it briefly exits the pelvis to enter the gluteal region, beneath the piriformis muscle via the infrapiriform notch (Figs. 56.1, 56.2, and 56.3). The nerve will then course between the sacrospinous and sacrotuberous ligaments, adjacent to the ischial spine. It reenters the pelvis through the lesser sciatic foramen to continue its course anteriorly through a fascial tunnel formed along the medial border of the obturator internus muscle known as Alcock's canal. The nerve provides three branches to innervate the perineum along its path that include the inferior rectal nerve, perineal branch, and dorsal branch.

The anatomical course of the pudendal nerve has been the subject of numerous investigations, and studies continue to report novel anatomic variations. These studies challenge the originally held belief of the pudendal nerve as a singular nerve with a consistent pathway through the pelvis. In contrast, this nerve can be quite complex with a number of well-described variations. Knowledge of these variances can aid in the appropriate management of patients requiring intervention through nerve blockade or surgery.

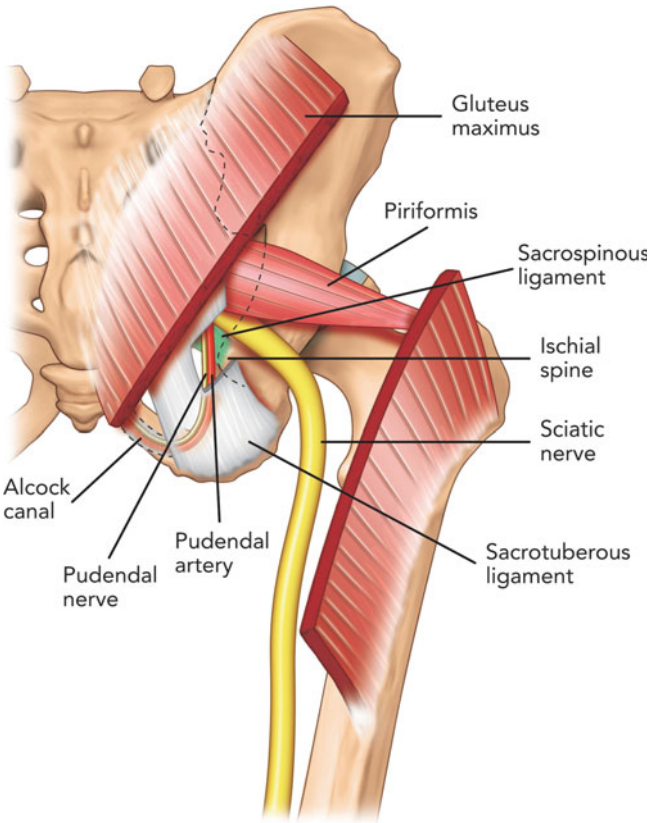


Fig. 56.1 Posterior view of pelvis, showing the piriformis muscle and the neurovascular bundle deep to it. The pudendal nerve and artery run between the sacrospinous and sacrotuberous ligaments (Reproduced with permission from Philip Peng Educational Series)

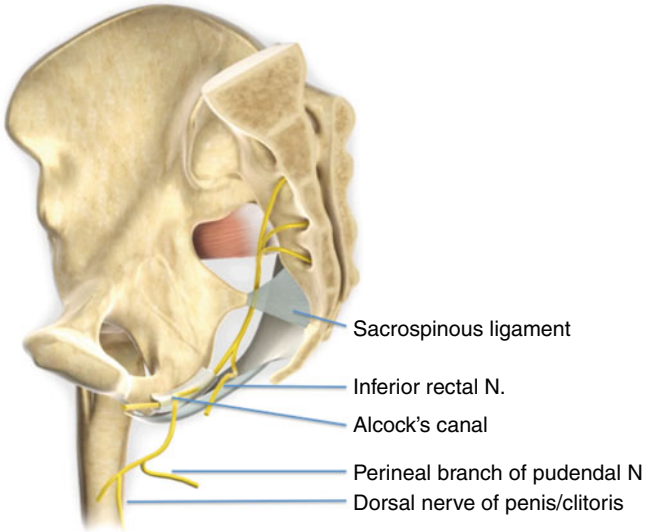


Fig. 56.2 Pudendal nerve is seen arising from S2–S4 and exiting pelvis to enter gluteal region through the greater sciatic foramen. The nerve gives rise to the inferior rectal nerve, perineal nerve, and the dorsal nerve of the penis or clitoris. The inferior rectal nerve branches from the pudendal nerve prior to Alcock's canal. *N* nerve (Reproduced with permission from Philip Peng Educational Series)

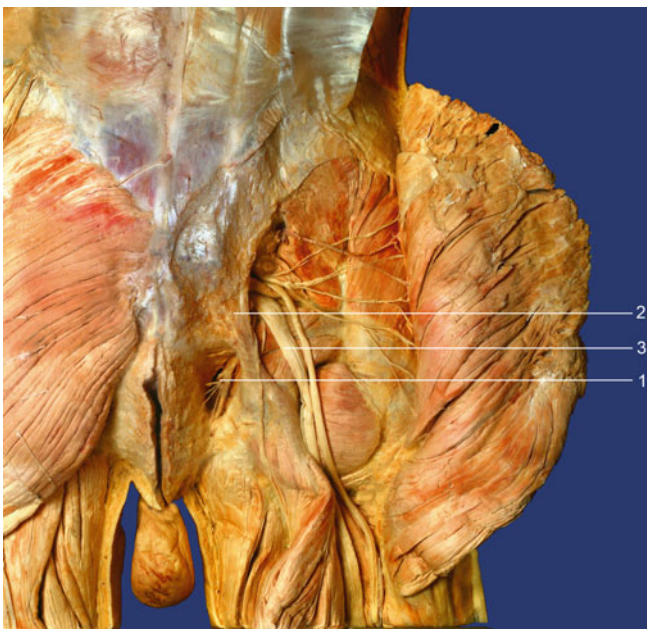


Fig. 56.3 Anatomic specimen. (*1*) Pudendal nerve and pudendal vessels in the ischiorectal fossa, (*2*) sacrotuberous ligament, (*3*) sciatic nerve (With permission from Dr. Danilo Jankovic)

Nerve Roots

The ventral rami of S2, 3, and 4 commonly form the pudendal nerve, yet contributions from S1 and S5 nerve roots have been documented [4, 6–10].

Frequently, the L4 nerve root of the lumbosacral plexus is divided between the lumbar and sacral plexuses, acting as a boundary root. On occasion, the L3 or L5 nerve root may serve as the boundary root, leading to a shift in the nerve roots composition of the plexus involving upper or lower nerve roots or prefixed or postfixed plexus, respectively. These alterations could influence nerve root contributions to pudendal nerve formation. This is exemplified in an anatomic study of 20 adult cadaveric specimens that sought to clarify the nerve root formation of the pudendal nerve in these plexus variations [11].

Of 20 cadaveric specimens, 9 normal lumbosacral plexuses were found, in which the S2 and S3 nerve roots contributed to pudendal nerve formation. In 8 prefixed plexuses, S1 and S2 nerve roots predominated the formation of the nerve, while in the postfixed plexus type, S3 and S4 nerve roots predominated nerve formation. Overall, the S2 nerve root participated in pudendal nerve formation in 17 of 20 cases [11].

A separate anatomical investigation by O'Brichere revealed that the S2 and S4 nerve roots are major contributors to formation of the pudendal nerve [10]. Conversely, Robert et al. discovered that the pudendal nerve is usually formed by S3, with contributions from S2 and 4 [8]. Clearly, variation in pudendal nerve formation is common. This can influence the success of interventions aimed to affect pudendal nerve function at the level of the nerve roots.

Nerve Trunks

The notion that foundational nerve roots consistently combine to form a single pudendal nerve is mistaken. In fact, much of what has been described in anatomical studies reveals that contributing nerve roots may either combine to form a single pudendal nerve or form between two and three "trunks," which may or may not combine to form the pudendal nerve and its terminal branches (inferior rectal, perineal, and dorsal branches) [9].

The frequency of occurrences of trunk types within the gluteal region has been investigated in several anatomical studies [2–4, 9, 10, 12–16]. Although separate research groups propose different classification schemes of the trunks, some generalizations can be drawn. The occurrence of the pudendal nerve as a single common trunk has been reported with the most frequency, occurring between 29 and 96 % of anatomical dissections. The occurrence as two trunks has been reported between 4 and 45.5 % and that of three trunks between 6 and 19.5 % in the literature [3].

Morphology and Anatomical Relationships at the Ischial Spine

A comprehensive knowledge of the nerve's anatomy at the level of the ischial spine is important when performing pudendal nerve blocks. This is a possible site of pudendal nerve entrapment and an accessible area for pudendal nerve blockade through either blind or image-guided approaches.

After exiting the pelvis from the infrapiriform notch, the pudendal nerve crosses over the posterior aspect of either the sacrospinous ligament or the ischial spine. Pirro et al. studied the pudendal nerves of 20 cadaveric specimens and observed that the nerve crosses the sacrospinous ligament in 80 % of cases, while in 15 % of cases it crossed the ischial spine. The remaining specimens contained multi-trunked nerves crossing both the ischial spine and the sacrospinous ligament [3].

The relationship of the pudendal artery to the nerve has also been examined. In 80 % of cases, the pudendal nerve lay medial to the artery, while in 10 % of cases, the nerve lay lateral to the artery. Remaining anatomical relationships observed include having the artery lie between two trunks, and in 7.5 % of cases, the artery crossed the nerve [3]. Knowledge of the pudendal nerve's position relative to the pudendal artery is useful when using ultrasound to guide neural blockade as the nerve itself is typically not easily visualized through a transgluteal approach. Identification of the artery as a pulsatile structure or via Doppler imaging can help clarify the most likely location of the nerve.

The pudendal nerve's mean diameter at the level of the ischial spine has been reported between 2 and 6 mm [3].

Nerve Branches

The pudendal nerve divides into three branches: the inferior rectal branch, the perineal branch, and the dorsal nerve of the penis/clitoris (Fig. 56.4a, b) [17].

The inferior rectal branch descends from its origin in the pelvis to occupy the lower half of the ischiorectal fossa. It further subdivides into cutaneous branches, which supply sensory innervation to the anal canal and the skin around the anus. Sensory innervation may also include the skin of the scrotum. The inferior rectal branch provides the main motor innervation to the external anal sphincter, and investigations have also documented occasional motor innervation to the levator ani muscle as well, through an "accessory rectal nerve" [6, 18]. The inferior rectal branch has been observed to pierce through the sacrospinous ligament as it proceeds to the ischiorectal fossa, which can be a possible site of entrapment [2].

The perineal branch of the pudendal nerve divides into superficial and deep branches. The superficial branch provides sensory innervation through the posterior scrotal/posterior labial nerve, which contributes to the innervation of the posterior aspect of the scrotum or labia majora [9]. This branch may join the sensory branches of inferior rectal branch [17]. The deep branch supplies motor innervation to the muscles of the pelvic floor and the deep perineal pouch. The external urethral sphincter receives voluntary innervation from this branch. Other muscles that receive motor innervation include the transverse perinei, bulbospongiosus, and ischiocavernosus muscles [19, 20]. Motor innervation has also been reported to include the anterior part of the external anal sphincter and the levator ani muscle [20].

The dorsal nerve of the penis or clitoris is the final branch of the pudendal nerve. It supplies the erectile tissue of the corpus cavernosum and of the crus penis/clitoris. This branch supplies sensory innervation to the skin over the dorsal and lateral aspects of the penis and clitoris [9].

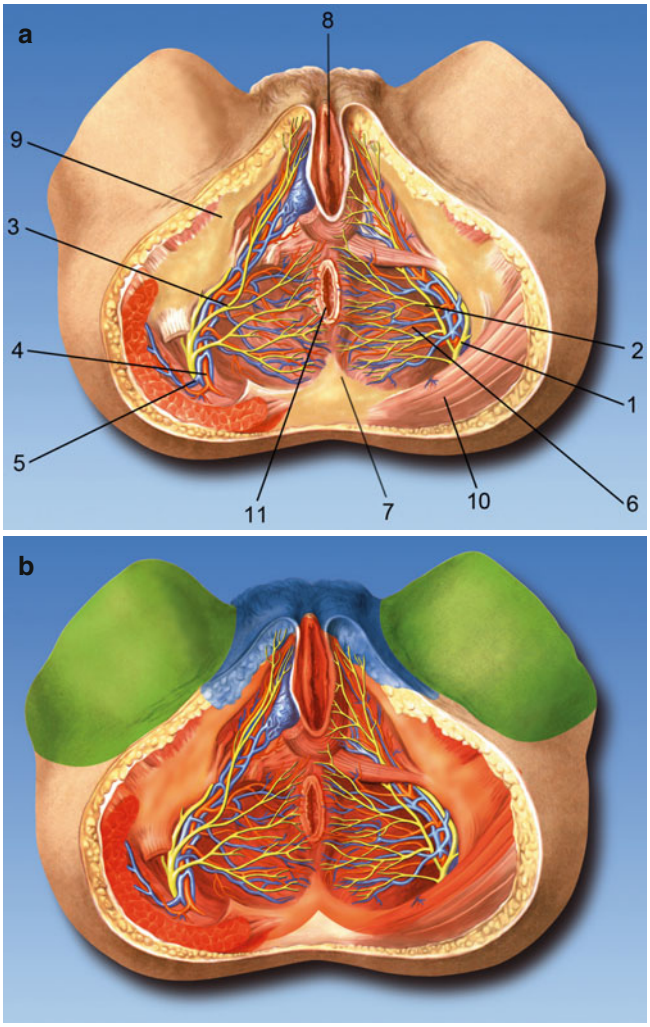


Fig. 56.4 (**a**) Anatomy. (*1*) Pudendal nerve, (*2*) inferior rectal nerves, (*3*) perineal nerves, (*4*) internal pudendal artery, (*5*) internal pudendal veins,(*6*) inferior rectal artery, (*7*) ischiorectal fossa, (*8*) vaginal orifice, (*9*) ischial tuberosity, (*10*) gluteus maximus muscle, (*11*) anus. (**b**) Skin innervations area of the pudendal nerve (*red*), ilioinguinal and genitofemoral nerves (*blue*), and obturator nerve (*green*) (With permission from Dr. Danilo Jankovic)

Alcock's Canal

Distal to the sacrotuberous and sacrospinous ligaments, the pudendal nerve reenters the pelvis through the lesser sciatic foramen and enters Alcock's canal [21]. This canal lies medial to the obturator internus muscle and is formed by a splitting of the muscle's fascia into a medial and lateral layer. The medial layer covers the pudendal neurovascular bundle and fuses below with obturator fascia. The lateral layer is continuous with the obturator fascia [5]. The length of the canal has been measured from 1.4 to 1.8 cm in adults, ending at a distance of 2–3 cm from the inferior border of the symphysis pubis [5]. The canal has been described to lie 2.5–4.5 cm from the inferior border of the ischial tuberosity.

Early anatomical studies report that the pudendal nerve and its branches course together through Alcock's canal and exit by piercing its fascial walls [5, 21]. As described by Shafik et al., the dorsal and perineal branches of the pudendal nerve exit the canal by piercing the obturator fascia at the canal's distal end [5]. The inferior rectal nerve showed some variation in that it either did not enter the canal or entered the canal, but then pierced the proximal aspect of the canal to enter the ischioanal fossa.

A more recent cadaveric study has challenged the previous anatomical descriptions of Alcock's canal. Furtmuller et al. performed dissections of 12 formalin-embedded cadavers, using a medial intrapelvic approach to map the course of the pudendal nerve and its branches [9]. Contrary to the classical description of the exiting pattern of the perineal and dorsal branches, they found that these branches do not exit the canal together at its distal end. Instead, it was discovered that the dorsal branch of the pudendal nerve exited 15–18 mm more anterior along the inferior pubic ramus than the perineal branch did, with its own distinct second exit from the canal. Furthermore, the dorsal nerve branch was found to take a higher course within the pelvis than the perineal branch. Rather than exiting the pelvis at the base of the urogenital diaphragm, the dorsal branch typically exited 12 mm lateral to the pubic symphysis in the parasymphyseal space [9]. This higher course of the dorsal nerve extends this branch in a horizontal fashion from the ischial spine to the parasymphyseal space, traveling separately from the perineal branch in the most superior aspect of Alcock's canal.

Urogenital Diaphragm

The dorsal branch of the pudendal nerve pierces through the superior fascia of the urogenital diaphragm once it reaches the inferior pubic ramus. Beyond this point, the nerve travels in a pouch that is defined by the crus of the penis/clitoris anteriorly and inferiorly. The inferior pubic ramus forms the posterior and superior border. The branch may then pierce either the inferior fascia of the urogenital diaphragm or pierce above the inferior transverse pubic ligament. Once exited the pelvis at this point, the nerve travels anterior to the pubic bone in a groove known as the "sulcus nervi dorsalis penis/clitoris." It then deflects ventrally to innervate the penis or clitoris [9, 22].

Indications

Surgical Anesthesia and Analgesia

There have been a number of investigations evaluating the utility of this block for hemorrhoidectomy, as the postoperative pain of this procedure can be very severe [23–29]. Pudendal nerve blockade has been found to confer substantial benefits for pain control over other types of analgesia, such as neuraxial blocks, general anesthesia, or nonspecific local anesthetic infiltration to the soft tissues of the perineum [23, 24, 26, 29]. Additionally, the use of this block is associated with reduced length of patient stay in hospital, reduced oral analgesic consumption, and improved patient satisfaction over other methods of analgesia [23, 24, 28, 29]. Urinary retention after hemorrhoidectomy is a common and undesirable side effect of anal surgery, as well as with neuraxial anesthetic techniques [30, 31]. The use of pudendal nerve blockade has been shown to significantly reduce this particular postoperative complication [26, 28, 32].

The evaluation of benefits of pudendal nerve blockade has also been investigated for urological procedures such as penile prosthesis surgery [33, 34], hypospadias repair, and circumcision in pediatric population [35, 36], prostate biopsy [37–39], and placement of prostate HDR brachytherapy [40].

The use of pudendal nerve blockade has been described for gynecologic surgical procedures such as placement of suburethral tape and colpoperineorrhaphy [41, 42], yet reports of its use are not as robust as for its use in obstetrical practice. Pudendal nerve blockade, however, has not proven useful to reduce pain after transvaginal pelvic reconstructive surgery [43].

Obstetrical Anesthesia and Analgesia

The use of pudendal nerve blockade during labor has typically been reserved for the second stage of labor. During this stage, pain is experienced in the perineum and becomes somatic, innervated by the S2 to S4 nerve roots and the pudendal nerves.

Pudendal nerve block was likely more commonly used prior to the introduction of epidural anesthesia techniques. However, it can still be employed when neuraxial techniques are contraindicated or if sacral sparring occurs during epidural catheter use. This nerve block has been described for facilitating instrumented deliveries, episiotomies, repair of perineal tears, and McDonald cerclages for incompetent cervices [44–48].

Pudendal Neuralgia

Pudendal neuralgia is an uncommon cause of perineal pain that may result from compression of the nerve or its trunks along its course through the pelvis. Patients will typically describe pain in the perineal area that can include the clitoris, penis, vulva, and perianal area. This may be accompanied by a foreign body sensation within the vagina or rectum. Sitting on a flat surface, which transfers pressure from the soft tissues within the perineum to the pudendal nerve, may exacerbate pain. Conversely, pain may be relieved by standing or sitting on a toilet seat, which relieves that pressure [49].

Diagnosis of this pelvic pain syndrome is challenging, given the lack of a commonly accepted diagnostic test. To assist in identification of the subset of pudendal neuralgia-pudendal entrapment neuropathy (PNE), the Nantes criteria have been proposed which list clinical inclusion and exclusion criteria. Essential criteria include: (1) pain in the anatomical territory of the pudendal nerve, (2) symptoms worsened by sitting, (3) patient is not woken at night by pain, (4) no objective sensory loss on clinical examination, and 5) positive anesthetic pudendal nerve block [50].

Pudendal nerve blockade may satisfy the last essential Nantes criterion if the pain is relieved for the duration of the local anesthetic. However, as described in the original article, a positive diagnostic block may not be specific for pudendal neuralgia, as alternative causes of the perineal pain will be anesthetized if they are situated within the nerve's territory. The typical clinical practice is to inject both local anesthetic and steroid around the nerve. For patient with entrapment neuropathy, the duration of relief will be expected to outlast the effect of local anesthetic.

A negative diagnostic block may not rule out pudendal neuralgia if the nerve is anesthetized distal to the site of entrapment. For example, one may not achieve pain relief through a block performed at Alcock's canal when the site of entrapment is more proximal at the ischial spine [50].

Block Techniques

Transvaginal Technique

The pudendal nerve can be blocked transvaginally through a "blind" technique using the ischial spine as an anatomical landmark (Fig. 56.5). To perform the block, the distal end of an introducer is used to guide the needle toward the pudendal nerve, which allows for infiltrating needles to be advanced 1.0–1.5 cm beyond their distal openings. Introducers described in the literature include the Iowa trumpet or Kobak needle and needle guide [51].

The introducer is first placed against the vaginal mucosa, inferior to the ischial spine. In obstetrical anesthesia literature, the guide is to be held parallel to the delivery table [51]. The needle is advanced into the vaginal mucosa and 1 mL of local anesthetic is infiltrated. The needle is then advanced further until contact is made with the sacrospinous ligament, where another 3 mL of local anesthetic is injected. Care should be taken at this point to first aspirate for blood to help exclude intravascular injection prior to injection as the pudendal vessels will be in close proximity. The needle is then passed through the ligament into the loose areolar tissue posterior to it, where another 3 mL of local anesthetic is deposited prior to aspiration. These steps are then repeated, but with the introducer placed superior to the ischial spine so as to ensure adequate spread around the pudendal nerve [52].

Transperineal Technique

This approach has been described in the literature together with the use of nerve stimulation and has mainly focused on providing analgesia for either perineal surgical procedures or for management of pudendal neuralgia.

The techniques described commonly include stimulation of the pudendal nerve adjacent to the ischial spine to elicit a contraction of the external anal sphincter and perineal muscles. The ischial spine can be localized by palpation of the ischial spine by inserting a finger through the vagina or rectum. Once this anatomical landmark is identified, a needle is guided to this point through the skin overlying the ischiorectal fossa (Fig. 56.6). The skin entry point can vary between descriptions. However, maintaining anal sphincter and perineal muscle contraction while diminishing the stimulating current to 0.5–0.6 mA is typically used to optimize the final needle tip position [25, 27, 53].

It should be noted that anal sphincter contraction alone might not be sufficient for a satisfactory pudendal nerve block, as this may indicate that only the inferior rectal nerve branch is being stimulated. Contraction of the pelvic floor muscles is more desirable as it indicates that the perineal branch is also being stimulated, signifying that the pudendal nerve itself, rather than individual branches, is being contacted.

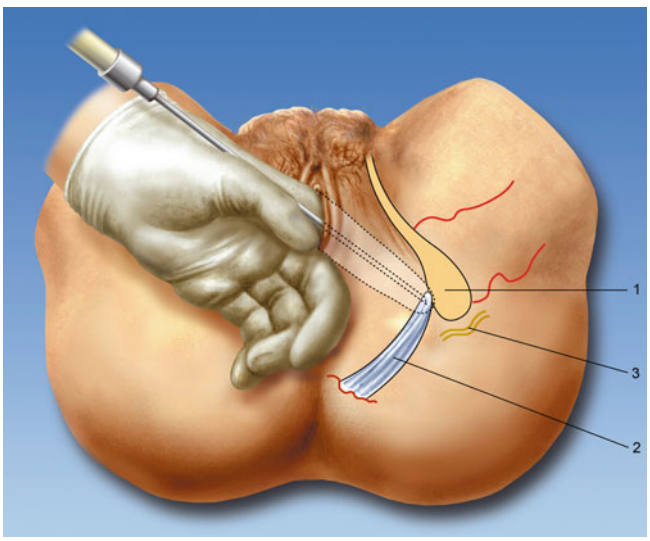


Fig. 56.5 Transvaginal access. (*1*) Ischial spine, (*2*) sacrospinous ligament, (*3*) pudendal nerve (With permission from Dr. Danilo Jankovic)

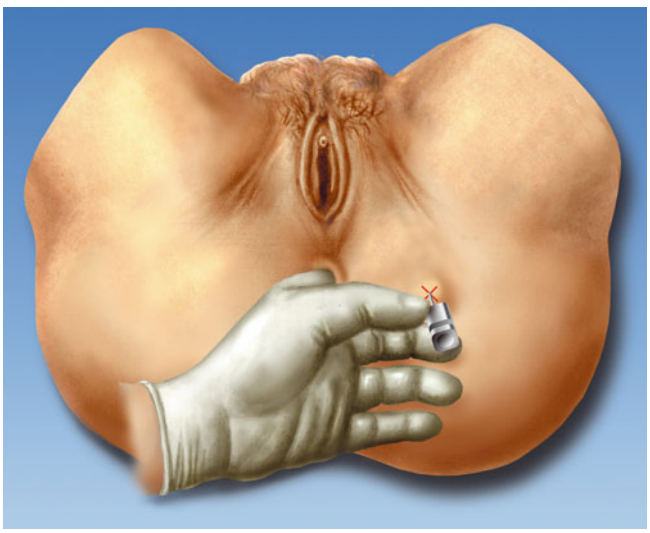


Fig. 56.6 Transperineal access. Rectal palpation of the ischial spine with the index finger. The needle is introduced into the ischiorectal fossa (With permission from Dr. Danilo Jankovic)

Transgluteal Approach

Fluoroscopic Guided

Blockade of the nerve is accomplished by targeting the nerve within the gluteal region as it courses adjacent to the ischial spine (Fig. 56.7).

Patients are placed in a prone position. A fluoroscope is then positioned over the targeted side of blockade to obtain an oblique view 5-20° to the side to be blocked. This view exposes the ischial spine more clearly, avoiding the overlapping with the pelvic brim [54, 55]. Once the ischial spine is identified, a skin entry point on the buttock is marked at the tip of the ischial spine. After skin infiltration with local anesthetic is achieved, a spinal needle can be advanced, coaxial to the fluoroscopic beam, until it contacts the bony surface of the spine. At this juncture, a lateral fluoroscopic view can be obtained to confirm appropriate needle depth and contact with the spine. Once satisfied, 1 mL of contrast medium can be injected to confirm appropriate soft tissue spread [54, 55]. Once complete, injection of the chosen solution can take place.

Contrast spread patterns described include spread in an irregular or round pattern at the tip of the ischial spine. Additionally, spread can occur along the ipsilateral obturator internus muscle, sacrotuberous ligament, or sacrospinous ligament. Investigators have not described any particular correlation between pattern of spread and success of sensory blockade [54].

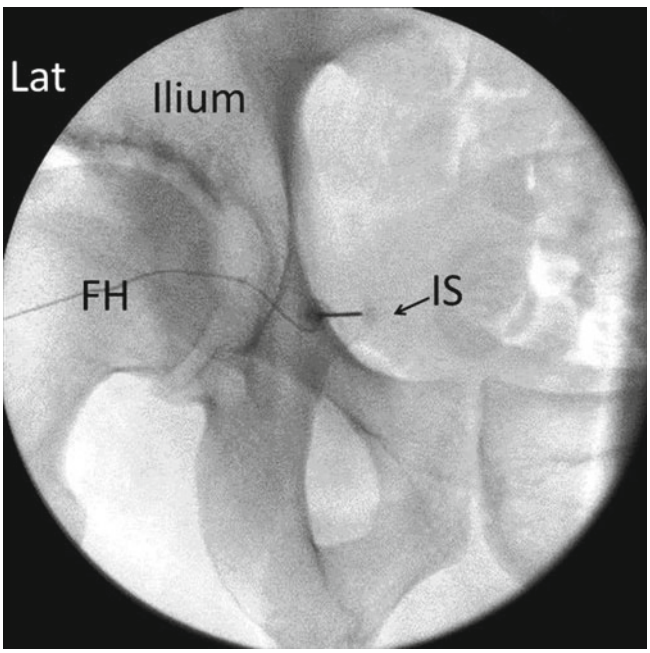


Fig. 56.7 Fluoroscopy guided pudendal nerve block. *FH* femoral head, *Lat* lateral, *IS* tip of the ischial spine (Reproduced with permission from Philip Peng Educational Series)

Ultrasound Guided

The use of an ultrasound-guided approach to block the pudendal nerve has been described in the literature [12, 14, 56, 57]. The use of ultrasound allows for the visualization of soft tissues, needle advancement, and live spread of injectate around the target structures. The target at the level of ischial spine is the interligamentous plane, which is defined by soft tissue, not bony, landmark.

This technique has been validated using sensory change as endpoint [56] and compared against the use of the fluoroscopic guided approach in a randomized trial [57]. The use of ultrasound has proven to be as accurate as the use of fluoroscopy, yet ultrasound guidance requires more procedure time.

Patients are placed in a prone position, and a curvilinear transducer with a low frequency (2–5 MHz) is required because of greater tissue depths. The transducer is first positioned over the ilium at the level of posterior superior iliac spine (PSIS). The ilium appears as a straight, hyperechoic line descending laterally (Figs. 56.8 and 56.9). As scanning continues caudally at the level of greater sciatic notch, the hyperechoic line of the ilium starts to regress from the medial aspect of the screen. The lateral aspect of the ultrasound screen transitions to a curved hyperechoic line revealing the posterior aspect of the acetabulum. At this point, two muscular layers can be identified: the gluteus maximus and the piriformis muscles.

Moving the probe in the caudal direction to the ischial spine reveals four changes in sonographic image: the curved

posterior portion of the acetabulum transitions to the straight ischial spine, disappearance of the piriformis muscle, appearance of a dense hyperechoic line extending medially from the ischial spine, and appearance of the pudendal artery. The most likely location of the pudendal nerve is medial to this artery, and careful scanning may reveal its fascicular structure.

Once the anatomy is identified as best possible, a needle is advanced medial to the probe at a steep angle, using an in-plane approach. Owing to the steep angle of needle advancement, the needle insertion point should be 2 cm away from the probe. Movement of the tissues or spread of injectate may be used to act as surrogate markers for locating the needle tip position.

The needle will eventually pierce through the sacrotuberous ligament, which may provide sturdy resistance to advancement. One may feel a "pop" sensation as the needle passes through. At this juncture, injection can begin under direct ultrasound visualization. Ideally, spread of the injectate is medial to the pudendal artery and is contained between the sacrotuberous and sacrospinous ligaments. If the injectate does not follow this pattern, the needle can be repositioned. There is no investigation on the optimal injectate. The authors preferred plain bupivacaine (to avoid jeopardizing circulation to the entrapped nerve) and steroid, e.g., 4 mL 0.25 % bupivacaine and 40 mg Depo-Medrol®.

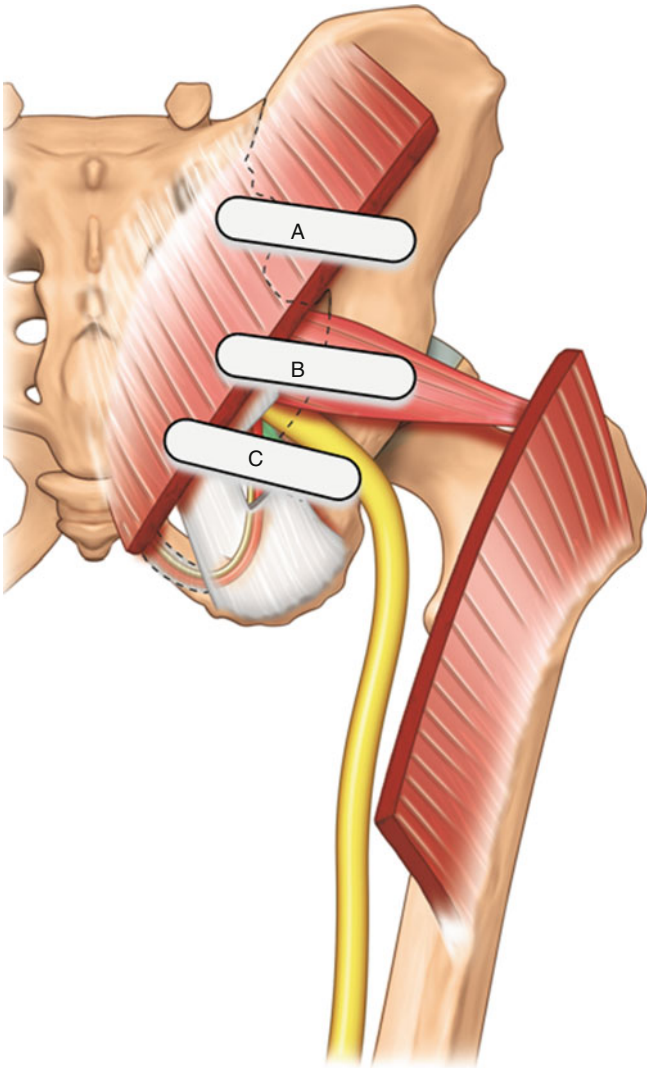


Fig. 56.8 Three positions of the ultrasound probe: (*A*) the ilium at the level of the posterior superior iliac spine, (*B*) at the level of the greater sciatic notch, and (*C*) at the level of the ischial spine (Reproduced with permission from Philip Peng Educational Series)

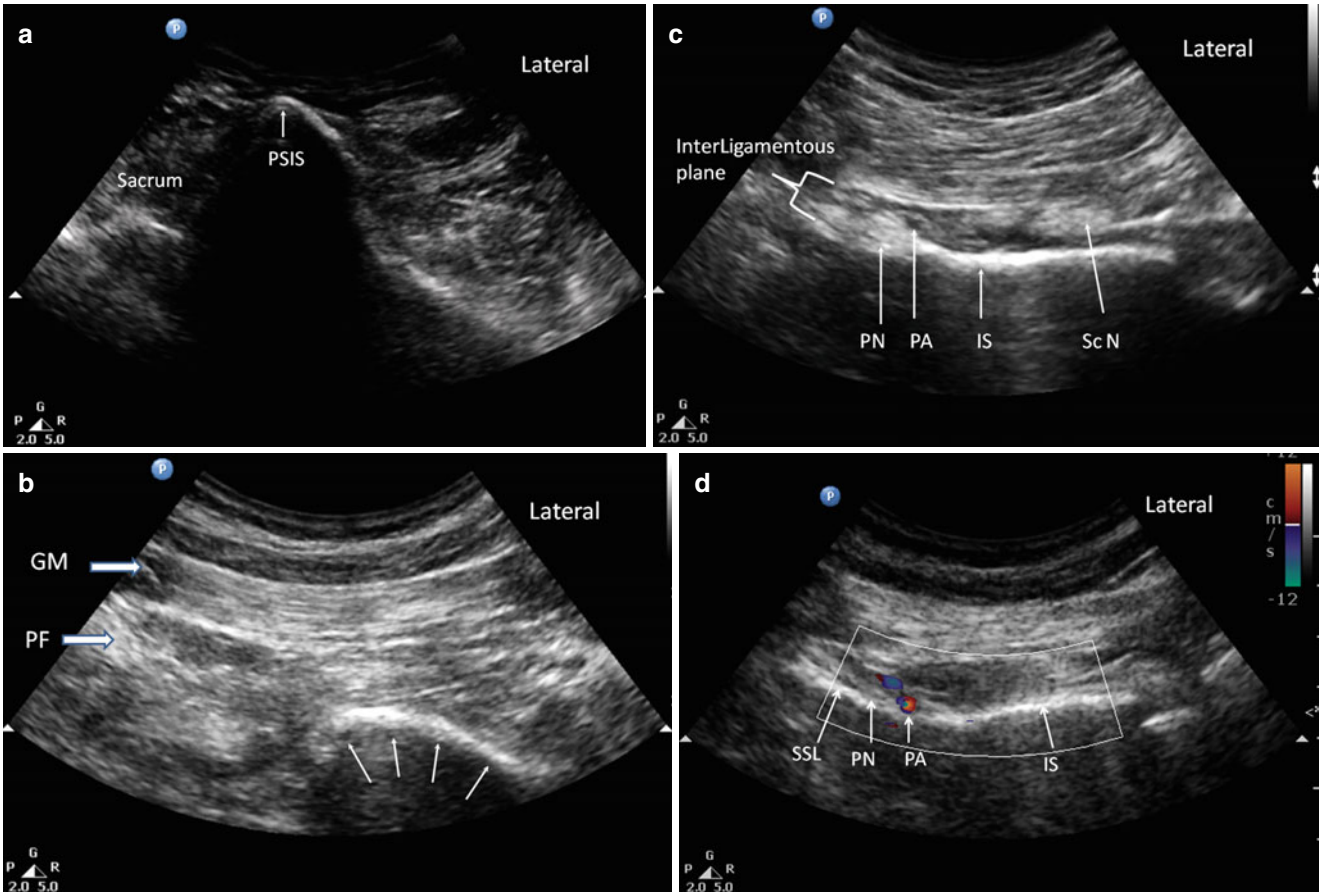


Fig. 56.9 (**a**) Ultrasound image at probe position A. (**b**) Ultrasound image at probe position B. *Line arrows* indicate the ischium, which is curved as it forms the posterior portion of the acetabulum. (**c**) Ultrasound image at probe position C. (**d**) Color-flow Doppler to show pudendal artery. *GM* gluteus maximus, *IS* ischial spine, *PA* pudendal artery, *PF* piriformis muscle, *PN* pudendal nerve, *PSIS* posterior superior iliac spine, *ScN* sciatic nerve, *SSL* sacrospinous ligament (Reprinted with permission from Philip Peng Educational Series)

CT Guided

This technique is more resource intensive compared to previously described methods. However, the advantage in using CT lies in its ability to more clearly define the anatomy of the pelvis. Use of CT scanning can not only block the pudendal nerve at the ischial spine, but also within Alcock's canal which other previously described techniques cannot accomplish owing to the canal's depth within the pelvis.

To perform a block at the level of the ischial spine, patients are placed in a prone position. Scanning can be performed using 2.5–5-mm-thick scans of the pelvis from the acetabular roof to the pubic symphysis. This can provide identification of the ischial spine, sacrotuberous and sacrospinous ligaments, pudendal bundle, and the falciform ligament of the sacrotuberous ligament [58–61]. Needles are passed transgluteally toward the caudal portion of the ischial spine, near the pudendal neurovascular bundle. One may feel the tip of the needle traverse the sacrotuberous ligament. Once the needle tip is in satisfactory position, contrast is injected to confirm appropriate spread. Ideally, contrast will surround the pudendal bundle between the sacrotuberous and sacrospinous ligaments. Once this is confirmed, the pudendal block can be performed [58–61].

To block the pudendal nerve within Alcock's canal, CT scanning is performed at the level of the pubic symphysis, which allows visualization of the obturator internus muscle inferior to the ischial spine. The pudendal neurovascular bundle can be identified on the medial aspect of the obturator internus muscle. A needle is passed in a transgluteal approach and placed in the fatty tissue immediately lateral to the obturator fascia. Contrast is injected to visualize the pudendal nerve at the entrance of Alcock's canal [58–61].

MRI Guided

Pudendal nerve blockade through magnetic resonance neurography (MRN) has been described [63–65]. As with CT-guided techniques, using MRI is resource intensive which limits it use. However, the advantage again lies with excellent tissue resolution to image the target nerve for accurate needle placement and injection. MRI also allows for the visualization of oblique needle paths through the use of multiplanar imaging capabilities [64]. Studies using MRN for pudendal nerve blockade have been limited to the diagnosis and therapy of chronic pelvic pain syndromes.

MRN can be especially useful to block the pudendal nerve within Alcock's canal, if clinically indicated [64]. The sensitivity of MRI at 1.5 T can allow operators to identify if an injection to Alcock's canal occurred inside or outside of the canal. If the injectate were seen to spread medial to the canal, it would be unlikely that the nerve was appropriately anesthetized, as the obturator internus fascia would protect the nerve from any drug effects. As such, the needle can be repositioned to facilitate a successful block.

Equipment and Solutions

Reports describing the use of pudendal nerve blockade have tremendous variability in the types and volumes of injectates used. Needle types are also inconsistent, but typically depend on the type of guidance employed to perform the block (e.g., transvaginal with introducer, ultrasound, or MRN).

Table 56.1 provides a brief survey of needle types and injectates used to perform pudendal nerve blocks according to the approach used.

Table 56.1 Technical details of various approaches of pudendal nerve blockade

Approach	Indication	Needle gauge and type	Injectate
Transvaginal	Obstetrical analgesia	22-gauge, 150-mm needle via tubular introducer	9 mL 1 % lidocaine [52]
	Obstetrical analgesia	Not specified	20 mL 1 % lidocaine [66]
	Obstetrical analgesia	Not specified	20 mL 1 % mepivacaine [67]
	Obstetrical analgesia	Not specified	5 mL 2 % prilocaine [44]
Transperineal	Coloperineorrhaphy	100-mm stimulating needle	10 mL 0.25 % bupivacaine [42]
	Episiotomy analgesia	100-mm stimulating needle	15 mL 0.75 % ropivacaine [47]
	Chronic pelvic pain and anorectal surgery	22-gauge, 100-mm stimulating needle	5 mL 0.25 % bupivacaine [53]
	Transrectal ultrasound guided prostate biopsy	22-gauge spinal needle	10 mL 1 % prilocaine [38]
Transgluteal fluoroscopy	Chronic perineal pain	25-gauge, 3.5″ spinal needle	3 mL 0.38 % ropivacaine and 20 mg triamcinolone [54]
Transgluteal ultrasound	Pudendal neuralgia	22-gauge, 120-mm insulated stimulating needle	5 mL 0.25 % bupivacaine in 1:200,000 epinephrine and 40 mg methylprednisolone [68]
Transgluteal CT	Pudendal neuralgia	22-gauge spinal needle	4 mL 1 % lidocaine and 1 mL triamcinolone [62]
	Pudendal neuralgia	22-gauge spinal needle	1 mL 2 % lidocaine and 1 mL methylprednisolone [59]
	Pudendal neuralgia	22-gauge spinal needle	3 mL 0.25 % bupivacaine and 1 mL of 40 mg/mL methylprednisolone [60]
Transgluteal MRI	Pudendal neuralgia	20-gauge, 100-mm MRI Chiba needle	1.5 mL of 0.25 mL triamcinolone 40 and 1.25 mL 0.5 % bupivacaine [65]

Complications

When performing pudendal nerve blockade for obstetrical anesthesia, the most frequently observed complication reported has been block failure [51]. When used during the second stage of labor, failure rates of the block have ranged from 10 to 50 % in the literature [45]. This may be due to failure of the local anesthetic to reach the nerve or improper timing of the block placement. If the block is placed as the fetal head is crowning, nerve blockade may not be effective in time for an episiotomy and may only be fully established in time for the repair [51].

Other more common complications can include unintended blockade of adjacent nerves. If pudendal nerve blockade is performed at the ischial spine, local anesthetic spread to the sciatic nerve may lead to sensory and motor blockade of the lower limb [57, 60]. Depending on the patient setting, this could lead to delays in ambulation, risk of falls, or delayed discharge from hospital or clinic. A randomized controlled study comparing fluoroscopic and ultrasound-guided pudendal nerve blockade through a transgluteal approach revealed an incidence of sciatic nerve sensory loss in 7/23 fluoroscopic guided procedures and 3/23 ultrasound-guided procedures. Motor weakness in the form of foot drop was noted in two patients for each group [57].

The posterior femoral cutaneous nerve is another nerve in close proximity to the pudendal nerve at the level of the ischial spine that also provides sensory innervation to the perineum. If a pudendal nerve blockade is being used to assist in the diagnosis of a pelvic pain syndrome, local anesthetic spread to the posterior femoral cutaneous nerve may lead to false-positive results [64].

The pudendal nerve provides motor innervation to the urethral sphincter and external anal sphincter, and loss of muscle tone may lead to temporary incontinence of bladder or bowel function. In the study comparing fluoroscopic to ultrasound-guided techniques of pudendal nerve blockade, only one patient of 23 experienced bladder incontinence with bilateral pudendal nerve blockade [57]. Although this may be an infrequent occurrence, patients should be made aware of this possibility.

Practitioners and patients should also be made aware of the uncommon, yet serious complications that are possible through pudendal nerve blockade. When used for labor analgesia, cases of fetal distress and neonatal local anesthetic toxicity have been documented. Presentations of the neonates with local anesthetic toxicity have included hypotonia, apnea, bradycardia, cyanosis, prolonged QT interval, and seizure activity [69, 70]. Factors that may increase the risk of this event include fetal ion trapping in the presence of acidosis and increased local anesthetic vascular uptake from the perineum during labor [69, 70].

Introduction of infection after transvaginal blocks for labor analgesia has been reported due to seeding of bacteria into soft tissues from vaginal mucosa. This can lead to serious morbidity and mortality, with two deaths having been reported in the literature [71, 72]. Abscess formation has been reported posterior to the hip joint, into the gluteal musculature, or the retropsoal space [71]. Of note, authors of these reports have highlighted the risk of delays in diagnosis as the clinical presentation can be initially confused with normal postpartum pain from sacroiliac joint strain or trochanteric bursitis [52, 71, 73].

The formation of significant hematoma after pudendal artery puncture has also been described in the literature in conjunction with infection [52, 73]. After blind infiltration for labor analgesia, an infected retroperitoneal hematoma along the iliac and psoas muscles has been reported, which extended from the midpelvis to the infrarenal fossa [73]. Infection rather than blood loss was the principal concern in this case, however.

References

1. Chan CL, Ponsford S, Scott SM, Swash M, Lunniss PJ. Contribution of the pudendal nerve to sensation of the distal rectum. Br J Surg. 2005;92(7):859–65.
2. Mahakkanukrauh P, Surin P, Vaidhayakarn P. Anatomical study of the pudendal nerve adjacent to the sacrospinous ligament. Clin Anat. 2005;18(3):200–5.
3. Pirro N, Sielezneff I, Le Corroller T, Ouaissi M, Sastre B, Champsaur P. Surgical anatomy of the extrapelvic part of the pudendal nerve and its applications for clinical practice. Surg and Radiol Anat: SRA. 2009;31(10):769–73.
4. Schraffordt SE, Tjandra JJ, Eizenberg N, Dwyer PL. Anatomy of the pudendal nerve and its terminal branches: a cadaver study. ANZ J Surg. 2004;74(1–2):23–6.
5. Shafik A, Doss SH. Pudendal canal: surgical anatomy and clinical implications. Am Surg. 1999;65(2):176–80.
6. Shafik A, el-Sherif M, Youssef A, Olfat ES. Surgical anatomy of the pudendal nerve and its clinical implications. Clin Anat. 1995;8(2):110–5.
7. Montoya TI, Calver L, Carrick KS, Prats J, Corton MM. Anatomic relationships of the pudendal nerve branches. Am J Obstetr Gynecol. 2011;205(5):504.e501–505.
8. Robert R, Prat-Pradal D, Labat JJ, Bensignor M, Raoul S, Rebai R, Leborgne J. Anatomic basis of chronic perineal pain: role of the pudendal nerve. Surg Radiol Anat SRA. 1998;20(2):93–8.
9. Furtmuller GJ, McKenna CA, Ebmer J, Dellon AL. Pudendal nerve 3-dimensional illustration gives insight into surgical approaches. Ann Plastic Surg. 2014;73(6):670–8.
10. O'Bichere A, Green C, Phillips RK. New, simple approach for maximal pudendal nerve exposure: anomalies and prospects for functional reconstruction. Dis Colon Rectum. 2000;43(7):956–60.
11. Matejcik V. Surgical location and anatomical variations of pudendal nerve. ANZ J Surg. 2012;82(12):935–8.
12. Gruber H, Kovacs P, Piegger J, Brenner E. New, simple, ultrasound-guided infiltration of the pudendal nerve: topographic basics. Dis Colon Rectum. 2001;44(9):1376–80.
13. Gustafson KJ, Zelkovic PF, Feng AH, Draper CE, Bodner DR, Grill WM. Fascicular anatomy and surgical access of the human pudendal nerve. World J Urol. 2005;23(6):411–8.
14. Kovacs P, Gruber H, Piegger J, Bodner G. New, simple, ultrasound-guided infiltration of the pudendal nerve: ultrasonographic technique. Dis Colon Rectum. 2001;44(9):1381–5.
15. Olszewski J. Variations of the pudendal nerve in man. Folia Morphol. 1982;41(2):245–52.
16. Sikorski A, Olszewski J, Miekos E. Anatomical considerations of selective pudendal neurectomy. Int Urol Nephrol. 1987;19(2):159–63.
17. Shafik A. Neuronal innervation of urethral and anal sphincters: surgical anatomy and clinical implications. Curr Opin Obstet Gynecol. 2000;12(5):387–98.
18. Grigorescu BA, Lazarou G, Olson TR, Downie SA, Powers K, Greston WM, Mikhail MS. Innervation of the levator ani muscles: description of the nerve branches to the pubococcygeus, iliococcygeus, and puborectalis muscles. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19(1):107–16.
19. Shah AP, Mevcha A, Wilby D, Alatsatianos A, Hardman JC, Jacques S, Wilton JC. Continence and micturition: an anatomical basis. Clin Anat. 2014;18(3):173–7.
20. Bharucha AE. Pelvic floor: anatomy and function. Neurogastroenterol Motil Off J Eur Gastrointest Motil Soc. 2006;18(7):507–19.
21. Colebunders B, Matthew MK, Broer N, Persing JA, Dellon AL. Benjamin Alcock and the pudendal canal. J Reconstr Microsurg. 2011;27(6):349–54.
22. Hruby S, Ebmer J, Dellon AL, Aszmann OC. Anatomy of pudendal nerve at urogenital diaphragm – new critical site for nerve entrapment. Urology. 2005;66(5):949–52.
23. Tepetes K, Symeonidis D, Christodoulidis G, Spyridakis M, Hatzitheofilou K. Pudendal nerve block versus local anesthesia for harmonic scalpel hemorrhoidectomy: a prospective randomized study. Tech Coloproctol. 2010;14 Suppl 1:S1–3.
24. Castellvi J, Sueiras A, Espinosa J, Vallet J, Gil V, Pi F. Ligasure versus diathermy hemorrhoidectomy under spinal anesthesia or pudendal block with ropivacaine: a randomized prospective clinical study with 1-year follow-up. Int J Colorectal Dis. 2009;24(9):1011–8.
25. Naja Z, Ziade MF, Lonnqvist PA. Nerve stimulator guided pudendal nerve block decreases posthemorrhoidectomy pain. Can J Anaesth. 2005;52(1):62–8.
26. Imbelloni LE, Beato L, Beato C, Cordeiro JA, de Souza DD. Bilateral pudendal nerves block for postoperative analgesia with 0.25 % S75:R25 bupivacaine. Pilot study on outpatient hemorrhoidectomy. Rev Bras Anestesiol. 2005;55(6):614–21.
27. Bolandard F, Bazin JE. Nerve stimulator guided pudendal nerve blocks. Can J Anaesth. 2005;52(7):773; author reply 773–4.
28. Gabrielli F, Cioffi U, Chiarelli M, Guttadauro A, De Simone M. Hemorrhoidectomy with posterior perineal block: experience with 400 cases. Dis Colon Rectum. 2000;43(6):809–12.
29. Naja Z, El-Rajab M, Al-Tannir M, Ziade F, Zbibo R, Oweidat M, Lonnqvist PA. Nerve stimulator guided pudendal nerve block versus general anesthesia for hemorrhoidectomy. Can J Anaesth. 2006;53(6):579–85.
30. Petros JG, Bradley TM. Factors influencing postoperative urinary retention in patients undergoing surgery for benign anorectal disease. Am J Surg. 1990;159(4):374–6.
31. Pybus DA, D'Bras BE, Goulding G, Liberman H, Torda TA. Postoperative analgesia for haemorrhoid surgery. Anaesth Intensive Care. 1983;11(1):27–30.
32. Kim J, Lee DS, Jang SM, Shim MC, Jee DL. The effect of pudendal block on voiding after hemorrhoidectomy. Dis Colon Rectum. 2005;48(3):518–23.
33. Dos Reis JM, Glina S, Da Silva MF, Furlan V. Penile prosthesis surgery with the patient under local regional anesthesia. J Urol. 1993;150(4):1179–81.
34. Kaufman JJ. Penile prosthetic surgery under local anesthesia. J Urol. 1982;128(6):1190–1.
35. Naja Z, Al-Tannir MA, Faysal W, Daoud N, Ziade F, El-Rajab M. A comparison of pudendal block vs dorsal penile nerve block for circumcision in children: a randomised controlled trial. Anaesthesia. 2011;66(9):802–7.
36. Naja ZM, Ziade FM, Kamel R, El-Kayali S, Daoud N, El-Rajab MA. The effectiveness of pudendal nerve block versus caudal block anesthesia for hypospadias in children. Anesth Analg. 2013;117(6):1401–7.
37. Adsan O, Inal G, Ozdogan L, Kaygisiz O, Ugurlu O, Cetinkaya M. Unilateral pudendal nerve blockade for relief of all pain during transrectal ultrasound-guided biopsy of the prostate: a randomized, double-blind, placebo-controlled study. Urology. 2004;64(3):528–31.
38. Inal G, Adsan O, Ugurlu O, Kaygisiz O, Kosan M, Cetinkaya M. Comparison of four different anesthesia methods for relief of all pain during transrectal ultrasound-guided prostate biopsy. Int Urol Nephrol. 2008;40(2):335–9.
39. Iremashvili VV, Chepurov AK, Kobaladze KM, Gamidov SI. Periprostatic local anesthesia with pudendal block for transperineal ultrasound-guided prostate biopsy: a randomized trial. Urology. 2010;75(5):1023–7.
40. Schenck M, Schenck C, Rubben H, Stuschke M, Schneider T, Eisenhardt A, Rossi R. Pudendal nerve block in HDR-brachytherapy patients: do we really need general or regional anesthesia? World J Urol. 2013;31(2):417–21.

41. Darcq C, Ayadi M, Dissait V, Piech JJ, Anton-Bousquet MC, Mansoor A. Pudendal nerve block: local anaesthesia for insertion of suburethral tape: preliminary study of 9 cases. Progres en urologie journal de l'Association francaise d'urologie et de la Societe francaise d'urologie. 2005;15(1):49–52; discussion 52.
42. Ismail MT, Elshmaa NS. Pre-emptive analgesia by nerve stimulator guided pudendal nerve block for posterior colpoperineorrhaphy. Eur J Obstet Gynecol Reprod Biol. 2012;163(2):200–3.
43. Abramov Y, Sand PK, Gandhi S, Botros SM, Miller JJ, Koh EK, Goldberg RP. The effect of preemptive pudendal nerve blockade on pain after transvaginal pelvic reconstructive surgery. Obstet Gynecol. 2005;106(4):782–8.
44. Arslan M, Yazici G, Dilek U. Pudendal nerve block for pain relief in episiotomy repair. Int J Gynaecol Obstetr Off Organ Int Fed Gynaecol Obstetr. 2004;87(2):151–2.
45. Novikova N, Cluver C. Local anaesthetic nerve block for pain management in labour. Cochrane Database Syst Rev 2012;(4):CD009200.
46. Volmanen P, Palomaki O, Ahonen J. Alternatives to neuraxial analgesia for labor. Curr Opin Anaesthesiol. 2011;24(3):235–41.
47. Aissaoui Y, Bruyere R, Mustapha H, Bry D, Kamili ND, Miller C. A randomized controlled trial of pudendal nerve block for pain relief after episiotomy. Anesth Analg. 2008;107(2):625–9.
48. McCulloch B, Bergen S, Pielet B, Keller J, Elrad H. McDonald cerclage under pudendal nerve block. Am J Obstet Gynecol. 1993;168(2):499–502.
49. Peng PWH, Antolak SJ, Gordon AS. Pudnedal Neuralgia. In: Goldstein I, Pukall C, Goldstein A (Eds). Female Sexual Pain Disorders: Evaluation and Management. 1st ed. Wiley-Blackwell Publisher, Oxford, UK; 2009:112–8.
50. Labat JJ, Riant T, Robert R, Amarenco G, Lefaucheur JP, Rigaud J. Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (Nantes criteria). NeurourolUrodyn. 2008;27(4):306–10.
51. King JC, Sherline DM. Paracervical and pudendal block. Clin Obstet Gynecol. 1981;24(2):587–95.
52. Cunningham FG, Williams JW. Williams obstetrics. 23rd ed. New York: McGraw-Hill Medical; 2010. p. 444–63.
53. Kim SH, Song SG, Paek OJ, Lee HJ, Park DH, Lee JK. Nerve-stimulator-guided pudendal nerve block by pararectal approach. Colorectal Dis Off J Assoc Coloproctol Great Brit Ireland. 2012;14(5):611–5.
54. Choi SS, Lee PB, Kim YC, Kim HJ, Lee SC. C-arm-guided pudendal nerve block: a new technique. Int J Clin Pract. 2006;60(5):553–6.
55. Prat-Pradal D, Metge L, Gagnard-Landra C, Mares P, Dauzat M, Godlewski G. Anatomical basis of transgluteal pudendal nerve block. Surg R Anat: SRA. 2009;31(4):289–93.
56. Rofaeel A, Peng P, Louis I, Chan V. Feasibility of real-time ultrasound for pudendal nerve block in patients with chronic perineal pain. Reg Anesth Pain Med. 2008;33(2):139–45.
57. Bellingham GA, Bhatia A, Chan CW, Peng PW. Randomized controlled trial comparing pudendal nerve block under ultrasound and fluoroscopic guidance. Reg Anesth Pain Med. 2012;37(3):262–6.
58. Filippiadis DK, Velonakis G, Mazioti A, Alexopoulou E, Malagari A, Brountzos E, Kelekis N, Kelekis A. CT-guided percutaneous infiltration for the treatment of Alcock's neuralgia. Pain Physician. 2011;14(2):211–5.
59. Fanucci E, Manenti G, Ursone A, Fusco N, Mylonakou I, D'Urso S, Simonetti G. Role of interventional radiology in pudendal neuralgia: a description of techniques and review of the literature. Radiol Med. 2009;114(3):425–36.
60. Hough DM, Wittenberg KH, Pawlina W, Maus TP, King BF, Vrtiska TJ, Farrell MA, Antolak Jr SJ. Chronic perineal pain caused by pudendal nerve entrapment: anatomy and CT-guided perineural injection technique. AJR Am J Roentgenol. 2003;181(2):561–7.
61. Thoumas D, Leroi AM, Mauillon J, Muller JM, Benozio M, Denis P, Freger P. Pudendal neuralgia: CT-guided pudendal nerve block technique. Abdom Imaging. 1999;24(3):309–12.
62. Calvillo O, Skaribas IM, Rockett C. Computed tomography-guided pudendal nerve block. A new diagnostic approach to long-term anoperineal pain: a report of two cases. Reg Anesth Pain Med. 2000;25(4):420–3.
63. Filler AG. Diagnosis and treatment of pudendal nerve entrapment syndrome subtypes: imaging, injections, and minimal access surgery. Neurosurg Focus. 2009;26(2):E9.
64. Fritz J, Chhabra A, Wang KC, Carrino JA. Magnetic resonance neurography-guided nerve blocks for the diagnosis and treatment of chronic pelvic pain syndrome. Neuroimaging Clin N Am. 2014;24(1):211–34.
65. Schelhorn J, Habenicht U, Malessa R, Dannenberg C. Magnetic resonance imaging-guided perineural therapy as a treatment option in young adults with pudendal nerve entrapment syndrome. Clin Neuroradiol. 2013;23(2):161–3.
66. Hutchins CJ. Spinal analgesia for instrumental delivery. A comparison with pudendal nerve block. Anaesthesia. 1980;35(4):376–7.
67. Schierup L, Schmidt JF, Torp Jensen A, Rye BA. Pudendal block in vaginal deliveries. Mepivacaine with and without epinephrine. Acta Obstet Gynecol Scand. 1988;67(3):195–7.
68. 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.
69. Bozynski ME, Rubarth LB, Patel JA. Lidocaine toxicity after maternal pudendal anesthesia in a term infant with fetal distress. Am J Perinatol. 1987;4(2):164–6.
70. Pages H, de la Gastine B, Quedru-Aboane J, Guillemin MG, Lelong-Boulouard V, Guillois B. Lidocaine intoxication in newborn following maternal pudendal anesthesia: report of three cases. J Gynecol Obstet Biol Reprod. 2008;37(4):415–8.
71. Wenger DR, Gitchell RG. Severe infections following pudendal block anesthesia: need for orthopaedic awareness. J Bone Joint Surg Am. 1973;55(1):202–7.
72. Svancarek W, Chirino O, Schaefer Jr G, Blythe JG. Retropsoas and subgluteal abscesses following paracervical and pudendal anesthesia. JAMA. 1977;237(9):892–4.
73. Kurzel RB, Au AH, Rooholamini SA. Retroperitoneal hematoma as a complication of pudendal block. Diagnosis made by computed tomography. West J Med. 1996;164(6):523–5.

Chapter 57

Superior Hypogastric Plexus and Ganglion Impar Block

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_57, © Springer International Publishing Switzerland 2015

Anatomy [1–3]

The superior hypogastric plexus (alternatively referred to as plexus hypogastricus and presacral nerve) constitutes the most caudal of the great plexuses of the sympathetic nervous system (Fig. 57.1). The superior hypogastric plexus represents the pelvic extension of the abdominal sympathetic nervous system. Its (preganglionic) cells of origin are located chiefly in the lower thoracic and upper two lumbar levels of intermediolateral column of the spinal cord.

Axonal fibers exit these regions as a white rami communicantes to synapse in the paravertebral lumbar sympathetic chain and preaortic ganglia and plexuses. Postganglionic fibers emerge from these regions and, together with contributions from the parasympathetic sacral ganglia, form the superior hypogastric plexus.

The superior hypogastric plexus is a bilateral retroperitoneal structure situated at the level of the lower third of the L5 vertebral body and the upper third of the S1 vertebral body, at the sacral promontory and close to the bifurcation of the common iliac vessels (Fig. 57.2a, b). The superior hypogastric plexus divides into the right and left hypogastric nerves, which descend lateral to the sigmoid colon and rectosigmoid junction to reach the two inferior hypogastric plexuses. The inferior hypogastric plexus is a bilateral structure situated on either side of the rectum, the lower portion of the bladder, and the prostate and seminal vesicles (in males) or the uterine cervix and vaginal fornices (in females) (Fig. 57.4).

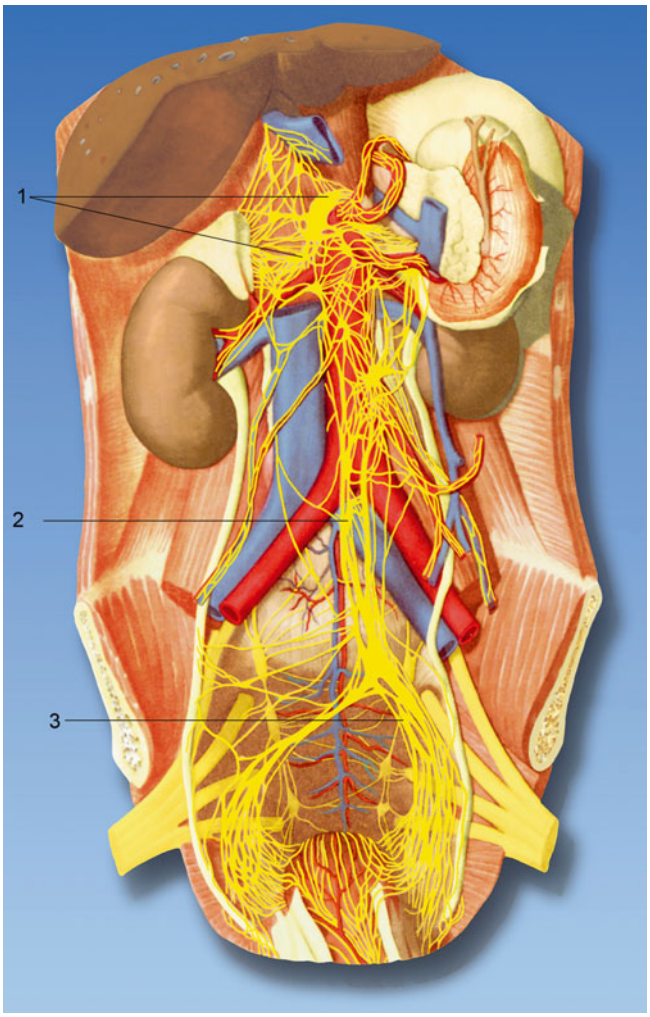


Fig. 57.1 Anatomy. (*1*) Celiac plexus, (*2*) superior hypogastric plexus, (*3*) inferior hypogastric plexus (With permission from Danilo Jankovic)

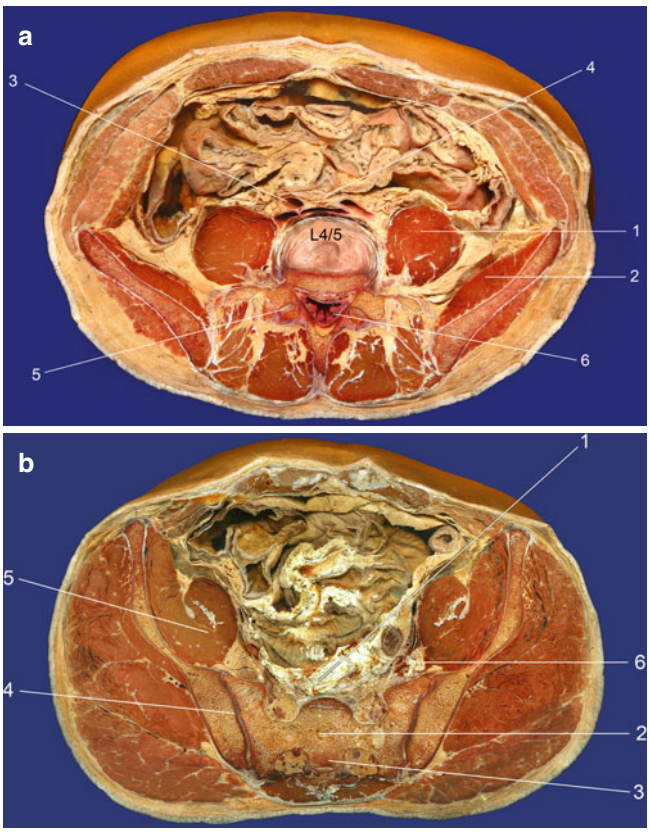


Fig. 57.2 (**a**) Hypogastric plexus. Transverse anatomic section through the spine at L4/5 level revealing the hypogastric plexus, vertebrae, and associated structures. (*1*) Psoas muscle, (*2*) iliacus muscle, (*3*) bifurcation iliac vessels, (*4*) hypogastric plexus, (*5*) articulatio intervertebralis, (*6*) cauda equina (With permission from Danilo Jankovic). (**b**) Transverse anatomic section. (*1*) Hypogastric plexus (*2*) sacrum, (*3*) sacral canal, (*4*) sacroiliac joint, (*5*) iliopsoas muscle, (*6*) lumbar plexus (With permission from Danilo Jankovic)

At the level of the pelvic inlet, the lumbar part of the sympathetic trunk becomes the sacral part of the sympathetic trunk. This lies behind the parietal peritoneum and the rectum in the parietal pelvic fascia and on the ventral surface of the sacrum immediately medial to the sacral foramina, with the vessels and nerves that course through them. The sacral part of the sympathetic trunk consists of three (but sometimes four or five) sacral ganglia, which have connections with the contralateral ganglia. From the two (or three) cranial ganglia emerge (two or three) sacral splanchnic nerves, with efferent (usually postganglionic) and afferent fibers to the inferior hypogastric plexus. The two sacral sympathetic trunks approach closer to each other caudally and join to form the ganglion impar (Walther ganglion), which is located in front of the coccyx (Fig. 57.3a, b). White rami communicantes are absent, but gray rami communicantes with postganglionic sympathetic fibers course from each of the sacral ganglia to the corresponding spinal nerves in the sacrococcygeal region. Along with branches of the sacrococcygeal plexus, these fibers reach vessels, sweat glands, erector muscles of the hair, cross-striated muscles, bones, and joints (Fig. 57.4).

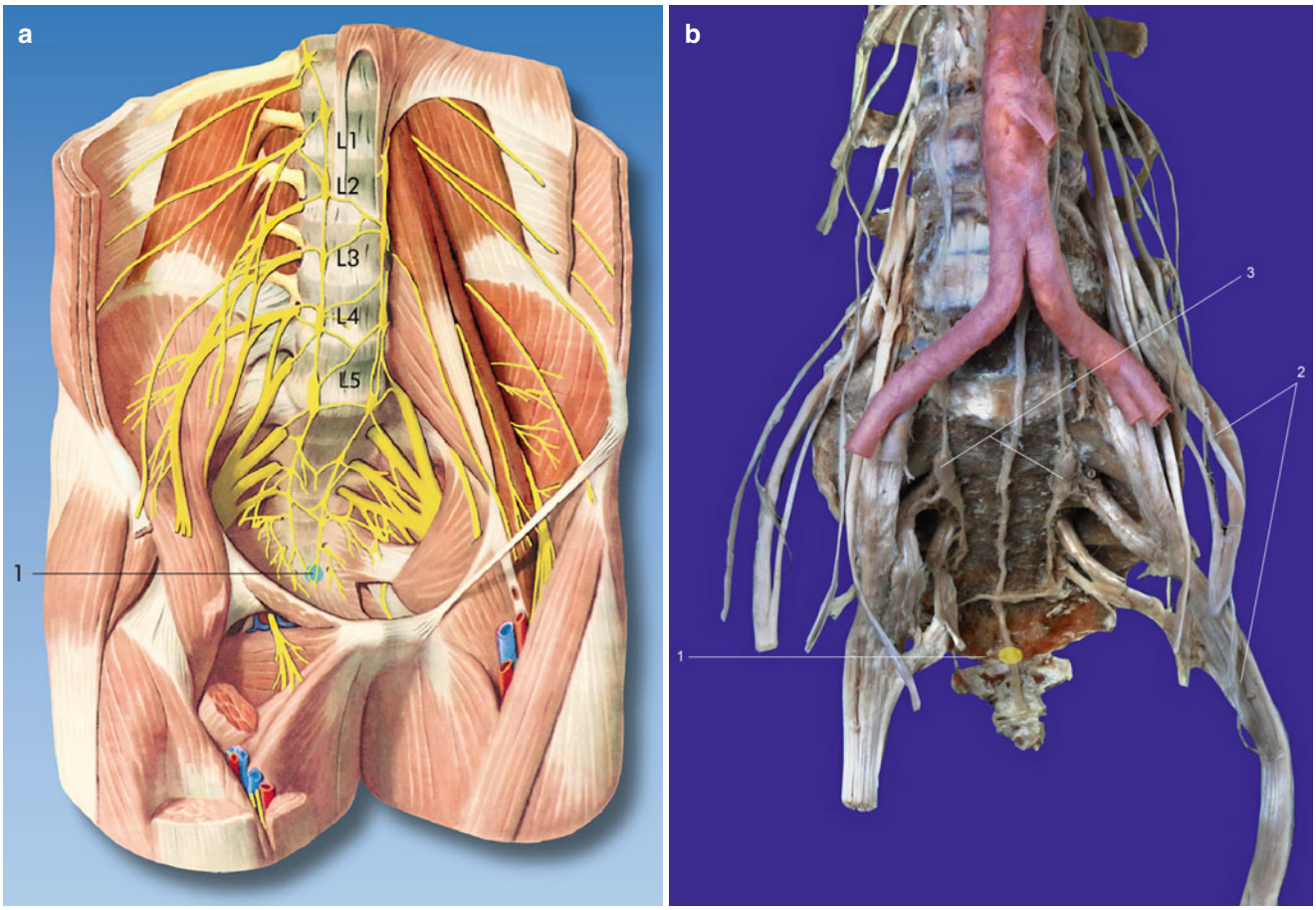


Fig. 57.3 (**a**) Ventral surface of the sacrococcygeal joint. (*1*) Ganglion impar (Walther's ganglion) (With permission from Danilo Jankovic). (**b**) (*1*) Ganglion impar, (*2*) lumbosacral plexus, (*3*) sacral sympathetic trunk (With permission from Danilo Jankovic)

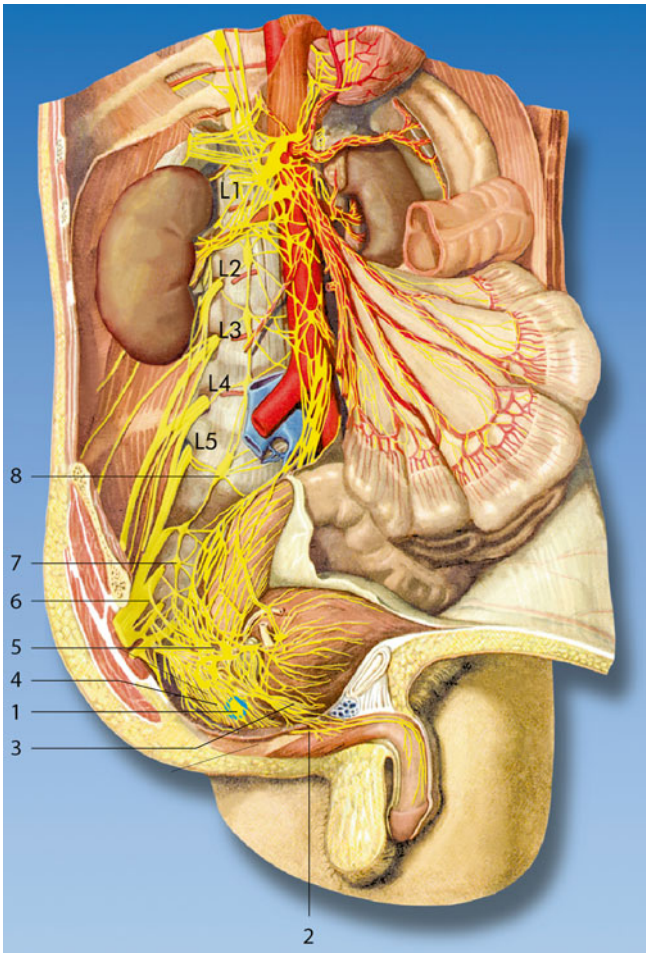


Fig. 57.4 Sacral ganglia and sacral plexus. (*1*) Ganglion impar, (*2*) pudendal nerve, (*3*) prostatic plexus, (*4*) rectal plexus, (*5*) ventral part of the inferior hypogastric plexus, (*6*) sacral plexus, (*7*) ganglion of the sympathetic trunk, (*8*) lumbosacral ganglion of the sympathetic trunk (With permission from Danilo Jankovic)

Superior Hypogastric Plexus Block

Injection of a local anesthetic or neurolytic in the region of the hypogastric plexus (pelvis region), one of the three large sympathetic plexuses.

Indications [4–10]

Diagnostic and Prognostic

1. Pelvic pain that has been unresponsive to more conservative interventions.
2. A diagnostic superior hypogastric plexus block may help distinguish between pelvic pain of visceral and somatic origin because it innervates only the pelvic viscera and vasculature; referred pain emanating from somatic structures will be unaffected.

Therapeutic

1. Neurolysis of the plexus to produce long-lasting pain relief in patients with pelvic cancer (cervical, proximal vaginal, uterine, ovarian testicular, prostatic, and rectal cancers)
2. Pain of oncologic origin (pain and tenesmus due to radiation injury and rectal anastomosis)
3. In patients with distal colonic or rectal inflammatory bowel disease
4. Penile pain after transurethral resection of the prostate
5. Chronic pelvic pain in the presence of endometriosis

Contraindications

1. Anticoagulant treatment
2. Infections and skin diseases in the injection area

Procedure

This block should only be carried out by experienced anesthetists, or under their supervision. Full information should be given to the patient.

Preparations

Check that the emergency equipment is complete and in working order; sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, emergency medication.

Materials

Fine 26-G needle, 25 mm long, for local anesthesia. Atraumatic 22-G needle, 0.7 × 120–140 mm (15°) with injection lead or spinal needle, 0.7 (0.9) × 120–140 mm (150 mm), 20–22-G syringes: 2, 5, and 10 mL. Disinfectant, swabs, compresses, sterile gloves and drape, flat, and firm pillow.

Patient Positioning

Prone position on a radiographic imaging table: support with a pillow in the lower abdomen (to eliminate lumbar lordosis). The patient's arms should be dangling. The patient should breathe with the mouth open, to reduce tension in the back muscles. This position is preferable, particularly when the block is carried out under radiographic control using an image intensifier.

Landmarks (Fig. 57.2a, b)

1. The location of L4–L5 interspace is approximated by palpation of the iliac crests and spinous processes and is then verified by fluoroscopy.
2. Skin wheals are raised 5–7 cm bilateral to the midline at the level of L4–L5 interspace.

Disinfection, generous local anesthetic infiltration of the injection channel, covering with a sterile drape, drawing up the local anesthetic, and testing the patency of the injection needle.

During the injection, the following points must be observed:

1. The person carrying out the injection must stand on the side being blocked.
2. Usually, the injection point should not be located more than 8 cm lateral to the midline and no more than 5 cm medial to it (the lateral side of the vertebra is more difficult to reach).
3. Paresthesias occur relatively frequently during introduction of the needle and indicate irritation of the lumbar somatic nerves.
4. There is a risk of perforating the dural cuff if the injection is made too far medially (epidural or subarachnoid injection).
5. Aspirate frequently and inject incrementally.
6. No neurolytics should be administered without precise confirmation of the needle position using radiographic control with an image intensifier.

Injection Technique [4–6]

1. A short-beveled needle with a depth marker is inserted through one of the skin wheals with the needle bevel directed toward the midline. From a position perpendicular in all planes to the skin, the needle is oriented about 30° caudad and 45° medial, so that its tip is directed toward the anterolateral aspect of the bottom of the L5 vertebral body.

   The iliac crest and the transverse process of L5, which is sometimes enlarged, are potential barriers to needle passage. If the transverse process of L5 is encountered during advancement of the needle, the needle is withdrawn to the subcutaneous tissue and is redirected slightly caudad or cephalad.
2. The needle is readvanced until the body of the L5 vertebra is encountered or until its tip is observed fluoroscopically to lie at its anterolateral aspect, at the depth of approximately 10–12.5 cm. If the vertebral body is encountered, gentle effort may be made to further advance the needle.
3. If this effort is unsuccessful, the needle is withdrawn and, without altering its cephalocaudal orientation, is redirected in a slightly less medial plane so that its tip is "walked off" the vertebral body. The needle tip is advanced about 1 cm past the depth at which contact with the body occurred, at which point a loss of resistance or "pop" may be felt, indicating that the needle tip has traversed the anterior fascial boundary of the ipsilateral psoas muscle and lies in the retroperitoneal space (Figs. 57.5, 57.6, and 57.7).

   A false loss of resistance can occur when the needle is positioned superficially between the psoas muscle and the quadratus lumborum muscle, leading to inadvertent block of the lumbar somatic nerves, with consequent numbness of the lower extremity during the period of effect of the local anesthetic. Injection of a neurolytic into this area without prior radiographic control of the position can have fatal consequences.
4. The contralateral needle is inserted in a similar manner, using the trajectory and the depth of the first needle as a rough guide.

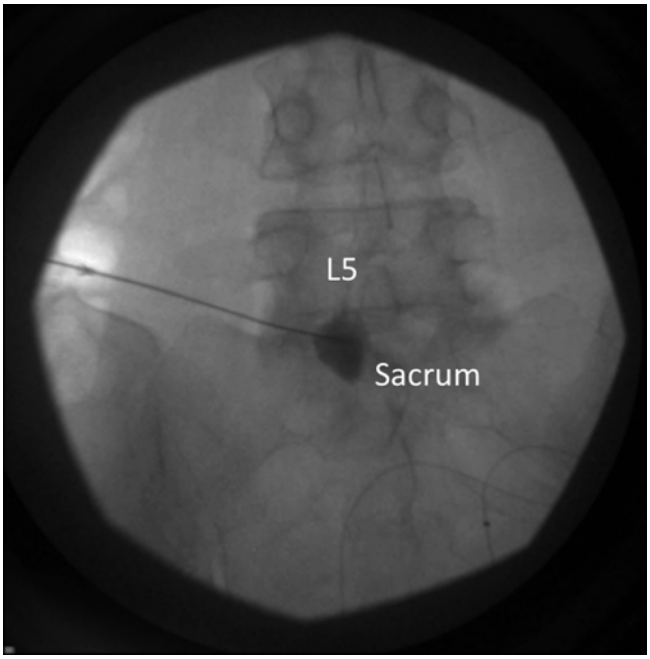


Fig. 57.5 Anteroposterior radiograph of the lumbosacral junction. The needle was seen inserted to the anterior aspect of the lumbosacral junction with contrast confirmation (With permission from Philip Peng)

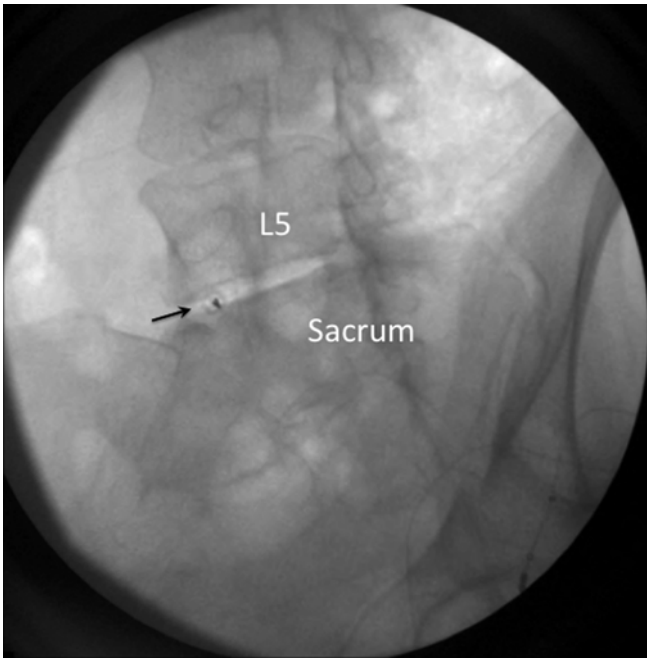


Fig. 57.7 Oblique radiograph of the lumbosacral junction. The spinal needle (indicated by the *arrow*) was inserted with transdiscal technique evident with the end-on view. Thus, only one needle inserted was required as this technique allowed the needle to reach the anterior aspect of lumbosacral junction (With permission from Philip Peng)

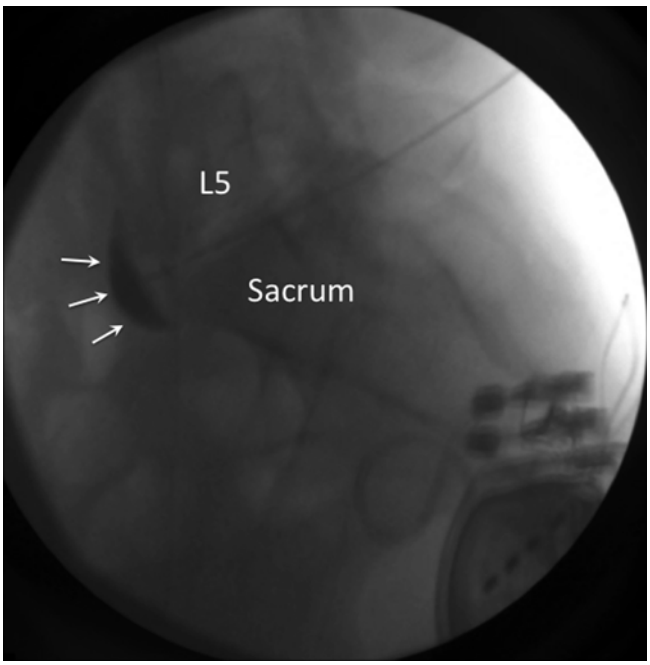


Fig. 57.6 Lateral radiograph of the lumbosacral junction. The contrast was outlined by the line *arrows* (With permission from Philip Peng)

Confirming the Correct Needle Position

1. Biplanar fluoroscopy is used during needle passage and to verify needle placement. Anteroposterior views should demonstrate the needle tips' locations at the level near the junction of the L5 and S1 vertebral bodies, and lateral views confirm placement of the needle tip just beyond the vertebral body's anterolateral margin (Figs. 57.5, 57.6, and 57.7).
2. The injection of 3–4 mL of water-soluble contrast medium through each needle is recommended to further verify accuracy of placement and an absence of spread in the vertebral column.
3. After careful aspiration testing at all levels, resistance-free incremental injection of the local anesthetic is carried out.

Intravascular injection, with a risk of subsequent hemorrhage and hematoma formation, is possible because of the proximity of the bifurcation of the common iliac vessels.

Alternative Modified Approaches

- Coaxial fluoroscopic imaging technique ("coaxial view," "tunnel view," "bull's-eye view" [11]
- Single-needle posterior approach with computed tomography guidance [12]
- Transdiscal approach with fluoroscopic guidance [13, 14] (Fig. 57.7)

Dosage [6–9]

Diagnostic

5–8 mL of local anesthetic with contrast medium through each needle—e.g., 0.5 prilocaine, 0.5 % mepivacaine, and 0.5 % lidocaine

Therapeutic

10 mL local anesthetic per needle (in the two-needle technique)—e.g., 0.2–0.375 % ropivacaine, and 0.25 % bupivacaine (0.25 % levobupivacaine)

Neurolytics

6–8 mL of 10 % aqueous phenol injected through each needle

Complications

Severe

1. Intravascular injection (proximity of the bifurcation of the common iliac vessels) with toxic reactions with local anesthetic or phenol toxicity or hematoma formation
2. Epidural or subarachnoid injection (less likely)

Potential

1. Retroperitoneal hemorrhage
2. Hemorrhage in the psoas area (with subsequent pain in the thigh and transient weakness in the quadriceps muscle)
3. Somatic nerve injury
4. Intraosseous or psoas injection
5. Visceral puncture (renal or ureteral injury accompanied by hematuria)
6. Bowel and uterus puncture
7. Back pain
8. Perforation of an intervertebral disc

Complications of Neurolytic Block

1. Injury of the lumbar somatic nerves
2. Ureteral stricture

Superior Hypogastric Plexus Block

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: *G* _____ *Length* _____ *cm*

i. v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs):* ☐ *Checked*

Patient: ☐ *Informed (what to do after block)*

Position: ☐ *Prone* ☐ *Lateral recumbent*

Injection level: ☐ *L* _____

Injection technique: ☐ *CT-guided* ☐ *X-ray image intensifier*

☐ *Loss of resistance*

Injection:

Local anesthetic: _____ *mL* _____ *%*

(in incremental doses)

Addition to LA: ☐ Yes _____ µg/mg ☐ No _____

Neurolytic: _____ *mL* _____ *%*

Addition: ☐ Yes ☐ No

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement right* _____ *°C left* _____ *°C*

Segments affected: L _____

Monitoring after block: ☐ < 1 h ☐ > 1 h

Time of discharge: _____

Motor/sensory status checked: _____

Complications:

☐ *None* ☐ *Intravascular injection*

☐ *Subarachnoid/epidural* ☐ *Drop in BP*

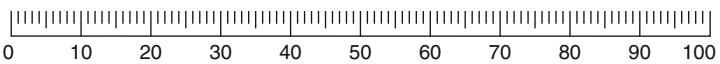
☐ *Other*

Subjective effect of the block : Duration: _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

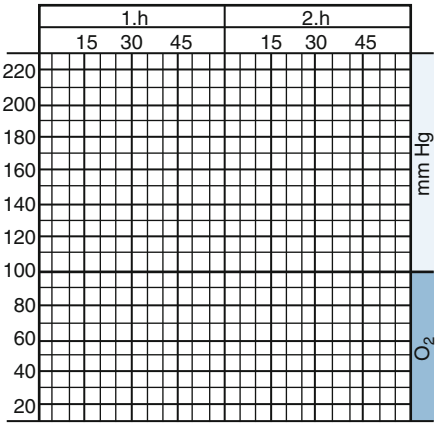
VISUAL ANALOG SCALE



Special notes:

With permission from Danilo Jankovic

Record and checklist



Ganglion Impar (Walther Ganglion) Block

Injection of a local anesthetic or neurolytic into the region of the most inferior (unpaired) ganglion of the sympathetic trunk, on the ventral side of the sacrococcygeal joint

Indications [15–17]

Diagnostic

1. Differential diagnosis of pain in the anorectal and perineal region
   Prognostic
2. In cancer pain, before a neurolytic block

Therapeutic

1. Pain conditions in the anorectal and perineal region
2. Injection of neurolytic agents as a palliative measure in malignant processes (rectum, cervix of the uterus, perineum, bladder, endometrium)

Contraindications

1. Anticoagulant treatment
2. Infections and skin diseases in the injection area
3. Advanced rectal carcinoma (causing blockage of the access route to the ganglion impar)

Procedure

This block should be carried out by experienced anesthetists. Full information for the patient is mandatory. Neurolytic agents should never be administered without precise localization of the needle position using radiographic guidance with an image intensifier. The method of choice is CT-guided injection.

Preparations

Check that the emergency equipment is complete and in working order; sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, emergency medication.

Materials

A fine 26-G needle for local anesthesia. A stable 70–80-mm-long 20–22-G needle. The needle shaft curves in a sickle shape (Fig. 57.8), so that the needle can pass atraumatically to the ventral surface of the sacrococcygeal joint. For intradiscal approach: 25-G spinal (Whitacre) needle with introducer.

Fig. 57.8 70–80-mm-long 20–22-G needle. The needle shaft is bent to form a sickle shape (With permission from Danilo Jankovic)

Patient Positioning

Prone or alternatively lateral recumbent

Localization

Palpation of the tip of the coccyx or sacrococcygeal joint. Disinfection, generous local anesthetic infiltration of the needle channel, covering with a sterile drape, drawing up the local anesthetic. The patency of the previously bent injection needle should be checked.

Injection Technique [15–18]

1. Approximately 3 cm laterally from the median sacral crest, thorough local anesthesia of the needle channel to a depth of ca. 3–4 cm is administered. After the onset of the local anesthetic effect, the sickle-shaped injection needle is introduced in a slightly cranial direction, toward the ventral surface of the sacrococcygeal joint (Fig. 57.9a, b).
2. Confirming the correct needle position. Fluoroscopy is used during needle passage and to verify needle placement. Retroperitoneal location of the needle is verified by observation of the spread of 2 mL of water-soluble contrast medium, which typically assumes a smooth-margined configuration resembling an apostrophe in the lateral plane (Fig. 57.10).
3. After an intravascular position of the needle (aspiration of blood) has been excluded, as well as an intrarectal position (aspiration of air), and after aspiration, incremental injection of the local anesthetic is carried out.

Transdiscal approach.

The procedure will be performed under sterile conditions with fluoroscopic guidance. The patient lies in the prone position with a pillow beneath the iliac crest.

The needle is inserted via the sacrococcygeal disc space. The puncture needle is initiated by the introducer needle through the skin, and then a 25-G spinal (Whitacre) needle is inserted through the introducer to minimize the risk of discitis. Retroperitoneal positioning of the needle is verified by observation of the spread of 2 mL of water-soluble contrast medium (Fig. 57.11).

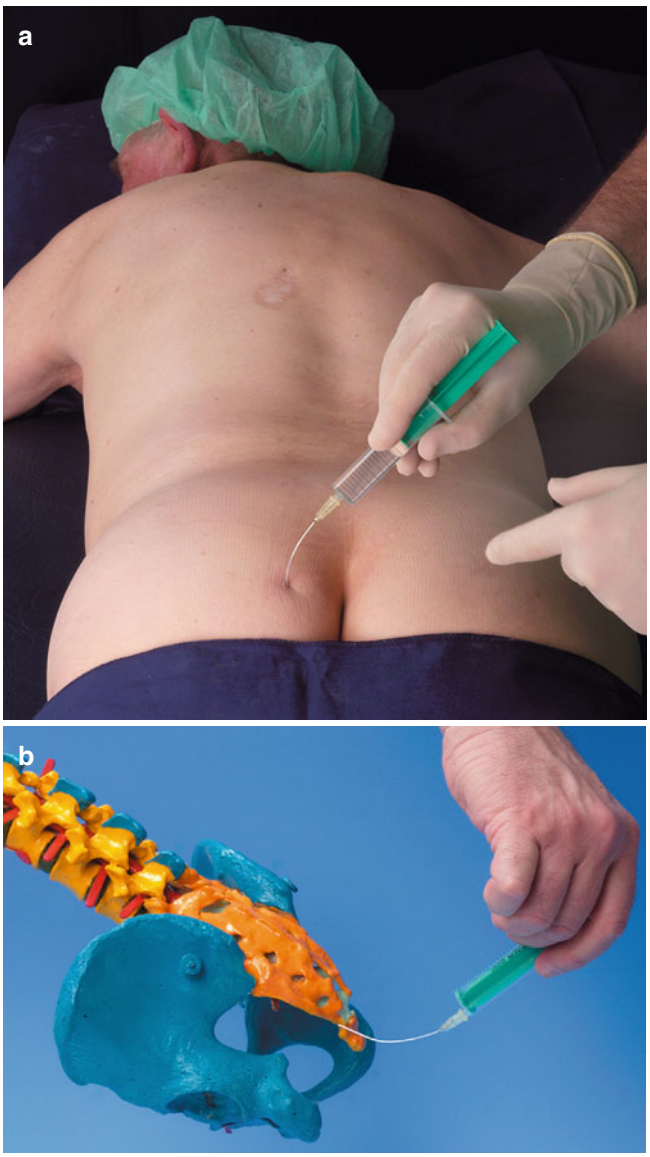


Fig. 57.9 (**a**) Introduction of the sickle-shaped curved injection needle in a slightly cranial direction toward the ventral surface of the sacrococcygeal joint (With permission from Danilo Jankovic). (**b**) In the skeleton (With permission from Danilo Jankovic)

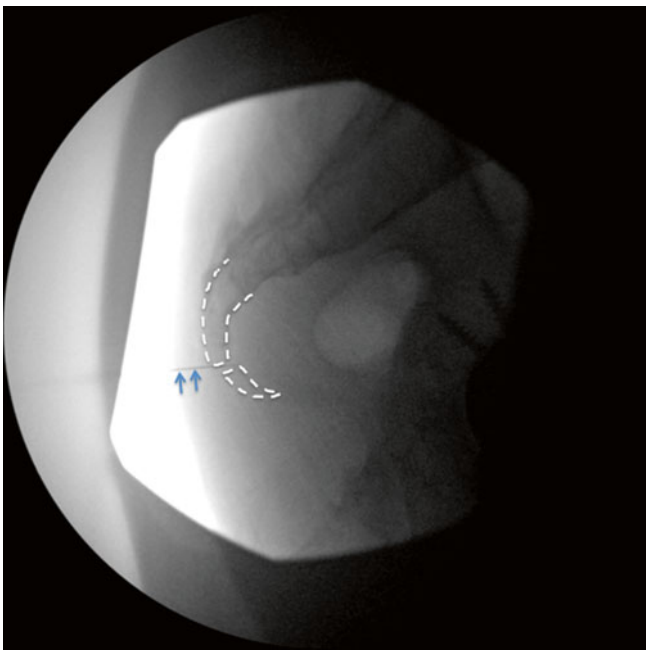


Fig. 57.10 Lateral radiograph of sacrum and the sacrococcygeal junction. The sacrum and coccyx were outlined by *dotted* line, and the needle was indicated by line *arrows* (With permission from Philip Peng)

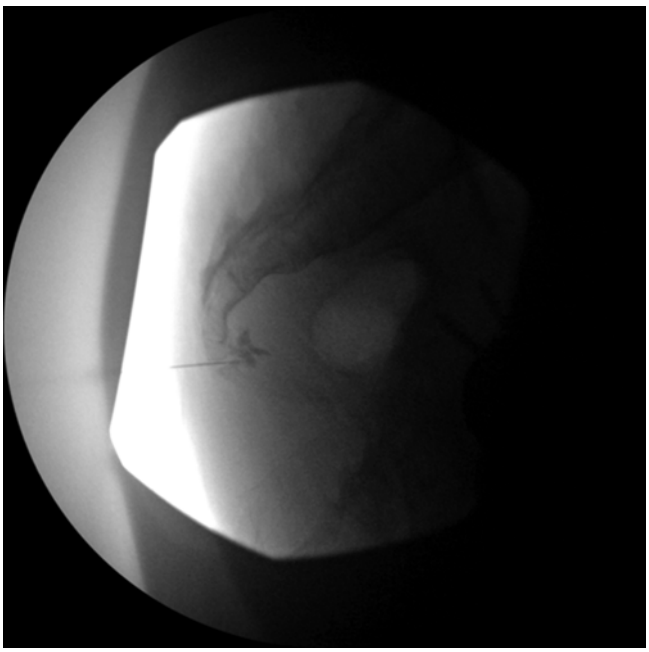


Fig. 57.11 Ganglion impar injection. The needle was inserted via the sacrococcygeal disc space. The puncture needle was initiated by the introducer needle through the skin, and then a 25-G spinal (Whitacre) needle was inserted through the introduced needle to minimize the risk of discitis. The needle position was further confirmed, with contrast (With permission from Philip Peng)

Dosage [15–18]

Diagnostic

5–10 mL local anesthetic—e.g., 1 % prilocaine or 1 % mepivacaine

Therapeutic

1. Local anesthetics: 5–10 mL local anesthetic—e.g., ropivacaine 0.2–0.375 % mixed with 40 mg triamcinolone
2. Neurolytics: 4–6 mL 10 % phenol

Complications

1. Intravascular injection
2. Rectal perforation
3. Subperiosteal position (becomes evident due to high resistance and severe pain during the injection)
4. Needle breakage (strong needles should be used)
5. Epidural/subarachnoidal
6. Infection (sterile precautions)

Ganglion impar block

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes _____

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Diagnostic* ☐ *Therapeutic*

Needle: *G* _____ *Length* _____ *cm, curved*

i. v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment (*drugs):* ☐ *Checked*

Patient: ☐ *Informed*

Contraindications excluded: ☐ _____

Position: ☐ *Prone* ☐ *Lateral recumbent*

Injection level: ☐ *Sacrococcygeal joint*

Injection technique: ☐ *X-ray image intensifier guidance* ☐ *CT-guided*

☐ *Ultrasound guided* ☐ *Transducer* ☐ *Linear* ☐ *Curved*

☐ *Transanococcygeal* ☐ *Other*

Injection:

Local anesthetic: _____ *mL* _____ *%*

(in incremental doses)

Addition to LA: ☐ Yes _____ ☐ No _____

Neurolytic: _____ *mL* _____ *%*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement before* _____ °*C after* _____ °*C*

Monitoring after block: ☐ <1 h ☐ >1 h

Time of discharge: _____

Complications:

☐ *None* ☐ *Intravascular injection*

☐ *Subperiosteal position* ☐ *Rectal perforation*

☐ *Infection* ☐ *Coccygeal pain*

Subjective effect of the block : Duration: _____

☐ *None* ☐ *Increased pain*

☐ *Reduced pain* ☐ *Relief of pain*

☐ *Visual analog scale*

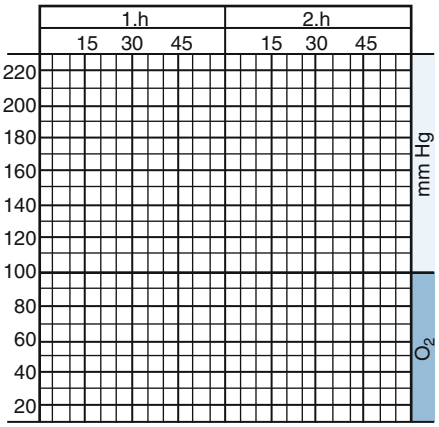
Special notes:

_____

_____

With permission from Danilo Jankovic

Record and checklist



References

1. Pütz R, Pabst R, editors. Sobota-Atlas der Anatomie des Menschen. 21th edition, Elsevier, Urban & Fischer. München, Wien, Baltimore; 2004.
2. Kahle W. Nervensystem und sinnesorgane. In: Taschenatlas der Anatomie. 7. vollständig überarbeitete Auflage. Stuttgart/New York: Georg Thieme Verlag; 2001.
3. Netter FH. Nervengeflechte und periphere Nerven. In: Krämer G (Hrsg.). Farbatlanten der Medizin (Band 5). Nervensystem I. Neuroanatomie und Physiologie. Stuttgart/New York: Georg Thieme Verlag; 1987.
4. Patt R, Plancarte R. Superior hypogastric plexus block: a new therapeutic approach for pelvic pain. In: Waldmann SD, editor. Interventional pain management. Philadelphia: WB. Saunders. Philadelphia, London, New York; 2001. p. 528–34.
5. Brown DL. Superior hypogastric plexus block. Sounders-Elsevier, Philadelphia, Pensylvania 19103; 2006. p. 325–31.
6. Kopacz DJ, Thompson GE. Celiac and hypogastric plexus. In: Cousins MJ, Bridenbaugh PO, editors. Neural blockade in clinical anesthesia and management of pain. Philadelphia: Lippincott-Raven; 1998. p. 470–2.
7. De Leon-Casasola O, Kent E, Lema MJ. Neurolytic superior hypogastric plexus block for chronic pelvic pain associated with cancer. Pain. 1993;54:145–51.
8. Plancarte R, Amescua C, Patt RB, Aldrete JA. Superior hypogastric plexus block for pelvic cancer pain. Anesthesiology. 1990;73:236.
9. Rosenberg SK, Tewari R, Boswell MV, Thompson GA, Seftel AD. Superior hypogastric plexus block successfully treats severe penile pain after transurethral resection of the prostate. Reg Anesth Pain Med. 1998;23:618–20.
10. Wechsler R, Maurer PM, Halpern FJ, Frank FD. Superior hypogastric plexus block for chronic pelvic pain in the presence of endometriosis: CT techniques and results. Radiology. 1995;196:103–6.
11. Stevens DS, Balatbat GR, Lee FMK. Coaxial imaging technique for superior hypogastric block. Reg Anesth Pain Med. 2000;25: 643–7.
12. Waldmann S, Wilson W, Kreps R. Superior hypogastric plexus block using a single needle and computed tomography guidance: description of a modified technique. Reg Anesth. 1991;16: 286–7.
13. Erdine S, Yucel A, Celik M, Talu GK. Transdiscal approach for hypogastric plexus block. Reg Anesth Pain Med. 2003;28:304–8.
14. Ina H, Kitoh TA. A new approach to superior hypogastric plexus block: Transvertebral disc L5-S1 technique. Reg Anesth. 1992; 17(Suppl):123 (abstr).
15. Patt RB, Plancarte R. Neurolytic blocks of the sympathetic axis. In: Patt RB, editor. Cancer pain. Philadelphia: JB. Lippincott; 1993.
16. Patt RB, Plancarte R. Superior hypogastric plexus and ganglion impar. In: Hahn MB, McQuillan PM, Sheplock GJ, editors. Regional anesthesia. St. Louis: Mosby; 1996.
17. Plancarte R, Amescua C, Patt RB, et al. Presacral blockade of Walther (ganglion impar). Anesthesiology. 1990;73:A 751.
18. Nebab EG, Florence IM. An alternative needle geometry for interruption of the ganglion impar. Anesthesiology. 1997;86:1213–4.

