
Key Points

- A thorough preanesthetic patient history helps identify any risk factors related to the nervous, respiratory, cardiovascular, gastrointestinal, and hematologic systems. A thorough physical exam will identify any potential pitfalls or unforeseen surprises that could affect the ease and effectiveness of the nerve block.
- Use of well-designed equipment, which is appropriate for the procedure, can increase the success of regional blocks. Today's anesthesiologists have a wide range of needles, perineural catheters, nerve stimulators, ultrasound machines/probes, and monitoring devices at their disposal.
- Unique complications are associated with specific blocks and block procedures. These can occur during the block or appear during the postoperative period. Vigilance and knowledge on the part of the anesthesiologist and proper monitoring can help in identifying and addressing block-related complications perioperatively
- Prevention of complications is the key to safe and effective local and regional anesthesia practice. A preanesthetic checklist, good anatomical knowledge, patient selection, and technical skill are factors that can prevent adverse events during or after a block.

John W. R. McIntyre (deceased).

B.T. Finucane, MB, BCh, BAO, FRCA, FRCPC (✉)
Department of Anesthesiology and Pain Medicine,
University of Alberta, Edmonton, AB, Canada
e-mail: bfinucane6@gmail.com

Introduction

We are now in the third edition of this book and Professor McIntyre's observations are still very relevant today and more so in view of the fact that we are emphasizing safe practice of local and regional anesthesia. I updated the information in this chapter but the lion's share of the credit for the writing should still go to Dr. McIntyre (Fig. 2.1) posthumously.

Every patient wishes to receive anesthesia care that is safe, in other words, "free from risk, not involving danger or mishap; and guaranteed against failure" [1]. The anesthesiologist will present a more realistic view to the patient. The personal view of the hoped-for care will be one in which the clinical outcome is satisfactory and has been achieved without complication (defined as "any additional circumstances making a situation more difficult" [1]) because performance has deviated from the ideal [2]. By this standard, most deviations are trivial or easily corrected by a perfect process, and outcome for the patient and a reasonably stress-free life for the providers are objectives for all anesthesiologists. The general objective here is to provide information that helps the clinician to minimize complications that may occur during the course of local and regional anesthesia practice. This information is presented under the following headings:

- Complication anticipation
- Equipment
- Behavioral factors and complications
- Complication recognition
- Complications of specific neural blockades
- Complications in the postoperative period
- Complication prevention

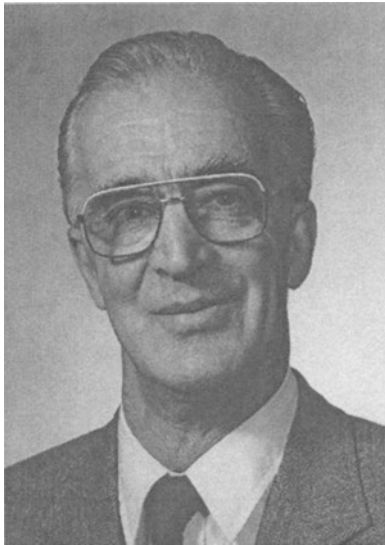


Fig. 2.1 Professor John W.R. McIntyre (1925–1998)

Complication Anticipation: Recognizing Precipitating Factors

The Preoperative Assessment: Patient History

Some anesthesiologists have a preconceived plan for regional anesthesia before they visit the patient; others gather information before considering what method of anesthesia is appropriate. The following paragraphs about the relationship between regional anesthesia and pathology are intended to aid recognition of potential complications for the patient under consideration and planning of anesthesia to avoid them.

The Nervous System

Fundamental issues to be settled during the preoperative visit are how the patient wishes to feel during the procedure and the anesthesiologist's opinion of how well the patient would tolerate the unusual sensations, the posture, and the environment. Whatever decision is made about pharmacologic support, it is absolutely essential that every patient has a clear understanding of reasonable expectations, once a plan has been made, and of the importance of revealing his or her own customary mood-altering medications. This is a convenient occasion to inquire about the patient's and relatives' previous experiences with local, regional, and general anesthesia.

Information should be sought regarding the presence of any degenerative axonal disease involving spinal cord, plexus, or nerve to be blocked and symptoms of thoracic outlet syndrome, spinal cord transaction, and lumbar lesions. Strong proponents of regional anesthesia have stated that a wide range of conditions—multiple sclerosis, Guillain–

Barré syndrome, residual poliomyelitis, and muscular dystrophy—are unaffected [3], although difficulty in a patient with Guillain–Barré syndrome has been reported [4]. However, there are reports of permanent neurologic deterioration in patients with unidentified preexisting problems [5–7]. Spinal anesthesia is an effective way of obtunding mass autonomic reflexes in patients with spinal cord transaction above T5, but a mass reflex has been described in a patient with an apparently appropriate block [8]. It must be concluded that the uncertainty of outcome when regional anesthesia is used in patients with established neurologic disease demands that the technique be used only when it is clearly advantageous for the patient. It is prudent to seek out symptoms of unrecognized neurologic abnormality when planning which anesthesia technique will be used. Parkinson's disease and epilepsy are not contraindications to regional anesthesia, provided they are habitually well controlled by medications, which should be continued during and after the operative period. This topic will be discussed in much greater detail in Chap. 9.

Thus far, the concerns addressed have largely involved the possibility of long-term neuronal damage and uncontrolled muscle activity, but the rapid changes in intracranial pressure during lumbar puncture can be dangerous [9, 10]. The lumbar extradural injection of 10 mL of fluid in two patients increased the intracranial pressure from 18.8 to 39.5 mmHg in the first patient and from 9.3 to 15.6 mmHg in the second patient [11]. Among patients at risk are those with head injuries, severe preeclampsia, and hydrocephalus.

A history of sleep apnea is more a reminder of the need for meticulous monitoring than a contraindication to regional anesthesia. In any case, patients may not recognize their own sleep apnea experiences. They are more likely to know of snoring, daytime hyper-somnolence, and restless sleep.

The Respiratory System

Preoperative pulmonary function tests do not identify definitive values predictive of hypoxia during regional anesthesia, but for practical purposes, if there are spirometric values <50 % of predicted, risk is increased [12]. It is certainly so if the values are FEV < 1.0 L, FVC < 15–20 mL/kg, FEV/FVC < 35 %, PEF < 100–200 L/min, and PCO₂ > 50 mmHg. Avoidance of the airway manipulation associated with general anesthesia and preserving coughing ability are advantageous for the patient with asthma or chronic obstructive pulmonary disease. Unfortunately, that can be more than offset by a magnitude of motor blockade that decreases vital capacity, expiratory reserve volume, maximum breathing capacity, and the ability to cough, all of which can result from anesthesia for abdominal surgery. If for some reason

the patient is particularly dependent on nasal breathing, as infants are, a block that is complicated by nasal congestion due to Horner's syndrome will cause respiratory difficulty.

Clinical assessment determines the need for acid–base and blood gas measurements. Hypoxia and acidosis enhance the central nervous system and cardiotoxicity of lidocaine [13–15]. In neonates, these effects are accentuated by poor compensation for metabolic acidosis.

The Cardiovascular System

Cardiac disease has profound implications for regional anesthesia, as it has for general anesthesia. Among the systems classifying the degree of cardiac risk, Detsky's modification of the Goldman index is useful (Table 2.1) [16]. However, this risk assessment is not patient specific, and there are individual asymptomatic patients with significant coronary artery disease that is unlikely to be detected. Also, chronic and relatively symptom-free chronic valvular dysfunction may lead to sudden and severe circulatory collapse [17]. There are many potential causes of myocardial infarction in patients undergoing extra cardiac surgery, as there are for other cardiovascular complications [18]. The role of dipyridamole-thallium scintigraphy and ambulatory (Holter) electrocardiography (ECG) has attracted interest [19, 20]; however, physiologic changes that can occur in a patient during the operative period and sub-

sets of patients to whom a specific test applies have yet to be identified with certainty [17].

When assessing the patient with cardiovascular problems for regional anesthesia and debating the addition, or perhaps sole use, of general anesthesia, the anesthesiologist must make predictions. These are the ability to satisfactorily control preload and afterload, myocardial oxygen supply, and demand and function. If one or more of these deviate from optimal limits, will the rate of change that may occur exceed the rate at which the therapeutic management can be developed?

The cardiac dysrhythmias of particular interest are the array of clinical disorders of sinus function (sick sinus syndrome). These are often associated with reduced automaticity of lower pacemakers and conduction disturbances. Local anesthetic drugs that diminish sinoatrial node activity, increase the cardiac refractory period, prolong the intracardiac conduction time, and lengthen the QRS complex will, in sufficient quantity, aggravate sinus node dysfunction.

It is important to realize that the pharmacokinetics of medications is influenced by certain cardiac defects. Patients with intracardiac right-to-left shunts are denied protection by the lungs, which normally sequester up to 80 % of the intravenous drug. If this is reduced, the likelihood of central nervous system toxicity is increased [21, 22].

The Gastrointestinal Tract

It is essential that the anesthesiologist obtain reliable information about the food and drink the patient has or will have taken preoperatively. A patient presenting for elective surgery will have received the customary institutional management, which may include one or more of the following: anticholinergic, histamine-receptor blocker (H₂), antacid, and benzamide derivative. Based on knowledge up to 1990, the following proposals have been made. First, solid food should *not* be taken on the day of surgery. Second, unrestricted clear fluids should be permitted until 3 h before scheduled surgery [23, 24].

In a study of the effect of epidural anesthesia on gastric emptying, measured by the absorption of acetaminophen from the upper small intestine, it appeared that block of sympathetic innervation of the stomach (T₆–10) did not affect gastric emptying [25]; however, epidural injection of morphine at the T₄ level delayed emptying. Nevertheless, with the onset of high spinal anesthesia, antiperistaltic movements and gastric regurgitation may occur and the ability to cough is reduced during a high blockade. Thus, the value of peripheral neural blockade for a patient with a potentially full stomach cannot be overestimated: subarachnoid and epidural anesthesia do not protect patients from aspiration. Similarly, paralysis of a recurrent laryngeal nerve, a complication of

Table 2.1 Detsky's modified multifactorial index arranged according to point value

Variables	Points
Class 4 angina ^a	20
Suspected critical aortic stenosis	20
Myocardial infarction within 6 months	10
Alveolar pulmonary edema within 1 week	10
Unstable angina within 3 months	10
Class 3 angina ^a	10
Emergency surgery	10
Myocardial infarction more than 6 months ago	5
Alveolar pulmonary edema ever	5
Sinus plus atrial premature beats or rhythm other than sinus on last preoperative electrocardiogram	5
More than five ventricular premature beats at any time before surgery	5
Poor general medical status ^b	5
Age over 70 years	5

Sources: Detsky et al. [16] Copyright 1986, American Medical Association. All rights reserved; Detsky et al. [17] Copyright 1986, Blackwell Publishing. All rights reserved with permission of Springer

^aCanadian Cardiovascular Society classification for angina

^bOxygen tension (PO₂) <60 mmHg; carbon dioxide tension (PCO₂) >50 mmHg; serum potassium <3.0 mEq/L; serum bicarbonate <20 mEq/L; serum urea nitrogen >50 mg/dL; serum creatinine >3 mg/dL; aspartate aminotransferase abnormality; signs of chronic liver disease; and/or patients bedridden from noncardiac causes

blockades in the neck region, predisposes patients to aspiration of gastric contents.

In a wide variety of abnormal circumstances, including trauma and near-term pregnancy, it is impossible to predict on the basis of the passage of time what the stomach contains. If the stomach is not empty, there are other vital considerations. In the presence of the blockade, the patient must be able to protect himself from aspiration; alternatively, in the presence of a failed blockade, it must be possible to administer a general anesthetic safely or to abandon the surgical procedure or delivery. Obstetric procedures usually brook no delay, and so it is mandatory that at some time well before the anticipated delivery date, the airway problems of pregnant patients be identified and plans made to cope with any eventuality.

The Hematologic System

Clotting Mechanisms

A regional anesthesia technique in which a hemorrhage cannot be detected readily and controlled by direct pressure is contraindicated in patients with a coagulation disorder, which might be attributed to diseases such as thrombocytopenia, hemophilia, and leukemia, or to drugs. Drugs having primary anticoagulant effects include unfractionated heparin, low-molecular-weight heparins, coumadin, and platelet inhibitors including aspirin, abciximab, clopidogrel, dipyridamole, anagrelide, ticlopidine, and tirifiban. Other drugs that to some degree influence coagulation are nonsteroidal anti-inflammatory medications, urokinase, phenprocoumon, and dextran 70.

Laboratory measurements determine the presence of a significant coagulation defect. Anticoagulation during heparin therapy is most often monitored by the activated clotting time. This method is not specific for a particular part of the coagulation cascade, and for diagnostic purposes, a variety of other tests are used: prothrombin (plasma thromboplastin) time, activated partial thromboplastin time, platelet count, and plasma fibrinogen concentration. Even in combination, however, these fail to provide a complete description of the status of the coagulation system. It is possible that viscoelastic methods are a convenient technique to monitor perioperative bleeding disorders [26].

Once a detailed history of drug use and laboratory measurements is available, a decision regarding the potential complications of central neural blockade, with or without catheter insertion, may be necessary, as may the influence of an anticoagulated state on postoperative developments.

Clinical experiences with these dilemmas have been comprehensively reviewed [27, 28], the conclusion being that performing epidural or spinal anesthesia in patients treated with drugs that may jeopardize the normal responses

of the clotting system to blood vessel damage is a concern. It is clear that major nerve-blocking techniques can be used in some patients who have received or will be receiving anticoagulant drugs. This success is not only dependent on an appreciation of the properties of different anticoagulant managements and a skilled regional anesthesia technique but also very careful postblockade monitoring. Thus, the advantages of the regional block envisaged must be carefully compared with other anesthesia techniques for the patient and the overall patient care available.

