Essentials of Regional Anesthesia

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Editors

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Springer

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For my wife of over 20 years, Kim Kaye, MD,
for her patience, love, and wisdom
in raising our two wonderful children,
Aaron and Rachel.

Alan David Kaye

To my mentors, students, and trainees
for their inspiration, and to my parents,
Dennis and Tanya, and my wife,
Zina Matlyuk-Urman, MD.

Richard D. Urman

I dedicate this book to my parents,
my husband Muthu, my sons Gopal
and Vijay, and all my colleagues and friends
for their constant encouragement.

Nalini Vadivelu

Foreword

The editors of *Essentials of Regional Anesthesia* have asked me to write a Foreword for the book. Their request is most likely due to my involvement in regional anesthesia and its growth in the last 40 years. I have written and published on regional anesthesia extensively during this period, and this opportunity allows me to reflect on the progress regional anesthesia has made since its inception in 1884.

Regional anesthesia came to the European medical scene with a bang, when Carl Koller experimented with cocaine for its use in eye surgery and presented the data of his experiments to the Ophthalmological Congress in Heidelberg in September, 1884. His findings were accepted readily by the surgeons, since they were having significant morbidity and mortality with Ether and Chloroform. During the ensuing years up to 1930, regional anesthesia was commonly practiced safely, both in Europe and North America. New techniques such as spinal block, epidural block and peripheral nerve block were developed during this period. The limitation of performing regional anesthesia was that there was no training program for any physician who wished to learn this practice. It was done on an ad hoc basis by the assistants to the surgeons and the nurses.

As the specialty of anesthesia started in 1933, the anesthesiologists preferred general anesthesia to regional anesthesia because general anesthesia was easier to learn. The scientific growth in monitoring and safety in anesthesia practice was the main reason for popularization of anesthesia techniques.

All the same, Mayo Clinic in 1930s and 1940s still did their major surgeries with spinal anesthesia or peripheral nerve blocks with intravenous sedation by a barbiturate. Gaston Labat worked at this institution and became very accomplished in regional anesthesia. He wrote the first book on the subject – *Regional Anesthesia: Its Technique and Clinical Application* – which even today is a seminal book for practitioners of regional anesthesia. He was uniquely responsible for forming a society called the "American Society of Regional Anesthesia" (ASRA) for interested regional anesthesia physicians. The society's deliberations and activities are well documented in the Wood Library Museum. ASRA started to wither with future scientific growth in general anesthesia, such as the introduction of Halothane, intravenous induction of anesthesia with a barbiturate, and the discovery of muscle

relaxants like Curare. This allowed the anesthesiologist to easily monitor the level of anesthesia necessary for surgery and not have the patient kept awake. Surgeons loved this change in anesthesia practice.

During the 1970s, anesthesia training was overwhelmingly for general anesthesia practice, and the instances in which physicians used regional anesthesia techniques were few and far between. In spite of this state of affairs, giant figures like John Bonica, Philip Bromage, Alon Winnie, Daniel Moore, and Donald Bridenbaugh fought hard to advocate for the practice of regional anesthesia for surgical, obstetric, or cancer pain patients. They were the teachers of regional anesthesia and started the training programs in their departments. This culminated in the revival of the American Society of Regional Anesthesia. The residents going into the anesthesia specialty loved to learn about regional anesthesia, and with their support, regional anesthesia practice flourished. ASRA created practical workshops and educational programs, and with their influence the society was able to spread its wings in Europe, Asia, and Latin America.

During the period 1975–2010, remarkable scientific advances were made in the techniques, local anesthetics, and devices that assist in the success of regional anesthesia. In the 1980s, the peripheral nerve stimulator was an advance in locating nerves, and in the last few years, assistance by ultrasound techniques was introduced. Both of these techniques have been well received by the practitioners of regional anesthesia. Many books and documentaries are now available for training the physician in regional anesthesia.

Alan David Kaye has provided me with the reason why this book was conceived. He explains it is for the clinicians, students, residents, fellows, and attendees to learn and train in the new technology of regional anesthesia practice. He has mostly chosen authors who are Directors of the Regional Anesthesia Fellowship programs; this provides a sound basis on which the trainee can get the most up-to-date information on the state of practice of regional anesthesia.

The book is divided into six sections, including general considerations, basic science and clinical practice, equipment, and the modern practice of regional anesthesia. There are 31 chapters most of the chapters are formatted consistently, such as (a) anatomy of the block (b) indications for the block (c) types of techniques available (d) clinical pearls (e) complications. The chapters commonly end with multiple-choice questions.

I find *Essentials of Regional Anesthesia* very well organized and timely and that it fills the void created by rapidly changing regional anesthesia practices. Even though there are many books available on regional anesthesia, the incorporation of ultrasound for each block is a unique feature of this book and will be well received by the trainees. I recommend this book for all libraries of anesthesia departments, including for training of residents and medical students.

Lubbock, TX, USA P. Prithvi Raj

Preface

The practice of regional anesthesia has undergone tremendous evolution in the past few decades. Until recently, older blind techniques were taught, and successful regional anesthetics were typically limited to a few extraordinary clinicians in each department of anesthesia worldwide. Ultrasound, electrical stimulation, fluoroscopy, and continuous catheters have contributed to a revolution in the fields of regional anesthesia and pain management. Advances in technology have changed these fields significantly, resulting in the development of formal regional anesthesia and pain fellowships.

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

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

New Orleans, LA, USA — Alan David Kaye
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Part I
General Principles of Regional Anesthesia Practice

1

General Considerations for Regional Anesthesia Practice

Edward R. Mariano • Karley J. Mariano

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Introduction

Setting up a regional anesthesia service requires a reliable and consistent product as well as a sound business plan. Technological advances in nerve stimulation, ultrasound guidance, and perineural catheters have led to rapid growth in the number and types of peripheral nerve block procedures available to regional anesthesia

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practitioners. Starting a new regional anesthesia program potentially adds monetary value to a facility's perioperative services by improving the quality of postoperative analgesia and recovery from surgery, thereby reducing perioperative costs and offering a competitive advantage over other surgical facilities. From the patient's perspective, a regional anesthesia program provides nonmonetary value by preventing pain and reducing the risk of nausea and vomiting after surgery. Unfortunately for anesthesiologists interested in starting a new regional anesthesia program, there are no evidence-based guidelines to follow.

While the argument in favor of developing a regional anesthesia program in terms of nonmonetary value is convincing, determining monetary value is vital to initiating any new program. There are start-up costs to consider, and expected revenues are typically delayed. How does the individual anesthesiologist convince his or her own anesthesiology group or hospital administrators that a new service that provides peripheral nerve blocks and acute pain management is worth the investment? In this era of cost-effective health-care delivery, all medical institutions, academic and private, are under similar financial pressures. For academic centers, the primary educational mission must be achieved in the setting of financial stability.

Reasons for Starting a Regional Anesthesia Program

As the definition of "outpatient" surgery continues to broaden, patients previously hospitalized for the same surgeries several years ago are now scheduled for discharge on the day of surgery. Lengthy hospitalizations for some major surgeries are gradually progressing to overnight admission. For example, total joint replacement postoperative protocols have been refined to the point that it is currently feasible to practice same-day discharge or short-stay admission for appropriate cases [1–3].

The causes of prolonged recovery after scheduled ambulatory surgery have been studied and are multifactorial. In addition to type of surgery, these factors include postoperative nausea and vomiting, as well as pain following general anesthesia [4]. The proper application of regional anesthesia techniques in the ambulatory setting can minimize or avoid these common side effects and decrease the time required for patients to meet predetermined discharge criteria [5–7].

According to the results of a survey conducted by Dr. Macario and colleagues, nausea, vomiting, and pain are among the main side effects patients prefer to avoid after anesthesia [8]. It is increasingly important to consider patient preferences in the current health care system. Ensuring high patient satisfaction will likely lead a patient to return to a particular health care system for future surgical services and potentially result in new referrals. Patients should be considered consumers with the right to the highest quality service, and they have choices regarding their health care. Anesthesiology groups or hospitals who offer regional anesthesia services can employ marketing strategies to outcompete other anesthesiology groups and hospitals that do not offer similar services.

Regional anesthesia is not "one size fits all" anesthesia. The combination of specific peripheral nerve block techniques can produce anesthesia and postoperative analgesia that is nearly as selective as the surgical procedure itself. In a large case series, Klein and colleagues have demonstrated that peripheral nerve blocks in the ambulatory setting lead to reductions in perioperative intravenous opioid use and high patient satisfaction and can be used in conjunction with oral opioid analgesics [9]. For the nonorthopedic patient, other regional anesthesia techniques may offer similar advantages [10–12].

In order to extend the duration of site-specific pain relief beyond the immediate perioperative period, continuous peripheral nerve blocks (CPNB) and perineural local anesthetic infusions are currently used for a wide variety of surgeries in the ambulatory environment [13–20]. Randomized, placebo-controlled studies have conclusively demonstrated significant reductions in patient-reported pain after shoulder, foot, and distal upper extremity surgery as a result of CPNB [18–21]. By providing superior analgesia at home, CPNB effectively reduces the need to hospitalize patients for pain control. Broader application of these advanced regional anesthesia techniques has contributed to the growing interest in ambulatory total joint replacement [1, 2, 22, 23].

Bringing Your "Product" to Market

Developing a new regional anesthesia program is like inventing a new product. In addition to ensuring the consistency and reliability of the product, the prospective clientele and demand for the product should be considered. Applying this analogy, it is essential to identify potential customers and their needs. The *patient* and *patient's family* are the most important customers, and improvements in the overall quality of postoperative recovery resulting from regional anesthesia offer meaningful benefits to them. S*urgeons* are clearly important customers to any anesthesiology practice, hospital, or surgical center. Surgeons' concerns regarding failed blocks, complications, and case delays must be addressed [24], and surgeons may rally behind a regional anesthesia program that improves operating room efficiency [25]. When a regional anesthesia program gains surgeon support, the dividends multiply. Since surgeons establish rapport with patients several days or weeks before the surgery, their recommendation in favor of regional anesthesia is likely to lead to higher utilization of these services.

Despite the common belief that surgeons represent the major obstacle to developing a regional anesthesia service, it is often the *anesthesiology practice* itself that requires the most convincing. Since the initial investment in money, training, and personnel is incurred by the practice, there must be tangible benefits from implementing a new service. The ability to recoup this cost is dependent on the model of regional anesthesia practice implemented and the anticipated volume of nerve block procedures. For a busy orthopedic hospital, a new regional anesthesia service may generate enough new revenue to support the salary of one dedicated regional

anesthesia provider per clinical day. However, this type of "block room" model is not appropriate to all practices, and every regional anesthesia service should be developed with consultation from the individual practice manager and billing service to ensure proper financial planning.

The hidden customer when developing a new regional anesthesia service is *hospital administration*. Often, hospital administration receives the requests to purchase the initial capital equipment (e.g., nerve stimulators, regional block carts, and ultrasound machines). Although these overhead expenses may be large, they tend to be *fixed* and nonrecurring [26]. Administrators must be assured that this investment will result in quality improvement and possibly financial return, either from cost containment (e.g., reductions in hospital stays or nursing labor), increased revenue (e.g., attracting new patients or insurance contracts), or both. It is important to note that cost savings generated by regional anesthesia and perioperative pain management tend to benefit the hospital financially and not the individual anesthesiologist or anesthesiology group. In light of this fact, it is reasonable and expected to request the financial support of administration when developing a comprehensive perioperative pain management service employing regional anesthesia techniques.

Determine if Regional Anesthesia Will Save Money

Strategies to decrease the total cost of providing perioperative services must focus on *variable* costs despite the fact that the overwhelming majority of costs associated with any surgical procedure are *fixed* [26].

For ambulatory surgery, the use of peripheral nerve block techniques leads to increases in PACU bypass and shorter time to discharge compared to general anesthesia [5–7]. Cost savings can result from decreased recovery time associated with PACU bypass by reducing nursing time and labor [27]. In a high-volume orthopedic surgery center specializing in regional anesthesia for anterior cruciate ligament reconstruction (ACLR), the odds of bypassing PACU are nearly four times higher when the patient receives regional anesthesia compared to general anesthesia [27]. In this setting, PACU bypass has been shown to reduce the average per-patient cost by approximately $420 (USD) [27].

The development of step-down (Phase II) recovery units alters traditional PACU nurse staffing since these units are not considered critical care units, and patients classified as Phase II may be staffed in a ratio of four or five patients to one nurse. By increasing PACU bypass from 0 to 40% with regional anesthesia, PACU staffing may be reduced by one full-time nurse in a typical surgical center employing full-time staff paid hourly with frequent overtime [28]. In practical terms, this increase in PACU bypass can reduce overtime and therefore ease the financial burden on short-staffed hospitals and surgery centers.

Shortening hospital stays and minimizing unplanned hospital admissions for outpatients are potential sources of cost savings. Cost containment is essential when dealing with insurers that only reimburse a fixed amount per surgical procedure regardless of charges. For scheduled outpatients undergoing ACLR, unplanned

admission to hospital adds $385 (USD) per patient [27]. When employing femoral CPNB for scheduled surgical procedures requiring inpatient care such as total knee arthroplasty (TKA), patients may meet criteria for discharge home sooner compared to conventional analgesic techniques [1].

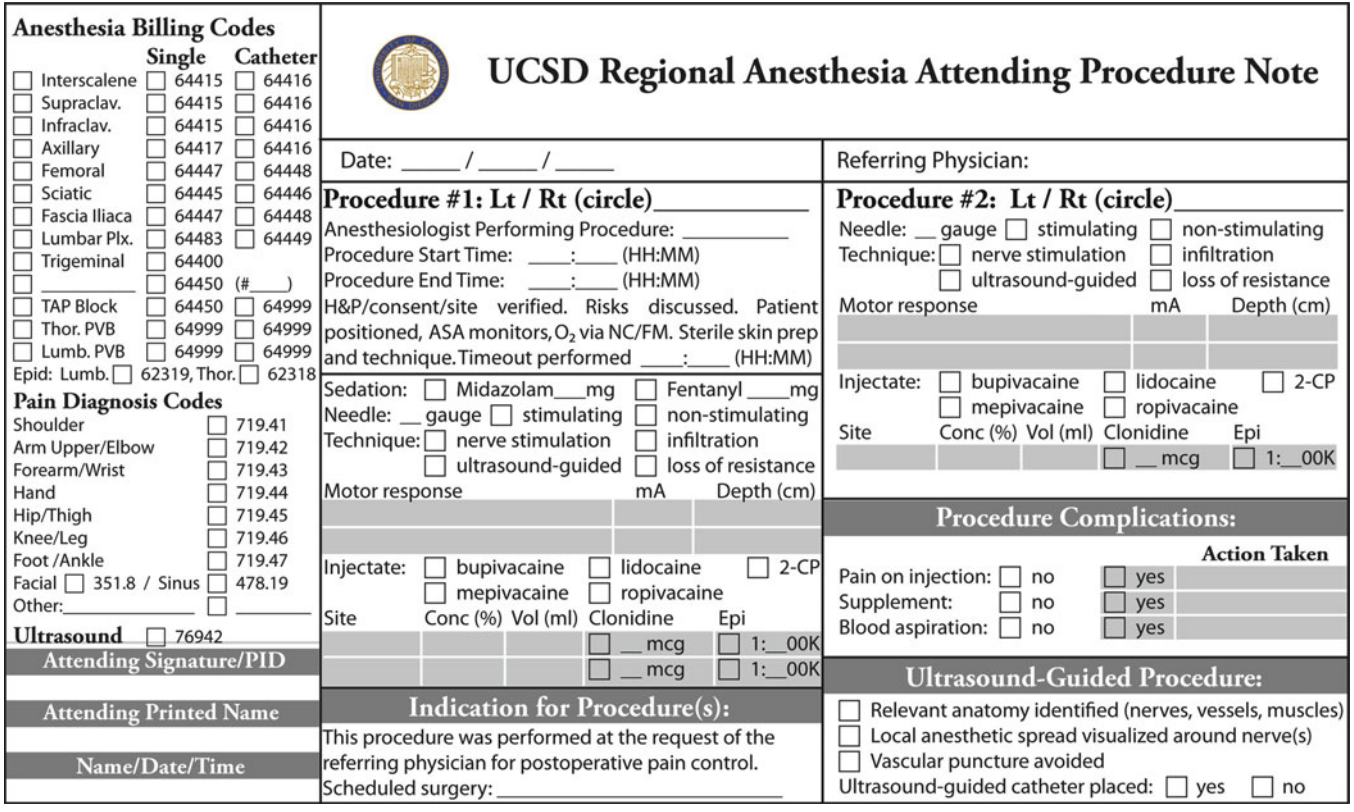
A major advantage of CPNB is that it may be provided on an outpatient basis unlike intravenous opioid patient-controlled analgesia or epidural analgesia. Shortening hospital stays for TKA by transitioning to outpatient femoral CPNB leads to a 34% decrease in overall hospitalization cost (US$ 2,682) due mostly to room and board savings [29]. According to data gathered from the National Hospital Discharge Survey, the rate of primary TKA in the United States nearly tripled between 1990 and 2002 to over 400,000 per year and is expected to increase [30]. Given this trend, the potential cost savings afforded by employing a regional anesthesia service for just TKA is impressive; extrapolating these data to all joint replacements and other major surgeries generates a staggering figure that should change surgical practice.

However, the decision to discharge a patient home early after TKA must be agreed upon by the entire health-care team, and proper patient selection is essential. For patients to be discharged home with a femoral CPNB after joint replacement, he or she must have a caretaker 24 h/day, an established outpatient physical therapy program, and close follow-up by a health care provider [1].

Bill Effectively for Professional Fees

The *charge* is the sum listed on a bill for services rendered. Taking a hands-on approach to billing professional fees will maximize charges and lead to revenue generation for the individual anesthesiologist and anesthesiology group. Billing strategies should be constantly reevaluated as regulations and procedural codes change, and actual billing practices will vary based on the individual institution, geographic location, and payor mix.

Use appropriate current procedural terminology (CPT) codes and modifiers. Anesthesia billing services should not be expected to interpret our handwritten procedure notes and deduce the appropriate codes for regional anesthesia because these procedures continue to evolve. For example, simply writing "infraclavicular block" on an anesthesia record may not be correctly coded as "64415 – brachial plexus block" unless the billing service happens to educate the staff in brachial plexus anatomy. To avoid confusion, consider doing your own coding on a standardized procedure note (Fig. 1.1). Common CPT codes for regional anesthesia are listed in Tables 1.1 and 1.2. Be aware that CPT codes are revised periodically, so make it a habit to review the updated CPT codes every year. When billing for nerve block procedures performed for postoperative pain management, include the distinct procedure modifier −59 to distinguish the block from the intraoperative anesthetic technique (e.g., 64416–59 for a brachial plexus catheter placed for postoperative pain management) [31]. This is especially important when the same provider performs the nerve block and the intraoperative anesthesia.



Anesthesia Billing Codes

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

Epid: Lumb. ☐ 62319, Thor. ☐ 62318

Pain Diagnosis Codes

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

Ultrasound ☐ 76942

Attending Signature/PID

Attending Printed Name

Name/Date/Time

UCSD Regional Anesthesia Attending Procedure Note

Date: _____ / _____ / _____

Referring Physician:

Procedure #1: Lt / Rt (circle)_____

Anesthesiologist Performing Procedure: _____

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

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

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

Sedation: ☐ Midazolam___mg ☐ Fentanyl ____mg

Needle: __ gauge ☐ stimulating ☐ non-stimulating

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

Motor response	mA	Depth (cm)

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

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

Indication for Procedure(s):

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

Scheduled surgery: _____

Procedure #2: Lt / Rt (circle)_____

Needle: __ gauge ☐ stimulating ☐ non-stimulating

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

Motor response	mA	Depth (cm)

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

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

Procedure Complications:

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

Ultrasound-Guided Procedure:

☐ Relevant anatomy identified (nerves, vessels, muscles)

☐ Local anesthetic spread visualized around nerve(s)

☐ Vascular puncture avoided

Ultrasound-guided catheter placed: ☐ yes ☐ no

Fig. 1.1 Example of a separate regional anesthesia procedure note

Table 1.1 Commonly-used CPT codes (2010) and suggested unit value charges (ASA Relative Value Guide) for single-injection nerve blocks

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

Table 1.2 Commonly-used CPT codes (2010) and suggested unit value charges (ASA Relative Value Guide) for continuous peripheral nerve blocks

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

Create a separate procedure note. Since postoperative pain procedures are not a part of intraoperative anesthetic care, it is helpful to develop a separate procedure note to document regional anesthesia techniques [32, 33]. The use of a different form physically separates the regional anesthesia procedure documentation from the documentation associated with the intraoperative anesthetic care and can even include common billing codes (Fig. 1.1). When designing new forms, involve your managers to ensure compliance with hospital policies and mandates from regulatory agencies. When using a separate form to document and bill for nerve blocks placed for postoperative pain management, the anesthesia record should not indicate "nerve block" as being part of the intraoperative anesthetic management. However, nerve blocks performed as the intraoperative anesthetic technique should be billed as such and not billed separately for postoperative pain management.

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

Billing for acute pain management and ultrasound guidance. Multimodal postoperative analgesia as part of integrated clinical care pathways has led to the evolution of some regional anesthesia services into acute pain management services [34]. Prior to January 2009, the CPT code for continuous nerve blocks included the period of routine follow-up (up to 10 days). Since then, the follow-up care has been unbundled, and daily evaluation and management (E&M) is currently a billable charge using 99231–99233 for established in-hospital consults. When utilizing real-time ultrasound guidance for nerve block procedures, use the CPT code 76942. In order

to charge appropriately for the use of ultrasound, the documentation should include an ultrasound image taken during the procedure and procedure-specific interpretation of findings. If long-term data storage is not available, consider printing an image and attaching it to the procedure note with a text annotation identifying relative anatomy, needle placement, injection of local anesthetic solution, and avoidance of complications. The modifier –26, when added to 76942, limits the ultrasound charge to professional fee only and should be used when the ultrasound equipment is owned by the hospital. Without the professional fee modifier, 76,942 includes a technical component charge for equipment storage and maintenance.

Foster a team approach to billing. Developing a good relationship with the people that send out claims and negotiate with insurance companies is essential. If regional anesthesia is new to an anesthesiology practice, meet with the billing service manager in person to clearly explain what regional anesthesia is, why it is performed, and what volume of procedures should be expected. If patient confidentiality is preserved, it may be feasible to have the billing manager observe regional anesthesia procedures and witness patients' postsurgical recovery. The more the billing service manager understands the indications and benefits of regional anesthesia, the better he or she is equipped to represent the anesthesiology group when appealing rejected claims.

Figure Out How to Make It Work

To make a regional anesthesia service successful, many pieces must fit together. All of the customers must be satisfied, including the patients, surgeons, and administrators. While patients may be satisfied by the superior pain control and improvement in the quality of postanesthesia recovery afforded by peripheral nerve blocks and CPNB, surgeons and administrators will also demand efficiency. In the busy outpatient surgery setting, the addition of a new regional anesthesia service does not have to detract from perioperative efficiency and may, in fact, contribute positively [25].

When regional anesthesia procedures are performed in a regional anesthesia induction area or "block room" while the preceding case is still in the operating room, anesthesia-controlled time is reduced compared to both general anesthesia and nerve blocks performed in the operating room [25, 35]. This parallel-processing model employing a block room may not work for every group practice or institution as it depends on the availability of resources and personnel [36]. For private anesthesia groups that function as a care team utilizing anesthesiologists and nurse anesthetists or academic anesthesiology departments with residents, a block room model is both feasible and recommended [33].

Regardless of the regional anesthesia service model employed, specially trained personnel in regional anesthesia techniques are necessary. When developing a service that utilizes specific procedural skills and advanced technology (e.g., surface ultrasound and CPNB), hiring and training staff with these skills is the most important first step. The use of ultrasound guidance for regional anesthesia, especially for CPNB, may offer advantages in terms of procedural efficiency [37–40] but requires

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

dedicated training [41, 42]. Not all anesthesiology residency training programs are providing adequate training in regional anesthesia techniques [43, 44]. The development of subspecialty regional anesthesia services at academic hospitals may lead to increased volume and complexity of procedures performed by residents [45, 46]. For private practice anesthesiologists, skills can be learned from partners who have received specialized training or continuing education courses that fulfill certain educational parameters [41].

With the increasing amount of technology employed in regional anesthesia procedures, centralizing supplies in a convenient location will contribute to effective time management. By storing all necessary supplies in one location, either a "block room" or regional anesthesia cart, performing peripheral nerve blocks and CPNB procedures can be as efficient as possible. Furthermore, getting all practitioners to conform to standardized supplies simplifies the ordering, storage, and preparation of equipment associated with each procedure.

A "block room" does not have to be a dedicated enclosed space and may be created out of existing clinical space in a preoperative holding area or postanesthesia care unit. At a minimum, this space should contain standard ASA monitoring, an oxygen source, and resuscitation equipment in addition to regional anesthesia supplies (Fig. 1.2). In addition, a portable regional anesthesia cart is advantageous because it can be transported from location to location when necessary.

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

Diligent Follow-up Is Key

Inserting the needle into the right place is the easiest part of regional anesthesia. The difficult part is developing an effective system of follow-up which is necessary for any program to succeed. A survey of outpatients with perineural catheters revealed that once-daily telephone calls from a health care provider is the optimal amount of contact, and 98% of patients are comfortable removing their own perineural catheters [47]. Only 4% would have liked a provider to remove his or her catheter, while 43% would have been satisfied with only written instructions and no person-to-person contact [47]. Patients with an ambulatory CPNB should be discharged with a caretaker (e.g., friend or family member) and should receive specific written instructions for their portable infusion device as well as provide a demonstration of the device function for the patient preoperatively. Written instructions should include expected CPNB issues (e.g., leakage and breakthrough pain) and contact information for a health care provider who will be available 24 h a day, 7 days a week. The patient should be followed on a daily basis until CPNB catheter removal, and each contact should be documented on a designated form [48].

For inpatients, a designated acute pain service provider or the anesthesiologist who performed the procedure should perform regular follow-up. Managing regional anesthesia patients, especially those with perineural catheters, is a team effort. A comprehensive clinical care team involving nurses, physical therapists, pharmacists, surgeons, and anesthesiologists can result in measurable patient benefits on a much broader scale [34].

A Guide to Team Building: Nursing Considerations in Regional Anesthesia

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

Collaboration fosters open communication with the staff [49] and prevents you, the physician, from feeling frustrated when managing nerve blocks and epidural catheters outside of the operating room. Nurses respond best to nurses; therefore, developing a model that allows for nurse–nurse interaction is best. Collaborate with nursing management and education to appoint at least one nurse for each unit, covering all shifts, to be a regional-specific educator who can function as a resource to his or her fellow nurses. You can educate these appointed nurses initially with the information you feel is important (e.g., functional anatomy and physiology of nerve blockade, expected effects, and untoward side effects), and they can continue this education into their unit. This will build the bridge you need for open communication, avoidance of unnecessary pages, and mutual respect among team members. If a local resource can answer the questions for which you most commonly receive calls (e.g., catheter leakage and pain not covered by the block), it will be easier for everyone involved and lead to faster intervention for the patient when necessary.

Approachability. Make yourself approachable and allow nurses to put a face to your name. When performing inpatient rounds, talk to the nurses and develop a professional relationship. Make sure the nurses know that you *want* them to call you about your patients and why they should. Remember that nurses are at the patient's bedside 24 h a day, 7 days a week, and are responsible for everything that happens to their patients. The two most common reasons that nurses call a physician are as follows (1) they are familiar with the situation and suspect that something is wrong, or (2) they are faced with a new situation beyond their comfort level and are requesting guidance. Nurses expect that the physician is there to help the patient and act as a guide when they are faced with unfamiliar clinical scenarios. Although there are times when contact may be inconvenient, it is important to respect the assessment of the nurse and discover the solution for the issue together. Later, you can address any underlying issues regarding education so the nurses may become more confident in managing regional anesthesia patients in the future.

Autonomy defines modern-day nursing. Recent nursing graduates are not part of the generation that simply followed orders. Finding a way to approach new ideas with nursing that allows for empowerment and self-sufficiency will avoid pushback and encourage more forward progress. Let the nursing staff determine their own educational goals. For example, find out from the nurses how much they want to know about regional anesthesia procedures, patient

(continued)

management, and technology. Remember that simply demanding something *never* works. This is the best way to generate resistance from nursing.

Lastly, *education* is what ties the above concepts together. Education is the key to improving nurse–physician relationships, especially when implementing regional anesthesia into nursing practice [49]. Modern-day nursing thrives on autonomy, and in order to prevent resistance from the nursing staff, one must educate a nurse to be efficient and productive when providing care for the patient with nerve blocks and continuous catheters. Highlight the benefits of regional anesthesia in reducing nursing interventions [50]. Undoubtedly, the educational process will likely start with you, the physician, then trickle down to nursing-specific education with dedicated nurse educators. Many hospitals throughout the nation already have pain resource nurses on individual units, and these nurses may be the ideal liaisons for you when disseminating regional anesthesia education.

Conclusion

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

Multiple-Choice Questions

Questions for General Considerations for Regional Anesthesia Practice:

1. Regarding the ways regional anesthesia provides "value" to an institution, all are true except:
   (a) Improving patient satisfaction with pain control and postoperative recovery
   (b) Creating a competitive edge versus other anesthesia groups and surgery centers
   (c) Reducing perioperative costs by decreasing the acuity of patients recovering from surgery and ensuring same-day discharge
   (d) Immediately generating new revenue

2. The cause of prolonged postanesthesia recovery is:
   (a) Postoperative nausea and vomiting
   (b) Acute postoperative pain
   (c) Multifactorial
   (d) Acute chronic pain

3. According to the study by Macario and colleagues, which side effect from anesthesia do patients most prefer to avoid?
   (a) Vomiting
   (b) Nausea
   (c) Pain
   (d) Gagging on the endotracheal tube

4. When considering regional anesthesia "customers," which of the following groups is most important?
   (a) Patients
   (b) Hospital administrators
   (c) Surgeons
   (d) Anesthesiology colleagues

5. Cost savings in the postoperative period attributable to regional anesthesia may result from all of the following except:
   (a) Reducing PACU length of stay
   (b) Avoiding unplanned hospitalization
   (c) Decreasing the patient to nurse ratio in PACU for bypass-eligible patients
   (d) Minimizing the need for pharmacologic interventions by PACU nurses

6. When designing the analgesic pathway for patients undergoing total knee arthroplasty, which of the following is false?
   (a) Intravenous patient-controlled opioid analgesia is typically administered on an inpatient basis.
   (b) Continuous nerve blocks may be managed effectively on an outpatient basis.
   (c) Epidural analgesia with local anesthetic solutions is most commonly maintained in the hospital setting.
   (d) None of the above.

7. Effectively billing for regional anesthesia procedures indicated for postoperative pain should involve which of the following?
   (a) Appropriate CPT coding.
   (b) Using a separate procedure note.
   (c) Including the distinct procedure modifier.
   (d) All of the above.

8. In a care team anesthesia delivery model, regional anesthesia may operate in the most efficient manner when:
   (a) Anesthesiologists are not familiar with regional anesthesia techniques
   (b) Equipment required for regional anesthesia is not centralized
   (c) Patients eligible for regional anesthesia are processed in "parallel"
   (d) Patients first learn about regional anesthesia on the day of surgery

9. When managing continuous nerve block catheters at home, which of the following is true?
   (a) Patients require a home nurse.
   (b) Patients must return to the hospital for catheter removal.
   (c) Patients should be called at home three times a day.
   (d) Patients should receive clear written and verbal instructions as well as contact information for a health care provider.

10. The clinical care team involved with managing patients with continuous regional anesthesia catheters should include:
   (a) Nursing
   (b) Pharmacists
   (c) Anesthesiologists
   (d) All of the above

11. Developing positive physician–nurse interactions when implementing a regional anesthesia program requires knowledge of all of the following except:
   (a) Collaboration
   (b) Assertiveness
   (c) Autonomy
   (d) Education

12. An nursing education program for regional anesthesia should include a discussion of:
   (a) Functional anatomy and physiology of nerve blockade
   (b) Expected effects of local anesthetics
   (c) Anticipated areas of pain not covered by blocks
   (d) All of the above

13. Nurses are most likely to call a physician about a regional anesthesia patient when:
   (a) They are familiar with the situation and suspect that something is wrong
   (b) They are bored
   (c) They are faced with a new situation beyond their comfort level and request guidance
   (d) (a) and (b)

14. Implementing a new regional anesthesia service requires all of the following except:
   (a) A master's degree in business administration (x)
   (b) Specialized training in regional anesthesia
   (c) Teamwork
   (d) Effective communication

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

Answers:

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

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2

Monitoring for Regional Anesthesia

Jeff Gadsden

Contents

Introduction

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

Regional anesthesia carries its own set of potential complications, principally nerve injury, systemic local anesthetic toxicity, and needle misadventure (e.g.,

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pneumothorax or arterial puncture). In general, the morbidity and mortality related to these adverse events are both less common and less severe than those associated with airway disasters, but catastrophic outcomes still occur [4]. There are a variety of monitors that are utilized during the performance of peripheral nerve block in order to avoid such complications, although their routine use varies greatly. In general, the adoption of consistent, objective monitoring to prevent injury during regional anesthesia has lagged behind monitoring efforts during general anesthesia. This chapter will focus on the basic setup that should be employed during each and every regional anesthetic, and the evidence base that supports the use of existing monitors.

Basic Setup

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

The use of standard monitors such as pulse oximetry, electrocardiography, and arterial blood pressure measurement are routinely recommended for any type of anesthetic (regional or general). Except in the obstetric population, neuraxial and peripheral nerve blocks are usually performed under some degree of sedation, both for patient comfort and to raise the threshold for local-anesthetic-induced seizures. As such, monitoring of oxygenation and ventilation are critical in order to detect hypoventilation, airway obstruction, and/or hypoxemia from excessive sedation. Pulse oximetry and frequent verbal contact with the patient are often sufficient to ensure adequate gas exchange; however, many centers employ capnography during peripheral nerve blockade in order to have a graphical representation of respiratory rate and guard against apnea.

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

Electrocardiography and blood pressure monitoring are essential in monitoring for early signs of cardiovascular systemic local-anesthetic toxicity. Cardiac toxicity from local anesthetics typically begins with myocardial depression followed by an increase in heart rate, blood pressure, and contractility that coincides with the onset of central nervous system excitement. As drug concentration increases, QRS intervals widen, and, particularly in the case of bupivacaine, ventricular arrhythmias

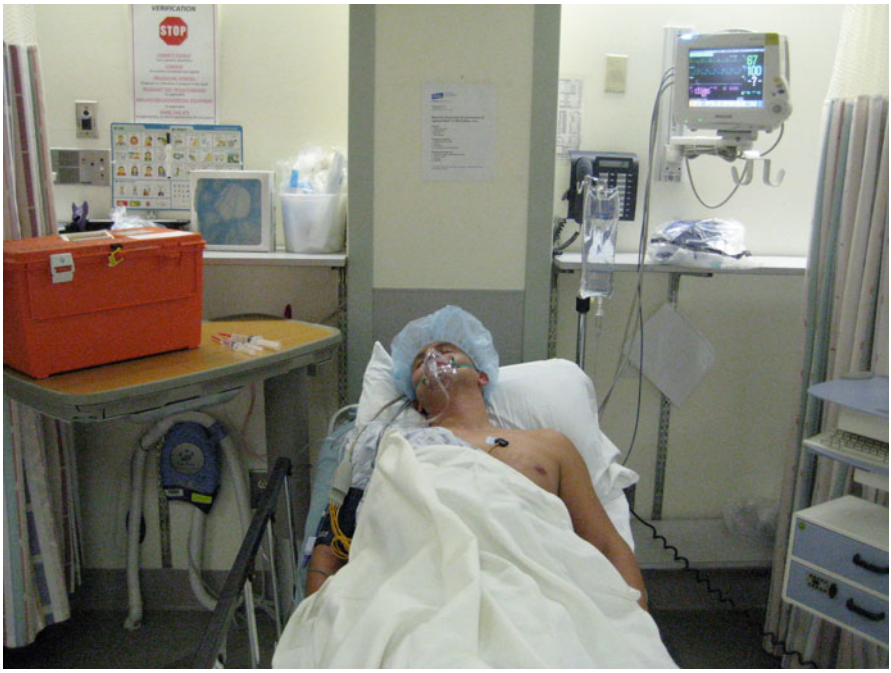

Fig. 2.1 Basic monitors and equipment required for regional anesthesia procedures. Note monitor displaying electrocardiography, pulse oximetry, and noninvasive blood pressure, as well as immediate availability of wall suction, bag-valve-mask, and emergency drugs

such as ventricular tachycardia or fibrillation occur. It is important to note that cardiac manifestations of systemic toxicity can precede the neurologic signs and symptoms, especially in the sedated patient, so vigilance for early changes in heart rate, blood pressure, and EKG morphology is vital [4]. An example of a patient with the basic monitors applied for regional anesthesia is illustrated in Fig. 2.1.

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

1. Self-inflating bag-valve-mask (i.e., Ambu® bag)
2. Suction
3. An oxygen source with facemask
4. Endotracheal tube(s), oral and/or nasal airways
5. Laryngoscope (tested and functioning)
6. Emergency drugs:

   - A "sleep" dose of induction agent (e.g., 20 ml of propofol)
   - Succinylcholine

- Atropine
- A vasopressor such as ephedrine or phenylephrine
- A 500-ml bag of intralipid for treatment of local-anesthetic systemic toxicity (this does not necessarily have to be bedside, but should be immediately available should the need arise to use it)

Specific Monitors

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

Aspiration, Fractionation, and Speed of Injection

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

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

A slow injection speed may protect against nerve injury as well. An association with injection pressure and fascicular rupture has been shown in large animals [11], and recent evidence has demonstrated that speed of injection is directly related to the risk of generating high pressure during femoral nerve blockade [12]. In this study, an injection speed of 10 ml/min carried a small incidence (6%) of pressure >1,000 mmHg, a threshold that has been associated with injury in rabbits and dogs. In contrast, speeds of 20 and 30 ml/min were associated with dangerous pressures 35 and 44% of the time, respectively.

Intravenous Markers

Early detection of rising levels of local anesthetic in the plasma is critical to avoiding systemic toxicity. Toxic symptoms can occur acutely (in the first seconds to minutes after injection), as is the case with an accidental intravascular needle/catheter insertion, or subacutely (minutes to hours later), which is due to gradual vascular absorption. Several studies have investigated the utility of premonitory symptoms as a means of early detection. Moore and colleagues found that sensitivity of a 1 mg/kg IV dose of lidocaine in unpremedicated volunteers was 100% in detecting early neurologic signs (e.g., tinnitus, perioral numbness, metallic taste) [13]. However, when given small doses of midazolam (1.5 mg) and fentanyl (75 mcg) prior to injection, the sensitivity dropped to 60%. Similar results have been found with other local anesthetics including bupivacaine, levobupivacaine, and 2-chloroprocaine [14, 15]. McCartney et al. found that a dose of 60 mg of ropivacaine did act as a reliable IV marker, even when volunteers were premedicated with 0.03 mg/kg of midazolam [16]. However, caution should be exercised when applying these results to clinical practice, as the use of such doses in elderly or frail patients may precipitate toxicity.

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

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

Concern has been raised about the use of epinephrine and vasoconstriction of small nutritive blood vessels in the nerve, which could potentially cause ischemia [22].

Epinephrine is known to produce a dose-dependent prolongation of neural blockade, the mechanism of which is partly thought to be due to nerve ischemia [23]. Combination with inherently vasodilating local anesthetics does not reverse this effect; Myers and Heckman demonstrated that the combination of lidocaine 2% and epinephrine 5 mcg/ml reduced rat sciatic endoneural blood flow by 78% [24]. However, more dilute concentrations of epinephrine may be beneficial: Partridge showed that epinephrine 2.5 mcg/ml applied to the rat sciatic nerve transiently increased neural blood flow by 20% for several minutes, before returning to baseline, suggesting that at reduced concentrations, the beta-adrenergic effects may predominate [25]. An ideal test dose might therefore be 6 ml of a local anesthetic solution containing 2.5 mcg/ml (15 mcg) of epinephrine.

Neurostimulation

Electrical nerve stimulation has been used as a means of nerve localization for three decades. While there is a lack of evidence showing improved block success or patient safety compared with the paresthesia technique, it remains a popular method [26]. However, its sensitivity as a means of neurolocalization has recently been questioned. Several experiments have found that needle-nerve contact and, in some cases, intraneural needle tip placement still require current intensities in excess of 0.5–1.0 mA in order to obtain a motor response [27–29]. This contradicts the common belief that "the closer the needle to the nerve, the stronger the twitch." On the other hand, extremely low current thresholds may be associated with intraneural needle tip positioning. Tsai et al. demonstrated in a pig model that, while extraneural current thresholds varied with distance to the nerve, motor responses obtained using currents less than 0.2 mA were always associated with intraneural needle tip positioning [30]. In another pig study, Voelckel et al. performed percutaneous sciatic nerve blocks using two different current intensities and examined the relationship to histologic changes [31]. Those nerves for which currents of 0.3–0.5 mA were accepted demonstrated no signs of injury; however, when the blocks were performed using currents less than 0.2 mA, 50% of nerves showed signs of inflammatory changes. More recently, Bigeleisen et al. provided clinical evidence that a motor response of 0.2 mA or less indicated intraneural needle placement in an ultrasound-guided supraclavicular block model [32]. While neurostimulation may not be a sensitive method of placing needles next to nerves (i.e., high current intensities may still be required if within the nerve), it appears to carry a high specificity for ruling out intraneural placement, using 0.2 mA as a cutoff for safe practice.

Ultrasonography

The use of ultrasound-guided nerve blockade has many apparent benefits, the most obvious being the ability to guide one's needle under real time toward the target.

Its use may also confer several safety advantages, chief among them being the ability to decrease the amount of local anesthetic used to effect a successful block. Among the first to demonstrate this were Casati et al., who showed in an up-and-down design that ultrasound guidance was able to reduce the minimum effective anesthetic volume required for a femoral block by 42% [33]. In an era where systemic toxicity from even "clinical doses" of local anesthetics is regularly published [34], this is not an insignificant finding. In addition, not all of the volume reduction benefits are of a systemic nature. Riazi et al. compared the effect of performing ultrasound-guided interscalene blocks using 5 ml vs. 20 ml of 0.5% ropivacaine on phrenic nerve palsy, and found the incidence of diaphragmatic paralysis to be 45 and 100% respectively, while pain scores and analgesic consumption in the first 24 h were identical [35]. Needless to say, this type of extreme volume reduction would be difficult without ultrasound guidance.

Ultrasound is a useful tool in demonstrating intraneural injection, as even volumes less than 1 ml can result in obvious nerve swelling on the ultrasound image [36, 37]. However, intraneural injection, despite common teaching, does not necessarily result in nerve injury, despite sometimes causing histologic changes suggestive of inflammation [36]. In fact, in two clinical studies of axillary and popliteal sciatic block in which over 80% of injections were within the epineurium, no patient had subsequent nerve injury [38, 39]. The surprising lack of injury with intraneural injection may be a result of needle deflection away from and between "tough" perineurium-surrounded fascicles within the nerve [40], as intrafascicular penetration is thought to be a mechanistic factor in nerve injury. As it turns out, the current resolution of ultrasound machines is not fine enough to detect intrafascicular vs. extrafascicular needle tip placement, and for that reason, may not be a sensitive monitor for preventing nerve injury. Ultrasound has not been shown to be superior to nerve stimulation alone with respect to patient safety [41–43].

Another potentially beneficial aspect of ultrasonography that seems intuitive is the avoidance of vascular, pleural, or nerve puncture. Just as the rate of carotid puncture during internal jugular cannulation appears to be reduced significantly when using ultrasound guidance [44], one large prospective audit of more than 7,000 peripheral nerve blocks showed a significant reduction in the incidence of inadvertent vascular puncture [43] There are, however, numerous reports of vascular and neural impalement despite the use of ultrasound [45, 46], as well as reports of pneumothoraces following brachial plexus block [47, 48]. These data suggest that, for the time being, either the technology or the manner in which it is being used is not foolproof. On the other hand, ultrasound has been advocated as a routine procedure to rule out pneumothorax prior to discharging patients home after supraclavicular blockade for ambulatory surgery [49].

Injection Pressure Monitoring

Hadzic et al. showed that high pressures (>25 psi) at the commencement of intraneural injections in dogs were associated with neurologic deficit and destruction of

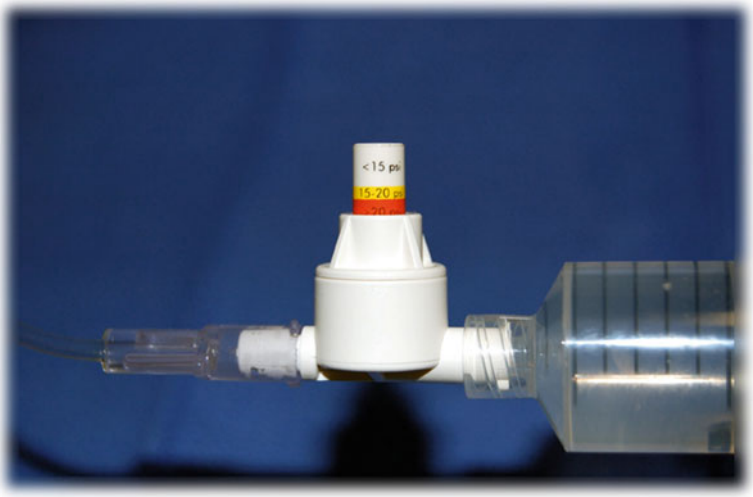

Fig. 2.2 A commercial in-line pressure transducer with graduated markings on the side of a piston, indicating pressure in psi (BSmart™, Concert Medical, Norwell, MA)

neural architecture [11]. This is likely due to the rupture of the fascicle after the expansion of these low-compliance spaces. In one blinded volunteer study of injection pressure, anesthesiologists were shown to be poor at judging the degree of force being exerted on the syringe plunger [50]. Therefore, the objective monitoring of injection pressure may be a useful modality. This can be achieved either through commercially available in-line devices (Fig. 2.2) or by the use of a "compressed air injection technique" [51]. By filling a 30-ml syringe with liquid and leaving 10 ml of air, a compression of the air component to no more than half its original volume (i.e., 5 ml) will ensure that injection pressure is kept at 20 psi or less. Since this technology is relatively new, evidence has not accumulated showing a safety advantage. However, since it is inexpensive and easy to use, and since the animal data are compelling, the reasons not to use it are few.

Pressure monitoring has other applications as well – a recent study showed that bilateral spread during lumbar plexus block occurred in 60% of patients exposed to injection pressures >20 psi compared with 0% in those with pressures <15 psi [52]. One patient had an epidural block with a T4 sensory level, a situation that could prove dangerous in a vulnerable patient.

Monitors of Consciousness and Cerebral Perfusion

When regional anesthesia is used alone (i.e., not in combination with general anesthesia), mild to moderate sedation is usually desirable for reasons of patient comfort,

anxiolysis, and amnesia. While monitoring the level of sedation can be achieved clinically, some have employed processed electroencephalography, such as the bispectral index (BIS), to titrate sedation to a specific depth. This may carry advantages for certain populations at risk for adverse outcomes as a result of oversedation. For example, one study of elderly patients undergoing repair of fractured hips under spinal anesthesia randomized subjects to receive propofol sedation titrated to a BIS of approximately 50 vs. a BIS of 80 or greater [53]. The incidence of postoperative delirium was significantly reduced in the group with the higher BIS target, 19% vs. 40%, suggesting that the use of clear BIS targets to minimize oversedation may aid in avoiding this all-too-common outcome.

Several observations have to be made with respect to monitors of brain activity and their use in regional anesthesia. First, neuraxial anesthesia itself is known to contribute to sedation and decrease the requirements for further anesthetic drugs [54]. This is effected by a combination of decreased afferent neural input to the reticular activating system, and a direct effect of local anesthetics in the cerebrospinal fluid [55, 56]. The spinal level of the block may be important: Nishikawa and colleagues demonstrated that a spinal resulting in a sensory block to T4 in unpremedicated patients resulted in a significant decrease from baseline BIS scores, whereas an L3 level did not [57]. Secondly, while propofol and midazolam both appear to predictably reduce BIS scores, nitrous oxide may not, even though the clinical effect is apparent. For instance, in patients who received epidural anesthesia for lower extremity surgery, increasing the concentration of nitrous oxide in oxygen (33, 50, and 67%) that was administered resulted in steadily decreasing sedation scores as judged by a blinded observer [58]. However, BIS scores remained unchanged throughout, suggesting that BIS may not be an effective monitor of level of consciousness in the presence of nitrous oxide.

Another group of patients that may benefit significantly from central nervous system monitoring during regional anesthesia are those that are positioned in the beach chair position. Used primarily for procedures on or about the shoulder, this position has been associated with several cases of perioperative ischemic cerebral events [59, 60]. These may be caused by underperfusion to the brain when brachial artery pressure, which is generally used to guide hemodynamic management during these cases, overestimates the cerebral perfusion pressure, as the pressure differential may be as much as 30 mmHg. A contributing factor is the frequent request by the surgeon to lower arterial blood pressure to reduce bleeding into the joint and improve visibility during arthroscopic shoulder surgery. Cerebral oximetry using near-infrared spectroscopy is a method by which cerebral tissue oxygenation can be continuously assessed noninvasively. A study comparing cerebral oxygenation using the beach chair vs. the lateral decubitus position for shoulder surgery showed that 80% of patients in the beach chair position had what were defined as "critical desaturation events," compared with 0% in the lateral decubitus position [61]. This group also experienced a sevenfold increase in the incidence of nausea and vomiting in the recovery room, although all other recovery variables were similar. Interestingly, the BIS scores between groups did not differ at any time, suggesting that oximetry is a more sensitive predictor of nausea and vomiting. The question that arises is

whether there might be more subtle neurologic changes in patients at risk (e.g., the elderly or hypertensive patients) that are occurring regularly without obvious signs in the immediate postoperative period. It is not known whether cerebral oximetry can be used to guide treatment in this population; patients were treated promptly in this study when the events were diagnosed and there was no control arm. Also, these patients all received general anesthesia; further research efforts should focus on conducting similar studies using regional anesthesia alone.

Clinical Pearls

- Minimize potential complications by employing the maximum number of monitors possible for every regional block, as they work in different, complementary ways.
- Slow, fractional injection at a rate of 10 ml/min or less reduces the peak plasma levels of local anesthetic and may reduce the possibility of nerve injury.
- Epinephrine 10–15 mcg, in a concentration of 2.5 mcg/ml, is the ideal test dose for most patients. The most sensitive and specific sign is a rise in systolic blood pressure of 20 mmHg or more, even in sedated patients.
- Use a nerve stimulator to help locate the nerve, and continue to decrease the current gradually once the needle is appropriately positioned. If a motor response is still obtained at 0.2 mA of current or less, the likelihood that the needle tip is intraneural is high. The needle should be withdrawn and repositioned.
- Ultrasonography significantly reduces the amount of local anesthetic required for a successful block, and can potentially improve safety by showing where injectate is spreading (and *not* spreading), although this is largely operator-dependent.
- High-pressure injections are associated with permanent nerve injury. Injection pressure monitoring is a useful modality to potentially protect against the injection of local anesthetic into a low-compliance space such as the fascicle and prevent devastating neurologic outcomes.
- The use of BIS to titrate sedation may help avoid postoperative delirium in elderly patients. Cerebral desaturation as detected by cerebral oximetry appears to predict postoperative nausea and vomiting.

Multiple-Choice Questions

1. Essential emergency drugs that must be immediately available during the performance of any regional anesthetic technique include:
   (a) Rocuronium
   (b) Glycopyrrolate
   (c) Dextrose 5%
   (d) Lipid emulsion

2. During performance of peripheral nerve blocks, hypoxia serves to:
   (a) Increase the free fraction of mepivacaine
   (b) Potentiate the cardiodepressive effects of lidocaine
   (c) Increase the metabolism of 2-chloroprocaine
   (d) Increase the therapeutic index of bupivacaine

3. Essential equipment for the performance of a femoral nerve block includes:
   (a) Electrocardiography
   (b) Nerve stimulation
   (c) Ultrasound
   (d) A laryngeal mask airway

4. Which of the following best describes the practice of aspirating on the syringe prior to injection of local anesthetics during peripheral nerve blockade?
   (a) Aspiration reliably detects intravascular needle placement.
   (b) Aspiration reliably prevents local anesthetic systemic toxicity.
   (c) Aspiration is not necessary during paravertebral blockade.
   (d) Despite little evidence of a safety advantage, the practice is still encouraged.

5. Which of the following is true regarding the use of local anesthetics as intravenous markers?
   (a) Premedication does not alter the perception of premonitory symptoms after a dose of IV lidocaine (1 mg/kg).
   (b) Volunteers premedicated with midazolam could reliably detect a 60-mg dose of intravenous ropivacaine.
   (c) All patients should receive an initial 100-mg dose of lidocaine through the block needle to test for intravenous absorption.
   (d) 2-Chloroprocaine has been shown to be a reliable IV marker.

6. Epinephrine is often added to local anesthetic solutions for all of the following reasons EXCEPT:
   (a) To truncate the peak plasma level of local anesthetic
   (b) To hasten the onset of the analgesic block
   (c) To prolong the duration of nerve blockade
   (d) To serve as an intravenous marker in case of intravascular absorption

7. In which of the following patients would the sensitivity of epinephrine as an intravenous marker be limited?
   (a) A 46-year-old woman with mitral valve prolapse for knee arthroscopy.
   (b) A 58-year-old man with asthma who has a comminuted fracture of the ulna.
   (c) A 38-year-old man with high cholesterol who presents for rotator cuff repair.
   (d) A healthy 29-year-old woman in active labor.

8. Which of the following is true regarding the use of nerve stimulation during peripheral nerve blockade?
   (a) There is a predictable relationship between needle tip-nerve distance and the current required to cause a motor response.
   (b) Nerve stimulation has little value in the era of ultrasonography.
   (c) The generation of a motor response at extremely low currents (i.e., <0.2 mA) has been associated with intraneural needle tip placement.
   (d) If a motor response is NOT generated at currents of 0.5 mA or greater, intraneural needle tip placement is not possible.

9. Which of the following regarding the use of ultrasonography during peripheral nerve blockade is true?
   (a) The minimal amount of local anesthetic volume that can be used for an effective block compared to nerve stimulator techniques is not significantly different.
   (b) Local anesthetic volumes can be reduced, albeit at the expense of duration and quality of the block.
   (c) Ultrasonography typically requires more local anesthetic than nerve stimulator techniques due to the use of multiple injections.
   (d) Inadvertent blockade of neighboring nerves such as the phrenic nerve can be avoided by reducing the volume through the use of ultrasound guidance during brachial plexus blockade.

10. Which of the following is NOT critical to observe during performance of all ultrasound-guided peripheral nerve blocks?
    (a) Expansion of the tissue at the time of local anesthetic injection at the target site.
    (b) Vasculature that is adjacent to the target site.
    (c) The tip of the nerve block needle.
    (d) The target nerve or plexus.

11. Which of the following regarding ultrasound guidance and intraneural injection is true?
    (a) Intraneural injection can be detected by ultrasonography.
    (b) Intrafascicular injection can be detected by ultrasonography.
    (c) Ultrasound guidance can predict clinical nerve injury reliably by detection of intraneural injection.
    (d) Ultrasound guidance prevents the entry of the needle tip into the epineurium.

12. Which of the following regarding intraneural injection is true?
    (a) Intraneural injection of even small amounts of local anesthetic leads to clinically detectable nerve injury.
    (b) Intraneural injection appears to cause histologic changes suggestive of inflammation, even if clinically detectable injury is not present.
    (c) Nerve fascicles are surprisingly easy to penetrate with a blunt-tipped needle.
    (d) Nerve injury following intraneural injection is almost invariably permanent.

13. Ultrasonography during peripheral nerve blocks has been shown to:
    (a) Reduce the incidence of nerve injury compared to nerve stimulation techniques
    (b) Reduce the likelihood of pneumothoraces compared to nerve stimulation techniques
    (c) Reduce the need for monitoring during nerve block performance
    (d) Reduce the incidence of accidental vascular puncture

14. The use of injection pressure monitoring:
    (a) May reduce the incidence of nerve injury
    (b) Is complicated and requires special equipment
    (c) Is unnecessary if the practitioner is experienced and can gauge "hand feel" for himself/herself
    (d) Should be used only for high-risk blocks such as interscalene brachial plexus blocks

15. Maintaining low (<15 psi) injection pressures has been associated with:
    (a) Improved outcomes in large-scale studies
    (b) Equivocal outcomes in animal studies
    (c) Reduced incidence of bilateral and high epidural spread during performance of lumbar plexus blocks
    (d) A reduction in the volume of local anesthetic required for lumbar plexus blocks

Answers:

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

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3

Regional Anesthesia in the Community Practice Setting

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Contents

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Introduction

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

By virtue of their training, scope of practice, and historical innovation, anesthesiologists are uniquely qualified and, indeed, expected to assume a leadership role in acute perioperative pain management. As the primary practitioners of regional techniques, anesthesiologists play a critical role in the delivery of state-of-the-art multimodal opioid-sparing techniques designed to maximize pain relief while minimizing side effects [4]. Within the specialty, this has led to a renaissance in the field of regional anesthesia. Yet, effectively responding to the many challenges presented by the expansion of anesthesia practice into the realm of pain management requires a conscious effort by practitioners, especially by those in community practice whose

formal training may not have adequately prepared them for this eventuality. Therefore, it is not surprising that despite evidence-based data to support their benefits, regional techniques appear to remain underutilized, especially in the community practice setting.

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

It is easy to appreciate that what actually constitutes "community practice" is an incredibly diverse reality. Practitioners may be solo or have any number of department members (which may include subspecialty-trained physicians, CRNA's, or nurse practitioners), with practice settings varying from hospitals to ambulatory surgery centers to office-based care. It would be impossible to address the unique issues of orthopedic pain management in each community practice circumstance. The intent of this chapter is to identify the common hurdles that exist in a community practice environment and present broad concepts and directions to overcome these hurdles to achieve a common goal: creating a culture of consistent and efficient acute pain management that extends beyond the operating room.

Identifying the Challenges

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

Institutional Challenges

Physicians in community practice are often wedged in a culture of conformity. A general anesthetic utilizing postoperative opiate therapy is reliable and requires less technical skill and minimal organizational adaptations. Institutions lacking leadership in acute pain medicine are poorly positioned to fully utilize the many

recent advances in this rapidly growing field. Furthermore, once a culture of medical practice is established, a transformation in this culture is difficult to accomplish. Implementing regional anesthesia-based acute pain protocols under these entrenched circumstances requires considerable effort and vision. If the institutional hierarchy fails to appreciate the many benefits of advanced pain management, this lack of support may make it difficult to obtain necessary staff, supplies, and equipment. This is especially true for expensive technology like ultrasound equipment.

Community practices also frequently lack accommodating facilities commonly encountered in academic environments, such as designated areas for the performance of regional blocks (block rooms) (Fig. 3.1). The optimal timing and location for regional anesthesia under these circumstances tends to be dictated by individual circumstances (nurse and anesthesia personnel staffing, room turnover times, patient flow within a facility, available equipment, etc.). In effort to overcome these infrastructural hurdles, physicians often must either perform regional techniques in less-than desirable locations or abort the prospect altogether.

Time Pressures

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

Surgeon Resistance

Any discussion of anesthesia choices in private practice must address the influence of surgeons, who are often considered either proponents or opponents of regional anesthesia. Just as surgical support for regional techniques can greatly facilitate their acceptance, resistance from surgical colleagues can be a significant hurdle. A 2002 survey of orthopedic surgeons found that the two principal reasons for not favoring regional anesthesia were OR delays and unpredictable success. The principal reasons for favoring regional anesthesia were less postoperative pain, decreased

Fig. 3.1 Photo of a block area. At our hospital, epidural and peripheral nerve blockade are frequently performed in the PACU. The block area is a dedicated patient location that includes full monitoring, the regional block ultrasound unit, stimulating catheters, and a fully stocked regional anesthesia cart. It is immediately adjacent to the operating room and allows rapid turnover with minimal distraction

nausea and vomiting and safety. If we can convince our surgical colleagues regarding the benefits of regional anesthesia, they may instead act as advocates in our mission to educate the public. These issues may be resolved with physician education, improvements in training, and organization of the regional anesthesia facility [6].

Deficiencies in Training

Few anesthesiologists in community practice have advanced clinical training in regional anesthesia or pain management. While the training of anesthesia residents is generally adequate for spinal and epidural techniques, exposure to peripheral nerve blocks continues to be deficient. Kopacz and Neal reported in 2002 that as many as 40% of anesthesiology residents in the United States may not be receiving the minimal required level of exposure to peripheral nerve blocks [7]. Given the large number of different regional techniques, it is apparent that few anesthesiologists will have sufficient experience during residency training with peripheral nerve blocks to feel confident as they enter community practice. Reflecting this narrowed comfort zone, German anesthesiologists who practice in small hospitals have been shown to rely heavily on basic regional techniques, in contrast to consultants at teaching institutions [8]. The explosive growth of perineural techniques has clearly outpaced the experience of many already in practice. Given these observations, it is not surprising that anesthesiologists in community practice have been noted to perform significantly fewer peripheral nerve blocks than those who practice in teaching institutions ($p = 0.05$) [9]. Finally, those who are trained in advanced pain therapies may be frustrated to find that many anesthesia colleagues in community practice may be uncomfortable or disinterested in providing cross-coverage for unfamiliar pain management techniques.

Personnel Issues

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

Patient Resistance

The public's fears and distorted perceptions of pain from needle passage, paralysis, and a wakeful state can also hinder the assimilation of regional techniques into daily practice. The public does not understand the risks and benefits of regional anesthesia. More problematic is the concept that anesthesiologists do not understand the general public's fears of regional anesthesia. This is evidenced by the finding that

anesthesiologists perceptions differed from the actual fears of interviewed patients. The anesthesiology community has not been successful in keeping the public well-informed regarding regional anesthesia. Future anesthesia-related educational programs should address the concerns of the public about anesthesia matters, particularly regional anesthesia [10].

Overcoming the Challenges

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

Create a Physical Environment Conducive to Regional Anesthesia

A block room can greatly facilitate the preoperative performance of regional techniques, and in one study resulted in an operating room time savings of over 20 min per case [11]. However, the economic feasibility of a dedicated block room is questionable, and a designated preoperative "block area" can be a reasonable alternative. Pressures to maintain OR flow and limit delays make the postanesthesia care unit (PACU) an excellent substitute for a block room if one does not exist. Consider isolating a single patient bay in a corner of the PACU to perform regional techniques preoperatively (Fig. 3.1). While many regional procedures can be performed with minimal assistance, each should be preceded by a "time-out." PACU nurses are exceptionally trained in monitoring and can serve as excellent assistants if dedicated personnel are unavailable. Furthermore, patients can be expeditiously transferred because of the PACU's close proximity to the OR. Regardless of locality, several regional anesthesia texts should be readily available wherever blocks are performed.

The efficiency of regional anesthesia is enhanced by keeping supplies together in a standardized "block cart," which has the additional advantages of being mobile and able to hold resuscitative equipment (Fig. 3.2). A sufficient supply of Intralipid should be stocked wherever local anesthetics are to be used. Lipid emulsion bolus followed by infusion represents a novel resuscitation method that has demonstrated efficacy in the treatment of local anesthetic toxicity [12]. Contents of a regional anesthesia cart should now include a 500-ml vial of 20% intralipid, 60-ml syringe, and a macrodrip infusion kit. A lipid rescue algorithm (Appendix 1) should be posted on this block cart (see Fig. 3.2) to aid the practitioner and to provide immediate visual cues in the event of an unintended intravascular injection. An educational website has been created (http://www.lipidrescue.org) and serves as an excellent instructional resource for physicians to learn about lipid emulsion therapy.

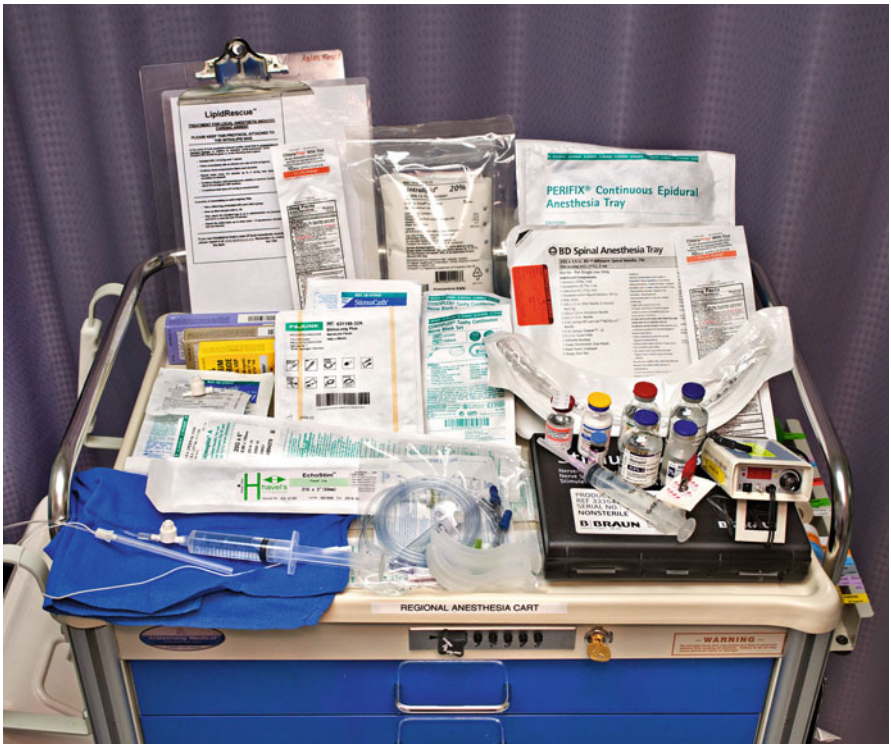

Fig. 3.2 Photo of the contents of a typical regional anesthesia cart. The cart includes catheters, stimulators, local anesthetic solutions, gowns, gloves, and prep solutions. Of importance, the cart is also stocked with resuscitative medications and Intralipid solutions for emergency treatment of local anesthetic-induced cardiotoxicity

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

Establish a Multidisciplinary Pain Management Team

Implementation of evidenced-based guidelines for pain management alone is inadequate to achieve advances in patient outcomes. A consistent and comprehensive approach to the management of acute pain involves the patient and every member of

their care team. Success of the service is predicated on collaboration among physicians, nurses, ancillary staff, and hospital administration. The cornerstone of this interdisciplinary effort is communication. Shortcomings in the effective management of acute pain can usually be overcome through efforts to improve communication, education, and coordination of care. Integrated collaborations between the medical, nursing, and ancillary staff are needed to achieve the full benefits of an improved analgesic regimen [2, 13]. It is useful to briefly consider how anesthesiologists may effectively interact with each component of this interdisciplinary effort.