Chapter 58

Paracervical (Uterosacral) Block

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_58, © Springer International Publishing Switzerland 2015

Before the established popularity of epidural anesthesia for labor and delivery, paracervical block was one of the most frequently used regional techniques administered for analgesia during the first stage of labor (see Chap. 42, Neuraxial anesthesia in obstetrics).

Anatomy

Pain from the uterus passes in sequence through uterine plexus, the pelvic ganglia and plexus, the hypogastric nerve, the superior hypogastric plexus, the lumbar and lower thoracic sympathetic chain, and the white rami communicantes, associated with the eleventh and twelfth nerves. Then, the pain stimuli pass through the posterior roots of these spinal nerves (T11 and T12) to enter the spinal cord (Figs. 58.1 and 58.2). Frankenhauser's ganglion, which contents all the visceral sensory nerve fibers from the uterus, cervix, and upper vagina, is located in the lateral fornix of the upper vaginal canal. It is that ganglion or plexus of nerves that is blocked by a paracervical block.

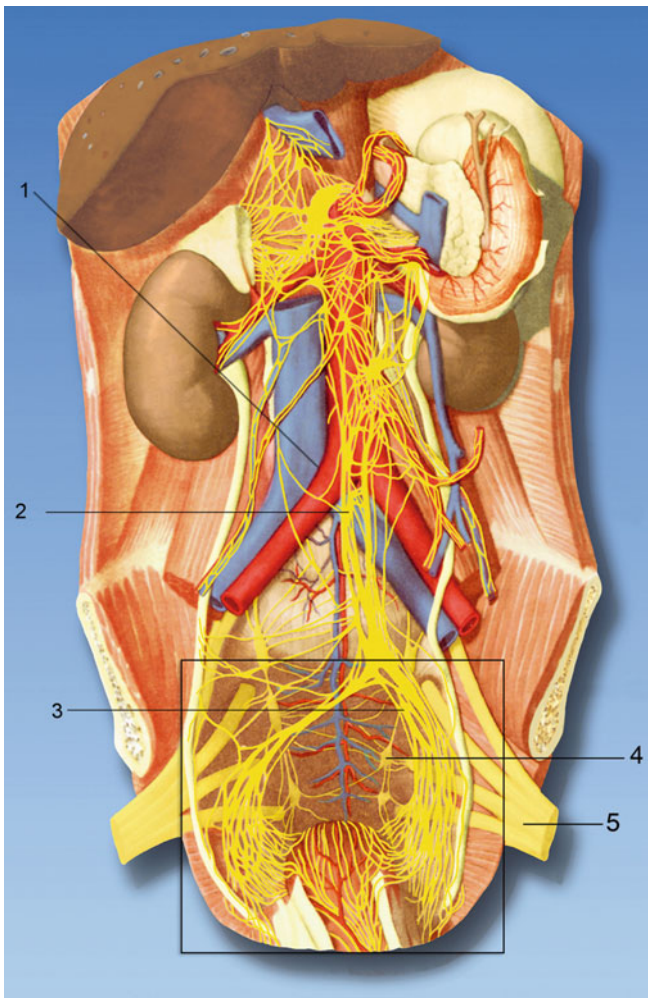


Fig. 58.1 Anatomy. (*1*) Aorta and inferior vena cava, (*2*) superior hypogastric plexus, (*3*) pelvic (inferior hypogastric plexus), (*4*) lumbosacral sympathetic ganglia (*5*) lumbosacral plexus (Reproduced with permission from Danilo Jankovic)

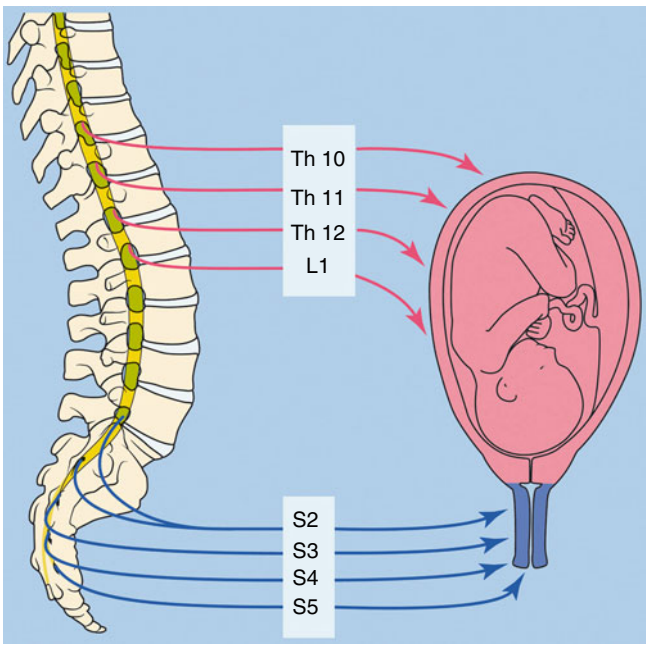


Fig. 58.2 Peripheral pain pathways during labor (Reproduced with permission from Danilo Jankovic)

Indications

Paracervical block has a limited role in modern obstetric practice, except in cases where central neural blockade is not feasible or is unavailable.

Surgical

1. Bilateral block may be used for dilatation of the cervix and uterine curettage.
2. Occasionally, it may be combined with pudendal nerve block and local infiltration to perform a vaginal hysterectomy.

Vaginal Delivery

Bilateral paracervical block alleviates the pain of the first stage of labor, and this is the most frequent use of this block.

Avoid: in prematurity, fetal distress, and uteroplacental insufficiency.

Therapeutic

1. Relief of severe dysmenorrhea
2. Endometritis and parametritis

Diagnostic

Differentiation of severe dysmenorrhea from other similar complaints which may be associated with menstruation.

Materials

The IOWA trumpet, Kobak instrument, is used as a guide for the placement of the needle (20-gauge, 12–14 cm needle) (S. Chap. 56, Pudendal nerve block).

Technique

Paracervical and uterosacral blocks are identical procedures and the local anesthetic solution bathes the same plexus and ganglia. If the solution is deposited at the 3 and 9 o'clock positions, the block is termed paracervical, and if the solution is deposited at the 4 and 8 o'clock positions, the block is termed uterosacral (Fig. 58.4). The pelvic (inferior hypogastric) ganglia and plexus are anesthetized when this block is performed.

Position and Landmarks

The patient is placed in the supine position with her legs apart and partially in the lithotomy position. The injection is made in the lateral fornices of the vagina.

1. Vaginal examination should be performed immediately before the block to determine the precise position of the presenting part.
2. Maternal and fetal vital signs should be determined immediately before the block.

Procedure

1. The needle is introduced through the submucosa into the lateral vaginal fornix (Fig. 58.3).

   The guide, with needle tip protected, is directed into the lateral fornices between 3 and 4 o'clock and between 8 and 9 o'clock, by the index and middle fingers, so that the tip of the guide does not depress the vaginal mucosa excessively and to determine the precise location of the needle and to protect the vaginal tissues and the fetal presenting part from inadvertent puncture by the advancing needle (Fig. 58.4).
2. The needle is then introduced into the uterosacral ligament.

   The needle should be inserted with a guard (e.g., an Iowa Trumpet) so that the needle point cannot protrude beyond the guard for more than 5–7 mm. This maneuver prevents inadvertent intravascular injection and also prevents damage to the fetal presenting part.
3. After careful aspiration at various levels (blood), the local anesthetic is injected on an incremental basis. Approximately 5–10 ml of the chosen local anesthetic should be used in each fornix for satisfactory paracervical block.
4. The needle is removed, the guide redirected to the other side of the cervix, and the injection repeated.

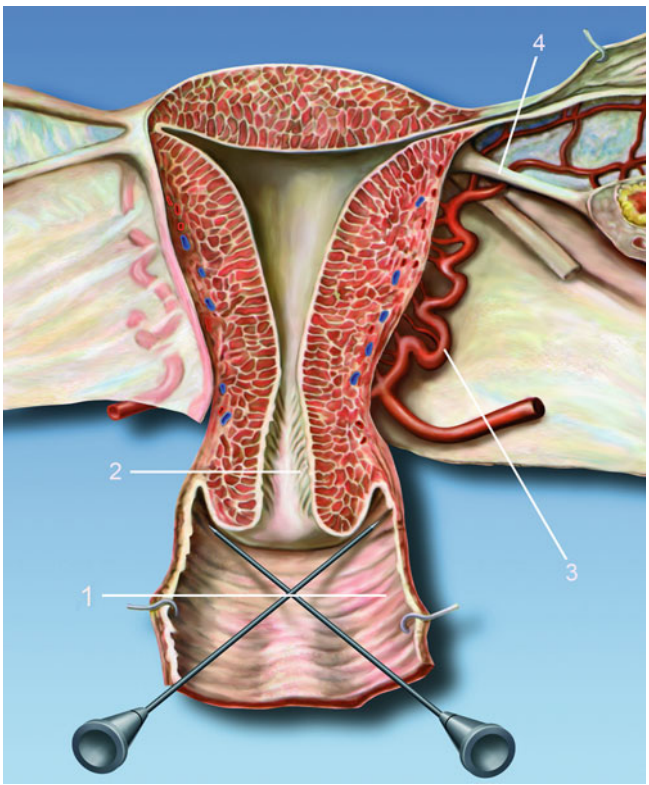


Fig. 58.3 The needle is introduced through the submucosa into the lateral vaginal fornix. (*1*) Lateral vaginal fornix, (*2*) ostium uteri, (*3*) uterine artery, (*4*) round ligament of uterus (Reproduced with permission from Danilo Jankovic)

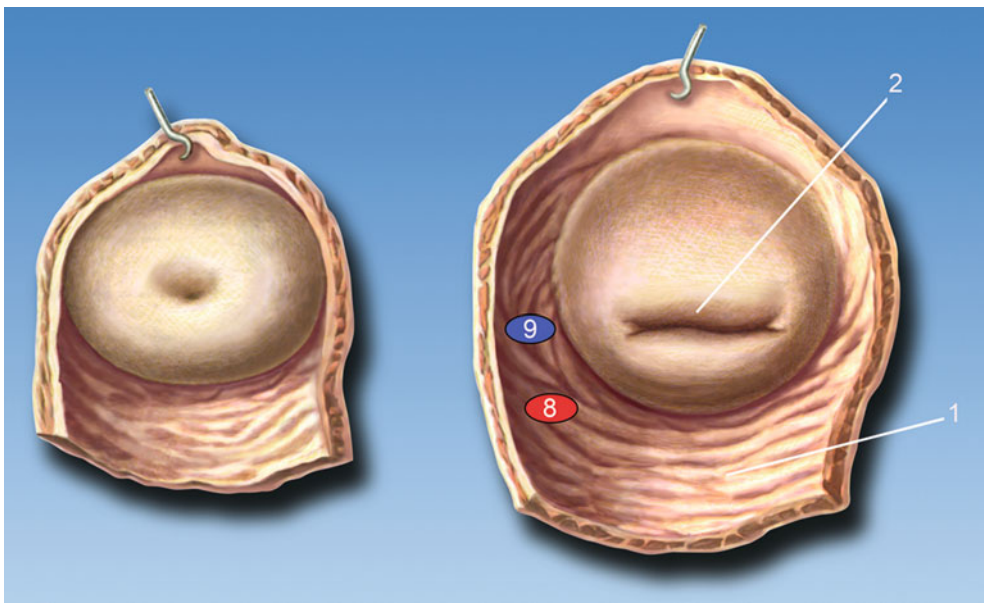


Fig. 58.4 The guide, with the needle tip protected, is directed into the lateral fornices between 3 and 4 o'clock and between 8 and 9 o'clock. (*1*) Vaginal fornix, (*2*) ostium uteri (Reproduced with permission from Danilo Jankovic)

Dosage

The duration of the analgesia will vary with the local anesthetic, its concentration, and the total dose used:

- 0. 25 % bupivacaine provides 90–150 min of analgesia.
- 1 % mepivacaine provides 90–150 min of analgesia.
- 1 % procaine or 2 % chloroprocaine provides 40–50 min of analgesia.

When performing a paracervical block, fetal heart rate and maternal uterine contractions should be monitored immediately preceding and for some time following the block.

Complications

1. Systemic toxic reactions (see Chap. 1).
2. Fetal distress, bradycardia, fetal acidosis, neonatal depression, and low Apgar scores. Paracervical injection may lead to high levels of local anesthetics in the fetus; up to 70 % of fetuses will have arrhythmias (principally bradycardia) within 10 min of injection.

   Fetal deaths from bupivacaine cardiotoxicity have also been reported.
3. Block of the sciatic nerve, neuritis of the sciatic nerves, or both. They may be a result of the needle being inserted further than 1. 5 cm beyond the vaginal mucosa, the use of great volume of local anesthetic solution, or a combination of these.
4. Hematoma of the parametrium. This occurs from trauma to a blood vessel during execution of the block. Finally, uterine artery hematoma, cervical abscess, and uterine trauma have been reported.
5. Injections have occasionally been made directly into the fetal scalp.

A transient decrease in intensity and/or frequency of uterine contractions could be associated with the local anesthetic, especially when epinephrine is used to perform the block.

Suggested Reading

1. Asling JH, Shnider SM, Margolis AJ, et al. Paracervical block anesthesia in obstetrics. II. Etiology of fetal bradycardia following paracervical block anesthesia. Am J Obstet Gynecol. 1971;107:626.
2. Bloom SL, Horswill CW, Curet LB. Effects of paracervical blocks on the fetus during labour. A prospective study with the use of direct fetal monitoring. Am J Obstet Gynecol. 1972;114:218.
3. Brownridge P, Cohen SE, Ward E. Neural blockade for obstetrics and gynecologic surgery. In: Cousins M, Bridenbaugh PO, editors. Neural blockade. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 574–5.
4. Cibils LA, Santonja-Lucas JJ. Clinical significance of fetal heart rate patterns during labour. III: effect of paracervical block anesthesia. Am J Obstet Gynecol. 1978;130:73.
5. Goins JR. Experience with mepivacaine paracervical block in an obstetric private practice. Am J Obstet Gynecol. 1992;167:342.
6. Grimes DA, Gates J. Deaths from paracervical anesthesia used first trimester abortion. N Engl J Med. 1976;295:1397.
7. Moore DC. Paracervical (uterosacral) Block. In: Moore DC, editor. Regional block. Springfield: Charles Thomas; 1976. p. 179–87.
8. Murphy PJ, Wright JD, Fitzgerald TB. Assessment of paracervical nerve block anesthesia during labour. Br Med J. 1970;1:526.
9. Parris WCV. Paracervical nerve. In: Hahn M, McQuillan PM, Sheplock GJ, editors. Regional anesthesia. St. Louis: Mosby; 1996. p. 273–7.
10. Rosefsky JB, Petersiel ME. Perinatal deaths associated with mepivacaine paracervical block anesthesia in labor. N Engl J Med. 1968;278:530.
11. Shnider SM, Asling JH, Hall JW, et al. Paracervical block anesthesia in obstetrics. I. Fetal complications and neonatal morbidity. Am J Obstet Gynecol. 1970;107:619.
12. Viscomi CM. Obstetric anesthesia. Paracervical block. In: Rathmell JP, Neal J, Viscomi CM, editors. Regional anesthesia. Philadelphia: Elsevier –Mosby; 2004. p. 173–85.

Part X
Lower Extremity Block

Chapter 59

Lumbar Plexus and Femoral Nerve Block. Traditional and Ultrasound Guided Techniques

Danilo Jankovic, MD (Traditional Techniques)
Director of Pain Management Centre DGC – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Jens Kessler, MD (✉) (Ultrasound Section)
Department of Anesthesiology, University Hospital Heidelberg, Heidelberg, Germany
e-mail: jens.kessler@med.uni-heidelberg.de

Andrew T. Gray, MD, PhD (Ultrasound Section)
Department of Anesthesia and Perioperative Care, University of California, San Francisco General Hospital, San Francisco, CA 94110, USA
e-mail: graya@anesthesia.ucsf.edu

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_59, © Springer International Publishing Switzerland 2015

Anatomy of the Lumbar Plexus, Sacral Plexus, and Coccygeal Plexus [1–5]

Danilo Jankovic

These plexuses, closely related to one another, are formed by the ventral branches of the lumbar, sacral, and coccygeal spinal nerves (Figs. 59.1, 59.2, 59.3, 59.4, and 59.5).

The lumbar plexus lies in front of the transverse processes of the lumbar vertebrae. It mainly arises from the ventral branches, the first three lumbar nerves, most of the fourth lumbar nerve, and the twelfth thoracic nerve (subcostal nerve).

The most important branches of the plexus are located in a fascial compartment that is enclosed ("sandwiched") by the quadratus lumborum, psoas major, and iliacus muscles. The first lumbar nerve, which contains a branch from the twelfth thoracic nerve, divides into an upper branch (iliohypogastric nerve and ilioinguinal nerve) and a lower branch (genitofemoral nerve).

Most of the second, third, and parts of the fourth lumbar nerves form ventral branches, from which the femoral nerve and obturator nerve branch off. The lateral femoral cutaneous nerve is formed from fibers of the ventral branches of L2/L3.

The caudal parts of the ventral branches of L4 and L5 combine to form the lumbosacral trunk. Together with the ventral branches of the first three sacral nerves and the upper part of the ventral branch of the fourth sacral nerve, the lumbosacral trunk forms the sacral plexus, the largest branch of which is the sciatic nerve. The lumbar plexus is also connected with the lumbar part of the sympathetic nervous system via two or three long communicating branches. The thickness of the ventral branches of the lumbar nerves increases markedly from the first to the fifth nerve (L1 has a diameter of ca. 2.5 mm, L2 is already ca. 4 mm, L3 and L4 are ca. 6 mm, and L5 is as large as 7 mm).

The coccygeal plexus arises from the lower part of the ventral branches of the fourth and fifth sacral nerves, as well as the coccygeal nerves.

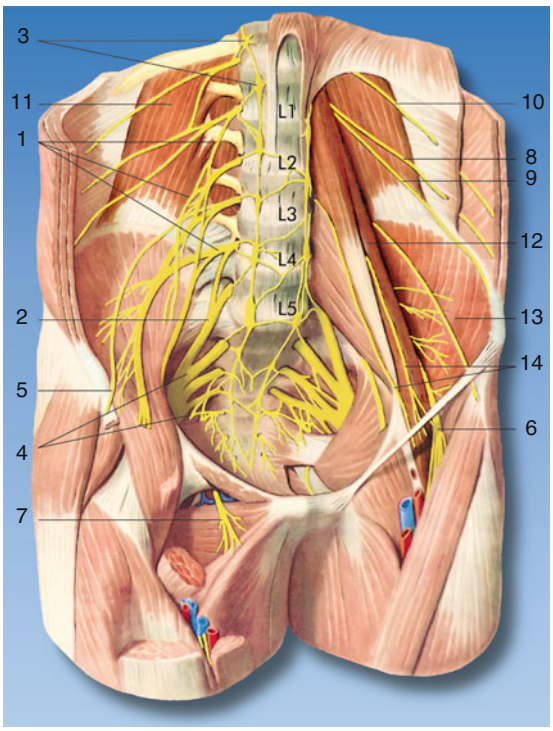


Fig. 59.1 Anatomy: lumbar plexus, sacral plexus, and coccygeal plexus. (*1*) Lumbar plexus, (*2*) lumbosacral trunk, (*3*) sympathetic trunk, (*4*) sacral plexus, (*5*) lateral femoral cutaneous nerve, (*6*) femoral nerve, (*7*) obturator nerve, (*8*) iliohypogastric nerve, (*9*) ilioinguinal nerve, (*10*) subcostal nerve, (*11*) quadratus lumborum muscle, (*12*) psoas major muscle, (*13*) iliacus muscle, (*14*) genitofemoral nerve (Reproduced with permission from Danilo Jankovic)

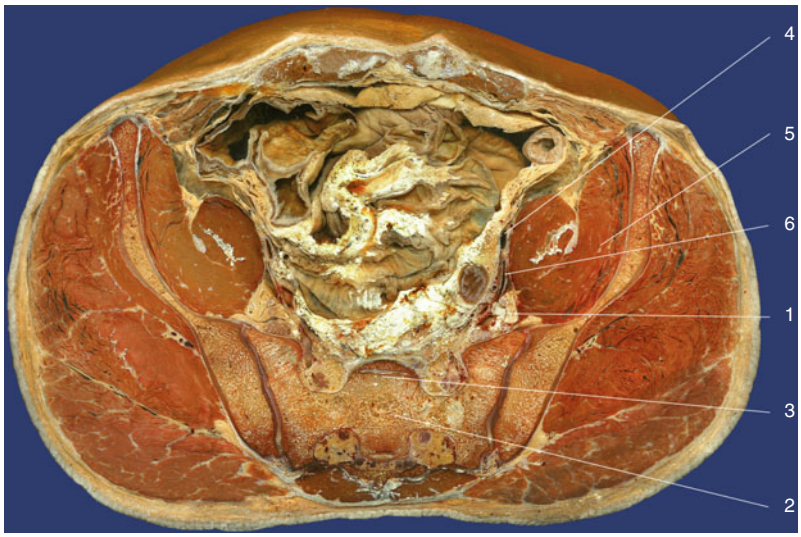


Fig. 59.2 Lumbar plexus. Transversal dissection. (*1*) Lumbar plexus, (*2*) os sacrum, (*3*) intervertebral disc, (*4*) left external iliac artery, (*5*) iliopsoas muscle, (*6*) left external iliac vein (Reproduced with permission from Danilo Jankovic)

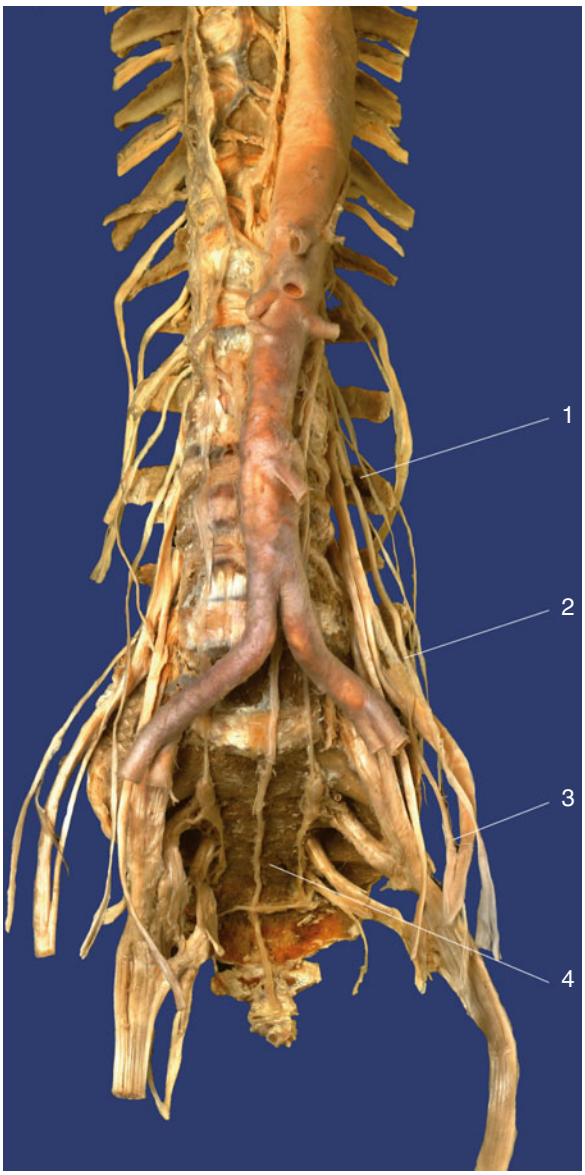


Fig. 59.3 Lumbosacral plexus. Ventral view. (*1*) Lumbar plexus, (*2*) lumbosacral trunk, (*3*) sacral plexus, (*4*) medial sacral artery: left and right side: sympathetic trunk (Reproduced with permission from Danilo Jankovic)

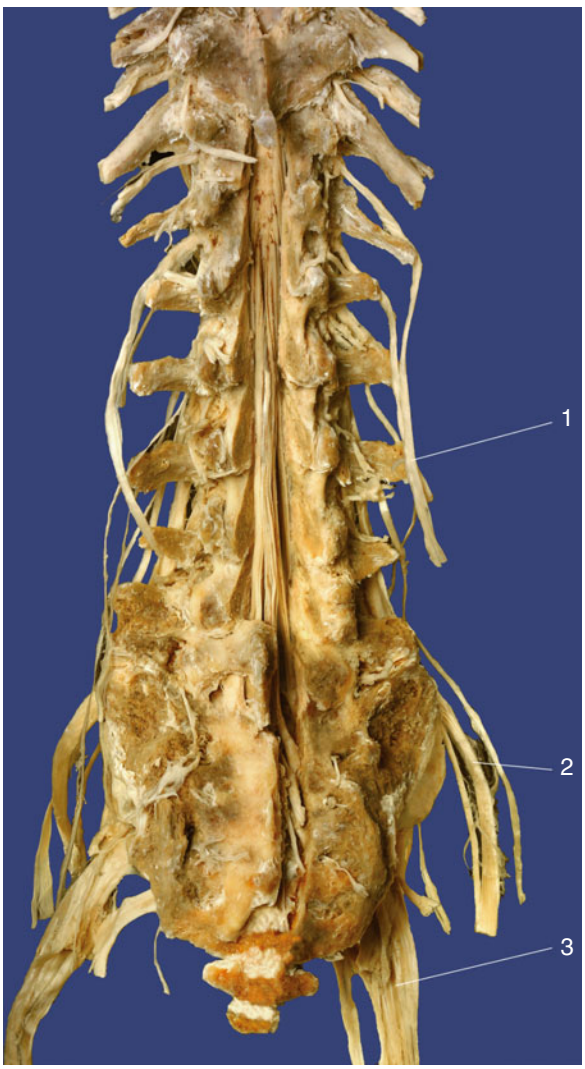


Fig. 59.4 Lumbosacral plexus. Dorsal view. (*1*) Lumbar plexus, (*2*) lumbosacral plexus, (*3*) sacral plexus (Reproduced with permission from Danilo Jankovic)

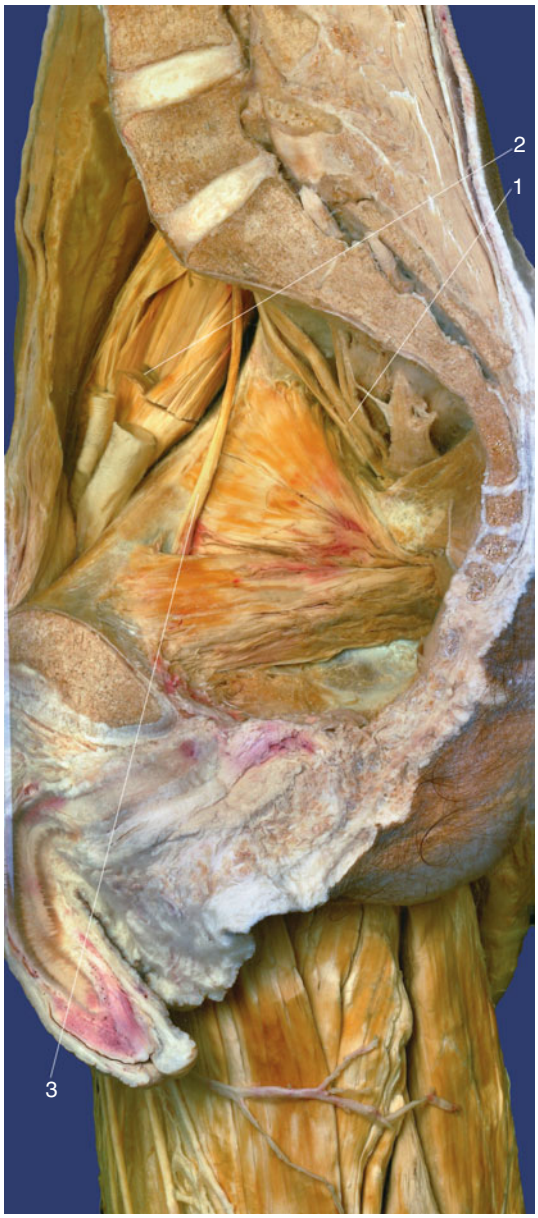


Fig. 59.5 Anatomy. Medial sagittal dissection. (*1*) Lumbosacral plexus, (*2*) iliopsoas muscle with femoral nerve, (*3*) obturator nerve (Reproduced with permission from Danilo Jankovic)

Lumbar Plexus Blocks

Two techniques are described that belong to the standard methods for blocking the lumbar plexus:

1. The caudal (ventral) psoas compartment block ("three-in-one" inguinal femoral paravascular block)
2. The cranial (dorsal) psoas compartment block

Inguinal Femoral Nerve Block ("Three-in-One" Block)

Danilo Jankovic

Introduction

The concept underlying lumbar plexus blocks is that the course of the neural network from the transverse processes to the inguinal ligament lies within a perivascular and perineural space. Like the epidural space, this space limits the spread of the local anesthetic and conducts it to the various nerves.

The initial description of the 3-in-1 block (an infero-anterior approach to the femoral, lateral femoral cutaneous, and obturator nerve) was published by Winnie et al. in 1973 involving a small number of patients. The authors postulated that a block of the entire lumbar plexus can be accomplished by a single perivascular injection slightly distal to the inguinal ligament. Consequently, a single injection should result in anesthesia of the femoral, the lateral femoral cutaneous, and obturator nerves (Figs. 59.6, 59.7, and 59.8). Winnie et al. suggested that the underlying mechanism of this regional anesthetic technique should be a cephalad distribution of the local anesthetic along the fascial layer. Within the connective tissue and neural sheath, the concentration and the volume of the local anesthetic determine the extent of the block's spread [6–8].

This hypothesis, however, was never confirmed clinically. Moreover, an MRI study clarified the spread of local anesthetic after an inguinal injection of local anesthetic lateral to the femoral artery and concluded that the distribution of local anesthetic follows a lateral and slightly medial direction but never a cephalad direction. One of the main proposed advantages of the 3-in-1 block was the ability to achieve block of the obturator nerve using this approach. In clinical practice, however, the obturator nerve (posterior branch) was never been shown to be anesthetized effectively using this approach [9–13]. Capdevila et al. reported that the local anesthetic used in 3-in-1 blocks spreads under the fascia iliaca but rarely to the lumbar plexus. Both techniques resulted in poor blocks of the obturator nerve [14].

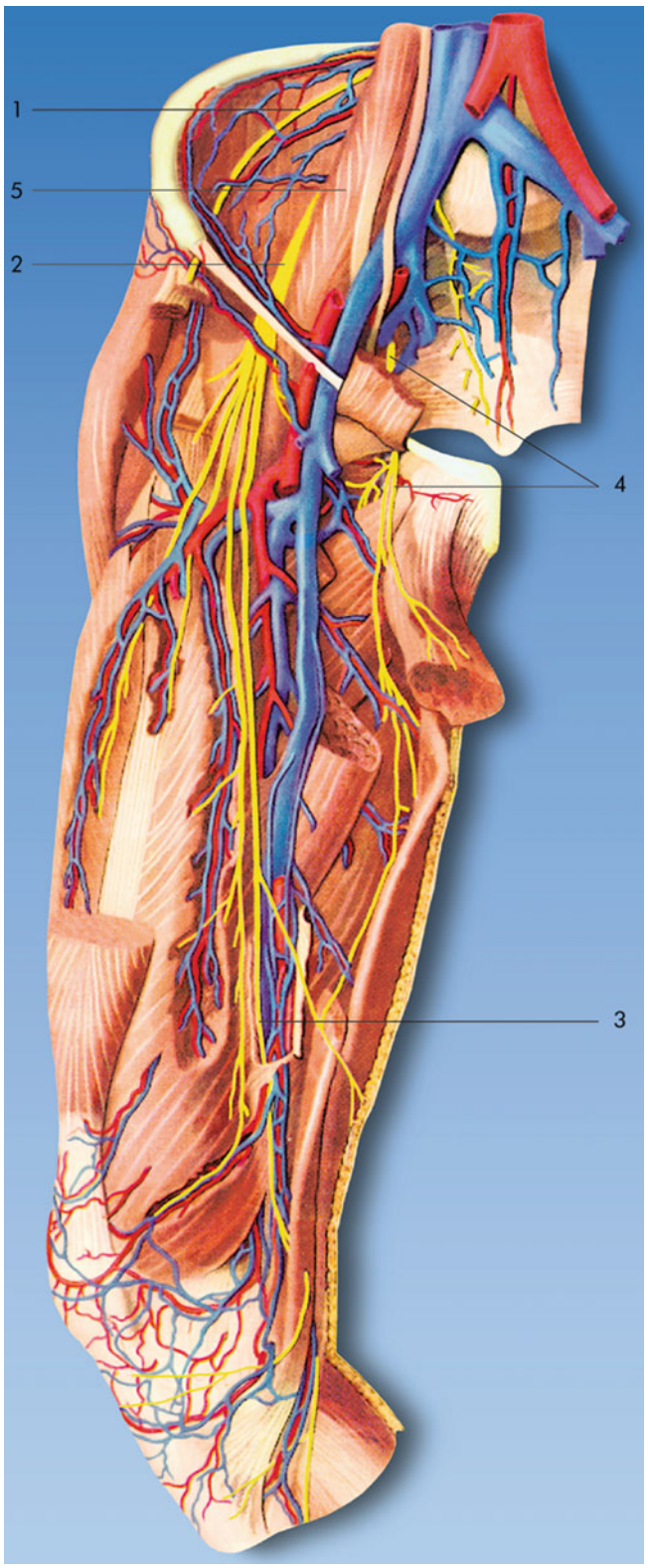


Fig. 59.6 Anatomy: femoral nerve, lateral femoral cutaneous nerve, and obturator nerve. (*1*) Lateral femoral cutaneous nerve, (*2*) femoral nerve, (*3*) saphenous nerve, (*4*) obturator nerve, (*5*) psoas major muscle (Reproduced with permission from Danilo Jankovic)

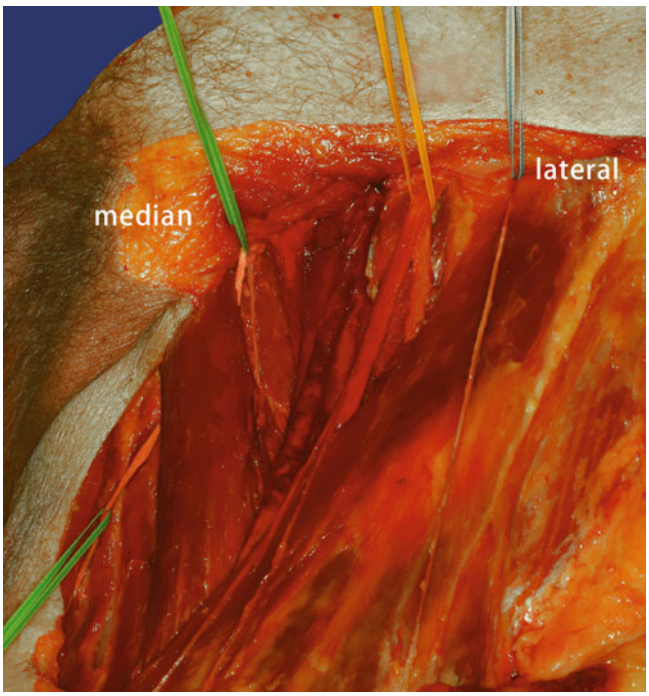


Fig. 59.7 (*1*) Femoral nerve (*yellow*), (*2*) lateral cutaneous femoral nerve (*blue*), (*3*) obturator nerve (*green*). In clinical practice, the obturator nerve (posterior branch) was never been shown to be anesthetized effectively (Reproduced with permission from Danilo Jankovic)

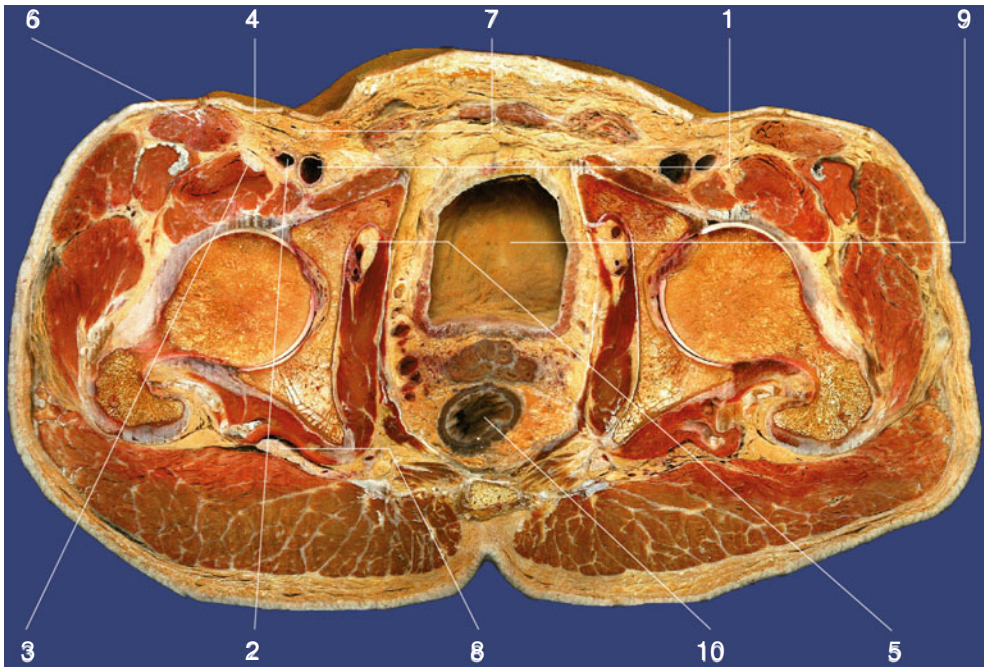


Fig. 59.8 Transversal dissection. (*1*) Femoral vein, (*2*) femoral artery, (*3*) femoral nerve, (*4*) lateral cutaneous femoral nerve, (*5*) obturator nerve with vasa obturatoria, (*6*) sartorius muscle, (*7*) fascia lata, (*8*) sciatic nerve, (*9*) vesica urinaria, (*10*) rectum (Reproduced with permission from Danilo Jankovic)

Functional Anatomy

The *femoral nerve* divides slightly distal to the inguinal ligament in several branches, which is the rationale for the nerve block needle to be inserted close to the distal ligament when performing the 3-in-1 block. The femoral nerve supplies motor branches to the quadriceps femoris, sartorius, and pectineus muscles, and its sensory branch (saphenous nerve) innervates the anteromedial side of the lower leg down to the medial ankle. Both the obturator nerve and the lateral femoral cutaneous nerve divide at variable levels from the femoral nerve (Figs. 59.6, 59.7, and 59.8).

The *lateral femoral cutaneous nerve* arises from the ventral branches of the L2 and L3 spinal nerves, passing lateral to the psoas muscle and then to the iliacus muscle. Covered by the fascia iliaca, it then runs to the region of the anterior superior iliac spine. It passes under the inguinal ligament and under the deep circumflex iliac artery and enters the thigh, where it lies under the superficial sheet of the fascia and divides into a thicker descending branch and a smaller posterior branch, which penetrate the fascia separately. The posterior branch runs posteriorly over the tensor fascia lata muscle and reaches the gluteal region. The anterior branch runs 3–5 cm below the inguinal ligament and then downwards along the anterior surface of the vastus lateralis muscle as far as the lateral knee area, where it sends off lateral branches (Figs. 59.6, 59.7, and 59.8).

The *obturator nerve* arises from the ventral branches of the L2–L4 spinal nerves.

The trunk runs downwards along the medial edge of the psoas muscle, passing behind the common iliac vessels to reach the pelvis and the obturator canal. Within the canal, it divides into its two end branches—the anterior and posterior branches. It provides the motor supply for the obturator externus muscle and the adductors of the thigh, sends off branches to the hip and knee joints and to the femur, and provides the sensory supply for a highly variable cutaneous area on the inside of the thigh and lower leg (Figs. 59.6, 59.7, and 59.8).

Advantages

1. Suitable for postoperative or post-traumatic analgesia and for therapeutic blocks
2. Suitable for patients in whom a unilateral block is desired—particularly in outpatient procedures

Disadvantages

1. Success is unpredictable.
2. Larger amounts of local anesthetic are necessary (particularly if the sciatic nerve is also being blocked).
3. The likelihood of systemic toxicity is increased.
4. Longer onset times have to be expected (surgical indications).
5. Despite larger amounts of local anesthetic, not all nerves in the plexus are blocked (e.g., the obturator nerve and lateral femoral cutaneous nerve).
6. For surgical procedures with ischemia or a tourniquet, neuraxial anesthesia is preferable.

Indications

Surgical [19–22]

1. Superficial surgical interventions in the innervated area: wound care, skin grafts, and muscle biopsies [17]
2. Analgesia for positioning for neuraxial block anesthesia in femoral neck fractures
3. Performing surgical interventions in the area of the lower extremity in ischemia or tourniquet, in combination with sciatic nerve block [15, 16]

   Larger volumes of local anesthetic have to be used here (toxicity!).
4. Outpatient procedures

Therapeutic

1. Postoperative pain therapy (e.g., after femoral neck, femoral shaft, tibial and patellar fractures, knee joint operations) [18]
2. Post-traumatic pain
3. Postoperative neurolysis or nerve reimplantations for better innervation
4. Early mobilization after hip or knee joint operations
5. Arterial occlusive disease and poor perfusion in the lower extremities
6. Complex regional pain syndrome (CRPS) types I and II
7. Postamputation pain
8. Edema in the leg after radiotherapy
9. Diabetic polyneuropathy
10. Knee joint arthritis
11. Elimination of adductor spasm in paraplegic patients

Block Series

A series of six to eight blocks is recommended. When there is evidence of improvement in the symptoms, additional blocks can also be carried out.

Prophylactic

1. Postoperative analgesia
2. Prophylaxis against postamputation pain
3. Prophylaxis against complex regional pain syndrome (CRPS)

Contraindications

Specific

1. Infections (e.g., osteomyelitis, pyoderma) or malignant diseases in the inguinal region
2. Local hematoma
3. Anticoagulant treatment
4. Distorted anatomy (due to prior surgical interventions or trauma to the inguinal and thigh region)

Relative

The decision should be taken after carefully weighing up the risks and benefits:

1. Hemorrhagic diathesis
2. Stable central nervous system disorders
3. Local nerve injury (when it is difficult to determine whether the cause is surgical or anesthetic)
4. Contralateral nerve paresis
5. Patients with a femoral bypass

Procedure

This block should be carried out by experienced anesthetists or under their supervision. Full information for the patient is mandatory.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, emergency medication.

Materials

Fine 26-G needle, 25-mm long, for local anesthesia.

Peripheral nerve stimulator.

Single-Shot Technique

50 (80)-mm-long atraumatic 22-G (15°) short-bevel insulated stimulating needle with injection lead ("immobile needle").

Continuous Technique*

Peripheral nerve stimulator.

Catheter kit (including a 50-80-mm 18-G (15°)) stimulating needle and catheter 0.45×0.85×400 mm or Tuohy set: 52 (102)-mm-long 18-G Tuohy needle with catheter.

Syringes: 2 and 20 mL.

Local anesthetics, disinfectant, swabs, compresses, sterile gloves, and drape.

*If technical difficulties arise, the catheter and Tuohy needle must always be removed simultaneously. A catheter must never be removed through the Tuohy needle (as the catheter may shear!).

Patient Positioning

Supine, with the thigh slightly abducted. The patient's ipsilateral hand lies under the head. The person carrying out the injection must stand on the side being blocked.

Landmarks

1. The femoral artery is palpated 1–2 cm distal to the inguinal ligament. It is held between the spread index and middle finger. The injection point lies about 1–1.5 cm laterally.
2. Skin prep, subcutaneous local anesthesia, sterile drapes; draw up local anesthetic into 20-mL syringes, check patency of injection needles and functioning of nerve stimulator, and attach electrodes.
3. Preliminary puncture with a large needle or stylet.

The quadriceps femoris muscle and the patella must be observed throughout the procedure.

Injection Technique

Single-Injection Technique

1. The injection is carried out in a cranial direction at an angle of about 30–40° to the skin surface, almost parallel to the course of the femoral artery.

   Stimulation current of 1–2 mA at 2 Hz is selected with a stimulus duration of 0.1 ms (Fig. 59.9). Usually, the nerve is at the depth of 12+/−4 mm.
2. The needle is advanced until contractions of the quadriceps femoris muscle and patellar movements become visible ("dancing patella"). Contractions of the sartorius muscle alone suggest incorrect positioning and are inadequate (Fig. 59.10).
3. Do not advance the needle further!

   The stimulation current is reduced to 0.2–0.3 mA. Slight twitching suggests that the stimulation needle is in the immediate vicinity of the nerve.
4. Aspiration test.
5. Test dose of 3 mL local anesthetic (e.g., 1 % prilocaine). During the injection, the twitching slowly disappears.
6. Incremental injection of a local anesthetic (injection–aspiration after each 3–4 mL).
7. After the injection, compression massage of the injection area is carried out and then flexing of the thigh for about 1 min (Fig. 59.11).
8. Careful cardiovascular monitoring.

During the injection, distal compression should be applied with the finger to encourage proximal spread of the local anesthetic.

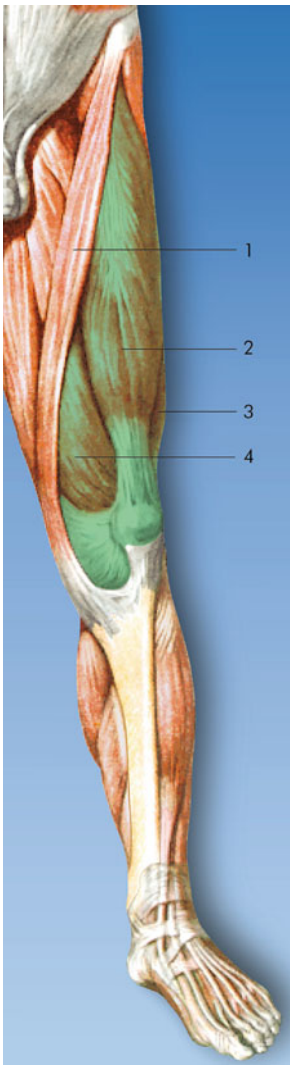


Fig. 59.10 Note the contractions of the quadriceps femoris muscle and patellar movements. (*1*) Sartorius muscle, (*2*) rectus femoris muscle, (*3*) vastus lateralis muscle, (*4*) vastus medialis muscle (Reproduced with permission from Danilo Jankovic)

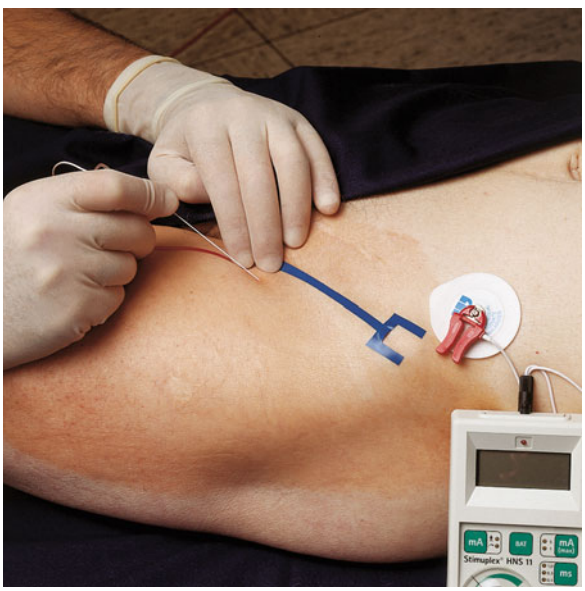


Fig. 59.9 Injection. Cranial direction, at an angle of about 30–40° to the skin surface (Reproduced with permission from Danilo Jankovic)

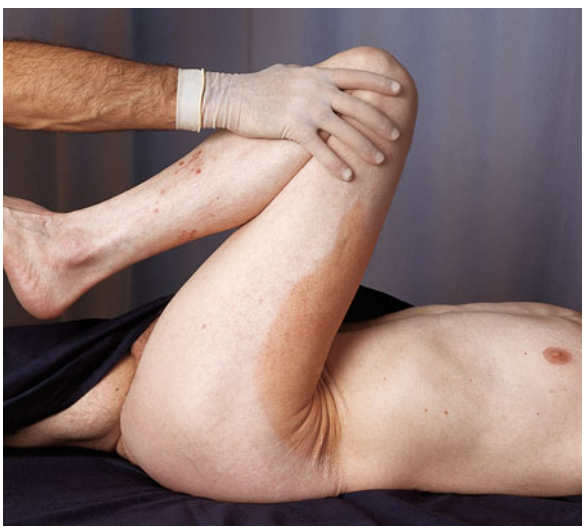


Fig. 59.11 After the injection: flexing the thigh for about 1 min (Reproduced with permission from Danilo Jankovic)

Distribution of Anesthesia

To produce complete anesthesia in the leg, it should be combined with a sciatic nerve block (Figs. 59.12 and 59.13) [15, 16].

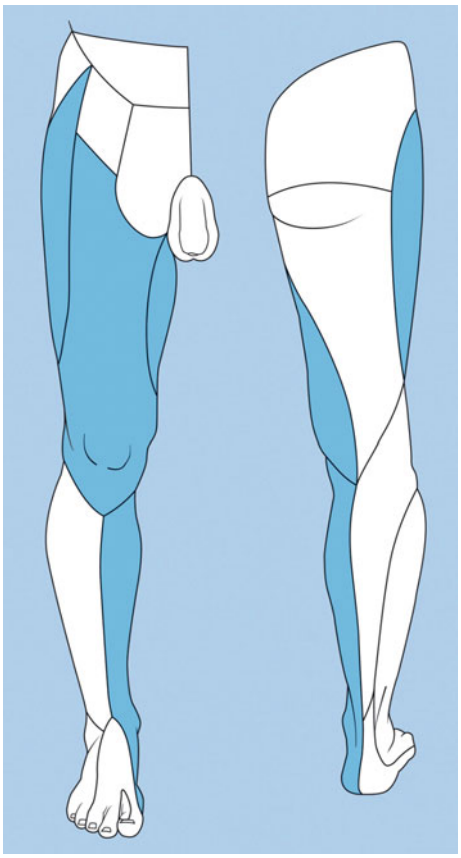


Fig. 59.12 The neural areas most frequently blocked after administration of a "three-in-one" block (Reproduced with permission from Danilo Jankovic)

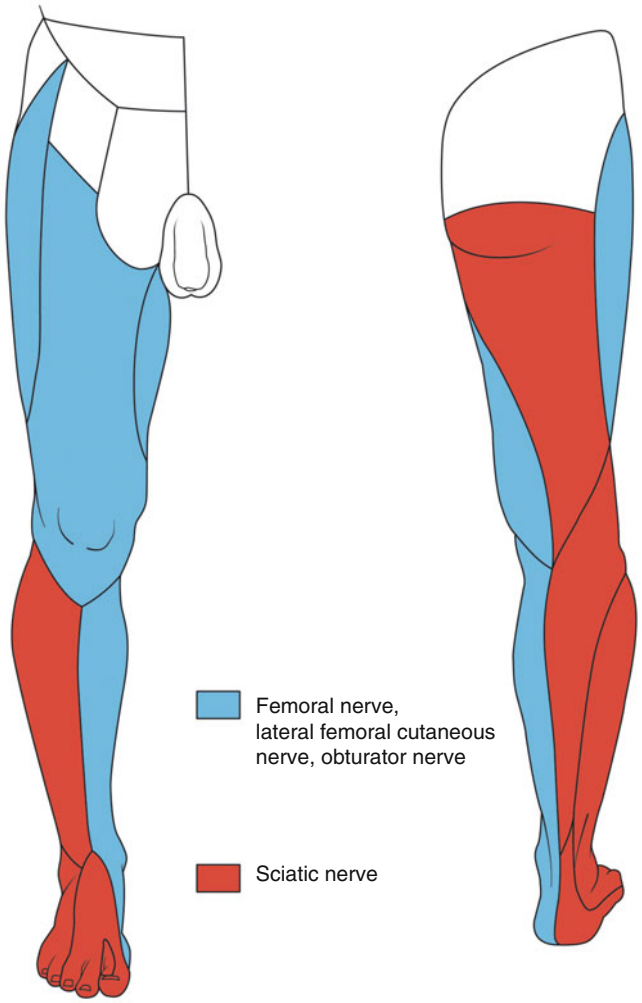


Fig. 59.13 Comparison of the innervation areas of the femoral nerve, lateral femoral cutaneous nerve, and obturator nerve (*blue*) with the innervation area of the sciatic nerve (*red*) (Reproduced with permission from Danilo Jankovic)

Continuous Technique

Either nonstimulating (conventional) or stimulating catheters can be used. The site is located in the same way as described for the unilateral technique. The injection is carried out about 2–2.5 cm below the inguinal ligament and 1–1.5 cm lateral to the femoral artery and in a cranial direction at an angle of about 30–40°. Using the Seldinger technique, the catheter is advanced at least 10 cm deep into the fascial compartment. An aspiration test, administration of a test dose, fixation of the catheter, and placement of a bacterial filter then follow. After aspirating again, the local anesthetic is given on an incremental basis.

Dosage

Surgical

1. 15–20 mL—single-injection femoral nerve block
2. 40–50 mL local anesthetic—e.g., 0.5–0.75 % ropivacaine, 0.5 % bupivacaine (0.5 % levobupivacaine), 1 % prilocaine, and 1 % mepivacaine

A combination of local anesthetics with longer-term and medium-term effect has proved particularly valuable for surgical indications—e.g., 1 % prilocaine (20 mL) + 0.5 to 0.75 % ropivacaine (20 mL) or 1 % prilocaine (20 mL) + 0.25 to 0.5 % bupivacaine (0.25–0.5 % levobupivacaine; 20 mL).

Therapeutic

10–15 mL local anesthetic—e.g., 0.2–0.375 % ropivacaine, 0.125–0.25 % bupivacaine (0.125–0.25 % levobupivacaine).

Important Notes for Outpatients

Long-lasting block can occur (even after administration of low-dose local anesthetics—e.g., 0.125 % bupivacaine or 0.2 % ropivacaine). The blocked leg can give way even 10–18 h after the injection. The patient must therefore use walking aids during this period. The same rules apply to the treatment of postamputation pain. During the period of effect of the local anesthetic, the patient should not wear a prosthesis.

A record must be kept of patient information and consent.

Continuous Technique [23, 24]

Test dose: 3–5 mL 1 % prilocaine (1 % mepivacaine).

Bolus administration: 30 mL.

0.5–0.75 % ropivacaine or 0.25–0.5 % bupivacaine.

Maintenance Dose

Intermittent administration.

15–20 mL of local anesthetic every 4–6 h (0.5–0.75 % ropivacaine or 0.25–0.5 % bupivacaine) after a prior test dose.

Reduction of the dose and/or adjustment of the interval, depending on the clinical picture.

Continuous Infusion

Infusion of the local anesthetic via the catheter should be started 30–60 min after the bolus dose. A test dose is obligatory.

Ropivacaine: 0.2–0.375 %	16–14 mL/h
	1 (max. 37.5 mg/h)
Levobupivacaine: 0.125–0.25 %	18–15 mL/h
Bupivacaine: 0.125 %	10–14 mL/h
Bupivacaine: 0.25 %	18–10 mL/h

If necessary, the infusion can be supplemented with bolus doses of 5–10 mL 0.5–0.75 % ropivacaine (0.25–0.5 % bupivacaine or 0.25 % levobupivacaine).

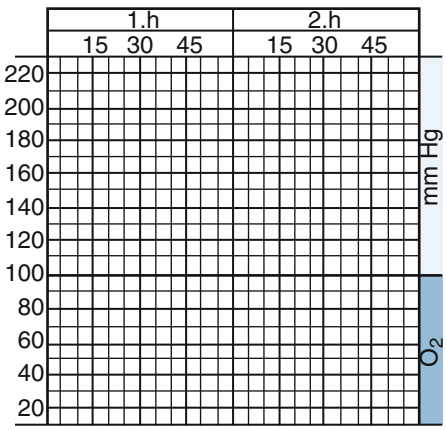
Individual adjustment of the dosage and period of treatment is essential.

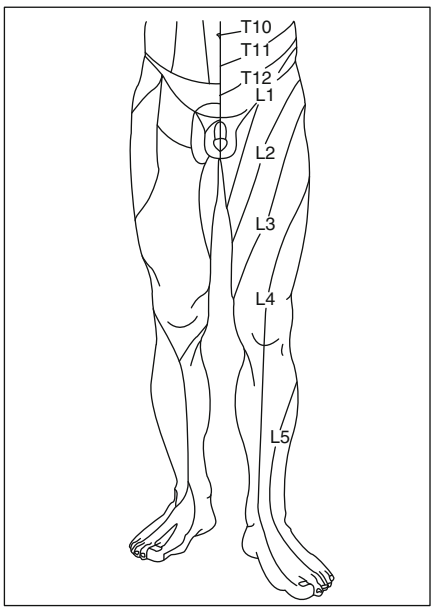
Complications

1. Nerve injuries (see Chap. 5)

   Traumatic nerve injury is a rare complication of this technique. It can occur as a result of the use of sharp needles (due to nerve puncture), intraneural or microvascular injury (hematoma and its sequelae), prolonged ischemia, as well as toxic effects of intraneurally injected local anesthetic (see Chap. 5). Probable effects of intraneural injection include a transient neurological deficit (unexpectedly prolonged block, lasting up to 10 days). A suspicion of intraneural needle positioning arises if there is strong twitching even at low levels of stimulation current (e.g., 0.2 mA) and if there is no cessation of the twitching after administration of the test dose. The local anesthetic may also be difficult to inject. Correction of the needle position is essential.
2. Intravascular injection (see Chap. 1)
3. CNS intoxication (see Chap. 1)
4. Infection in the injection area (continuous techniques)
5. Hematoma formation (note the obligatory prophylactic compression)

Record and checklist





Lumbosacral plexus and individual nerves in the plexus

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Diagnostic* ☐ *Therapeutic*

Needle: *G*____ *Length* _____ *cm* ☐ *15°* ☐ *30°* ☐ *Other*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed (behavior after block)*

Position: ☐ *Prone* ☐ *Lateral recumbent* ☐ *Sims-Position* ☐ *Sitting*

Injection: ☐ *Inguinal "3-in-one" block* ☐ *Dorsal psoas compartment block*

☐ *Sciatic nerve* ☐ *Femoral nerve* ☐ *Lateral cutaneous nerve of thigh*

☐ *Obturator nerve* ☐ *Ilioinguinal nerve / hypogastric nerve*

Location technique: ☐ *Electrostimulation* ☐ *Other*

☐ *Ultrasound- guided* ☐ *Transducer* ☐ *Linear* ☐ *Curved*

Approach: ☐ *In-plane* ☐ *Out of plane*

Plexus (nerve): ☐ *Located* ☐ *Aspiration test* ☐ *Test dose*

Injection:

Local anesthetic: _____ *mL* _____ *%*

(in incremental doses)

☐ *Inguinal "3-in-one" block* ____ *mL* ☐ *Dorsal psoas compartmt. block* ____ *mL*

☐ *Sciatic nerve* ____ *mL* ☐ *Femoral nerve* ____ *mL* ☐ *Lateral cutaneous nerve of thigh* ___ *mL* ☐ *Obturator nerve* ___ *mL* ☐ *Ilioinguinal nerve/hypogastric nerve* ___ *mL*

☐ Addition to LA _____ *µg/mg*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain (intraneural position?)*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement before* ____ *°C after* ____ *°C*

☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____ ☐ *Sensorimotor function checked*

Complications:

☐ *None* ☐ *Intravascular injection* ☐ *Signs of intoxication*

☐ *Hematoma* ☐ *Neurological complications* ☐ *Other*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

With permission from Danilo Jankovic

Psoas Compartment Block (Cheyen Approach)

Danilo Jankovic

The psoas compartment block represents a cranial and dorsal paravertebral access route to the lumbar plexus. The concept is to block the closely juxtaposed branches of the lumbar plexus and parts of the sacral plexus by injecting local anesthetic through a high-access route to the plexus (L4–L5) (see lumbar plexus anatomy). When the quality of the block is good, the area of distribution is comparable with that of the "three-in-one" block (see "3-in-1" femoral nerve block; Figs. 59.12 and 59.13). The most important branches of the plexus are located in a fascial compartment that is enclosed ("sandwiched") by the quadratus lumborum, psoas major, and iliacus muscles (Figs. 59.14 and 59.15). The following nerves are affected: lateral femoral cutaneous nerve, femoral nerve, genitofemoral nerve, obturator nerve, and parts of the sciatic and posterior femoral cutaneous nerve. A combination of this block with block of the sciatic nerve is necessary to achieve complete anesthesia of the lower extremity.

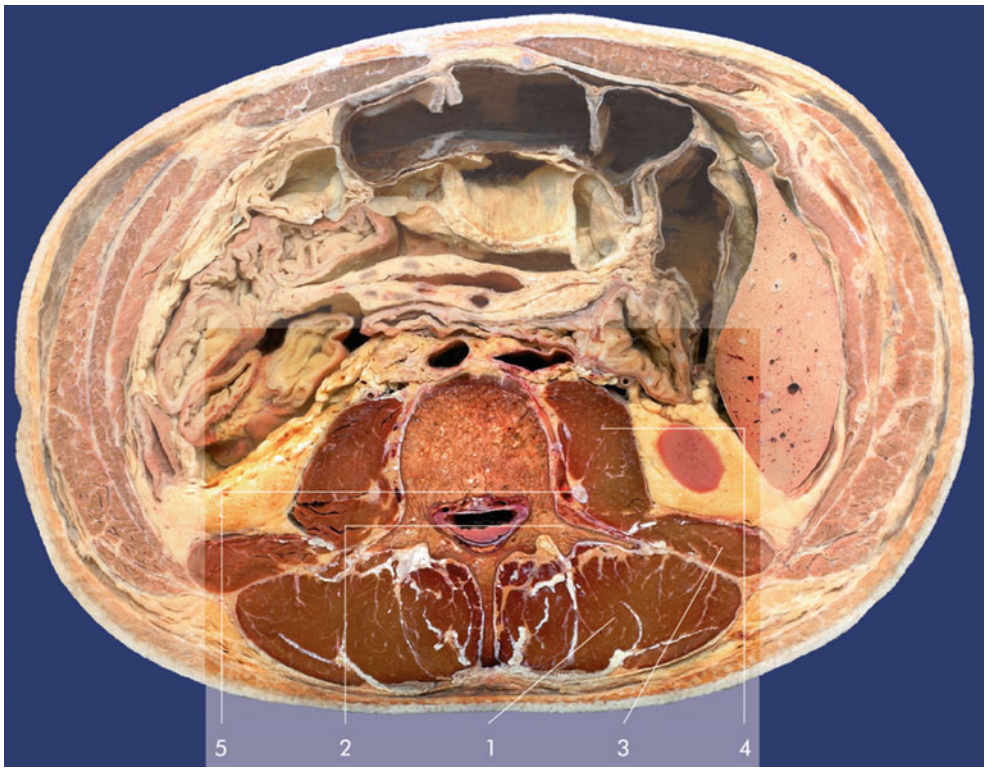


Fig. 59.14 Anatomy. Transversal dissection on the level of the L3/L4. (*1*) Erector spinae muscle, (*2*) transverse process, (*3*) quadratus lumborum muscle, (*4*) psoas major muscle, (*5*) lumbar plexus (Reproduced with permission from Danilo Jankovic)

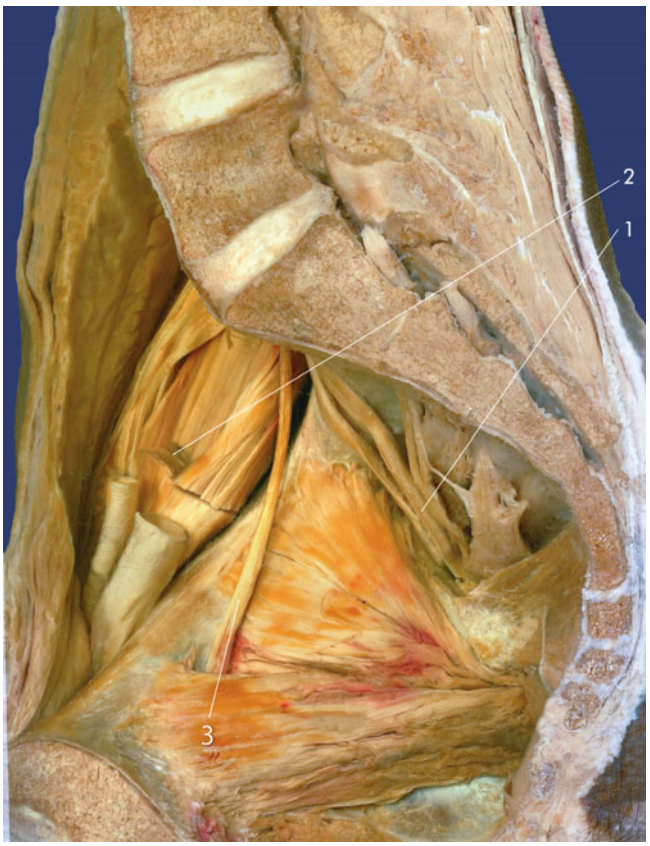


Fig. 59.15 Lumbosacral plexus. Median sagittal dissection. (*1*) Lumbosacral plexus at the level of L5–S3 segment, (*2*) iliopsoas muscle with femoral nerve, (*3*) obturator nerve (Reproduced with permission from Danilo Jankovic)

Advantages

1. Better block quality in comparison with the "three-in-one" block.
2. Suitable for patients in whom a unilateral block is desired, particularly in outpatient procedures.
3. The method is suitable for postoperative and post-traumatic analgesia and for therapeutic blocks.

Disadvantages

1. Success of the block is unpredictable.
2. Larger quantities of local anesthetic are needed (particularly if the sciatic nerve is also being anesthetized).
3. There is an increased likelihood of systemic toxicity.
   Because the placement of the needle is in the deep muscles, the potential for systemic toxicity is greater than it is with more superficial techniques.
4. The proximity of the lumbar nerve roots to the epidural space also carries risk of the epidural spread of the local anesthetics.
5. Slower onset must be expected (surgical indications).
6. For surgical procedures with ischemia or tourniquet, neuraxial anesthesia is preferable.

Indications

Surgical [27, 28]

1. As a continuous or single-shot block for all surgical procedures in the region of the lower extremity but in combination with a block of the sciatic nerve. A need for larger volumes of local anesthetics must be expected (toxicity!).
2. Outpatient procedures.

Therapeutic

1. Postoperative and post-traumatic pain therapy
2. Early mobilization after hip and knee operations
3. Arterial occlusive disease and poor perfusion of the lower extremities
4. Complex regional pain syndrome (CRPS), types I and II
5. Postsurgical neurolysis or nerve reimplantations for better innervation
6. Edema after radiotherapy

7. Postamputation pain
8. Diabetic polyneuropathy
9. Tumors and metastases in the hip joint and pelvis

Block Series

A series of six to eight blocks is recommended. When there is evidence of improvement in the symptoms, additional blocks can also be carried out.

Prophylactic

1. Postoperative analgesia
2. Prophylaxis against postamputation pain
3. Prophylaxis against complex regional pain syndrome

Contraindications

Specific

1. Infection or hematoma in the injection area
2. Anticoagulant treatment
3. Lesion in the nerves to be stimulated distal to the injection site

Relative

The decision should be taken after carefully weighing up the risks and benefits:

1. Hemorrhagic diathesis
2. Stable systemic neurological diseases
3. Local nerve injury (when there is doubt whether the fault lies with the surgeon or anesthesiologist)
4. Contralateral nerve paresis

Procedure

This block should be carried out by experienced anesthetists or under their supervision. Full information for the patient is mandatory.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, emergency medication.

Materials

Fine 26-G needle, 25-mm long, for local anesthesia.

Electrostimulation Technique

Peripheral nerve stimulator.

120-mm-long short-bevel insulated atraumatic 22-G needle (15°) with injection lead ("immobile needle").

Continuous Technique*

Tuohy set: 1.3 × 102 (152)-mm-long 18-G Tuohy insulated needle with catheter or 18-G (15°) insulated stimulating needle (1.3 × 110 mm with catheter).

Syringes: 2 and 20 mL.

Local anesthetics, disinfectant, swabs, compresses, sterile gloves, and drape.

*If technical difficulties arise, the catheter and Tuohy puncture needle are always removed simultaneously. A catheter must never be withdrawn through a Tuohy puncture needle that remains in place (because of catheter shearing).

Patient Positioning

Lateral decubitus or sitting, as in the position for neuraxial anesthesia; legs drawn up, with the leg being blocked on top.

Landmarks

The iliac crest and the midline of the spinous process are located. From the intersection between these (L4 spinous process), a line is drawn 3 cm caudally, and from the end of it, another line is drawn 5 cm laterally as far as the medial edge of the iliac crest and marked as the injection point (Fig. 59.16).

Skin prep, local anesthesia, sterile draping, drawing up the local anesthetic into 20-mL syringes, checking the patency of the injection needle and functioning of the nerve stimulator, and attaching the electrodes.

Preliminary puncture with a large needle or stylet.

> The quadriceps femoris muscle must be observed throughout the procedure.

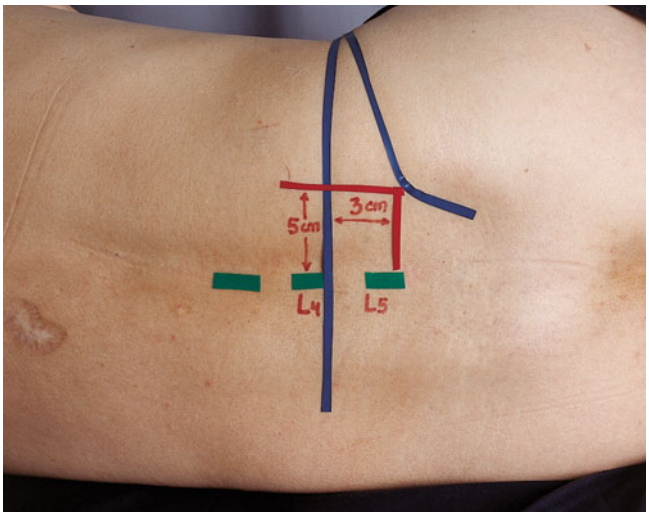


Fig. 59.16 Location (Reproduced with permission from Danilo Jankovic)

Injection Technique [29–31]

Single-Injection Technique

1. The palpating hand should be firmly pressed and anchored against the paraspinal muscles to facilitate the needle insertion and redirection of the needle when necessary.
2. Introduce an electrostimulation needle perpendicular to the skin surface until bone contact is made with the transverse process of L5 (Figs. 59.17a and 59.18).

   The location of the transverse process is very important. The lumbar plexus is situated at the depth of 8.35 cm in average in males (6.1–10.1 cm) and 7.1 cm in average in females (5.7–9.3 cm). The distance from the skin to the transverse process is variable and depends from the anatomy. The distance between the transverse process and the lumbar plexus is relatively constant (app. 2 cm) [32].

   It is then withdrawn slightly and advanced further cranially, past the transverse process (Figs. 59.17b and 59.18). Stimulation current of 1,5 mA at 2 Hz is selected with a stimulus duration of 0.1 ms.
3. Advance the needle further until contractions of the quadriceps femoris muscle become visible (usually at the depth of 6–8 cm).
4. Reduce the stimulation current to 0.5–1.0 mA. If contractions of the muscle are still visible at this level of current, the needle is in the correct position.
5. Do the aspiration test.
6. Test dose 3–5 mL of a local anesthetic.
7. Inject the local anesthetic incrementally (injection–aspiration after each 3–4 mL).
8. Do cardiovascular monitoring carefully.

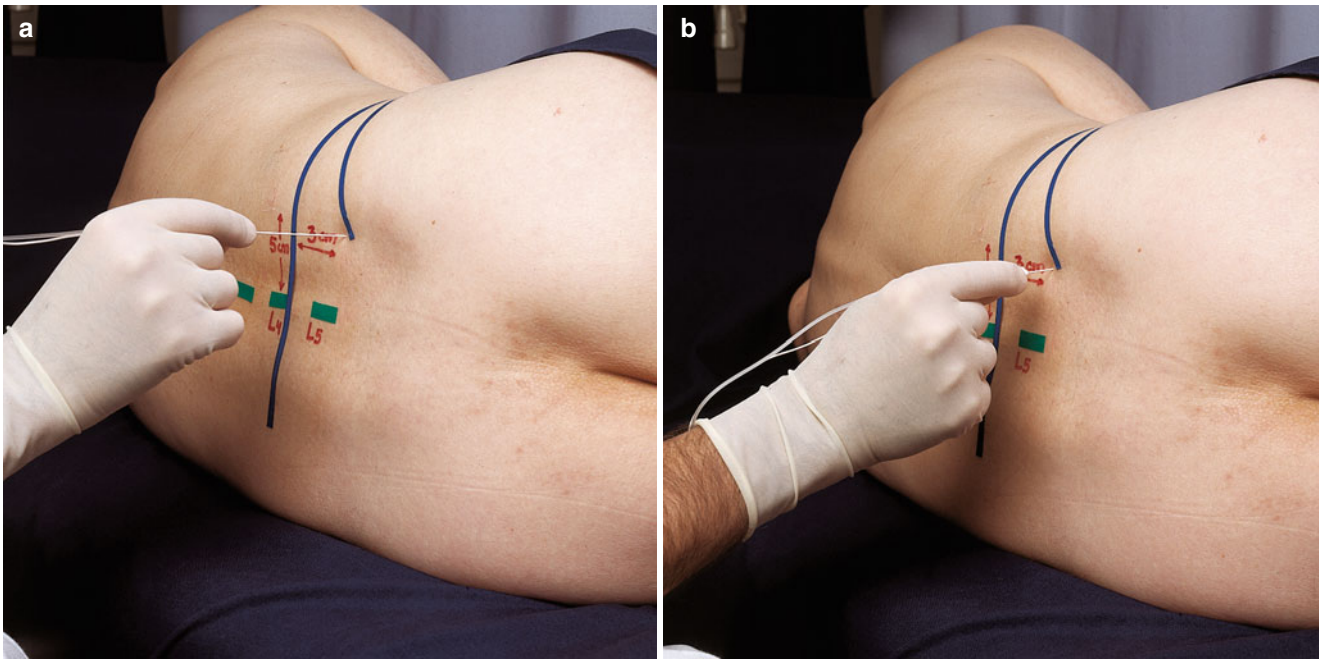


Fig. 59.17 (**a**) The injection needle is introduced perpendicular to the skin surface until bone contact is made with the transverse process of L5 (reproduced with permission from Danilo Jankovic). (**b**) The injection needle is advanced until contractions of the quadriceps femoris muscle become visible (Reproduced with permission from Danilo Jankovic)

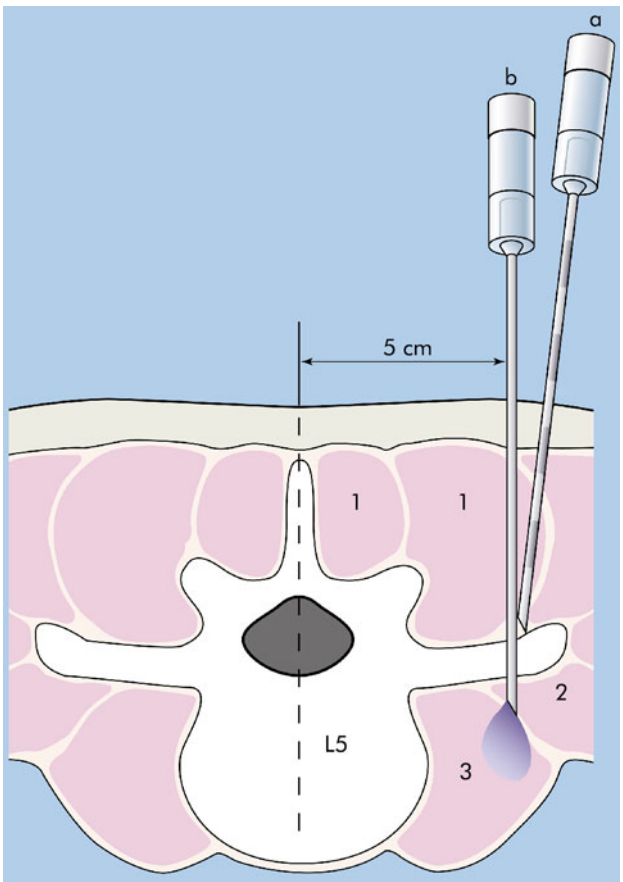


Fig. 59.18 Diagram: (*a*) contact with the transverse process of L5; (*b*) the needle is advanced past the transverse process until contractions of the quadriceps femoris muscle become visible. (*1*) Erector spinae muscle, (*2*) quadratus lumborum muscle, (*3*) psoas muscle (Reproduced with permission from Danilo Jankovic)

Continuous Technique [32–34]

The needle is attached to the nerve stimulator and to a syringe with local anesthetic.

A Tuohy, continuous block needle is inserted at the perpendicular angle to the skin and advanced until the quadriceps twitch response is obtained at 0.5–1.0 mA current. At this point, after aspiration test and injection of test dose, the initial volume of local anesthetic is injected (e.g.,15–25 mL), and the catheter is advanced ca. 8–10 cm beyond the needle tip into the fascial compartment. The needle is then withdrawn back to the skin level, while the catheter is simultaneously advanced. The opening of the needle should be oriented cephalad before threading the catheter. This method prevents inadvertent removal of the catheter and intravascular and intrathecal placement by negative aspiration test.

Dosage

Surgical

40–50 mL local anesthetic—e.g., 1 % prilocaine (20–30 mL) + 0.5 to 0.75 % ropivacaine (20 mL); 1 % prilocaine (20–30 mL) + 0.25 to 0.5 % bupivacaine (0.25–0.5 % levobupivacaine; 20 mL).

Therapeutic

10–20 mL local anesthetic—e.g., 0.2–0.375 % ropivacaine, 0.125–0.25 % bupivacaine (0.125–0.25 % levobupivacaine).

Important Notes for Outpatients (See "3-in-1" block)

Continuous Technique

Test dose: 3–5 mL 1 % prilocaine (1 % mepivacaine).

Bolus administration: 30 mL.

0.5–0.75 % ropivacaine or 0.25–0.5 % bupivacaine.

Maintenance Dose

Intermittent administration.

15–20 mL of local anesthetic every 4–6 h (0.5–0.75 % ropivacaine or 0.25–0.5 % bupivacaine) after a prior test dose.

Reduction of the dose and/or adjustment of the interval, depending on the clinical picture.

Continuous Infusion

Infusion of the local anesthetic via the catheter should be started 30–60 min after the bolus dose. A test dose is obligatory.

Ropivacaine: 0.2–0.375 %	16–14 mL/h
	1 (max. 37.5 mg/h)
Levobupivacaine: 0.125–0.25 %	18–15 mL/h
Bupivacaine: 0.125 %	10–14 mL/h
Bupivacaine: 0.25 %	18–10 mL/h

If necessary, the infusion can be supplemented with bolus doses of 5–10 mL 0.5–0.75 % ropivacaine (0.25–0.5 % bupivacaine or 0.25 % levobupivacaine).

Individual adjustment of the dosage and period of treatment is essential.

Complications

1. Nerve injury (extremely rare) (see Chap. 5, neurological complications..).
2. Intravascular injection
3. Local anesthetic toxicity [35, 36] (see Chap. 1)
4. Subarachnoid or epidural injection with the potential risk of high neuraxial anesthesia [37, 38] (see Chap. 41, neuraxial anesthesia)
5. Hypotension
6. Iliopsoas or renal hematoma formation [39–41]
7. Pneumatocoele [42]
8. Intra-abdominal injuries
9. Post-injection pain due to spasm in the lumbar paravertebral musculature

Ultrasound-Guided Femoral Nerve Block

Jens Kessler

The femoral nerve is the largest branch of the lumbar plexus and is located superficial to the iliopsoas muscle and deep to the fascia iliaca as a key landmark [44] inside a triangular shape [45] (Fig. 59.19). Close to its medial side, another two important landmarks can be detected: the femoral artery and the femoral vein. The differentiation of the hypoechoic muscle and the hyperechoic nerve is easy, whereas the differentiation of the hyperechoic borders of the nerve and the also hyperechoic fascia layers on top can be difficult. Tilting the probe to direct the ultrasound beam perpendicular to the femoral nerve influences its visibility due to anisotropic effects [51].

Textbooks describe these landmarks to be consistent, but there are also anatomic variations shown in literature [47, 49]. It is therefore important to compress the underlying vessels with the ultrasound transducer to differentiate between the artery and the vein. Furthermore, the correct position of the transducer for a femoral nerve block can be defined by scanning cephalad along the inguinal crease to the inguinal ligament (between the anterior superior iliac spine and the pubic tubercle) until the profunda femoris artery disappears and the branches of the femoral nerve are visible as a hyperechoic flattened oval structure next to the common femoral artery.

Figure 59.20 also shows an inguinal lymph node which is common in this region. Due to its central hyperechoic appearance, it is sometimes interpreted to be a part of a nerve. By scanning proximally and distally, the lymph node disappears, whereas the continuous structure of a nerve can be traced.

There are two important artifacts which can influence the block procedure of the femoral nerve (Fig. 59.21).

The "lateral shadowing" can delete important information of the ultrasound picture and is also able to imitate the profunda femoris artery. The "dorsal enhancement" is able to cause confusion by simulating a nonexisting part of the femoral nerve.

The femoral nerve block provides anesthesia and analgesia in areas supplying the femoral, lateral femoral cutaneous, infrapatellar, and saphenous nerves and also provides motor innervation to the quadriceps, pectineus, and sartorius muscles. In combination with a proximal sciatic nerve block, it provides complete analgesia below the knee joint. The patient is placed supine with the leg in the neutral position. After skin disinfection and appropriate covering of the transducer (Fig. 59.22) to maintain sterility, the transducer is placed along the inguinal region to detect the sonoanatomy with its typical landmarks.

The block needle can be inserted either in an out-of-plane (Fig. 59.22) or an in-plane (Fig. 59.23) approach by using a short-axis imaging.

Only for the insertion of a catheter, it has been shown that a long-axis in-plane approach results in a slightly faster onset of sensory anesthesia [48]. To reduce the incidence of needle–nerve contacts during femoral nerve blocks, the equally effective in-plane approach is recommended in the latest literature [50]. Due to lack of evidence, other authors suggest that for ultrasound-guided femoral catheter placement in a short-axis nerve imaging, operators should use the needle–probe alignment technique with which they are most familiar [46]. These recommendations support the findings by Michael Barrington describing quality-compromising behavior (QCB) types, especially QCB 1, the advancement of needles while not being visualized [43].

The ultrasound transducer should be a linear probe with a higher frequency (10–14 MHz) to be able to detect the superficial structures in high resolution. In patients with a higher body mass index, it can be necessary to use a low-frequency (2–5 MHz) curved array probe to achieve a better tissue penetration.

There are several studies investigating the optimum dose of the local anesthetic used for the block procedure [53, 54]. It depends on the type and concentration of the used medication. Some authors recommend to inject the local anesthetic around the nerve to achieve the well-known doughnut sign, but to date there is no evidence for clinical advantage to attempting to deposit LA circumferential to the femoral nerve relative to depositing it either above or below the nerve [52].

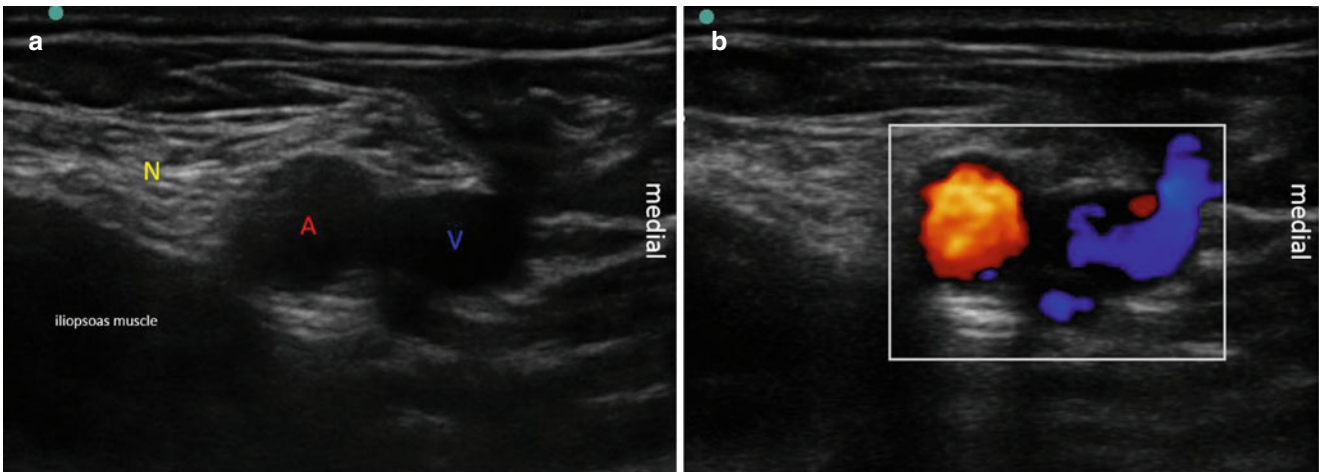


Fig. 59.19 (**a**) Femoral nerve (*N*) in the inguinal crease, femoral artery (*A*), and femoral vein (*V*) as landmarks. (**b**) Color Doppler as a confirmation method (With permission from Jens Kessler)

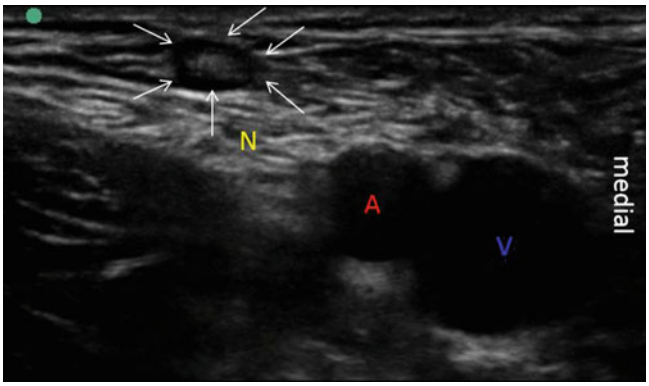


Fig. 59.20 Inguinal lymph node as artifact (With permission from Jens Kessler)

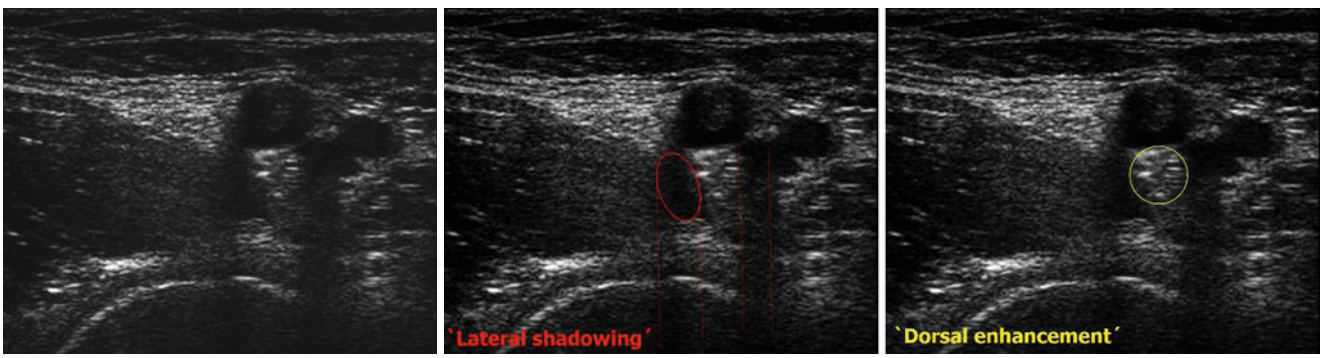


Fig. 59.21 Artifacts. Lateral shadowing (*red circle*); dorsal enhancement (*yellow circle*) (With permission from Jens Kessler)

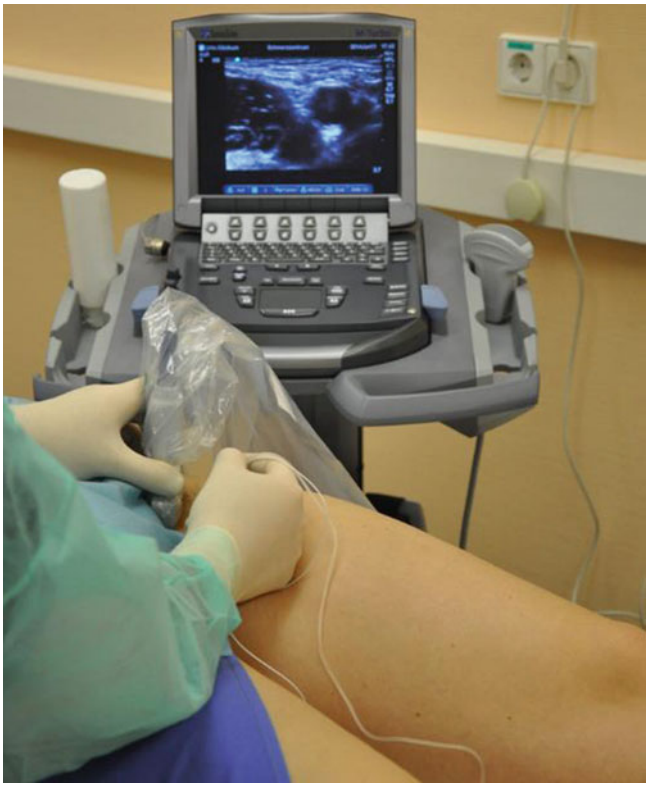


Fig. 59.22 Clinical setting with ergonomic positioning of the equipment for a femoral nerve block with an out-of-plane approach and a short-axis imaging (With permission from Jens Kessler)

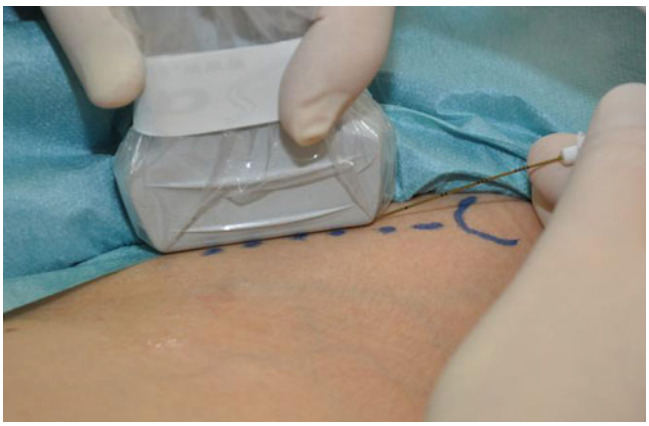


Fig. 59.23 The in-plane approach with an echogenic needle; the inguinal ligament and the anterior superior iliac spine are marked (With permission from Jens Kessler)

Ultrasound Imaging for Lumbar Plexus Blocks

Jens Kessler and Andrew T. Gray

Background and Methods

Indications for lumbar plexus block include surgery on the hip, femur, or knee. The patient position for the procedure can be supine, lateral decubitus (with the block side up), or prone. Of these positions, prone with a pillow under the abdomen is the most stable way to scan the lumbar plexus and paravertebral anatomy. Curved arrays will provide a broader view than linear arrays but have less scan line density as a function of depth. Because of the depth of the lumbar plexus and the attenuation of soft tissue, a low-frequency transducer (3–7 MHz) is recommended for imaging.

Level of the Block Procedure

The usual level of the block procedure is at L4–5 intertransverse space. More cephalad levels are not commonly approached because the lower poles of the kidneys extend to L3 and the psoas muscle is smaller in its mediolateral and anteroposterior diameters [3, 55]. Although approach to lumbar plexus block at L2–3 has been described [56], kidney injuries have been reported at or near this level [39]. During inspiration the kidneys will descend to a low position, with the lower pole of the right kidney slightly inferior to that of the left. These observations emphasize the importance of accurate level determination.

To establish the level of the block procedure, transverse process echoes can be traced from the sacrum upward [3]. The height of the intertransverse space averages 25 mm in the lumbar region, with the distance from L5 to S1 markedly smaller (about 13 mm). The lumbar roots average from 5 to 7 cm in mediolateral diameter in adults and exit the neuraxis at an angle of about 40° relative to the sagittal plane [57].

Depth

A significant problem with lumbar plexus block is how far to advance the needle. If the bone is not contacted and motor response not elicited, then there is a serious question of whether the needle is lateral to the transverse process or has advanced through the psoas muscle deep to the lumbar plexus. An additional concern is that the block needle is close to midline, risking epidural or subarachnoid placement.

The depth of the lumbar plexus ranges from 60 to 100 mm in adults of normal body habitus [32]. This broad range of values has led to development of anatomic rules for estimating the position of the lumbar plexus. The lumbar plexus is approximately 2 cm deep to the transverse process of L4 off its caudal edge (median value 18 mm for adults of either gender; [58]). Therefore, if the transverse process is contacted with the block needle, this can be used to walk off the caudal edge of the bone. Analogously, for thoracic paravertebral blocks, it is imperative to locate the transverse process before advancing the needle any further to prevent inadvertent pleural puncture [59]. Another anatomic rule is for needle advancement that the lumbar plexus is located at the junction of the posterior third and anterior two thirds of the psoas muscle [55]. The lumbar nerve roots that contribute to the lumbar plexus lie 5–6 mm deep to the intertransverse ligament [59]. If an online technique is utilized (scanning during needle placement and injection), it is important to observe layering of the injection deep to the transverse process to be sure it is deep to the intertransverse muscles.

Ultrasound Imaging of Paravertebral Anatomy

Just as practitioners of midline or paramedian techniques are familiar with the anatomy of the spinous processes, the practitioners of paravertebral and lumbar plexus blocks must be familiar with the anatomy of the transverse processes. Because the transverse processes cannot be palpated, estimation of their location is inherently more difficult. Furthermore, patient positioning can substantially change the relation and distances of these deep structures.

Similar to the way in which sonography can be used to estimate the depth and interspace for neuraxial blocks, ultrasound can be used to facilitate lumbar plexus block by identifying acoustic windows around bony structures. Paramedian longitudinal imaging planes are preferred for visualization of neuraxial structures [60]. Analogous imaging planes can be utilized for paravertebral structures [61].

Ultrasound can be used to estimate the depth and lateral extent of the transverse process for lumbar plexus block (Figs. 59.24 and 59.25). The bone shadow of the transverse process can be recognized by the bright reflection and distal extinction of the sound wave [3]. High receiver gain can help identify the bone shadows deep to the posterior surface of the transverse processes. From a practical standpoint, the easiest structure to identify is the lateral tip of the transverse process (the bone shadow that disappears laterally).

Direct imaging of the psoas muscle with ultrasound is consistently possible [62]. The normal muscle has echogenic fascial planes that may contain the lumbar plexus [31, 62]. The cephalad extent of the origin of the psoas muscle is normally at the T12 vertebral level.

The use of online sonography to guide lumbar plexus blocks can be difficult for several reasons. First, direct nerve imaging has only been consistently reported in pediatric patients less than 30 kg [63]. It has not been possible to visualize the lumbar plexus itself in adults because of the limited resolution of 3–7 MHz transducers. Second, the steep angle that the block needle must take to approach the lumbar plexus (close to perpendicular to the skin) results in poor needle tip visibility because backscatter rather than specular reflections are received by the ultrasound transducer [64]. Third, the manual dexterity and sterile precautions required for online use adds complexity without much benefit for this procedure. For these reasons, we generally prefer an offline imaging approach (scanning prior to needle placement) to estimate and mark the position of the transverse process and psoas muscle (Fig. 59.26).

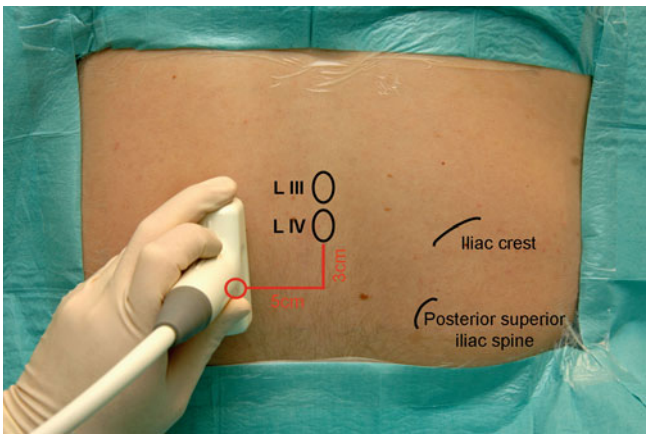


Fig. 59.24 External photograph illustrating the approximate position of the ultrasound transducer for imaging prior to lumbar plexus block. In this example, the tip of the transverse process will be imaged in short-axis view to estimate the position and depth of the lumbar plexus

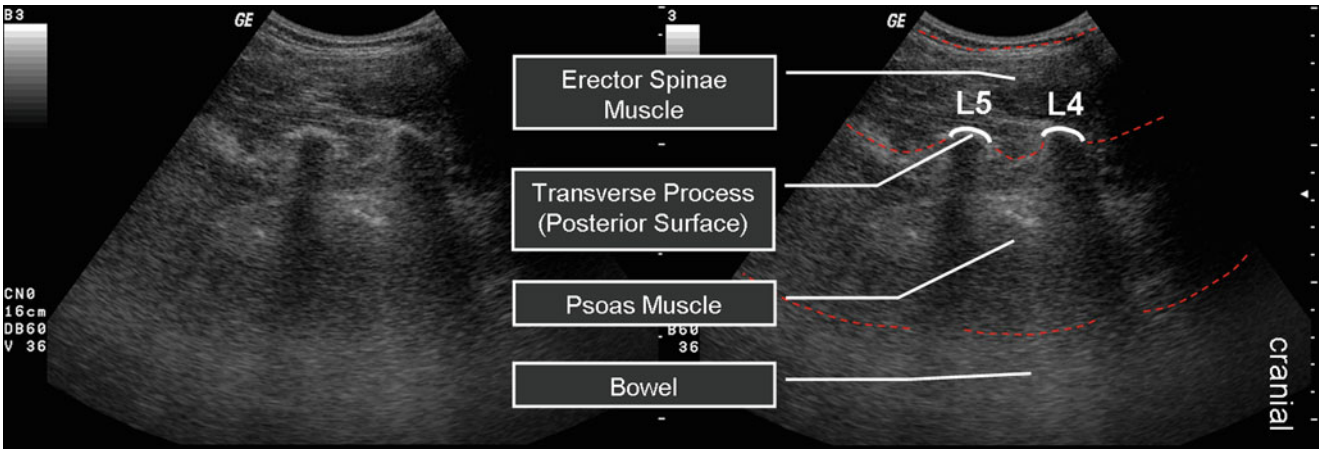


Fig. 59.25 Ultrasound image of the transverse process of L4 and L5 and the underlying psoas muscle. This sonogram was obtained with a curved 3.5 MHz transducer (Logiq 400 CL, General Electric). Depth markers are 1 cm apart

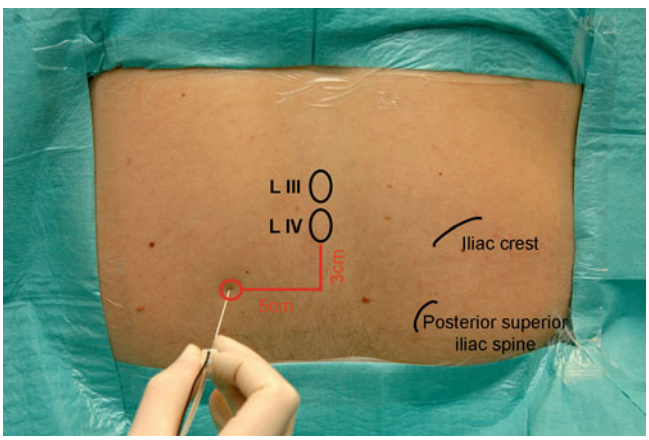


Fig. 59.26 Offline puncture with a nerve stimulation needle lateral to the tip of the transverse process of L5 after ultrasound scanning

Anatomic Variation and Limitations

Ultrasound imaging may be of benefit in guiding lumbar plexus blocks in patients with scoliosis, as it has been for neuraxial anesthetics [65–67]. However, the paravertebral anatomy of these patients has not been formally studied. The accuracy of ultrasonography in correctly identifying lumbar interspace levels is in the 71–76 % range for patients undergoing MRI to evaluate the lumbar spine [68, 69]. Lumbosacral transitional vertebrae are relatively common. An MRI study of 200 patients revealed 24 transitional vertebrae (15 cases of sacralization of L5 and nine cases of lumbarization of S1; [70]). Transitional vertebrae are associated with abnormal transverse process morphology, including complete or partial fusion to the sacrum [71]. Near the cephalad end of the lumbar region, it is common to have 11 pairs of ribs in the absence of associated anomalies; this situation occurs in 5–8 % of normal individuals [72]. These anatomic variations place limitations on the ability of any imaging modality to absolutely determine levels for block procedures.

Block Assessment

Lumbar plexus blocks can be assessed by testing sensation over the medial (L4), middle (L5), and lateral (S1) foot. Block of the contralateral side indicates neuraxial distribution of local anesthetic.

Risks

Risks of lumbar plexus block include incomplete block [73], inadvertent neuraxial block [32, 74], systemic toxicity [35, 75], bleeding [41, 57, 76, 77], infection, and nerve injury.

The failure rate of single-injection posterior lumbar plexus blocks is approximately 7 %, depending on the distribution evaluated [73]. Similarly, paravertebral block failures have been reported to be as high as 10 % [78, 79].