“Histaminoid” Reactions

Histaminoid refers to a reaction whose precise identity—histamine, prostaglandin, leukotremia, or kinin—is unknown. Few patients would recognize that term, and it is wiser to inquire of “allergy or sensitivity experiences.” This is particularly valuable information if the patient describes a situation that the anesthesiologist has contemplated repeating [29]. The patient’s story should not be discounted by attributing the reported events to epinephrine or a misplaced injection.

The dose or rate of administration does not affect the severity of a histaminoid reaction. Additionally, many studies have shown that reactions occur more often in patients with a history of atopy [30], but that a history of allergy is not predictive of severe clinical anaphylaxis [31]. The patient’s history, or lack of it, is important and may guide the anesthesiologist away from certain drugs; however, an unexpected reaction will challenge some anesthesiologists, somewhere, sometime, and that complication will demand immediate recognition and treatment.

Pseudocholinesterase Dysfunction

If a patient’s red cell cholinesterase is deficient or abnormal, drugs metabolized by that enzyme, such as 2-chloroprocaine, will be broken down more slowly, lowering the toxicity threshold [32, 33].

Methemoglobinemia

Drugs predisposing to methemoglobinemia are aniline dyes, nitrites, nitrates, sulfonamides, and antimalarial medications. It may also be associated with hemoglobinopathies and glucose-6-phosphate dehydrogenase deficiencies. The local anesthetics benzocaine, lidocaine, and prilocaine can contribute to methemoglobinemia.

Muscle Disease

Inquiries about muscular dystrophy, myasthenia gravis, and malignant hyperthermia are part of the preanesthetic evaluation, regardless of the contemplated anesthetic technique. It has been stated that neither amide nor ester-linked local anesthetics are contraindicated in such cases [34], we now have a clear message from the Malignant Hyperthermia Association of the United States (MHAUS) that all local

anesthetics in common use today are safe to use in patients at risk of malignant hyperthermia [35].

If the patient has a muscular dystrophy it is important to know because of associated problems that may be present, such as ECG abnormalities, but regional anesthesia is not contraindicated and may indeed be the technique of choice.

Diabetes

Diabetic patients usually announce their disease, but some leave the anesthesiologist to find out. It is important that the anesthesiologist knows that a patient is diabetic, because although neural blockade may be the technique of choice in some respects, the peripheral neuropathy and autonomic dysfunction associated with the disease have implications, particularly if they are in the area to be blocked. Preanesthetic symptoms and signs should be carefully documented.

Notably, a central conduction block limits the normal physiologic response to hypoglycemia and a diabetic patient can be unduly sensitive to the normal insulin regimen. This may complicate postoperative care [36, 37].

Miscellaneous Medications

Neural blockade complications clearly caused by drug interactions are rare, but possibilities can be taken into account during anesthesia planning and in diagnosing any complications detected later.

Aspirin

Aspirin therapy, because of its antiplatelet activity, may increase the risk of bleeding, which in association with central neural blockade, is potentially tragic. The effect of the drug on platelets is irreversible and lasts 7–10 days; thus, some assessment of platelet function should be made in aspirin-treated patients [38]. Today, measurement of the bleeding time is the only practical test of in vivo platelet function. It may return to normal 72 h after discontinuation of the drug, but in vitro platelet aggregation tests require much more time. If the bleeding time is 10 min or more, the clinician must weigh the relative disadvantages for that patient of other forms of anesthesia and analgesia.

Quinidine and Disopyramide

Laboratory studies showed that lidocaine metabolites and the metabolites of several antiarrhythmic agents had little effect on lidocaine protein binding. However, bupivacaine, quinidine, and disopyramide caused a significant increase in the lidocaine free fraction. These effects could cause unexpected drug-related complications [39].

Benzodiazepines

Diazepam enhances the cardiovascular toxicity associated with bupivacaine and verapamil [40]. Benzodiazepines mask

the early signs of systemic toxicity, so that the first evidence of problems may be cardiorespiratory depression.

Verapamil

Verapamil increases the toxicity of lidocaine and bupivacaine in mice [41], and cardiovascular collapse in patients has been reported [42].

Nifedipine

Nifedipine increases the toxicity of bupivacaine in dogs [43].

The Preanesthetic Visit: Physical Examination

The routine preoperative examination for anesthesia is described in many textbooks. The following paragraphs address matters that, although interesting at any time, are particularly important for the anesthesiologist contemplating performing a neural blockade. Positive answers to the following questions are not necessarily contraindications to regional anesthesia; indeed, they may support its selection, but they do indicate matters that must be given particular consideration.

Positioning for the Block

- Is the patient so large or heavy that a dangerous strain may be placed on tables, stools, and assistants unless special precautions are taken?

Blood Pressure

- Is the patient hypertensive or hypotensive?

Oxygenation

- Is the patient hypoxic?

Blood Volume

- Is the patient hypovolemic?

Infection

- Is there dystrophic skin or infection at the site of needle entry or infection in the needle track?
- Is there systemic infection in the body?
- Is the patient febrile?

Previous Surgery

- Are there scars anywhere indicating previous trauma or surgery that the patient has not mentioned?

Abdominal Masses

- Is an abdominal mass present that could impair venous return or respiration?
- Is there a gravid uterus beyond the first trimester that could impair venous return and influence the spread of subarachnoid injections?

Venous Access

- Will venous access for medications or fluids be easily obtained?

The Upper Airway

- In an emergency situation, can the anesthesiologist easily take control of the patient's airway, ventilate the patient, and prevent aspiration?

Technical Difficulty Performing the Proposed Block

- Will arthritis, amputation, or obesity hinder positioning the patient?
- Does obesity obscure bony landmarks?
- Is arthritis likely to hinder neural access?
- Are spinal defects, abnormalities of vertebral fusions, or foreign bodies present to hinder neural access?
- Can the arm be moved into a suitable position?
- Is there a hindrance to positioning a tourniquet?

Lymph Glands

- Are there axillary or femoral lymph glands in the needle path for the proposed block?
- Evaluating the Hemodynamic Status of the Limb
- Will a cast or other hindrance prevent monitoring of peripheral blood flow in a limb?

Conclusion

Surprises for an anesthesiologist in the block room are usually stressful, potentially hazardous for the patient, and may delay the operating room schedule. It is cautionary to realize that, in complex processes, be they medical care or industry, dangerous situations result from a sequence of events. Failure to obtain a certain item of information at the preanesthetic visit can be compounded by related events in the surgical or dental suite and the recovery area. The preoperative visit is the opportunity to plan the patient's anesthetic, be it a technique of regional anesthesia, general anesthesia, or a combination. A structured interview and examination is one facet of safe regional anesthesia practice.

Equipment

The objective for any attempted neural blockade is to produce the anesthesia required, and thus a major complication is block failure. Neural blockade may fail for pharmacologic or pharmacokinetic reasons, because the anesthesiologist lacks mental imagery of the anatomy, manual dexterity, or tactile sensitivity. Well-designed equipment does not make the user skilled, but it can diminish the complication of "failed spinal" and other complications associated with needle placement. The following is a collation of published data criteria believed to influence successful identification of the location for the anesthetic and of the complications associated with these attempts. Ultrasound-guided needle placement has greatly enhanced success rates of regional anesthesia particularly those involving peripheral nerves, in recent years.

Spinal Needles

Clinical Reports

The size of needles ranging from 18 to 25 gauge do not affect the success rate for subarachnoid tap [44, 45], and Whitacre 25 and 27 gauge, Quincke 25 gauge, and Sprotte have been used satisfactorily [46–49]. Thinner needles (29 and 30 gauge) have a greater tendency to deviate during their passage through ligamentous tissues, and an introducer through which those needles can be passed is essential [50–52].

Cerebrospinal fluid (CSF) spontaneous flow through a 29-gauge needle appears extremely slowly, if at all, even if the hub is clear plastic instead of metal. Similarly, injection of fluid can be accomplished only slowly, and drug distribution may be affected [51].

Spinal anesthesia in children can safely be done with 22- or 25-gauge spinal needles or the hollow stylet from a 24-gauge Angiocath.

Headache is primarily a complication of spinal tap in adults. An extensive and critical analysis of clinical reports concluded that the smallest gauge needle with a noncutting tip reduces its likelihood [53, 54]. Thus, choice of needle gauge is a compromise because using a very fine needle is more difficult. It has been suggested that when avoiding headache is paramount, Quincke or Whitacre 27 gauge are the needles of choice [55]. Waiting times for the appearance of CSF, with the patient in a lateral position using these needles were 10.8 ± 6.9 and 10.7 ± 6.8 s, respectively.

Laboratory Reports

Laboratory reports address the technical problems about which clinicians speculate and some complications to avoid. The conclusions are summarized next.

Changing the Needle Direction During Insertion

Deliberate change of direction of a needle is customarily done by almost complete withdrawal and subsequent reentry, and inadvertent deviation during advancement is misleading. A laboratory model demonstrated the occurrence of needle deviation and the influence of needle point design and gauge [56]. It was least with pencil-point spinal needles and greatest with beveled spinal needles. The needle deviation with beveled needles was consistent in direction as well as degree, in contrast to pencil-point tip configurations. Thus, rotating a beveled needle during insertion and redirection may hinder future identification of the epidural or subarachnoid space.

Resistance to Penetration of the Dura Mater

The human dura mater is relatively resistant to penetration by a long, beveled 21-gauge (80 × 0.8 mm) Quincke-Babcock needle [57]. After entering the epidural space (anatomically believed to vary from 1 to 7 mm in depth), depending on the site of insertion, the needle advanced 7–13 mm within it. This tenting of the dura mater is believed to be a potential hazard in the thoracic and cervical region because the spinal cord could be impacted.

Detection Time for CSF After Dural Puncture

Features that determine the effective use of spinal needles include rapid detectability of CSF and low resistance to injectate. Experiments with a wide variety of needles revealed that all Becton-Dickinson needles had a zero detection time [58]. The Quincke “Spinocan” 26 gauge and Portex pencil-point had the greatest delay, which at an artificial CSF pressure of 20–50 cm H₂O was approximately 8 s. The calculated relative resistance to flow through the needles varied from 0.21 (Becton-Dickinson Whitacre 22 gauge) to 2.91 (Quincke, Spinocan 26 gauge).

Rate of CSF Leak Following Dural Puncture

The rate of CSF loss through a dural puncture site can be measured in an *in vitro* model, and experiments demonstrated that, although more force was required to pierce the dura, CSF leakage from pencil-point needles was significantly less than that from Quincke needles of the same external diameter [59]. The authors concluded that the Whitacre 27-gauge needle lacks a clear advantage over the 25-gauge needle, which may be easier to use.

Needle Orifice Shape and Unintended Extra Dural Injection

A needle whose distal orifice is partially in and partially outside the subarachnoid space may deliver CSF from the hub, but only part of the injectate will be delivered into the subarachnoid space. The 22-gauge Whitacre needle is preferable

to long-orifice needles such as 22-gauge Sprotte, Quincke, and Diamond point [53, 60].

Epidural Needles

A suitable needle has the following characteristics: (1) easy penetration of ligaments, (2) minimally traumatic penetration, (3) minimal difficulty locating the epidural space, and (4) a lumen that facilitates epidural catheter placement. There are three needles that largely incorporate these features.

Tuohy Needle

The distal end is curved 20 degrees to direct a catheter into the epidural space. It must be introduced into the epidural space at least to the depth of the orifice. After a catheter has been inserted, it cannot be withdrawn without a serious risk of transaction.

Crawford Needle

This needle lacks a curved end and so must approach the epidural space obliquely if a catheter is to be inserted. It does not have to penetrate as deeply as the Tuohy needle into the space.

Whitacre Needles

Whitacre epidural needles have a blunt tip to reduce the likelihood of dural puncture. The eye of the needle is located laterally, so the distal end must be inserted well into the epidural space.

Needle sizes appropriate to the ages of children are as follows: [61] until 6–7 years, 20 gauge; from 7 to 10 years, 19 gauge; over 10 years, 19 or 18 gauge. A 16- or 18-gauge needle is customarily used in adults.

Combined Spinal and Epidural Techniques

The development of combined spinal and epidural (CSE) techniques since their inception in 1937 has been recently reviewed [62]. Various techniques, including conventional epidural, long spinal needles, catheters, and special devices, can be used. The double-segment technique involves the insertion of an epidural needle followed by a spinal needle inserted one or two segments below. The single-space technique (SST) requires an epidural needle insertion followed by a spinal needle insertion through its lumen once the epidural anesthesia solution has been injected. There are technical complications associated with the combined use of these devices as well as the individual ones, and sets specifically designed for SST have been designed.

Double-Lumen Needles

In this technique, a Tuohy needle has a parallel tube as a guide for a thinner spinal needle. There are two types—a bent parallel tube and a straight parallel tube. The bent parallel tube consists of a curved 20- to 22-gauge spinal needle of the same length as the Tuohy needle. The straight tube is fixed on the side of a Tuohy needle; the point of the guide is situated 1 cm behind the eye of the Tuohy needle. Spinal needles of normal length can be used. The double-lumen concept allows insertion of the epidural catheter before positioning of the spinal needle.

Another device is a conventional Tuohy needle to which has been added an additional aperture at the end of the longitudinal axis [52]. It is through this that a spinal needle on its way to the subarachnoid space will exit. Favorable clinical reports of CSE techniques have been supplemented by laboratory studies of flow characteristics of long spinal needles and the risk of catheter migration from the epidural space.

Flow Characteristics of Long Spinal Needles

The 120-mm, 26-gauge Braun Spinocan needle was compared in vitro with the 120 mm, 27-gauge Becton-Dickinson spinal needle. A pressure of 10 cm H₂O caused fluid to drop from the needle after 330 ± 14.8 and 129 ± 20.7 s, respectively. Clinical study findings were 33.5 and 10.85 s, respectively. The internal diameter of the 26-gauge needle is 0.23 mm and of the 27-gauge needle, 0.25 mm. The gauge value indicates the outer size, not the lumen [63].

Catheter Migration

An epiduroscopic study of cadavers demonstrated that the risk of epidural catheter migration through a dural puncture hole was very small. It was much less likely if the hole had been made by a 25-gauge spinal needle than with a Tuohy needle [64].