Senior Leadership

Coordination of a successful pain management program requires strong institutional support. Plans for major initiatives should be disseminated to the senior leadership at both medical and hospital board levels delineating the benefits of the service. Institutional support for pain management efforts is essential if additional staffing will be required and also necessary to obtain necessary supplies and equipment. It is of no small import in this regard that ultrasound guidance for regional anesthesia can often be viewed as an institutional revenue generator [14]. Any efforts that will look to maximize patient safety, improve patient care, enhance operating room efficiency, and decrease length of stay will certainly be embraced and highlight the efforts of the department toward developing new standards of practice and "service excellence."

Anesthesia Department

Establish a Core Group Within Your Ranks

Surprisingly, the greatest resistance to the successful integration of regional techniques in community practice may come from within the department of anesthesiology itself. A lack of interest or inexperience and consequent medicolegal concerns may lead some colleagues to oppose implementing techniques that are perceived to require greater technical skill. The collaborative effort for the success of the initiative needs to start within the department of anesthesiology and an important core group of partners is needed to support the formation of the regional anesthesia service. Establish a minimum level of proficiency within the department by creating opportunities to mentor partners with less experience with both didactic and practical instruction. Establishing a single primary location for block placement (such as the PACU) facilitates the education of other anesthesia team members, where members can gather together, learn each other's techniques, and share information. Creating a core group of partners promotes an infrastructure of technical support making these analgesic techniques available to all patients as well as allowing

the burden of work to be shared. There is encouraging evidence that motivated practitioners can successfully utilize even the most complex regional techniques in the community practice setting, as was recently shown for ambulatory continuous interscalene blocks [15].

Appoint a Leader/Physician Champion

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

Physician Organization

Surgeons

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

Surgeons can be the greatest advocates for the routine use of regional anesthesia and are the drivers of patient acceptance [16]. As noted above, patients will tend to be more receptive to regional techniques if they are introduced to the possibility by their surgeons. Identifying which surgeons are supportive of the initiative before implementing the service to the entire department will ensure acceptance of the techniques and increase success. The survey mentioned above regarding resistance to regional anesthesia among orthopedic surgeons provides some valuable insight

into the rationale involved [6]. Although surgeons reported predictable concerns with regional anesthesia regarding operating room delays and unpredictable success, when data were reanalyzed, investigators found that these perceptions of delays or success rate were surprisingly not predictive of their preferences for regional anesthesia [17]. Instead, they found that a surgeon's preference for peripheral nerve blocks for his or her own surgery strongly predicted their preference for his or her patients. Importantly, a significant number of surgeons would want peripheral nerve blocks for some surgical procedures but not others, probably based on perceptions of how painful a surgery may be. These data serve to emphasize the value of discussing procedure-specific anesthesia choices with surgeons, focusing on what they would want for their anesthetic if they were the patient and why.

Nonsurgeon Physicians

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

An often overlooked area of pain management in hospitals is the emergency room. There is ample evidence that pain continues to be inadequately managed in the ER setting and could be improved upon [18]. The early performance of a fascia iliaca block for patients with hip fractures, for example, is a safe and simple intervention that can control pain and minimize opioid use in a frail, elderly population [19]. Anesthesiologist attendance at an emergency room departmental meeting can be one means of educating emergency physicians and help expand the service beyond the operating room.

Nursing Staff

Optimal analgesia requires careful therapeutic fine-tuning to maximize the benefits and minimize the risks and side effects of therapy, necessitating an organized service beyond the operating room [20]. Nursing staff support is an implicit prerequisite to the viability of an anesthesia-based acute pain management service. While physician leadership is required to champion the goals of the service in a physician-directed nurse-delivered model, the nursing staff is empowered to assess, manage, and ultimately treat the patient. Regardless of the diversity that exists in the variety of anesthesia staffing models, this arrangement creates an infrastructure of support resulting in close patient surveillance preventing the occurrence of any analgesic gaps. Establishing this link allows advanced regional techniques to be safely utilized in any institutional setting.

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

It is important that the degree of insight by nurses into acute pain management modalities extends deeper than the physician orders. While written orders should clearly delineate nursing responsibilities, nurses should also understand the rationale for pain management choices and appreciate the nuances of each. Direct involvement by the department of anesthesiology in nursing education is one means of effectively preparing hospital staff for full participation in the management of acute pain. The didactic instruction should include a comprehensive description of the normal side effects and complications from regional anesthesia techniques, care for/troubleshoot catheters and infusion pumps, and the delineation of discharge instructions to patients (Appendix 7). A system for follow-up with outpatients must also be established (with a phone call from nursing generally being sufficient). A formal process of continuing education where the nursing staff is credited with continuing education units (CEU's) maintains the integrity of the service and ensures optimal nursing assessment and management skills.

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

Ancillary Staff

The department of physical therapy plays a crucial role in the transition from the acute postoperative period to eventual functional outcome. Better management of pain facilitates more aggressive physiotherapy regimens, which may improve outcomes and decrease hospital length of stays [21]. Physical therapists need to be educated regarding the potential for motor blockade with lower extremity regional techniques and how this may impact ambulation. The vigilance of a well-informed physical therapy department coupled with the use of ambulatory-assist devices (e.g., knee immobilizers) can minimize the risk of iatrogenic injuries, such as falls during ambulation.

While pharmacists are often viewed as being somewhat removed from direct patient care, their involvement is essential to a smoothly operating acute pain management system. Standardizing the volume and concentration of analgesic infusion

solutions can help reduce the risk of medication error. Stocking supplies of premixed standardized infusion agents in a convenient location (e.g., the PACU) is more efficient than an on-demand system for pharmacy and also helps to ensure the timely availability of solutions. Using appropriate sterile procedures, pharmacists may also be able to fractionate certain agents into clinically useful amounts (e.g., 1 mg. preservative-free clonidine into 100 μg single-dose volumes).

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

Multimodal anesthetic techniques can improve discharge predictability and accelerate discharge eligibility. If social services are not involved early in the patients' perioperative course, these advantages can go essentially unrecognized. Preoperative patient education sessions describing the perioperative course may help to overcome common social delays in discharge (nursing home placement, patient transportation, lack of home readiness by family members, patient concerns resulting in requests for extended hospital stay), facilitating early discharge planning. Engaging the social service department in a comprehensive patient care plan at the beginning of hospital admission allows for the timely discharge of patients [22].

The Public

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

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

Finally, it is essential that anesthesiologists rapidly and clearly communicate acute pain management plans during the preoperative visit. The general public has

many misconceptions regarding anesthesia and pain management that are often best discussed in a one-on-one manner [10].

Formulate and Implement an Acute Pain Management Plan

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

Multimodal Analgesia

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

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

These evidence-based recommendations serve to reinforce several points. First, overreliance on opioid analgesia in the postoperative period is to be avoided. Second, simple nonopioid measures like acetaminophen and NSAIDS/COXIBS should not be overlooked [23]. Third, whether employed for surgical anesthesia or not, regional blocks are an essential component in the optimal postoperative management of pain. Finally, any analgesic plan, including established clinical pathways, must be tailored to each individual patient.

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

local anesthetic (as a one-time procedure or continuously administered [24], which may allow for patient-controlled boluses) and intra-articular agents (e.g., intra-articular morphine) [25]. Another consideration is the preoperative administration of analgesics (usually orally) whose duration would be anticipated to extend into the postoperative period, such as extended-release opiates (e.g., extended-release oxycodone) or anti-inflammatories (e.g., celecoxib). Other less well-established adjunctive modalities such as ketamine, gabapentin, and clonidine are being actively investigated and may assume greater importance in the future. As recently demonstrated for pregabalin, there may also be significant promise for these and other agents in the prevention of chronic postoperative pain [26].

Clinical Pathways

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

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

Usually, multimodal pathways for orthopedic surgeries prominently feature regional anesthesia. Optimal management of pain, largely accomplished through the addition of regional techniques, can help minimize complications while facilitating aggressive physiotherapy, which can result in improved functional outcomes and decreased hospital length of stays [27]. Rather than assume a lead role in the design of standardized protocols, physicians in community practice are encouraged to investigate the current practice at academic centers. Many leaders in the development orthopedic care maps have published their experiences (e.g., the Mayo Clinic) [28]. Analyzing and adapting such protocols from academic centers, which have been used successfully on a large scale, is likely to prove safe and effective in the community hospital environment [29] (Appendix 8). Recent updated evidence-based recommendations are also available for several common orthopedic procedures on the PROSPECT website (http://www.postoppain.org) and published in recent review articles [13, 30].

Judicious Use of Regional Blocks

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

Situations where basic blocks result in obvious patient benefits (the "low-hanging fruit") should be considered to be the foundation for regional acceptance within an institution. It is easy, for example, to generate a consensus of support for femoral block after total knee arthroplasty. Momentum generated through a single routine can then be used to further promote regional techniques for other indications.

In the community practice environment, management should be streamlined whenever possible. While combinations of peripheral blocks may be necessary to provide complete pain relief following certain surgeries, single block approaches are generally more practical. The lack of functional improvement with the addition of sciatic block following total knee arthroplasty, for example, makes femoral block alone an attractive choice in community practice [31]. Likewise, although catheter techniques can provide superior long-term pain relief, single-shot blocks are generally preferred unless severe pain is expected to extend for several days.

Conceptually, the approach to regional blocks in a community practice setting is often starkly pragmatic when compared to an academic environment. Practitioners should thoughtfully consider specific regional blocks in light of the following three "ideal" attributes: a single injection site, short needle (50 mm or less), and supine positioning. Blocks that have high success rates with single injections are clearly preferable to blocks that rely on delivery of local anesthetic to multiple locations. Supra- and infraclavicular blocks are thereby able to be performed more expeditiously than multiple-stimulation axillary block. Efficacy can also be improved through knowledge of optimal target responses for successful block with single injection sites (i.e., posterior cord stimulation with infraclavicular block [32] and tibial nerve stimulation for popliteal block [33]). Blocks that can be done using short needles are able to be more quickly performed and tend to be associated with fewer needle passes, less patient discomfort, and possibly lower complication rates. The ability to maintain the supine position generally allows for patient care to proceed along a usual flow, despite sometimes necessitating the use of longer needles (e.g., lateral popliteal block [33] or anterior sciatic block [34]).

Keys to Success with Regional Anesthesia in Community Practice

Given the realities presented above, it is apparent that the successful performance of regional techniques is critical to an anesthesia-based acute pain service. Yet the modern community practice environment can often make these techniques seem

impractical, if not impossible, to put into practice. Successfully performing and expanding the use of regional anesthesia under such circumstances requires a pragmatic approach, which can be summarized as follows:

Operate Within the "Comfort Zone"

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

In general, and especially with new approaches to acute pain, it is ideal that these modalities require minimal attention outside of the operating room and normal working hours. The availability of concomitant intravenous patient controlled analgesia (IV PCA), in particular, is a major consolation when initiating more advanced nonopioid pain management modalities (i.e., single-injection or continuous nerve blocks).The patient-titrated nature of IV PCA has the advantages of minimizing nursing care while being capable of independently providing adequate postoperative analgesia. The extent of IV PCA use (or more accurately, the extent to which it was not used) also to some degree reflects the efficacy of nonopioid techniques being simultaneously utilized. Once a "comfort zone" for the concomitant use of postoperative opioids is established, a transition to extended-release oral opiates as seen in published analgesic care maps can obviate the need for parenteral use and its consequent side effects (Appendix 8).

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

Learn in a Logical Progression

Given the large number of different regional techniques, it is apparent that few anesthesiologists will have sufficient experience during residency with peripheral nerve blocks to feel broadly confident as they enter community practice. Considered in its proper perspective, regional anesthesia training must be viewed as an introduction to a lifelong commitment to further learning. Just as an anesthesiologist must acquire experience when a new inhalational agent is marketed, they should approach overcoming deficiencies in regional anesthesia training with the same intellectual curiosity. Effectively removing surgical pain from the equation along with the unpleasant side effects of opioids is where regional anesthesia has evolved. The explosive growth that orthopedic anesthesia has witnessed should not mandate specialty training

for regional techniques to be implemented in community practice. Just as we have not created a subspecialty for the placement of arterial lines or administration of total intravenous anesthesia (TIVA), we do not need specialty training for perioperative blocks. Every anesthesia provider should be able to perform these techniques if they are willing to choose so.

It is easy to appreciate that some regional procedures (e.g., spinal anesthesia) are more readily mastered than others. All anesthesiologists possess some regional skills and should therefore strive to expand their regional anesthesia practice in a stepwise manner. They should take care not to violate institutional or their own personal comfort zones, but rather seek to reasonably expand these zones. With this concept in mind, regional procedures have been classified into basic, intermediate, and advanced categories [35]. An awareness of this stratification can help practitioners develop competence and confidence with regional techniques in a logical progression. Proficiency with manual skills is developed through practice, and skills learned with one block will generally build confidence with all regional procedures. Anesthesiologists should liberally utilize regional techniques in appropriate clinical situations, not just when it is crucial that they work.

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

Incorporate Ultrasound into Your Practice

Anatomical diversity in patients coupled with a challenging body habitus has led some practitioners with marginal regional experience to navigate through an attempt at regional blockade with trepidation in a "poke and hope" approach. Unpredictable block success, patient discomfort, and technical delays will negatively reinforce future attempts at perineural techniques.

Advances in the science of regional anesthesia have seen the technique of nerve location progress from utilizing paresthesias to nerve stimulation to ultrasound guidance. Ultrasound guidance of regional anesthesia is currently an area of intense interest and has created the potential of simplifying peripheral nerve blockade. The prediction of Dr. Alon Winnie many years ago was: "Sooner or later someone

will make a sufficiently close examination of the anatomy involved, so that exact techniques will be developed" [36]. While it is not yet viewed as the gold standard, the literature suggests that this technology may be capable of improving the efficiency and efficacy of regional blocks [37]. Compared to nerve stimulation techniques, ultrasound-guided blocks are performed more quickly, using less local anesthetic, with fewer needle passes as well as a reduced incidence of vascular puncture [38]. The increase in current thresholds caused by the injection of conducting solutions hampers the ability to instantly reinject local anesthetic after a failed block. By confirming local anesthetic spread around the target nerve or perivascular anatomy, ultrasound can overcome this phenomenon of electrical interference and offers practitioners a powerful tool for block rescue and the potential for increased block success. Furthermore, ultrasound guidance provides the practitioner with a renewed opportunity to perform interventions on patients difficult to stimulate with the peripheral nerve stimulator (i.e., diabetic patients).

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

Keep Regional Blocks in Proper Perspective

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

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

Dealing with Block Failures

Plans for regional anesthesia often suffer from a failure to consider reasonable alternatives in a timely manner. Visualizing success with regional anesthesia is in many ways similar to management of the airway. If plan A (laryngoscopy) does not meet with success, then plan B (LMA) and even C (fiberoptic bronchoscopy, etc.) should be pursued. Likewise, if certain regional techniques are not proceeding smoothly, they can be appropriately followed by "plan B" blocks. Difficulties with infraclavicular or femoral blocks can be expeditiously addressed by performing axillary and fascia iliaca blocks, respectively. Wound infiltration with local anesthetic by the surgeon is usually a reasonable plan C option.

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

In the event of a true block failure that becomes evident in the postanesthesia care unit (PACU), reattempting the same block is usually not considered prudent. However, incomplete pain relief in some anatomic regions may be adequately covered by similar techniques. Failure of interscalene and femoral blocks, for example, can be safely and effectively followed by suprascapular [39] and fascia iliaca blocks [19], respectively. More selective distal blocks are often ideal following the failure of a more proximal block (e.g., ulnar, median, or radial blocks at the elbow after failed brachial plexus blocks).

Be Cost-Conscious

Anesthesiologists must be knowledgeable regarding the hospital cost of supplies and consistently choose cost-efficient means of providing pain control. While few supplies are essential, practitioners are faced with a number of important choices whenever regional techniques are contemplated. For example, either nonstimulating or comparatively expensive stimulating catheters may be utilized for continuous femoral nerve block after total knee arthroplasty, yet the evidence would indicate that the two appear to be equally effective [40]. Opponents of ultrasound will claim that the initial investment in machinery is prohibitively expensive. However, the use of ultrasound can replace the need for stimulating needles and stimulating catheters leading practitioners to use simple thin-walled needles at a fraction of the cost. This can result in cost savings, which can efficiently recover the initial investment. Costs may also be reduced through the use of a prep sponge and sterile towel pack instead of a commercially manufactured block tray, choosing bupivacaine over ropivacaine as circumstances permit, and utilizing reusable pumps as opposed to disposable infusion devices.

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

Avoid Delays (Even the Perception of Delays)

The production pressures mentioned above require that practitioners ensure that regional techniques not be perceived as a cause of delays. On the contrary, a systematic multimodal approach to acute pain management, which includes regional analgesia, should be viewed as the ideal strategy to improve efficiency through "fast-tracking" (bypass of phase 1 recovery) and speeding discharge readiness [41].

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

In an effort to avoid delays, anesthesiologists in community practice may elect to perform regional anesthesia in anesthetized or heavily sedated patients. Practice has been noted to vary widely in this regard. While performing regional anesthesia on insensate patients may ensure guaranteed cooperation and maximize flexibility in the timing of these procedures, it may also expose the patient and practitioner to unnecessary risk. Anesthesiologists should be aware of the recent practice advisory on this subject [42]. In this advisory, the authors acknowledge that the decision to perform regional anesthesia under these circumstances is "controversial, complicated, and must be made in the absence of traditional forms of evidence-based medicine." Notably, interscalene block is the only regional technique explicitly contraindicated in anesthetized or heavily sedated patients.

Documentation

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

such that any pain management issues within the institution are naturally directed to the department.

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

Most institutions require that patients provide written informed consent for anesthesia care, which is separate from surgical care. Practitioners may wish to obtain additional consent for pain management procedures, which can be considered apart from surgical anesthesia care. Procedures performed for postoperative pain are considered separate from the anesthesia care provided for surgery. As such, these procedures should be documented on a form separate from the anesthesia record. The key elements to a standardized peripheral nerve block procedure note form have been described and analyzed [43]. Dedicated procedure notes have been developed for both peripheral nerve blockade [43] and neuraxial techniques [44], which can be readily combined into a single form (Appendix 9).

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

Following Through on an Acute Pain Management Course

Proper follow-through is a duty of ownership and critical to the long-term success of any patient care program. Efforts by anesthesiologists which clearly extend to the conclusion of care are necessary to maximize benefits and minimize risks associated with acute pain management, and will ensure the highest levels of satisfaction from both patients and surgeons.

Follow-Through for Outpatients

Adequate analgesia is an obvious prerequisite for ambulatory surgery, where inadequate pain control has been shown to be a common reason for prolonged

postoperative stays and unanticipated admissions. Furthermore, it is essential to anticipate pain-related issues that may become evident following discharge in ambulatory patients as inadequate pain management has been shown to be a leading and preventable cause for readmissions [45]. In ambulatory surgery, regional techniques including single-injection and continuous perineural catheters provide improved analgesia, less opioid-related side effects, and the potential for earlier discharge [46].

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

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

Follow-Up for Inpatients

Hospitalized patients, by virtue of their higher acuity of illness and injury, may stand to benefit the most from the effective management of pain through minimizing complications and possibly preventing chronic pain. Following up on inpatients is a primary function of an acute pain service. It has been repeatedly acknowledged that there is no consensus regarding the optimal structure or function of an acute pain service [47]. In the diverse reality of community practice, an acute pain service may take many forms but must at least consist of involved physician (e.g., anesthesia) and nursing personnel.

Nurses are at the core of inpatient follow-up and are empowered to assume the leading role in assessing and treating postoperative pain. Regular assessment of pain, commonly every 4 h utilizing a 0–10 pain rating scale, is noted on pain assessment flow sheets which serve to track the "5th vital sign" (i.e., pain) over time and

record responses to treatment (Appendix 6), although such documentation is now often computerized. Multimodal treatment of pain based on scores >4 is usually included in standing pain management orders. This approach has been used successfully in many practice settings and shown to result in improved pain control and patient satisfaction, but can also be associated with an increased incidence of opioid-induced oversedation [48]. This oversedation is usually preceded by a gradual decrease in the patient's level of consciousness, which underscores the critical importance of frequent clinical assessment by nursing.

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

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

Although the acute pain service in many community practice settings is not a formal, distinct entity, prompt 24-h coverage is essential. Instructions for appropriate contact of anesthesia personnel should be included in all pain management orders. An acute pain service beeper can help maintain continuity of communication within a system. If in-house anesthesia coverage is available, then an on-call physician manages overnight pain-related issues. If in-house overnight coverage is not available, then a mechanism that provides for off-hour patient evaluation needs to be devised. One solution is to specifically train selected night shift nursing

personnel to evaluate and troubleshoot common issues concerning acute pain management (for continuous infusions, for example, this would include occlusion alarms, catheter disconnections, and evaluation of skin entry sites).

Management of Complications

The ideal management of complications begins with the tacit acknowledgement that complications are inevitable. Having realistic preoperative discussions with patients regarding potential complications, obtaining meaningful written informed consent, and keeping accurate records comprise the foundations of appropriately dealing with adverse events. The traditional model of anesthesia care involved the placement of regional techniques with the "occasional" participation in postoperative pain management. The surgeons' office was frequently used as the "middle man" to manage block-related complications. Unhappy patients coupled with a lack of knowledge regarding block-related sequelae created an adversarial relationship between the two working disciplines. Adopting a "patient-centric" approach where the anesthesiologist collaborates closely with the surgical staff on any postoperative block-related issues creates a cooperative approach to the management of complications. Furthermore, taking ownership of our interventions will certainly result in a more vigilant approach improving procedural efficacy.

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

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

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

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

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

Quality Improvement

A process for quality improvement (QI), also commonly referred to as quality management (QM), is a fundamental requirement of all health-care organizations. Although QI for the department of anesthesiology largely concerns the operative period, in the case of an anesthesiology-based acute pain service, it must extend through the entire duration of management. Quality improvement efforts allow for clinically significant data concerning pain management to be collected and monitored with the goal of improving performance and enhancing patient safety. The American Society of Anesthesiologists website is an excellent resource regarding quality improvement (http://www.asahq.org). The Quality Management Template found at the ASA website, developed by ASA committees and provided without charge, serves as an indispensable guide to implementing a quality improvement program in any practice setting [49].

The ready availability of occurrence reporting forms is a key element in the consistent self-reporting of adverse events. For cases in the operating room, reporting forms are often attached to the anesthesia record. Similarly, anesthesia-specific incident reporting forms should be immediately at hand as nurses and anesthesiologists are engaged in following through on an acute pain management plan. While occurrence forms are usually completed manually, if large amounts of data will require analysis, it is advisable that these forms be capable of being scanned. A number of computer-ready process improvement tracking tools are commercially available, with several examples provided in the ASA's Quality Management Template. Although self-reporting of adverse outcomes has inherent weaknesses, it has been shown to be more reliable than medical chart review or incident reports and tends to be successful in environments where it is perceived that participation may result in improved patient care [50].

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

Conclusion

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

Clinical Pearls

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

Ultrasound Pearls

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

Multiple Choice Questions

1. All of the following are examples of interventions used in a standard multimodal analgesic pathway except:
   (a) Acetaminophen
   (b) NSAID's
   (c) Continuous spinal–epidural with local anesthetic
   (d) Spinal anesthetic with continuous femoral block
   (e) General anesthetic with rapid sequence induction

2. The two principal reasons for not favoring regional anesthesia when surveying orthopedic surgeons are:
   (a) Operating room delay and excessive motor block
   (b) Operating room delay and high injection pressures
   (c) Unpredictable success and medicolegal complications
   (d) Unpredictable success and operating room delay
   (e) Medicolegal complications and operating room delay

3. Success of a regional anesthesia service is predicated on:
   (a) Collaboration with ancillary staff
   (b) Implementation of evidenced-based guidelines for pain management
   (c) Minimizing wrong-sided blocks with the performance of a "time-out"
   (d) Avoiding operating room delays
   (e) All of the above

4. Contents of a standardized regional anesthesia block cart should include all of the following except:
   (a) Resuscitative medications
   (b) Endotracheal tubes
   (c) Intralipid
   (d) EMLA cream
   (e) Ester local anesthetics

5. Contents necessary for a successful resuscitation with lipid rescue include all of the following except:
   (a) 20% Intralipid
   (b) Macrodrip infusion kit
   (c) 60 cc syringe
   (d) Propofol

6. Regional techniques for ambulatory surgery result in all of the following except:
   (a) Improved analgesia
   (b) Less opioid-related side effects
   (c) Potential to bypass the postanesthesia care unit
   (d) Increase use of antiemetics
   (e) Reduced incidence of readmission

7. Coordination of a successful pain management program requires strong institutional support. Didactic instruction by the department of anesthesiology in nursing education should consist of:
   (a) Care for/troubleshoot catheters and infusion pumps
   (b) Expecting quadriceps weakness as a normal component of a femoral block
   (c) How to administer Intralipid for resuscitation of local anesthetic toxicity
   (d) Delineation of discharge instructions
8. All of the following factors may explain why anesthesiologists in community practice perform fewer peripheral nerve blocks as compared to practitioners in academic institutions except:
   (a) Lack of an accommodating infrastructure
   (b) Deficient exposure during residency training
   (c) Time pressures
   (d) Patient request
   (e) Lack of assistance
9. Regional anesthetic techniques can improve discharge predictability and accelerate discharge eligibility. Social service involvement early in the patients perioperative course can:
   (a) Overcome delays in nursing home placement
   (b) Arrange for patient transportation
   (c) Anticipate lack of home readiness by family members facilitating timely discharge
   (d) Addressing patient concerns resulting in requests for extended hospital stay
   (e) All of the above
10. Useful approaches when dealing with block-related complications include:
   (a) Having realistic preoperative discussions with patients regarding potential complications
   (b) Obtaining meaningful written informed consent
   (c) Keeping accurate records
   (d) All of the above

Answers:

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

Appendix 1: Lipid Rescue Algorithm

LipidRescue™

TREATMENT FOR LOCAL ANESTHETIC-INDUCED CARDIAC ARREST

PLEASE KEEP THIS PROTOCOL ATTACHED TO THE INTRALIPID BAG

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

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

In practice, in resuscitating an adult weighing 70kg:

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

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

Appendix 2: Pain Management Log Book

Anesthesiology Postoperative Pain Management Procedure Record

Postoperative pain management specifically requested by _____

Medical indication (e.g. pain location) _____

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

Team members present: _____ **Correct side and site** ( )

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

Needle:_____ Gauge / Length_____mm ☐ Quincke ☐ Pencil-point
☐ Insulated ☐ Tuohy ☐ Short-bevel ☐ Other:_____

Single-Injection Techniques

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

Continuous Techniques

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

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

Performed by: _____ _____ _____ _____
Name Signature Date Time

Billings Clinic

Patient Identification

Appendix 3: Pain Management Order Sheet

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

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

PAIN MANAGEMENT ORDER SHEET
INTRAVENOUS PCA

(Recommended for patients over 40 kg)

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

DATE: _____ **TIME:** _____

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

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

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

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

   ☐ Oxygen via nasal cannula at _____ L/min

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

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

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

6. **OTHER** instructions: _____
   _____

Signature: _____ # _____ Beeper # _____
Orders verified by: _____ RN _____ RN

1PO

Rev. 4/07 #1-369

Appendix 4: Pain Management Order Sheet

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

PAIN MANAGEMENT ORDER SHEET
CONTINUOUS REGIONAL ANALGESIA

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

Allergies: _____

Height: _____

Weight: _____lb _____ kg ☐ Actual ☐ Estimated

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

Date: _____ **Time:** _____

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

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

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

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

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

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

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

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

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

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

Signature: _____ #_____Telephone # _____ Beeper # _____

Orders verified by: _____ RN _____ RN

1PO

#1-370 Rev. 4/07

Appendix 5: Pain Management Order Sheet

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

Name:
DOB: 00 / 00 / 00 Age: Sex:
Acct#: 0000000 Religion:
MR#: 000000000
Attending MD:
Admitted on: 00 / 00 / 00

PAIN MANAGEMENT ORDER SHEET
EPIDURAL INFUSION

Allergies: _____

Height: _____ Weight: _____ **kg** Pregnant: □Yes □ No Breast Feeding: □ Yes □ No

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

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

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

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

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

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

Fentany 4 mcg/mL +
bupivacaine 0.04%
Continuous Rate: _____ mL/hr
Demand Dose (PCEA):
☐ 3mL every 10 minutes
☐ 5mL every 10 minutes
☐ 5mL every 15 minutes
☐ _____mL every _____minutes

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

MAINTAIN IV saline lock for duration of epidural infusion.

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

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

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

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

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

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

Date: _____ Time: _____

Signature: _____ # _____ Beeper #: _____

Orders verified by: _____ RN _____ RN

IPO

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

Appendix 6: Nursing Assessment Flow Sheet

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

Date	Time	INIT	INIT

Discontinued:

Date	Time	Waste	INIT	INIT

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

Date	Time	INIT	INIT

Discontinued:

Date	Time	Waste	INIT	INIT

ONLY PATIENTS ARE ALLOWED TO PUSH BUTTON

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

Appendix 7: Patient Instruction Sheet for Outpatients Receiving Regional Blocks

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

Pain Management Flow Sheet (PMFS)

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

Signature/Title	Initial	Signature/Title	Initial

Allergies: _____

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

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

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

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

Pain level Scale
0 – 10

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

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

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

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

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

Cardiac Monitor
N = No
Y = Yes

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

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

3PMF

#1-341 Rev 4/07

Appendix 8: Post-op Multimodal Pain Management Orders

Patient Instruction Sheet for Outpatients Receiving Regional Blocks

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

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

It is normal after regional blocks to experience:

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

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

* Temporary hoarseness, a droopy eyelid, and difficulty swallowing

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

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

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

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

Local XXX-XXXX

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

Appendix 9: Anesthesiology Postoperative Pain Management Procedure Record

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

POST-OP
MULTIMODAL PAIN MANAGEMENT ORDERS

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

Allergies: _____

Height: _____ Weight: _____ **kg**

Pregnant: - Yes No Breast Feeding: - Yes No

In conjunction with CRA (See CRA order form)

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

Date: _____ Time: _____ Signature _____ LIP #_____

1PO

1PO

Appendix 10: Outpatient Postoperative Contact Form

Patient Identification

Anesthesiology Postoperative Pain Management Procedure Record

Postoperative pain management specifically requested by _____

Medical indication (e.g. pain location) _____

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

Needle: _____ Gauge / Length _____ mm ☐ Quincke ☐ Pencil-point ☐ Insulated ☐ Tuohy ☐ Short-bevel ☐ Other: _____

Single-Injection Techniques

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

Continuous Techniques

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

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

Performed by: _____ _____ _____ _____

Name Signature Date Time

Appendix 11: Peel-and-Stick Form

Billings Clinic.

Patient Identification

Outpatient Postoperative Contact Form

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

Telephone Interview

Date/Time of Callback: _____

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

_____

_____

_____

Did you meet and talk with your anesthesiologist before surgery?

_____

Do you have any other questions, comments, or suggestions?

_____

_____

_____

_____

Actions

Actions taken by RN:

_____

_____

_____

_____

_____

☐ Unable to contact by telephone. Card sent to address above on _____ (date)

RN: Signature _____ Printed Name _____

HUNTINGTON HOSPITAL
ACUTE PAIN MANAGEMENT SERVICE

_____ PCA
_____ E P I
_____ CRA

Surgeon: _____ Diagnosis code: _____

Anesthesiologist: _____

Operation: _____ ROOM NO. | |

Date of service: _____

HUNTINGTON HOSPITAL ACUTE PAIN MANAGEMENT SERVICE: THERAPY INITIATION

CHECK ONE:

☐ I. V. PCA	☐ EPIDURAL/NEURAXIAL	☐ PERIPHERAL NERVE BLOCK
CPT: 01997 **INITIAL SETTINGS:** ☐ Morphine ☐ Hydromorphone Continuous Rate: _____ mg./hr. Demand Dose _____ mg. Lockout Interval _____ Min. 4 Hr. Dose Limit _____	CPT ☐ Thoracic 62318 + 99231 ☐ Lumbar 62319 + 99231 ☐ Postop Pain Rx only (Daily Mgmt.) 01996 ☐ Postop Visit (Single Shot) 99231 ☐ Blood Patch 62273 **INITIAL SETTINGS:** ☐ Continuous Rate: ___ml./hr./Titrate ___ to ___ Bupivacaine: _____ % Ropivacaine _____% + Fentanyl ___ mcg./ml., Hydromorphone___mcg./ml. or Preserv. Free Morphine ___mcg./ml. ☐ PCEA Dose _____ ml. Delay _____ Min. Other: _____	Brachial Plexus ☐ Single shot 64415 ☐-59 ☐ Continuous 64416 ☐-22 Sciatic ☐ Single shot 64445 ☐ Continuous 64446 Femoral ☐ Single shot 64447 ☐ Continuous 64448 Psoas ☐ Continuous 64449 Bolus _____ ml. Continuous Rate: _____ ml./hr. Ropivacaine _____ % Other _____ During Placement: ☐ Yes ☐ No Heme ☐ Yes ☐ No Paresthesia ☐ Yes ☐ No Pain on Injection ☐ Yes ☐ No Low Resistance to Inj. PCRA Dose _____ ml. Delay _____ Min.

☐ Procedure Explained to Patient Including Risks/Benefits/Alternatives. Patient Consents to Procedure.

POSTOP DAY # ___

HUNTINGTON HOSPITAL · ACUTE PAIN MANAGEMENT SERVICE

SUBJECTIVE: _____

Date: _____

OBJECTIVE: Pain Score: _____ /10

☐ PCA ☐ Epidural ☐ Peripheral nerve block ☐ Single shot neuraxial

☐ Vital signs stable ☐ Alert & oriented ☐ No motor/sensory block ☐ Nausea ☐ Pruritus ☐ Headache

Time: _____

☐ Bromage Score _____

ASSESSMENT/PLAN: ☐ Continue current Rx ☐ Catheter removed, tip intact ☐ Further pain Rx plan _____

Provider Signature: _____

COMMENTS: _____

POSTOP DAY # ___

HUNTINGTON HOSPITAL · ACUTE PAIN MANAGEMENT SERVICE

SUBJECTIVE: _____

Date: _____

OBJECTIVE: Pain Score: _____ /10

☐ PCA ☐ Epidural ☐ Peripheral nerve block

☐ Vital signs stable ☐ Alert & oriented ☐ No motor/sensory block ☐ Nausea ☐ Pruritus ☐ Headache

Time: _____

☐ Bromage Score _____

ASSESSMENT/PLAN: ☐ Continue current Rx ☐ Catheter removed, tip intact ☐ Further pain Rx plan _____

Provider Signature: _____

COMMENTS: _____

POSTOP DAY # ___

HUNTINGTON HOSPITAL · ACUTE PAIN MANAGEMENT SERVICE

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☐ Bromage Score _____

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Provider Signature: _____

COMMENTS: _____

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Part II
Basic Science and Clinical Practice

4

The Anatomy of Pain

Harry J. Gould III • Alan David Kaye

Contents

In years past, the prevailing approach to providing pain control was focused on identifying underlying etiologies or pathologic syndromes, e.g., low back pain, trigeminal neuralgia, and cancer pain, that produce the pain. While treating the presumed source of the pain, attempts to improve the accompanying discomfort relied largely on the use of non-opioid medications and the limited use of opioid and adjuvant analgesics. Over the past 25 years, however, there has been a dramatic increase

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in our understanding of the nervous system and how stimuli associated with actual or potential tissue injury are transduced, transmitted, modulated, perceived, and interpreted to form the basis for initiating appropriate evasive or protective behavior, thereby avoiding or limiting injury. Our current bank of knowledge has led to the recognition that (1) pain in the chronic state is in itself a disease deserving consideration, assessment, and management, (2) pain is not a single entity but a complex, multifaceted experience that warrants detailed and comprehensive evaluation to elucidate symptoms that may reflect specific associated mechanisms amenable to targeted treatment [1, 2], and (3) treatment modalities and management approaches not heretofore considered can be effective and can improve the quality of life for those suffering with pain. This chapter will provide a brief overview of the anatomy of pain that forms the basis for current practice.

Considerations of General Organization

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

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

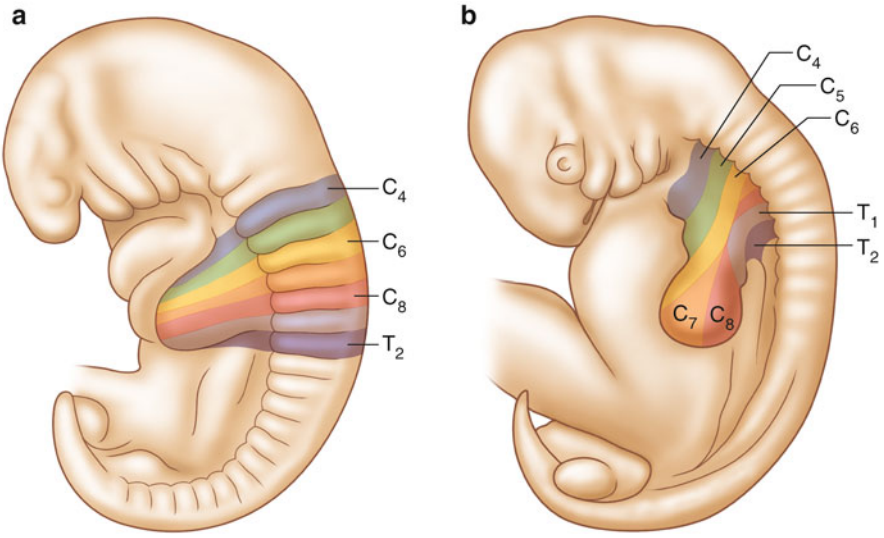

Fig. 4.1 Diagrams of the dermatomal pattern during early development of the pectoral limb bud

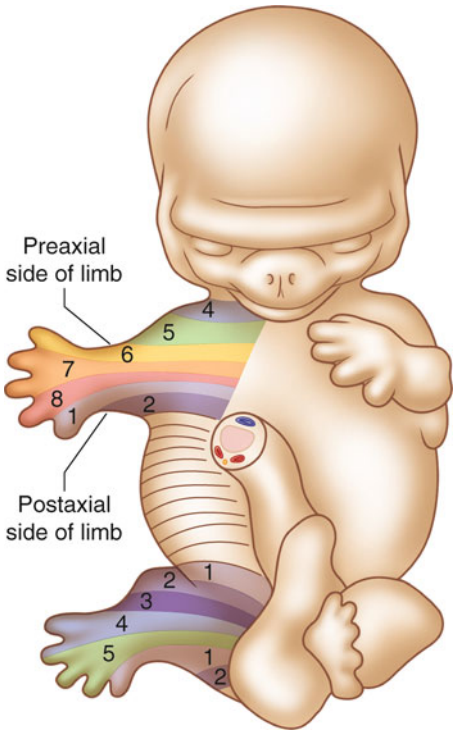

Fig. 4.2 Diagram of the ordered segmental distribution of peripheral afferents during an early stage of development

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

Axons that travel in close proximity to each other are packaged into nerve bundles that provide the conduits for neuronal traffic. Neurons innervating somatic derivatives of several dermatomal levels are packaged together and course through branches of spinal nerves that are distributed to the body wall and appendages (Figs. 4.7 and 4.8). The paths taken by neurons that innervate derivatives of the lateral plate mesoderm are less well defined in that they are variable and can course along blood vessels through elements of branches of the somatic nerves and through splanchnic components of the sympathetic nervous system (Fig. 4.9). These conduits ensure coverage of visceral tissues, smooth muscles, and glands located both in the body wall and in the core regions of the body. As long as the peripheral nerves

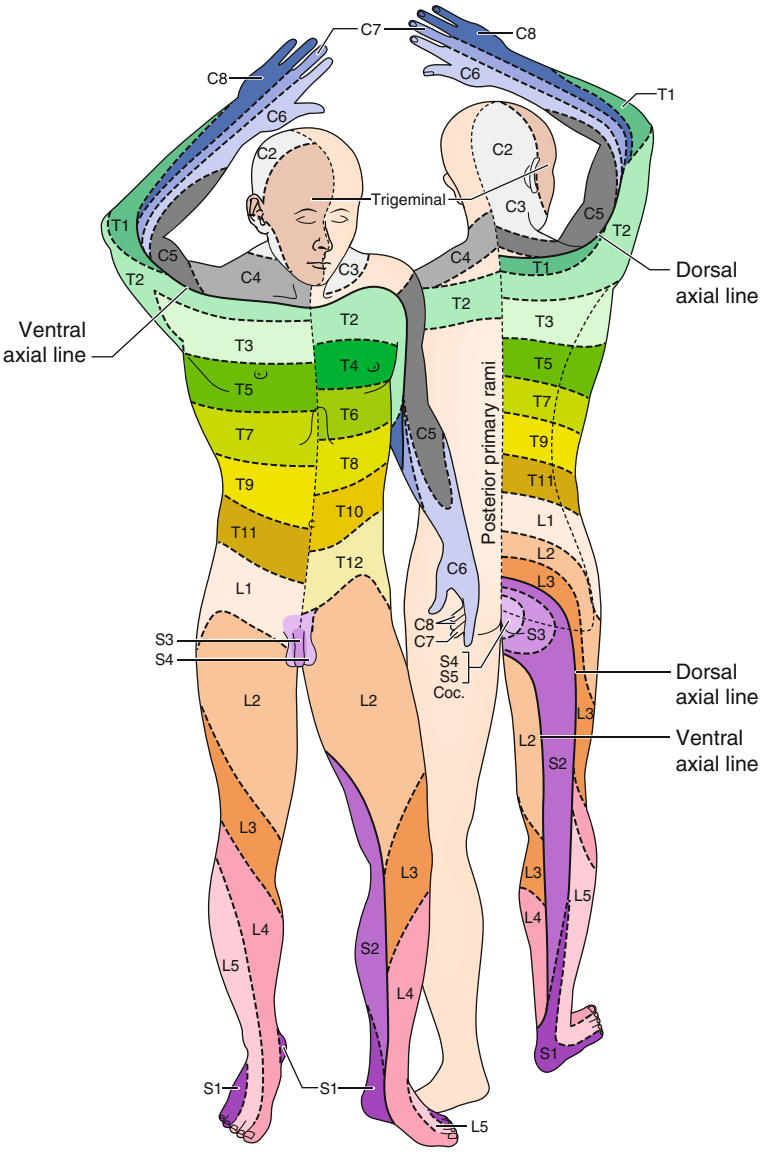

Fig. 4.3 Dermatomal distribution in the adult. Overlapping distribution of segments is indicated for the trunk (adapted from Lockhart RD, Hamilton GF, Fyfe FW. Anatomy of the human body. Philadelphia: J.B. Lippincott Company; 1972)

are intact, damage to an individual nerve root will not result in complete loss of sensation in the area supplied by the damaged root. By contrast, damage to a peripheral nerve will result in a complete loss of sensation in the area served. An understanding of the differences between the patterns of dermatomal and peripheral nerve, thus, is important in assessing localization of site of injury and for determining the effect of diagnostic and therapeutic interventions.

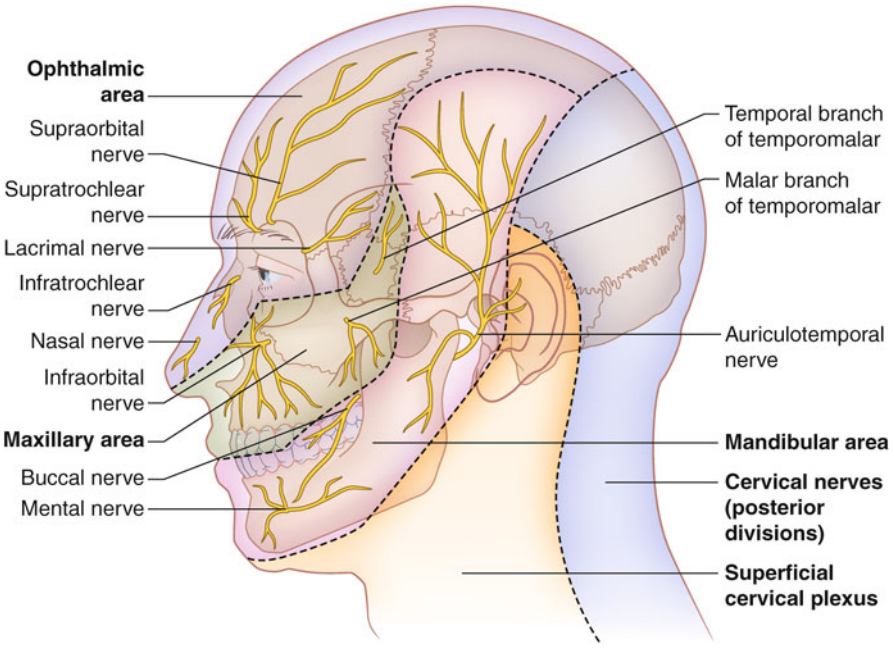

Fig. 4.4 Distribution of sensory nerves to the head (adapted from Gray's Anatomy, 1966)

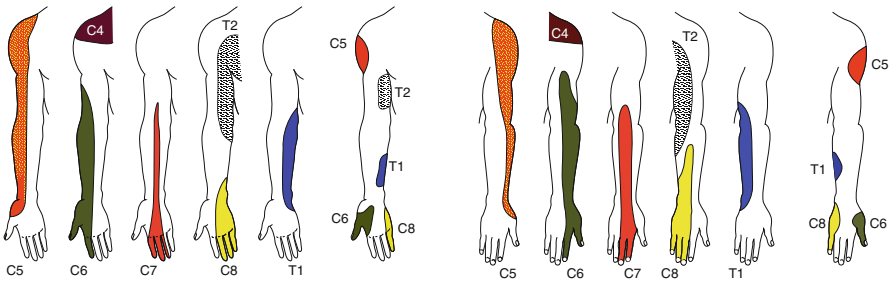

Fig. 4.5 Dermatomal distribution of the anterior (*left*) and posterior (*right*) upper extremity, showing areas supplied by only one segmental level (illustrations 6 and 12 from *left*) (adapted from Lockhart RD, Hamilton GF, Fyfe FW. Anatomy of the human body. Philadelphia: J.B. Lippincott Company;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

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

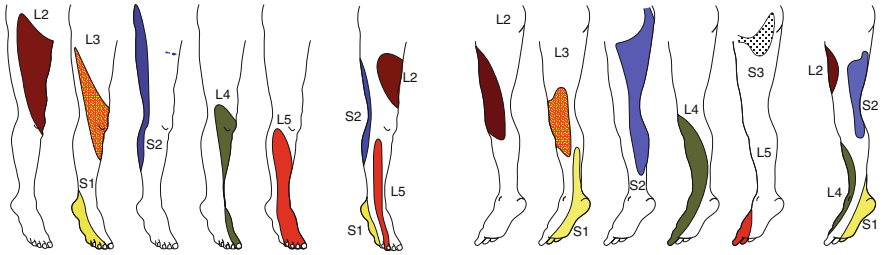

Fig. 4.6 Dermatomal distribution of the anterolateral (*left*) and posteromedial (*right*) lower extremity, showing areas supplied by only one segmental level (illustrations 6 and 12 from *left*) (adapted from Lockhart RD, Hamilton GF, Fyfe FW. Anatomy of the human body. Philadelphia: J.B. Lippincott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

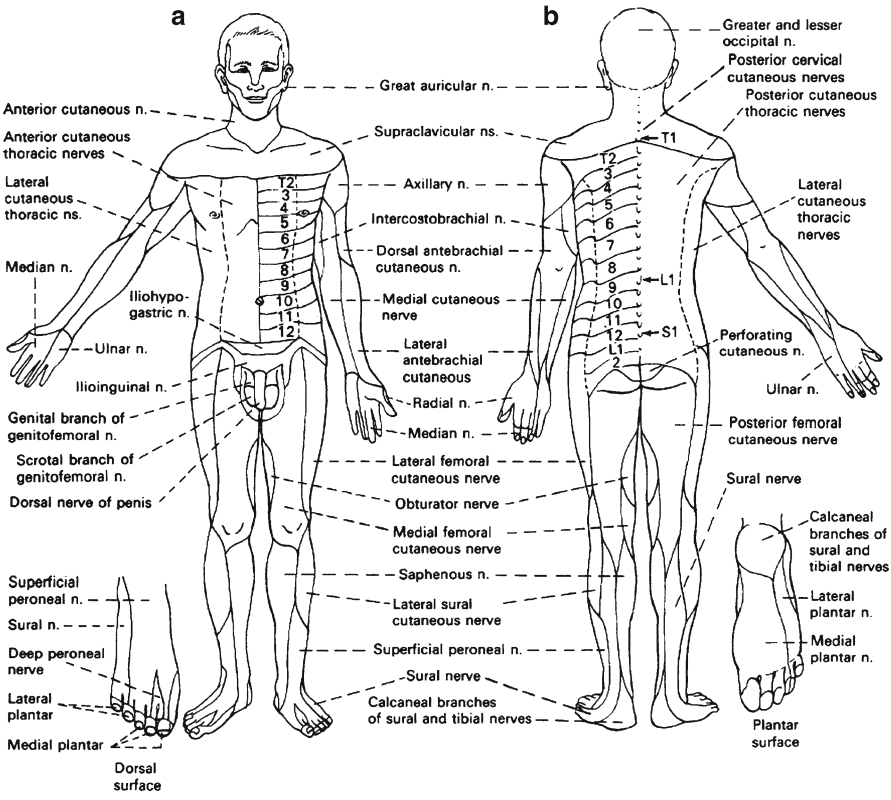

Fig. 4.7 The cutaneous fields of peripheral nerves (n.). (**a**) Anterior view. (**b**) Posterior view. In both figures, the numbers on the trunk refer to the intercostal nerves (modified from Haymaker W, Woodhall B. Peripheral nerve injuries: principles of diagnosis. Philadelphia: WB Saunders; 1945)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

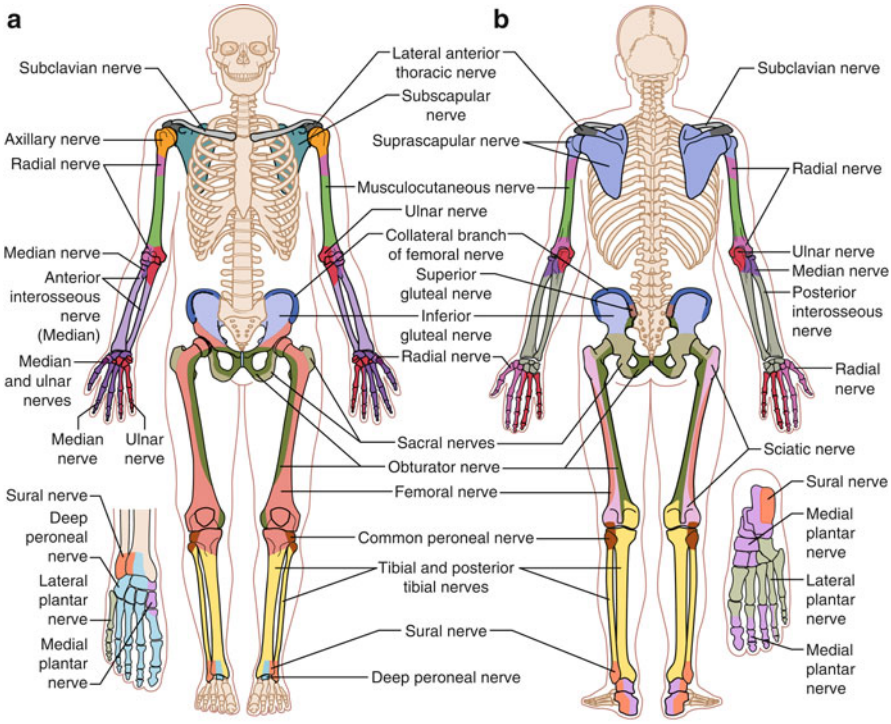

Fig. 4.8 The peripheral nerve supply of the skeleton. (**a**) Anterior view. (**b**) Posterior view. The various peripheral nerve fields are indicated by different patterns (modified from Dejérine J. Sémiologie du système nerveux. Paris: Masson; 1914) (adapted from LeResche L, Bonica's management of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

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

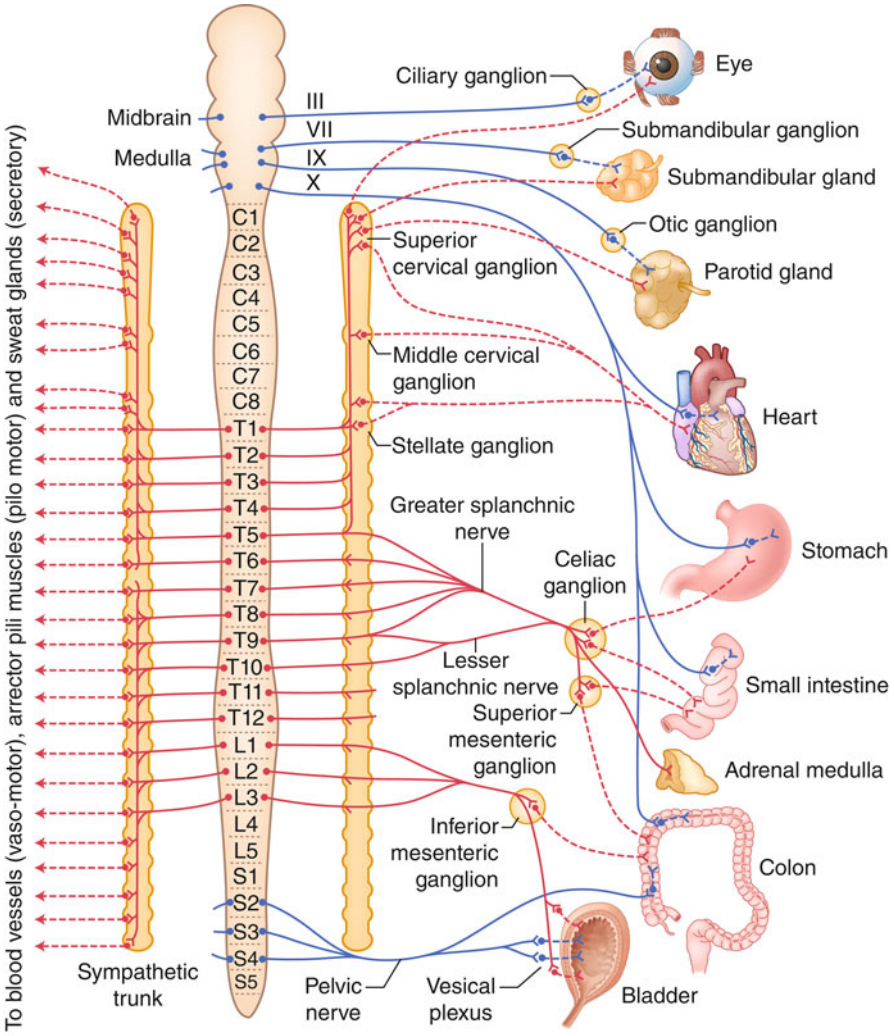

Fig. 4.9 Schematic diagram showing general arrangement of the autonomic system. The sympathetic components are shown in *red*, while the parasympathetic components are in *blue*. *Solid lines* represent preganglionic fibers; *broken lines* indicate postganglionic fibers. The sympathetic fibers to the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

Stimulus Transduction and Transmission

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

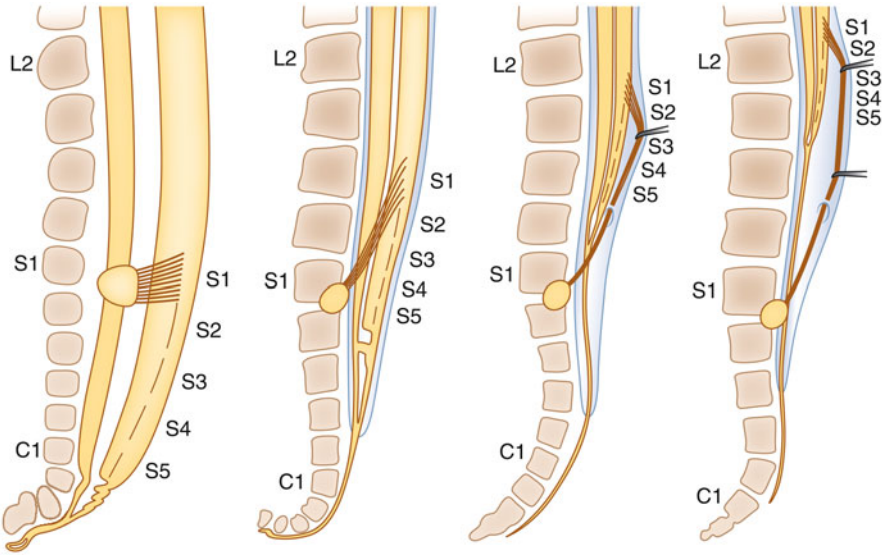

Fig. 4.10 Four successive stages in development of the caudal end of the human spinal cord (after Streeter). They show the formation of the filum terminale and the progressive obliquity of the first sacral nerve which is caused by the differential growth of the spinal cord and vertebral column. From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

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

Free nerve endings comprise the distal terminals of nociceptive neurons. They are distributed within the epidermis of the skin, deep tissues, elements of the

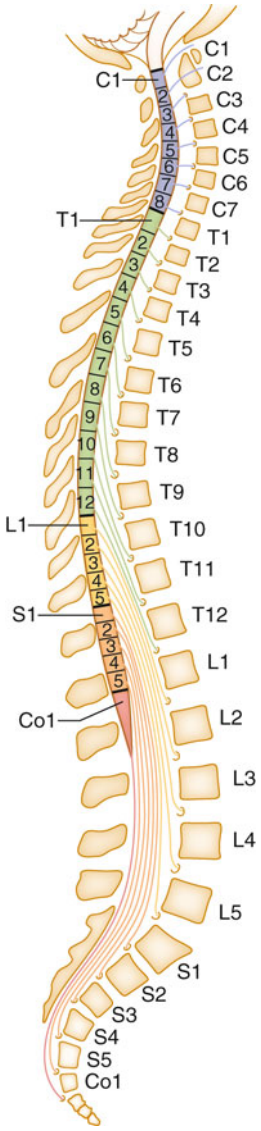

Fig. 4.11 Diagram of the position of the spinal cord levels in relation to the vertebral bodies and spinous processes of the vertebral column

musculoskeletal system, and internal visceral organs. The nociceptors are optimally positioned to monitor changes in the thermal, mechanical, and chemical environment of every region of the body. Potentially injurious stimuli, when present in the peripheral tissues, trigger the release of a myriad of chemical mediators that set into motion a constellation of events that alters the membrane permeability of afferent nerve terminals to charged ions. Among the inciting nociceptive events are the release of potassium ions, protons, and bradykinin and the initiation of the arachidonic acid cascade which leads to the production of prostaglandins and leukotrienes.