There is a substantial incidence of inadvertent neuraxial block with lumbar plexus procedures. In particular, epidural spread from lumbar plexus injection has been recognized as a relatively common event (about 6.5 % of lumbar plexus blocks; [32]). More medial approaches to lumbar plexus block are associated with higher incidence of epidural distributions [31]. Subarachnoid blocks also are possible [74].

Because lumbar plexus block usually involves high-volume injection through a needle in fixed position, systemic toxicity can be a significant issue. Manifestations of systemic toxicity have included seizures and cardiac arrest [35, 75]. These concerning outcomes have emphasized the importance of incremental injection and intermittent aspiration.

Serious bleeding complications with lumbar plexus blocks include renal subcapsular hematoma and psoas hematoma with resultant lumbar plexopathy [39, 76]. Several cases of delayed retroperitoneal hematoma have been reported after lumbar plexus block when anticoagulation was initiated after the block procedure [41, 77]. These cases have generated concern as to whether lumbar plexus block is indicated when anticoagulation is anticipated in the postoperative period.

The systemic toxicity of local anesthetics is probably reduced with ultrasound guidance. The reasons include lower volumes of local anesthetic solutions and less vascular punctures, and in the event that intravascular injections do occur, they are easily recognized with sonography [80, 81]. The dynamic feedback from ultrasound during online needle placement and injection also may influence this risk. Because less vascular punctures have been reported when ultrasound imaging is utilized, presumably less bleeding complications occur. Aberrant distributions of injected local anesthetic should be recognized and less likely with low volumes of local anesthetic injection. However, with offline technique the injections will not be visualized and therefore the usual precautions for intravascular injection are mandatory.

Although the use of ultrasound imaging for lumbar plexus block is new, complications already have been reported. Subarachnoid placement of a catheter intended for lumbar plexus anesthesia occurred when ultrasound imaging and nerve stimulation were combined for guidance [37]. Events such as this emphasize limitations with these approaches and underscore the continued need for clinical awareness of potential complications.

Alternatives

Lumbar plexus block has compared favorably to other anesthetic techniques. Lumbar plexus blocks reduce pain and blood loss after major lower extremity procedures compared with general anesthesia alone [82]. Lumbar plexus anesthesia has several advantages over spinal or epidural anesthesia, including less hypotension, urinary retention, and bleeding issues. Lumbar plexus block provides more extensive anesthesia than femoral block, although for some surgical procedures, posterior lumbar plexus blocks offer no advantages over three-in-one femoral blocks [83].

Conclusion

Ultrasound imaging can be used to guide lumbar plexus block in identifying the transverse processes and psoas muscle. Although direct nerve imaging can be used for guidance for other regional block procedures, this is not routinely possible for the lumbar plexus in adults. Ultrasound imaging of bony and muscular landmarks may be especially useful for patients with spinal deformities or obesity, although suboptimal imaging can sometimes be encountered. It is not clear at this time if sonographic guidance, which is relatively new, will reduce the risks of lumbar plexus block. However, this

technology has shown tremendous promise for improving efficacy of block procedures and may improve safety.

References

1. Farny J, Drolet P, Girard M. Anatomy of the posterior approach to the lumbar plexus block. Can J Anaesth. 1994;41:480–5.
2. Hanna M, Peat SJ, D'Costa F. Lumbar plexus block: an anatomical study. Anesthesia. 1993;48:675–8.
3. Kirchmair L, Entner T, Wissel J, et al. A study of the paravertebral anatomy for ultrasound guided posterior lumbar plexus block. Anesth Analg. 2001;93:477–8.
4. Netter FH. Nervous system. Part 1. Anatomy and physiology. The Ciba collection of medical illustrations. West Caldwell, NJ; 1991. Plates 1–16.
5. Jankovic D. Anatomie des plexus lumbalis, sacralis und coccygeus. In: Jankovic D, editor. Regionalblockaden & Infiltrationstherapie. 4th ed. Berlin: ABW; 2007. p. 316–8.
6. Winnie AP, Ramamurthy S, Durani Z. The inguinal paravascular technic of lumbar plexus anesthesia: the "3-in-1" block. Anesth Analg. 1973;52:989–96.
7. Winnie A. Regional anesthesia. Surg Clin North Am. 1975; 55:861–92.
8. Winnie A, Ramamurthy S, Durrani Z, Radonjic R. Plexus blocks for lower extremity surgery. Anesthesiol Rev. 1974;1:11–6.
9. Ritter J. Femoral nerve "sheath" for inguinal paravascular lumbar plexus block is not found in human cadavers. J Clin Anesth. 1995;7:470–3.
10. Marhofer P, Nasel C, Sitzwohl C, Karpal S. Magnetic resonance imaging of the distribution of local anesthetic during the three-in-one block. Anesth Analg. 2000;90:119–24.
11. Lang S, Yip R, Chang P, Gerard M. The femoral 3-in-1 block revisited. J Clin Anesth. 1993;5:292–6.
12. Lang S. Electromyographic comparison of obturator nerve block to 3-in-1 block. Anesth Analg. 1996;83:436–7.
13. Cauhepe C, Oliver M, Colombani R, et al. The "3-in-1" block: myth or reality? Ann Fr Anesth Reanim. 1989;8:376–8.
14. Capdevila X, Biboulet P, Bouregba M, et al. Comparison of the three-in-one and fascia iliaca compartment blocks in adults: Clinical and radiographic analysis. Anesth Analg. 1998;86:1039–44.
15. Elmas C, Atanassoff P. Combined inguinal paravascular (3 in 1) and sciatic nerve blocks for lower limb surgery. Reg Anesth. 1993;18:88–92.
16. Misra U, Pridie AK, McClymont C, Bower S. Plasma concentrations of bupivacaine following combined sciatic and femoral 3 in 1 nerve blocks in open knee surgery. Br J Anaesth. 1991;66:310–3.
17. Berkowitz A, Rosenberg H. Femoral block with mepivacaine for muscle biopsy in malignant hyperthermia patients. Anesthesiology. 1985;62:651–2.
18. Singelyn FJ, Gouverneur JM. The continuous "3-in-1" block as postoperative pain treatment after hip, femoral shaft or knee surgery: a large scale study of efficacy and side effects. Anesthesiology. 1994;81:1064.
19. Hirst GC, Lang SA, Dust WN, Cassidy D, Yip RW. Femoral nerve block. Single injection versus continuous infusion for total knee arthroplasty. Reg Anesth. 1996;21(4):292–7.
20. Ringrase NH, Cross MJ. Femoral nerve block in knee joint surgery. Am J Sports Med. 1984;12:398–402.
21. Rooks M, Fleming LL. Evaluation of acute knee injuries with sciatic femoral nerve blocks. Clin Orthop. 1983;179:185–8.
22. Singelyn F, Vanderelst P, Gouverneur J. Extended femoral nerve sheath block after total hip arthroplasty: continuous versus patient-controlled techniques. Anesth Analg. 2001;92:455–9.
23. Capdevila X, Biboulet P, Morau D, Bernard N, Deschodt J, Lopez S, d'Athis F. Continuous three-in-one block for postoperative pain after lower limb orthopaedic surgery: Where do the catheters go? Anesth Analg. 2002;94:1001–6.
24. Rosenblatt R. Continuous femoral anesthesia for lower extremity surgery. Anesth Analg. 1980;59:631.
25. Frerk CM. Palsy after femoral nerve block. Anaesthesia. 1988;43:167–8.
26. Lynch J. Prolonged motor weakness after femoral nerve block with bupivacaine 0,5 %. Anaesthesia. 1990;45:421.
27. Indelli PF, Grant SA, Nielsen K, et al. Regional anesthesia in hip surgery. Clin Orthop Relat Res. 2005;441:250–5.
28. Bogoch ER, Hence M, Mackenzie T, et al. Lumbar paravertebral nerve block in the management of pain after total hip and knee arthroplasty: a randomized controlled trial. J Arthroplasty. 2002;17:398–401.
29. Robards C, Hadzic A. Lumbar plexus block. In: Hadzic A, editor. Textbook of regional anesthesia and acute pain management. New York, Chicago, Sydney, Toronto; 2007. p. 481–8.
30. Chayen D, Nathan H, Clayen M. The psoas compartment block. Anesthesiology. 1976;45:95–9.
31. Mannion S, Barrett J, Kelly D, et al. A description of the spread of injectate after psoas compartment block using magnetic resonance imaging. Reg Anesth Pain Med. 2005;30:567–71.
32. Capdevila X, Macaire P, Dadure C, Choquet O, Biboulet P, Ryckwaert YD, D'Athis F. Continuous psoas compartment block for postoperative analgesia after total hip arthroplasty: new landmarks, technical guidelines and clinical evaluation. Anesth Analg. 2002;94:1606–13.
33. Chelly JE, Casati A, Al-Samsam T, et al. Continuous lumbar plexus block for acute postoperative pain management after open reduction and internal fixation of acetabular fractures. J Orthop Trauma. 2003;17:362–7.
34. Blumenthal S, Ekatodramis G, Borgeat A. Ropivacaine plasma concentrations are similar during continuous lumbar plexus blockade using two techniques: pharmacokinetics or pharmacodynamics? Can J Anaesth. 2004;51:851.
35. Huet O, Eyrolle LJ, Mazoit JX, et al. Cardiac arrest after injection of ropivacaine for posterior lumbar plexus blockade. Anesthesiology. 2003;99:1451–3.
36. Pham-Dang C, Beaumont S, Floch H, et al. Acute toxic accident following lumbar plexus block with bupivacaine. Ann Fr Anesth Reanim. 2000;19:356–9.
37. Litz RJ, Vicent O, Wiessner D, et al. Misplacement of a psoas compartment catheter in the subarachnoid space. Reg Anesth Pain Med. 2004;29:60–4.
38. Gentilli M, Aveline C, Bonnet F. Total spinal anesthesia after posterior lumbar plexus block. Ann Fr Anesth Reanim. 1998;17:740–2.
39. Aida S, Takahashi H, Shimoji K. Renal subcapsular hematoma after lumbar plexus block. Anesthesiology. 1996;84:452–5.
40. Hsy DT. Delayed retroperitoneal hematoma after failed lumbar plexus block. Br J Anaesth. 2005;93:589–91.
41. Aveline C, Bonnet F. Delayed retroperitoneal hematoma after failed lumbar plexus block. Br J Anaesth. 2004;93:589–91.
42. Reddy MB. Pneumocoele following psoas compartment block. Anesthesia. 2002;57:938–9.
43. Barrington MJ, Wong DM, Slater B, Ivanusic JJ, Ovens M. Ultrasound-guided regional anesthesia: how much practice do novices require before achieving competency in ultrasound needle visualization using a cadaver model. Reg Anesth Pain Med. 2012;37(3):334–9. doi:10.1097/AAP.0b013e3182475fba.
44. Chin KJ, Tse C, Chan V. Ultrasonographic identification of an anomalous femoral nerve: the fascia iliaca as a key landmark. Anesthesiology. 2011;115(5):1104. doi:10.1097/ALN.0b013e3182241d72.
45. Fanara B, Christophe JL, Boillot A, Tatu L, Jochum D, Henri Y, Berthier F, Samain E. Ultrasound guidance of needle tip position for

femoral nerve blockade: an observational study. Eur J Anaesthesiol. 2014;31(1):23–9. doi:10.1097/01.EJA.0000435016.83813.aa.
46. Fredrickson MJ, Danesh-Clough TK. Ultrasound-guided femoral catheter placement: a randomised comparison of the in-plane and out-of-plane techniques. Anaesthesia. 2013;68(4):382–90. doi:10.1111/anae.12119.
47. Hocking G. Anomalous positioning of femoral artery and vein–even 'constant' landmarks can be inconsistent. Anaesth Intensive Care. 2011;39(2):312–3.
48. Mariano ER, Kim TE, Funck N, Walters T, Wagner MJ, Harrison TK, Giori N, Woolson S, Ganaway T, Howard SK. A randomized comparison of long-and short-axis imaging for in-plane ultrasound-guided femoral perineural catheter insertion. J Ultrasound Med. 2013;32(1):149–56.
49. Muhly WT, Orebaugh SL. Ultrasound evaluation of the anatomy of the vessels in relation to the femoral nerve at the femoral crease. Surg Radiol Anat. 2011;33(6):491–4. doi:10.1007/s00276-010-0755-9.
50. Ruiz A, Sala-Blanch X, Martinez-Ocon J, Carretero MJ, Sanchez-Etayo G, Hadzic A. Incidence of intraneural needle insertion in ultrasound-guided femoral nerve block: a comparison between the out-of-plane versus the in-plane approaches. Rev Esp Anestesiol Reanim. 2014;61(2):73–7. doi:10.1016/j.redar.2013.09.023.
51. 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. doi:10.1016/j.rapm.2005.05.012.
52. Szucs S, Morau D, Sultan SF, Iohom G, Shorten G. A comparison of three techniques (local anesthetic deposited circumferential to vs. above vs. below the nerve) for ultrasound guided femoral nerve block. BMC Anesthesiol. 2014;14:6. doi:10.1186/1471-2253-14-6.
53. Taha AM, Abd-Elmaksoud AM. Ropivacaine in ultrasound-guided femoral nerve block: what is the minimal effective anaesthetic concentration (EC90)? Anaesthesia. 2014;69(7):678–82. doi:10.1111/anae.12607.
54. Watson MJ, Walker E, Rowell S, Halliday S, Lumsden MA, Higgins M, Binning A, McConnachie A. Femoral nerve block for pain relief in hip fracture: a dose finding study. Anaesthesia. 2014;69(7):683–6. doi:10.1111/anae.12683.
55. Kirchmair L, Entner T, Kapral S, Mitterschiffthaler G. Ultrasound guidance for the psoas compartment block: an imaging study. Anesth Analg. 2002;94:706–10.
56. Hanna MH, Peat SJ, D'Costa F. Lumbar plexus block: an anatomical study. Anaesthesia. 1993;48:675–8.
57. Ebraheim NA, Xu R, Huntoon M, Yeasting RA. Location of the extraforaminal lumbar nerve roots. An anatomic study. Clin Orthop Relat Res. 1997;340:230–5.
58. Karmakar MK. Thoracic paravertebral block. Anesthesiology. 2001;95:771–80.
59. Gu Y, Ebraheim NA, Xu R, Rezcallah AT, Yeasting RA. Anatomic considerations of the posterolateral lumbar disk region. Orthopedics. 2001;24:56–8.
60. Grau T, Leipold RW, Horter J, Conradi R, Martin EO, Motsch J. Paramedian access to the epidural space: the optimum window for ultrasound imaging. J Clin Anesth. 2001;13:213–7.
61. Pusch F, Wildling E, Klimscha W, Weinstabl C. Sonographic measurement of needle insertion depth in paravertebral blocks in women. Br J Anaesth. 2000;85:841–3.
62. King AD, Hine AL, McDonald C, Abrahams P. The ultrasound appearance of the normal psoas muscle. Clin Radiol. 1993;48:316–8.
63. Kirchmair L, Enna B, Mitterschiffthaler G, Moriggl B, Greher M, Marhofer P, Kapral S, Gassner I. Lumbar plexus in children. A sonographic study and its relevance to pediatric regional anesthesia. Anesthesiology. 2004;101:445–50.
64. Gray AT, Collins AB, Schafhalter-Zoppoth I. An introduction to femoral nerve and associated lumbar plexus nerve blocks under ultrasonic guidance. Tech Reg Anesth Pain Manag. 2004;8:155–63.
65. Suzuki S, Yamamuro T, Shikata J, Shimizu K, Iida H. Ultrasound measurement of vertebral rotation in idiopathic scoliosis. J Bone Joint Surg Br. 1989;71:252–5.
66. Yeo ST, French R. Combined spinal-epidural in the obstetric patient with Harrington rods assisted by ultrasonography. Br J Anaesth. 1999;83:670–2.
67. McLeod A, Roche A, Fennelly M. Case series: Ultrasonography may assist epidural insertion in scoliosis patients. Can J Anaesth. 2005;52:717–20.
68. Furness G, Reilly MP, Kuchi S. An evaluation of ultrasound imaging for identification of lumbar intervertebral level. Anaesthesia. 2002;57:277–80.
69. Watson MJ, Evans S, Thorp JM. Could ultrasonography be used by an anaesthetist to identify a specified lumbar interspace before spinal anaesthesia? Br J Anaesth. 2003;90:509–11.
70. Hahn PY, Strobel JJ, Hahn FJ. Verification of lumbosacral segments on MR images: identification of transitional vertebrae. Radiology. 1992;182:580–1.
71. Castellvi AE, Goldstein LA, Chan DP. Lumbosacral transitional vertebrae and their relationship with lumbar extradural defects. Spine. 1984;9:493–5.
72. Glass RB, Norton KI, Mitre SA, Kang E. Pediatric ribs: a spectrum of abnormalities. Radiographics. 2002;22:87–104.
73. Capdevila X, Coimbra C, Choquet O. Approaches to the lumbar plexus: success, risks, and outcome. Reg Anesth Pain Med. 2005;30:150–62.
74. Pousman RM, Mansoor Z, Sciard D. Total spinal anesthetic after continuous posterior lumbar plexus block. Anesthesiology. 2003;98:1281–2.
75. Breslin DS, Martin G, Macleod DB, D'ercole F, Grant SA. Central nervous system toxicity following the administration of levobupivacaine for lumbar plexus block: A report of two cases. Reg Anesth Pain Med. 2003;28:144–7.
76. Klein SM, D'Ercole F, Greengrass RA, Warner DS. Enoxaparin associated with psoas hematoma and lumbar plexopathy after lumbar plexus block. Anesthesiology. 1997;87:1576–9.
77. Weller RS, Gerancher JC, Crews JC, Wade KL. Extensive retroperitoneal hematoma without neurologic deficit in two patients who underwent lumbar plexus block and were later anticoagulated. Anesthesiology. 2003;98:581–5.
78. Klein SM, Greengrass RA, Weltz C, Warner DS. Paravertebral somatic nerve block for outpatient inguinal herniorrhaphy: an expanded case report of 22 patients. Reg Anesth Pain Med. 1998;23:306–10.
79. Lonnqvist PA, MacKenzie J, Soni AK, Conacher ID. Paravertebral blockade. Failure rate and complications. Anaesthesia. 1995;50:813–5.
80. Marhofer P, Schrogendorfer K, Wallner T, Koinig H, Mayer N, Kapral S. Ultrasonographic guidance reduces the amount of local anesthetic for 3-in-1 blocks. Reg Anesth Pain Med. 1998;23:584–8.
81. Sandhu NS, Bahniwal CS, Capan LM. Feasibility of an infraclavicular block with a reduced volume of lidocaine with sonographic guidance. J Ultrasound Med. 2006;25:51–6.
82. Stevens RD, Van Gessel E, Flory N, Fournier R, Gamulin Z. Lumbar plexus block reduces pain and blood loss associated with total hip arthroplasty. Anesthesiology. 2000;93:115–21.
83. Kaloul I, Guay J, Cote C, Fallaha M. The posterior lumbar plexus (psoas compartment) block and the three-in-one femoral nerve block provide similar postoperative analgesia after total knee replacement. Can J Anaesth. 2004;51:45–51.

Chapter 60

Proximal Sciatic Nerve Block-Ultrasound Guided

Faraj W. Abdallah (✉)
Department of Anesthesia, St. Michael's Hospital, University of Toronto, 30 Bond Street, Toronto, ON, M5B 1 W8, Canada
e-mail: AbdallahF@smh.ca

Vincent W. Chan, MD, FRCPC
Department of Anesthesia, Toronto Western Hospital, University of Toronto, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: mail2vincechan@aol.com

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_60,

Anatomy

The sciatic nerve is the largest peripheral nerve in the human body and is a branch of the lumbosacral plexus formed by the L4, L5 lumbar nerves and the ventral rami of S1–3 sacral nerves [30] (Fig. 60.1). The proximal portion of the sciatic nerve runs deep in the gluteal region between the gluteus maximus muscle posteriorly and the quadratus femoris, obturator internus, and gemelli muscles anteriorly [18] and is accompanied by the posterior femoral cutaneous nerve and the inferior gluteal artery. The sciatic nerve exits the pelvis through the greater sciatic notch, below the piriformis muscle, descending between the ischial tuberosity and greater trochanter of the femur [26]. The sciatic nerve then runs along the posterior thigh, anterior to the biceps femoris muscle, and bifurcates into the common peroneal nerve and tibial nerve in the distal third of the femur [14]. The level at which the sciatic nerve bifurcates is variable and may occur at anywhere between the piriformis muscle and the popliteal fossa [27].

The sciatic nerve provides sensory and motor innervation to the lower extremity. Its articular branches innervate the hip joint through the posterior capsule, the posterior knee joint, and the ankle joint [14, 18, 26]. Its muscular branches innervate muscles in the gluteal area (gluteus maximus, medius, and minimus muscles) and the thigh (adductor magnus, long and short heads of biceps femoris, semitendinosus, semimembranosus). Distally, the sciatic nerve provides innervation to the leg, ankle, and foot.

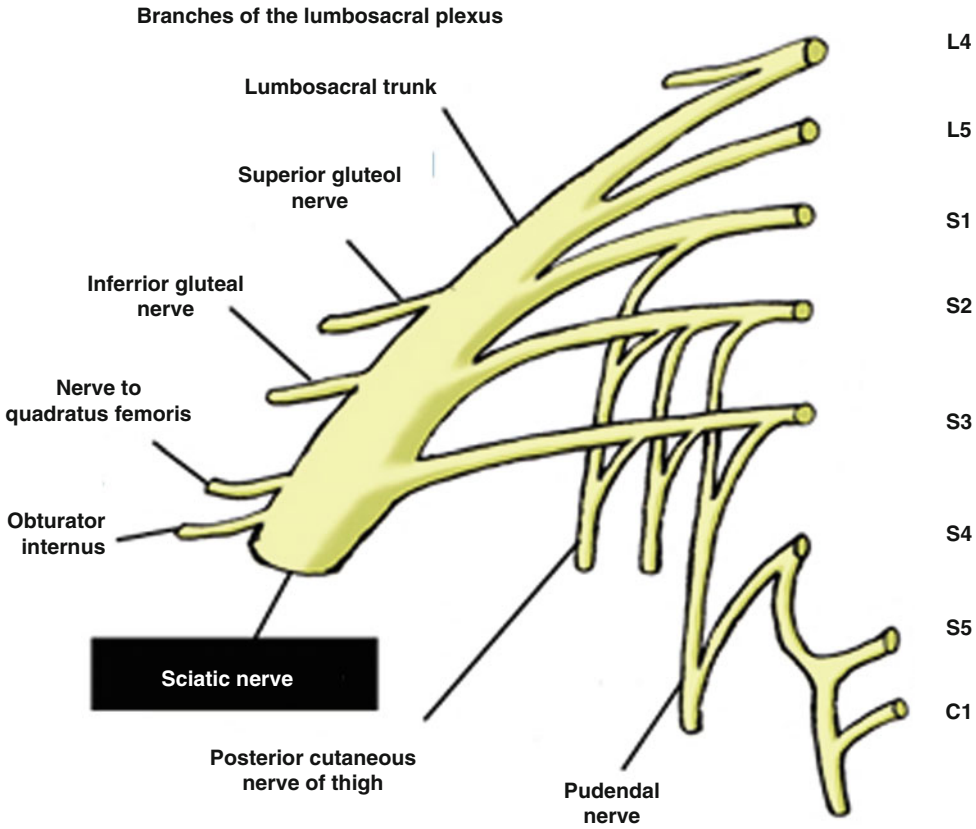


Fig. 60.1 Lumbosacral plexus (Figures are reproduced with permission of www.USRA.ca)

Ultrasound-Guided Block Technique

This chapter will limit the description of ultrasound-guided sciatic nerve block techniques to approaches in the gluteal, infragluteal, and the proximal thigh regions.

Gluteal Approach

Patient Positioning

(a) Place the patient in the semiprone (Sims) position with the hip and knee flexed and the operative side up.
(b) Identify the greater trochanter (GT) of the femur, the posterior superior iliac spine (PSIS), and the sacral hiatus (SH) (Fig. 60.2).

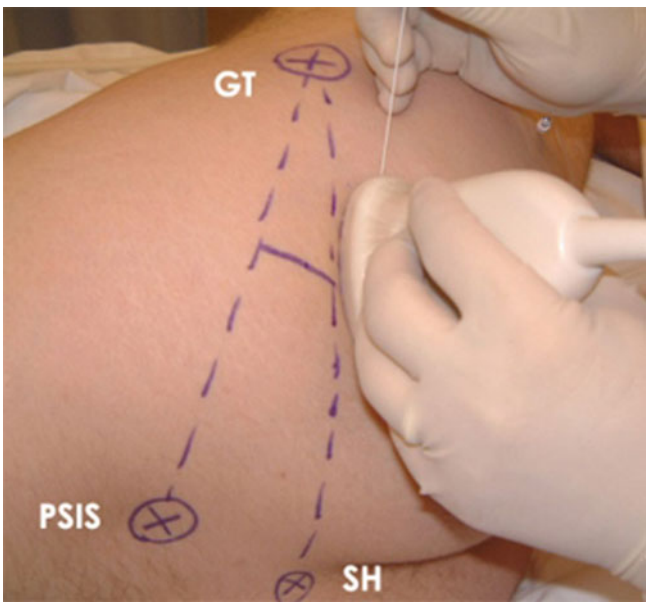


Fig. 60.2 Gluteal approach surface landmarks. *GT* greater trochanter of the femur, *PSIS* posterior superior iliac spine, *SH* sacral hiatus (Figures are reproduced with permission of www.USRA.ca)

Sonographic Imaging

(c) Sterilize the skin and scan with a low-frequency curvilinear 2–5-MHz ultrasound transducer covered by a sterile adhesive dressing.
(d) Place the transducer in the transverse plane on the buttock at approximately the level of the ischial tuberosity.
(e) Optimize the machine setting to capture a short-axis view of the sciatic nerve. A depth >4 cm is usually required.
(f) Start by moving the transducer cephalad from the ischial tuberosity level to identify the internal bony landmark ischial bone which casts a wide bony acoustic shadow. The most medial aspect of this bone is the ischial spine.
(g) Often another bony landmark, the greater trochanter or the hip joint, is seen lateral to the ischial bone.
(h) Next locate the muscular landmarks, the gluteus maximus muscle (the outer muscle layer) and the superior and inferior gemellus muscles (the inner muscle layers) that are all superficial and posterior to the ischial bone.
(i) The sciatic nerve is expected to be visualized deep (anterior) to the gluteus maximus muscle layer and superficial (posterior) to the superior and inferior gemellus muscles as well as the ischial bone.
(j) The sciatic nerve in the gluteal region is usually hyperechoic (bright), wide and thin in the cross-sectional (short-axis) view (Fig. 60.3).
(k) The pulsatile inferior gluteal artery is often visualized in close proximity to the sciatic nerve in this region. Use color Doppler to confirm.
(l) It may also be possible to locate the internal pudendal vessels (artery and vein) near the ischial spine that are significantly medial to the sciatic nerve.
(m) Scan the nerve proximally and distally to follow its course and confirm its identity in both the short-axis and long-axis views.

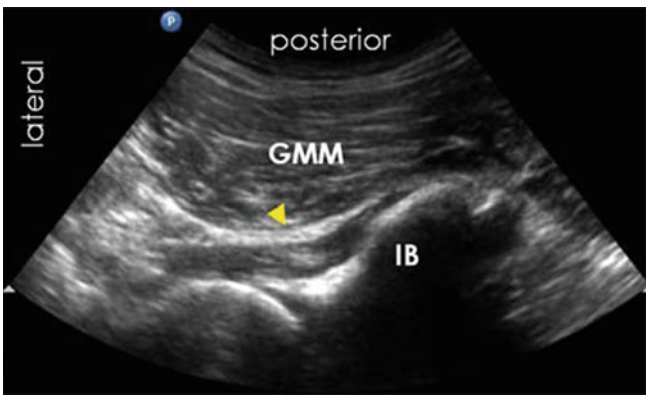


Fig. 60.3 Gluteal approach sonographic appearance. *Arrow* designates sciatic nerve. *IB* ischial bone, *GMM* gluteus maximus muscle (Figures are reproduced with permission of www.USRA.ca)

Needling

(n) Ultrasound-guided gluteal sciatic nerve block is considered an advanced block technique.

(o) The gluteal sciatic nerve block is amenable to both in-plane (IP) and out-of-plane (OOP) approaches. One may prefer the OOP approach for blocking this nerve in this deep location because the block needle travels the shortest skin to nerve distance. Also, the OOP approach is often used for catheter insertion. For single-shot injection, following local anesthetic skin infiltration, an 80–100-mm 22G insulated block needle is inserted often under combined ultrasound and nerve stimulation guidance because ultrasound alone may not always visualize accurately the needle tip or nerve location. After obtaining a satisfactory ultrasound and nerve stimulation response endpoint, a bolus of local anesthetic, 15–20 mL, is injected incrementally for postoperative analgesia. A dilute bupivacaine (0.25 % or less) or ropivacaine (0.5 % or less) solution is the local anesthetic of choice to achieve analgesia without a prolonged foot drop.

(p) For the IP approach, the block needle is advanced in a lateral-to-medial direction through the gluteus maximus muscle, along the long axis of the transducer, in the plane of the ultrasound beam. This allows needle visualization in real-time during needle advancement.

(q) The technical challenge with this block often relates to needle-tip visualization which is hampered by the steep angle of needle insertion. Hydrolocation, i.e., injecting small volumes of local anesthetic (1–2 mL) is recommended to serve as a surrogate marker of the needle-tip position.

(r) Another technical challenge is to visualize the nerve in short axis since it may appear thin and wide. In this case, it is helpful to rotate the transducer 90° to locate the nerve in the long-axis view which can show a hyperechoic longitudinal structure running cephalad and caudad.

(s) Electrical nerve stimulation to elicit twitches in the muscle groups innervated by the tibial nerve and/or peroneal nerve further confirms needle-to-nerve contact.

(t) The ideal endpoint for local anesthetic injection is to achieve a circumferential spread pattern around the sciatic nerve. Needle-tip position may be adjusted as needed to achieve this endpoint. However, it is not always practical or possible to deposit local anesthetic deep to the nerve and one must be cautious to avoid multiple needle attempts and the potential risk of needle trauma to the nerve.

(u) For the OOP approach, the sciatic nerve image is aligned at the midpoint of the transducer and the block needle is inserted inferior to the transducer to reach the nerve target.

(v) Needle visualization: As in the section "Gluteal approach" subsection "Needling" q, above.

(w) Electric stimulation: As in the section "Gluteal approach" subsection "Needling" s, above.

(x) Injection endpoint: As in the section "Gluteal approach" subsection "Needling" t, above.

Subgluteal Approach

Patient Positioning

(a) Place the patient in the semiprone (Sims) position with the hip and knee flexed and the operative side up.

(b) Identify the GT of the femur and the ischial tuberosity (IT) (Fig. 60.4).

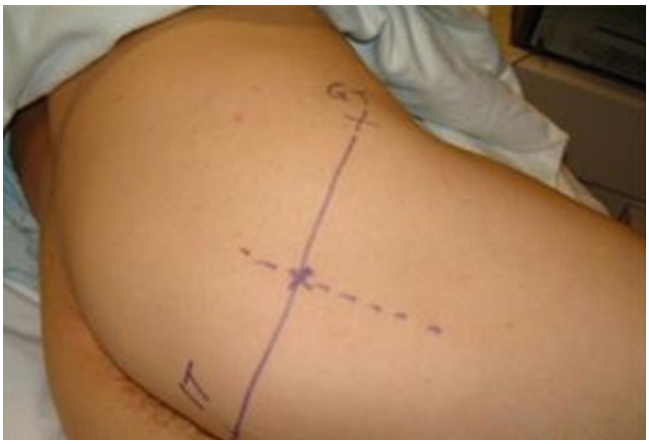


Fig. 60.4 Infragluteal approach surface landmarks. *GT* greater trochanter of the femur, *IT* ischial tuberosity (Figures are reproduced with permission of www.USRA.ca)

Sonographic Imaging

(c) Sterilize the skin and scan with a low-frequency curvilinear 2–5-MHz ultrasound transducer covered by a sterile adhesive dressing.
(d) Place the transducer oblique over the subgluteal region of the buttock to capture a cross-sectional view of the sciatic nerve.
(e) Optimize the machine setting to proper depth; 3–4 cm is often required.
(f) Identify the internal bony landmarks, the greater trochanter (GT) laterally, and the ischial tuberosity (IT) medially.
(g) Identify the muscular landmarks, the gluteus maximus muscle (GMM), the outer muscle layer, and the quadratus femoris muscle (QFM), the inner muscle layer.
(h) The sciatic nerve often appears hyperechoic and elliptical in the subgluteal region. It is expected to be visualized in the fascial plane between the GMM and the QFM (Fig. 60.5).
(i) The sciatic nerve is also expected to be found between the GT laterally and IT medially.
(j) Scan the nerve proximally and distally to follow its course and confirm its identity.
(k) If nerve visualization is difficult in the short axis, it is helpful to turn the patient prone and scan to visualize the sciatic nerve in long axis.

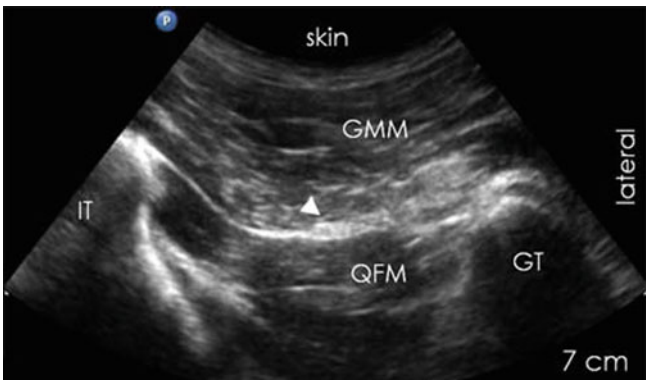


Fig. 60.5 Infragluteal approach sonographic appearance. *GMM* gluteus maximus muscle, *GT* greater trochanter of the femur, *IT* ischial tuberosity, *QFM* quadratus femoris muscle. The sciatic nerve is indicated by the *arrow* (Figures are reproduced with permission of www.USRA.ca)

Needling

(l) Ultrasound-guided subgluteal sciatic nerve block is considered an intermediate level block technique. The sciatic nerve in this region is often less deep compared to the gluteal and proximal thigh regions.
(m) The subgluteal sciatic nerve block is amenable to both in-plane and out-of-plane approaches. The block requires a shorter needle, e.g., a 50- or 80-mm 22G insulated block needle, and a total volume of 15–20-mL volume of local anesthetic is used for postoperative analgesia.
(n) Visualization of the sciatic nerve in this region can also be challenging because of the required depth of beam penetration and the attenuation of ultrasound energy by the overlying adipose tissue layers.
(o) Needle visualization: as in the section "Gluteal approach" subsection "Needling" q, above.
(p) Electric stimulation: as in the section "Gluteal approach" subsection "Needling" s, above.
(q) Injection endpoint: as in the section "Gluteal approach" subsection "Needling" t, above. Exercise caution to avoid multiple needle attempts and potential nerve trauma.

Proximal Thigh (Anterior) Approach

Patient Positioning

(a) Place the patient supine with the hip externally rotated and the hip and knee slightly flexed (Fig. 60.6).
(b) The sciatic nerve in the proximal thigh is expected to lie between the medial thigh compartment containing the adductor muscles (adductor longus, brevis, and magnus muscles) and the posterior thigh compartment containing the biceps femoris muscle.

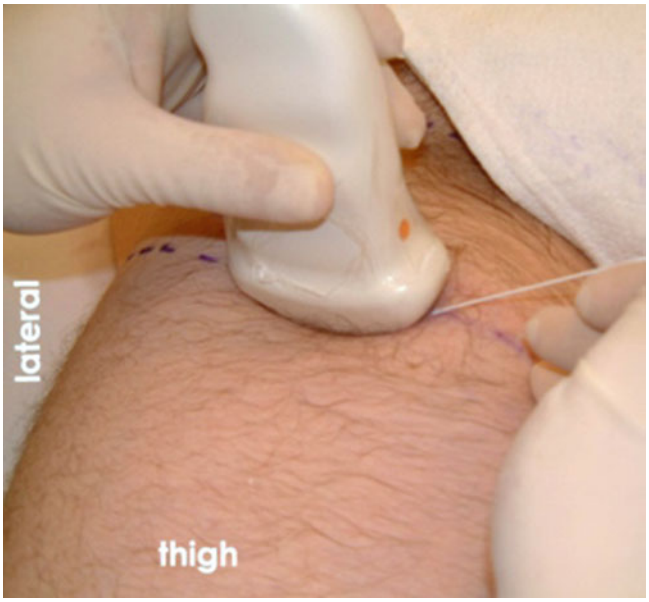


Fig. 60.6 Anterior approach surface landmarks (Figures are reproduced with permission of www.USRA.ca)

Sonographic Imaging

(c) Sterilize the skin and scan with a low-frequency curvilinear 2–5-MHz ultrasound transducer covered by a sterile adhesive dressing.
(d) Place the transducer transverse over the proximal thigh, approximately 8 cm from the inguinal crease, to capture a cross-sectional view of the sciatic nerve (Fig. 60.6).
(e) Optimize the machine setting and focus to a depth usually >6 cm.
(f) Identify the femur, a bony landmark that casts a curved hyperechoic outline and an underlying acoustic shadow.
(g) When scanned more proximally (cephalad), the femur bone shadow changes from curved to a flat surface. This represents the lesser trochanter of the femur. Aim to visualize the sciatic nerve caudad to the lesser trochanter.
(h) Identify the muscular landmarks, the anterior compartment group of muscles (adductor muscles) medially and the muscle in the posterior compartment of the thigh, biceps femoris muscle more distally, and the gluteus maximus muscle (GMM) more proximally.
(i) Identify the sciatic nerve that is posterior to the femur (i.e., the sciatic nerve image is situated farther away from the skin than the femur). The nerve is also between the adductor muscle group and the posterior GMM (more proximally) or the BFM (more distally).
(j) Viewed in short axis, the sciatic nerve in the proximal thigh often appears hyperechoic and elliptical (Fig. 60.7).
(k) If the femoral bone shadow obstructs the view of the sciatic nerve, move the transducer more medially. In essence, the transducer is now positioned in an anteroposterior direction because the hip is externally rotated. The sciatic nerve is visualized posterior to the femur.
(l) Scan the nerve proximally and distally to follow its course and confirm its identity.
(m) The sciatic nerve in the proximal thigh may be difficult to visualize because of the required depth of ultrasound beam penetration and the requirement for lower-frequency transducer.

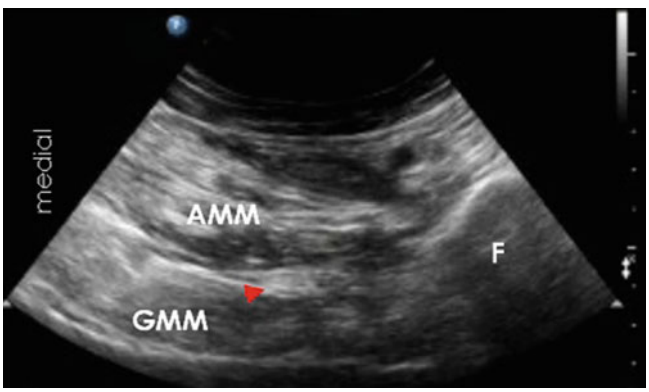


Fig. 60.7 Anterior approach sonographic appearance. *AMM* adductor magnus muscles, *GMM* gluteus maximus muscle, *F* femur. The sciatic nerve is indicated by the *arrow* (Figures are reproduced with permission of www.USRA.ca)

Needling

(n) Ultrasound-guided sciatic nerve block in the proximal thigh is considered an advanced block technique because of the required depth of needle insertion.
(o) The sciatic nerve block in this region is amenable to both in-plane and out-of-plane approaches and follows the same skin sterilization procedure and local anesthetic injection volume. A long needle, e.g., a 80–120-mm 22-G insulated block needle is required depending on the size of the thigh.
(p) For the in-plane approach, the needle is advanced in a medial-to-lateral, anterior-to-posterior direction, along the long axis of the transducer, in the plane of the ultrasound beam.
(q) With the hip externally rotated, the femoral neurovascular bundle is now situated laterally and is far away from the path of needle insertion thus unlikely unintentional vascular puncture.
(r) Needle visualization: as in the section "Gluteal approach" subsection "Needling" q, above. As steep angle is required for needle insertion, needle-tip and shaft visualization is very challenging.
(s) Electric stimulation: as in the section "Gluteal approach" subsection "Needling" s, above.
(t) Injection endpoint: as in the section "Gluteal approach" subsection "Needling" t, above.
(u) For the out-of-plane approach, the sciatic nerve is aligned at the midpoint of the transducer and the needle is also inserted inferior to the probe.
(v) Similar approach to follow needle-tip position (tissue movement and hydrolocation), to confirm needle-to-nerve contact (electrical stimulation), and to inject local anesthetic is recommended as for IP approach.

Clinical Utility

The proximal sciatic nerve block (SNB) can provide analgesic benefits following several orthopedic procedures, including hip arthroplasty [6], below knee amputation [10], as well as foot and ankle surgery [12]. Nonetheless, it is most commonly used in the setting of total knee arthroplasty (TKA) [3], in combination with femoral nerve block. Though a few observational studies suggested that SNB effectively treats posterior knee pain following TKA [5, 28], evidence supporting the routine administration of SNB has been weak [2, 21], and the question of whether SNB should routinely be added to femoral nerve block remains unanswered [1, 17]. However, additional relevant trials have been published recently [8, 9, 15, 19, 23–25, 29, 31], enabling further attempts at answering this question. This section of the chapter will quantitatively examine the evidence supporting the use of SNB in TKA.

We systematically reviewed the literature to investigate whether single-shot SNB reduces rest pain at 6 h postoperatively. This time point limits the effect of heterogeneity of the treatment effect introduced by using local anesthetics of varying doses and durations of action in the trials reviewed. Furthermore, this time point has been frequently selected to assess analgesic outcomes in trials evaluating analgesic effects of nerve blocks following TKA [2, 11, 13, 21]. We also assessed the cumulative opioid medications consumed during the 0–12-h interval postoperatively. For the purpose of this chapter, pain was measured on a visual analog scale (VAS; 10-cm scale where 0 = no pain, 10 = worst pain), while all opioid medications consumed during the first 12 h postoperatively were converted into equianalgesic doses of intravenous (IV) morphine [7].

A total of five randomized controlled trials published between 1998 and 2014 addressed this question [4, 9, 16, 23, 29]. Data relating to a total of 256 patients were available for analysis: 129 patients in the SNB group and 127 in the control group. Both groups received femoral nerve block. Data relating to postoperative rest pain at 6 h were available from four trials [4, 16, 23, 29]. Compared to control, SNB reduced rest pain VAS scores at 6 h postoperatively by 1.38 cm [−2.48, −0.27] (P=0.01) (Fig. 60.8). Additionally, data relating to postoperative morphine equivalent consumption during the 0–12-h interval were available from four trials [4, 16, 23, 29]. Compared to control, SNB reduced the postoperative IV morphine equivalent consumption by 7.32 mg [−14.09, −0.54], (P=0.03) during the 0–12-h interval postoperatively (Fig. 60.9).

This quantitative review of evidence suggests that adding single-shot SNB to a femoral nerve block improves analgesia following TKA by reducing rest pain severity for at least 6 h and decreasing the 0–12-h postoperative opioid consumption. These results contradict earlier evidence provided by two meta-analyses [21, 13], as well as two systematic reviews [2, 11], which concluded that adding SNB to femoral nerve block in patients undergoing TKA was not associated with additional benefits. Though not included in this analysis, using a catheter-based infusion seems to prolong the SNB benefits observed [22, 20]. Despite these benefits, earlier concerns regarding SNB-related injury, surgical injury, and delayed mobilization still apply [1, 17].

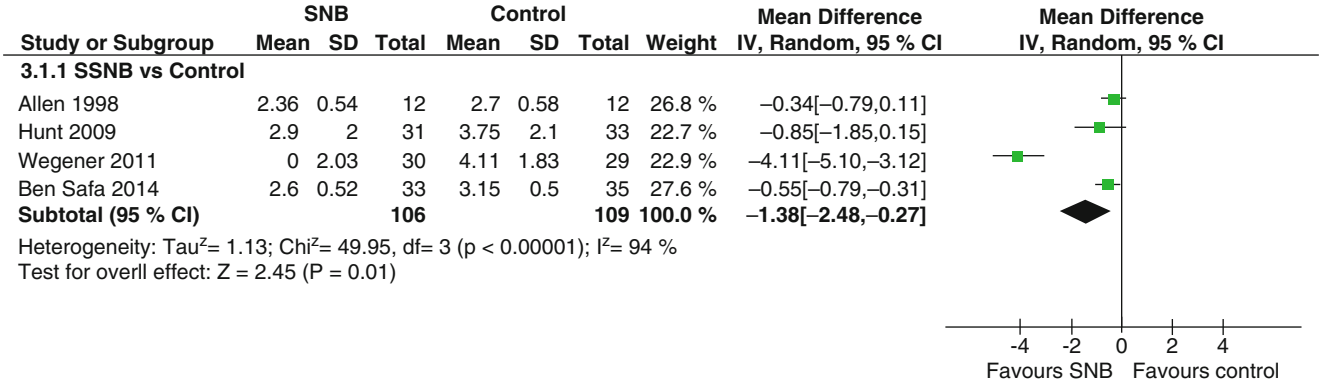


Fig. 60.8 Rest pain at 6-h forest plot. The sample size, mean, standard deviations, and the pooled estimates of the mean difference are shown. The 95 % CIs are shown as *lines* for individual studies and as *diamonds* for pooled estimates (Figures are reproduced with permission of www.USRA.ca)

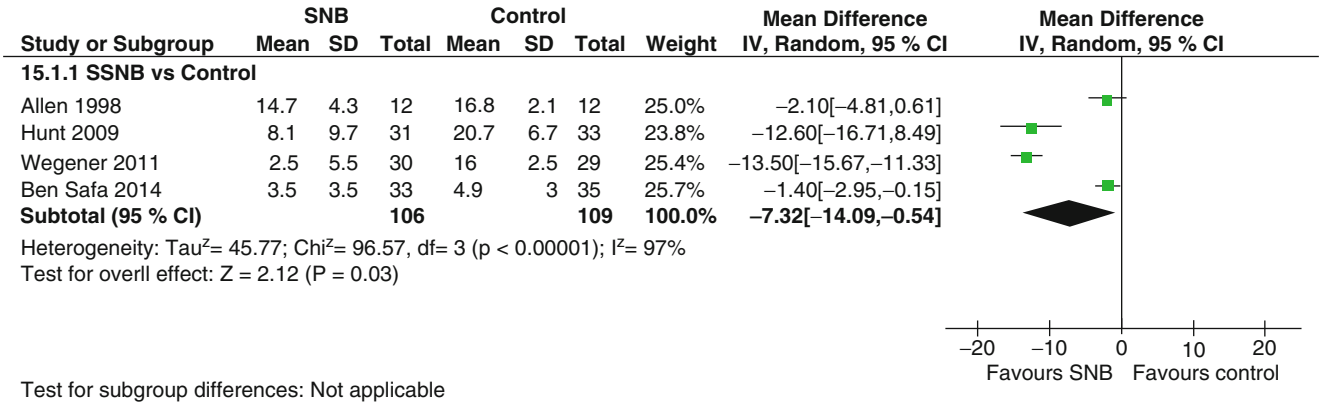


Fig. 60.9 IV morphine equivalent consumption for the 0–12-h interval. The sample size, mean, standard deviations, and the pooled estimates of the mean difference are shown. The 95 % CIs are shown as *lines* for individual studies and as *diamonds* for pooled estimates (Figures are reproduced with permission of www.USRA.ca)

References

1. Abdallah FW, Brull R. Sciatic nerve block for analgesia after total knee arthroplasty: the jury is still out. Reg Anesth Pain Med. 2012;37(1):122–3.
2. Abdallah FW, Brull R. Is sciatic nerve block advantageous when combined with femoral nerve block for postoperative analgesia following total knee arthroplasty? A systematic review. Reg Anesth Pain Med. 2011;36(5):493–8.
3. Aguirre J, Neudoerfer C, Ekatodramis G, Borgeat A. The sciatic nerve should be blocked to optimize postoperative analgesia after knee arthroplasty. Eur J Anaesthesiol. 2009;26(5):439–40.
4. Allen HW, Liu SS, Ware PD, Nairn CS, Owens BD. Peripheral nerve blocks improve analgesia after total knee replacement surgery. Anesth Analg. 1998;87(1):93–7.
5. Ben-David B, Schmalenberger K, Chelly JE. Analgesia after total knee arthroplasty: is continuous sciatic blockade needed in addition to continuous femoral blockade? Anesth Analg. 2004;98(3):747–9.
6. Buckenmaier III CC, Xenos JS, Nilsen SM. Lumbar plexus block with perineural catheter and sciatic nerve block for total hip arthroplasty. J Arthroplasty. 2002;17(4):499–502.
7. Canadian Pharmacists Association. Compendium of pharmaceuticals and specialties: the Canadian drug reference for health professionals. 45th ed. Ottawa: Canadian Pharmacists Assoc; 2010.
8. Cappelleri G, Ghisi D, Fanelli A, Albertin A, Somalvico F, Aldegheri G. Does continuous sciatic nerve block improve postoperative analgesia and early rehabilitation after total knee arthroplasty? A prospective, randomized, double-blinded study. Reg Anesth Pain Med. 2011;36(5):489–92.
9. Carvalho R, Calixto L, Braganca JP. Effect of a single shot sciatic nerve block combined with a continuous femoral block on pain scores after knee arthroplasty. A randomized controlled trial. Open J Anesthesiol. 2012;2:107–12.
10. Connolly C, Coventry D. Combined sciatic/femoral block followed by sciatic infusion of ropivacaine 2 mg/ml for below knee amputation; a feasibility study. Reg Anesth Pain Med. 1998;23(3):81–6.
11. Fischer HBJ, Simanski CJP, Sharp C, Bonnet F, Camu F, Neugebauer EAM, Rawal N, Joshi GP, Schug SA, Kehlet H. A procedure-specific systematic review and consensus recommendations for postoperative analgesia following total knee arthroplasty. Anaesthesia. 2008;63(10):1105–23.
12. Fournier R, Faust A, Chassot O, Gamulin Z. Levobupivacaine 0.5 % provides longer analgesia after sciatic nerve block using the Labat approach than the same dose of ropivacaine in foot and ankle surgery. Anesth Analg. 2010;110(5):1486–9.
13. 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.
14. Gaertner E, Fouche E, Choquet O, Hadzic A, Vloka J. Sciatic nerve block. In: Hadzic A, editor. Textbook of regional anesthesia and acute pain management. 1st ed. New York: McGraw-Hill, Medical Pub; 2007. p. 517–32.
15. Gi E, Yamauchi M, Yamakage M, Kikuchi C, Shimizu H, Okada Y, Kawamura S, Suzuki T. Effects of local infiltration analgesia for posterior knee pain after total knee arthroplasty: comparison with sciatic nerve block. J Anesth. 2014;28:696–701.
16. Hunt K, Bourne M, Mariani EM. Single-injection femoral and sciatic nerve blocks for pain control after total knee arthroplasty. J Arthroplasty. 2009;24(4):533–8.
17. Ilfeld BM, Madison SJ. The sciatic nerve and knee arthroplasty: to block, or not to block-that is the question. Reg Anesth Pain Med. 2011;36(5):421–3.
18. Madan K, Wasan AD. Sciatic nerve block. In: Lennard TA, Vivian DG, Walkowski SD, Singla AK, editors. Pain procedures in clinical practice. 3rd ed. Philadelphia: Elsevier Health Sciences; 2011. p. 261–6.
19. Mahadevan D, Walter RP, Minto G, Gale TC, McAllen CJ, Oldman M. Combined femoral and sciatic nerve block vs combined femoral and periarticular infiltration in total knee arthroplasty: a randomized controlled trial. J Arthroplasty. 2012;27(10):1806–11.
20. Morin A, Kratz C, Eberhart LH, Dinges G, Heider E, Schwarz N, Eisenhardt G, Geldner G, Wulf H. Postoperative analgesia and functional recovery after total-knee replacement: comparison of a continuous posterior lumbar plexus (psoas compartment) block, a continuous femoral nerve block, and the combination of a continuous femoral and sciatic nerve block. Reg Anesth Pain Med. 2005;30(5):434–45.
21. Paul J, Arya A, Hurlburt L, Cheng J, Thabane L, Tidy A, Murthy Y. Femoral nerve block improves analgesia outcomes after total knee arthroplasty: a meta-analysis of randomized controlled trials. Anesthesiology. 2010;113(5):1144–62.
22. Pham Dang C, Gautheron E, Guilley J, Fernandez M, Waast D, Volteau C, Nguyen JM, Pinaud M. The value of adding sciatic block to continuous femoral block for analgesia after total knee replacement. Reg Anesth Pain Med. 2005;30(2):128–33.
23. Safa B, Gollish J, Haslam L, McCartney CJ. Comparing the effects of single shot sciatic nerve block versus posterior capsule local anesthetic infiltration on analgesia and functional outcome after total knee arthroplasty: a prospective randomized, double-blinded, controlled trial. J Arthroplasty. 2014;29:1149–53.
24. Serrano A, Santiveri X, Bisbe E, Ortiz P, Puig L, Castillo J. Analgesic efficacy of associating a sciatic block to a femoral block in the postoperative period of total knee arthroplasty: 8AP4-9. Eur J Anaesthesiol. 2011;28:119–20.
25. Sinha SK, Abrams JH, Arumugam S, D'Alessio J, Freitas DG, Barnett JT, Weller RS, Weller RS. Femoral nerve block with selective tibial nerve block provides effective analgesia without foot drop after total knee arthroplasty: a prospective, randomized, observer-blinded study. Anesth Analg. 2012;115(1):202–6.
26. Tsui BC. Sciatic and popliteal blocks. In: Tsui BC, Bhargava R, Chan V, editors. Atlas of ultrasound-and nerve stimulation-guided regional anesthesia. 1st ed. New York: Springer; 2007. p. 171–203.
27. Vloka JD, Hadzic A, April E, Thys DM. The division of the sciatic nerve in the popliteal fossa: anatomical implications for popliteal nerve blockade. Anesth Analg. 2001;92(1):215–7.
28. Weber A, Fournier R, Van Gessel E, Gamulin Z. Sciatic nerve block and the improvement of femoral nerve block analgesia after total knee replacement. Eur J Anaesthesiol. 2002;19(11):834–6.
29. Wegener JT, van Ooij B, van Dijk CN, Hollmann MW, Preckel B, Stevens MF. Value of single-injection or continuous sciatic nerve block in addition to a continuous femoral nerve block in patients undergoing total knee arthroplasty: a prospective, randomized, controlled trial. Reg Anesth Pain Med. 2011;36(5):481–8.
30. Williams A. Pelvic girdle gluteal region and hip joint area. In: Standring S, editor. Gray's anatomy: the anatomical basis of clinical practice. 39th ed. Philadelphia: Elsevier; 2004.
31. Yoshinuma H, Hara K, Sugimura K, Makino S, Nakamura K, Osamu K. Sciatic nerve block provide superior analgesia for popliteal pain after total knee arthroplasty: 8AP7-1. Eur J Anaesthesiol. 2013;30:134.

Chapter 61

Proximal Sciatic Nerve Block-Traditional Technique

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Battista Borghi, (Subgluteal Access Route Section)
Anesthesia Research Unit, Instituti Orthopedici Rizzoli, Bologna, Italy
e-mail: battista.borghi@ior.it

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_61,

Traditional Techniques

1. *Classic dorsal transgluteal technique (Labat technique)*
2. *Proximal thigh (anterior technique)*
3. *Subgluteal block*

Definition

Block of the largest of the four nerves supplying the leg at the lower end of the lumbosacral plexus, after it exits from the greater sciatic foramen or infrapiriform foramen.

Anatomy

The sciatic nerve arises from the ventral branches of the spinal nerves from L4 to S3. Exiting from the pelvic cavity at the lower edge of the piriformis muscle (in about 2 % of individuals, the nerve pierces the piriformis) (Fig. 61.1b), its 16–20-mm-thick trunk runs between the ischial tuberosity and the greater trochanter; turns downward over the gemelli, the obturator internus tendon, and the quadratus femoris, which separate it from the hip joint; and leaves the buttock to enter the thigh beneath the lower border of the gluteus maximus (Figs. 61.1a, b and 61.2).

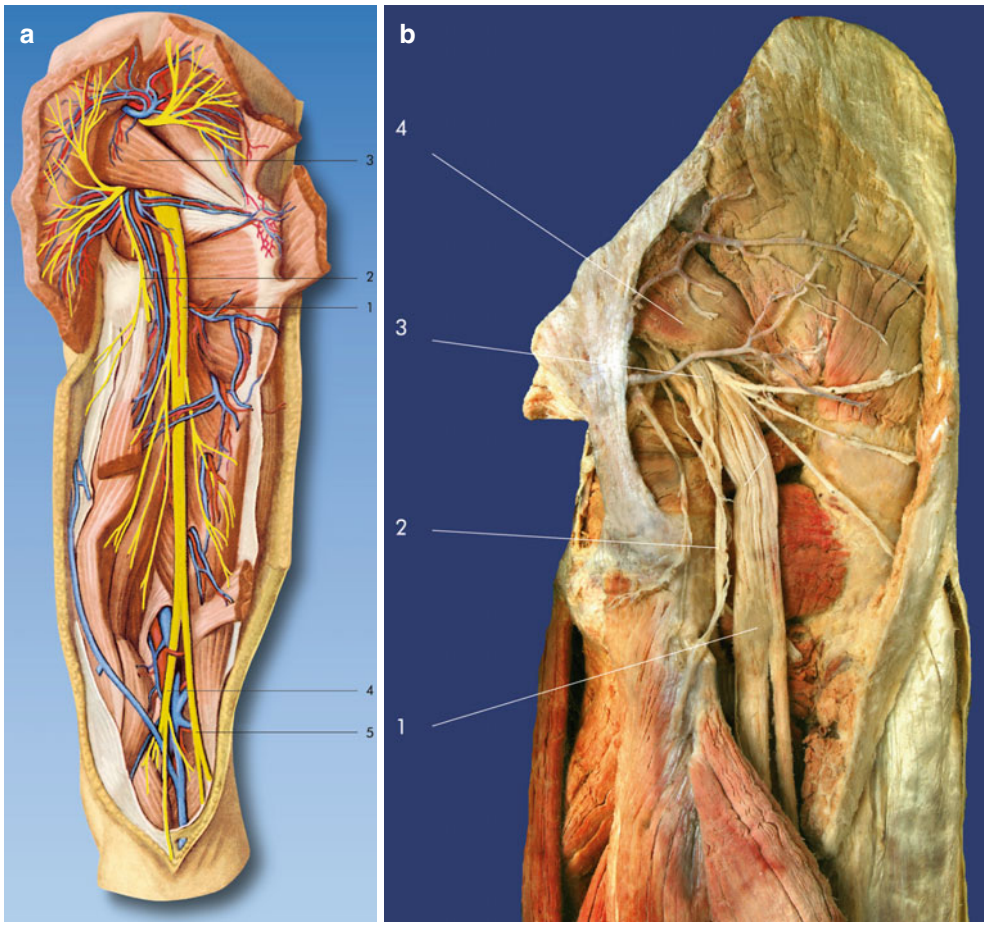


Fig. 61.1 Anatomy of the sciatic nerve. (**a**) (*1*) Sciatic nerve, (*2*) posterior femoral cutaneous nerve, (*3*) piriformis muscle, (*4*) tibial nerve, (*5*) common peroneal (fibular) nerve (Reproduced with permission from Danilo Jankovic). (**b**) (*1*) Sciatic nerve, (*2*) posterior cutaneous femoris nerve, (*3*) infrapiriform foramen with sciatic nerve and gluteal inferior nerve,(*4*) piriformis muscle (Reproduced with permission from Danilo Jankovic)

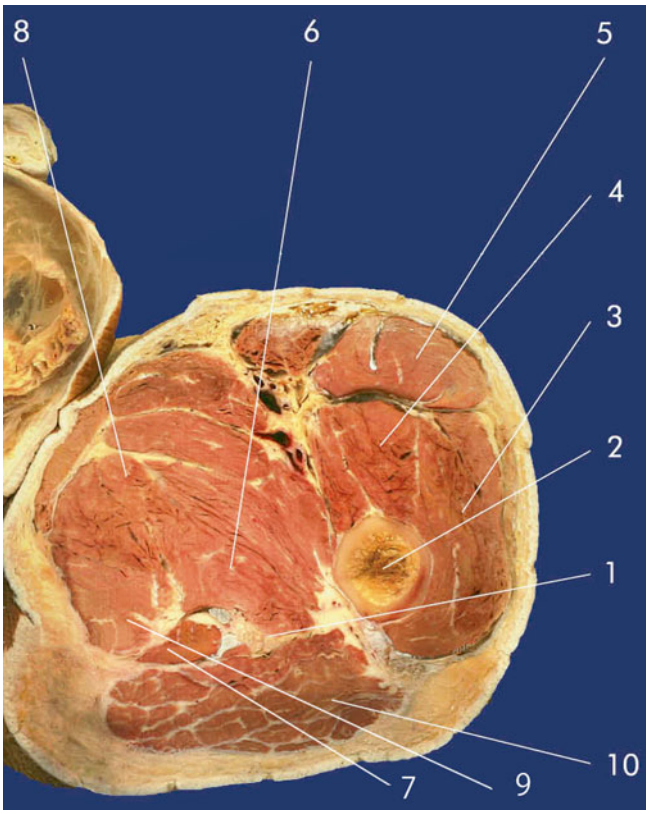


Fig. 61.2 Transversal dissection. (*1*) Sciatic nerve, (*2*) femur, (*3*) vastus lateralis muscle (*4*) vastus intermedius muscle, (*5*) rectus femoris muscle, (*6*) semimembranosus muscle, (*7*) semitendinosus muscle, (*8*) adductor brevis muscle, (*9*) adductor magnus muscle, (*10*) gluteus maximus muscle (Reproduced with permission from Danilo Jankovic)

Distal to this, the nerve lies on the posterior surface of the adductor magnus muscle, where it is covered by the flexor muscle originating from the ischial tuberosity and thus extends as far as the popliteal fossa (Fig. 61.2). Here it lies slightly laterally and above the popliteal vein and artery, with thick popliteal fascia overlying it. At the proximal end of the popliteal fossa, the nerve usually divides into the thicker tibial nerve, which continues the trunk and the smaller common peroneal (fibular) nerve (Fig. 61.1).

The sensory branches of the nerve innervate the dorsal thigh, the dorsolateral lower leg, and lateral half of the foot, the hip, and knee joint, as well as the femur.

Its muscular branches are responsible for supplying the biceps femoris, semimembranosus, semitendinosus, and adductor magnus muscles.

Indications

Surgical

- Superficial procedures in the innervated area.
- Carrying out surgical procedures in the region of the lower extremity under tourniquet, but in combination with a block of the lumbar plexus ("three-in-one" block or dorsal psoas compartment block). A need for larger volumes of local anesthetics must be expected (toxicity!).

Therapeutic

An isolated block of the sciatic nerve is rarely indicated. A combination with block of the lumbar plexus or femoral nerve is recommended.

Block Series

A series of six to eight blocks is recommended. When there is evidence of improvement in the symptoms, additional blocks can also be carried out.

Contraindications

Specific

- Infection or hematoma in the injection area
- Anticoagulant treatment
- Lesion in the nerves to be blocked distal to the injection site

Relative

The decision should be taken after carefully weighing up the risks and benefits:

- Hemorrhagic diathesis
- Stable central nervous system diseases
- Local nerve injury

Procedure

This block should be carried out by experienced anesthetists or under their supervision. Full prior information for the patient is mandatory.

Preparations

Check that the emergency equipment is complete and in working order. Sterile precautions, intravenous access, ECG monitoring, pulse oximetry, intubation kit, ventilation facilities, emergency medication.

Materials

Peripheral nerve stimulator

Single-Shot Technique

Fine 26-G needle, 25 mm long, for local anesthesia.

80–100 mm long (120–150 mm for ventral access), short-bevel insulated stimulating atraumatic 22-G needle (15°) with injection lead ("immobile needle")

Continuous Technique

Tuohy set: 102-mm-long 18-G Tuohy needle with catheter Catheter kit: 18-G needle (80–110 mm, 15°) with catheter.

Syringes: 2, 10, and 20 mL.

Local anesthetics, disinfectant, swabs, compresses, sterile gloves, and drape.

Classic Dorsal Transgluteal Technique (Labat Technique)

Patient Positioning (Fig. 61.3a)

Lateral decubitus, with the leg being blocked on top (Sims position).

The upper leg is bent at the hip and knee joints and the upper knee lies on the table. The lower leg is straight.

Landmarks (Fig. 61.3b)

The important landmarks are: the greater trochanter and posterior superior iliac spine (and/or sacral hiatus). The greater trochanter and posterior superior iliac spine are located. From the midpoint of the connecting line, a line is drawn medially and the injection point is marked at 5 cm (Labat line).

To check this, another line connecting the greater trochanter and the sacral hiatus is bisected (Winnie line). The two points should coincide.

Skin prep, local anesthesia, sterile draping, drawing up local anesthetic into a 20-mL syringe, checking patency of the injection needle and correct functioning of the nerve stimulator, attaching the electrodes.

Preliminary puncture with a large needle or stylet.

During the procedure, the biceps femoris, semimembranosus and semitendinosus muscles, and the foot must be observed.

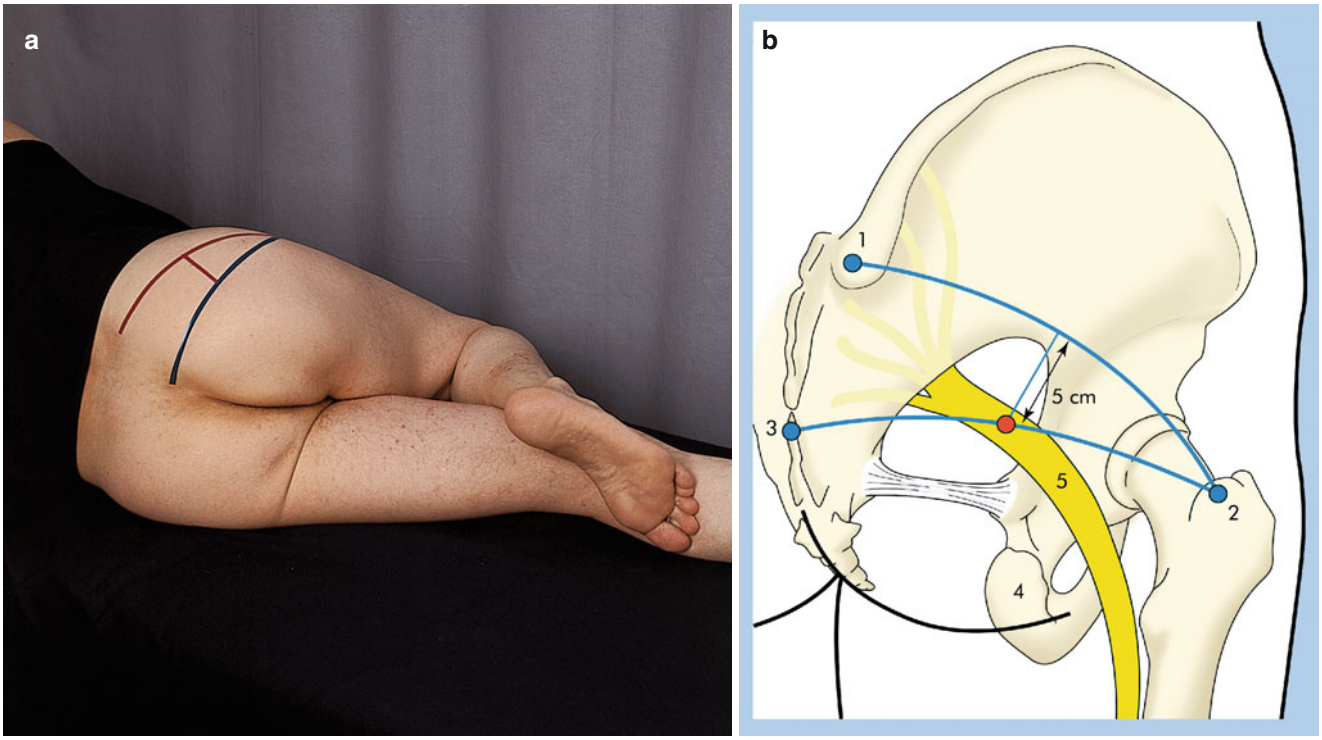


Fig. 61.3 (**a**) Classic dorsal transgluteal technique (positioning) (Reproduced with permission from Danilo Jankovic). (**b**) Landmarks. (*1*) Posterior superior iliac spine, (*2*) greater trochanter, (*3*) sacral hiatus, (*4*) ischial tuberosity, (*5*) sciatic nerve (Reproduced with permission from Danilo Jankovic)

Injection Technique

1. The injection needle is introduced perpendicular to the skin surface (Fig. 61.4).

   Stimulation current of 1 mA at 2 Hz is selected with a stimulus duration of 0.1 ms.
2. After about 1–4 cm, there should be direct stimulation of the gluteus maximus muscle.
3. At a depth of about 5 cm, contractions of the biceps femoris, semimembranosus and semitendinosus muscles are produced (Fig. 61.5).

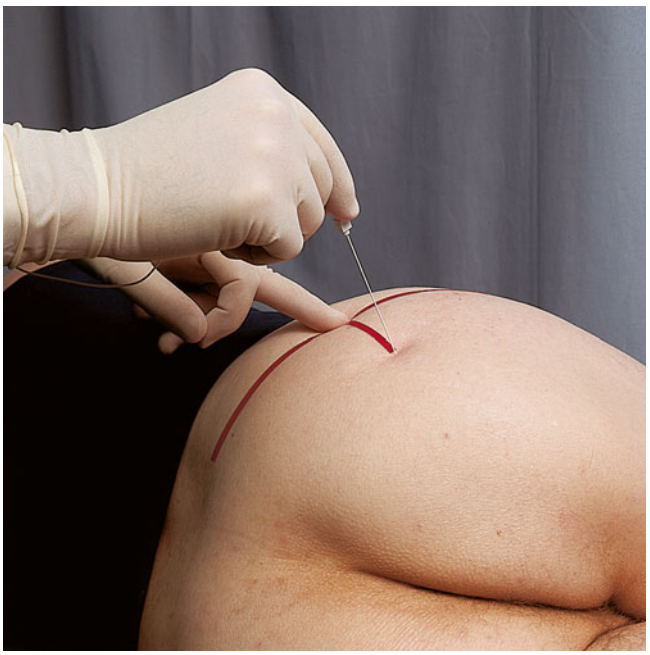


Fig. 61.4 The injection needle is introduced perpendicular to the skin surface (Reproduced with permission from Danilo Jankovic)

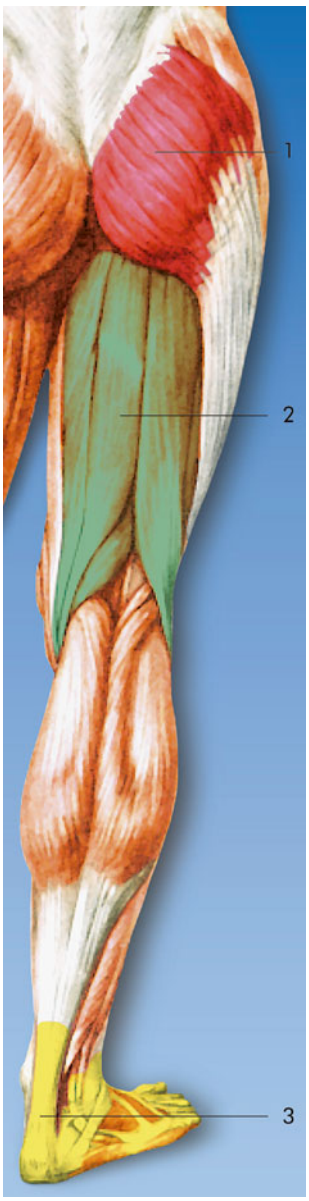


Fig. 61.5 Sequence of muscle contractions. (*1*) Gluteus maximus muscle; (*2*) semitendinosus muscle, semimembranosus muscle, biceps femoris muscle; (*3*) plantar/dorsal flexion of the foot (Reproduced with permission from Danilo Jankovic)

4. After the needle is advanced further, at a depth of about 6–8 cm, there is plantar and dorsal flexion of the foot as a response to the stimulus from the tibial or peroneal part of the sciatic nerve.
5. Do not advance the needle any further.
6. The stimulation current is reduced to 0.3 mA. Slight twitching suggests that the needle is positioned in the immediate vicinity of the nerve.
7. Aspiration test.
8. Test dose of 3-mL local anesthetic (e.g., 1 % lidocaine, mepivacaine, or prilocaine). During the injection, the twitching should slowly disappear.
9. Incremental injection of a local anesthetic (injection–aspiration after each 3–4 mL).
10. Careful cardiovascular monitoring.

The area of anesthesia is shown in Fig. 61.6.

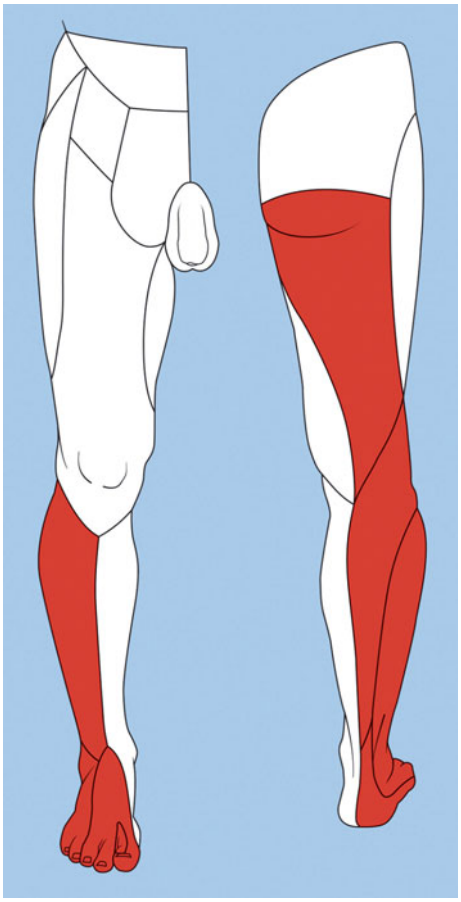


Fig. 61.6 Cutaneous innervated area of the sciatic nerve (Reproduced with permission from Danilo Jankovic)

Problem Situations

- Bone contact at a depth of 8 cm without visible twitching. The injection needle should be withdrawn and the direction should be altered laterally.
- Intraneural positioning:

The following signs suggest intraneural positioning of the injection needle:

– Strong twitching (even at a stimulant current of 0.2 mA).
– No disappearance of the twitching during injection of a test dose.
– High resistance and severe pain during the injection.
– The injection must be stopped immediately and the needle must be withdrawn.

Continuous Technique

The injection is carried out as in the single-shot technique.

An 18-G Tuohy needle 102 mm long and a short-bevel stimulating atraumatic needle 110 mm long are usually used as stimulation needles. After correct stimulation and aspiration, a test dose is injected. The catheter is then advanced ca. 3 cm beyond the end of the container, and the stimulation needle is slowly withdrawn, while the thumb and index finger of the left hand simultaneously hold the catheter at the injection site. A bacterial filter is then placed, and the catheter is fixed with a skin suture and dressing.

Anterior Approach

Patient Positioning

A supine position that is comfortable for the patient, with slight outward rotation of the leg being blocked.

Landmarks

Important landmarks are the anterior superior iliac spine, pubic tubercle, and greater trochanter. Two lines are drawn for orientation:

- A line connecting the anterior superior iliac spine with the pubic tubercle, which is marked into thirds.
- A second line parallel to the first, from the greater trochanter across the thigh.

A perpendicular line is drawn from the intersection of the medial and central third of the upper inguinal ligament line to the parallel line and marked as the injection point (Fig. 61.7a, b).

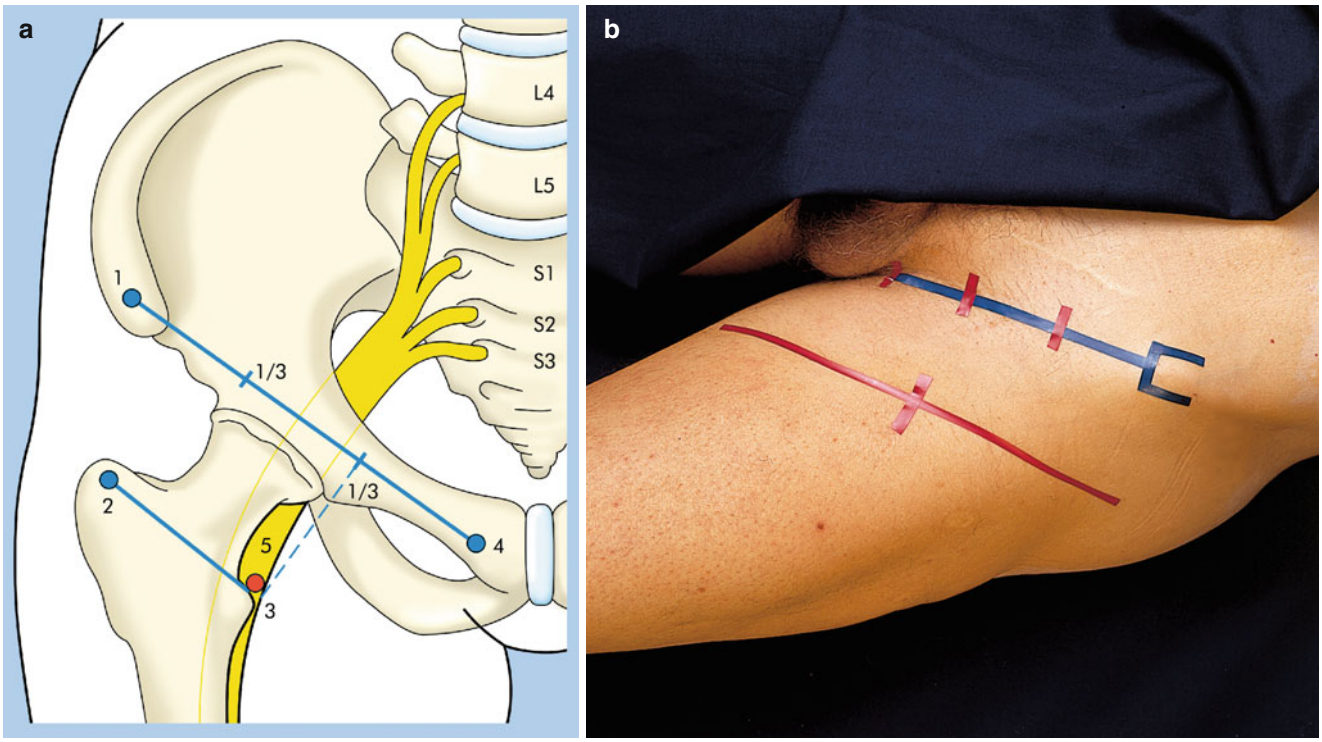


Fig. 61.7 (**a**) Anterior approach. Location (diagram). (*1*) Anterior superior iliac spine, (*2*) greater trochanter, (*3*) injection site, (*4*) pubic tubercle, (*5*) sciatic nerve (Reproduced with permission from Danilo Jankovic). (**b**) Location (in patient) (Reproduced with permission from Danilo Jankovic)

Injection Technique

A 22-G (15°) short-bevel insulated stimulating atraumatic needle 120–150 mm long, with an injection lead, is advanced perpendicular to the skin until bone contact is made with the femur. The needle is then withdrawn slightly and introduced about 5 cm deeper, past the medial border of the femur.

The correct needle position is confirmed when paresthesias or twitches are produced during electrostimulation. After aspiration and administration of a test dose, incremental injection of a local anesthetic is carried out.

Subgluteal Access Route (Di Benedetto–Borghi Approach)

Battista Borghi

The subgluteal block of the sciatic nerve has the advantage over the classic posterior transgluteal technique in that it is less stressful to the patient during the procedure, as the sciatic nerve has a more superficial course in the subgluteal region than in the gluteal region. This access route also makes it easier to place and fix a catheter for postoperative analgesia.

Procedure

Patient Positioning

Lateral decubitus, with the leg being blocked on top (Sims position; Fig. 61.3a).

Landmarks

From the midpoint of a line connecting the greater trochanter and the ischial tuberosity, a second line is drawn to the upper edge of the popliteal fossa (known as the "sciatic line"). The injection site is located ca. 3–4 cm caudal to this (Fig. 61.8). If the patient is lying in the Sims position, for easier guidance, one can palpate a groove along this line between the semitendinosus muscle and the biceps femoris muscle,

In this technique, the distance between the skin and the sciatic nerve is shorter (4.7 cm) than in Labat's classic transgluteal access route (6.7 cm).

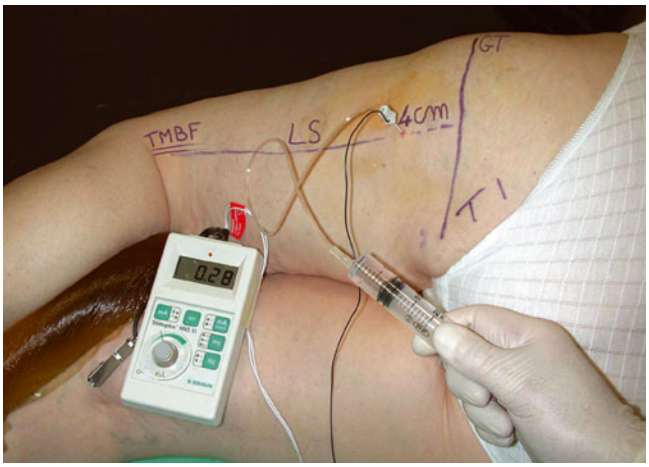


Fig. 61.8 Subgluteal approach. Landmarks. *GT* greater trochanter, *TI* ischial tuberosity, *LS* sciatic line, *TMBF* tendon of the biceps femoris muscle (Reproduced with permission from Battista Borghi)

Preparations

Skin prep, local anesthesia, sterile draping, drawing up local anesthetic, checking the patency of the injection needle and correct functioning of the nerve stimulator, attaching the electrodes.

Injection

Preliminary puncture with a large needle or stylet.

During the procedure, the biceps femoris, semimembranosus and semitendinosus muscles, and the foot must be observed.

An injection needle ca. 50 (80) mm long is introduced perpendicular to the skin (Fig. 61.9a). Stimulation current is applied at 1–1.5 mA at 2 Hz with a stimulus duration 0.1 ms. At a depth of ca. 4 cm, plantar flexion and dorsiflexion of the foot occur in response to the stimulation of the tibial or peroneal parts of the sciatic nerve. The needle should not be advanced any further. The stimulation current is reduced to 0.3 mA. Slight twitching indicates that the stimulation needle is located in the immediate vicinity of the nerve. After an aspiration test, a test dose (e.g. 3 mL 1 % prilocaine) is injected and incremental injection of the local anesthetic follows.

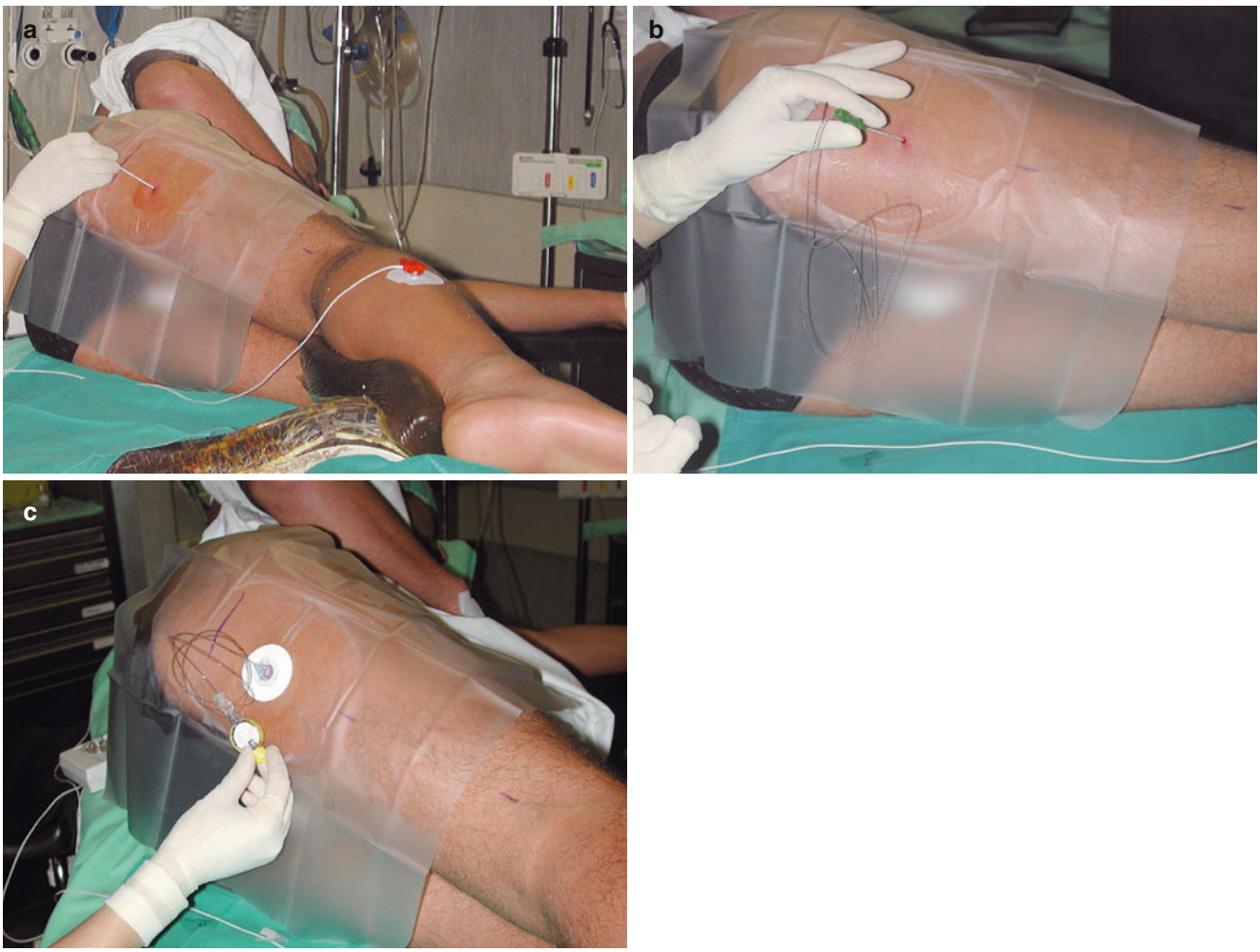


Fig. 61.9 (**a**) Introducing the needle (Reproduced with permission from Battista Borghi). (**b**) Introducing the catheter (Reproduced with permission from Battista Borghi). (**c**) Fixing the catheter and placing a bacterial filter (Reproduced with permission from Battista Borghi)

Continuous Subgluteal Block of the Sciatic Nerve

After skin prep, an adhesive sterile transparent drape with a hole is applied.

Materials

Tuohy continuous set: 52 (102)-mm-long 18-G Tuohy needle with a catheter

Catheter kit: 18-G short-bevel insulated atraumatic needle 80–110 mm (15°) long with a catheter

Injection

The injection is carried out as in the single-shot technique. After injection of 5 mL 0.9 % saline, the catheter is introduced through the already positioned needle. The catheter is advanced ca. 3–4 cm beyond the end of the needle or cannula (Fig. 61.9b). After removal of the needle or cannula, fixing of the catheter and placement of a bacterial filter (Fig. 61.9c), after careful aspiration, and injection of a test dose, bolus administration of the local anesthetic is carried out.

Dosage

Surgical

30–40-mL local anesthetic – e.g., 0.5–0.75 % ropivacaine, 0.5 % bupivacaine (0.5 % levobupivacaine), 1 % prilocaine, 1 % mepivacaine.

A combination of long-duration and medium-duration local anesthetics has proved particularly useful for surgical indications.

Continuous Administration

0.2–0.375 % ropivacaine

5–15 mL/h (max. 37.5 mg/h)

Alternatively, as a bolus dose: 0.2–0.375 % ropivacaine, 10–30 mL

Subgluteal Access

Patient-controlled analgesia (PCA): baseline rate of 4 mL/h 0.4 % ropivacaine, 0.25 % levobupivacaine, and 0.25 % bupivacaine

Bolus dose of 2 mL

Lockout time 10 min

Therapeutic

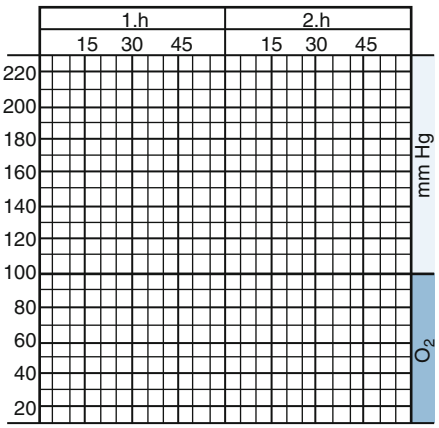
10–20-mL local anesthetic – e.g., 0.2–0.375 % ropivacaine, 0.125–0.25 % bupivacaine

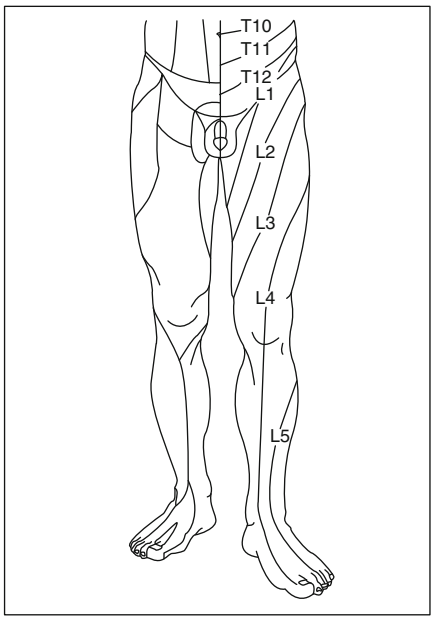
Complications

Complications are rare, but possible:

- Nerve injury,
- Intravascular injection
- CNS toxicity
- Infection in the area of the injection
- Hematoma formation

Record and checklist





Lumbosacral plexus and individual nerves in the plexus

Block no. ☐ Right ☐ Left

Name: _____ Date: _____

Diagnosis: _____

Premedication: ☐ No ☐ Yes

Neurological abnormalities: ☐ No ☐ Yes _____

Purpose of block: ☐ *Surgical* ☐ *Diagnostic* ☐ *Therapeutic*

Needle: *G* ____ *Length* _____ *cm* ☐ *15°* ☐ *30°* ☐ *Other*

i.v. access: ☐ *Yes*

Monitoring: ☐ *ECG* ☐ *Pulse oximetry*

Ventilation facilities: ☐ *Yes (equipment checked)*

Emergency equipment *(drugs):* ☐ *Checked*

Patient: ☐ *Informed (behavior after block)*

Position: ☐ *Prone* ☐ *Lateral recumbent* ☐ *Sims-Position* ☐ *Sitting*

Injection: ☐ *Inguinal "3-in-one" block* ☐ *Dorsal psoas compartment block*

☐ *Sciatic nerve* ☐ *Femoral nerve* ☐ *Lateral cutaneous nerve of thigh*

☐ *Obturator nerve* ☐ *Ilioinguinal nerve / hypogastric nerve*

Location technique: ☐ *Electrostimulation* ☐ *Other*

☐ *Ultrasound- guided* ☐ *Transducer* ☐ *Linear* ☐ *Curved*

Approach: ☐ *In-plane* ☐ *Out of plane*

Plexus (nerve): ☐ *Located* ☐ *Aspiration test* ☐ *Test dose*

Injection:

Local anesthetic: _____ *mL* _____ *%*

(in incremental doses)

☐ *Inguinal "3-in-one" block* ____ *mL* ☐ *Dorsal psoas compartmt. block* ____ *mL*

☐ *Sciatic nerve* ____ *mL* ☐ *Femoral nerve* ____ *mL* ☐ *Lateral cutaneous nerve of thigh* ___ *mL* ☐ *Obturator nerve* ___ *mL* ☐ *Ilioinguinal nerve/hypogastric nerve* ___ *mL*

☐ Addition to LA _____ *µg/mg*

Patient's remarks during injection:

☐ *None* ☐ *Paresthesias* ☐ *Warmth* ☐ *Pain (intraneural position?)*

Nerve area: _____

Objective block effect after 15 min:

☐ *Cold test* ☐ *Temperature measurement before* ____ *°C after* ____ *°C*

☐ *Sensory* ☐ *Motor*

Monitoring after block: ☐ *< 1 h* ☐ *> 1 h*

Time of discharge: _____ ☐ *Sensorimotor function checked*

Complications:

☐ *None* ☐ *Intravascular injection* ☐ *Signs of intoxication*

☐ *Hematoma* ☐ *Neurological complications* ☐ *Other*

Subjective effects of the block: *Duration:* _____

☐ *None* ☐ *Increased pain* ☐ *Reduced pain* ☐ *Relief of pain*

VISUAL ANALOG SCALE

0 10 20 30 40 50 60 70 80 90 100

Special notes:

With permission from Danilo Jankovic

Suggested Reading

1. Bridenbaugh PO, Wedel DJ. The lower extremity: somatic blockade. In: Cousins MJ, Bridenbaugh DL, editors. Neural blockade in clinical anesthesia and management of pain. 3rd ed. Philadelphia-New York: Lippincott-Raven; 1998. p. 375–94.
2. Capdevilla X, Ponrouch M, Choquet O. Continuous peripheral nerve blocks in clinical practice. Curr Opin Anaesthesiol. 2008;21(5):619–23.
3. Casati A, et al. Using stimulating catheters for continuous sciatic nerve block shortens onset time of surgical block and minimizes postoperative consumption of pain medication after halux valgus repair as compared with conventional nonstimulating catheters. Anesth Analg. 2005;101(4):1192–7.
4. di Benedetto P, Casati A, Bertini L. Continuous subgluteal sciatic nerve block after orthopedic foot and ankle surgery: comparison of two infusion techniques. Reg Anesth Pain Med. 2002;27(2):168–72.
5. di B, Bertini L, Casati A, Borghi B, et al. A new posterior approach to the sciatic nerve block. A prospective,randomized comparison with the classical posterior approach. Anesth Analg. 2001;93: 1040–4.
6. Elmas C, Atanassoff P. Combined inguinal paravascular (3 in 1) and sciatU.ic nerve blocks for lower limb surgery. Reg Anesth. 1993;18:88–92.
7. Grabtree EC, et al. A method to estimate the depth of the sciatic nerve during subgluteal block by using thigh diameter as a guide. Reg Anesth Pain Med. 2006;31(4):358–62.
8. Hadzic A. Peripheral nerve blocks. Sciatic block. New York: Mc Graw-Hill; 2012, p. 229–248.
9. Jankovic D. Sciatic nerve block. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Textbook and color atlas. Malden: Blackwell-Malden, Massachusetts 02148-5020, Oxford, Carlton, Victoria; 2004. p. 230–7.
10. Misra U, Pridie AK, McClymont C, et al. Plasma concentrations of bupivacaine following combined sciatic and femoral 3in1 nerve blocks in open knee surgery. Br J Anesth. 1991;66:310–3.
11. Tran D, Clemente A, Finlayson RJ. A review of approaches and techniques for lower extremity nerve blocks. Can J Anesth. 2007;54(11):922–34.

Chapter 62

Popliteal Sciatic Nerve Block

Amit Pawa
Consultant Anaesthetist, Lead for Regional Anaesthesia, Guy's and St Thomas' NHS Foundation Trust, London, UK

Ki Jinn Chin, MBBS, MMed., FANZCA, FRCPC (✉)
Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, M5T 2S8, Canada
e-mail: Kijinn.chin@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_62, © Springer International Publishing Switzerland 2015

Introduction

The popliteal sciatic nerve block is a well-established and popular technique of peripheral nerve blockade in the lower limb. It is a versatile block in that it may be performed in varying patient positions (prone, lateral, or supine) and using different approaches (lateral to medial, posterior to anterior). The landmark-guided technique uses neurostimulation to locate the nerves and is both effective and easy to perform. The advent of ultrasound imaging though has both revolutionized our approach to the popliteal sciatic nerve block [1] and enhanced our understanding of neural anatomy. In particular, the concept of a paraneural sheath in this region is now well accepted [2, 3] and has changed the way we think about "intraneural" and "extraneural" injections [4].

Indications

- Anesthesia or analgesia of the lower limb below the knee, except the medial aspect of the calf/ankle/foot which is supplied by the saphenous nerve
- Anesthesia or analgesia of the posterior aspect of the knee
- Notes
  - The primary advantage over a more proximal sciatic nerve block is preservation of the ability to flex at the knee, which allows more mobility, e.g., ambulation with crutches.
  - If immediate postoperative ambulation is anticipated, the patient will require a surgical boot or cast to overcome the issue of foot drop.
  - The popliteal block must be combined with a saphenous nerve block for complete sensory block of the lower leg and foot.
  - Additional provisions for anesthesia will have to be made if a thigh tourniquet is to be used during surgery, i.e., general anesthesia or neuraxial block.
  - Heel protection with soft padding during the duration of the block is essential to avoid the development of a pressure sore.

Contraindications

- Absolute
  - There are no absolute contraindications specific to this block. Generic contraindications such as patient refusal, allergy to local anesthetics, and local infection at the site of injection apply as usual.
- Relative
  - Preexisting neuropathy in the distribution of the sciatic nerve.
  - Traumatic injuries of the lower leg where there is a concern of compartment syndrome. This may be overcome by adequate postoperative monitoring for clinical features of compartment syndrome other than patient-reported pain.
  - Coagulation abnormalities are not a significant contraindication. The use of ultrasound should prevent vascular puncture. There are no major blood vessels in the immediate vicinity of the target nerves (the popliteal artery and vein are usually more than 2 cm anterior to the nerves at the level of injection) that are at risk during the landmark-guided approach. It can however be a deep block that traverses muscle (biceps femoris) when performed at the mid-thigh level.

Clinical Anatomy of the Sciatic Nerve in the Popliteal Fossa

The sciatic nerve (L4, 5, S1–3) is the largest nerve in the body and exits the pelvis as a structure consisting of two nerve bundles: the more medially placed tibial nerve and the common peroneal (fibular) nerve lying laterally. These two bundles are enclosed within a common paraneural sheath [3, 5]. At its origin, it is broad and flat, but as it passes peripherally, it becomes more rounded. The branching of this sciatic nerve bundle into the two separate nerves occurs at a variable location during its course in the posterior aspect of the thigh, but it has usually occurred within 8–10 cm of the popliteal crease.

The popliteal fossa is a closely packed compartment through which all of the nerves and vessels pass from the thigh to the leg posteriorly. The biceps femoris muscle forms the upper lateral border. The upper medial border is formed by the muscle of semimembranosus and by the tendon of the semitendinosus (Anatomy Fig. 62.1). Appearing from between the biceps femoris and semimembranosus are the two heads of gastrocnemius, which form the lower medial and lateral muscular borders of the fossa. Within the popliteal fossa are the popliteal artery (which terminates as the anterior and posterior tibial arteries), popliteal vein, and the tibial and common peroneal nerves (Anatomy Fig. 62.2).

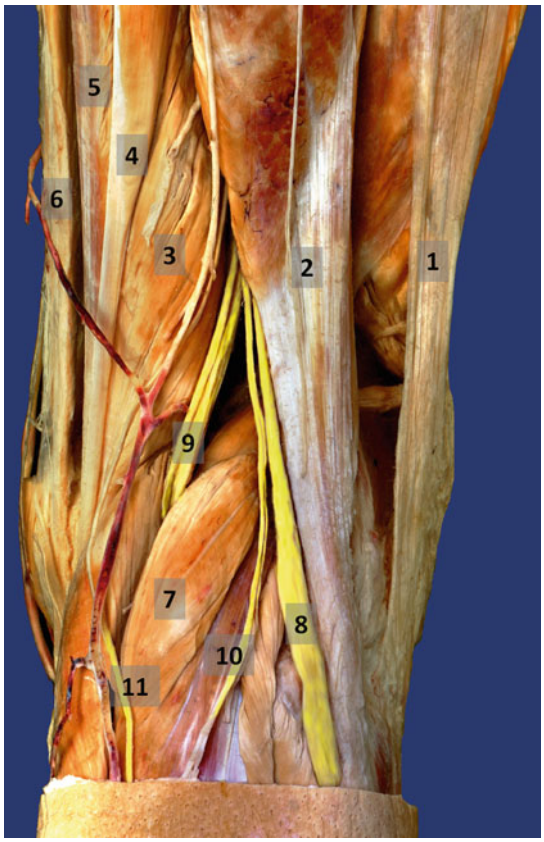


Fig. 62.1 Tibial and common peroneal (fibular) nerve in the popliteal area. (*1*) Iliotibial tract, (*2*) biceps femoris muscle, (*3*) semimembranosus muscle, (*4*) semitendinosus muscle, (*5*) gracilis muscle, (*6*) sartorius muscle, (*7*) gastrocnemius muscle, (*8*) common peroneal(fibular) nerve, (*9*) tibial nerve, (*10*) lateral sural cutaneous nerve (originates from peroneal nerve), (*11*) medial sural cutaneous nerve (originates from tibial nerve) (With permission from Danilo Jankovic)

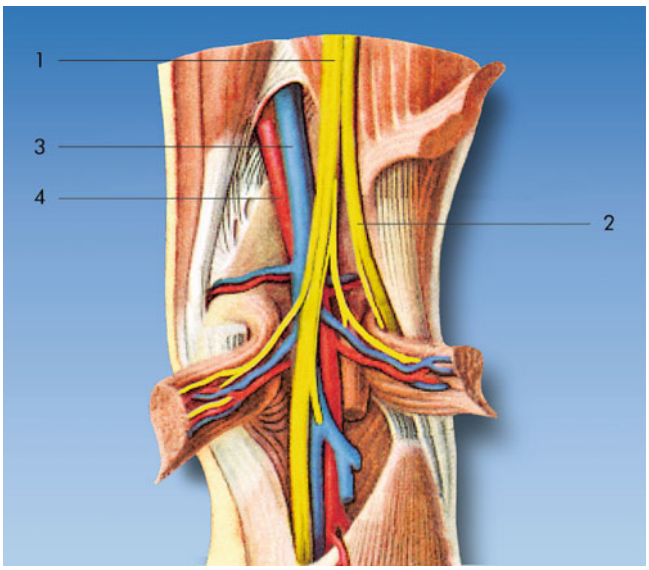


Fig. 62.2 Tibial and common peroneal (fibular) nerve in the popliteal fossa. (*1*) Tibial nerve, (*2*) common peroneal (fibular) nerve, (*3*) popliteal artery, (*4*) popliteal vein (With permission from Danilo Jankovic)

Tibial Nerve

The tibial nerve is the larger of the two sciatic divisions, being almost twice as thick as the common peroneal (fibular) nerve. It traverses the popliteal fossa lying posterior and slightly lateral to the popliteal vessels.