Complications Associated with Spinal and Epidural Catheters

1. *Insufficient length* to reach from the exit site to the shoulder.
2. *Venous penetration*. The lumen must be sufficient for aspiration. A stylet in the catheter must not project out of the tip.
3. *Dural penetration*. The lumen must be sufficient for aspiration. A stylet in the catheter must not project out of the tip. A closed round-ended catheter with side openings makes penetration less likely.
4. *Kinking*. This is less likely with currently manufactured catheters and with the redesigned version of the Raczy catheter [65].

5. *Knotting*. Interval marking of the catheter is a useful guide to the catheter length within the subarachnoid or epidural space and discourages coiling.
6. *Difficult withdrawal*. A clinical study of forces necessary for lumbar extradural catheter removal (range 1.57 ± 0.96 to 3.78 ± 2.8 N) and literature review indicated that the original approach to the space was inconsequential. However, the withdrawal force required was greater with the patient sitting than in the lateral position. Thus, the flexed lateral position was recommended for removal [66, 67]. This opinion is controversial. It has been recommended that the patient be in the same position used for insertion when it is removed [68].

Devices for Peripheral Nerve Blockade

Complications of nerve blockade include intravascular injection, intraneural injection, and failure to locate the nerve to be blocked. Breakage at a weak junction between the hub and stem is unlikely with modern needles, although in some circumstances a security bead can be a useful precaution.

Intravascular needle placement may be impossible to detect by aspiration if the needle lumen is very fine, and a translucent hub is of little help. This has implications for resuscitation arrangements established for minor surgical or dental procedures performed in offices and clinics. Intraneural injection is unlikely, but needles with side ports provide some protection from that event.

Paresthesias are quite common and unwelcome during the conduct of a central neural blockade especially spinal anesthesia, but in the past peripheral nerves were often deliberately located by eliciting paresthesias with the needle. This crude method of identifying peripheral nerves is no longer necessary with the advent of neurostimulation and more recently, ultrasound-guided regional anesthesia techniques. The causal relationship between paresthesia elicited in this manner and neural damage is controversial, and no statistically significant clinical data indicate that such stimulation produces neuropathy [69]. The animal experiments upon which claims for potential neuropathy are based did not represent clinical practice, although a clinician can never be absolutely certain that the tip of the needle is not actually within a nerve. Indeed, the sterile flexible infusion line between syringe and needle is there to help immobilize the needle when it is in position.

Concerns about mechanically produced paresthesia popularized the introduction of nerve stimulation to locate and identify peripheral nerves. The needle should ideally be insulated by Teflon coating in order to enhance opportunities to place the needle tip close to the nerve. Paresthesias may occur when the instrument is in use, but its purpose is to elicit visible contraction in a muscle served by the nerve to be blocked.

Ideally, the nerve stimulator should have the following characteristics [70]:

1. Constant current output
2. Clear meter reading to 0.1 mA
3. Variable output
4. Linear output
5. Clearly marked polarity
6. Short pulse width
7. Pulse of 1 Hz
8. Battery indicator
9. High-quality alligator clips
10. High- and low-output settings

Instruments designed for testing neuromuscular transmission do not usually indicate voltage or current at the site of stimulation and so are disadvantageous because they control only voltage, whereas it is current that causes a nerve to depolarize [71]. It is possible to elicit a muscle response when the needle is some distance from the nerve unless the stimulus current is less than 0.5 mA [72]. The concept is attractive and popular with some practitioners, but definitive evidence of its superiority over other methods is lacking and the occurrence of serious complications has been reported [69].

Another technique to safely identify the site for injection is visualizing the anatomy by ultrasonography. Not only can this increase the likelihood of successful neural blockade, but it reduces the incidence of pneumothorax associated with the supraclavicular approach to brachial plexus blockade [73].

Resuscitation Supplies

Cardiovascular failure, with or without respiratory failure, is a rare complication of regional blockade whether for head, trunk, or limbs. If competent treatment is not *immediately* available, however, the result will be permanent cerebral damage or death.

ASRA guidelines require the following medications and equipment to be immediately available when performing any regional anesthesia procedure:

Intravenous access and fluids, a tipping trolley, an oxygen supply, and resuscitation drugs and equipment must be available. The equipment must include an anesthesia machine as a source of oxygen, a means of lung ventilation, a laryngoscope, oropharyngeal airways, cuffed endotracheal tubes, a stilette, and continuous suction. Benzodiazepine, propofol, suxamethonium, ephedrine, epinephrine, atropine, and Lipid Emulsion 20 % should be immediately available. For complete details, please refer to the ASRA Practice Advisory on Local Anesthetic Systemic Toxicity [74].

Those are the basic requirements of the caregivers trained to provide advanced cardiopulmonary resuscitation and must be present when neural blockade is attempted in the hospital,

“block” clinic, or indeed anywhere. They are just as necessary in the office where a minor procedure is to be done under neural blockade. Not only must equipment be there, but the persons present should be trained to use it. In light of the magnitude of the potential tragedy, they should be able to communicate with extramural help while continuing their efforts at cardiopulmonary resuscitation. In other words, the anesthesiologist must always be accompanied by a trained assistant when performing regional anesthesia.

Behavioral Factors and Complications

The behavioral factors that lead to complications are of several categories. A lapse of safe habit is the routine failure to check effectively the identity and concentration of fluid to be injected. Another is the lack of a routine method of distinguishing between syringes. An unsafe habit could be the use of an air-filled syringe to identify the epidural space of a child. Other potential causes have been reviewed and in general are referred to as *vigilance decrement*, *vigilance* being a state of maximal and psychological readiness to react to a situation [75–77]. These can be the cause of temporarily breaking a safe habit or creating an unsafe habit or of missing evidence of a complication. It is an important feature of complication avoidance that anesthesiologists be aware of these behavioral pitfalls and to discipline themselves accordingly, while establishing safe work scheduling.

Effects of Sleep Deprivation

Sleep deprivation can dramatically impair performance of monitoring tasks, whether the signals are presented in an auditory or visual mode—and particularly if the task is not cognitively exciting. A cumulative sleep debt incurred over days has a detrimental effect; however, there are wide individual differences in responses to acute or chronic sleep loss. Ideally, anesthesiologists should objectively establish their own limitations because an anesthesiologist who has been working most of the night may feel remarkably awake, perhaps euphoric, in the morning, although studies have documented reduced performance, and in the afternoons the situation will have further deteriorated. Napping is not necessarily helpful, particularly if it occurs during a period of REM sleep.

A recommendation supported by evidence from a variety of subjects, including anesthesiologists, for the anesthesiologist who has been working most of the night and is scheduled for a full day’s work is this: “Do not work [78]. If work is mandatory do not nap for only 2 h. If 4 h is possible, accept it but be prepared for some remaining performance decrement.”

The Effects of Fatigue

Hours of continuous cognitively challenging work result in fatigue. The effects of fatigue are accentuated by sleep deprivation and influenced by the position of the activity in the individual's circadian rhythm. Published data support the contention that a fatigued anesthesiologist may be careless and less likely to detect perioperative complications or to respond optimally to evolving clinical situations [78].

The Hazard of Boredom

A task that is repetitious, uneventful, uninteresting, and undemanding is boring. In such a case, the anesthesiologist has too little work. It is a problem shared by many other real-life responsible tasks and results in inappropriate automatic behavior, vigilance decrement, inappropriate interest, and a general feeling of fatigue. Thus, the low-workload situation, similar to the high-workload state, can cause performance decrement, and thus complications, because evidence of their development is overlooked. Anesthesiologists periodically change their location in the operating room or converse with operating room companions, probably in an unconscious effort to maintain vigilance by increasing sensory input [76]. An unседated patient under regional anesthesia is sometimes a highly entertaining and educational source of information and social commentary, thus keeping the anesthesiologist close by. During boring cases, the addition of occupations completely unrelated to patient care demand a time-sharing technique that must be learned, and even then their impact on an individual's vigilance for clinically important matters is variable and very difficult to predict. Thus, while reading or listening to personal music in the operating room is common behavior it is difficult to judge if these practices interfere with patient care.

The Influences of Physical and Mental Factors

An anesthesiologist is sometimes anxious in the operating room, but when this is compounded by personal anxieties, planning, decision making, and monitoring may be adversely affected. Substance abuse reduces vigilance and psychomotor performance and there is strong evidence that hangovers from alcohol and marijuana have similar effects. Recent work suggests that pilots should wait at least 14 h after drinking alcohol before flying, although it is constituent aromatic substances in some beverages that are more likely to cause a problem.

Work Environment

The physical environment for conducting hospital surgery under regional anesthesia is similar to that for general anes-

thesia in that monitor displays should be discernible from the variety of positions assumed by the anesthesiologist during the course of the procedure [76].

Recently, verbal communications were found to be responsible for 37 % of events that could have resulted in patient deterioration or death in an intensive care unit, supporting other anecdotal reports of communication errors [78]. This confirms the need for an established routine to check the identity and concentration of fluids to be injected in every hospital or clinic location where neural blockades are done or existing blockades reinforced.

Small clinics and professional offices may differ from the hospital environment in one significant respect. In an acute emergency, persons performing cardiopulmonary resuscitation may be unable to communicate with outside help without discontinuing their lifesaving activity, and in some countries or states such behavior is illegal. Protection of patients demands an arrangement that avoids such a situation by ensuring a communication system that can be instantly and conveniently activated.

The "mental environment" in which neural blockade and surgery are performed is as important as the physical environment. It is salutary that anesthesiologists, who are sometimes confronted with injured patients who have suffered because the response to industrial production pressures was to ignore certain defenses against injury, can find themselves faced with the same decision as the industrial worker—and even under similar production pressures. These pressures may be temptations for personal gain or generated by surgeons, dentists, or institutional managers. A recent study concluded that pressure from internal and external sources is a reality for many anesthesiologists and is perceived, in some cases, to have resulted in unsafe actions being performed [79]. The implication is that any effort to increase anesthesia and surgical productivity should be based on methods other than reducing safe practices. Any attempt to achieve it by introducing new technology should be accompanied by a careful analysis and, if necessary, education of the person using it [80].

Complication Recognition During Neural Blockade and Surgery

Sharing Human and Instrumental Monitoring

Regional anesthesia conducted expertly on the basis of a careful medical history and examination of the patient is safe, but complications can occur [81–93]. Signs and symptoms, listed by body systems, are matched with the human and instrumental monitoring techniques used for their detection in Table 2.2.

The role of the patient is included, as is the anesthesiologist's direct or monitor-assisted sensing. If heavy sedation or a supplementary general anesthetic is used, the clinical

Table 2.2 Complication recognition

Symptoms and signs to be detected	Detection methods
Nervous system events	
• Peroneal numbness and tingling	Patient: Assuming there is no language barrier, the patient may report any of these spontaneously but should be initially instructed to report any unusual sensation
• Dizziness, tinnitus	Anesthesiologist: Communication with the patient and observation
• Hearing impairment	Instrument: Instruments do not identify these sensations for the anesthesiologist
• Headache	
• Reduced vision	
• Diplopia	
• Taste in mouth	
• Dysphagia	
• Coughing and sneezing	
• Nausea	
• Throat numbness	
• Dysphasia	
• Pain and paresthesia	
• Faintness	
• Restlessness	
Postural pressure or tension on peripheral nerves	Patient: An unreliable source of information
	Anesthesiologist: Power of observation
	Instrument: Limited in application. A pulse oximeter at a limb periphery may indirectly indicate a threat to nerve or plexus
Horner's syndrome	Patient: Reports unusual feeling
	Anesthesiologist: Observation
	Instrument: –
Phrenic nerve paralysis	Patient: Reports unusual feelings
	Anesthesiologist: Observation
	Instrument: SpO ₂ value may diminish
Recurrent laryngeal nerve block	Patient: Reports unusual feelings
	Anesthesiologist: Observation
	Instrument: –
Presence or absence of CSF in hub of needle or dripping from it	Patient: –
	Anesthesiologist: Observation. After dural puncture, the delay before the first drop of CSF appeared was approximately 11 s for a 27-gauge Becton-Dickinson spinal needle, and 33 s for a 26-gauge Braun needle [63]
	There is considerable variation among commercially available spinal needles [58]. Such details regarding needles used for blocks other than central neural blockade are unavailable
	Instrument: –
Loss of resistance to injection (epidural space detection)	Patient: –
	Anesthesiologist: Observation
	Instrument: Pressure variations in the injection system can be digitized and displayed to show an exponential pressure decline [94]
Blood reaching the hub of a needle and not pulsating	Patient: –
	Anesthesiologist: Observation. Note, blood will take substantially longer than CSF to pass through a spinal, or other, narrow bore needle
	There will be interpatient variability. Thus, a “bloody tap” is evidence that the needle is in a vein or hematoma, but absence of blood is not necessarily definitive evidence that drug will not be injected intravascularly
	Instrument: –
Cerebral function	Patient: Reports unusual sensation
	Anesthesiologist: Conversation or intermittent questioning of patient
	Instrument: –
Evidence of planned neural blockade	Patient: Report of unusual sensations
	Anesthesiologist: Questioning and examining the patient
	Instrument: Thermography and plethysmography

(continued)

Table 2.2 (continued)

