Brendan T. Finucane
Ban C.H. Tsui
Editors

Complications of Regional Anesthesia

Principles of Safe Practice in Local and Regional Anesthesia

Third Edition

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We would like to dedicate this edition of the book to our patients, our teachers, our students, and our families.

Brendan T. Finucane, MB, BCh, BAO, FRCA, FRCPC
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Preface

We are now ready to publish the third edition of ***Complications of Regional Anesthesia*** which was first published 17 years ago. The title remains the same but we have added the subtitle, *Principles of Safe Practice in Local and Regional Anesthesia,* to stress the relatively new emphasis and importance on safety and prevention and to broaden our horizons to include some discussion about the practice and the administration of not just **Regional** but also **Local Anesthesia**.

We have made some significant changes to the book which we hope you approve. First of all this is a much more comprehensive edition going from 24 to 35 chapters, and we have also divided the book into seven separate parts based mostly on logic. In the opening part entitled **General** considerations, we started out with a chapter on the History of Regional Anesthesia which seemed like a good place to start. We also addressed the issue of Safety of Regional Anesthesia. It is difficult to discuss much about regional and local anesthesia without mentioning toxicity of local anesthetics which has been a problem with regional and local anesthesia since its inception more than 130 years ago, and we finished up that section with a good discussion of Outcomes comparing Regional and General Anesthesia. In the second part we addressed **Special** considerations, which includes a chapter on Mechanisms of Nerve injury, Infection, Catheter techniques, and the whole issue of regional anesthesia in the presence of neurologic disease and how to evaluate neurologic injury following regional anesthesia. We then dedicated several chapters to **Specific** blocks involving anatomic regions of the body specifically addressing safety and management of adverse events. We dedicated the next part to specific **Patient Populations**—the young, the old, the pregnant, obese, and those suffering from chronic pain. The next part is new territory for us and is entitled **Special Environments**. We invited a group of practitioners, mostly surgeons, who frequently use local anesthetics in their practices, to share their expertise and experiences with us. Among this group of specialists are dentists, ophthalmologists, emergency room physicians, orthopedists, and plastic surgeons. We have a lot to learn by sharing our experiences using local and regional anesthesia with specialists outside our own discipline and they from us. We dedicated a part to **Morbidity Studies** and this part includes writers from across the world adding an **International** flavor, as we are sometimes accused of being too insular in North America. We dedicated the final part to **Medical Legal Aspects** of Local and Regional Anesthesia, which we must realistically face in the modern world of this twenty-first century.

Labat, in the 1920s, was the first fully trained specialist in Regional Anesthesia, and he influenced the leaders of this new emerging specialty of anesthesiology to use regional anesthesia in their practices. Most anesthesiologists at that time opted for general anesthesia because of its predictability. Tremendous advances have been made in Regional Anesthesia in the past 30 or 40 years, so much so that most anesthesiologists in the modern era have become interested in regional anesthesia again because there is far more predictability in the practice of regional anesthesia than ever before. We can now actually see what we are doing instead of blindly seeking neural targets, based on our knowledge of anatomy. Most anesthesiologists fully appreciate the enormous benefits of regional anesthesia to patients especially in the postoperative period but also long term. However, despite good practice, we encounter problems

and unforeseen circumstances, so practitioners must be fully aware of the many pitfalls and complications associated with the practice of regional anesthesia even though we have made enormous advances in recent years.

This edition is much more comprehensive than our previous efforts and more inclusive and there are more pages, tables, diagrams, and colored illustrations. This text is also comprehensively referenced. As in previous editions, there is some repetition and that is inevitable. However, it is refreshing to compare anesthesia practitioners' experiences from around the world and from outside our own discipline. Local Anesthetic Systemic Toxicity (LAST) is a very common theme among all who practice Local and Regional Anesthesia, and we have learned a lot about prevention and treatment of this malady in the past 30 years. Fortunately most of the complications we have discussed are rare and all too often we appear to shoulder the blame for injuries that we did not cause in the first place.

Our main emphasis is on safety and prevention of injury in the practice of local and regional anesthesia, and we have called upon a great variety of experts from around the world to share their experiences with us. We hope you appreciate the changes we have and as always we welcome your critique and recommendations for improvement.

There is one other important change I have made in this edition and that is I have invited my colleague and friend from the Department of Anesthesiology and Pain Medicine from the University of Alberta to co-edit this edition of the text with me. He has contributed enormously to our knowledge of local and regional anesthesia in the past two decades and helped a great deal with this latest version.

Edmonton, AB, Canada — Brendan T. Finucane
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To my wife, Eliza, and my children, Jenkin and Jeremy—the real loves of my life. Without their support and understanding, I could not have completed this demanding project. I would also like to dedicate this opus to my parents, Woon-Tak and Kau-Wan, for their love and guidance throughout my life.

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Part I

General Considerations

The History of Local and Regional Anesthesia

1

Brendan T. Finucane

Key Points

- The discovery of the local anesthetic properties of cocaine by *Koller* in 1884 was one of the most important discoveries in the history of Medicine and revolutionized the practice of Ophthalmology, Dentistry, Anesthesia, and Surgery.
- Chemists studied the pharmacological properties of cocaine and developed a series of synthetic local anesthetic compounds which were less toxic than cocaine and more predictable and efficacious.
- Systemic toxicity to local anesthetics continues to be an issue, but we have seen a significant reduction in the incidence of this problem and great advances in prevention and management.
- Spinal anesthesia was first introduced by *Bier* in 1884 and today remains one of the most reliable and safe techniques used in regional anesthesia more than 120 years after it was first introduced.
- *Bier* also introduced Intravenous Regional Anesthesia in 1908 (Bier Block) and this technique has also withstood the test of time and remains one of the most reliable techniques for short surgical procedures involving the upper extremity.
- A succession of leading figures in regional anesthesia have introduced and developed a number of safe and effective local and regional techniques, including epidural anesthesia and numerous peripheral nerve blocks. The lives of these great contributors to local and regional anesthesia are highlighted in this chapter, all of whom also wrote classic textbooks on the subject of regional anesthesia.
- The introduction of nerve stimulation more than 40 years ago represented a significant advance in the practice of regional anesthesia and the importance of this advance is emphasized in this chapter.
- The recent introduction of ultrasonography has transformed regional anesthesia practice, increasing safety and precision of nerve blocks.