It then passes between the two heads of the gastrocnemius muscle to the upper edge of the soleus muscle, giving off the medial sural cutaneous nerve which unites with the lateral sural cutaneous nerve (a branch of the common peroneal nerve) to form the sural nerve. The tibial nerve continues distally between the posterior tibial muscle and the soleus muscle and runs together with the posterior tibial artery through the calf musculature, between the medial malleolus and the calcaneus, and to the medial side of the foot joint (Anatomy Fig. 62.3). It divides into its two end branches, the medial and lateral plantar nerves, behind the medial malleolus. These pass under the flexor retinaculum to the sole of the foot and provide it with its sensory innervation.

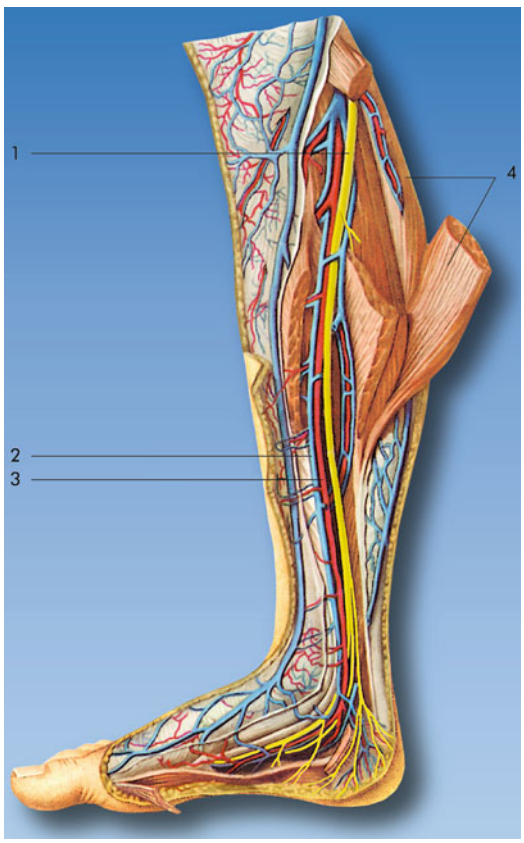


Fig. 62.3 The course of the tibial nerve through the leg and foot. It runs with the posterior tibial artery through the calf to lie behind the medial malleolus, where it divides into the medial and lateral plantar nerves. (*1*) Tibial nerve, (*2*) posterior tibial vein, (*3*) posterior tibial artery, (*4*) gastrocnemius muscle (With permission from Danilo Jankovic)

Common Peroneal (Fibular) Nerve

After separating from the tibial nerve, the common peroneal (fibular) nerve runs along the medial edge of the biceps femoris muscle over the lateral head of the gastrocnemius muscle to the lateral angle of the popliteal fossa. At the neck of the fibula, it passes to the lateral surface of the bone. Before entering the peroneus longus muscle, which originates here, it divides into the superficial peroneal nerve (which is mainly sensory innervation of the foot) and the deep peroneal nerve (mainly motor innervation of the foot).

Up to the point at which it divides, its small branches supply the short head of the biceps femoris muscle, the lateral and posterior parts of the joint capsule, and the tibiofibular joint.

The common peroneal nerve also gives off the lateral sural cutaneous nerve (Anatomy Fig. 62.1). The anterior branch of the lateral sural cutaneous nerve runs subcutaneously on the lateral surface of the lower leg as far as the lateral malleolus and supplies sensory innervation to the posterior and lateral aspects of the lower leg. Its posterior branch unites with the medial sural cutaneous nerve (branch of the tibial nerve) in the middle of the lower leg to form the sural nerve, although it can also occasionally continue as a separate branch as far as the lateral malleolus.

Sonoanatomy of the Sciatic Nerve in the Popliteal Fossa

The sciatic nerve is imaged by placing the ultrasound probe in a transverse orientation on the posterior aspect of the patient's leg, in the popliteal fossa (Sonoanatomy Figs. 62.4 and 62.5). It is recommended that a systematic scan be performed starting at the level of the popliteal skin crease (where structures are most superficial) and moving proximally or cephalad (the traceback approach) [6]. On the lateral aspect of the image, the muscle bulk is made up of biceps femoris, and medially, the muscles of semitendinosus and semimembranosus (Sonoanatomy Fig. 62.6). The popliteal vessels are identified as dark hypoechoic circular structures and can be distinguished by the fact that the artery is pulsatile and the vein is compressible. Color Doppler can also be used for additional confirmation (Sonoanatomy Fig. 62.7). The hyperechoic tibial nerve is located superficial (posterior) to the popliteal vessels and is most commonly lateral to the vein. As the probe is moved proximally, the smaller common peroneal (fibular) nerve will be seen to move from the lateral aspect of the thigh toward the midline to join the tibial nerve to form the sciatic nerve within a common (paraneural) sheath (Sonoanatomy Fig. 62.8).

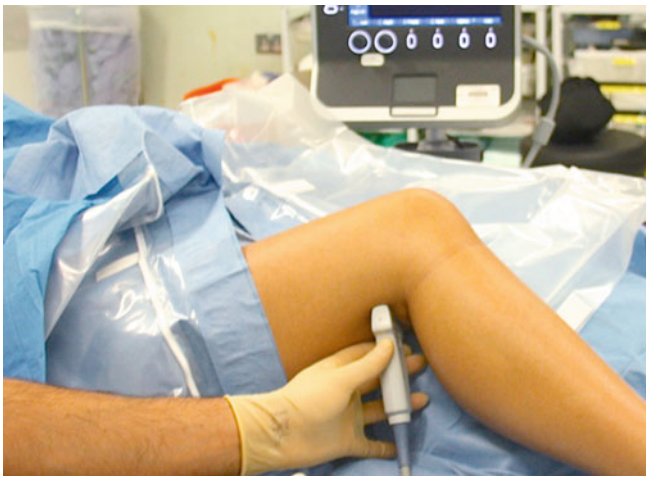


Fig. 62.4 Ultrasound probe position in the popliteal fossa with the patient in the supine position (With permission from Dr. Ki Jinn Chin)

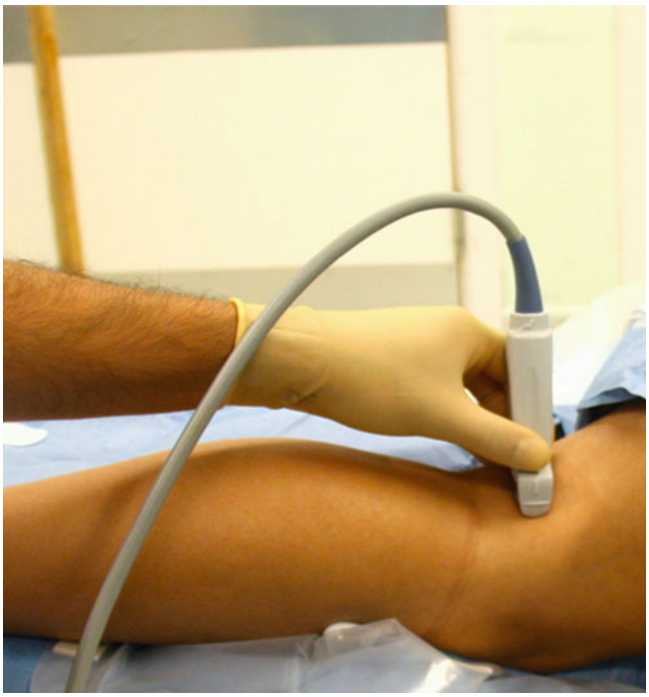


Fig. 62.5 Ultrasound probe position in the popliteal fossa with the patient in the prone position (With permission from Dr. Ki Jinn Chin)

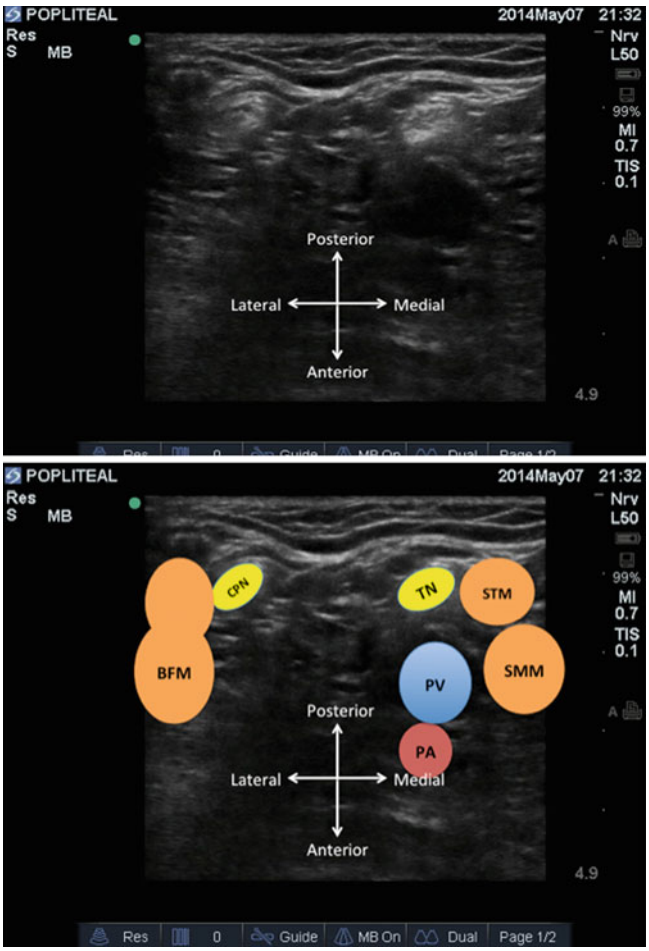


Fig. 62.6 Transverse view of the popliteal fossa and associated structures. The tibial nerve (*TN*) is located superficial to the popliteal artery (*PA*) and vein (*PV*). The common peroneal nerve (*CPN*) is visible lateral to the tibial nerve. *BFM* biceps femoris muscle, *STM* semimembranosus muscle, *SMM* semimembranosus muscle (With permission from Dr. Ki Jinn Chin)

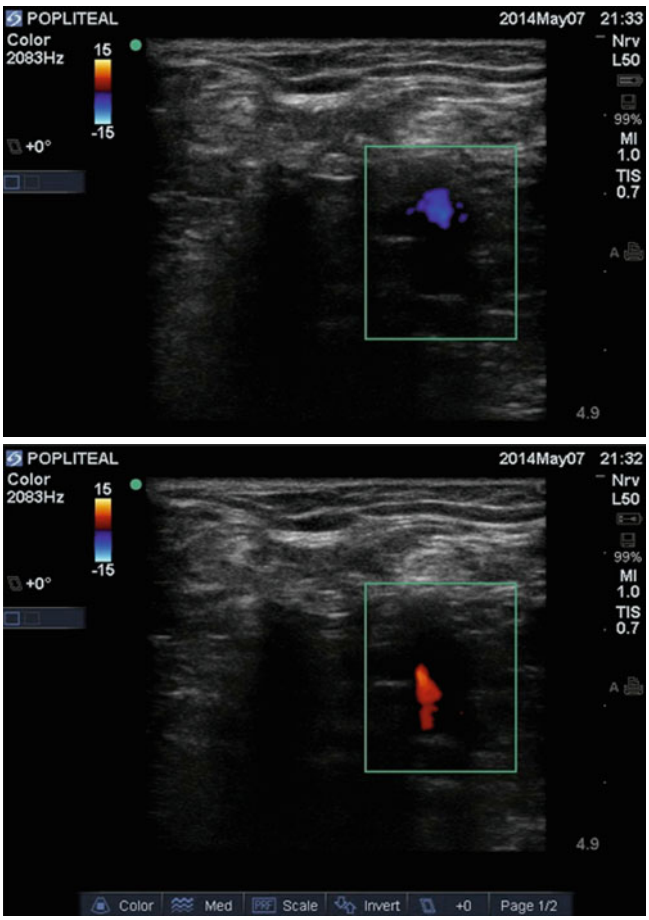


Fig. 62.7 Color Doppler may be used to help identify the popliteal vein (*upper image*) and artery (*lower image*) (With permission from Dr. Ki Jinn Chin)

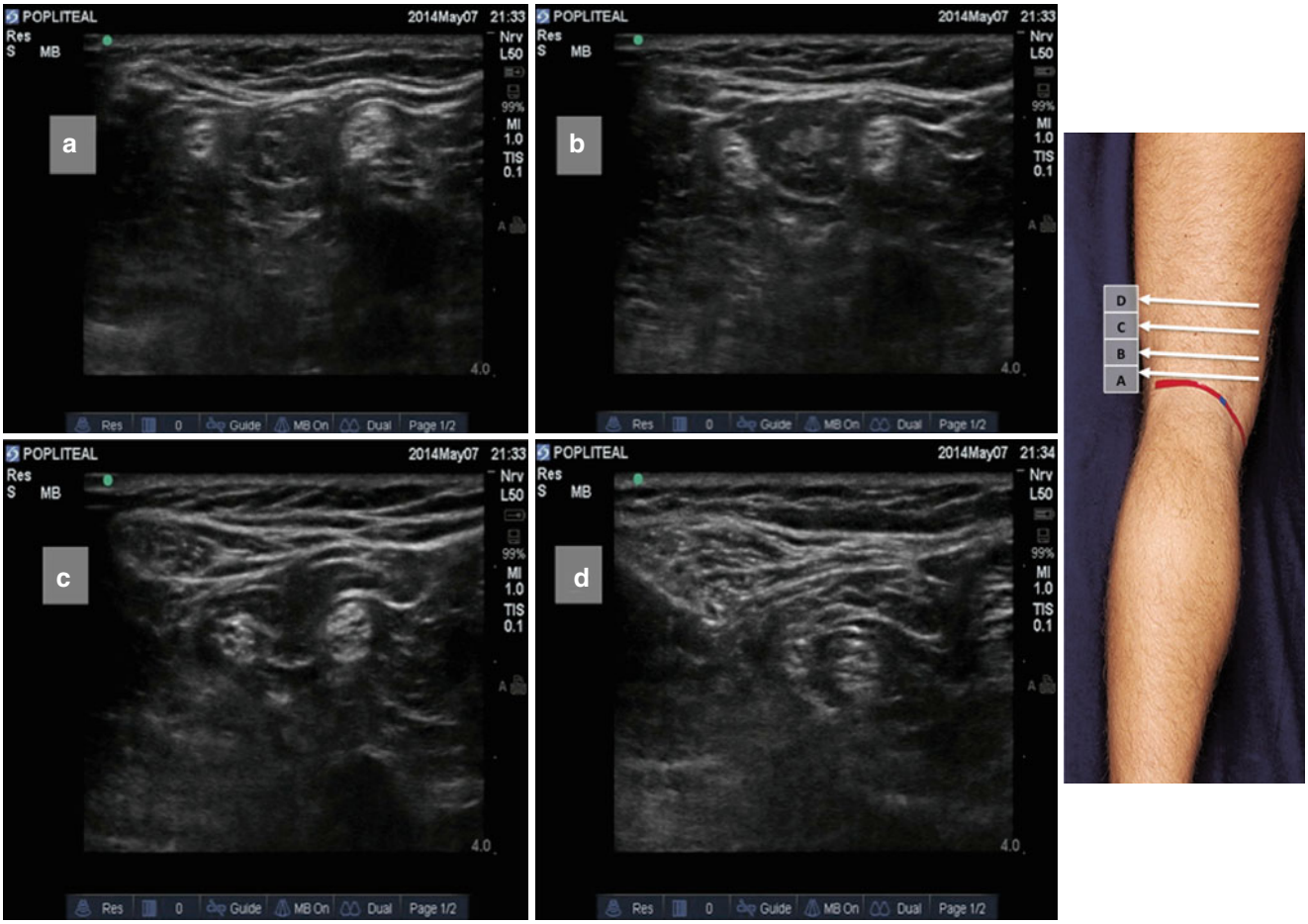


Fig. 62.8 Scanning the sciatic nerve in the popliteal fossa from distal to proximal using the traceback method. The positions of the ultrasound in Figs. **a** to **d** correspond to the positions indicated in the figure on the right side. The tibial nerve (nerve on the *right side*) and common peroneal nerve (nerve on the *left side*) of the sciatic nerve will be seen to approach each other and join within a common sheath (With permission from Dr. Ki Jinn Chin)

Clinical Pearls for Optimal Imaging of the Popliteal Sciatic Nerve

- The neural structures are often more laterally positioned in the popliteal fossa than initially anticipated; bear this in mind with initial probe placement.
- Use color Doppler to identify the popliteal vessels. Use minimal pressure initially to ensure that the popliteal vein is not occluded.
- The traceback method allows you to identify the point at which the common peroneal (fibular) nerve unites with the tibial nerve to form the common sciatic nerve. Blockade at this point is ideal.
- The tibial and common peroneal (fibular) nerves rarely lie in a parallel plane to the skin surface and are therefore usually poorly visible if the ultrasound probe is placed perpendicular to the skin surface. The probe will need to be tilted (often in a plantar direction) to a varying degree to increase the echogenicity and visibility of the nerves (a phenomenon known as anisotropy).

Technique: Landmark-Guided Approach (Single Shot)

Required Supplies and Equipment

- Disinfectant solution and swabs for skin preparation
- Sterile gloves and drapes
- Short-beveled 22-G block needle with extension tubing
  - 50–80 mm (posterior approach)
  - 80–100-mm needle (lateral approach)
- Local anesthetic of choice in 10- or 20-ml syringes
- Lidocaine 1–2 % in 3-ml syringe with a 25–27-G hypodermic needle for skin infiltration (at operator's discretion)
- Equipment and supplies for managing life-threatening acute complications, including intralipid for local anesthetic systemic toxicity
- Drugs for intravenous sedation during the block (at operator's discretion)

Preparation of Patient

- Obtain informed consent for the block.
- Explain expected clinical course including care of the insensate limb and managing the transition to systemic analgesia.
- Establish intravenous access, supplemental oxygen delivery, and standard monitors (ECG, noninvasive blood pressure, pulse oximetry).
- Perform a time-out to confirm patient identity and site and side of surgery.

Block Performance

There are two main landmark-guided approaches that may be used.

1. The posterior popliteal block
2. The lateral popliteal block

Landmark-Guided Popliteal Nerve Block: Posterior Approach

Patient Position

- The patient is placed prone with little to no flexion at the knees and in a neutral position—internal or external rotation at the hips should be avoided. This is done by placing a small soft roll under the ankles or by allowing the feet to dangle off the foot of the bed.
- A right-handed operator should always stand on the left side of the patient, to allow use of the dominant hand for needling, and vice versa.

Surface Landmarks

- Different methods of determining the appropriate needle insertion point have been described [7], but all are based upon the principle of targeting the sciatic nerve in the mid-thigh, proximal to its bifurcation into tibial and peroneal nerves.
- The modified intertendinous approach [7] (LMG Fig. 62.9):
  - Palpate the upper (cephalad) borders of the popliteal fossa, which are formed by the tendons and muscle bellies of the semimembranosus and semitendinosus (medial) and the biceps femoris (lateral). Identification of these landmarks can be facilitated by asking the patient to flex their knee against resistance.
  - Trace the borders to where they meet at the apex of the popliteal fossa—this is the site of needle insertion.
- Alternative method (LMG Fig. 62.9):
  - Draw a transverse line between the lateral and medial epicondyles of the femur and mark its midpoint.
  - From a point 1 cm lateral to the midpoint of this first line, draw a second line perpendicular to the first one, extending 5–7 cm in a cephalad direction—this is the site of needle insertion.

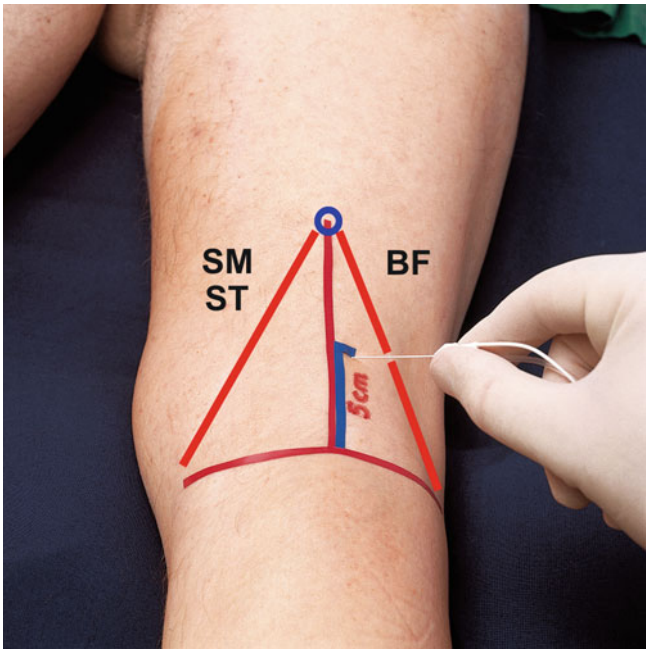


Fig. 62.9 Surface landmarks for the posterior approach to the popliteal nerve block. A transverse line between the lateral and medial epicondyles of the femur (corresponding approximately to the popliteal crease) is drawn and its midpoint marked. The upper borders of the popliteal fossa, formed by semimembranosus (*SM*), semitendinosus (*ST*), and biceps femoris (*BF*) muscles, are palpated and marked. There are two possible insertion points: (1) at the apex of the popliteal fossa (the modified intertendinous approach) or (2) at a point 5–7 cm cephalad and 1 cm lateral to the midpoint of the transverse line between femoral epicondyles (With permission from Danilo Jankovic)

Needle Insertion and Injection Technique

- Disinfect the skin, drape the area appropriately, prime the block needle and tubing with local anesthetic solution, and attach a nerve stimulator at an initial current setting of 1–2 mA, pulse duration of 0.1–0.3 ms, and frequency of 1–2 Hz.
- Fixing the skin over the skin insertion point with the second and third fingers of the nondominant hand, insert a 50–80-mm block needle at a 45–60° angle in a cephalad direction and parallel to the long axis of the leg (LMG Fig. 62.10).
- Advance the needle slowly and steadily until plantar flexion and inversion (tibial nerve, medial aspect of sciatic nerve) or dorsiflexion and eversion (peroneal nerve, lateral aspect) of the foot is obtained at a current threshold of 0.3–0.5 mA.

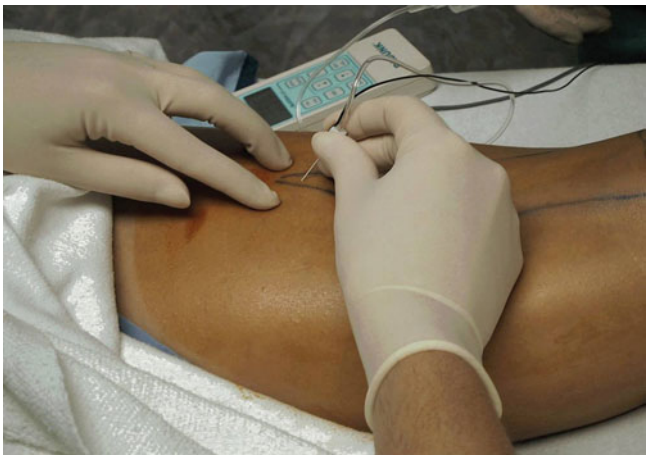


Fig. 62.10 Needle insertion in the surface landmark-guided posterior approach to the popliteal nerve block. The needle is advanced at 45–60° angle in a cephalad direction until an appropriate motor response is obtained at 0.3–0.5 mA

- Incremental injection of local anesthetic is performed with intermittent aspiration to exclude intravascular injection.
- If no motor response is obtained despite inserting the needle to its full depth, the needle should be withdrawn to the skin and redirected in a slightly medial or lateral direction.

Landmark-Guided Popliteal Nerve Block: Lateral Approach

Patient Position

- The patient is placed supine; the knee may be slightly flexed by placing a soft roll underneath it.
- The operator stands on the side of the patient that is to be blocked.

Surface Landmarks (LMG Fig. 62.11)

- The groove between the lateral (posterior) edge of vastus lateralis and biceps femoris is identified; this can be accentuated by asking the patient to extend the knee against resistance.
- A point in the groove 7 cm proximal to the lateral femoral epicondyle is marked—this is the initial needle insertion point. Alternatively, a point 8 cm proximal to the popliteal crease in the groove can also be used.

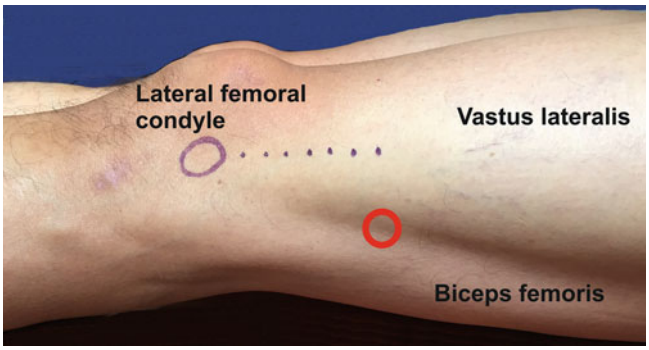


Fig. 62.11 Surface landmarks for the lateral approach to the popliteal nerve block. The intermuscular groove between vastus lateralis (*VL*) and biceps femoris (*BF*) muscles is palpated. The needle insertion point (*red circle*) is in this groove, either 7 cm proximal to the lateral femoral epicondyle or 8 cm proximal to the popliteal crease

Needle Insertion and Injection Technique

- Disinfect the skin, drape the area appropriately, prime the block needle and tubing with local anesthetic solution, and attach a nerve stimulator at an initial current setting of 1.5 mA, pulse duration of 0.1–0.3 ms, and frequency of 1–2 Hz.
- Fixing the skin over the skin insertion point with the second and third fingers of the nondominant hand, insert an 80–100-mm block needle perpendicular to the skin in the horizontal plane (LMG Fig. 62.12).

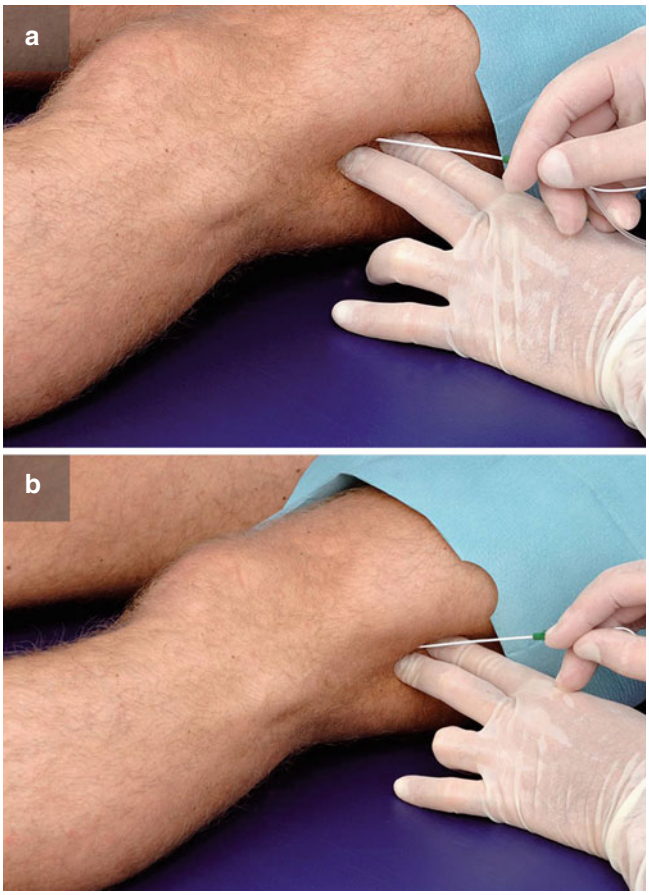


Fig. 62.12 Needle insertion in the surface landmark-guided lateral approach to the popliteal nerve block. The needle is initially advanced perpendicular to the skin to contact the femur (**a**) and the depth noted. The needle is then withdrawn to the skin and redirected posteriorly under the femur (**b**) until an appropriate motor response is obtained at 0.3–0.5 mA (With permission from Danilo Jankovic)

- Advance the needle slowly and steadily until the femur is contacted. The depth of insertion is noted—the sciatic nerve is expected to lie 1–2 cm deeper, posterior to the femur (LMG Fig. 62.13).

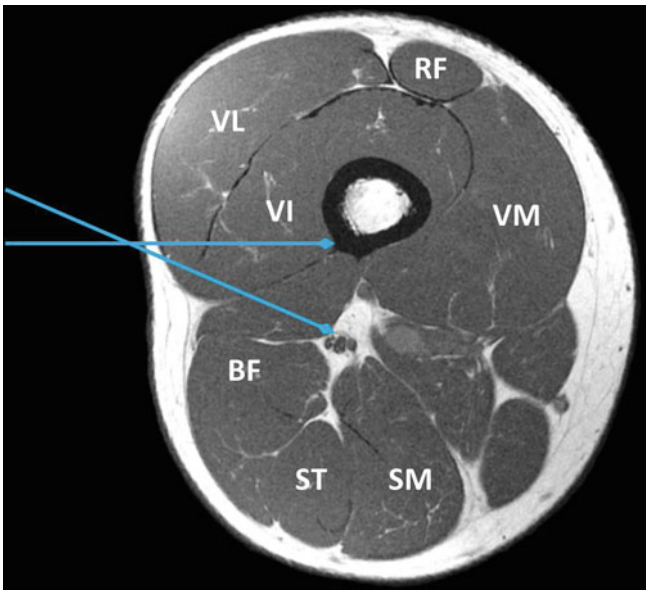


Fig. 62.13 MRI cross section of the thigh indicating the lateral approach to the sciatic nerve. The needle is initially advanced perpendicular to the skin to contact the femur, before being redirected posteriorly at approximately 30° angle to reach the sciatic nerve. *BF* biceps femoris, *RF* rectus femoris, *SM* semimembranosus, *ST* semitendinosus, *VI* vastus intermedius, *VL* vastus lateralis, *VM* vastus medialis

- The needle is then withdrawn to the skin and redirected approximately 30° posteriorly (downward) and advanced posteriorly to (under) the femur until plantar flexion and inversion (tibial nerve, medial aspect of sciatic nerve) or dorsiflexion and eversion (peroneal nerve, lateral aspect) of the foot is obtained at a current threshold of 0.2–0.5 mA.
- Biceps femoris twitches should not be accepted as they may represent local muscle stimulation. If the appropriate motor response is not obtained within 2 cm of cessation of the biceps femoris twitches, it is likely that the nerve has been missed and the needle should be withdrawn and redirected at a shallower or steeper angle.
- Once an appropriate distal motor response has been obtained, incremental injection of local anesthetic is performed with intermittent aspiration to exclude intravascular injection.

Continuous Nerve Block Technique

This is identical to the single-shot technique except that a peripheral nerve block catheter kit is used instead. Either the posterior or lateral approach may be used, although the authors favor the posterior approach as the catheter is more likely to advance within the paraneural sheath along the long axis of the nerve.

The introducer needle is advanced as described above until the desired motor response is obtained. If a stimulating catheter is being used, 5–10 ml of 5 % dextrose solution can be injected through the introducer needle to distend the paraneural sheath. This should also result in augmentation of the motor response. Injection of other solutions will abolish the motor response.

The catheter is then advanced 3–5 cm beyond the tip of the introducer needle, looking to maintain a motor response from the sciatic nerve at current thresholds of at least 0.2 mA and up to 0.8–1 mA. The needle is then withdrawn and the catheter fixed in place in the usual manner.

Technique: Ultrasound-Guided Approach (Single Shot)

Required Supplies and Equipment

- Disinfectant solution and swabs for skin preparation
- Sterile gloves, drapes, and ultrasound probe cover
- Linear 38–50-mm, high-frequency ultrasound probe (at least 7–13 MHz) and machine
- Nerve stimulator (at operator's discretion)
- Short-beveled 22-G block needle with extension tubing
  - 50–80 mm (posterior approach)
  - 80–100-mm needle (lateral approach)
- Local anesthetic of choice in 10- or 20-ml syringes
- Lidocaine 1–2 % in a 3-ml syringe with a 25–27-G hypodermic needle for skin infiltration (at operator's discretion)
- Equipment and supplies for managing life-threatening acute complications, including intralipid for local anesthetic systemic toxicity (LAST)
- Drugs for intravenous sedation during the block (at operator's discretion)

Preparation of Patient

- Obtain informed consent for the block.
- Explain expected clinical course including care of the insensate limb and managing the transition to systemic analgesia.
- Establish intravenous access, supplemental oxygen delivery, and monitoring (ECG, noninvasive blood pressure, pulse oximetry).
- Perform time-out to confirm patient identity and site and side of surgery.

Block Performance

Patient Position

The patient may be placed in 1 of 3 positions:

1. Prone position
2. Supine position
3. Lateral position

- The prone position is probably the most ergonomic one for the operator. The patient is placed prone with little to no flexion at the knees and in a neutral position—internal or external rotation at the hips should be avoided. This is done by placing a small soft roll under the ankles or by allowing the feet to dangle off the foot of the bed.
- The supine position is useful if there are hindrances to turning the patient prone (morbid obesity, leg in a plaster cast,

etc.). To allow probe access to the popliteal fossa, the lower leg may be elevated by placing a roll or other support (e.g., a padded Mayo stand) underneath the calf. The leg may also be simply flexed at the knee, in which case access can be further improved by slight internal rotation at the hip.
- The lateral decubitus position is a third alternative to the prone and supine positions. The operative leg should be uppermost and extended at the knee, with padding (e.g., a small pillow) placed between the knees for comfort.
- Regardless of patient position, attention should be paid to ergonomics to ensure the operator, needle, and machine are aligned to minimize unnecessary movements during the procedure.

Probe Position and Image Optimization

- Place the probe in a transverse orientation above the popliteal crease to obtain a short-axis view of the popliteal fossa.
- Select appropriate depth of field (normally up to 5 cm), gain, and focus.
- Ensure the orientation marker is correctly positioned.
- Use the traceback method described previously to obtain a transverse, short-axis view of the sciatic nerve just distal to its bifurcation.
- Note that with the patient in the supine position, the ultrasound image and needle movements will appear inverted or "upside down" on the screen relative to the actual patient position. The orientation of the image on the screen can also be inverted using the machine settings to compensate for this, or the operator may choose to mentally compensate when redirecting the needle.

Needle Insertion and Injection Technique

- Disinfect the skin, drape the area appropriately, apply the cover to the ultrasound probe, prime the block needle and tubing with local anesthetic solution, and, if being used, attach a nerve stimulator at an initial current setting of 0.5–6 mA, pulse duration 0.1–0.3 ms, and frequency of 1–2 Hz.
- The needle can be inserted either in-plane or out-of-plane to the ultrasound probe and beam (see below).
- If a nerve stimulator is to be used, there should be no twitches elicited below 0.2 mA. This helps to exclude intraneural needle placement. It may not be necessary to reposition the needle just to obtain a minimum current threshold (<0.5 mA) if the injectate is clearly observed to spread within the paraneural sheath and around the nerves.
- The nerves should be carefully observed during injection to exclude intraneural injection, which is indicated by an increase in their cross-sectional area.

Out-of-Plane Needling Technique

- The out-of-plane approach may be used with the patient in either the prone or lateral position.
- Insert the needle perpendicular to the ultrasound probe and parallel to the long axis of the nerve (USG Fig. 62.14).

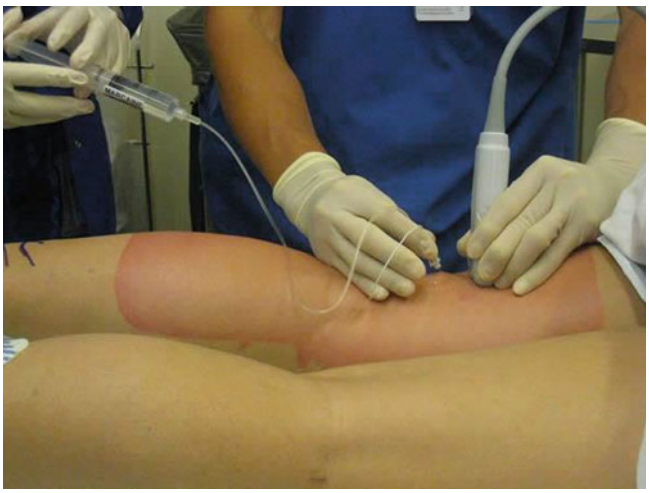


Fig. 62.14 Out-of-plane needle insertion in the popliteal nerve block with the patient in the prone position. As the needle is inserted progressively deeper, the probe must be scanned proximally to track the needle tip (With permission from Dr. Ki Jinn Chin)

- The needle can be challenging to visualize with this technique, and at best, it will appear in short axis on the screen as a white dot (USG Fig. 62.15). However, progress of the needle tip can usually be easily tracked by tissue movement as it penetrates the various layers.
- Care must be taken to distinguish between the shaft and the tip of the needle. To aid needle tip identification, small "side-to-side" or "jiggling" movements can be made and the resultant tissue movement observed to identify the tip.
- Small aliquots (0.5–1 ml) of fluid (5 % dextrose if neurostimulation is being used as it preserves the motor response) can be injected to "hydrolocate" the tip of the needle.
- The aim is to place the needle tip within the paraneural sheath and between the tibial and common peroneal (fibular) nerves and to observe spread of local anesthetic within the sheath. Do not aim directly for the neural structures. Local anesthetic spread within the sheath should be observed (USG Fig. 62.15).

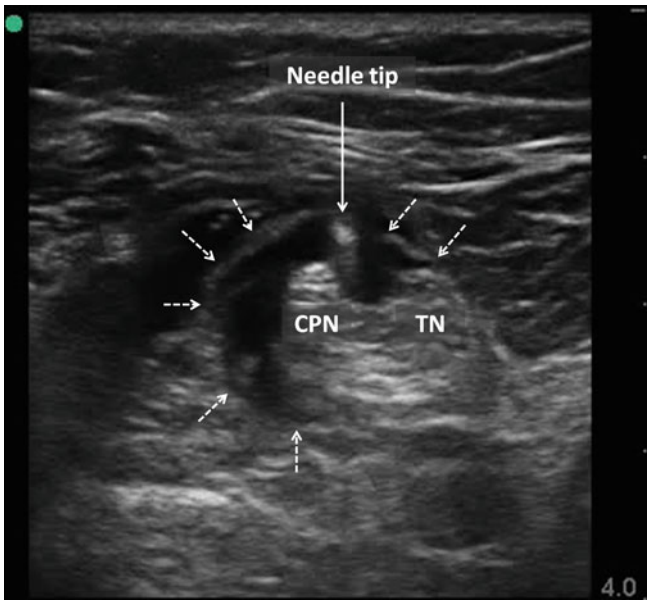


Fig. 62.15 Transverse short-axis view of the common peroneal nerve (*CPN*) and tibial nerve (*TN*) in the popliteal fossa. The needle has been inserted out of plane to the probe and the tip is within the paraneural sheath (*dashed arrows*). Injected local anesthetic has distended the sheath and has begun to spread around the nerves, outlining them and increasing their visibility (With permission from Dr. Ki Jinn Chin)

In-Plane Needling Technique

- The in-plane approach may be used with the patient in either the prone (USG Fig. 62.16), lateral, or supine (USG Fig. 62.17) position.

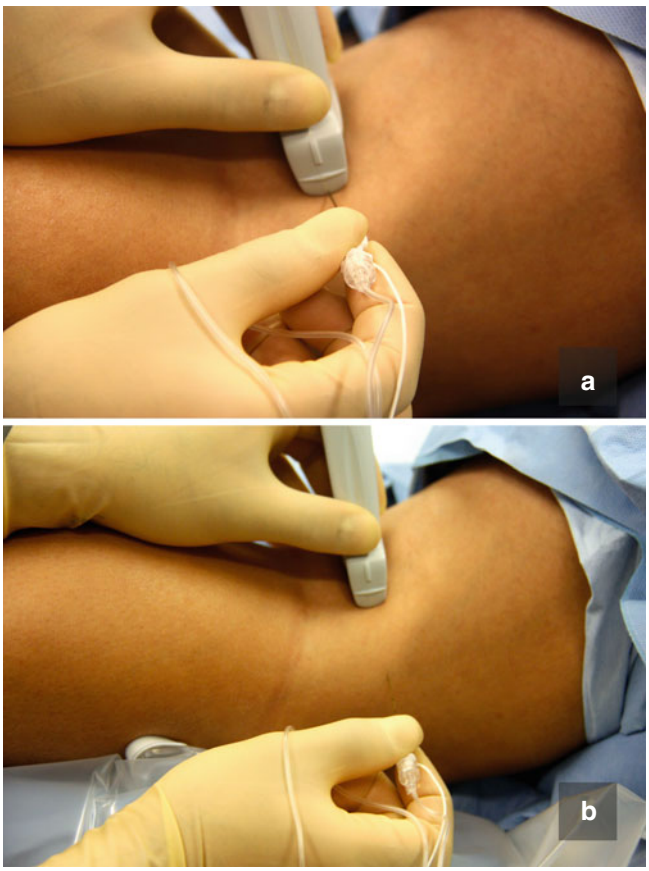


Fig. 62.16 In-plane needle insertion in the popliteal nerve block with the patient in prone position. The needle may be inserted (**a**) immediately adjacent to the probe or (**b**) in the intermuscular groove between the vastus lateralis and biceps femoris. The second approach maximizes needle visibility as the shaft will be perpendicular to the ultrasound beam (With permission from Dr. Ki Jinn Chin)

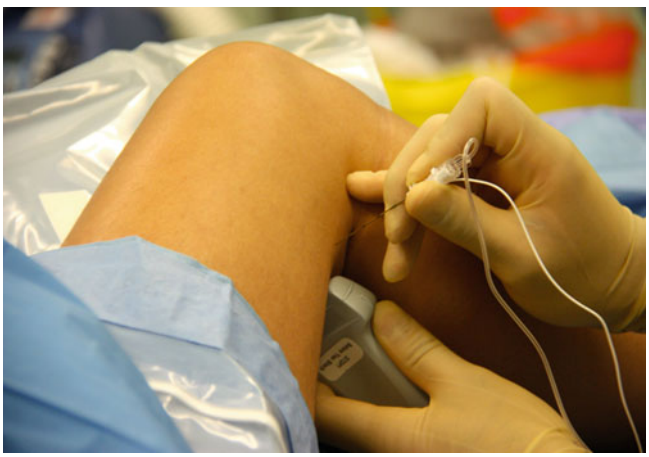


Fig. 62.17 In-plane needle insertion in the popliteal nerve block with the patient in the supine position. The knee has been flexed and rotated slightly inward to facilitate probe placement (With permission from Dr. Ki Jinn Chin)

- The needle is inserted from the lateral aspect of the patient's thigh, either immediately adjacent to the probe (USG Fig. 62.16a) or in the intermuscular groove between the biceps femoris and vastus lateralis (USG Fig. 62.16b). The second approach may allow better needle visualization as the shaft will be perpendicular to the ultrasound beam.
- It is essential to constantly visualize the tip of the needle as it advances, and small sliding movements of the probe should be made as needed to ensure needle-beam alignment.
- The aim is to place the needle tip within the paraneural sheath and between the tibial and common peroneal (fibular) nerves and to observe spread of local anesthetic within the sheath. Do not aim directly for the neural structures. Local anesthetic spread within the sheath should be observed (USG Fig. 62.18).

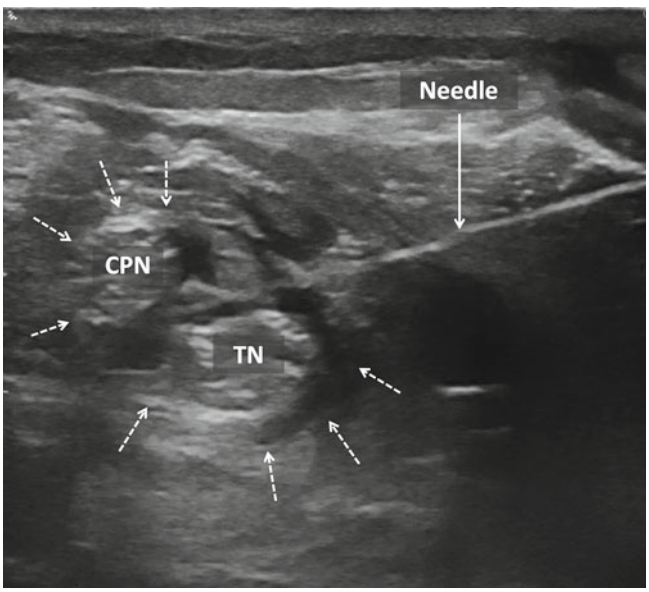


Fig. 62.18 Transverse short-axis view of the common peroneal nerve (*CPN*) and tibial nerve (*TN*) in the popliteal fossa. The needle has been inserted in-plane to the probe, and the tip is within the paraneural sheath (*dashed arrows*) and between the two nerves. Injected local anesthetic is spreading within the paraneural sheath and beginning to outline the nerves (With permission from Dr. Ki Jinn Chin)

Continuous Nerve Block Technique

There are three possible approaches for siting a catheter in the popliteal fossa under ultrasound guidance [8].

These are:

1. Needle in plane with the nerve viewed in the short axis
2. Needle out of plane with the nerve viewed in the short axis
3. Needle in plane with the nerve viewed in the long axis

Even after considerable mastery of ultrasound-guided regional anesthesia, it is a challenge to maintain in-plane visualization of the needle, nerve, and catheter during the procedure [8, 9], so the authors do not recommend using the third approach for catheter insertion.

Needle in Plane with Nerve Viewed in Short Axis

This is identical to the single-shot in-plane technique except that a peripheral nerve block catheter kit is used instead. Either the posterior or lateral approach may be used.

The introducer needle is advanced as described above, and an initial bolus of either local anesthetic or 5 % dextrose solution can be used to distend the paraneural sheath. The catheter is then advanced through the introducer needle no more than 2–3 cm beyond the tip. The introducer needle is then withdrawn. Local anesthetic injection is observed through the catheter under ultrasound guidance, and the catheter can then be withdrawn slightly if required to achieve the optimal position.

The catheter is fixed in place in the usual manner.

Needle Out of Plane with Nerve Viewed in Short Axis

This is identical to the single-shot out-of-plane technique except that a peripheral nerve block catheter kit is used instead. Either the posterior or lateral approach may be used, though technically, it may be easier to perform with the patient in the prone position. This is the authors' preferred technique as the catheter can be advanced further within the paraneural sheath along the long axis of the nerve, which reduces the risk of dislodgement.

The introducer needle is advanced as described above and an initial bolus of either local anesthetic or 5 % dextrose solution can be used to distend the paraneural sheath. The catheter is then advanced through the introducer needle 3–5 cm beyond the tip. Local anesthetic injection is observed through the catheter under ultrasound guidance to confirm optimal position. If necessary, in addition, a proximal scan should be able to confirm perineural catheter placement. The introducer needle is withdrawn and the catheter is fixed in place in the usual manner.

The areas of cutaneous innervation of the individual nerves discussed in this chapter are illustrated in Fig. 62.19.

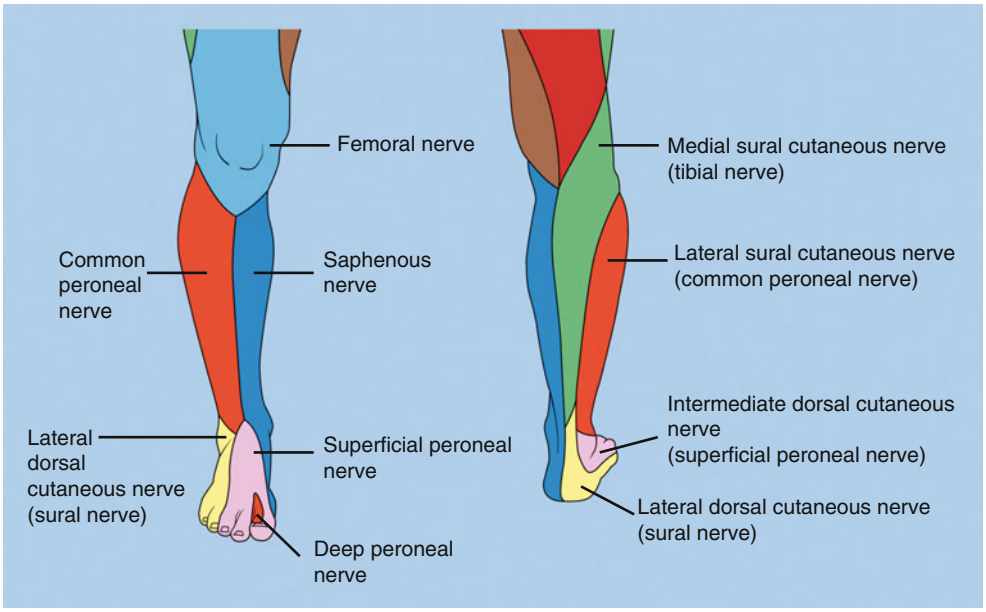


Fig. 62.19 Sensory innervation areas in the lower leg (Reproduced with permission from Danilo Jankovic)

Local Anesthetic Dosages

Single-Shot Block

- A volume of 30–40 ml (0.4–0.5 ml/kg) is generally recommended for the landmark-guided approach to popliteal nerve block [7, 10].
- The choice of local anesthetic and concentration depends on the desired speed of onset and duration. Commonly used local anesthetics include:
  - A 1:1 mixture of an intermediate-acting local anesthetic (e.g., 2 % mepivacaine or lidocaine) and a long-acting local anesthetic (e.g., 0.5 % bupivacaine), which will have a faster onset than bupivacaine alone and a longer duration than lidocaine alone.
  - 0.25–0.5 % bupivacaine.
  - 0.5–0.75 % ropivacaine.
- Epinephrine may be added in a concentration of 2.5 mcg/ml as a marker of intravascular injection and to reduce systemic vascular absorption.

Continuous Popliteal Sciatic Nerve Block

- 0.2–0.4 % ropivacaine at 4–8 ml/h [11].
- 0.125–0.25 % bupivacaine at 4–8 ml/h.
- PCA boluses of 2–5 ml may be added if this function is available on the infusion pump [11, 12].
- Both infusion rates and bolus volumes should be titrated to achieve the optimal balance between adequate analgesia and an excessively dense sensory and motor block.

Complications and Adverse Effects

- Complications associated with popliteal nerve block are uncommon.
- Potential complications are similar to that of any peripheral nerve block and include:
  - Local bruising and pain at the site of needle insertion
  - Hematoma formation
  - Intravascular injection
  - Local anesthetic systemic toxicity (LAST)
  - Neurologic injury and deficit
- Patients should be counseled on appropriate care of the insensate limb, and in particular the heel should be protected against pressure injury.

References

1. Perlas A, Wong P, Abdallah F, Hazrati L-N, Tse C, Chan V. Ultrasound-guided popliteal block through a common paraneural sheath versus conventional injection: a prospective, randomized, double-blind study. Reg Anesth Pain Med. 2013;38:218–25.
2. Franco CD. Connective tissues associated with peripheral nerves. Reg Anesth Pain Med. 2012;37:363–5.
3. Karmakar MK, Shariat AN, Pangthipampai P, Chen J. High-definition ultrasound imaging defines the paraneural sheath and the fascial compartments surrounding the sciatic nerve at the popliteal fossa. Reg Anesth Pain Med. 2013;38:447–51.
4. Sala-Blanch X, Vandepitte C, Laur JJ, Horan P, Xu D, Reina MA, Karmakar MK, Clark TB, Hadzic A. A practical review of perineural versus intraneural injections: a call for standard nomenclature. Int Anesthesiol Clin. 2011;49:1–12.
5. Andersen HL, Andersen SL, Tranum-Jensen J. Injection inside the paraneural sheath of the sciatic nerve: direct comparison among ultrasound imaging, macroscopic anatomy, and histologic analysis. Reg Anesth Pain Med. 2012;37:410–4.
6. Tsui BCH, Finucane BT. The importance of ultrasound landmarks: a "traceback" approach using the popliteal blood vessels for identification of the sciatic nerve. Reg Anesth Pain Med. 2006;31:481–2.
7. Nader A, Kendall MC, Candido KD, Benzon H, McCarthy RJ. A randomized comparison of a modified intertendinous and classic posterior approach to popliteal sciatic nerve block. Anesth Analg. 2009;108:359–63.
8. Ilfeld BM, Fredrickson MJ, Mariano ER. Ultrasound-guided perineural catheter insertion: three approaches but few illuminating data. Reg Anesth Pain Med. 2010;35:123–6.
9. Edward Kim T, Howard SK, Funck N, Kyle Harrison T, Walters TL, Wagner MJ, Ganaway T, Mullens J, Lehnert B, Mariano ER. A randomized comparison of long-axis and short-axis imaging for in-plane ultrasound-guided popliteal-sciatic perineural catheter insertion. J Anesth. 2014;28:854–60. doi:10.1007/s00540-014-1832-8.
10. Taboada M, Rodríguez J, Valiño C, Carceller J, Bascuas B, Oliveira J, Alvarez J, Gude F, Atanassoff PG. What is the minimum effective volume of local anesthetic required for sciatic nerve blockade? A prospective, randomized comparison between a popliteal and a subgluteal approach. Anesth Analg. 2006;102:593–7.
11. Ilfeld BM, Loland VJ, Gerancher JC, Wadhwa AN, Renehan EM, Sessler DI, Shuster JJ, Theriaque DW, Maldonado RC, Mariano ER. PAINfRE Investigators: the effects of varying local anesthetic concentration and volume on continuous popliteal sciatic nerve blocks: a dual-center, randomized, controlled study. Anesth Analg. 2008;107:701–7.
12. Paqueron X, Narchi P, Mazoit J-X, Singelyn F, Bénichou A, Macaire P. A randomized, observer-blinded determination of the median effective volume of local anesthetic required to anesthetize the sciatic nerve in the popliteal fossa for stimulating and nonstimulating perineural catheters. Reg Anesth Pain Med. 2009;34:290–5.

Chapter 63

The Adductor Canal Block

Jeffrey Ghassemi, MD, MPH (✉)
HS Assistant Clinical Professor, Department of Anesthesiology and Perioperative Care, University of California, San Francisco, CA, USA
e-mail: ghassemij@anesthesia.ucsf.edu

Andrew T. Gray, MD, PhD
Department of Anesthesia and Perioperative Care, Room 3C-38, San Francisco General Hospital, University of California, San Francisco, CA 94110, USA
e-mail: graya@anesthesia.ucsf.edu

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_63,

Key Points

- Not a definitive anesthetic (part of multimodal analgesia).
- Provides better disposition than femoral block (but can still cause some impairment in motor strength).
- Optimal location along the adductor canal in the thigh is still yet to be established.
- Femoral nerve block may be a more shallow alternative in morbidly obese patients.
- Questions remain and will be the subject of future clinical research on the ACB.

Introduction

The adductor canal block (ACB), or more precisely the saphenous nerve block in the adductor canal, is a regional technique that capitalizes on the anatomic relationship of branches of the femoral nerve as they course through the adductor canal in order to achieve anesthesia and analgesia for lower extremity surgery. While the technique itself is not new, the reported analgesic effect and motor-sparing benefit of this block for surgical procedures about the knee has recently drawn much attention.

Indications

Blocking the saphenous nerve as it travels within the adductor canal may be used in combination with a sciatic nerve block to complement anesthesia for various procedures of the lower leg. As previously mentioned, the ACB also has shown promise in providing anesthesia and analgesia in a similar fashion to the traditional femoral nerve block for open and arthroscopic procedures on the knee. Either a single-shot or continuous technique may be used.

Contraindications

Relative contraindications for ACB may include prior surgery at the thigh (i.e., vascular or skin grafts), local infection, tumor, or preexisting neuropathy.

Anatomy (Fig. 63.1a–d)

The adductor canal (canalis adductorius, Hunter's canal, subsartorial canal) is a musculoaponeurotic tunnel in the middle third of the thigh, extending from the distal apex of the femoral triangle—defined by the crossing of the medial margin of the adductor longus muscle and the medial margin of the sartorius muscle—to the adductor hiatus, an opening in the adductor magnus [1, 2]. The muscular boundaries of the canal are the sartorius (anterior), vastus medialis (anterolateral), and adductor longus/magnus (posteromedial). The vastoadductor membrane, which varies in thickness, is a continuum of the aponeurotic roof of the canal that covers the distal part of the canal [3]. The canal contains the femoral artery and vein, the descending genicular artery and muscular branches of the femoral artery, the nerve to the vastus medialis, and the saphenous nerve.

The saphenous nerve is the largest and longest branch of the femoral nerve. It is a pure sensory nerve of the medial leg distal to the knee and arises from the posterior division of the femoral nerve. It courses lateral to the femoral artery in the proximal thigh, along the roof of the adductor canal, and then crosses anteriorly over the femoral artery to lie medial to it in the distal aspect of the canal [4]. The saphenous nerve enters the adductor canal after most of the motor branches of the femoral nerve have divided. The exception is the nerve to the vastus medialis, a motor nerve that travels with the saphenous nerve in the adductor canal under the sartorius muscle and anterolateral to the femoral artery [5, 6].

After entering the adductor canal, the saphenous nerve can divide either within the canal or distally in the subsartorial fat [7]. It gives off the infrapatellar branch and other cutaneous branches, which may anastomose with cutaneous branches from the obturator nerve and medial cutaneous nerve of the thigh to form a subsartorial plexus [7–9]. The infrapatellar branch can either run parallel to the saphenous nerve or pierce the distal part of the sartorius muscle. The saphenous nerve consistently emerges into the subcutaneous tissue between the tendons of the sartorius and gracilis muscles [10, 11]. The saphenous nerve travels with the saphenous branch of the descending genicular artery just beneath the sartorius muscle [12–14]. This occurs where the femoral artery descends through the adductor hiatus into the popliteal fossa.

The posterior articular branch of the obturator nerve either enters the distal part of the adductor canal or penetrates the adductor magnus muscle on its way to the posterior knee capsule in the popliteal fossa [7–9, 15]. This nerve branch may contribute to the analgesic effect of the adductor canal block for knee surgery. However, the course of the nerve in the distal thigh and its relation to the adductor canal varies, and the nerve may not have any meaningful clinical role following saphenous nerve blocks in the adductor canal.

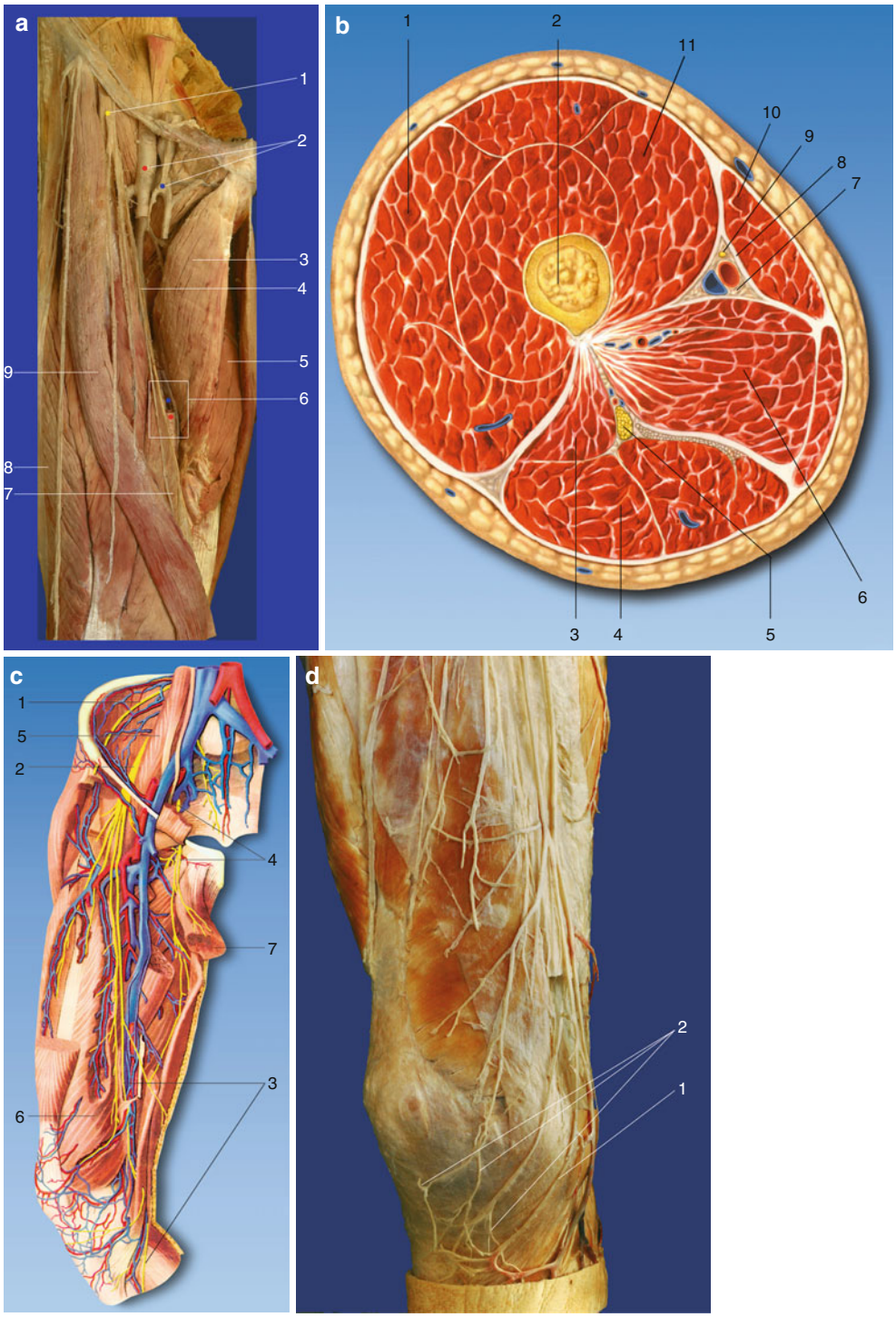


Fig. 63.1 (**a**) Dissection of front of thigh and adductor region. (*1*) Femoral nerve, (*2*) femoral artery and vein, (*3*) adductor longus muscle, (*4*) saphenous nerve, (*5*) adductor magnus muscle, (*6*) vastoadductor membrane and adductor canal with saphenous nerve and femoral artery and vein, (*7*) vastus medialis muscle, (*8*) vastus lateralis muscle, (*9*) sartorius muscle (With permission from Danilo Jankovic). (**b**) Cross-sectional depiction of the thigh. (*1*) Vastus lateralis muscle, (*2*) femur, (*3*) biceps femoris (short head), (*4*) biceps femoris (long head), (*5*) sciatic nerve, (*6*) adductor muscles, (*7*) femoral artery–nerve, (*8*) vastoadductor membrane, (*9*) saphenous nerve, (*10*) sartorius muscle, (*11*) vastus medialis muscle (With permission from Danilo Jankovic). (**c**) Depiction of medial thigh. (*1*) Lateral femoral cutaneous nerve, (*2*) femoral nerve, (*3*) saphenous nerve, (*4*) obturator nerve, (*5*) psoas major muscle, (*6*) vastus medialis muscle, (*7*) adductor longus muscle (With permission from Danilo Jankovic). (**d**) Medial view of cadaveric thigh. (*1*) Saphenous nerve (infrapatellar branch), (*2*) superior medial and inferior medial genicular arteries (With permission from Danilo Jankovic)

Only a few of the nerves of the subsartorial plexus, specifically the saphenous nerve, infrapatellar nerve, and the nerve to the vastus medialis muscle, can be identified with ultrasound imaging. It is unclear to what extent all the plexus or just the saphenous nerve and its branches are involved in adductor canal block.

Landmarks

Based on the aforementioned anatomical definition of the adductor canal, identification by external surface landmarks can prove misleading. To assume that a position halfway between the base of the patella and the anterior superior iliac spine is within the canal can be erroneous and may mislead clinicians to choose a needle entry site proximal to the entrance of the canal within the femoral triangle [2]. Rather, the adductor canal may be reliably identified using sonographic landmarks (vastoadductor membrane, sartorius muscle, adductor longus muscle, vastus medialis muscle, and femoral vessels; Fig. 63.2).

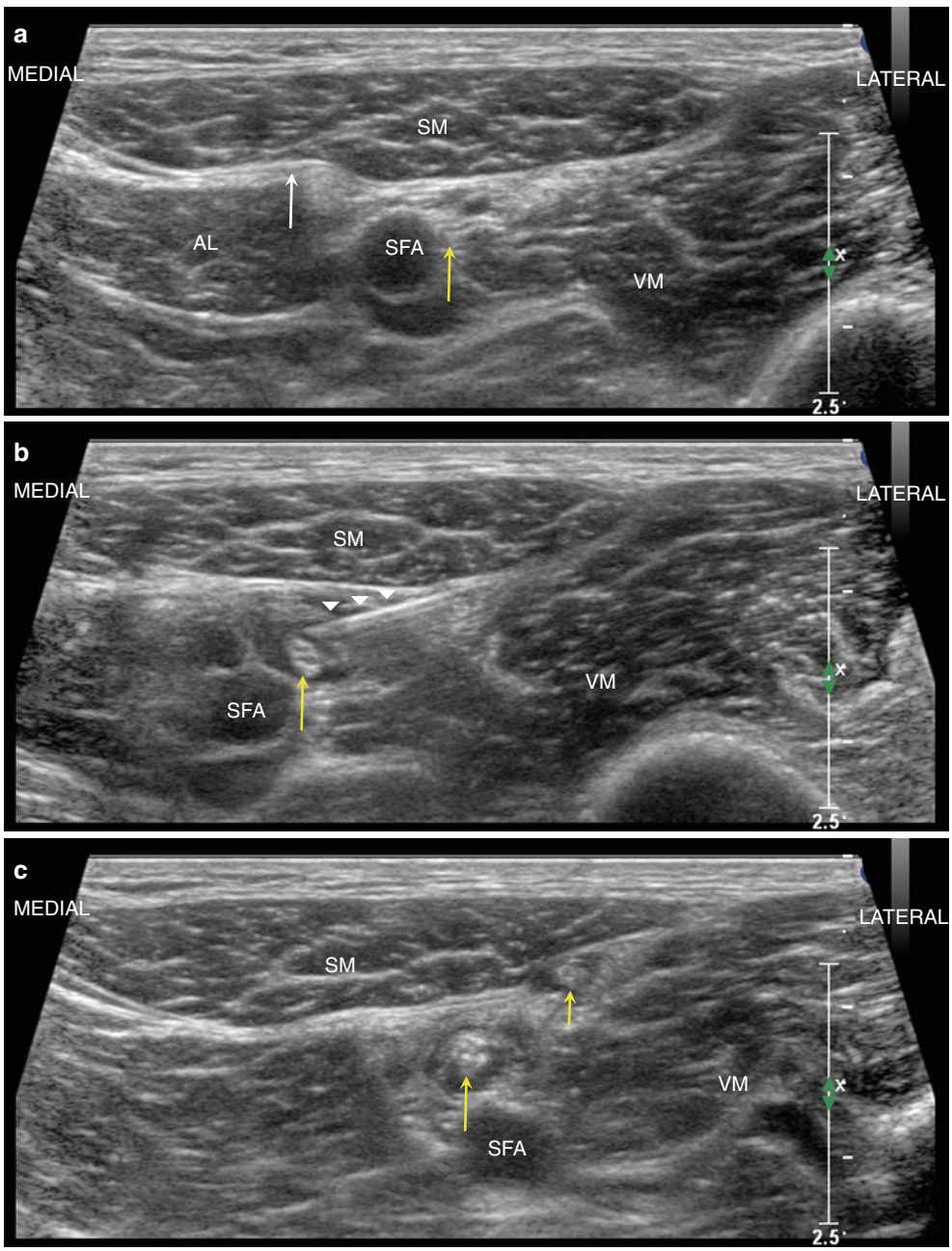


Fig. 63.2 Serial sonograms demonstrating adductor canal block. Panel (**a**) baseline sonogram prior to injection (pre-scan). The saphenous nerve (*long yellow arrow*) lies deep to the sartorius muscle (*SM*) and hyperechoic vastoadductor membrane (*white arrow*) and is adjacent to the superficial femoral artery (*SFA*); adductor longus muscle (*AL*). Panel (**b**) in-plane approach to adductor canal block (the needle is shown with *white arrowheads*). Panel (**c**) distributions after injection tracked distally in the thigh demonstrating local anesthetic surrounding the saphenous (*long yellow arrow*) and infrapatellar (*short yellow arrow*) nerves. *VM* vastus medialis

A high-frequency linear ultrasound probe is placed on the medial aspect of the thigh using a transverse plane of imaging. The saphenous nerve can be identified in its short axis as it courses alongside the femoral artery in the adductor canal. As it travels distally in the thigh, the femoral artery, which appears immediately posterior to the sartorius muscle, eventually "dives" deep and moves away from the muscle plane of the sartorius toward the posterior aspect of the thigh where it becomes the popliteal artery [4]. This point is considered to be the adductor hiatus and the distal extent of the adductor canal.

Equipment

A standard regional anesthesia tray may include the following:

- Ultrasound machine with linear transducer (8–14 MHz), sterile cover, and acoustic coupling gel
- A 38-mm 25-gauge needle for skin infiltration
- A syringe containing 5–15 mL of local anesthetic
- A 50–70 mm, 22-gauge echogenic needle
- Sterile gloves
- A standard nerve catheter tray prepared with needle, catheter, and sterile drape (for continuous technique).

Suggested Block Technique

After placement of routine monitors, peripheral venous access, and sedation (if required), the patient is positioned supine with the operative leg slightly flexed at the knee and externally rotated; this will facilitate coverage of the saphenous nerve and superficial femoral artery by the sartorius muscle (Fig. 63.3). Sterile skin preparation should always occur prior to block.

As previously mentioned, ultrasound can be used to guide the saphenous nerve block in the adductor canal and anywhere along its course. The choice of approach may be somewhat arbitrary—although too proximal, an approach can anesthetize motor branches of the femoral nerve, while too distal an approach may theoretically risk paresthesia, nerve puncture, or nerve entrapment [16–19]. Despite much debate regarding the anatomy and nomenclature of the adductor canal, present clinical studies have failed to show significant differences when comparing various locations and approaches to these regional blocks in the thigh [16, 20, 21].

A myriad of techniques have been proposed based on external vs. internal landmarks, nerve stimulation vs. ultrasound, and the amount of total local anesthetic used [2, 4, 16]. While debate and clinical research is ongoing, this section presents a safe and effective approach for blocking the saphenous nerve in the adductor canal.

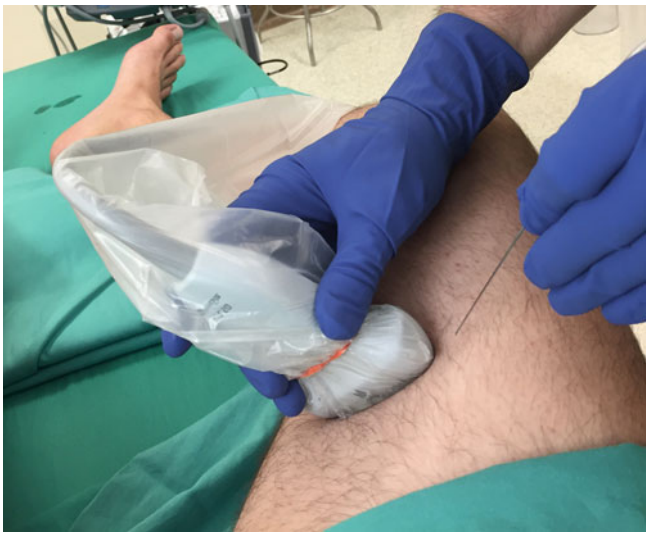


Fig. 63.3 External photography showing ultrasound-guided, in-plane approach to adductor canal block in the thigh (Photograph courtesy of Adam B. Collins, MD)

Single Injection

Place the ultrasound machine on the opposite side of the bed so that the block site and display are both in front of the operator. Perform the ACB with the sartorius muscle viewed in short axis while advancing the needle in the plane of imaging. The needle approach is from the anterior side at a level sonographically determined within the adductor canal, typically with the superficial femoral artery directly in the midpoint and deep to the sartorius muscle (Figs. 63.2 and 63.4). Because of the relatively steep angle of insertion through the sartorius muscle, an echogenic needle is typically selected with this approach [3]. This steep approach may prove problematic in the morbidly obese patient, in which case the femoral nerve block may be a shallower alternative. However, as long as the echogenic vastoadductor membrane is visualized sonographically under the posterior border of the sartorius muscle, success can usually be achieved with this block.

Once through skin and the subcutaneous tissues, the needle is placed through the sartorius muscle to enter the plane deep to the muscle (i.e., transsartorial approach; Fig. 63.4). There may be a loss of resistance as the needle tip crosses the vastoadductor membrane. Local anesthetic is injected within this plane, adjacent to the femoral artery. While the bevel of the needle initially faces the transducer to improve needle-tip visibility, rotating it 180° (bevel down) and advancing the needle just above the artery can extend distribution of local anesthetic under the sartorius muscle [3].

Another approach to the saphenous nerve involves advancing the needle in plane, with a short-axis view, through the superficial part of the vastus medialis muscle in a lateral-to-medial direction deep to the sartorius muscle and vastoadductor membrane. This may provide improved needle-tip visibility and minimize vascular puncture. The needle tip is then placed next to the saphenous nerve and local anesthetic injected [3].

While much of the clinical pharmacology of injection volume in relation to onset, duration, and degree of motor blockade is still under investigation, volumes of 5–15 mL are commonly used for the single-shot ACB.

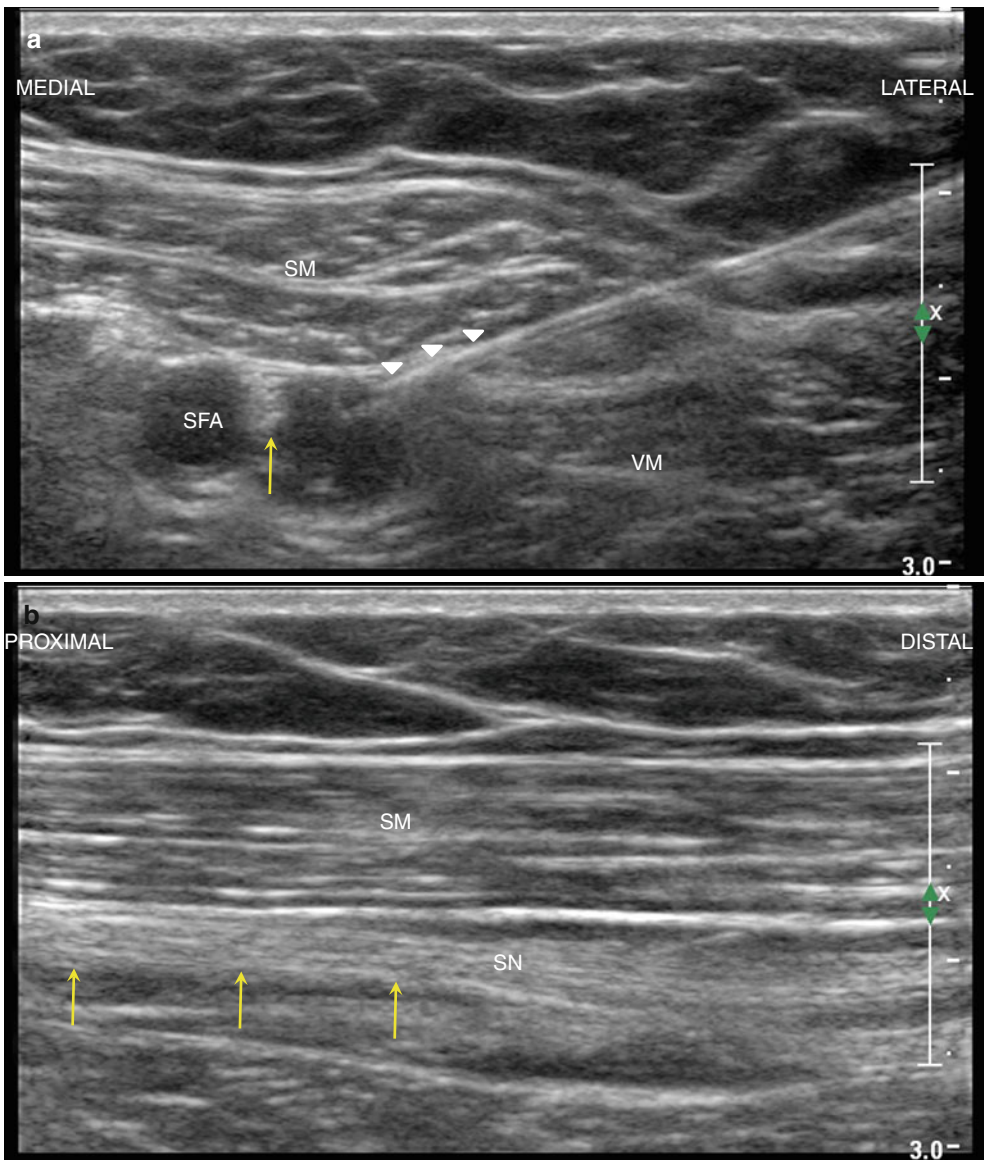


Fig. 63.4 Serial sonograms demonstrating in-plane approach to adductor canal block deep to the sartorius muscle (*SM*). Panel (**a**) needle tip in plane for block injection (*white arrowheads*). Panel (**b**) long-axis view of the saphenous nerve (*SN*, *long yellow arrows*) after injection demonstrating local anesthetic tracking along the nerve course in the thigh

Continuous Technique

The continuous technique is similar to the single-injection approach. After dilation of the adductor canal with sterile saline or local anesthetic, advance the catheter 1–3 cm beyond the needle tip posterior to the nerve under direct ultrasound visualization (if possible). Withdraw the needle carefully while gently advancing the catheter to prevent recoil of the catheter from the insertion site. Catheter tip position may be assessed by injecting 0.5 mL of air via the catheter under ultrasound imaging [22]. The catheter should then be secured either by tunneling or applying a biocompatible tissue adhesive and occlusive adhesive dressing.

If the catheter is placed preoperatively, there should be communication with the surgical team regarding the location of the catheter, as a more distal approach may lie within the planned sterile surgical field or underneath the thigh tourniquet. The presence of a continuous catheter under the pressure of a tourniquet may cause pressure damage to the nerves, although this notion has not yet been tested by formal research. There is no formal study to support the belief that a tourniquet increases the incidence of catheter dislodgement, and observations may actually show that a thigh tourniquet reinforces the security of the catheter [23]. There is also concern that, given intraoperative manipulation of the knee, catheters placed preoperatively may be at risk for migration or dislodgement out of the adductor canal. Neither catheter type (rigid stimulating vs. flexible) vs. nor subcutaneous tunneling has shown to have an effect on migration within the canal [24].

Block Assessment

There are several sonographic signs of an effective ACB. Imaging should demonstrate the distribution of hypoechoic local anesthetic solution around the saphenous nerve in short- and long-axis views (Fig. 63.5). Specifically, the injectate should spread in a semilunar fashion on the anteromedial aspect of the femoral artery and saphenous nerve with displacement of the artery. If, upon injection, the sartorius muscle is lifted before the femoral artery is displaced, then the needle tip is likely intramuscular and should be repositioned. Finally, the saphenous nerve should appear more hyperechoic following the injection because of acoustic enhancement from the hypoechoic local anesthetic solution [3].

Clinically, the medial leg and foot will show signs of cutaneous anesthesia, with the distal extent of sensory blockade usually reaching the base of the great toe [25]. As the nerve to the vastus medialis muscle lies within the adductor canal, there may be a component of motor weakness associated with a successful ACB, though this was shown to be clinically insignificant in healthy volunteers receiving the ACB [26].

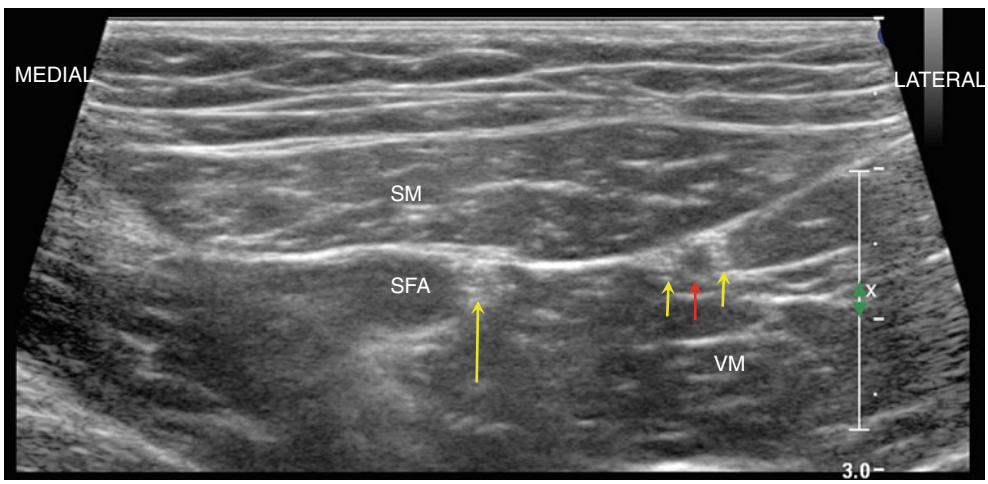


Fig. 63.5 Sonogram showing nerves to the vastus medialis (*VM*) (*short yellow arrows*) entering this muscle together with a corresponding small artery (*short red arrow*) that arises from the superficial femoral artery (*SFA*). The saphenous nerve (*long yellow arrow*) lies deep to the sartorius muscle (*SM*)

Patient Disposition

Although motor nerves to the vastus medialis muscle lie within the adductor canal, human volunteer studies have shown that adductor canal blocks spare quadriceps motor strength to a much greater degree than femoral blocks [26, 27]. This quadriceps strength advantage may extend to the clinical setting after total knee arthroplasty [28]. Ambulation advantages have also been observed with the ACB [29–31]. Despite such promise, there exists the potential for proximal spread of local anesthetic to the common femoral nerve, even with small volumes, resulting in clinically significant quadriceps weakness [17]. Moreover, isolated quadriceps weakness may occur from nerve injury or compression, surgical trauma, pain, compartment syndrome, or muscle injury. The potential for quadriceps weakness following the ACB, even if reduced compared to a femoral nerve block, necessitates careful postoperative monitoring with fall precautions.

Choice of Local Anesthetic

Single Injection

The choice of the type and concentration of local anesthetic for the ACB, as with other regional blocks, should be based on whether it is planned for surgical anesthesia or post-op analgesia, as well as the duration required. An example for surgical anesthesia would be 0.5 % ropivacaine or bupivacaine, whereas a lesser concentration (i.e., 0.25 %) may be used when the goal is for postoperative analgesia. While the optimal volume is still debated, 5–15 mL of local anesthetic is commonly used.

Continuous Technique

Like for the single-injection technique, the optimal dose of local anesthetic solution to maintain a continuous adductor canal block has not yet been determined. A dilute solution of long-acting local anesthetic, such as 0.2 % ropivacaine or 0.125 % bupivacaine, is commonly used, with rates of 4–8 mL/h.

Complications

As with other nerve blocks, hematoma, vascular puncture, nerve injury, and infection are potential complications with ACB. Because of the proximity of the saphenous nerve to the femoral artery, there is an inherent risk of vascular puncture when advancing the needle tip toward the nerve. Also, it is theoretically possible to place the needle tip through or into one or more of the nerve branches within the adductor canal. However, because these nerve branches are small and easily displaced in the soft tissue, this risk appears to be low in clinical practice [3].

Considerations

Rising concerns over quadriceps weakness and fall risk—a source of complications and costs for patients and health systems—have prompted a shift at some institutions from femoral nerve blockade to the ACB [32–34]. This shift is bolstered by recent research suggesting comparable analgesia with less motor weakness to a femoral blockade, and superior analgesia when compared to no block, for surgical procedures of the knee including TKA and knee arthroscopy (i.e., ACL repair, meniscectomy) [35]. Preliminary research also shows further systems benefits, with a decreased provider workload with implementation of ACB for TKA pathways when compared to femoral nerve catheters [36].

Table 63.1 presents further questions for consideration and research regarding the role of ACB for surgical procedures of the knee. Such questions may need to be addressed prior to supporting a universal change from femoral nerve blockade to ACB for these patients.

Proponents of the ACB support incorporating this block in their TKA pathways, even if it does not provide enough analgesia per se. They argue that many institutions follow an integrated multimodal analgesic protocol, as defined by the American Society of Anesthesiologists, whereby two or more analgesic modalities with different mechanisms of action are used to provide superior analgesia and minimize side effects and adverse events [37]. Similarly, adding the ACB to a protocol of local infiltration analgesia further improves pain scores and ambulation in this setting [38].

Table 63.1 Considerations and areas for further investigation

Role of multimodal analgesia
Local infiltration analgesia
Strength vs. mobility (ambulation)
Ideal location to inject
Ideal volume to inject
Delayed weakness
Fall risk
Placement of the block relative to thigh tourniquet
Tunneled vs. non-tunneled catheters

Summary

By understanding the anatomic relationships within the thigh, the ultrasound-guided ACB is a relatively easy block to perform for surgical procedures of the lower extremity below the knee when combined with a sciatic nerve block. The ACB has also shown promise in providing a similar degree of analgesia for surgical procedures of the knee while offering a motor-sparing benefit as compared to the femoral nerve block. While further research is necessary to answer outstanding questions about this block's potential for knee procedures, such as total knee arthroplasty, the ultrasound-guided ACB offers the potential to spare the major motor branches of the femoral nerve and reliably anesthetize its major sensory branch, the saphenous nerve.

References

1. Gray H, Lewis WH. Anatomy of the human body. New York: Bartleby.com; 2000, S.I.
2. Bendtsen TF, Moriggl B, Chan V, Pedersen EM, Borglum J. Redefining the adductor canal block. Reg Anesth Pain Med. 2014;39(5):442–3.
3. Andersen HL, Gray AT (2012) The saphenous nerve block. ASRA News. http://www.asra.com/Newsletters/feb-2012.pdf. Accessed 16 Oct 2014.
4. Manickam B, Perlas A, Duggan E, Brull R, Chan V, 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.
5. Thiranagama R. Nerve supply of the human vastus medialis muscle. J Anat. 1990;170:193–8.
6. Jojima H, Whiteside LA, Ogata K. Anatomic consideration of nerve supply to the vastus medialis in knee surgery. Clin Orthop Relat Res. 2004;423:157–60.
7. Horner G, Dellon AL. Innervation of the human knee joint and implications for surgery. Clin Orthop Relat Res. 1994;301:221–6.
8. Gardner E. The innervation of the knee joint. Anat Rec. 1948;101(1):109–30.
9. Strandring S, Gray H. The anatomical basis of clinical practice. London: Churchill Livingstone; 2008.
10. Hunter LY, Louis DS, Ricciardi JR, O'Connor GA. The saphenous nerve: its course and importance in medial arthrotomy. Am J Sports Med. 1979;7(4):227–30.
11. Arthornthurasook A, Gaew-Im K. Study of the infrapatellar nerve. Am J Sports Med. 1988;16(1):57–9.
12. Scheibel MT, Schmidt W, Thomas M, Von Salis-Soglio G. A detailed anatomical description of the subvastus region and its clinical relevance for the subvastus approach in total knee arthroplasty. Surg Radiol Anat. 2002;24(1):6–12.
13. Krombach J, Gray AT. Sonography for saphenous nerve block near the adductor canal. Reg Anesth Pain Med. 2007;32:369–70.
14. Tsui BC, Ozelsel T. Ultrasound-guided transsartorial perifemoral artery approach for saphenous nerve block. Reg Anesth Pain Med. 2009;34(2):177–8.
15. Kumka M. Critical sites of entrapment of the posterior division of the obturator nerve: anatomical considerations. J Can Chiropr Assoc. 2010;54(1):33–42.
16. Mariano ER, Kim E, Wagner MJ, Funck N, Karrison TK, Walters T, Giori N, Woolson S, Ganaway T, Howard SK. A randomized comparison of proximal and distal ultrasound-guided adductor canal catheter insertion sites for knee arthroplasty. J Ultrasound Med. 2014;33:1653–62.
17. Veal C, Auyong DB, Hanson NA, Allen CJ, Strodtbeck W. Delayed quadriceps weakness after continuous adductor canal block for total knee arthroplasty: a case report. Acta Anaesthesiol Scand. 2014;58:362–4.
18. Porr J, Chrobak K, Muir B. Entrapment of the saphenous nerve at the adductor canal affecting the infrapatellar branch – a report on two cases. J Can Chiropr Assoc. 2013;57(4):341–9.
19. Romanoff ME, Cory Jr PC, Kalenak A, Keyser GC, Marshall WK. Saphenous nerve entrapment at the adductor canal. Am J Sports Med. 1989;17(4):478–81.
20. Head SJ, Leung RC, Hackman GP, Seib R, Rondi K, Schwarz SK. Ultrasound-guided saphenous nerve block – within versus distal to the adductor canal: a proof-of-principle randomized trial. Can J Anaesth. 2014. doi:10.1007/s12630-014-0255-1.
21. Adoni A, Paraskeuopoulos T, Saranteas T, Sidiropoulou T, Mastrokalos D, Kostopanagiotou G. Prospective randomized comparison between ultrasound-guided saphenous nerve block within and distal to the adductor canal with low volume of local anesthetic. J Anaesthesiol Clin Pharmacol. 2014;30(3):378–82.
22. Kan JM, Harrison TK, Kim TE, Howard SK, Kou A, Mariano ER. An in vitro study to evaluate the utility of the "air test" to infer perineural catheter tip position. J Ultrasound Med. 2013;32(3):529–33.
23. Moore DM, O'Gara A, Duggan M. Continuous saphenous nerve block for total knee arthroplasty: when and how? Reg Anesth Pain Med. 2013;38(4):370–1.
24. Leng JC, Harrison TK, Miller B, Howard SK, Conroy M, Udani A, Shum C, Mariano ER. A pilot study to assess adductor canal catheter tip migration in a cadaver model. J Anesth. 2014. doi:10.1007/s00540-014-1922-7.
25. Benzon HT, Sharma S, Calimaran A. Comparison of the different approaches to saphenous nerve block. Anesthesiology. 2005;102:633–8.
26. Jaeger P, Nielsen ZJ, Henningsen MH, Hilsted KL, Mathiesen O, Dahl JB. Adductor canal block versus femoral nerve block and quadriceps strength: a randomized, double-blind, placebo-controlled, crossover study in healthy volunteers. Anesthesiology. 2013;118(2):409–15.
27. Kwofie MK, Shastri UD, Gadsden JC, Sinha SK, Abrams JH, Xu D, Salviz EA. The effects of ultrasound-guided adductor canal block vs. femoral nerve block on quadriceps strength and fall risk: a blinded, randomized trial of volunteers. Reg Anesth Pain Med. 2013;38:321–5.
28. Lund J, Jenstrup MT, Jaeger P, Sorensen AM, Dahl JB. Continuous adductor-canal-blockade for adjuvant post-operative analgesia after major knee surgery: preliminary results. Acta Anaesthesiol Scand. 2011;55(1):14–9.
29. Jaeger P, Zaric D, Fomsgaard JS, Hilsted KL, Bjerregaard J, Gyrn J, Mathiesen O, Larsen TK, Dahl JB. Adductor canal block vs. femoral nerve block for analgesia after total knee arthroplasty: a randomized, double-blind study. Reg Anesth Pain Med. 2013;38:526–32.
30. Hanson NA, Allen CJ, Hostetter LS, Nagy R, Derby RE, Slee AE, Arslan A, Auyong DB. Continuous ultrasound-guided adductor canal block for total knee arthroplasty: a randomized, double-blind trial. Anesth Analg. 2014;118(6):1370–7.
31. Mudumbai SC, Kim TE, Howard SK, Workman JJ, Giori N, Woolson S, Ganaway T, King R, Mariano ER. Continuous adductor canal blocks are superior to continuous femoral nerve blocks in promoting early ambulation after TKA. Clin Orthop Relat Res. 2014;472:1377–83.
32. Charous MT, Madison SJ, Suresh PH, Sandhu NS, Loland VJ, Mariano ER, Donohue MC, Dutton PH, Ferguson EJ, Ilfeld

BM. Continuous femoral nerve blocks: varying local anesthetic delivery method (bolus vs. basal) to minimize quadriceps motor block while maintaining sensory block. Anesthesiology. 2011;115:774–81.
33. Ilfeld BM, Duke KB, Donohue MC. The association between lower extremity continuous peripheral nerve blocks and patient falls after knee and hip arthroplasty. Anesth Analg. 2010;111:2552–4.
34. Memtsoudis SG, Dy CJ, Ma Y, Chiu YL, Della Valle AG, Mazumdar M. In-hospital patient falls after total joint arthroplasty: incidence, demographics, and risk factors in the United States. J Arthroplasty. 2012;27:823–8.
35. Espelund M, Grevstad U, Jaeger P, Holmich P, Kjeldsen L, Mathiesen O, Dahl JB. Adductor canal blockade for moderate to severe pain after arthroscopic knee surgery: a randomized controlled trial. Acta Anaesthesiol Scand. 2014;58:1220–7.
36. Rasmussen M, Kim E, Kim TE, Howard SK, Mudumbai S, Giori NJ, Woolson S, Ganaway T, Mariano ER. A retrospective comparative provider workload analysis for femoral nerve and adductor canal catheters following knee arthroplasty. J Anesth. 2014. doi:10.1007/s00540-014-1910-y.
37. Mariano ER, Perlas A. Adductor canal block for total knee arthroplasty: the perfect recipe or just one ingredient? Anesthesiology. 2014;120(3):530–2.
38. Andersen HL, Gyrn J, Moller L, Christensen B, Zaric D. Continuous saphenous nerve block as supplement to single-dose local infiltration analgesia for postoperative pain management after total knee arthroplasty. Reg Anesth Pain Med. 2013;38:106–11.

Chapter 64

Lateral Femoral Cutaneous Nerve Block

Paul Tumber
Department of Anesthesia, Toronto Western Hospital, University of Toronto, Toronto, ON, Canada

Philip W.H. Peng, MBBS, FRCPC (✉)
Department of Anesthesia, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_64,

The lateral femoral cutaneous nerve (LFCN) originates from the dorsal divisions of the second and third lumbar nerve roots (Fig. 64.1). It provides sensory innervation to the anterior and lateral aspects of the thigh to the level of the knee (Fig. 64.2). Neural blockade of LFCN may be useful for surgical procedures involving the thigh such as skin grafting, but more commonly a LFCN block is used in the diagnosis and treatment of meralgia paresthetica [1]. There is a great deal of variability in the appearance and course of the LFCN, and thus ultrasonography is helpful in the performance of neural blockade [2].

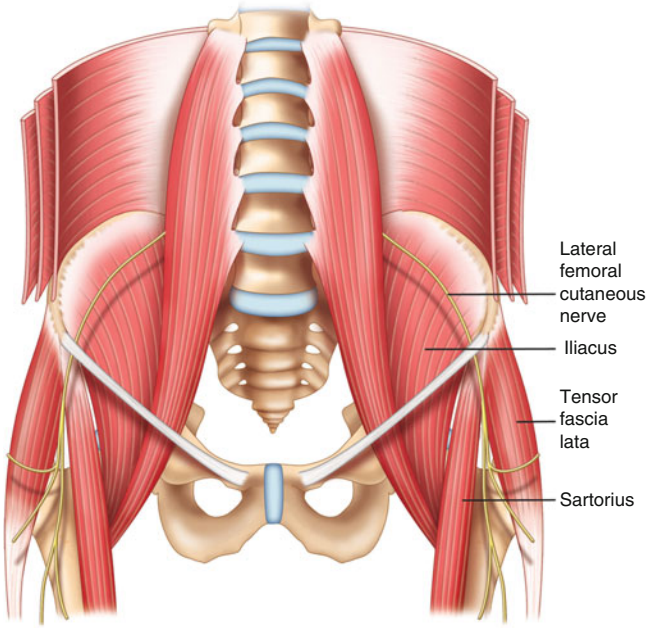


Fig. 64.1 Schematic diagram showing the pathway of a typical course of lateral femoral cutaneous nerve. Note that the nerve courses beneath the inguinal ligament and runs superficially to the sartorius muscle and then in between this muscle and tensor fascia lata muscle (Reproduced with permission from Philip Peng Educational Series)

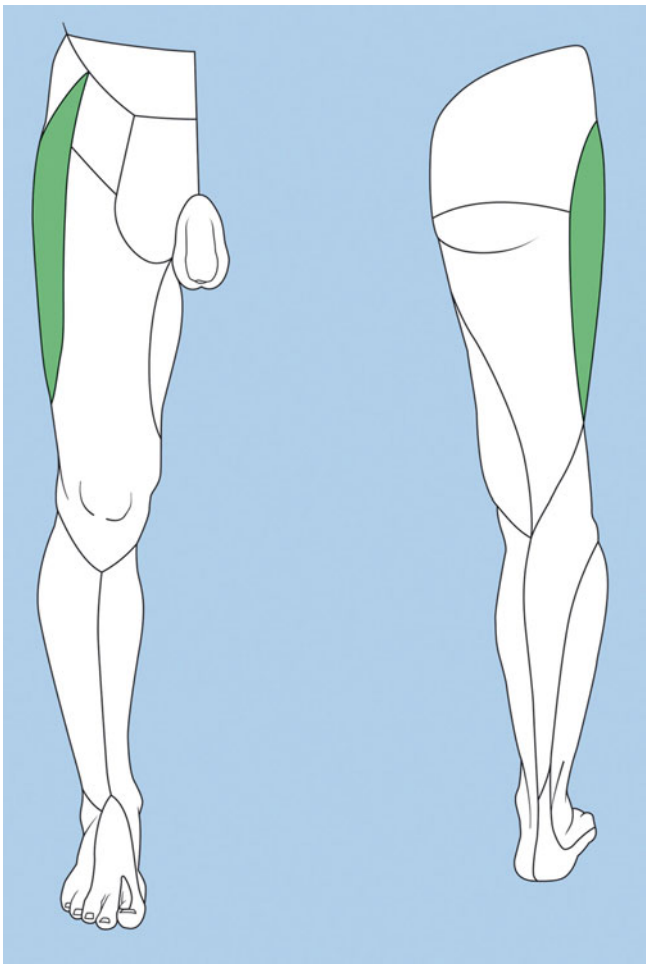


Fig. 64.2 Dermatome of lateral femoral cutaneous nerve (Reproduced with permission from Danilo Jankovic)

Anatomy

After forming near the lumbar spine, the LFCN emerges from the lateral border of the psoas major muscle and crosses the iliacus muscle obliquely under the fascia iliaca (Fig. 64.3). Before it reaches the level of the anterior superior iliac spine (ASIS), it crosses under the deep circumflex artery [3, 4].

The LFCN usually emerges from under the inguinal ligament (IL) medial to the ASIS at a variable distance (average 29 mm; range 0–73 mm) [3, 5–7]. In patients with meralgia paresthetica, however, a recent study using high-resolution ultrasonography revealed the nerve to be closer than normal patients to the ASIS, with a mean distance of 5.2 mm (SD 4.6 mm) [6]. A few anatomical variations that one should bear in mind in scanning the LFCN: it may course over or through the IL rather than under it, it may branch before crossing the IL in up to 28 % of cases, and it may pass over or posterior the ASIS in 4–29 % of cadavers [8, 9]. The average diameter of LFCN was found to be 3.2±0.7 mm [4] although there is no established normal value for cross-sectional area.

Once the LFCN passes under the IL, it usually enters the thigh superficial to the sartorius muscle confined by the fascia lata, although in 22 % of cases the LFCN may pass through the sartorius muscle [4] or at times medial to it [8]. Thereafter the LFCN is visualized in a plane between the tensor fascia lata muscle and the sartorius muscle. It will normally divide into an anterior and a posterior branch and will pierce the fascia lata in order to innervate the skin.

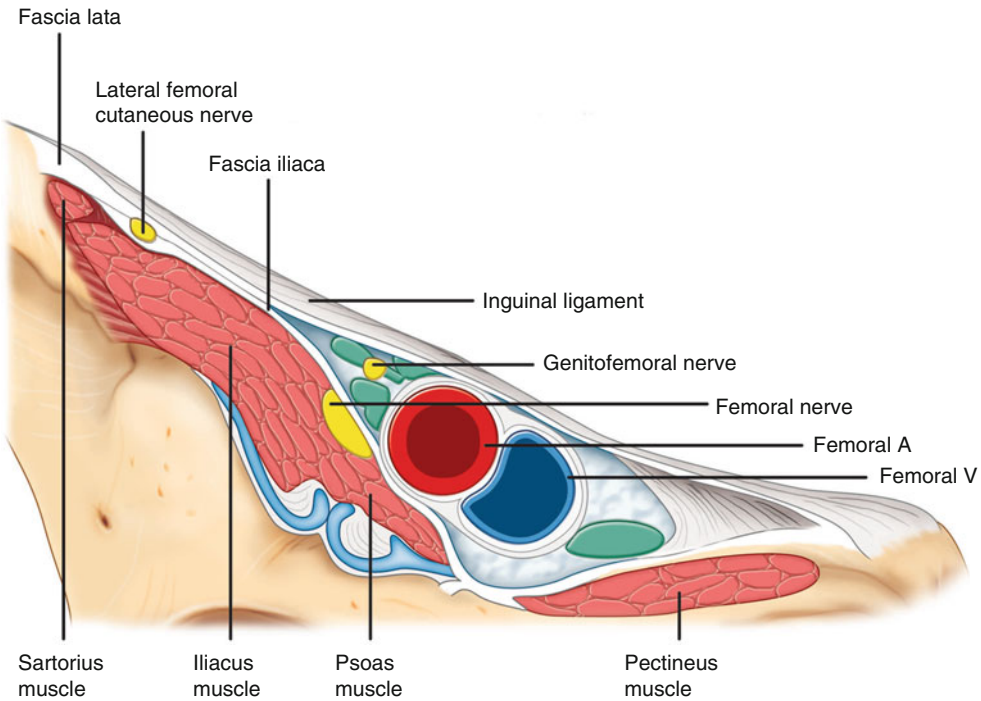


Fig. 64.3 Nerves at the inguinal area (Reproduced with permission from Philip Peng Educational Series)

Sonoanatomy

The LFCN can be difficult to visualize with ultrasound since it is a small nerve, is often surrounded by connective tissue, and has a variable anatomic course. It may appear round or oval shaped and may be hyperechoic or hypoechoic or have a mixed pattern (Fig. 64.4). In many cases of meralgia paresthetica, a portion of the nerve will appear enlarged or swollen (pseudoneuroma) although this can be an incidental finding in the occasional normal patient (Fig. 64.5) [6]. In order to locate the LFCN accurately, high-resolution ultrasonography (HRUS) with 18 MHz high-frequency probes has been utilized [6, 7]. The LFCN is best visualized inferior to the ASIS in a connective tissue plane between the sartorius and tensor fascia lata. However, in order to achieve therapeutic relief of meralgia paresthetica, it is important to block the LFCN as close to the IL as possible, which is often considered the source of irritation or compression of the nerve. Thus it is important to dynamically scan a wide area when performing a LFCN block.