Symptoms and signs to be detected	Detection methods
Evidence of unexpected neural blockade	Patient: Report of unusual sensations and/or motor function Anesthesiologist: Observation of blockade area and the patient Instruments: Sphygmomanometer, ECG, pulse meter
Vagal stimulation	Patient: Faintness or loss of consciousness Anesthesiologist: Observations Instruments: ECG, pulse oximeter, pulse meter, sphygmomanometer
Respiratory system events	Patient: Dyspnea may be reported but in general patients seem unaware of the significance of respiratory changes, and, if they have been sedated, unaware of them
• Respiratory rate changes	Anesthesiologist: Observations are valuable but are unlikely to assess function accurately or continuously
• Tidal volume change	Instruments: Pulse oximetry is a late indicator of respiratory dysfunction, relative to end-tidal capnography
• Apnea	The stethoscope in the operating room or PARR is now more of a diagnostic tool to identify such things as atelectasis and pneumothorax than a monitor of respiration but a paratracheal audible respiratory monitor has been described [95]
• Stertor	
• Respiratory obstruction	
• Dyspnea	
• Bronchospasm	
Erroneous gas delivery to patient	Patient: Comments may be made about odor Anesthesiologist: Observation of patient behavior Instrument: An Fio ₂ monitor with functioning alarms is quicker and more reliable than patient or anesthesiologist
Cardiovascular system events	
Hypotension	Patient: –
Hypertension	Anesthesiologist: Sensing error is large Instrument: Automated direct or indirect measurement
Bradycardia	Patient: –
Tachycardia	Anesthesiologist: Accurate observation is possible but may be intermittent. Instruments: A variety is available to provide this information continuously
Cardiac arrhythmia	Patient: The patient may state their heart is beating irregularly Anesthesiologist: Clinical observation Instrument: Pulse oximeter and precordial stethoscope will indicate irregularity. The ECG provides continuous information upon which a diagnosis can be based
Asystole	Patient: – Anesthesiologist: Suspicion is aroused if at that moment the finger is on a pulse or a precordial stethoscope is in use Instrument: An ECG is a continuous and definitive indicator A pulse oximeter can raise a delayed but serious suspicion
Increased or decreased central venous pressure	Patient: Symptoms relative to cardiopulmonary function may be announced Anesthesiologist: Clinical events indicate a possibility Instrument: Central venous pressure measurement
Cyanosis	Patient: – Anesthesiologist: Visual acuity and environmental circumstances create an undesirable error of assessment Instruments: Pulse oximetry and blood gas measurements
Muscle events	
These range from twitching of facial muscles to convulsive movements of major muscle masses	Patient: – Anesthesiologist: Observations Instrument: –
Body temperature events	
Hypothermia	Patient: Patients are aware of cold sometimes but are often poor judges of their real body temperature. There is strong evidence that not only do spinal and epidural anesthesia impair central and peripheral regulatory controls but are not perceived by the patient [96–99] Anesthesiologist: The observations of the patient may be an unreliable assessment of temperature because shivering is not occurring and, depending on the area felt, the skin may feel warm Instrument: Thermometry

situation changes radically. The cost–benefit picture of a specific regional anesthesia plan must be estimated in light of these factors. This is followed by an account of the documented complications for different neural blockades. It would be possible to create monitoring algorithms for individual blocks, but in this author’s opinion, such focusing of patient care would be detrimental to the patient’s safety because unrelated events might be ignored, threatening though they might be. It is important to realize that, although monitoring devices are invaluable, an astute anesthesiologist will detect signs that are precursors to the resulting events detected by the device. This anticipatory information enables therapy to begin sooner.

Monitoring Devices

Contemporary recommendations for monitoring of patients under regional anesthesia include the cardiovascular and respiratory systems and body temperature. Whatever the combination of human and instrumental monitoring might be, its purpose is to recognize complications before damage to the patient is inevitable. A vital question is, during what period of patient care should monitoring be in progress? It may not be surprising that reported serious complications threatening patient outcome have occurred any time from the onset of attempted neural blockade until surgery has been in progress for several hours, or even when the patient is in the recovery area [90]. In some instances, a complication has been detected much later. Accordingly, it is prudent to monitor patients carefully from entry into the block room until the effects of the blockade have ended.

When instrumental monitors are used, they should be calibrated correctly and located so that there can be a planned balance of visual attention between patient and instruments, and access by audible alarms. If they are to be used optimally for the early detection of complications, however, the characteristics of these essential pieces of equipment must be appreciated. The following paragraphs concentrate on these limitations but should not undermine their clinical value for caregivers.

Pulse Oximetry [100–106]

Pulse oximeters require a pulse at the site of measurement and provide only a crude indication of peripheral perfusion. Blood flow is barely required. It has been shown that peripheral blood flow can be reduced to only 10 % of normal before the pulse oximeter has difficulty estimating a saturation [107]. It does not justify assumptions regarding cardiac output, arterial blood pressure, or cardiac rhythm, which must be assessed by other means. Regarding respiration, a normal

saturation measurement when the patient breathes an increased inspired oxygen concentration does not confirm adequacy of ventilation. The hypoxemia that would otherwise accompany the rising carbon dioxide tension is masked. Most pulse oximeters make measurements and calculations that provide oxygen saturation. The more popular definition of O_2 saturation is functional saturation, which is the concentration of oxy-hemoglobin divided by the concentration of hemoglobin plus reduced hemoglobin: $\text{Functional saturation} = O_2\text{Hb}/(\text{RHb} + O_2\text{Hb})$

The met or CO-Hb concentrations used in the algorithms are estimations for the population under consideration; however, the presence of a large percentage of those abnormal hemoglobin’s can cause erroneous readings of saturation and mask serious hypoxia.

Regional anesthesia can produce profound changes of sympathetic nerve activity in different parts of the body. Evidence has been presented that pulse oximetry during lumbar epidural anesthesia gives falsely low readings when the sensor is placed on a finger [108].

Capnography [109–112]

Carbon dioxide production, pulmonary circulation, and ventilation are necessary to produce a normal capnogram. Change in the end-tidal carbon dioxide ($ETCO_2$) value can have a cardiovascular or respiratory origin, but it is as a monitor of spontaneous breathing that the capnograph has its role in regional anesthesia.

End-tidal capnography sampling in the spontaneously breathing, unintubated patient may be from inside a plastic oxygen mask, a nasal cannula, or a catheter tip in the nasopharynx. The numeric value of the $ETCO_2$ and its relationship to the arterial CO_2 pressure is influenced by oxygen delivery, ventilation–perfusion ratio, and sampling errors. The value of such monitoring, beyond respiratory rate indication and apnea detection, has been a contentious matter [113–115]. There have been very favorable recent reports of its use in adults and children, but certain provisos apply [116–120]. Small differences in sampling technique affect the accuracy of the values measured, so the technique requires expert evaluation where it is in use. A gas temperature–flow relationship in the nostril has been proposed as a monitor of respiration and refuted [121, 122]. Previous attempts to utilize such a relationship were unsuccessful.

Cardiac Rate and Rhythm

A normal ECG can be recorded from a patient who is profoundly hypotensive, hypoxic, or hypercapnic, so although it is valuable as an indicator of heart rate and rhythm, it is a

very late indicator of other threatening complications, even if the patient is conscious. Nevertheless, it provides potentially useful diagnostic information not provided by peripheral pulse-activated devices.

This information is more valuable for the diagnosis of arrhythmias than detection of myocardial ischemia, even if a modified V5 lead is used and the right arm electrode of lead I is placed over a position on the intersection of the left anterior axillary line and the fifth intercostal space and the ground electrode is placed on the left shoulder. The principal guides to cardiac ischemic complications are data gathered from monitoring and management of heart rate, mean arterial pressure, hemoglobin concentration, and saturation.

ECG monitoring should be used for major surgery and for patients at cardiac risk, but for routine cases the use of an ECG in preference to a pulse oximeter or capnograph is controversial. Many anesthesiologists favor pulse oximetry or capnography.

Systemic Arterial Pressure

The anesthesiologist predicts an acceptable blood pressure range for the patient and selects the methods of measurement on the basis of the anticipated margin of error. Invasive direct methods have their own sources of error but are more accurate than noninvasive techniques. Although invasive direct methods are possible during regional anesthesia and necessary for major surgery in very poor-risk patients, indirect methods are used for most patients.

Manual Indirect Measurement of Blood Pressure

Methods usually involve the application of a cuff (20 % larger than the diameter of the arm), applied snugly to the upper arm. After inflation to above the anticipated systemic pressure, it should be deflated, reducing the pressure at 2–4 mmHg per heartbeat. Detection of the returning pulse by palpation or oximeter provides a crude estimate, as do oscillations of aneroid manometers or mercury columns.

The Korotkoff method of detection requires a sensor under the cuff and over an artery, enabling the Korotkoff sounds to be heard. Although the pressures measured may differ from intra-arterial values by only a few millimeters of mercury, systolic, diastolic, and mean arterial pressures may be over- or underestimated by up to 30 % [123]. During anesthesia and surgery, the patient's cardiovascular status changes and the magnitude, and even the direction, of error may change [124].

Correlation with direct arterial pressure measurement is poor [125, 126]. Additionally, even if the blood pressure remains unchanged, alterations in the vascular tone in the

limb, such as may be produced by vasopressor agents, alter Korotkoff sounds. When the patient is very vasoconstricted or hypotensive, Korotkoff sounds are difficult to detect and the palpatory method is reassuring rather than accurate [127].

Automated Oscillometric Measurement

The inflatable cuff functions as a sensor supplying a pressure transducer within the instrument. The varying oscillations and cuff pressures are analyzed electronically to determine systolic, diastolic, and mean arterial pressures. Comparisons with pressures in the aorta or a peripheral artery have been made [128–131], and these devices are accurate to ± 10 mmHg. Another study demonstrated a good correlation only for systolic pressures [132]. Oscillometric diastolic pressures have been found to be higher; however, in a survey of six commercially available devices, errors ranged from –30 to +40 % for mean arterial pressures [124]. In general, low pressures were overestimated and high pressures were underestimated. If the patient has cardiac arrhythmia, results may be erroneous.

There is no doubt that automated sphygmomanometers are invaluable, providing blood pressure readings regularly and frequently, particularly when the patient is otherwise inaccessible. However, the anticipated accuracy of measurement does not always meet the anesthesiologist's requirements, and invasive methods are preferable, assuming they are conducted skillfully with the proper equipment. If electronic transducer-amplifier systems are not available, mean arterial pressure may be measured by a calibrated aneroid gauge [133].

Plethysmography

The finger arterial pressure device (Finapres) consists of a small finger cuff containing an inflatable bladder and an infrared plethysmograph volume transducer that can provide continuous monitoring. It seems that performance is better on a thumb than a finger [134], and studies have shown the Finapres to be as good as, if not better than, noninvasive oscillometric devices as compared with direct arterial pressure readings [135]. However, lacking precision, the instrument has not been recommended as a substitute for invasive arterial pressure measurement [135]. Since then, it has been shown that even small degrees of cuff misapplication contribute to measurement error as compared with intra-arterial cannulation. A comparative study of patients undergoing spinal anesthesia for lower segment cesarean delivery revealed many inconsistencies in some patients, and it was concluded that the Finapres was unsatisfactory for patients in whom sudden hypotension was a threat to outcome [136]. Problems with its use have been reviewed [137].

Thermometry

The location of the sensor is important if it is to be used as a predictor of temperature at a site other than its location. The ideal place for a probe is the lower third to fourth of the esophagus, but this site, similar to the nasopharynx, tympanic membrane, and rectum, is uncomfortable for conscious or even mildly sedated patients. The axilla of an adducted area is a useful site for the patient under regional anesthesia, reading approximately 0.5 °C less than the oral temperature.

Liquid crystal skin thermometers have been evaluated and are potentially useful as trend indicators during surgery, because they can conveniently be applied to the skin. They are susceptible to drafts, and it is recommended that, before changing exclusively to such a device, it be standardized using a thermocouple method in parallel until adequate experience has been obtained in that working environment [138].

Conclusion

Conventional practice demands that certain monitoring devices be used routinely; however, funding for them competes in society with all the nonmedical and medical factors that contribute to health in that society. Accordingly, any application for funds and decisions on the dispensation of a global budget must be supported by a valid justification. These are challenging tasks. Outcome studies designed to predict individual risk of complications must be based on very large population [139, 140]. They are very expensive and can be confounded to a greater or lesser extent by learning contamination bias during their implementation [141]. Practitioners sometimes develop or improve clinical skills when using a device, and that change affects patient care when the device is not in use. The argument that once learning has occurred with the aid of a monitor the monitor is no longer necessary is invalid, because reinforcement of the learning will be necessary. Additionally, even if convincing studies demonstrating a lack of change in patient outcome were presented, the question of anesthesiologist outcome remains to be addressed. Do these simple monitoring devices render the task less stressful for anesthesiologists and enable them to be more effective members of the hospital personnel and better citizens, once the working day or night is over?

The template proposed for assessing the efficacy of diagnostic imaging [142] has been modified for the assessment of anesthesia technology [139] and has five components: (1) technical efficacy, (2) diagnostic efficacy, (3) diagnostic thinking efficacy and therapeutic efficacy, (4) patient outcome, and (5) societal efficacy. As new devices become commercially available, future studies will be based on the specific problems embraced by regional anesthesia.

Critical features of introducing any new device into the workplace are new educational requirements and the attitudes of the potential users, which will be strongly influenced by the design features, additional work, its perceived value, and health factors [80].

Complications of Specific Neural Blockades

The wide variety of symptoms and signs of complications associated with regional blockade have been described as, “When sorrows come, they come not single spies/But in battalions” (Hamlet: Act 4, Scene 5) so the anesthesiologist must be encouraged to take an overall view of the patient. Nevertheless, initially the emphasis is on the complications of the neural blockade under consideration, because of their role in determining the final anesthesia plan and the matters uppermost in the mind of the anesthesiologist while monitoring that procedure and diagnosing complications during its conduct. Some sources of complications are shared by all patients and will not be described repeatedly for each block (e.g., airway obstruction, drug toxicity, epinephrine side effects, and neural damage).

Airway Obstruction

Traditionally in some institutions, nurses familiar to the patient kept the patient comfortable during major surgery under regional anesthesia. The patient was wide awake, and this was considered an important feature; however, tolerance of the procedure and cooperation must be ensured, not only for the success of the procedure but for satisfaction of all concerned. The choices range from complete consciousness, through a mild state of cortical depression in which the patient is calm and tranquil, to a drug-induced sleep or even general anesthesia supplemented by the regional blockade. The last is usually necessary for infants and children; there are more options for adult patients.