Definitions

Regional anesthesia is defined as the selective blockade of a nerve or group of nerves supplying an area of the body such as a limb(s) or an eye, using local anesthetics, thereby allowing a surgeon to operate on a patient without the need for full general anesthesia. *Local* anesthesia is a non-selective blockade of a smaller area of the body by infiltrating with local anesthesia directly into the skin, subcutaneous, and deeper tissues, without any attempt to target a particular nerve. *Topical* anesthesia refers to anesthesia of the skin or mucous membranes which occurs following topical application of a local anesthetic.

A number of different approaches to regional anesthesia were tried before and after general anesthesia was introduced in 1846, but none of them were satisfactory. These included: nerve compression, refrigeration, alcohol injections, acupuncture, and ether sprays, but no real progress was made until the discovery of local anesthetics.

Of course in order to perform local and regional anesthesia, we must have a delivery system. Therefore, you should know that *Sir Francis Rynd* performed the first nerve block injection for the treatment of trigeminal neuralgia using morphine dripped through a cannula and this took place in the Meath Hospital in Dublin, Ireland, in 1844 [1]. *Alexander Wood* improved on this by producing a hollow needle in 1853 [2]. And the hypodermic syringe, known in Europe as the *Pravaz* syringe, was introduced in 1853 [3].

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The Discovery of Local Anesthetics

It has been known for centuries that the chewing of the coca leaf resulted in numbness of the tongue and lips. *Gaedeke* extracted the active principle of the coca leaf in 1855 and named it erythroxyline [4]. In 1858, the Austrian government sent the frigate Novara on an expedition around the world. A trade expert on board named Dr.Scherzer took samples of the coca leaf and upon return gave them to a knowledgeable chemist at the University of Gottingen in Germany, named Wohler. Dr.Wohler and his assistant *Niemann* isolated the crystal extract from the coca leaf and named the alkaloid cocaine [5].

Moreno y Maiz, a Peruvian army surgeon, saw the potential of sensory anesthesia with cocaine in a manual he wrote for the military in 1868 [6]. *Van Anrep* in 1879 observed the local numbing effects of cocaine on the throat and the dilation of the pupil upon local application to the eye, but he did not observe that the conjunctiva was anesthetized [7]. However, *Karl Koller* (Fig.1.1) put all this information together and discovered the local anesthetic properties of cocaine [8]. This happening deserves the full details.

Koller had studied cocaine in depth as a result of his friendship with *Freud* when they were in Vienna, so he was very knowledgeable about the compound. He was also highly motivated to find a suitable analgesic for patients undergoing eye surgery. General anesthesia was not used by ophthalmologists for cataract surgery because of severe post-operative nausea and vomiting frequently associated with its use, so most cataracts were performed without any anesthesia. Following is an extract from Koller's own writing on the topic:

> *The unsuitability of general narcosis for eye operations; for not only is the co-operation of the patient greatly desirable in these operations, but the sequelae of general narcosis-vomiting, retching and general restlessness-are frequently such as to constitute a grave danger to the operated eye; and this was especially the case at the time when narcosis was not skilfully administered as it is now, by trained experts. Eye operations were formerly being done without any anesthesia whatsoever* [9]

Fig. 1.1 Karl Koller (1857–1944). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

Following is a description of cataract surgery performed without anesthesia in 1882:

"*It was like a red-hot needle in yer eye whilst they was doing it. But he wasn't long about it. Oh no. if he had been long I couldn't ha' beared it. He wasn't a minute more than three quarters of an hour at the outside*"—an old man's description of his cataract operation to Thomas Hardy and his wife on their visit to Dorsetshire in 1882 [10].

Freud and *Koller* both worked at the same hospital in Vienna, and in the summer of 1884, *Freud* planned a trip to Germany and asked *Koller* if he would continue clinical research on cocaine in his absence. *Koller* agreed to do so. *Freud* had left some of the powdered cocaine to continue the experiments. *Koller* allowed one of his colleagues (Engel) to taste the cocaine and Engel said: "*how that numbs the tongue*". *Koller* immediately said: "*Yes that has been noticed by everyone that has eaten it' and in the moment it flashed upon me that I was carrying in my pocket the local anesthetic for which I had searched some years earlier.*"

Koller went straight to his laboratory and asked his assistant for a guinea pig for the experiment. This moment was observed by Dr.Gaertner, an assistant in Stricker's laboratory, who said the following. "*A few grains of cocaine were dissolved in a small quantity of distilled water. A large lively frog was selected from the aquarium and held immobile in a cloth, and now a drop of the solution was trickled into one of the protruding eyes. At intervals of a few seconds the reflex of the cornea was tested by touching the eye with a needle...After about a minute came the great historic moment, I do not hesitate to designate it as such. The frog permitted his cornea to be touched and even injured with out a trace of reflex action or attempt to protect himself, where as the other eye responded with the usual reflex action to the slightest touch. 'Now it was necessary to go one step further and to repeat the experiment upon a human being. We trickled the solution under the upraised lids of each other's eyes. Then we put a mirror before us, took a pin in hand and tried to touch the cornea with its head. Almost simultaneously we could joyously assure ourselves, "I can't feel a thing*".