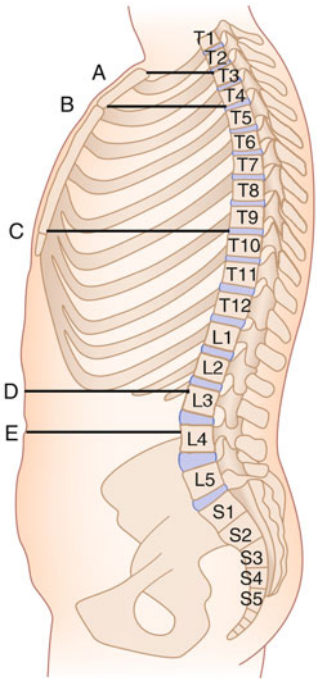

Fig. 4.12 A lateral view of the body, showing the vertebral levels of certain landmarks on the anterior thoracic and abdominal walls: (**a**) suprasternal notch, (**b**) sternal angle, (**c**) xiphisternal joint, (**d**) subcostal line, and (**e**) umbilicus (adapted from Crafts RC. A textbook of human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

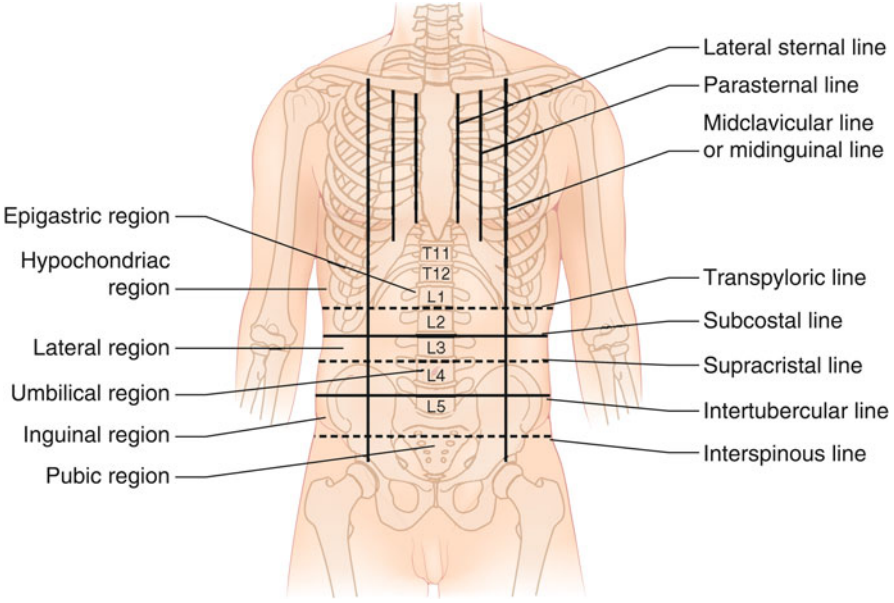

Fig. 4.13 Lines of reference on anterior thoracic and abdominal walls (adapted from Crafts RC. A textbook of human anatomy. 2nd ed. New York: Wiley; 1979)

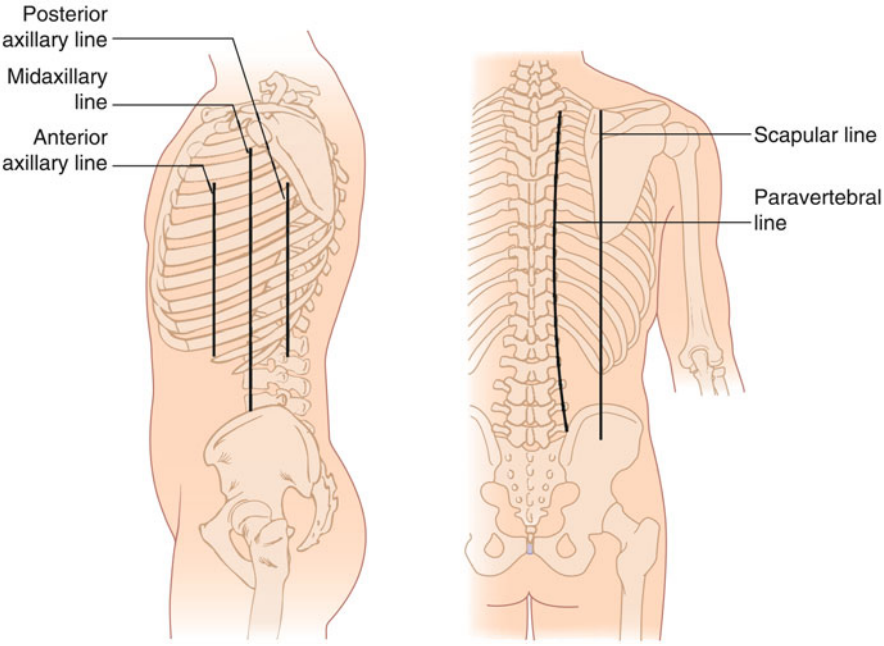

Fig. 4.14 Lines of reference on lateral and posterior chest wall (adapted from Crafts RC. A textbook of human anatomy. 2nd ed. New York: Wiley; 1979)

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

Axons of the Aδ system range in diameter from 1 to 6 μm and are ensheathed by a thin layer of myelin [4]. The myelin provides a supportive and trophic effect for axons, and in addition to insulating axons within a nerve bundle from each other for the maintenance of temporal and spatial integrity of the signal, it serves to enhance conduction velocity. The Aδ axons are supported by cell bodies that measure 25–30 μm in diameter and serve small receptive fields. They respond to relatively

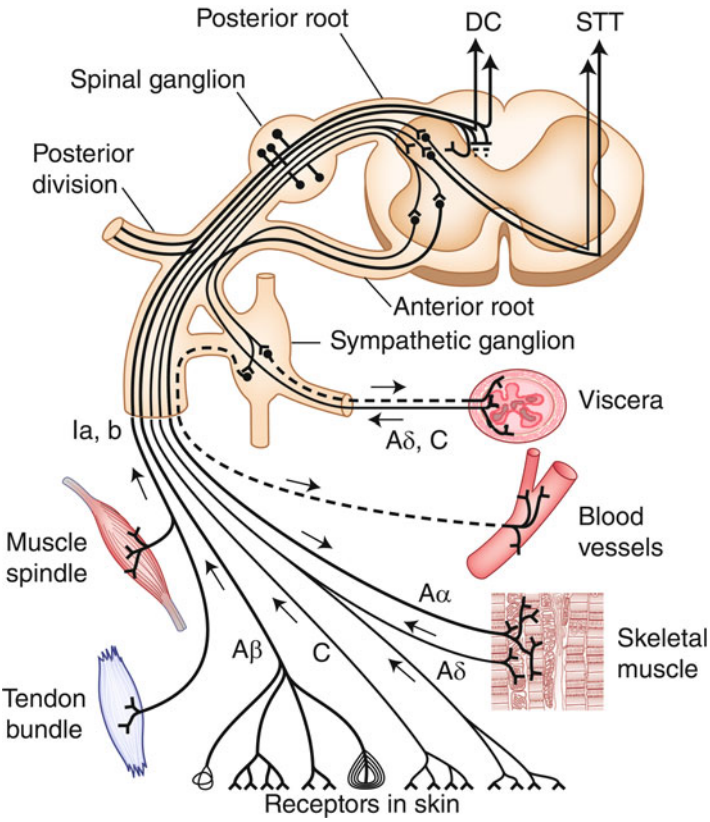

Fig. 4.15 A simplified schema of a spinal nerve and the different types of fibers contained therein (DC, dorsal columns; STT, spinothalamic tract) (adapted from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's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

low levels of noxious stimulation and conduct impulses at velocities between 5 and 30 m/sec. Although they respond preferentially to mechanical stimulation, they also respond to noxious heat. As the axons approach the spinal cord, they diverge from the main nerve trunk and enter the dorsal root where they course by their cell bodies in the DRG and enter the spinal cord to terminate on neurons in Rexed laminae I, II, III, V, and X [8] (Fig. 4.19). The axons of the C fiber system are unmyelinated [5]. They are supported by DRG neurons measuring 10–15 μm in diameter, serve larger receptive fields than those served by A-delta fibers, require a higher stimulus intensity to initiate an action potential, and convey information at velocities between 0.5 and 2 m/s. C fibers respond to polymodal stimuli but preferentially respond to noxious heat. Their central elements course medially in the dorsal root and terminate on neurons

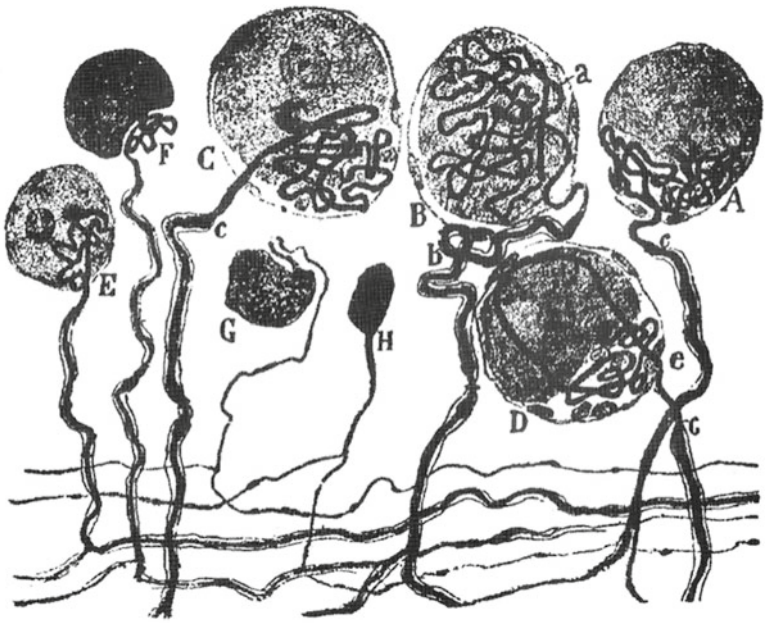

Fig. 4.16 Large and small DRG cell somata, the T-stem axon with its glomerulus, and the T-junction (adapted from Cajal, 1911, p. 428)

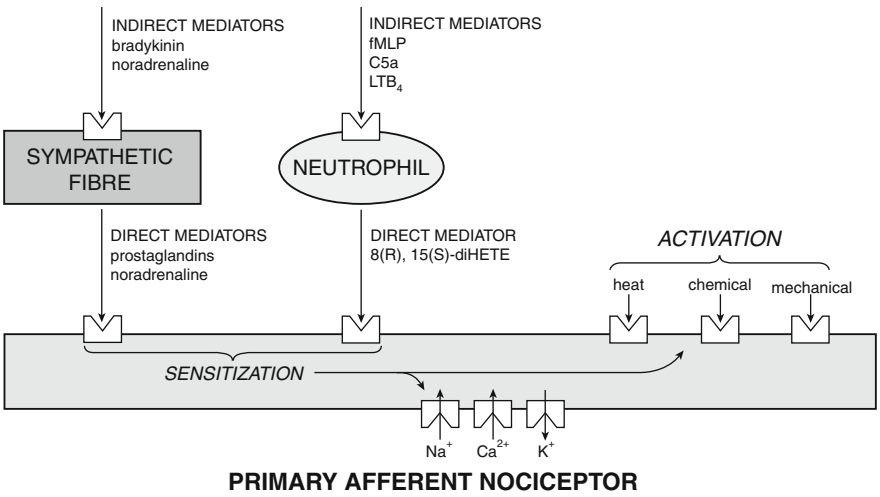

Fig. 4.17 Direct and indirect mechanisms by which inflammatory mediators sensitize primary afferent nociceptors (adapted from Levine JD, Reichling DB. Peripheral mechanisms of inflammatory pain. In: Wall PD, Melzack R, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 59–84)

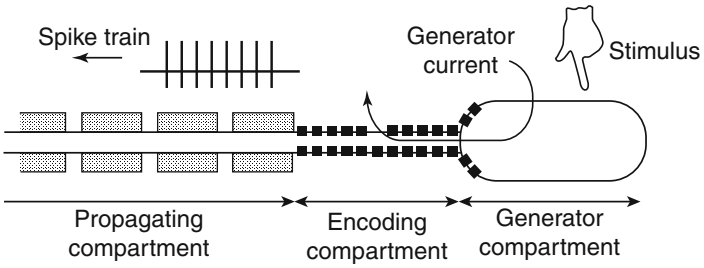

Fig. 4.18 Sketch of sensory ending showing generator, encoding, and propagating compartments (adapted from Devor M, Seltzer Z. The pathophysiology of damaged peripheral nerves. In: Wall PD, Melzack R, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 129–64)

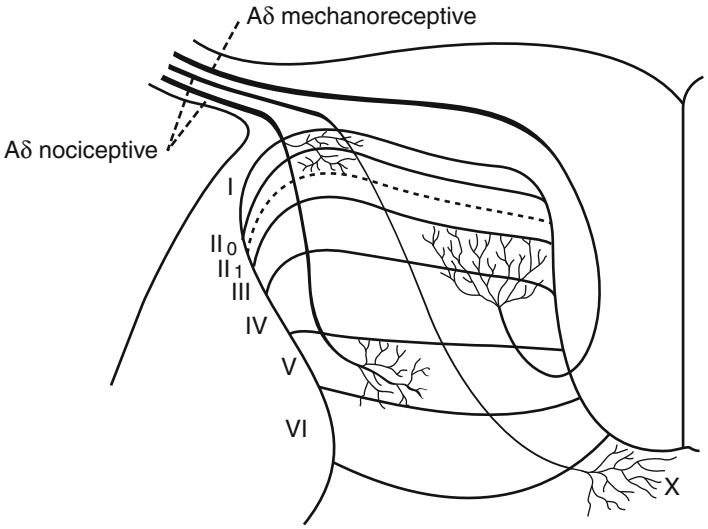

Fig. 4.19 Schematic diagrams of the course and termination of collaterals of the Aδ cutaneous fibers in the dorsal horn of the spinal cord (adapted from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's management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

in Rexed lamina I, the outer portion of lamina II, and lamina V [8] (Fig. 4.20). Upon entering the spinal cord, the axons of the primary nociceptors ascend and descend in the zone of Lissauer. The majority of these fibers ascend approximately two spinal levels before terminating in the dorsal horn.

In the resting state, the free nerve endings of nociceptive afferents maintain a polarized membrane with a higher concentration of sodium ions outside the cell.

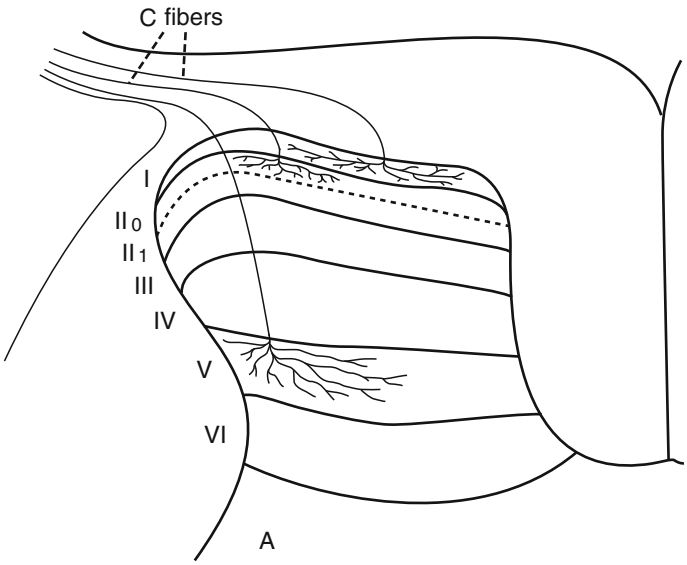

Fig. 4.20 Schematic diagrams of the course and termination of collaterals of unmyelinated C fibers in the dorsal horn of the spinal cord (adapted from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's management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

Noxious heat (>45–55°C), cold (8–25°C), mechanical (pressure or distention; 60 g/mm^2), or chemically mediated stimuli increase the permeability of the membrane to charged ions, thereby setting up a generator current (Fig. 4.18) that leads to a subsequent shift in the electrochemical gradient and voltage across the membrane [7, 9]. The change in voltage alters the configuration of voltage-gated channels, allowing entry of predominantly sodium ions into the cell in exchange for potassium ions, and the initiation of an action potential, which is propagated along the axon to the central nervous system. In myelinated axons like those of the Aβ and Aδδ system, the excitable membrane that supports the propagation of action potentials is found only in the intervals between adjacent segments of myelin, called nodes of Ranvier, where there is a high density of sodium channels. Since membrane depolarization occurs at the nodes of Ranvier, impulses "jump" from one node to the next in a saltatory fashion, resulting in rapid conduction of the action potential (Fig. 4.21). The fiber diameter and the internodal distance are primary determinants of the conduction velocity of the axon. In unmyelinated axons like those of the C fiber system, sodium channels are distributed along the entire length of the axon (Fig. 4.21). Depolarization is propagated contiguously between adjacent membrane segments, resulting in the slowest impulse conduction of any system. After the passage of the action potential, the electrochemical gradient is reestablished through energy-dependent sodium/potassium pumps (Na^+/K^+, ATPase) that transport sodium ions out of the cell in exchange for potassium ions.

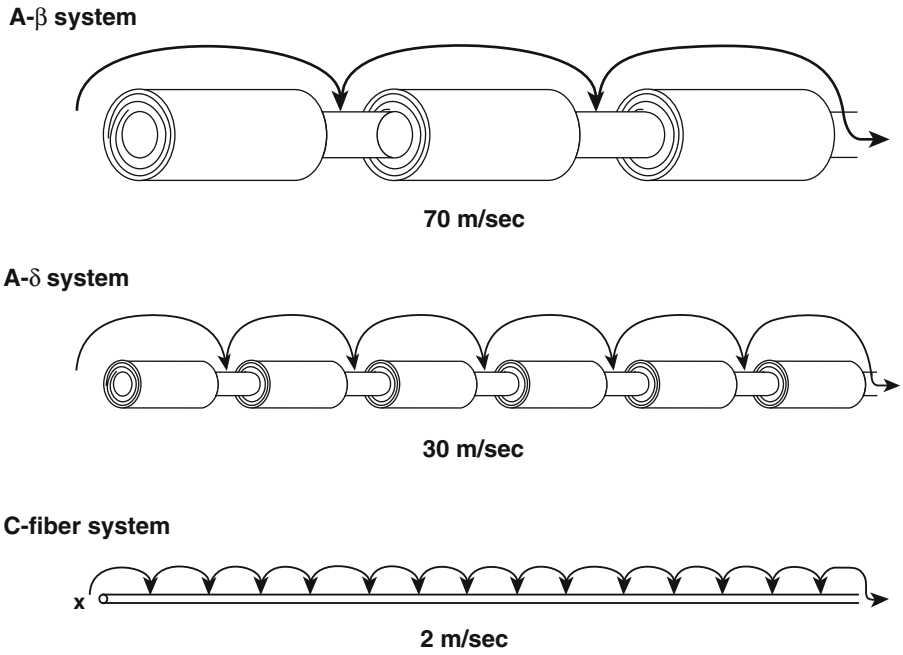

Fig. 4.21 Diagram depicts the relationship between fiber diameter, myelination, and conduction velocity in the peripheral nervous system (adapted from Gould HJ III. Understanding pain: what it is, why it happens, and how it's managed. New York: American Academy of Neurology Press, Demos; 2007)

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

By contrast, when non-noxious mechanical stimuli are presented to specialized afferent end organs, e.g., Pacinian and Meissner corpuscles, Ruffini endings, and Merkel cells (Fig. 4.22), the membrane permeability of large (>25 μm in diameter), low-threshold neurons of the Aβ system is similarly altered, thus initiating action

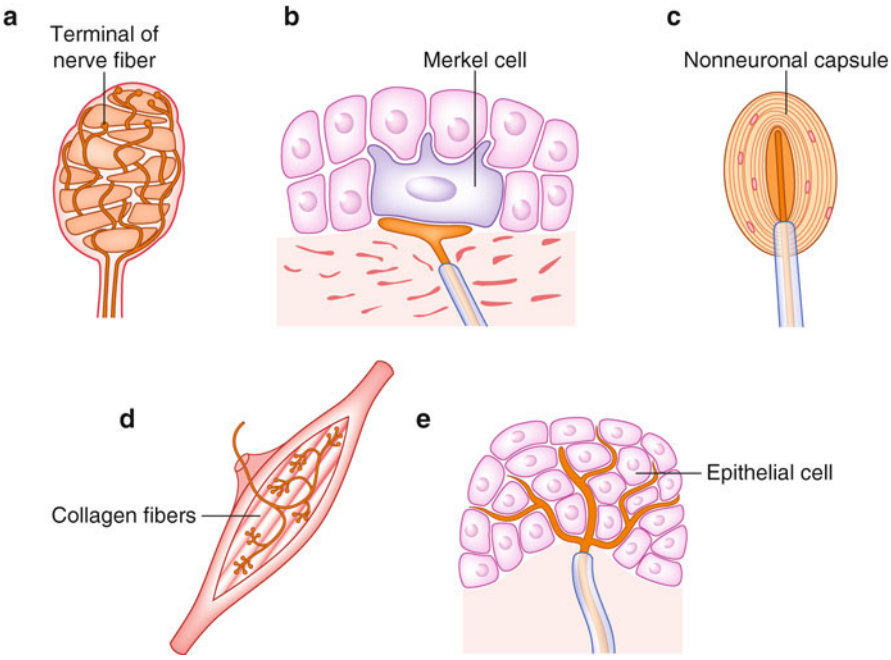

Fig. 4.22 Morphological features of somatosensory receptors, including the variation in non-neural components. (**a**) Meissner corpuscles are composed of axonal loops, separated by non-neuronal, supporting cells; (**b**) Merkel disks are characterized by the close association between afferent axons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

potentials conveying information of a non-noxious tactile nature [5]. These action potentials are conducted along large, 6–12-μm diameter myelinated axons at velocities between 30 and 70 m/s. Upon arriving at the spinal cord, the axons enter the cuneate and gracile fasciculi and ascend ipsilaterally in the spinal cord to terminate in the cuneate and gracile nuclei of the caudal medulla. Axons arising from neurons located in the cuneate and gracile nuclei then cross the midline of the neuraxis and ascend in the medial lemniscus to terminate in the lateral portion of the ventral posterior nucleus of the thalamus (VPN). Collaterals from the Aβ afferents also project into the dorsal horn where they terminate in Rexed laminae III, IV, and V and, through stimulation of inhibitory interneurons, can reduce the intensity of nociceptive signals allowed through the dorsal horn (Fig. 4.23). Similar low-threshold tactile afferents that arise from the head course in branches of the trigeminal nerve and enter the central nervous system at the level of the pons. These afferents terminate

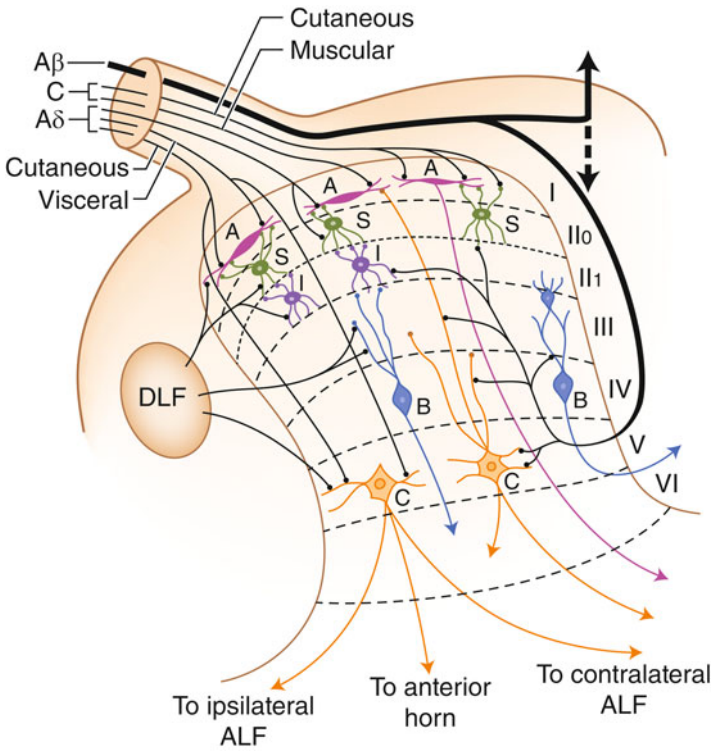

Fig. 4.23 Simplified schematic cross-section diagram of input and output of the dorsal horn of the spinal cord as well as interneurons and axonal terminals of descending control systems (adapted from Terman GW, Bonica JJ. Spinal mechanisms and their modulation. In: Loeser JD, Butler SH, Chapman CR, Turk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

in the principal sensory nucleus. Axons arising from the principal sensory nucleus cross the midline, join the medial lemniscus, and ascend through the rostral brain stem to terminate in the medial portion of the VPN.

Stimulus Modulation

Upon entry of the central gray matter, primary afferents release stored excitatory neurotransmitters, thereby relaying the initial nociceptive signal to either wide dynamic range or nociceptive-specific neurons of the dorsal horn (Fig. 4.24). Through this connection, the modality and the temporal and spatial aspects of the nociceptive signal are integrated. The sum of that integration is then transmitted to higher levels of the nervous system for further processing. The wide dynamic

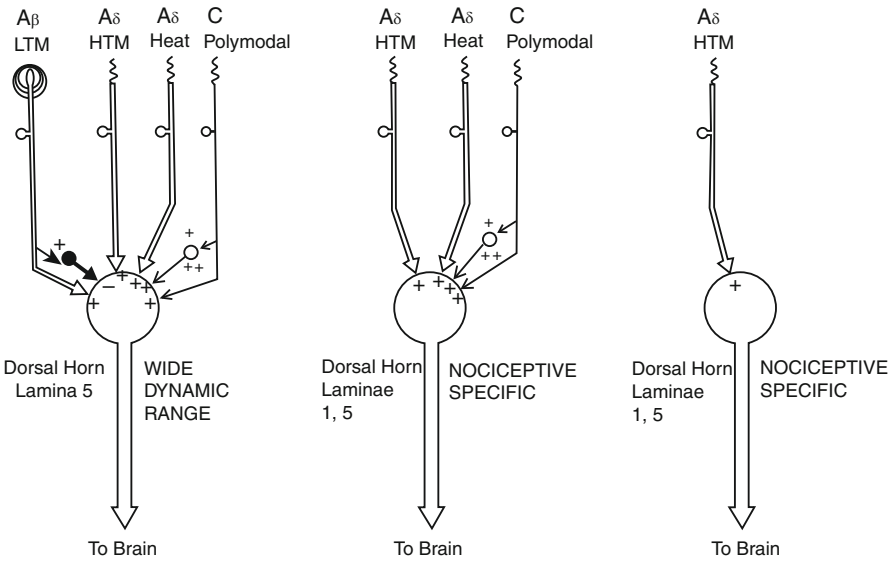

Fig. 4.24 Three types of nociceptive cells in the dorsal horn, their inputs from primary afferents, their location in the spinal cord, and their output to ascending systems. Wide dynamic range neurons receive inputs from low-threshold mechanoreceptive primary afferents (LTM), high-threshold mechanoreceptive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

range neurons are found primarily in lamina V and are responsible for much of the information that is transmitted to the brain stem and thalamus. These neurons receive not only polymodal inputs from high-threshold mechanical and heat-sensitive Aδ and C fiber nociceptors but also inputs from collaterals of non-nociceptive, low-threshold mechanical Aβ afferents and local internuncial neurons of the dorsal horn. They have a moderate threshold for initiating an impulse and are responsible for signals related to itch and flutter. Inputs to the wide dynamic range neurons provide the essential segmental framework for the "gate control theory" proposed by Melzack and Wall [18] whereby impulses transmitted by low-threshold mechanoreceptors can reduce the nociceptive signal that is relayed to higher integrative levels for conscious perception (Fig. 4.25). By comparison, nociceptive-specific neurons are located in laminae I and V and receive inputs only from high-threshold mechanical and heat-sensitive Aδ and C fiber nociceptors. Nociceptive-specific neurons receive inputs that may be either polymodal or modality specific and possess the capability of supporting after discharges. "Silent nociceptors" are a special group of nociceptive-specific neurons that become active only during periods of inflammation and tissue injury and provide a means for amplifying the nociceptive signal.

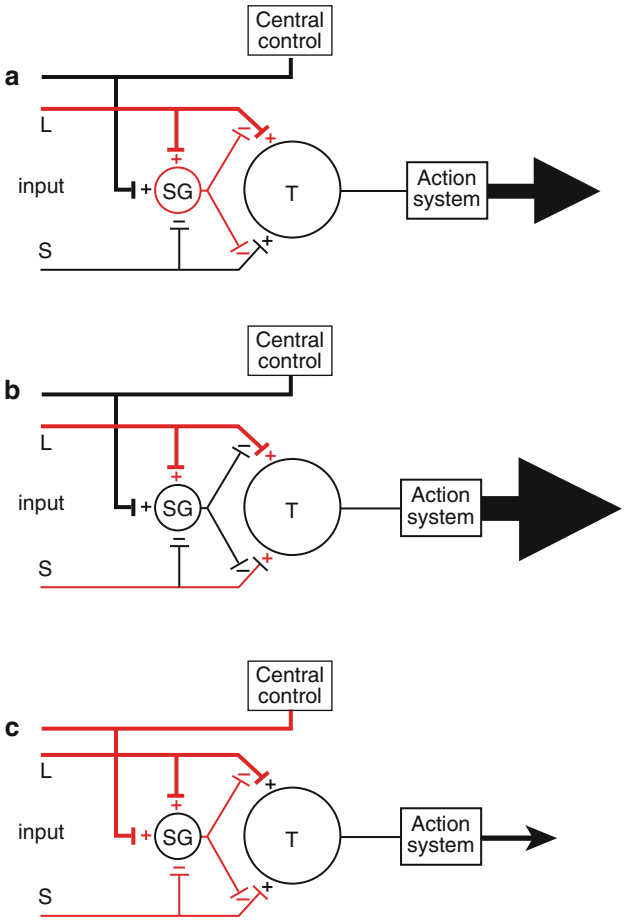

Fig. 4.25 Modified schematic diagram of the "gate control theory" of Melzack and Wall, 1965. SG represents an interneuron in the substantia gelatinosa of the dorsal horn. T represents a cell that transmits the nociceptive signal for higher central processing

When an action potential that is initiated by a nociceptive stimulus reaches the central afferent terminal, calcium enters the synaptic bouton through voltage-gated calcium channels (Fig. 4.26). In the presence of calcium, vesicles containing excitatory neurotransmitters, such as glutamate, aspartate, calcitonin gene-related peptide (CGRP), substance P (sP), neurokinin, vasoactive intestinal peptide (VIP), neuropeptide Y (NP-Y), galanin, or somatostatin, fuse with the terminal cell membrane and release their contents into the synaptic cleft [4, 5]. The neurotransmitters cross the synaptic cleft, recognize receptors on the postsynaptic relay cell, and, through specific stoichiometric interaction, alter the membrane properties of ligand-gated receptors on the receiving neuron. The ligand–receptor interaction initiates a

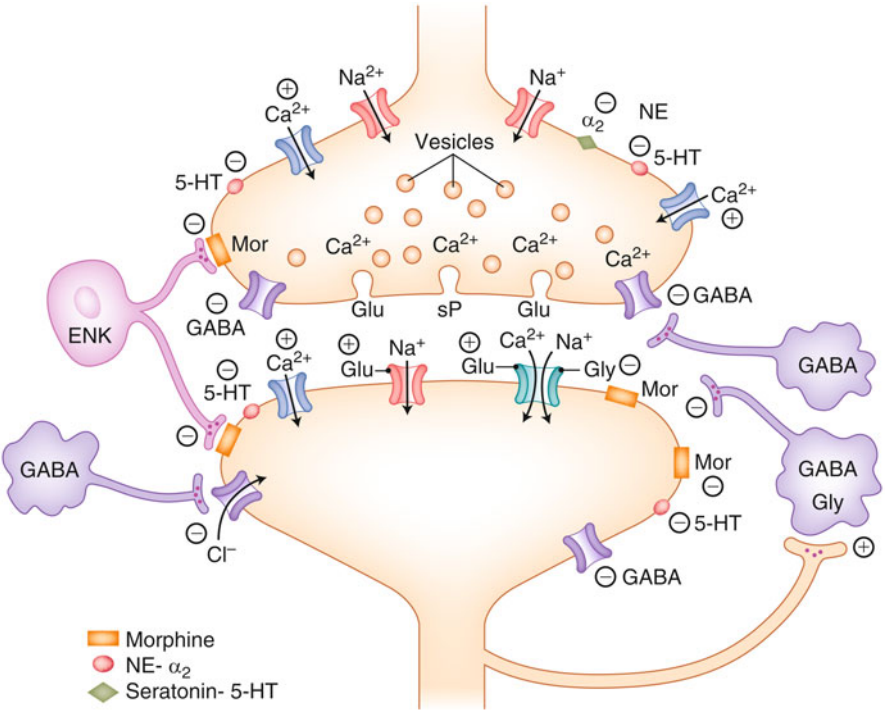

Fig. 4.26 Schematic depiction of a primary afferent synapse on a relay neuron in the dorsal horn of the spinal cord. ENK, enkephalinergic neuron; NE, norepinephrine; 5-HT, serotonin; Glu, glutamate; Gly, glycine; MOR, μ-opioid receptor; and GABA, gamma (γ)-aminobutyric acid (adapted from Gould HJ III.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

cascade of intracellular events that enables the triggering of the next impulse in the chain. The stability of the synapse is reestablished either by removal of the neurotransmitter from the synaptic cleft through enzymatic degradation, through reuptake into the presynaptic terminal or transport into astrocytes that support the synapse [10], or through the activation of processes that inhibit synaptic transmission. One such process is the collateral activation of inhibitory interneurons within the dorsal horn that release inhibitory neurotransmitters such as glycine and gamma (γ)-aminobutyric acid (GABA). These transmitters inhibit further release of excitatory neurotransmitters from the presynaptic terminal and stabilize the postsynaptic cell. It is the critical balance between the excitatory components of the afferent pathway whose role is to ensure transmission of the signal warning of impending injury. It is the inhibitory components that through amplification can enhance or through suppression can reduce the amount of nociceptive signal that is allowed to pass to higher levels of the nervous system and be perceived at any given time (Fig. 4.27).

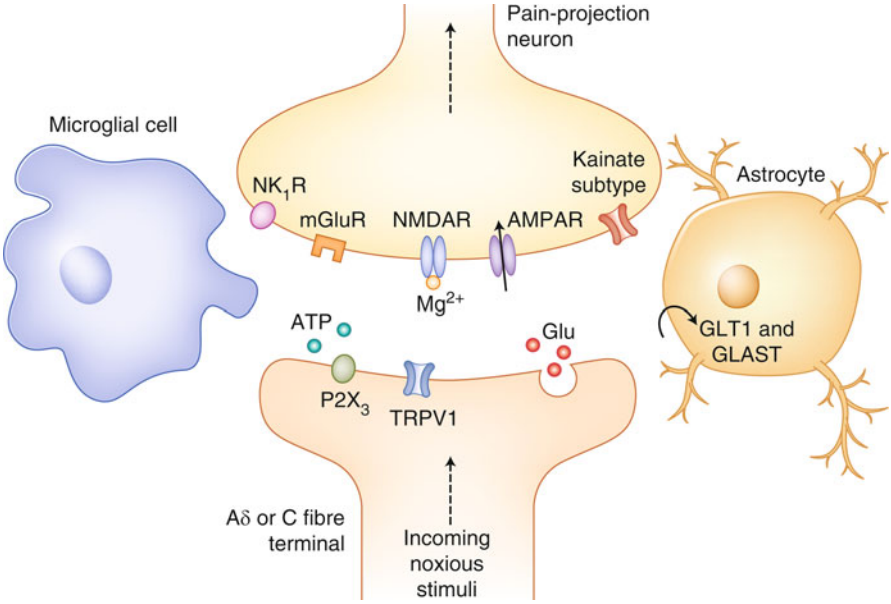

Fig. 4.27 Schematic depiction of the glial contribution to the processing of the primary afferent signal. Under healthy circumstances, low-frequency activation of Aδ and C fiber nociceptors by mild noxious stimuli leads to glutamate (Glu) release from the central presynaptic afferent nerve terminals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

Stimulus Perception and Interpretation

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

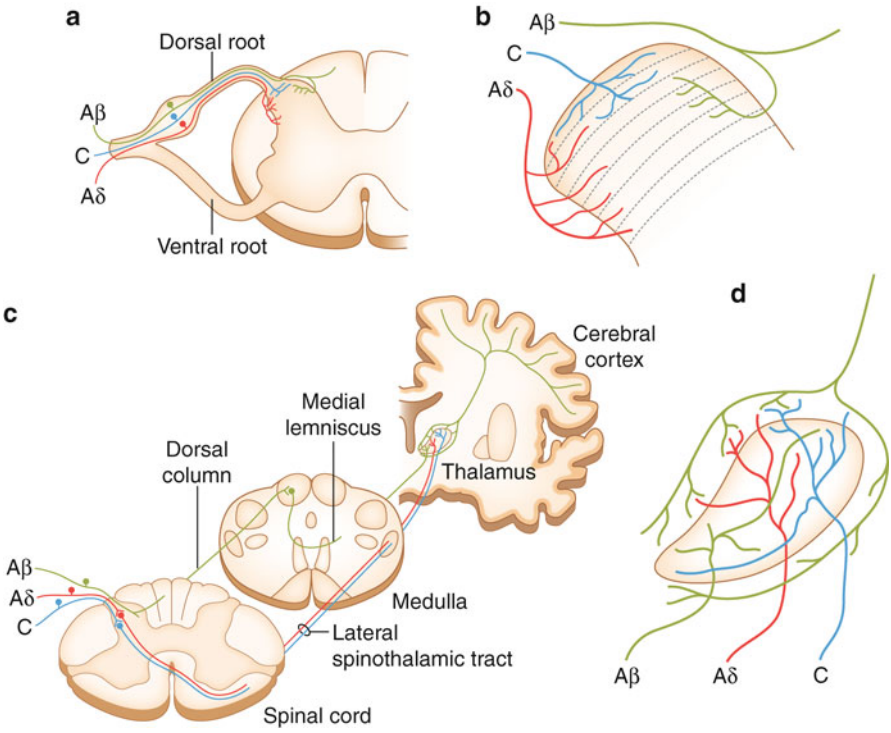

Fig. 4.28 Distribution of pathways involved in transmitting nociceptive information from peripheral nerves to higher levels of the brain for processing. (**a**) Depicts sensory nerves passing through dorsal roots en route to points of termination in the dorsal horn of the spinal cord and medulla. Specific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

somatotopically organized, and modality specific. The second component, the paleospinothalamic tract, provides the basis for the affective and modulatory components of pain and is associated with the C fiber system. Its projections are more diffusely organized. In addition to projecting to the VPN, the paleospinothalamic tract provides collateral connections to the nuclei of the rostral ventromedial medulla (RVM), the lateral tegmental nucleus (LTN), the periaqueductal gray (PAG), the posterior and intralaminar nuclei of the thalamus, the basal telencephalic regions, limbic and paralimbic forebrain, amygdala, fornix, habenula, septal nuclei, and the hypothalamus. Upon termination in the thalamus, the nociceptive signal is consciously perceived [11].

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

Stimulus Modulation and Behavioral Response

The hypothalamus monitors basal body functions, such as thirst, hunger, satiety, sexual function, blood pressure, temperature, and emotion, and influences behavior based on conscious and subconscious information sent from cortex and from various body organs to maintain normal body function. Hypothalamic modulation of the behavioral response can be affected through the release of several hormones, including vasopressin, corticotropin-releasing factor (CRF), and pituitary adrenocorticotropic hormone (ACTH), that act centrally or peripherally to produce direct or indirect activity on pain-transmitting neurons. The process of modulation occurs through direct projections that affect the activity of enkephalinergic neurons of the PAG, the norepinephrine-containing neurons of the LTN, the serotonergic neurons of the RVM, and the neurons in the entry zones that receive primary afferent input [8, 12]. Projections from the RVM and the LTN descend through the brain stem and the dorsolateral funiculus of the spinal cord and synapse on the terminals of the primary afferent neurons and on inhibitory enkephalinergic and GABAergic interneurons of the dorsal horn, thereby indirectly affecting the transmission of nociceptive signals through the dorsal horn (Fig. 4.29). These projections can block the release of neurotransmitter from the primary afferent terminals, stimulate local inhibitory interneurons, or stabilize the membrane of the relay neurons and thus suppress the amount of nociceptive signal that is allowed to pass through the dorsal horn en route to higher integrative centers. Depending on the state of the individual, modulation of these descending systems can produce the opposite effect through

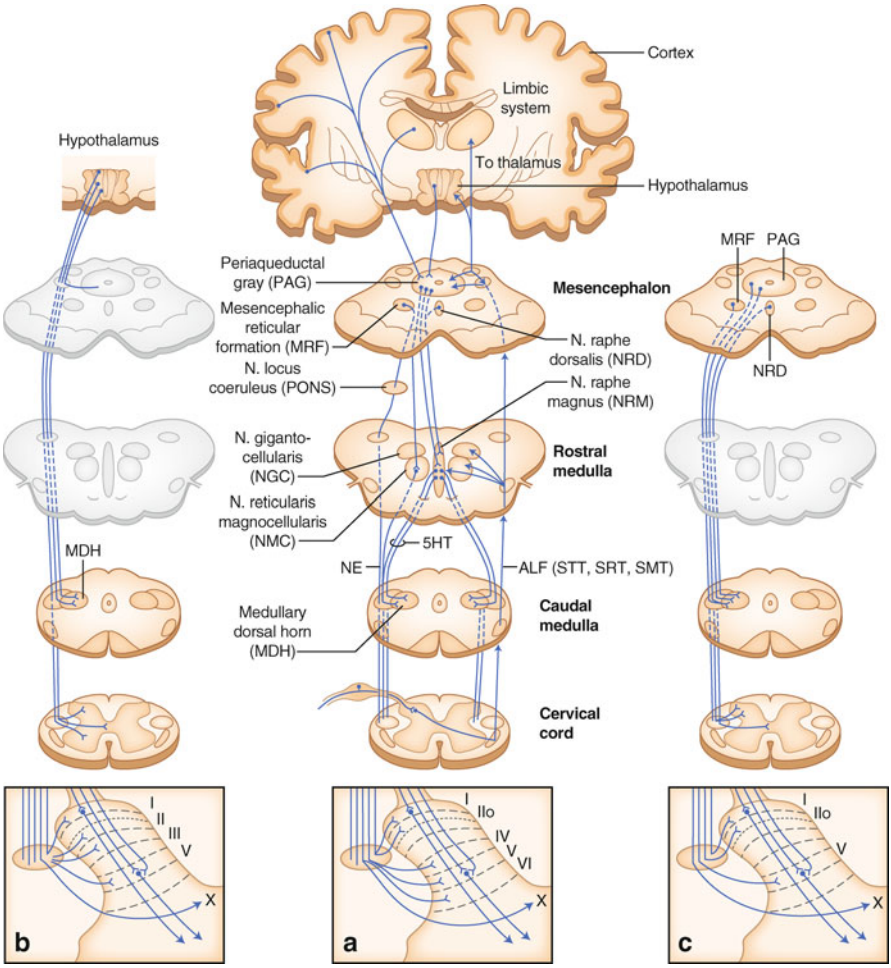

Fig. 4.29 Descending endogenous pain inhibitory systems. (**a**) The most extensively studied and probably the most important descending system, composed of four tiered parts. The ascending anterolateral fasciculus (ALF), composed of the spinothalamic, spinoreticular, and spinomesencephalic tracts, has important inputs into the nucleus raphe magnus (NRM), nucleus magnocellularis (NMC), nucleus reticularis gigantocellularis (NGC), and the periaqueductal gray (PAG) via the nucleus cuneiformis. The ALF also has input to the medullary/pontine reticular formation, the nucleus raphe dorsalis (NRD), and the mesencephalic reticular formation (MRF). The PAG receives input from such rostral structures as the frontal and insular cortices and other parts of the cerebrum involved in cognition, and from the limbic system, thalamus, and hypothalamus, which sends β-endorphin axons to the PAG. The locus coeruleus in the pons is a major source of noradrenergic input to the PAG and dorsal horn (tract-labeled NE). These mesencephalic structures (PAG, NRD, MRF) contain enkephalin (ENK), dynorphin (DYN), serotonin (5-HT), and neurotensin (NT) neurons, but only the latter two send axons that project to NRM and NGC. Here, they synapse with neurons that are primarily serotonergic, whose axons project to the medullary dorsal horn and descend in the dorsolateral funiculus to send terminals to all laminae of the spinal gray (the densest populations are found in laminae I, II, and V of the dorsal horn and the motor neuron

reduction of the level of direct inhibitory input or through the disinhibition of local inhibitory circuits, thus amplifying nociceptive signals and augmenting the likelihood that additional signals of a painful nature will be transmitted to the thalamus for perception [8].

For optimum survival, it is important to prepare the organism for an appropriate behavioral response and return the monitoring system to optimum levels of functioning in anticipation of additional warnings. This function is built into the nervous system. Since pain may well signal a threat to the survival of at least a part of an individual, painful stimuli automatically prepare the individual for rapid assessment of the afferent stimulus and the initiation of defensive "fight-or-flight" behavior through activation of the sympathetic nervous system (Fig. 4.30). The sympathetic nervous system controls blood pressure, heart and breathing rate, and the volume of blood that flows to specific tissues – more to voluntary muscles, heart, and lungs and less to the intestinal system and skin. The neurotransmitter that is released to produce these responses is norepinephrine. When released in the vicinity of peripheral afferent nerve terminals, impulse generation is made easier. The sympathetic tone is modulated through descending cortical and hypothalamic projections that determine the firing frequency of preganglionic sympathetic neurons located in the intermediolateral cell column of the spinal gray matter from C8 (T1) to L1–2 levels of the spinal cord.

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

Fig. 4.29 (continued) pools of lamina IX). The projection from NRM is bilateral, whereas the projection from NGC is ipsilateral. Noradrenergic fibers descend and project to the medullary dorsal horn and then descend in the dorsolateral funiculus of the spinal cord to send terminals to laminae I, II, IV through VI, and X. (**b**) A simplistic schema to show the direct hypothalamospinal descending control system, which originates in the medial and paraventricular hypothalamic nuclei. This descending system consists of vasopressin and oxytocin neurons (and perhaps some enkephalinergic neurons), which not only send terminals predominantly to laminae I and X but also provide sparse input into laminae II and III and the lateral part of lamina V, as well as the homologous area in the medullary dorsal horn. (**c**) Direct PAG-spinal projection system, which bypasses the medullary nuclei and projects directly to the medullary dorsal horn and then descends in the dorsolateral funiculus to send terminals to laminae I, II_o, V, and X. Most of the axons are serotonergic and noradrenergic (adapted from Terman GW, Bonica JJ. Spinal mechanisms and their modulation. In: Loeser JD, Butler SH, Chapman CR, Turk DC, editors. Bonica's management of pain. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 73–152)

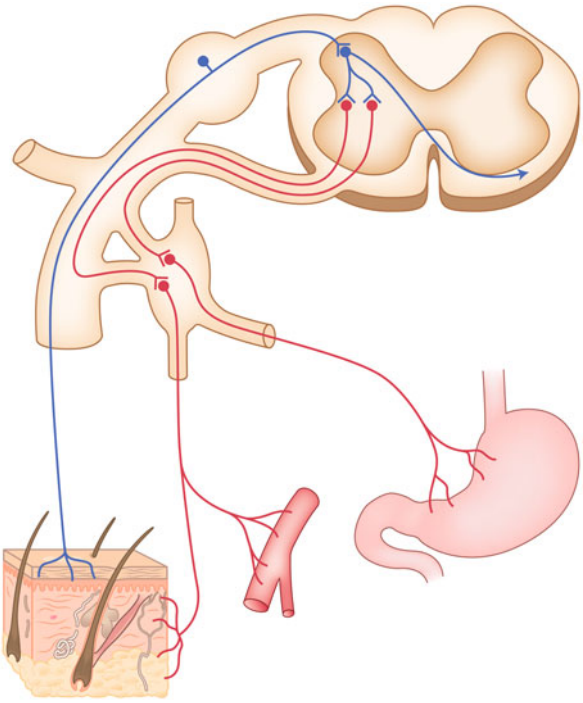

Fig. 4.30 Schematic depiction of sympathetic efferent projections in red that contribute to the "fight-or-flight" response to a nociceptive stimulus (from Gould 2007, modified from Byers MR, Bonica JJ. Peripheral pain mechanisms and nociceptor plasticity. In: Loeser JD, Butler SH, Chapman CR, Turk DC,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

Pathway Alterations Following Injury

After an injury, it is important to be aware of the area that has been injured so as not to subject it to further trauma that could exacerbate the injury. Mechanisms to enhance sensitivity in an injured region are also present in the normal nervous system, thereby aiding in recovery by increasing vigilance of the wound during the healing process. A significant portion of stimulus enhancement occurs during the process of peripheral sensitization. Peripheral sensitization is a by-product of the inflammation that is part of the mechanism of repair. It is present following injury and continues through the time that the wound has healed [13]. The process is initiated when tissue is injured by a thermal, mechanical, or chemical stimulus. Chemical mediators of inflammation are released from tissues in and around the site of injury. These chemicals increase blood flow to the injured area, carrying cells that engulf and destroy non-viable tissues and infectious agents, and increase levels of oxygen and nutrients necessary for repair. The inflammatory cells sequester particulate by-products of the cleanup and remove the by-products of metabolism. This process directly sensitizes the local nociceptors at the site of injury and, through the release of neurochemicals from collateral free nerve endings, indirectly sensitizes free nerve

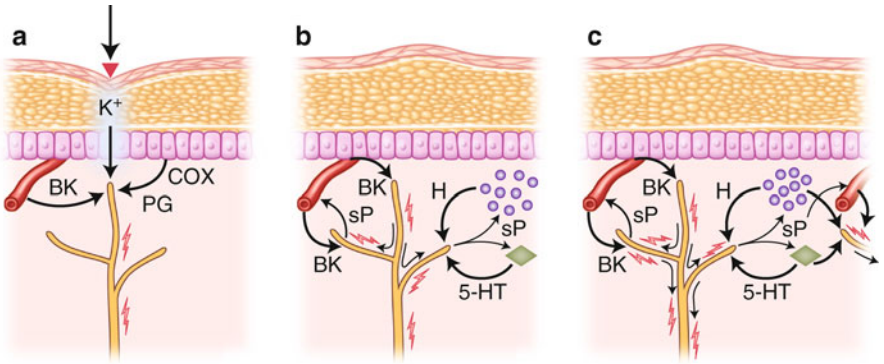

Fig. 4.31 Inflammatory enhancement of the pain signal. (**a**) Shows that noxious stimulation (*arrow*) results in the local release of protons (K^+) and inflammatory chemicals, bradykinin (BK) and prostaglandins (PG). A nociceptive signal is initiated and transmitted to the spinal cord. (**b**) Illustrates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

endings in adjacent tissues (Fig. 4.31). Consequently, the threshold for peripheral nociceptors is lowered, which increases the likelihood that a warning signal will be generated in a primary afferent nerve cell.

Abnormal sites for generation of a nociceptive signal that lead to repetitive firing and spontaneously generated pain can also develop when nerves are injured [7]. The mechanisms of injury vary, resulting in unique alterations in the normal function and integrity of the nerve. The alterations can include the destruction or damage to the neuronal cell bodies, the axons with their central and/or peripheral processes, the specialized endings in the peripheral tissues, and the supportive glial and Schwann cell elements as a result of toxic, metabolic, infectious, traumatic, and congenital processes involving either the central or the peripheral nervous system. Such changes potentially lead to alterations in the numbers and relative densities of ion channels responsible for cellular excitability. In a significant portion of the population, neuronal injury results in such changes that make it possible for impulses to be generated at abnormal sites along the course of an axon rather than just at the generator zone of nerve terminals and at synapses (Fig. 4.32). Because of altered numbers, types, and distribution of ion channels, spontaneous channel openings allow the entry of sufficient sodium ions into the axon to depolarize the membrane [7, 14]. In the periphery, the wave of depolarization proceeds away from the active site both toward the spinal cord and toward the body surface [15]. When a nerve impulse reaches the free endings of the afferent nerve terminal, neurochemical

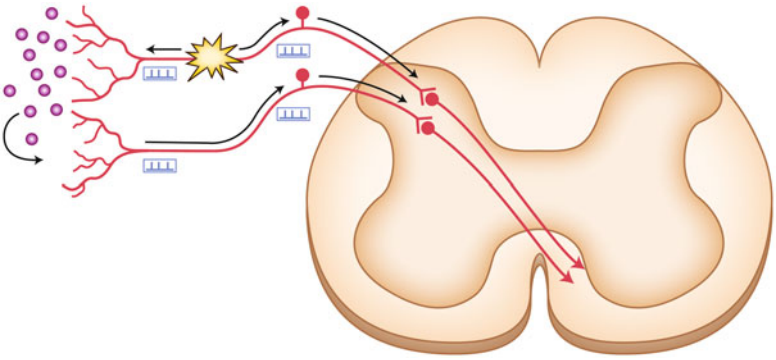

Fig. 4.32 Ectopic firing of injured nerve cells and peripheral sensitization. When peripheral nerves are injured, nerve impulses can be generated spontaneously at abnormal sites along the axon. An impulse is transmitted to the spinal cord and brain in the normal fashion but, in addition, is transmitted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

mediators are released from the terminals as described earlier, resulting in peripheral sensitization of the adjacent nerve terminals and the generation of nerve signals as a result of either noxious or non-noxious stimuli. The signals then project centrally; reach the spinal cord, thalamus, and cortex; and are perceived as pain in the region of the body served by the aberrantly firing nerve. The resulting perception of pain can thus occur in the absence of a noxious stimulus being delivered to the body at the time of perception.

Enhanced peripheral activity associated with tissue injury, especially in individuals susceptible to developing neuropathic pain related to nerve injury, potentially lays the foundation for the development of persistent or permanent pain states. The regular and frequent signals are passed to the central nervous system, and through a process of central sensitization called "windup," the repetitive firing of peripheral C fibers produces a gradual increase in the perception of a stimulus irrespective of an increase in stimulus intensity [16]. This phenomenon effectively increases the likelihood that a stimulus will be relayed to levels of cognitive perception through a sensitization of relay neurons in the dorsal horn of the spinal cord. If the process of central sensitization is allowed to persist, high levels of glutamate remain in the synaptic cleft (Fig. 4.33). When glutamate concentration remains high due to repetitive firing of primary afferent neurons, the depolarized postsynaptic membrane in the presence of increased levels of glycine, released from local inhibitory interneurons, stimulates the opening of N-methyl-D-aspartate (NMDA) glutamate receptors. The activated receptor allows entry of calcium as well as sodium into the postsynaptic cell. Other voltage-gated calcium channels present in the relay cell membrane also are activated and allow the entry of additional calcium into the relay

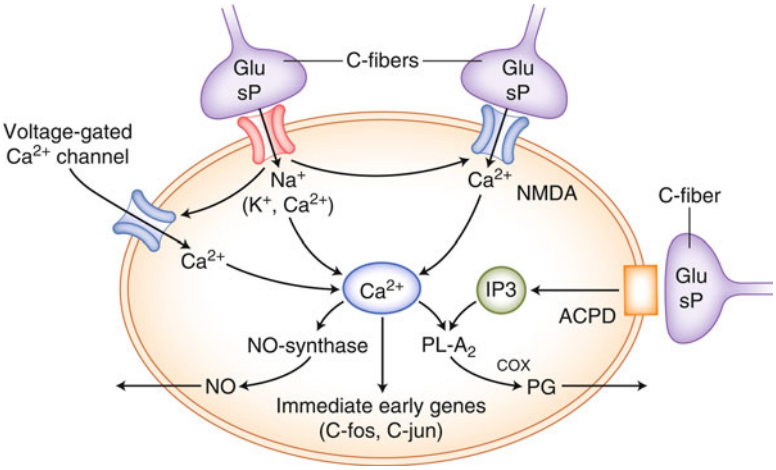

Fig. 4.33 Repetitive stimulation results in the activation of NMDA glutaminergic receptors and voltage-gated calcium channels. The entry of excess calcium stimulates the synthesis of nitric oxide and prostaglandins that are released from the neuron, resulting in sensitization of neighboring relay neurons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

neurons. Excess levels of intracellular calcium enhance the production of nitric oxide and prostaglandins that are released into the local neuropil. These mediators decrease the firing threshold of adjacent relay neurons, making it possible for them to reach firing threshold upon receiving an input generated by any level of stimulation (Fig. 4.34). In addition, neuromodulating chemicals that are released from hyperactive relay neurons can affect additional transmitter release from the primary afferent neuron and affect the release of cytokines, neurotransmitters, and trophic agents from local microglia and astrocytes (Fig. 4.35) [10]. The resulting cascade of events enhances the likelihood that both noxious and non-noxious stimuli will be sufficient to initiate transmission of a nociceptive signal to higher levels of the nervous system. Finally, continued high levels of intracellular calcium may initiate the process of protein synthesis, providing a basis for generating new and permanent neuronal connections and establish the basic framework for permanent hypersensitivity or centrally generated pain [17].

Clearly, the processing of painful signals within the nervous system is complex and involves many components that function sequentially and simultaneously to enhance survival of the individual. The system provides many fail-safe assurances to ensure the integrity of the warning system to protect against serious injury, yet these assurances provide problems and frustration in achieving complete or even adequate pain relief. To achieve the best possible treatment of pain, all components must be considered as possible sources for pain generation and possible avenues for

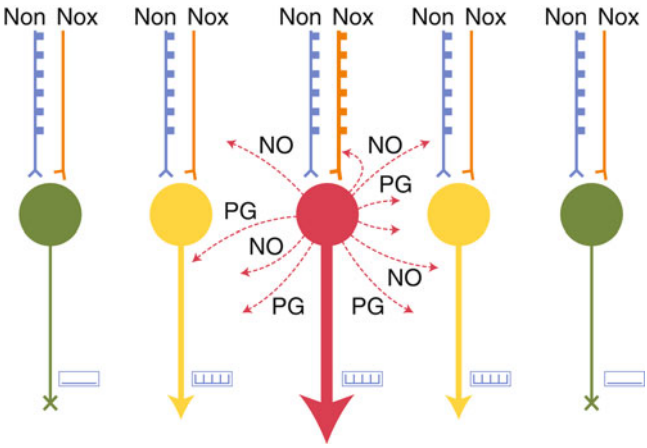

Fig. 4.34 Central sensitization enhances the transmission of a nociceptive signal. When nociceptive signals (Nox) repeatedly cause relay neurons to fire (*arrowhead*), prostaglandins (PG) and nitric oxide (NO) are released from the relay neuron (*red*), as illustrated in Fig. 4.32. PG and NO sensitize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

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

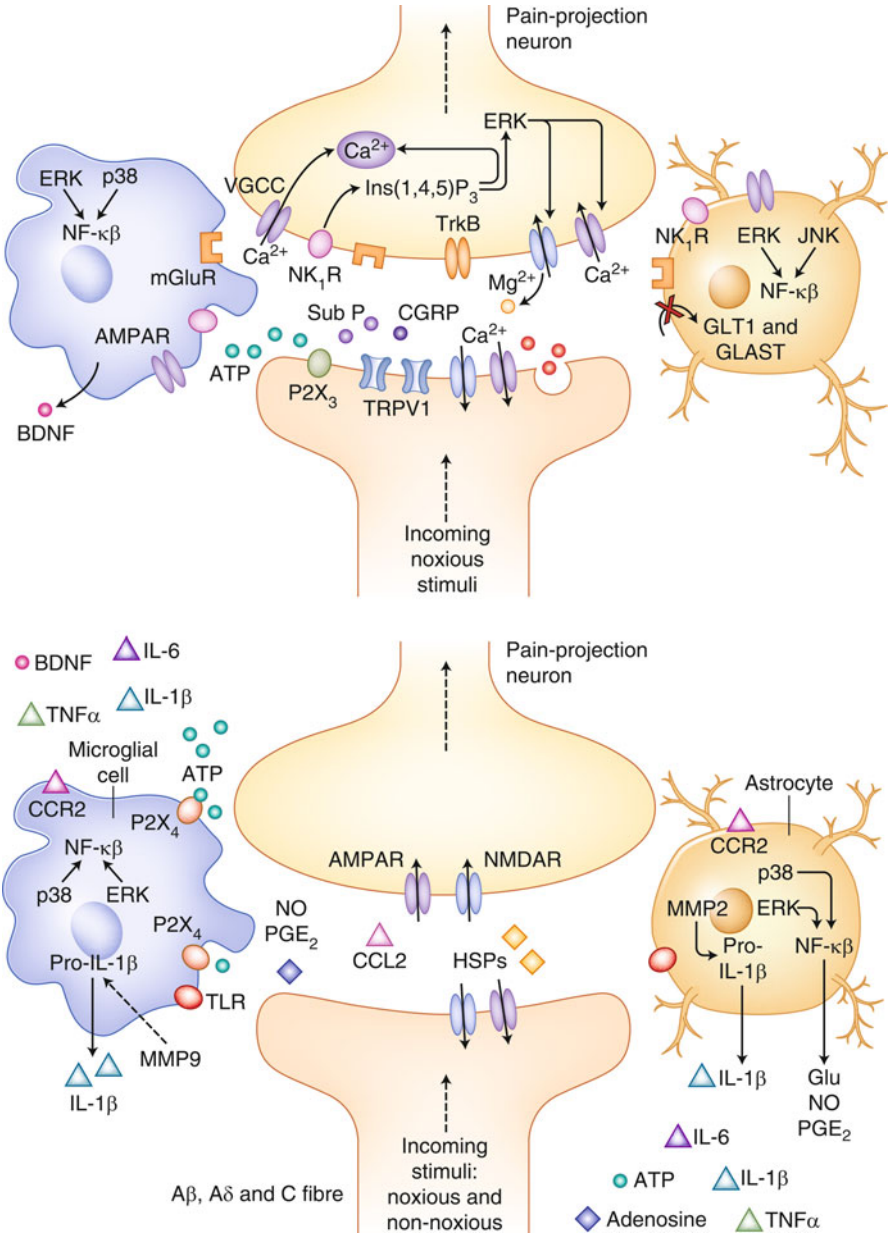

Fig. 4.35 Schematic depiction of the role of glia in processing repetitive nociceptive input and pain processing during inflammation. After repetitive synaptic communication, which can occur after a short barrage of nociceptive afferent input, there is an increase in the responsiveness of dorsal horn pain-projection neurons to subsequent stimuli (known as central sensitization). A co-release of glutamate and neurotransmitters such as substance P (sub P) and calcitonin gene-related peptide (CGRP) mediates NMDAR activation, leading to voltage-gated Ca^{2+} currents (VGCCs). In addition, inositol-1,4,5-triphosphate ($Ins(1,4,5,)P_3$) signaling and mitogen-activated protein kinases,

pain control. Knowledge of the anatomical and physiological basis for nociceptive processing and an understanding of the most likely sites where damage and intervention can occur is essential for providing optimum care for your patients.

Multiple-Choice Questions

1. Inhibitory interneurons within the dorsal horn release inhibitory neurotransmitters such as:
   (a) Glycine and gamma (γ)-aminobutyric acid (GABA)
   (b) Glutamate and aspartate
   (c) Calcitonin gene-related peptide (CGRP), galanin, and substance P (sP)
   (d) Neurokinin, vasoactive intestinal peptide (VIP), and neuropeptide Y (NP-Y)

2. There are two components of the lateral spinothalamic pathway:
   (a) Neospinothalamic tract and paleospinothalamic tract
   (b) Subthalamic tract and cerebellar vermis tract
   (c) Anterior and posterior longitudinal tract
   (d) Neocerebellar and tuberculum tract

3. When glutamate concentration remains high due to repetitive firing of primary afferent neurons, the depolarized postsynaptic membrane in the presence of increased levels of glycine, released from local inhibitory interneurons, stimulates the opening of:
   (a) Serotonin receptors
   (b) Bradykinin receptors
   (c) Muscarinic receptors
   (d) N-Methyl-D-aspartate (NMDA) glutamate receptors

4. Inputs to the wide dynamic range neurons provide the essential segmental framework for the "gate control theory" proposed by:
   (a) Melzack and Wall (1965)
   (b) Racz and Raj (1971)
   (c) Bonica (1958)
   (d) Lema (1986)

5. The gate control theory:
   (a) Is completely false.
   (b) States that impulses transmitted by low-threshold mechanoreceptors can reduce the nociceptive signal that is relayed to higher integrative levels for conscious perception.
   (c) Explains the mechanism of the gamma reflex loop.
   (d) Is the basis of our understanding of saltatory conduction.

6. The regular and frequent signals which can be passed to the central nervous system and through a process of central sensitization are called:
   (a) "Windup"
   (b) Diffusion
   (c) Archicerebellum redundancy
   (d) Schmidt–Lanterman syndrome

7. The consequences of "windup" include:
   (a) Quicker reflexes.
   (b) Increased micturition and defecation.
   (c) The repetitive firing of peripheral C fibers which produces a gradual increase in the perception of a stimulus irrespective of an increase in stimulus intensity.
   (d) The sequential discharge of β fibers which produces γ-mediated pain.

8. Unique structures, which are depolarized by stimuli in response to tissue damage:
   (a) Touch receptors
   (b) Nociceptors
   (c) Temperature receptors
   (d) Chloride channels

9. As the axons approach the spinal cord, they diverge from the main nerve trunk and enter the dorsal root where they course by their cell bodies in the DRG and enter the spinal cord to terminate on neurons in:
   (a) Rexed laminae I and II
   (b) Rexed laminae III and V
   (c) Rexed laminae X
   (d) All of the above

10. The axons of the C fiber system:
    (a) Are unmyelinated.
    (b) Are myelinated.
    (c) Are never found in the peripheral nerves of the somatic sensory system.
    (d) Have fast conduction velocity of over 20 m/s.

11. Neurons in the ventral posterior nucleus of the thalamus (VPN) relay the nociceptive signal to:
    (a) The primary somatosensory cortex
    (b) The secondary somatosensory cortex
    (c) The inferotemporal and frontal cortices
    (d) All of the above

12. After an injury, a significant portion of stimulus enhancement can occur during the process of peripheral sensitization and is limited to injury by:
    (a) Thermal stimulus
    (b) Mechanical stimulus
    (c) Chemical stimulus
    (d) All of the above

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

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

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

Answers:

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

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5

Practical Pharmacology in Regional Anesthesia

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Contents

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Introduction

Local anesthetics are the pharmacologic cornerstone of regional anesthesia producing reversible and complete blockade of neuronal transmission when applied near the axons. Their application results in complete interruption of nerve impulse conduction, allowing abolition of sensation from the area innervated by the corresponding nerves and leading also to motor block. A number of compounds with local anesthetic activity occur in nature such as cocaine, eugenol derived from plants, tetrodotoxin derived from fish species in the family *Teraodontiformes*, and saxitoxin derived from algae (*dinoflagellates*). The first reported medicinal use of a drug as a local anesthetic occurred in 1884 when Carl Koller used cocaine to anesthetize the eye by topical application.

This chapter describes the basic chemical structure of local anesthetics, the basic receptor pharmacology, and gives an overview over pharmacologic properties of the different drugs. Clinical use, advantages, and side effects are compared. Finally, some clinical pearls are highlighted, and local anesthetic toxicity is described.

Local Anesthetics

Chemical Structure

Local anesthetic molecules are comprised of three basic building blocks: a hydrophobic aromatic ring, a hydrophilic tertiary amine, and an intermediate chain connecting the two. Hydrocarbon chain length varies between 6 and 9 Å. The chemical connection between the intermediate chain and the aromatic ring divides local anesthetics in "esters" and "amides" depending on whether the hydrocarbon chain is joined to the benzene-derived moiety by an ester or an amide linkage (Fig. 5.1). The type of linkage is important as it determines how local anesthetics are metabolized. Moreover, this chemical differentiation is clinically relevant because the amides are more stable and have less risk of allergic reaction than the esters (Table 5.1).

Site of Action and Nerve Conduction

Sodium Channel Structure

The human sodium channel is a transmembrane protein composed of three subunits forming a voltage-sensitive and sodium-selective channel [1] (Fig. 5.2). Different isoforms are expressed in different tissues (muscle, heart, central nervous system, peripheral nervous system, etc.) [2]. Mutations with different sensitivity to local anesthetics are possible and have been shown in the experimental but not (yet) in clinical setting [3].

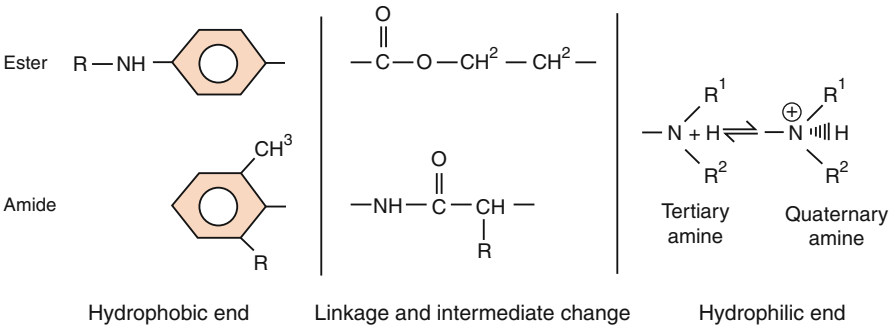
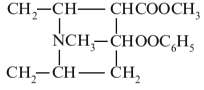
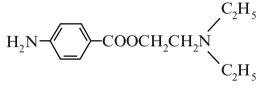
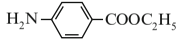
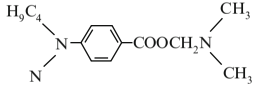
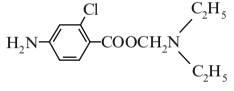
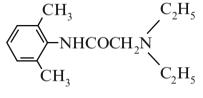
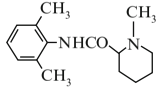
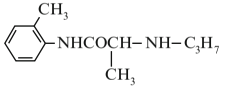
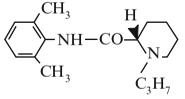
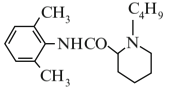
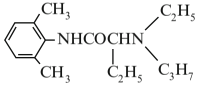


Fig. 5.1 Typical structure of local ester and amide anesthetic molecules: a practical approach to regional anesthesia. 4th ed; Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13: 978-0-7817-6854-2. p 2

Table 5.1 Physiochemical properties of local anesthetics: a practical approach to regional anesthesia, 4th ed. Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13:978-0-7817-6854-2. p 3

Drug (brand name)	Type (year introduced)	Chemical structure	Relative in vitro potency			
			Rat sciatic nerve	pK_a	Partition coefficient[a]	Plasma protein binding
Cocaine	Ester		–	8.6	–	92
Procaine (Novocaine)	Ester (1905)		1	8.9	1.7	5.8
Benzocaine	Ester (1900)		–	3.5	81	–
Tetracaine (Pontocaine)	Ester (1930)		8	8.5	221	75.6
2-Chloroprocaine (Nesacaine)	Ester (1952)		1	8.7	9.0	NA
Lidocaine (Xylocaine)	Amide (1944)		2	7.72	2.4	64.3
Mepivacaine (Carbocaine, Polocaine)	Amide (1957)		2	7.6	21	77.5

Prilocaine (Citanest)	Amide (1960)		2	7.7	25	55
Ropivacine (Naropin) Amide (1995)	Amide (1995)		4	8.1	115	95
Bupivacaine (Marcaine, Amide (1963) Sensorcaine) Levobupivacaine (Chirocaine)	Amide (1963)		8	8.1	346	95.6
Etidocaine (Durnest)	Amide (1972)		8	7.74	800	94

[a] Otanol: buffer pH 7.4

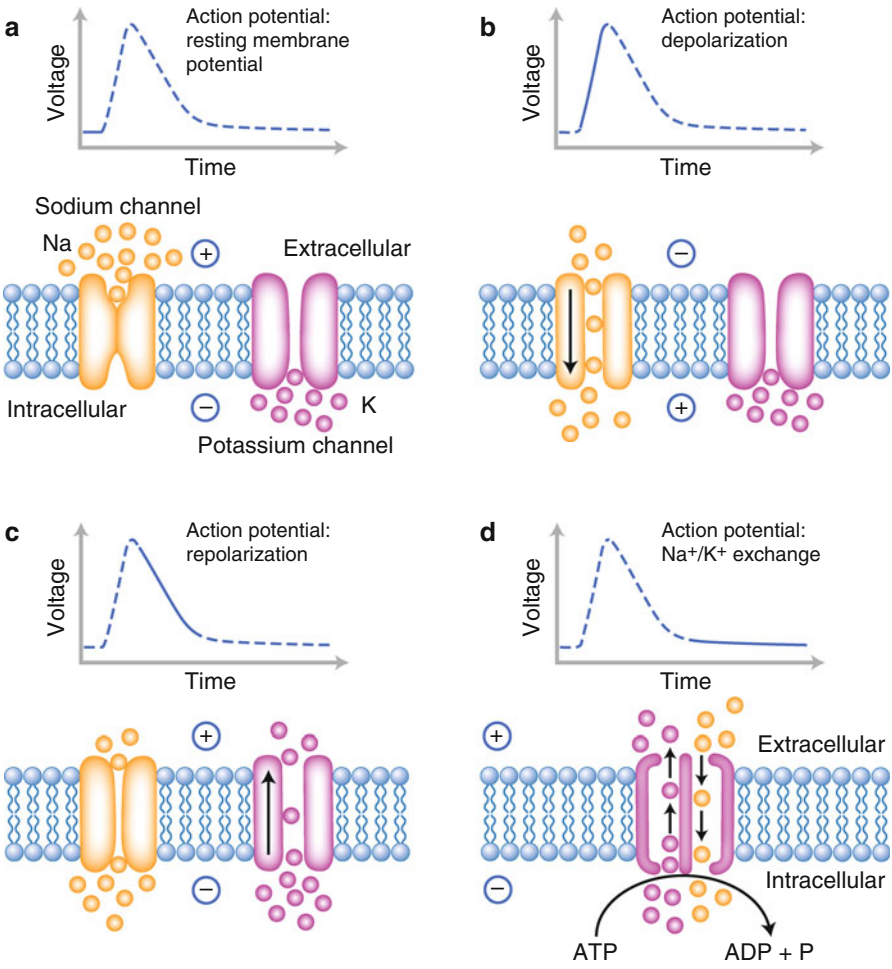


Fig. 5.2 Sodium and potassium channel function and ion movements during nerve depolarization: a practical approach to regional anesthesia. 4th ed; Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13: 978-0-7817-6854-2. p 6

Conduction

With electrical excitation of the neuron, a depolarizing stimulus is conducted down an axon. A stimulus of significant magnitude changes the negative resting potential from −70 mV toward −55 mV, the threshold required for complete depolarization: sodium channels in the cell membrane are activated and open permitting Na^+ ions to

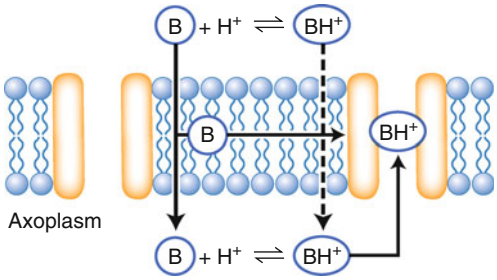


Fig. 5.3 Model of local anesthetic interaction with the sodium channel. A practical approach to regional anesthesia. 4th ed; Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13: 978-0-7817-6854-2. p 7

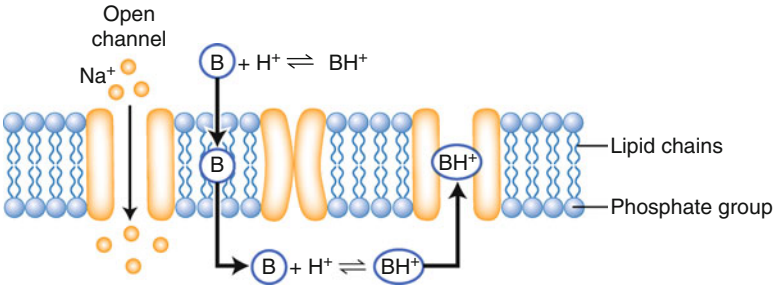


Fig. 5.4 Mechanism of action of local anesthetics. Regional anesthesia. The requisites in anaesthesiology. 1st ed. Rathmell Japmes P, Elsevier Mosby 2004, Philadelphia; ISBN 0-323-02042-9. p.17

move down their electrochemical gradient intracellularly and locally "depolarize" the axonal membrane. This influx of cations rapidly changes the membrane potential to +35 mV. The resultant propagation of voltage change down the axon is defined as the action potential. Local anesthetic molecules traverse the cell membrane and then block the sodium channel from within the cell (Fig. 5.3) blocking propagation of the action along the nerve.