From the anesthesiologist’s point of view, some warning signs and symptoms are obtunded in unconscious patients. If the patient is heavily sedated, as opposed to tracheally intubated under general anesthesia, management of respiratory obstruction may be needed. In the awake state, the upper airway muscles help keep the airway patent. In the supine posture, airway patency increases in response to greater airway resistance. During normal sleep, muscle activity is reduced and can be supplemented by drugs such as alcohol, benzodiazepines, and barbiturates [143–145]. Thus, respiratory obstruction is a potential problem throughout the procedure that must be immediately recognized and successfully managed. This hazard is compounded in patients who normally experience episodes of sleep apnea, from the influence of deafferentation and central effects of the local anesthetic agent, including respiratory depression.

Local Anesthetic Focal Complications

In a conscious, unседated patient, the first symptoms or signs of focal complications are drowsiness or light-headedness. As toxic activity increases, the characteristic sequence is circumoral and lingual numbness, tinnitus, visual disturbances, dysarthria, and restlessness. Muscular twitching, often facial, progresses to convulsions, coma, and respiratory and circulatory depression. The quantity of drug reaching activity sites and time after injection are influenced not only by distribution, elimination, and drug characteristics, but by the site of injection. Sometimes all the vital systems are depressed simultaneously. This dangerous situation is compounded by inability of the patient to report symptoms. In the case of pregnant patients at term, neonatal depression can occur and hypotonia has a prominent role [146–148]. Bradycardia, heart block, and ventricular tachycardia have been reported [149, 150].

Epinephrine Complications

Epinephrine complications in regional anesthesia are related to vasoconstriction at the site of the injected fluid. As such, they are more likely to be evidenced in the postoperative period. However, if absorbed into the general circulation at the time of neural blockade, temporary hypertension is associated with tachycardia or reflex bradycardia. Cardiac arrhythmias, including ventricular fibrillation, occur when the quantity entering the general circulation is sufficient.

Complications of Neural Blockade

The complications of neural blockade are directly related to the anatomy of the route of the needle and the body into which fluid or air has been introduced. Thus, the anesthesiologist with a good mental image of the relevant anatomy can predict events that may occur, particularly if the preoperative visit has been informative. Those events comprise a mix of the symptoms and signs outlined as complications to be recognized during neural blockade, surgery, and recovery. Risks depend not only on the skill and care of the anesthesiologist, but also on the drugs, equipment, the environment, and unanticipated scenarios. Their early detection and management depend on the competence of all those with care responsibilities and their performance. In view of this multifactorial situation, it is virtually impossible to know the chances of a specific complication for a specific patient, although low reported incidences can be an encouraging guide. Table 2.3 lists the complications that have been associated with various neural blockades and can be correlated with previous sections about detection methods. Complications associated with narcotics are described elsewhere in this volume. The complications identified have been gathered largely from references [61, 71, 72, 81–93].

Miscellaneous Neural Blockade Complications

Neural blockades are created at a wide variety of sites in the upper and lower limbs, the lumbar and sacral nerves, the scalp, and nerves supplying the mandible and maxilla. These complications are similar in character, and on occasion their development is sudden and severe.

- Vascular penetration and hematoma
- Vascular penetration followed by the local anesthetic focal complications (LAFC) that may culminate in cardiac and respiratory arrest
- Neural trauma
- Local vasoactive effects of epinephrine resulting in gangrene
- Cardiac arrhythmias produced by epinephrine
- Bradycardia

Complications in the Postoperative Period

Patients who have been neurally blocked or received centrally administered opioids require meticulous surveillance if complications are to be detected while therapy has an excellent chance of being effective. Specific training of personnel is necessary for these tasks.

Admitting the Patient: History and Physical Examination

The activities of caregivers in recovery rooms and intensive care units have much in common, and there is anecdotal as well as research evidence in intensive care units that a significant complication is failure of communication between physicians and nurses [78]. This complication can occur in recovery rooms as well. The nurse accepting responsibility for a patient from the operating room is entitled to a report of the baseline data about vital systems and other information that relates to the neurally blocked patient. Presented verbally with a completed written protocol, recovery room complications may be a continuation of intraoperating room or in-transit events on new developments. They manifest themselves in several categories.

Cardiovascular System

- Blood pressure, pulse rate, and cardiac rhythm: when vasopressor drugs were administered, and whether their waning effect will unmask residual sympathetic blockade or hypovolemia
- Details of any evidence of circulatory overload during surgical irrigation of the bladder

Table 2.3 Complications of neural blockade^a

Orbital regional blockade	
Local effect by needle, catheter, or injected volume	Conductor blockade effects
<ul style="list-style-type: none"> • Venous penetration causing retrobulbar hematoma 	<ul style="list-style-type: none"> • Brain stem anesthesia associated with optic nerve sheath penetration resulting in
<ul style="list-style-type: none"> • Arterial penetration causing a retrobulbar hematoma and local ischemia • Vascular occlusion of the central retinal artery • Optic nerve penetration • Penetration of the globe • Penetration of the optic stem • Oculo-cardiac reflex 	<ul style="list-style-type: none"> • Increasing or decreasing cardiovascular vital signs, pulmonary edema, cardiac arrest, shivering, convulsions, hyperreflexia, hemiplegia, paraplegia, quadriplegia, contralateral amaurosis, contralateral oculomotor paralysis, facial palsy, deafness, vertigo, aphasia, loss of neck muscle power, loss of consciousness, vagolysis, respiratory depression, apnea
Cervical plexus blockade complications	
<ul style="list-style-type: none"> • Entry to epidural space • Entry to subarachnoid space • Intravenous penetration • Intra-arterial penetration • Penetration of esophagus (associated with the anterior approach to the ganglion) • Pneumothorax (especially on the patient's right side) • Nasal congestion 	<ul style="list-style-type: none"> • High spinal anesthesia with cardiovascular and respiratory failure • Aphasia and hemiparesis • Blindness
Supraclavicular brachial plexus blockade complications	
<ul style="list-style-type: none"> • Vascular penetration of subclavian and axillary arteries or veins, the vertebral artery, and external jugular vein. Ischemic arm problems may develop, particularly in children • Penetration of apical pleura, causing a pneumothorax 	<ul style="list-style-type: none"> • Stellate ganglion block producing Horner's syndrome • Phrenic nerve block which in children impairs respiration • Recurrent laryngeal in block causing hoarseness and possibility of aspiration
<ul style="list-style-type: none"> • Epidural space entry • Subarachnoid space entry • Nerve trauma • Vasovagal episodes in patients in the sitting position 	<ul style="list-style-type: none"> • Epidural anesthesia with cardiovascular and respiratory depression • Spinal anesthesia with cardiovascular and respiratory depression
Infraclavicular brachial plexus blockade complications	
<ul style="list-style-type: none"> • Axillary artery puncture, sometimes with a brief vascular insufficiency • Venous penetration causing a hematoma • Apical pleura penetration and ensuing pneumothorax is possible but unusual 	
Epidural blockade complications	
<ul style="list-style-type: none"> • Epidural vessel penetration • Epidural hematoma • Dural puncture • Back pain • Neural trauma • Air embolism (especially in children) if an air-filled syringe has been used to locate the epidural space 	<ul style="list-style-type: none"> • Hypotension • Respiratory depression failure • Bradycardia • Total spinal anesthesia • Horner's syndrome • Trigeminal nerve paralysis
If a catheter has been inserted:	
<ul style="list-style-type: none"> • Subdural space catheterization • Intravascular catheterization • Infection • Headache associated with supplementary injections 	
Caudal epidural blockade complications	
<ul style="list-style-type: none"> • Subcutaneous injection • Penetration of dura mater 	<ul style="list-style-type: none"> • Accidental spinal anesthesia with cardiovascular and respiratory involvement

(continued)

Table 2.3 (continued)

Orbital regional blockade	
• Penetration into epidural vein	• Urinary retention
• Hematoma	
• Intraosseous penetration	
• Pelvic visceral penetration	
• Infection, particularly if a caudal-epidural catheter is in situ	
Subarachnoid block complications	
• Epidural vessel penetration	• Total spinal anesthesia
• Epidural hematoma	• Hypotension
• Neural trauma	• Respiratory depression/failure
• Headache	• Dyspnea
	• Bradycardia/asystole
Intercostal nerve blockade complications	
• Pneumothorax	• Hemodynamic depression
• Penetration of intercostal vessels	• Respiratory depression/failure
• Penetration of pleural space	• Depressed cough reflex
• Entry to paravertebral space	• Blockade of spinal nerves
• Entry to epidural space	
• Entry to subarachnoid space	
Intravenous regional anesthesia (IVRA, Bier's block) complications	
Local effect by needle, catheter, or tourniquet	Conductor blockade effects
• Tourniquet discomfort	
• Tourniquet leak	
• Tourniquet release less than 20 min after local anesthetic injection	
• Vomiting followed by aspiration of recent food or drink	
• Neural damage caused by prolonged tourniquet time, or the cuff too close to the elbow joint	
• Necrosis caused by ischemia created in an already injured limb	
Thoracic paravertebral anesthesia	
Local effect by needle catheter	Conductor blockade effects
• Paravertebral vessel puncture	• Hypotension
• Pneumothorax	• Respiratory paralysis
• Intrapleural catheter placement or migration	• Epidural analgesia
	• Horner's syndrome (possibly bilateral)
• Headache	• Phrenic nerve paralysis (possibly bilateral)
• Sepsis	
• Intercostal nerve trauma and pain	

^aFor an explanation of central effects, see the section Local Anesthetic Focal Complications

- Fluid balance
- Perfusion of peripheral vascular beds

Respiratory System

- Respiratory rate, tidal volume, and apparent oxygenation
- Administration of respiratory depressant drugs epidurally or by any other route, and any antidote administration
- Airway management in the operating room

Central Nervous System

- Sedative or analgesic drugs

- The likelihood that the patient will arouse before sensory and motor block have disappeared and then will become agitated
- State of consciousness and responses to sensory stimuli
- Analgesia preparations for recovery room sojourn
- Antinausea preparations administered

Peripheral Nervous System

- The existing neural blockade and when it is expected to have disappeared
- The nerves in an anesthetized area that need protection (e.g., ulnar or lateral peroneal nerves)
- An epidural or subarachnoid catheter in situ

Bladder Distention

- Presence of a urinary catheter, its drainage, and the state of the bladder
- Perioperative Anticoagulant Therapy
- The drugs administered and anticipated effects on prothrombin time or other measurements of coagulation

Endocrine Pathology

- Diabetes and its management in the operating room
- Steroid medications given in the operating room or elsewhere

Body Temperature

- Evidence of hypo- or hyperthermia

Muscle Activity

- Restlessness
- Shivering
- Muscle twitching

Monitoring the Patient

The demand for recognition of complications in the recovery room is similar in most respects to recognition in the operating room and as described in a previous section. It is a judicious combination of human and instrumental sensing, the former being the fundamental component of recovery room care. Analysis of recovery room complications in adults and children reveals that they were identifiable largely by clinical observation rather than instrumental monitoring [151]. Nevertheless, certain instruments are invaluable for recovery room care because they provide for patients at risk; they provide more precise information and supply the caregiver with continuous vital information. Instruments invaluable for recovery room care are an ECG, pulse meter, pulse oximeter, automated sphygmomanometer, thermometer, and stethoscope.

Complications monitoring include evaluation of respiration, hemodynamics, level of consciousness, adequacy of analgesia, degree of motor blockade, and other side effects on admission to the postanesthesia recovery room. There are certain complications for which early detection, followed by early diagnosis and treatment, reduces the chance of a permanent neurologic deficit. They are those associated with central neural blockade, and presenting symptoms include backache [152, 153]; pain in thighs, calves, or buttocks [153]; headache; muscle twitching; and increase in neural blockade or its failure to regress. The detection of these complications can be made difficult by postoperative sedation [154], and analgesia and the normal variation in block duration. Although these complications, indicative

of a wide variety of pathology, are chronologically related to the neural blockade, they may be attributable to concomitant pathology [155], and headache accompanying epidural supplementation can be attributable to an increase in intracranial pressure during labor [156], trauma, or another intracranial lesion.

Discharging the Patient

Ambulatory patients are discharged home with a companion when the effects of the neural blockade have worn off and complications such as nausea, pain, and dizziness have been treated. Exceptions are patients who have had dental and very minor surgical procedures, for whom the residual effects of sedation determine fitness for discharge from the office or clinic, rather than the disappearance of neural blockade effects. Subsequent complications are detected by a follow-up call, visit 24 h later, or an emergency communication from patient or relative.

The situation for hospitalized patients who have often received a central neural blockade is somewhat different. When the neural blockade has worn off; pain and nausea have been treated; pharmacologic, neurologic, cardiovascular, and respiratory concerns resolved, the patient is transferred to a ward or intensive care unit. It is there that the delayed, but potentially permanent, complications occur 2 or 3 days later, whose outcome is determined by the time between detection and therapeutic intervention. The presenting symptoms and signs associated with different complications often include pain and evidence of increasing (rather than decreasing) neural blockade that may end in permanent disability. These complications are discussed elsewhere in this volume. It suffices to say that it is likely to be helpful if patient and caregivers are aware of the need to keep in touch regarding symptoms of these rare complications.

Complication Prevention

Complication reduction, and ultimate abolition, depends on consistent application of current knowledge and skills to patient care plus further development of expertise. In 1940, the leading article of the first issue of the *Journal of the American Society of Anesthesiologists* (today *Anesthesiology*) concluded with this statement: “The important decision is what man shall give the anesthetic [in contrast to the drug or technique]” [157]. The implications for training and practice remain.

The baseline competence reached at the inception of independent anesthetic practice is established by certifying authorities but the significant variation among certificants probably represents other training programs [158]. This is partly attributable to limited clinical experience and, particularly relevant for regional anesthesia, a possibly doubtful correlation between knowledge and skills [159–161].

Thus, any further move toward complication prevention must, among other things, include better regional anesthesia training. This is occurring on several counts. Virtual reality techniques that register in a three-dimensional manner on a computer screen can radically change the pattern of training [159]. Mental imagery of anatomy is an integral part of the anatomic reasoning while performing neural blockade. Three-dimensional computer-based methods of presenting anatomic relations have great potential for overcoming existing limitations of conventional teaching [162]. Last, more critical evaluation techniques can assess training methods and establish levels of competence reached in manual skills [163, 164]. However, improvement in the training of future anesthesiologists does little to reduce complications perpetuated by recently training and established anesthesiologists.