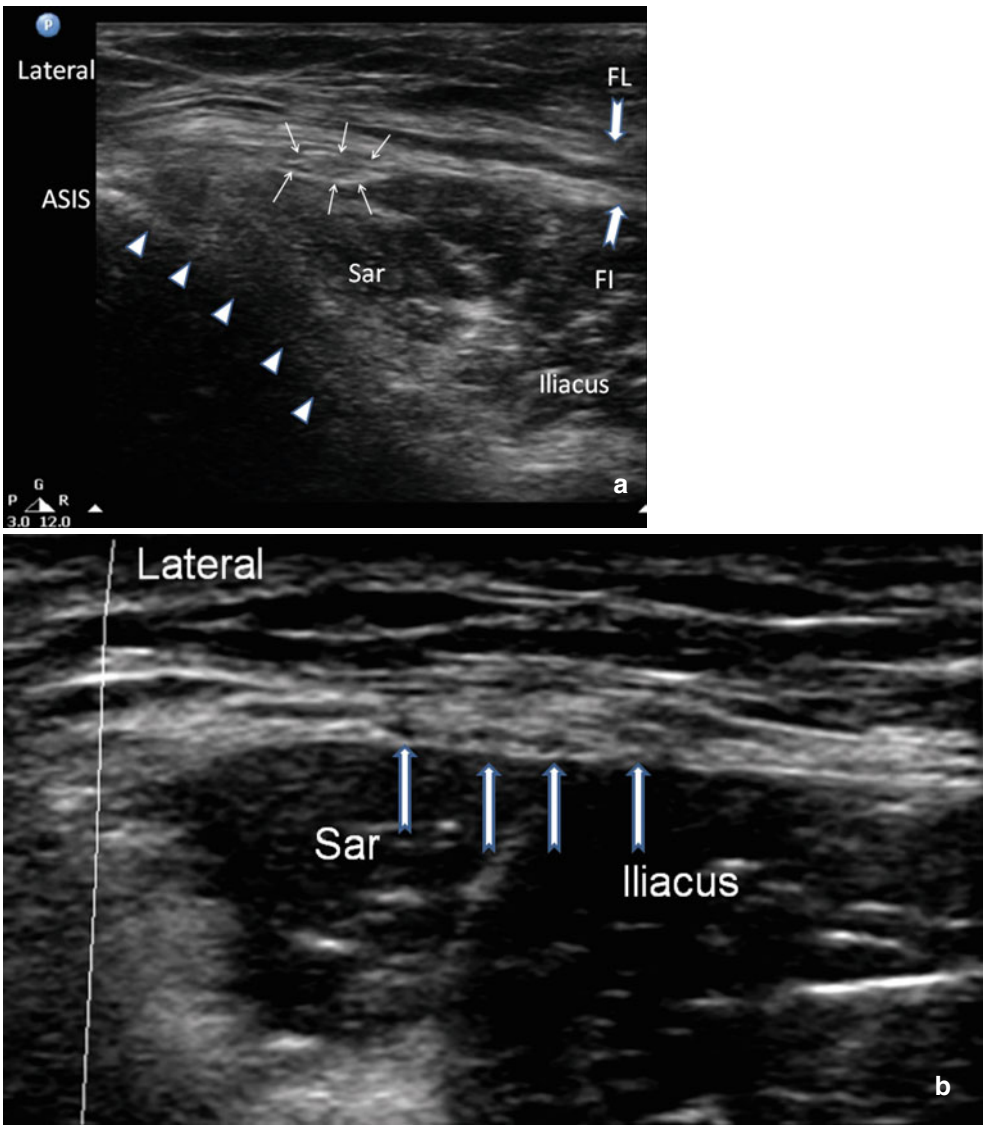


Fig. 64.4 Ultrasonographic picture showing different morphology of the lateral femoral cutaneous nerve (LFCN). It can be in form of hyperechoic or honeycomb appearance as indicated by *line arrows* in figure (**a**). The fascia is indicated by *bold arrows* (*FL* fascia lata, *FI* fascia iliaca); the ilium is indicated by *solid arrowheads*; *SAR* sartorius muscle; *ASIS* anterior superior iliac spine. The LFCN may branch into smaller nerves and appears as hypoechoic structures (*solid line arrows*) in figure (**b**) (Reproduced with permission from Philip Peng Educational Series)

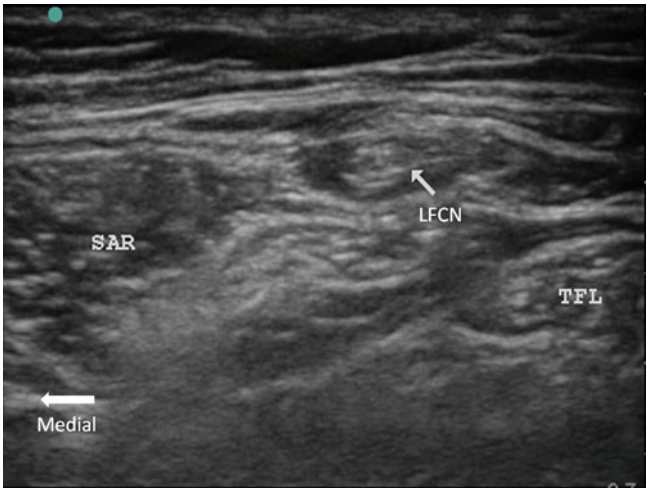


Fig. 64.5 The ultrasound appearance of the LFCN in a patient with meralgia paresthetica. The nerve appears large and swollen, embedded in the adipose tissue in the grove formed by the tensor fascia lata (TFL) and sartorius (SAR) (Reproduced with permission from Philip Peng Educational Series)

Ultrasound-Guided Block Technique

The patient is placed supine and the ASIS and the inguinal ligament are palpated. A high-frequency linear array (6–13 MHz) ultrasound probe is transversely placed with the lateral end of the probe revealing the bony shadow of the ASIS. It is oriented with the medial end slightly angled inferiorly, thus displaying the long axis of the IL. The probe is then moved caudally and the sartorius muscle will be seen as a small triangular-shaped muscle that enlarges as the scan proceeds away from the ASIS.

At some point along this dynamic scan, the small LFCN may be seen in a short-axis view, deep to the fascia lata and superficial to the sartorius muscle. If it is difficult to visualize the nerve near the ASIS, one can look for the LFCN superficially in the connective tissue and adipose-filled plane formed by the fascia lata between the tensor fascia lata muscle and the sartorius. The LFCN can then be traced proximally back to the ASIS–IL region. To assist ultrasonographic confirmation of the nerve, it is helpful to hydrodissect the connective tissue plane surrounding the structure with small 0.5 cc aliquots of normal saline. If electrical stimulation of the LFCN is desired to confirm sensory paresthesia prior to injection [10, 11], then a 5 % dextrose solution is used instead of normal saline. To facilitate the nerve location before the scanning, a transdermal nerve stimulator may be used [12].

Once the LFCN has been identified in the short axis near the ASIS, a needle can be advanced in-plane with the ultrasound probe (Fig. 64.6). Alternatively, the needle may be placed in an out-of-plane approach from caudal to cephalad. The depth to the target can be measured and a 25-G 1.5-in. needle is often sufficient. Prior to any injection, color Doppler imaging is utilized to insure blood vessels are avoided. For a LFCN block, 3–5 cc of injectate is satisfactory. For the treatment of meralgia paresthetica, 40 mg of methylprednisolone is added [13, 2]. The injectate can easily be visualized on real-time ultrasound scanning, and this can help one determine how much volume to use to accomplish circumferential and proximal spread toward the desired ASIS–IL location.

A similar ultrasonographic technique to localize the LFCN has been used successfully in the treatment of meralgia paresthetica with pulsed radiofrequency [14, 15].

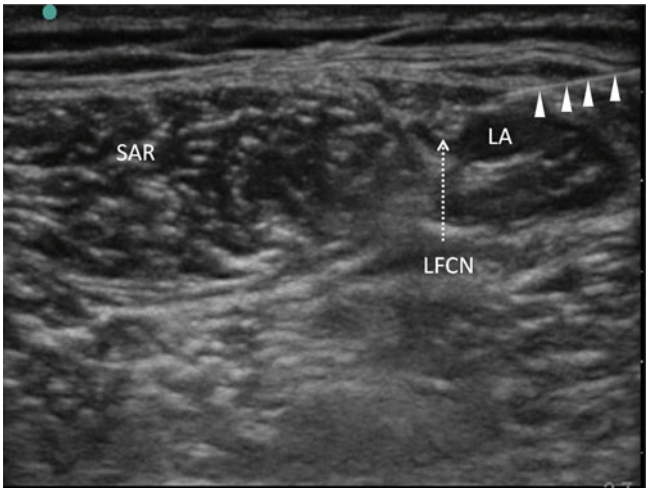


Fig. 64.6 Post-injection ultrasonographic picture of LFCN; the needle was inserted in-plane and was indicated by *arrowheads*; *LA* local anesthetic, *SAR* sartorius muscle (Reproduced with permission from Philip Peng Educational Series

References

1. Hui GK, Peng PW. Meralgia paresthetica: what an anesthesiologist needs to know. Reg Anesth Pain Med. 2011;36(2):156–61.
2. Tagliafico A, Serafini G, Lacelli F, Perrone N, Valsania V, Martinoli C. Ultrasound-guided treatment of meralgia paresthetica (lateral femoral cutaneous neuropathy): technical description and results of treatment in 20 consecutive patients. J Ultrasound Med. 2011;30(10):1341–6.
3. Damarey B, Demondion X, Boutry N, Kim HJ, Wavreille G, Cotten A. Sonographic assessment of the lateral femoral cutaneous nerve. J Clin Ultrasound. 2009;37(2):89–95.
4. Dias Filho LC, Valença MM, Guimarães Filho FA, et al. Lateral femoral cutaneous neuralgia: an anatomical insight. Clin Anat. 2003;16(4):309–16.
5. Bodner G, Bernathova M, Galiano K, Putz D, Martinoli C, Felfernig M. Ultrasound of the lateral femoral cutaneous nerve: normal findings in a cadaver and in volunteers. Reg Anesth Pain Med. 2009;34(3):265–8.
6. Moritz T, Prosch H, Berzaczy D, et al. Common anatomical variation in patients with idiopathic meralgia paresthetica: a high resolution ultrasound case-control study. Pain Physician. 2013;16(3):E287–93.
7. Zhu J, Zhao Y, Liu F, Huang Y, Shao J, Hu B. Ultrasound of the lateral femoral cutaneous nerve in asymptomatic adults. BMC Musculoskelet Disord. 2012;13:227.
8. Aszmann OC, Dellon ES, Dellon AL. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. Plast Reconstr Surg. 1997;100(3):600–4.
9. Murata Y, Takahashi K, Yamagata M, Shimada Y, Moriya H. The anatomy of the lateral femoral cutaneous nerve, with special reference to the harvesting of iliac bone graft. J Bone Joint Surg Am. 2000;82(5):746–7.
10. Shannon J, Lang SA, Yip RW, Gerard M. Lateral femoral cutaneous nerve block revisited. A nerve stimulator technique. Reg Anesth. 1995;20(2):100–4.
11. Corujo A, Franco CD, Williams JM. The sensory territory of the lateral cutaneous nerve of the thigh as determined by anatomic dissections and ultrasound-guided blocks. Reg Anesth Pain Med. 2012;37(5):561–4.
12. Ng I, Vaghadia H, Choi PT, Helmy N. Ultrasound imaging accurately identifies the lateral femoral cutaneous nerve. Anesth Analg. 2008;107(3):1070–4.
13. Tumber PS, Bhatia A, Chan VW. Ultrasound-guided lateral femoral cutaneous nerve block for meralgia paresthetica. Anesth Analg. 2008;106(3):1021–2.
14. Patijn J, Mekhail N, Hayek S, Lataster A, van Kleef M, Van Zundert J. Meralgia paresthetica. Pain Pract. 2011;11(3):302–8.
15. Fowler IM, Tucker AA, Mendez RJ. Treatment of meralgia paresthetica with ultrasound-guided pulsed radiofrequency ablation of the lateral femoral cutaneous nerve. Pain Pract. 2012;12(5):394–8.

Chapter 65

Obturator Nerve

Jens Kessler, MD
Department of Anaesthesiology, University Hospital Heidelberg, Heidelberg, Germany
e-mail: jens.kessler@med.uni-heidelberg.de

Content

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_65,

The obturator nerve originates from the anterior divisions of the L2–4 ventral rami and innervates most of the adductor muscles. Furthermore it sends articular branches to the knee joint and innervates a small dermatome on the medial side of the knee with a high variability [1]. When the obturator nerve enters the medial aspect of the thigh, it divides into very thin and flat divisions which are named after their anatomical relation to the adductor brevis muscle: the anterior division of the obturator muscle runs in between the adductor longus and brevis muscle, and the posterior division runs in between the adductor brevis and magnus muscle.

The sonographic imaging of these small branches is only possible because of the difference in echogenicity between the hypoechoic muscles and the hyperechoic, bright white appearance of the nervous structures [6] (Fig. 65.1).

Indications for an obturator nerve block are the suppression of the adductor reflex for transurethral lateral bladder wall resections [4], adjunct to femoral nerve blocks for postoperative pain management of the medial knee joint [7] and the treatment of adductor spasm.

The block is performed in supine position with slight abduction of the hip and external rotation of the thigh. After skin disinfection and appropriate covering of the transducer (Fig. 65.2) to maintain sterility, the transducer is placed in the medial thigh to visualize the obturator divisions and the adductor brevis muscle in a short-axis view. It is also possible to start with the femoral nerve, artery, and—by compression—vein, moving medially and slightly caudal to the pectineus and the adductor muscles.

Color Doppler can help detecting the adjacent obturator arteries to avoid hemorrhage due to unintentional vessel puncture. Both divisions should be blocked separately, beginning with the posterior division. In case of inadequate visualization of both divisions, the local anesthetic can be injected between the three adductor muscles [3, 5].

A linear transducer with a higher frequency would be appropriate for this kind of nerve block. Both out-of-plane [2] and in-plane approaches are possible to inject the local anesthetic around the divisions by using only one needle insertion point.

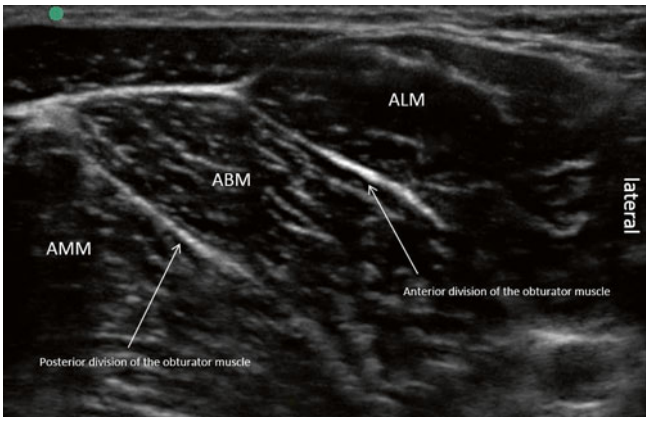


Fig. 65.1 Anterior and posterior divisions of the obturator nerve between the adductor muscles (*ALM* adductor longus muscle, *ABM* adductor brevis muscle, *AMM* adductor magnus muscle) (With permission from Jens Kessler)

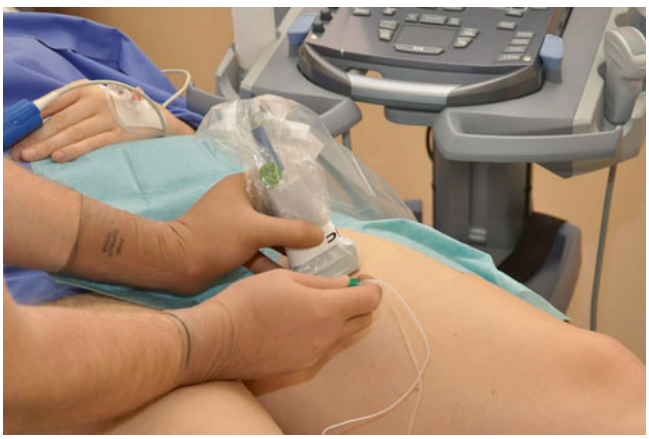


Fig. 65.2 Out-of-plane approach to obturator nerve block in the medial thigh (With permission from Jens Kessler)

References

1. Bouaziz H, Vial F, Jochum D, Macalou D, Heck M, Meuret P, Braun M, Laxenaire MC. An evaluation of the cutaneous distribution after obturator nerve block. Anesth Analg. 2002;94(2):445–9, table of contents.
2. Ishiyama T, Kotoda M, Asano N, Ikemoto K, Masamune T, Matsukawa T. Ultrasound-guided out-of-plane obturator nerve block. Anaesthesia. 2013;68(10):1074–5. doi:10.1111/anae.12432.
3. Lee SH, Jeong CW, Lee HJ, Yoon MH, Kim WM. Ultrasound guided obturator nerve block: a single interfascial injection technique. J Anesth. 2011;25(6):923–6. doi:10.1007/s00540-011-1228-y.
4. Manassero A, Bossolasco M, Ugues S, Palmisano S, De Bonis U, Coletta G. Ultrasound-guided obturator nerve block: interfascial injection versus a neurostimulation-assisted technique. Reg Anesth Pain Med. 2012;37(1):67–71. doi:10.1097/AAP.0b013e31823e77d5.
5. Sinha SK, Abrams JH, Houle TT, Weller RS. Ultrasound-guided obturator nerve block: an interfascial injection approach without nerve stimulation. Reg Anesth Pain Med. 2009;34(3):261–4. doi:10.1097/AAP.0b013e3181a32c4d.
6. Soong J, Schafhalter-Zoppoth I, Gray AT. Sonographic imaging of the obturator nerve for regional block. Reg Anesth Pain Med. 2007;32(2):146–51. doi:10.1016/j.rapm.2006.10.012.
7. Westergaard B, Jensen K, Lenz K, Bendtsen TF, Vazin M, Tanggaard K, Worm BS, Krogsgaard M, Borglum J. A randomised controlled trial of ultrasound-guided blockade of the saphenous nerve and the posterior branch of the obturator nerve for postoperative analgesia after day-case knee arthroscopy. Anaesthesia. 2014;69(12):1337–44. doi:10.1111/anae.12794.

Chapter 66

Peripheral Nerve Block in the Ankle Joint Region

Philip W.H. Peng , MBBS, FRCPC
Department of Anesthesia, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_66, © Springer International Publishing Switzerland 2015

This refers to the blockade of the following nerves: tibial nerve, sural nerve, saphenous nerve, and superficial and deep peroneal (fibular) nerves.

Anatomy

Tibial Nerve (Fig. 66.1a, b)

The tibial nerve in the ankle region is the direct continuation of the tibial nerve distal to the bifurcation of the sciatic nerve in the popliteal fossa. The nerve descends through the calf sandwiched between gastrocnemius/soleus and the deep flexors. It reaches the distal lower leg posterior to the medial malleolus running deep to the flexor retinaculum and is usually posterior to the artery. It gives off medial calcaneal branches to the heel and divides into its two end branches, the medial and lateral plantar nerves, which pass to the sole of the foot and provide it with its sensory supply.

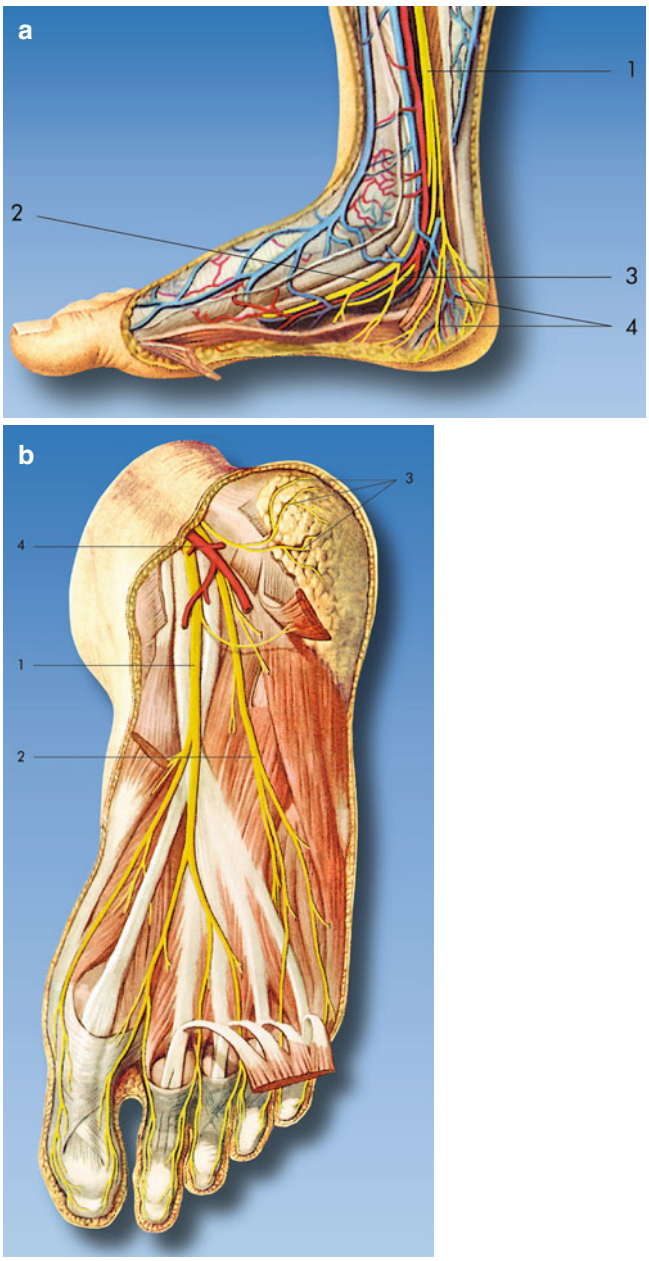


Fig. 66.1 (**a**) Tibial nerve. (*1*) Tibial nerve, (*2*) medial plantar nerve, (*3*) lateral plantar nerve, (*4*) calcaneal branches (With permission from Danilo Jankovic). (**b**) Tibial nerve – sole of the foot. (*1*) Medial plantar nerve, (*2*) lateral plantar nerve, (*3*) medial calcaneal branches, (*4*) posterior tibial artery (With permission from Danilo Jankovic)

Superficial Peroneal (Fibular) Nerve
(Figs. 66.2 and 66.3)

This nerve arises from the common peroneal (fibular) nerve, runs through the peroneus longus muscle, and extends between the peroneus longus and brevis muscles. In the distal half of the leg, the nerve is sandwiched between peroneus brevis and extensor digitorum longus in the intermuscular septum that separates the anterior and lateral compartments of the leg and gradually ascends into a superficial location before eventually piercing the crural fascia. Subcutaneously, or still at the subfascial level, it divides into the thicker medial dorsal cutaneous nerve and the smaller intermediate dorsal cutaneous nerve, providing the sensory supply for the skin on the back of the foot and the toes.

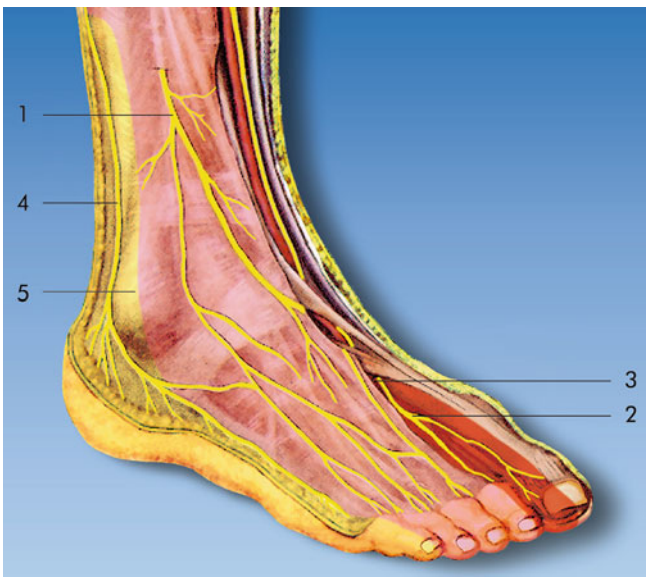


Fig. 66.2 Common, superficial, and deep peroneal (fibular) nerve and sural nerve. Cutaneous innervation areas of the back of the foot (lateral). (*1*) Superficial peroneal (fibular) nerve, (*2*) deep peroneal (fibular) nerve, (*3*) dorsalis pedis artery, (*4*) sural nerve, (*5*) lateral malleolus (With permission from Danilo Jankovic)

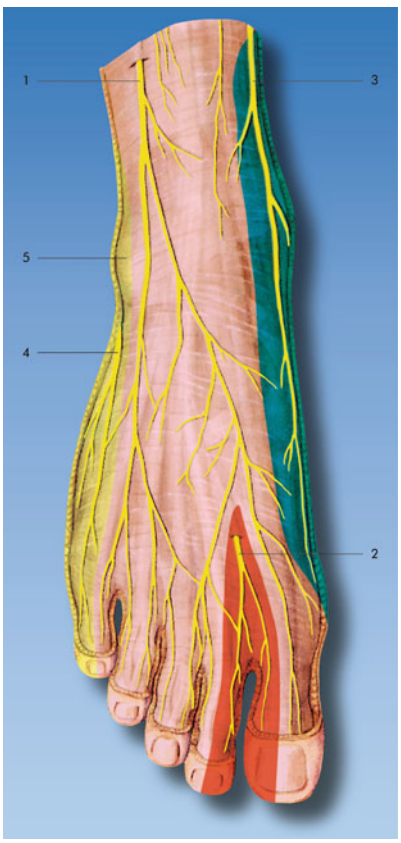


Fig. 66.3 Cutaneous innervation areas in the region of the back of the foot (from the front). (*1*) Superficial peroneal (fibular) nerve, (*2*) deep peroneal (fibular) nerve, (*3*) saphenous nerve, (*4*) lateral dorsal cutaneous nerve (sural nerve), (*5*) lateral malleolus (With permission from Danilo Jankovic)

Deep Peroneal (Fibular) Nerve (Figs. 66.2, 66.3, and 66.4)

This nerve runs between the tibialis anterior muscle and the extensor hallucis longus muscle in the direction of the ankle, where it divides into a medial and a lateral end branch. The medial end branch continues in the same direction as the nerve trunk and passes with the dorsalis pedis artery to the first interosseous space, crossing under the tendon of the extensor hallucis brevis muscle to the distal end of the interosseous space. Here it joins with a strand of the superficial peroneal nerve and divides into the end branches for the facing sides of the dorsal surfaces of the first and second toes. The lateral end branch turns laterally and supplies the extensor digitorum brevis muscle, sending off three interosseous nerves.

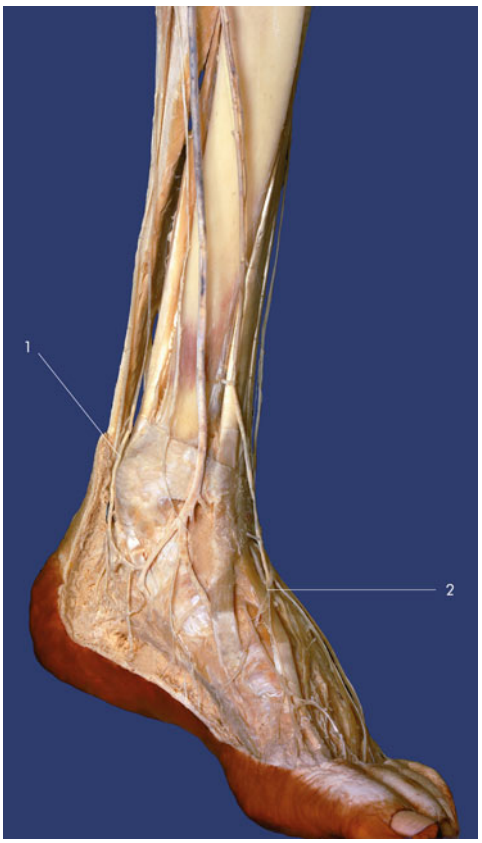


Fig. 66.4 (*1*) Tibial nerve, (*2*) deep peroneal nerve (With permission from Danilo Jankovic)

Sural Nerve (Figs. 66.2 and 66.3)

The medial sural cutaneous nerve arises in the proximal part of the popliteal fossa, runs down between the two heads of the gastrocnemius muscle, and joins the peroneal communicating branch (lateral sural nerve) to form the sural nerve. Accompanied by the small saphenous vein, the sural nerve runs behind the lateral malleolus and is contained within the same superficial fascial sheath. It passes as the lateral dorsal cutaneous nerve along the lateral side of the foot, where it gives off a connecting branch to the intermediate dorsal cutaneous nerve and ends as the dorsalis digiti minimi nerve on the lateral edge of the dorsum of the small toe.

Behind the lateral malleolus, it sends off branches (the lateral calcaneal branches) to the skin there and at the heel. The branches for the lateral side of the ankle, for the anterior capsular wall, and for the tarsal sinus originate proximal to the malleolus.

Saphenous Nerve (Fig. 66.3)

Below the knee, it runs along the tibial surface close to the great saphenous vein. Its relationship to the saphenous vein is inconstant, and the nerve may be either posterior or anterior to the vein. A common fascia has been described that envelops both the vein and the nerve in the distal third of the calf. At the level of the lower leg, the saphenous nerve courses along the medial side of and anterior to the medial malleolus and sends off branches to the skin of the medial side of the foot. It usually ends in the metatarsal area, without reaching the big toe.

Sonoanatomy

As all the five ankle nerves are superficial nerves, linear high-frequency ultrasound probe is used.

Tibial Nerve (Fig. 66.5)

The patient is in the supine position with hip in external rotation, so that the medial side of the ankle is facing upward. The ultrasound probe is placed in a transverse orientation just proximal to the prominence of the medial malleolus. The main landmark is the posterior tibial artery and is usually accompanied by two venae comitantes. The tibial nerve is a round hyperechoic structure usually posterior to the artery and rest on the fascia of flexor hallucis longus. By extending and flexing the big toe, the tibial nerve can be seen moving up and down, a maneuver to help identifying the tibial nerve.

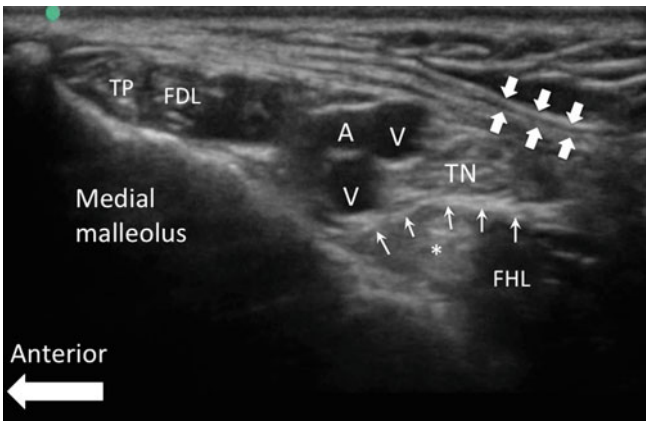


Fig. 66.5 Sonography of the tibial nerve at the level of medial malleolus. The flexor retinaculum is indicated by the *block arrows*. From anterior to posterior, the structures are tibialis posterior tendon (*TP*), flexor digitorum longus tendon and muscle (*FDL*), posterior tibial artery (*A*) and veins (*V*), tibial nerve (*TN*), and flexor hallucis longus (*FHL*). Please note the tibial nerve is usually seen resting on the fascia overlying FHL (*line arrows*). The tendon of the FHL (*) is deep to the fascia and should not be mistaken as the tibial nerve (Reproduced with permission from Philip Peng Educational Series)

Superficial Peroneal (Fibular) Nerve (Fig. 66.6)

With the patient in the supine position, the knee is flexed and the hip rotated inward, to make the lateral aspect of the leg accessible. The probe is placed in a transverse orientation on the lateral aspect of the lower one third of the leg above the subcutaneous part of the fibula. The superficial peroneal nerve is visible as a small triangular hyperechoic structure in the intermuscular septum between the peroneus brevis and extensor digitorum longus muscles just deep to the crural fascia. Its identity can be confirmed by sliding the probe distally and observing the nerve rise through the crural fascia to eventually lie above it in a superficial location.

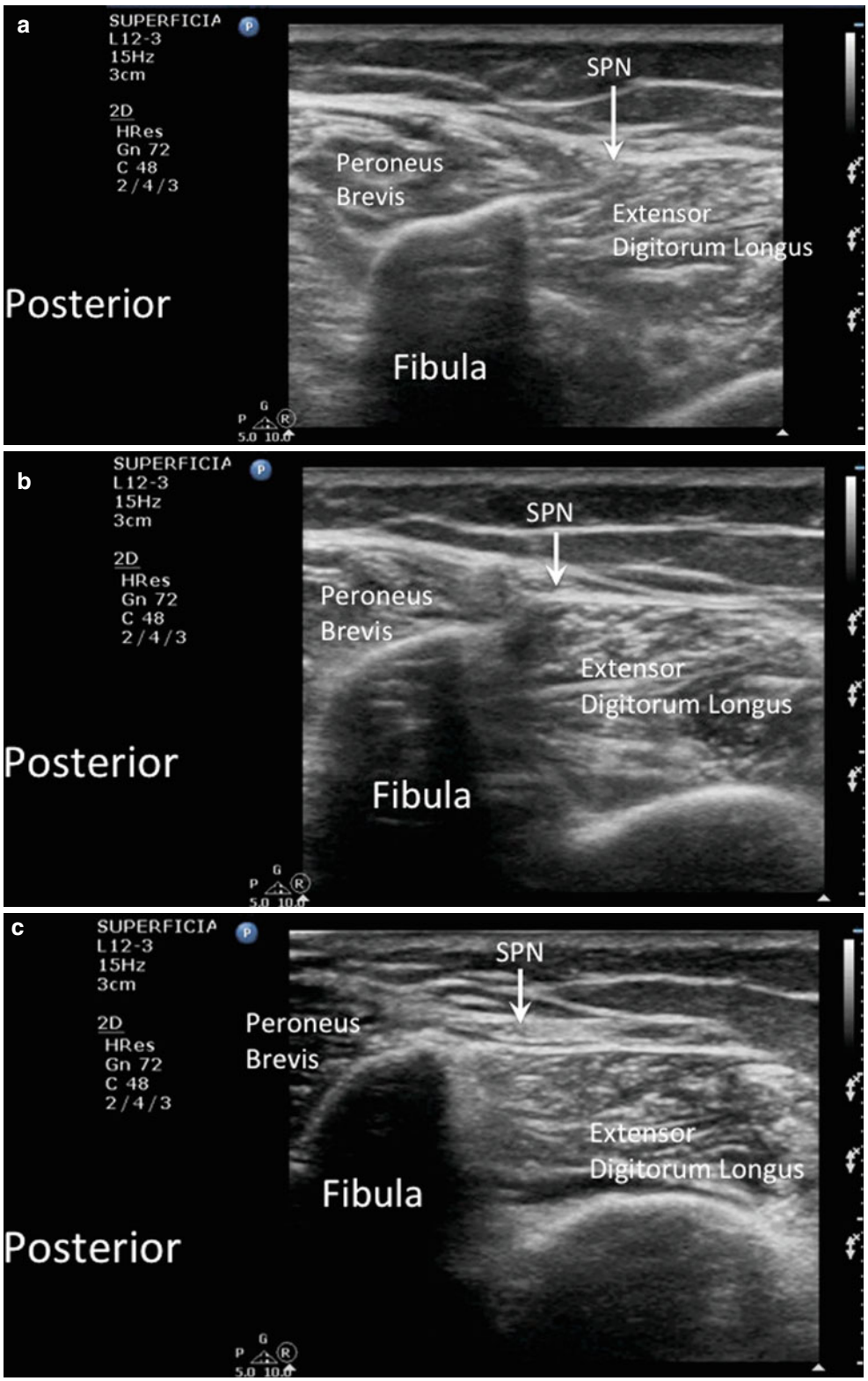


Fig. 66.6 Sonography of the superficial peroneal (fibula) nerve (SPN) at three different levels in the distal third of the leg. (**a**) At the level where the fibula is covered by peroneus brevis. At this level, the SPN is seen in the intermuscular septum between peroneus brevis and extensor digitorum longus deep to the crural fascia. (**b**) Advancing the ultrasound probe in the distal direction reveals the SPN embedded within the crural fascia. (**c**) Moving the probe further distally shows the SPN superficial to the crural fascia (Reproduced with permission from Philip Peng Educational Series)

Deep Peroneal (Fibular) Nerve (Fig. 66.7)

The patient is placed in supine position with the leg in a neutral position. Place the probe in a transverse orientation on the anterior surface of the ankle at the intermalleolar line (that is proximal to the ankle joint). The landmark is the anterior tibial/dorsalis pedis artery, which is usually deep to the extensor hallucis longus. The deep peroneal nerve is sometimes, but not always, visible as a small hyperechoic structure lateral to the artery in the same tissue plane. It is not essential to visualize the nerve to perform this block; the main reason to visualize it is so as to avoid inadvertently piercing it with the block needle.

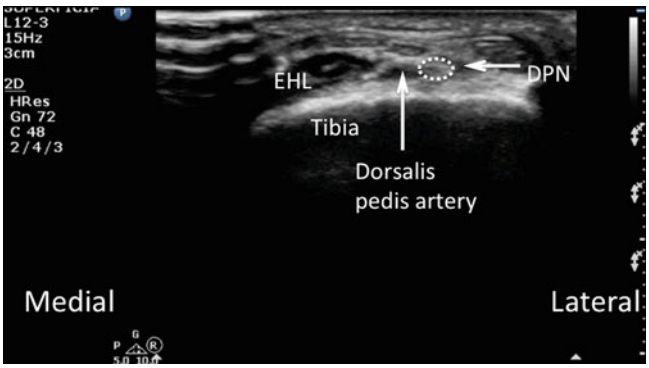


Fig. 66.7 Sonography of deep peroneal (fibula) nerve at the low tibial nerve. The nerve is seen lateral to the dorsalis pedis artery which is deep to the extensor hallucis longus (*EHL*) (Reproduced with permission from Philip Peng Educational Series)

Sural Nerve (Fig. 66.8)

The patient is placed in lateral position with the knee flexed. The probe is placed in a transverse orientation across the groove between the lower fibula and Achilles tendon with minimal pressure to avoid compression of the lesser saphenous vein. Both the vein and the nerve are contained with the fascial plane between the fibula and Achilles tendon. The sural nerve is a small hyperechoic round structure lying adjacent to the lesser saphenous vein in the same fascial subcutaneous plane. If the nerve is not visible, local anesthetic may be injected around the vein.

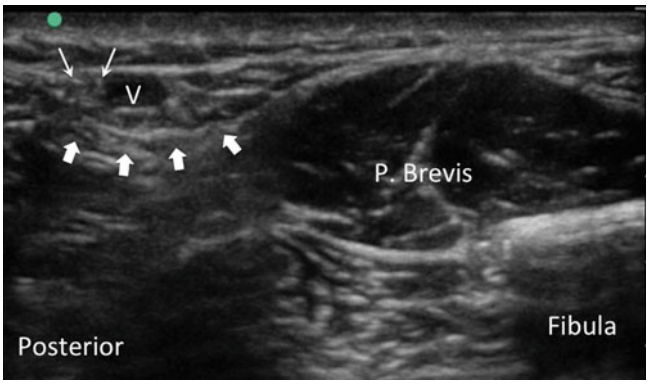


Fig. 66.8 Sonography of the sural nerve (indicated by *line arrows*). It is accompanied by the lesser saphenous vein and both are located within the fascia plane (*block arrows*) posterior to the peroneus brevis muscle (Reproduced with permission from Philip Peng Educational Series)

Saphenous Nerve (Fig. 66.9)

The position is the same as that for tibial nerve scanning. The probe is placed gently just proximal to the medial malleolus in the approximate location of the greater saphenous vein. The vein is the primary landmark. The saphenous nerve, which is a small hyperechoic structure, is not always clearly visible and does not have a constant relationship to the vein. However, the saphenous vein indicates the plane where the saphenous nerve is.

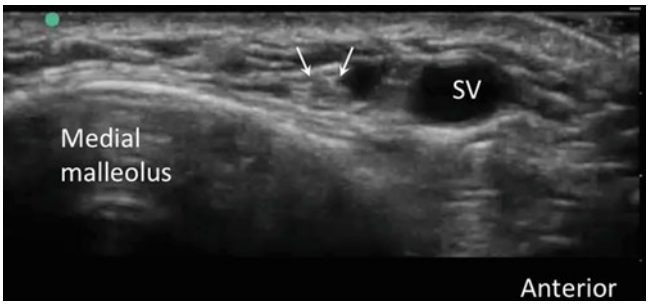


Fig. 66.9 Sonography of the saphenous nerve (*line arrows*). It lies within the same fascia plane with the greater saphenous vein (Reproduced with permission from Philip Peng Educational Series)

Preparation for the Block

Before performing the procedure, full prior information for the patient is mandatory. Since the location of nerves is superficial, a 30-mm 25-G needle should be sufficient. The procedure is performed with sterile technique.

Landmark-Guided Technique

Care must be taken to avoid nerve injury, as paresthesias are not available as a warning sign here.

Tibial Nerve

Patient Positioning

Prone, with a pillow under the ankle (or the patient may be seated)

Landmarks

Medial malleolus, posterior tibial artery

Injection Technique (Fig. 66.4, 66.10, and 66.11)

Lateral to the palpated pulse of the posterior tibial artery, a fine 25-G needle, 30 mm long, is introduced at a right angle to the posterior side of the tibia and just posterior to the posterior tibial artery. After paresthesias are elicited and after a negative aspiration test, 5 mL of local anesthetic is injected. If paresthesias cannot be elicited, then after reaching the posterior tibia, the needle is withdrawn for about 1 cm, and 5–10 mL of local anesthetic is injected.

Another technique is to carry out perpendicular puncture of the skin at the level of the medial malleolus, dorsal and then ventral to the posterior tibial artery, and to distribute the total dose of local anesthetic in two equal halves on each side.

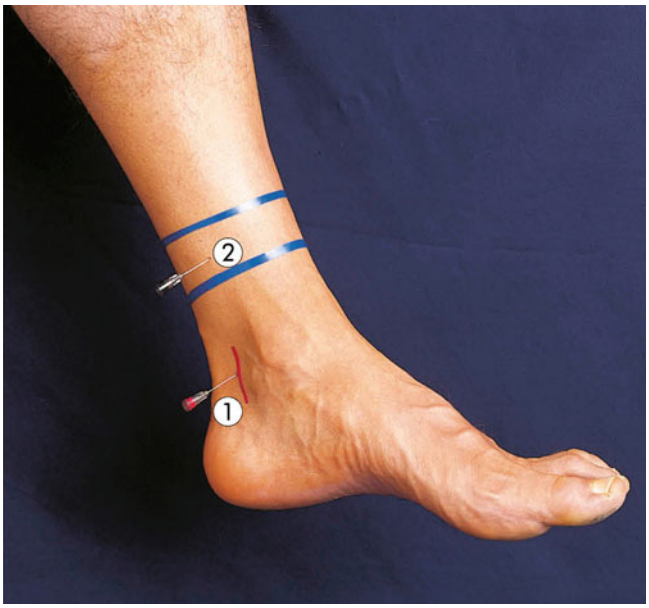


Fig. 66.10 Tibial nerve (*1*) (*red needle*) and posterior tibial artery (*red*), (*2*) saphenous nerve (*black needle*) (With permission from Danilo Jankovic)

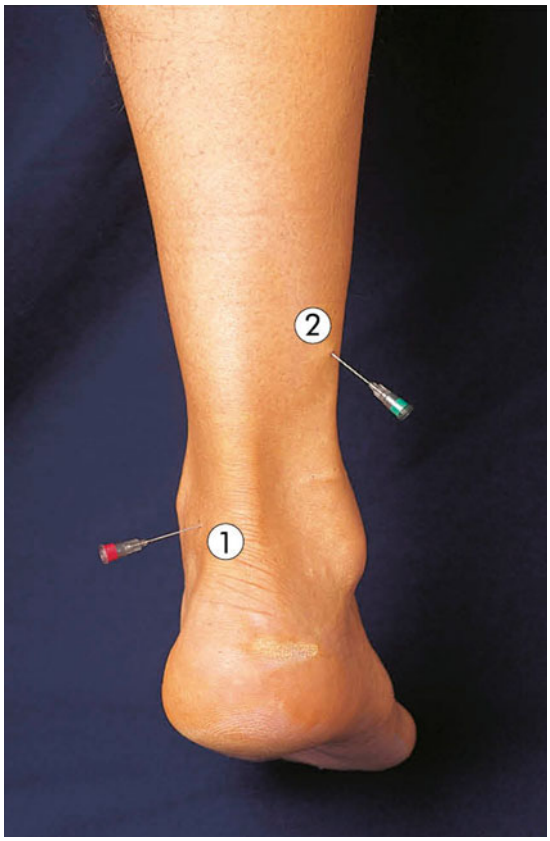


Fig. 66.11 Tibial nerve (*1*) (*red needle*) and (*2*) sural nerve (*green needle*) (With permission from Danilo Jankovic)

Deep Peroneal Nerve

Patient Positioning

Supine or sitting

Landmarks

Dorsalis pedis artery, proximal back of the foot

Injection Technique (Fig. 66.4 and 66.12)

A fine 25-G injection needle, 30 mm long, is introduced perpendicular to the skin surface; 5 mL of the local anesthetic is injected on each side, first lateral to the artery and then medial to it.

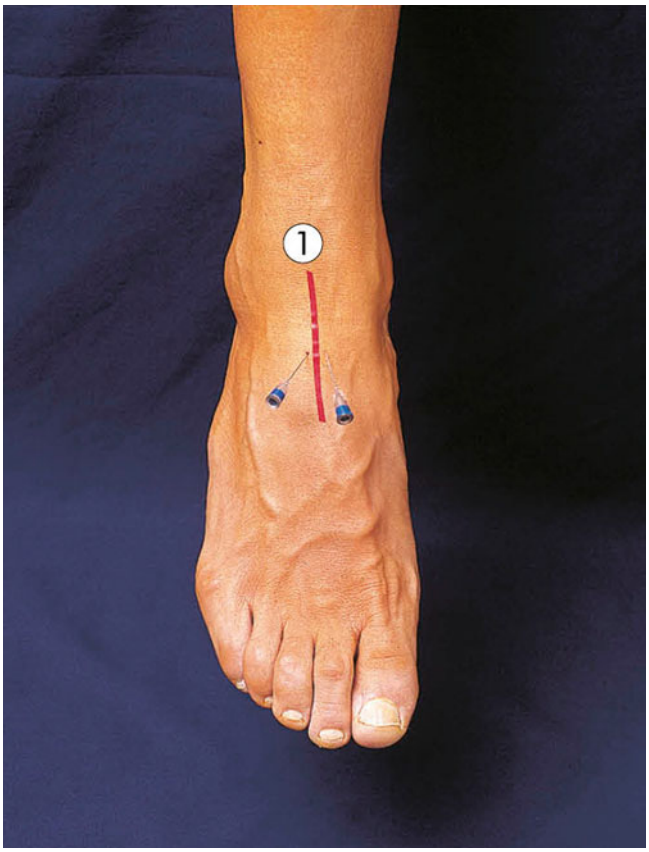


Fig. 66.12 Deep peroneal (fibular) nerve. (*1*) Dorsalis pedis artery (With permission from Danilo Jankovic)

Sural Nerve and Superficial Peroneal Nerve

Patient Positioning

Supine or sitting

Landmarks

Lateral malleolus

Injection Technique (Figs. 66.13 and 66.8)

About 10 cm above the lateral malleolus, parallel to the upper ankle, fan-shaped subcutaneous infiltration of the Achilles tendon is carried out as far as the edge of the tibia, using about 10 mL of local anesthetic.

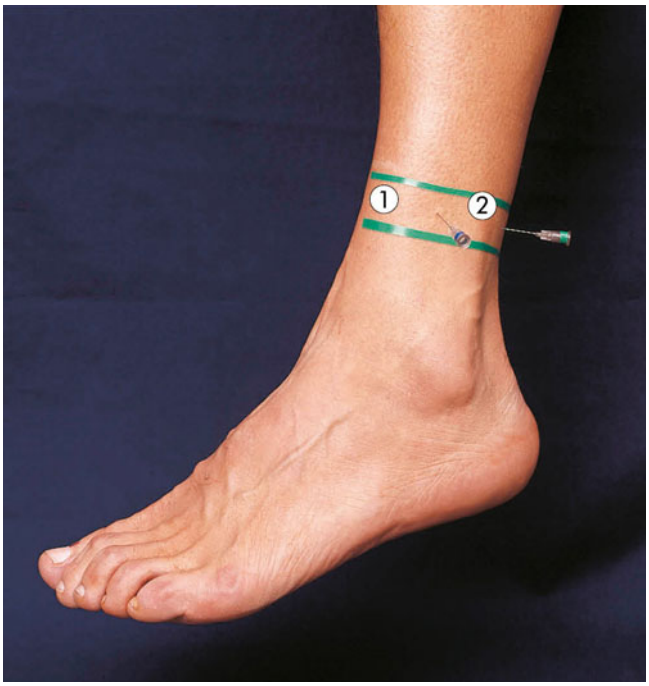


Fig. 66.13 (*1*) Superficial peroneal (fibular) nerve (*blue needle*) and (*2*) sural nerve (*green needle*) (With permission from Danilo Jankovic)

Saphenous Nerve

Patient Positioning

Supine or sitting

Landmarks

Medial malleolus

Injection Technique (Fig. 66.10)

About 10 cm above the medial malleolus, 5–10 mL of local anesthetic is injected subcutaneously around the long saphenous vein and, in a fan-shaped fashion, in a mediolateral direction.

The cutaneous innervation areas of the individual nerves are shown in Fig. 66.14.

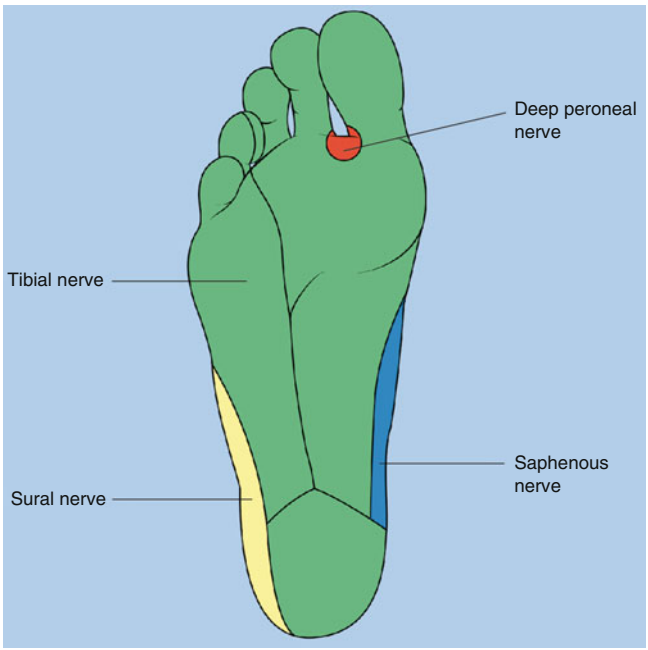


Fig. 66.14 Cutaneous innervation areas in the region of the sole of the foot (With permission from Danilo Jankovic)

Dosage and Local Anesthetic for Landmark Technique

Tibial Nerve and Deep Peroneal Nerve

5–10 mL of local anesthetic

Sural Nerve, Superficial Peroneal Nerve, Saphenous Nerve

10 (15)–20 mL of local anesthetic (subcutaneous fan-shaped infiltration)

Local Anesthetics

0.5–0.75 % ropivacaine, 0.25–0.5 % bupivacaine (0.25–0.5 % levobupivacaine), 1 % prilocaine, and 1 % mepivacaine

Ultrasound-Guided Technique

Tibial Nerve

Both in-plane and out-of-plane needle approach to the nerve are feasible. The nerve lies within a fascial sheath, and injecting the local anesthetic within this sheath will produce a characteristic circumferential spread of local anesthetic around the nerve. Eight to ten milliliter of local anesthetic is sufficient to produce a good block.

Deep Peroneal Nerve

The key landmark is the artery and it is not essential to visualize the nerve to perform this block. An in-plane or out-of-plane needle approach may be used, although out-of-plane allows a more direct needle entry for a superficial nerve. The needle tip should be directed to either side of the artery, and local anesthetic should be injected to distend the plane in which the artery lies. Three mL of local anesthetic is sufficient for a good block.

Superficial Peroneal Nerve

The nerve can be blocked where it lies under the crural fascia using either an in-plane or out-of-plane approach. In-plane approach is preferred as it allows easier entry into the narrow fascial plane. Five milliliter of local anesthetic should be sufficient to establish a good block.

Sural Nerve

If the nerve is not visible, local anesthetic may be injected around the vein. Even if both are not visible, local anesthetic can be injected into the fascial plane between Achilles tendon and peroneus tendon. The nerve may be approached using an in-plane or out-of-plane technique, taking care not to pierce the nerve itself. Five milliliter of local anesthetic is injected in the fascial plane of the nerve.

Saphenous Nerve

The vein is the primary landmark and light pressure from the ultrasound probe should be applied. To enhance the visualization of the vein, a tourniquet around the calf can be used. The saphenous nerve, which is a small hyperechoic structure, is not always clearly visible and does not have a constant relationship to the vein. However, it reflects the plane that the local anesthetic infiltration should be directed. The volume of local anesthetic is 3–5 mL.

Suggested Reading

1. Jankovic D. Blocking peripheral nerves in the ankle joint region. In: Jankovic D, editor. Regional nerve blocks & infiltration therapy. Textbook and color atlas. 3rd ed. Malden: Blackwell-Malden, Massachusetts, Oxford, Carlton, Victoria; 2004. p. 254–8.
2. Husni A, Chin KJ. Ankle. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide. Volume 1. Peripheral structures. Philip Peng educational series. 1st ed. iBook, CA: Apple Inc.; 2013. p. 74–88.
3. Hoerster W. Blockaden peripherer Nerven im Bereich des Kniegelenkes; Blockaden im Bereich des Fußgelenkes; In: Hoerster W. Kreuscher H, Niesel HC, editors. Regionalanaesthesie. Stuttgart: Fischer Verlag; 1981. p. 124–399.
4. Kofoed H. Peripheral nerve blocks at the knee and ankle in operations for common foot disorders. Clin Orthop. 1982;168:97–101.

Part XI

Lower Extremity Musculoskeletal Injection

Chapter 67

Hip: Intra-articular and Trochanteric Bursitis Injections

Philip W.H. Peng , MBBS, FRCPC
Department of Anesthesia, Toronto Western Hospital, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_67, © Springer International Publishing Switzerland 2015

Pain in the hip region can be a manifestation of a broad range of intra-articular or extra-articular pathologies. The two common pain conditions referred to an anesthesiologist for intervention are osteoarthritis of the hip and greater trochanteric pain syndrome (GTPS).

Intra-articular hip injection is mainly considered for pain related to osteoarthritis, rheumatoid arthritis, and acetabular labral tears. Although radiographic evidence of osteoarthritis of the hip is present in about 5 % of the population over 65 years old, not all patients are symptomatic. Patients indicated for intra-articular injection are those with moderate to severe pain and disability, with poor response to conservative management and those nonsurgical candidate either because of age or comorbidity. Injected medications may include corticosteroids, local anesthetics, and viscosupplements.

The accuracies of the landmark-based technique for intra-articular hip injection range from 52 to 80 %. In addition, the risk of piercing the femoral nerve from the "blind" anterior approach has been reported as high as 27 %. In contrast, the accuracy of ultrasound-guided injection has been validated with contrast-enhanced fluoroscopy or computed tomography scan, which showed the accuracy of 97–100 %.

The analgesic effectiveness of intra-articular hip steroid injection has been examined in five randomized controlled trials (RCTs) and all injections were performed under image guidance (fluoroscopy-3; ultrasound-2). Four are positive trials with improvement in pain and functions. Current data from available RCTs and other uncontrolled studies demonstrates strong evidence that steroid injection can provide a short-term reduction in pain.

GTPS affects approximately 18 % of the adults in community settings and is more common in female. In the past, the pain over the trochanteric area was termed as trochanteric bursitis. Current literature suggests that the etiology of pain is similar to rotator cuff disease of the shoulder. It is now believed that tendinopathy of the gluteus medius and/or gluteus minimus plays an important role. Both MRI and ultrasound are very useful for the evaluation of the gluteal tendinopathy, tendon tears, or presence of bursitis. Thus, injection for GTPS is directed to peritendon space of the gluteus medius or minimus tendons.

Literature on the GTPS injection reveals that the injection technique used is predominately landmark-based with needle directed to the "bursa." Multiple case series have been published examining the effect of injection as the primary treatment modality. The quality of the studies is poor and most of the studies did not use the visual analog pain scale (VAS) as outcome measure. Subjective improvement and achieving a return to the patient's baseline activity level ranged from 49 to 100 %.

One randomized trial examined a fluoroscopic-guided against "blind" injection without any placebo or nontreatment control. Both groups were comparable in outcomes (>30 % reduction in VAS score and >50 % with positive global perceived effects) at 3-month assessment. A pragmatic, multicenter, open label, randomized clinical trial evaluated the effect of corticosteroid injections compared with expectant treatment (usual care) in patients with GTPS in a primary care setting. They found a superior outcome in the injection group at 3-month follow-up (number needed to treat = 5). The injection group is superior in both pain scores (rest and with activity) and Western Ontario and McMaster osteoarthritis index (WOMAC) pain and function scores.

Anatomy

The hip is a synovial "ball-and-socket" joint formed by the articulation of the femoral head and the acetabulum. The depth and surface of the acetabular cavity is augmented by the acetabular labrum, a fibrocartilaginous ring attached directly to the rim of the acetabulum (Fig. 67.1). At any position of hip motion, approximately 40 % of the articular surface of the femoral head is covered by the acetabulum. The stability to the hip joint attributes to the strong joint capsule and several powerful para-articular ligaments (Figs. 67.2 and 67.3). The capsule covers the outer surface of the labrum and insert distally to the intertrochanteric region and posterior aspect of the femoral neck (Fig. 67.4). The anterior joint capsule is composed of two layers, anterior and posterior, that are separated by the anterior recess of the joint space (Fig. 67.5). Each layer is lined by only a minute synovial membrane. The anterior layer runs caudally and inserts on the intertrochanteric line where it blends with the periosteum. Many fibers are reflected upward, covering the femoral neck, to form the posterior layer of the joint capsule, which ends at the caudal edge of the articular cartilage of the femoral head.

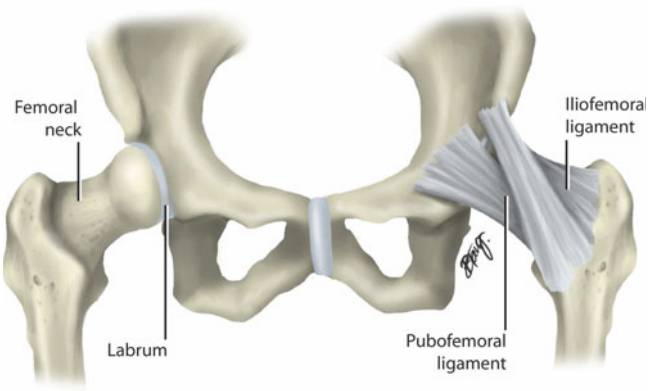


Fig. 67.1 This figure shows the hip joint and the labrum (left) and the ligaments in the anterior hip joint (Reproduced with permission from Philip Peng Educational Series)

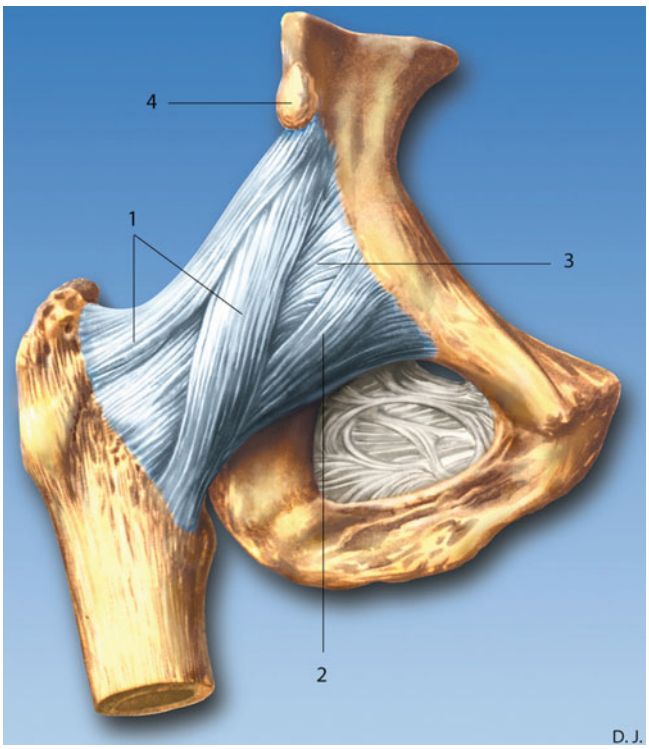


Fig. 67.2 Ligaments of the hip joint (ventral view). (*1*) Iliofemoral ligament, (*2*) pubofemoral ligament, (*3*) articular capsule, (*4*) anterior inferior iliac spine (Reproduced with permission from Danilo Jankovic)

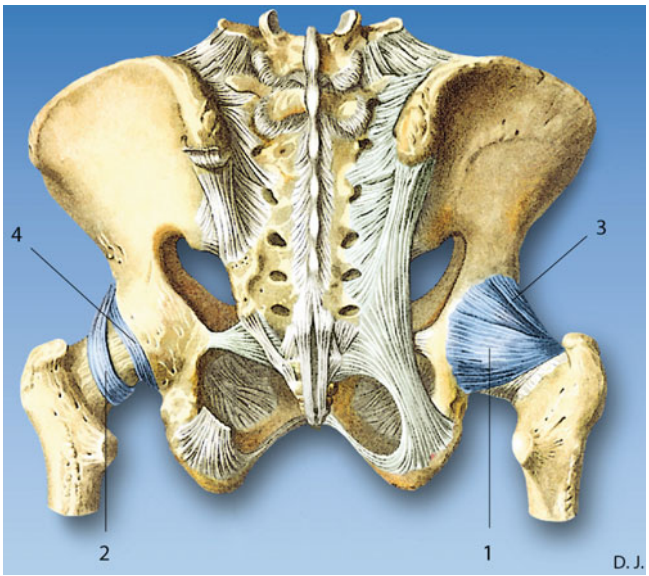


Fig. 67.3 Ligaments of the hip joint (dorsal view). (*1*) Ischiofemoral ligament, (*2*) orbicular zone, (*3*) iliofemoral ligament, (*4*) acetabular labrum (Reproduced with permission from Danilo Jankovic)

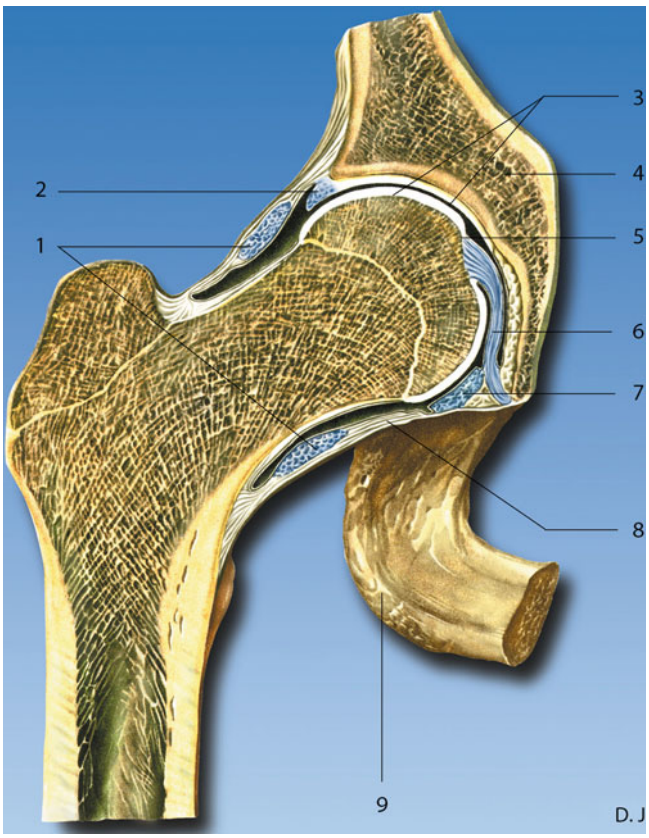


Fig. 67.4 Dissection through the hip joint. (*1*) Orbicular zone, (*2*) acetabular labrum, (*3*) articular cartilage, (*4*) os coxae, (*5*) cavum articulare, (*6*) ligament of head of femur, (*7*) transverse acetabular ligament, (*8*) joint capsule, (*9*) tuber ischiadicum (Reproduced with permission from Danilo Jankovic)

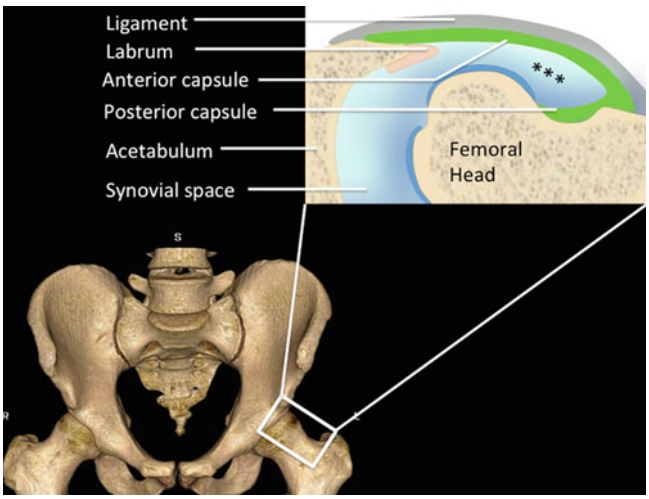


Fig. 67.5 Figure shows the anterior synovial recess (***). Under normal circumstances, the amount of synovial fluid in the recess is kept at a minimum. This figure shows a hip with effusion for demonstration (Reproduced with permission from Philip Peng Educational Series)

The capsule is reinforced by the extracapsular hip ligaments: iliofemoral, ischiofemoral, and pubofemoral ligaments (Figs. 67.2 and 67.3). In the anterior hip regions, the structures from medial to lateral are pectineus muscle, femoral neurovascular bundle, iliopsoas muscle and tendon, rectus femoris, and sartorius muscle (Figs. 67.6, 67.7, 67.8, and 67.9).

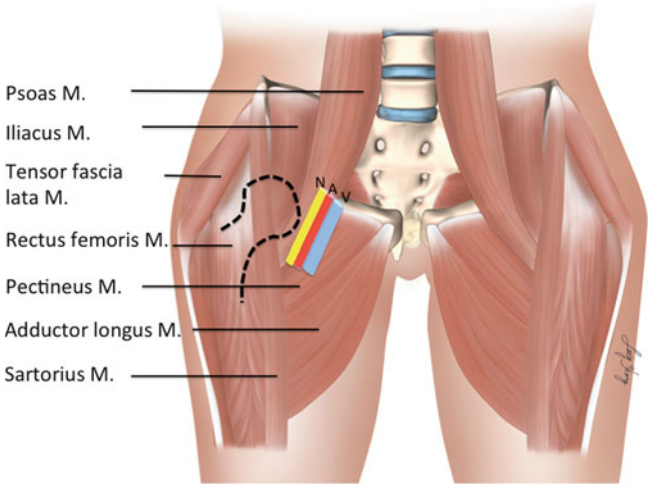


Fig. 67.6 Muscles (*M*) around hip joint. The femoral head and neck (in *dotted line*) and the schematic of femoral neurovascular bundle are shown here for reference. *V* femoral vein, *A* femoral artery, *N* femoral nerve (Reproduced with permission from Philip Peng Educational Series)

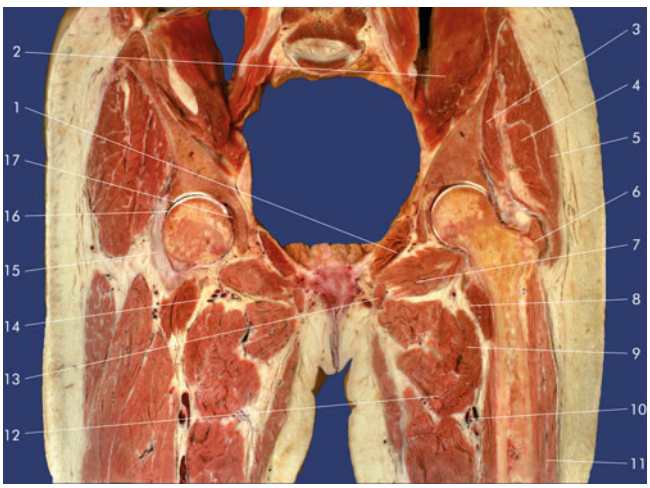


Fig. 67.7 Frontal dissection through the pelvis. (*1*) Obturator internus muscle, (*2*) iliacus muscle, (*3*) gluteus minimus muscle, (*4*) gluteus medius muscle, (*5*) gluteus maximus muscle, (*6*) greater trochanter, (*7*) obturator externus muscle, (*8*) iliopsoas muscle, (*9*) pectineus muscle, (*10*) deep femoral artery, (*11*) vastus lateralis muscle, (*12*) adductor brevis muscle, (*13*) pubic bone (inferior ramus), (*14*) medial femoral circumflex artery, (*15*) iliofemoral ligament, (*16*) acetabular labrum, (*17*) ligament of head of femur (Reproduced with permission from Danilo Jankovic)

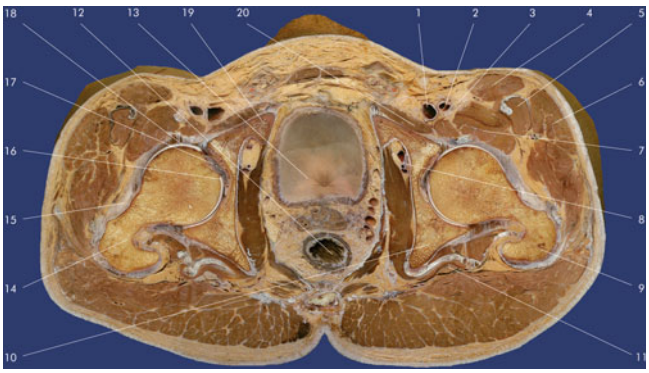


Fig. 67.8 Transversal dissection through the hip joint. (*1*) Femoral vein, (*2*) femoral artery, (*3*) femoral nerve, (*4*) sartorius muscle, (*5*) rectus femoris muscle, (*6*) tensor fasciae latae muscle, (*7*) iliopsoas muscle, (*8*) obturator nerves with vasa obturatoria, (*9*) greater trochanter, (*10*) ischial spine, (*11*) sciatic nerve, (*12*) rectum, (*13*) bladder, (*14*) neck of the femur, (*15*) orbicular zone, (*16*) fovea capitis, (*17*) ligamentum of head of femur, (*18*) acetabular labrum, (*19*) pubic bone, (*20*) pectineus muscle (Reproduced with permission from Danilo Jankovic)

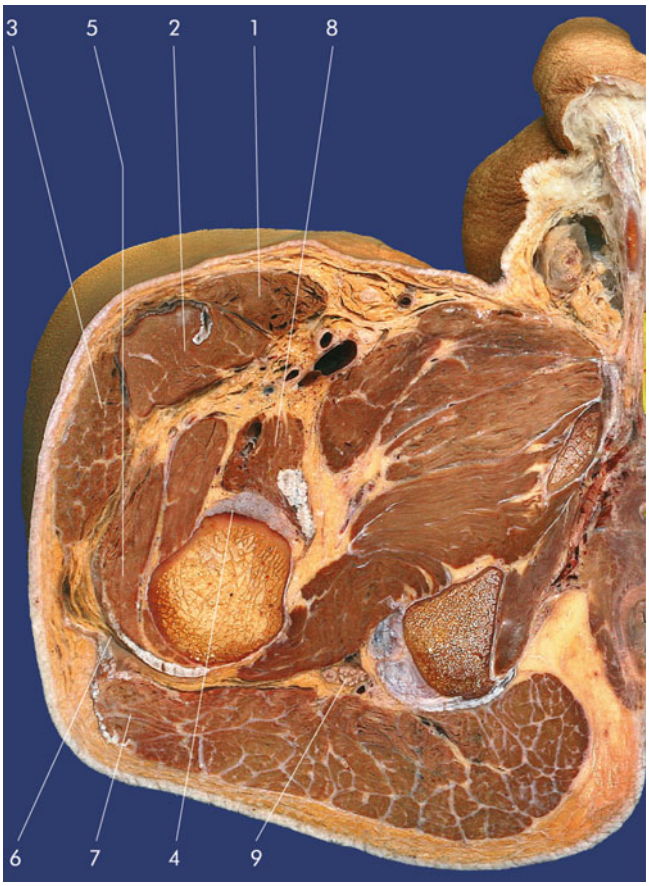


Fig. 67.9 Transversal dissection. (*1*) Sartorius muscle, (*2*) rectus femoris muscle, (*3*) tensor fasciae latae muscle, (*4*) iliofemoral ligament, (*5*) vastus lateralis muscle, (*6*) iliotibial tractus, (*7*) gluteus maximus muscle, (*8*) iliopsoas muscle, (*9*) sciatic nerve (Reproduced with permission from Danilo Jankovic)

The greater trochanter (GT) comprises of four facets: anterior, lateral, superoposterior, and posterior (Fig. 67.10). The anterior and posterior tendons of the gluteus medius insert into the lateral and superoposterior facets respectively. The tendon of the gluteus minimus inserts into the anterior facet. The muscles in the lateral region are divided into two layers. The superficial layer comprises the tensor fascia lata, and gluteus maximus muscle with the triangular interval between these two muscles is filled with fascia lata (Fig. 67.11). The iliotibial (IT) tract is a thickening of the fascia lata commencing at the level of greater trochanter, where three quarter of gluteus maximus muscle and tensor fascia lata are inserted into it. The deep layer comprises of gluteus medius and minimus muscle. Three bursae are described consistently in this region (Fig. 67.12), namely, the subgluteus maximus bursa (SMaB), the submedius bursa (SMeB), and the subminimus bursa (SMiB). The function of the bursae is to serve as a cushion against friction between tendons and fascia lata. The tendons of gluteus minimus and medius can be considered as the rotator cuffs of the hip joint with the IT band and fascia lata as the coracoacromial arch. In both situations, the tendons are covered with bursa against friction.

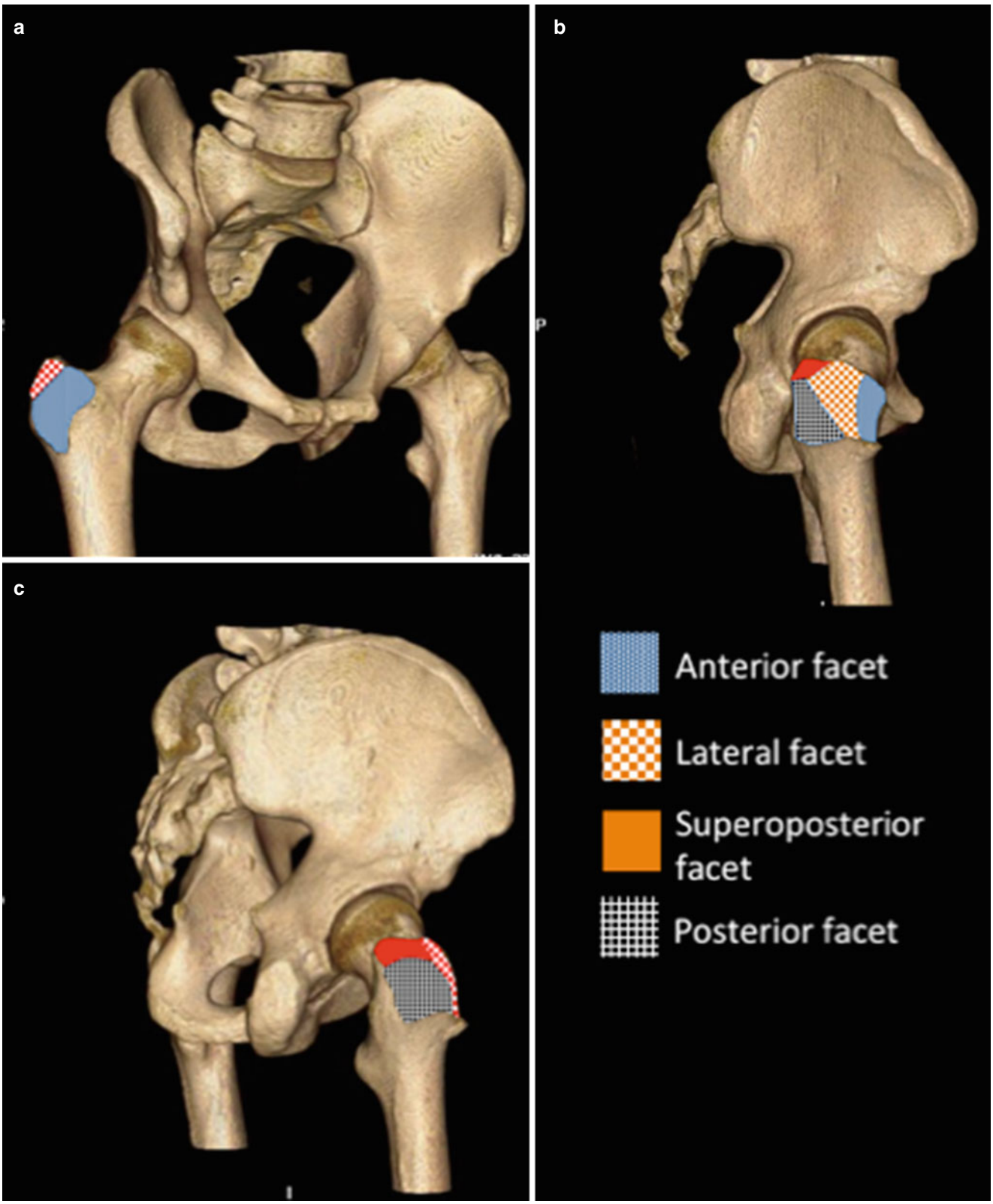


Fig. 67.10 Figure shows the four facets of great trochanter (Reproduced with permission from Philip Peng Educational Series)

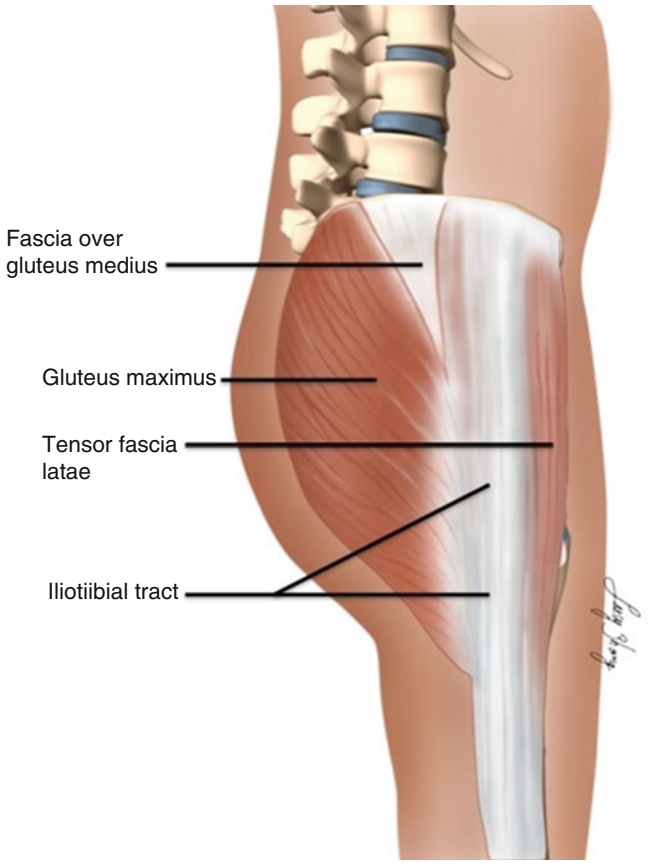


Fig. 67.11 Figure shows the muscles and fascia in the lateral hip region (Reproduced with permission from Philip Peng Educational Series)

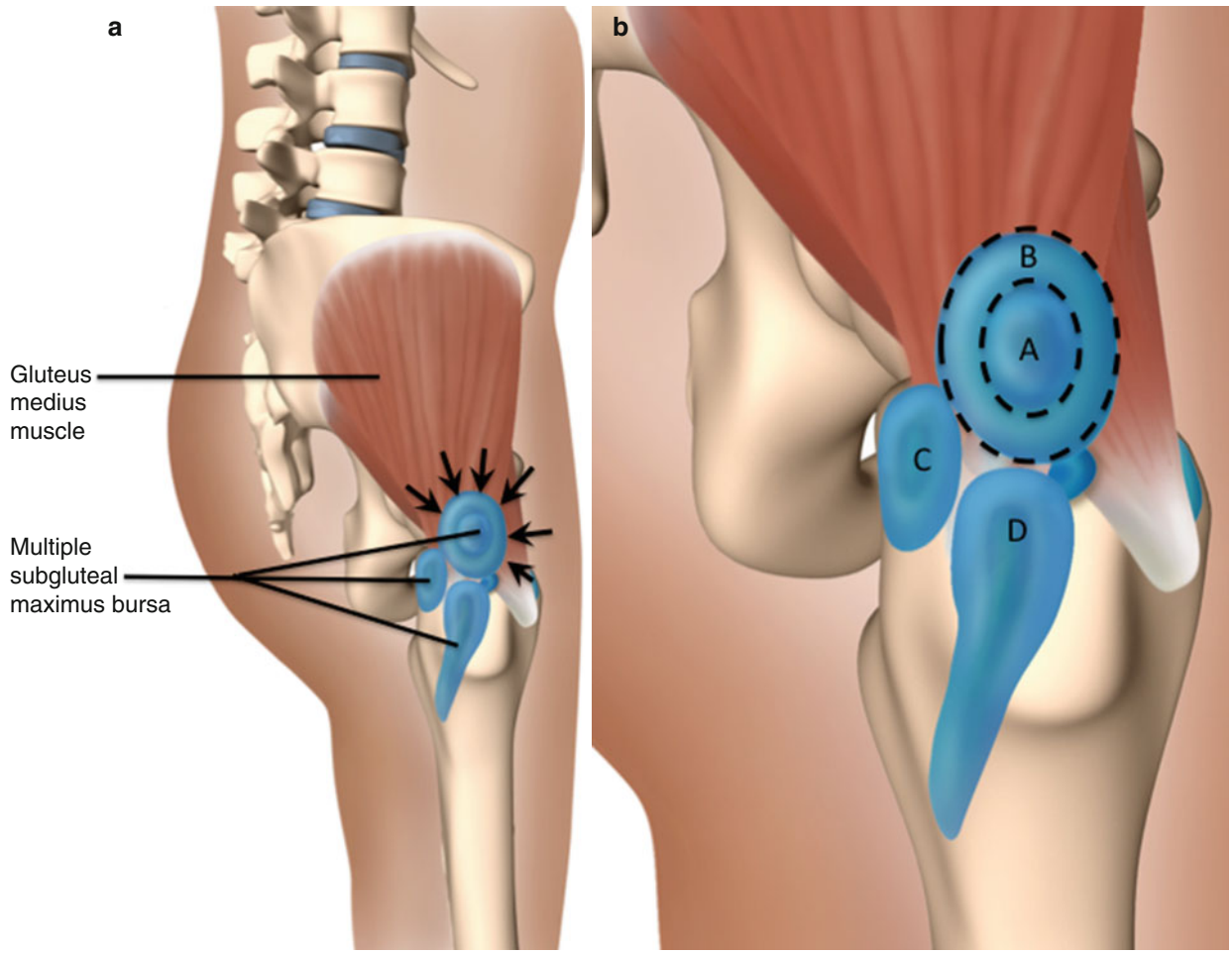


Fig. 67.12 (**a**–**e**) Figures show the bursae in the lateral hip region layer by layer. Figure b is a close up of Figure a. *A* superficial subgluteal maximus bursa, *B* deep subgluteal maximus bursa, *C* secondary deep subgluteal maximus bursa, *D* gluteofemoral bursa, *M* muscle (Reproduced with permission from Philip Peng Educational Series)

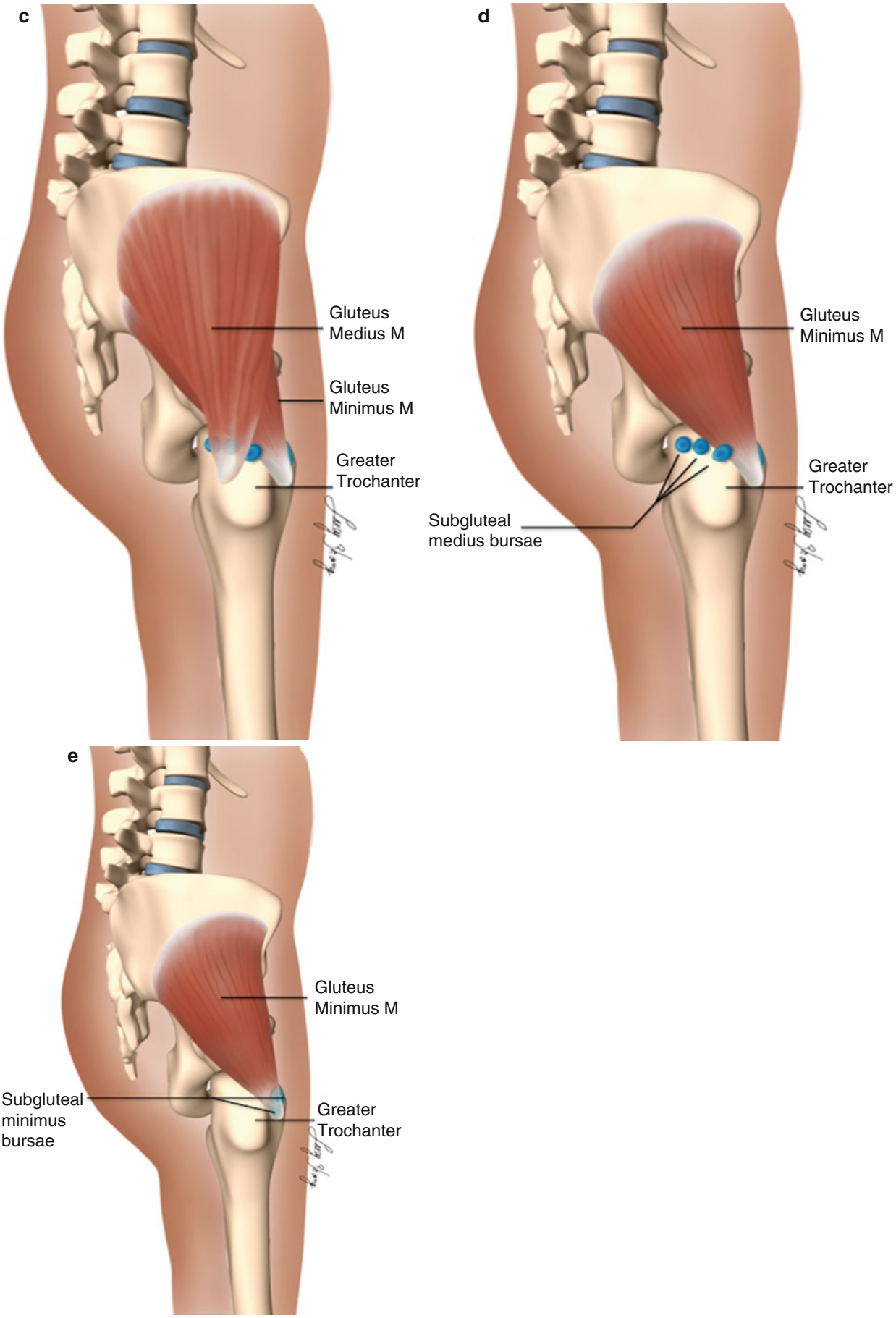


Fig. 67.12 (continued)

Sonoanatomy

The details of the hip joint cannot be visualized by ultrasound. However, the anterior region of the hip, including the anterior acetabulum, labrum, femoral head, and neck as well as the anterior recess, can be revealed with ultrasound. The anterior recess is of particular importance for intra-articular injection as it communicates with hip joint. The technique for revealing the anterior recess of hip joint is anterior oblique sagittal technique, that is, the probe position is aligned with the axis of the femoral neck. Both curvilinear and linear probe can be used, but the author prefers a curvilinear probe. The patient is placed in supine position with the hip in neutral position. The first scan is to locate the femoral neurovascular bundle in the infrainguinal region. The femoral head and acetabulum can usually be seen underneath the iliopsoas muscle and its tendon (Fig. 67.13a). The probe is then rotated to align with the femoral neck such that the following structures can be defined: femoral head, neck, joint capsule, and anterior recess (Fig. 67.13b). The target for intra-articular injection of the hip is the junction of femoral head and neck inside the anterior recess.

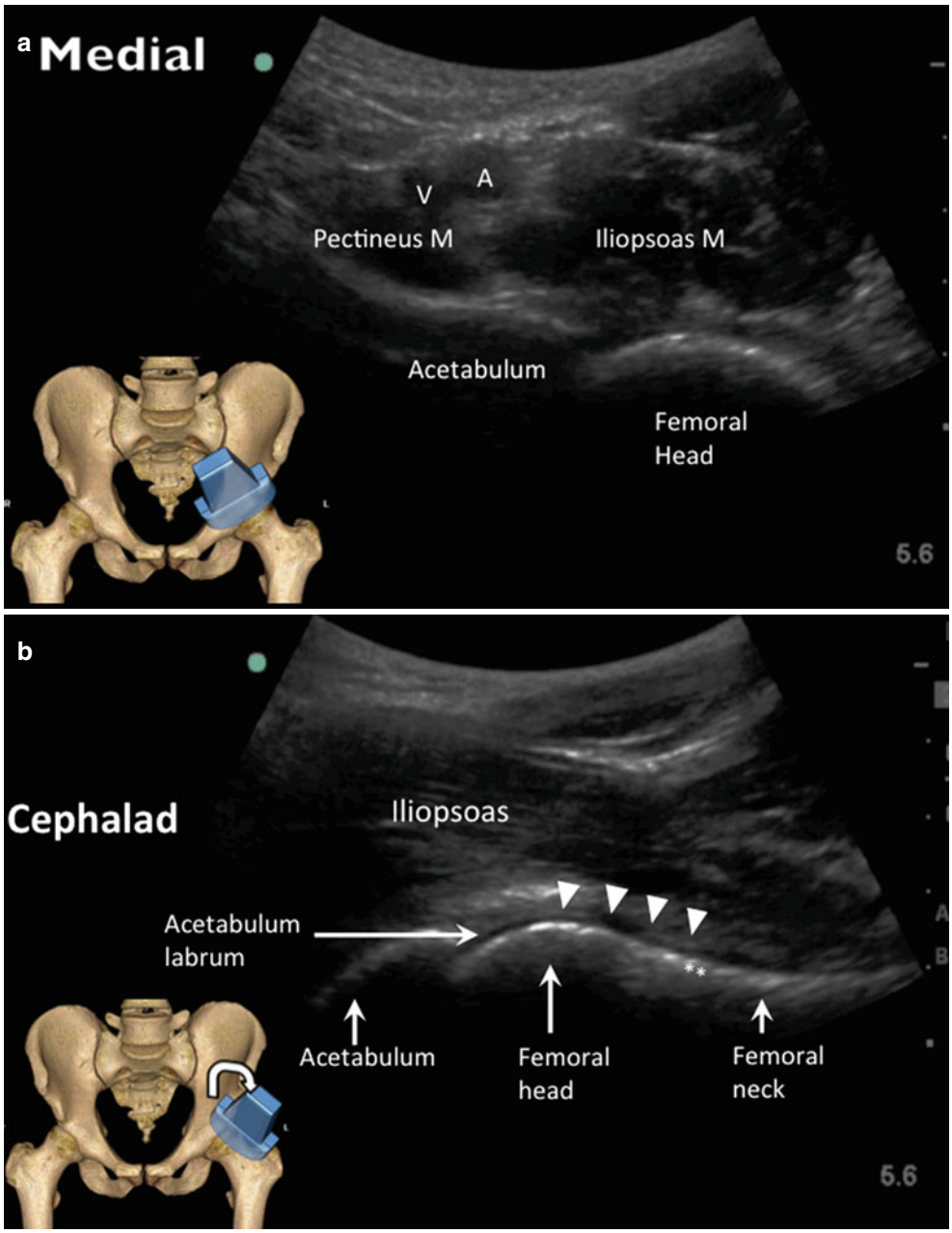


Fig. 67.13 (**a**) Sonoanatomy of the infrainguinal hip region. The position of the transducer is shown in the insert. *A* femoral artery, *V* femoral vein. (**b**) Sonoanatomy of the anterior hip region when the transducer is placed in the long axis of the femoral neck. The *arrow heads* indicate the anterior recess. The position of the transducer is shown in the insert (Reproduced with permission from Philip Peng Educational Series)

The targets for injection for GTPS are the tendons of gluteus medius or gluteus minimus. A linear probe is used and the patient is placed in lateral position with the affected side in the nondependent position. The greater trochanter is scanned in short axis. The junction between the anterior and lateral facet is usually well demarcated by the sharp boney edge (Fig. 67.14). Moving the probe to the anterior facet reveals the gluteus minimus tendon (Fig. 67.15). When the probe is moved over the lateral facet and then turned 90° to obtain a long axis view of the lateral facet, the following structures should be seen in this view: greater trochanter, fascia lata, and tendon of gluteus medius (Fig. 67.16).

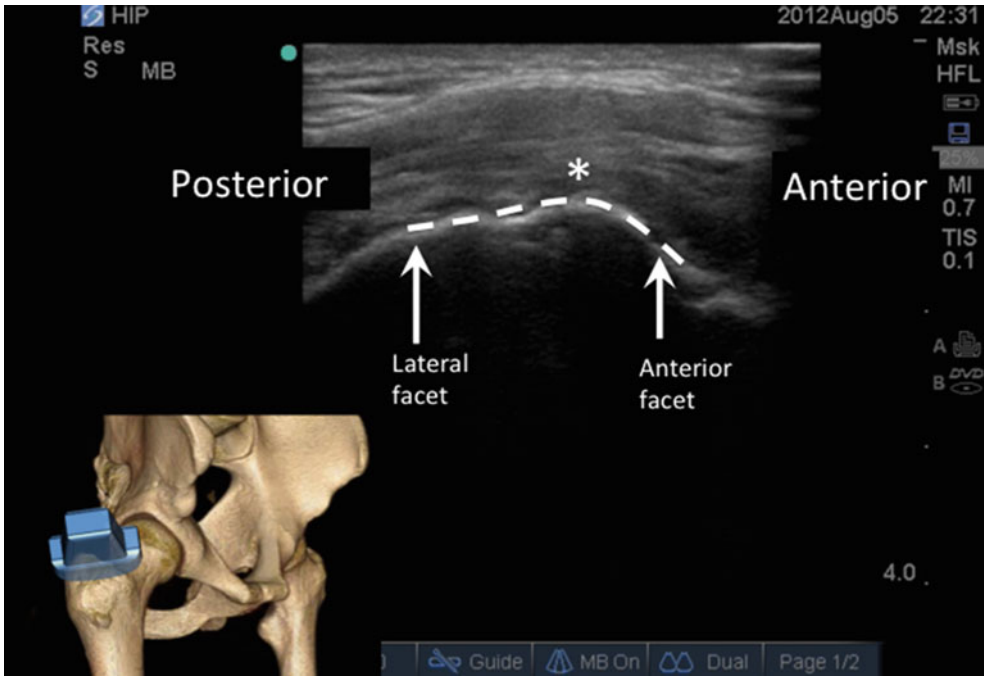


Fig. 67.14 Ultrasonography shows the junction (*) between the anterior and lateral facets of the greater trochanter (*dotted line*). The position of the transducer is shown in the insert (Reproduced with permission from Philip Peng Educational Series)

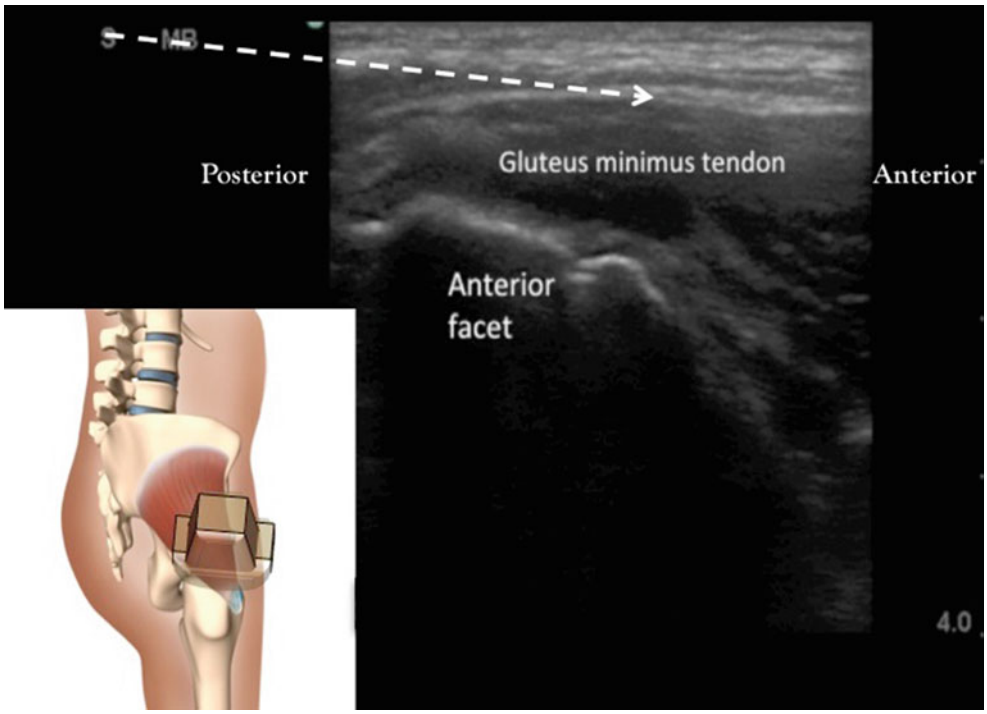


Fig. 67.15 Ultrasonography shows the gluteus medius tendon in short axis. The position of the transducer is shown in the insert. *Dotted arrow* shows the needle insertion path (Reproduced with permission from Philip Peng Educational Series)

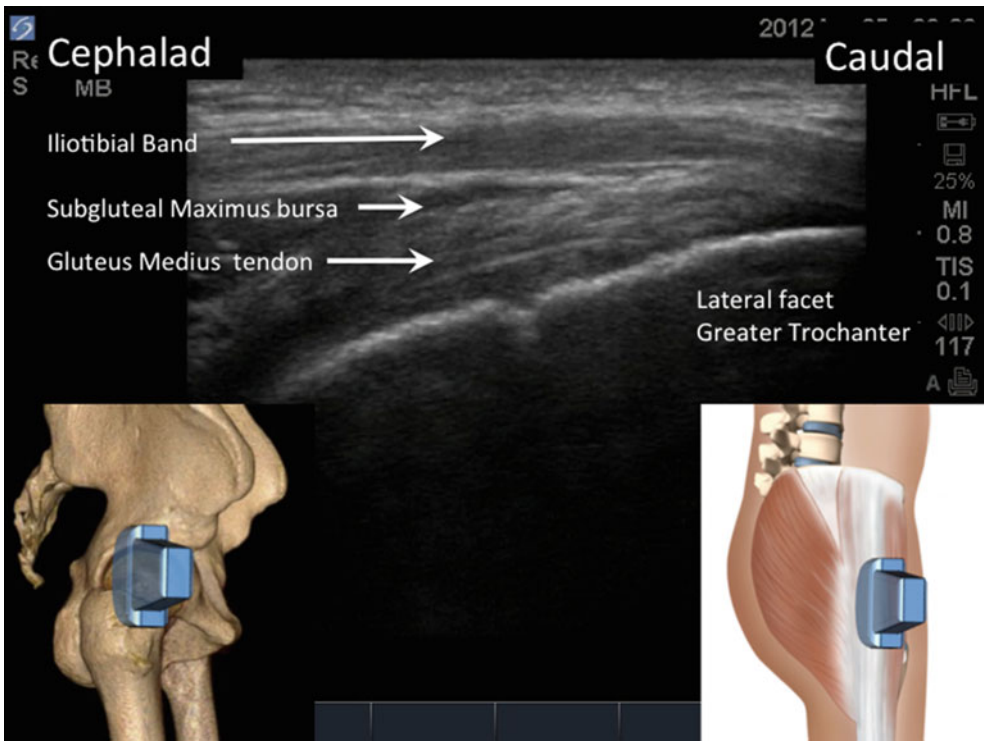


Fig. 67.16 Ultrasonography shows the subgluteal maximus bursa. Note that the axis of the transducer is aligned with the long axis of the iliotibial band as shown in the insert (Reproduced with permission from Philip Peng Educational Series)

Injection Technique

Intra-articular Injection

The anterior recess of hip joint is revealed with anterior oblique sagittal technique. A 3.5-in. 22-G needle is inserted in plane from lateral to medial direction to the junction of femoral head and neck. Before the needle insertion, it is advisable to obtain a Doppler scan to "scout" the vessel in the vicinity of the needle path to avoid inadvertent vessel injury. The volume of injectate is 5 mL, either local anesthetic with steroid (40 mg DepoMedrol) or hyaluronic acid.