This information was obtained from Koller's daughter who went through his papers after his death and found notes

her father had left about the actual discovery. This information was published in the Psychoanalytic Quarterly in 1963 some 20 years after *Koller's* death in 1944 [11].

Koller's discovery had an enormous impact immediately. Within 1 year of his discovery, cocaine was used in all parts of the developed world for cataract surgery. *Koller* was just 27 years of age when he made the discovery that led to the widespread use of local anesthetics all over the world. Local anesthetics are still among the most important and frequently used medications in Medicine, Surgery, and Dentistry and Anesthesia today. It is interesting to note that *Morton* gave his first public demonstration of etherization when he was 27 years old. By the turn of the twentieth century, General, Local, Regional, and Topical Anesthesia had all been discovered.

Evolution of Local Anesthetics

It soon became apparent that cocaine was a very toxic substance, and between 1884 and 1891, 200 cases of toxicity had been reported and as many as 13 deaths had occurred [11]. Cocaine was also an addictive substance. Chemists and pharmacologists studied the structure of cocaine and this led to the introduction of the first synthetic local anesthetic, novocaine [12], (later to be named *procaine*) in 1904. *Procaine* was an *ester* compound, and although much less toxic than cocaine, was not the most reliable local anesthetic, was quite short acting, and was somewhat unstable when sterilized and was associated with allergies. In the ensuing years, numerous local anesthetics were tested with variable results, but *procaine*, even with its limitations, was still considered to be the gold standard for almost 50 years. In the 1940s, *Löfgren* and *Lundqvist* from Sweden experimented with local anesthetic compounds and discovered *Xylocaine* (LL30), also known as lidocaine, an *amino-amide* compound which proved to be an outstanding local anesthetic [13]. Lidocaine was the prototype and quickly replaced procaine (novocaine) as the gold standard of local anesthetics. These compounds proved to be very stable and allergies occurred rarely. To this very day, *Xylocaine* is still considered the gold standard of local anesthetics and it is interesting that its discovery, like the local anesthetic effects of cocaine, was first uncovered by tasting! (*Löfgren* used taste to determine which local anesthetic compound was better than another—from the book entitled, "Xylocaine: a discovery, a drama, an industry," by *Lindqvist and Sundling* [14].)

Systemic toxicity was a problem with all local anesthetics from the very beginning and continues to be a problem to this day. The most serious reactions occur when local anesthetics are injected into the circulation (in error). Although the *amino-amide* compounds proved to be highly effective and relatively safe, the duration of action was a limiting factor with their use. The addition of epinephrine prolonged the duration of action of these compounds significantly, but the maximum reliable duration was only about 2–4 h for most major nerve blocks. The search continued for the ideal local anesthetic. In 1957, *Bo Af Ekenstam* introduced a new group of long-acting local anesthetics and these were the *pipecholylxylidine* compounds represented prominently by bupivacaine [15]. This group of compounds presented a new set of problems in that they were highly toxic not just to the central nervous system (CNS), but also to the cardiovascular system. Etidocaine and bupivacaine were the first *pipecholylxylidine* compounds used clinically and were approved for use in humans in the early 1960s, first in Europe and later in the United States. They were characterized by a markedly increased duration of action compared to lidocaine and were initially received with great enthusiasm. Etidocaine was much faster acting than bupivacaine because it was highly lipid-soluble, but was associated with profound motor blockade that sometimes outlasted the sensory blockade, which was very disturbing to some patients. This unusual problem was only one factor that led to etidocaine being relegated to the shelf. In 1979, *Albright* wrote a powerful editorial exposing the dangers of both etidocaine and bupivacaine[16]. Both of these local anesthetics were associated with numerous deaths in both the United States and the United Kingdom due to selective and lethal cardio-toxicity that did not come to light for more than 10 years after the drugs were first approved for clinical use. A number of the fatalities reported with these compounds occurred in healthy young patients and a high percentage of these fatalities occurred in young parturients. Unlike the *amino-amides* and *amino esters*, the *pipecholyl xylidine* compounds caused serious cardiac toxicity at blood levels close to those associated with CNS toxicity. Furthermore, treatment of both CNS and cardiac toxicity was very difficult and required prolonged and aggressive resuscitation as these compounds were highly lipid-soluble and attached firmly to both CNS and cardiac receptors. This episode led to a major investigation of these compounds by the FDA and restrictions were placed on the use of these compounds thereafter. The practice of regional anesthesia and use of local anesthetics was carefully scrutinized by the leaders in the field of regional anesthesia, which led to a series of safety guidelines published by the American Society of Regional Anesthesia. Furthermore, the academic anesthesia community was again challenged to produce a safe and reliable local anesthetic.

Just as the anesthesia community was recovering from the bupivacaine/etidocaine tragedy it was faced with another toxicity problem, this time associated with the use of 2-chloroprocaine (Nesacaine-CE). This ester compound was synthesized in 1949 and promoted by *Foldes* for obstetric anesthesia based on a greatly reduced potential for systemic toxicity [17]. *Ansbro* et al. estimated that the risk of systemic toxicity was 1/20 that of lidocaine when injected epidurally[18]. It became very popular in obstetric anesthesia because the risk to the fetus from trans-placental transfer was practically eliminated. In the early 1980s, there were reports of serious neural deficits following accidental subarachnoid injection of 2-chloroprocaine in

obstetric patients. The formulation of 2-chloroprocaine used contained preservatives (sodium bisulfite) and was not intended for subarachnoid use. The controversy continued for years afterwards as to whether the neural deficits were caused by the local anesthetic itself or the preservative. Eventually, a preservative-free chloroprocaine was introduced and is now being used for spinal anesthesia in ambulatory patients in some medical centers in the United States.