Repolarization

The sodium channel is inactivated after a few milliseconds by a time-dependent change in conformation closing an inactivation gate (Fig. 5.4). The inactivated state cannot conduct Na^+ and is not reopened if further stimulated (refractory period). Thereafter, the Na^+ channel changes further to the closed (resting) state. In this state, it cannot conduct Na^+ ions, but with a sufficiently strong stimulus, will convert the channel to the open state.

Binding of Local Anesthetics

Local anesthetics do not bind to a classical "receptor"; it is more a "binding" site which is located within the sodium channel near its intracellular opening [3]. It is, on the one hand, a hydrophobic region to which the hydrophobic part of the local anesthetic molecule "binds," on the other hand, a hydrophilic region with which the quaternary amine interacts. Any change in amino acid sequence can prevent local anesthetics from being effective.

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

Pharmacodynamics and Physiochemical Properties of Local Anesthetics

Potency

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

Phasic Block

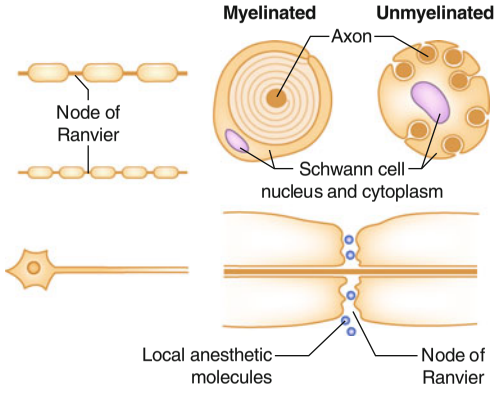
The faster a nerve is stimulated, the lower the concentration of local anesthetic is needed to produce a blockade (in vitro). This observation is called phasic block or rate-dependent block. Typically, phasic block occurs with more hydrophobic (potent) local anesthetics. They show a greater difference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

Anesthetic Block in Dependency of Nerve/Axon Exposed

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

Table 5.2 Axon classification[a]. A practical approach to regional anesthesia. 4th ed. Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13:978-0-7817-6854-2. p 9

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



[a]Human axons are classified by size, presence or absence of myelin, and function, in vitro, small unmyellinated axons are most resistant to local anesthetic blockade, whereas large myelinated axons are the most sensitive. In vivo, however, the sensitivity to local anesthetic block is different for reasons that are not fully understood (See chapter clinical pharmacology of local anesthetics). "+" indicates the relative sensitivity to local anesthetic block.

- Unmyelinated axons: the concentration of local anesthetic required to block conduction of unmyelinated axons decreases with increasing length of nerve exposed to the local anesthetic.
- Myelinated axons: myelin consists of Schwann cell plasma membranes wrapped around axons. There are gaps, called nodes of Ranvier, at fixed intervals between the myelinated areas. Myelination results in much faster conduction velocities because the axonal membrane needs to be only depolarized at the node. This process is called saltatory conduction.
- Unmyelinated axons (C fibers) are in vitro the most resistant to local anesthetic blockade, followed by large (Aα, Aβ fibers) and small (B fibers) myelinated axons [4]. Intermediate-size myelinated axons (Aδ, Aγ fibers) are the easiest axons to block in vitro.

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

Acid–Base and pK_a

Local anesthetics (except benzocaine) are weak bases (pK_a = 7.6–9.0) that are commercially prepared as an acidic solution, typically at pH 4–5. The pK_a defines the pH, where half of the drug is ionized (positively charged form, conjugate acid) and half is nonionized (base). The ionized and nonionized forms have different, but important, clinical effects. The nonionized form penetrates the nerve membrane, while the ionized form binds to proteins on the intracellular side of the sodium channel (Fig. 5.5). The percentage of each form present in a solution or in the tissue depends on the pH of the solution or tissue and can be calculated from the Henderson-Hasselbalch equation:

$$\text{p}K_a = \text{pH} - \log(\text{base})/(\text{acid}).$$

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

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

Sodium bicarbonate can be added to local anesthetic solutions to raise the pH of the solution, thereby increasing the nonionized form. Other factors being similar, local anesthetics with more basic pK_a have a slower onset of blockade effect due to the lesser amount of nonionized local anesthetic molecules at physiologic pH. This relative lack of the nonionized form impairs local anesthetic movement across the cell membrane and thus delays block onset (Fig. 5.5).

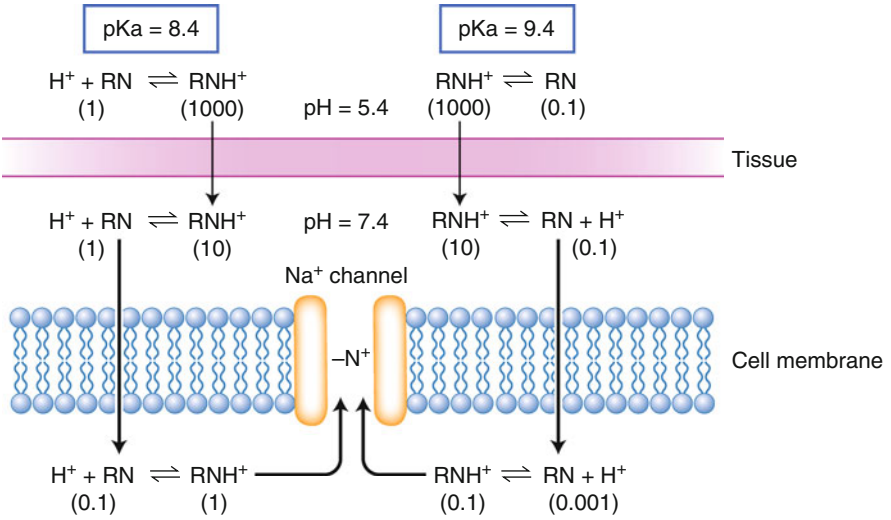


Fig. 5.5 Effect of ionization on activity. Regional anesthesia. The requisites in anaesthesiology. 1st ed. Rathmell Japmes P, Elsevier Mosby 2004, Philadelphia; ISBN 0-323-02042-9. p.18

Hydrophobicity

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

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

Protein Binding

One of the most important clinical characteristics of local anesthetics is its duration of action, which correlates with the degree of local anesthetic protein binding (typically to albumin and α-1-acid-gylcoprotein). Binding to plasma protein varies between 5 and 95%. In general, more hydrophobic drugs have higher protein binding. However, plasma protein binding do not correlate necessarily with tissue protein binding.

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

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

Metabolism

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

Summary

The comprehension of the principles described in this chapter is essential to understand local anesthetic clinical pharmacology. However, one should keep in mind that the clinical setting is much more complicated as there are multiple influencing factors not present in vitro studies.

Clinical Pharmacology of Local Anesthetics

Factors Determining Block Quality

Block Onset

The proximity of the injected local anesthetic to the nerve is the most important factor determining block onset; the nearer to the nerve, the shorter the time required to diffuse into the nerve (Fig. 5.6).

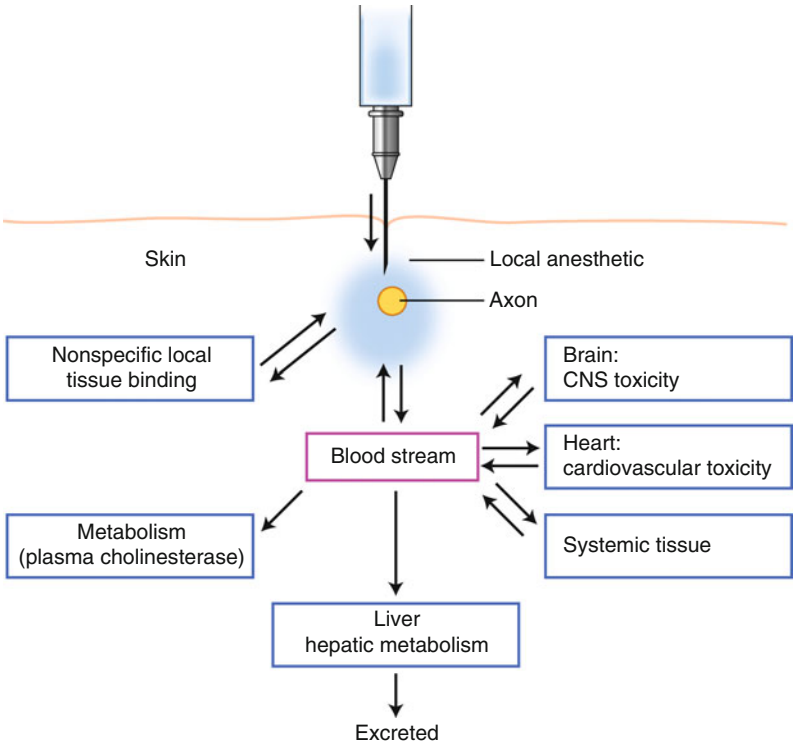


Fig. 5.6 Disposition of sites for local anesthetics following peripheral nerve blocks. A practical approach to regional anesthesia, 4th ed. Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13: 978-0-7817-6854-2. p 12

The total local anesthetic dose and not the volume or concentration determines the onset time, the duration, and the intensity of the nerve block [5].

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

Block Duration

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

The choice of local anesthetic greatly influences block duration; hydrophobic local anesthetics have a slower clearance compared to hydrophilic local anesthetics. Moreover, hydrophobic compounds have a higher potency. These two factors are

responsible for a longer-lasting block. Furthermore, local anesthetics show variable vascular effects on local blood vessels. Vasoconstriction will reduce clearance, impairing its transport from the injection site. High concentrations of local anesthetics lead to a vasodilation increasing local blood flow and consequently their own clearance. But with decreasing concentration, vasoconstriction is present reducing clearance and increasing the duration of the block. Individual differences are listed below.

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

Block Potency

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

Individual Local Anesthetics

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

Ester Local Anesthetics

Cocaine

Topical mucous membrane applications of cocaine (4% solution) result in very rapid anesthesia and vasoconstriction. At excessive doses, vasoconstrictive properties lead to hypertension, coronary ischemia, and arrhythmias. Mixtures of lidocaine with phenylephrine or oxymetazoline are safer alternatives to cocaine for anesthetizing and vasoconstricting mucous membranes. Attention must be paid not to mix cocaine with other vasoconstrictors (phenylephrine) because of the increased risk of acute myocardial infarction [6].

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

Table 5.3 Local anesthetic drug clinical doses. A practical approach to regional anesthesia. 4th ed. Mulroy Michael F; Wolters Kluwer/Lippincott Williams & Wilkins 2009, Philadelphia; ISBN-13:978-0-7817-6854-2. p 17

	Epidural[f]						Maximum recommended doses			
							Plain		With epinephrine	
Drug {brand name}	Topical[f] (%)	Spinal[f] (%)	Surgical[f] (%)	Obstetric[f] (%)	Peripheral nerve block (%)	Intravenous regional (%)	Total	mg/kg	Total	mg/kg
Cocaine	4	NA	NA	–	NA	NA	200	1.5	–	–
Benzocaine	5–20	NA	NA	–	**NA**	NA	—	—	—	—
Short duration										
Procaine (Novocaine)	NA	10	NI	NI	1	NI	500	—	—	—
2–Chloroprocaine (nesacaine)	NI	NA	2–3	2–3	1–2	NI	800	11	1,000	14
Intermediate duration										
Lidocaine (Xylocaine)	4	5	1.5 2	1.5 2[a]	0.5 1	0.5	300	4.5	500	7
Mepivacaine (Carbocaine, Polocaine)	NA	NA	1 1.5 2	NI	1	NA	400	—	550	*e*
Prillocatine (Citanest)	NA	NA	2–3	NI	1	0.5	—	—	500	—
Long duration										
Ropivacaine (Naropin)	NA	0.5[b]	0.75, 1[c]	0.2	0.5	NA	250	—	250	3
Bupivacaine (Marcaine, Sensorcaine)	NA	0.5 0.5	0.5 0.5	0.125[e] 0.125[e]	0.25 0.25 0.5[a]	0.25[b] 0.5	175	—	225	3

(continued)

Table 5.3 (continued)

Drug {brand name}	Epidural[f]						Maximum recommended doses			
							Plain		With epinephrine	
	Topical[f] (%)	Spinal[f] (%)	Surgical[f] (%)	Obstetric[f] (%)	Peripheral nerve block (%)	Intravenous regional (%)	Total	mg/kg	Total	mg/kg
Levobuplvacaine (Chirocaine)										
Etidocaine (Duranest)	NA	NA	1 1.5	NI	1	NI	300	4	400	6
Tetracaine (Pontocaine)	1–2	1	NA	NA	NA	NA				

Drugs are grouped in general duration of action. Concentrations listed are those recommended for particular application
NA not available, *NI* not indicated, *PDR* Physicians' Desk Reference
[a]Produces motor blockade suitable for cesarean delivery
[b]Not approved for this use
[c]For single injection only; lower concentrations should be used for follow-up injections of catheters
[d]Not prepared commercially; must be diluted at time of use
[e]Specific dose for epinep hrine-containing solution not identified; this is largest described dose
[f]Preservative free solutions only

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

Procaine

Procaine was the first synthetic local anesthetic used clinically. Unfortunately, procaine combines a short duration and limited tissue penetration. Procaine is still occasionally used for skin infiltration (0.25–1.0%) and short duration (30–45 min) spinal anesthesia (50–100 mg), although discharge readiness may be slightly longer than that seen with equipotent doses of spinal lidocaine. The block after spinal anesthesia is shorter compared to the block induced by lidocaine but has a higher failure rate (inadequate sensory block). On the other hand, less transient neurologic symptoms (TNS) have been reported [7]. Procaine is ineffective when used topically and is not reliable for epidural anesthesia. It is not recommended for peripheral block since it has a very slow onset time paired with a short-acting time. Procaine is metabolized in the plasma by the cholinesterase; its elimination half-life is approximately 8 min.

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

2-Chloroprocaine

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

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

Tetracaine

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

mucosal application. Metabolism is slower compared to procaine; therefore, the risk of systemic toxicity is greater.

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

Benzocaine

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

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

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

Amide Local Anesthetics

Lidocaine

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

Lidocaine 5% has been implicated in the occurrence of cauda equina syndrome with the use of small-diameter microcatheters for continuous spinal anesthesia. Spinal microcatheters have since then been withdrawn from the US market. Single-shot spinal anesthesia can be associated with TNS, the etiology of which is uncertain [8, 9].

Mepivacaine

Mepivacaine has similar pharmacokinetic profile to lidocaine, with slightly longer duration and better tissue penetration. Chemically, it is a cyclic tertiary amine like bupivacaine and ropivacaine. It is used primarily for intermediate-duration infiltration, peripheral, epidural, and spinal nerve blocks in Europe. It has a mild vasoconstricting effect which may be responsible for its longer duration compared to

lidocaine. Mepivacaine is not used anymore in obstetric epidural anesthesia since this drug is poorly metabolized in the fetus and neonate and may be responsible for lower neurobehavioral score in the first days of life [10].

Prilocaine

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

Etidocaine

Etidocaine is a derivate of lidocaine. Different chemical changes in the structure make etidocaine very hydrophilic. It is available in the USA as 1, 1.5, or 2% solutions. Thus, it is rarely used in contemporary practice. Its onset is similar to lidocaine, but its high protein binding is similar to bupivacaine, as are its duration of action and cardiac toxicity profile. Clinical potency is similar to that of mepivacaine with 2.5% solutions commonly used in the epidural space and 1% solutions for the performance of peripheral nerve blocks.

Articaine

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

Bupivacaine

Bupivacaine was the first long-acting amide local anesthetic. Chemical structure makes bupivacaine significantly more hydrophobic than mepivacaine and lidocaine, slower in onset but of longer duration. Bupivacaine is highly protein bound, which is consistent with long duration and potential for cardiotoxicity. Indeed, the cardiotoxicity of bupivacaine prompted the development of ropivacaine and L-bupivacaine.

Bupivacaine is popular for use in a wide array of applications, including infiltration (0.25%), peripheral nerve blocks (0.375–0.5%), spinal (0.5 and 0.75%), and epidural (0.5 and 0.75%) anesthesia. Because of systemic toxicity, it is not used for IV regional anesthesia.

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

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

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

Levobupivacaine

Levobupivacaine is the levorotatory enantiomer of bupivacaine. Commercial bupivacaine is a racemic mixture of both enantiomers (R and S). Levobupivacaine is approximately equivalent to its racemic mixture for its use in regional anesthesia. Cardiac toxicity and CNS studies in animals and healthy volunteers indicated that levobupivacaine is approximately 35% less cardiotoxic compared to racemic bupivacaine [11, 12]. Levobupivacaine is used in the same concentrations, doses, and applications as racemic bupivacaine.

Ropivacaine

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

This drug was specifically designed and formulated to minimize cardiotoxicity [13, 14]. At higher concentration (anesthetic), its potency is equivalent to that of bupivacaine [15]. At lower concentration (analgesic), ropivacaine was shown to be 40% less potent than bupivacaine [16]. The clinical experience for peripheral blocks shows that at equivalent doses ropivacaine and bupivacaine produce similar onset and quality of block, but it can be stated that bupivacaine has a significantly longer duration. Ropivacaine is primarily used in epidural anesthesia/analgesia and peripheral nerve block applications. Ropivacaine appears to be approximately 40% less cardiotoxic as compared to racemic bupivacaine in animal models [13]. Ropivacaine produces vasoconstriction at clinically used concentrations for peripheral nerve blocks explaining the little advantage of adding epinephrine to additionally prolong peripheral nerve block or epidural analgesia [17].

Adjuvants

Sodium Bicarbonate

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

Hyaluronidase

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

Vasoconstrictors

Adding epinephrine leads to vasoconstriction and thereby local blood flow and drug clearance are decreased. This prolongs block duration and decreases local anesthetic plasma concentration following spinal, epidural, and peripheral nerve blocks [18]. Lower peak plasma concentration decreases the risk for toxicity. However, epinephrine does not provide protection if accidental intravascular local anesthetic injection occurs [19].

Clonidine

Alpha-2-adrenergic agonists are analgesic drugs in their own right and have been shown to inhibit both C fibers and A fibers and to modestly inhibit local anesthetic clearance [20, 21]. When added to local anesthetics, clonidine prolongs sensory block during peripheral, central neuraxial, and intravenous regional anesthesia to a degree comparable to that produced by epinephrine. However, unlike epinephrine, clonidine does not prolong motor block when administered orally, as when added to the intrathecal local anesthetic [22].

Opioids

When added to short-duration local anesthetics used for spinal anesthesia, short-acting opioids (fentanyl and sufentanil) prolong and intensify sensory block without prolonging motor block or time to void, which is particularly advantageous for

ambulatory spinal anesthesia [23]. However, postanesthesia nausea and vomiting, itching can be a problem [24]. When added to local anesthetics or peripheral nerve block, fentanyl has also been shown to prolong sensory block, but at the expense for significantly slowing onset in some studies [25].

When added to intrathecal local anesthetics, the peak plasma concentrations for sufentanil occur between 20 and 30 min and are greater than what is necessary for postoperative analgesia [14]. This explains the many reports of "early" respiratory depression in mothers [15] and fetal heart rate abnormalities in infants when sufentanil is added to intrathecal local anesthetics for labor analgesia or cesarean section [26].

Depot Local Anesthetic Preparations

Depot preparations of local anesthetics are interesting because they would allow using long-acting anesthetics without the need for catheters and pumps.

Gels, polymer microspheres, liposomes, and oil-water emulsions have been studied in animal models to produce long-acting anesthetic blocks [27]. To date, clinical convincing results are still lacking.

Complications of Regional Anesthesia

Introduction

Overall incidence of neuropathy after peripheral nerve block varies from 0 to >5%. Studies which used closed claims databases ranked neuropathy at the second place, with 16% of all claims [28]. In a prospective French study, incidence of major neurologic adverse reactions was estimated at 3.5/10,000 [29]. Peripheral nerve damages following either spinal anesthesia or peripheral nerve blockades represented >50% of severe adverse reactions in this investigation.

Permanent injuries after regional anesthesia are rare [30–32]. Most surveys with large cohorts are retrospective [33, 34] or related to closed claims analysis [35, 36]. Few studies are prospective but focus on specific adverse reactions inducing limitation in their interpretation [29, 37, 38].

The largest recent clinical study was a voluntary reporting model used in France [29]. Data of 158,083 different blocks from 487 anesthesiologists were collected and analyzed. The incidence of serious complications such as central or peripheral nerve injury, seizure, death, etc. was described as 3.5/10,000 blocks. The risk of deaths was shown to be 1/400,000 regional blocks. All but one occurred during spinal anesthesia.

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

Systemic Toxicity

Systemic toxicity is a significant and potentially dangerous problem [39]. Beside a local toxicity, an increase of the local anesthetic plasma concentration may lead to systemic toxicity, mainly neurologic and cardiovascular ones. Such an increase in local anesthetic plasmatic concentration may be related to inadvertent intravascular injection with a consecutive sudden plasmatic peak of concentration. The most frequent cause of systemic toxicity is related to a high and rapid resorption of local anesthetics through perinervous vessels. Toxicity occurs first in the CNS and then in the cardiovascular system (Fig. 5.7).

CNS Toxicity

The incidence of seizures varies between 0.2 and 1/1,000 cases and according to the anesthetic regional procedure [40, 41]. The clinical manifestation largely depends on the velocity of plasma concentration increment: a slow increase shows clear and reproducible series of typical CNS signs and symptoms. A rapid increase leads to generalized seizures as first clinical manifestation.

Sedatives and hypnotics such as propofol, benzodiazepines, and barbiturates raise seizure threshold and help protecting the CNS [42, 43].

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

The prevention and the treatment of CNS toxicity should be done according to published recommendations [44, 45].

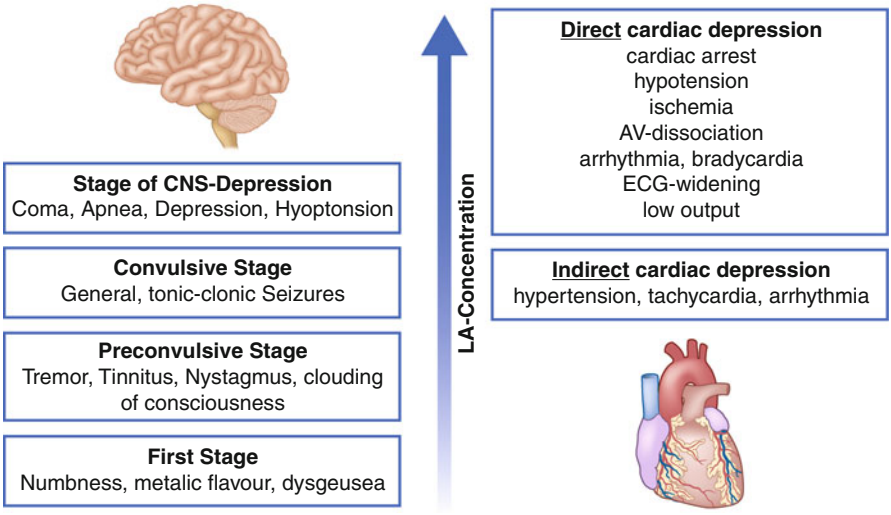


Fig. 5.7 Signs and symptoms of local anesthetics toxicity

Table 5.4 Classification of nerve injuries

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

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

Cardiac Toxicity

Estimated incidence of cardiac arrest related to local anesthetics varies between 1.8 and 3.1/10,000 cases [40, 46].

High plasma concentration of local anesthetics is needed to cause significant cardiovascular toxicity. This may occur, when the local anesthetic is injected intravenously, but a quick resuscitation is also possible. The therapeutic/cardiotoxic ratio is lower for hydrophobic local anesthetics (bupivacaine) compared to hydrophilic local anesthetics. Hydrophilic local anesthetics dissociate only after a greater amount of time from their binding sites; therefore, Na^+ channels are blocked when the next depolarization arrives. Cardiac toxicity can manifest as either malignant dysrhythmias (ventricular fibrillation), pulseless electrical activity, or asystolia [19, 42, 47].

Cardiac toxicity should be prevented [45], but in case of patients experiencing signs or symptoms of local anesthetic systemic toxicity (LAST), treatment should be done according to the ASRA guidelines 2010 [44, 48].

Often, the doses of epinephrine in this setting are higher [19, 42, 47, 49]. Intralipid seems to be effective mainly in case of bupivacaine toxicity. A review about models and mechanisms of local anesthetic cardiac toxicity and a review of clinical presentations of local anesthetic systemic toxicity over the last 30 years have recently been published [50, 51].

Prevention of Toxicity

Toxicity depends on total dose of local anesthetic injected, type of local anesthetic, speed and site of injection, combination with adjuncts, patient's medical history, and concomitant use of other drugs leading to dangerous interactions, particularly with drugs presenting a hepatic metabolism action (hepatic blood flow modification, cytochrome P450 action, etc.). Interactions have been described among local anesthetics

and β-blockers, amiodarone, cimetidine, and volatile agents [52–56]. Calculation of the optimal dose taking into account patient's age, pharmacokinetic and pharmacodynamic interactions with concomitant disease, and other drugs could be probably useful [57]. Development of nerve localization by ultrasonographic technique is thought to help reaching such objectives by limiting the volume of local anesthetic needed to block nerves [58]. However, clinical practice has shown that such a technique cannot always prevent intravascular injection or quick reabsorption [59].

Recently, a good summary about prevention of local anesthetic systemic toxicity (LAST) has been published in a series of articles dealing with LAST [45].

Local Tissue Toxicity

Nerve Injury/Transient Neurologic Syndrome

Direct nerve injury from local anesthetic is receiving increased scrutiny, particularly with regard to spinal anesthesia [60, 61]. Toxicity can result from either local anesthetics themselves or from additives, preservatives, antiseptics, or the pH of the formulations. The mechanism of local anesthetic-induced neurotoxicity is multifactorial [60, 62]. Direct nerve injury is evident when isolated nerves are exposed to high concentration of local anesthetics, particularly lidocaine and tetracaine. Local anesthetics also change the biologic milieu surrounding neurons, including localized alteration of prostaglandin production, altering ionic permeability and changes in neural blood flow.

Compared with bupivacaine, lidocaine has a significantly greater potential for direct neurotoxicity, particularly when isolated nerves are exposed to high concentrations of lidocaine over long periods of time. Hyperbaric 5% lidocaine and tetracaine have been associated with cauda equina syndrome after continuous spinal anesthesia. In these cases, spinal microcatheters were used to administer supernormal doses (up to 300 mg) of hyperbaric 5% lidocaine. Because spinal microcatheters (25–32 gauge) greatly limit the speed of drug administration, badly distributed local anesthetics presumably pooled near the catheter tip. As a result of the lordotic lumbar spine curvature, higher concentration of lidocaine remained in the lumbosacral cistern [62, 63].

Single-shot spinal anesthesia can cause transient pain (TNS), manifest as back and posterior leg discomfort with radicular symptoms lasting 1–3 days after spinal anesthesia. The etiology of TNS is unclear, but some have speculated that this syndrome represents a form of neurotoxicity. Transient neurologic symptoms occur more frequently with lidocaine than bupivacaine, which may relate to lidocaines greater neurotoxicity in isolated nerve preparations [36, 64–67]. Additionally, several risk factors (lidocaine, lithotomy position, out-patient status, arthroscopic knee surgery, and obesity) for developing TNS have been identified [64, 65].

Needle Trauma

Recent ultrasonographic data have shown that injections between epineurium and perineurium did not produce significant neural injury [68]. If injection pressure is low (less than 12 psi), intraneural injection does not necessarily result in permanent injury but can lead to severe injury if pressures are high [69].

Studies over the last years have demonstrated that the correlation between needle-nerve proximity and the current necessary to elicit a motor response is poor and not always reliable, despite the high success rate of neurostimulation and its low complication rate [70, 71]. Moreover, also eliciting paresthesia has surprisingly poor correlation with nerve proximity [72, 73]. Case reports of intraneural, intravasal, and other complications despite the use of ultrasound have shown that also this promising technique does not guarantee a complete visualization of the targeted nerve to avoid further complications [74]. The best way to avoid needle-induced nerve trauma is to avoid long bevel needle and perpendicular needle approaches to the nerve.

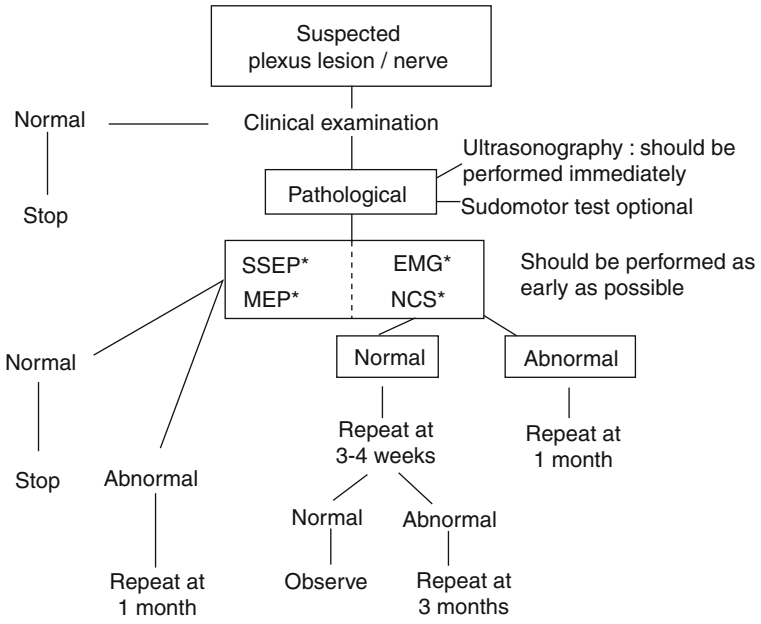
Clinical symptomatology of perimedullar complication following central nervous block is variable. Spinal cord injury can occur even while a patient did not complain of any paresthesia during puncture [75, 76]. Different risk factors have been identified to explain the occurrence of this complication [60]. Epidural hematoma can cause paraplegia following neuraxial anesthesia in patients concomitantly anticoagulated with low-molecular-weight heparin. Other causes of neural injury include positioning injuries, surgical trauma, and injuries related to the use of a limb tourniquet.

Guidelines on management of such complications following both central and peripheral nerve blocks have recently been published by the American Society of Regional Anesthesia [60]. Decision-making algorithms have been proposed to help the clinician in case of neuropathy occurrence [61, 77] (Fig. 5.8).

Myotoxicity

Skeletal muscle toxicity is a rare and uncommon side effect of local anesthetic drugs. Intramuscular injections of these agents regularly result in reversible myonecrosis [78]. The extent of muscle damage is dose dependent and worsens with serial or continuous administration. This problem is probably underestimated as incidence of symptomatic clinical forms is unknown. Experimental studies have concluded that all LA cause muscular damages with concentration use in daily practice. The extent of such damage depends on pharmacological properties of each local anesthetic, dose injected, and site of injection [79].

Animal studies in pigs showed lower mean damage score in muscles exposed to ropivacaine compared to exposure to bupivacaine [80, 81]. Stereospecificity of the drug seems also to play an important role in Ca^{2+} metabolism, which has been shown



* The choice of the examination will be done according to clinical condition and neuro physiologist 's recommendations

Fig. 5.8 Algorithm recommended to be performed in case of suspected plexus/nerve lesion

to be important in myotoxicity [82]. First reports of muscular dysfunction were related to retrobulbar injection of local anesthetics.

Bupivacaine seems to be the most toxic local anesthetic. Phenomena of apoptosis have been described only with bupivacaine but not with other LA [81, 83]. Interactions with the Ca^{2+} metabolism seem to be a key pathway and explain most damage [82, 84]. Also, changes in the mitochondrial metabolism induced by local anesthetics have been reported [83, 85, 86]. These effects are less pronounced with ropivacaine, a less lipophilic local anesthetic, compared with bupivacaine on heart cell preparation [87], but this was not shown in rat psoas muscle [88]. A recent study has concluded that mitochondrial bioenergetics alterations with bupivacaine were more severe in young rats compared to adults [89].

Chondrotoxicity

Complications from the use of pain pumps in orthopedic surgery have recently received considerable interest. Human and animal studies have reported on the chondrotoxicity of intra-articular application of bupivacaine [90–92]. Postarthroscopic glenohumeral chondrolysis is a noninfectious entity associated with factors

including use of radiofrequency tumoral instruments and intra-articular pain pumps that administer bupivacaine [93]. Also, the viability of bovine articular chondrocytes after exposure to corticosteroids alone or with lidocaine in a simulated inflammatory environment was assessed. The results showed a dose-dependent and time-dependent decrease in chondrocyte viability after exposure to methylprednisolone. The combination with lidocaine was toxic, with virtually no cells surviving the treatment [94]. Continuous 0.5% bupivacaine exposure was shown to have a clear detrimental effect on chondrocytes in an in vitro model [95]. There is a growing amount of evidence that intra-articular administration of bupivacaine is chondrotoxic, especially at a higher concentration and with a prolonged exposure. More studies are needed to clarify this issue.

Allergy

Allergic reactions may occur from preservatives added to some local anesthetics (sulfites and methylparaben). Actual allergic reactions to local anesthetics are quite rare but are more common with ester local anesthetics compared to amides [96]. This is likely due to the breakdown products of ester local anesthetics, such as PABA. There are only a few convincing reports of allergic reactions to preservative-free amide local anesthetics.

If there is a history suggestive of true allergy, it may be worthwhile to perform allergy testing to preservative-free local anesthetics. Measurement of plasma esterase, which is increased in the event of "true" allergy, is useful. Skin testing is often performed to prospectively identify patients with local anesthetic allergy [97].

Bleeding Complications

This issue deals mainly with neuraxial blocks. Epidural (1:150,000 cases) or intrathecal (1:200,000 cases) hematomas can cause devastating neurologic injury. The increased use of antithrombotic prophylaxis has increased this risk after epidural/spinal anesthesia to 1:1,000–1:10,000. The ASRA has recently reviewed the risks attendant to performance of regional blocks in the anticoagulated patient and refreshed its guidelines [98, 99] which are also to be found in their website (www.asra.com). Patients may develop sensory changes, progressive weakness and/or back pain. Confirmatory diagnosis with neuraxial imaging (CT and MRI) must be obtained in conjunction with immediate neurosurgical consultation. If more than 8 h pass between symptom onset and decompression, the likelihood of a full or partial recovery decreases dramatically.

Medicamentous Coagulopathy

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

Recently, the ASRA published new guidelines for regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy [98].

Infection

Infection is a seldom complication in regional anesthesia. Risk factors are indwelling catheters left in place for more than 5 days, immunocompromised patients, catheters in trauma patients, and lack of perioperative antibiotics [30].

Peripheral Nerve Blocks

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

Central Neuraxial Blocks

Single-shot spinal and epidural anesthesia have a low risk of infection, but this risk seems to be higher than for peripheral nerve blocks. The incidence of meningitis after spinal anesthesia is estimated at less than 1:40,000; the risk of abscess after epidural anesthesia is less than 1:10,000 (Lit 2). Risk factors are the use of indwelling catheters and bacteremia [100].

Clinical Pearls

- Nerve-blocking potency of local anesthetics increases with increasing molecular weight and lipid solubility [101].
- The effectiveness of local anesthetics is influenced by the dose, site of administration, additives, temperature, and pregnancy [101].
- The plasma [101] concentrations of local anesthetics is depending on the injection technique, place of injection, and addition of adjuvants to local anesthetics.

- In laboratory experiments, most local anesthetics will only produce cardiovascular toxicity after the blood concentration has exceeded three times that necessary to produce seizures [50].
- True allergic reactions to preservative-free amide-type local anesthetics are rare [96].
- True anaphylaxis is more common with ester local anesthetics that are metabolized directly to PABA than to amide local anesthetics [96].
- Some patients may react to preservatives, such as methylparaben, used in local anesthetics.
- In contrast to other shorter-acting amide local anesthetics, bupivacaine, levobupivacaine, and ropivacaine have a motor-sparing effect; they produce less motor block for a comparable degree of sensory analgesia.
- It is well accepted that lipid solubility usually goes hand in hand with local anesthetic potency. All things being equal, greater lipid solubility is related to increasing length of the aliphatic chain on the amino ring.
- Intraepidurally administered opioids reduce intraoperative requirements for volatile anesthetics significantly more compared to their intravenous administration. This proves site-specific action in the epidural space.
- Exceeding a total dose of 0.25 mg of epinephrine may be associated with cardiac arrhythmias.
- Adding epinephrine to spinal anesthetics will prolong motor blockade and delay the return of bladder function, thus preventing patients from achieving discharge criteria.
- When clonidine is used in combination with opiates, the analgesic effects are additive, but not synergistic. Thus, patients require a smaller total dose of narcotics and have a decreased incidence of oxygen desaturation with equivalent analgesia.
- Generally, the bigger the size of the nerve fibers, the greater the amount of local anesthetic solution required to block conduction. Thus, fibers of small size are blocked sooner than those of larger diameter.
- The B fibers of the autonomic system constitute an exception of this rule: even though they are myelinated fibers, a minimum concentration of local anesthetic solution is required to produce an effective blockade.
- This explains why the sympathetic blockade is observed before the onset of sensory or motor blockade.
- The onset time of local anesthetic is influenced by the molecules pK_a (the higher the pK_a, the slower the onset time of the nerve block in a physiologic environment) and diffusibility [101].
- The ability to cross cell membrane depends on the molecular weight and the liposolubility of the molecule.
- The nonionized form of the molecule is more lipid soluble than the ionized one; therefore, it can cross more readily the cell membrane but diffuses less easily.
- The duration of the action of local anesthetic solutions depends on the protein binding as well as the clearance from the injection site.

- The closer the pK_a of local anesthetic is to physiologic pH, the shorter the onset time of the nerve block [101].
- Increasing the lipophilicity of local anesthetic increases its potency and toxicity, whereas protein binding is proportional to the duration of action of the local anesthetic.
- Sensory-motor differentiation is based on the different size and myelinization of the nerve fibers involved in pain conduction (Aδ and C) as compared to those involved in motor function (Aα).
- Postoperative maintenance is best performed with low concentration of a long-acting agent, like 0.2% ropivacaine, 0.125–0.2% levobupivacaine.
- Local toxicity with neurotoxicity primarily occurs in cases of intraneural injection rather than normal applications of clinically relevant concentrations of local anesthetics [102].
- To decrease the risk of nerve injury, utmost care should be taken during nerve localization; excessively high concentrations of local anesthetic and high injection pressures should be avoided [102].
- The larger the fascicle, the greater is the risk of accidental intraneural injection because large fascicles are easily speared by the needle.
- Injections into epineurium or perineural tissue do not result in significant injection resistance.
- When injection is difficult (injection pressures >20 psi), the injection should be stopped because of the risk of intraneural needle position [102].
- It is suggested that nerve stimulation with current intensity of 0.2–0.5 mA (0.1 ms) indicates close needle-nerve placement [103].
- Stimulation with current intensity of ≤0.2 mA may be associated with intraneural needle placement.
- Motor response to nerve stimulation may be absent even when the needle is inserted intraneurally [68].

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6

Anticoagulation and Regional Anesthesia Concerns

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Introduction

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

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

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

Coagulation Physiology

Under normal circumstances, a tight equilibrium between clotting and bleeding is maintained. A complex interplay between activators, cofactors, inhibitors, and feedback loops exists. Hemostasis involves three simultaneous processes [4] (1) primary hemostasis, (2) secondary hemostasis, and (3) fibrinolysis.

Platelets play a critical role in primary hemostasis [4, 5]. After needle trauma, a friable platelet plug is created in order to stop bleeding. A platelet is a cell fragment, which is surrounded by a coat of glycoproteins; these glycoproteins adhere to injured endothelium. Von Willebrand factor (vWF), a protein found in subendothelial tissues, facilitates platelet adhesion. Platelets express a glycoprotein Ib receptor that binds to vWF. Upon adhesion, the platelet activates. Platelets change shape, initiate a granule release reaction, and express glycoprotein IIb/IIIa receptors. Phosphatidyl serine is flipped to the outer side of the platelet. Plasma clotting factors

interact with the activated platelet surface. Fibrinogen adheres to the GpIIb/GpIIIa receptors. The process is highly regulated, and endothelial cells prevent platelet aggregation outside the area of vascular injury.

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

Coagulation must be restricted to the area of injury; control occurs via three types of inhibitory pathways [4, 6] (1) antithrombin III, (2) thrombomodulin, and (3) tissue factor inhibitor. Antithrombin III inhibits several factors, particularly Xa. Thrombomodulin binds to thrombin and activates proteins C and S, which causes proteolysis of factors Va and VIIIa. Tissue factor pathway inhibitor blocks factor Xa via a negative feedback loop. The enzymatic degradation of fibrin is governed by the fibrinolytic system. Plasmin is a proteolytic enzyme that digests fibrin, fibrinogen, factor V, factor VIII, prothrombin, and factor XII. Plasmin is derived from plasminogen. Tissue-type plasminogen activator is a serine protease that binds to a fibrin clot and activates plasminogen. Proteolysis is confined to the clot itself.

Coagulation Pathophysiology

Coagulation pathophysiology clinically manifests as a hemorrhagic or thrombotic disorder. Hemorrhagic disorders may be hereditary or acquired disorders of hemostasis. The three most common hereditary coagulation disorders are von Willebrand's disease, Hemophilia A, and Hemophilia B [7, 8].

Von Willebrand's disease is the most common hereditary bleeding disorder, afflicting 1–3% of the general population. There may be a quantitative or qualitative impairment in von Willebrand factor [8]. Platelets lose their adhesive properties, and factor VIII levels are reduced. The condition is inherited as an autosomal dominant disorder. Patients develop bruising and mucosal bleeding. Prolonged epistaxis or menorrhagia may occur. Surgical bleeding is localized to the area of injury, and distant site bleeding is uncommon. Platelet function analyses, PFA-100, can diagnose the problem. Specialized assays, such as one that directly measures the von Willebrand factor antigen, can confirm the diagnosis [8]. Treatment involves desmopressin and factor replacement.

Hemophilia A is a bleeding disorder resulting from a defect in factor VIII:C, a cofactor involved in the activation of factor X [9, 10]. The disease primarily afflicts men since the disease is X-linked. All patients with hemophilia A have normal plasma concentrations of vWF. Life-threatening hemorrhage can occur. Spontaneous bleeding into joints, neural compartments, and intracranial structures can occur. Central nervous system hemorrhaging is associated with a 30% mortality rate. The activated partial thromboplastin time (aPTT) is abnormal. The prothrombin or bleeding times are normal. Disease severity is proportional to plasma concentrations of factor VIII. Factor replacement is essential in planning surgical procedures. Factor VIII [10] replacement can be plasma derived or recombinant [9]. Recombinant VIIa, a "universal" hemostatic agent, can be used to control surgical or trauma-associated bleeding in patients with hemophilia A. Hemophilia B is a bleeding disorder resulting from a defect in factor IX. Inheritance and clinical patterns are indistinguishable from hemophilia A. Treatment requires plasma-derived or recombinant factor IX.

Vitamin K deficiency develops with malnutrition, fat malabsorption, antibiotic usage, and liver disease [5, 11]. Microsomal carboxylase, a liver enzyme dependent on vitamin K, is necessary to convert factors II, VII, IX, and X into their functionally active forms. Vitamin K deficiency leads to a reduction in these factors and, consequently, a bleeding diathesis. Patients develop melena, hematuria, ecchymoses, and hematomas [5, 11]. Supplemental vitamin K can be given 4–8 h prior to a procedure in the at-risk patient.

Liver disease [4] can lead to thrombocytopenia, platelet dysfunction, reduced production of clotting factors, increased factor consumption, and increased fibrinolysis. A continuum of bleeding disorders may result, and all stages of liver disease increase the risk of bleeding [4, 12]. Preprocedural screening of liver disease patients may include hemoglobin, PT, aPTT, platelet count, platelet function analysis, and fibrinogen level. Management strategies include vitamin K supplementation, fresh frozen plasma, platelets, and cryoprecipitate. Vitamin K supplementation may be sufficient in patients with biliary tract disorders [12].

Renal disease causes hemostatic defects due to multiple reasons [13]. Defects in platelets, subendothelial metabolism, and platelet vessel interactions occur. Renal disease augments the effects of antiplatelet drugs and low-molecular-weight heparins. A comprehensive coagulation profile must be performed in renal failure patients. Bleeding time may be useful to assess bleeding risk. Elevations in the PT or aPTT denote the effect of other clotting problems. Coagulapathy [13] may be treated with dialysis, anemia correction, desmopressin, cryoprecipitate, estrogens, and avoidance of antiplatelet drugs.

Drugs that Impair Hemostasis

Cyclooxygenase inhibitors disrupt the formation of thromboxane A2, which disturbs vasoconstriction and secondary platelet aggregation. A primary platelet plug may still form and be adequate for small injuries; however, this will not be

sufficient for stopping surgically induced bleeding. Aspirin irreversibly inhibits cyclooxygenase for the life of platelet, whereas nonsteroidals reversibly inhibit this enzyme. Procedural postponement may not be necessary, except in situations whereby bleeding times are excessively prolonged. Another option is to have patients withhold aspirin for 7–10 days and NSAIDs for 3–5 days [14, 15].

Thienopyridine inhibitors, such as clopidogrel, ticlopidine, and prasugrel, interfere with primary and secondary platelet aggregation [16]. These agents interfere with ADP binding and subsequent activation of the GpIIb/GpIIIb receptor complex. Clopidogrel interferes with platelets to platelet, platelet to fibrinogen, and platelet to endothelium interactions. Clopidogrel reaches a steady state in 7 days, but is reversible. Clopidogrel prolongs bleeding time more than aspirin. Purpura and epistaxis can occur in up to 5% of patients. Serious bleeding occurs at a rate of 1–2% [16, 22]. Recent studies suggest bleeding is more common with prasugrel. Extra caution with this medication is suggested as its clinical use is increasing.

Glycoprotein receptor antagonists interfere with the final common pathway of platelet aggregation and cross-linking [17]. GpIIb/GpIIIa receptor antagonists are often used in the management of acute coronary syndromes. Platelet function normalizes 8 to 24 h after stopping the infusion. During the infusion period, significant bleeding 2% of the time, and the majority of patients develop some degree of bleeding. These agents are not encountered in the outpatient setting, but are important in the perioperative setting. Elective surgery should be delayed for 24–48 h following abciximab and 4–8 h after tirofiban. Eptifibatide is another product in the same class that would be expected to have similar bleeding risks.

Warfarin [11, 18] is an oral anticoagulant that interferes with the carboxylation of vitamin K–dependent coagulation factors. Vitamin K-dependent factors, II, VII, IX, and X, become depleted. The intensity of warfarin therapy depends on the proportion of inactive factors and factor half-lives. Factor VII has the shortest half life and is most likely to affect the prothrombin time/international normalized ratio. When factor VII is at 40%, the INR approaches 1.5 [18]. Hemostasis is presumed to be normal at an INR $\leq$ 1.5. Age, diet, race, drug interactions, gender, body weight, and comorbidities influence the response to warfarin. A warfarin overdose manifests as ecchymoses and mucosal bleeding. Most surgical procedures can be carried out at an INR of 1.5. However, many practitioners will hold warfarin for 4–5 days prior to a surgical procedure. Balancing the risks of withdrawing anticoagulation in patients with a history of stroke or venous thromboembolic disease prior to the performing of surgery is a challenge.

Heparin [19] is a glycosaminoglycan that is widely used for anticoagulation during surgical procedures. Unfractionated heparin has a heterogeneous range of molecular weights with correspondingly heterogeneous anticoagulant properties. Heparin interacts with antithrombin III, which inactivates factors IIa, Xa, IXa, XIa, and XIIa. The anticoagulant effect is nonlinear. Low doses have a half-life of 60 min, and high doses have a half-life of up to 150 min. Significant bleeding has been reported in association with different types of heparin therapy. Subcutaneous hemorrhages and deep tissue hematomas may occur. Spontaneous bleeding may occur, even with an aPTT 1.5–2 times the normal.

Low-molecular-weight heparin [20] has a longer half-life, compared to unfractionated heparin. These products include enoxaparin, dalteparin, ardeparin, danaparoid, tinzaparin, and nadroparin. No blood test can adequately monitor the anticoagulant effect. LMWH primarily is associated with anti-Xa activity. Two to four hours following subcutaneous administration, therapeutic levels are reached, and up to 50% of this effect can be maintained at 12 h. Abnormal renal function, advanced age, and concomitant NSAID use enhance the effects of LMWH. LMWH is commonly used to reduce the risk of venous thromboembolism (VTE).

Herbal medications [21] such as garlic, ginkgo, and ginseng may cause bleeding. Garlic irreversibly inhibits platelet aggregation and has fibrinolytic activity. Ginkgo inhibits platelet-activating factor. Ginseng interferes with platelet aggregation. Several other herbal medications have been implicated as antihemostatic agents, including feverfew, green tea, horse chestnut, cat's claw, ginger, fenugreek, and chamomile. Fish oil (omega-3 fatty acid) supplements have been reported to increase the risk for significant bleeding. While well-controlled studies have not provided solid evidence for increased risk of clinically significant bleeding with fish oil, clinicians should be aware of the possibility of increased risk when used in conjunction with anticoagulants.

SSRIs and Other Antidepressants

Selective serotonin reuptake inhibitors (SSRIs) include fluoxetine (Prozac), fluvoxamine (Luvox), paroxetine (Paxil), sertraline (Zoloft), citalopram (Celexa), clomipramine (Anafranil), and trazodone (Desyrel). Nonselective serotonin reuptake inhibitors include amitriptyline (Elavil), imipramine (Tofranil), and doxepin (Sinequan). These medications are commonly used for depression, anxiety, and other psychological conditions. Research has shown that SSRIs can increase the risk of bleeding when combined with NSAIDs, aspirin, or anticoagulants. Depletion of serotonin (5-HT) levels in platelets inhibits induced aggregation and has the potential to prevent formation of clots. Clinicians should be aware of the potential of increased bleeding when these medications are combined with other anticoagulants.

Other Antiplatelet Drugs

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

Thrombin Inhibitors

Reversible inhibitors of thrombin may carry a lower risk of bleeding as compared to high-dose intravenous heparin. Recombinant hirudin derivatives (Desirudin, Lepirudin, Bivalirudin) reversibly inhibit free and clot-bound thrombin. They are an alternative to heparin in patients with unstable angina undergoing percutaneous coronary interventions. They may have a lower risk of major hemorrhage as compared to heparin. The half life is approximately 1 h. Serum levels decrease to zero 2 h after discontinuation. Argatroban, an L-arginine derivative, also provides reversible thrombin inhibition. It is used in the treatment and prophylaxis of heparin induced thrombocytopenia. These agents have an elimination half-life of 21 min – aPTT levels normalize within 2 h after discontinuation.

Newer Anticoagulants

Fondaparinux is a new selective factor Xa inhibitor. It has 100% bioavailability, without significant metabolism or nonspecific binding. Anticoagulation is more predictable and has been approved for venous thromboembolism prophylaxis following orthopedic surgery. This agent may reduce the risk of VTE by 50%, as compared to LMWH.

Bleeding Complications in Association with Interventional Pain Practice and Regional Anesthesia

The incidence of spontaneous spinal hematoma is extremely rare with an estimate of 1 patient per 1,000,000 patients per year [3, 23]. The incidence of clinically significant spinal hematoma has been estimated with 95% confidence to be <1 of 150,000 epidural anesthetics and <1 of 220,000 spinal anesthetics for noncardiac surgical cases. Moen et al. [4] estimated the risk of epidural hematoma following epidural placement in the obstetric population to be 1 in 200,000. Ruppen et al. estimated the risk of epidural hematoma following epidural analgesia for labor to be 1 in 168,000. For cardiac surgery, with profound anticoagulation during cardiopulmonary bypass, the estimated risk with 95% confidence may increase to 1:1,500 for epidural blockade, and to 1:3,600 for spinal blockade [3, 5, 6, 25]. In patients undergoing lower limb orthopedic surgery, with epidural analgesia, the incidence of spinal hematoma has been reported as 1:3,600 and 1:6,600 [4]. Hence, anticoagulation increases the risk of spinal hematoma during the performance of neuraxial procedures [22–25].

Epidural catheterization and regional blocks have been successfully carried in patients with hemophilia A, when factor VIII replacement was carried out [26, 27]. Epidural catheterization has been successfully performed in patients with von Willebrand's disease when vWF:Ag and RiCof levels met certain thresholds [28, 29]. Advanced liver disease, with associated portal hypertension and hypersplenism, poses a unique risk in procedure-related bleeding. Epidural hematomas following catheterization have been reported in patients with mild liver disease [30]. In renal disease, a delayed epidural hematoma has been reported. In this patient, coagulation studies and bleeding history were normal [31].

Anticoagulation increases the risk of bleeding. Spontaneous bleeding occurs in 3–7% of patients receiving warfarin [32]. Bleeding occurs in less than 3% of patients receiving fractionated or unfractionated heparin [32]. Thrombolytics present the greatest risk of bleeding with 6–30% [32]. In this background, procedure-related bleeding risk increases with anticoagulation.

The relative risk of neuraxial procedures in the presence of anticoagulation has been estimated [33]. There is no increased risk in the presence of aspirin therapy. Traumatic insertion increases the relative risk to 11. Traumatic insertion in the presence of systemic heparinization increases the relative risk to 111. Aspirin and intravenous heparin therapy increase the risk to 26. Timing of heparinization can influence the risk of bleeding. If heparinization is started within 1 h of a neuraxial procedure, the risk is 25. If heparinization is delayed more than 1 h, then the risk drops to 2.

Aspirin and NSAIDs are not contraindicated in neuraxial or nonneuraxial procedures [2]. Vandermeulen [34] only identified two hematomas related to aspirin and indomethacin in his series of 61 patients. The American Society of Regional Anesthesia has adapted guidelines for thienopyridine derivatives [2], such as clopidogrel. Clopidogrel should be held for 7 days prior to a neuraxial procedure [2]. Neuraxial procedures should be avoided for 4 weeks following a glycoprotein receptor antagonist [2]. Thienopyridine agents have been implicated in bleeding following lumbar sympathetic blocks and cervical epidural steroid injections [35, 36].

Oral anticoagulation is a contraindication to neuraxial anesthesia [2]. Atraumatic epidural catheterization in an anticoagulated patient has lead to paraplegia [37]. The American Society of Regional Anesthesia suggests that warfarin should be stopped 4–5 days prior to neuraxial procedures [2]. The prothrombin time and international normalized ratio should be checked prior to the neuraxial block. There is no "safe" INR for performing neuraxial procedures [1], but ideally, an INR less than 1.3 should be sought. In the setting of thrombolytics, neuraxial procedures should be avoided [1, 2].

Vandermeulen et al. [34] reported 30 cases of epidural hematoma in patients receiving fractionated or unfractionated heparin therapy. For unfractionated heparin, the ASRA guidelines [2] suggest that neuraxial techniques should be avoided in patients with concomitant coagulopathies, heparinization should be delayed for 1 h after needle placement, indwelling catheters should be removed 2–4 h after the last heparin dose, reheparinization should be delayed for 1 h after catheter removal, postoperative neurological monitoring with minimal use of local anesthetics is

mandatory, and caution should be exercised if needle insertion is difficult or traumatic. Similar guidelines should be considered for nonneuraxis procedures [2].

The recommendations for the perioperative administration of low-molecular-weight heparin by the American Society of Regional Anesthesia2 advise avoidance of concomitant antiplatelet and oral anticoagulants, significant increase in the risk of spinal hematomas with traumatic needle insertions, needle placement delay of 12 hours following the last dose of prophylactic LMWH, and needle placement delay of 24 hours following the last dose of therapeutic LMWH. LMWH should be held at least 24 h following surgery if an indwelling catheter is in place. The catheter should be removed prior to initiation of LMWH therapy, and this therapy should not be started for at least 2 h after catheter removal.

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

Bleeding Risk Assessment

A bleeding risk score (Fig. 6.1) can be estimated based on the potential hazards of bleeding, associated with specific anticoagulants and bleeding disorders. The goal of this scoring system is to provide a framework for physicians and nonphysicians to quickly assess the bleeding risk in specific patients. In a surgical center or pain procedure suite, patient flow is rapid. A bleeding risk instrument enables nonphysicians to quickly bring a potential bleeding problem to the attention of the physician.

Technique-specific factors may influence the risk and consequences of bleeding [1] (Fig. 6.2). These factors depend on whether the target structure is near a major vascular or neurological structure or is in a confined space [1]. The type of needle used and the number of passes required for the procedure will influence the risk of bleeding [1]. The caliber of the needle, the use of fluoroscopy and contrast, and the use of aspiration are factors that influence the risk and recognition of bleeding [1]. Finally, a procedure that is a “single shot” may have a lower risk of bleeding compared to a continuous infusion [1].

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

Fig. 6.1 Components of bleeding risk associated with interventional pain procedure (adapted from ref. [1])

Hemostasis	Modifying factors	Score
Normal	None	2
Normal	History of self-limited, transient bleeding disorder	4
Normal	Normal coagulation studies despite the intake of medications that theoretically may affect hemostasis	6 (nutraceuticals, serotonin reuptake inhibitors)
Normal	Normal coagulation studies after discontinuation of known anticoagulants (the score may be modified, depending on when the drug was stopped relative to the period of drug effect)	6 (e.g., warfarin was stopped 5 days earlier, aspirin was stopped 7-10 days earlier, heparin infusion held for >6 hours) 8 (e.g., aspirin was stopped 3 days earlier) 10 (e.g. warfarin was stopped 2 days earlier, heparin infusion was stopped 4 hours earlier) 6-10 (e.g., factor or blood product replacement therapy in specific acquired and congenital bleeding disorder
Abnormal	Active consumption of anticoagulants that cannot be held (the score may be modified based on the specific anticoagulant and abnormal coagulation studies)	10 (low dose aspirin, NSAIDS) 12 (subcutaneous heparin, low dose coumadin (INR<1.4), medium-high dose aspirin, ticlopidine, clopidogrel) 14 (low molecular weight heparin, coumadin (INR 1.5-2, Gp IIb/Gp IIIa inhibitors) 16 (intravenous heparin bolus, coumadin (INR 2-3)) 16-18 (thrombin inhibitors) 18 (high dose intravenous heparinization and warfarin, INR >3).

Fig. 6.2 Components of bleeding risk associated with impaired hemostasis and anticoagulant (adapted from ref. [1])

		20 (thrombolytics)
Abnormal	Known history of medical bleeding disorder (the score may be modified if there is a history of easy bruisability, deep versus superficial bleeding episodes, or spontaneous versus traumaticallyinduced bleeding episodes)	10 (thrombocytopenia >80,000) 12 (thrombocytopenia <80,000, idiopathic thrombocytopenic purpura, renal failure-uremia) 12-14(von Willebrand disease, depending severity) 14 (vitamin K deficiency 14-18 (Hemophilia A and B depending on severity of factor deficiency) 14-18 (liver disease, depending on severity)
Abnormal	Known history of significant bleeding with procedures but cause not identified	18
Abnormal	Major hemorrhage due to incompetent coagulation system	20 (disseminated intravascular coagulation)

Fig. 6.2 (continued)

Physicians can estimate this bleeding risk based on patient- and technique-specific factors by using a bleeding risk score. This score may support clinical decision making about whether to cancel or carry out the procedure [1]. Even then, patient flow is rapid in interventional pain management – prone to the possibility of failing to identify patients at an elevated bleeding risk. The bleeding risk score can be helpful to physicians, nonphysicians, and health-care teams to identify patients at risk of bleeding. A numerical bleeding score has been developed with the intent of facilitating health-care communication and improving patient safety.

Neuraxial procedures are safe in the absence of anticoagulation, with respect to the development of a spinal hematoma. It suggests that neuraxial procedures can be safe in the presence of aspirin or even nonsteroidal anti-inflammatory drugs. This systematic review suggests that no definitive conclusion can be reached regarding minimizing spinal hematoma or major bleeding risk, in the setting of anticoagulant therapy or impaired hemostasis. In this scenario, guidelines and recommendations based on sensible practices are advised. An adequately powered study to truly ascertain the risk of bleeding, following an interventional spine procedure in the setting of anticoagulation or impaired hemostasis, would require a very large number of subjects. This would put many patients at risk and would be unethical. The FDA Medwatch system [38] alerted practitioners to the risks of spinal hematoma in the setting of low-molecular-weight heparin. This resulted in modification of American practices. The German guidelines [39] were more conservative with respect to

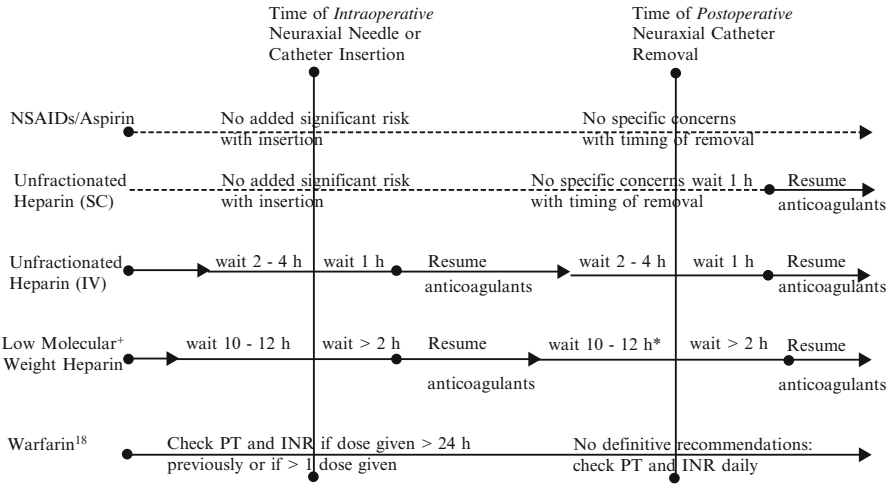

Fig. 6.3 Recommendations for anticoagulated patients undergoing image-guided spinal procedures (adapted from ref. [40])

neuraxial anesthesia and low-molecular-weight heparin, and they reported fewer cases of spinal hematoma. Thus, there is a precedent for the use of guidelines or mathematical estimations of risk [24], such as those proposed by the American Society of Regional Anesthesia [2] (Fig. 6.3). Nonetheless, spinal hematomas can still arise with close adherence to guidelines. Furthermore, interventional spine procedures are more heterogeneous with respect to anatomical localization, technique, practitioner expertise, and needle or catheter type. A prior narrative review [1] suggested utilizing a bleeding risk score/assessment to determine the safety of performing an interventional pain procedure in a patient with impaired hemostasis. These strategies, in the absence of sufficient information, may be incorporated into clinical decision making.