GTPS Injection

For the peritendinous injection of the gluteus medius, a long axis scan of the gluteus medius tendon with the insertion to the lateral facet of the greater trochanter is required. A 3.5-in. 22-G needle is inserted in plane from cephalad to caudal direction aiming at the space between the fascia lata and the gluteus medius tendon. The volume of injectate is 5 mL, with local anesthetic with steroid (40 mg DepoMedrol). For the peritendinous injection of the gluteus minimus, the probe is placed over the anterior facet and the tendon is viewed in short axis. A 3.5-in. 22-G needle is inserted in plane from lateral to medial direction directing toward either the superficial or deep aspects of the tendon. The volume of injectate is 5 mL, either local anesthetic with steroid (40 mg DepoMedrol).

Complication

Potential complications of hip injection are similar to that of other intra-articular injections: increase in pain, hematoma, infection, and systematic effect of steroids. In particular, there is a concern about the increase in risk of infection following hip arthroplasty in patients who received hip injection prior to arthroplasty. Four retrospective studies have examined the increased infection risk of intra-articular hip injection to subsequent hip replacement. The earliest publication revealed that the rates of superficial and deep infection were 30 and 10 % compared with 7.5 and 0 % of the matched cohort (the group without prior steroid injection of hip). However, subsequent three publications did not confirm the increased risk from intra-articular injection. One paper suggested an interval of less than 6 weeks between the injection and hip replacement is a risk factor for deep infection.

The adverse effect injection for GRPS is minimal. The patient may experience tenderness in the injection sites but of short term.

Suggested Reading

1. Peng PWH. Ultrasound-guided interventional procedures in pain medicine. A review of anatomy, sonoanatomy, and procedures. Part IV: Hip. Reg Anesth Pain Med. 2013;38:264–73.
2. Peng PW. Hip. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide. Volume 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, CA: Apple Inc.; 2014. p. 77–85.
3. McCarthy J, Noble P, Aluisio FV, Schuck M, Wright J, Lee JA. Anatomy, pathologic features, and treatment of acetabular labral tears. Clin Orthop Relat Res. 2003;406:38–47.
4. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. Anesth Analg. 2009; 108:1662–70.
5. Martinoli C, Bianchi S. Hip. In: Baert AL, Knauth M, Sartor K, editors. Ultrasound of the musculoskeletal system. Heidelberg: Springer; 2007. p. 551–610.
6. Pfirrmann CWA, Chung CB, Theumann NH, Trudell DJ, Resnick D. Greater trochanter of the hip: attachment of the abductor mechanism and a complex of three bursae-MR imaging and MR bursography in cadavers and MR imaging in asymptomatic volunteers. Radiology. 2001;221:469–77.
7. Silva F, Adams T, Feinstein J, Arroyo RA. Trochanteric bursitis: refuting the myth of inflammation. J Clin Rheumatol. 2008; 14:82–6.
8. Woodley SJ, Mercer SR, Nicholson HD. Morphology of the bursae associated with the greater trochanter of the femur. J Bone Joint Surg Am. 2008;90:284–94.
9. Leopold SA, Battista V, Oliverio JA. Safety and efficacy of intraarticular hip injection using anatomic landmarks. Clin Orthop Relat Res. 2001;391:192–7.
10. Smith J, Hurdle MFB, Weingarten TN. Accuracy of sonographically guided intra-articular injections in the native adult hip. J Ultrasound Med. 2009;28:329–35.
11. Cohen S, Strassels S, Foster L, et al. Comparison of fluoroscopically guided and blind corticosteroid injections for greater trochanteric pain syndrome: multicentre randomised controlled trial. BMJ. 2009;338:986–8.
12. Brinks A, van Rijn RM, Willemsen SP, et al. Corticosteroid injections for greater trochanteric pain syndrome: a randomized controlled trial in primary care. Ann Fam Med. 2011;9:226–34.

Chapter 68

Knee Joint: Intra-articular Injection

Philip W.H. Peng, MBBS, FRCPC
Department of Anesthesia, Toronto Western Hospital, University of Toronto, McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, M5T 2S8, Canada
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_68,

Clinical Relevant Anatomy

Knee Joint [1, 3, 5, 7]

The knee joint (Fig. 68.1), the largest joint in the human body, is a hinge joint that allows bending and stretching. The femoral condyles and tibial condyles are parts of the joint. The knee joint space includes the space between and around the condyles, includes the femoropatellar joint behind the patella superiorly, and then communicates freely with the suprapatellar bursa between the tendon of the quadratus femoris muscle and the femur. The flaccid, soft capsule is thin anteriorly and laterally and is strengthened by ligaments. In the anterior capsular wall, the patella – a large sesamoid bone – is embedded in the tendon of the quadriceps femoris muscle.

Ligaments in the Knee Joint [3, 7]

The stability of the knee is mainly maintained by various knee ligaments. The patellar ligament is a continuation of the quadriceps ligament and passes from the patella to the tibial tuberosity. The lateral patellar retinaculum is formed from fibers of the vastus lateralis and rectus femoris muscles, and fibers from the iliotibial tract also radiate into it. The medial patellar retinaculum mainly arises from fibers from the vastus medialis (Figs. 68.2 and 68.3).

The collateral ligaments prevent overextension of the knee joint, as well as abduction and adduction. The broad collateral tibial ligament stretches between the medial condyles of the femur and the tibia and is firmly adherent to the medial meniscus. The round fibular collateral ligament is not firmly attached to the joint capsule (Figs. 68.2 and 68.3).

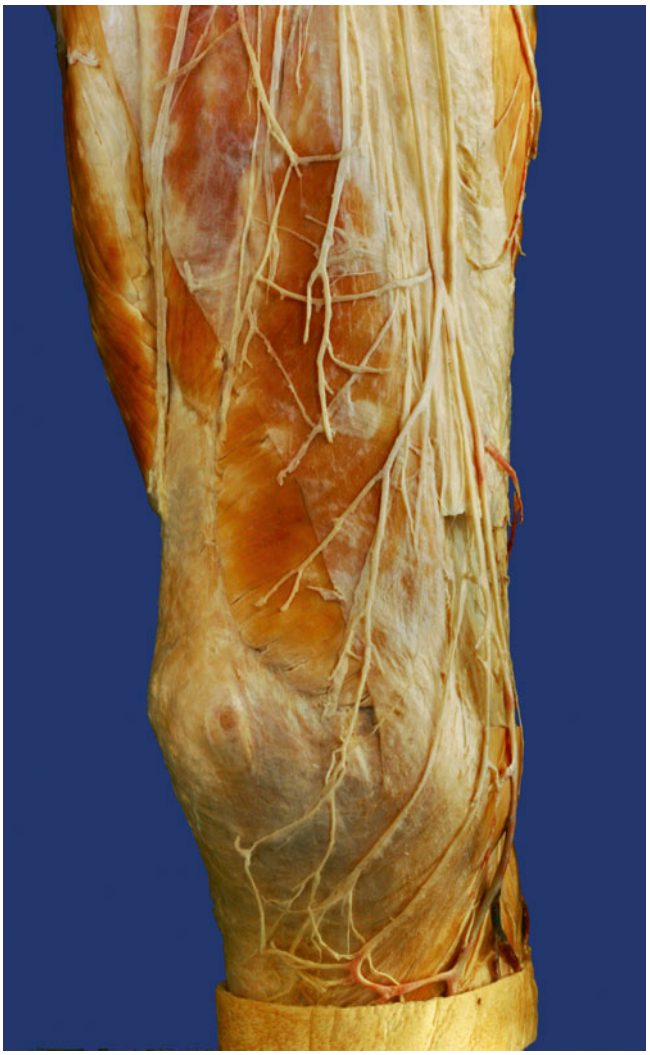


Fig. 68.1 Knee joint area (Reproduced with permission from Danilo Jankovic)

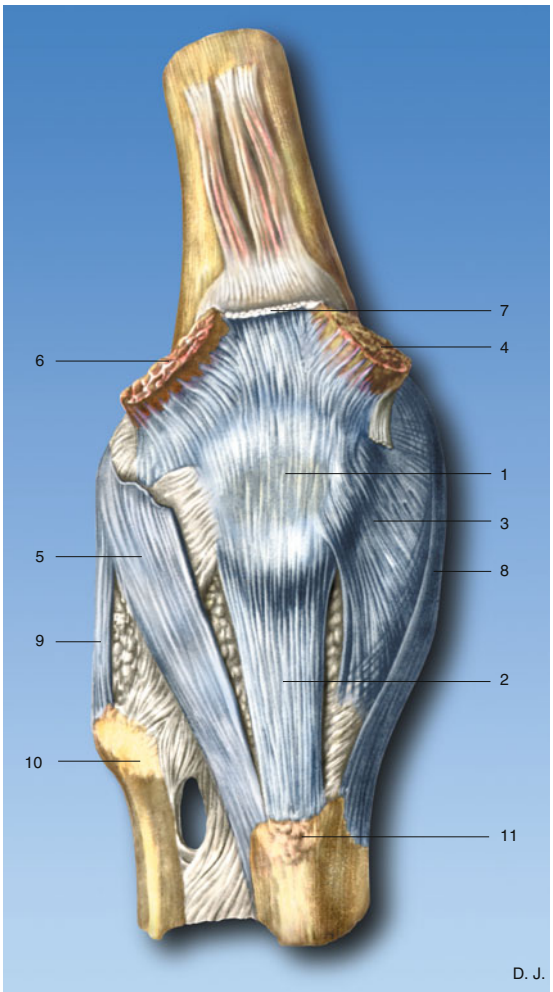


Fig. 68.2 (*1*) Patella, (*2*) ligamentum patellae, (*3*) medial patellar retinaculum, (*4*) vastus medialis muscle, (*5*) lateral patellar retinaculum, (*6*) vastus lateralis muscle, (*7*) tendon of the rectus femoris muscle, (*8*) tibial collateral ligament, (*9*) fibular collateral ligament, (*10*) head of fibula, (*11*) tibial tubercle (Reproduced with permission from Danilo Jankovic)

The cruciate ligaments (anterior and posterior) serve to maintain contact during rotational movements (Figs. 68.2 and 68.3). The menisci (lateral and medial) consist of connective tissue with abundant collagenous fibrous material and embedded cartilage-like cells (Fig. 68.3).

The infrapatellar fat pad (Hoffa's fat pad) forms the anterior part of the medial septum, which together with the infrapatellar synovial plica and cruciate ligaments separates the femorotibial joints from one another. From the medial and lateral margin of the articular surface of the patella, the synovial membrane forms twofold — the alar folds – which project inside the joint and cover the fat pad (Fig. 68.4).

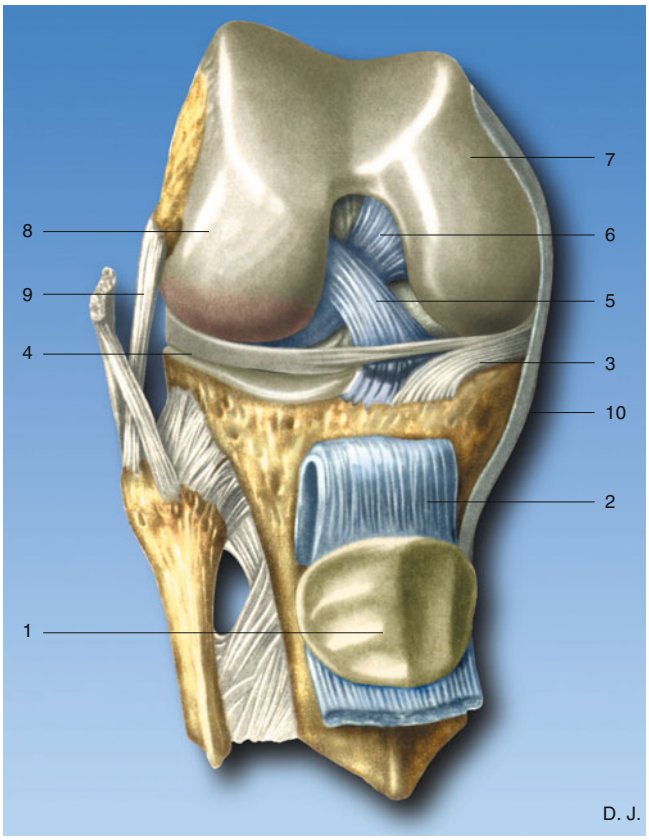


Fig. 68.3 (*1*) Articular surface of the patella, (*2*) ligamentum patellae, (*3*) medial meniscus, (*4*) lateral meniscus, (*5*) anterior cruciate ligament, (*6*) posterior cruciate ligament, (*7*) medial epicondyle, (*8*) lateral epicondyle, (*9*) fibular collateral ligament (lateral ligament), (*10*) tibial collateral ligament (lateral ligament) (Reproduced with permission from Danilo Jankovic)

Vascular Supply and Innervation of the Knee Joint [7]

The vascular anastomosis for the knee joint is formed by the terminal branches of ten vessels:

- From above: from the lateral circumflex femoral artery (descending branch) and the descending genicular artery from the femoral artery.
- At the level of the joint: five branches are contributed from the popliteal artery.
- From below: three branches of leg arteries, from the anterior and posterior tibial arteries and from the fibular artery.

The above arteries are accompanied by the veins with the same names (Fig. 68.5a–d).

The innervation of the knee joint is provided by numerous nerves: joint branches from the femoral nerve reach the knee via neural branches supplying the vastus muscles and via the saphenous nerve, via articular branches from the posterior branch of the obturator nerve, and via articular branches from the sciatic nerve (tibial and common fibular [peroneal] nerves) (Figs. 68.5a–d and 68.6).

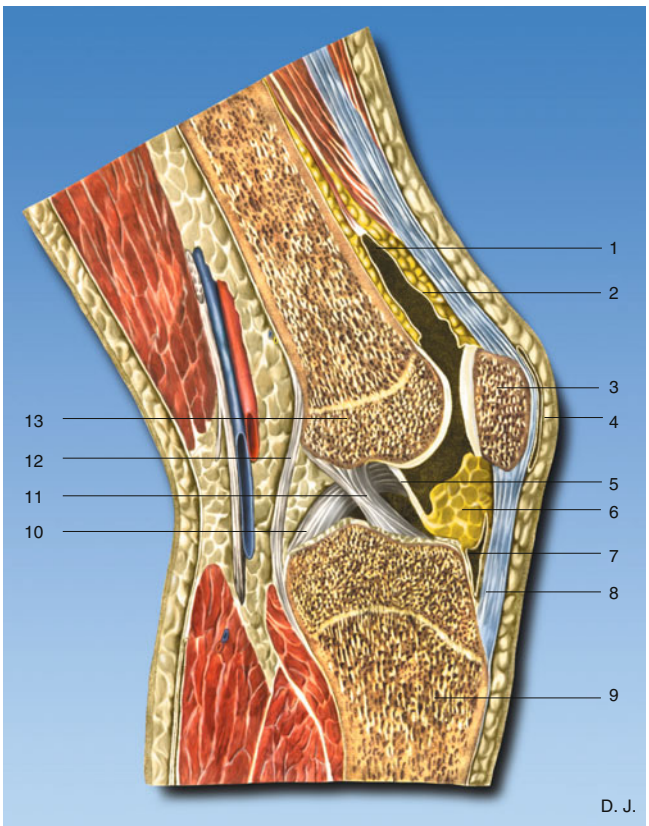


Fig. 68.4 (*1*) Suprapatellar bursa, (*2*) corpus adiposum suprapatellare, (*3*) patella, (*4*) prepatellar bursa, (*5*) synovial fold, (*6*) infrapatellar fat pad (Hoffa's fat pad), (*7*) infrapatellar bursa, (*8*) ligamentum patellae, (*9*) tibia, (*10*) posterior cruciate ligament, (*11*) anterior cruciate ligament, (*12*) articular capsule, (*13*) lateral epicondyle (Reproduced with permission from Danilo Jankovic)

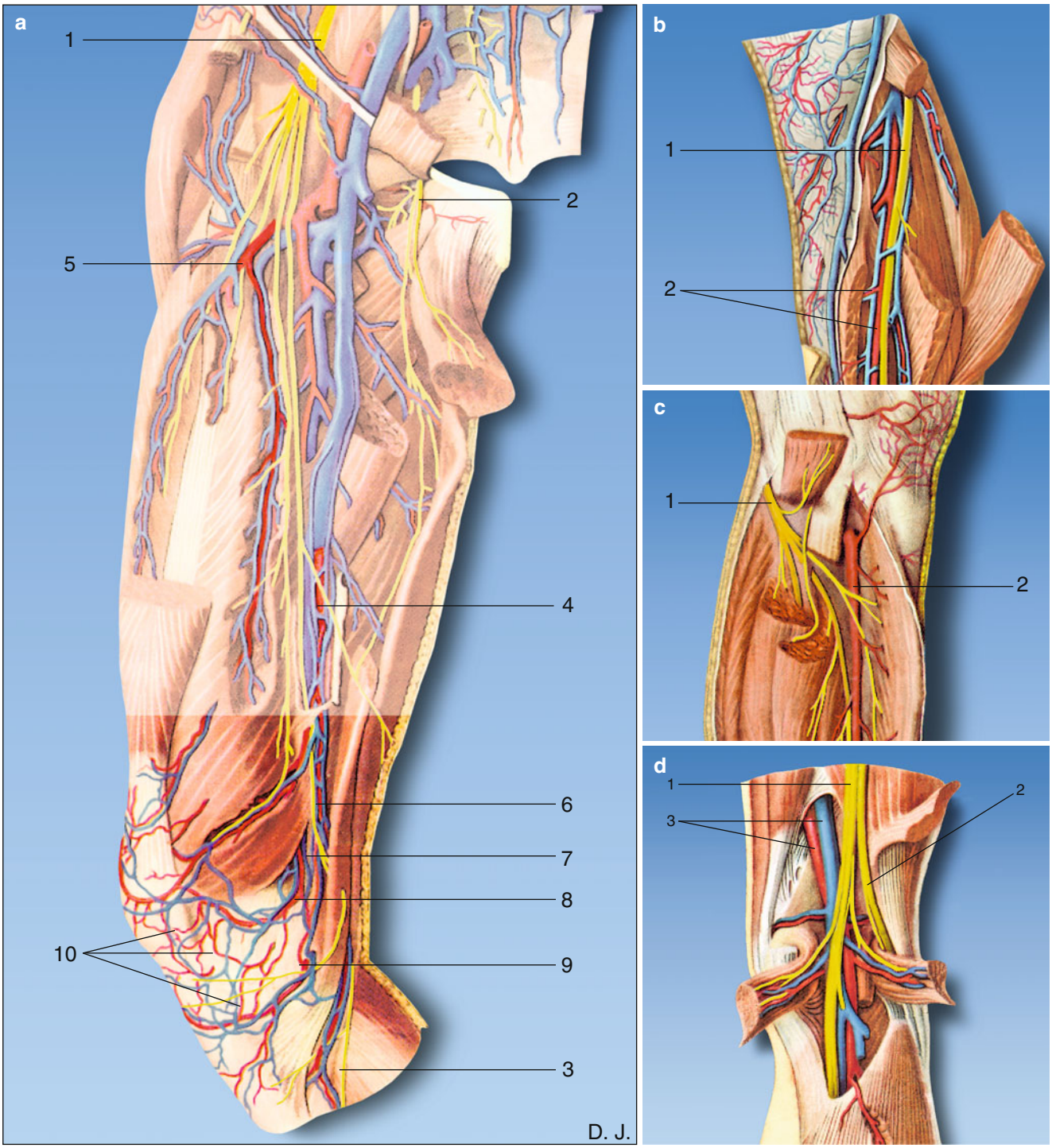


Fig. 68.5 (**a**–**d**) Vascular supply and innervation of the knee joint. (**a**) Anterior view. (*1*) Femoral nerve, (*2*) obturator nerve, (*3*) saphenous nerve, (*4*) femoral artery, (*5*) lateral femoral circumflex artery (descending branch), (*6*) descending genicular artery, (*7*) articular branch, (*8*) superior medial genicular artery, (*9*) inferior medial genicular artery, (*10*) rete patellae. (**b**) Medial view. (*1*) Tibial nerve, (*2*) posterior tibial artery and vein. (**c**) Antero-lateral view. (*1*) Common peroneal (fibular) nerve, (*2*) anterior tibial artery. (**d**) Popliteal fossa. (*1*) Tibial nerve, (*2*) common peroneal (fibular) nerve, (*3*) popliteal artery and vein (Reproduced with permission from Danilo Jankovic)

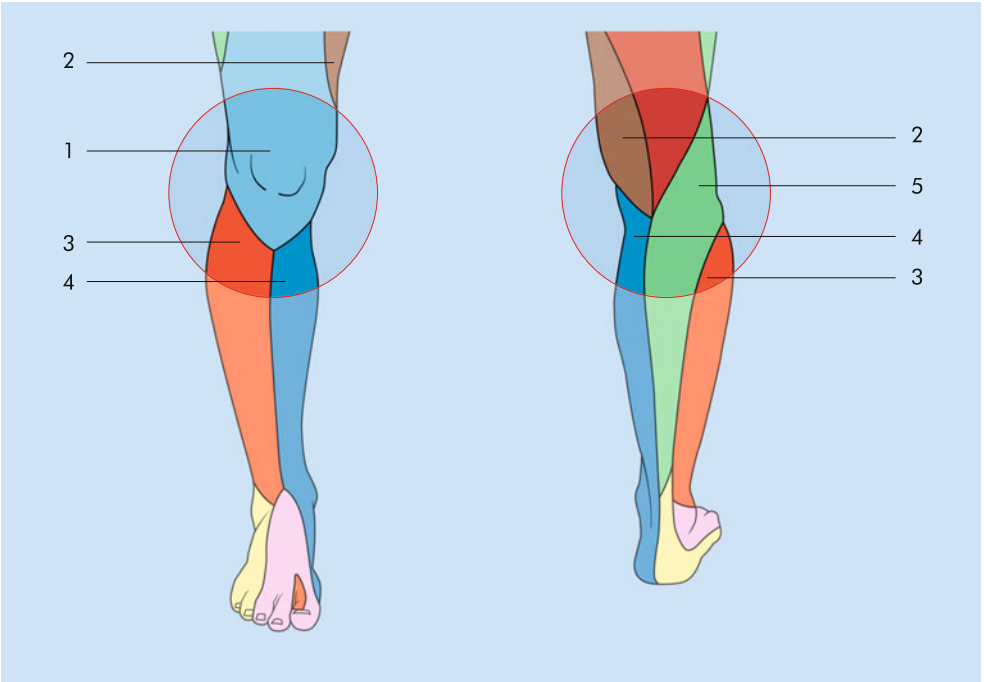


Fig. 68.6 Knee joint. Cutaneous innervation area. (*1*) Femoral nerve, (*2*) obturator nerve, (*3*) common peroneal (fibular) nerve, (*4*) saphenous nerve, (*5*) tibial nerve (Reproduced with permission from Danilo Jankovic)

Sonoanatomy

When examining the knee joint, the patient is placed in supine position with the knee slightly flexed 20°–30° on a bolster to keep the quadriceps tendon taut [8, 9]. A high-frequency (6–12 MHz) linear transducer is usually used. The target is the suprapatellar bursa, which communicates with the knee joint and can be revealed by placing the ultrasound probe just above the patella in long axis with the quadriceps tendon (Fig. 68.7a–c). The suprapatellar bursa appears as a thin hypoechoic line between the suprapatellar and prefemoral fat pads. Voluntary contraction of the quadriceps may help identify smaller effusions. The medial and lateral recesses may be examined in a transverse view by the side of the patella (Fig. 68.8).

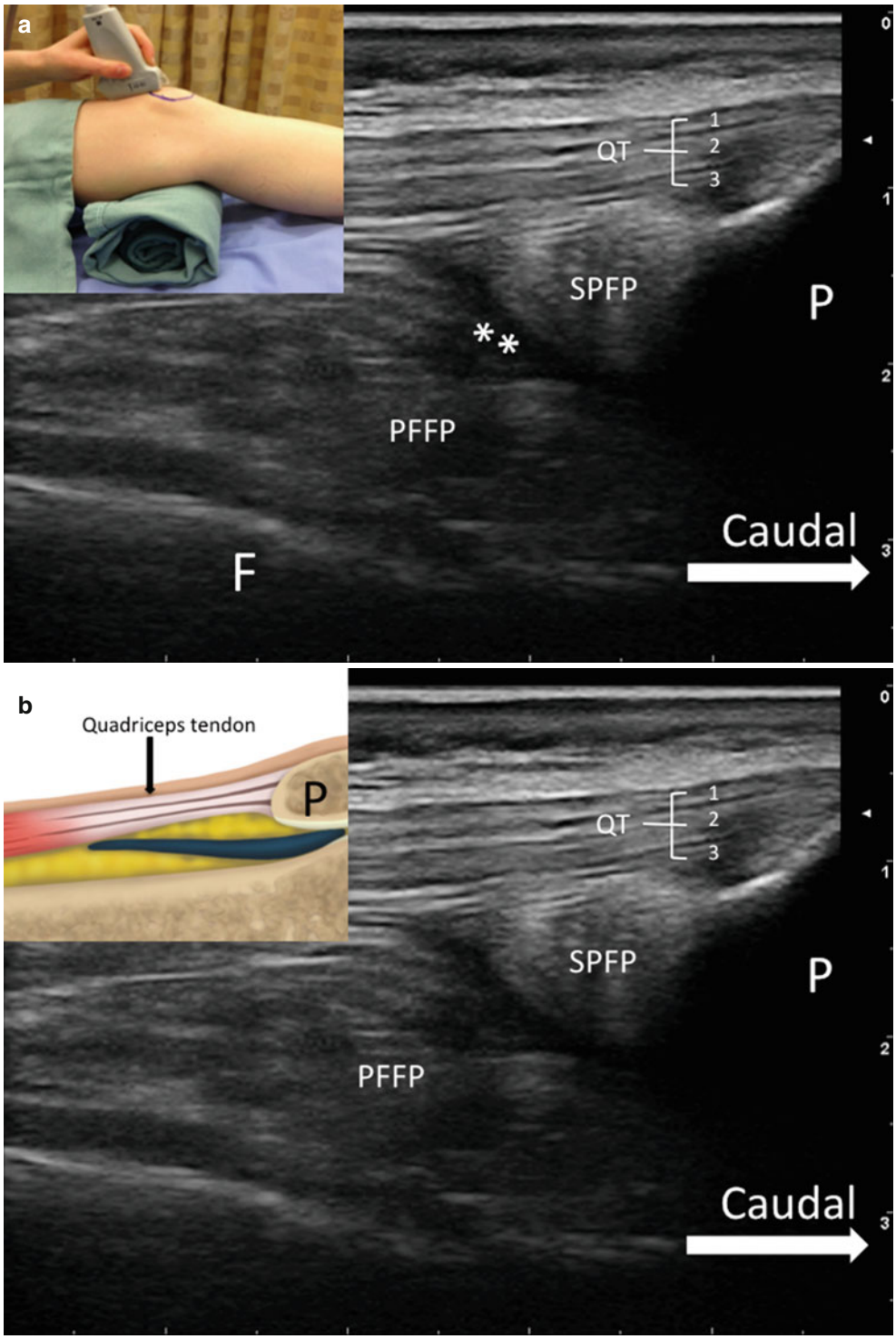


Fig. 68.7 (**a**–**c**) (**a**) Sonogram of the suprapatellar view of the normal knee. The insert showed the position of the patient and the ultrasound probe. (**b**) Sonogram of the details of quadriceps tendon (*QT*). (**c**) Sonogram of the suprapatellar view of a patient with knee effusion. Note the presence of effusion fluid filling the space between prefemoral fat pad and quadriceps tendon. *SPFP* suprapatellar fat pad, *PFFP* prefemoral fat pad, *P* patella, *F* femur, ** indicates the suprapatellar recess (Reproduced with permission from Dr. Philip Peng from Philip Peng Educational Series)

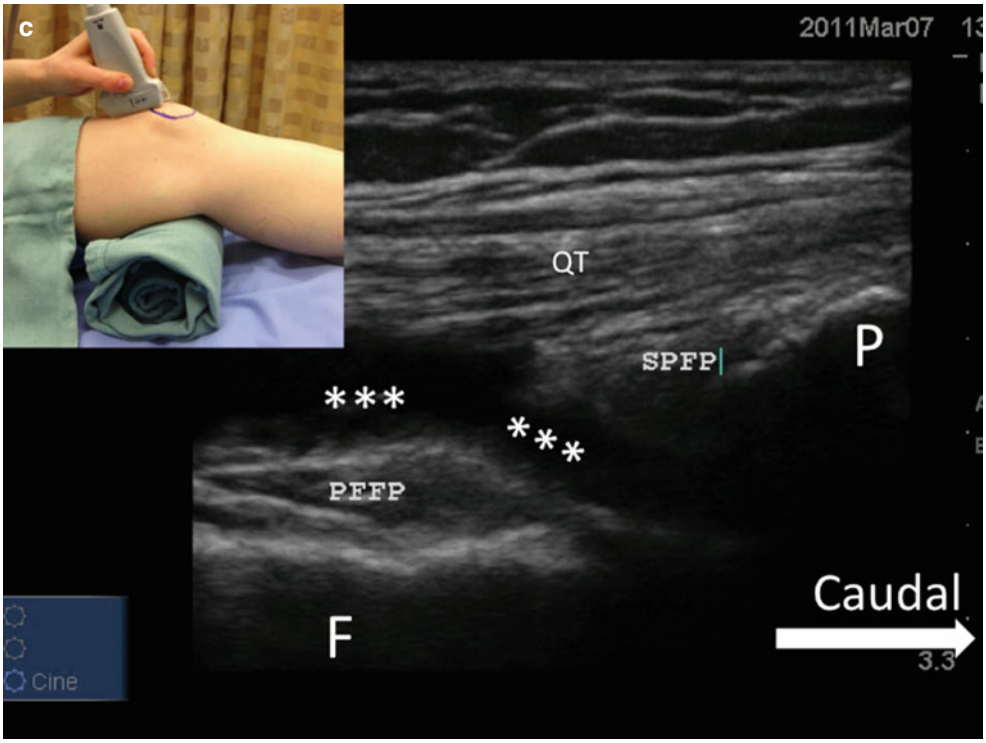


Fig. 68.7 (continued)

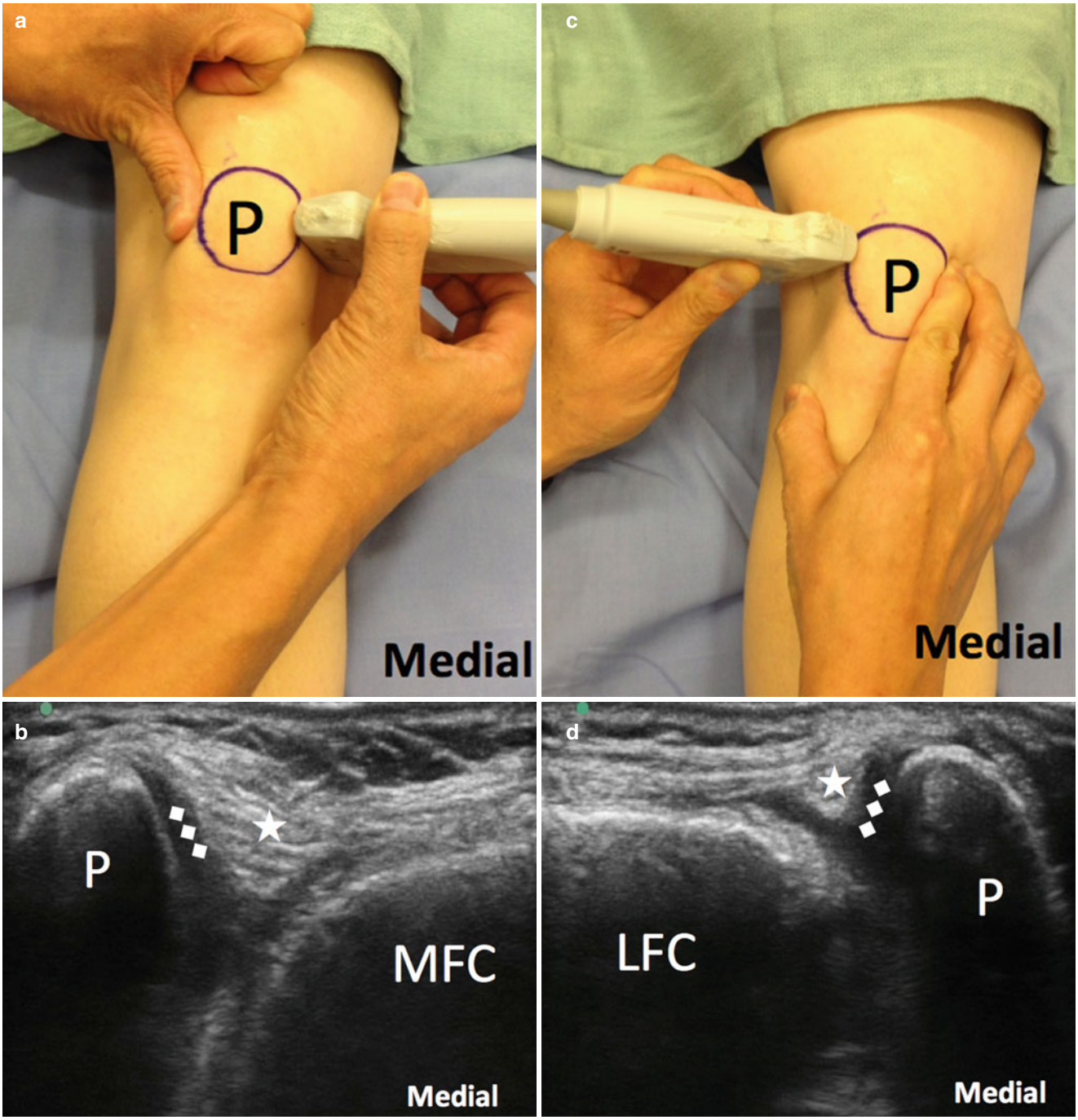


Fig. 68.8 (**a**, **c**) Pictures showed the position of the ultrasound probes and the manipulation of the patella by the examiner. The patella was pushed to the medial and lateral sides respectively in (**a**, **c**). The respective sonograms (**b**, **d**) showed the medial and lateral views respectively. *MFC* medial femoral condyle, *LFC* lateral femoral condyle, *stars* indicated the Hoffa fat pad, *chain of trapezoid* indicated the cartilage (Reproduced with permission from Dr. Philip Peng from Philip Peng Educational Series)

Indications and Contraindications

The main indication for intra-articular injection is the osteoarthritis of the knee which efficacy has been examined in multiple systematic reviews and guidelines [10–13]. The patient reports gradually arising or sudden-onset pain and swelling of the knee. The pain may be ventral and/or dorsal, as the knee is in the area of dermatomes L3/4 and S1/2. The symptoms are exacerbated by weight-bearing activities, and after resting phases, the knee joint may stiffen. Swelling and synovial thickening are palpable at the physical examination. There is a capsular-pattern movement restriction, with flexion being more severely restricted than extension.

Similar to other intra-articular injection, knee injection is contraindicated in patients with infections and skin diseases in the puncture area as well as those receiving anticoagulant treatment.

Procedure

Landmark-Based Technique

There are various techniques for intra-articular knee joint injections. Two techniques will be discussed.

The first one is through medial access [2, 4, 6, 7], in which the patient is placed in supine position with the knee bent by approximately 45° using a knee roll. In this position, the knee joint is well opened at the front and the accessory patellar ligaments are not particularly tensed passively. The patella is pushed laterally with the thumb and raised slightly. A 23-G $2\frac{3}{8}$-inch needle is introduced almost horizontally at the transition from the middle to the lower third of the patella. The trajectory of the needle is directed toward the intercondylar fossa so that the needle is slid into the joint. The joint space is reached after 3–5 cm (depending on the anatomy). Following aspiration, the injection is carried out, and this must only be done if there is no resistance (Fig. 68.9). If there is greater injection resistance (infrapatellar fat pad or cruciate ligaments), the direction must be corrected. The injection can also be carried out in the same way lateral to the patellar ligament.

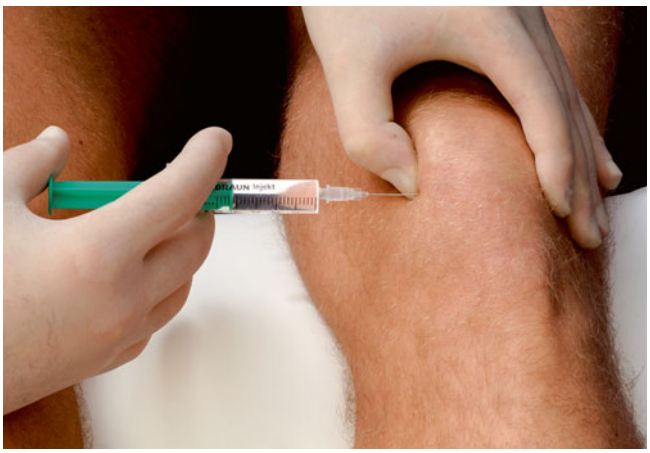


Fig. 68.9 Knee joint injection. Medial access. Transition from the middle to lower third of the patella (medially or laterally) (reproduced with permission from Danilo Jankovic)

Another approach is through ventromedial access. The patient is seated on the examination table, with the lower leg hanging in a relaxed position. There is a fossula medial to the patellar ligament and lateral to the medial condyle of the femur. The needle is introduced into the fossula at an angle of approximately 60° (in a slightly medial direction toward the intercondylar eminence) (Fig. 68.10).

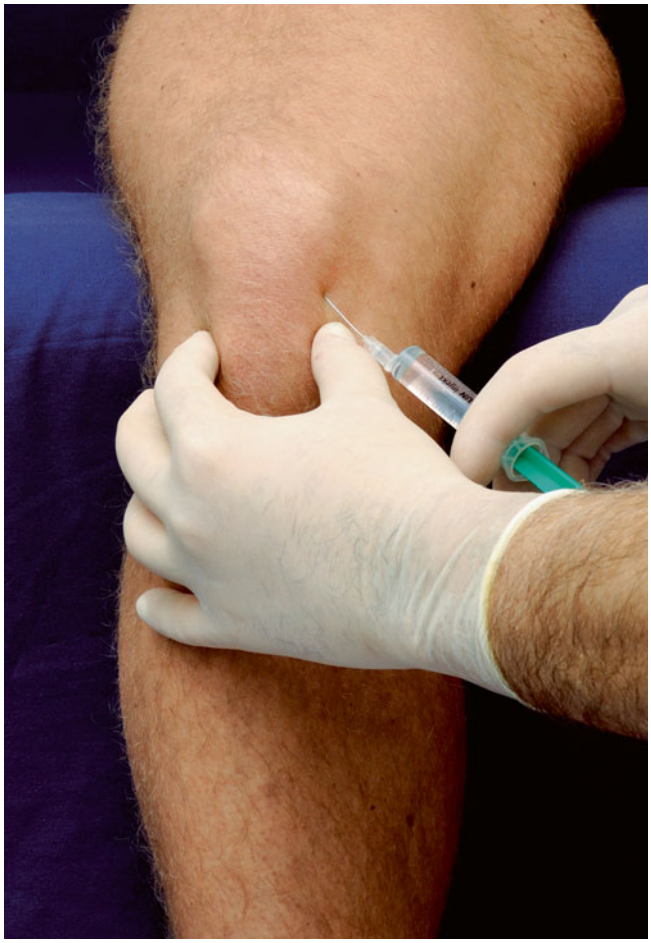


Fig. 68.10 Knee joint injection. Ventromedial access. Sitting position. The lower leg is hanging in a relaxed position. The needle is introduced at the angle of approximately 60° (in a slightly medial direction) (Reproduced with permission from Danilo Jankovic)

Ultrasound-Guided Approach [9–17]

Pooled data on the accuracy rates of landmark-based versus ultrasound guidance techniques are 77.8 % and 95.8 % respectively. In performing landmark-guided injection, the presence of an effusion, not the presence of loss of resistance, greatly enhances the accuracy. This was reflected by a cadaver study examining the reasons for the failure of the landmark-based injection, and most of the inaccuracies were due to the injection into the Hoffa's fat pad (81 %).

Suprapatellar approach is described here, in which the patient is placed in supine position with the knee slightly flexed and supported. Following sterile preparation, the ultrasound probe is placed over junction of the quadriceps tendon and patella to reveal the suprapatellar recess (SPR) (Fig. 68.10). In the situation when the synovial fluid is scant, a couple of maneuvers can be used to augment the SPR. One is to ask the patient to perform isometric contraction of the quadriceps and the other is to apply pressure in the parapatellar space to squeeze the synovial fluid to the SPR. Once the SPR is seen, the ultrasound probe is rotated 90° above the patella. This maneuver allows the needle to puncture the retinaculum instead, thus avoiding needle trauma to the quadriceps tendon (Fig. 68.11).

A 20- or 22-G needle is inserted from lateral to medial in-plane toward the SPR. Alternatively, the ultrasound probe is rotated 45° with the cephalad end directed to the lateral side (superolateral position). In the presence of effusion, aspiration of synovial fluid should always be considered.

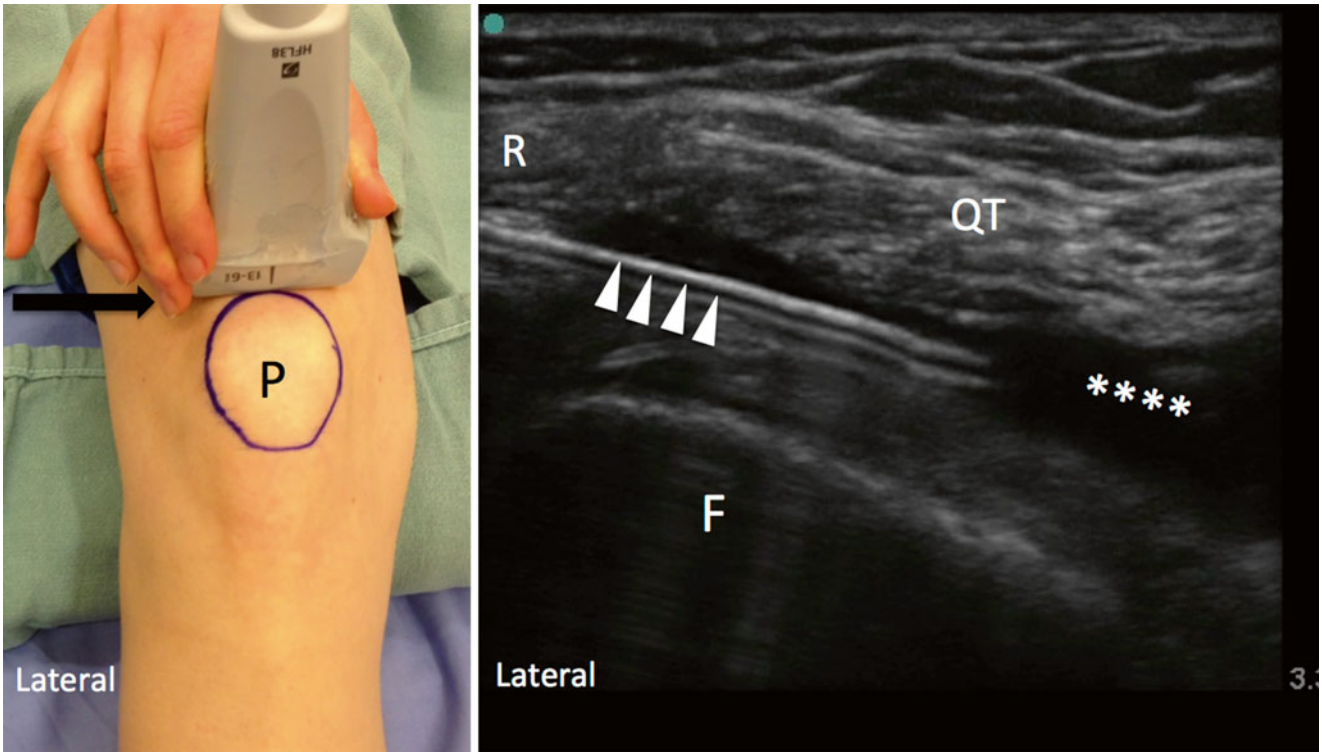


Fig. 68.11 Picture showed the injection technique. The ultrasound probe is placed between the patella and quadriceps tendon initially and then turn 90° upon visualization of the suprapatellar recess. The needle is then approached from lateral to medial to avoid puncturing the quadriceps tendon. The needle is indicated by the *arrowheads* and the suprapatellar recess by *asterisks* (****). *R* retinaculum, *Q* quadriceps tendon, *F* femur (Reproduced with permission from Dr. Philip Peng from Philip Peng Educational Series)

Dosage

The volume of injectate is a mixture of 5 mL of corticosteroid and local anesthetic (40 mg methylprednisolone or triamcinolone diluted in 5 mL of local anesthetic) or hyaluronic acid.

Side Effects and Complications

- Infection (prophylaxis: extremely strict asepsis).
- A temporary increase in pain may occur in approximately 25 % of the patients (who should be informed about this).
- Hematoma formation (lateral access).

References

1. Netter FH. Knee joint-anatomy. In: Firbas H, Kaplan F, hrsg. Farbatlanten der Medizin (Band7) Bewegungsapparat I. Anatomie. Stuttgart/New York: Thieme; 1992.
2. Pellegrini R, Schmitz H, Zohmann A. Schmerzzbehandlung mit Xyloneural (3.Auflage). Wiena: Uhlen Verlag Wien; 1996.
3. Thiel W. Photographischer Atlas der Praktischen Anatomie (2.Auflage). Berlin, Heidelberg, New York, Hongkong, Tokio: Thiel Springer Verlag; 2003.
4. Tilscher H, Eder M. Infiltrationstherapie (3.Auflage). Stuttgart: Hippocrates; 1996.
5. Travel JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual, vol. 2. Baltimore: Williams & Wilkins; 1992.
6. Waldmann S. Pain management injection techniques. Philadelphia, London, New York, St. Louis, Sydney, Toronto: Waldmann-Saunders; 2000.
7. Jankovic D. Kniegelenk-intraartikuläre Injektionen. In Jankovic: Regionalblockaden & Infiltrationstherapie. Berlin: ABW-Verlag; 2008. p. 435–9.
8. Peng PWH, Shankar H. Ultrasound-guided interventional procedures in pain medicine. A review of anatomy, sonoanatomy, and procedures. Part V: Knee joint. Reg Anesth Pain Med. 2014;39(5):368–80.
9. Peng PW. Knee intra-articular injection. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide. Volume 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, CA: Apple Inc.; 2014. p. 90–8.
10. Neustadt DH. Intra-articular injections for osteoarthritis of the knee. Clev Clin J Med. 2006;73:897–911.
11. Bellamy N, Campbell J, Robinson V, Gee T, Bourne R, Wells G. Intraarticular corticosteroid for treatment of osteoarthritis of the knee. Cochrane Database Syst Rev. 2006;(2):CD005328.
12. Cheng OT, Souzdalnitski D, Vrooman B, Cheng J. Evidence-based knee injections for the management of arthritis. Pain Med. 2012;13:740–53.
13. American Academy of Orthopaedic Surgeons. Treatment of osteoarthritis of the knee: evidence-based guideline. http://www.aaos.org.proxy.lib.mcw.edu/research/guidelines/TreatmentofOsteoarthritisoftheKneeGuideline.pdf. Accessed 2 Dec 2013.
14. Daley EL, Bajaj S, Bisson LJ, Cole BJ. Improving injection accuracy of the elbow, knee, and shoulder: does injection site and imaging make a difference? A systematic review. Am J Sports Med. 2011;39:656–62.
15. Berkoff DJ, Miller LE, Block JE. Clinical utility of ultrasound guidance for intra-articular knee injections: a review. Clin Interv Aging. 2012;7:89–95.
16. Esenyel C, Demirhan M, Esenyel M, et al. Comparison of four different intra-articular injection sites in the knee: a cadaver study. Knee Surg Sports Traumatol Arthrosc. 2007;15:573–7.
17. Hermans J, Bierma-Zeinstra SM, Bos PK, Verhaar JA, Reijman M. The most accurate approach for intra-articular needle placement in the knee joint: a systematic review. Semin Arthritis Rheum. 2011;41:106–15.

Chapter 69

Talotibial (Talocrural) and Subtalar Intra-articular Injection

Philip W.H. Peng, MBBS, FRCPC (✉)
McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, Canada M5T 2S8
e-mail: philip.peng@uhn.ca

Danilo Jankovic, MD
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_69, © Springer International Publishing Switzerland 2015

Anatomy [1–3]

The bones of the foot are divided into the tarsus, metatarsus, and toes (Fig. 69.1a, b). The tarsus consists of seven bones (tarsal bones): the talus, calcaneus, navicular, and cuboid bones, and three cuneiform bones. The metatarsus consists of the five metatarsal bones. The toes are formed by the phalanges. A distinction is made in the foot joints between the upper ankle joint (the talocrural joint) and the lower ankle joint (subtalar and talocalcaneonavicular joints) (Fig. 69.2).

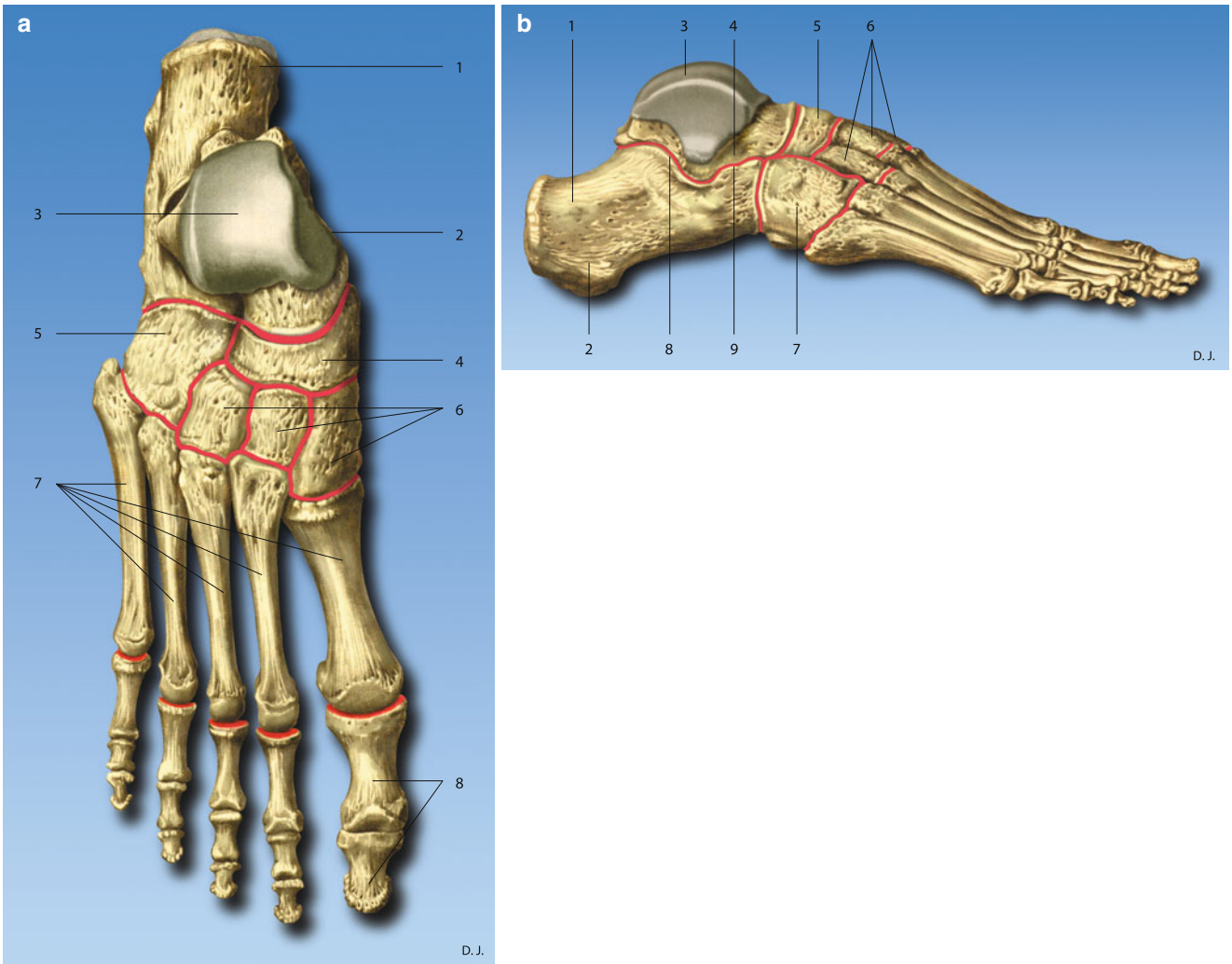


Fig. 69.1 (**a**) Foot skeleton. Dorsal view.(*1*) Calcaneus, (*2*) talus, (*3*) trochlea tali, (*4*) navicular bone, (*5*) cuboid bone, (*6*) cuneiform bone, (*7*) metatarsal bones, (*8*) phalanges (Reproduced with permission from Danilo Jankovic). (**b**) Foot skeleton. Lateral view. (*1*) calcaneus, (*2*) tuber calcaneus, (*3*) talus, (*4*) sinus tarsi, (*5*) navicular bone, (*6*) cuneiform bones, (*7*) cuboid bone, (*8*) subtalar joint, (*9*) talocalcaneonavicular joint (Reproduced with permission from Danilo Jankovic)

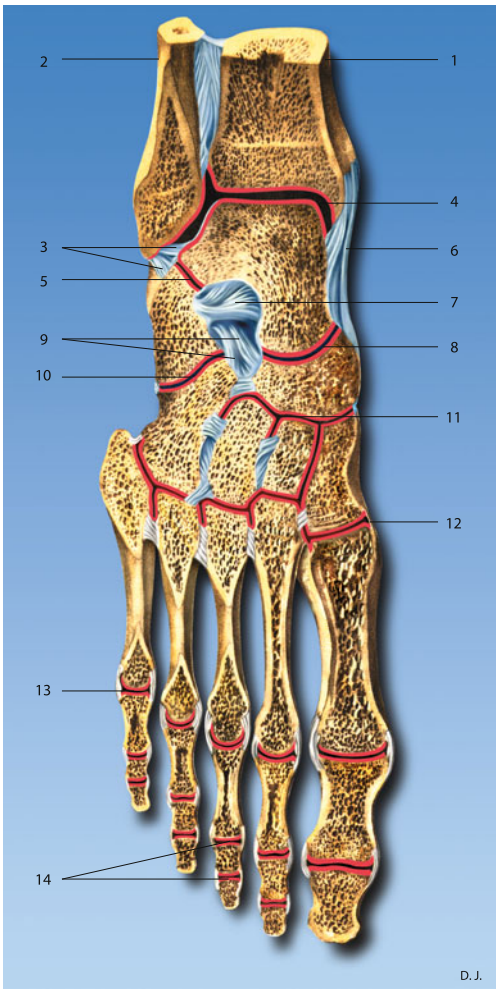


Fig. 69.2 (*1*) Tibia, (*2*) fibula, (*3*) posterior talofibular ligament, (*4*) talocrural joint, (*5*) subtalar joint, (*6*) deltoid ligament, (*7*) talocalcaneus ligament, (*8*) talonavicular ligament, (*9*) bifurcated ligament, (*10*) calcaneocuboid ligament, (*11*) cuneonavicular ligament, (*12*) tarsometatarsal ligament, (*13*) metatarsophalangeal ligament, (*14*) articulationes digiti (Reproduced with permission from Danilo Jankovic)

The joint surfaces of the talocrural joint are formed by the malleolar mortise (mortise joint) at the trochlea of the talus, with its superior facet and medial and lateral malleolar facets. The ligaments of the upper ankle joint are the medial collateral ligament (deltoid ligament), anterior talofibular ligament, posterior talofibular ligament, and calcaneofibular ligament (Figs. 69.3 and 69.4a, b).

The lower ankle joint consists of two separate joints – the subtalar joint, which forms the posterior part of the lower ankle joint, and the talocalcaneonavicular joint, which forms the anterior part of it. These two separate joints act together. The joint surfaces in the subtalar joint are formed by the talus and calcaneus (Figs. 69.3 and 69.4a, b).

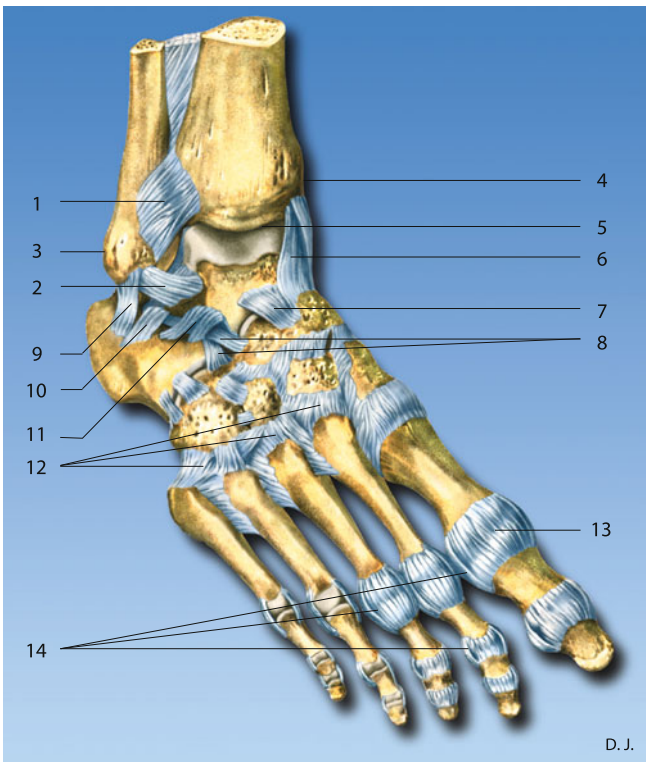


Fig. 69.3 Foot joints. Lateral view. (*1*) Anterior tibiofibular joint, (*2*) anterior talofibular ligament, (*3*) lateral malleolus, (*4*) medial malleolus, (*5*) talocrural joint, (*6*) deltoid ligament (medial collateral ligament), (*7*) talonavicular ligament, (*8*) bifurcated ligament, (*9*) calcaneofibular ligament, (*10*) talocalcanean ligament, (*11*) interosseous talocalcaneal ligament, (*12*) dorsal tarsometatarsal ligaments, (*13*) articular capsule, (*14*) collateral ligaments (Reproduced with permission from Danilo Jankovic)

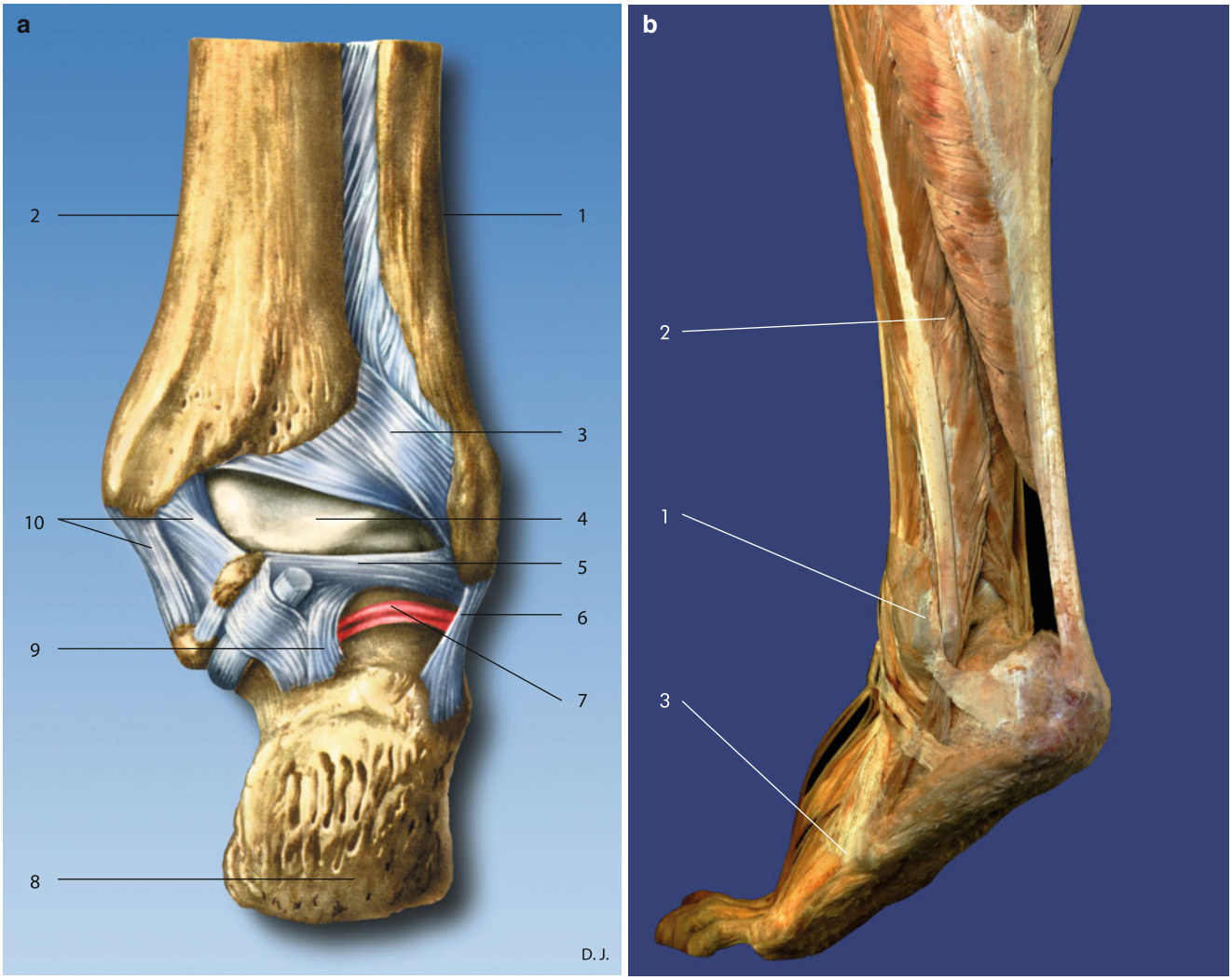


Fig. 69.4 (**a**) A Foot joints. Dorsal view. (*1*) Fibula, (*2*) tibia, (*3*) posterior tibiofibular ligament, (*4*) talus, (*5*) posterior talofibular ligament, (*6*) calcaneofibular ligament, (*7*) subtalar joint, (*8*) calcaneus, (*9*) posterior talocalcaneal joint, (*10*) deltoid ligament (Reproduced with permission from Danilo Jankovic). (**b**) Ankle joint. Lateral view. (*1*) Lateral malleolus, (*2*) peroneal muscles, (*3*) tuberosity of the metatarsal bone 5. With tendon of the peroneus brevis and tertius muscle (Reproduced with permission from Danilo Jankovic)

The vascular supply to the ankle is from branches of the *posterior tibial artery* (the medial and lateral plantar arteries) and from the *anterior tibial artery* (the dorsalis pedis artery). The ankle is innervated from the *tibial nerve* (lateral and medial plantar nerves and calcaneal branches) and from the *deep fibular (peroneal) nerve* (Fig. 69.5a–c).

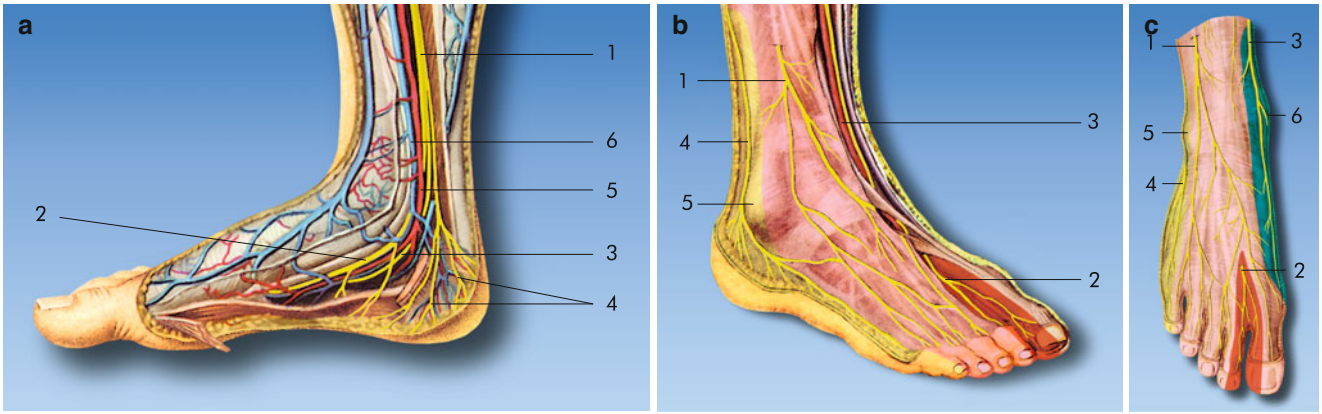


Fig. 69.5 (**a**) Foot. Vascular supply and innervation. (*1*) Tibial nerve, (*2*) medial plantar nerve, (*3*) lateral plantar nerve, (*4*) calcaneal branches, (*5*) posterior tibial artery, (*6*) posterior tibial vein (Reproduced with permission from Danilo Jankovic). (**b**) Cutaneous innervation areas of the back of the foot. Lateral view. (*1*) Superficial peroneal(fibular) nerve, (*2*) deep peroneal(fibular) nerve, (*3*) anterior tibial artery, (*4*) sural nerve, (*5*) lateral malleolus (Reproduced with permission from Danilo Jankovic). (**c**) Cutaneous innervation areas in the region of the back of the foot (from the front). (*1*) Superficial peroneal (fibular) nerve, (*2*) deep peroneal (fibular) nerve, (*3*) saphenous nerve, (*4*) sural nerve, (*5*) lateral malleolus, (*6*) medial malleolus (Reproduced with permission from Danilo Jankovic)

Sonoanatomy [4–6]

For tibiotalar or talocrural joint, the target is the anterior tibiotalar recess between the tibialis anterior tendon and medial malleolus (Fig. 69.6). The patient is placed in supine position with the knee flexed 90° and the foot flat on the table or in semi-recumbent with the ankle in a plantar-flexed position. A linear (8–12 MHz) ultrasound probe is placed over the tibialis anterior tendon first. By moving the probe to the medial direction, the fat pad in the anterior recess of the tibiotalar joint is revealed (Fig. 69.7).

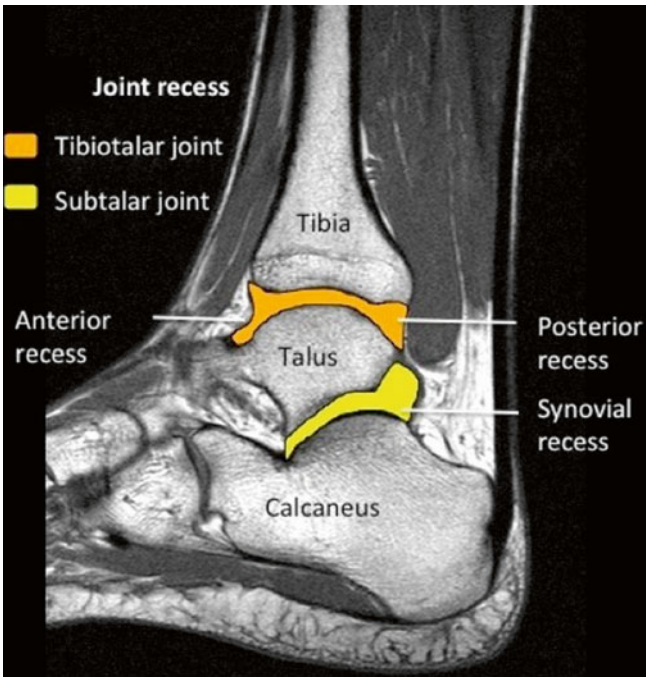


Fig. 69.6 Joint recess for tibiotalar joint and subtalar joint (Reproduced with permission from Philip Peng Educational Series)

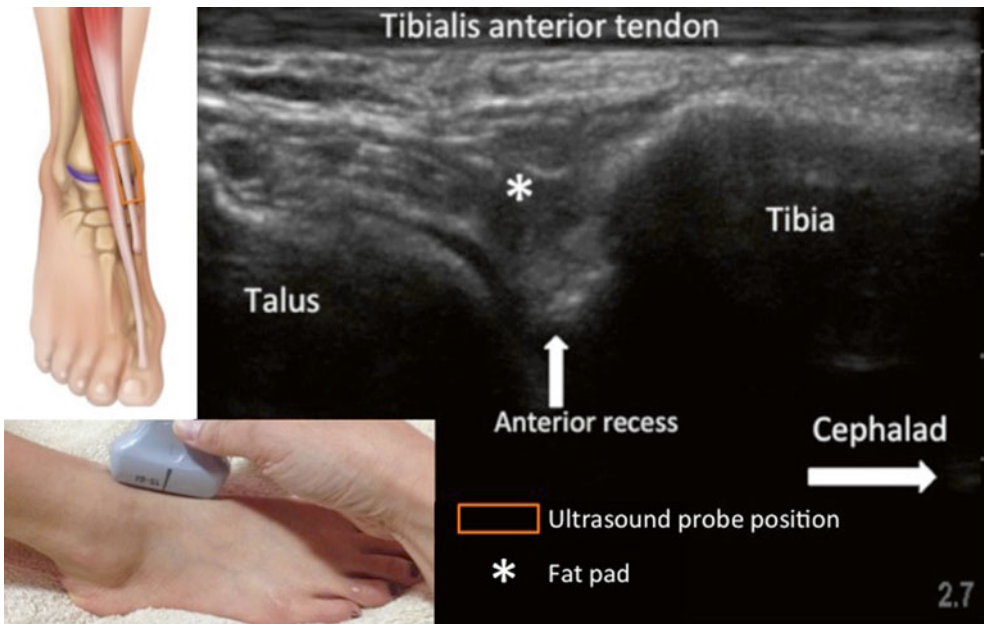


Fig. 69.7 Sonogram of the anterior aspect of tibiotalar joint. The inserts on the left side show the position of the ultrasound probe (*orange rectangle*) (Reproduced with permission from Philip Peng Educational Series)

For subtalar joint, the joint space can be revealed with either medial or lateral approach. The author-preferred approach is the lateral approach. The patient is placed in lateral position (injection side as nondependent side) and a small pillow is placed on the medial aspect of ankle. A linear (8–12 MHz) ultrasound probe is placed anterior to lateral malleolus right on sinus tarsi (Fig. 69.8). The probe is then moved and tilted in posterior direction to reveal the subtalar joint (Fig. 69.9).

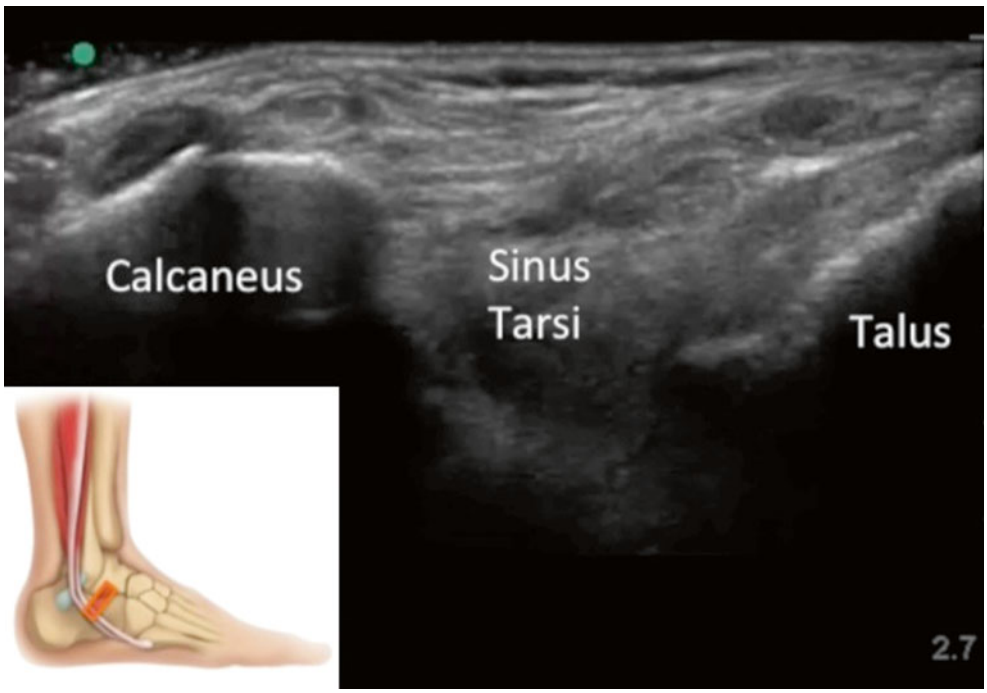


Fig. 69.8 Sonogram of sinus tarsi (Reproduced with permission from Philip Peng Educational Series)

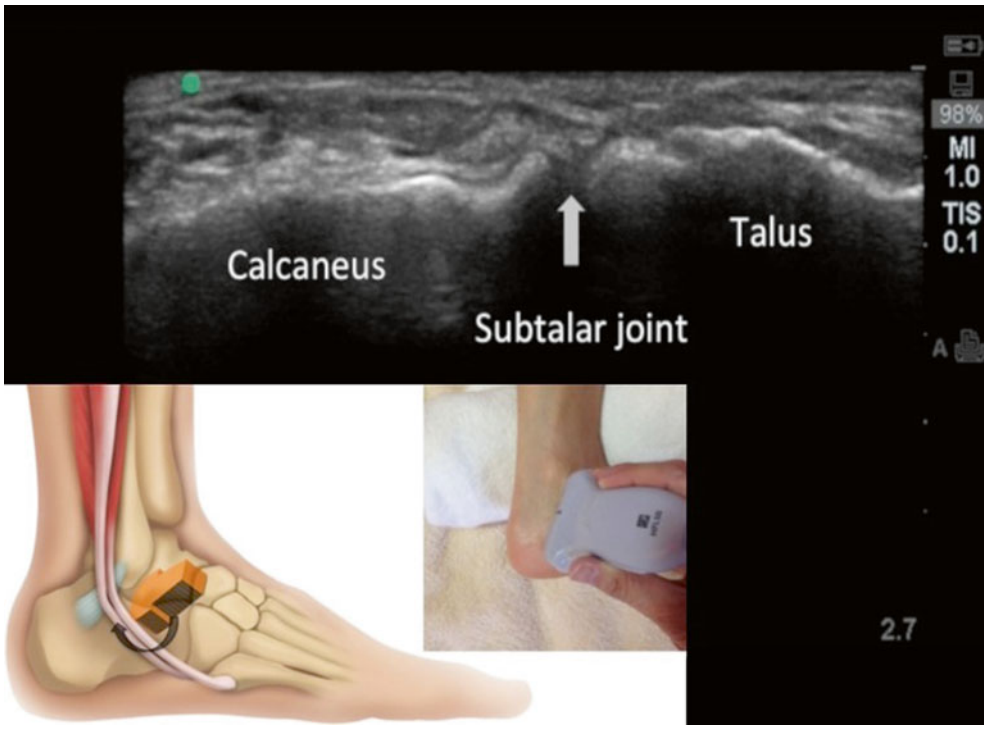


Fig. 69.9 Sonogram of the lateral aspect of posterior subtalar joint. The inserts show the position of the ultrasound probe. The probe position is similar to that of sinus tarsi but moved and tilted toward the lateral malleolus (Reproduced with permission from Philip Peng Educational Series)

Indications and Contraindications

The main indication is osteoarthritis, especially posttraumatic. Arthrosis of the ankle joint is rare in the absence of a causative factor (e.g., fracture, instability, or posture-related joint strain). The patient reports local pain and swelling in the ankle region. The examination reveals capsular-pattern movement restriction, with plantar flexion more severely restricted than dorsal flexion. Contraindications are similar to other joint injections, such as infection in the skin around the joint area and in patients who are taking anticoagulants.

Procedure [7–11]

Literature supports that ultrasound-guided ankle injections are much more accurate than that with landmark guidance alone. In one cadaver study, the investigators directly compared the accuracy of ultrasound with landmark-guided injection in tibiotalar joint. The accuracies were 100 % and 85 % respectively. In a validation study, the accuracy of subtalar joint under ultrasound guidance was 100 %. Ultrasound should be utilized when a definitive diagnosis needs to be established or hyaluronic acid is being utilized.

For tibiotalar or talocrural joint, the target is the anterior tibiotalar recess between tibialis anterior tendon and medial malleolus. The patient position and the ultrasound probe are described in the sonoanatomy section. Both in-plane and out-of-plane techniques have been described.

For out-of-plane technique, ultrasound probe is placed over the extensor hallucis longus and a 1.5-in. 25-G needle is inserted medial to the tibialis anterior tendon into the anterior recess. The injectate should be seen spread deep to the fat fad. In the presence of joint effusion, one should consider a larger gauge needle for aspiration. A total volume of 3–5 mL of injectate is sufficient, either a mixture of local anesthetic with steroid (2 % lidocaine and 40 mg depoMedrol) or a commercial available hyaluronic acid solution (Fig. 69.10). For in-plane technique, the ultrasound probe is first placed over the tibialis anterior tendon first. The probe is moved slightly in medial direction to reveal the anterior joint recess. A 3.5-in. 22-G needle is inserted in-plane deep to the fat pad (Fig. 69.11).

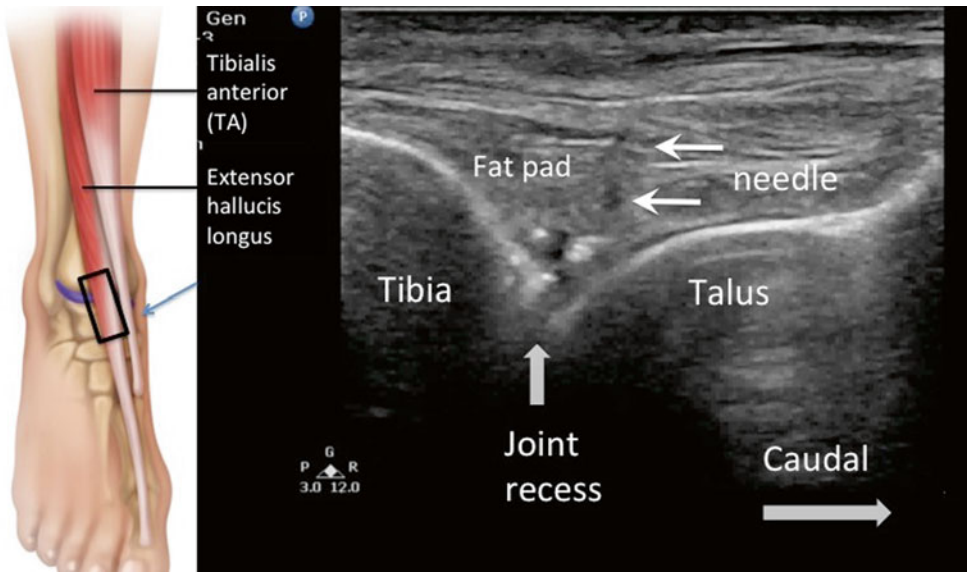


Fig. 69.10 Sonogram shows out-of-plane needle insertion for tibiotalar joint. The *line arrows* indicate the needle path. The insert shows the anatomy and the ultrasound probe position (Reproduced with permission from Philip Peng Educational Series)

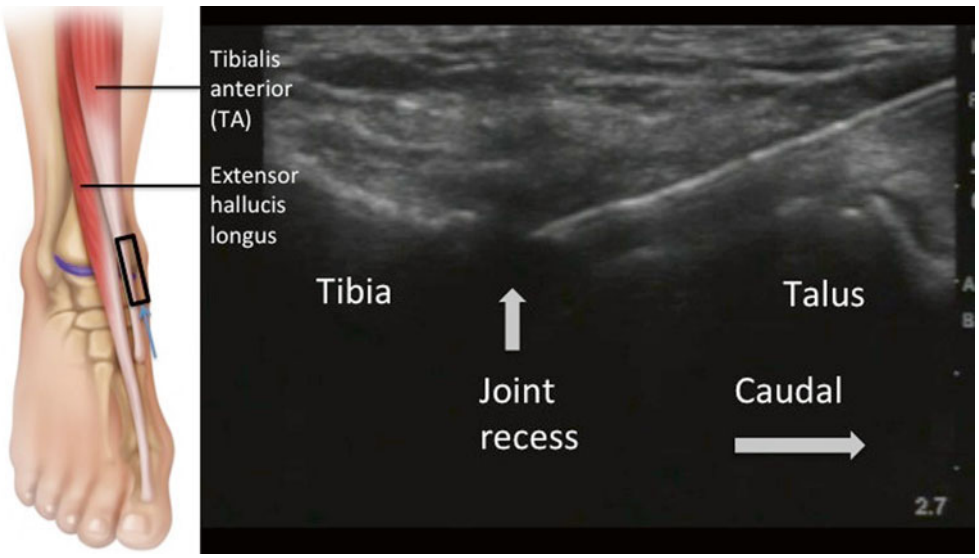


Fig. 69.11 Sonogram shows in-plane needle insertion for tibiotalar joint. The insert shows the anatomy and the ultrasound probe position (Reproduced with permission from Philip Peng Educational Series)

For subtalar joint, the approach used is lateral approach to the posterior subtalar joint. Once the subtalar joint is visualized, a 1.5-in. 25-G needle in inserted out-of-plane from posterior to anterior direction (Figs. 69.12 and 69.13). A total volume of 1–2 mL of either a mixture of local anesthetic with steroid (2 % lidocaine and 20 mg depoMedrol) or a commercial available hyaluronic acid solution is sufficient.

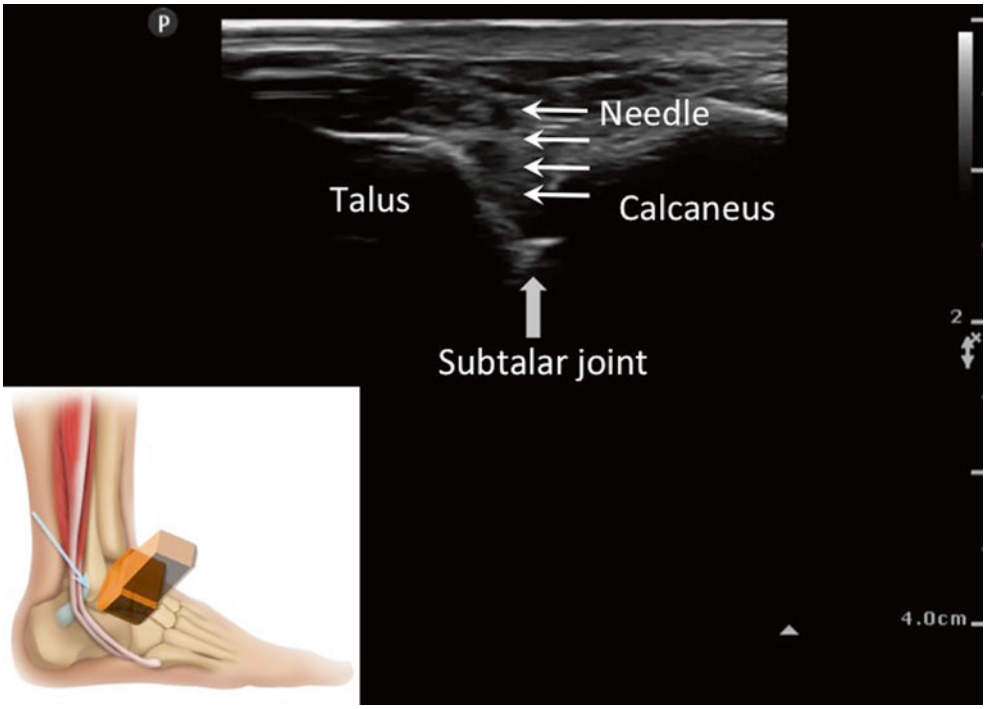


Fig. 69.12 Sonogram shows out-of-plane needle insertion for subtalar joint. The insert shows the anatomy and the ultrasound probe position. The *line arrows* indicate the needle path (Reproduced with permission from Philip Peng Educational Series)

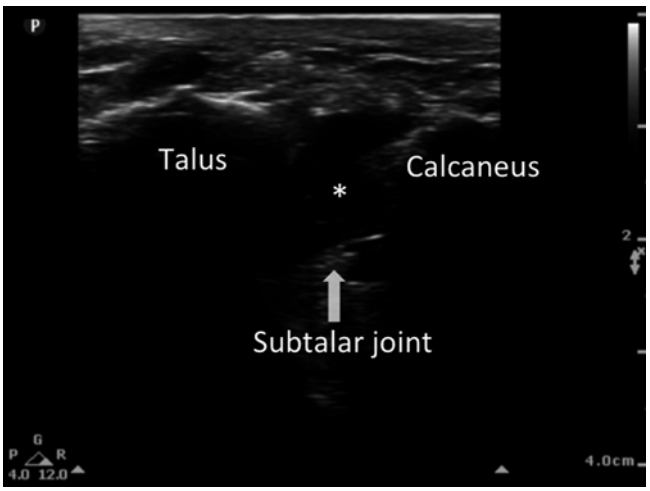


Fig. 69.13 Sonogram shows the pooling of injectate (*) following injection (Reproduced with permission from Philip Peng Educational Series)

Side Effects and Complications

- Infection (prophylaxis: extremely strict asepsis).
- Injury to cartilage or bone.
- A temporary increase in pain may occur in approximately 25 % of the patients (who should be informed about this).
- Hematoma formation.

References

1. Jankovic D. Oberes und unteres Sprunggelenk-Intraartikuläre Injektionen. In Jankovic: Regionalblockaden & Infiltrationstherapie. Berlin: ABW-Verlag; 2008. p. 440–5.
2. Netter FH. Ankle-anatomy. In: Firbas H, Kaplan F, hrsg. Farbatlanten der Medizin (Band7) Bewegungsapparat I. Anatomie. Stuttgart/New York: Thieme; 1992.
3. Thiel W. Photographischer Atlas der Praktischen Anatomie (2.Auflage). Berlin, Heidelberg, New York, Hongkong, Tokio: Thiel-Springer Verlag; 2003.
4. Pellegrini R, Schmitz H, Zohmann A. Schmerzzbehandlung mit Xyloneural (3.Auflage). Wiena: Uhlen Verlag Wien; 1996.
5. Tilscher H, Eder M. Infiltrationstherapie (3.Auflage). Stuttgart: Hippocrates; 1996.
6. Villarubis JM. Handbuch der Infiltrationen im Bewegungsapparat und bei Sportverletzungen. München: Unas Verlag. U. Bayer; 1989.
7. Cunnington J, Marshall N, Hide G, Bracewell C, Isaacs J, Platt P, Kane D. A randomized, double-blind, controlled study of ultrasound-guided corticosteroid injection into the joint of patients with inflammatory arthritis. Arthritis Rheum. 2010;62(7):1862–9.
8. Gilliland CA, Salazar LD, Borchers JR. Ultrasound versus anatomic guidance for intra-articular and periarticular injection: a systematic review. Phys Sportsmed. 2011;39:121–13.
9. Wisniewski SJ, Smith J, Patterson DG, Carmichael SW, Pawlina W. Ultrasound-guided versus nonguided tibiotalar joint and sinus tarsi injections: a cadaveric study. PM R. 2010;2:277–81.
10. Smith J, Finnoff JT, Henning PT, Turner NS. Accuracy of sonographically guided posterior subtalar joint injections. Comparison of 3 techniques. J Ultrasound Med. 2009;28:1549–57.
11. Candler SA, Hurdle MFB, Peng PW. Hip. In: Peng PWH, editor. Ultrasound for pain medicine intervention. A practical guide. Volume 3. Musculoskeletal pain. Philip Peng educational series. 1st ed. iBook, CA: Apple Inc.; 2014. p. 105–13.

Chapter 70

Metatarsophalangeal Joints and Morton's Neuroma Injections

Danilo Jankovic, MD (✉)
Director of Pain Management Centre DGS – Cologne – Huerth, Luxemburger Str. 323-325, 50354 Cologne – Huerth, Germany
e-mail: danilo@jankovic1.de

Philip W.H. Peng, MBBS, FRCPC
McL 2-405 Toronto Western Hospital, 399 Bathurst Street, Toronto, ON, Canada M5T 2S8
e-mail: philip.peng@uhn.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_70, © Springer International Publishing Switzerland 2015

Intra-articular Injection into the Hallux Metatarsophalangeal Joint

Anatomy [1–3]

The metatarsophalangeal and interphalangeal joints in the foot are divided into the basal, medial, and terminal joints. The basal joints are ball-and-socket joints in shape, and their functioning is restricted by collateral ligaments. The medial and terminal joints have the shape of pure hinge joints (ginglymus, cylindric joint) (Fig. 70.1). The metatarso-phalangeal joint connects to metatarsosesamoid articulations, which can be the source of hallucal sesamoid pain [4].

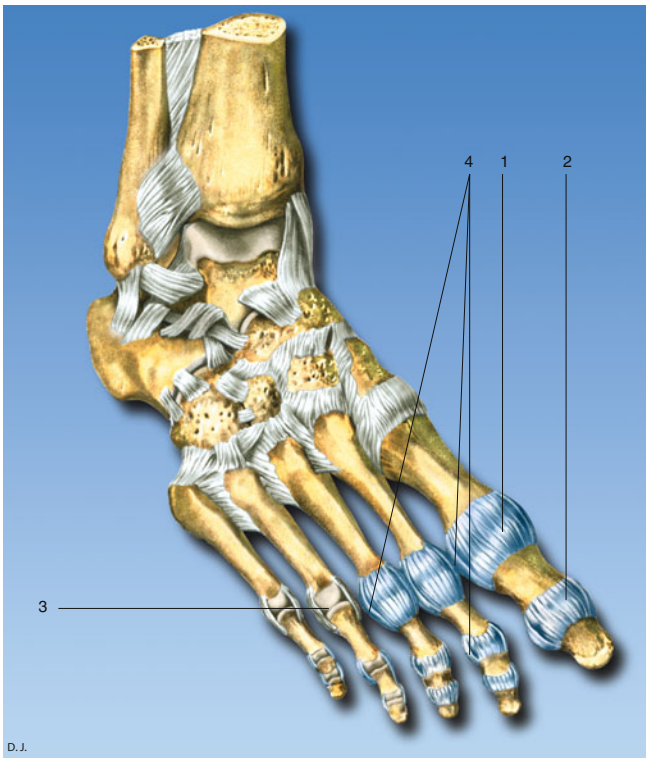


Fig. 70.1 (*1*) Joint capsule of the metatarsophalangeal joint, (*2*) capsule of the articulatio hallucis (*3*) metatarsophalangeal joint,(*4*) collateral ligaments (Reproduced with permission from Dr. Danilo Jankovic)

Indications

- Rheumatology (rheumatoid arthritis, gouty arthritis)
- Orthopedics (hallux rigidus, hallux valgus, pain in the metatarsophalangeal joint of the hallux, hallucal sesamoid pain)

Procedure

Patient Positioning

Supine position with flexed knee joint, or sitting (with the foot placed on a small footstool).

Materials

Sterile precautions, fine 25-mm-long 26-G needle, swabs, compresses, tuberculin syringe, local anesthetic, glucocorticoid if needed

Strict Asepsis

Thorough and broad skin disinfection, drying and covering of the injection site with a fenestrated drape. Local anesthesia (skin infiltration)

Injection Techniques

Landmark Based [1, 5, 6]

The articular space can be palpated during movement of the large toe (Fig. 70.2). The needle is introduced horizontal to the surface of the skin between the head of metatarsal I and the base of the proximal phalanx of the great toe (Fig. 70.2). The depth of injection is 0.5–1.0 cm. The injection must only be carried out when there is no resistance.

Ultrasound Guidance

The accuracy of landmark-guided technique is at best 65 %, while the accuracy for ultrasound guidance is 100 % [4, 7–9]. A linear probe with a small footprint (6–13 MHz) is used and applied to the dorsal aspect of metatarsophalangeal joint just medial to the extensor hallucis longus tendon (Fig. 70.3). The needle is introduced out-of-plane from medial to lateral [4]. A successful injection will result in spread of the injectate to the first metatarsophalangeal joint recess.

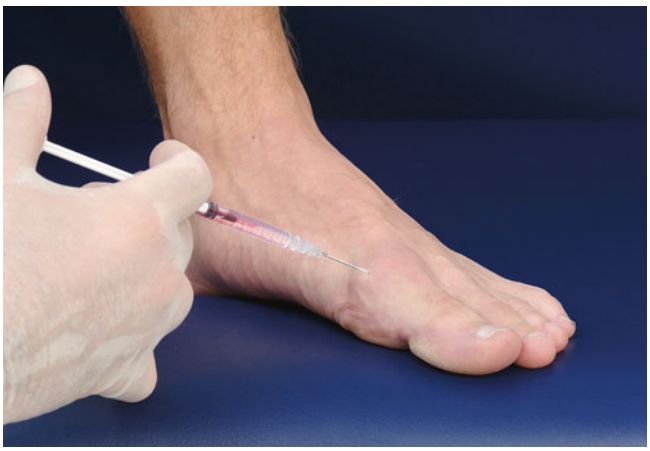


Fig. 70.2 Metatarsophalangeal joint. Injection. The needle is introduced horizontal to the surface of the skin between the head of metatarsal I and the base of the proximal phalanx of the great toe (Reproduced with permission from Dr. Danilo Jankovic)

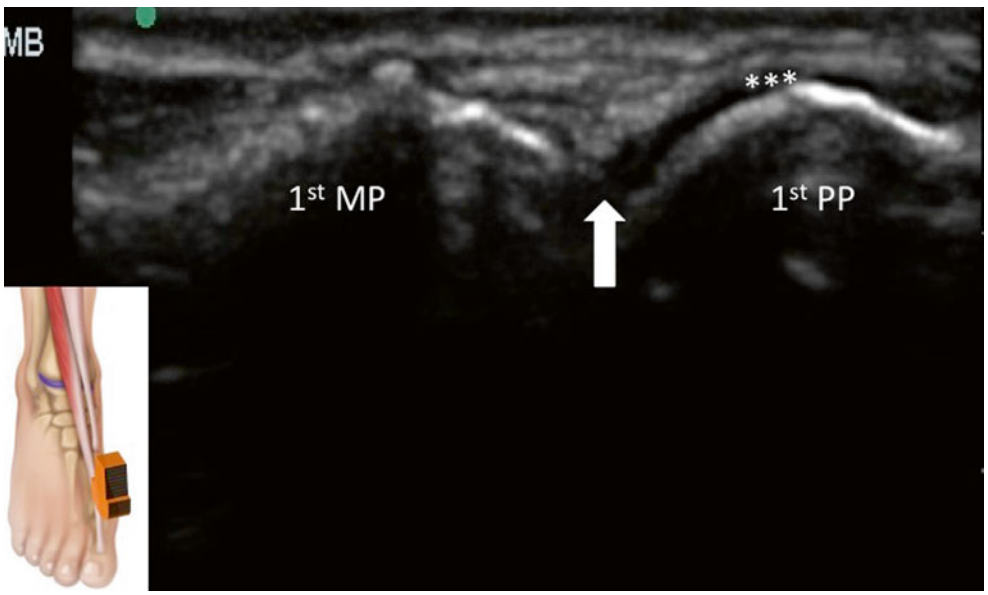


Fig. 70.3 Sonography of the long axis view of the first metatarsophalangeal joint (MTPJ). The ultrasound probe position was indicated in the insert in the left lower corner. The MTPJ was indicated by the *bold arrow*. *1st PP* first proximal phalanx, *1st MT* first metatarsal, ** metatarsophalangeal joint recess (Reproduced with permission from Philip Peng Educational Series)

Dosage

1.0–1.5 mL local anesthetic — e.g., 0.5–0.75 % ropivacaine (mixed with 20 mg methylprednisolone if appropriate)

Side Effects and Complications

- Infection (prophylaxis: strictest possible asepsis)
- Injury to cartilage or bone
- A temporary increase in pain may occur in approximately 25 % of the patients (who should be informed about this
- Hematoma formation

Injections of the Morton's Neuroma

In *1935, Dudley Morton* described two structural variants of the foot skeleton that are regularly found either individually or together in patients who report metatarsalgia [6, 10–12].

- The most frequent variant was hypermobility of metatarsal I (in the tarsometatarsal joint) with simultaneously slack long plantar ligaments. The hypermobility of *metatarsal I* overstrains the *tibialis posterior* and *flexor digitorum longus* muscles.
- The second variant involved a metatarsal I that was relatively too short (occurring in approximately 40 % of the population). This leads to overstraining of the *peroneus longus* and more rarely of the peroneus brevis muscles [6, 10].

Metatarsalgia is not an anatomic diagnosis. Primary metatarsalgia can be triggered by the following factors: static, congenital, hallux valgus, or surgical procedures. Secondary metatarsalgia can be provoked by trauma, sesamoiditis, or neurogenic diseases [6, 10].

Morton's neuroma is a pain syndrome in the sole of the foot due to pressure injury or formation of a fusiform pseudoneuroma on a *digital nerve* (sensory branch of the tibial nerve) in the region of the second, third, or fourth interdigital space (Fig. 70.4). This involves neuralgiform, often burning pain on the sole of the foot, usually in the region of the head of the third and fourth metatarsal bone and in the corresponding two toes — initially during walking and later also at rest. In addition, there is often local pressure and compression pain in the corresponding bones. Physical examination shows a click when the metatarsal heads are squeezed together (Mulder's sign) [13, 14]. *Muscular dysfunction* due to a Morton anomaly in the foot can affect the vastus medialis, gluteus medius, and gluteus minimus in addition to the fibularis muscles [6, 10–12].

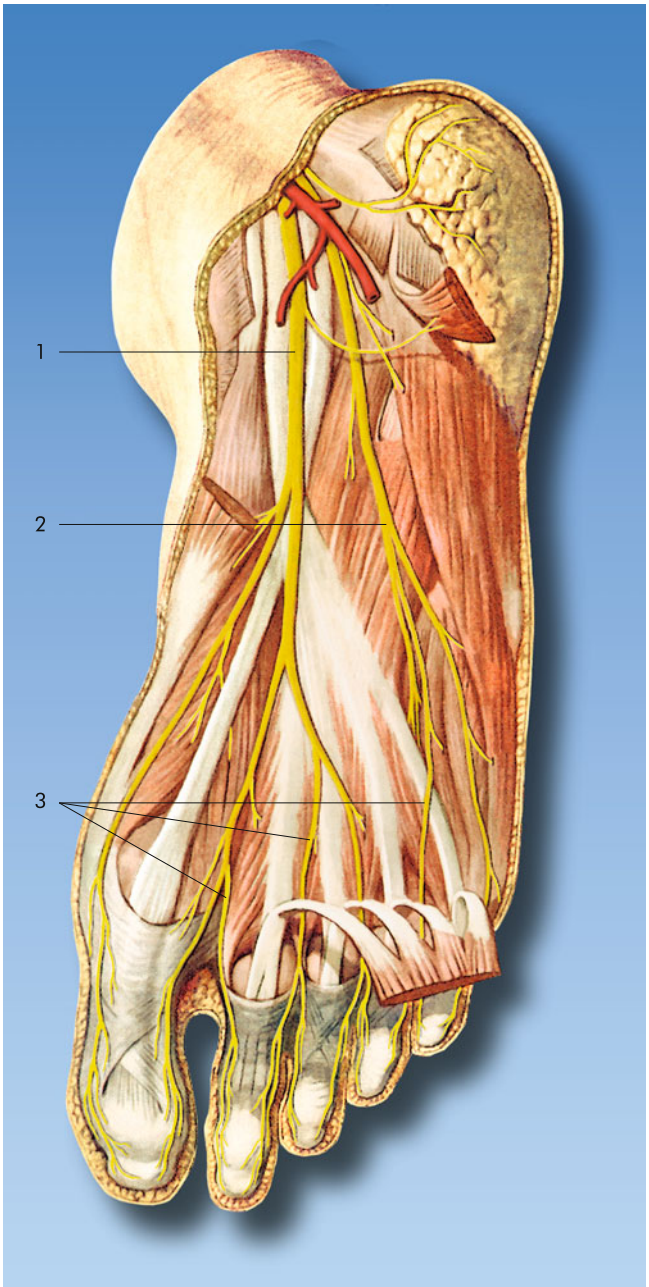


Fig. 70.4 Sole of the foot (tibial nerve). (*1*) Medial plantar nerve, (*2*) lateral plantar nerve, (*3*) joint capsule of the metatarsophalangeal joint with connecting plantar digital nerves (Reproduced with permission from Danilo Jankovic)

Procedure

Patient Positioning

Supine position with knee extended and the foot is supported with a pillow [13, 15, 16].

Materials

Sterile precautions, fine 25-mm long 26-G needle, swabs, compresses, 3 mL or tuberculin syringe, local anesthetic, glucocorticoid, or neurolytic agents as necessary (phenol or alcohol) [17]

Strict Asepsis

Thorough and broad skin disinfection, drying and covering of the injection site with a fenestrated drape. Local anesthesia (skin infiltration)

Injection Technique

A linear probe (6–13 MHz) is applied to the metatarsal heads over the lateral aspect of the foot to reveal the 4th and 5th metatarsal heads. The probe is then moved to the medial direction with the 3rd and 4th metatarsal heads in the center of the screen. With the assistant squeezing the metatarsal, the neuroma will appear as a hypoechoic structures (Fig. 70.5). The needle is inserted as out-of-plane to deposit the 1–2 mL of local anesthetic and steroids around the neuroma (Fig. 70.6). If an intraneural phenolysis is performed, the needle is inserted into the neuroma and a small amount (increment of 0.1 mL up to 0.4 mL) neurolytic agent can be injected. Real-time ultrasound scanning is important to avoid the spilling of the agents to the surrounding tissue.