When all the controversy about systemic and neural toxicity of local anesthetics subsided, most clinicians agreed that, despite the toxicity potential of bupivacaine, it was otherwise an excellent local anesthetic.

This discussion brings us into the world of stereochemistry[19]. If we take a closer look at the chemistry of bupivacaine, we find that it is a *chiral* compound and can exist in two forms (enantiomers) depending on how each one responds to polarized light. Enantiomers have identical physical properties and have the same chemical formula and the only way they differ is in how they respond to polarized light. The enantiomer is dextrorotatory R (+) if polarized light is rotated to the right and levorotatory S (−) if rotated to the left. Bupivacaine is a racemic mixture containing equal parts of both enantiomers that neutralize each other and therefore do not rotate the plane of polarized light. In the process of studying stereochemistry, investigators learned that the S enantiomer of bupivacaine was less cardiotoxic. The S enantiomer was produced and marketed as levo-bupivacaine (Chirocaine) and proved to be less likely to cause cardiotoxicity. Ropivacaine was subsequently introduced after in-depth study and it too is the S enantiomer and theoretically even less toxic than levo-bupivacaine.

The pharmaceutical industry invested a huge amount of Research and Development funds into the development of the *chiral* compounds and it is unlikely that they will invest much more in this area of research at least in the near future. Yet there is a serious need for a good short-acting local anesthetic for spinal anesthesia in ambulatory surgery. There is still some discomfort among clinicians about using 2-chloroprocaine in spinal anesthesia. And after 50 years of apparent safe use, 5 % lidocaine is no longer acceptable as a spinal anesthetic as a result of reports of Transient Neurologic Symptoms in a significant number of patients following its use [20]. Also, more serious side effects have been reported with lidocaine 5 %, following subarachnoid injection through continuous micro-catheters.

Although the issue of systemic toxicity to local anesthetics continues to be a permanent risk, a great breakthrough has taken place recently in the treatment of this malady. Like many advances in medicine, it was accidentally discovered that systemic injections of lipids acted as a sponge which soaked up lipid-soluble medications and quickly and efficiently reduced the concentration of these toxic compounds in the circulation [21]. This is a brief summary of the history of local anesthetics without which we could not have Regional Anesthesia. Please refer to Chap. 3 for a more complete discussion of Local Anesthetics and Systemic Toxicity of local anesthetics.

The Birth of Regional Anesthesia

The same year that *Koller* discovered local anesthetics (1884), *Halsted* performed a brachial plexus block in a patient in the United States and so began the practice of Regional Anesthesia using injectable local anesthetics [22].

Leonard Corning (Fig.1.2), a neurologist from New York, was most likely the first person to perform spinal anesthesia, but apparently was not fully aware that he had done so at the time [23]. He described an experiment on a dog in which he injected 1.18 mL of 2 % cocaine hydrochloride into the space "situated between the spinous processes of two inferior dorsal vertebrae" with the result that the animal did not react for several hours afterwards if a stimulus was applied from a powerful faradic battery or through pinching or pricking the hind limbs. He did a similar experiment on a human with the same results and concluded the following: *Corning* actually believed that cocaine injected into the region between the two spinous processes was absorbed by the veins and 'then transferred to the substance of the cord and gave rise to anesthesia of the sensory and perhaps motor tracts of the same'. He said this in his own writings. *Corning* was more interested in relieving pain than he was of producing anesthesia. *Corning* was a prolific writer, and in 1894, he described 'The irrigation of the cauda equina with medicinal fluids…' "*I became impressed with the desirability of introducing remedies directly in to the spinal canal with a view to produc-*

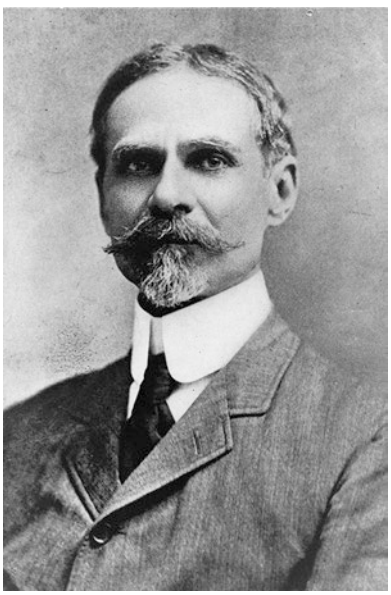

Fig. 1.2 James Leonard Corning (1855–1923). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

ing still more powerful impressions on the cord and more especially on its lower segment." Probably, the reasons why *Corning* did not make the connection between the injection of the local anesthetic and spinal anesthesia was that when he inserted a needle he always had a syringe attached to it. So he never saw CSF drip back and therefore perhaps did not appreciate that he was in the subarachnoid space on some of these occasions, which would explain some of his observations. However, he still deserves the credit for the first subarachnoid injection of a local anesthetic.

Corning published one of the first textbooks on Local Anesthesia in 1886 [24], and the first textbook on pain in 1894 [25], but nothing came of his suggested use of spinal anesthesia for surgery.