Contrasting characteristics of two competing perspectives of safe practice are presented in Table 2.4 [165].

Anesthesiologists' attitudes consistent with the same characteristics of normal accident theory have been documented [166]. These reflect certain problems facing persons who wish to implement factors supporting a high reliability theory, for example, the five hazardous thinking patterns: antiauthority, impulsivity, invulnerability, macho, and resignation.

Ever since anesthesia has been practiced, a variety of case reports and collations of mortality and morbidity have been published under the auspices of individuals, groups, or institutions. Nevertheless, controversy and democracy have remained preeminent, and resistance "on principle" to exter-

nal imposition of medical practice was firmly entrenched until the last 25 years, when such independence was seriously challenged and many anesthesiologists perceived certain changes to be in their own interests as well as those of patients.

The Department of Anesthesia of Harvard Medical School, Boston, in 1986 published specific, detailed, mandatory standards for minimal patient monitoring during anesthesia [167]. These were to be implemented in its nine component teaching hospital departments and published for the interest of other practitioners, organizations, and institutions. The motivation was anesthetic complications that incurred substantial financial settlements and that were thought to have been preventable and strongly influenced by a report of critical incidents. Included in those standards were these references to regional anesthesia:

- An attending or resident anesthesiologist or nurse anesthesiologist shall be present in the operating room at all times during its conduct.
- The arterial blood pressure and heart rate shall be measured at least every 5 min, where not clinically impractical.
- The ECG shall be continuously displayed from the institution of anesthesia until preparing to leave the anesthetizing location, unless clinically impractical.

The effect of these standards on complications of regional anesthesia has not been published, but there has been a favorable association between the adoption of the standards and diminishing cost of malpractice insurance.

The Canadian Anesthesiologists' Society (CAS) has promoted its guidelines to the practice of anesthesia for close to 20 years [168]. A standard is a definite level of excellence or adequacy demanded by an organization. Clinical practice guidelines (CPGs) are systematically developed statements to inform practitioners about appropriate care in specific clinical circumstances. Implicit in this is planned avoidance of complications. The word *guidelines*, as opposed to *standards*, was used advisedly, because although mandatory requirements could be reasonable for a hospital or group of institutions, it was deemed inappropriate to address all Canadian anesthesiologists in such a manner. The Canadian guidelines promulgated for regional anesthesia in 1996 are as follows.

Patient Monitoring

The only indispensable monitor is the presence, at all times, of an appropriately trained and experienced physician. Mechanical and electronic monitors are, at best, aids to vigilance. Such devices help the anesthesiologist to ensure the integrity of the vital organs, and in particular the adequacy of

Table 2.4 Competing perspectives on safety

High reliability theory
• Complications can be prevented through good organization and management
• Safety is the priority of the organization
• Duplicating tasks and devices increase safety
• Continuous quality improvement with simulations creates and maintains safety
• Trial-and-error learning from complications can be effective
Normal accidents theory
• Complications are inevitable in any complex system
• Safety is only a competing objective, "We cannot necessarily do that here"
• Duplication encourages risks and reduces safety
• Discipline and socialization are incompatible with democratic values
• Organizations cannot train for the unimagined. "Intuition is better than algorithms"
• Learning efforts from critical incidents and complications are crippled by faulty reporting and denial of responsibility

Source: Modified from Sagan. Copyright 1993, Princeton University Press, 1995 paperback edition. Reprinted by permission of Princeton University Press [165]. With permission of Springer

tissue perfusion and oxygenation. The healthcare facility is responsible for the provision and maintenance of monitoring equipment that meets current published equipment standards.

The chief of anesthesiology is responsible for advising the healthcare facility on the procurement of monitoring equipment and for establishing policies for monitoring to help ensure patient safety.

The anesthesiologist is responsible for monitoring patients receiving care and *must ensure* that appropriate monitoring equipment is available and working properly. *A preanesthetic checklist (such as found in Table 2.5 or equivalent) must be completed before initiation of anesthesia.* Monitoring guidelines for standard patient care apply to all patients receiving regional anesthesia or intravenous sedation.

Monitoring equipment may be classified either as *required* for each anesthetized patient (i.e., the device is attached, or dedicated exclusively, to each patient) or *immediately available* (the device is available for the anesthetized patient without inappropriate delay).

Required Equipment

- Pulse oximeter
- Apparatus to measure blood pressure
- Stethoscope, precordial, esophageal, or paratracheal
- ECG monitor
- Capnograph for an intubated patient
- Apparatus to measure temperature
- Appropriate lighting to visualize the exposed portion of the patient

Table 2.5 Preanesthetic checklist

A. Gas pipelines	D. Vacuum system Suction adequate
Secure connections between terminal units (outlets) and anesthetic machine.	E. Scavenging system Correctly connected to patient circuit and functioning
B. Anesthetic machine	F. Routine equipment
1. Turn on machine master switch and all other necessary electrical equipment	1. Airway Functioning laryngoscope (backup available)
Line oxygen (40–60 psi) (275–415 kPa)	Appropriate tracheal tubes: patency of lumen and integrity of cuff
Line nitrous oxide (40–60 psi) (275–415 kPa)	Appropriate oropharyngeal airways
Adequate reserve cylinder oxygen pressure	Stylet
Adequate reserve cylinder nitrous oxide content	Magill forceps
Check for leaks and turn off cylinders	2. IV supplies
Flow meter function of oxygen and nitrous oxide over the working range	3. Blood pressure cuff of appropriate size
2. Vaporizer filled	4. Stethoscope
Filling ports pin-indexed and closed Ensure “on/off” function and turn off	5. ECG monitor
3. Functioning oxygen bypass (flush)	6. Pulse oximeter
4. Functioning oxygen fail-safe device	7. Capnograph
5. Oxygen analyzer calibrated and turned on functioning mixer (where available)	8. Temperature monitor
Attempt to create a hypoxic O ₂ /N ₂ O mixture and/or verify correct changes in flow alarm	9. Functioning low- and high-pressure alarm
6. Functioning common fresh gas outlet	G. Drugs
7. Ventilator function verified	1. Adequate supply of frequently used drugs and IV solutions
8. Backup ventilation equipment available and functioning	2. Appropriate doses of drugs in labeled syringes
If an anesthesiologist uses the same machine in successive cases, departmental policy may permit performing an abbreviated checklist between cases	H. Location of special equipment in each anesthetizing location
C. Breathing circuit	1. Defibrillators
1. Correct assembly of circuit to be used	2. Emergency drugs
2. Patient circuit connected to common fresh gas outlet	3. Difficult intubation kit
3. Oxygen flow meter turned on	
4. Check for exit of fresh gas at face mask pressurizes. Check for leaks and integrity at circuit (e.g., Pethick test for coaxial)	
5. Functioning high-pressure relief valve	
6. Unidirectional valves and soda lime	
7. Functioning adjustable pressure relief valve	

Immediately Available Equipment

- Peripheral nerve stimulator
- Respirometer (tidal volume)

It is recognized that brief interruptions of continuous monitoring may be unavoidable. Furthermore, there are certain circumstances when a monitor may fail; thus, continuous vigilance by the anesthesiologist is essential.

The use of agent-specific anesthetic gas monitors is encouraged.

Epidural Anesthesia During Childbirth

Experience since publication of the guidelines in the September 1986 issue of the CAS newsletter has shown that the incidence of major complications associated with continuous low-dose epidural infusion for obstetric analgesia is extremely low. Consequently, it is not necessary for an anesthesiologist to remain physically present or immediately available during maintenance of continuous infusion epidural analgesia. Instead, the following requirements suffice: (1) an appropriate protocol for the management of these epidurals is in place; (2) an anesthesiologist can be contacted for the purpose of advice and direction.

In contrast to continuous infusion epidural analgesia, bolus injection of local anesthetic into the epidural space can be associated with immediate life-threatening complications. In recognition of this, the CAS recommends the following:

- When a bolus dose of local anesthetic is injected into the epidural space, an anesthesiologist must be available to intervene appropriately should complications arise.
- The intent of the phrase *available to intervene appropriately* is that individual departments of anesthesiology shall make their own determinations of *availability* and *appropriateness*. This determination must be made after each individual department of anesthesiology has considered the possible risks of bolus injection of local anesthetic and the methods of dealing with any emergency situation that might arise from the performance of the procedure in their facility.

Practice of Anesthesia Outside a Hospital

The basic principles, training requirements, techniques, equipment, and drugs used for the practice of anesthesia are noted in other sections of the guidelines. The following guidelines are for certain aspects peculiar to anesthetic practice outside a hospital.

Patient Selection

Patients should be classified by physical status in a manner similar to that in use by the American Society of Anesthesiologists (ASA). Usually, only patients in the ASA classifications I and II should be considered for an anesthetic outside a hospital. Patients in classification III may be accepted under certain circumstances.

Preoperative Considerations

The patient must have had a recent and recorded history, physical examination, and appropriate laboratory investigations. This may be performed by another physician or anesthesiologist. The duration of fasting before anesthesia should conform to the previously stated guidelines. The patient should be given an information sheet with pre- and postanesthetic instructions.

Conduct of Anesthesia

The anesthetic and recovery facilities shall conform to hospital standards published by the Canadian Standards Association, as defined in other sections. The standards of care and monitoring shall be the same in all anesthetizing locations. The Canadian guidelines are comprehensive and include the organization of hospital anesthesia services, the responsibilities of the chief of anesthesiology, and anesthetic equipment and anesthetizing locations.

Intuitively, CPGs are useful for collaboration with lay persons in a managerial capacity and with physicians, and they have been generated for a variety of reasons [169], including quality assurance and the assistance of practitioners in their decision making. However, a cause-and-effect relationship between guidelines and anesthesia complications has been neither demonstrated nor sought [170]. Indeed, formal evaluation of CPGs in Canada is rare, and there is concern that CPGs, lacking policies to ensure compliance, will be ineffective. It is expected that guidelines unsupported by peer review and prominent personalities will be ignored; nevertheless, whether referred to as *audit*, *quality assurance*, or *continuous quality improvement* or CPG, developments continue. It is noteworthy that it was insistence of the government of the United Kingdom that motivated the Confidential Enquiry into Perioperative Deaths there, and pressures elsewhere for establishing actual standards of practice come from governments, insurers, and the general public. In a definitive analysis of guidelines [170], the need for a clear target if they are to be effective improvers of patient care is emphasized and that they must be oriented to practitioners, managers, and planners as well as other stakeholders. Achieving consensus is itself a difficult task, but guidelines for this process have been promulgated [171].

Conclusion

Safety—avoidance of complications—in regional anesthesia is dependent on the cooperative efforts of anesthesiologists, other care providers, and persons with management responsibilities. The deficiencies at any moment in time may be inadequacies in the state of the art or defects in what is a very complex system. It may be that differences between general and regional anesthesia detected in comparative studies are affected by factors in the patient care systems other than differences intrinsic to the techniques.

In 1858, the redoubtable John Snow published rules for chloroform administration. These were not rules in the regulatory sense but advice or recommendations from a respected figure. What would have been his views about competing perspectives on safety will remain unknown; however, his efforts for the greater good of patients can be emulated by taking advantage of superior opportunities to promote safe regional anesthesia practice, not only by improving training, practice, and research but by international dissemination of information.