Conclusion

Interventional pain physicians perform procedures that carry a finite amount of bleeding risk. Many patients have congenital or acquired bleeding disorders and/or are taking anticoagulants. Patient safety can be compromised if this information is overlooked. Understanding hemostatic disorders and anticoagulation and the bleeding risk of interventional procedures is imperative to improving patient safety. Pain physicians and anesthesiologist must familiarize themselves with this subject matter in order to make informed decisions about interventional procedures.

Communicating this risk benefit analysis with the patient is imperative, for the purposes of informed consent and shared decision making. Patients must understand that there is no way to reduce the risk of a spinal hematoma or major bleeding

to zero. Practitioners may face difficult decisions about stopping the anticoagulant, correcting a hemostatic disorder, modifying the interventional technique, or pursuing less-invasive pain therapies.

A model has been proposed, wherein the patient- and technique-specific bleeding risks can be added to provide an overall risk of bleeding score [1]. When utilized, physicians and nonphysicians may quickly and efficiently calculate this overall risk and communicate this vital piece of information among themselves. This overall risk score may facilitate clinical decision making and improve patient safety [1].

Based on the author's review of the literature, there is high-level evidence for continuing aspirin, even prior to an interlaminar epidural steroid; the benefits outweigh the risks. Also supported by the literature are the following (1) restarting heparin therapy following epidural catheterization during cardiac surgery, (2) withholding nonsteroidal anti-inflammatory drugs prior to a procedure, (3) reducing risk of major bleeding with once-daily low-molecular-weight heparin, (4) restarting coumadin therapy, and (5) safety of neuraxial procedures, have been provided. These recommendations are specific but cannot be generalized to all clinical scenarios. Clinical guidelines and mathematical estimations of risk may partially fill this void. To help with decision making, an overall risk of bleeding risk score has been proposed by a narrative review [2]. This score may support clinical decision making about whether to cancel or carry out the procedure [2].

Clinical Pearls

- Discontinue chronic warfarin therapy 4–5 days before spinal procedure – the INR should be within the normal range at time of procedure.
- No contraindications with aspirin or NSAIDs.
- Thienopyridine derivatives (clopidogrel and ticlopidine) should be discontinued at least 7 days prior to procedure.
- GpIIb/IIIa inhibitors should be discontinued to allow recovery of platelet function prior to procedure (8 h for tirofiban and eptifibatide, 24–48 h for abciximab).
- Thrombolytics/fibrinolytics: an extremely high risk of bleeding; neuraxial techniques should be avoided; no recommendations for timing of catheter removal in those patients who unexpectedly receive fibrinolytic or thrombolytic therapy. Follow fibrinogen level and observe for signs of neural compression.
- LMWH: Delay procedure at least 12 h from the last dose of thromboprophylaxis LMWH dose. For "treatment" dosing of LMWH, at least 24 h should elapse prior to procedure. LMWH should not be administered within 24 h after the procedure.
- Unfractionated SQ heparin: There are no contraindications to neuraxial procedure if total daily dose is less than 10,000 U. For higher dosing regimens, manage according to intravenous heparin guidelines.
- Unfractionated IV heparin: Delay spinal puncture 2–4 h after last dose; document normal aPTT. Heparin may be restarted 1 h following procedure.

- There should be a low threshold for obtaining an MRI if a patient demonstrates progressive neurological impairment.
- The incidence of clinically significant spinal hematoma has been estimated to be <1 of 150,000 epidural anesthetics and <1 of 220,000 spinal anesthetics for non-cardiac surgical cases.

Multiple-Choice Questions

1. A bleeding risk score is useful for estimating which of the following?
   (a) Physicians can estimate bleeding risk based on patient- and technique-specific factors.
   (b) Physicians can estimate the amount of blood a patient may lose during an intervention pain procedure.
   (c) This score may support clinical decision making about whether to cancel or carry out a procedure.
   (d) (a) and (c).

2. Neuraxial procedures are usually safe, with respect to the development of a spinal hematoma, in the absence of which of the following?
   (a) Warfarin
   (b) Aspirin or nonsteroidal anti-inflammatory drugs
   (c) Low-molecular-weight heparin
   (d) (a) and (d)

3. Which of the following is a contraindication to neuraxial anesthesia?
   (a) Oral anticoagulation
   (b) Thrombolytics
   (c) Unfractionated heparin
   (d) All of the above

4. Which of the following are considered thienopyridine derivatives?
   (a) Clopidogrel
   (b) Dipyridamole
   (c) Lepirudin
   (d) Fondaparinux

5. Neuraxial procedures should be avoided for 4 weeks following a glycoprotein receptor antagonist. Which of the following is not a glycoprotein receptor antagonist?
   (a) Abciximab
   (b) Tirofiban
   (c) Eptifibatide
   (d) Prasugrel

6. Which of the following can lead to platelet dysfunction and hemostatic defects?
   (a) Renal disease
   (b) Liver disease
   (c) Vitamin K deficiency
   (d) All of the above

7. Which of the following is considered the "universal" hemostatic agent and can be used to control surgical or trauma-associated bleeding in patients with hemophilia A?
   (a) Recombinant VIIa
   (b) Recombinant VIa
   (c) Recombinant IVa
   (d) Recombinant VIIIa

8. Which of the following is *not* a characteristic of von Willebrand's disease?
   (a) It is the most common hereditary bleeding disorder.
   (b) Afflicts 1–3% of the general population.
   (c) Factor VIII levels are reduced.
   (d) The condition is not an autosomal dominant disorder.

9. Which of the following are accurate concerning interventional pain management?
   (a) Is an emerging specialty.
   (b) Uses procedures to diagnose and treat chronic pain.
   (c) Perform procedures percutaneously that often carry a risk of bleeding.
   (d) All of the above.

10. Which of the following usually occurs after needle trauma?
    (a) A platelet plug is created in order to stop bleeding.
    (b) Glycoproteins adhere to injured endothelium.
    (c) Von Willebrand factor (vWF), a protein found in subendothelial tissues, facilitates platelet adhesion.
    (d) All of the above.

11. Which of the following are true concerning cyclooxygenase inhibitors?
    (a) They disrupt the formation of thromboxane A2.
    (b) They stimulate the production of thromboxane A2.
    (c) NSAIDs irreversibly inhibit cyclooxygenase for the life of platelet.
    (d) Aspirin reversibly inhibits this enzyme.

12. Thienopyridine inhibitors interfere with primary and secondary platelet aggregation due to ADP binding and subsequent activation of the GpIIb/GpIIIb receptor complex. Which of the following is not a thienopyridine inhibitor?
    (a) Heparin
    (b) Prasugrel
    (c) Ticlopidine
    (d) All of the above are thienopyridine inhibitors

13. Clopidogrel should be held for how many days prior to a neuraxial procedure?
    (a) 9 days
    (b) 2 days
    (c) 5 days
    (d) 7 days
14. Ideally, what should be sought prior to the neuraxial block?
    (a) An INR less than 1.3
    (b) An INR less than 2.3
    (c) An INR less than 2.5
    (d) An INR less than 3
15. With regard to communicating the risk benefit analysis with patients, which of the following is true?
    (a) Patients must understand that there is no way to reduce the risk of a spinal hematoma or major bleeding episode to zero.
    (b) Difficult decisions about stopping anticoagulants may need to be made.
    (c) Pursuing less-invasive pain therapies may be a safer choice for a patient with hemostatic disorders.
    (d) All of the above are true.

Answers:

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

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Part III
Equipment for Regional Anesthesia

7

Equipment and Clinical Practice: Aids to Localization of Peripheral Nerves

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Introduction

Identifying nerve location begins by identifying standard anatomical landmarks, which are used as a basis for subsequent invasive needle exploration. The successful endpoint used may be anatomical (transarterial axillary block), ultrasonographic (real-time imaging), or functional [a sensory paresthesia or a motor response to electrical nerve stimulation (NS)].

In the 1960s, electrical nerve stimulation techniques were developed. Even more recently, small, battery-operated, portable handheld devices have been introduced [1–3].

Subsequently, peripheral nerve stimulation (PNS) enjoys widespread use, with a proven clinical efficacy and safety record. Electrical stimulus of the nerve is based on factors such as conductive area of the electrode, resistance to electrical stimulation, distance between skin and nerve, current flow, and pulse duration (Fig. 7.1) [4].

Stimulation

A weak direct current (DC) electrical current is supplied to the stimulating needle by an oscillating (square wave) current generator (the nerve stimulator). Assuming that a square pulse of current is used to stimulate the nerve, the total energy (charge)

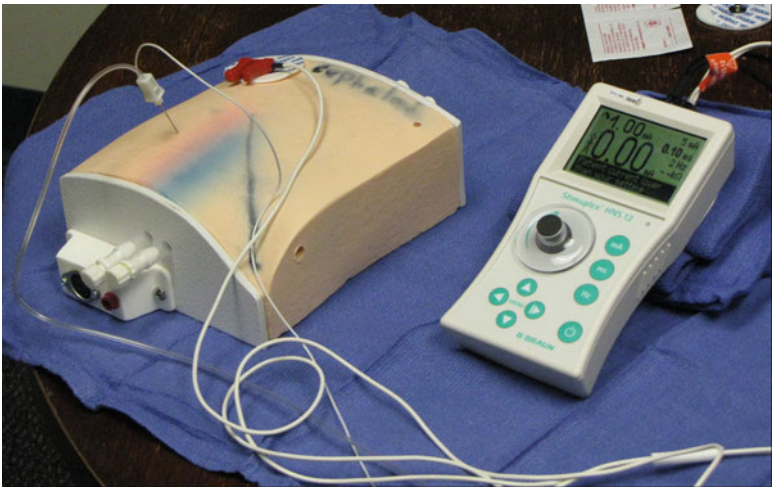

Fig. 7.1 Nerve stimulator, with stimulating needle/ground lead, attached to a simulated model

applied to the nerve is the product of the current intensity and the pulse duration. The actual current output by the stimulator is calculated as voltage output (voltage [V]/impedance [Q] = output [mA]) (Ohm's law) [5].

Technique

The current is pulsed, typically at a frequency (*f*) of 1–2 Hz (cycles/s). Initial current amplitude (amperage) of 1–2 mA with a pulse duration of 0.1–0.2 ms is then directed to the stimulating needle, which is then inserted through the skin and slowly advanced toward the expected anatomical location of the targeted nerve. The current is slowly decreased when the appropriate motor response is elicited. Motor contractions that occur at a low current (usually 0.2–0.5 mA) indicate that the needle tip is in close proximity to the nerve. In practice, no motor response will occur if the needle tip is greater than 1 cm from the targeted nerve: PNS is used to refine the search endpoint, guiding the needle through the final 5 mm or so.

PNS is thus limited in application to mixed peripheral nerves, as a motor response endpoint is desired. Although pure sensory nerves may be stimulated, ultimately obtaining a sensory paresthesia, this is not commonly performed clinically.

Electrophysiology

Energy

The amount of electrical energy required to propagate a nerve impulse is the product of the stimulus strength (mA) and current duration (ms). For any nerve type, there is a minimum current strength required in order to generate an impulse – this is referred to as the rheobase. Below this minimum level, an impulse will not be generated. The chronaxie is defined as the stimulus duration needed for impulse generation, when employing a current strength of twice the rheobase. Myelinated fibers are much more sensitive and require less electrical energy for stimulation (having shorter chronaxie) than unmyelinated fibers (chronaxie of alpha fibers, 50–100; delta fibers, 170; C fibers, 400).

The large (alpha) motor fibers are much more readily stimulated than the smaller Δ (delta) fibers or C fibers responsible for pain sensation. However, when a higher intensity current is used (e.g., greater than 1.0 mA), preferential stimulation of the motor fibers may be lost, and uncomfortable paresthesia-like stimulation often occurs. Alternatively, significantly less current is required to elicit a motor response with a stimulus of longer duration (e.g., >0.5 mA). Thus, using a shorter impulse duration of 0.1 ms will allow for motor nerve stimulation without initiating painful C fiber activity.

Polarity

Less electrical energy is required if the (negative) cathode is close to the nerve since with a negative stimulating needle, the direction of current flow induces some direct depolarization. The reverse is true with an anodal (positive) needle since the direction of flow induces hyperpolarization of the target nerve. This, in turn, requires a higher current to stimulate the nerve. For these reasons, the needle polarity is designated negative by default. The site of placement of the positive (return) electrode, however, is probably irrelevant, as long as quality grounding electrodes are used and good electrical contact is made.

Distance

The relationship between the constant current stimulus intensity and the distance from the nerve is governed by Coulomb's law:

$$I = K\left(\frac{Q}{r^2}\right),$$

where I is the current required to stimulate the nerve, K is a constant, Q is the minimal current needed for stimulation, and r is the distance from the stimulus to the nerve.

The presence of the inverse square means that a current of very high intensity is required as the needle moves away from the nerve. Conversely, a nerve will only be stimulated when the needle is close to it. The initial current should therefore be set at 1–2 mA with an impulse duration of 0.1 ms. Generally, a motor response is elicited when the needle is within 5–10 mm from the nerve, using a current set at around 0.5 mA. This suggests that the needle tip is 1–2 mm from the motor nerve, signifying that a subsequent block will be satisfactory. If a muscle twitch is generated at a current strength of less than 0.2 mA, the stimulating needle may have penetrated the epineurium, thus risking a subsequent intraneural injection. It is therefore important to ensure that the muscle twitch disappears at or higher than a current of 0.2 mA.

Stimulus Frequency

As the needle is advanced, a muscle twitch by the stimulating current indicates that the needle is approaching the target nerve. If the frequency of impulses is too low, the nerve may be inadvertently penetrated. If the frequency is too high, painful muscle twitches (tetany) may be induced. A frequency of 2 Hz (cycles/s) is a good compromise as well as a suggested needle advancement speed of approximately 1 mm/s [6].

Summary

A peripheral nerve stimulator should provide as a minimum:

1. A square wave impulse with a duration of 0.1 ms.
2. The negative lead connected to the stimulating needle.
3. 2 Hz frequency.
4. Initial current level of 1–2 mA, seeking the nerve.
5. A final current level of 0.3–0.6 mA, positioning the needle tip close to the nerve.
6. Current delivery down to 0.1–0.2 mA, to ensure no intraneural stimulation.

Additional safety features include:

1. Accurate current delivery in the range of 0–5.0 mA.
2. Constant current square wave pulse.
3. Display of current flowing into the patient as well as that delivered internally from the device.
4. Open circuit alarm.
5. Excessive impedance alarm.
6. Low battery alarm.
7. Internal malfunction alarm [5].

New Developments in Nerve Stimulation

Percutaneous Electrode Guidance

The percutaneous electrode guidance (PEG) technique is a modification of transcutaneous NS: A transcutaneous nerve electrode coupled to a nerve stimulator can be used to locate an underlying nerve by passing the superficial electrode over standard anatomic landmarks. Cutaneous stimulation of the underlying nerve occurs at nerve stimulator settings between 2 and 10 mA, with a 0.1-ms pulse duration (alternatively, 0–5 mA, pulse duration 0.2–1.0 ms). Cutaneous stimulation benefits from a longer pulse duration (0.2–1.0 ms), which enables an electrical motor response at a lower current. Since much of the electrolocution is initially done by the probe, which indents the skin toward the nerve, the stimulating needle tip (inserted from within the outer PEG cannula) travels only a short distance in order to finally contact the nerve. Skin indentation during the performance of the PEG technique allows for a decrease in impedance as well as a maximal increase in electrical conductance. Thus, PEG has the net effect of eliciting a motor response with minimal discomfort to the patient [5, 7, 8].

Sequential Electrical Nerve Stimulation

Presently, current amplitude (amperage) is continuously varied, deliberately maintaining a constant frequency and pulse duration (one degree of freedom). Therefore, only one constant fixed pulse duration has been used (e.g., 0.1 or 0.2 ms). Some newer nerve stimulators allow the pulse duration to be preset at different fixed pulse widths (e.g., 0.05, 0.1, 0.3, 0.5, or 1.0 ms). However, this pulse duration cannot be easily varied during the actual block performance. Urmey and Grossi [9] recently evaluated a novel technique for nerve localization utilizing an electrical nerve stimulator programmed to deliver sequenced electrical nerve stimuli (SENS). The nerve stimulator generated alternating sequential electrical pulses of differing pulse durations at an overall set frequency of 3 Hz (3 cycles/s). Repeating pulse duration sequences of 0.1, 0.3, and 1.0 ms (shortest to longest) were generated, with 1/3-s period intervals separating each pulse.

Selective attenuation of the applied current resulted in the three pulses having more equivalent charges. In each case, the needle was advanced at an initial current amplitude of 1 mA until appropriate motor responses (MR) occurred. If 1 MR/s or 2 MR/s were noted, the needle was continually advanced until all 3 MR/s were visible. Current was then decreased until MR/s decreased to 1 or 2. At this point, the needle was again advanced slowly. When 3 MR/s occurred at ≤0.5 mA, indicating that the 0.1-ms pulse was stimulating the nerve, final needle position was held constant. Prior to final injection, current was then slowly decreased with the needle held immobile.

Conventionally, increasing the current flow has been the only parameter used to increase stimulation range since it directly enables stimulation at a greater distance from the nerve. Additionally, with SENS, pulse durations of 0.3–1.0 ms were used almost simultaneously to increase the range, in distance, of successful stimulation at a given current amplitude. Therefore, higher pulse durations increase sensitivity for successful NS with the stimulator needle at a distance, whereas specificity is then enhanced by decreasing the pulse duration down to the standard 0.1 ms. By employing sequential long and short pulses, successful neurostimulation was able to occur at a much greater needle to nerve distance. Prior to SENS, these elicited motor responses did not occur with the standard 0.1-ms pulses. Thus, the near simultaneous variance of two separate parameters (applied current together with pulse width duration) enhanced successful PNS of the targeted motor nerve [5].

Regional Anesthesia Equipment Tray

When performing a regional anesthetic procedure, it is often useful to have a kit of supplies needed to perform the block. Not only does this provide organization and efficiency while performing, but can also be added as a separate billable charge bundle, as the equipment used is not part of the typical general anesthetic protocol,

leading to possible revenue [10]. Depending on the institution, regional anesthetic equipment kits can include, but are not limited to nasal cannulae, EKG electrodes, pulse oximeter, needles, syringes, stopcock, sterile gloves, midazolam and fentanyl for sedation, and insulated nerve block needles. When placing a continuous nerve block catheter, the kit may be expanded to include a nerve block catheter, sterile drapes, sterile dressing, and a local anesthetic infusion device.

Disposable Versus Reusable Equipment Trays

Both disposable and reusable equipment trays have their benefits and risks. Disposable trays have the benefit of decreasing cross contamination and infection risk, however, may be costly. Reusable equipment trays require facilities to sterilize them after every use. Although reusable equipment trays allow for less waste, it represents a significant initial cost investment.

Skin Preparation

Infection is a rare complication associated with regional anesthetic procedures but is a concern nonetheless. The concern is greater with the growing popularity of perineural catheters. Two common antiseptics used to prepare the skin for the procedure are povidone-iodine and chlorhexidine. While both may kill organisms on the skin, there is a wealth of literature supporting the use of chlorhexidine over povidone-iodine. Chlorhexidine was demonstrated to have more rapid of onset and more efficacy at antisepsis with longer duration when being used for epidural or even central venous catheter or arterial line placement [11–15].

Syringes

Syringes are not only useful for dispensing the local anesthetic but are also useful in locating the epidural space in neuraxial blocks.

Large syringes have the benefit of holding larger volumes of local anesthetic but have less precision in volume injection. Larger syringes also have the benefit of not having to disconnect and reconnect syringes when injecting large volume of anesthetic.

It is a common belief that intraneural injection can be avoided by the sensation of resistance during injection. It is also commonly believed that smaller volume syringes enable the clinician to better feel this resistance. In a recent animal study, the conclusion was that syringe feel was no better than chance at detecting intraneural injection when using a 20-mL syringe [16].

Glass Syringes

Glass syringes are commonly used when identifying the epidural space. The glass syringe provides little friction between the internal wall and piston of the syringe, allowing the clinician to feel a loss of resistance upon entering the epidural space. Newer plastic syringes have been developed with lubrication that may be adequate for locating the epidural space as well.

Needles

There are several different needles employed during regional anesthetic procedures. Each needle provides different advantages.

Stimulating Needles

Stimulating needles or also known as insulated needles have a protective nonconducting sheath over the shaft of the needle, with the exception of the tip. By applying an electrical current to the needle using a nerve stimulator, it is possible to stimulate a nerve as the tip of the needle comes in proximity of the nerve.

When comparing nerve blocks performed with nerve stimulator with stimulating needles and with ultrasound alone, studies have demonstrated quicker onset of sensory and motor blockade and longer duration with US guidance compared to peripheral nerve stimulation alone [17, 18].

Needle Gauge

In general, a 22-G needle is used to place single shot nerve blocks. Needles with smaller gauge improve patient comfort while placing the block; however, they are more difficult to inject through. These needles are usually used for local infiltration at the skin.

Larger bore needles can be useful when placing catheters. An 18-G Tuohy needle is commonly used to place perineural catheters.

Needle Bevel

There have been several studies concerning mechanical trauma in regard to the needle bevel. Short-beveled and <45° needles may have less incidence of nerve trauma: however, when nerve injury does occur, it may be more severe compared to long-beveled needles, >45° [19–22].

Spinal Needles

Spinal needles are designed to penetrate the dura when performing a subarachnoid block. They are usually 25–27 gauge in bore size. 25-G needles are often used for spinal anesthetic techniques. A 27-G needle is often used for combined spinal–epidural procedures as the small bore of the needle may make traversing the subcutaneous tissues, muscles, and ligaments difficult. These needles often are designed with a stylet to prevent a tissue plug from forming while traversing the tissue in between the skin and the subarachnoid space.

Postdural puncture headache is a well-described side effect of placing a subarachnoid block. There is data that supports that the type of needle used may affect the incidence of postdural puncture headache. One large meta-analysis concluded that the incidence of postdural puncture headache was decreased when using a smaller bore needle and noncutting needles [23]. Greene and Whitacre point needles have rounded tips and are less likely to cause PDPH. It is theorized that the tip spreads rather than severing the longitudinal fibers of the dura, thus, causing less damage.

Epidural Needles

Epidural needles are larger bore needles. They have a stylet to prevent a tissue plug while advancing the needle through tissue. The function of an epidural needle is to provide a channel to locate the epidural space via loss of resistance or hanging drop techniques. The channel of the needle also functions as a channel to thread a catheter.

Tuohy needles are commonly used to find and catheterized the epidural space. They have a curved tip so that a catheter will exit the needle to the side rather than straight out the shaft but will still allow a spinal needle to exit along the same direction. Because of the curved tip, the needle may not traverse in the same direction as the force applied to it. Knowledge of direction of needle tip will allow the practitioner to predict the direction the needle is going and also manipulate the direction a catheter is being advanced passed the tip.

With the increasing popularity of perineural catheters, stimulating Tuohy needles have been developed in efforts to locate a nerve using electrical stimulation and allowing a catheter to be placed in close proximity to it.

Catheters

Infusion catheters have been used for continuous infusion of local anesthetic into the epidural space. Catheters have been constructed with several different properties. They can be single port or multiorifice. Single-port catheters have their opening at the distal tip of the catheter. With single-port catheters, test injectate is all expelled from the single orifice, allowing for a more reliable test dose to the same area,

should the area be intravascular or intrathecal. Multiorifice catheters may be less likely to plug given the multiple openings and may be more reliable for detection of intrathecal or intravascular placement via aspiration.

Newer catheters may be made of different materials, leading to differences in stiffness and flexibility. Metal reinforced catheters may be difficult to kink; however, they are not MRI compatible and should be noted when placed.

More recently, perineural catheters have become popular to provide anesthesia and analgesia for orthopedic procedures. Brachial plexus catheters have been shown to have favorable outcomes [24]. Interscalene catheters have been shown to decrease pain and opioid usage after shoulder surgery [25].

Catheters usually come in a 20–22-G size. Smaller gauge catheters may increase resistance to infusing medication through. They also may be more difficult to thread and more likely to kink.

Recently, catheters have been developed that are able to conduct an electrical current. By applying an electrical current along a catheter, the nerve it is placed by can be stimulated, thereby, theoretically demonstrating a close proximity to that nerve. Recent data comparing stimulating catheters to nonstimulating catheters demonstrated that the stimulating catheters may lead to better analgesic quality [26]. Another study showed that infraclavicular catheters showed a higher block success using stimulating catheters and ultrasound guidance compared to placement of either stimulating or nonstimulating catheter without ultrasound [27].

Infusion Devices

With the plethora of infusion devices available today, it can be difficult in choosing one device that is ideal for all situations. Infusion devices vary in their infusion rate accuracy and consistency, programmability, infusate capacity, disposability, and price [28].

Elastomeric devices and spring-powered are simple, depending creating a tensile force to eject the infusate into the catheter. These devices may be cheaper, but tend to be inaccurate with their infusion rates. The rate of infusion changed with the amount of infusate left in the reservoir. Electronic pumps may be more expensive, but tend to be more accurate and consistent. They may have an added patient-controlled bolus function, but are not as disposable as its elastomeric counterparts.

Multiple-Choice Questions

1. Identifying nerve location can be:
   (a) Anatomic
   (b) Ultrasonographic
   (c) Functional
   (d) All of the above

2. Electrical nerve stimulation is based on:
   (a) Conductive area
   (b) Resistance
   (c) Distance
   (d) All of the above

3. A nerve stimulator
   (a) Applies a constant current impulse:
   (b) Charge = (I) X (pulse duration)
   (c) Utilizes an oscillating current
   (d) (b) and (c)

4. Nerve stimulator current:
   (a) Is pulsed at 1–2 Hz (cycles/s)
   (b) Starts at 10 mA
   (c) Pulse duration of 0.1–0.2 ms
   (d) (a) and (c)

5. Motor contractions:
   (a) Occur at low current (0.2–0.5 mA)
   (b) Do not occur greater than 1 cm from needle tip to nerve
   (c) Start at a current of 1–2 mA
   (d) All of the above

6. Pure sensory nerves:
   (a) May be stimulated
   (b) Endpoint is a sensory paresthesia
   (c) Are not commonly stimulated clinically
   (d) All of the above

7. When using a high-intensity current (>1 mA):
   (a) Motor and sensory stimulation occurs
   (b) Painful C fiber activity occurs
   (c) More current is required with longer duration (>0.5 mA)
   (d) (a) and (b)

8. True statements regarding needle polarity and grounding are:
   (a) Needle is negative by default
   (b) The grounding electrode should be applied within 6 in. of the target nerve
   (c) A positive needle requires a higher stimulating current
   (d) (a) and (c)

9. Chlorhexidine is more advantageous over povidone–iodine in regard to antisepsis because it:
   (a) Is more rapid in onset
   (b) More efficacious
   (c) Has longer duration antimicrobial activity
   (d) All of the above

10. Short-beveled needles compared to long-beveled needles:
    (a) Have less incidence of nerve trauma
    (b) Have less severe nerve trauma when trauma occurs
    (c) Are $>45°$
    (d) Are more comfortable for the patient

11. Postdural puncture headache:
    (a) Increases in incidence with smaller gauge needles
    (b) Is less likely with Whitacre and Greene needles
    (c) Is less likely with cutting edge needles
    (d) In unrelated to the gauge or edge of the needle

12. Tuohy needles:
    (a) Have an orifice at the tip in line with the shaft of the needle
    (b) Are nonstimulating needles
    (c) Traverse through tissue in a straight line
    (d) Are larger bore needles that allow advancement of a catheter through its shaft

13. Single-port catheters compared to multiorifice catheters:
    (a) Are more reliable when tested with test injectate
    (b) Are more reliable when aspirated for return of fluid
    (c) Are less likely to become obstructed by a plug
    (d) (a) and (c)

14. Stimulating catheters compared to nonstimulating catheters:
    (a) Are equivalent in analgesic quality
    (b) Improve block success when used in conjunction with ultrasound when placing infraclavicular blocks
    (c) Are only single orifice
    (d) (a) and (c)

15. Elastomeric infusion devices:
    (a) Are cheap and accurate
    (b) Have faster infusion rates when the reservoir is full
    (c) Have slower infusion rates when the reservoir is close to empty
    (d) (b) and (c)

Answers:

1. d
2. d
3. d
4. d
5. d
6. d
7. d
8. d

9. d
10. a
11. b
12. d
13. a
14. b
15. d

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8

Principles of Sonography

Paul E. Bigeleisen • Steven Orebaugh

Content

The frequency of medical ultrasound ranges between 2 and 15 MHz. The speed of sound in tissues is 1,500 m/s yielding a wavelength of about 1 mm. This limits the imaging of nerves to structures that are at least 1 mm in diameter. Most nerves, veins, and arteries of interest are larger (3–15 mm).

Many factors determine the quality and resolution of an ultrasound image. These include the frequency of the sound wave, the signal strength, attenuation of the beam by tissue, refraction by tissue interfaces, and surface scattering and diffraction by fat. In addition, high-frequency beams which provide the highest resolution images are rapidly attenuated as the sound wave traverses the tissue (Fig. 8.1). For this reason, the best images, such as the brachial plexus, are limited to depths of 4 cm or less. Deeper structures, 5–15 cm, such as the sciatic nerve and lumbar plexus are more difficult to image. In some cases, bone shadows, such as in spinal imaging, impede the formation of an image of the nerve tissue, making ultrasound less useful for neuraxial imaging.

The image formed on ultrasound is very sensitive to the angle that the beam forms with the target nerve. This angle is called the angle of insonation. The reason for this is that the nerve itself contains fat and is often also surrounded by fat (Fig. 8.2).

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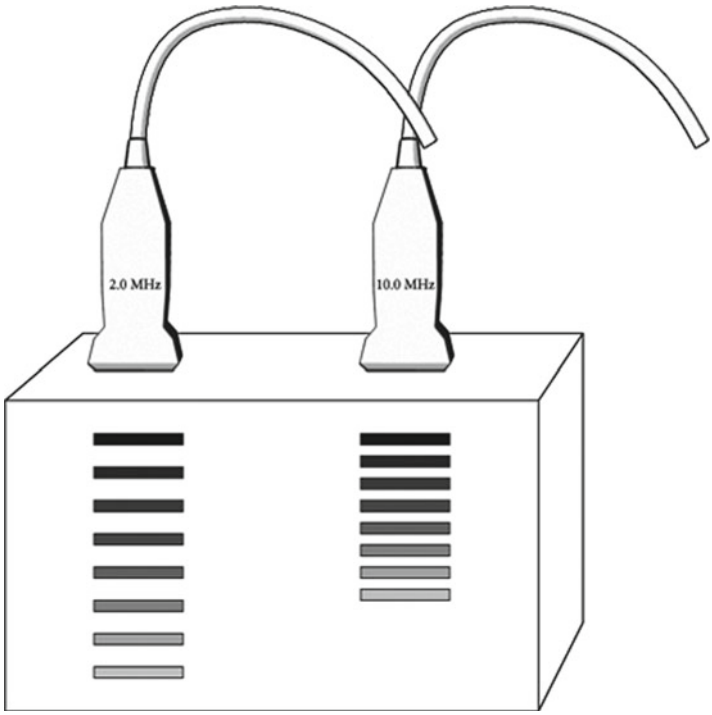

Fig. 8.1 Attenuation of the beam as a function of the frequency

Thus, at some angles of insonation, the nerve may blend into its fatty background. Changing the angle of insonation by only a few degrees (toggling) may make the difference between creating an excellent image and an image in which the nerve is invisible (Fig. 8.3). To obtain the best image, the probe should be placed on the patient's skin so that the area of interest (nerve) is in the middle of the screen, both from left to right and from top to bottom. For deep structures, compressing the tissue will make the distance from the transducer to the target nerve smaller. In this case, the beam will be attenuated less, and the nerve will be more readily imaged. Rotating or toggling the probe may also improve the image.

Modern ultrasound platforms allow the user to adjust the gain (brightness) of the image in the near field and far field independently. This allows the user to improve the image of structures at different depths in the field. The gain should be set so that the background is mostly black and the nerves of interest are white (hyperechoic). In the case where the nerves themselves are black (hypoechoic), the gain should be adjusted so that the tissue immediately around the nerve is white (hyperechoic). Many machines have an auto gain button which does this automatically for the user.

The user may also adjust the contrast of the image. The formal term for this is dynamic range compression. Most users prefer to have a large discrimination (increased contrast) between black and white structures. By adjusting the contrast,

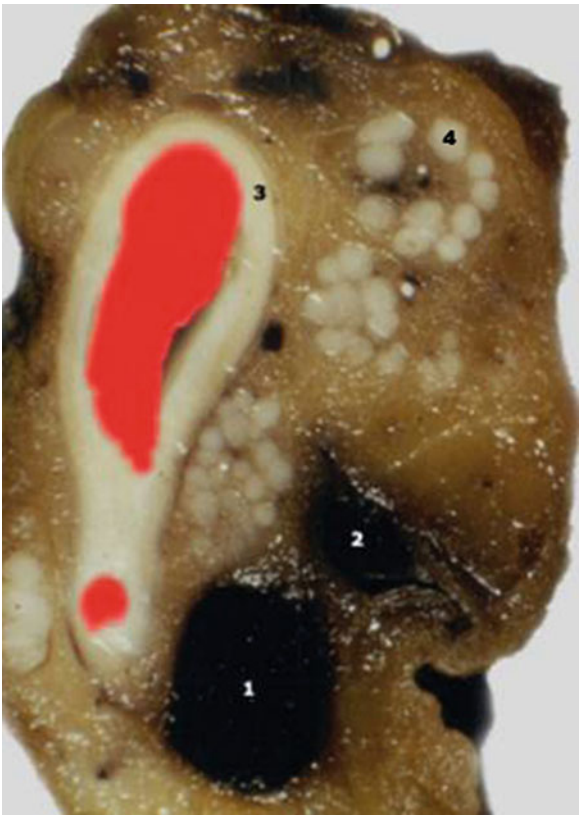

Fig. 8.2 (1) Axillary vein; (2) basilic vein; (3) wall of axillary artery; and (4) nerve fascicles

black structures (hypoechoic) become blacker and white structures (hypoechoic) become whiter.

All machines allow the user to adjust the depth to which the probe penetrates the tissue as well as the frequency of the beam. For high frequencies (10–15 MHz), the depth can be adjusted from 1.5 to 6 cm. For intermediate frequencies (5–10 MHz), the depth can be adjusted from 4 to 10 cm, and for low frequencies (2–5 MHz), the depth can be adjusted from 6 to 15 cm. The depth should be set so that the area of interest (nerve) is in the middle of the screen. Some machines adjust the focal zone as the user adjusts the depth of field setting. Other machines allow the user to set the focal length independent of the field depth (Fig. 8.4). If the user is adjusting the focal zone, the focal zone should be set at a depth that corresponds to the area of interest (nerve) in the image.

Proficiency in ultrasound-guided nerve block requires the user to distinguish nerves from bones, arteries, veins, and muscles. Arteries are black (hypoechoic) and pulsatile. They are not easily compressed. Veins are also hypoechoic but can be easily compressed. Bones are white (hyperechoic). Muscle usually has a black background with white speckles (fat) interspersed. It can be very difficult in obese patients

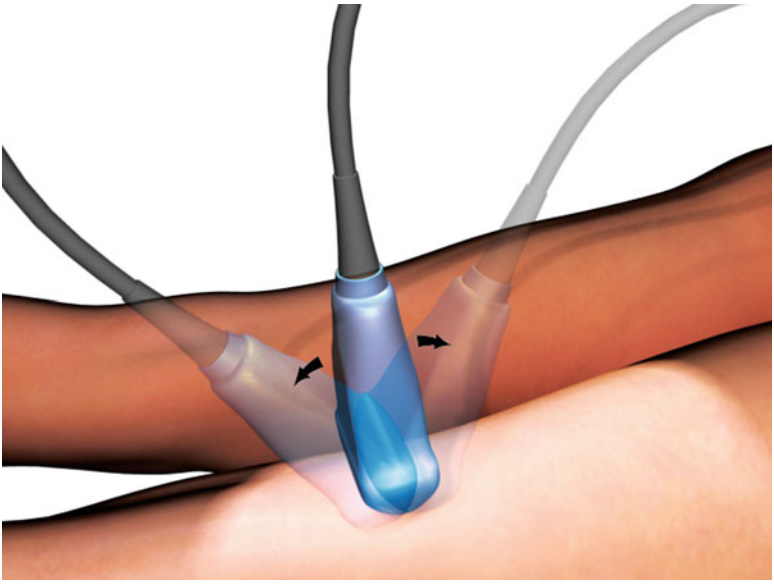

Fig. 8.3 Toggling the probe to improve the image

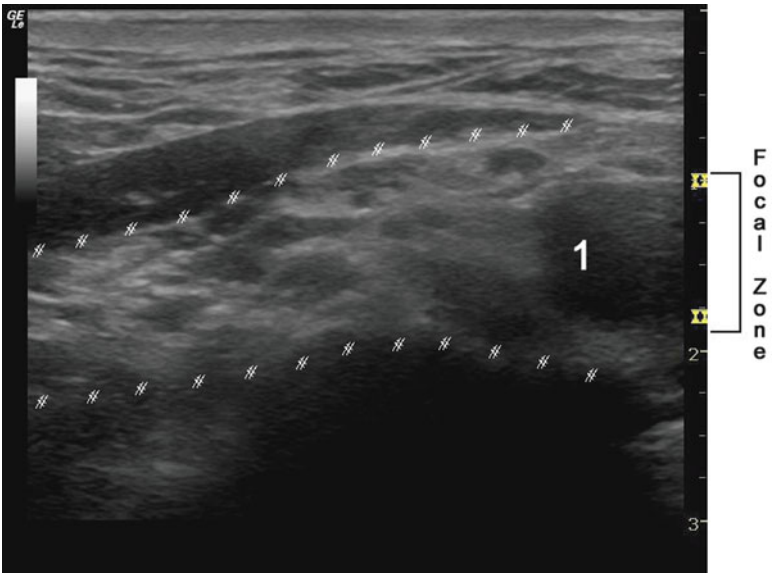

Fig. 8.4 Focal zone adjusted to the region of interest in the supraclavicular fossa

to tell the difference between muscle and nerves. In this setting, the user may wish to use a nerve stimulator to confirm the identity of the nerve. The user may also employ color flow Doppler imaging to differentiate hypoechoic nerves from vessels.

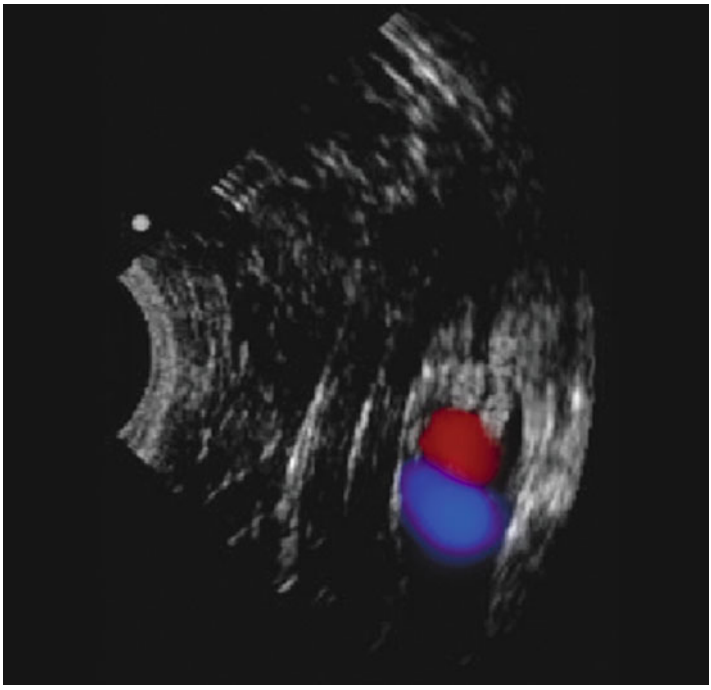

Fig. 8.5 Color flow Doppler Image. *Blue*: flow away from the probe and *red*: flow toward the probe

Blood flowing toward the transducer is always colored red. Blood flowing away from the transducer is always colored blue (Fig. 8.5). Measuring the speed of the blood flow will also help the user differentiate venous from arterial blood flow.

Transducer elements can be arranged in linear (rectangular probe) or curved arrays (curved probe). Linear arrays are used for superficial, high-resolution imaging (brachial plexus). Linear arrays form a rectangular image (Fig. 8.4). Curved arrays are used for deeper structures such as the sciatic nerve in the buttocks. These arrays form an image in the shape of a slice of pizza (Fig. 8.5). All modern machines also have a feature called compound beam imaging. This is a simple form of tomography that can greatly enhance the image of a nerve. Compound beam imaging focuses the beam on the area immediately around the structure of interest (nerve) in the middle of the screen and averages out effects due to surface scattering, refraction, and diffraction.

Once the image is formed, the user must insert a needle into the tissue adjacent to or in the nerve. In most cases, the needle will form an angle between 30 and 60° to the incident beam. In this setting, a lot of the incident beam is not reflected back to the transducer, and the needle can be difficult to image (Fig. 8.6). Usually, the operator can inject a small amount of local anesthetic to give the operator some idea of where the tip of the needle lies. This technique is called sono-location.

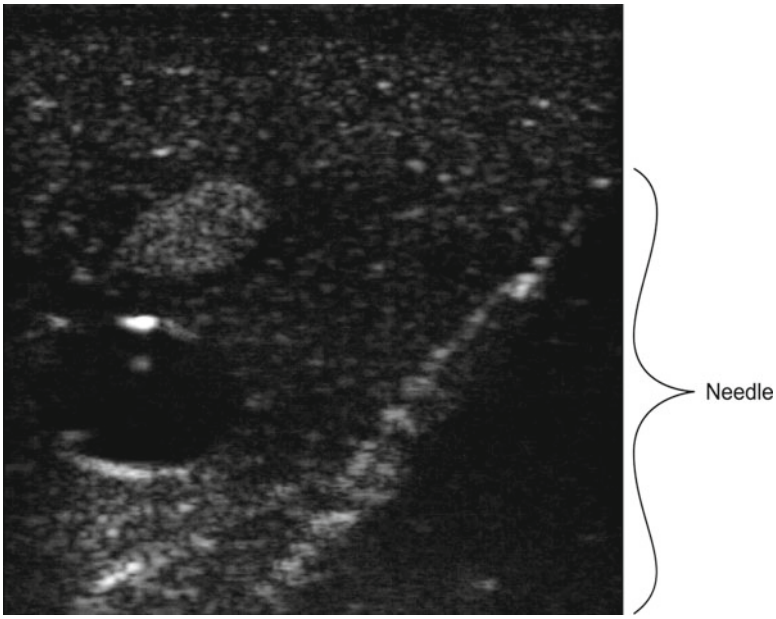

Fig. 8.6 Reflection of the ultrasound beam from a needle at 45° of insonation

Several companies (Life-Tech and Pajunk) make needles that markedly improve the image of the needle in this setting by creating a reflective texture in the surface of the needle near its tip. Another company, Ultrasonix, makes a system that uses GPS tracking to form a virtual image of the needle tip. In the Ultrasonix system, the user can always track the tip of the needle relative to any structure of interest. Other companies make needle guides that help the user keep the needle in the beam of the probe. All of these devices add to the cost of performing ultrasound-guided nerve block. These devices are particularly useful for deep blocks and for training novices. The user must weigh the utility of each device against the improvement in safety and efficacy that each device provides.

ESRA Test Questions

1. Which frequency ranges are used for ultrasound imaging of nerves?
   (a) 2–15 MHz
   (b) 2–15 GHz
   (c) 2–15 KHz
   (d) 2–15 THz
   (e) None of the above

2. Which of the following are responsible for ultrasound beam attenuation in tissue?
   (a) Frequency
   (b) Depth
   (c) Amplitude extinction coefficient
   (d) Tissue type
   (e) All of the above

3. Distortion and degradation of an ultrasound image may be caused by:
   (a) Diffraction
   (b) Refraction
   (c) Backscattering from an irregular surface
   (d) Beam dispersion
   (e) All of the above

4. The formal term for contrast adjustment in an ultrasound image is:
   (a) Amplitude gain
   (b) Dynamic range compression
   (c) Grey scale adjustment
   (d) Tissue harmonic imaging
   (e) None of the above

5. The cause of multiple images of the same needle in an ultrasound scan is due to:
   (a) Reverberation of the sound wave within the needle
   (b) Diffraction
   (c) Ghosting
   (d) Tissue anisotropy
   (e) All of the above

6. The quality of an ultrasound image may be improved by:
   (a) Toggling
   (b) Changing the frequency of the probe
   (c) Changing the focal length of the probe
   (d) Changing the dynamic range compression
   (e) All of the above

7. On color flow Doppler imaging, red indicates that the blood flow:
   (a) Is towards the probe
   (b) Away from the probe
   (c) Is arterial
   (d) Is venous
   (e) Is turbulent

8. Which tissue most strongly attenuates the ultrasound beam?
   (a) Fat
   (b) Bone
   (c) Muscle
   (d) Blood
   (e) Air

Answers:

1. a
2. e
3. e
4. b
5. e
6. e
7. a
8. b

9

Ultrasound-Guided Peripheral Nerve Blockade

David M. Polaner • Alan Bielsky

Contents

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Introduction

The introduction of ultrasonography into peripheral nerve blockade techniques has provided today's anesthesiologist with a new tool for managing a myriad of perioperative situations. Due to the wide range of blocks performed, and the inability to blind observers when studying blocks, it is difficult to determine if ultrasound-guided blocks are better than other techniques [1]. The currently accepted opinion is that ultrasound-guided nerve blocks are certainly as effective, safe, and efficient as other techniques, including surface landmark base techniques and neurostimulation techniques. Randomized, controlled trials have assessed individual ultrasound-guided blocks and do seem to suggest a reduced risk of vascular puncture and possibly improved quality of block, onset time of block, and time to perform the block. Additionally, some investigations have suggested that ultrasound guidance may permit successful blockade with lower volumes of local anesthetics, which might have implications regarding reduced risk for toxicity [2, 3]. Nevertheless, all of these data remain preliminary, as large-scale comparison studies have yet to be performed.

Basic principles of ultrasound-guided peripheral nerve blockade require an understanding of nomenclature, physics, and descriptions of probe manipulation and the orientation of the probe relative to the needle. An ultrasound probe uses a cyclic sound pressure beam which penetrates a medium, and then measures the reflection signature, creating an image [4]. This permits the operator to visualize the inner structural details of many media, including soft tissue.

When describing an ultrasound image, one uses the terms hyperechoic and hypoechoic. Hyperechoic refers to a bright, white appearance of structures, while hypoechoic refers to a darker, duller appearance of structures. Anechoic refers to a completely dark appearance of structures. Typically, tendons, nerves, and fascia appear as hyperechoic, while fat and muscle appear as heterogeneous, hypoechoic structures. Fluid, which exists in arteries and veins, appears as anechoic. Air produces a bright, hyperechoic image (Fig. 9.1).

In order to optimize images with the tools available on ultrasound machines, one must understand some basic principles of ultrasound. The ultrasound wave frequency can be chosen both by probe selection and by changing the settings on the machine itself. Higher-frequency beams improve axial resolution, which is the ability to distinguish between two objects at different depths in line with the axis of the beam [5]. Thus, increasing the frequency of the ultrasound probe (10–13 MHz) will improve resolution of superficial structures at the expense of visualizing deeper structures. Conversely, a lower frequency will improve image quality of deeper structures at the

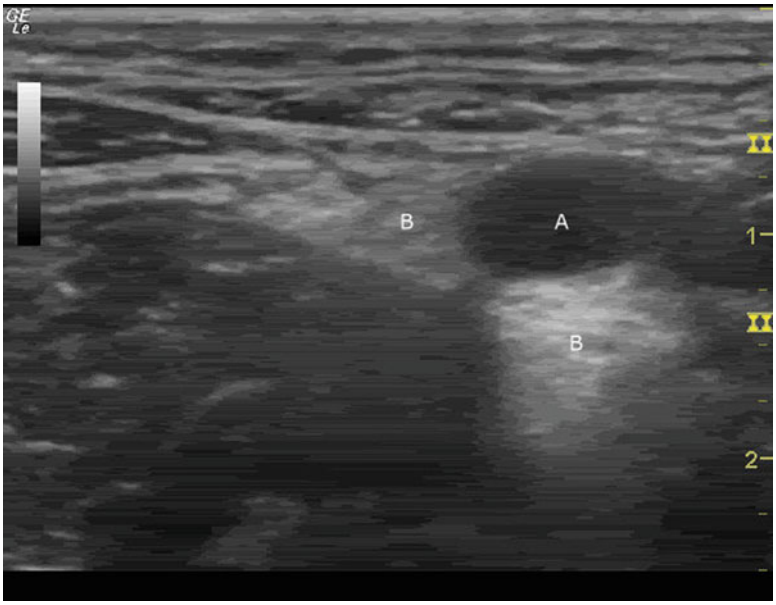

Fig. 9.1 Image of hyper- and hypoechoic. (**a**) The fluid-filled femoral artery appears as a *dark*, hypoechoic structure and (**b**) the fascial structure appears *bright*, and as such, is hyperechoic

expense of resolution of more superficial structures. The term "gain" refers to the degree to which the ultrasound machine amplifies returning ultrasound waves, making them appear brighter. Gain will increase the brightness of the entire image, but also increases background noise artifact. "Time gain compensation" is a form of gain manipulation that allows the operator to adjust the gain at specific depths in the field. Time gain compensation is useful in filtering out background noise and focusing on the depth of the target, though it may make visualization of the needle more difficult. Altering depth penetration also can be used to enhance the image. Once a target is identified, if a greater-than-necessary depth is selected, the target will appear small due to the change in aspect ratio of the image. If the depth is too shallow, the target may be obscured. The final manipulation is "focus," which allows the operator to place the focal zone of the beam at various points in the field to limit beam divergence, thereby improving lateral resolution, which is the ability to distinguish between two structures that sit side by side [6, 7].

The basic motions of probe manipulation are pressure, rotation, alignment, and tilt (Fig. 9.2). The needle direction in relation to the ultrasound beam can be described as in-plane or out-of-plane (Fig. 9.3). It is also useful, before needle placement, to establish the ultrasound probe's markings in relation to the right and left sides of the screen.

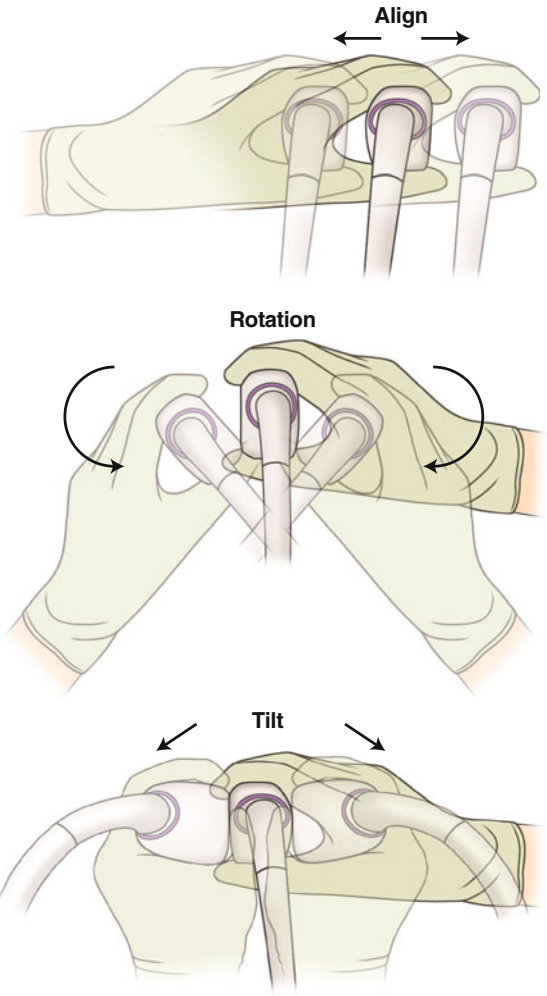

Fig. 9.2 The basic principles of ultrasound probe manipulation are pressure, rotation, alignment, and tilting

Interscalene Block

The use of ultrasound guidance in the placement of interscalene peripheral nerve blockade has been validated by studies addressing success rate, block quality, and time to perform the block [8–10]. The interscalene nerve block aims to inject local anesthetic at the level of the trunks in the brachial plexus, thereby providing anesthesia to the upper arms and shoulder.

The trunks of the brachial plexus are most effectively accessed at the level of C6, at which location cadaver studies have shown a minimum distance of 23 mm from skin to vertebral foramen [11]. Here, the plexus passes through a compartment formed by the fascia-encased anterior and middle scalene muscles (Figure. 9.4).

To perform an interscalene nerve block with ultrasound guidance, the patient is placed supine with the head tilted between 30 and 45° away from the side of the

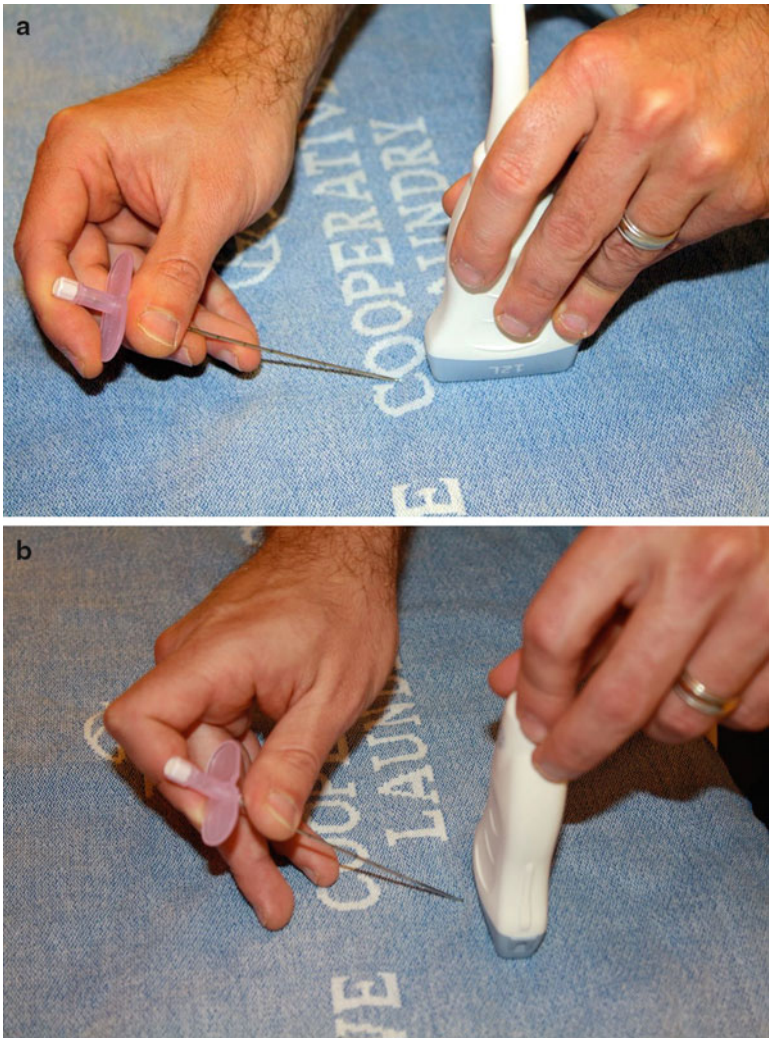

Fig. 9.3 (**a**) The needle is in-plane with the ultrasound probe. (**b**) The needle is out-of-plane with the ultrasound probe

block. After sterile preparation of the skin and the ultrasound probe, visual inspection reveals the sternocleidomastoid muscle and the thyroid prominence, which is slightly above C6 level. A linear probe is placed on the skin overlying the sternocleidomastoid at this level in an axial oblique plane in order for the ultrasound beam to transect the plexus (Fig. 9.5). Initial ultrasonographic anatomic landmarks include the sternocleidomastoid, which can be identified by its tapering appearance as one examines more laterally. The carotid artery and jugular artery can be recognized as pulsatile, hypoechoic, round structures (Fig. 9.6). Moving laterally, the anterior and middle scalene muscles appear in cross section, noticeable by their round, striated nature. In between these two muscles, potentially between their hyperechoic-appearing

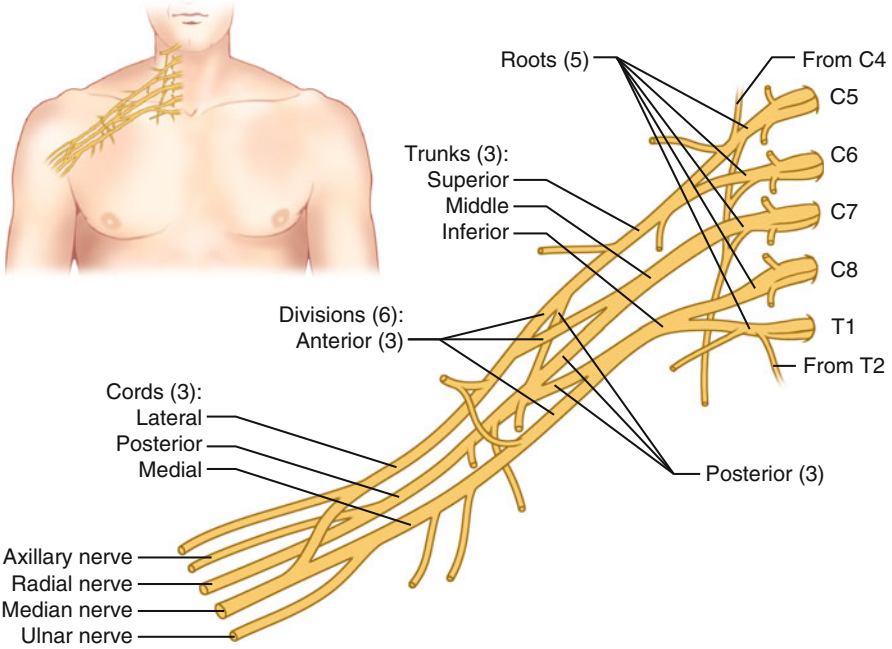

Fig. 9.4 The brachial plexus

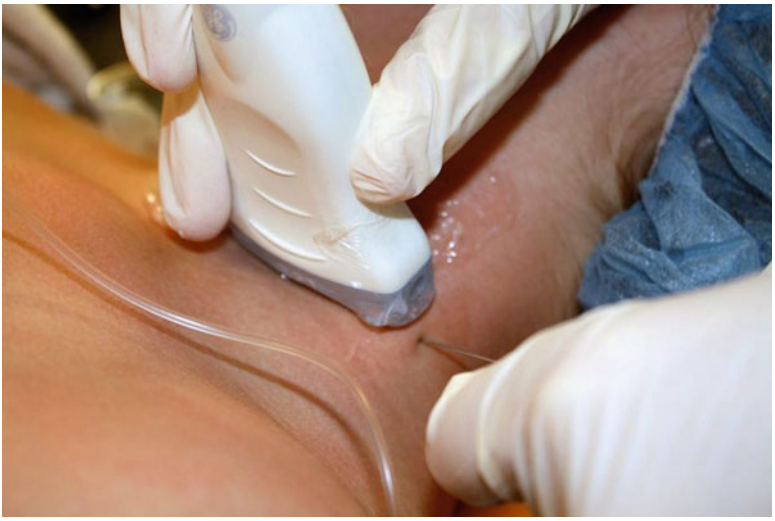

Fig. 9.5 Picture of someone doing an interscalene

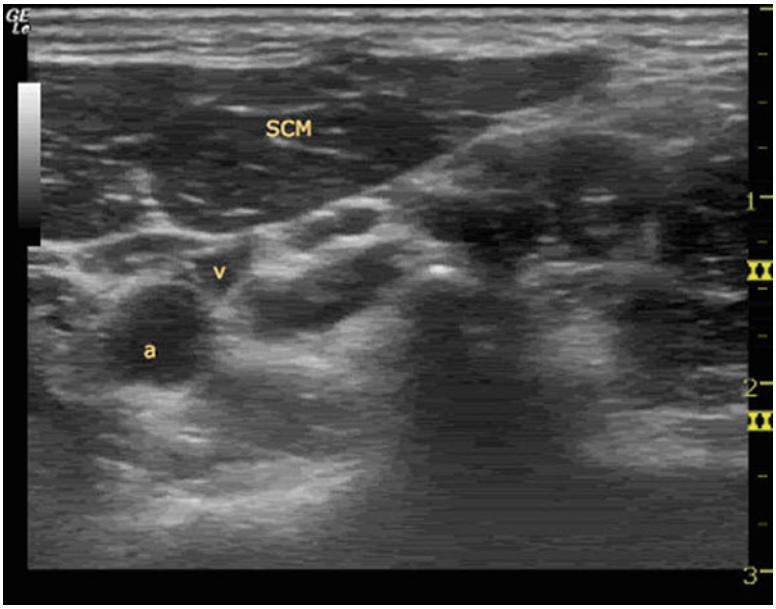

Fig. 9.6 IJ, carotid, scm. Relationship between the carotid artery (*a*), jugular vein (*v*), and sternocleidomastoid (*scm*)

investing fascia, lay the trunks of the brachial plexus. Adjustments should be made to the ultrasound image in order to maximize frequency and minimize field depth. The trunks appear as round, hypoechoic structures that may be separated by hyperechoic fascial septae (Fig. 9.7). Their stacked linear orientation has been described as resembling a snowman.

A 1- to 2-cm needle is placed at the lateral border of the linear ultrasound probe and passed medially toward the trunks under constant ultrasound visualization. The needle tip should be visualized passing lateral to the sternocleidomastoid and skirt along the medial border of the middle scalene fascia until it reaches the midpoint of the viewed trunks (Fig. 9.8). After aspiration, a small test dose of local anesthetic should be administered, with good spread being visualized around the trunks, and not in muscle or vascular tissue.

Complications from interscalene nerve blockade can be dramatic due to the proximity to major vascular and neuraxial structures. Pneumothorax, spinal cord injection with resultant permanent paralysis, epidural injection, intrathecal injection, and intravascular injection are all concerns, and although rare, have been reported [12, 13]. Additionally, neck hematoma and sepsis have been described [14]. Persistent neuropathy after interscalene block has been assessed prospectively and does occur with an incidence of between 4 and 16% within the first week after the block and only 0.1–0.2% permanently [15]. These numbers were not different for ultrasound-guided versus nerve stimulator-guided blocks. More common side effects may include a Horner's-type syndrome, transient vocal changes, and transient phrenic

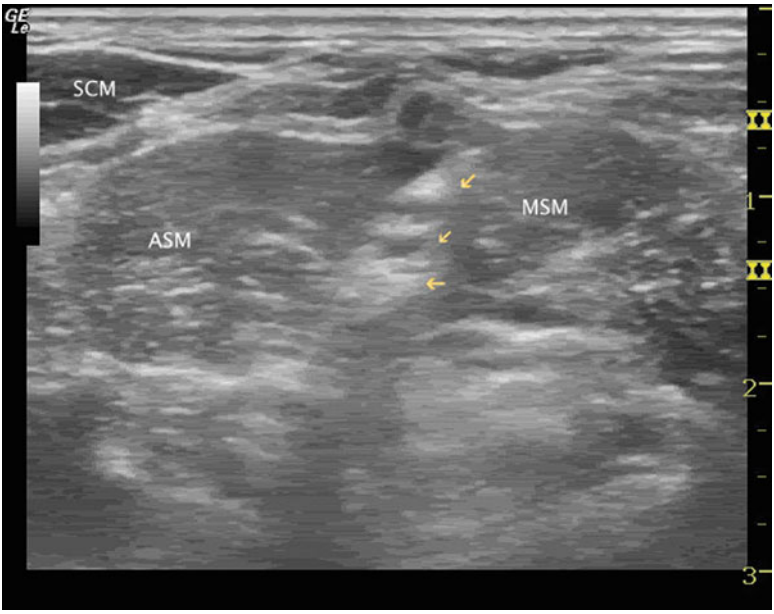

Fig. 9.7 The interscalene block. View of the trunks of the brachial plexus in the interscalene nerve block. Displayed are the sternocleidomastoid (*scm*), the anterior scalene muscle (*asm*), the middle scalene muscle (*msm*), and the trunks of the brachial plexus (*arrows*)

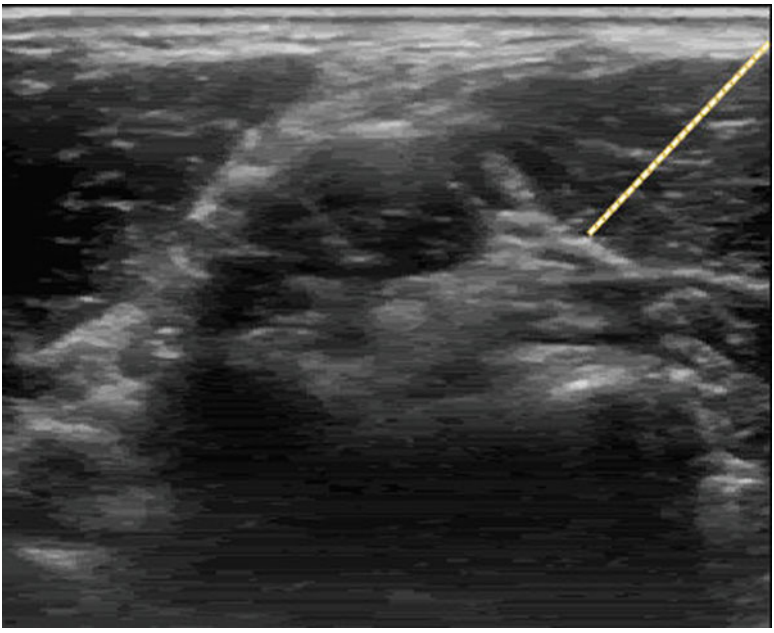

Fig. 9.8 Interscalene injection. Needle trajectory of the interscalene nerve block (*dashed line*)

blockade. As such, patients should be advised of these phenomena preoperatively, as to alleviate postoperative concerns. Whereas phrenic nerve paresis is nearly universal when nerve stimulation is used to guide needle placement and may produce significant hypoxemia or respiratory insufficiency in susceptible subjects, one recent report suggests that ultrasound guidance may dramatically reduce its incidence [16].