The Discovery of Spinal Anesthesia by Bier

Another dramatic breakthrough occurred in Regional Anesthesia in 1898 and that was the first recording of spinal anesthesia in a human by *August Bier* [26]. *Bier* was influenced by his senior mentor surgeon *Heinrich Quincke* who studied in depth the anatomy of the spinal canal and the spinal nerves and who pioneered the technique of lumbar puncture and treated patients with hydrocephalus and tuberculous meningitis by performing lumbar puncture as a therapeutic intervention [27]. Figure 1.3 shows a picture of *August Bier*, one of the great figures of surgery in Germany. He was born near Waldeck in Germany in 1861. He was educated in Berlin and Leipzig and graduated from medical school at Kiel in 1889 and dedicated his life to surgery and he worked as an assistant to the famous surgeon Professor *Friedrich von Esmarch* [28]. In 1898, Bier worked with *Heinrich Irenaeus Quincke*. He was also familiar with *Koller's* work with cocaine. It is likely that he put the two ideas together and developed the technique of spinal anesthesia, a technique that we perform today in much the same way it was performed by *Bier* 119 years ago. Bier anticipated that the injection of cocaine into the subarachnoid space would result in anesthesia of the lower body. He described his technique in 6 patients using 10–20 mg of cocaine and the first of these experiments occurred on August 16 1898. *Bier* was not happy with the initial results because the patients had intractable headaches and many of them were vomiting for days afterwards. *Bier* decided that he needed to experiment a little more before suggesting that this was a viable and safe technique. In his opinion, the results were not much better than those achieved with chloroform. *Bier* asked his colleague *Hildebrandt* to perform spinal anesthesia on him. *Hildebrandt* obliged but had trouble attaching the syringe containing the cocaine to the needle, and by the time he did so, most of the CSF had drained from the spinal canal and no anesthesia developed. *Hildebrandt* obliged *Bier* by inviting him to perform spinal anesthesia on him. *Bier* successfully performed a lumbar puncture on his colleague and then injected 5 mg of cocaine and obtained a very satisfactory spinal block, and to prove the success of this block, they performed a number of tests including pulling the pubic hair, hard pressure on and pulling of the testes, and a sharp blow with an iron on the shin! These experiments which began at 7.30 PM in the evening were followed by dinner, wine, and cigars. Both volunteers suffered headaches and nausea and vomiting for a day or 2 afterwards. *Bier's* symptoms of headache and dizziness were relieved when he lay down and could easily be attributed to leakage of CSF, and those of *Hildebrandt*, which included vomiting, suggest that meningeal irritation may have been the cause. *Bier* was quite discouraged by his observations and did not feel justified in continuing his work on patients without further animal work. *Bier* published the first paper on spinal anesthesia in 1899 and this was followed by another paper on this topic 3 months later by *Tuffier* in France [29]. *Tuffier* was more enthusiastic about his experiences and reports from America soon after supported this. One of the first reports of spinal anesthesia performed in the United States was written up by *Matas* et al. from Charity Hospital in New Orleans in the United States on December 18 1899 [30]. The technique was not widely practiced until newer and safer local anesthetics were introduced.

Fig. 1.3 Professor August Bier (1861–1949). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

While we can all agree that the discovery of local anesthetics truly heralded the dawning of regional anesthesia, the discovery of spinal anesthesia was a huge advance. As mentioned before, the novelty and enthusiasm of general anesthesia was waning especially when deaths were reported and so spinal anesthesia was greeted with great enthusiasm by the surgeons, who were not used to the profound degree of muscle relaxation associated with its use, especially when performing abdominal surgery.

Spinal anesthesia was the mainstay of regional anesthesia for the first 20 years or so of its use. During that time, great advances were made in the physiology and pharmacology of spinal anesthesia. The concept of baricity was introduced [31], new local anesthetic mixtures were used, and spinal anesthesia was found to be highly successful especially for procedures involving the lower abdomen, perineum, and lower extremity. Continuous techniques were used first using a malleable needle and subsequently continuous catheters were inserted for prolonged surgery. The great advantage of spinal anesthesia was the profound muscle relaxation associated with its use particularly for abdominal surgery. At the same time, the major drawback even today is the problem of spinal headache which, even with greatly advanced needle technology, continues to tarnish the reputation of a technique that has withstood the test of time.

Sir Robert Macintosh (Fig.1.4) was one of the great proponents of spinal anesthesia and wrote a remarkable handbook named *Lumbar Puncture and Spinal Anesthesia*, which has amazing illustrations and is still available today [32]. The fourth edition was published in 1978 by Lee and Atkinson and many more editions have been published since then. Spinal anesthesia was very popular in Great Britain until a very highly publicized tragedy involving spinal anesthesia was reported in the British Press (*Times*) in 1947 (*Wooley and Roe*) [33]. In this case, two patients in adjoining operating rooms remained permanently paralyzed following spinal anesthesia for relatively minor procedures. This report put an end to spinal anesthesia in the United Kingdom (UK) for the ensuing 50 years. *Sir Robert Macintosh* testified at the trial. The doctor involved in these cases was acquitted at trial. Spinal anesthesia came under serious scrutiny in the United States a few years later when a report by a prominent (former British) neurologist (*Foster Kennedy*) inferred that spinal anesthesia was associated with permanent paralysis, based on his observations in a series of 12 cases of paralysis following spinal anesthesia [34]. However, *Kennedy's* allegations were proven to be incorrect in a subsequent report by *Dripps and Vandam,* when they published one of the first major outcomes studies of spinal anesthesia involving more than 10,000 cases [35]. These authors definitively proved that spinal anesthesia was rarely associated with paralysis.