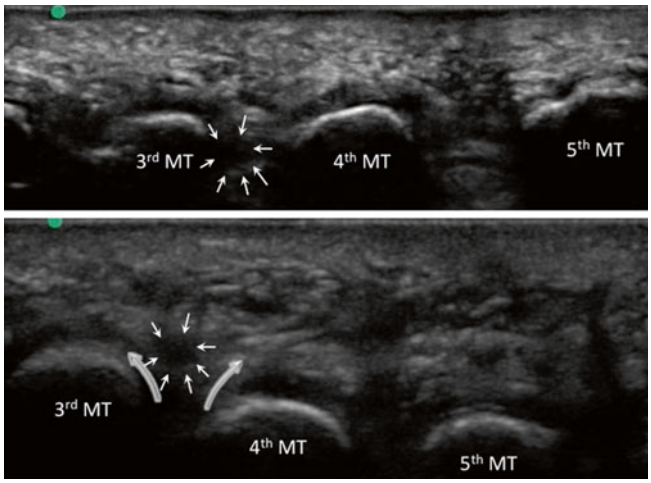


Fig. 70.5 Sonography of the Morton's neuroma. Short axis view of the forefoot when the ultrasound probe was applied over the metatarsal heads on the plantar surface of the foot. The upper figure showed the neuroma (*line arrows*) as a hypoechoic structure between the 3rd and 4th metatarsals (MT) at the resting state. On squeezing the metatarsal heads together (Mulder test), the neuroma was squeezed out of the metatarsal heads (Reproduced with permission from Philip Peng Educational Series)

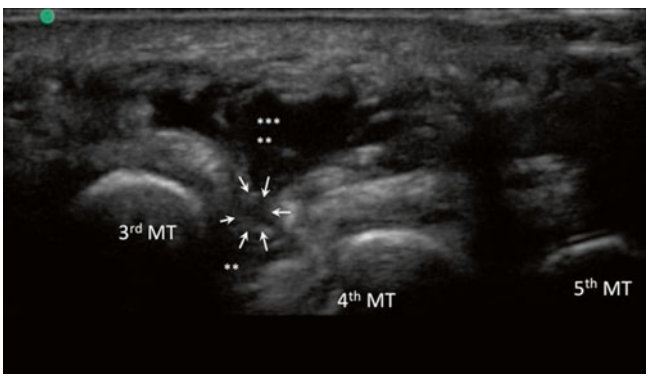


Fig. 70.6 Sonography of the Morton's neuroma (*line arrows*) following the hydrodissection with local anesthetic (**). The picture is the short axis view of the forefoot when the ultrasound probe was applied over the metatarsal heads on the plantar surface of foot. MT-metatarsal (Reproduced with permission from Philip Peng Educational Series)

Dosage

1–2 mL e.g., 0.5–0.75 % ropivacaine, mixed with methylprednisolone 20 mg

0.2–0.4 mL for neurolytic agent

Complications

See above.

If neurolytic agent is injected, the potential risk is increase in pain locally due to the spilling of agent.

References

1. Jankovic D. Großzehegrundgelenk, Metatarsophalangealgelenke (Metatarsalgie)intraartikuläre Injektionen. In Jankovic: Regionalblockaden & Infiltrationstherapie. Berlin: ABW-Verlag; 2008. p. 446–8.
2. Netter FH. Anatomy. In: Firbas H, Kaplan F, hrsg. Farbatlanten der Medizin (Band7) Bewegungsapparat I. Anatomie. New York: Thieme Stuttgart; 1992.
3. Thiel W. Photographischer Atlas der Praktischen Anatomie (2.Auflage). Berlin, Heidelberg, New York, Hongkong, Tokio: Thiel-Springer Verlag; 2003.
4. Wempe MK, Sellon JL, Sayeed YA, Smith J. Feasibility of first metatarsophalangeal joint injections for sesamoid disorders: a cadaveric investigation. PM R. 2012;4:556–60.
5. Tilscher H, Eder M. Infiltrationstherapie (3.Auflage). Stuttgart: Hippocrates; 1996.
6. Travel JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual, vol. 2. Baltimore: Williams & Wilkins; 1992.
7. Reach JS, Easley ME, Chuckpaiwong B, Nunley JA. Accuracy of ultrasound guided injections in the foot and ankle. Foot Ankle Int. 2009;30:239–42.
8. Rogers CJ, Cianca J. Musculoskeletal ultrasound of the ankle and foot. Phys Med Rehabil Clin N Am. 2010;21:549–57.
9. Koski JM, Hermunen HS, Kilponen VM, Saarakkala SJ, Hakulinen UK, Heikkinen JO. Verification of palpation-guided intra-articular injections using glucocorticoid-air-saline mixture and ultrasound imaging (GAS-graphy). Clin Exp Rheumatol. 2006;24:247–52.
10. Calliet R. Foot and ankle pain. 2nd ed. Philadelphia: F.A. Davis Company; 1983.
11. Morton DJ. The human foot. Its evolution, physiology and functional disorders. New York: Columbia University Press; 1935.
12. Mumenthaler M. Läsionen peripherer Nerven. Morton-Neuralgie. In: Mumenthaler M, editor. Neurologie (7.Auflage). New York: Georg Thieme, Stuttgart; 1982.
13. Torriani M, Kattapuram SV. Dynamic sonography of the forefoot: the sonographic Mulder sign. AJR Am J Roentgenol. 2003;180:1121–3.
14. Lee KS. Musculoskeletal ultrasound: how to evaluate for Morton's neuroma. AJR Am J Roentgenol. 2009;193:W172.
15. Quinn TJ, Jacobson JA, Craig JG, van Holsbeeck MT. Sonography of Morton's neuromas. AJR Am J Roentgenol. 2000;174:1723–8.
16. Yap LP, McNally E. Patient's assessment of discomfort during ultrasound-guided injection of Morton's neuroma: selecting the optimal approach. J Clin Ultrasound. 2012;40:330–4.
17. Hughes RJ, Ali K, Jones H, Kendall S, Connell DA. Treatment of Morton's neuroma with alcohol injection under sonographic guidance: follow-up of 101 cases. AJR Am J Roentgenol. 2007;186:1535–9.

Part XII

Regional Block for Children

Chapter 71

Pediatric Peripheral Nerve Block: Upper Limb

Sara S.K. Cheung, MBBS, FHKCA, FHKAM
Department of Anaesthesia, Pamela Youde Nethersole Eastern Hospital, Hong Kong, China

University of Alberta, Edmonton, AB, Canada
e-mail: cheungsk3@gmail.com

Heather Y.Z. Ting, MD, FRCPC
Department of Anesthesiology and Pain Medicine, University of Alberta, Edmonton, AB, Canada
e-mail: yizhenhe@ualberta.ca

Ban C.H. Tsui, MSc, MD, FRCPC (✉)
Department of Anesthesiology and Pain Medicine, University of Alberta, Edmonton, AB, Canada

University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada
e-mail: btsui@ualberta.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_71,

Background

The brachial plexus (Fig. 71.1) is derived from the ventral rami of C5–T1 (with occasional contributions from C4 and T2). After leaving the intervertebral foramina, the roots travel between the anterior and middle scalene muscles, where they become three trunks (upper, middle, and lower). The trunks then emerge from the scalene muscles and lay cephaloposterior to the subclavian artery as they cross the base of the posterior triangle of neck. Each trunk forms anterior and posterior divisions as they pass over the lateral border of the first rib. At the apex of axilla, these divisions unite to form three cords, which are named according to their relative positions to the axillary artery (lateral, medial, and posterior). After leaving the axilla, the cords give rise to terminal branches which supply the muscles and skin of the upper limb.

In children, upper limb blocks are performed under general anesthesia or heavy sedation, although in cooperative and older children, it may be possible to perform these blocks awake or under light sedation. Since monitoring paresthesia is precluded by general anesthesia/heavy sedation or the child is too young to verbalize paresthesia, the use of nerve stimulation is essential for localization of nerve and avoiding intraneural injection. In addition, ultrasound allows direct visualization of the needle and neural structures and has further improved the success rate and safety of regional blocks in children.

Several approaches for brachial plexus block have been described, namely, interscalene, supraclavicular, infraclavicular, and axillary. Blockade of a single peripheral nerve is possible distal to the axilla. Different approaches should be used depending on the site of the surgery. For example, interscalene block provides the best coverage for shoulder surgery, while more distal techniques are indicated for surgery of the forearm and hand. General complications of brachial plexus block include infection at the needle insertion site, bleeding/hematoma formation, nerve injury, and local anesthetic toxicity. For more proximal blocks (e.g., interscalene), specific complications, such as phrenic nerve palsy, Horner's syndrome, recurrent laryngeal nerve palsy, epidural/intrathecal injection, and vertebral artery injection can occur. Pneumothorax is also a potential complication with supraclavicular and infraclavicular blocks; however, the increasing use of ultrasound has lowered the risk of these complications significantly.

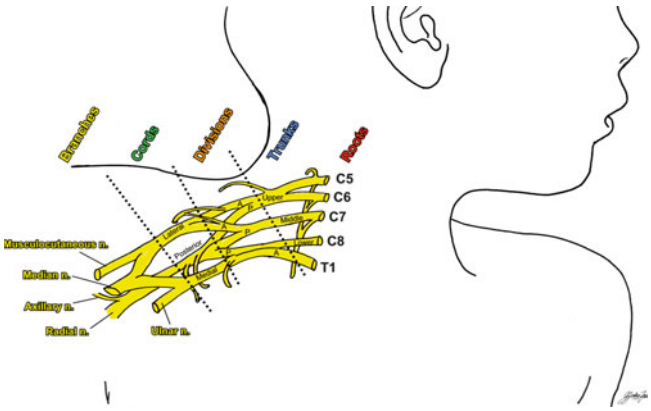


Fig. 71.1 Anatomy of the brachial plexus. *A* anterior divisions, *P* posterior divisions

Interscalene Block

Introduction, Indications, and Complications

The interscalene block targets the roots and proximal trunks of the brachial plexus between the anterior and middle scalene muscles at the level of the cricoid cartilage (C6) (Fig. 71.2). It is indicated for surgery of the shoulder and upper arm since it reliably anesthetizes the axillary and musculocutaneous nerves, although the hand and fingers are often spared. Phrenic nerve block is a major complication in infants and young children due to greater dependence on diaphragmatic function. Other risks include vertebral artery puncture and injection, epidural or intrathecal injection, Horner's syndrome, recurrent laryngeal nerve blockade, and hematoma formation in the neck due to arterial or venous puncture. A combined nerve stimulation and ultrasound-guided approach should be used to minimize complications.

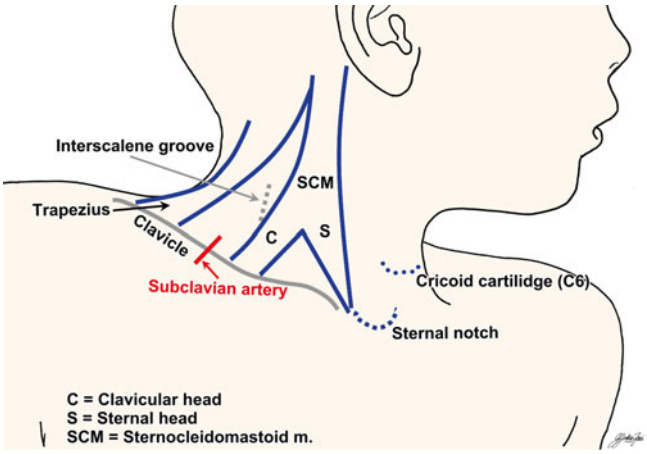


Fig. 71.2 Patient positioning and surface landmarks for interscalene brachial plexus block

Patient Positioning, Preparation, Equipment, and Dosage

The patient is placed with the head turned approximately 45° away from the side to be blocked. For parascalene block, a rolled towel is placed underneath the shoulders. The skin is cleaned with antiseptic solution. If ultrasound guidance is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G insulated short-beveled needle is used. Recommended local anesthetics are 0.125–0.25 % bupivacaine or 0.1–0.2 % ropivacaine. Using 0.25 % bupivacaine at 0.2–0.3 mL/kg is a reasonable option for this block. The duration of sensory block averages 15 ± 4.5 h, independent of the type and concentration of the local anesthetic used. Epinephrine 1:200,000 may be added to detect intravascular injection.

Nerve Stimulation Technique

Different approaches have been described for this block. For interscalene approach, the point of needle insertion is at the level of cricoid cartilage (C6) within the interscalene groove (Fig. 71.2). The interscalene groove is located between the anterior and middle scalene muscles posterior to the lateral border of the sternocleidomastoid muscle (SCM), slightly above the point where the sternal and clavicular heads of the SCM separate. The line between the point of needle insertion and the cricoid cartilage should cross the Chassaignac's tubercle (anterior tubercle of the transverse process of the C6 vertebra), which can be felt easily. The needle is inserted at 60° to skin and directed medially, posteriorly, and caudally to prevent inadvertent puncture of the vertebral artery or epidural/intrathecal space. The initial current is set at 0.8–1.0 mA (2 Hz, 100–300 μs) and then gradually reduced to a threshold current of 0.2–0.4 mA (0.1–0.2 ms) after eliciting appropriate motor responses. Response at a current >0.4 mA indicates that the needle is too far away from the plexus; a current ≤0.2 mA signifies intraneural placement.

Another approach to this block is the parascalene approach [1]. A line is drawn between the midpoint of clavicle and the Chassaignac's tubercle. The needle is inserted two thirds of the way down this line and advanced posteriorly until twitches are seen. The risk of vascular puncture, Horner's syndrome, and phrenic nerve block is lower with this technique, but the external jugular vein may be penetrated.

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) hockey stick or linear transducer probe is suitable for this block. The probe is placed on the neck in an axial oblique view at the level of the cricoid cartilage (C6) (Fig. 71.3). The anechoic great vessels (common carotid artery and internal jugular vein) and the overlying triangular-shaped sternocleidomastoid muscle are identified first. If necessary, color Doppler can be used to locate the vessels. Move the probe proximally and distally to identify the roots/trunks of the brachial plexus (which commonly seen as three round or oval-shaped hypoechoic structures) in the interscalene groove between the anterior and middle scalene muscles. Occasionally, the vertebral artery can be seen deep to the plexus and anterior to the C6 transverse process. Extra caution should be exercised not to confuse the artery with a nerve and inject into it. Visualization of the neural structures can be difficult in small children, so a "traceback" approach is recommended. In the traceback approach, the probe is placed in a coronal oblique plane at the upper border of the clavicle. The brachial plexus at this point appears as "a bunch of grapes," superolateral to the subclavian artery. The plexus is then traced back to the interscalene region by scanning in a cephalad direction (Fig. 71.4).

The needle is inserted either in-plane or out-of-plane, although the in-plane approach is preferred to ensure visualization of the needle, thereby minimizing the risk of complications. For the in-plane approach, the needle is inserted in a lateral-to-medial fashion into the interscalene groove. When using the out-of-plane approach, the plexus is centered in the middle of the screen, and the needle is inserted cranial to the probe at the midline. Direct the needle tip, which appears as bright dot on the screen, in a "walk-down" manner (see Ref. [1]) in proximity to the nerves. Nerve structures can be confirmed by nerve stimulation. A test dose with D5W is useful to visualize the spread and confirm nerve localization. After negative aspiration for blood or CSF, local anesthetic is deposited to achieve good spread surrounding the nerves within the interscalene groove. The depth should be less than 1–2 cm, even in teenage adolescents.

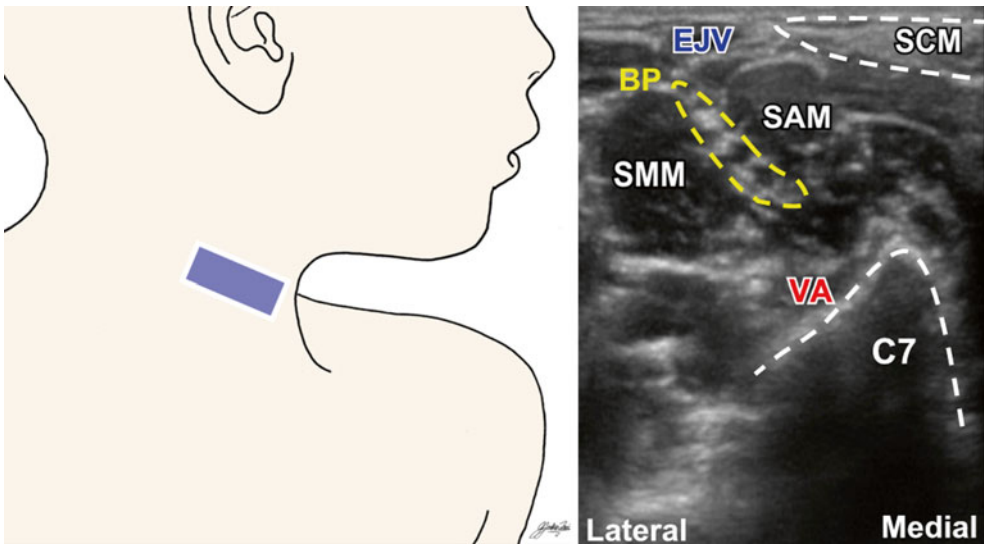
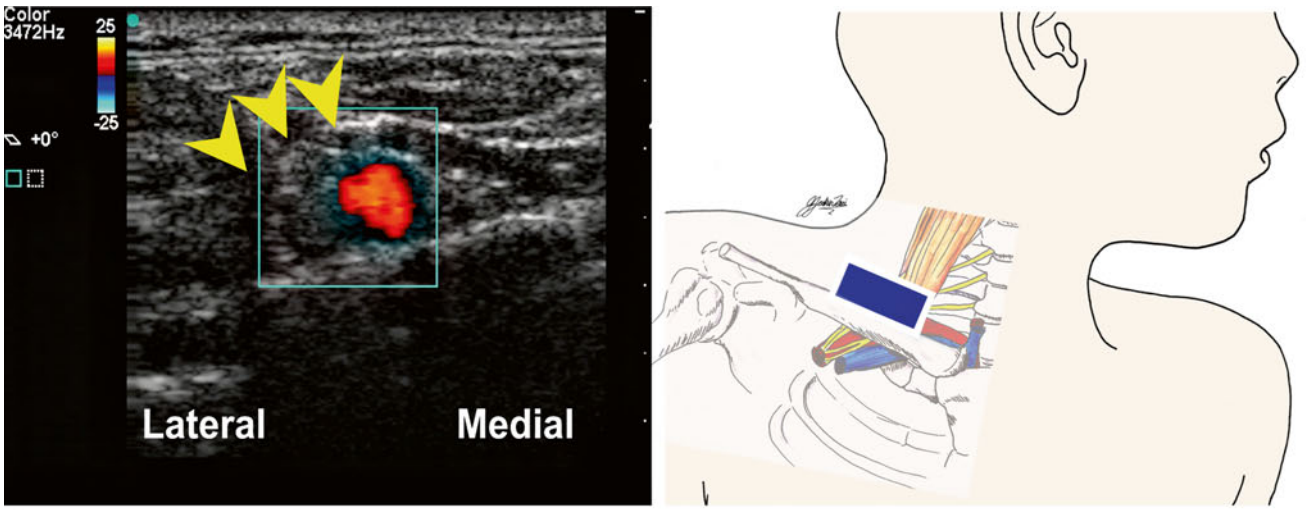
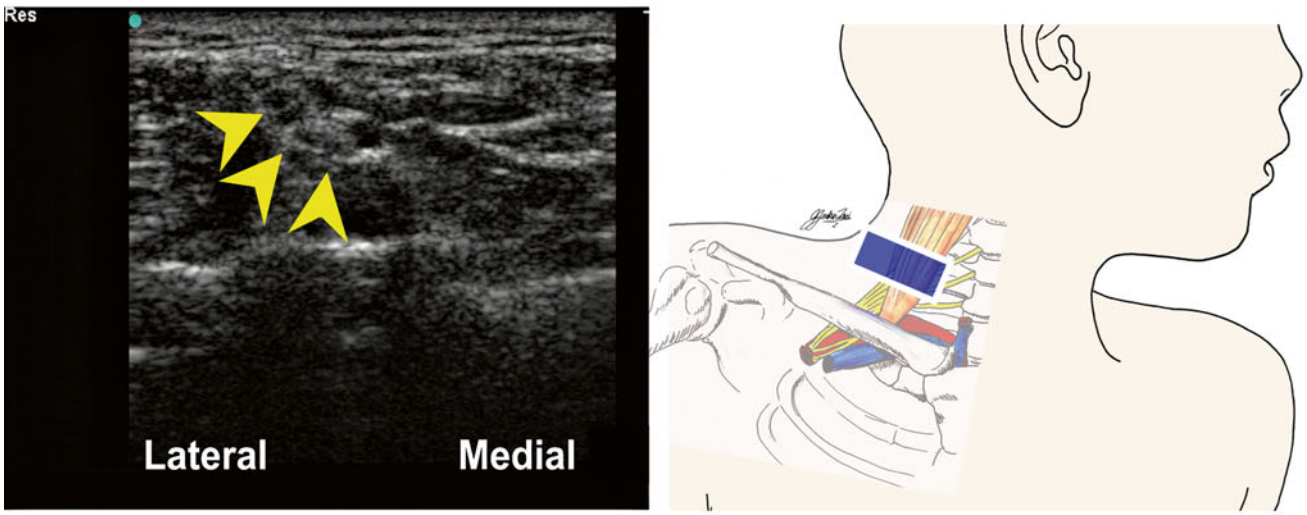


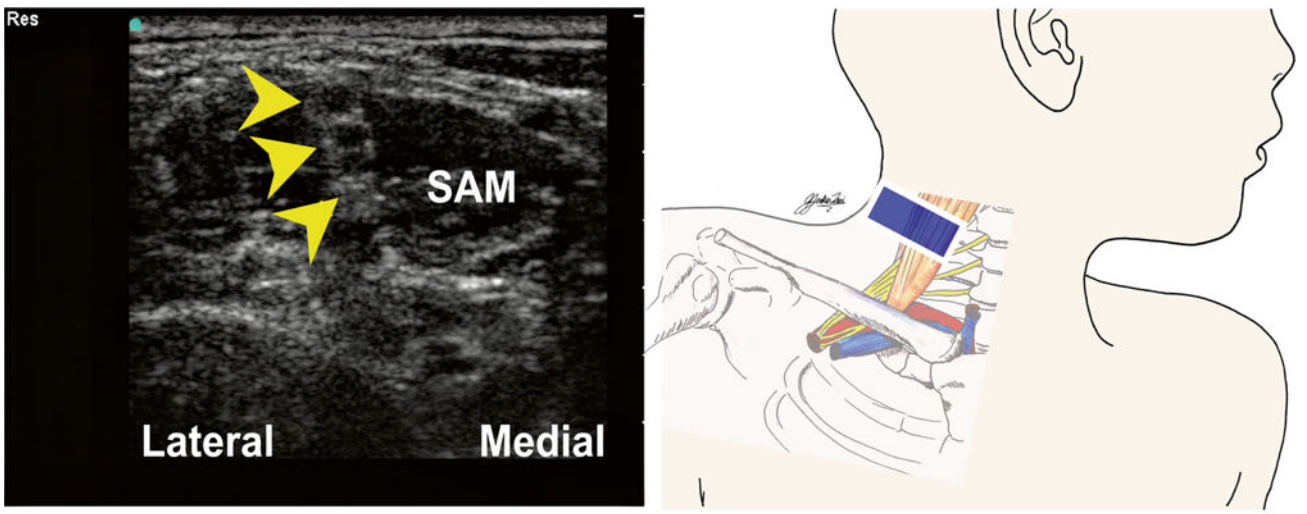
Fig. 71.3 Ultrasound image of the brachial plexus at the interscalene groove. *EJV* external jugular vein, *SCM* sternocleidomastoid muscle *SAM* scalenus anterior muscle *SMM* scalenus medius muscle, *VA* vertebral artery, *BP* brachial plexus, *C7* C7 vertebra



1. Locate the subclavian artery



2. Place the artery and plexus trunks/divisions centrally and track upward



3. Tilt the probe upward and downward to obtain the best view

Fig. 71.4 Ultrasound traceback approach for interscalene block. *SAM* scalenus anterior muscle; arrowheads show location of brachial plexus

Supraclavicular Block

Introduction, Indications, and Complications

This block targets the trunks and/or divisions of the brachial plexus where they are located cephaloposterior to the subclavian artery above the first rib (Fig. 71.5). The rapid onset of this block offers the most reliable blockade of the brachial plexus for anesthesia and analgesia of the entire upper extremity, especially the elbow, forearm, and hand. Because of the high risk of pneumothorax due to the proximity of the apex of the lung to the brachial plexus, an ultrasound-guided approach is strongly recommended.

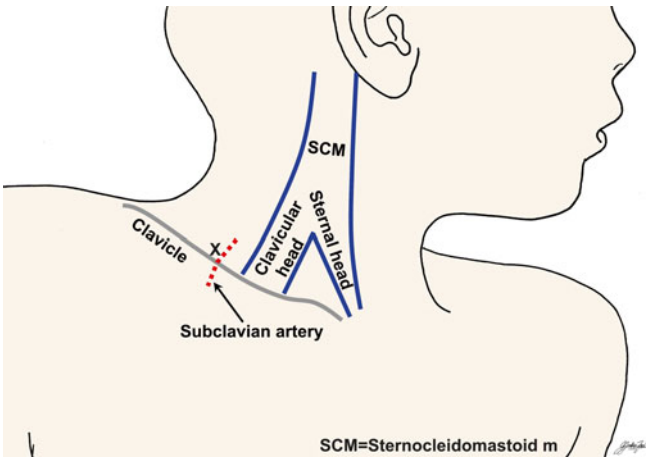


Fig. 71.5 Patient positioning and surface landmarks for supraclavicular brachial plexus block. *X* indicates the point of needle entry

Patient Positioning, Preparation, Equipment, and Dosage

The head of the patient is turned to the contralateral side. The arm is placed on the side, and the shoulder is pushed backward on to the mattress and down toward the feet (Fig. 71.5). The skin is cleaned with antiseptic solution. If the ultrasound approach is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G insulated needle is used. Depth of insertion is related to the age and weight of the patient in a nonlinear manner. For a 10-kg child, the depth of insertion is about 10 mm. For every 10 kg increase in weight, the depth of insertion increases 3 mm until the child reaches 50 kg. After that, advance 1 mm for every 10 kg increase in weight. The maximum depth should not exceed 35 mm. The required depth of penetration is usually less than 1 cm for children and 1–2 cm for teenagers.

Recommended local anesthetics are 0.25–0.5 % bupivacaine, 0.2 % ropivacaine, and 2 % lidocaine. Blockade at this level can be achieved with volumes as low as 0.15–0.2 mL/ kg.

Nerve Stimulation Technique

The needle insertion point is located 1 cm above the midpoint of the clavicle posterolateral to the subclavian artery. The subclavian artery pulsation serves as the landmark for localization of the plexus. The current is initially set at 0.8 mA (2 Hz, 100–300 μs) and then gradually reduced to a threshold current of 0.2–0.4 mA (0.1–0.2 ms) after obtaining appropriate response. Motor response at a current $\leq$0.2 mA indicates intraneural placement, and the needle should be withdrawn. The spread of local anesthetic solution in children may be greater than for adults since, in children, the fascia adheres less to the nerve trunks. This increases the likelihood of a successful block with any motor response.

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) hockey stick probe is ideal for small children. For older and/or obese children, a small footprint curved array probe (8-5 MHz) is a better option. The probe is first placed in a coronal oblique plane at the lateral end of the upper border of the clavicle. It is then moved medially until the subclavian artery is seen. The subclavian artery is anechoic, hypodense, pulsatile, and round; its identity can be confirmed by color Doppler. The plexus is located superior and lateral to the artery above the first rib and appears as a "bunch of grapes" outlined by a hyperechoic fascia sheath (Fig. 71.6). Below the artery, the first rib appears as a hyperechoic structure with a hypoechoic acoustic shadow, while the lung pleura is accompanied by a hyperechoic shadow due to air artifacts.

The needle is inserted immediately above the clavicle in a lateral-to-medial direction at a shallow angle. An in-plane approach is strongly recommended to ensure visualization of the needle tip at all times so as to minimize the risks of pneumothorax and vascular puncture. A test dose of D5W is used to visualize the spread and confirm nerve localization. Local anesthetic is first deposited into the "corner pocket" (the corner between the subclavian artery and the first rib). This way, the plexus is often lifted up and away from the pleura so as to reduce the chance of pleural puncture upon subsequent injection. The needle may then be repositioned to achieve good local anesthetic spread around the nerves within the fascia.

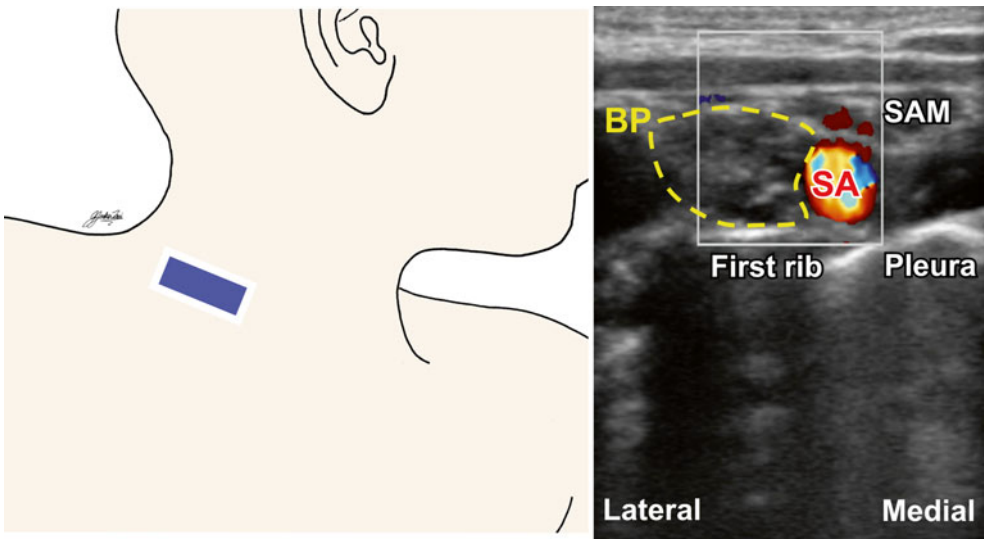


Fig. 71.6 Ultrasound image of the brachial plexus at the supraclavicular level. *BP* brachial plexus, *SAM* scalenus anterior muscle, *SA* subclavian artery

Infraclavicular Block

Introduction, Indications, and Complications

This block targets the cords of the brachial plexus where they surround the axillary artery. In this region, the three cords are arranged around the artery in the following manner: lateral cord cephalad, posterior cord posterior, and medial cord caudad. This block is indicated for surgery on the upper arm, elbow, forearm, and hand. Continuous infusion of local anesthetic via an infraclavicular catheter provides excellent postoperative analgesia for major upper limb surgery and is preferred over supraclavicular and axillary catheters because of ease of placement and securement.

Patient Positioning, Preparation, Equipment, and Dosage

A pillow is placed underneath the patient's shoulder. The elbow is flexed with the hand resting on the abdomen or with the arm resting at the side. Alternatively, the arm can be abducted and externally rotated with the elbow flexed. This maneuver has the advantage of stretching the cords and bringing them closer to the surface, enhancing the ultrasonographic appearance as well as facilitating local anesthetic spread. The skin is cleaned with antiseptic solution. If ultrasound guidance is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G needle is commonly used for this block. An insulated needle should be chosen if nerve stimulation is used. The depth of penetration is usually within 2–3 cm at a lateral location inferior to the coracoid process. Recommended local anesthetic doses are 0.5 mL/kg of 0.2–0.5 % ropivacaine or 0.25–0.5 % bupivacaine. Concentrations may need to be reduced in very small children to obtain a volume of at least 5 mL for this block.

Nerve Stimulation Technique

A lateral approach is recommended where the point of needle insertion is approximately 0.5–1 cm inferior and slightly medial to the coracoid process (Fig. 71.7). At this point, the pleura is further away so the risk of pneumothorax is lower compared to a more medial injection site. The needle is advanced in a vertical direction until distal motor response (hand or wrist flexion) is obtained. The current is initially set at 0.8 mA (2 Hz, 100–300 μs) and then gradually reduced to a threshold current of 0.2–0.4 mA. Twitching of the pectoralis muscles indicates the needle is too shallow, while bone (rib) contact means it is too deep, and appropriate needle adjustment should be made. Careful aspiration is crucial to rule out pneumothorax or arterial/venous puncture.

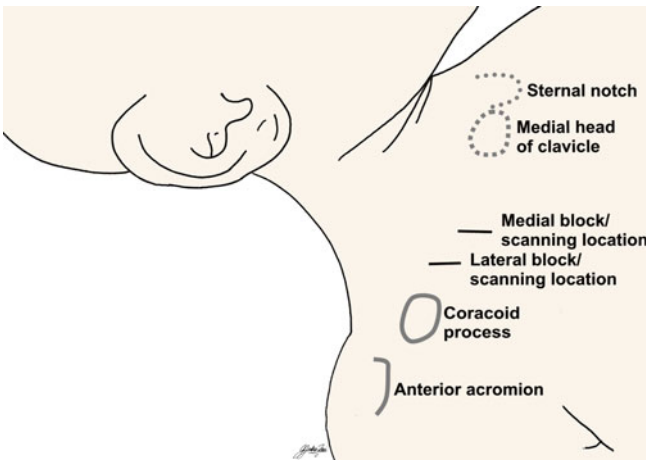


Fig. 71.7 Patient positioning and surface landmarks for infraclavicular brachial plexus block

Ultrasound-Guided Technique

A small footprint linear array transducer (13-6 MHz frequency) is ideal for young children. In older or larger children, a curved array transducer (8-5 MHz frequency) is desirable to allow greater depth of penetration.

A lateral block location is recommended. The probe is placed in a parasagittal plane below and slightly medial to the coracoid process. Scan medially and laterally to locate the axillary neurovascular bundle, which sits underneath the pectoralis major and minor muscles (Fig. 71.8). The axillary artery can be identified as a round or oval-shaped pulsatile structure. The axillary vein is almost always medial and caudad to the artery and is irregularly shaped. Color Doppler can be used to identify the vessel in cases of doubt. At this point, the cords, which are seen as hyperechoic oval structures, can be found posterolateral to the artery. The medial cord may be difficult to identify because it may be hidden between the axillary artery and vein and can be posterior or even slightly cephalad to the artery.

If a medial approach is to be used, the probe is positioned at the midpoint of the line between the anterior acromion and jugular notch. It is important to maintain the pleura and needle in view at all times during the time of needle insertion.

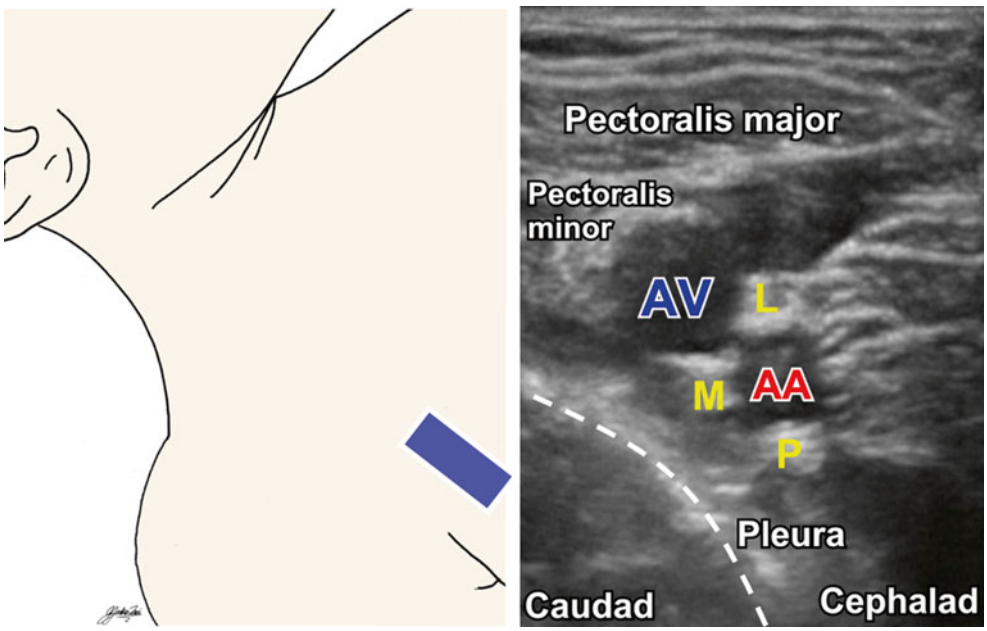


Fig. 71.8 Ultrasound image of the brachial plexus at the infraclavicular level at a lateral scanning location. *AV* axillary vein, *AA* axillary artery, *L* lateral cord, *M* medial cord, *P* posterior cord

The pleura usually appears as a hypoechoic cavity outlined by a hyperechoic line and is often located proximal to the vessels and plexus (Fig. 71.9).

Both in-plane and out-of-plane approaches can be used for this block. The in-plane approach is strongly recommended, especially when a more medial block location is chosen, because it allows visualization of the needle tip and shaft, thereby minimizing the risk of pleural puncture. The needle is inserted at the cephalad end of the probe at a 45–60° angle to the skin and advanced caudally. The needle is then directed to the posterior cord, and local anesthetic is deposited around it. This often results in a "U-shaped" spread around the artery and hence complete blockade of the plexus. If the spread is deemed inadequate, a further dose of local anesthetic is deposited as the needle is withdrawn to the lateral cord position. Another injection between the artery and the vein may be needed to ensure blockade of the medial cord. Nerve stimulation offers additional confirmation of the neural structure and is recommended. A test dose of D5W prior to injection of local anesthetic can visualize spread and confirm nerve localization.

Occasionally, the out-of-plane approach is required when there is not enough space for the in-plane needle insertion between the probe and the clavicle, especially in very small children. This approach also has the advantage of a reduced length of needle path for better patient comfort if the block is to be performed on a conscious child. A 45° angled needle insertion is used so that the distances between the needle insertion point, probe, and brachial plexus are equal (i.e., forming a right-angled triangle). The needle then "walks down" to reach the posterior cord while the needle tip is being tracked.

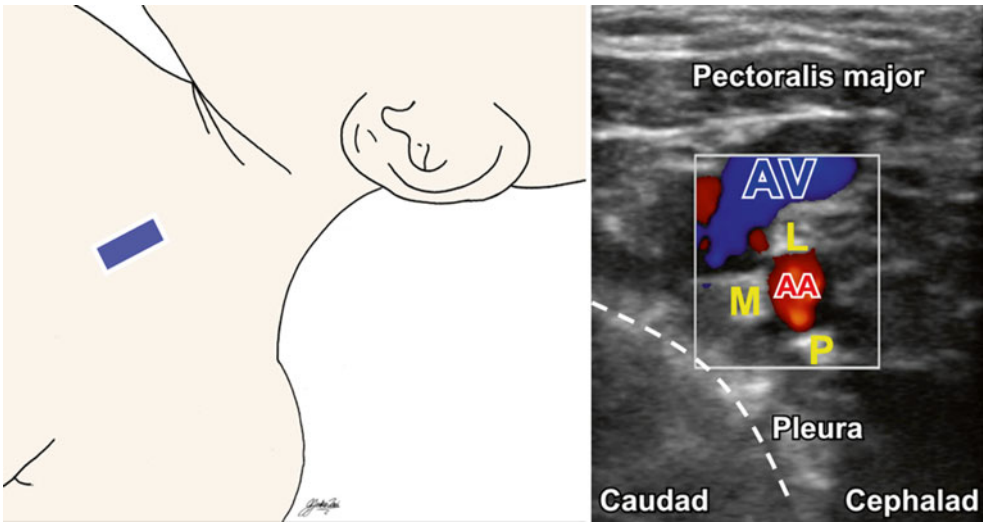


Fig. 71.9 Ultrasound image of the brachial plexus at the infraclavicular level at a medial scanning location. *AV* axillary vein, *AA* axillary artery, *L* lateral cord, *M* medial cord, *P* posterior cord

Axillary Block

Introduction, Indications, and Complications

This block targets the terminal branches of the brachial plexus (median, ulnar, radial, and musculocutaneous nerves) where they are in close relation to the axillary artery at the apex of axilla. The musculocutaneous nerve often leaves the plexus proximal to this point and travels between the biceps and coracobrachialis muscles; therefore, it must be blocked with a separate injection. Axillary block is indicated in surgery of the elbow, forearm, wrist, or hand. Traditionally, this block has been considered safer than blocks at more proximal locations because it is free from the pleura, vertebral artery, and phrenic nerve. However, hematoma formation causing nerve compression can occur occasionally in children. When performed blind, the success rate of this block is only 70–80 % [2], but the use of ultrasound, especially when combining with nerve stimulation, can improve nerve localization and hence the success rate significantly.

Patient Positioning, Preparation, Equipment, and Dosage

The arm of the patient is abducted 70–80° and externally rotated. The skin is cleaned with antiseptic solution. If ultrasound is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G insulated needle is typically used. The recommended dose of local anesthetic for an ultrasound-guided technique is 0.2–0.3 mL/kg of 0.25–0.5 % bupivacaine, ropivacaine, or levobupivacaine and 0.5–1.0 mL for the musculocutaneous nerve. Higher volumes are needed if nerve stimulation is used.

Nerve Stimulation Technique

The axillary artery is first palpated at the apex of axilla. The needle is introduced at an approximately 45° angle to the skin at the upper edge of the axillary artery, pointing cephalad toward the midpoint of clavicle (Fig. 71.10). Advance the needle until a "pop" or "give" is felt as the needle enters the neurovascular sheath. Pulsations in the needle indicate that the needle tip is in immediate vicinity of the artery. An initial current is applied at 0.8 mA (2 Hz, 100 μs) and then gradually reduced to a threshold current of 0.4 mA (0.1 ms) after obtaining a distal motor response in the hand, wrist, or forearm. Local anesthetic is deposited after careful aspiration to rule out any intra-arterial/intravenous placement. A second injection can be made at the lower edge of the artery in a similar fashion ("two-puncture technique").

To block the musculocutaneous nerve, direct the needle (using the same needle insertion site) toward the belly of the coracobrachialis muscle and inject local anesthetic. Elbow flexion can be elicited if nerve simulation is used.

If a tourniquet is required for the surgery, the intercostobrachial nerve should be blocked; this can be done by subcutaneous injection of local anesthetic across the medial surface of the upper arm.

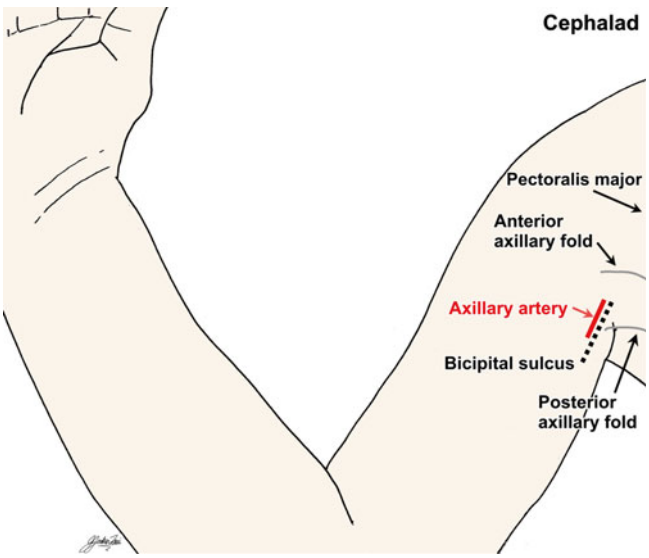


Fig. 71.10 Patient positioning and surface landmarks for axillary block of the brachial plexus

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) linear probe is suitable for this block. In small children, a small footprint ("hockey stick") probe is desirable. The probe is placed in a transverse plane along the axillary crease and scanned as proximally as possible to obtain the best transverse view of the neurovascular bundle surrounded by the biceps brachii, coracobrachialis, and triceps muscles. The anechoic axillary artery can be identified as a pulsatile circular structure, while the anechoic axillary vein(s) is irregular in shape, compressible, and usually more superficial. The nerves, which appear as round- or oval-shaped structures with a honeycomb-like appearance, are situated around the artery (Fig. 71.11). The median nerve can be found between the artery and biceps brachii muscle and is located superficial to the artery. The ulnar nerve lies between the artery and the triceps muscle and is also superficial to the artery. The radial nerve is deep to the artery at the midline and can sometimes be difficult to locate. The musculocutaneous nerve, whose appearance can vary from round or oval to flat, can usually be found in the plane between the biceps and the coracobrachialis muscles. If necessary, each of these terminal branches can be traced distally for confirmation. The use of nerve stimulation can also help identify individual nerves according to the corresponding motor responses.

The needle is inserted either in-plane or out-of-plane in relation to the probe. Commonly, multiple injections and needle redirections are required to ensure circumferential spread of local anesthetic around each individual nerve. For the in-plane approach, the needle is inserted at an acute angle (20–30°) to the skin in a superior-to-inferior direction in parallel to the long axis of the probe. The needle is initially directed underneath the artery to reach the radial nerve. It is recommended to block the radial nerve first to minimize image distortion from the spread of local anesthetic. After that, the needle is withdrawn and redirected to deposit local anesthetic around the ulnar and the median nerves. A test dose with D5W is useful to visualize the spread and confirm nerve localization prior to injection of local anesthetic. If an out-of-plane approach is used, the needle is inserted approximately 1 cm away from the midpoint of the probe at a 30–45° angle from the skin to reach the nerves.

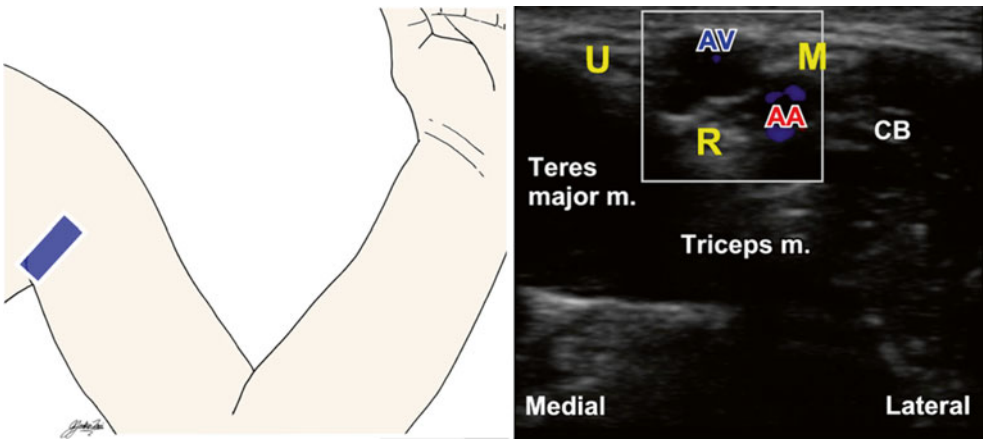


Fig. 71.11 Ultrasound image of the brachial plexus at the axilla. *AV* axillary vein, *AA* axillary artery, *CB* coracobrachialis, *U* ulnar nerve, *R* radial nerve, *M* median nerve

Distal Nerve Block of the Upper Extremity

Individual peripheral nerve blocks of the upper extremity can be used as rescue blocks to supplement incomplete brachial plexus blocks. Occasionally, they are performed to provide anesthesia for surgical procedures which involve a territory supplied by a single peripheral nerve or to prolong postoperative analgesia.

Median Nerve

Introduction, Indications, and Complications

The median nerve innervates muscles which produce flexion and opposition of the thumb, middle, and index fingers, as well as pronation and flexion of the wrist. The nerve is derived from the lateral and medial cords, carrying fibers from all roots (C5–T1). Distal to the axilla, it descends lateral to the brachial artery in the upper arm. It then crosses over to lie medial to the artery at the mid-humeral level. At the antecubital fossa, the nerve remains medial to the brachial artery (Fig. 71.12) and anterior to the brachialis muscle. After that, it pierces through bicipital aponeurosis to enter the forearm to supply all the muscles in the anterior compartment except the flexor carpi ulnaris and the medial half of the flexor digitorum profundus. At the wrist, it passes deep to the flexor retinaculum near the midline, lying between the flexor carpi radialis tendon and the palmaris longus tendon, before entering the hand to supply the thenar muscles and lateral two lumbricals. The cutaneous branches supply the palmar aspects of first three digits and the lateral half of the fourth digit.

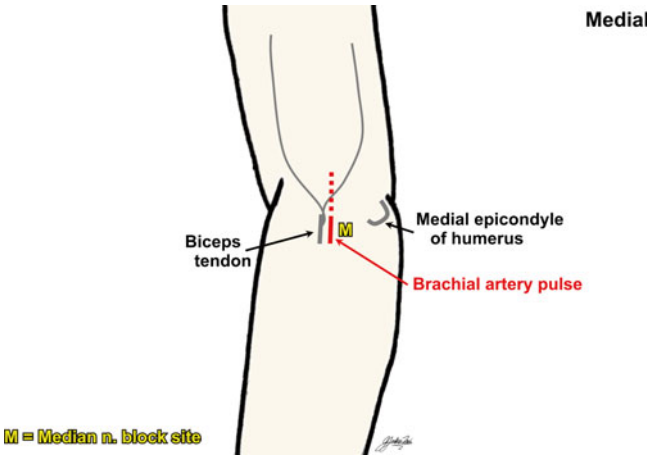


Fig. 71.12 Surface anatomy and landmarks for median nerve block

Patient Positioning, Preparation, Equipment, and Dosage

The arm is rested on an arm board with the elbow slightly flexed. Prepare the skin and, if ultrasound is used, the probe. A 50-mm short-beveled needle (insulated if using nerve stimulation) should be used. A volume of 1–3 mL of 0.25–0.5 % ropivacaine or bupivacaine is adequate for this block.

Nerve Stimulation Technique

The needle is inserted immediately medial to the brachial artery at the antecubital fossa (Fig. 71.12). Advance slowly until appropriate motor responses are observed (flexion in the lateral three digits, wrist flexion, or forearm pronation). The nerve should be superficial to the skin.

Ultrasound-Guided Technique

A high-frequency (13-6 MHz), small footprint ("hockey stick") probe should be used. The probe is placed in the axial plane medial to the biceps tendon at or near the antecubital fossa. The nerve, which appears as oval-peanut shaped and is often larger than the artery, should be seen lying medially to the brachial artery (Fig. 71.13). Both in-plane and out-of-plane approaches can be used. When using the in-plane approach, the needle is inserted medial to the probe to reduce the chance of puncturing the brachial artery. A test dose of D5W to observe the spread is recommended before local anesthetic is deposited around the nerve.

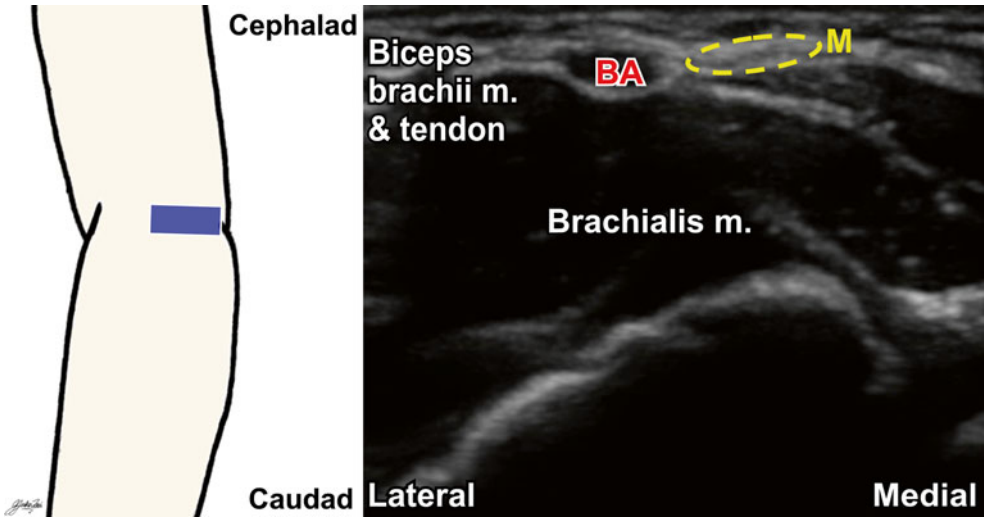


Fig. 71.13 Ultrasound image of the median nerve at the antecubital fossa. *BA* brachial artery, *M* median nerve

Ulnar Nerve

Introduction, Indications, and Complications

The ulnar nerve innervates muscles that produce flexion of the fourth and fifth fingers and ulnar deviation of wrist. It is derived from the medial cord, carrying fibers from roots C7 to T1. Distal to the axilla, the ulnar nerve descends medial to the brachial artery in the upper arm before passing posterior to the medial epicondyle in the condylar groove. The nerve then courses anteriorly in the forearm, lying on the surface of flexor digitorum profundus, deep to the flexor digitorum superficialis, and lateral to the flexor carpi ulnaris, to supply the flexor carpi ulnaris and medial half of the flexor digitorum profundus. The nerve runs medial to the ulnar artery, approaching the artery at the mid-forearm (Fig. 71.14). At the wrist, the nerve crosses superficial to the flexor retinaculum, between the ulnar artery (laterally) and the flexor carpi ulnaris tendon (medially), to supply all intrinsic muscles of hand except the thenar muscles and the lateral two lumbricals. The cutaneous branches supply the dorsal and palmar aspects of the fifth and the medial half the fourth finger.

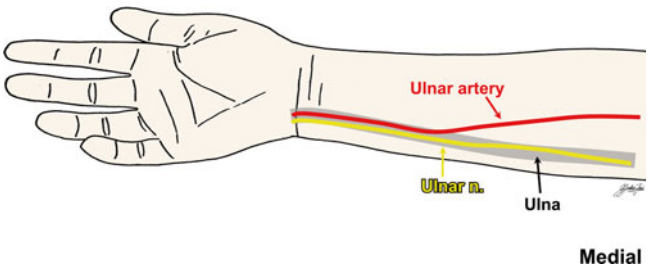


Fig. 71.14 Surface anatomy and landmarks for ulnar nerve block

Patient Positioning, Preparation, Equipment, and Dosage

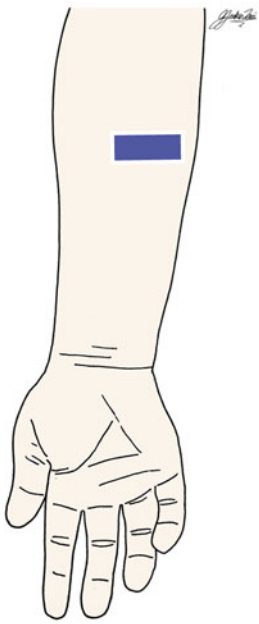
The elbow is flexed with the shoulder externally rotated and the forearm supinated. Prepare the skin and, if ultrasound is used, the probe. A 50-mm short-beveled needle (insulated if using nerve stimulation) should be used. A volume of 1–3 mL of 0.25–0.5 % ropivacaine or bupivacaine is adequate for this block.

Nerve Stimulation Technique

The needle is inserted 45° to the skin, 1–3 cm distal (or alternatively, 2–3 cm proximal) to the condylar groove between the medial epicondyle and olecranon process, pointing proximally in the direction of the groove. Advance slowly until appropriate motor responses are observed (flexion of the fourth and fifth fingers and/or ulnar deviation of the wrist). The nerve should be superficial at this point.

Ultrasound-Guided Technique

A high-frequency (13-6 MHz), small footprint ("hockey stick") probe should be used. The probe is placed in the axial plane across the flexor carpi ulnaris muscle on the forearm. The nerve, which appears as oval or round shape, should be seen lying medially to the ulnar artery (Fig. 71.15). Its location can be confirmed by scanning up and down the forearm to observe the nerve and the artery approaching each other at the mid-forearm. Both in-plane and out-of-plane approaches can be used. It is best to target the nerve when it separates from the artery to minimize the risk of arterial puncture during needle insertion. A test dose of D5W to observe the spread is recommended before local anesthetic is deposited around the nerve.



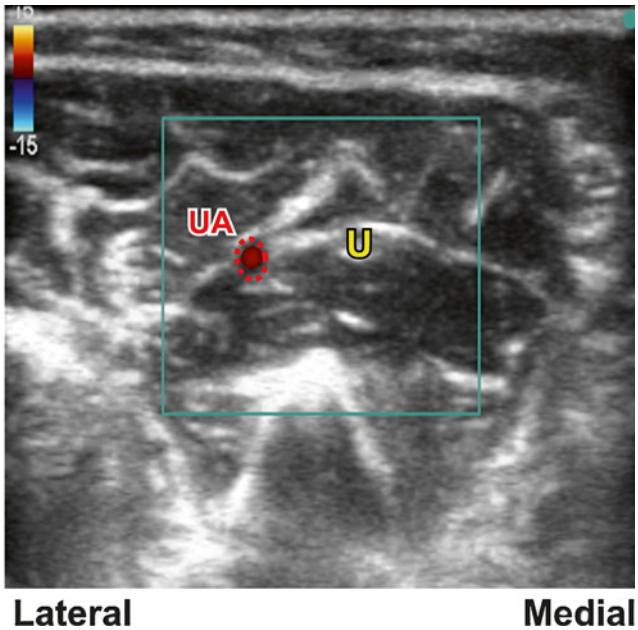


Fig. 71.15 Ultrasound image of the ulnar nerve in the forearm. *UA* ulnar artery, *U* ulnar nerve

Radial Nerve

Introduction, Indications, and Complications

The radial nerve innervates muscles that produce extension of the wrist and fingers. The nerve is derived from the posterior cord, carrying fibers from roots C5 to C8. After leaving the axilla, the nerve runs between two heads of the triceps muscle. It then travels in the radial groove of the humerus, posteromedial to the deep brachial artery. As it approaches the elbow, it crosses over the lateral epicondyle to enter the anterior compartment of the forearm, where it runs between the brachialis and brachioradialis muscles. The nerve divides into deep (motor) and superficial (sensory) branches as it passes in front of the lateral epicondyle. The motor branches supply the muscles of the posterior compartments of the arm and forearm, while the cutaneous branches supply the posterior arm and forearm as well as the dorsum of the hand except the medial and dorsal aspects of the lateral three and a half fingers up to the distal interphalangeal crease.

Patient Positioning, Preparation, Equipment, and Dosage

The arm is placed slightly abducted with the elbow flexed approximately 30°. Prepare the skin and, if ultrasound is used, the probe. A 50-mm short-beveled needle (insulated if using nerve stimulation) should be used. A volume of 1–3 mL of 0.25–0.5 % ropivacaine or bupivacaine is adequate for this block.

Nerve Stimulation Technique

The needle is inserted in the groove between the biceps brachii tendon and the brachioradialis muscle at the intercondylar line, pointing cephalad (Fig. 71.16). Advance the needle until appropriate motor response(s) (extension of the wrist and fingers) is observed. The nerve is usually superficial.

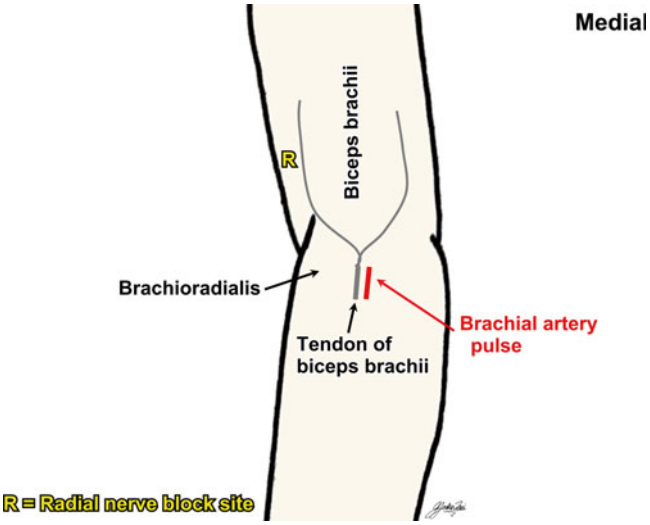


Fig. 71.16 Surface anatomy and landmarks for radial nerve block

Ultrasound-Guided Technique

A high-frequency (13-6 MHz), small footprint ("hockey stick") probe should be used. With the probe placed in the axial plane on the arm, the block can be performed at more proximal or distal locations. Proximally, the nerve can be located in the radial groove at the posterior humerus, where it travels with the deep brachial artery (Fig. 71.17). Distally, the nerve can be found in the groove between the biceps brachii tendon and the brachioradialis muscle at the lateral aspect of the distal upper arm (at the level of the supracondylar ridge). It can be confirmed by scanning further downward to look for where the nerve splits into two branches.

Both in-plane and out-of-plane approaches can be used. With an in-plane approach, the needle is inserted in an anterior-to-posterior direction to avoid puncturing the deep brachial artery. The out-of-plane approach causes less discomfort and is recommended in awake patients. The needle is inserted a small distance away from the midpoint of the probe, directing cephalad. A test dose of D5W to observe the spread is helpful before depositing local anesthetic around the nerve.

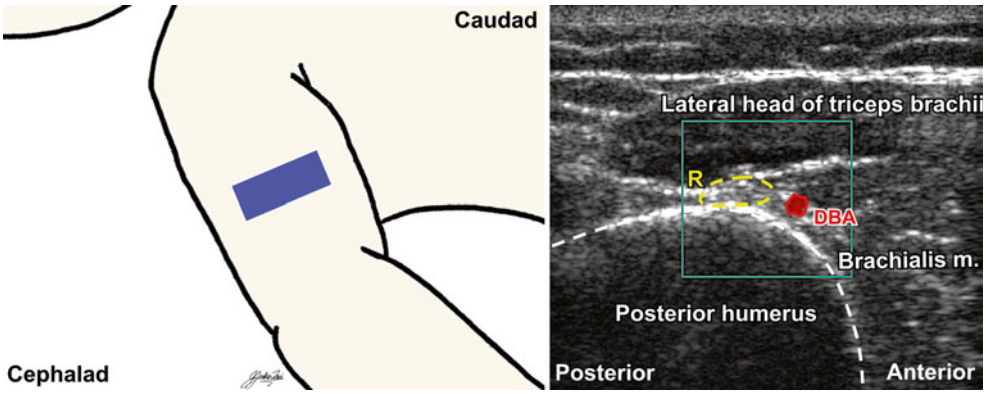


Fig. 71.17 Ultrasound image of the location of the radial nerve. *R* radial nerve, *DBA* deep brachial (profunda brachii) artery

Acknowledgments This chapter was written using Drs. Tsui and Suresh's textbook, *Pediatric Atlas of Ultrasound and Nerve Stimulation-Guided Regional Anesthesia* 1st edition (Springer) as a template. The authors thank Jenkin Tsui for providing the original artwork for the figures, Alex Kwan for the assistance with creating and organizing the figures, and Dr. Gareth Corry for the assistance with manuscript preparation and management.

References

1. Dalens B, Vanneuville G, Tanguy A. A new parascalene approach to the brachial plexus in children: comparison with the supraclavicular approach. Anesth Analg. 1987;66:1264–71.
2. Carre P, Joly A, Cluzel FB, Wodey E, Lucas MM, Ecoffey C. Axillary block in children: single or multiple injection? Paediatr Anaesth. 2000;10:35–9.

Suggested Reading

Jankovic D, Wells C. Regional nerve blocks: textbook and color atlas. 2nd ed. Wiley; 2001.
Lin E, Gaur A, Jones M, Ahmed A. Sonoanatomy for anaesthetists. Cambridge University Press; 2012.
Marhofer P. Ultrasound guidance for nerve blocks: principles and practical implementation. Oxford University Press; 2008.
Peutrell JM, Mather SJ. Regional anesthesia for babies and children. Oxford University Press; 1997.
Saint-Maurice C, Schulte Steinberg O, Armitage E, editors. Regional anaesthesia in children. Appleton & Lange, Mediglobe; 1990.
Tsui BC. Atlas of ultrasound and nerve stimulation-guided regional anesthesia. Springer; 2007.

Chapter 72

Pediatric Peripheral Nerve Block: Lower Limb

Sara S.K. Cheung, MBBS, FHKCA, FHKAM
Department of Anaesthesia, Pamela Youde Nethersole Eastern Hospital, Hong Kong, China

University of Alberta, Edmonton, AB, Canada
e-mail: cheungsk3@gmail.com

Heather Y.Z. Ting, MD, FRCPC
Department of Anesthesiology and Pain Medicine, University of Alberta, Edmonton, AB, Canada
e-mail: yizhenhe@ualberta.ca

Ban C.H. Tsui, MSc, MD, FRCPC (✉)
Department of Anesthesiology and Pain Medicine, University of Alberta and University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada
e-mail: btsui@ualberta.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_72, © Springer International Publishing Switzerland 2015

Femoral Nerve Block

Introduction, Indications, and Complications

The femoral nerve is a mixed sensory and motor nerve. The motor fibers supply the quadriceps femoris, sartorius, and pectineus muscles, while the sensory fibers innervate the anterior thigh and medial aspect of the knee and lower leg. The nerve is formed from the posterior divisions of the L2–L4 roots. After emerging at the lateral border of psoas, it descends between the psoas and iliacus muscles underneath the fascia iliaca and runs beneath the inguinal ligament into the thigh, lateral to the femoral sheath.

Femoral nerve block is a relatively safe and easy block that is performed commonly in children. It is indicated in the management of femoral fracture, providing pain relief and muscle relaxation to facilitate transport, examination, and manipulation of the fracture. It also offers postoperative analgesia for surgery on the knee and quadriceps tendon. Occasionally, it is used to provide surgical anesthesia for muscle biopsy and skin grafting of the anterior thigh. Complete anesthesia below the mid-thigh can be achieved if it is performed in combination with a sciatic nerve block.

Patient Positioning, Preparation, Equipment, and Dosage

The patient lies supine and the leg is slightly externally rotated. The skin is cleaned with antiseptic solution. If ultrasound is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G short-beveled needle is used. An insulated needle should be chosen if nerve stimulation is used. The recommended local anesthetic dose is 0.2–0.5 mL/kg of 0.25 % bupivacaine or 0.2 % ropivacaine without exceeding the toxic dose limits (2 mg/kg for bupivacaine and 3–4 mg/kg for ropivacaine without epinephrine). The dose for the femoral block should be decreased if an additional sciatic nerve block is planned.

Nerve Stimulation Technique

The point of needle insertion is approximately 0.5–1 cm lateral to the femoral artery at the inguinal crease (which is about 0.5–1 cm below the inguinal ligament) (Fig. 72.1). This insertion point may vary according to the age and weight of the child. The needle is inserted at 30–45° to the skin, pointing cephalad. Advance the needle until "patellar twitch" (contraction of quadriceps femoris muscle) is obtained. The current is initially set at 0.8–1 mA (1 Hz, 0.1–0.2 ms) and then gradually reduced to a threshold current of 0.5 mA. Currents less than 0.3 mA indicate intraneural placement, and the needle should be withdrawn slightly. As the needle is advanced, two "pops" or "gives" can be felt as the needle passes through the fascia lata and fascia iliaca, respectively. Local anesthetic is injected after the second "pop" following negative aspiration. Depending on the age and size of the patient, the depth of insertion is usually around 0.5–1 cm.

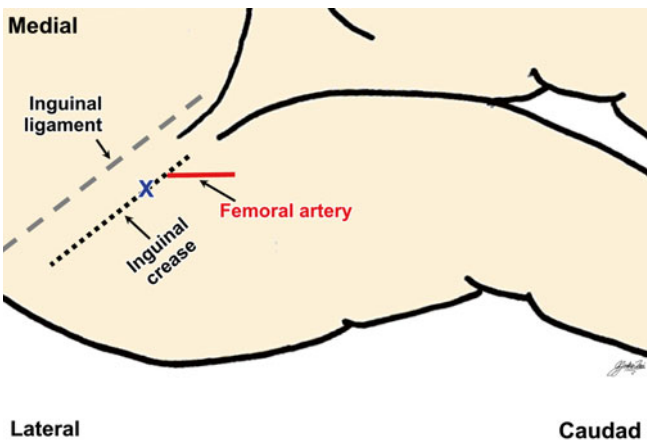


Fig. 72.1 Surface anatomy and landmarks for femoral nerve block

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) linear probe is suitable for this block. In small children, a small footprint ("hockey stick") probe is desirable. The probe is placed in a transverse plane along the inguinal crease to obtain a transverse view of the femoral artery and the vein (Fig. 72.2). The nerve, which often appears oval or triangular shaped, is located lateral to the femoral artery and just underneath the hyperechoic fascia iliaca. Color Doppler may be used to identify the vessels. The femoral artery is anechoic, circular, and pulsatile, whereas the femoral vein is more irregular in shape, compressible, and located medial to the artery. If localization of the nerve is difficult, scan distally to look for the profunda femoris artery and trace it proximally to the point where it joins the femoral artery. The femoral nerve is usually found lateral to the artery.

Both in-plane and out-of-plane techniques can be used. For the in-plane approach, the needle is inserted at the lateral edge of the probe and angled medially to reach the nerve. Two "pops" or "gives" are felt as the needle penetrates the fascia lata and fascia iliaca. When using the out-of-plane approach, the nerve is centered in the middle of the screen, and the needle is inserted at 0.5–1 cm away from the midpoint of the caudad edge of the probe at 45° to the skin. The needle then "walks down" to reach the nerve while maintaining the needle tip in view. A test dose of D5W can visualize spread and confirm nerve localization prior to injection of local anesthetic.

Lateral Cutaneous Nerve Block

Introduction, Indications, and Complications

The lateral cutaneous nerve of the thigh is a pure sensory nerve that innervates the lateral thigh up to the knee. It arises from the posterior branches of the L2 and L3 roots. After emerging from the lateral border of the psoas muscle, the nerve runs obliquely towards the anterior superior iliac spine (ASIS) beneath the fascia iliaca. It then passes underneath the inguinal ligament at a point approximately one fingerbreadth medial to the ASIS and then enters the thigh deep to the fascia lata, where it divides into anterior and posterior branches to supply the skin of the lateral thigh.

This block is indicated for procedures involving the lateral thigh, for example, muscle biopsy, skin grafting, and hip or femur operations with lateral incisions. Very often, it is performed in conjunction with femoral and sciatic nerve blocks for upper thigh surgery. Occasionally, it is used to diagnose meralgia paresthetica, a condition that presents with pain and numbness of the outer thigh as a result of the entrapment of the lateral cutaneous nerve of the thigh.

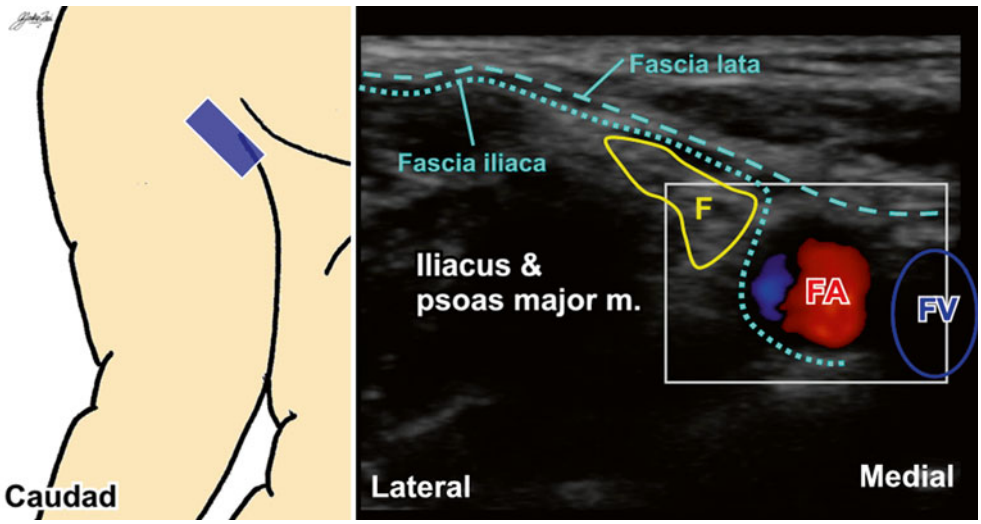


Fig. 72.2 Ultrasound image of major anatomical structures surrounding the femoral nerve. *F* femoral nerve, *FA* femoral artery, *FV* femoral vein

Patient Positioning, Preparation, Equipment, and Dosage

The child lies supine with the leg externally rotated. Clean the skin with antiseptic solution. If ultrasound is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G short-beveled needle is used. Local anesthetics of choice are 0.25 % bupivacaine and 0.2 % ropivacaine. A volume of 1–3 mL (up to a maximum of 10 mL) is adequate.

Landmark Technique

Nerve stimulation is not used for this block since the nerve is purely sensory and monitoring paresthesia is often impossible for children. The point of needle insertion for the landmark technique is approximately 0.5–1 cm (depending on the age and size of the child) medial to the ASIS and below the inguinal ligament. Advance the needle until a "pop" is felt as the fascia lata is penetrated. Local anesthetic is deposited at this point.

Ultrasound-Guided Technique

A small footprint ("hockey stick") high-frequency (13-6 MHz) probe is used. The probe is placed in a transverse plane along the inguinal crease with the lateral end over the ASIS. The nerve, which is often oval shaped, is usually seen 0.5–1 cm medial and inferior to the ASIS, between the fascia lata and fascia iliaca (Fig. 72.3). The nerve is superficial and can be difficult to identify. The hyperechoic nerve will be better visualized after injection of hypoechoic D5W or local anesthetic, which expands the fascial plane.

Both in-plane and out-of-plane techniques can be used. For the in-plane approach, the needle is inserted in either a lateral-to-medial or medial-to-lateral direction. When using the out-of-plane approach, the nerve is centered in the middle of the screen, and the needle is inserted 1 cm away from the caudad edge of the probe at 45° to the skin. The needle then "walks down" to reach the nerve between the fascial planes while maintaining the needle tip in view. A test dose of D5W can visualize spread and confirm nerve localization prior to injection of local anesthetic.

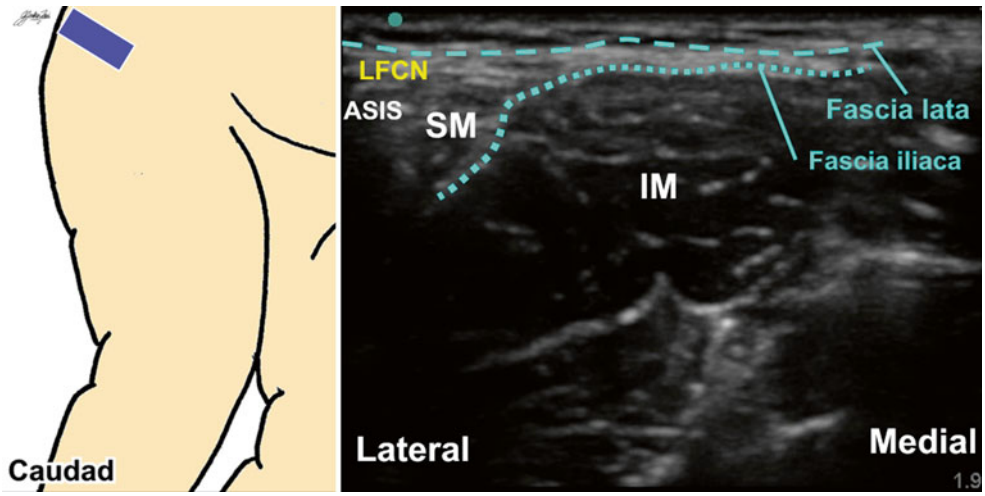


Fig. 72.3 Ultrasound image of the lateral femoral cutaneous nerve (*LFCN*) between the fascia lata and fascia iliaca. *ASIS* anterior superior iliac spine, *SM* sartorius muscle, *IM* iliacus muscle

Obturator Nerve Block

Introduction, Indications, and Complications

The obturator nerve is a mixed sensory and motor nerve. The motor fibers supply the adductor muscles (adductor longus, adductor brevis, adductor magnus, and gracilis). The sensory fibers innervate the medial thigh and the hip and knee joints. The nerve is derived from anterior divisions of the L2–L4 roots. After emerging from the inner border of the psoas, the nerve descends medially and posteriorly in the pelvis, close to the lateral wall of the bladder. The nerve exits the pelvis via the obturator foramen, splitting into anterior and posterior branches which enter the thigh. The anterior branch runs between the adductor longus and adductor brevis, supplying the adductor muscles, the hip joint, and the skin of the medial thigh. The posterior branch travels between the adductor brevis and adductor magnus, supplying the adductors and the knee joint.

Indications for obturator nerve block include relieving adductor spasm and treatment of pain in the hip and knee when combined with femoral block. Several approaches have been described to block the obturator nerve, for example, the psoas compartment block, iliaca compartment block, and the 3-in-1 block. The success rates of these approaches vary, and some approaches can be difficult to perform. Also, accidental puncture of the obturator arteries is a potentially life-threatening complication. The use of ultrasound allows isolated blockade of the nerve with increased success and safety and is highly recommended.

Patient Positioning, Preparation, Equipment, and Dosage

The leg is externally rotated and the knee slightly flexed to expose the medial thigh. The skin is cleaned with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if ultrasound is used.

A 50-mm, 22-G insulated needle is used. Choices of local anesthetic include 0.25 % bupivacaine and 0.2 % ropivacaine. Usually, a volume of 0.25 mL/kg (maximum 10 mL) is required for each interfascial plane. The depth is approximately 4–6 cm.

Nerve Stimulation Technique

The point of needle insertion is 0.5–1 cm (depending on the age and size of the child) lateral and caudal to the pubic tubercle (Fig. 72.4), where the nerve just passes underneath the inguinal ligament before bifurcation. Advance the needle until it contacts the superior pubic ramus and then walk the needle off the inferior border of the superior pubic ramus to enter the obturator foramen. Redirect the needle laterally and cephalad and advance slowly until twitches of the adductor muscles are observed. The initial current is set at 0.8–1 mA (1 Hz, 0.1–0.2 ms) and decreased slowly to a threshold current of 0.5 mA. Local anesthetic is deposited after careful aspiration.

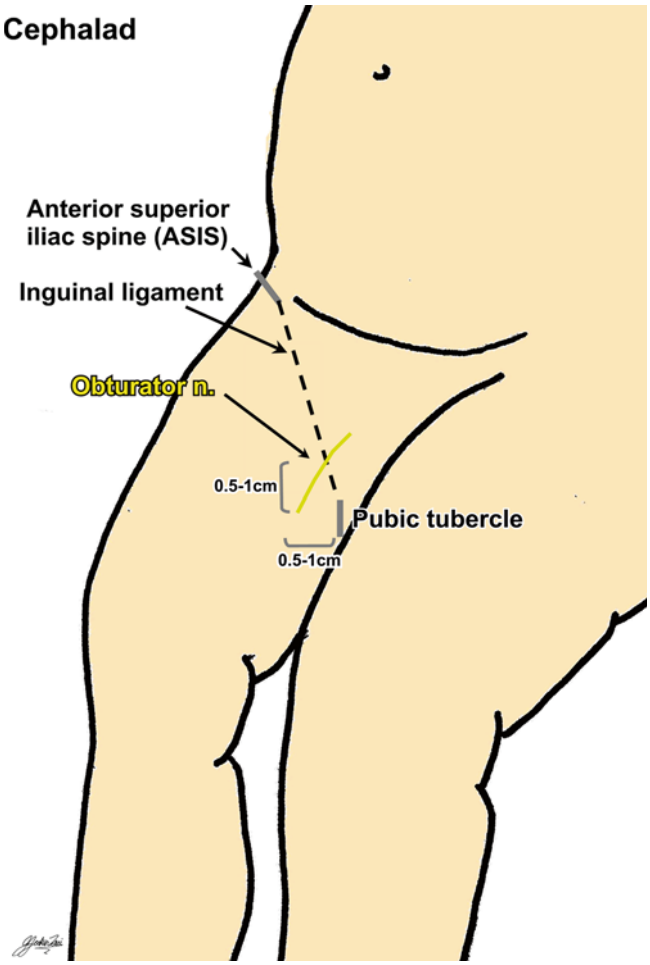


Fig. 72.4 Surface anatomy for obturator nerve block

Ultrasound-Guided Technique

A 13-6 MHz linear probe is suitable for this block. The probe is placed in a transverse plane below the inguinal crease (around 2–3 cm inferior to the inguinal ligament) (Fig. 72.5). After locating the femoral vessels, scan medially to identify the pectineus muscle which is medial to the femoral vein. The three adductor muscles (adductor longus, adductor brevis, and adductor magnus, from superficial to deep) can be found medial to the pectineus. As the branches of the obturator nerve are usually too small to be seen, the aim for this technique is to perform two fascial blocks (injection between the fascial planes through which the branches pass).

An in-plane approach is recommended. The needle is inserted at the lateral edge of the probe and angled medially to first reach the hyperechoic fascial plane (which contains the anterior branch of the obturator nerve) between the adductor brevis and adductor magnus. Half of the local anesthetic is deposited here. After that, the needle is advanced further into the deeper plane (which contains the posterior branch) between the adductor brevis and adductor longus, and the rest of the drug is injected. A test dose with D5W prior to injection of local anesthetic is useful to visualize spread and confirm position of the needle tip. Attention should be paid not to puncture the femoral and circumflex vessels.

Saphenous Nerve Block

Introduction, Indications, and Complications

The saphenous nerve is a purely sensory nerve which innervates the medial aspect of the lower leg and foot. It is the terminal branch of the femoral nerve and takes its origin from the L3–L4 roots. Together with the femoral artery, the saphenous nerve travels through the adductor canal in the mid to lower thigh, where it lies underneath the sartorius muscle. The nerve then emerges subcutaneously just below the knee and runs along the medial side of the lower leg, accompanied by the saphenous vein anterolaterally.

This block is indicated for operations on the medial aspects of the lower leg and foot. When combined with a sciatic nerve block, it provides complete blockade of the whole lower leg and foot for major foot and ankle surgery.

Patient Positioning, Preparation, Equipment, and Dosage

The leg is externally rotated with the knee slightly flexed to expose the medial thigh. The skin is cleaned with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if ultrasound is used.

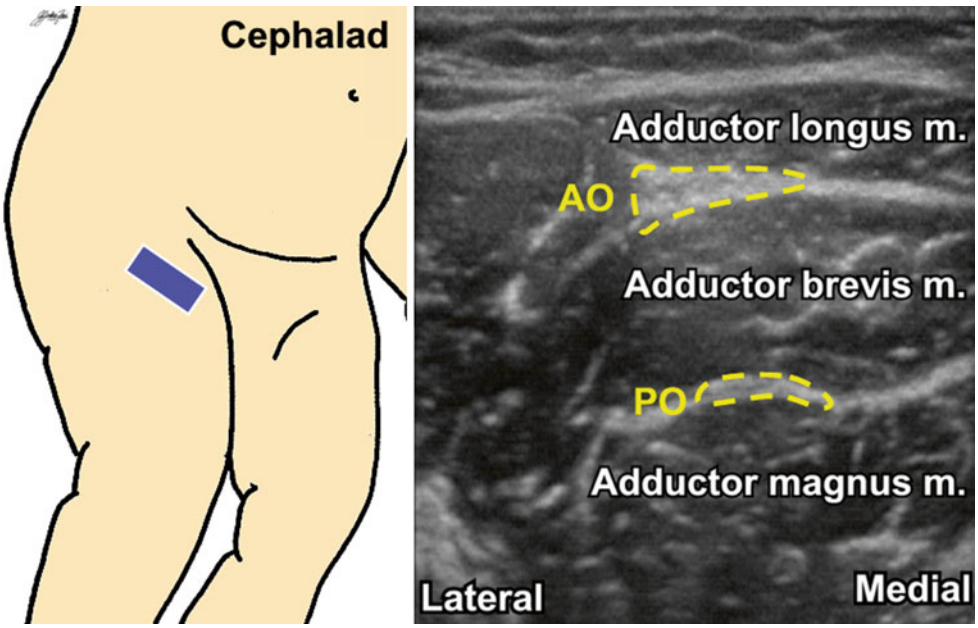


Fig. 72.5 Ultrasound image of the adductor muscles and anterior (*AO*) and posterior (*PO*) obturator nerves

A 50-mm, 22-G needle is used. Alternatively, a 20-G Tuohy pediatric epidural needle is also suitable for this block. The recommended local anesthetic dose is 0.15–0.25 mL/kg (maximum 10 mL) of 0.25 % bupivacaine or 0.2 % ropivacaine. Too much volume can increase the intracompartment pressure (especially for adductor canal block), resulting in nerve injury, and is not recommended.

Landmark Technique

Nerve stimulation is not used for this block since the nerve is purely sensory, and monitoring paresthesia is often impossible in children.

Different locations have been described to block the saphenous nerve [1, 2], including a field block around the tibial tuberosity, but the success rates vary due to significant branching of the nerve below the knee. The transsartorial approach has shown to be the most effective approach for this block. The point of needle insertion is one fingerbreadth above the patella on the sartorius muscle at the medial side of the lower thigh (Fig. 72.6). Direct the needle 45° caudally into the sartorius muscle and advance slowly until a "pop" or give is felt as the needle passes through the sartorius fascia (usually at a depth of 1–2 cm, depending on the age and size of the child).

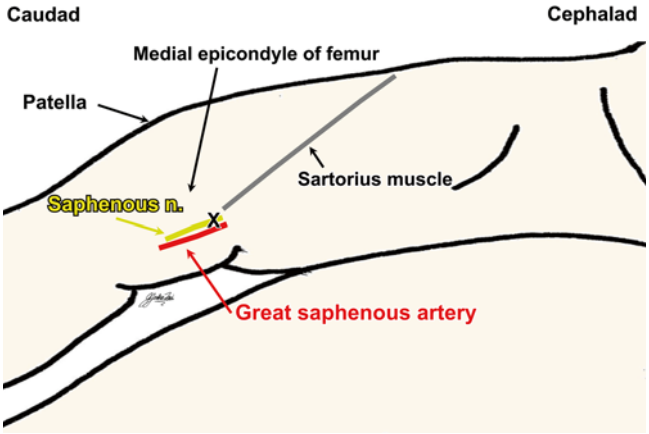


Fig. 72.6 Surface anatomy for saphenous nerve block. The *X* indicates the site of needle insertion for the landmark-guided approach

Ultrasound-Guided Technique

Similar to the landmark technique, various approaches have been suggested for blocking the saphenous nerve using ultrasound, including the paravenous approaches (either at the level of the proximal tibia or ankle) and the adductor canal block [3–6]. The latter offers more reliable blockade, not only because the landmark—the femoral artery—is easier to visualize, but less branching occurs at a more proximal location.

A 13-6 MHz linear probe is used. The probe is placed in a transverse plane over the anteromedial surface of the mid to lower thigh to obtain a transverse view of the femoral artery underneath the sartorius muscle (Fig. 72.7). Trace the artery distally to the point just before it starts to descend posteriorly to form the popliteal artery. At this point, the vastus medialis is lateral to the artery, and the adductor magus is medial. The nerve, which is often too small to be seen, usually lays anterolateral to the artery in the fascial plane between the sartorius and the vastus medialis. Beneath the vastoadductor membrane, the nerve crosses the descending genicular artery to lie medial to the artery.

An in-plane approach is commonly performed for this block. The needle is inserted in a lateral-to-medial direction and directed to the medial side of the femoral artery in the fascial plane between the artery and the sartorius muscle. A test dose of D5W can expand the fascial plane and confirm needle-tip location prior to injection of local anesthetic.

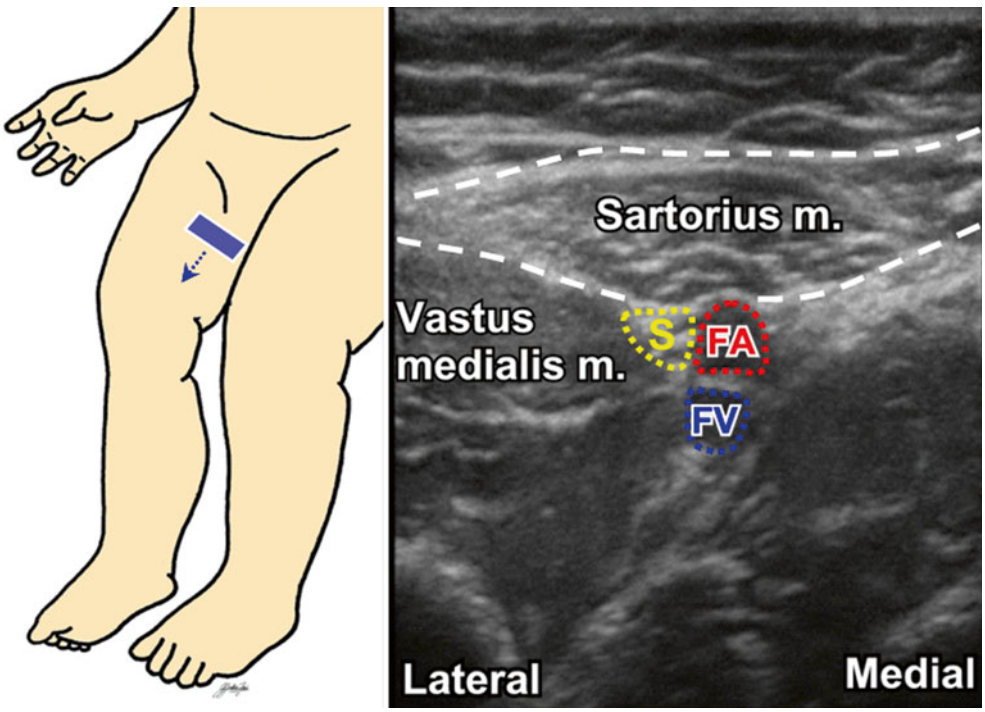


Fig. 72.7 Ultrasound image showing the saphenous nerve in the adductor canal. Often, the probe is moved distally until the femoral artery is further away from the nerve. *S* saphenous nerve, *FA* femoral artery, *FV* femoral vein

Sciatic Nerve Block

Introduction, Indications, and Complications

The sciatic nerve is the largest peripheral nerve in the body. Derived from the L4–S3 roots, it exits the pelvis along with the posterior cutaneous nerve of the thigh via the greater sciatic foramen underneath the piriformis. It then passes midway between the greater trochanter of the femur and ischial tuberosity and descends along the back of the thigh. Proximally, the nerve lies deep to the gluteus maximus, after which it lies underneath the long head of the biceps femoris muscle. In the distal thigh, the nerve divides into the tibial nerve medially and common peroneal nerve laterally. The sciatic nerve supplies the posterior aspect of thigh, the hamstring muscles, the entire lower leg (except the territory supplied by the saphenous nerve), as well as the hip and knee joints.

Blockade of the sciatic nerve can be achieved proximally or distally depending on the site of the surgery and is commonly performed together with a femoral/saphenous block to provide anesthesia or analgesia for surgery on the knee, lower leg, and foot. Although nerve stimulation and ultrasound-guided techniques are described individually, it is strongly recommended that both techniques be used in combination for more accurate localization of the nerve and better monitoring for intraneural injection.

Posterior Gluteal (LABAT) Approach

This approach aims to block the sciatic nerve proximally at the gluteal region. This can be technically difficult since the nerve lies deep in the muscles.

Patient Positioning, Preparation, Equipment, and Dosage

The child lies in the lateral decubitus position with the hip and knee flexed. Clean the skin with antiseptic solution. Prepare the ultrasound probe surface by applying a sterile adhesive dressing.

A 50–100-mm, 22-G needle is used. Choices of local anesthetic are 0.2 % ropivacaine, 0.25 % bupivacaine, and 0.25 % levobupivacaine. Lower concentrations should be used in children aged less than six. A volume of 0.25–0.5 mL/kg (without exceeding the toxic dose limit) is recommended. Needle insertion depth is approximately 1 mm per kg for a child 20–40 kg.

Nerve Stimulation Technique

A line is drawn between the greater trochanter and the posterior superior iliac spine. A perpendicular line is drawn from the midpoint of the first line to intersect a third line drawn between the greater trochanter and the sacral hiatus (Fig. 72.8). The needle is inserted at the point of intersection perpendicularly to the skin and advanced until (a) twitches of the hamstring or calf muscles, (b) plantar flexion/inversion of the ankle, or (c) extension of the toes are observed. Twitches in the foot and toes indicate stimulation of the tibial or common peroneal nerves (rather than the proximal branches) and are preferred. The initial current is set at 1–1.5 mA (2 Hz, 0.1–0.2 ms) and then gradually reduced to a threshold current of 0.4 mA. Local anesthetic is injected after negative aspiration.

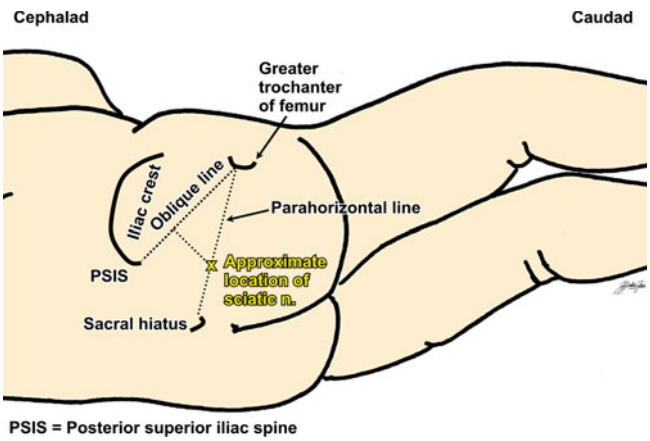


Fig. 72.8 Surface anatomy of the posterior gluteal approach for sciatic nerve block

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) linear probe is used for small children. For older and/or obese children, a curved-array low-frequency (5-2 MHz) probe is preferred. The probe is placed firmly on the gluteal region to obtain the best transverse view of the sciatic nerve (Fig. 72.9). The nerve, which is hyperechoic and appears wide and flat in this view, is situated underneath the large gluteus maximus muscle and superficial to the ischial bone. If there is difficulty in recognizing the nerve, locate it in the subgluteal region and trace proximally.

Both in-plane and out-of-plane approaches can be used. For the in-plane approach, the needle is inserted from the lateral edge of the probe and directed medially to reach the nerve. When using the out-of-plane approach, center the nerve in the middle of the screen, and insert the needle caudal to the probe at a steep angle. In both approaches, extra attention should be paid to avoid puncturing the inferior gluteal artery (adjacent to the nerve) or the internal pudendal vessels (adjacent to the ischial spine). A test dose of D5W can visualize spread and confirm nerve localization. Local anesthetic is injected after negative aspiration. For an optimal block, aim for a hypoechoic local anesthetic fluid collection around the hyperechoic nerve.

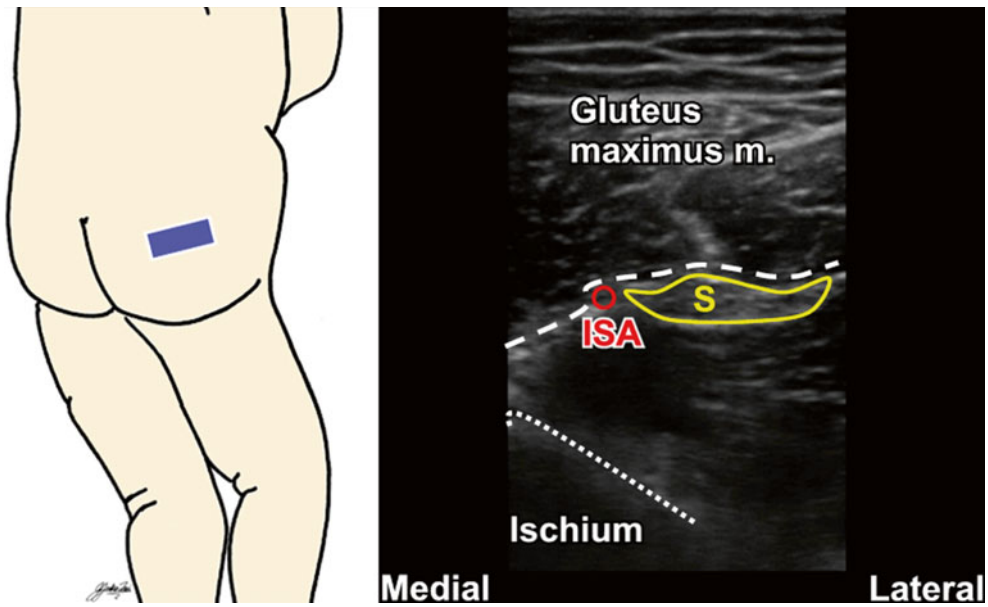


Fig. 72.9 Ultrasound image of posterior gluteal sciatic nerve block location. *S* sciatic nerve, *IGA* inferior subgluteal artery

Infragluteal/Subgluteal Approach

This approach aims to block the nerve at the subgluteal region.

Patient Positioning, Preparation, Equipment, and Dosage

The child lies in the lateral decubitus position with the hip and knee flexed. Clean the skin with antiseptic solution. Prepare the ultrasound probe surface by applying a sterile adhesive dressing.

A 50–100-mm, 22-G needle is suitable for this block. Similar to the previous approach, the recommended local anesthetic dose is 0.25–0.5 mL/kg (without exceeding the toxic dose limit) of 0.2 % ropivacaine, 0.25 % bupivacaine, or 0.25 % levobupivacaine.

Nerve Stimulation Technique

The point of needle insertion is the midpoint between the greater trochanter and the ischial tuberosity (Fig. 72.10). The needle is inserted perpendicularly to the skin and advanced until twitches of the hamstring, calf muscles, ankle, or toes are observed. The current is initially set at 1–1.5 mA and then slowly reduced. Local anesthetic is deposited when a threshold current of 0.4 mA (2 Hz, 0.1–0.2 ms) is reached. A loss of resistance may be felt when the needle punctures the common sheath of the sciatic nerve.

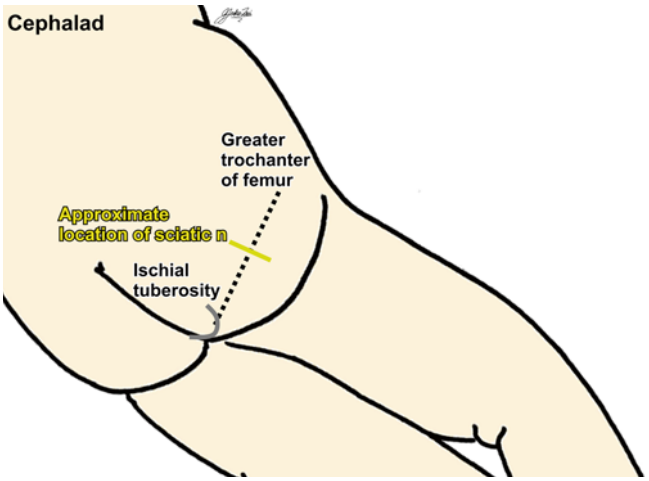


Fig. 72.10 Surface anatomy for subgluteal approach to sciatic nerve block

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) linear probe is used for small children. For older and/or obese children, a curved-array low-frequency (5-2 MHz) probe is preferred. The probe is placed on the subgluteal region to obtain a transverse view of the hyperechoic, elliptical nerve lying between the greater trochanter (laterally) and the ischial tuberosity (medially) (Fig. 72.11). In young children, the bony landmarks may be relatively hypoechoic and not very distinct on ultrasound. If difficulty is encountered in recognition of the nerve, it can first be located in the popliteal fossa and then traced proximally. Both in-plane and out-of-plane approaches can be used. Local anesthetic is injected after negative aspiration and a test dose of D5W.

Anterior Approach

Ultrasound-guided anterior sciatic nerve block has been described for adults [7, 8] and is indicated for use with patients that cannot be positioned laterally. Anterior sciatic nerve blocks are not normally carried out in children since pediatric patients are usually under general anesthesia and can therefore be turned or positioned to allow an approach with a shorter needle depth.

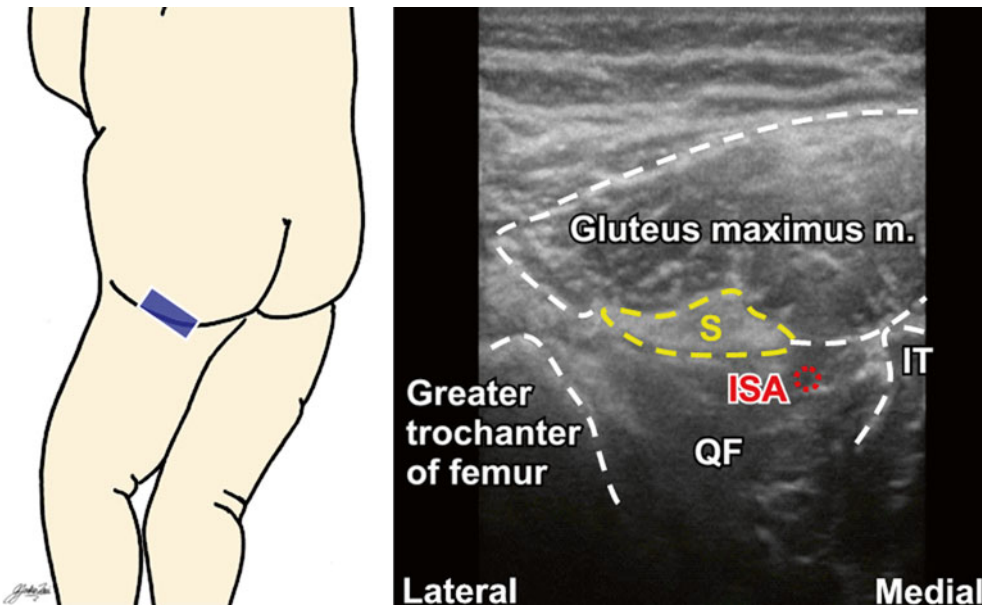


Fig. 72.11 Ultrasound image subgluteal sciatic nerve block location. *S* sciatic nerve, *ISA* inferior subgluteal artery, *QF* quadratus femoris muscle

Popliteal Approach

This approach aims to block the nerve at a superficial level in the popliteal fossa just before the bifurcation. This is the most commonly performed approach to the sciatic block for surgery on the lower leg and foot.

Patient Positioning, Preparation, Equipment, and Dosage

The child lies in the lateral or prone position with the knee extended. The foot is kept off the bed if nerve stimulation is used. The skin is cleaned with antiseptic solution. Prepare the ultrasound probe surface by applying a sterile adhesive dressing.

A 50-mm, 22-G short-beveled block needle is used. The recommended local anesthetic dose is 0.25–0.5 mL/kg (without exceeding the toxic dose limit) of 0.2 % ropivacaine, 0.25 % bupivacaine, or 0.25 % levobupivacaine.