References

1. The Oxford english dictionary. New York: Oxford University Press; 1971.
2. MB W, Slagle J. Human factors research in anesthesia patient safety. Proc AMIA Symp. 2001;756–60.
3. Crawford JS, James FM, Nolte H. Regional anaesthesia for patients with chronic neurological disease and similar conditions. *Anaesthesia*. 1981;36:821–2.
4. Perel A, Reches A, Davidson JT. Anaesthesia in the Guillain-Barre syndrome. A case report and recommendations. *Anaesthesia*. 1977;32:257–60.
5. Ballin NC. Paraplegia following epidural analgesia. *Anaesthesia*. 1981;36:952–3.
6. Chaudhari LS, Kop BR, Dhruva AJ. Paraplegia and epidural analgesia. *Anaesthesia*. 1978;33:722–5.
7. Kopp SL, Jacobs AK, Hebl JR. Regional anesthesia in patients with pre-existing neurologic disease. *Reg Anesth Pain Med*. 2015;40:467–78.
8. Lambert DH, Deane RS, Mazuzan Jr JE. Anesthesia and the control of blood pressure in patients with spinal cord injury. *Anesth Analg*. 1982;61:344–8.
9. Duffy GP. Lumbar puncture in the presence of raised intracranial pressure. *Br Med J*. 1969;1:407–9.
10. Richards PG, Towu-Aghantse E. Dangers of lumbar puncture. *Br Med J (Clin Res Ed)*. 1986;292:605–6.
11. Hilt H, Gramm HJ, Link J. Changes in intracranial pressure associated with extradural anaesthesia. *Br J Anaesth*. 1986;58:676–80.
12. Brown LK. Surgical considerations: effects of surgery on lung function, preoperative evaluation. In: Miller A, editor. *Pulmonary function tests: a guide for the student and house officer*. Orlando: Grune and Stratton; 1987.
13. Englesson S, Matousek M. Central nervous system effects of local anaesthetic agents. *Br J Anaesth*. 1975;47(Suppl):241–6.
14. Freysz M, Timour Q, Bertrix L, Loufoua J, Aupetit JF, Faucon G. Bupivacaine hastens the ischemia-induced decrease of the electrical ventricular fibrillation threshold. *Anesth Analg*. 1995;80:657–63.
15. Rosen MA, Thigpen JW, Shnider SM, Foutz SE, Levinson G, Koike M. Bupivacaine-induced cardiotoxicity in hypoxic and acidotic sheep. *Anesth Analg*. 1985;64:1089–96.
16. Detsky AS, Abrams HB, Forbath N, Scott JG, Hilliard JR. Cardiac assessment for patients undergoing noncardiac surgery. A multifactorial clinical risk index. *Arch Intern Med*. 1986;146:2131–4.
17. Detsky AS, Abrams HB, McLaughlin JR, Drucker DJ, Sasson Z, Johnston N, Scott JG, Forbath N, Hilliard JR. Predicting cardiac complications in patients undergoing non-cardiac surgery. *J Gen Intern Med*. 1986;1:211–9.
18. Mangano DT. Perioperative cardiac morbidity. *Anesthesiology*. 1990;72:153–84.
19. Fleisher LA, Rosenbaum SH, Nelson AH, Jain D, Wackers FJ, Zaret BL. Preoperative dipyridamole thallium imaging and ambulatory electrocardiographic monitoring as a predictor of perioperative cardiac events and long-term outcome. *Anesthesiology*. 1995;83:906–17.
20. DT M. Preoperative risk assessment: many studies, few solutions. Is a cardiac risk assessment paradigm possible? *Anesthesiology*. 1995;83:897–901.
21. Jorfeldt L, Lewis DH, Lofstrom JB, Post C. Lung uptake of lidocaine in healthy volunteers. *Acta Anaesthesiol Scand*. 1979;23:567–74.
22. Lofstrom JB. Tissue distribution of local anesthetics with special reference to the lung. *Int Anesthesiol Clin*. 1978;16:53–71.
23. Goresky GV, Maltby JR. Fasting guidelines for elective surgical patients. *Can J Anaesth*. 1990;37:493–5.
24. Splinter WM, Stewart JA, Muir JG. Large volumes of apple juice preoperatively do not affect gastric pH and volume in children. *Can J Anaesth*. 1990;37:36–9.
25. Thoren T, Wattwil M. Effects on gastric emptying of thoracic epidural analgesia with morphine or bupivacaine. *Anesth Analg*. 1988;67:687–94.
26. Hett DA, Walker D, Pilkington SN, Smith DC. Sonoclot analysis. *Br J Anaesth*. 1995;75:771–6.
27. Vandermeulen EP, Van AH, Vermeylen J. Anticoagulants and spinal-epidural anesthesia. *Anesth Analg*. 1994;79:1165–77.
28. Horlocker TT, Wedel DJ, Rowlingson JC, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy. (ASRA Evidence-Based Guidelines, 3rd Edition). *Reg Anesth Pain Med*. 2010;35:64–101.
29. McKinnon RP, Wildsmith JA. Histaminoid reactions in anaesthesia. *Br J Anaesth*. 1995;74:217–28.
30. Dundee JW, Fee JP, McDonald JR, Clarke RS. Frequency of atopy and allergy in an anaesthetic patient population. *Br J Anaesth*. 1978;50:793–8.
31. Fisher MM, Outhred A, Bowey CJ. Can clinical anaphylaxis to anaesthetic drugs be predicted from allergic history? *Br J Anaesth*. 1987;59:690–2.
32. Kuhnert BR, Philipson EH, Pimental R, Kuhnert PM. A prolonged chloroprocaine epidural block in a postpartum patient with abnormal pseudocholinesterase. *Anesthesiology*. 1982;56:477–8.
33. Smith AR, Hur D, Resano F. Grand mal seizures after 2-chloroprocaine epidural anesthesia in a patient with plasma cholinesterase deficiency. *Anesth Analg*. 1987;66:677–8.
34. Paasuke RT, Brownell AK. Amide local anaesthetics and malignant hyperthermia. *Can Anaesth Soc J*. 1986;33:126–9.
35. Malignant Hyperthermia Association of the United States. 2015. www.mhaus.org/dentoral.html.
36. Romano E, Gullo A. Hypoglycaemic coma following epidural analgesia. *Anaesthesia*. 1980;35:1084–6.
37. Traynor C, Paterson JL, Ward ID, Morgan M, Hall GM. Effects of extradural analgesia and vagal blockade on the metabolic and

- endocrine response to upper abdominal surgery. *Br J Anaesth.* 1982;54:319–23.
38. Macdonald R. Aspirin and extradural blocks. *Br J Anaesth.* 1991;66:1–3.
 39. McNamara PJ, Slaughter RL, Pieper JA, Wyman MG, Lalka D. Factors influencing serum protein binding of lidocaine in humans. *Anesth Analg.* 1981;60:395–400.
 40. Yong CL, Kunka RL, Bates TR. Factors affecting the plasma protein binding of verapamil and norverapamil in man. *Res Commun Chem Pathol Pharmacol.* 1980;30:329–39.
 41. Tallman Jr RD, Rosenblatt RM, Weaver JM, Wang YL. Verapamil increases the toxicity of local anesthetics. *J Clin Pharmacol.* 1988;28:317–21.
 42. Collier C. Verapamil and epidural bupivacaine. *Anaesth Intensive Care.* 1985;13:101.
 43. Howie MB, Mortimer W, Candler EM, McSweeney TD, Frolicher DA. Does nifedipine enhance the cardiovascular depressive effects of bupivacaine? *Reg Anesth.* 1989;14:19–25.
 44. Manchikanti L, Hadley C, Markwell SJ, Colliver JA. A retrospective analysis of failed spinal anesthetic attempts in a community hospital. *Anesth Analg.* 1987;66:363–6.
 45. Munhall RJ, Sukhani R, Winnie AP. Incidence and etiology of failed spinal anesthetics in a university hospital: a prospective study. *Anesth Analg.* 1988;67:843–8.
 46. Campbell DC, Douglas MJ, Pavy TJ, Merrick P, Flanagan ML, McMorland GH. Comparison of the 25-gauge Whitacre with the 24-gauge Sprotte spinal needle for elective caesarean section: cost implications. *Can J Anaesth.* 1993;40:1131–5.
 47. Kang SB, Goodnough DE, Lee YK. Spinal anesthesia with 27-gauge needles for ambulatory surgery patients. *Anesthesiology.* 1990;73:A2.
 48. Kang SB, Romeyn RL, Shenton DW. Spinal anesthesia with 27-gauge needles for outpatient knee arthroscopy in patients 12 to 18 years old. *Anesthesiology.* 1991;75:A1103.
 49. Parker RK, DeLeo BC, White PF. Spinal anesthesia: 25 GA Quincke vs 25 GA or 27 GA Whitacre needles. *Anesthesiology.* 1992;77:A485.
 50. Dahl JB, Schultz P, Anker-Møller E, Christensen EF, Staunstrup HG, Carlsson P. Spinal anaesthesia in young patients using a 29-gauge needle: technical considerations and an evaluation of postoperative complaints compared with general anaesthesia. *Br J Anaesth.* 1990;64:178–82.
 51. Lesser P, Bembridge M, Lyons G, Macdonald R. An evaluation of a 30-gauge needle for spinal anaesthesia for caesarean section. *Anaesthesia.* 1990;45:767–8.
 52. Lifschitz R, Jedeikin R. Spinal epidural anaesthesia. A new combination system. *Anaesthesia.* 1992;47:503–5.
 53. Fritz T, Mitthew A. How to establish accurate estimates of the effectiveness of three spinal needles in decreasing postdural puncture headache (PDPH) and other complications. A meta analysis. *Reg Anesth.* 1994;19:4.
 54. Halpern S, Preston R. Postdural puncture headache and spinal needle design. Metaanalyses. *Anesthesiology.* 1994;81:1376–83.
 55. Lynch J, Kasper SM, Strick K, Topalidis K, Schaaf H, Zech D, Krings-Ernst I. The use of Quincke and Whitacre 27-gauge needles in orthopedic patients: incidence of failed spinal anesthesia and postdural puncture headache. *Anesth Analg.* 1994;79:124–8.
 56. Kopacz DJ, Allen HW. Comparison of needle deviation during regional anesthetic techniques in a laboratory model. *Anesth Analg.* 1995;81:630–3.
 57. Zarzur E, Goncalves JJ. The resistance of the human dura mater to needle penetration. *Reg Anesth.* 1992;17:216–8.
 58. Carson DF, Serpell MG. Clinical characteristics of commonly used spinal needles. *Anaesthesia.* 1995;50:523–5.
 59. Westbrook JL, Uncles DR, Sitzman BT, Carrie LE. Comparison of the force required for dural puncture with different spinal needles and subsequent leakage of cerebrospinal fluid. *Anesth Analg.* 1994;79:769–72.
 60. Sayeed YG, Sosis M, Braverman B. An in vitro investigation of the relationship between spinal needle design and failed spinal anesthesia. *Reg Anesth.* 1993;18:85.
 61. Dalens BJ. Epidural anesthesia. In: BJ D, editor. *Pediatric regional anesthesia.* Boca Raton: CRC Press; 1990. p. 386.
 62. Felsby S, Juelsgaard P. Combined spinal and epidural anesthesia. *Anesth Analg.* 1995;80:821–6.
 63. Patel M, Samsoun G, Swami A, Morgan BM. Flow characteristics of long spinal needles. *Anaesthesia.* 1994;49:223–5.
 64. Holmstrom B, Rawal N, Axelsson K, Nydahl PA. Risk of catheter migration during combined spinal epidural block: percutaneous epiduroscopy study. *Anesth Analg.* 1995;80:747–53.
 65. Racz GB, Sabonghy M, Gintautas J, Kline WM. Intractable pain therapy using a new epidural catheter. *JAMA.* 1982;248:579–81.
 66. Boey SK, Carrie LE. Withdrawal forces during removal of lumbar extradural catheters. *Br J Anaesth.* 1994;73:833–5.
 67. Frankhouser PL. Hazard of a new epidural catheter. *Anesthesiology.* 1983;58:593.
 68. Morris GN. Removal of lumbar extradural catheters. *Br J Anaesth.* 1995;74:722.
 69. Moore DC, Mulroy MF, Thompson GE. Peripheral nerve damage and regional anaesthesia. *Br J Anaesth.* 1994;73:435–6.
 70. Ford DJ, Pither CE, Raj PP. Electrical characteristics of nerve stimulators: implications for nerve localization. *Reg Anesth.* 1984;9:42.
 71. Neal JM, Barrington MJ, Brull R, Hadzic A, Hebl JR, Horlocker TT, Huntoon MA, Kopp SL, Rathmell JP, Watson JC. The second ASRA practice advisory on neurologic complications associated with regional anesthesia and pain medicine: executive summary 2015. *Reg Anesth Pain Med.* 2015;40:401–30.
 72. Prithvi Raj P. *Clinical practice of regional anesthesia.* New York: Churchill Livingstone; 1991. p. 167.
 73. Kapral S, Krafft P, Eibenberger K, Fitzgerald R, Gosch M, Weinstabl C. Ultrasound-guided supraclavicular approach for regional anesthesia of the brachial plexus. *Anesth Analg.* 1994;78:507–13.
 74. Neal JM, Bernards CM, Butterworth JF, et al. ASRA practice advisory on local anesthetic systemic toxicity. *Reg Anesth Pain Med.* 2010;35:152–61.
 75. Gaba DM. Human error in anesthetic mishaps. *Int Anesthesiol Clin.* 1989;27:137–47.
 76. McIntyre JW. Implication of anaesthesiologists' varying location during surgery. *Int J Clin Monit Comput.* 1995;12:33–6.
 77. Weinger MB, Englund CE. Ergonomic and human factors affecting anesthetic vigilance and monitoring performance in the operating room environment. *Anesthesiology.* 1990;73:995–1021.
 78. Donchin Y, Gopher D, Olin M, Badihi Y, Biesky M, Sprung CL, Pizov R, Cotev S. A look into the nature and causes of human errors in the intensive care unit. *Crit Care Med.* 1995;23:294–300.
 79. Gaba DM, Howard SK, Jump B. Production pressure in the work environment. *California anesthesiologists' attitudes and experiences.* *Anesthesiology.* 1994;81:488–500.
 80. McIntyre JW. Anaesthesia monitoring: the human factors component of technology transfer. *Int J Clin Monit Comput.* 1993;10:23–9.
 81. Beers RA, Thomas PS, Martin RJ, Gorji R. Severe lumbar back pain following epidural injection of local anesthetic for epidural anesthesia. Case report and literature review. *Reg Anesth.* 1995;20:69–74.
 82. Crosby E, St-Jean B, Reid D, Elliott RD. Obstetrical anaesthesia and analgesia in chronic spinal cord-injured women. *Can J Anaesth.* 1992;39:487–94.
 83. Douglas MJ. Potential complications of spinal and epidural anesthesia for obstetrics. *Semin Perinatol.* 1991;15:368–74.