Fig.1.4 Professor Macintosh (1897–1989). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

Evolution of Regional Anesthesia

Regional anesthesia was greeted with great enthusiasm by surgeons at least initially because it gave them a sense of independence and autonomy because they did not have to rely on someone else to induce unconsciousness, which in those days could take as long as 30 min in the best of hands. The surgeon was now in control of his/her own destiny. This worked very well with spinal anesthesia, but not so well with other forms of regional anesthesia because the discipline of regional anesthesia was still in its infancy. Sometimes we forget that regional anesthesia was originally firmly in the domain of surgery.

Intravenous Regional Anesthesia (The Bier Block)

Bier's name is also associated with another remarkable regional anesthesia technique known as the *Bier* block [36]. *Bier* was mentored by *Friedrich von Esmarch*, a famous German surgeon who introduced the Esmarch bandage. One of *Bier's* other important discoveries was the use of passive hyperemia using the Esmarch bandage to treat tuberculous bones and joints in 1907. This likely led to his idea of intravenous regional anesthesia. This idea was not very practical initially because it required a venous cut-down at the elbow. Sixty years later, a simple modification of *Bier's* technique by *C Mck Holmes* established the *Bier* block as one of the most reliable regional anesthesia techniques for upper extremity surgery of short duration [37]. Instead of using a cut-down, *Mck Holmes* inserted a plastic cannula into the venous system and the local anesthetic was injected below an inflated tourniquet. *The Bier block* or intravenous regional anesthesia remains one of the most reliable forms of regional anesthesia of the upper extremity for procedures lasting 45 min or less. The technique can also be used for lower extremity surgery, but not as reliably or safely.

Regional Anesthesia-Pre-emptive Analgesia

One of the early enthusiasts of regional anesthesia in America was *George Crile*, the founder of the Cleveland Clinic [38]. His theory of "anoci-association" was quite advanced at that time.

He recognized that patients still responded to noxious stimuli under general anesthesia, but that this response was blocked in patients who had combined regional/general anesthesia. He theorized that by preventing the noxious stimuli from reaching the brain, he prevented "surgical shock" in some patients. This theory was formulated in 1908 and was the forerunner of a more recent theory of 'pre-emptive analgesia' put forward by *Woolf* et al. in 1993, proving in animals at least, that we can prevent or greatly reduce 'wind up', altering in a positive way the metabolic response to trauma and greatly reduce or prevent the risk of chronic pain following surgery [39].

Peripheral Nerve Blockade

Victor Pauchet (1869–1936) was another great pioneer of regional anesthesia in France in the early 1900s and wrote a text book on the subject of regional anesthesia and fostered the idea of using peripheral nerve blocks in surgery, including intercostal and paravertebral blocks in addition to spinal anesthesia [40]. *Gaston Labat* was one of Pauchet's trainees [41]. In 1920, *Charles Mayo* was visiting *Pauchet* in his hospital in Paris demonstrating some surgical techniques[42]. *Mayo* was quite impressed by *Labat's* skill set in regional anesthesia and invited him to Rochester, Minnesota in the USA, to teach regional anesthesia to his colleagues. *Labat* impressed a number of the doctors at Mayo, but his tenure there was short, but he did manage to publish an outstanding textbook entitled: *Regional Anesthesia-Techniques and Application* (on the basic principles of regional anesthesia) in 1922. This text book is still considered to be one of the classic textbooks ever published on the topic of Regional Anesthesia. *Labat* moved to New York to Bellevue hospital and worked with and taught *Emery Rovenstine* the principles of regional anesthesia. *Labat* was a great teacher of regional and his book was by today's standards a medical best seller with more than 10,000 copies sold during his lifetime. *Labat* had a significant following in New York and his enthusiasm as a teacher of regional anesthesia led to the formation of the American Society of Regional Anesthesia (ASRA) in 1923. This group consisted mostly of surgeons in the beginning, but with time specialists in anesthesia dominated the group. *Labat* was the first physician to dedicate his career solely to regional anesthesia. He was initially trained as a surgeon, but spent most of his career performing, teaching, and writing about regional anesthesia. *Labat* died from complications following a cholecystectomy in New York in 1934. ASRA was disbanded in 1939 and was reformed again in 1975 by *Alon Winnie*, *Don Bridenbaugh*, *Harold Carron*, *Jordan Katz*, and *Pritvi Raj* (Founding Fathers). *Labat's* name is memorialized by the annual award (Medal) given by the ASRA for outstanding contributions to Regional Anesthesia.

Epidural Anesthesia

Sicard and Cathelin injected cocaine into the epidural space caudally in 1901 [43, 44]. *Fidel Pages-Mirave* described the lumbar approach to the epidural space in 1923 [45]. *Dogliotti* popularized the technique in the 1930s when he described the "loss of resistance technique" [46] and *Curbelo* introduced continuous epidural anesthesia in 1949 [47]. *Hingson* popularized continuous caudal anesthesia in obstetrics anesthesia in the 1940s [48]. The progress of regional anesthesia was slow, but the technique of spinal anesthesia was always an important technique in the hands of most anesthesiologists.

There were a number of strong proponents of regional anesthesia in Europe and North America in the middle of the last century, but a few names deserve special mention. Regional anesthesia was one of those pursuits that required the most enthusiastic followers because, with the exception of spinal and epidural anesthesia, there were not many followers especially when it came to peripheral nerve blocks. Most practitioners preferred general anesthesia because it was far more predictable and easier to perform.

Development of Regional Anesthesia Post WW II

Danny Moore from the Mason Clinic published an outstanding textbook on Regional Anesthesia in 1953 entitled: *Regional Block* [49]. It was the most popular book on the topic of regional anesthesia since *Labat's* classic textbook was first published in 1922. In this book, *Moore* described how to perform most regional anesthesia nerve blocks and promoted regional anesthesia on a very broad scale. *Moore* also published a very good textbook on *Complications of Regional Anesthesia (1955)* [50]. He trained a large number of residents and fellows in regional anesthesia from around the world. He led the renaissance in regional anesthesia in the USA in the post-WW II for close to 50 years and was a legend in his own time.