Supraclavicular Nerve Block

After fading away from the anesthesiologist's armamentarium due to an elevated risk of pneumothorax, the ultrasound-guided supraclavicular approach to the brachial plexus has gained widespread acceptance due to the ability to easily visualize and inject structures in this area. Subsequent analysis has shown exceedingly low incidences of pneumothorax when ultrasound is applied [17]. The supraclavicular nerve block aims to anesthetize the divisions of the brachial plexus as they pass over the first rib, under the clavicle (Fig. 9.9). Here, the divisions are located posterolateral to the subclavian artery, medial to the middle scalene muscle, and superior to

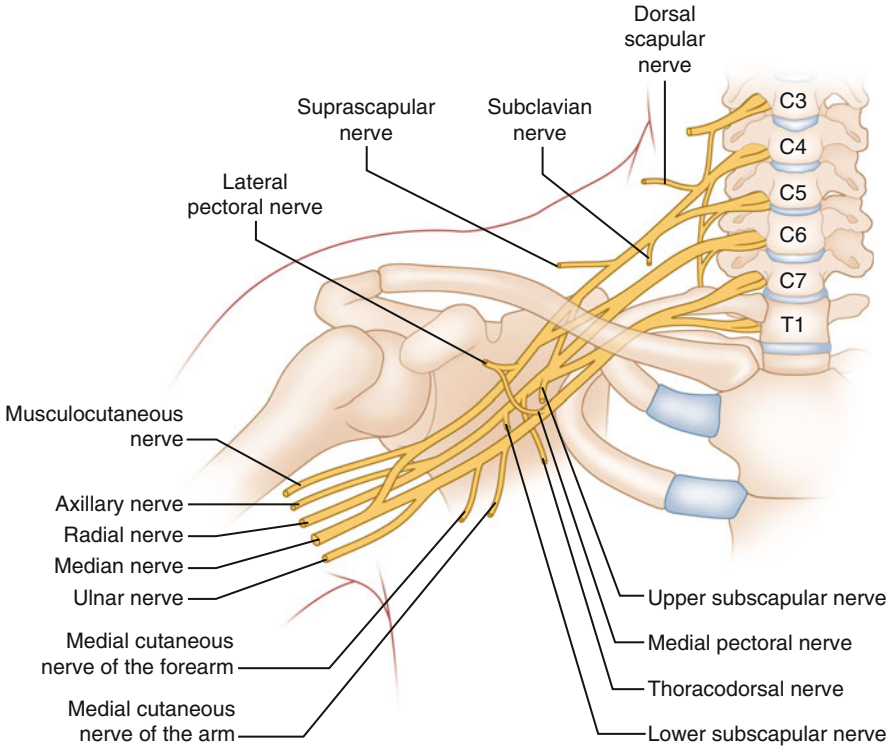

Fig. 9.9 Passage of the brachial plexus in the supraclavicular area

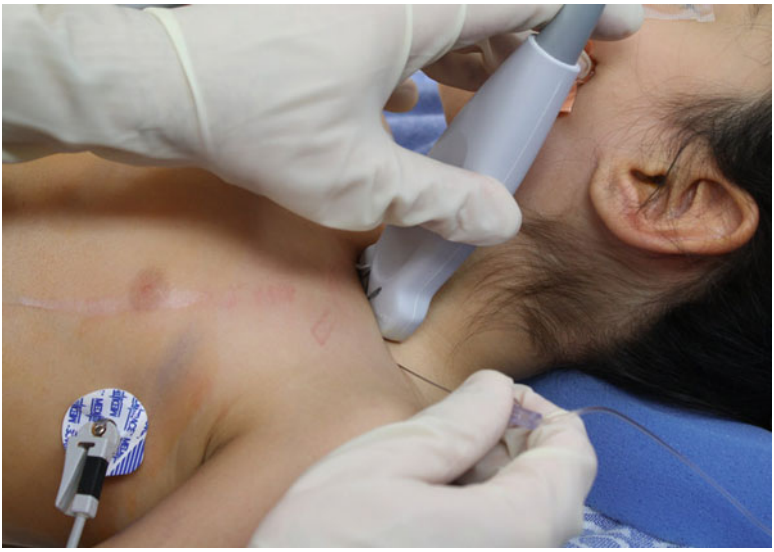

Fig. 9.10 Photo of performance of the supraclavicular block

both the first rib and the pleura [18, 19]. The supraclavicular nerve block provides fast-acting and dense anesthesia for procedures distal to the midhumerus.

To perform the block, the operator stands at either the head of the bed or facing the ipsilateral shoulder. The patient's head is turned between 30 and 45° away from the side to be blocked. After sterile preparation of the skin and probe, the supraclavicular fossa is visually identified, noting the sternocleidomastoid muscles, clavicle, and coracoid process. A high-frequency linear probe is placed in a coronal oblique plane, which can be approximated by orienting the probe roughly parallel to the clavicle. Some may find it most easy to "step off" the clavicle and let the probe seat into the supraclavicular fossa (Fig. 9.10).

Ultrasound examination begins by locating the subclavian artery in the short axis view, where, with appropriate rotation and tilting, the artery will appear as a round, pulsating, hypoechoic structure. At this point, one will also see the hypoechoic first rib underneath the artery and, possibly, the pleura. Ultrasonographically, the pleura will have a mixed pattern of hyper- and hypoechoic signals due to the presence of air in the interstitium and will move with respiration, while the rib will not. Lateral to the subclavian artery, the operator will appreciate the middle scalene, which is scanned in short axis and is notable for its often-striated appearance. In between the subclavian and the middle scalene lie the divisions of the brachial plexus, which appear as a hypoechoic, grape-cluster-like structure (Fig. 9.11).

After identification of the brachial plexus in the supraclavicular fossa, the ultrasound image is optimized by increasing frequency and decreasing image depth to focus on the plexus and the first rib. The block needle is then advanced under constant visualization in an in-plane fashion along the medial border of the middle scalene, toward the lateral portion of the plexus. In order to obtain proper needle position, it is often necessary to go through the middle scalene muscle (Fig. 9.12).

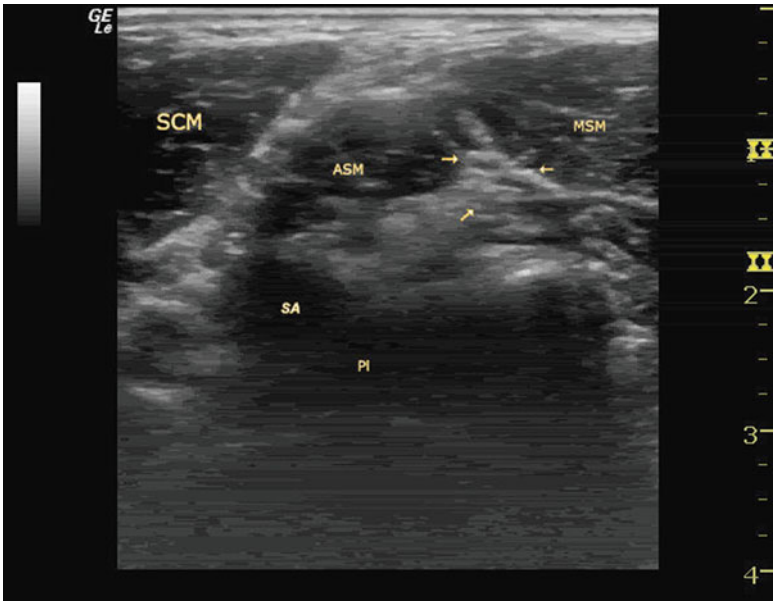

Fig. 9.11 View of the brachial plexus in the Supraclavicular fossa. Note the sternocleidomastoid (*scm*), subclavian artery (*SA*), anterior scalene muscle (*asm*), middle scalene muscle (*msm*), first rib shadow and pleura (*pl*), and the brachial plexus divisions (*arrows*)

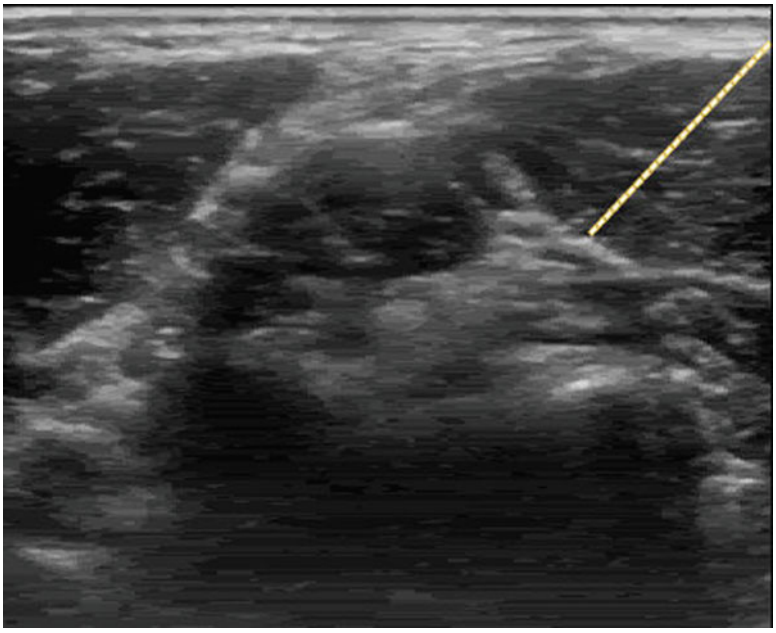

Fig. 9.12 The needle trajectory (*yellow dashed line*) for supraclavicular nerve block

Here, a test dose reveals spread of the anesthesia in the fascial layer surrounding the divisions of the brachial plexus. If indicated, subtle movements can be used to penetrate small fascial layers in order to provide adequate local anesthetic spread. An alternative approach is to use a medial to lateral approach, thereby further avoiding potential pleural puncture. In this approach, the plexus is identified in a similar fashion, but the needle is introduced in the inferomedial portion of the probe. Particular attention must be paid here to avoiding puncture of the subclavian artery, as it may be directly in the trajectory of the needle [20].

Inherent to the supraclavicular nerve block is the risk of pneumothorax, which is likely reduced by ultrasound guidance. Other risks include intravascular injection with resultant local anesthetic toxicity, neck hematoma, and abscess. In the largest cohort reported to date, the incidence of accidental vascular puncture and transient sensory deficits were both 0.4%. Horner Syndrome and hemidiaphragmatic paresis occurred in 1% and there were no pneumothoraces [17].

Infraclavicular Nerve Block

The infraclavicular nerve block targets the brachial plexus as it emerges from underneath the clavicle. Here, the lateral, medial, and posterior cords surround the axillary artery and are easily identified by ultrasound examination. This block is utilized in surgeries distal to the midhumerus. Though quite similar to the supraclavicular nerve block, it offers benefits of diminished risk of phrenic nerve blockade and ease of catheter placement [21]. The ultrasound-guided infraclavicular nerve block has shown similar, if not improved, efficacy compared with the axillary approach to the plexus as well as greater patient comfort and willingness to undergo the same procedure [22, 23].

To perform the infraclavicular nerve block, the operator preferably stands at the head of the bed. Standard monitors are applied to the patient, and general anesthesia or sedation is administered. Visual inspection reveals the sternocleidomastoid, the clavicle, and the coracoid process. It may be helpful to mark these points on the patient with a soft-tip marker. Additionally, it may be useful to abduct the arm to 90°, externally rotate the shoulder, and flex the elbow (Fig. 9.13). This action may bring the plexus closer to the skin, allowing the ultrasound image to be optimized [24].

After sterile prep, a linear ultrasound probe is placed in a coronal plane in the infraclavicular fossa. Of note, the ultrasound beam will likely require a lower frequency (8–10 MHz) as it must penetrate deeper than for other blocks. The hypoechoic clavicle is identified, and then the axillary artery is visualized in cross section. Rotation and tilting may be used to develop the artery's round, pulsating image. Once this is achieved, one can appreciate the two muscle layers immediately anterior to the artery, comprised of the pectoralis major and pectoralis minor. The image depth and frequency are then optimized. It is important to orient oneself to the location of the beam, specifically which side is caudal and which side is rostral. Other landmarks to note may be the heterogeneous lung pleura, which move with inspiration.

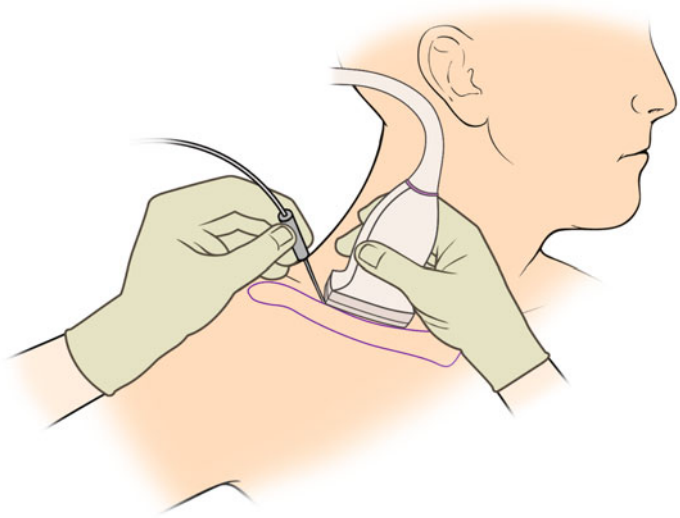

Fig. 9.13 Performing the supraclavicular block

In the infraclavicular fossa, the cords of the brachial plexus appear as hyperechoic starlike structures surrounding the hypoechoic axillary artery. Vincent Chan classically described the positions of the nerve cords in relation to the axillary artery in terms of a clock face with the lateral cord located cranially at 09:00 h, the posterior cord between 06:00 and 07:00 h, and the medial cord lying at 04:00–05:00 h, often between the axillary artery and vein (Figs. 9.14 and 9.15) [20, 25].

Using an in-plane approach, a needle is guided from either the rostral or caudal end of the ultrasound probe, under constant visualization. Of note, it is often easier to approach from the rostral end of the probe, as a 45° angle is usually sufficient to reach the posterior cord initially, and after injection, draw back to the lateral cord and inject further local anesthetic. When the needle reaches the posterior cord, injection may result in the "double bubble sign," which consists of the hypoechoic axillary artery anteriorly and the spreading local anesthetic posteriorly [23]. One may see additional spread of the local anesthetic in a U-shaped fashion along the posterior border of the axillary artery.

Complications associated with the infraclavicular nerve block include hematoma, infection, vascular puncture, and local anesthetic toxicity. Pneumothorax is avoided by maintaining the needle in the sagittal plane and avoiding medial movement. Minor dysesthesia has been noted in 2% of patients in large cohorts, though permanent nerve injury is only rarely described following this block [21, 22].

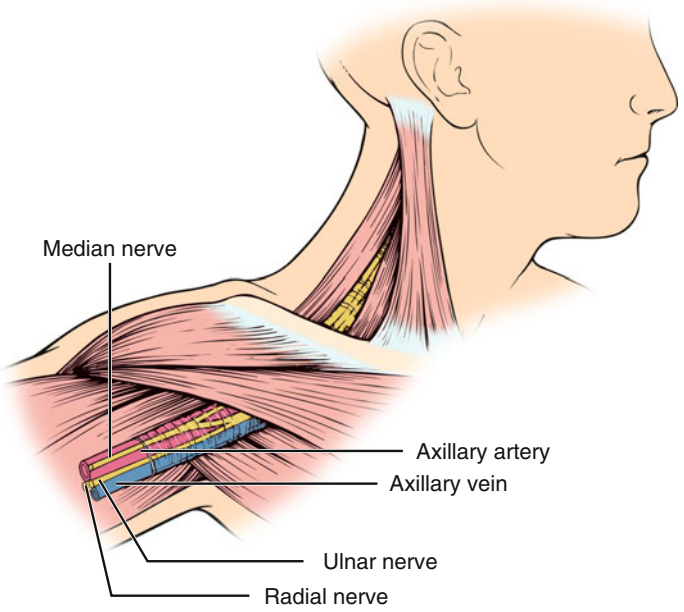

Fig. 9.14 Location of the cords around the axillary artery

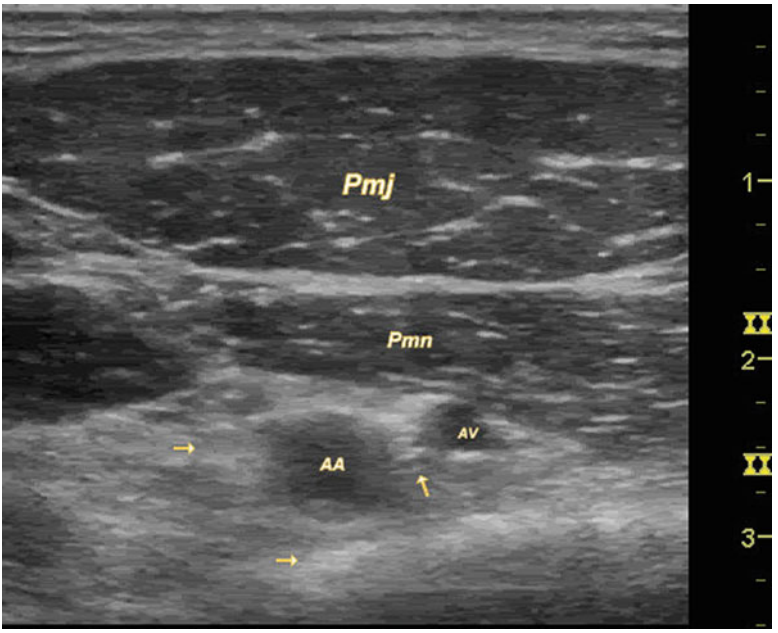

Fig. 9.15 Ultrasound appearance of the infraclavicular nerve block. The pectoralis major (*pmj*) and pectoralis minor (*pmn*) cover the brachial plexus noted by *arrows*, which surrounds the axillary artery (*AA*). The small axillary vein (*AV*) is located caudal to the artery

Axillary Nerve Block

A time-tested approach to the brachial plexus exists in the axillary nerve block. Here, the brachial plexus is blocked at the level of terminal nerves as they pass through the axilla. This technique lends itself to ultrasound guidance due to the superficial orientation of the plexus. Of note, this is an excellent "starter" block for newcomers to ultrasound-guided blocks, due to the easy visualization of structures, the ability to handle the needle with multiple passes under ultrasound guidance, and the lack of critical structures to avoid. Much like the infraclavicular block, the axillary nerve block provides anesthesia for extremities distal to the midhumerus.

To perform the axillary nerve block, the arm is abducted to 90° and externally rotated. After sterile prep, a linear probe is placed in a parasagittal orientation in the axilla (Fig. 9.16). On initial exam, one notices the striated biceps and triceps muscles and the pulsating axillary artery. As the probe is centered over the axillary artery, the image is optimized by increasing frequency, adjusting gain, and reducing depth. The median, radial, and ulnar nerves are visualized as heterogeneous, typically honeycomb-shaped structures around the artery. While there does tend to be variation in location of the nerves, the median nerve is located most laterally in close proximity to the biceps, the radial nerve tends to lie deep to the axillary artery, and the ulnar nerve lies medially, close to both the triceps muscle and the axillary vein (Fig. 9.17) [26].

An in-plane approach is used to guide a needle to the plexus. There seems to be a consensus that multiple injection passes are needed to provide adequate anesthesia to the distal upper extremity [27–29]. Typically, one may insert the needle from the lateral aspect of the brachial plexus and advance the needle to the radial nerve,

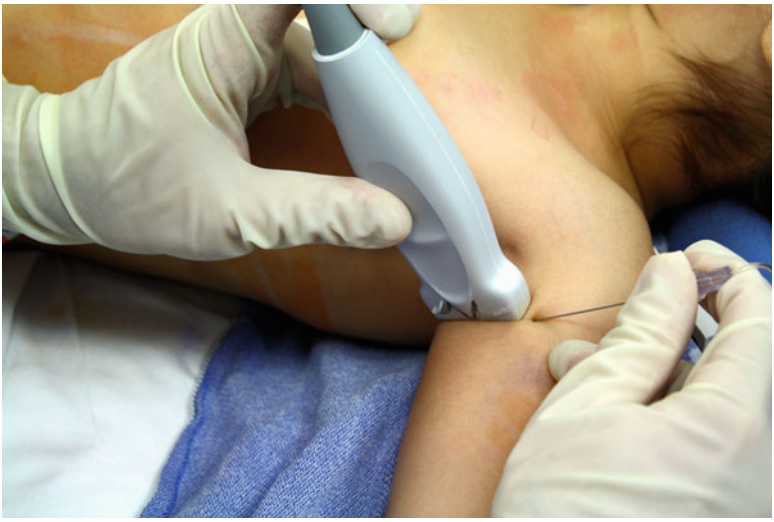

Fig. 9.16 Performing the axillary nerve block

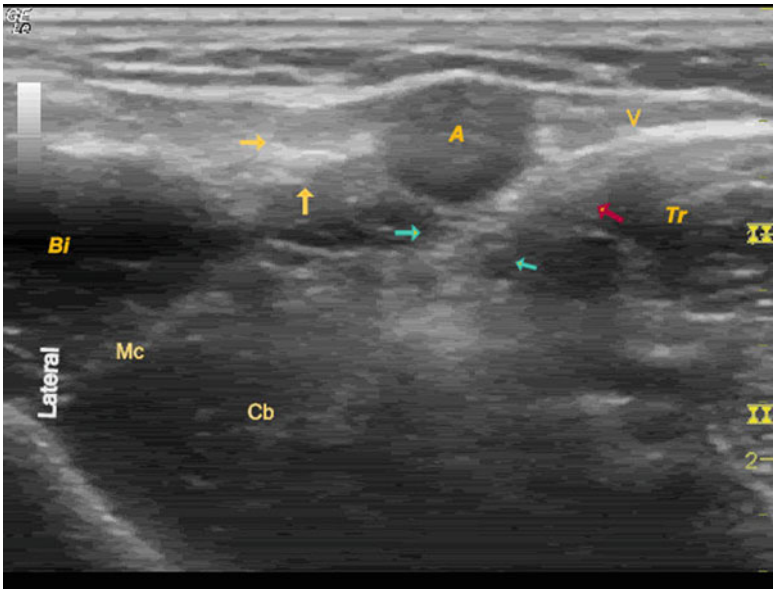

Fig. 9.17 The ultrasonography of the axillary nerve block. The axillary artery (*A*) is located centrally, bounded laterally by the biceps muscle (*Bi*) and medially by the triceps muscle (*Tr*) and compressible axillary vein (*V*). The terminal nerves surrounding the artery are the median nerve (*yellow arrows*), the radial nerve (*blue arrows*), and the ulnar nerve (*red arrow*). The musculocutaneous nerve (*Mc*) is separately blocked as it runs in between the biceps and the coracobrachialis (*Cb*)

which lies underneath the axillary artery, typically at a 06:00 h position. The needle is then withdrawn to the 09:00 h position, where the median nerve is injected. The current evidence suggests that selective ulnar nerve injection is not necessary for block success because diffusion of the solution within tissue planes will produce adequate blockade of that nerve [27, 30]. Block success seems to be improved when it is easy to visualize the spread of hypoechoic local anesthetic around the nerve bundles [20].

A specific problem frequently encountered with the axillary plexus block is the early (proximal) exit of the musculocutaneous nerve, which innervates the surface of the medial arm. This frequently necessitates the direct blocking of the musculocutaneous nerve, which courses through the body of the coracobrachialis muscle. To block the nerve here, one simply places the probe on the coracobrachialis muscle in a cross-sectional fashion and finds the nerve by its hyperechoic signature within the muscle mass. A small volume of local anesthetic then is placed at this site [31].

Complications of the axillary nerve block include hematoma, infection, vascular puncture, and local anesthetic toxicity. Nerve injury from this block is exceedingly rare [21].

Femoral Nerve Block

The femoral nerve block is utilized for procedures involving the anterior thigh and knee. This block is easy to perform given the size of the nerve sheath and its adjacent structures and finds many uses in orthopedic practice. The femoral nerve is blocked in the proximal thigh as it exits below the inguinal ligament and above both the psoas and iliacus muscles. It is easily identifiable as it courses laterally to the femoral artery, surrounded in a triangular fashion by the artery and iliopectinate ligament medially, fascia lata superiorly, and iliacus inferiorly (Fig. 9.18).

To perform the block, the patient is placed supine, with the legs in a neutral position. After sterile preparation, a linear probe is placed in a transverse orientation just distal to the inguinal ligament (Fig. 9.19). The first and most prominent landmark is the pulsating, hypoechoic femoral artery. The operator then moves the probe distally to observe the takeoff of the profunda femoris artery from the femoral artery. Blockade of the femoral nerve should be proximal to this landmark. The nerve is identified as a hyperechoic, heterogeneous structure located laterally to the artery. It often appears as a "comet trail," which is due to its surrounding fascia lata and iliacus muscle [32]. If difficulty is encountered visualizing the nerve, the probe can be tilted in a caudal-rostral fashion until the image improves (Fig. 9.20) [33].

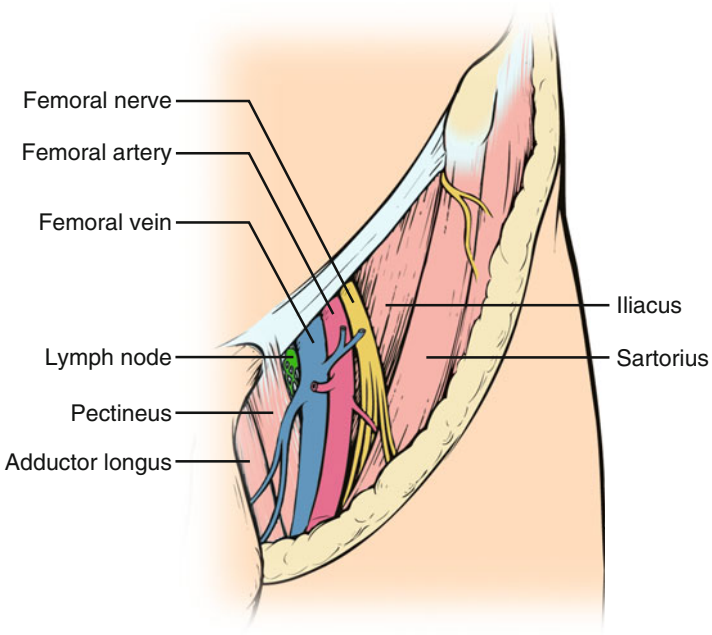

Fig. 9.18 The femoral nerve and its relation to the artery and vein

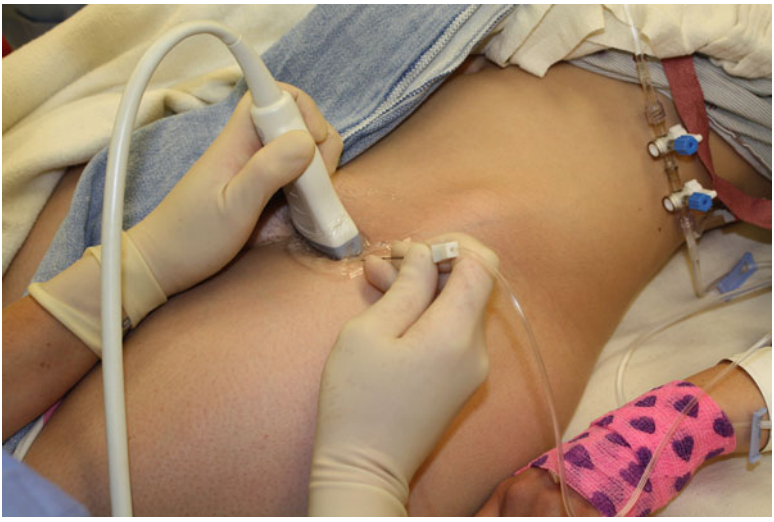

Fig. 9.19 Performing the femoral nerve block

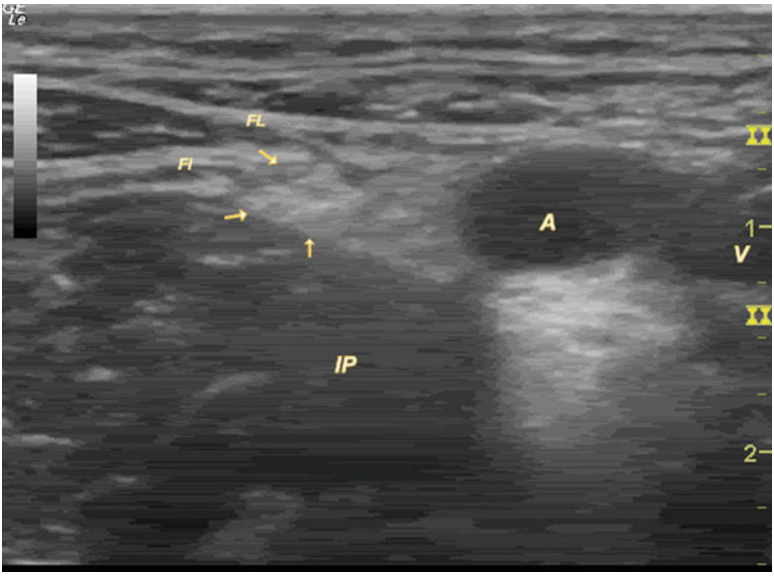

Fig. 9.20 Ultrasonographic appearance of the femoral nerve. The femoral nerve (*arrows*) is observed superior to the iliopsoas muscle (*IP*) and lateral to the femoral artery (*A*) and femoral vein (*V*). It is encased by the fascia iliaca (*FI*) and bounded superiorly by the fascia lata (*FL*)

The image is optimized using frequencies between 8 and 10 MHz, by adjusting depth and gain. In an in-plane approach, the needle is passed to the inferolateral border of the "comet tail." Here, local anesthetic is deposited. Frequently, with injection, the round, hyperechoic nerve becomes more visible. If there is inadequate

spread of local anesthetic medially, one can reposition the needle on top of the nerve and continue injection, though it is wise to stay below the hyperechoic, linear-appearing fascia iliaca.

Complications of the femoral nerve block include hematoma, abscess, vascular puncture, and local anesthetic toxicity. Transient and permanent nerve injury after femoral nerve blockade is exceedingly rare [21].

Sciatic Nerve Block

While it is a large nerve, ultrasound imaging of the sciatic nerve is impeded by its depth and by the large amounts of tissues that surround it in its proximal region. For this reason, one must consider whether a proximal or distal sciatic nerve block is appropriate. If anesthesia of the posterior thigh is required, a proximal sciatic block should be performed. If anesthesia is only required below the knee, a popliteal fossa sciatic nerve block is sufficient.

The sciatic nerve is formed from the L4, L5, and S1–S3 nerve roots, leaving the pelvis via the greater sciatic foramen, deep to the gluteus maximus, and along the medial side of the femur [34]. The sciatic nerve splits in the popliteal fossa to form the tibial and peroneal nerves, which provides innervation to the lower extremity distal to the knee. As such, the sciatic nerve can be blocked proximally, near the gluteus maximus, or distally in the popliteal fossa.

Sciatic nerve blockade near the gluteus maximus poses challenges of depth to anesthesia guidance. To perform the block at this level, the patient is placed in a lateral position with the hips and knees flexed. In order to obtain an image, a lower-frequency (2–5 MHz) curved array probe is placed just inferior to the buttock in line with the ischial tuberosity and greater trochanter [35]. Here the sciatic nerve will be found deep to the gluteus maximus and medial to the ischial tuberosity. The depth of the nerve varies between 3 and 5 cm. The nerve appears as an elliptical hyperechoic structure that may be difficult to distinguish from the fascia surrounding the gluteus maximus (Fig. 9.21). For this reason, a stimulating needle may be of benefit in confirming the position [36]. Using an in-plane or out-of-plane technique, an insulated stimulating needle is passed to the nerve. It is wise to anticipate that a longer-length needle may be needed. Once the needle has reached the sciatic nerve, neurostimulation may reveal the need for readjustment.

At the midthigh, the sciatic nerve becomes more superficial, though it may still be difficult to see in patients with more muscle or adipose mass. In this setting, a lower-frequency (6–10 MHz) linear probe is used. The patient's leg is flexed at the knee and positioned so that the posterior aspect of the thigh is accessible to probe placement. The probe is then applied laterally to the posterior aspect of the thigh. Anatomic structures of note in the view are the biceps femoris, adductor magnus, semitendinosus muscle, and semitendinosus muscle (Fig. 9.22). The sciatic nerve appears as a honeycombed oval or elliptical structure deep to these muscles [36, 37]. If further confirmation is needed, one can use the "scan down-scan up" technique in which the operator scans, in the same plane, distally to observe the bifurcation of

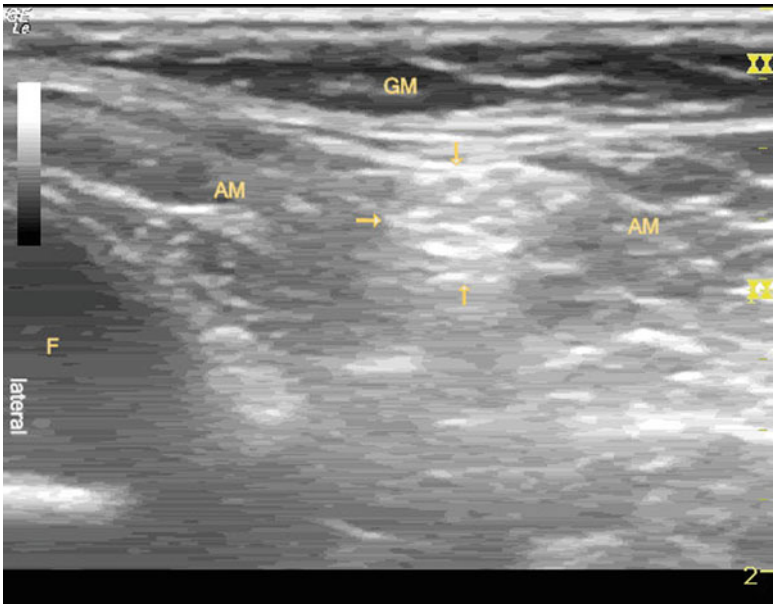

Fig. 9.21 The proximal sciatic nerve. The sciatic nerve (*arrows*) is bounded superficially by the gluteus maximus (*GM*) and deeper by the adductor magnus muscles (*AM*). The hypoechoic femur (*F*) serves as the lateral point of reference

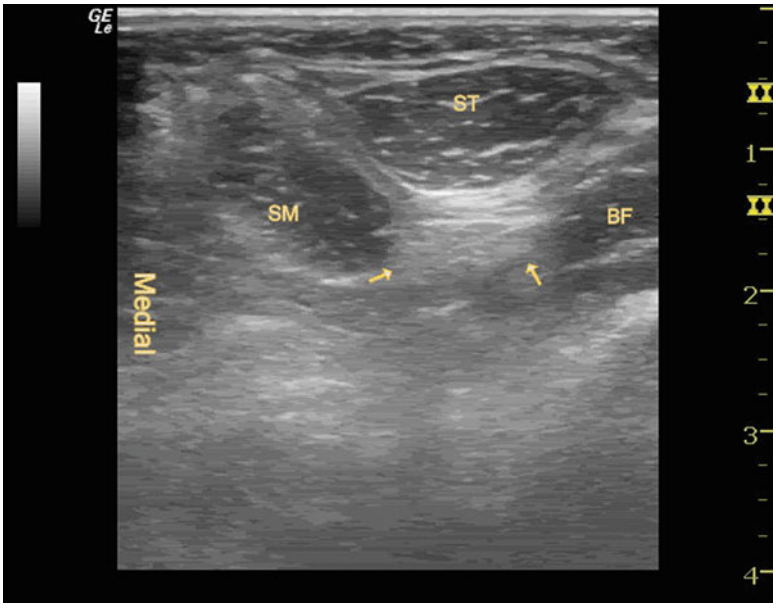

Fig. 9.22 The midthigh sciatic nerve. The sciatic nerve (*arrows*) lying deep to the semimembranosus (*SM*), semitendinosus (*ST*), and biceps femoris (*BF*)

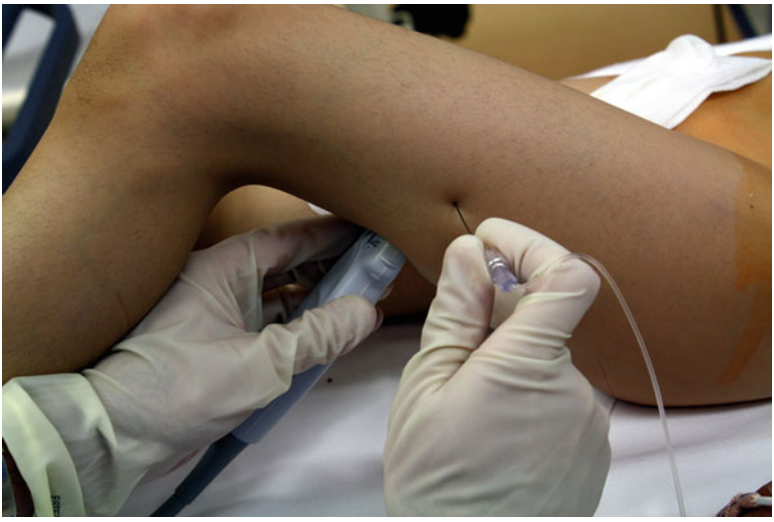

Fig. 9.23 The trans-vastus approach for needle placement

the sciatic nerve into the tibial and peroneal nerves. The "scan back" portion then tracks the nerve proximally and proceeds with needle placement. Needle insertion can be in-plane or out-of-plane. It is often useful to use an in-plane, trans-vastus lateralis approach. For this technique, the needle is inserted in-plane and passed in a lateral fashion from the side of the thigh (Fig. 9.23). An advantage of the trans-vastus approach is that the needle angle remains constant, allowing the operator to optimize the needle image and then advance in that optimized plane.

The popliteal fossa provides an easy location to perform sciatic nerve blockade, though it will not provide anesthesia to the posterior thigh proximally to the popliteal fossa. Here, the thick muscular tissues part, allowing easy visualization of the sciatic nerve. The patient is placed in either a lateral position or a supine position with the hip flexed and the knee flexed. An 8- to 10-MHz probe is placed transversely at the posterior portion of the popliteal crease. The tendons of the semimebranosus and semitendinosus are identified medially, and the biceps femoris is located laterally. Additional landmarks noted by ultrasound exam include the anechoic, pulsatile popliteal artery and the compressible popliteal vein. The vessels are located medially to the sciatic nerve, which appears as a hyperechoic, round structure with hypoechoic honeycombing (Fig. 9.24) [18]. Needle choice is made, and the needle can be introduced in an in-plane or out-of-plane fashion. Again, as in the midthigh region, the in-plane needle insertion can be performed in a trans-vastus approach or in an angular approach from the side of the probe. The "scan back" technique is often useful for this approach as well, noting the separate tibial and peroneal nerves and then moving the transducer proximally until their point of bifurcation from the single sciatic nerve is identified (Fig. 9.25).

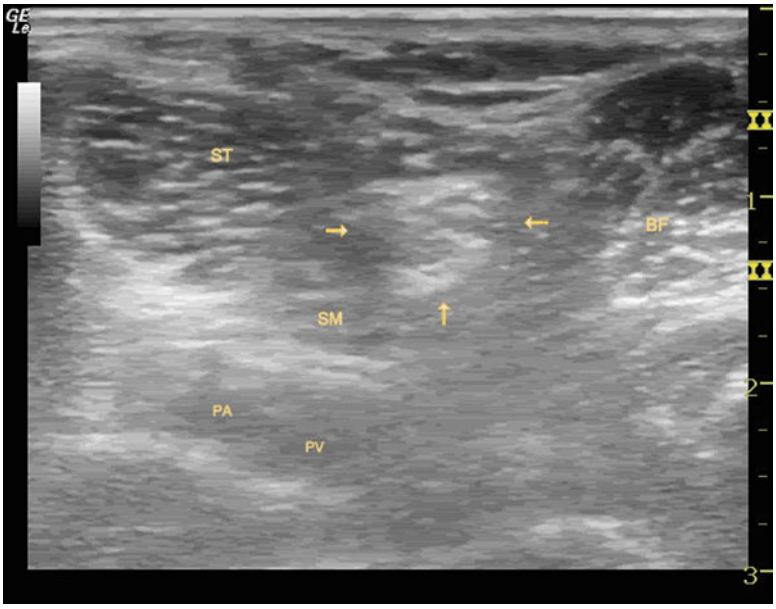

Fig. 9.24 The popliteal fossa. The sciatic nerve (*arrows*) is bounded laterally by the semitendinosus (*ST*) and semimembranosus (*SM*) muscles and medially by the biceps femoris muscles (*BF*). The popliteal artery (*PA*) and popliteal vein (*PV*) are also seen

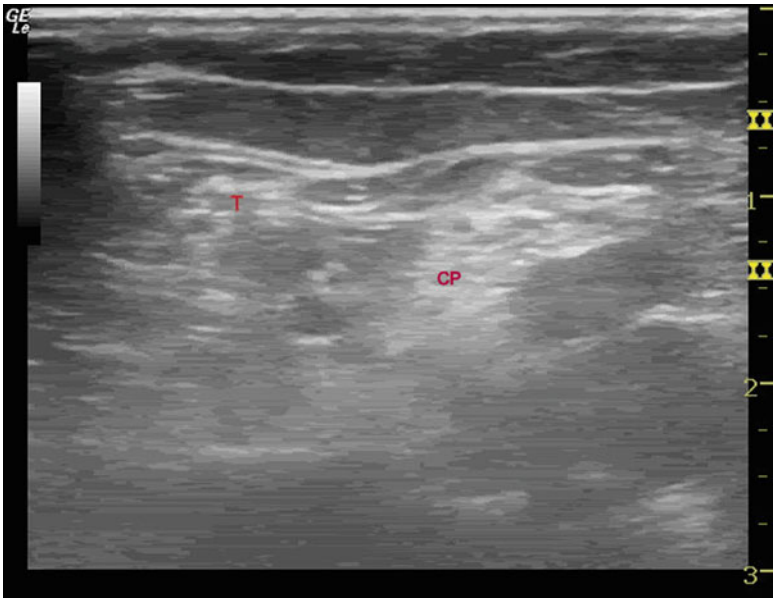

Fig. 9.25 The bifurcation of the sciatic nerve into the common peroneal (*CP*) and tibial nerves (*T*)

Complications

Complications of the sciatic nerve block include hematoma, abscess, vascular puncture, and local anesthetic toxicity. Transient and permanent nerve injury after sciatic nerve blockade is exceedingly low [21].

Saphenous Nerve Block

The saphenous nerve, a terminal branch of the posterior division of the femoral nerve, provides innervation to the medial aspect of the upper thigh, lower leg, ankle, and foot [38]. As the nerve exits the adductor canal, it courses along with the saphenous branch of the descending genicular artery [39]. The nerve then gives off an infrapatellar branch innervating the knee, and a sartorial branch, which courses to the posterior portion of the knee.

The saphenous nerve is easily blocked in the midthigh, owing to its course along with the sartorius muscle, the femoral artery, and more distally, the descending genicular artery. The patient is placed supine, and after sterile prep, the probe is placed in a transverse fashion on the anterior thigh, midway between the inguinal crease and knee. Typically, owing to the deeper location of the nerve, a frequency between 6 and 1 MHz is selected. Initial ultrasound examination will identify, most superficially and medially, the sartorius muscle, and laterally and deeper, the vastus medialis. The nerve can be identified as it courses laterally and deep to the sartorius muscle, in an anterolateral relation to the femoral artery. Here, the nerve will appear as a hyperechoic, starlike structure that directly abuts the artery (Fig. 9.26). If difficulty is encountered, the probe can be moved more distally in order to attempt to image the nerve as it exits the adductor canal, though it may change its orientation to both the sartorius and the descending genicular artery in this view. The nerve can also be identified by a “scan back technique” in which the operator locates the femoral nerve and artery in the inguinal crease and then traces these structures down to the midthigh, thereby identifying the saphenous nerve [40].

Needle selection is based on the depth of the nerve on ultrasound examination, and an in-plane approach is used from the lateral edge of the ultrasound probe. After the needle reaches the saphenous nerve, aspiration is performed to exclude vascular placement, and local anesthetic is injected incrementally. At times, the needle may be repositioned to ensure injection into the fascial sheath running in between the vastus medialis and the sartorius.

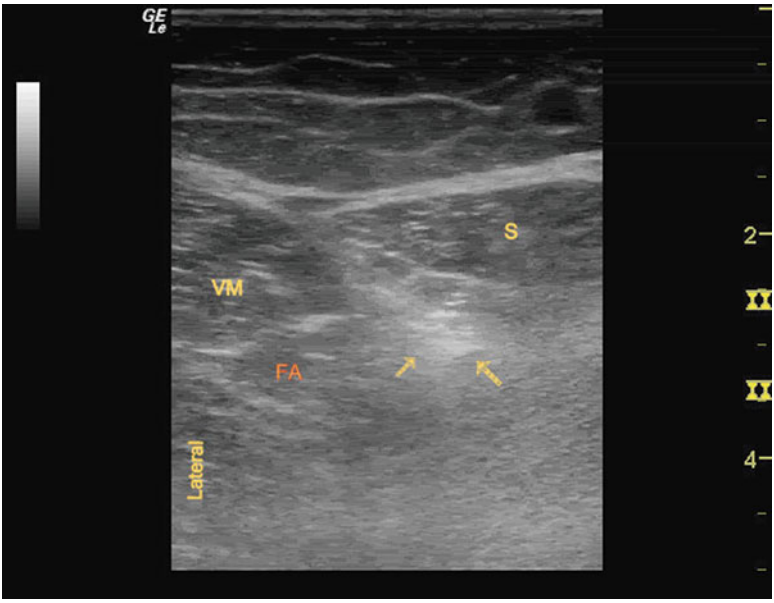

Fig. 9.26 The trans-sartorial saphenous block. The saphenous nerve (*arrows*) in relation to the vastus medialis (*VM*), the femoral artery (*FA*), and the sartorius (*S*)

Truncal Blocks

Ilioinguinal Iliohypogastric Nerve Block

The ilioinguinal nerve provides the upper medial part of the thigh and the upper part of the genitalia, while the iliohypogastric nerve provides sensation to the buttock and abdominal wall above the pubis [41]. As such, the ilioinguinal iliohypogastric nerve block (IIIHNB) can provide analgesia for procedures including inguinal hernia repair, orchiopexy, and hydrocele repair [41]. The ilioinguinal and iliohypogastric nerves are terminal portions of the L1 root that emerge from the lateral border of the psoas major muscle, cross the quadratus lumborum muscle obliquely, and perforate the transverse abdominis muscle, where they course together in the plane between the internal oblique and transverse abdominis (Fig. 9.27) [20].

The nerve block is performed with the patient supine. After sterile prep of the skin and probe, the probe is placed in a transverse fashion directly medial to the anterior superior iliac spine (ASIS). Here it is useful to tilt and rotate the probe so that it runs parallel to a line drawn between the ASIS and umbilicus. The operator then gently moves the probe medially, "rolling off" the ASIS. Ultrasound examination will reveal, laterally, the hypoechoic shadow of the ASIS and the three layers of musculature including the external oblique, internal oblique, and transverse abdominis. Below the transverse abdominis, the bowel can be seen. The ilioinguinal and

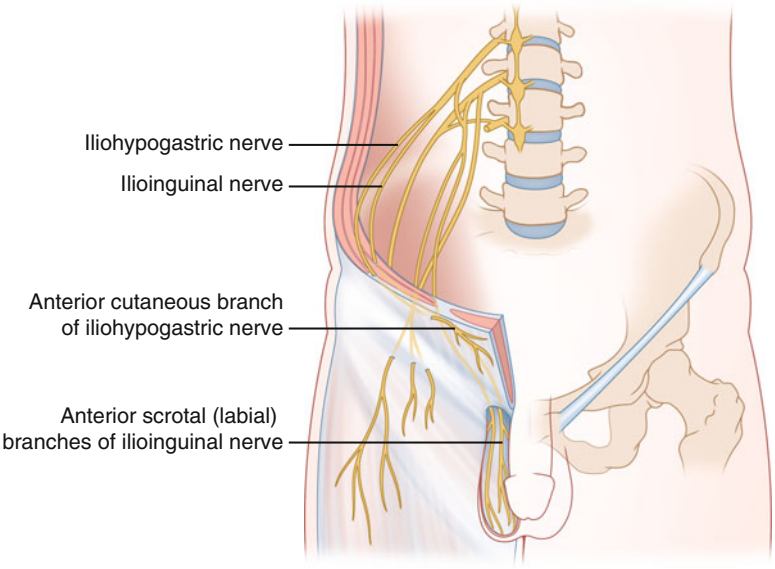

Fig. 9.27 Cartoon of the course of the ilioinguinal and iliohypogastric nerves

iliohypogastric nerves can be visualized as elliptical honeycomb structures that run between the internal oblique and transverse abdominis (Fig. 9.28). They can often be mistaken for vascular structures due to their round, hypoechoic centers.

After frequency, depth, and gain are adjusted to optimize the image, a needle is passed from the lateral border of the probe toward the nerve (Fig. 9.29). After an aspiration is performed to rule out vascular placement of the needle, a small test dose is injected. Ultrasound examination of correct placement reveals a lemon-shaped appearance of local spread around the nerve bundle. Incorrect placement of the needle will often result in a round appearance of local anesthetic spread, typical of intramuscular injection.

Specific complications associated with IIIHNB are bowel hematoma, bowel puncture, pelvic hematoma, femoral nerve block, and local anesthetic toxicity [41]. As such, it is prudent to constantly visualize the tip of the needle with specific attention to the depth of both the needle and bowel contents.

Transverse Abdominis Plane Block

The transverse abdominis plane (TAP) block can be used as an analgesic supplement in procedures involving the abdominal wall and anterior parietal peritoneum [41]. The transverse abdominis plane exists between the internal oblique and transverse abdominis muscles and consists of an interconnected plexus of nerves comprised of the somatic afferents of T8–L1 (Fig. 9.30) [42, 43].

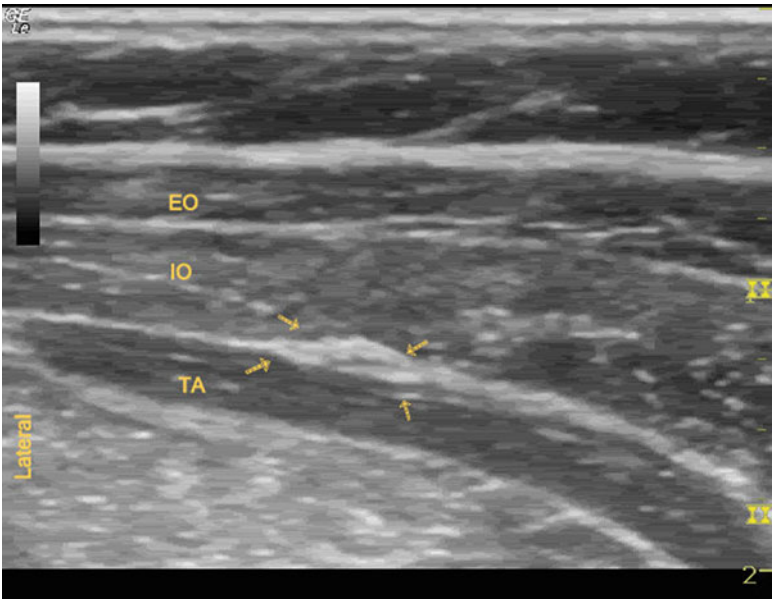

Fig. 9.28 Ultrasound of the ilioinguinal and iliohypogastric nerves. The ilioinguinal and iliohypogastric nerves (*arrows*) lie in between the internal oblique (*IO*) and transverse abdominis muscle (*TA*). Also pictured is the external oblique

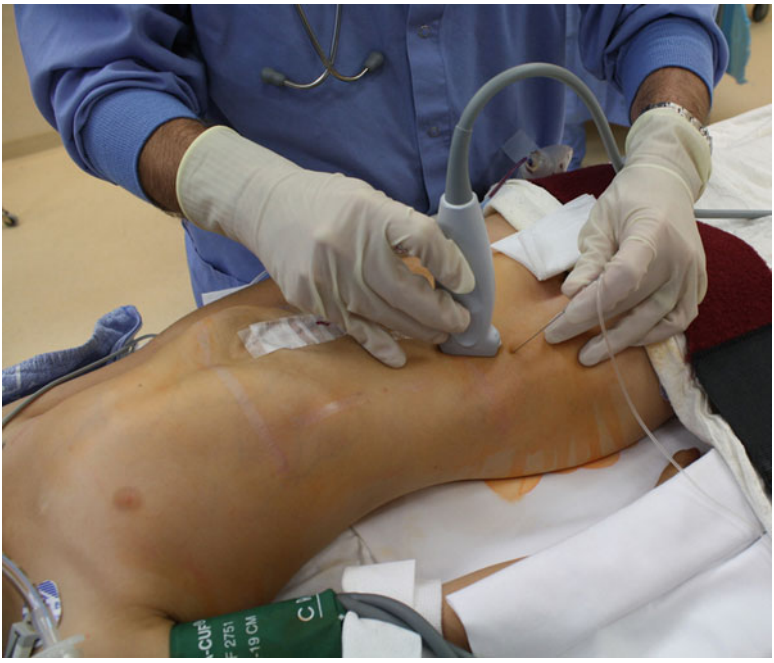

Fig. 9.29 Performing the ilioinguinal iliohypogastric nerve block

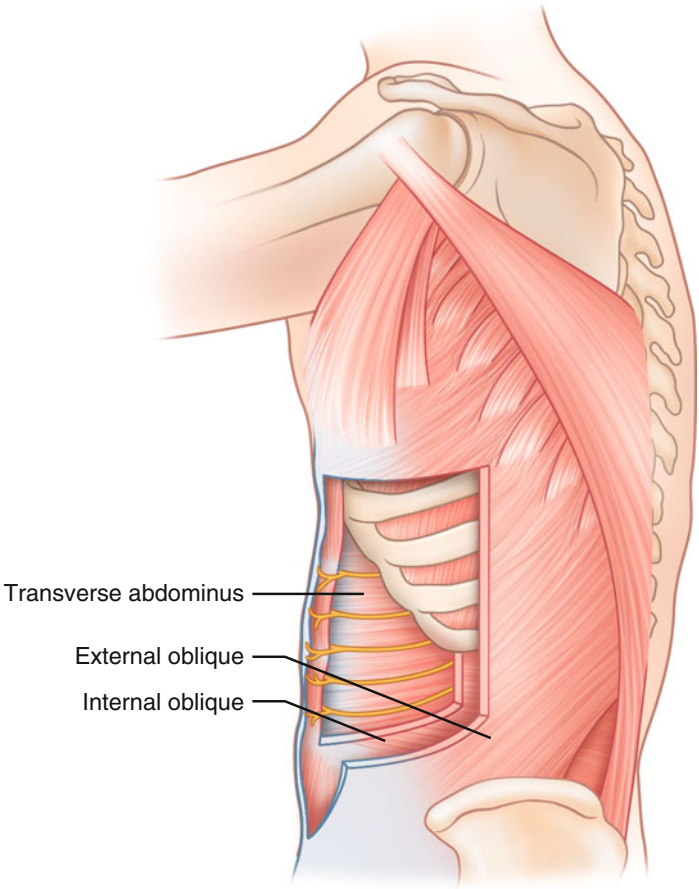

Fig. 9.30 The TAP plexus

To perform the TAP block, a linear probe is selected. After sterile prep, the probe is placed on the abdomen, approximately at the level of the planned incision, in a transverse fashion just lateral to midline. Initial ultrasound examination will identify the large, ellipse-like muscular structure of the rectus abdominis muscles (Fig. 9.31). Once this is identified, the probe is moved laterally, noting the edge of the rectus abdominis muscle. Directly adjacent to the lateral edge of the rectus abdominis muscle, the three linear layers of muscle can be visualized, consisting of the external oblique most superficially, then the internal oblique, and deepest, the transverse abdominis (Fig. 9.32). Once identified, depth, focus, and frequency (typically 12–14 MHz) are adjusted to optimize the image. Of note, as the probe is moved more laterally, the latissimus dorsi will obscure the fascial planes, and the external and internal oblique may become aponeurotic [44]. The block is ideally performed as lateral as possible on the abdominal wall, but medial to the latissimus dorsi.

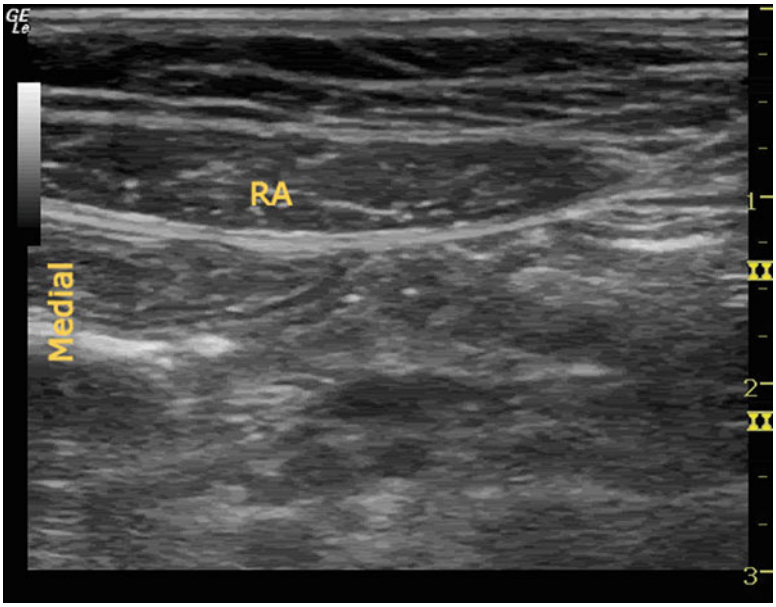

Fig. 9.31 The cross-sectional ultrasonographic appearance of the rectus abdominis

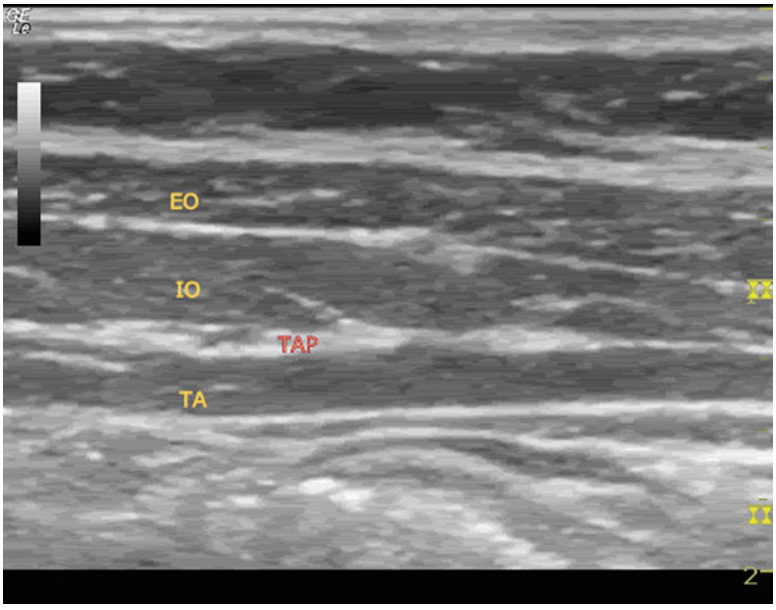

Fig. 9.32 TAP block. The external oblique muscle (*EO*), internal oblique muscle (*IO*), and transverse abdominis muscle (*TA*) are visualized. The transverse abdominis plane (*TAP*) lie between the internal oblique and transverse abdominis muscle

The needle is introduced in-plane from the medial edge of the ultrasound probe. Under constant visualization, it is passed into the layer between the internal oblique muscle and the transverse abdominis muscle. After aspiration, a test dose injection is performed. Ideal visualization of local spread will show a "lemon"-shaped spread of local anesthetic between fascial planes, as opposed to the more circular-shaped appearance of an intramuscular injection [45].

Potential complications of the TAP block include intravascular injection, local anesthetic toxicity, peritoneal puncture with or without visceral injury, and infection at the injection site [46, 47].

Rectus Sheath Block

The rectus sheath block can provide analgesia for procedures involving the anterior abdominal wall such as vertical midline laparotomy and laparoscopy. Its advantages include large and recognizable size of the rectus muscle and lack of large vascular structures in that area [48].

The ventral roots of T6–L1 innervate the central portion of the abdominal wall and lie between the belly of the rectus abdominis muscle and, posteriorly, the fascia of the rectus sheath. Here, an injection of local anesthetic will spread in a caudocephalad manner, anesthetizing the terminal branches of the nerves [49].

To perform the nerve block, the patient is placed supine. A linear probe is selected, with typical frequency ranging from 10 to 12 MHz. After sterile preparation of the skin and probe, the probe is placed in a transverse fashion along the lateral edge of the umbilicus. Here, the rectus abdominis muscle is identified in cross section, and the image is optimized by decreasing depth and selecting the best focus depth and frequency. The probe is then directed laterally to identify the lateral, beak-like border of the rectus muscle. Directly posterior to the muscle lies the hyperechoic fascia of the rectus sheath (Fig. 9.33).

Needle selection is based on the depth of the border of the rectus muscle and rectus sheath. In an in-plane fashion, a needle is introduced from the lateral edge of the rectus muscle and is placed between the rectus muscle and the posterior rectus sheath fascia. After aspirating to rule out intravascular needle placement, a test dose injection will reveal an ellipse-like spread of local anesthetic in the fascial plane. Incorrect intramuscular placement of the needle will result in the local anesthetic injection forming a circular appearance. Once correct needle placement is confirmed, the remaining local anesthetic is injected with the goal of separating the rectus muscle and sheath [50].

Potential complications of the rectus sheath block include intravascular injection, local anesthetic toxicity, rectus sheath hematoma, peritoneal puncture with or without visceral injury, and infection at the injection site.

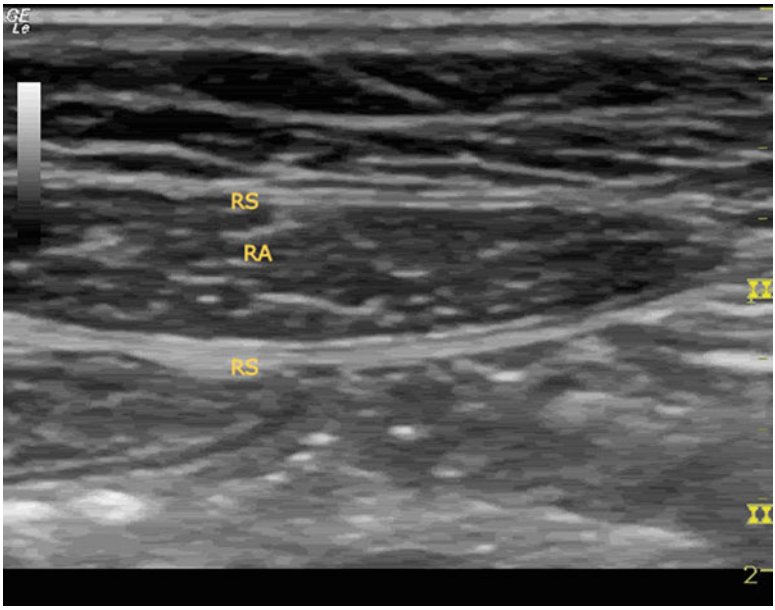

Fig. 9.33 The rectus sheath (*RM*) is encapsulated anteriorly and posteriorly by the rectus sheath (*RS*)

Peripheral Nerve Blockade in Children

Peripheral nerve blockade in the pediatric setting has undergone a slower evolution when compared with the adult world. Before the development of ultrasound-guided peripheral nerve blockade techniques, significant challenges in pediatric patients included the difficulty in targeting nerve structures that course dangerously closely to other critical structures, the constant risk of local anesthetic toxicity with smaller children and higher volume blocks, and the concern of nerve injury when performing a nerve block on a heavily sedated or completely anesthetized patient [51]. The development of ultrasound-guided techniques and subsequent large-scale evaluation of outcomes have led to both increased numbers of peripheral nerve blocks performed in children and clinical data proving efficacy and safety [52].

The pediatric patient offers several advantages over the adult patient in relation to ultrasonography. Typically, the child has less adipose tissue and has smaller structures, which allows the operator to utilize higher frequencies and improve resolution and image quality of blocks that are often difficult in adults. Secondly, with the exception of interscalene nerve blockade, peripheral nerve blocks in a heavily sedated or anesthetized child have been shown to be safe, which provides the operator with an immobile patient [53]. A disadvantage encountered when performing ultrasound-guided regional nerve blocks on children is related mainly to size. In this setting, more specific equipment may be needed, including “hockey-stick” probes

and short block needles. Additionally, there is often limited application space for ultrasound probe placement on the skin in small children.

Performing nerve blocks provides an excellent analgesic alternative to systemic pain medication. In light of the fragile nature of infants and children, peripheral nerve blockade can provide a safe, long-lasting analgesic devoid of the risk of respiratory depression and hemodynamic instability. Current trends support this claim, as the gap between neuraxial blockade, once the most commonly practiced regional anesthetic option, and peripheral nerve blockade in pediatric patients narrows [52].

Concerns of nerve injury when performing peripheral nerve blockade under a heavily sedated or completely anesthetized child remain an area of investigation. Currently, this risk has been determined to be acceptably low, with the exception of interscalene nerve blockade. The American Society of Regional Anesthesia and Pain Medicine has issued a practice advisory stating "Regardless of wakefulness, infants and children may be unable to communicate symptoms of potential peripheral nerve injury. However, uncontrolled movement may increase the risk of injury. Therefore, placement of peripheral nerve blocks in children undergoing general anesthesia or heavy sedation may be appropriate after duly considering individual risk-to-benefit ratio" [53].

Systemic local anesthetic toxicity has always been a concern when placing regional blocks in infants and children. Ultrasound guidance has been proven to reduce the volume of local anesthetic needed for block success, thereby significantly reducing this risk to an acceptable level [54].

Lastly, anesthesia and neurotoxicity to the developing central nervous system has become an area of controversial investigation with apparent areas of concern [55]. If there is indeed a problem, it seems logical that reducing the depth of required anesthesia by employing safe and effective peripheral nerve blockade techniques will be an emerging tool in perfecting the care of children and infants.