Nerve Stimulation Technique

The popliteal fossa is bordered superolaterally by the biceps femoris, superomedially by the semimembranosus and semitendinosus, and inferiorly by the medial and lateral heads of the gastrocnemius (Fig. 72.12). It is divided by the popliteal crease in the middle, creating the superior and inferior triangles of the popliteal fossa. The sciatic nerve runs lateral and superficial to the popliteal vessels and then branches into tibial nerve (medially) and common peroneal nerve (laterally) near the upper apex of the fossa. According to an age-related formula [9], the nerve bifurcates at a mean distance of 4.1 cm ± 0.8 cm in children under 8 years and at 5.8 cm ± 1.3 cm in children older than 8 years. Some studies have shown that this age-related distance is not consistent and found wide variability in the location of the bifurcation point [10]. Thus, the use of nerve stimulation together with ultrasound guidance is recommended to obtain a higher success rate of blocking the sciatic nerve before it divides or blocking the individual nerves distal to the bifurcation.

The point of needle insertion is midpoint between the semimembranosus and semitendinosus tendons and the biceps femoris tendon in the superior popliteal triangle, immediately lateral to the popliteal pulse, with the distance from the popliteal cease according to the weight of the child: 1 cm if <10 kg, 2 cm if 10–20 kg, etc. [11]. Introduce the needle at 45° to the skin, pointing cephalad. With an initial current of 1 mA, the needle is advanced slowly until twitches in the foot and toes (especially tibial nerve response) are observed. A "pop" or give may be felt as the needle penetrates the popliteal membrane. Local anesthetic is deposited after aspiration when a threshold current of 0.4 mA is reached.

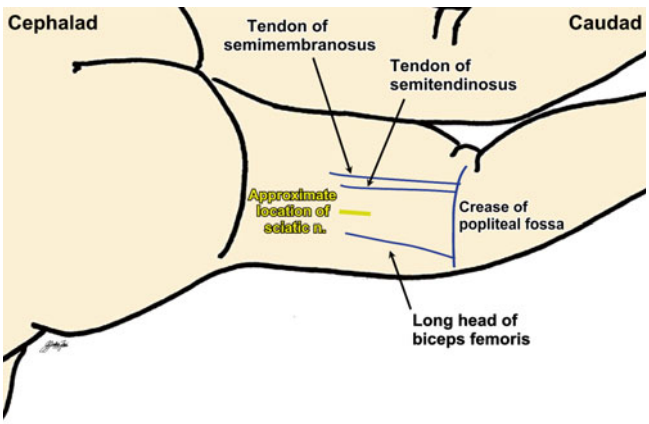


Fig. 72.12 Surface anatomy of the popliteal fossa

Ultrasound-Guided Technique

A small footprint ("hockey stick") high-frequency (13-6 MHz) probe is used. The probe is placed in a transverse plane along the popliteal cease to identify the popliteal vessels (color Doppler can be used). The tibial and common peroneal nerves, which are hyperechoic and appear round or oval, are usually seen superficial and lateral to the popliteal vessels (Fig. 72.13). Scan proximally to observe the two nerves converging to become the sciatic nerve. Both in-plane and out-of-plane approaches can be used, although the out-of-plane approach is preferred. The probe is placed at the bifurcation point with the nerve in the center of the screen. Insert the needle 45° to the skin and caudal to the probe. The distance between the probe and needle insertion point should be the same as the distance between the nerve and skin surface; the needle can then be "walked down" to reach the nerve while keeping the needle tip in view. Local anesthetic is deposited after negative aspiration. For optimal blockade, aim for a circumferential spread around both branches of the sciatic nerve.

Ankle Blocks

Introduction, Indications, and Complications

Five nerves supply the foot: the posterior tibial, deep peroneal, superficial peroneal, sural, and saphenous nerves. The first four are derived from the sciatic nerve, while the saphenous is from the femoral nerve. The posterior tibial nerve is responsible for plantar flexion and cutaneous sensation of the sole of the foot. The deep peroneal nerve is responsible for ankle and toe extension and cutaneous sensation of the web space between the first and second toes. The other three nerves are mainly sensory: the superficial peroneal nerve innervates the dorsum of the foot (except the area supplied by the deep peroneal), the sural innervates the heel and lateral malleolus, and the saphenous innervates the anteromedial aspect. Blocking some or all of the nerves is commonly performed for foot and toe surgery.

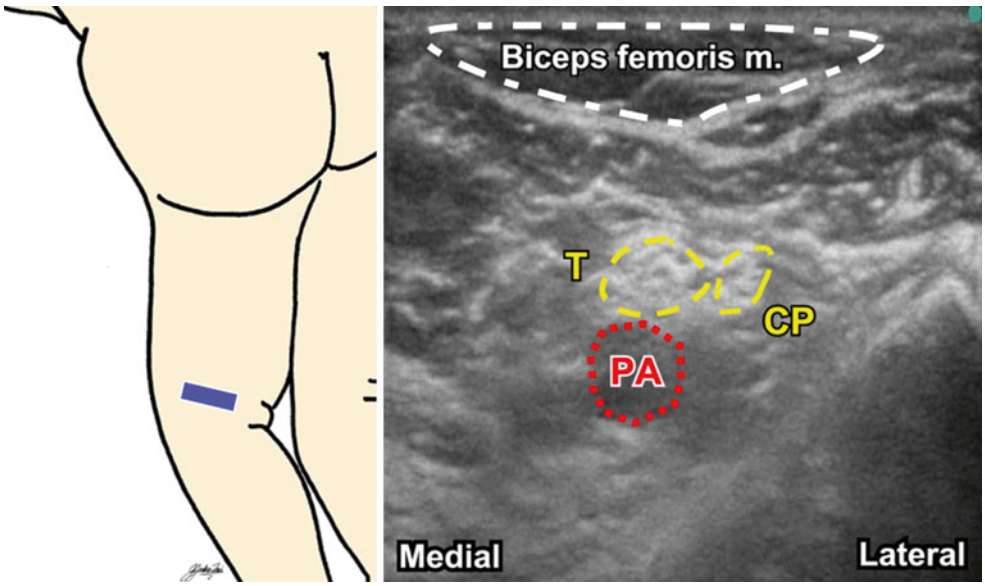


Fig. 72.13 Ultrasound image of the popliteal sciatic nerve block location. *T* tibial nerve, *CP* common peroneal nerve, *PA* popliteal artery

Patient Positioning, Preparation, Equipment, and Dosage

The foot is raised and supported on a pillow. The skin is cleaned with antiseptic solution. If ultrasound is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 25–27-G short-beveled or hypodermic needle is suitable for these blocks. The recommended local anesthetic dose is 0.1 mL/kg of 0.25 % bupivacaine or 0.2 % ropivacaine (a volume of 1–3 mL is usually adequate for each block). Epinephrine should not be added because of the presence of end arteries.

Nerve Stimulation Technique

Among the five nerves that supply the foot, only the posterior tibial and deep peroneal have motor fibers. Since the others are sensory, nerve stimulation is usually not necessary.

Posterior Tibial Nerve

The needle is inserted immediately posterior to the posterior tibial pulse (Fig. 72.14), which is about midway between the medial malleolus and the medial border of the Achilles tendon. With an initial current of 0.8 mA (2 Hz, 0.1–0.3 ms), the needle is advanced until plantar flexion of the toes is observed. Local anesthetic is injected after aspiration when a threshold current of 0.4 mA is reached.

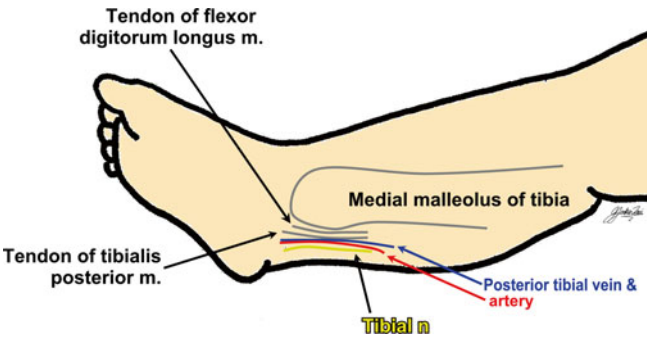


Fig. 72.14 Surface anatomy and landmarks for posterior tibial nerve block

Deep Peroneal Nerve

The needle is inserted perpendicular to the skin with a slight anterior tilt at the mid-foot, lateral to the extensor hallucis longus tendon and immediately lateral to the anterior tibial pulse (Fig. 72.15). Observe for toe extension upon nerve stimulation. Local anesthetic is deposited when twitches are seen at a current of 0.4 mA.

Superficial Peroneal Nerve

Local anesthetic is infiltrated subcutaneously along the line between the malleoli.

Saphenous Nerve

Local anesthetic is infiltrated subcutaneously above and anterior to the medial malleolus.

Sural Nerve

The needle is inserted midway between the lateral malleolus and the Achilles tendon.

Ultrasound-Guided Technique

While the more superficial nerves (superficial peroneal, sural, and saphenous) can be blocked reliably using the landmark technique, blockade of the deeper nerves (posterior tibial and deep peroneal) can be facilitated by the use of ultrasound. A high-frequency (13-6 MHz) small footprint "hockey stick" transducer is preferred.

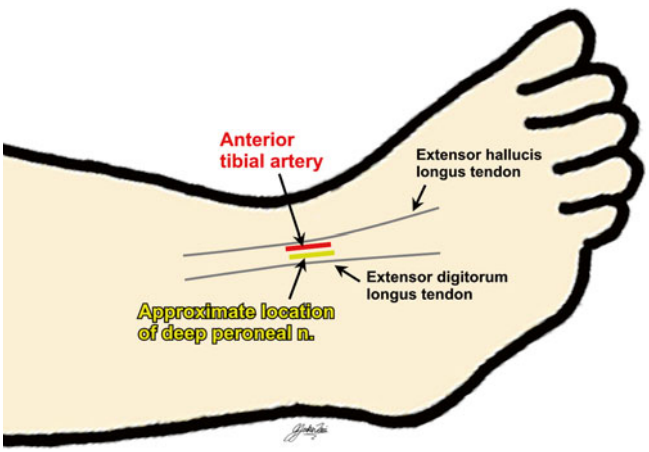


Fig. 72.15 Surface anatomy and landmarks for deep peroneal nerve block

Posterior Tibial Nerve

The probe is placed in a transverse plane posterior to medial malleolus to locate the posterior tibial artery (color Doppler can be used). The nerve, which is round or oval with a honeycomb appearance, is found posterior and deep to the artery (Fig. 72.16). Local anesthetic is deposited at the point before it divides into the medial and lateral plantar nerves.

Deep Peroneal Nerve

The probe is placed in a transverse plane on the dorsum of the foot along the line between the malleoli to locate the deep anterior tibial artery (color Doppler can be used). The nerve, which is small and may be difficult to visualize, is found lateral to the artery (Fig. 72.17).

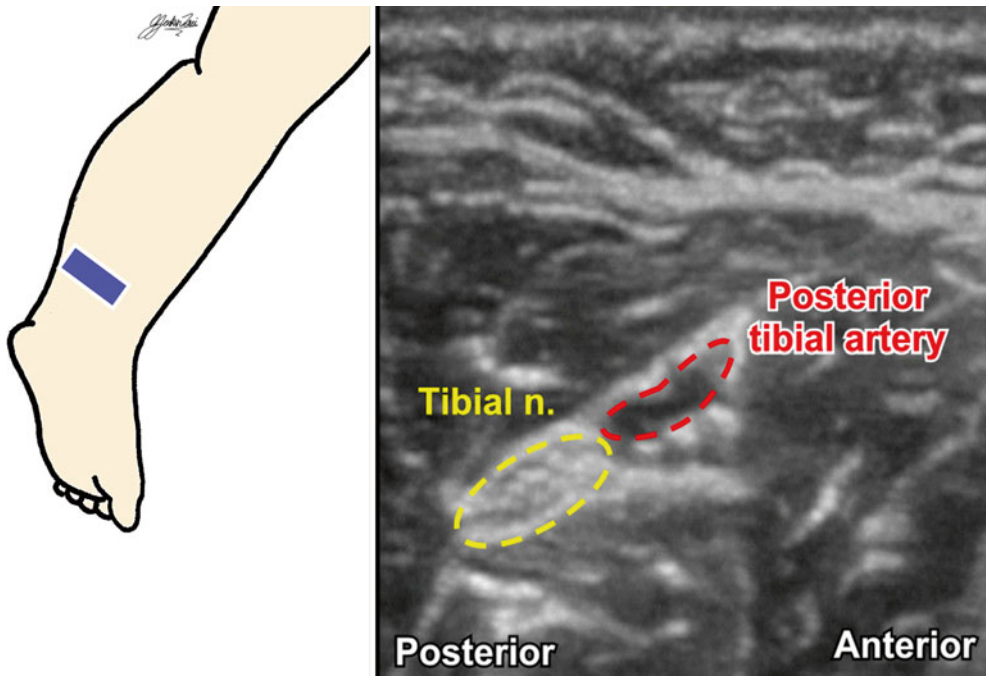


Fig. 72.16 Ultrasound image of major anatomical structures surrounding the tibial nerve

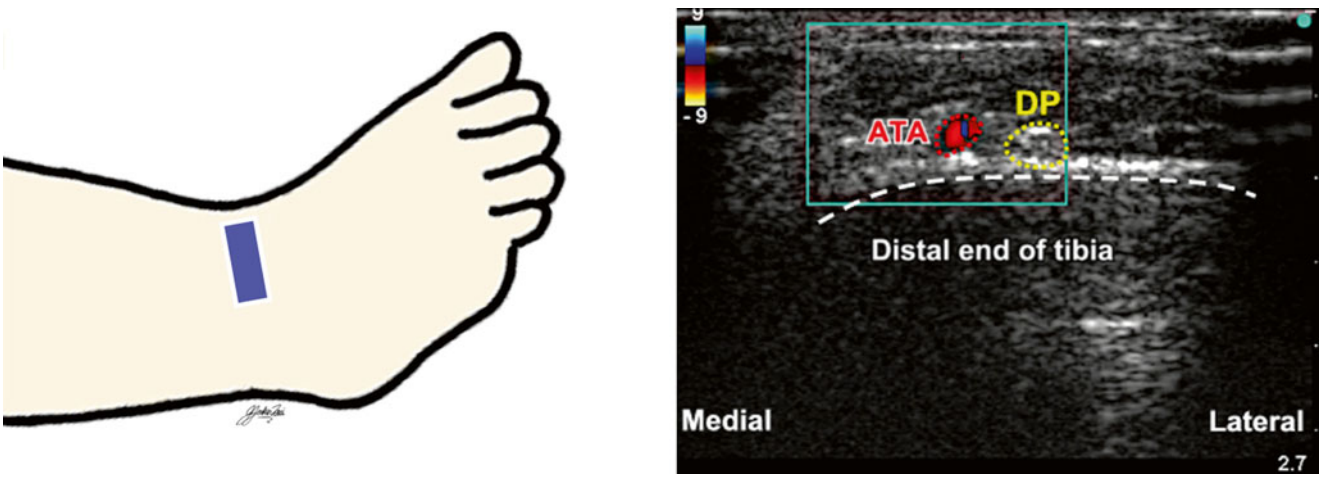


Fig. 72.17 Ultrasound image of major anatomical structures surrounding the deep peroneal nerve. *ATA* anterior tibial artery, *DP* deep peroneal nerve

Although both in-plane and out-of-plane techniques can be used for these blocks, the out-of-plane approach is often easier due to the limited space at the ankle. A "donut sign" upon injection of local anesthetic indicates good circumferential spread around the nerve.

Acknowledgments This chapter was written using Drs. Tsui and Suresh's textbook *Pediatric Atlas of Ultrasound and Nerve Stimulation-Guided Regional Anesthesia* 1st edition (Springer) as a template. The authors thank Jenkin Tsui for providing original artwork for the figures, Alex Kwan for assistance with creating and organizing the figures, and Dr. Gareth Corry for assistance with manuscript preparation and management.

References

1. Benzon HT, Sharma S, Calimaran A. Comparison of the different approaches to saphenous nerve block. Anesthesiology. 2005;102:633–8.
2. van der Wal M, Lang SA, Yip RW. Transsartorial approach for saphenous nerve block. Can J Anaesth. 1993;40:542–6.
3. Gray AT, Collins AB. Ultrasound-guided saphenous nerve block. Reg Anesth Pain Med. 2003;28:148.
4. Manickam B, Perlas A, Duggan E, Brull R, Chan VW, Ramlogan R. Feasibility and efficacy of ultrasound-guided block of the saphenous nerve in the adductor canal. Reg Anesth Pain Med. 2009;34:578–80.
5. Miller BR. Ultrasound-guided proximal tibial paravenous saphenous nerve block in pediatric patients. Paediatr Anaesth. 2010;20:1059–60.
6. Tsui BC, Ozelsel T. Ultrasound-guided transsartorial perifemoral artery approach for saphenous nerve block. Reg Anesth Pain Med. 2009;34:177–8.
7. Dolan J. Ultrasound-guided anterior sciatic nerve block in the proximal thigh: an in-plane approach improving the needle view and respecting fascial planes. Br J Anaesth. 2013;110:319–20.
8. Tsui BC, Ozelsel T. Ultrasound-guided anterior sciatic nerve block using a longitudinal approach: "expanding the view". Reg Anesth Pain Med. 2008;33:275–6.
9. Berniere J, Schrayer S, Piana F, Vialle R, Murat I. A new formula of age-related anatomical landmarks for blockade of the sciatic nerve in the popliteal fossa in children using the posterior approach. Paediatr Anaesth. 2008;18:602–5.
10. Schwemmer U, Markus CK, Greim CA, Brederlau J, Trautner H, Roewer N. Sonographic imaging of the sciatic nerve and its division in the popliteal fossa in children. Paediatr Anaesth. 2004;14:1005–8.
11. Oberndorfer U, Marhofer P, Bosenberg A, Willschke H, Felfernig M, Weintraud M, Kapral S, Kettner SC. Ultrasonographic guidance for sciatic and femoral nerve blocks in children. Br J Anaesth. 2007;98:797–801.

Suggested Reading

Jankovic D. Regional nerve blocks and infiltration therapy. Textbook and color atlas. 3rd ed. Hoboken: Malden: Blackwell; 2004.

Peutrell JM, Mather SJ. Regional anesthesia for babies and children. New York: Oxford University Press; 1997.

Saint-Maurice C, Schulte Steinberg O, Armitage E, editors. Regional anaesthesia in children. Norwalk: Appleton & Lange, Mediglobe; 1990.

Tsui BC. Atlas of ultrasound and nerve stimulation-guided regional anesthesia. New York: Springer; 2007.

Chapter 73

Pediatric Nerve Blockade: Trunk and Neuraxial

Heather Y.Z. Ting, MD, FRCPC (✉)
University of Alberta Hospital, Edmonton, AB, Canada
e-mail: yizhenhe@ualberta.ca

Sara S.K. Cheung, MBBS, FHKCA, FHKAM
University of Alberta, Edmonton, AB, Canada

Department of Anaesthesia, Pamela Youde Nethersole Eastern Hospital, Hong Kong, China
e-mail: cheungsk3@gmail.com

Ban C.H. Tsui, MSc, MD, FRCPC
Department of Anesthesiology and Pain Medicine, University of Alberta Hospital and Stollery Children's Hospital, Edmonton, AB, Canada
e-mail: btsui@ualberta.ca

Contents

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4_73,

Lumbar Plexus Block

Introduction, Indications, and Complications

The lumbar plexus is formed by the ventral rami of the lumbar roots L1–L4 with contributions from T12. The plexus is embedded in the substance of the psoas muscle. From there, the anterior and posterior divisions reunite and emerge as individual branches of nerves: ilioinguinal and iliohypogastric (L1), genitofemoral (L1 and L2), lateral cutaneous nerve of the thigh (L2 and L3), obturator (anterior divisions of L2, L3, and L4), and femoral (posterior divisions of L2, L3, and L4). Deposition of local anesthetic in the fascial plane within the psoas muscle produces blockade of the lower limb in the distribution of these nerves, i.e., the anterolateral and medial thigh, the knee, and the medial aspect of the lower leg.

This block, also known as psoas sheath block, is indicated for surgery in the distribution of the lumbar plexus (e.g., groin and anterolateral thigh). When combined with a sciatic nerve block, the entire leg can be anesthetized. It is especially useful in patients contraindicated to neuraxial block. However, it is a complex block with potential serious complications, including retroperitoneal hematoma, perforation of retroperitoneal structures, systemic toxicity, and unintentional epidural/intrathecal injection. The use of this block should be well justified, especially when simpler and safer alternatives are present. A combined approach with nerve stimulation and ultrasound guidance can improve accuracy and safety, especially for older children.

Patient Positioning, Preparation, Equipment, and Dosage

The child lies in the lateral position with hips and knees flexed. The skin is cleaned with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if an ultrasound approach is used.

A 50-mm, 22–25-G, short-beveled needle is used. The recommended local anesthetic dose is 0.2–0.5 mL/kg of 0.25 % bupivacaine or 0.2 % ropivacaine (without exceeding the toxic limit).

Nerve Stimulation Technique

Winnie's approach [1] describes the point of needle insertion as the intersection of the intercristal line (a line joining the upper borders of the iliac crests) and a line parallel to the midline and crossing the ipsilateral posterior superior iliac spine (PSIS) (Fig. 73.1).

The needle is inserted perpendicularly to the skin and advanced slowly until twitches of the quadriceps muscle are observed. The initial current is set at 1–2 mA (2 Hz) and then gradually reduced. Local anesthetic is injected after negative aspiration when the threshold current of 0.5 mA is reached. The skin-to-plexus distance correlates with weight for children aged 3–12 years old and ranging from 1.24 to 1.74 mm/kg [2].

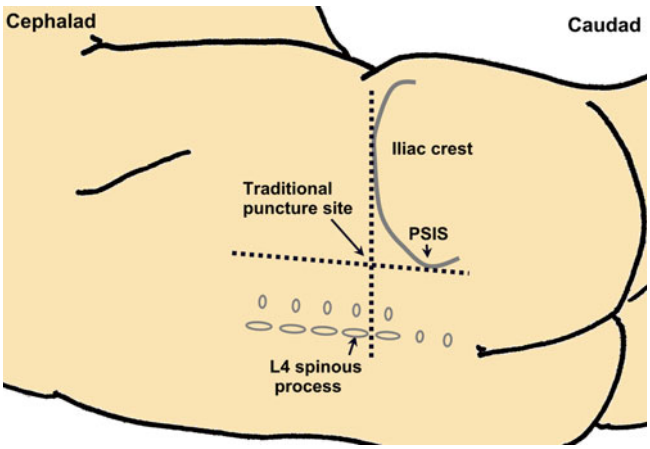


Fig. 73.1 Surface anatomy for lumbar plexus block

Ultrasound-Guided Technique

A 13-6 MHz linear "hockey stick" probe is commonly used for infants and young children. A lower-frequency (5-2 MHz) curved array probe is required for deeper penetration in older children. The probe is placed longitudinally just lateral to the midline to identify the sacrum. Scan proximally to locate the L5 transverse process and the L4/L5 interspace. From there, the probe is rotated 90° to obtain a transverse view of the spinous process, the erector spinae muscle (superficial and lateral to the spinous process), and the quadratus lumborum muscle (lateral and deep to the erector spinae muscle) (Fig. 73.2). The psoas muscle, which appears slightly hypoechoic with multiple hyperechogenic striations within (i.e., a "starry night" appearance), is located deep to the quadratus lumborum muscle, and the plexus can be found in the muscle belly of the psoas. The nerves often appear as hypoechoic "dots" surrounded by hyperechoic connective tissue. Note that the lower pole of the kidney is usually found at L2/L3 level in adolescents and can reach as low as L4/L5 in young children.

If the probe is placed longitudinally to view the transverse processes of L3/L4/L5, the psoas muscle can be seen through the acoustic window between the transverse processes, deep to the erector spinae muscle. The plexus lies within the posterior third of the psoas.

The transverse processes and the plexus may be difficult to visualize in older children because of the greater depth and poorer differentiation between muscles and nerves. It may be necessary to switch between transverse and longitudinal scanning to survey the area. Occasionally, the more superficial L3/L4 interspace can be used for better resolution.

Both in-plane and out-of-plane approaches can be used. When using the in-plane approach, the probe is placed longitudinally. The needle is inserted from the inferior end of the probe and directed cephalad to reach the psoas muscle between the L4 and L5 transverse processes. For the out-of-plane approach, the probe is placed in a transverse plane at L4/L5 interspace. The needle is introduced at 45–60° to the skin, 1 cm from the caudad end of the probe. Walk the needle down to reach the plexus while keeping the needle tip in view. Confirm the plexus using nerve stimulation.

For the "offline" technique, the lateral edges of the L4 and L5 transverse processes are marked, and the depth to transverse process is noted during the pre-procedural scan. The needle is inserted perpendicularly to the skin to a depth approximately 1 cm beyond the recorded depth of the transverse process. Locate the plexus using nerve stimulation.

In all cases, avoid directing the needle medially, which may result in unintentional epidural/intrathecal injection. Local anesthetic is injected after negative aspiration. A "flow-like" pattern within the psoas compartment may be seen.

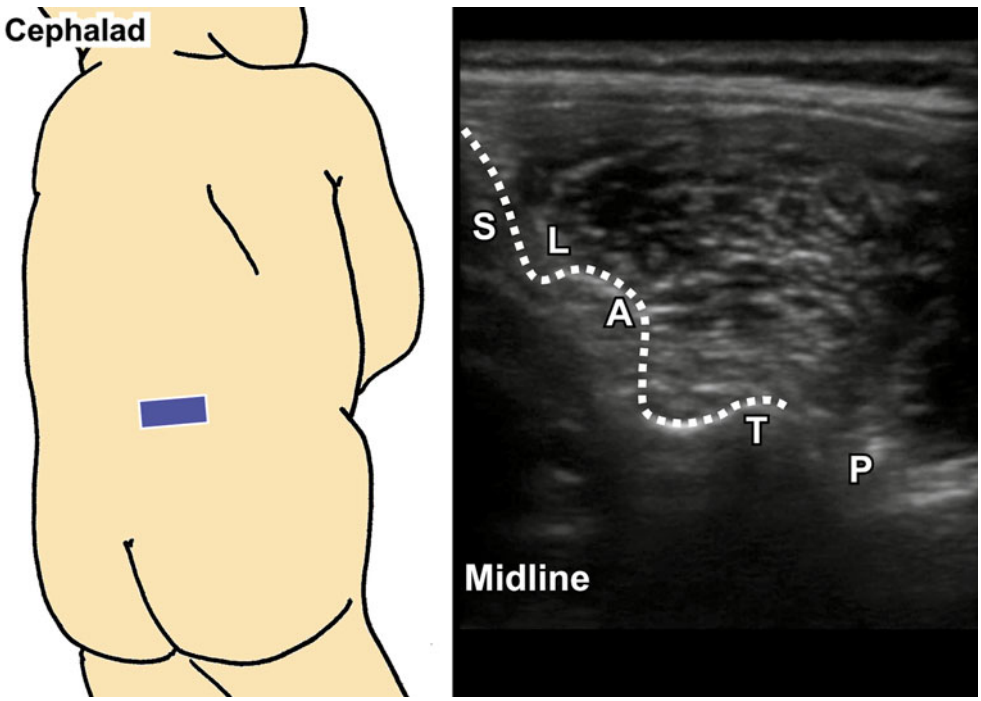


Fig. 73.2 Ultrasound image of major anatomical structures in the lumbar plexus. *S* spinous process, *L* lamina, *A* articular process, *T* transverse process, *P* psoas muscle

Paravertebral Block

Introduction, Indications, and Complications

The paravertebral space is a wedge-shaped region on either side of the vertebral column which contains the spinal nerves as they exit the intervertebral foramina. In the thoracic region, the paravertebral space is bounded medially by the vertebral body, intervertebral disc and foramen, and spinous processes; anterolaterally by the parietal pleura; and posteriorly by the anterior surface of the transverse process and superior costotransverse ligament. In the lumbar region, the space is bounded anteriorly by the psoas muscle. The paravertebral space communicates medially with the epidural space and, in the thoracic region, laterally with the intercostal space. By depositing local anesthetic into the paravertebral space, the spinal nerves on the operative side can be anesthetized, producing ipsilateral somatic and sympathetic blockade similar to a "unilateral epidural," although true epidural spread is possible due to the extension of the dural cuff. In addition to single-injection blocks, catheters can be placed to provide analgesia by continuous infusion.

This block is indicated for unilateral surgical procedures. For thoracic paravertebral block, it is commonly performed for breast and renal surgery, thoracotomy, and pain management for rib fractures, whereas lumbar paravertebral block is used in lower limb surgeries. It is an advanced block with potential serious complications, including total spinal anesthesia and, in the case of thoracic blocks, pneumothorax. The use of ultrasound has the advantages of improving the accuracy of needle placement and visualizing spread, while at the same time minimizing inadvertent puncture of the pleura or neuraxial structures.

Patient Positioning, Preparation, Equipment, and Dosage

Under general anesthesia or heavy sedation to avoid any movement, the child lies in the prone or lateral decubitus position with the back assuming a kyphotic posture. The skin is cleaned with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if the ultrasound approach is used.

A 22-G, 50-mm insulated needle is commonly used. Alternatively, an epidural set with an 18–22 G Tuohy needle can be used if seeking loss of resistance. Choices of local anesthetic include 0.125–0.25 % bupivacaine. A volume of 0.5 mL/kg with or without epinephrine (without exceeding the toxic dose) is required to cover a vertical spread of 4–5 vertebral levels. A smaller volume of 0.1 mL/kg should be used if blocks are performed individually at each level.

Landmark Technique

Palpate for the spinous processes in the midline. Locate the bony landmarks: T7 vertebra at the tip of the scapula, C7 vertebra with the most prominent spinous process, L4 vertebra on the intercristal line (L5/S1 for neonates), and S1 on the line between two PSIS (Fig. 73.3). Mark the spinous processes on the skin. Next, locate and mark the transverse processes which are 1–2.5 cm lateral to the spinous processes, depending on the child's age (see equation below). In the thoracic region, the transverse processes are generally located at one intervertebral space higher than the corresponding spinous process because of the inferior angulation of the spinous processes. In the lumbar region, the transverse processes are located lateral to the corresponding spinous process.

The needle is inserted perpendicularly to the skin to contact the transverse process. The depth of the transverse process is noted. Redirect the needle 10° superiorly or inferiorly to walk off the transverse process and advance slightly deeper (to a maximum of 1 cm). Angle the needle slightly medially to minimize risk of puncturing the lung (excessive medial angulation may result in intraforaminal injection and should be avoided). A "give," indicating a loss of resistance, is felt as the needle punctures the costotransverse ligament. When using the epidural set, loss of resistance to air or D5W can be elicited. The depth of the paravertebral space has been found to correlate with the patient's body weight. Formulae determining the distance of the thoracic paravertebral space from the skin and the spinous process have been developed [3]:

- Depth to epidural space (mm) = 0.48 × body weight (kg) + 18.7
- Distance from midline (spinous process) (mm) = 0.12 × body weight (kg) + 10.2

The lumbar paravertebral block is technically similar to the thoracic paravertebral block. However, injection at each level is recommended for effective coverage since the vertical spread of local anesthetic is limited at the lumbar level.

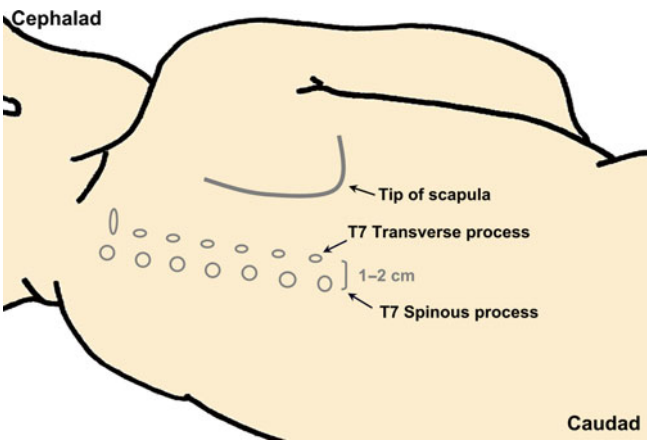


Fig. 73.3 Surface anatomy for thoracic paravertebral block

Nerve Stimulation Technique

Nerve stimulation for paravertebral block has been reported but is not commonly used for nerve localization. If nerve stimulation is used, a current of 1.5–3 mA is applied after the needle is introduced. Advance the needle slowly until appropriate motor responses (twitches in the ipsilateral thoracic/abdominal wall for thoracic paravertebral block or twitches in the ipsilateral quadriceps muscle for lumbar paravertebral block) are observed. The current is then decreased gradually, and local anesthetic is injected when a threshold current of 0.4–0.6 mA is reached. Bilateral motor responses indicate possible intrathecal/subdural placement or epidural placement when higher-threshold currents are reached.

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) linear probe is used. Both "offline" and real-time scanning can be used. The "offline" approach involves performing a pre-procedural scan to locate the point of needle insertion, depth of the paravertebral space, and needle trajectory prior to needling. The real-time approach involves performing needle insertion under real-time ultrasound guidance and is more commonly used nowadays. Similar to adults, the visibility of the paravertebral space is limited in children older than 3 years old because of the small acoustic window that results from ossification of bony elements.

The probe is placed in the transverse plane in the midline to identify the spinous process and laminae with hyperechoic outline and acoustic shadowing (Fig. 73.4 for thoracic;

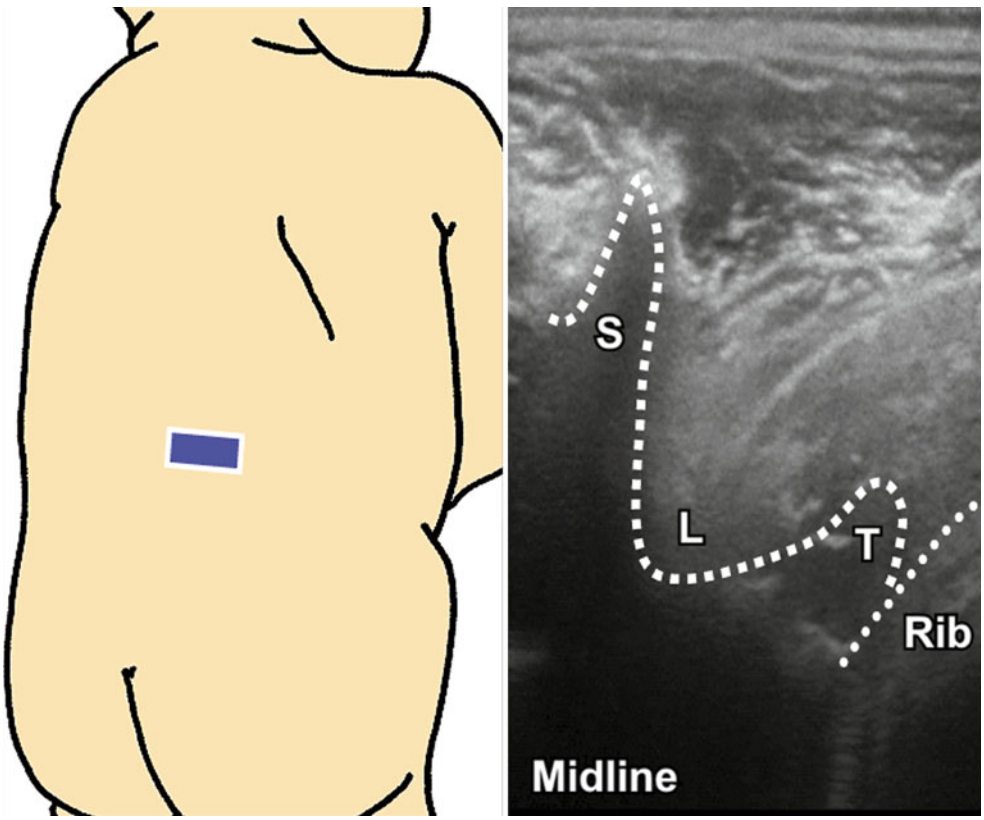


Fig. 73.4 Ultrasound image of transverse view at T9–T10. *S* spinous process, *L* lamina, *T* transverse process

Fig. 73.2 for lumbar). Locate the transverse process laterally. The lateral distance and depth of the transverse process are noted. Rotate the probe to the midline longitudinal plane to again look for the spinous processes. Scan laterally to identify the lamina, articular processes, and transverse processes (and ribs in the thoracic region) from medial to lateral. For thoracic region, the transverse processes appear as short rectangular structures with hyperechoic outlines and acoustic shadowing, as compared to the overlapping lines for lamina and "lumps" for articular processes (Fig. 73.5). The ribs often have a roundish outline instead of rectangular. The pleura appears as a hyperechoic line under the ribs. In the thoracic region, the paravertebral space can be found deep to the transverse processes between the intercostal muscle and parietal pleura. In the lumbar region, the lumbar plexus lies in the "psoas compartment" between the psoas major and quadratus lumborum muscles. The depth to parietal pleura (for thoracic paravertebral) or psoas major (for lumbar paravertebral) is noted.

Both in-plane and out-of-plane approaches can be used for blocks under real-time ultrasound guidance. If possible, the probe should be placed in the longitudinal plane when using the in-plane approach and the transverse plane when using the out-of-plane approach to avoid excessive medial angulation of the needle. Local anesthetic is deposited into the paravertebral space after negative aspiration for blood or air. For thoracic paravertebral blocks, the pleura will appear to be pushed down by the hypoechoic local anesthetic. For lumbar paravertebral block, the injectate is seen posterior to the psoas. Vertical spread (especially thoracic paravertebral block) to other levels can be seen.

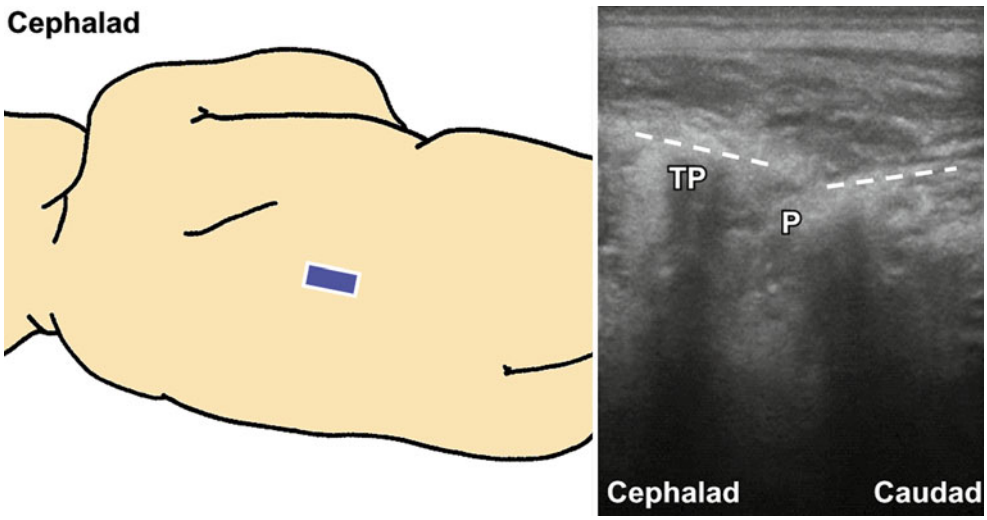


Fig. 73.5 Ultrasound image of midline longitudinal view at T9–T10. *TP* transverse process, *P* paravertebral space

Intercostal Block

Introduction, Indications, and Complications

After emerging from the intervertebral foramina, the thoracic spinal nerves divide into dorsal and ventral rami. The former supply the paravertebral region, while the latter give rise to intercostal nerves. There are 11 pairs of intercostal nerves (T1–T11) and one pair of subcostal nerves (T12). An intercostal nerve runs initially between the parietal pleura and the intercostal membrane and then passes through the membrane near the angle of the rib, travelling between the innermost and internal intercostal muscles. The nerve gives rise to the lateral cutaneous branch (which innervates the lateral trunk) near the midaxillary line and terminates as the anterior cutaneous branch (which innervates the anterior trunk) anteriorly. Within the intercostal space, the nerve travels in the costal groove underneath the intercostal vein and artery.

This block is used in pain management for rib fractures, thoracotomies, hepatobiliary and gastric surgery, and minor procedures such as chest tube insertion. Systemic toxicity is a potential risk, especially if multiple blocks are performed, since systemic absorption can be significant in the intercostal region. Pneumothorax is another possible complication.

Patient Positioning, Preparation, Equipment, and Dosage

The child lies in a lateral decubitus position. The prone position is preferred for bilateral block. The arms are extended forward so that the scapulae move laterally away from midline. The skin is cleaned with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if an ultrasound approach is used.

A 50-mm, 22–24-G short-beveled needle can be used. Extension tubing is attached to the needle to minimize movement of the needle upon respiration. Choices of local anesthetic include 0.25–1 % lidocaine and 0.125–0.25 % bupivacaine, depending on the required duration of action. A volume of 0.5–1 mL of local anesthetic is usually adequate for each level. More diluted solution should be used for small children to avoid overdose. Epinephrine 1:400,000 (with a maximum dose of 4 μg/kg) can be added to decrease systemic absorption.

Landmark Technique

Similar to paravertebral block, nerve stimulation is not commonly used for nerve localization. Depending on the location of the incision (or area of injury for fracture ribs), determine the level of the block. Multiple blocks at consecutive levels are usually needed for effective analgesia.

The midaxillary line runs longitudinally from the axilla. The posterior axillary line runs midway between the midaxillary line and the spinous processes. The lower border of the rib marks the needle insertion site for the nerve of the same level. The point of needle insertion is at the inferior border of the rib at the posterior axillary line (Fig. 73.6). With the fingers of one hand retracting the skin up and over the rib, the needle is inserted at a 20° cephalad angle with the bevel facing cephalad. Advance the needle slowly until it contacts the bone. Withdraw the needle slightly and walk off the inferior border of the rib. Apply a constant pressure to the syringe. A sudden loss of resistance can be felt when the needle enters the intercostal space. Local anesthetic is injected after negative aspiration for blood and air.

Nerve Stimulation Technique

Not commonly used for this block.

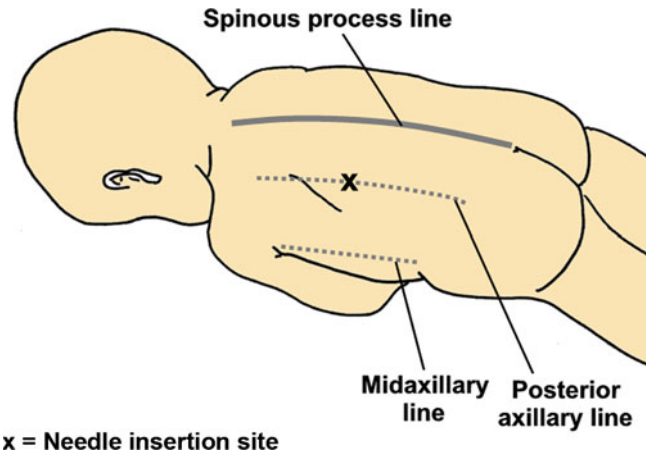


Fig. 73.6 Surface anatomy for intercostal nerve block (posterior approach)

Ultrasound-Guided Technique

A high-frequency (13-6 MHz) linear probe is used. The probe is placed along the mid- or posterior axillary line to obtain a cross-sectional view of two ribs with the intercostal muscle layers and pleura in between (Fig. 73.7). The ribs have a hyperechoic outline with acoustic shadowing, while the pleura is distinguished by its sliding movement with respiration and the hypoechoic pleural cavity underneath. The intercostal vessels, which are situated between internal and innermost intercostal muscles, can be identified on color Doppler. The intercostal nerve is found next to the vessels.

Both in-plane and out-of-plane approaches can be used. For the in-plane approach, the needle is introduced at the inferior end of the probe and directed cephalad to the nerve between the internal and innermost intercostal muscles. For the out-of-plane approach, the needle is inserted at the inferior border of the rib to reach the nerve. Local anesthetic is injected following aspiration and injection of a test dose of D5W to visualize the spread which will expand the fascial plane.

Rectus Sheath Block

Introduction, Indications, and Complications

The rectus sheath is formed from the aponeuroses of the three abdominal muscles—external oblique, internal oblique, and transversus abdominis—and encloses the rectus abdominis muscles. The left and right sheaths join at the linea alba in the midline. Above the arcuate line, the rectus muscles sit on the posterior rectus sheath derived from the aponeurosis of the internal oblique and transversus abdominis. Below the arcuate line, the posterior rectus sheath terminates and all three aponeuroses pass superficial to the rectus muscles. The lower seven intercostal nerves (T6–T12), together with the L1 nerve, innervate the anterior abdominal wall. The nerves course anteriorly, lying between the internal oblique and transversus abdominis muscles, and enter the rectus sheath at the posterolateral edge of the rectus abdominis muscle. They then penetrate through the muscle anteriorly to supply the skin.

This block is indicated for midline abdominal incisions (e.g., epigastric and umbilical hernia repair). By placing

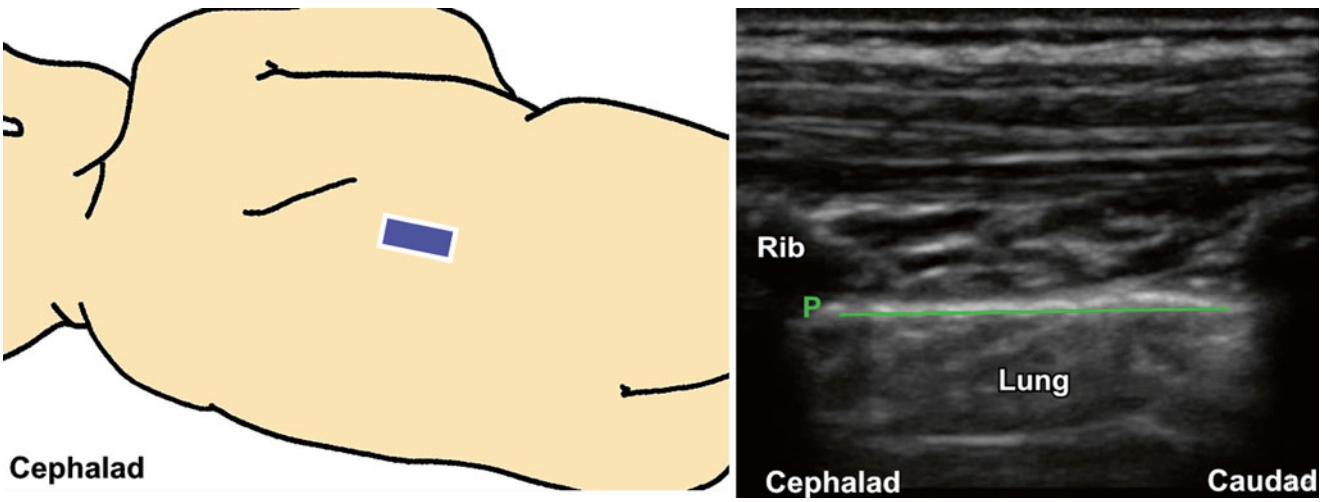


Fig. 73.7 Ultrasound image of the intercostal space. *P* pleura

local anesthetic between the rectus muscle and the posterior rectus sheath bilaterally, the paraumbilical area can be anesthetized. Although not discussed here, an umbilical nerve block can be performed for umbilical hernia repair [4]. Potential complications include peritoneal puncture and puncture of inferior epigastric vessels causing hematoma. The use of ultrasound can reduce these risks and is recommended.

Patient Positioning, Preparation, Equipment, and Dosage

With the child in supine position, the skin is cleaned with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if an ultrasound approach is used.

A 50-mm, 22-G short-beveled needle is used. Choices of local anesthetic include 0.2 % ropivacaine and 0.25–0.5 % bupivacaine. For children aged up to 7 years, 0.2 mL/kg 0.2 % ropivacaine should be used. For older children, 0.2–0.3 mL/kg 0.5 % levobupivacaine is required. The depth of the posterior sheath is usually less than 1 cm.

Landmark Technique

The point of needle insertion is at the lateral edge of the rectus abdominis muscle at the level of the umbilicus (Fig. 73.8). The needle is inserted at 60° to the skin, pointing medially. Advance the needle slowly until a "pop" or give is felt, indicating a loss of resistance as the needle pierces the anterior rectus sheath. Local anesthetic is deposited after aspiration. The block is repeated on the other side.

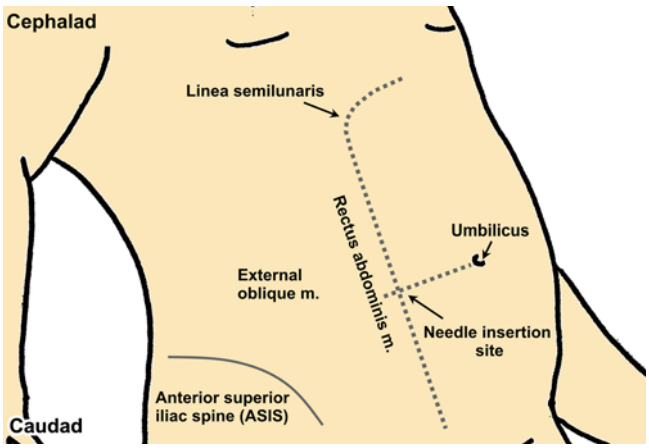


Fig. 73.8 Surface anatomy for rectus sheath block

Nerve Stimulation Technique

Nerve stimulation is rarely performed with these blocks.

Ultrasound-Guided Technique

A linear (13-6 MHz) probe is used for a penetration depth of a few cm, depending on the child's age. The probe is placed in a transverse plane on the anterior abdominal wall to first obtain the view of the three muscle layers: external oblique, internal oblique, and transversus abdominis (Fig. 73.9). Scan medially to identify the rectus muscle, which is oval shaped and enveloped by the hyperechoic rectus sheath. The muscle lies medial to the internal oblique on the same plane. Color Doppler can be used to visualize the inferior epigastric vessels which run through the rectus muscle. The target site for local anesthetic is the plane between the rectus muscle and the posterior rectus sheath (the nerve is too small to be seen).

An in-plane approach is preferred. The needle is inserted in a lateral-to-medial direction and directed to the plane between the rectus muscle and the posterior rectus sheath [5]. Avoid entering the belly of the rectus muscle (which contains the inferior epigastric vessels) by approaching from the lateral side of the muscle. A test dose of D5W is injected to confirm needle-tip position and visualize spread. Local anesthetic is deposited after negative aspiration. To achieve optimal blockade, the block can be performed at the upper and lower ends of the incision bilaterally (i.e., a total of four blocks).

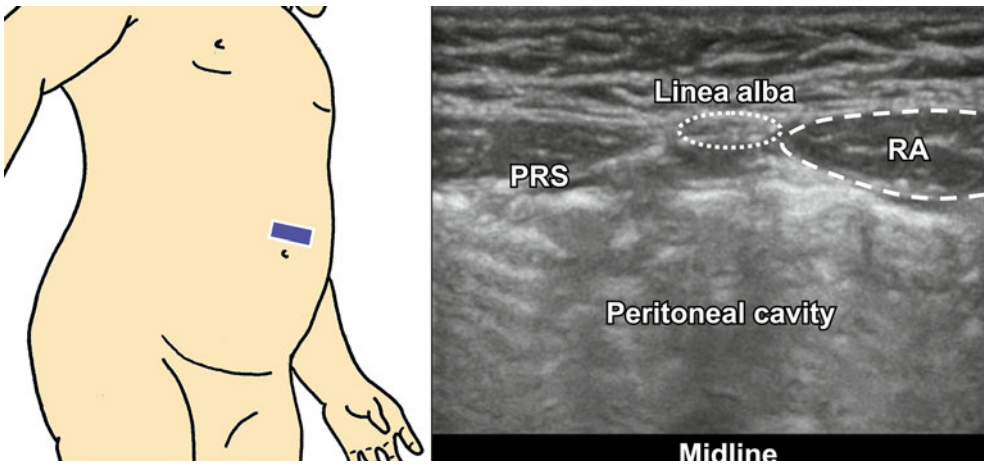


Fig. 73.9 Ultrasound image of the abdominal wall and rectus sheath. *PRS* posterior rectus sheath, *RA* rectus abdominis muscle

Transversus Abdominis Plane (TAP) Block

Introduction, Indications, and Complications

The transversus abdominis plane (TAP) is the fascial plane between the internal oblique and transversus abdominis muscles in the anterior abdominal wall. It is traversed by the lower seven intercostal nerves (T6–T12) and the L1 nerve, which innervate the anterior abdominal wall. Deposition of local anesthetic in this plane results in unilateral blockade of the skin, muscle, and parietal peritoneum of the anterior abdominal wall. This block is indicated for treatment of postoperative pain for various abdominal and pelvic surgeries and is an alternative to epidural analgesia in children with contraindications to neuraxial block. It may also substitute ilioinguinal and iliohypogastric block since blockade of the L1 nerve can be achieved.

Patient Positioning, Preparation, Equipment, and Dosage

The child can be either supine or lateral, although a more posterior approach permits a more proximal block location for better results. Clean the skin with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if an ultrasound approach is used.

A 50-mm, 22-G short-beveled needle is used. The recommended local anesthetic dose is 0.2–0.3 mL/kg of 0.25 % bupivacaine or 0.2 % ropivacaine on each side (up to a maximum of 3 mg/kg).

Landmark Technique

The triangle of Petit—an area at the lateral abdominal wall bounded anteriorly by the external oblique, posteriorly by the latissimus dorsi, and inferiorly by the iliac crest—is identified (Fig. 73.10). The internal oblique muscle forms the floor of this triangle, so it is the only area of the abdominal wall where this muscle can be localized directly. Since palpation of the triangle of Petit in an anesthetized child can be difficult, an ultrasound-guided block may be a better option.

The point of needle insertion is in the triangle at the point just anterior to the attachment of the latissimus dorsi to the external lip of the iliac crest [6]. The needle is introduced perpendicularly to the skin. Walk off the iliac crest when the needle contacts bone. Advance slowly until a "pop" or give is felt as the needle penetrates the internal oblique muscle to enter the TAP. Local anesthetic is injected after negative aspiration. The block is repeated on the other side for a midline or transverse incision.

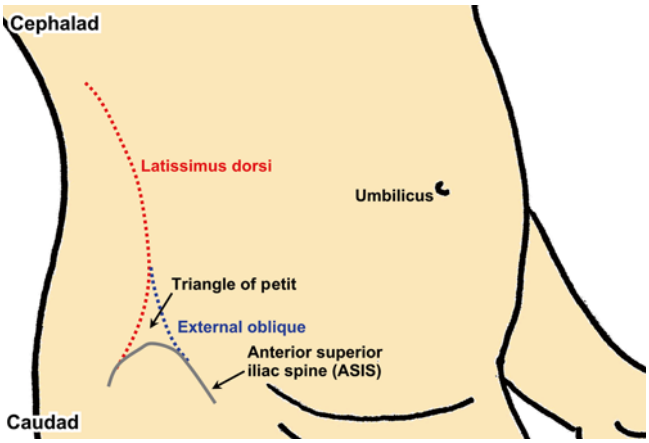


Fig. 73.10 Surface anatomy for transversus abdominis plane block

Nerve Stimulation Technique

Nerve stimulation is not used for these blocks.

Ultrasound-Guided Technique

A linear (13-6 MHz) probe is used for a penetration depth of a few cm. Occasionally, a curved probe is needed for obese patients. The probe is placed in a transverse plane on the midaxillary line, between the costal margin and the iliac crest, to visualize the three muscle layers separated by the hyperechoic fascia: external oblique, internal oblique, and transversus abdominis (Fig. 73.11). The internal oblique is the widest muscle layer, while the transversus abdominis is the thinnest. The peritoneum and bowel are seen underneath the transversalis fascia deep to the transversus abdominis.

Both in-plane and out-of-plane approaches can be used. For the in-plane approach, the needle can be inserted in a medial-to-lateral or lateral-to-medial direction to reach the TAP. A "pop" or give can be felt as the needle passes through the fascial plane. Local anesthetic is deposited after negative aspiration and following injection of a test dose of D5W to visualize spread and confirm needle-tip position. When using an out-of-plane approach, the needle is introduced at the cephalad edge of the probe and directed caudally [7]. The needle then "walks down" to reach the TAP while keeping the needle tip in view.

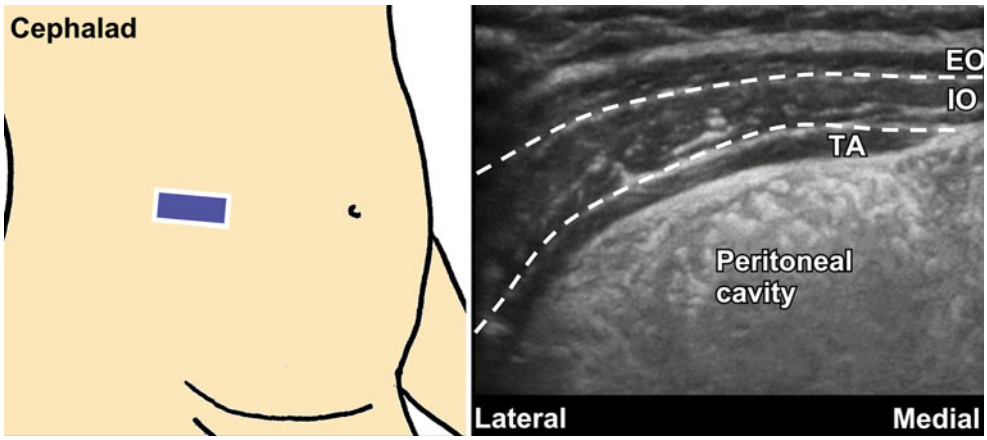


Fig. 73.11 Ultrasound images of the abdominal muscles and transversus abdominis plane. *EO* external oblique, *IO* internal oblique, *TA* transversus abdominis muscle

Ilioinguinal and Iliohypogastric Nerve Blocks

Introduction, Indications, and Complications

The ilioinguinal and iliohypogastric nerves are derived from the anterior division of the L1 nerve root. At or near the level of the anterior superior iliac spine (ASIS), the nerves pierce the transversus abdominis to lie between it and the internal oblique muscle in the transversus abdominis plane (TAP). The iliohypogastric nerve runs within the TAP plane to supply the inguinal region, while the ilioinguinal nerve travels anteroinferiorly and pierces the internal oblique muscle at the superficial inguinal ring to supply the root of the penis, scrotum, and a small area of the medial thigh below the inguinal ligament.

These blocks are commonly indicated for inguinal operations; however, they are inadequate to provide surgical anesthesia for hernia repair per se because they do not cover areas innervated by the genitofemoral nerve and contralateral branches. In addition, the spermatic cord and peritoneum are not anesthetized. Alternatively, a TAP block can be performed to block the nerves proximally.

Patient Positioning, Preparation, Equipment, and Dosage

With the child lying supine, the skin is cleaned with antiseptic solution. If an ultrasound approach is used, prepare the probe surface by applying a sterile adhesive dressing.

A 50-mm, 22–27-G short-beveled needle is used. Recommended local anesthetics are 0.25 % bupivacaine and 0.5 % bupivacaine with a volume of 0.3–0.5 mL/kg (or 0.2 mL/kg into each fascial plane if the nerves are not visible).

Landmark Technique

The classical description of needle insertion is 0.5–2 cm medial and 0.5–2 cm below the ASIS (Fig. 73.12). Distance to ASIS varies depending on the age and size of the child. The needle is introduced perpendicular to the skin and advanced slowly. Two "clicks" are felt as the needle passes through the external and internal oblique muscles (often, only one "click" is felt). Following negative aspiration, local anesthetic is deposited after the first "click" (between the external oblique and the internal oblique muscles) and second "click" (between the internal oblique and transversus abdominis). Inject local anesthetic from cephalad to caudad in a fan-like pattern. Upon withdrawal of the needle, 0.5–1 mL of local anesthetic is deposited subcutaneously. Alternatively, the needle can be inserted at 45° angle to the midline at 2.5 mm from the ASIS on a line between the ASIS and the umbilicus (Fig. 73.12) [8]. However, it is important to point out that, in our practice, we prefer to stay as close to the ASIS as possible (one finger-breadth of patient's index finger size).

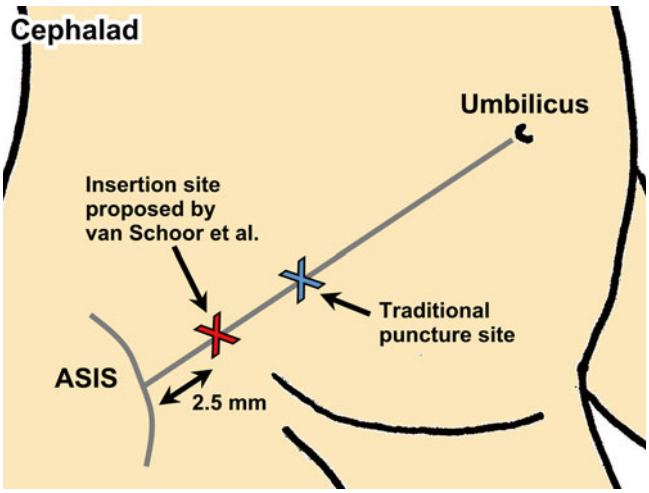


Fig. 73.12 Surface anatomy for ilioinguinal nerve block. *ASIS* anterior superior iliac spine

Nerve Stimulation Technique

Nerve stimulation is rarely performed with these blocks.

Ultrasound-Guided Technique

A linear (13-6 MHz) probe is commonly used. In older/obese children, a curved probe may be necessary. The probe is placed just medial to the ASIS, in an oblique plane parallel to a line joining the ASIS and the umbilicus (Fig. 73.13). The three muscle layers (external oblique, internal oblique, and transversus abdominis) are identified. The peristalsis of the bowel can be seen underneath the peritoneum. The ilioinguinal and iliohypogastric nerves, which are often seen side by side as hyperechoic ovals, can be found either in the fascial plane between the external and internal oblique muscles or between the internal oblique and transversus abdominis. The deep circumflex iliac artery (as identified on color Doppler) often lies adjacent to the nerves in the same plane.

Both in-plane and out-of-plane approaches can be used. For the in-plane approach, the needle is introduced in a medial-to-lateral direction at the medial end of the probe and angled laterally to reach the nerves at a shallow angle. "Clicks" can be felt as the needle passes through the fascial layers. If the nerves are not seen well, a fascial block can be performed to deposit local anesthetic at the two fascial planes that carry the nerves. A test dose with D5W prior to injection of local anesthetic is useful in visualizing spread as well as confirming the position of the needle tip. When using the out-of-plane approach, the needle is inserted at 45° to the skin at the same distance as the depth of the nerves and "walked down" to reach the target while keeping the needle tip in view. Local anesthetic is deposited after negative aspiration.

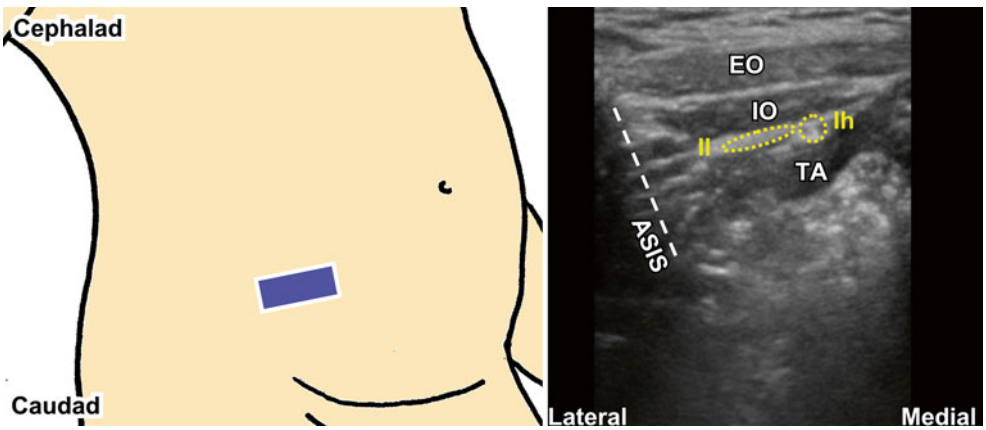


Fig. 73.13 Ultrasound images of the ilioinguinal and iliohypogastric nerves and major landmarks. *EO* external oblique, *IO* internal oblique, *TA* transversus abdominis muscle, *Il* ilioinguinal nerve, *Ih* iliohypogastric nerve, *ASIS* anterior superior iliac spine

Epidural Anesthesia

Lumbar and Thoracic Epidural Anesthesia

Introduction, Indications, and Complications

Several age-specific differences exist between adult and pediatric patients in terms of anatomy. The conus medullaris ends at a lower level in younger children (L2–L3 in infants, L1–L2 in 1-year-olds) compared to adults (T12–L2) [9]. Likewise, the dural sac terminates at a more caudad position in neonates and infants (S3) compared to adults (S1). The sacrum is incompletely ossified, and the sacral intervertebral spaces can be felt. The epidural space is loosely packed, allowing an epidural catheter to be threaded cranially to the thoracic level easily from lumbar or caudal approaches. The depth to epidural space is approximately 1 cm in neonates.

In children, performing an epidural block is technically more difficult because the landmarks and the distance between tissue planes are smaller. Also, the procedure often has to be performed under general anesthesia or heavy sedation, rendering monitoring for sensory warning signs of spinal cord placement impossible, further increasing the risk of spinal cord injury. Nowadays, the use of nerve stimulation and ultrasound allows better localization of the epidural space, improving the success rate and safety of epidural insertion. However, the use of ultrasound is limited for children over 1 year old because visualization of the epidural space is difficult. Therefore, placement of a thoracic epidural catheter via the direct thoracic approach is rarely performed. A safer way to block the thoracic dermatomes is to insert an epidural catheter at the lumbar or caudal level and thread it to the thoracic level under guidance of the epidural stimulation test.

Epidural anesthesia is indicated for treatment of postoperative pain in major surgery involving the thorax, abdomen, pelvis, and lower limb. Occasionally, it is used to provide surgical anesthesia. Although both single-injection and continuous catheter techniques can be used, the continuous catheter technique offers prolonged postoperative analgesia and is more commonly performed. Contraindications of epidural insertion include patient refusal, deranged coagulation, local infection or septicemia, uncorrected hypovolemia, and raised intracranial pressure.

Patient Positioning, Preparation, Equipment, and Dosage

The child lies in the lateral position. A sitting position can be adopted if the child is awake and cooperative. Clean the skin with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if an ultrasound approach is used.

The size of the Tuohy needle and syringe is chosen according to the size of the child. A 50-mm, 19-G Tuohy needle is used for smaller children, while an 18-G needle is suitable for larger children. The recommended local anesthetic dose is 0.5–1 mL/kg of 0.25 % bupivacaine or 0.5 % ropivacaine as a loading dose. Otherwise, the commonly used infusion rate for neonates is 0.2 mL/kg and for infants and children is 0.4 mL/kg per hour with 0.1 % bupivacaine plus 1 μg/mL fentanyl.

Landmark Technique

Palpate the spinous processes along the midline (two separate bony landmarks may be felt in infants because the neural arches are incompletely fused). Locate the bony landmarks: T7 vertebra at the tip of the scapula, C7 vertebra with the most prominent spinous process, L4 vertebra on the intercristal line (for older children) (Fig. 73.3), and S2 on the line between two PSISs (Fig. 73.1). Use these landmarks to identify the target intervertebral space.

Both median and paramedian approaches can be used. The median approach is typically used in the lumbar region, while the paramedian approach is commonly performed for thoracic epidurals since the median approach requires an acute insertion angle due to the angle of the spines. The paramedian technique in the thoracic region requires a higher skill level and is more difficult to master. The technique involves inserting the Tuohy needle 1–2 fingerbreadths (patient's finger) lateral to the spinous process. The needle is advanced perpendicularly to the skin until it contacts the lamina and is then angled 15° medially. From there, the needle is redirected cephalad and "walked off" the lamina slowly into the epidural space.

Loss of resistance to either air or saline can be used to identify the epidural space. Identification of the epidural space is more difficult in infants because the ligamentum flavum is soft and therefore less resistant. Alternatively, the hanging drop technique can be used to identify the space but it is less accurate in sedated patients in a lateral position. Catheter placement is confirmed by the electrical epidural stimulation test. Local anesthetic is injected after negative aspiration for blood and cerebrospinal fluid (CSF). If in doubt of intravascular placement, administer a test dose of epinephrine (0.5 μg/kg) and observe for specific ECG changes (i.e., >25 % increase in T wave or ST segment changes, irrespective of chosen lead). For lumbar epidurals, the distance from the skin to the epidural space can be estimated as 1 mm/kg or [10 + (age in years × 2)] mm.

Epidural Stimulation Test and ECG Monitoring Technique

The epidural stimulation test (Tsui test) [10] can confirm epidural catheter placement and has a positive predictive value of 80–100 %. The test allows guidance of catheters to an appropriate position within two segmental levels. Moreover, it is useful for detecting intrathecal, subdural, or intravascular catheter placement. Additionally, the test can be applied on epidural needles to confirm correct needle placement [11].

The test is based on the current versus nerve–distance relationship. By stimulating the spinal nerve roots via the epidural stimulating catheter, motor responses observed upon a current of 1–10 mA indicate that the epidural catheter tip is within 1–2 cm from the nerve roots. The location of the motor responses (upper limbs, trunk, or lower limbs) also represents the segmental level of the catheter tip. Twitches upon a current of <1 mA signifies the tip is either abutting a nerve root or is in the intrathecal or subdural space. To exclude intravascular placement, a test dose of local anesthetic is injected. Motor response intensity observed at the same current indicates intravascular injection.

To perform the test, connect the epidural stimulating catheter to a nerve stimulator via an electrode adaptor such as Johans ECG adaptor. Prime the catheter and adaptor with normal saline. The grounding anode is attached to the child's body surface (upper limbs for lumbar epidurals and lower limbs for thoracic), while the cathode is connected to the metal hub of the adaptor. The nerve stimulator is initially set to a frequency of 2 Hz and a pulse width of 0.2 ms. Increase the current slowly (up to 10 mA for lumbar and caudal catheters and 17 mA for thoracic catheters) until motor responses of the corresponding segment are observed, confirming epidural placement. Interpretations of various responses to the test are shown in Table 73.1.

The ECG monitoring technique is used to confirm catheter tip position for paralyzed patients or when local anesthetics have been given via the epidural catheter [12]. Firstly, attach left-leg and left-arm ECG leads according to their standard positions. Next, place the right-arm lead on the child's back at the target segmental level and obtain a reference ECG waveform. Then, the right-arm lead is attached to the metal hub of the electrode adaptor to obtain the epidural ECG waveform. Compare the amplitude of epidural ECG to that of the reference ECG. The amplitudes of the two waveforms will be similar if the tip of the epidural catheter is within two vertebral spaces of the target segmental level. It is important to note that this technique cannot detect intrathecal or intravascular catheter placement.

Ultrasound-Guided Technique

The use of ultrasound allows evaluation of the location, depth, and direction to the epidural space to guide epidural needle insertion. This reduces the number of puncture attempts and improves safety. In the pediatric population, neuraxial structures are better visualized in children up to 3 months of age.

A low-frequency (5-2 MHz) probe is usually required for deeper penetration. Different structures can be viewed with different ultrasound scanning planes. The lower lumbar region has the largest acoustic window compared to high lumbar or thoracic regions. Flexing the spine helps to open the acoustic window for better visualization of neuraxial structures. Common scan planes are:

- Transverse scan (Fig. 73.14a): The spinous process and the lamina appear as an inverted "V" with hypoechoic shadows. In young children, the hyperechoic dura containing anechoic CSF can be visualized inside the spinal canal. The cauda equina may appear as fibrillar structure. In the thoracic region, the hypoechoic spinal cord may be seen.
- Paramedian longitudinal scan (Fig. 73.14b): The lamina and the facet are identified with intervertebral spaces in between. Within the intervertebral spaces, the hyperechoic dura can be found with underlying anechoic CSF. The ligamentum flavum, which marks the epidural space, may be seen on the dorsal aspect of dura. This scan plane provides the best acoustic window with good visibility of the dura.
- Midline longitudinal scan (Fig. 73.14c): The spinous processes are seen with interspinous spaces. In the thoracic region, the long spinous processes obscure the acoustic window through the interspinous spaces.

Both "offline" (pre-procedural) scanning and real-time approaches can be used. Pre-procedural scanning can be used to locate the point of needle insertion, depth of epidural space, and needle trajectory prior to needling.

When performing "offline" scanning, both transverse paramedian and longitudinal paramedian scanning can be

Table 73.1 Motor responses and currents associated with catheter location during electrical epidural stimulation test

Catheter location	Current	Motor response
Subcutaneous	N/A	No motor response
Subdural	<1 mA	Bilateral (many segments)
Subarachnoid	<1 mA	Unilateral or bilateral
Epidural space		
Non-intravascular	1–15 mA (threshold increases upon local anesthetic injection)[a]	Unilateral or bilateral
Intravascular	1–15 mA (no change in threshold upon local anesthetic injection)	Unilateral or bilateral
Against nerve root	<1 mA	Unilateral

[a]These currents are more reliable for caudal and lumbar placement; thoracic placement may require higher upper limits (e.g., 17 mA). Lower threshold limits apply to both catheter and needle placement

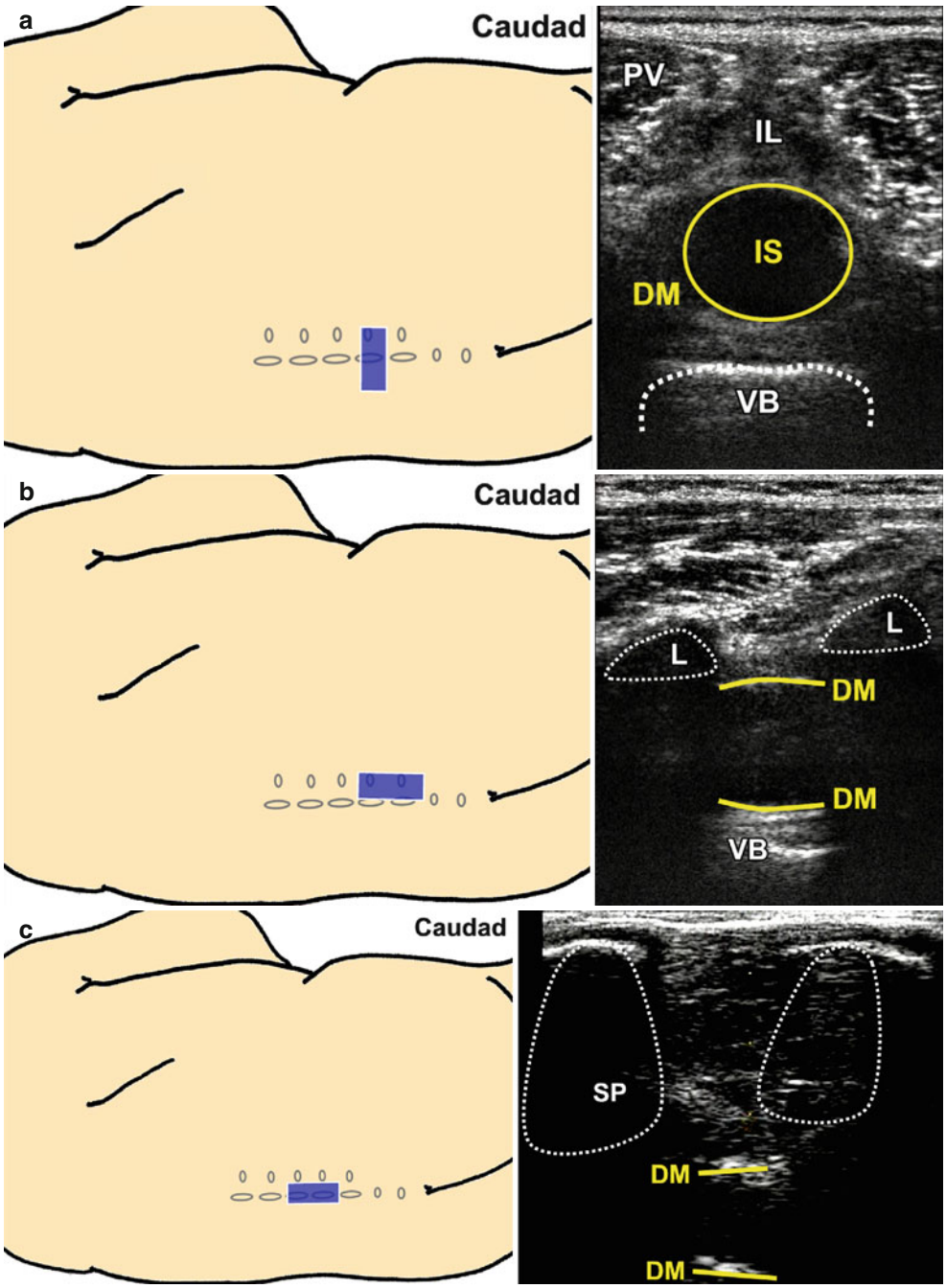


Fig. 73.14 Sonographic appearance of the lumbar spine in a 10-year-old. (**a**) Transverse view; (**b**) paramedian longitudinal view; (**c**) midline longitudinal view. *PV* paravertebral muscles, *IL* intraspinous ligament, *IS* intrathecal space, *DM* dura mater, *VB* vertebral body, *L* lamina

helpful to survey the anatomy. The neuraxial anatomy is evaluated, and the location and depth of the epidural space is assessed. The rule of thumb is that if the dura is seen, the depth of the epidural space is slightly less than the skin-to-dura distance, and if only the lamina is seen, the depth of the epidural space is slightly greater than skin-to-lamina distance.

For the real-time approach, the probe is placed (ideally by an assistant; alternatively, an automotive device can be used) in the longitudinal paramedian plane. The needle is inserted at the midline. The needle (midline) and probe (paramedian) are out-of-plane, so only the needle tip may be visualized. Advance the needle while feeling for loss of resistance. The needle may be seen piercing through the ligamentum flavum. To confirm the correct placement of the needle into the epidural space, look for dural movement and widening of the epidural space upon injection of saline. Introduce the catheter once needle position is confirmed. The tip of the catheter may be identified indirectly by dural movement and directly by local anesthetic spread.

Caudal Epidural Anesthesia

Introduction, Indications, and Complications

Caudal epidurals can be performed either as single-injection blocks or as a means to thread a catheter to a more cranial location. Single-injection caudal blocks will anesthetize the lumbar and sacral regions; indications include urogenital and lower limb surgeries. Penetration of the sacral hiatus and sacrococcygeal membrane (indicated by a "pop") is a reliable indicator of correct placement.

Patient Positioning, Preparation, Equipment, and Dosage

The patient is placed in the lateral position. Clean the skin with antiseptic solution. Prepare the probe surface by applying a sterile adhesive dressing if an ultrasound approach is used. If nerve stimulation is being used, an insulated 22 G needle will be required. The recommended local anesthetic dose is 0.5–1 mL/kg of 0.25 % bupivacaine or 0.5 % ropivacaine as a single injection. The infusion rate is similar to lumbar and thoracic epidural (see above).

The iliac crests and posterior superior iliac spines are identified. In older children, the median sacral crest and sacral hiatus may also be identified. The sacral hiatus is at the midpoint between the sacral cornua, which can be palpated ~5 cm above the tip of the coccyx (Fig. 73.15).

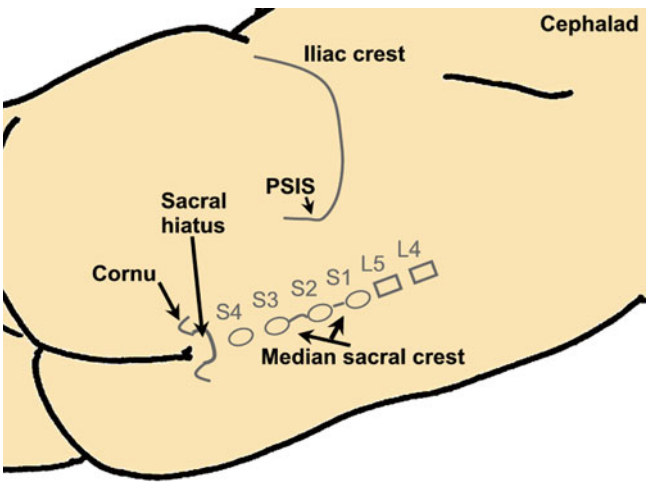


Fig. 73.15 Surface landmarks for caudal epidural needle insertion

Nerve Stimulation Technique

The nerve stimulator should be set to a low frequency (1–2 Hz) and pulse width (0.1–0.2 ms). Insert the needle perpendicular to the skin at the level of the sacral cornua. As the needle is advanced, a distinct "pop" or give is felt, indicating entry into the caudal epidural space. The current is increased until contraction of the anal sphincter (S2–S4) is seen. The threshold current will generally be between 1 and 15 mA (Table 73.1) [13]. Incorrect motor responses, such as localized twitches in the gluteal or back muscles, and higher currents (>8 mA) are suggestive of subcutaneous needle-tip placement. Following negative CSF aspiration, a test dose of epinephrine (0.5 μg/kg) should be delivered to identify inadvertent intravascular placement; this will appear as distinct ECG changes (i.e., >25 % T wave or ST segment increase). Other indicators of incorrect needle placement include subcutaneous bulging or resistance upon injection of local anesthetic.

Ultrasound-Guided Technique

Linear probes can be used for most patients. For older patients, curvilinear array probes with lower frequencies may provide better views in older or obese patients. Small footprint ("hockey stick") probes can be used in smaller patients.

For most patients, scanning should begin with a 13-6 MHz linear probe in a short-axis, transverse view at the S5 level. This will allow visualization of the sacral cornua, dorsal surface of the sacrum, posterior sacrococcygeal ligament, and sacral hiatus. Following skin puncture, the probe is rotated 90° to obtain a longitudinal (long-axis) view between the cornua. This will facilitate an in-plane view of needle trajectory toward the sacrococcygeal ligament. The distinctive "pop" will indicate needle entry into the epidural space. Rotate the probe to a transverse view to show the needle in short axis (dot on the screen) between the sacrococcygeal ligament and pelvic (ventral) surface of the sacrum. Limited ossification in young infants (<1 year old) will allow a 13-6 MHz linear probe to be placed over the mid-sacral level; longitudinal views will allow visualization of needle trajectory. Neuraxial structures at the level of the sacral hiatus appear as follows:

- Transverse view (Fig. 73.16a): The sacral cornua appear as hyperechoic, inverted U-shaped structures. Two hyperechoic bands deep to and between the cornua are the posterior sacrococcygeal membrane and dorsum of the pelvic surface of the sacrum. Between these two structures lies the hypoechoic sacral hiatus.
- Longitudinal view (Fig. 73.16b): The sacrococcygeal ligament is angled caudally and appears broad, hyperechoic, and linear. The dorsal surface of the sacrum, deep and cephalad to the ligament, is hyperechoic, while the dorsum of the ventral sacral surface is dark at the bottom of the image with a hypoechogenic region (sacral hiatus) between the sacrococcygeal ligament and sacral bone.

Upon injection, local anesthetic will appear as an expansion of hypoechogenicity and will also cause movement of the dura. There is a risk of dural puncture when performing caudal epidurals. The "Whoosh" [14] and "Swoosh" [15] tests, electrical stimulation [16], and Doppler imaging [17] can be used to determine whether injection is in the intrathecal or epidural space.

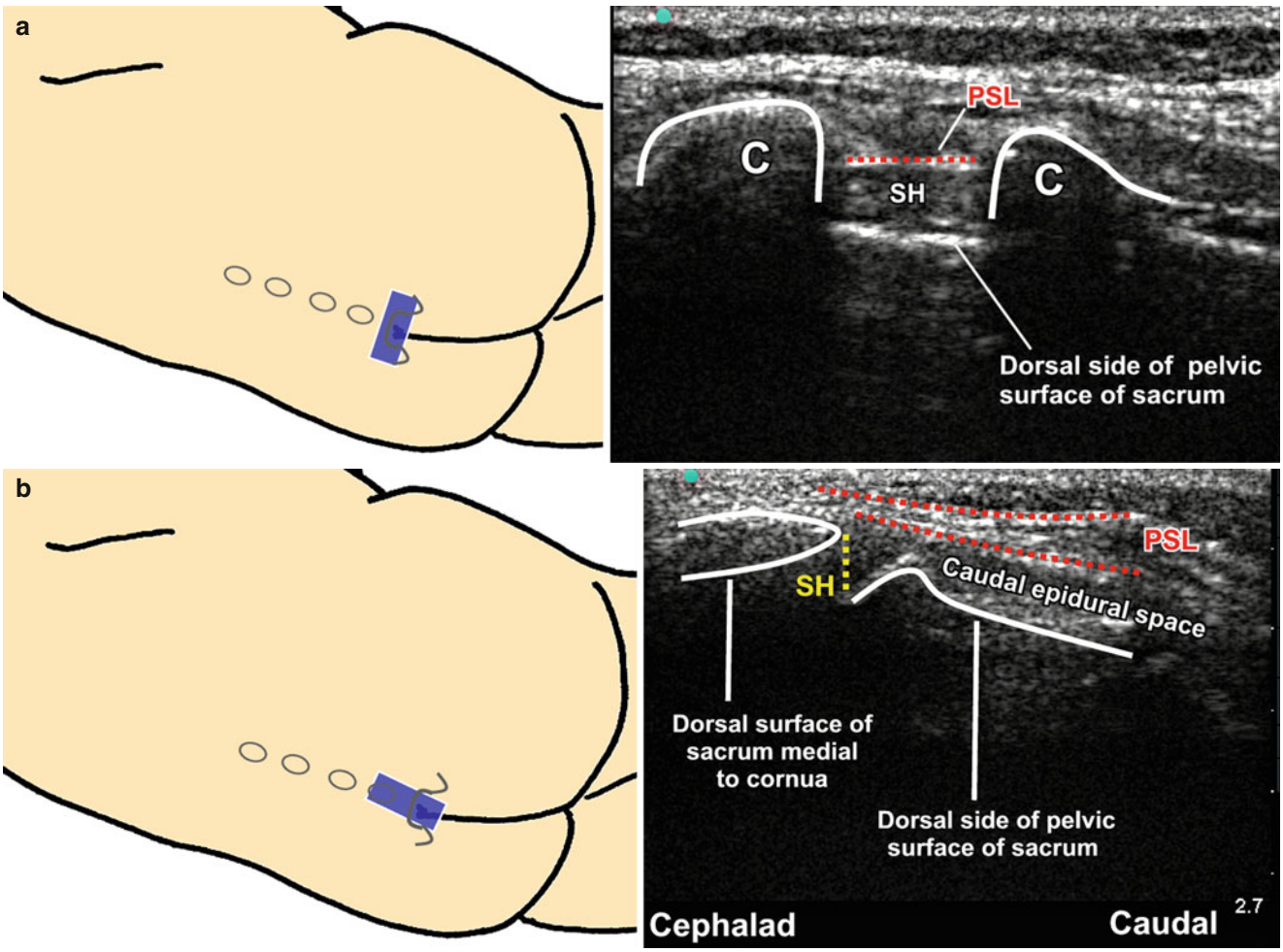


Fig. 73.16 Sonographic appearance of the sacral region. (**a**) Transverse view; (**b**) longitudinal view. *C* sacral cornu, *SH* sacral hiatus, *PSL* posterior sacrococcygeal ligament

Spinal Anesthesia

Introduction, Indications, and Complications

In the pediatric population, spinal anesthesia is used primarily to avoid complications and risks associated with general anesthesia. Spinal blocks are generally performed in neonates or small infants, especially preterm babies with a history of apnea, bradycardia, and/or chronic lung disease who would otherwise receive a general anesthetic. Spinal blockade offers some important advantages over other forms of anesthesia, including avoidance of airway manipulation, rapid onset, and speedy postoperative recovery. Despite being used mostly in infants, spinal anesthesia can be administered to pediatric patients of all ages for a wide range of surgical procedures [18].

In pediatric patients, the depth to the subarachnoid space is significantly reduced in younger children; estimates of this depth increase from 10 to 15 mm at birth to 20, 25, and >30 mm at 3, 5, and 10 years old, respectively [19–21]. Finally, CSF volume is greater in infants compared to adults (4 mL/kg vs. 2 mL/kg), and, in infants, a greater proportion of CSF is in the spinal canal [22]. These differences may explain observations of shorter duration of action of spinal anesthetics in infants despite large anesthetic doses.

Spinal blocks are used primarily for lower abdominal, urologic, and lower extremity surgery. Important considerations to keep in mind include:

1. Coagulation status: Neonates and infants less than 1 year old do not have the necessary clinical history to reveal coagulation insufficiency. Before performing spinal anesthesia on an ex-preterm neonate, it is recommended to obtain coagulation status; test results should be compared to age-specific ranges [23].
2. Surgery duration: When used as a sole, single-injection anesthetic, spinal block will last up to approximately 90 min. If surgery is anticipated to last more than 1 h, supplementary sedation or general anesthetic may be required.

Absolute contraindications to spinal block in children include patient/parental refusal, confirmed or suspected coagulopathy, local or systemic infection, hypovolemia, and increased intracranial pressure. Other contraindications are spinal abnormalities, degeneration or disease affecting the central nervous system, and presence of ventriculoperitoneal shunts or intrathecal catheters.

The most common complications of pediatric spinal anesthesia are need for multiple attempts, sensory/motor block failure requiring supplementary anesthetic, and anesthesia wearing off before surgery is complete. Less common complications include bleeding or hematoma, infection, allergic reaction, local anesthetic toxicity, cardiovascular problems, and nerve injury. When tetracaine is used, there is a risk of methemoglobinemia.

Patient Positioning, Preparation, Equipment, and Dosage

Thirty minutes before the spinal block is given, topicalize the skin using *e*utectic *m*ixture *l*ocal *a*nesthetic (EMLA) cream (2.5 % each lidocaine and prilocaine) can be applied 1 h prior to the procedure. The topicalizing agent may be covered with a Tegaderm® dressing. Intravenous (i.v.) access should be established before proceeding with the spinal to (a) avoid delay or complications arising from difficulty gaining vascular access, (b) allow premedication with atropine (10 μg/kg) prior to administering the spinal, and (c) facilitate easier management of unforeseen complications of the spinal, including high/total spinal or profound apnea.

The patient may be placed in a lateral decubitus or sitting position, depending on the anesthesiologist's preference. A trained assistant will be needed to help in both cases.

1. Lateral position: With the patient's legs flexed at the knees and hips, the neck and shoulders are flexed forward gently while maintaining an open airway. The flexed position allows palpation of bony landmarks and "opens" the spine, allowing easier access to the area between the spinous processes. The lateral position may aid in creating a better flexed position in an awake infant or sedated older child.
2. Sitting position: The assistant holds the patient in a sitting position, keeping the patient's hips flexed and head flexed forward. Cooperative older children may assume the sitting position themselves with support from the assistant. The sitting position is advantageous since it increases CSF pressure in the lumbar spine, improving CSF flow through the spinal needle.

Three important landmarks are identified (Fig. 73.17):

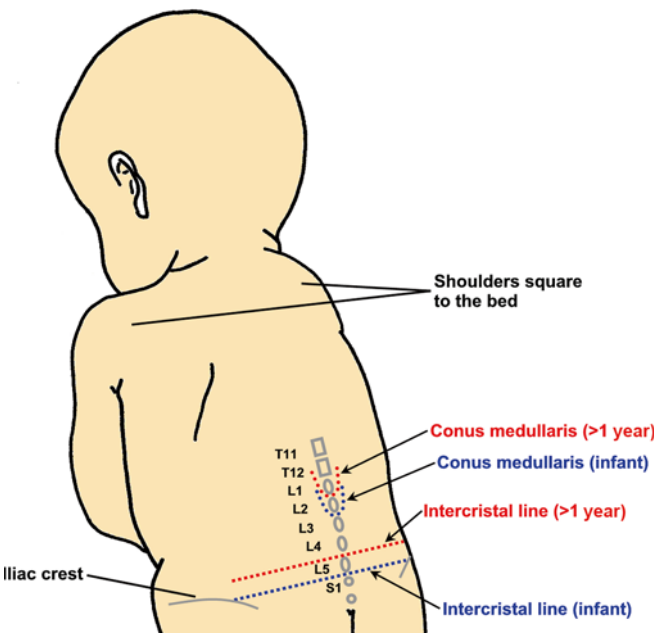


Fig. 73.17 Surface landmarks for spinal anesthesia. Positions of the conus medullaris and intercristal line are shown for infants less than 1 year old (*blue*) and children over 1 year old (*red*)

Table 73.2 Common local anesthetics and adjuvants for pediatric spinal anesthesia

Local anesthetic	*Dose*
Bupivacaine	0.3–1.0 mg/kg
Tetracaine	0.4–1.0 mg/kg
Additives	*Dose*
Epinephrine (extend block duration)	0.01 mL/kg of 1:100,000 diluted solution (epinephrine washout of a tuberculin syringe may be preferred to standard intrathecal dose)
Clonidine (extend block duration)	1 μg/kg
Morphine (pain control)	5 μg/kg for postoperative analgesia in general pediatric procedures (e.g., scoliosis repair) 10 μg/kg for cardiac surgical patients who will be ventilated postoperatively

- Spinous processes: Used to locate midline and assess for abnormalities in spinal curvature. Because of delayed fusion, the spinous process may be palpated as two adjacent bony structures in neonates and infants.
- Iliac crests: The intercristal line crosses the L5–S1 interspace in neonates and infants less than 1 year old and the L4–L5 interspace in children older than 1 year.
- Shoulders: Both shoulders should remain square to the bed, ensuring proper trunk flexion and alignment of the thoracic and lumbar spine.

Although various spinal needles are available in pediatric sizes, a 25-mm, 25-G pencil-point needle is recommended. Alternatively, a 38-mm, 22-G Quincke spinal needle may be used. An introducer is not necessary in neonates and younger infants.

Intrathecal drugs that have been used for spinal anesthesia in the pediatric population include bupivacaine, tetracaine, ropivacaine, and levobupivacaine. These drugs may be used as sole anesthetics or combined with sedatives or general anesthesia. The most common local anesthetics for pediatric spinal block are bupivacaine and tetracaine. A dose of 0.4–1.0 mg/kg of either drug will provide adequate surgical anesthesia. In our experience, a 1 mg/kg (=0.2 mL/kg) dose of preservative-free plain bupivacaine 0.5 % will provide 60 min of surgical anesthesia for inguinal hernia repair. Commonly used adjuvants include morphine, fentanyl, clonidine, epinephrine, neostigmine, and dextrose. Table 73.2 contains a list of suggested local anesthetics and adjuvants.

Landmark Technique

As with all regional blocks, observe strict aseptic technique. Place an absorbent pad between the warming blanket and the patient; this will absorb any fluids resulting from a post-block bowel movement. After sterilizing the skin, a sterile, clear plastic drape is used to cover the injection area. It is recommended that the anesthesiologist be seated for stability and enhanced dexterity when performing a spinal on an awake patient in the lateral decubitus position. If skin has not been topicalized with EMLA cream, the skin may be infiltrated with a 27–30-G needle and 1 % lidocaine prior to inserting the spinal needle. For lumbar puncture, use a midline approach with a short, 25–27-G styletted spinal needle. Upon puncture of the dura, a distinctive "pop" may or may not be felt since, in children, the ligamentum flavum is soft. The stylet should be removed occasionally to examine for CSF flow. Although initial CSF may contain some blood, the fluid should be clear before injecting anesthetic. A 1-mL syringe can be used to inject slowly (e.g., over a 15–20-s period). The barbotage method should be avoided since it may cause high levels of motor block. After the block has been administered, avoid elevating the patient's legs or trunk over their head. If a rapidly rising block level is observed, place the patient in the reverse Trendelenburg position to avoid further cranial spread of local anesthetic.

Sensory and motor assessment can be difficult in pediatric patients, particularly neonates, small children, and sedated patients. For infants, a cold stimulus (e.g., ice in a glove) can be used. A Bromage score [24] can usually be obtained for children over 2 years of age.

Ultrasound-Guided Technique

Sonographic assessment of neuraxial structures is not absolutely necessary; however, "offline" pre-procedural transverse and longitudinal views offer the following benefits:

1. Lumbar and sacral levels can be identified prior to dural puncture.
2. Spinous processes can be identified, enabling optimal needle trajectory to be estimated.
3. Position of the conus medullaris can be determined.
4. Depth to subarachnoid space can be estimated, as can the distance between the skin and dura (this distance can be narrow (6–8 mm) in neonates and infants [19]).
5. Real-time color-flow Doppler may be used to distinguish epidural and intrathecal injection [17].

In infants and younger children, a high-frequency (13-6 MHz) probe can be used to obtain excellent resolution; in adolescents, neuraxial structures are deeper and may require that a lower-frequency probe (e.g., 5-2 MHz curvilinear) be used to obtain good resolution. Wide-footprint linear-array transducers (13-6 MHz) offer good median or paramedian longitudinal views. Resolution will be best (>80 %) in children younger than 3 months and will decline in quality gradually to around 30–40 % by around 8 years of age.

Acknowledgements This chapter was written using Drs. Tsui and Suresh's textbook *Pediatric Atlas of Ultrasound and Nerve Stimulation-Guided Regional Anesthesia* 1st edition (Springer) as a template. The authors thank Jenkin Tsui for providing original artwork for the figures, Alex Kwan for assistance with creating and organizing the figures, and Dr. Gareth Corry for assistance with manuscript preparation and management.

References

1. Winnie AP, Ramamurthy S, Durrani Z. The inguinal paravascular technic of lumbar plexus anesthesia: the "3-in-1 block". Anesth Analg. 1973;52:989–96.
2. Kirchmair L, Enna B, Mitterschiffthaler G, Moriggl B, Greher M, Marhofer P, Kapral S, Gassner I. Lumbar plexus in children. A sonographic study and its relevance to pediatric regional anesthesia. Anesthesiology. 2004;101:445–50.
3. Lonnqvist PA. Continuous paravertebral block in children. Initial experience. Anaesthesia. 1992;47:607–9.
4. de Jose Maria B, Gotzens V, Mabrok M. Ultrasound-guided umbilical nerve block in children: a brief description of a new approach. Paediatr Anaesth. 2007;17:44–50.
5. Willschke H, Bosenberg A, Marhofer P, Johnston S, Kettner SC, Wanzel O, Kapral S. Ultrasonography-guided rectus sheath block in paediatric anaesthesia–a new approach to an old technique. Br J Anaesth. 2006;97:244–9.
6. Rafi AN. Abdominal field block: a new approach via the lumbar triangle. Anaesthesia. 2001;56:1024–6.
7. Laghari ZA, Harmon D. Ultrasound-guided transabdominus plane block. J Clin Anesth. 2008;20:156–8.
8. van Schoor AN, Boon JM, Bosenberg AT, Abrahams PH, Meiring JH. Anatomical considerations of the pediatric ilioinguinal/iliohypogastric nerve block. Paediatr Anaesth. 2005;15:371–7.
9. Kesler H, Dias MS, Kalapos P. Termination of the normal conus medullaris in children: a whole-spine magnetic resonance imaging study. Neurosurg Focus. 2007;23:E7.
10. Tsui BC, Gupta S, Finucane B. Confirmation of epidural catheter placement using nerve stimulation. Can J Anaesth. 1998;45:640–4.
11. Tsui BC, Wagner A, Cave D, Seal R. Threshold current for an insulated epidural needle in pediatric patients. Anesth Analg. 2004;99:694–6.
12. Tsui BC. Thoracic epidural catheter placement in infants via the caudal approach under electrocardiographic guidance: simplification of the original technique. Anesth Analg. 2004;98:273.
13. Tsui BC, Tarkkila P, Gupta S, Kearney R. Confirmation of caudal needle placement using nerve stimulation. Anesthesiology. 1999;91:374–8.
14. Lewis MP, Thomas P, Wilson LF, Mulholland RC. The 'whoosh' test. A clinical test to confirm correct needle placement in caudal epidural injections. Anaesthesia. 1992;47:57–8.
15. Orme RM, Berg SJ. The 'swoosh' test-an evaluation of a modified 'whoosh' test in children. Br J Anaesth. 2003;90:62–5.
16. Tsui BC, Wagner AM, Cunningham K, Perry S, Desai S, Seal R. Can continuous low current electrical stimulation distinguish insulated needle position in the epidural and intrathecal spaces in pediatric patients? Paediatr Anaesth. 2005;15:959–63.
17. Tsui B, Leipoldt C, Desai S. Color flow Doppler ultrasonography can distinguish caudal epidural injection from intrathecal injection. Anesth Analg. 2013;116:1376–9.
18. Tobias JD. Spinal anaesthesia in infants and children. Paediatr Anaesth. 2000;10:5–16.
19. Arthurs OJ, Murray M, Zubier M, Tooley J, Kelsall W. Ultrasonographic determination of neonatal spinal canal depth. Arch Dis Child Fetal Neonatal Ed. 2008;93:F451–4.
20. Bonadio WA, Smith DS, Metrou M, Dewitz B. Estimating lumbar-puncture depth in children. N Engl J Med. 1988;319:952–3.
21. Shenkman Z, Rathaus V, Jedeikin R, Konen O, Hoppenstein D, Snyder M, Freud E. The distance from the skin to the subarachnoid space can be predicted in premature and former-premature infants. Can J Anaesth. 2004;51:160–2.
22. Cote CJ, Lerman J, Todres ID. A practice of anesthesia for infants and children. Philadelphia: Saunders Elsevier; 2009. p. 877.
23. De Saint BL, Simon L, Laplace C, Egu JF, Hamza J. Preoperative coagulation tests in former preterm infants undergoing spinal anaesthesia. Paediatr Anaesth. 2002;12:304–7.
24. Bromage PR. A comparison of the hydrochloride and carbon dioxide salts of lidocaine and prilocaine in epidural analgesia. Acta Anaesthesiol Scand Suppl. 1965;16:55–69.

Suggested Reading

Jankovic D, Wells C. Regional nerve blocks: textbook and color atlas. 2nd ed. Berlin: Wiley; 2001.

Peutrell JM, Mather SJ. Regional anesthesia for babies and children. Oxford: Oxford University Press; 1997.

Saint-Maurice C, Schulte Steinberg O, Armitage E, editors. Regional anaesthesia in children. Norwalk: Appleton & Lange, Mediglobe; 1990.

Tsui BC. Atlas of ultrasound and nerve stimulation-guided regional anesthesia. New York: Springer; 2007.

Index

D. Jankovic, P. Peng, *Regional Nerve Blocks in Anesthesia and Pain Therapy: Traditional and Ultrasound-Guided Techniques*,
DOI 10.1007/978-3-319-05131-4, © Springer International Publishing Switzerland 2015

T