84. Finucane BT. Regional anaesthesia: complications and techniques. *Can J Anaesth.* 1991;38:R3-16.
85. Fox MA, Webb RK, Singleton R, Ludbrook G, WB R. The Australian Incident Monitoring Study. Problems with regional anaesthesia: an analysis of 2000 incident reports. *Anaesth Intensive Care.* 1993;21:646-9.
86. Goldman LJ. Complications in regional anaesthesia. *Paediatr Anaesth.* 1995;5:3-9.
87. Gribomont BF. Sudden complications in regional anesthesia. *Acta Anaesthesiol Belg.* 1988;39:165-70.
88. Harris AP, Michitsch RU. Anesthesia and analgesia for labor. *Curr Opin Obstet Gynecol.* 1992;4:813-7.
89. Liu S, Carpenter RL, JM N. Epidural anesthesia and analgesia. Their role in postoperative outcome. *Anesthesiology.* 1995;82:1474-506.
90. McIntyre JW. Monitoring regional anaesthesia. *Int J Clin Monit Comput.* 1990;7:241-7.
91. Peyton PJ. Complications of continuous spinal anaesthesia. *Anaesth Intensive Care.* 1992;20:417-25.
92. Rubin AP. Complications of local anaesthesia for ophthalmic surgery. *Br J Anaesth.* 1995;75:93-6.
93. Wong DH. Regional anaesthesia for intraocular surgery. *Can J Anaesth.* 1993;40:635-57.
94. Rodiera J, Calabuig R, Aliaga L, Espinosa W, Hobeich F, Oferil F, Gual A. Mathematical analysis of epidural space location. *Int J Clin Monit Comput.* 1995;12:213-7.
95. Eldor J, Adler D, Mahler Y, Davidson JT. Para-tracheal audible respiratory monitor (PTARM). *Can J Anaesth.* 1987;34:328.
96. Giesbrecht GG. Human thermoregulatory inhibition by regional anesthesia. *Anesthesiology.* 1994;81:277-81.
97. Joris J, Ozaki M, Sessler DI, Hardy AF, Lamy M, McGuire J, Blanchard D, Schroeder M, Moayeri A. Epidural anesthesia impairs both central and peripheral thermoregulatory control during general anesthesia. *Anesthesiology.* 1994;80:268-77.
98. Ozaki M, Kurz A, Sessler DI, Lenhardt R, Schroeder M, Moayeri A, Noyes KM, Rotheneder E. Thermoregulatory thresholds during epidural and spinal anesthesia. *Anesthesiology.* 1994;81:282-8.
99. Vassilieff N, Rosencher N, Sessler DI, Conseiller C. Shivering threshold during spinal anesthesia is reduced in elderly patients. *Anesthesiology.* 1995;83:1162-6.
100. Clayton DG, Webb RK, Ralston AC, Duthie D, Runciman WB. A comparison of the performance of 20 pulse oximeters under conditions of poor perfusion. *Anaesthesia.* 1991;46:3-10.
101. Clayton DG, Webb RK, Ralston AC, Duthie D, WB R. Pulse oximeter probes. A comparison between finger, nose, ear and forehead probes under conditions of poor perfusion. *Anaesthesia.* 1991;46:260-5.
102. Hutton P, Clutton-Brock T. The benefits and pitfalls of pulse oximetry. *BMJ.* 1993;307:457-8.
103. Ralston AC, Webb RK, Runciman WB. Potential errors in pulse oximetry. I. Pulse oximeter evaluation. *Anaesthesia.* 1991;46:202-6.
104. Ralston AC, Webb RK, Runciman WB. Potential errors in pulse oximetry. III: effects of interferences, dyes, dyshaemoglobins and other pigments. *Anaesthesia.* 1991;46:291-5.
105. Runciman WB, Webb RK, Barker L, Currie M. The Australian Incident Monitoring Study. The pulse oximeter: applications and limitations--an analysis of 2000 incident reports. *Anaesth Intensive Care.* 1993;21:543-50.
106. Webb RK, Ralston AC, Runciman WB. Potential errors in pulse oximetry. II. Effects of changes in saturation and signal quality. *Anaesthesia.* 1991;46:207-12.
107. Tremper KK. Interpretation of non-invasive oxygen and carbon dioxide data. *Can J Anaesth.* 1990;37:Slxxvii-Slxxxviii.
108. Peduto VA, Tani R, Pani S. Pulse oximetry during lumbar epidural anesthesia: reliability of values measured at the hand and the foot. *Anesth Analg.* 1994;78:921-4.
109. Campbell FA, McLeod ME, Bissonnette B, Swartz JS. End-tidal carbon dioxide measurement in infants and children during and after general anesthesia. *Can J Anaesth.* 1994;41:107-10.
110. Paulus DA. Capnography. *Int Anesthesiol Clin.* 1989;27:167-75.
111. Stock MC. Noninvasive carbon dioxide monitoring. *Crit Care Clin.* 1988;4:511-26.
112. Williamson JA, Webb RK, Cockings J, Morgan C. The Australian Incident Monitoring Study. The capnograph: applications and limitations--an analysis of 2000 incident reports. *Anaesth Intensive Care.* 1993;21:551-7.
113. Dunphy JA. Accuracy of expired carbon dioxide partial pressure sampled from a nasal cannula. *Anesthesiology.* 1988;67:960-1.
114. Goldman JM. Accuracy of expired carbon dioxide partial pressure sampled from a nasal cannula. *Anesthesiology.* 1988;68:961.
115. Urmey WF. Accuracy of expired carbon dioxide partial pressure sampled from a nasal cannula. *Anesthesiology.* 1988;68:961.
116. Abramo TJ, Cowan MR, Scott SM, Primm PA, Wiebe RA, Signs M. Comparison of pediatric end-tidal CO₂ measured with nasal/oral cannula circuit and capillary PCO₂. *Am J Emerg Med.* 1995;13:30-3.
117. Barton CW, Wang ES. Correlation of end-tidal CO₂ measurements to arterial PaCO₂ in nonintubated patients. *Ann Emerg Med.* 1994;23:560-3.
118. Flanagan JF, Garrett JS, McDuffee A, Tobias JD. Noninvasive monitoring of end-tidal carbon dioxide tension via nasal cannulas in spontaneously breathing children with profound hypocarbia. *Crit Care Med.* 1995;23:1140-2.
119. Oberg B, Waldau T, Larsen VH. The effect of nasal oxygen flow and catheter position on the accuracy of end-tidal carbon dioxide measurements by a pharyngeal catheter in un-intubated, spontaneously breathing subjects. *Anaesthesia.* 1995;50:695-8.
120. Tobias JD, Flanagan JF, Wheeler TJ, Garrett JS, Burney C. Noninvasive monitoring of end-tidal CO₂ via nasal cannulas in spontaneously breathing children during the perioperative period. *Crit Care Med.* 1994;22:1805-8.
121. Amin HM, Cigada M, Fordyce WE, EM C. Noninvasive monitoring of respiratory volume. Experimental evaluation of a breath monitoring device. *Anaesthesia.* 1993;48:608-10.
122. Drummond GB. Breath monitoring device. *Anaesthesia.* 1994;49:552.
123. Van Bergen FH, Weatherhead DS, Treloar AE, Dobkin AB, Buckley JJ. Comparison of indirect and direct methods of measuring arterial blood pressure. *Circulation.* 1954;10:481-90.
124. Rutten AJ, Ilsley AH, Skowronski GA, Runciman WB. A comparative study of the measurement of mean arterial blood pressure using automatic oscillometers, arterial cannulation and auscultation. *Anaesth Intensive Care.* 1986;14:58-65.
125. Bruner JM, Krenis LJ, Kunsman JM, Sherman AP. Comparison of direct and indirect methods of measuring arterial blood pressure, part II. *Med Instrum.* 1981;15:97-101.
126. Bruner JM, Krenis LJ, Kunsman JM, Sherman AP. Comparison of direct and indirect measuring arterial blood pressure. *Med Instrum.* 1981;15:11-21.
127. Cohn JN. Blood pressure measurement in shock. Mechanism of inaccuracy in auscultatory and palpatory methods. *JAMA.* 1967;199:118-22.
128. Borow KM, Newburger JW. Noninvasive estimation of central aortic pressure using the oscillometric method for analyzing systemic artery pulsatile blood flow: comparative study of indirect systolic, diastolic, and mean brachial artery pressure with simultaneous direct ascending aortic pressure measurements. *Am Heart J.* 1982;103:879-86.
129. Hutton P, Dye J, Prys-Roberts C. An assessment of the Dinamap 845. *Anaesthesia.* 1984;39:261-7.
130. Ramsey III M. Noninvasive automatic determination of mean arterial pressure. *Med Biol Eng Comput.* 1979;17:11-8.

131. Yelderman M, Ream AK. Indirect measurement of mean blood pressure in the anesthetized patient. *Anesthesiology*. 1979;50:253–6.
132. Nystrom E, Reid KH, Bennett R, Couture L, Edmonds Jr HL. A comparison of two automated indirect arterial blood pressure meters: with recordings from a radial arterial catheter in anesthetized surgical patients. *Anesthesiology*. 1985;62:526–30.
133. Runciman WB, Ludbrook GL. Monitoring. In: WS N, DJ R, Smith G, editors. *Anaesthesia*. 2nd ed. Cambridge: Blackwell Science; 1994. p. 711.
134. Kurki T, Smith NT, Head N, Dec-Silver H, Quinn A. Noninvasive continuous blood pressure measurement from the finger: optimal measurement conditions and factors affecting reliability. *J Clin Monit*. 1987;3:6–13.
135. Jones RD, Kornberg JP, Roulson CJ, Visram AR, Irwin MG. The Finapres 2300e finger cuff. The influence of cuff application on the accuracy of blood pressure measurement. *Anaesthesia*. 1993;48:611–5.
136. Wilkes MP, Bennett A, Hall P, Lewis M, Clutton-Brock TH. Comparison of invasive and non-invasive measurement of continuous arterial pressure using the Finapres in patients undergoing spinal anaesthesia for lower segment caesarean section. *Br J Anaesth*. 1994;73:738–43.
137. Lake CL. *Clinical monitoring for anesthesia and critical care*. 2nd ed. Philadelphia: WB Saunders; 1994. p. 106.
138. MacKenzie R, Asbury AJ. Clinical evaluation of liquid crystal skin thermometers. *Br J Anaesth*. 1994;72:246–9.
139. Byrick RJ, Cohen MM. Technology assessment of anaesthesia monitors: problems and future directions. *Can J Anaesth*. 1995;42:234–9.
140. Myles PS, Williams NJ, Powell J. Predicting outcome in anaesthesia: understanding statistical methods. *Anaesth Intensive Care*. 1994;22:447–53.
141. Roizen MF, Toledano A. Technology assessment and the “learning contamination” bias. *Anesth Analg*. 1994;79:410–2.
142. Fryback DG, Thornbury JR. The efficacy of diagnostic imaging. *Med Decis Making*. 1991;11:88–94.
143. Drummond GB. Influence of thiopentone on upper airway muscles. *Br J Anaesth*. 1989;63:12–21.
144. Montravers P, Dureuil B, Desmots JM. Effects of i.v. midazolam on upper airway resistance. *Br J Anaesth*. 1992;68:27–31.
145. Nishino T, Kochi T. Effects of sedation produced by thiopentone on responses to nasal occlusion in female adults. *Br J Anaesth*. 1993;71:388–92.
146. Abboud TK, Khoo SS, Miller F, Doan T, Henriksen EH. Maternal, fetal, and neonatal responses after epidural anesthesia with bupivacaine, 2-chloroprocaine, or lidocaine. *Anesth Analg*. 1982;61:638–44.
147. Kuhnert BR, Harrison MJ, Linn PL, Kuhnert PM. Effects of maternal epidural anesthesia on neonatal behavior. *Anesth Analg*. 1984;63:301–8.
148. Wiener PC, Hogg MI, Rosen M. Neonatal respiration, feeding and neurobehavioural state. Effects of intrapartum bupivacaine, pethidine and pethidine reversed by naloxone. *Anaesthesia*. 1979;34:996–1004.
149. Garner L, Stirt JA, Finholt DA. Heart block after intravenous lidocaine in an infant. *Can Anaesth Soc J*. 1985;32:425–8.
150. Van Dorsten JP, Miller FC. Fetal heart rate changes after accidental intrauterine lidocaine. *Obstet Gynecol*. 1981;57:257–60.
151. Wood CE, Goresky GV, Klassen KA, Kuwahara B, Neil SG. Complications of continuous epidural infusions for postoperative analgesia in children. *Can J Anaesth*. 1994;41:613–20.
152. Bougher RJ, Ramage D. Spinal subdural haematoma following combined spinal-epidural anaesthesia. *Anaesth Intensive Care*. 1995;23:111–3.
153. Tarkkila P, Huhtala J, Tuominen M. Transient radicular irritation after spinal anaesthesia with hyperbaric 5 % lignocaine. *Br J Anaesth*. 1995;74:328–9.
154. Horlocker TT, Cabanela ME, Wedel DJ. Does postoperative epidural analgesia increase the risk of peroneal nerve palsy after total knee arthroplasty? *Anesth Analg*. 1994;79:495–500.
155. Mills GH, Howell SJ, Richmond MN. Spinal cord compression immediately following, but unrelated to, epidural analgesia. *Anaesthesia*. 1994;49:954–6.
156. Murthy BV, Fogarty DJ, Fitzpatrick K, Brady MM. Headache during epidural top-ups in labour—a sign of reduced intracranial compliance. *Anaesth Intensive Care*. 1995;23:744–6.
157. Haggard HW. The place of the anesthetist in American medicine. *Anesthesiology*. 1940;1:1.
158. Slogoff S, Hughes FP, Hug Jr CC, Longnecker DE, Saidman LJ. A demonstration of validity for certification by the American Board of Anesthesiology. *Acad Med*. 1994;69:740–6.
159. Burt DE. Virtual reality in anaesthesia. *Br J Anaesth*. 1995;75:472–80.
160. Duncan PG, Cohen MM, Yip R. Clinical experiences associated with anesthesia training. *Ann RCPSC*. 1993;26:363.
161. Sivarajan M, Miller E, Hardy C, Herr G, Liu P, Willenkin R, Cullen B. Objective evaluation of clinical performance and correlation with knowledge. *Anesth Analg*. 1984;63:603–7.
162. Rosse C. The potential of computerized representations of anatomy in the training of health care providers. *Acad Med*. 1995;70:499–505.
163. Ellis FR. Measurement of competence. *Br J Anaesth*. 1995;75:673–4.
164. Kestin IG. A statistical approach to measuring the competence of anaesthetic trainees at practical procedures. *Br J Anaesth*. 1995;75:805–9.
165. Sagan SD. *The limits of safety*. Princeton University Press: Princeton; 1993. p. 46
166. Rudge BA. Decision-making in anaesthesia. *Anaesth Intensive Care*. 1995;23:597–9.
167. Eichhorn JH, Cooper JB, Cullen DJ, Maier WR, Philip JH, Seeman RG. Standards for patient monitoring during anesthesia at Harvard Medical School. *JAMA*. 1986;256:1017–20.
168. Merchant R, Chartrand D, Dain S, et al. Guidelines to the practice of anesthesia—revised edition. *Can J Anaesth*. 2012;59:1.
169. Battista RN, Hodge MJ. Clinical practice guidelines: between science and art. *CMAJ*. 1993;148:385–9.
170. Carter AO, Battista RN, Hodge MJ, Lewis S, Basinski A, Davis D. Report on activities and attitudes of organizations active in the clinical practice guidelines field. *CMAJ*. 1995;153:901–7.
171. Lomas J. Words without action? The production, dissemination, and impact of consensus recommendations. *Annu Rev Public Health*. 1991;12:41–65.