Clinical Pearls

Interscalene Block

- The trunks of the plexus lie lateral to the easily identifiable sternocleidomastoid (SCM). The tapered tip of the SCM lies directly above and medial to the anterior scalene muscle.
- It is easy to mistake the lateral border of the SCM for the brachial plexus. This can be avoided by identification of the anterior scalene muscle.
- The anterior scalene muscle can be small as the probe is moved rostrally. It may be beneficial to move the probe caudally in order to identify it.
- If the brachial plexus is not visualized in the interscalene position, gently roll the probe down to the supraclavicular position, and then trace the plexus back to the interscalene position.

Supraclavicular Nerve Block

- The plexus at this level appears as a "bunch of grapes" as opposed to the more linear, "snowman" appearance in the interscalene block.
- The probe should be tilted in order to transect the brachial plexus at this level.
- It is often easy to mistake the fascia enveloping the sternocleidomastoid for the brachial plexus. It is important, here, to visualize the SCM, the anterior scalene, and the middle scalene in order to correctly identify the brachial plexus lying between the anterior and middle scalene.

Infraclavicular Nerve Block

- This is a deeper block; therefore, a curvilinear probe with lower frequencies may be necessary to penetrate tissue.
- If there is difficulty in locating the brachial plexus in the infraclavicular fossa, it is often helpful to identify the brachial plexus in the supraclavicular fossa and then mark on the skin where the plexus is located there. This surface landmark can now serve as the medial border of the area to scan in the infraclavicular fossa.
- In children, it may be difficult to pass a needle at a correct angle in between the probe and the clavicle. If this is the case, it is acceptable to use an in-plane needle placement from the inferior edge of the probe.

Femoral Nerve Block

- The optimal block location is proximal to the femoral artery bifurcation.
- Pressure on the transducer is often useful in obtaining the image of the femoral artery.
- The point of injection must always be below the fascia lata.
- A small amount of rostral tilt may improve the image.

Sciatic Nerve Block

- Lower-frequency probes may be useful as the block is performed more proximally.
- When introducing the needle from the lateral thigh, first measure the depth of the nerve itself, then use that distance from the probe as the insertion site on the lateral thigh.
- Probe tilting may improve the image as you are "transecting" the nerve at different points of the thigh.

- The bifurcation of the sciatic nerve into the tibial and peroneal nerves should be visualized during the popliteal fossa block (Fig. 9.25). The nerve should be blocked proximal to this bifurcation, or the common peroneal and tibial nerves should be blocked with separate injections.

Ilioinguinal Iliohypogastric Nerve Block

- It is often useful to "roll off" the iliac crest in order to situate the probe in its proper alignment.
- Useful landmarks may include the thick iliacus muscles which lie below the transverse abdominis muscles.
- The nerves may appear similar to vasculature. Doppler interrogation may be used to determine whether it is a vascular or nervous structure.

Transverse Abdominis Plane Block

- The TAP block provides analgesia for abdominal wall and peritoneal pain. Supplementation with NSAIDs and opiates should be added for visceral pain.
- Higher-frequency probes will improve the resolution of the image to the superficial nature.
- The TAP block should be performed as lateral as possible on the abdominal wall, specifically where the latissimus dorsi muscle begins to obscure the three muscle layers.
- The rectus abdominis muscle can serve as an excellent landmark medially because this is where the three muscle layers become evident.

Rectus Sheath Block

- The rectus sheath is easily visualized in the midline.
- A lateral approach may be easier as it allows the operator to avoid the thick rectus muscle.

Multiple-Choice Questions

1. Axial resolution refers to:
   (a) The ability to distinguish two points side by side
   (b) The ability to distinguish two points in the same line of axis
   (c) The ability to minimize background noise
   (d) The optimal depth in order to visualize a structure

2. The interscalene block aims to inject the brachial plexus between:
   (a) The sternocleidomastoid and the first rib
   (b) The omohyoid muscle and the sternocleidomastoid
   (c) The anterior and middle scalene muscles
   (d) The pectoralis minor and axillary artery

3. When placing an ultrasound-guided supraclavicular nerve block, the pulsating artery found at the inferior portion of the target area is the:
   (a) Subclavian artery
   (b) External carotid artery
   (c) Internal carotid artery
   (d) Vertebral artery

4. After placing an axillary nerve block, the patient has sensory sparing of the medial upper arm. The nerve responsible for this is:
   (a) The radial nerve
   (b) The ulnar nerve
   (c) The median nerve
   (d) The musculocutaneous nerve

5. The femoral nerve is bounded medially by the femoral artery, inferiorly by the iliacus muscle, and superiorly by the hyperechoic:
   (a) Iliopectinate ligament
   (b) Fascia lata
   (c) Femoral nerve
   (d) Psoas muscle

6. After performing a popliteal fossa–sciatic nerve block for an ankle procedure, the patient is experiencing pain on the dorsum of the foot. A likely scenario of failure is:
   (a) The femoral nerve has been mistakenly blocked
   (b) The saphenous nerve was not adequately anesthetized
   (c) There is typically sparing of anesthesia in this location
   (d) The block occurred distal to the bifurcation of the sciatic, only including the tibial nerve

7. The “trackback” technique involves locating the saphenous nerve related to its branching from the:
   (a) Femoral nerve
   (b) Sural nerve
   (c) Sciatic nerve
   (d) Obturator nerve

8. The ilioinguinal and iliohypogastric nerves are blocked as they course between the:
   (a) Transverse abdominis muscle and the parietal peritoneum
   (b) External and internal oblique muscles
   (c) Internal oblique and transverse abdominis muscles
   (d) Iliacus and transverse abdominis muscles

9. When utilizing a TAP block for a laparoscopic appendectomy, the following is false:
   (a) Parenteral opiates or NSAIDs are useful for the treatment of uncovered visceral pain
   (b) The anterior abdominal wall is anesthetized by the TAP block
   (c) The anterior parietal peritoneum is blocked by the TAP block
   (d) The TAP block should cover components of visceral pain

10. When injecting a local anesthetic solution during ultrasound-guided peripheral nerve block placement, you notice large hyperechoic artifacts filling the target area. The likely problem is:
   (a) Vascular perforation
   (b) Air has been injected through the needle
   (c) Intraneural injection
   (d) Vasospasm

11. While performing a TAP block, injection reveals a round, circular spread of injectate in an area presumed to lie between the transverse abdominis and internal oblique muscles. The block fails. The likely problem is:
   (a) This was an intramuscular injection
   (b) This was an intravascular injection
   (c) The nerve was missed
   (d) The volume of local anesthetic was insufficient

12. While performing an interscalene nerve block, the image appears as bright, causing difficulty in identifying neural structures. An appropriate control to manipulate would be:
   (a) The depth
   (b) The frequency
   (c) The gain
   (d) The focus position

13. While placing a femoral nerve block, difficulty is encountered in identifying the needle placement. Injection of a small amount of local anesthetic results in a widening of the femoral nerve image. A likely scenario is:
   (a) Intraneural injection
   (b) Correct placement
   (c) Intravascular injection
   (d) Air injection

14. While placing a femoral nerve block, you notice an asymmetric, expanding hypoechoic element next to the nerve and artery. This is likely:
   (a) Extravasation of a small amount of local anesthetic from the needle
   (b) Separation of the fascial plains
   (c) Mild damage to and extravasation of the lymphatic system
   (d) Formation of a hematoma from the femoral artery

15. While performing an interscalene block, it is difficult to obtain an acceptable image of the axillary artery and brachial plexus due to the depth of the structures.

The following would be appropriate manipulations of the ultrasound machines EXCEPT:
(a) Decreasing frequency
(b) Increasing frequency
(c) Manipulating the TGC
(d) Increasing depth

Answers:

1. b – Axial resolution refers to the ability to distinguish two points in the same line of axis. Higher-frequency beams improve axial resolution at the cost of poorer depth penetration and visualization.
2. d – The interscalene block aims to inject the brachial plexus at the level of the trunks, which usually exists at C6. Here, the trunks lie between the anterior and middle scalene, though the anterior scalene may appear small.
3. a – The brachial plexus follows a similar course to the subclavian artery, which becomes the axillary artery. As such, this is a useful landmark in locating the brachial plexus.
4. d – The musculocutaneous nerve is frequently missed in the axillary nerve block. It can be visualized with an ultrasound and injected separately, or accessed by directly injecting into the body of the coracobrachialis muscle.
5. b – The fascia lata is the superior border of the femoral nerve sheath. It separates from the fascia iliaca at the femoral nerve and rejoins laterally.
6. d – Blocking the sciatic nerve in the popliteal fossa must be performed proximal to the bifurcation of the sciatic nerve into the tibial and peroneal nerves.
7. a – The saphenous nerve is a branch of the femoral nerve. As such, it is often responsible for medial foot pain when only a sciatic block is performed.
8. c – Much like the TAP block, the ilioinguinal and iliohypogastric nerves course between the internal oblique muscle and the transverse abdominis muscle.
9. d – While the TAP block anesthetized the terminal branches of T8–T11, it does not typically block visceral pain. As such, the anterior abdominal wall and the parietal peritoneum will likely be covered, but visceral pain must be addressed with either parenteral medicines or additional neuraxial anesthetics.
10. b – As air is hyperechoic, even small amounts can significantly worsen imaging when injected. Therefore, it is essential to always flush needles before ultrasound-guided peripheral nerve blockade.
11. a – The TAP block relies on anesthetic deposition between two fascial layers. As such, the injectate should appear as an elliptical spread of hypoechoic fluid. Round-appearing injectate is frequently indicative of intramuscular injection.
12. c – Gain refers to the amplification of received signals. As such, too much gain leads to an amplification of background noise and, in this case, should be lowered.
13. a – Widening of the nerve image is often indicative of intraneural injection, hich has been associated with nerve injury.

14. d – Inadvertent vascular puncture is often seen as a hypoechoic expansion of the perivascular space.
15. b – By decreasing frequency and depth, deeper structures are better imaged. TGC may help optimize the gain on attenuated deeper structures.

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10

Sonopathology

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Contents

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Introduction

Ultrasound-guided regional anesthesia (UGRA) is an emerging subspecialty of regional anesthesia. The use of ultrasound (US) to visualize the needle, nerve, and spread of local anesthetic has energized the community and increased the popularity of regional anesthesia techniques. During the normal conduct of UGRA, the anesthesiologist will frequently be confronted with aberrant and atypical anatomy. The term *sonopathology* has been used to describe such unexpected anatomy and pathology [1]. The following chapter will review clinically relevant principles related to challenging and interesting sonoanatomy. Selected examples and images of sonopathology are presented with the intention of allowing regional anesthesiologists to appreciate variations of anatomy, distinguish artifact from pathology, and utilize this information to expand their roles as perioperative physicians.

Nerves

Ultrasound scanning for regional anesthesia occasionally reveals abnormalities of the peripheral nervous system. Anatomic variations, inflammation within or surrounding the nerves, and nerve tumors are examples of findings that have been reported. These unexpected discoveries could interfere with successful performance of a nerve block or potentially alter the anesthetic plan.

Anatomy

Cadaver studies have demonstrated that up to 40% of the population has a situation in which the brachial plexus does not completely pass through the interscalene groove [2]. Anatomic variations include passage of individual branches or the entire plexus through the body of the anterior scalene muscle. For example, the components of the plexus in Fig. 10.1 are unlikely to be contained within a common compartment. These anomalies have significant implications for block success, in that the anterior and middle scalene muscles are traditionally used as reference points to aid in the location of the brachial plexus. Multiple separate injections around discrete nerves may be required for complete blockade. The sonographic recognition of such anomalies may allow immediate targeting of the intramuscular and split component of brachial plexus.

Similar anatomic aberrancies have been noted in the supraclavicular, axillary, and femoral regions. The divisions of the brachial plexus at the supraclavicular level normally are bundled at the lateral border of the subclavian artery. However, the ultrasound image in Fig. 10.2 depicts individual nerve branches that course medial to the artery. A more recent cadaver study revealed axillary brachial plexus abnormalities that include anomalous nerve location, as well as the presence of additional vasculature, such as multiple veins (Fig. 10.3) or a double axillary artery [3].

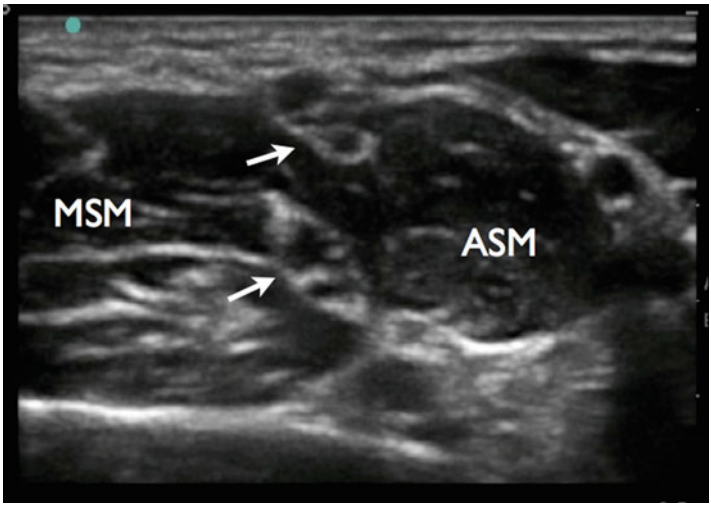

Fig. 10.1 Components of the brachial plexus at the midneck level. There is no interscalene groove. Nerves appear to be located in separate compartments, with muscle tissue seen dividing the plexus. *ASM* anterior scalene muscle, *MSM* middle scalene muscle, *arrows* brachial plexus

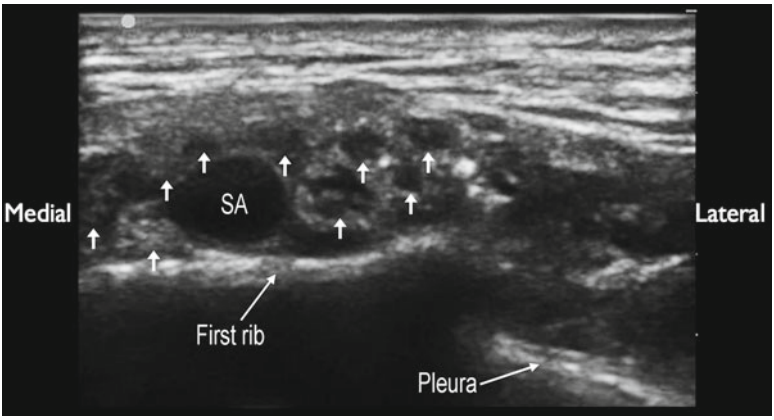

Fig. 10.2 Atypical presentation of brachial plexus at supraclavicular level. Nerves are seen medial to the artery. *SA* subclavian artery, *small arrows* brachial plexus

Figure 10.4 is an excellent example in which the femoral nerve is noted to be embedded in the body of the iliopsoas muscle, rather than within the compartment bordered by the fascia iliaca, iliopsoas muscle, and femoral artery. Further, the nerve in this particular patient is located more lateral to the femoral artery than usual. It is conceivable that the iliopsoas muscle may serve as a barrier to local anesthetic

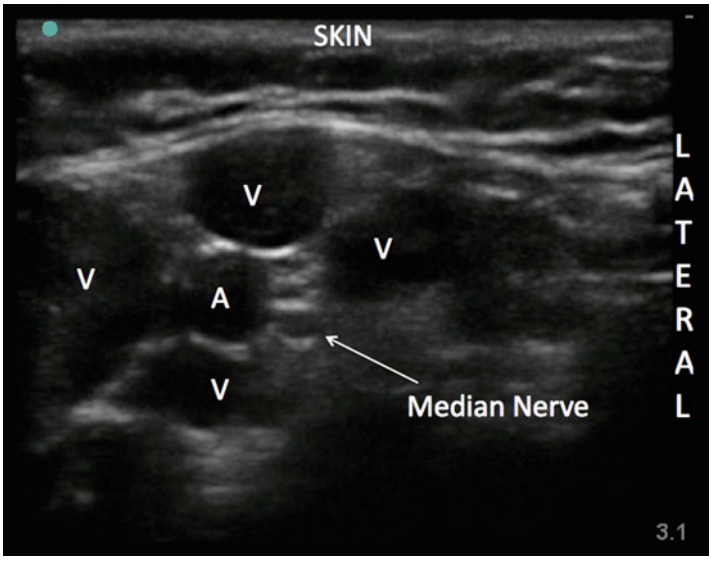

Fig. 10.3 Brachial plexus at axillary level. Multiple large veins surround the artery and nerves. This image comes from a patient with a distal arm A–V fistula that was pressurizing the veins. *V* vein, *A* artery

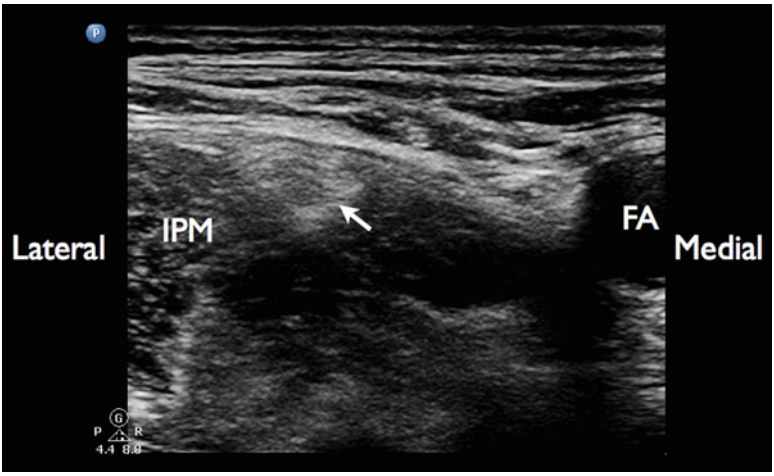

Fig. 10.4 Femoral nerve imbedded within the iliopsoas muscle. *IPM* iliopsoas muscle, *FA* femoral artery, *arrow* femoral nerve

spread, thus mandating an atypical intramuscular type of injection. In this situation, ultrasound guidance would likely decrease the number of needle passes in comparison to a landmark approach. That is, a traditional landmark approach would likely place the needle more medial and superficial than that actually required.

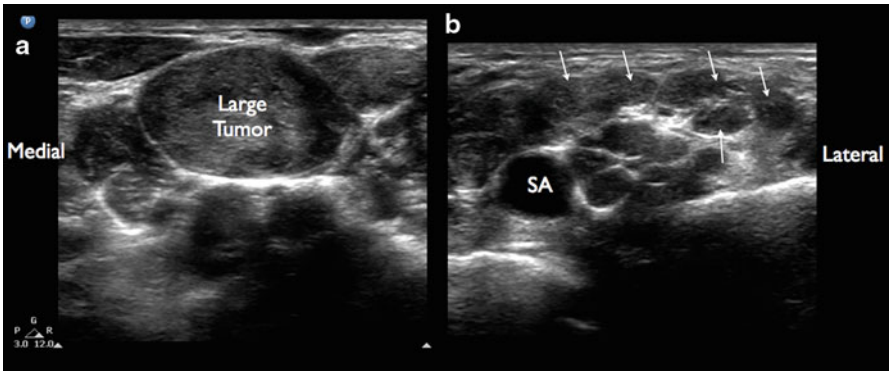

Fig. 10.5 (**a**) Large tumor seen at the midneck (interscalene) level of the brachial plexus in a patient with neurofibromatosis. (**b**) Tumors can also be seen at the supraclavicular level in the same patient. *SA* subclavian artery, *arrows* nerve tumors

Tumors

Neurofibromatosis is a genetic disorder characterized by the predisposition to nerve sheath tumors [4]. Lesions most commonly seen are neurofibromas and schwannomas. Figure 10.5a and b depicts nerve sheath tumors found on ultrasonography of the interscalene and supraclavicular regions, respectively, in a patient with a known diagnosis of neurofibromatosis. The lesions appear quite large on ultrasound, despite a normal physical examination. Our knowledge of the effects of local anesthetics in patients with this disorder is limited, so anesthetic management requires an individualized assessment of potential risks and benefits. The tumors in Fig. 10.5a and b presented obvious technical challenges for a safe and successful brachial plexus block. Therefore, a nerve block was not attempted, and the patient underwent general anesthesia.

Neuritis

Inflammation of the nerve can be seen on ultrasound. Nerves that appear swollen, enlarged, or distorted may be manifesting an inflammatory neuritis [5]. Scanning of the same nerve on the contralateral side allows comparison to help verify the diagnosis. Clinical signs and symptoms also contribute to the evaluation. Neuritis is a relative contraindication to nerve blocks given that potential dangers of local anesthetic on inflamed nerves are unknown.

Blood Vessels

Correct identification of peripheral nerves requires a dynamic ultrasound evaluation of their relationship with surrounding structures such as muscle, bone, fascia, arteries, and veins. Many nerves are located in close proximity to major blood vessels. The ability to correctly identify vascular structures is critical for safe and effective UGRA. Recognition of vascular abnormalities reveals important information that may affect surgical or anesthetic management.

Occlusive Disease

Causes of vascular occlusion include deep vein thrombosis, arterial thrombi or emboli, atherosclerosis, tumor invasion, and external compression from tumors, lymphadenopathy, or edema. Such findings on routine scanning for nerve blocks should lead one to consider possible perioperative implications. Discovery of a plaque within the femoral artery (Fig. 10.6) in a patient with known risk factors for vascular disease may have little impact on the anesthetic management, but it does justify a discussion with the patient and referral for a definitive evaluation. Alternatively, significant carotid artery stenosis (Fig. 10.7) in a patient scheduled for elective shoulder surgery in the beach chair position raises concerns about adequate cerebral perfusion under general anesthesia. Intraoperative monitoring strategies and blood pressure goals may need to be reconsidered, and severe disease could warrant delay to allow proper referral and investigation.

Because US waves are easily transmitted through fluid, blood vessels should appear, in short axis, as anechoic (black) round structures. Veins can be distinguished from arteries by observing pulsation and assessing compressibility. Color Doppler is another useful tool to assess flow within a structure. A deep vein thrombosis (DVT) can be diagnosed with ultrasonography and may be discovered incidentally during peripheral nerve block procedures [6]. The vein may contain hyperechoic material, be resistant to compression, and exhibit limitations, or complete obstruction, to blood flow as seen on color Doppler (Fig. 10.8).

Vascular Anomalies

Anomalous vascular anatomy can create confusion during UGRA. Arteries and veins can vary greatly in size, number, and location. Not uncommonly, unexpected vessels are discovered in the intended path of the block needle (Fig. 10.9). Performing systematic probe maneuvers (pressure, alignment, rotation, and tilting) and utilizing color Doppler can unmask blood vessels, helping to avoid inadvertent puncture. Avoiding unnecessary vascular punctures adds quality to the nerve block by decreasing hematoma formation and reducing the possibility of compressive neuropathy. The large blood vessel present in Fig. 10.9 was likely an atypical transverse cervical artery (TCA). Like its counterpart in the femoral region (the lateral circumflex

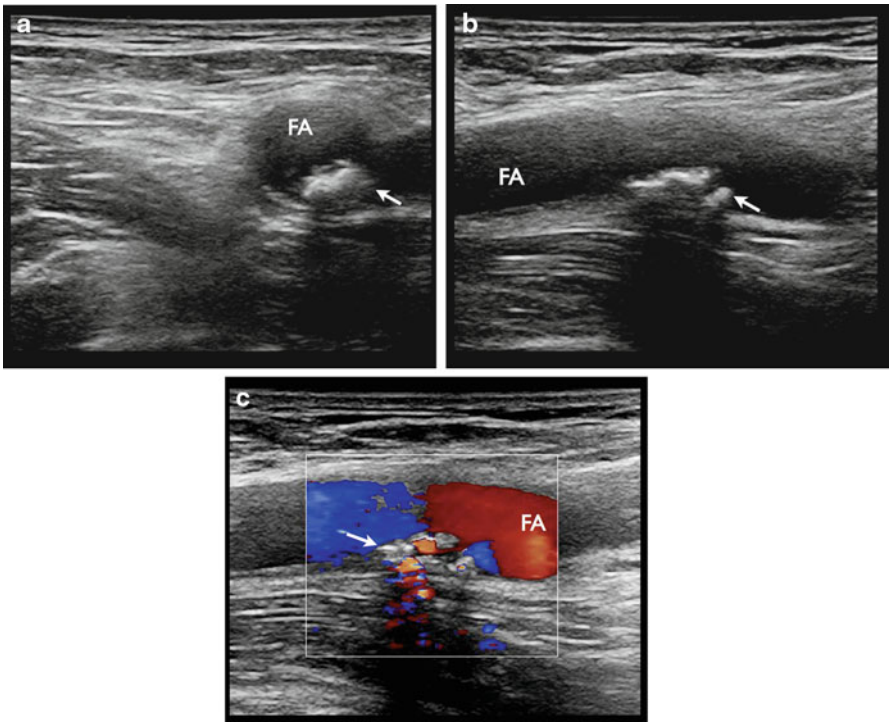

Fig. 10.6 (**a**) Short-axis view of the femoral artery with thrombus seen within lumen. (**b**) Long-axis view of the same artery. Notice the posterior acoustic shadowing that appears deep to the lesion. (**c**) Long-axis view with color Doppler demonstrates partial occlusion to blood flow. *FA* femoral artery, *arrow* thrombus

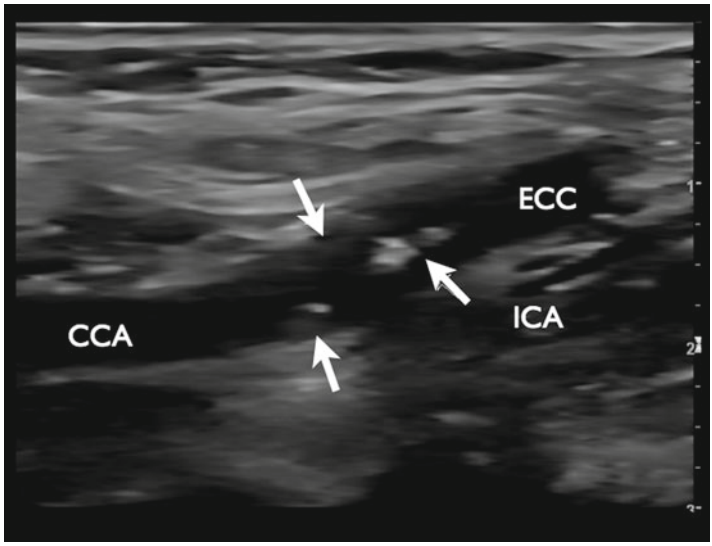

Fig. 10.7 Carotid artery plaque. *CCA* common carotid artery, *ECC* external carotid artery, *ICA* internal carotid artery, *arrows* plaque

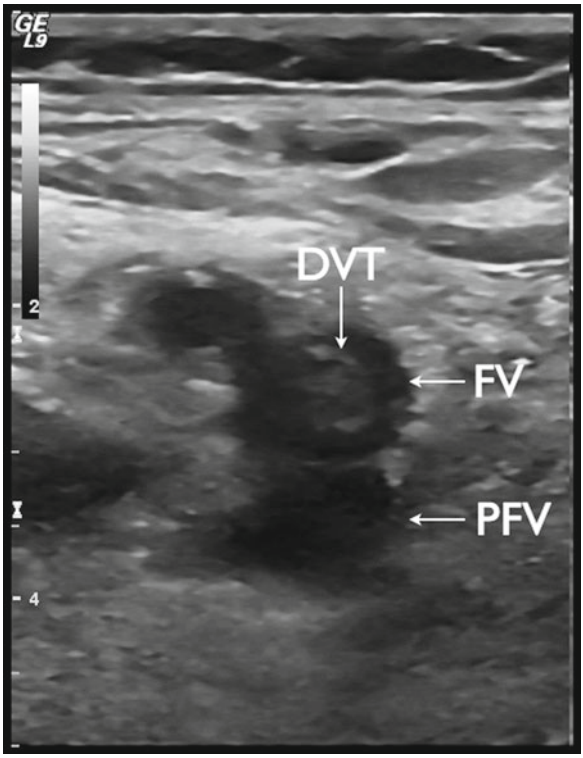

Fig. 10.8 Deep vein thrombosis of the proximal thigh. *DVT* deep vein thrombosis, *FV* femoral vein, *PFV* profunda femoris vein

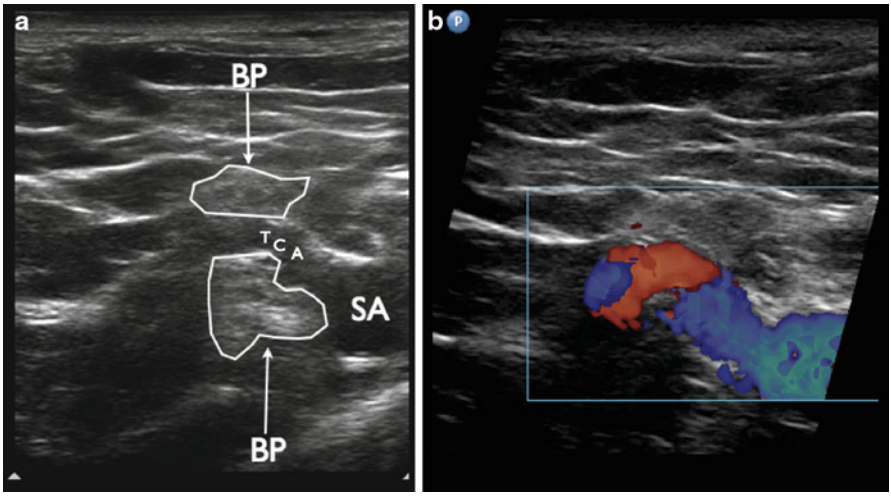

Fig. 10.9 (**a**) Supraclavicular view of the brachial plexus with an arterial branch located between the nerve bundles. (**b**) Color Doppler illustrates flow within the artery. *BP* brachial plexus, *SA* subclavian artery, *TCA* transverse cervical artery

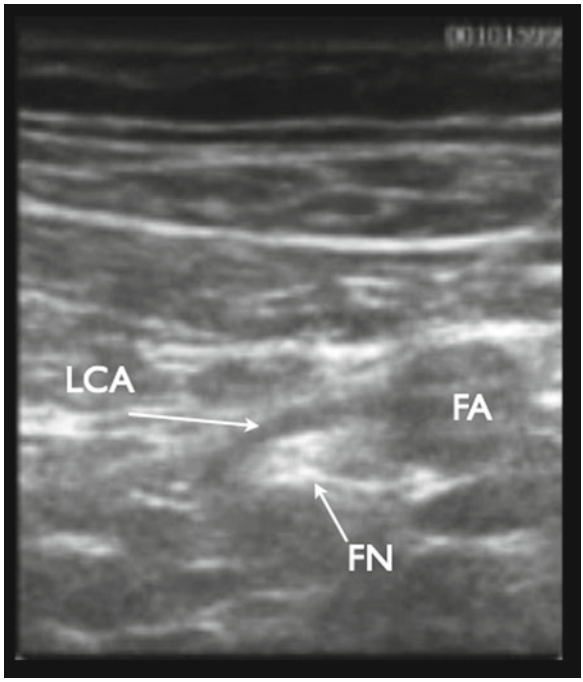

Fig. 10.10 A branch of the femoral artery (presumed to be the lateral circumflex femoral artery) is seen superficial to the femoral nerve. An alternate view should be obtained to avoid vascular puncture. *LCA* lateral circumflex femoral artery, *FN* femoral nerve, *FA* femoral artery

femoral artery, seen in Fig. 10.10), the TCA often prevents the safe approach of the needle using the standard in-plane needle insertion techniques. When such anatomy is identified, a modification of the block or needle approach can be made.

Vascular tumors such as hemangiomas, glomangiomas, hemangioendotheliomas, hemangiopericytomas, and angiosarcomas may be encountered during UGRA [1]. Characteristics of these structures on US can be highly variable, as they may appear hyperechoic or hypoechoic and have either a homogenous or heterogeneous pattern [7]. Additionally, arteriovenous malformations and aneurysms should be considered if abnormal vasculature is observed. Although definitive diagnosis and treatment of such findings should be deferred to an appropriate specialist, anesthesiologists performing UGRA should be able to distinguish pathologic vasculature from normal anatomy.

Other Tissues

Musculoskeletal

Bone, muscle, and tendons can exhibit abnormal findings. Tendons and nerves have a similar appearance on US and can often be confused. Figure 10.11 illustrates how the median nerve and surrounding tendons of the distal forearm appear

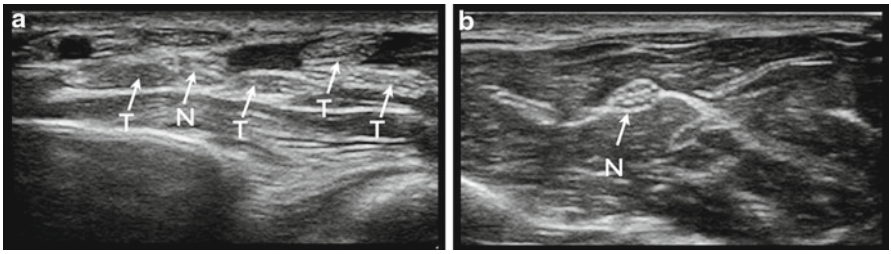

Fig. 10.11 (**a**) Image of the median nerve at the wrist. Notice that tendons and nerves appear nearly identical. (**b**) The median nerve in the same patient is easy to see in isolation at a slightly more proximal location. *T* tendon, *N* median nerve

nearly identical on a static US image, but as the transducer is moved proximal, the nerve becomes readily apparent. Appreciation of anatomic relationships, especially with dynamic US scanning, is often necessary to distinguish tissues with similar acoustic appearances. Ultrasound examination of the tendons occasionally reveals inflammation or tears. Muscles generally appear hypoechoic with visible linear hyperechoic striations within the body. However, atrophy is characterized by deposition of fibrous and adipose tissue within the muscle, which causes a generalized hyperechoic appearance. This explains why nerve visualization may be difficult in sedentary or immobilized patients. Bones highly attenuate ultrasound energy, thus generating a strong superficial echogenic signal with acoustic shadowing below. Although fractures can often be appreciated, the echogenic nature of the bone cortex makes thorough evaluation with US very challenging. Bone anomalies such as an absent first rib or the presence of a cervical rib can be found incidentally when scanning the brachial plexus [8].

Tumors

Tumors of the bone, muscle, and soft tissue include fibromas, sarcomas, and lipomas. Appearance can be variable in terms of echogenicity and architecture. The inability to compress or visualize flow on color Doppler helps to identify such tumors as nonvascular structures.

Lymphadenopathy

Lymph nodes (Fig. 10.12) can be visualized with US, especially during femoral, interscalene, and axillary nerve blocks as they frequently reside in the subcutaneous tissue. Incidental discovery of malignant lymphadenopathy has been reported [9].

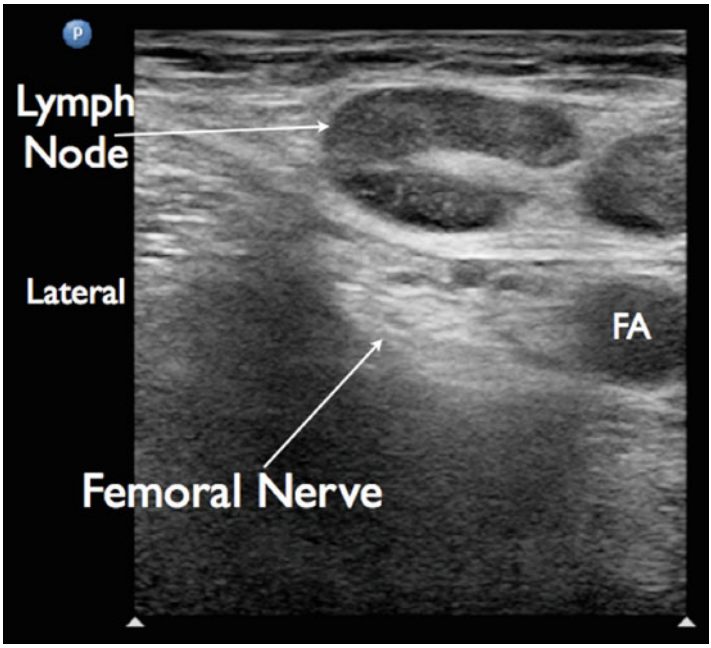

Fig. 10.12 Large lymph node superficial to the femoral nerve. This lesion was noted during preparation for a continuous nerve block in a patient undergoing a total knee replacement. The continuous nerve block was abandoned in favor of a single injection. *FA* femoral artery

They appear as round or oval, anechoic structures with a hyperechoic border. Lack of flow and inability to compress them distinguish lymph nodes from vessels. Small lymph nodes are usually insignificant and pose only minor obstacles. Care should be taken to avoid confusing them with nerves, especially in the axilla, where the nerves appear as anechoic circles surrounding the artery. Profound lymphadenopathy may suggest an insidious underlying process and should prompt further investigation.

Viscera

Several common visceral pathologic states are seen in the context of UGRA. The thyroid gland is often identified as having nodules (Fig. 10.13). A nodule is defined as a discrete lesion within the thyroid gland that is sonographically distinguishable from the adjacent parenchyma [10]. The ability to distinguish malignant vs. benign thyroid disease by ultrasound is limited even for an advanced trained practitioner [10]. Given that thyroid lesions are often asymptomatic, the anesthesiologist may be the first to identify such lesions during the performance of brachial plexus blocks

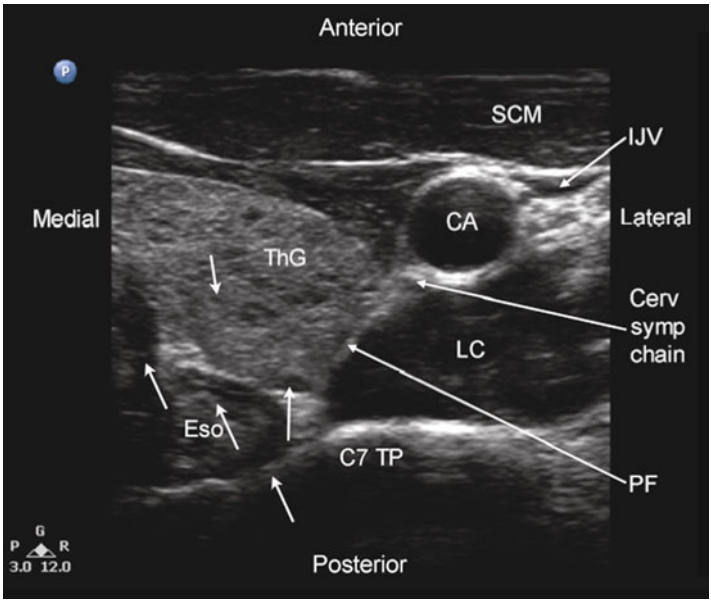

Fig. 10.13 Thyroid nodules. A multinodular thyroid was discovered during the performance of a stellate ganglion block. This is a short-axis image at the level of C7 showing multiple hypoechoic nodules (*unlabeled arrows*) in the thyroid gland parenchyma. *SCM* sternocleidomastoid muscle, *IJV* internal jugular vein, *Eso* esophagus, *C7TP* transverse process of the C7 vertebral body, *ThG* thyroid gland, *Cerv symp* cervical sympathetic chain, *PF* prevertebral fascia, *CA* carotid artery, *LC* longus coli muscle

above the clavicle. With appropriate referral, fine needle aspiration by a radiologist is usually the recommended diagnostic intervention.

Femoral, incisional, and inguinal hernias may be identified during the performance of femoral, TAP, and rectus sheath blocks. Ultrasound is extremely sensitive and specific for the diagnosis of inguinal and femoral hernias [11]. Nonincarcerated hernias are usually seen as moving heterogeneous echogenicities consisting of a hernia sac [12]. Fluid in the hernia sac will cast an anechoic layer that may outline contained intestinal or omental structures. The ability to identify a hernia sac containing possible bowel contents is important from the perspective of pure diagnosis, as well as the avoidance of needle-related bowel injury.

Fluid Collections

Cysts, ganglia, seromas, and abscesses can all resemble lymph nodes. Of these, it is most important to be able to identify an abscess, which is most likely to affect the

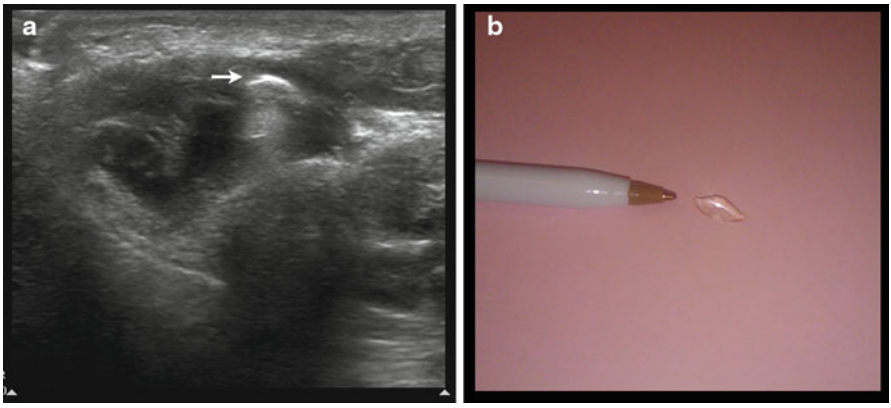

Fig. 10.14 (**a**) Piece of glass embedded in a patient's hand. (**b**) Same piece of glass following excision. *Arrow* glass

decision to perform regional anesthesia and may warrant additional treatment. Abscesses usually have an irregular and echogenic border with a heterogeneous pattern interiorly that signifies the purulent debris contained within [8]. Alternatively, cysts, ganglia, and seromas tend to be round or oval with a homogenous anechoic center [8].

Foreign Bodies

Wood, metal, and glass objects are among the most common foreign bodies that can become lodged below the skin [13]. Wood splinters are likely to create a dropout shadow on the US screen, obscuring visualization of deeper structures. Metal and glass tend to be highly echogenic and may produce a "comet tail." This artifact is caused by multiple reflections within a small but highly reflective object, resulting in a long hyperechoic line extending downward along the US beam [14]. Figure 10.14 depicts a sliver of glass in the palm of a patient who presented for surgical excision of this foreign body. Ultrasound scanning by an anesthesiologist allowed precise marking and depth measurement. This information was requested by the surgeon given that the glass was not palpable on physical exam.

Air

Air is highly resistant to ultrasound transmission. Even small air bubbles injected during a nerve block can significantly obscure the target structures. The presence of

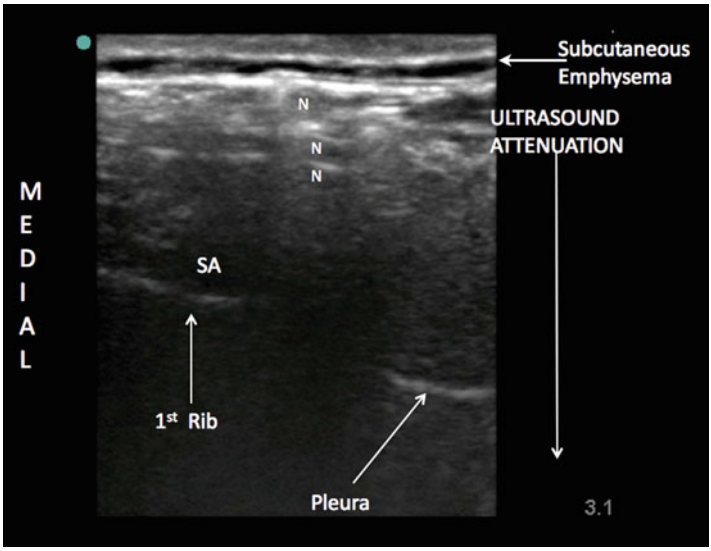

Fig. 10.15 Supraclavicular view of the brachial plexus. Image is severely degraded due to the presence of subcutaneous air, leading to the diagnosis of a pneumothorax. *N* nerve, *SA* subclavian artery

preexisting air may indicate a pathologic process. Imaging of the structures in the supraclavicular view in Fig. 10.15 is degraded due to subcutaneous air. This unexpected finding in a stable trauma patient presenting for semielective arm surgery suggested a possible missed diagnosis of a pneumothorax. Suspicion of a pneumothorax warrants prompt evaluation, for which ultrasound is a powerful diagnostic tool. The pleura is easily visualized through the intercostal space, and the sliding of the visceral and parietal pleurae generates a distinctive ultrasound image. Further confirmation of pleural sliding can be made with motion mode (M-mode) US, a modality that utilizes a higher frame rate and is thus highly effective at evaluating moving structures [8]. The characteristic "seashore sign" on M-mode US is caused by lung sliding and is a normal finding. When sliding is absent, the image created is referred to as the "stratosphere sign," which suggests the presence of a pneumothorax (Fig. 10.16). The patient in Fig. 10.15 was indeed found to have a pneumothorax, and a preoperative chest tube was placed.

M-mode can also detect diaphragm movement, which is particularly useful in patients receiving an interscalene block [15]. If preexisting contralateral diaphragm paralysis is suspected, information obtained from M-mode US could significantly impact the anesthetic plan [15].

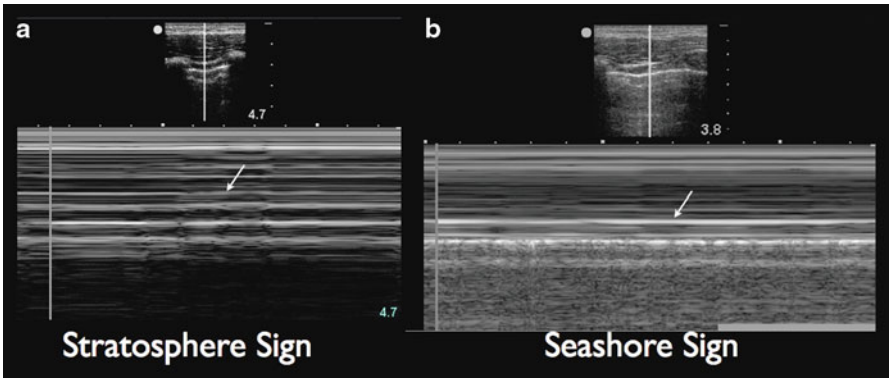

Fig. 10.16 M-mode US can be used to identify a pneumothorax. (**a**) The "stratosphere sign" describes an image that appears homogenous above and below the pleural line. This occurs because pleural sliding is absent in patients with a pneumothorax. (**b**) The "seashore sign" indicates no pneumothorax. Pleural sliding creates a granular pattern below the pleural line. *Arrows* pleural line

Controversy

Does this discussion of sonopathology reach beyond our scope of practice as anesthesiologists? Some may argue that using UGRA as an opportunity to make diagnoses oversteps the boundaries of the practice of anesthesiology and potentially endangers patients and exposes us to unnecessary liability. Although the use of TEE by anesthesiologists to make perioperative decisions is well accepted, an extensive certification process exists to ensure that clinicians meet specific standards. However, no such certification currently exists for UGRA. Steps have been taken to establish guidelines for training physicians to perform UGRA, but the scope of practice remains undefined [16].

The American Society of Anesthesiologists describes regional anesthesia as the practice of medicine [17]. As perioperative physicians we have an obligation to provide optimal patient care based on the information we can reasonably obtain. Anesthesiologists are not necessarily trained in cardiology or pulmonology, yet it is well within our scope of practice to auscultate the heart, review a chest X-ray, and assess the lungs in order to derive information that will impact perioperative patient care. We believe that it is reasonable to regard the ultrasound transducer as an extension of the stethoscope, thereby modernizing the physical exam [18]. Physicians in various areas of medicine including emergency medicine, obstetrics, rheumatology, and critical care are utilizing ultrasound for timely evaluation of patients. Our specialty should be no different.

Summary

When US is used routinely for peripheral nerve blocks, a multitude of anatomic and pathologic aberrancies will inevitably present themselves. Vascular and nerve lesions may preclude injection of local anesthetic and/or necessitate further evaluation. Unexpected findings with anesthetic implications involving other organ systems may also occur. Sonopathology encountered during UGRA can range from the trivial, curious anomaly to potentially devastating disease. Although the role of the anesthesiologist in this setting is controversial, the ability to recognize sonopathology allows the adjustment of the anesthetic plan accordingly and the facilitation of an appropriate intervention or consultation.

Clinical Pearls

- Sonographic imaging is within the role of the anesthesiologist.
- Systematic scanning prior to nerve blockade should be utilized to evaluate the anatomy. Manickam et al. describe a "4D" approach to the systematic survey: direct visualization of structures, dynamic evaluation to determine identity and course, Doppler assessment to visualize blood flow, and different-anatomical-site evaluation in the event of anatomic challenges [18].
- Degraded US images frequently occur due to technical limitations or body habitus but may also result from pathology. If repositioning and machine adjustment do not improve image quality, one must be suspicious of a pathologic lesion.
- Posterior acoustic shadowing (dropout shadow) is created by anechoic material including bone, calcifications, and foreign bodies (wood, metal, and glass).
- Atherosclerosis is extremely common and will be seen frequently during sonography of all regions of the body.
- The anesthetic plan should be flexible. If UGRA is planned, utilize the US to help guide the care of the patient. Unexpected findings can and should impact the anesthetic plan.
- Communicate unexpected findings with surgical colleagues and refer patients for further evaluation by a specialist if appropriate. Effective communication exemplifies the role the perioperative physician.

Multiple-Choice Questions

1. Anomalous location of which artery is most likely to affect brachial plexus block with the supraclavicular approach?
   (a) Circumflex scapular artery
   (b) Thoracoacromial artery
   (c) Suprascapular artery
   (d) Transverse cervical artery

2. True statements regarding M-mode US include all of the following EXCEPT:
   (a) The "seashore sign" is a normal finding.
   (b) It can be used to identify phrenic nerve paralysis.
   (c) It requires use of a high-frequency transducer.
   (d) It is more sensitive than chest X-ray for detection of a pneumothorax.

3. A needle that appears bent on the screen can be explained by:
   (a) The bayonet effect
   (b) Reverberation artifact
   (c) Refraction artifact
   (d) The comet tail effect

4. Which of the following explains the presence of a series of bright lines that sometimes appear parallel to the needle?
   (a) The bayonet effect
   (b) Reverberation artifact
   (c) Refraction artifact
   (d) The comet tail effect

5. All of the following will cause a brighter image EXCEPT:
   (a) Increased gain
   (b) Edema
   (c) Injected air
   (d) Calcification

6. Posterior acoustic shadowing:
   (a) Can be distinguished from "dropout shadow" by adjusting the frequency
   (b) Is generally less pronounced when created from bone rather than air
   (c) May help identify a vascular plaque
   (d) All of the above

7. Methods of confirming the presence of DVT include:
   (a) Applying pressure to assess compressibility
   (b) Utilizing Doppler color flow to characterize the lesion
   (c) Imaging the vessel in long axis as well as short axis
   (d) All of the above

8. Useful transducer maneuvers to optimize the ultrasound image include all of the following EXCEPT:
   (a) Pressure
   (b) Sliding
   (c) Rotation
   (d) Tracking

9. Which of the following statements regarding anatomical variation is true?
   (a) The incidence of neural anatomical variants of the brachial plexus ranges from 17% to 35%.
   (b) Nerve anomalies of the lower extremities are more common than above the clavicle.
   (c) Anatomic variations of the brachial plexus can usually be predicted based on clinical signs.
   (d) All of the above.

10. Detection of a nerve tumor during UGRA is important because:
   (a) Peripheral nerve tumors have a relatively high incidence of malignancy.
   (b) The increased sensitivity to local anesthetics in these patients is well documented.
   (c) The risk of bleeding or intravascular injection is likely higher.
   (d) All of the above.

11. Findings that can be appreciated during UGRA include all of the following EXCEPT:
   (a) Bone fractures
   (b) Elevated CVP
   (c) Tendonitis
   (d) Diabetic peripheral neuropathy

12. All of the following statements regarding ultrasound detection of lymphadenopathy are true EXCEPT:
   (a) Enlarged lymph nodes are most commonly discovered in the cervical, inguinal, and axillary regions.
   (b) Ultrasound can help distinguish between benign and malignant lymph nodes.
   (c) Ultrasound is no better than palpation in detecting lymphadenopathy.
   (d) Lack of compressibility can help distinguish lymph nodes from blood vessels.

13. Lymph nodes are most likely to be confused with which of the following nerves?
   (a) Femoral nerve at inguinal crease
   (b) Ulnar nerve within axillary sheath
   (c) Sciatic nerve at the popliteal fossa
   (d) Median nerve in proximal forearm

14. Which of the following statements regarding visceral sonopathology is true?
   (a) Ultrasound can be helpful to differentiate an indirect vs. direct inguinal hernia.
   (b) Fluid contained within a hernia sac may obscure the view of intestinal structures.
   (c) Although a hernia may be discovered incidentally during UGRA, ultrasound is neither a sensitive nor specific diagnostic tool for diagnosis of hernias.
   (d) Ultrasound can usually distinguish malignant from benign thyroid nodules, but fine needle aspiration is generally recommended to confirm the diagnosis.

15. All anesthesiologists practicing UGRA should be able to:
    (a) Distinguish a DVT from a vascular tumor
    (b) Recognize lymphadenopathy that is significant enough to warrant referral to a specialist
    (c) Quantify the degree of carotid stenosis due to an observed plaque
    (d) Unequivocally identify the roots of the brachial plexus prior to interscalene nerve block injection

Answers:

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

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Part IV
Techniques for Regional Anesthesia

11

Neuraxial Blockade: Subarachnoid Anesthesia

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Introduction

The administration of a local anesthetic in the subarachnoid space acts on the spinal roots causing reversible blockage of these nerves. It is a classic technique that has been refined over time and expanded in its practical applications. The development of new drugs and special techniques has been crucial and has greatly influenced the use of spinal anesthesia and its indications.

Anatomy of the Spine

The spine consists of 33 vertebrae: seven cervical, 12 thoracic, five lumbar, five that are fused to form the sacrum, and four fused to form the coccyx. The spine has four curves. The thoracic and sacral curves have concave forward curvature and are primary curvatures formed at birth. The cervical and lumbar curves have forward convex curvatures and are secondary curvatures developed after birth. When the patient is supine, the highest point is in L3 and the lowest point is in T5 (Fig. 11.1).

The vertebrae consist of two essential parts: an anterior solid segment or body and a posterior segment or arch. The arch is divided on each side into a pedicle attached to the body and a lamina at the back. The spinous process extends backward from the junction of the two laminae. The junction of the pedicles and laminae forms the transverse process, which extends outward from each side of the arch. The pedicles of each vertebral arch are notched forming an incomplete ring, the intervertebral foramen. The spinal nerves enter and exit through these holes from each side of the vertebral canal (Fig. 11.2).

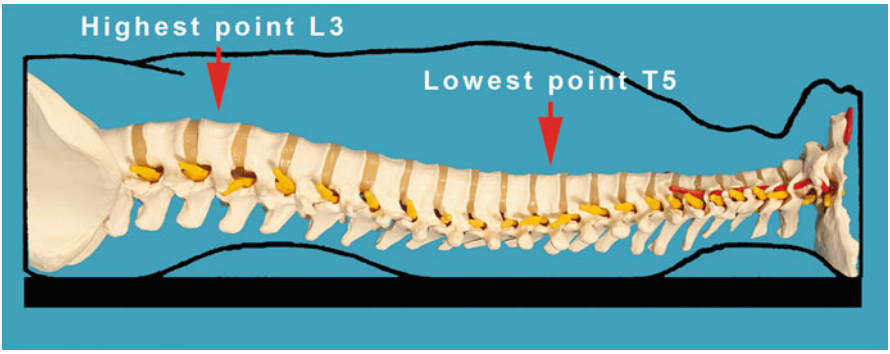

Fig. 11.1 Highest and lowest points on the spine in the supine position

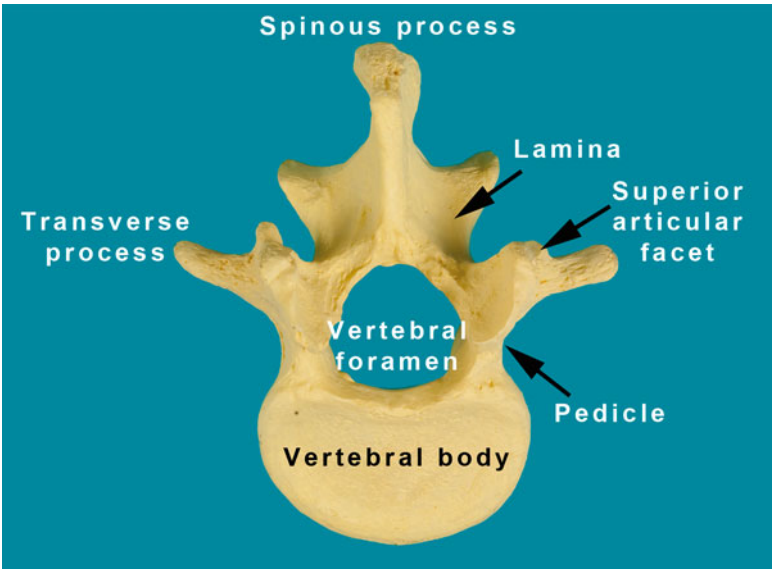

Fig. 11.2 The vertebrae

The Intervertebral Discs

The intervertebral discs are a pad of fibrous cartilage between adjacent surfaces of the vertebral bodies. They provide mobility and shock absorption to the spine.

Five Ligaments

Five ligaments connect the vertebral processes: The supraspinous ligament connects the tips of the spinous processes,and the interspinous ligament connects the spinous processes.

The ligamentum flavum connects the laminae of adjacent vertebrae and consists of elastic fibers. It becomes progressively thicker from front to back, and it is easily recognized by the increased resistance to the passage of the needle. The other two ligaments are the posterior longitudinal ligament and the anterior longitudinal ligament.

The Epidural Space

The epidural space extends from the foramen magnum to the sacral hiatus. It is bounded by the posterior longitudinal ligament at the sides by the pedicles and the intervertebral foramen and posteriorly by the ligamentum flavum. It contains nerve roots, venous plexuses, arteries, and fat.

The Spinal Cord

The spinal cord originates in the brainstem and continues through the occipital foramen magnum ending in the conus medullaris. This distal end ranges from L3 in infants up to the bottom of L1 in adults due to differences in growth between the bony spinal canal and central nervous system. It ends at the conus medullaris from where the lumbar nerve, sacral and coccygeal roots emerge to form the cauda equina (horse tail). It is in this area (below L2) that spinal needles are inserted.

Meninges and Spaces

Meninges and spaces include the pia mater, which is the innermost layer closely attached to the spinal cord and brain. It ends as terminal filum and is highly vascularized. The arachnoid is an avascular membrane tightly attached to the outermost layer, the dura mater. It seems that the arachnoid acts as a major barrier to the flow of drugs from the cerebrospinal fluid (CSF); thus, it would be responsible for 90% of the drug resistance to migration [1, 2].

The dura mater is the third and outermost membrane of the spinal canal. It is the continuation of the cranial dura mater, extending from foramen magnum to S2 (Fig. 11.3).

The subarachnoid space lies between the pia mater and the arachnoid. In it is found the CSF, the spinal nerves, a network of trabeculae between the two membranes, and blood vessels supplying the spinal cord. It extends from S2 to the cerebral ventricles. The subdural space is a virtual space between the dura and the arachnoid that contains small amounts of serous fluid that allows the membranes to move past each other.

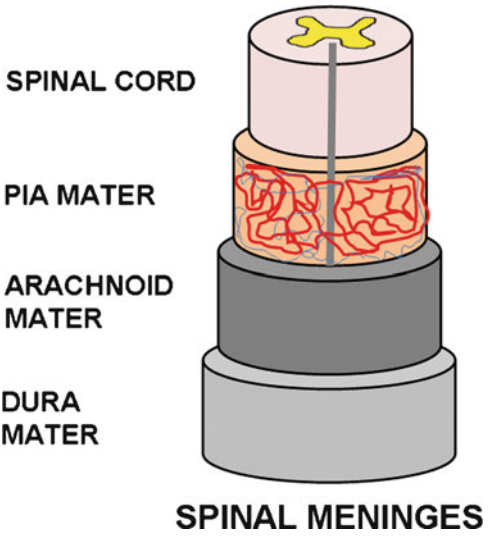

Fig. 11.3 Spinal meninges

Table 11.1 Physical properties of CSF

• Clear, Liquid, colorless
• Specific Gravity: 1,003–1,009 at 37ºC
• Total volume: 120–150 ml
• Spinal CSF volume: 25–30 ml
• Ventricular CSF volume: 60–75 ml
• Average pressure: 100–150 cm of water
• pH: 7.6

The Cerebrospinal Fluid (CSF)

The cerebrospinal fluid (CSF) is formed continuously at a rate of 450 ml/day by way of the secretion or plasma ultrafiltration from the choroidal arterial plexus located in the lateral ventricle and the third and fourth ventricle. The CSF is reabsorbed into the bloodstream through the arachnoid villi and granulations, which protrude from the dura mater to be in contact with the endothelium of the cerebral venous sinuses. The CSF is a determinant of the effects of intrathecally administered substances because all the drugs injected into the subarachnoid space are diluted in the CSF before reaching their target in the spinal cord. It has been noted that the volume of CSF is one of the most important factors affecting the level of sensory block and duration of spinal anesthesia [3, 4]. The volume of CSF varies from one individual to another and, with the exception of weight, is not related to the anthropometric values clinically available (Table 11.1).

Thirty-One Pairs of Spinal Nerves

Thirty-one pairs of spinal nerves emerge from the spinal cord by the anterior and posterior roots. Each spinal nerve innervates a specific area of skin or dermatome and skeletal muscles.

The Anterior Spinal Root

The anterior spinal root is efferent and contains:

1. Motor fibers (voluntary muscles).
2. Preganglionic sympathetic fibers (T1–L2) join spinal nerves to form the sympathetic chain. The sympathetic chain extends along the entire column (anterolateral side of the vertebral bodies). It gives rise to the stellate ganglion, splanchnic nerves, and celiac plexus.

The Posterior Spinal Root

The posterior spinal root is afferent. All afferent impulses from the body, including viscera, pass through the posterior roots. Each has a dorsal root ganglion (Fig. 11.5).

Physiology

Neural Blockade

The small-diameter unmyelinated (sympathetic) fibers are blocked more easily than larger myelinated fibers (sensory and motor). As a result, the level of autonomic blockade extends two or three segments above the sensory block, and in the same way, the sensory block extends one to four segments above the motor block. Weaker concentrations of local anesthetic can produce sensory block without causing motor paralysis. The sequence of the blockade of nerve fibers is generally in the following order: (1) vasomotor block, (2) pain, (3) touch, (4) motor, (5) pressure, (6) proprioception.

Cardiovascular Physiology

The nerve block produces sympathectomy two to four dermatomes above the sensory level. This causes arterial and venous vasodilatation, the venodilator effect being predominant because smooth muscle in the arterial side retains a considerable degree of autonomous tone. This causes a decrease in systemic vascular

resistance, venous return, and cardiac output, all of which contribute to lower blood pressure. Vasodilation in the lower extremities can be compensated by vasoconstriction in the upper extremities. However, with high thoracic anesthetic levels, vasoconstriction in upper extremities and in the splanchnic bed may be highly reduced, and this can lead to significant hemodynamic instability. Similarly, heart rate in a high neuraxial block decreases by the blockade of sympathetic cardioaccelerator fibers (T1–T5), giving rise to a predominant vagal parasympathetic tone. Furthermore, the reduction in venous return induced by spinal anesthesia paradoxically increases vagal tone, and this leads to a marked bradycardia and possible asystole [5].

Respiratory Physiology

The anesthesia in low spinal sites has no effect on ventilation. If the blockade reaches to thoracic areas, there is a paralysis of the intercostal muscles. This has little effect on ventilation since diaphragmatic breathing is regulated by the phrenic nerve. However, the patient may complain of difficulty breathing, and in patients with inadequate respiratory reserve, ventilation might be insufficient. The paralysis of both intercostal and abdominal muscles decreases the patient's ability to cough and clear secretions.

Digestive Physiology and Function

Gastrointestinal hyperperistaltism occurs as a consequence of unopposed parasympathetic activity (vagal). It can cause nausea and vomiting but responds well to atropine.

Genitourinary Physiology and Function

Sacral blockade produces an atonic bladder able to retain large volumes of urine. Efferent sympathetic blockade (T5–L1) causes an increased sphincter tone producing retention. The risk of urinary retention seems lower under spinal anesthesia with a short half-life [6, 7]. The most prudent approach is to avoid excessive use of crystalloid intravenous solutions [8].

Thermoregulation Issues

Hypothermia due to spinal anesthesia is caused by the redistribution of heat as a direct result of vasodilation accompanying sympathetic block [9–11]. This is the

most important cause of core hypothermia during the first hour [11]. Hypothermia can remain in patients who undergo major operations under spinal anesthesia if sympathetic blockade persists. Hypothermia risk factors are as follows: age less than 1 month, low temperature in the operating room, second- and third-degree burns, combined general and spinal anesthesia, age over 70, low temperature of the patient before induction, low body weight, and large blood loss, in that order [12]. To reduce the risk of intraoperative hypothermia, several strategies are recommended: (a) Monitor core temperature. (b) Perform active heating with air blankets (as treatment or, in certain cases, as a prophylactic treatment. (c) Heat fluids to approximately 37°C. (d) Maintain the temperature of the operating room to over 25°C. (e) Cover the skin to reduce cutaneous heat loss [10]. (f) Avoid high spinal blocks where possible [13].

Techniques

Preparation is important before the injection of spinal anesthesia. The site where the technique is performed must be equipped with an oxygen source, as well as immediate access to emergency drugs and equipment for resuscitation and intubation. In addition, the patient should be monitored and sedated so he is both comfortable and cooperative.

Spinal needles (Fig. 11.4) are classified into two major categories: those that have conical points and separate the dural fibers (Fig. 11.5) and the ones that cut the dura (Fig. 11.6). Among the former are the Whitacre and Sprotte needles (Fig. 11.5), and the latter will encompass the Quincke-Babcock needles (Fig. 11.6). With the needles that have conical-shaped tips and side openings, the incidence of post-dural puncture headaches (PDPH) decreases [14]. The incidence of PDPH decreases with fine-gauge needles, although it also may increase if numerous attempts to puncture are made since thin needles produce worse tactile sensation during needle placement.