John Bonica was another great proponent of regional anesthesia for Obstetric patients and published an outstanding book on this topic entitled *Principles and Practice of Obstetric Analgesia and Anesthesia* [51]. He also promoted the use of regional anesthesia for chronic pain therapy and wrote two definitive textbooks on these topics, both of which are anesthesia classics. In 1990, Pope John Paul II requested a copy of his book entitled *The Management of Pain* [52]. *John Bonica* was a pioneer in the discipline of chronic pain and was the leader in establishing one of the first multi-disciplinary Pain Centers in the world. He is also a founding member of the International Association for the Study of Pain (IASP).

Fig. 1.5 Professor Philip Bromage (1920–2013). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

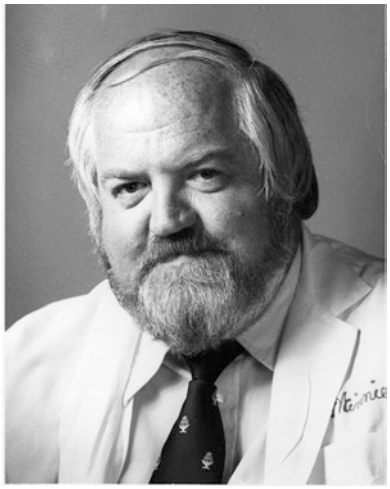

Fig. 1.6 Professor Alon Winnie (1932–2015). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

Although spinal anesthesia became obsolete in the UK in the 1950s, there was a great interest in epidural anesthesia and one of the outstanding pioneers of epidural anesthesia in the UK was *Philip Bromage* (Fig.1.5), who worked under the tutelage of *J Alfred Lee* in South-End-On-Sea in the UK. *Bromage* wrote the definitive textbook on epidural anesthesia and was a leading expert in epidural and regional anesthesia both in Europe and North America. His first text book was entitled *Spinal Epidural Anesthesia* [53]. *Bromage* moved to Montreal in 1956 and succeeded *Harold Griffith* as the Chair of Anesthesia at McGill University. He wrote the definitive textbook on *Epidural Anesthesia* in 1978 and it is today considered a classic[54]. He was very active in ASRA for many years, was a prolific writer, and a leading authority on the physiology and pharmacology of epidural anesthesia and the use of epidural and spinal opioids. He also deserves much credit for the promotion of epidural anesthesia for obstetric anesthesia in the 1960s. This new enthusiasm about epidural anesthesia for obstetrics attracted more interest in regional anesthesia also.

In the late 1960s, another great proponent of regional anesthesia emerged and that was *Alon Winnie* (Fig.1.6), who was an extraordinary teacher of regional anesthesia. Brachial plexus anesthesia was one of the great challenges to all enthusiasts of regional anesthesia. Even in the best of hands, most honest reporters could not achieve anything near 100 % success. *Winnie* described a new approach named the interscalene method and convinced most of us that the brachial plexus was contained in a single sheath, and if you could reliably place a needle in that sheath, you would have a high degree of success. His textbook entitled *Plexus Anesthesia: Perivascular Techniques of Brachial Plexus Block* is a classic and has the most spectacular illustrations [55]. *Alon Winnie* attracted a large number of new enthusiasts to regional anesthesia and he, *Don Bridenbaugh, Harold Carron, Jordan Katz*, and *Pritvi Raj* reformed the American Society of Regional Anesthesia (ASRA) and the first official meeting of that group occurred in 1975. In 2015, we celebrated 40 years of the newly formed ASRA (1975) during which tremendous advances were made in the discipline of regional anesthesia.

Pritvi Raj deserves special mention in the evolution of Regional Anesthesia. He popularized and promoted the idea of nerve stimulation to first identify the proximity of a needle to a nerve, and secondly, to actually identify which nerve was being stimulated based on a motor response. This was a major step forward because for the first time we had objective evidence indicating that a probing needle was in close proximity to a nerve based on the motor response. The first report about the use of nerve stimulation as an aid to regional anesthesia was published in 1973 [56]. The science of electro-location has evolved over the ensuing decades that it has been used and is still being used in some major anesthesia teaching centers in North America today. *Ban Tsui* has contributed enormously to our understanding of the science of electro-location today and was the first to use nerve stimulation to verify entry into the epidural space at any level. His textbook on ultrasound and nerve stimulation-guided regional anesthesia [57] is one of the most popular regional anesthesia textbooks published recently.

Nicholas Greene (Fig.1.7) was one of the great proponents of spinal anesthesia in the United States and his textbook entitled the *Physiology of Spinal Anesthesia* is one of the finest monographs ever published in the anesthesia literature and today remains a great resource in the understanding of all aspects of spinal anesthesia [58]. *Greene* was famous for his

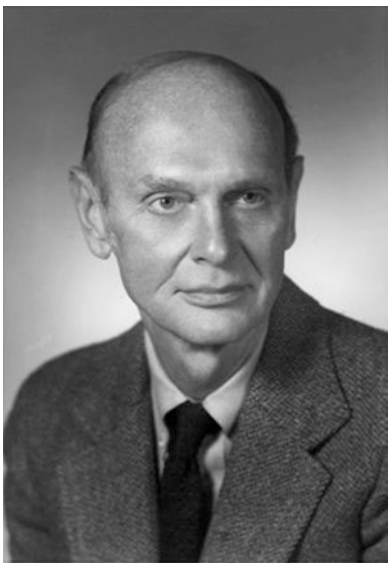

Fig. 1.7 Professor Nicholas Greene-(1922–2004). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

much quoted adage about spinal anesthesia when he said: "*position is everything in life, but especially in spinal anesthesia*". His lectures and publications on the topic of spinal anesthesia were outstanding and memorable experiences for those of us who were lucky enough to witness them.

The practice of regional anesthesia remained dormant in the UK for about 50 years after the *Wooley and Roe case*, but the French and Nordic countries were strong proponents of Regional Anesthesia. *Torsten Gordh* from Sweden was a leader in the use of regional anesthesia in his country and was among the first to test lidocaine clinically after *Löfgren's* discovery and demonstrated that lidocaine was a significant improvement on other available local anesthetics at the time [59].

Bruce Scott from Edinburgh deserves most of the credit for the revival of regional anesthesia in the UK and deservedly was named the founder and first President of the European Society of Regional Anesthesia in 1979 [60]. *Benjamin Covino* (Fig. 1.8), former Head of Research at ASTRA laboratories, was trained in regional anesthesia by *Bruce Scott*. *Covino* subsequently became one of the leading authorities on local anesthetics worldwide, and through his leadership, promoted research towards the introduction of newer, safer, long-acting local anesthetics. His textbook on local anesthetics is outstanding and concise and without a doubt is considered a classic today [61, 62].

Regional Anesthesia in the Modern Era

One of the greatest advances in regional anesthesia in recent years was the introduction of ultrasound technology to help identify peripheral nerves in regional anesthesia. This technology was first demonstrated in Europe and popularized in North America by *Vincent Chan* [63], and subsequently by *Ban Tsui* who recently published an outstanding publication entitled: *Atlas of Ultrasound and Nerve Stimulation-Guided Regional Anesthesia* [57]. The Regional Anesthesia Societies around the world(ASRA, ESRA, LASRA, AOSRA, AFSRA) deserve a great deal of credit also for hosting numerous workshops promoting the use of Ultrasound-guided regional anesthesia.

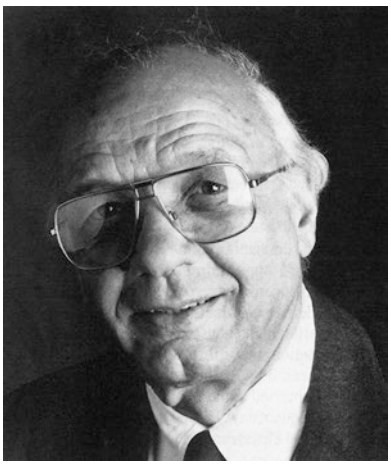

Fig. 1.8 Professor Benjamin Covino-(1931–1961). All images presented in this chapter are at the courtesy of the Wood Library-Museum of Anesthesiology, Schaumburg, Illinois, USA

When one reflects on the progress that has been made in Regional Anesthesia since *Koller's* discovery of Local Anesthetics in 1884 just over 130 years ago, we realize how far we have come. When *Halsted* performed that first brachial block in 1884, he had the advantage of direct vision of the brachial plexus. For about 100 years, we inserted our needles blindly towards peripheral nerves based on knowledge of anatomy alone and that indeed was a very "hit and miss affair". Today, we can actually see the nerve that we wish to block and see the needle as it advances towards its target and then see and observe the results of the subsequent injection. One has to wonder how we can improve on that in the future. Without a doubt, there will be some improvement.

There are many other names that deserve mention in this brief history of local and regional anesthesia, but this chapter should be a good introduction to this fascinating subject. For a more complete history of local and regional anesthesia, we refer you to the definitive text on that topic entitled The Wondrous Story of Anesthesia [64].

Summary

The history of Local and Regional anesthesia is one of the most interesting chapters in the annals of the history of medicine and deserves special mention any time the

history of anesthesia is discussed. *Koller's eureka* moment in 1884 changed the practice of Ophthalmology overnight and sparked a new era in local and regional anesthesia in ophthalmology, dentistry, surgery, and anesthesia. Spinal anesthesia has changed very little in over 100 years of use and remains one of the most reliable techniques in anesthesia today. We have made great strides in recent years to relieve the scourge of acute postoperative pain by applying regional anesthesia techniques prior to and during surgery. We still have a long way to go before we develop reliable methods of relieving chronic pain, but we already know that the judicious use of local anesthetics, pre-emptively in some procedures, reduces the incidence of chronic pain following surgery.

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Regional Anesthesia Safety

2

John W.R. McIntyre and Brendan T. Finucane

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).

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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

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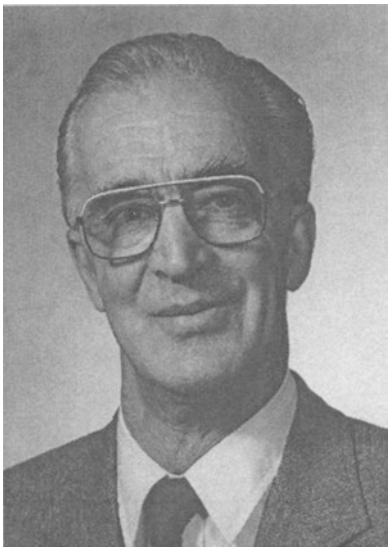

Fig. 2.1 Professor John W.R. McIntyre (1925–1998)

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 PCO_2 > 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 subsets of patients to whom a specific test applies have yet to be identified with certainty [17].

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

[a]Canadian Cardiovascular Society classification for angina

[b]Oxygen tension (PO_2) <60 mmHg; carbon dioxide tension (PCO_2) >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

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

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 H_2O 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 H_2O 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 Racz 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.