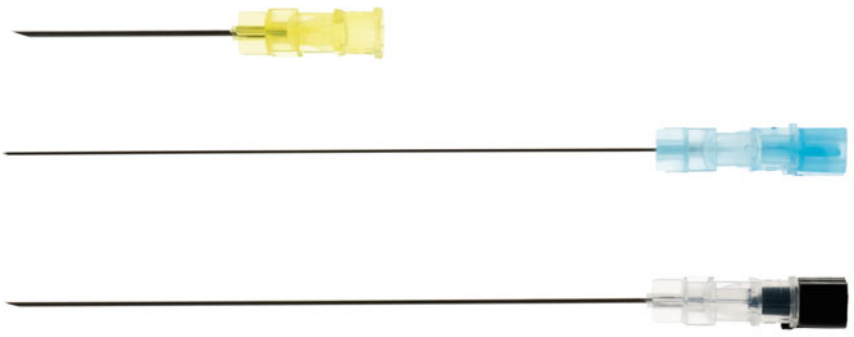

Fig. 11.4 Spinal anesthesia needle types

Fig. 11.5 Quincke-Babcock needles (sharp point)

Fig. 11.6 Whitacre and Sprotte needles (conical point)

Position of the Patient

The choice of position for spinal anesthesia is influenced by a combination of several factors: the preference of the anesthesiologist, patient characteristics, and the baricity of local anesthetic solutions in conjunction with the surgical site.

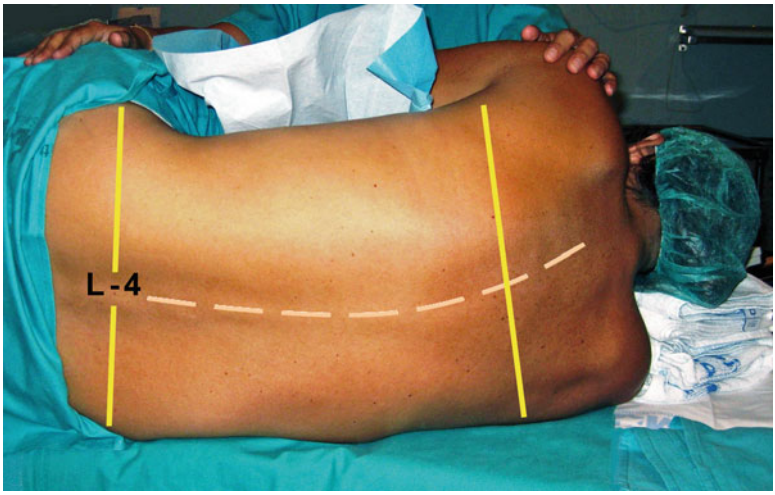

Fig. 11.7 Lateral decubitus position

Lateral Decubitus Position

The patient is placed with the affected side down if a hyperbaric solution is used or with the affected side up if the anesthetic solution is hypobaric. The spine should be horizontal and parallel to the edge of the table or bed. Maximum deflection of the column must be obtained with the knees bending to the chest, and the chin should be flexed down into the chest (Fig. 11.7).

Seated Position

The head and shoulders are bent down over the trunk, feet resting on a stool, and the patient's back should be near the edge of the table or bed. One must have an assistant to stabilize the patient, who should not be oversedated (Fig. 11.8). Many anesthesiologists prefer this position because it helps to identify the midline, especially in obese patients. The line connecting the upper edges of the iliac crests crosses the vertebral body of L4 or the L3–L4 interspaces (Fig. 11.9). If the patient is left in this position for several minutes (with hyperbaric anesthetic solution), a block to the sacral dermatomes (saddle block) is obtained. To achieve a higher spinal block, the patient is placed in supine position immediately after the intrathecal injection. One must be aware of the arterial pressure while the patient is sitting since this position favors the decrease in venous return due to sympathetic effects of spinal block.

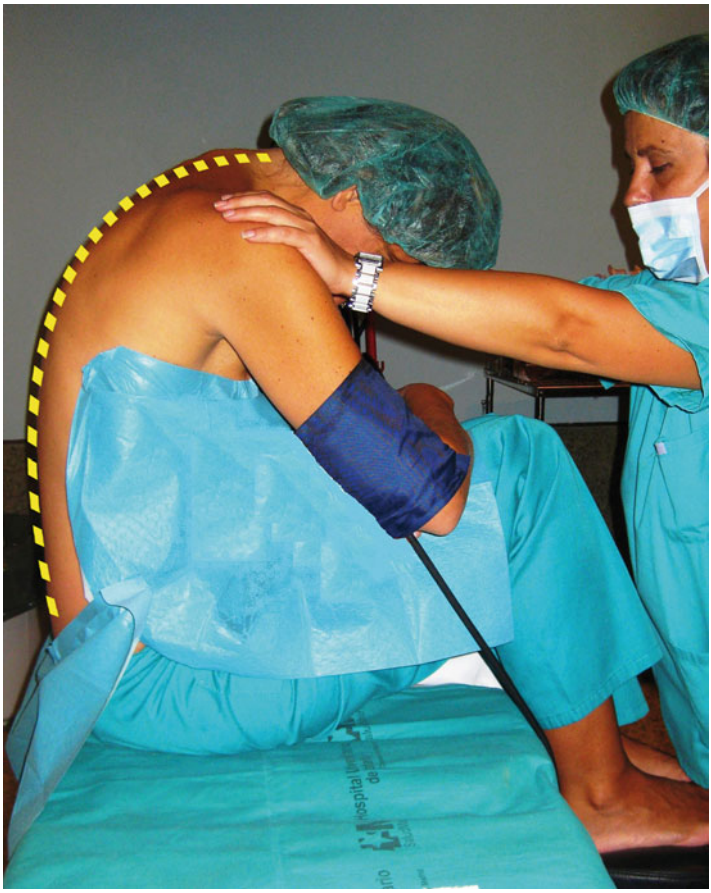

Fig. 11.8 Seated position

Prone or Jackknife Position

It is used in conjunction with hypobaric anesthesia for procedures in the rectum, perineum, and anus. It is possible to use this position both for surgery and anesthesia (Fig. 11.10).

Anatomic Approach

In general, for spinal anesthesia, spaces L3–L4 and L4–L5 are used. To avoid traumatic puncture of the conus medullaris, the puncture should be below L2. A large

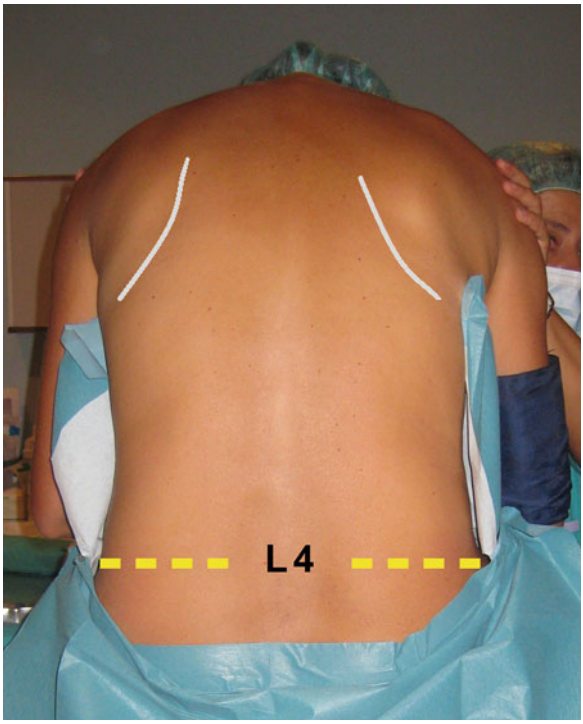

Fig. 11.9 Reference lines and vertebral body of L4

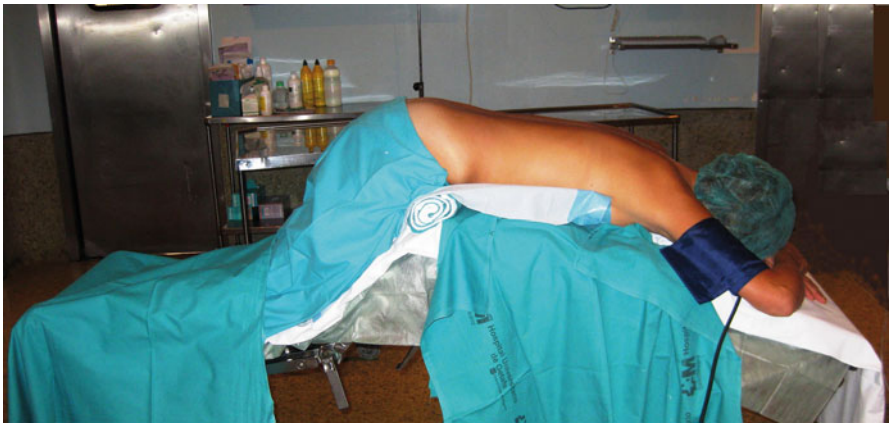

Fig. 11.10 Prone or jackknife position

area of skin should be prepared, avoiding contamination with any antiseptic solution that could be potentially neurotoxic. This should be followed with 1% lidocaine at the site of the spinal needle puncture.

Midline Approach

Using the midline pathway decreases the lumbar lordosis by inserting the spinal needle between adjacent spinous processes. By palpation, the spinous and interspinous spaces are located. A subcutaneous wheal with local anesthetic is then used. The needle used for infiltration with local anesthetic is also used to verify the alignment between the spinous processes. It is necessary to use a spinal needle introducer when the needles are small (24–25 gauges with a pencil tip). If the introducer is properly placed, it should be firmly inserted between the fibers of the interspinous ligament. A spinal needle is inserted through the introducer along its cephalic angulation. The insertion should be slow to heighten the sense of passing through tissue planes to notice the characteristic change when the needle passes through the ligamentum flavum and the dura. The stylet must be placed to prevent obstruction of the aperture with tissue. After the stylet is removed, CSF must appear. If no CSF appears, the spinal needle is rotated at 90° increments until the CSF is noticed. Flow of CSF confirms the position of the needle into the subarachnoid space, and that the tip of the needle has come into contact with part of the cauda equina. If the patient describes a paresthesia at any time, needle advancement should be stopped. Normally, paresthesia is usually transient and mild and serves as an indication that the subarachnoid space has been reached. The stylet is then removed, and appearance of CSF should be observed. If the paresthesia has been resolved, a local anesthetic injection should follow. If paresthesia recurs on injecting the local anesthetic, under no circumstances should local anesthetic be injected. It will be necessary to remove the needle and insert it again. The most common cause of not obtaining CSF is that the needle was inserted away from the midline. Another common mistake is insertion of the needle at an excessive cephalad angulation. During the injection, a gentle aspiration of 0.1 or 0.2 ml of CSF to confirm the position in the subarachnoid space could be performed before the local anesthetic injection. The midline approach is suitable for most patients, is easy to learn, and also provides a relatively avascular approach (Fig. 11.11a).

Paramedian Approach

This route is useful in patients who cannot bend properly or when the interspinous ligament is ossified. Local anesthetic is injected 1 cm lateral and 1 cm caudal to the superior spinous process. The needle with the introducer is directed medially and in a slightly cephalad direction and is passed laterally to the supraspinous ligament. The most common error, as occurs in the medial approach, is to go in an excessively cephalad direction at the insertion. Nevertheless, if a contact with the vertebral

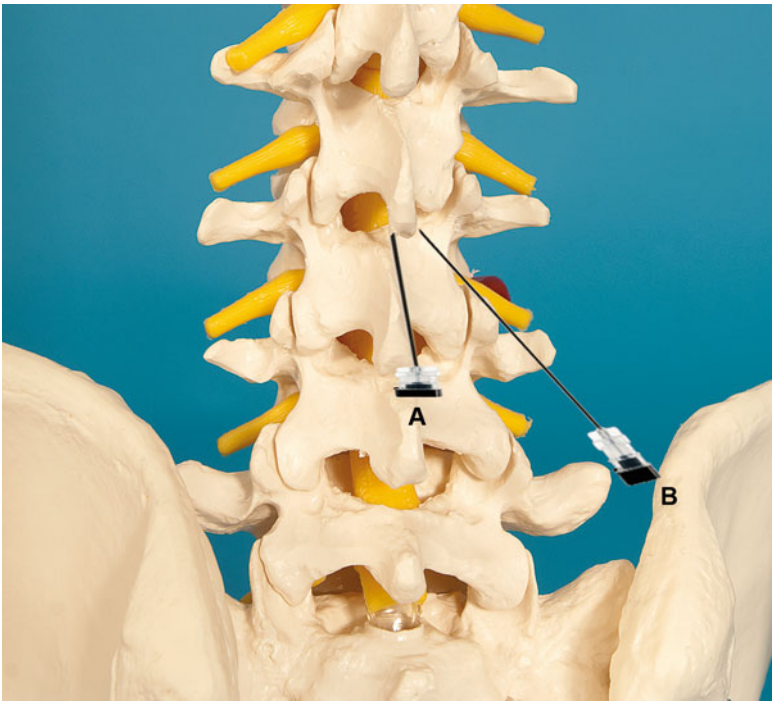

Fig. 11.11 Midline and paramedian approach

lamina is made, the needle should be redirected and introduced in a medial and cephalad direction. As in the case of the midline pathway, the characteristic feeling of the passage through the ligament and the dura can be perceived, although the needle requires a greater depth of insertion. Once CSF is obtained, the injection is performed in the same manner as described in the midline approach (Fig. 11.11b).

Lumbosacral Approach (Taylor)

The lumbosacral vertebral foramen is the largest of the spine. In patients in which the approaches described above do not allow entry into the spinal canal due to calcification, or fusion of the intervertebral spaces, the lateral oblique pathway L5–S1 space (or Taylor approximation) can be the most appropriate to reach the subarachnoid space. It can be used in a seated, lateral, or prone position. The posterosuperior iliac spine is identified, and skin is marked 1 cm medial and 1 cm caudal. Also identified and marked is the intervertebral space L5–S1. A spinal needle 120–125 mm long is inserted because the oblique angle creates a great distance to reach the subarachnoid space. A cutaneous wheal is created. Then, the needle is inserted and is

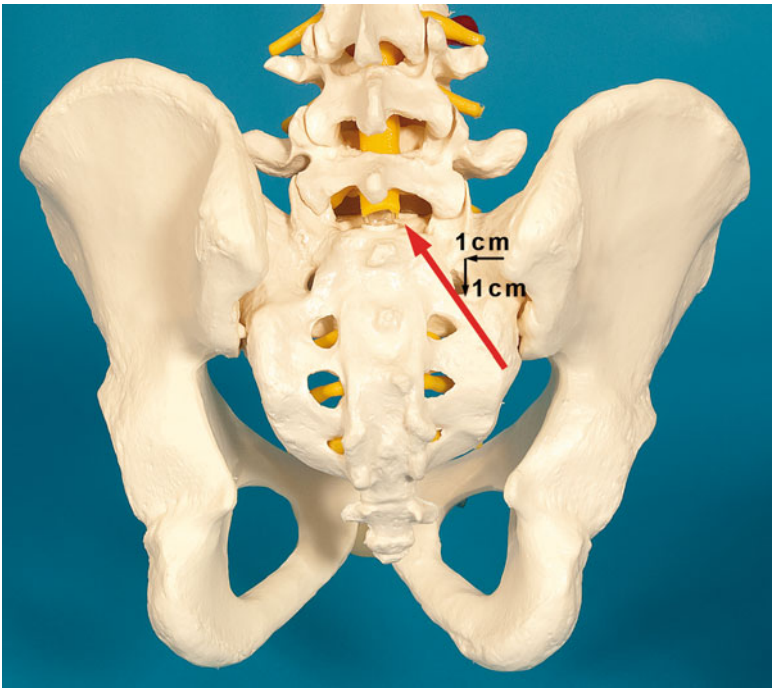

Fig. 11.12 Lumbosacral approach (Taylor)

directed 45° medial and 45° caudal to the L5–S1 space. Changes of resistance to the passage of the needle through the ligamentum flavum and dura mater are the same as in the medial pathway (Fig. 11.12).

Pharmacology

General Considerations

Local anesthetics are substances capable of producing a reversible block of the conduction in nerve fibers. Its chemical structure is an aromatic radical attached to an amine structure with a link that can be ester or amide. Amino ester anesthetics are procaine and tetracaine; the amino amides are lidocaine, prilocaine, mepivacaine, bupivacaine, and ropivacaine. The most important clinical properties of local anesthetics are potency, onset, and duration of action. They are differentiated by lipid solubility, pKa, and protein binding. They may be short duration of action or long

duration of action. The anesthetics of short duration (≤90 min) are procaine, lidocaine, and mepivacaine. Those of long duration are tetracaine, bupivacaine, ropivacaine, and levobupivacaine.

Local Anesthetics for Spinal Anesthesia

Procaine

It has been used as a local anesthetic for 100 years. Lidocaine replaced it by providing a faster onset and longer duration of action. The recent association of lidocaine with the appearance of transient neurological symptoms (TNS) has renewed interest in the use of procaine in spinal anesthesia because it seems to cause fewer TNS, but it produces more frequent failure of the blockade and also nausea. Additionally, the recuperation period is greater than with lidocaine [15].

Chloroprocaine

It is also a short-acting local anesthetic. In clinical studies with volunteers, it has been shown to have a profile similar to lidocaine, with a lower incidence of TNS.

Lidocaine

It is considered to be a local anesthetic of intermediate/short-term action. It has historically been the most widely used as a local anesthetic for spinal anesthesia. Its use has greatly diminished because of the incidence of TNS, which varies between 15 and 33% depending on the type of surgery [3].

Mepivacaine

It is also a local anesthetic of short to intermediate duration of action. It has a lower incidence of TNS (3–6%) compared with lidocaine.

Bupivacaine

It is the anesthetic most widely used for long-term action. The extent and duration of the blockade is dose dependent. However, it has a great variability due to its high lipid solubility. It is cardiotoxic, but the doses used in spinal anesthesia (maximum 20 mg) are too small to produce toxicity. In ambulatory surgery, it has been used in low doses as an alternative to lidocaine, albeit with wide variability and high failure rate [16].

Table 11.2 Doses and duration of local anesthetic in spinal anesthesia

Anesthetic	Dose (mg)	To T10 (mg)	To T4 (mg)	Duration (min)	With epinephrine (min)
Procaine	50–200	125	200	45	60
Chlorprocaine	30–100	15–40	40–100	80	–
Lidocaine	25–100	50–75	75–100	60–75	75–100
Mepivacaine	30–60	60	80	70–90	120–180
Tetracaine	5–20	8–14	14–20	70–90	100–150
Bupivacaine	5–20	8–12	12–20	90–110	100–150
Ropivacaine	8–25	12–18	18–25	80–120	–
Levobupivacaine	5–20	8–10	12–20	90–120	100–150

Ropivacaine and Levobupivacaine

These are two long-term anesthetics that have been developed as an alternative to bupivacaine for epidural anesthesia and nerve blocks due to their reduced cardiotoxicity. For spinal anesthesia, cardiotoxicity is not a relevant clinical issue because of the low dose used. Ropivacaine has lower potency than bupivacaine but also allows early motor recovery. Levobupivacaine is the L-enantiomer of bupivacaine. It is no longer marketed in the USA.

Tetracaine

It is the prototype of the ester type of local anesthetic of long duration. Compared with bupivacaine, it is more potent and has longer life, but it seems to produce more hypotension [17] (see Table 11.2).

Local Anesthetic Additives

There are two main reasons for using additives with local anesthetics: to improve the quality and duration of the spinal block and to decrease the dose of local anesthetic injected, thereby reducing the cardiovascular effects and improving the clinical profile of the spinal block [1]. Alpha-adrenergic agents and opioids are substances that are associated most frequently with local anesthetics.

Vasoconstrictors

These act by reducing the elimination of local anesthetics due to the vasoconstriction they produce, thus reducing their absorption into the systemic circulation.

Epinephrine

Its vasoconstrictor action is due to the direct alpha-adrenergic effect [17]. Clinically, the effectiveness of intrathecal epinephrine depends on the local anesthetic with which it is associated [17–20]. The recommended dose is 0.2–0.3 mg epinephrine. Large doses of epinephrine may decrease blood flow to the spinal cord, whereas doses of 0.2 mg appear to not affect the blood supply to the spinal cord [18–20]. However, they can contribute to the development of transient neurological symptoms (TNS), [21]. On the other hand, the intrathecal epinephrine significantly delays the return of the sacral autonomic function and the capacity for spontaneous urination [22].

Phenylephrine

It is used clinically in doses of 5 mg. Its use has declined since it seems that TNS increases when associated with tetracaine.

Opioids

These interact synergistically because they block afferent stimuli in action sites different from those of local anesthetics. However, they also produce different side effects such as itching, nausea, vomiting, and respiratory depression, all in a dose-dependent manner.

Morphine

It has a slow onset of action (30–60 min) and a prolonged duration of action, providing extensive postoperative analgesia. Doses of 0.1–0.2 mg provided an extensive spinal analgesia for up to 24 h for different surgical procedures such as cesarean delivery, radical prostatectomy, hysterectomy, and total hip arthroplasty. With these low doses, the risk of respiratory depression is quite rare. However, the minimum dose needed in total knee arthroplasty is 0.3–0.5 mg. With these doses, the side effects such as nausea, vomiting, urinary retention, and pruritus increased significantly compared with lower doses. The risk of respiratory depression is dose dependent.

Fentanyl

It is the most commonly used intrathecal opioid. Its lipophilic profile allows a quick onset of action (5–10 min) and intermediate duration of action (60–120 min).

Doses of fentanyl of 20–25 mcg, in association with lidocaine or bupivacaine, prolong the duration of anesthesia without increasing complete sensory, motor, and bladder recovery time.

Determinants of Intrathecal Local Anesthetic Distribution

Baricity

Baricity is a ratio that compares the density of one solution in another. In the case of spinal anesthesia, it is defined as the ratio between the density of the local anesthetic solution compared with the density of the patient's CSF at 37°C. Local anesthetic solutions that have the same density as the CSF are called isobaric. Local anesthetic solutions with a higher density than the CSF fluid are called hyperbaric, while solutions with a lower density than the CSF fluid are called hypobaric. Hyperbaric solutions will flow in the direction of gravity and settle in the most dependent areas of the intrathecal space, while hypobaric mixtures will rise in relation to gravitational pull. The effects of gravity are determined by the choice of the patient's position and in the supine position on the curvatures of the spine. There is wide variability in the density of CSF among the various population subgroups. Therefore, a local anesthetic solution can be isobaric in one individual while hypobaric in another.

Hyperbaric Spinal Anesthesia

Hyperbaric solutions are prepared by mixing the local anesthetic solution with glucose. With the patient in supine position, hyperbaric solutions tend to be distributed by gravity to the most sloped points of the thoracic (T6–T7) and sacral (S2) curves. If an anesthetic hyperbaric solution is injected into a patient in a seated position, its distribution will be restricted during 5–10 min to the lumbosacral dermatomes, thereby producing a "saddle block." Similarly, if hyperbaric solutions are injected in the lateral supine position with the surgical side sloped down, and remains in this position for 10–15 min, it is possible to get a unilateral spinal anesthesia.

Hypobaric Spinal Anesthesia

Commercial solutions are prepared by diluting isobaric solutions with sterile distilled water. Although it is less commonly used for perineal and perirectal surgical procedures performed with the patient in the prone position or "jackknife" position, it provides significant advantages in hip surgery in lateral position.

Isobaric Spinal Anesthesia

These solutions are not completely isobaric but tend to be slightly hypobaric. Consequently, depending on the patient's position during the injection and during surgery, the solution can behave unpredictably rather than as a true isobaric solution. Isobaric solutions are not distributed far from the initial point of injection and are particularly useful when sensory block in the high thoracic dermatomes is not desirable.

Dose, Volume, and Concentration

The importance of concentration, dose, and volume of different local anesthetic solutions on the extent of spinal block has been considered. A change in one of the variables causes changes in the others. However, it appears that the dose (as weight in mg) is the most important of the three. The volume and concentration are not as important. The greater the dose of local anesthetic injected, the greater the extent and the duration of the blockade [23, 24].

Intervertebral Space for the Injection

Different researchers have studied the importance of injection site in the extension of the spinal anesthesia. It seems that the effect of the injection site is superseded by the baricity of the anesthetic solution and the position during the injection.

Position of the Side Holes of the Spinal Needle

This is of importance when using pencil-point needles with a side opening (Whitacre and Sprotte). If the hole is directed either caudal or cranial, it appears that it may affect the extent and duration of the spinal anesthesia differently [25].

Age

With increasing age, there appears to be a tendency to increase the extent of spinal anesthesia. This seems to be related to decreased CSF and to the demyelination that occurs in elderly patients [17, 26, 27].

Height

There is no significant correlation between height and extension of the anesthesia [17].

Table 11.3 Dermatomal levels of spinal anesthesia for common surgical procedures

Procedure	Dermatomal
Hysterectomy, cesarean delivery, inguinal herniorrhaphy, appendectomy	T4
TURP(transurethral resection of the prostate), cistoscopy, hysteroscopy, total hip replacement, femoral popliteal bypass, varicose vein stripping	T10
Lower extremity surgery with tourniquet use. Knee replacement and arthroscopy. Below knee amputation	T8–T10
Foot and ankle surgery	L1
Perirectal and perineal	S2–S5

Body Mass Index

It seems that there is a tendency toward an increase in the extension of the blockade in obese patients, but a significant statistical correlation has not yet been observed. The most likely mechanism is the compression in the subarachnoid space due to an increase in abdominal mass that produces a decrease in the CSF causing the extent of the spinal anesthesia to be higher. This also occurs in pregnant patients [28, 29].

Indications and Contraindications

Indications

The anesthesiologist must determine the correct segmental level for surgery to be performed and also assess that the physiological effects of the required anesthetic level are not harmful to the patient. Visceral sensitivity and viscerosomatic reflexes have spinal segmental levels that are much higher than what could be predicted from skin dermatomes. Table 11.3 shows the levels needed for common surgical interventions.

Contraindications

- The most important contraindications for spinal anesthesia are patient refusal and increased intracranial pressure. Other contraindications may be infection of the puncture site, severe hypovolemia, or coagulation disorder. For patients with preexisting neurological diseases (peripheral neuropathies, demyelinating diseases), it is controversial because there is no clinical study showing that spinal anesthesia worsens these diseases. Rather, it appears that the contraindication is largely based on legal considerations.

Complications

Cardiovascular Side Effects

The most frequent and severe side effects are hypotension and bradycardia. There is an incidence of hypotension around 33% in nonobstetric populations [3]. Risk factors for hypotension in the nonobstetric population include block height above T5, age over 40, systolic blood pressure less than 120 mmHg, and chronic hypertension. The severity of the decline in blood pressure correlates with the height of the blockade and with the patient's intravascular volume [30]. Prophylactic measures to prevent hypotension include prehydration with crystalloids or colloids or administration of vasoactive drugs [1, 3]. The crystalloid solutions are quickly distributed from intravascular to extravascular space. It has been observed that the administration of Ringer's lactate during the induction of spinal anesthesia is more effective than when it is administered 20 min before [31]. Administration of large volumes of crystalloids (more than 1 l) does not seem to offer great additional benefits over small volumes (250 ml) and may be harmful in patients with limited cardiopulmonary reserve [3]. Prophylactic treatment with vasoactive agents may be more effective than prehydration for the prevention of hypotension [32]. Ephedrine, in intravenous increments of 5–10 mg, produces increased cardiac output in addition to vasoconstriction. Phenylephrine causes increased peripheral vascular resistance but can decrease the frequency and cardiac output. It would be second choice, especially if tachycardia is present.

The incidence of bradycardia is about 13% in nonobstetric populations. The heart rate decreases with a high block height by blockade of cardioaccelerator sympathetic fibers and a decrease in venous return, leading to a predominant vagal tone. Risk factors favoring bradycardia include block height above T5, decreased age (American Society of Anesthesiologists physical status) ASA I, average heart rate <60 BPM, prolonged PR interval, and treatment with beta blockers. Cardiovascular collapse associated with spinal anesthesia is not common and often is preceded by bradycardia and hypotension. This rare but severe complication seems mainly related to decreased venous return to the heart which activates vagal tone. The incidence of cardiac arrest is difficult to determine and depends on the interpretation of data and definitions. Large observational studies indicate an incidence of (0.04–10)/10,000 [33]. The excessive sedation and a delay in treatment with vasoactive drugs may exacerbate the effects of hypotension and bradycardia. Therefore, treatment should be immediate and aggressive. In addition to treatment with crystalloids and/or colloids, treatment should be continued in steps with atropine (0.5–1 mg), ephedrine (25–30 mg), and epinephrine (0.2–0.3 mg) [33].

Total Spinal Anesthesia

Total spinal anesthesia occurs when the local anesthetic spreads so high that there is a sensory block of all the spinal cord beyond the cervical region. Subsequent to a complete sympathetic block, severe hypotension and bradycardia occur, followed

by respiratory arrest and loss of consciousness. Fortunately, when the local anesthetic spreads so cephalad, the total amount is low, and the motor paralysis is limited and of short duration. Recognition and prompt treatment are essential to prevent cardiac arrest and hypoxic brain injury. Supportive treatment for the duration of the blockade includes vasopressors, atropine, and fluids, in addition to oxygen and controlled ventilation. Morbidity and mortality should not occur if ventilation and circulation are maintained until the blockade is resolved [34].

Subdural Anesthesia

The subdural space is a potential space between the dura and arachnoid, which contains only small amounts of serous fluid that allows the two membranes to move over each other. On rare occasions, during the course of spinal or epidural anesthesia, local anesthetics may be injected into this space. If the amount of local anesthetic is small (spinal anesthesia), the result is an extensive but minimal anesthesia and may explain many cases of failed spinal anesthesia. If the dose was injected for epidural anesthesia, a wide spreading of the local anesthetic into the subdural compartment can occur with an unexpected spread of the sensory and motor blockade with symptoms resembling total spinal anesthesia.

Spinal Hematoma

The formation of a hematoma within the spinal canal can produce spinal cord compression and ischemic damage. The hematoma can occur in patients with normal coagulation because of damage from the needle or catheter in the epidural venous plexus, but the risk is increased in patients with impaired hemostasis. It is estimated that the incidence of hematoma is less than 1/150,000 in the case of epidural puncture and 1/22,000 in the case of subarachnoid puncture. Suspect a potential hematoma problem when a spinal block is unusually long. Early detection is critical because a delay of more than 8 h in decompressing the spinal cord worsens the prognosis [35, 36].

Infectious Complications

These may occur as localized infection of the skin, spinal abscess, or meningitis. Spinal abscess manifests itself as back pain accompanied by radicular pain, motor deficits, and fever. The diagnosis is made with an MRI. Treatment includes intravenous antibiotics and drainage/surgical decompression.

Neurological Complications

The incidence of neurological injury in large series of spinal and epidural anesthesia ranges from 0.03 to 0.1% [37, 38]. Blunt trauma from the needle causes paresthesia. Intraneural injection causes a more severe paresthesia and worsens nerve injury. Therefore, when a paresthesia occurs while performing a spinal puncture, the advancement of the needle should be stopped, and the local anesthetic injection should be discontinued by removing the needle and waiting for the disappearance of paresthesia. Laboratory studies suggest that all local anesthetics are potentially neurotoxic, but clinical experience suggests that nerve injury induced by these agents is rare. The syndrome of the cauda equina is associated with the use of microcatheters during continuous spinal anesthesia, as well as with 5% lidocaine [39]. Transient neurological symptoms or transient radicular irritation is a syndrome that manifests itself with back pain that radiates to the thighs and lower extremities after spinal anesthesia. The pain on a scale 1–10 has an average value of 6.2. Most patients report an onset of symptoms from 12 to 24 h after surgery for a period of between 6 h and 4 days. The neurological examination is standard [37]. Included among the risk factors are ambulatory surgery, surgical position (in lithotomy, knee arthroscopy), and obesity. One of the most important risk factors is the use of lidocaine. Although all local anesthetics can produce TNS, lidocaine is the one with a higher incidence. Although this is a transitory situation, its symptoms can be very disabling. Treatment is symptomatic with anti-inflammatory analgesics and opiates [37].

Hearing Loss

Incidence is 0.4–0.5%. This complication has been described with increasing frequency. It has been demonstrated by audiometry [40]. It is believed to be due to loss of CSF after lumbar puncture and lower CSF pressure that is transmitted to the perilymph [41].

Nausea

The most frequent causes are hypotension, the predominance of vagal tone that produces sympathetic blockade leading to gastrointestinal hyperactivity and the use of intrathecal opioids.

Post-dural Puncture Headache [42]

This is a common complication of spinal anesthesia. The mechanism appears to be related to the leak of CSF through the puncture site. The leak causes a decrease in CSF pressure and traction of the intracranial structures when the patient changes

position from supine to seated [1]. It is an intense occipital headache radiating to the posterior cervical region and may be accompanied by nausea, vomiting, and photophobia. The main characteristic is the positional nature. Other neurological symptoms, such as diplopia and hearing loss, may indicate a severe case of dural puncture headache. It occurs between 12 and 48 h after puncture and usually lasts no more than 2–4 days. It is more common in the young and females. Maternity cases present the greatest risk, and this relates to the type of needle employed or to the increase in abdominal pressure.

The differential diagnosis with regard to other types of headaches, such as meningitis, subarachnoid hemorrhage, and those associated with eclampsia and preeclampsia, should be made. During recent years, the incidence of post-dural puncture headache has decreased, thanks to the use of smaller needles and the introduction of pencil-point needles [43].

Social factors influence treatment because it is a headache that is relieved by supine position and worsened with movement. Thus, it does not affect a woman who has to care for her newborn child in the same way as it would a recently operated patient who cannot move (e.g., trauma patients). Therefore, the treatment of dural puncture headache is, initially, rest, hydration, and treatment with NSAIDs. The treatment of choice is the epidural blood patch. If the headache is severe and lasts more than 24 h, a treatment should be initiated consisting of a spinal epidural injection of 10–15 ml of the patient's sterilized blood. The effectiveness is between 70 and 98% but may require a second blood patch. After the completion of the blood patch, the patient should be lying down for 1–2 h.

Clinical Pearls

Anatomy of the Spine

- The ligamentum flavum is easily recognized by the increased resistance to the passage of the needle.
- The spinal needles are inserted below L2.
- The arachnoid acts as a major barrier to the flow of drugs from the CSF.
- The subdural space is a virtual space between the dura and the arachnoid.
- The volume of CSF is one of the most important factors affecting the level of sensory block and duration of spinal anesthesia.
- Each spinal nerve innervates a specific area of skin or dermatome and skeletal muscles.

Physiology

- The heart rate and the blood pressure in a high neuraxial block decrease by the reduction in venous return that also increases the vagal tone, and this leads to a marked hypotension, bradycardia, and possible asystole.

- If the blockade reaches to thoracic areas, ventilation might be insufficient.
- Hypothermia is caused by the redistribution of heat as a direct result of vasodilation accompanying sympathetic block.

Techniques

- Appropriate sedation may be used although it is important to communicate with the patient.
- Patient positioning and subsequent repositioning must be carefully considered in real time.
- The technique should be done in the shortest time possible without excessive haste. Refrain from making multiple punctures.
- The site should have prompt access to equipment for resuscitation and intubation.

Pharmacology

- Spinal blockade anesthetics should produce (a) a rapid onset to facilitate the start of surgery, (b) a duration commensurate with the length of the surgical procedure, and (c) a recovery that facilitates the expected recovery time and patient discharge.
- Lidocaine use has decreased due to the incidence of TNS, which varies between 15 and 33% depending on the type of surgery.
- Additives to the anesthetic improve the quality and duration of the spinal block and decrease the dose of the local anesthetic injected, thereby reducing the cardiovascular effects and improving the clinical profile of the spinal block.
- The risk of respiratory depression with intrathecal morphine is dose dependent.
- The three most important factors in determining distribution of local anesthetics are baricity, position of the patient during and just after injection, and dose.
- The anesthesiologist must determine the correct segmental level for surgery and must also assess that the physiological effects are not harmful to the patient.
- The contraindications for spinal anesthesia are patient refusal, increased intracranial pressure, infection of the puncture site, severe hypovolemia, or coagulation disorder.

Complications

- The severity of the decline in blood pressure correlates with the height of the blockade and with the patient's intravascular volume.
- A large volume of crystalloids does not offer great benefits over small volumes and may be harmful in patients with limited cardiopulmonary reserve.

- Cardiovascular collapse associated with spinal anesthesia is not uncommon and often is preceded by bradycardia and hypotension.
- A potential hematoma should be suspected when a spinal block is unusually long.
- When a paresthesia occurs while performing a spinal puncture, the advancement of the needle and the local anesthetic injection should be stopped, and disappearance of paresthesia should be awaited.
- If neurological damage is suspected, immediate diagnosis is essential.
- TNS is a syndrome with back pain that radiates to the thighs and lower extremities after spinal anesthesia. The risk factors are the use of lidocaine and surgical position.
- The main characteristic of post-dural puncture headache (PDPH) is the positional nature. The treatment of choice is the epidural blood patch.

Multiple-Choice Questions

1. A female, 60 years old, is perineorrhaphy operated on in the lithotomy position under spinal anesthesia. The next day, she complains of severe low back pain radiating down her legs. What is the most common cause of this pain?
   (a) TNS (transient neurological symptoms)
   (b) Epidural spinal hematoma
   (c) Epidural abscess
   (d) Decubitus

2. What is the path you should follow?
   (a) Consult with a neurologist
   (b) MRI
   (c) CT at 24 h
   (d) Expect spontaneous resolution

3. Which of the following is true of post-dural puncture headache?
   (a) Commences within 12–48 h of dural puncture.
   (b) Traction of the intracranial structures appears when the patient changes position from supine to seated.
   (c) The technique of blood patch is between 70 and 98% effective. Some cases require a second patch.
   (d) All of the above.

4. What is the minimum level to be achieved for spinal anesthesia in cesarean delivery?
   (a) T1
   (b) T4
   (c) T10
   (d) T8–T10

5. In relation to spinal anesthesia and "transient neurological symptoms" (TNS):
   (a) One of the most important risk factors is the use of lidocaine.
   (b) Onset of symptoms appears between 6 h and 4 days after surgery.
   (c) Although this is a transitory situation, its symptoms can be very disabling.
   (d) All of the above.

6. In a neonate, the spinal cord terminates at the lower border of:
   (a) T12
   (b) L1
   (c) L2
   (d) L3

7. In the case of a patient sitting upright with his arms by his side, a line drawn between the tips of the scapulae will correspond to the vertebral body of:
   (a) T6
   (b) T7
   (c) T8
   (d) T9

8. Indicate which of the statements is false:
   (a) The subarachnoid space lies between the pia mater and arachnoids.
   (b) The subdural space is a virtual space between the dura and the arachnoids.
   (c) C Spinal CSF volume is approximately 25 ml.
   (d) Ventricular CSF volume is approximately 150 ml.

9. Risk factors for hypotension in the nonobstetric population include:
   (a) Block height greater than T5.
   (b) Systolic blood pressure less than 120 mmHg.
   (c) Chronic hypertension.
   (d) All of the above.

10. Which of the following is true of the baricity of anesthetic solutions?
    (a) Local anesthetic solutions that have the same density as the CSF are called hyperbaric.
    (b) Local anesthetic solutions with a higher density than the CSF fluid are called isobaric.
    (c) Hyperbaric solutions are distributed to areas most nondependent on intrathecal space while the hypobaric are distributed to dependent intrathecal areas.
    (d) The effects of gravity are determined by the choice of the patient's position and in the supine position on the curvatures of the spine.

11. The line connecting the upper edges of the iliac crest crosses the vertebral body of:
    (a) Interspaces L2–L3
    (b) L3
    (c) L4
    (d) Interspaces L4–L5

Answers:

1. a
2. a
3. d
4. b
5. d
6. d
7. b
8. d
9. d
10. d
11. d

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12

Neuraxial Blockade: Epidural Anesthesia

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Introduction

The first epidural injection was performed in 1901 by Jean-Athanase Sicard and Ferdinand Catheline through caudal route. Touhy needle was developed for continuous spinal catheter technique and later adapted for epidural anesthesia by Manual Martinez Curbelo. Its popularity increased due to the possible serious neurological sequelae of spinal injections and the availability of long-acting local anesthetic agents such as bupivacaine. As a result of its versatility, it certainly remains a popular regional anesthetic technique in the USA and UK. It can be used as a sole anesthetic or for extended analgesia extending from the neck downward covering various surgical subspecialties. It is also a useful technique in the management of chronic pain syndromes and cancer pain. Epidural space is approachable at all levels of the vertebral column, and it provides segmental analgesia. This is yet another favorable feature.

Epidural analgesia has shown to be beneficial in reducing postoperative complications following surgery, and these include reduced cardiovascular, pulmonary, and metabolic dysfunctions. It promotes improved wound healing and decreases incidence of venous thrombosis [1].

In comparison with spinal, epidural injection takes longer time to perform, is technically more challenging, slow in onset, and the motor blockade produced is less dense. The incidence of post-dural-puncture headache (PDPH) is significantly higher than with spinal injections especially when spinal injections are performed with fine gauge needles that split (instead of cutting) the dural fibers, e.g., Whitacre needle.

Anatomy of Epidural Space

An understanding of the anatomy of the epidural space is essential to safely and successfully utilize it for the purposes of performing this regional anesthetic technique. An effort to create a mental three-dimensional anatomical image could prove invaluable in mastering the technique.

The epidural is also known as the extradural or peridural space and extends all along the spinal column from the base of skull to the tip of the sacrum. It encircles the dura from the dural reflections at the foramen magnum cranially down to the sacrococcygeal ligament inferiorly. It is thinnest in the cervical region (2 mm) and thickest in the lumbar region (6 mm).

The vertebral column is made up of 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 3–5 coccygeal vertebrae. The latter two are fused together to form the sacrum and the coccyx, respectively. Though the morphology of the five types of vertebrae differs considerably with regard to size and shape, the basic components for the vertebra remain same. These are the anterior body, lateral pedicles, and posterior spinous process. The lamina and the pedicle form the posterolateral structures (Fig. 12.1). The size and shape of the vertebrae vary as we move down along the vertebral column from the cervical to sacral region (Fig. 12.3), which has implications on the technique of needle insertion into the epidural space. Of notable importance is the variation in angle of the spinous process at the various levels. In the cervical and lumbar regions, the spinous process is almost horizontal which permits a midline approach to the space, whereas in the thoracic region, these processes are more acutely angled; a paramedian approach is easier than a midline approach.

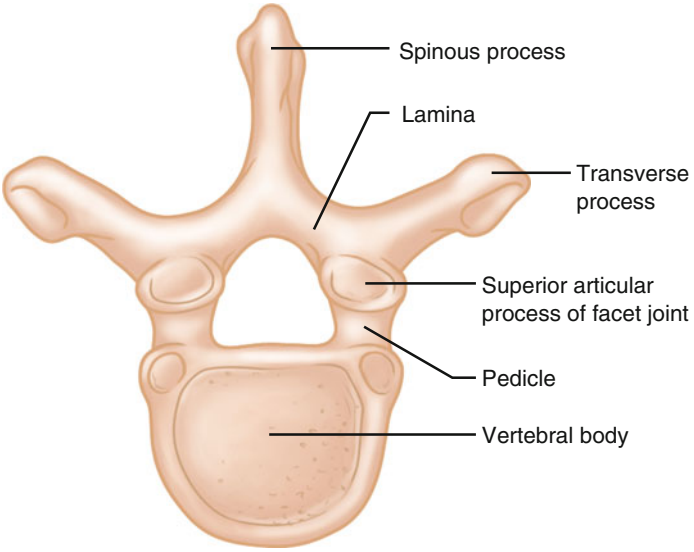

Fig. 12.1 Posterolateral structures

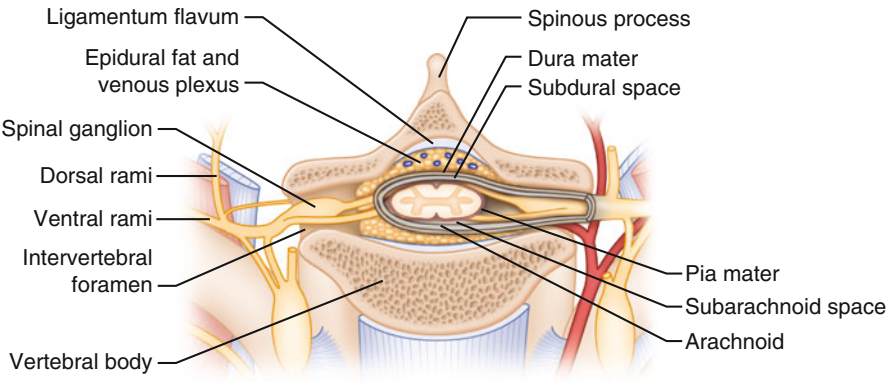

Fig. 12.2 Contents of the epidural space

The anatomical borders of the epidural space are superiorly the foramen magnum, inferiorly the sacrococcygeal ligament, and anteriorly the vertebral bodies and intervertebral disks, and the inferior border is formed by the sacrococcygeal ligament which covers the sacral hiatus and fuses with the coccyx.

Ligaments

There are three important ligaments that provide posterior support for the vertebral column and are important when accessing the space as the epidural needle introduced through the midline will pass though all the three. The *supraspinous* ligament is a continuation of the ligamentum nuchae, and this thin structure that runs all along the vertebral column joining the tips of the adjacent spinous processes. Its thickness increases gradually from above downward, and it is maximum in the lumbar region. The *interspinous ligaments* lie beneath the supraspinous ligament and connect adjacent spinous processes. They are thin and inconsequential. The *ligamentum flavum* is a midline structure, which is paired and usually fused in the midline. It is much thicker and offers resistance to a needle passing through it (when the two halves are separate, then it can lead to difficulties in identifying the epidural space by the midline approach with increased risk of dural tap). The *ligamentum flavum* is thinnest in the cervical region and thickens in the thoracic and lumbar region. This is the only ligament that is encountered in the paramedian approach to the epidural space.

Contents of the Epidural Space (Fig. 12.2)

Epidural space contains *fat*, *epidural veins*, *spinal nerve roots*, and *connective tissue*. *Epidural fat* lies between the dura and the vertebral canal and surrounds the spinal cord. It might have a protective role in reducing accidental dural tap during epidural

Table 12.1 Anatomical landmarks for epidural siting

Surface marking	Vertebral level
Vertebra prominence	C7
Root of spine of scapula	T3
Inferior angle of scapula	T7
Rib margin 10 cm from midline	L1
Superior aspect of iliac crest	L4
Posterior superior iliac spine	S2

needle insertion. To some extent, this fat can potentially act to modify the effect of drugs injected into the epidural space depending on their lipid solubility. However, the exact role played by this is not very clear. *Epidural venous plexus* is composed of a network of valveless veins known as Batson plexus. They form a reticular network in the epidural space and can transmit pressure fluctuations in the thorax and abdomen as happens during coughing, straining, or during pregnancy. In pregnancy, especially during active labor, these epidural plexuses become highly engorged reducing the volume of the epidural space. The spinal nerve roots lie in the epidural space and, as they exit the spinal cord, carry a short length of the dura, which forms a cuff around these roots. Finally, *the connective tissue* loosely arranged in the epidural space may have some bands and poorly defined septae which can rarely interfere with passing of a catheter or spread of local anesthetic solutions.

Surface Anatomy

Surface landmarks (Table 12.1) and palpation are most commonly used to identify intervertebral level, although both lack accuracy. The vertebra prominens is the most prominent structure noticeable descending down the vertebral column. The other useful surface landmarks are demonstrated in Fig. 12.3.

Special Anatomical Considerations in Caudal Epidural

Although the termination of the spinal cord (conus medullaris) is generally at the level of L2, the cauda equina extends for a variable distance below this (see Fig. 12.4) and remains encased within the dural sac. The dural sac extends down into the sacral canal. Epidural space can be accessed here in the form of caudal epidural anesthesia. Access to the epidural space is via the sacral hiatus (see Fig. 12.5). The sacral hiatus is the area of S5 (there is significant individual variability) where the spinous process is absent. The sacral hiatus can be identified cephalad to the coccyx and in-between the two sacral cornua.

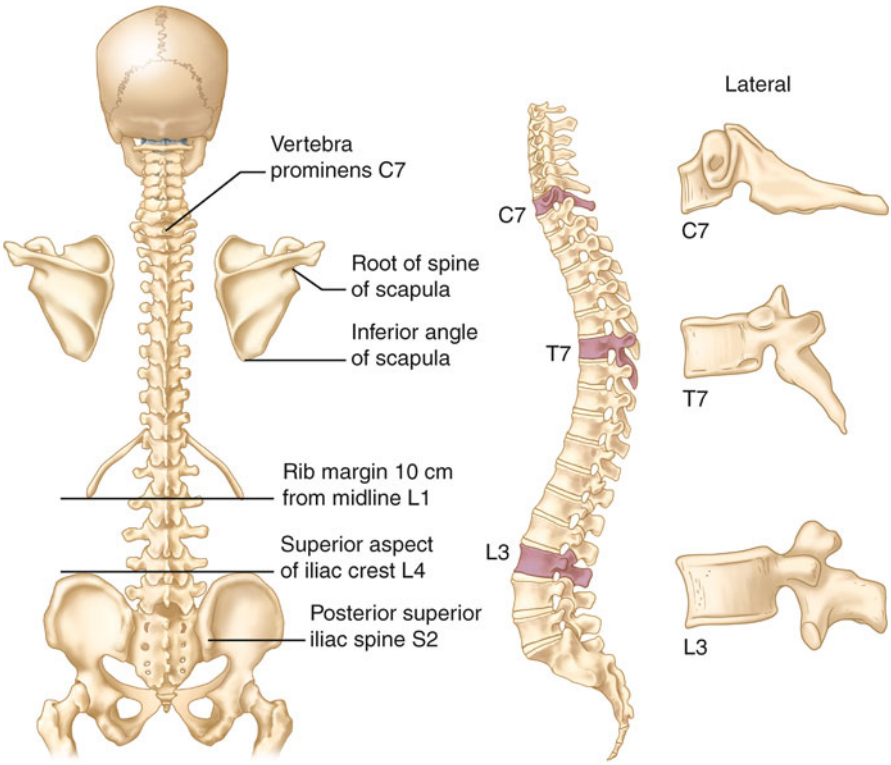

Fig. 12.3 Surface landmarks

An important point to remember is that in some patients, variations in anatomy result in the coccyx and therefore, the sacral hiatus lying in very close proximity to the anus, which may increase the infection risk of caudal anesthesia.

Physiological Effects of Epidural Blockade [2]

Physiological effects of epidural local anesthetic injections are similar to that of subarachnoid injections. The key difference is in the onset time and the segmental nature of the block produced which is related to the restricted epidural spread of the drugs injected. This is an advantage when a slow-controlled establishment of blockade is required in a given clinical situation (patients with cardiovascular or respiratory illnesses).

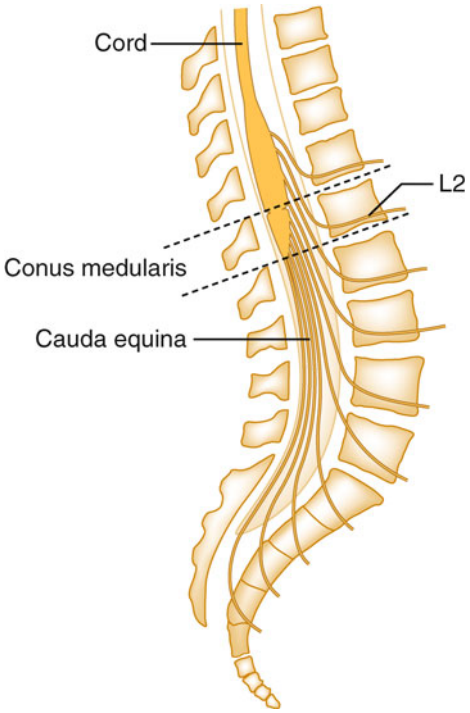

Fig. 12.4 Cauda equina

Nervous System

The principle of epidural anesthesia is based on the fact that the spinal nerve roots exiting the spinal cord are amenable to blockade with local anesthetic drugs as they pass through the epidural space (see Fig. 12.2).

Autonomic Nervous System

Epidural blockade affects both the sympathetic and peripheral nervous systems. The sympathetic nerves exit the spinal cord between T1 and L2, and blockade of these nerves frequently results in vasodilatation and subsequent hypotension. Blockade at T1–T5 may affect cardiac branches resulting in a reduction in myocardial oxygen demand by reducing inotropy and chronotropy.

Peripheral Nervous System

The dermatomal distribution of the sensory nerves is shown in Fig. 12.6. Epidurals provide a segmental blockade of the nervous system with caudal and cephalad

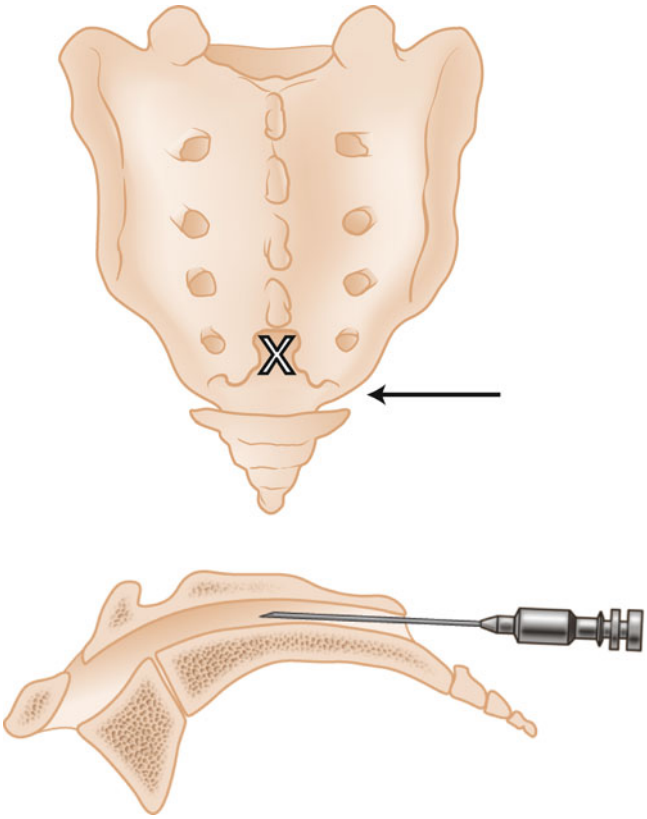

Fig. 12.5 Needle insertion

spread from the point of insertion. This is in contrast to spinal anesthesia, which generally provides complete neural blockade below and, to a variable level above, the level of injection.

Cardiovascular System

The cardiovascular effects of epidural anesthesia depend on the level of block produced and are due to sympathetic system blockade. It also depends on the type and dosage of agents used. Extensive epidural blockade with local anesthetic agents will lead to reduction in blood pressure due to vasodilatation reduced venous return and reduced adrenal medullary secretions. Compensatory vasoconstriction in upper part of body can lead to baroreceptor-mediated reflex bradycardia [3]. This feature of epidurals can be useful in that a selective lumbar or thoracic blockade has the benefit of providing hemodynamic stability as a result of compensatory vasoconstriction in unblocked segments.

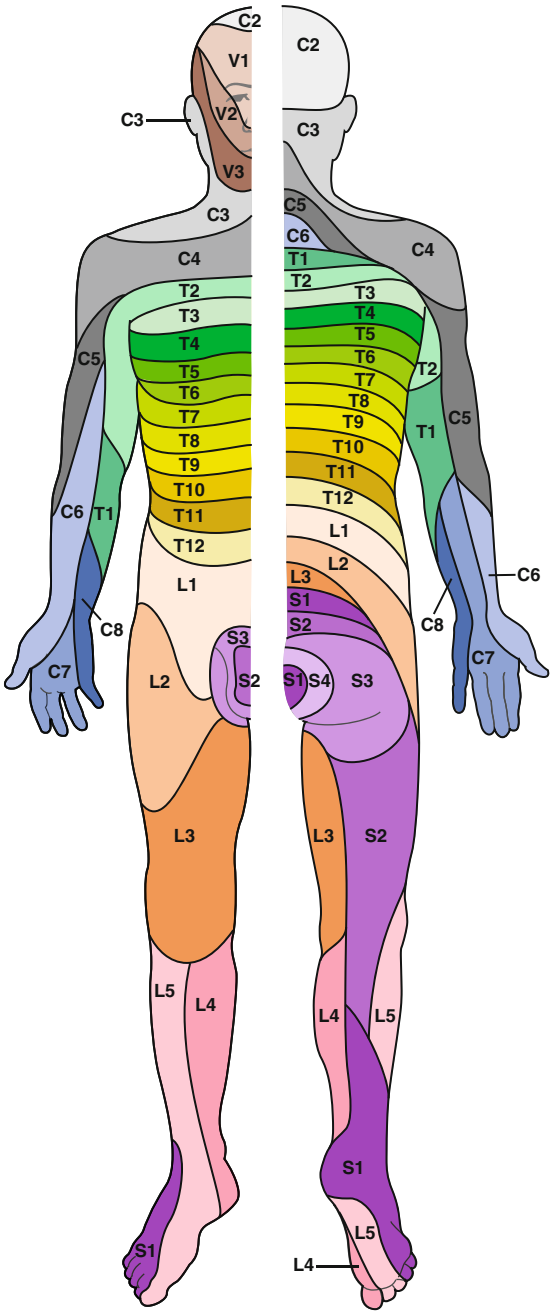

Fig. 12.6 The dermatomal distribution of the sensory nerves

A reduction in heart rate may be observed with high thoracic blockade, and this is due to inhibition of the cardioaccelerator fibers of T1–T5. There is a theoretical benefit of improved coronary oxygenation with reduction in heart rate and coronary

vasodilatation with selective thoracic epidural provided the blood pressure is maintained either spontaneously or by pharmacological means.

Respiratory System

Epidural blockade has minimal effect on respiratory function unless the level of block produced is too high. A high thoracic block can cause distress to patients as they become unable to breathe or feel that they are breathing as a result of sensory block. Diaphragmatic paralysis occurs only if the block level is above C5 (phrenic nerve C3–C5), which is not very common. On the contrary, epidural analgesia improves respiratory function if used for postoperative analgesia. The analgesia with comparatively lower density of motor block helps patient to be pain free with minimal effect on the pulmonary function reducing the risk of postoperative respiratory dysfunction.

Gastrointestinal System

Gastrointestinal effects are due to alterations in the autonomic nervous system. The splanchnic nerve blockade (T5–L1) produced can lead to unopposed parasympathetic activity culminating in increased GI secretions, hypermotility, and relaxation of sphincters. These effects could be beneficial in that a small contracted bowel improves access during bowel surgery. An increase in visceral perfusion and early return of postoperative GI motility are preferable following operation on the gut. Nausea and vomiting observed are secondary to increased vagal tone and reduction in blood pressure.

Genitourinary System

Epidural anesthesia has no direct effect on renal function. However, a sacral (S2–S4) blockade can lead to urine retention, which might necessitate catheterization of the bladder.

Effect on Thermoregulation

Shivering is commonly observed after epidural injections. The exact mechanism still remains obscure. Suggested mechanisms include vasodilatation and reduction in core body temperature and disruption of normal thermoregulatory mechanism.

The latter is a result of differential nerve blockade, which allows selective conduction of cold sensation to the thermoregulatory center or blocking descending inhibitory pathways to the spinal cord.

Indications

These can be broadly divided into three major categories: obstetric analgesia, surgical analgesia/anesthesia, and chronic pain interventions.

Obstetric Analgesia

Epidural anesthesia has been used for the treatment of the frequently excruciating pain of labor for over 40 years. A number of controversies exist about the effect it has on labor, but despite this, its use has become increasingly commonplace over the years. In addition to its usefulness in providing analgesia during labor, the epidural catheter that is sited during labor can be "topped up" with a more concentrated local anesthetic solution in order to provide anesthesia for a caesarean section or any other operative interventions. Epidural analgesia can also be extended to provide postoperative analgesia as well.

Although the validity of obtaining informed consent for a procedure when a patient is in severe distress is questionable, it is generally accepted that a number of important points are discussed with the patient prior to performing an epidural labor pain. Common as well as serious risks/side effects should be discussed. Commonly occurring side effects include PDPH, failure, hypotension, shivering, temporary muscle weakness, and urinary retention, whereas rare but serious side effects include risk of nerve damage, epidural hematoma, high/total spinal, and infection. Possible risks specific to epidurals in labor include prolongation of labor, increased risk of instrumental deliveries, and need for continuous fetal monitoring.

Adjunct to General Anesthesia

Epidural catheters or injections may be sited prior to surgery at various levels in the vertebral column in order to provide both intraoperative analgesia and postoperative analgesia.

Cervical Epidurals

Though not used generally for anything other than treatment of radicular pain in the upper limbs, it may be occasionally used to provide analgesia for surgery in the same areas. Due to the narrow epidural space and the increased depth of the space

from the skin, this technique should only be practiced by those experienced in the technique. If local anesthetics are used, partial blockade of the phrenic nerve will result in a rise in the $PaCO_2$. This respiratory inhibition may contraindicate the block in certain patient groups such as those with chronic obstructive airway disease.

Thoracic Epidurals

These are particularly useful for patients who will have large abdominal or thoracic incisions. As well as providing analgesia, the sympathetic block may reduce the myocardial oxygen demand and reduce the risk of postoperative myocardial ischemia. It may also reduce the incidence of ileus. Due to relative sparing of the lower dermatomes, early postoperative ambulation may be facilitated.

These beneficial effects must be balanced with the risks of performing the procedure particularly in the following patient groups:

1. The shocked patient: in this situation, the loss of sympathetic tone may result in unacceptably severe hypotension with fatal consequences.
2. Those likely to suffer major blood loss – the derangement in clotting may make the indwelling epidural catheter a potential risk for epidural hematoma.

Lumbar Epidurals

As mentioned previously, these are ideal for labor pain and caesarean section but can also be utilized for other surgeries on abdomen and lower limbs. The ensuing sympathetic block may be useful to improve tissue perfusion in the lower limb following vascular surgery or plastic surgery.

The use of a combined spinal–epidural (CSE) technique may be desirable in a procedure where rapid onset, complete anesthesia is required but where the procedure may last longer than the duration of the spinal component alone. In such a situation, the epidural component may be utilized to prolong the anesthesia with the need to resort to general anesthesia. When CSE is used, it is prudent to avoid higher lumbar level approaches to avoid accidental damage to the spinal cord by the lumbar puncture needle.

Yet another use of epidural is when a lumbar puncture becomes technically difficult because of poor anatomical landmarks or difficult patient positioning. In this situation, identifying the epidural space first with an epidural needle, followed by the lumbar puncture using a higher gauge (thinner) spinal needle through the epidural needle, can rescue the spinal block. This technique avoids multiple attempts and use of lower gauge (thicker) spinal needles and can potentially reduce the incidence of PDPH.

For Management of Chronic Painful Conditions

Injection of steroids into the epidural space is frequently used to treat radicular pain and low back pain. Previously it was the radicular pain was thought to be due to nerve compression. In contrast, recent work suggests that it may be due to release of inflammatory markers from the damaged intervertebral disk. Corticosteroid injection into the epidural space is thought to inhibit the inflammatory process [35]. Delivering the steroids directly to the injured area reduces the systemic effects of the steroids and increases the concentration of the drug at the site where its action is most required. These injections are commonly used to treat nonspecific radiculitis, spinal canal stenosis, and vertebral compression fracture resulting in radicular pain. Its use has also been documented in postlaminectomy syndrome, postherpetic and posttraumatic neuralgia, diabetic neuropathy, and myofascial pain.

Spinal Cord Stimulation [4]

The epidural route space is utilized for the placement of stimulator electrodes for the treatment of certain kinds of neuropathic pain. Access to the space and subsequent catheter placement can be made through a needle or via an open laminotomy. Spinal cord stimulation has also been used successfully to treat conditions such as urine and fecal incontinences.

Contraindications

The only absolute contraindication is patient refusal although there are a number of situations when the risks of the procedure will outweigh the potential benefits. The conditions that relatively contraindicate epidural anesthesia are described below.

Compromised Hemodynamic States

In patients who are shocked through either trauma or sepsis, administration of neuraxial blockade can pose serious risks. It is risky for those with any fixed cardiac output state such as aortic stenosis as well. This group of patients is unable to compensate for the loss in SVR by increasing their cardiac output. Cardiac arrest may ensue secondary to reduced coronary perfusion, and resuscitation is particularly difficult in this set of patients.

Coagulopathy and Bleeding

Patients with uncorrected clotting disorders and major blood loss are at risk of epidural hematoma formation. Epidural hematoma is a surgical emergency, and the clot will have to be evacuated without delay as the increased pressure in the epidural space can compromise the viability of the spinal cord.

Local infection or inflammation around the site of desired catheter insertion might risk the spread of the infection into the epidural space. History of allergy to the drugs used is another contraindication. However, this can be overcome by using alternate drugs.

Epidural: The Procedure

The insertion of the epidural catheter is the starting point of closer monitoring and care. The clinical area where the patient is admitted should have appropriate facilities and qualified staff to look after the patient. If these are not guaranteed, then the procedure should never be undertaken.

Equipment

The equipment used depends on whether the injection is performed alone or in combination with spinal and whether a single shot or continuous extendable technique is used. These include needles, loss of resistance syringes (LOR), catheters, filters, and connectors. Now these are available as presterilized packs.

Epidural Needles

Though there are a range of available epidural needles (Touhy, Husted, and Crawford), the most widely used one is the Touhy needle. The main difference between these needles is the angle of the blunt tip, which varies from 15 to 30°. Figure 12.7 shows the different type of needles and their tips.

A standard Touhy needle consists of a 8-cm metal shaft with marking at 1 cm intervals attached to a hub, taking the total length to 10 cm. A wing or flange is attached to the hub, which helps in stabilizing the needle while it is inserted into the epidural space. In some, this flange is fused with hub, and in others it is attached to the needle just prior to insertion. The markings on the metallic shaft help to measure the depth of needle tip from the skin.

The tip of the Touhy is rounded and pointed upward, and this is called a Huber point. The deflected bevel top makes the cutting surface approximately perpendicular to the needle shaft. This design reduces the coring of tissue and septa. The bevel at the tip of the needle reduces the risk of puncture of the dura. It also has the effect of angling the direction of the catheter, which is introduced through it upward.

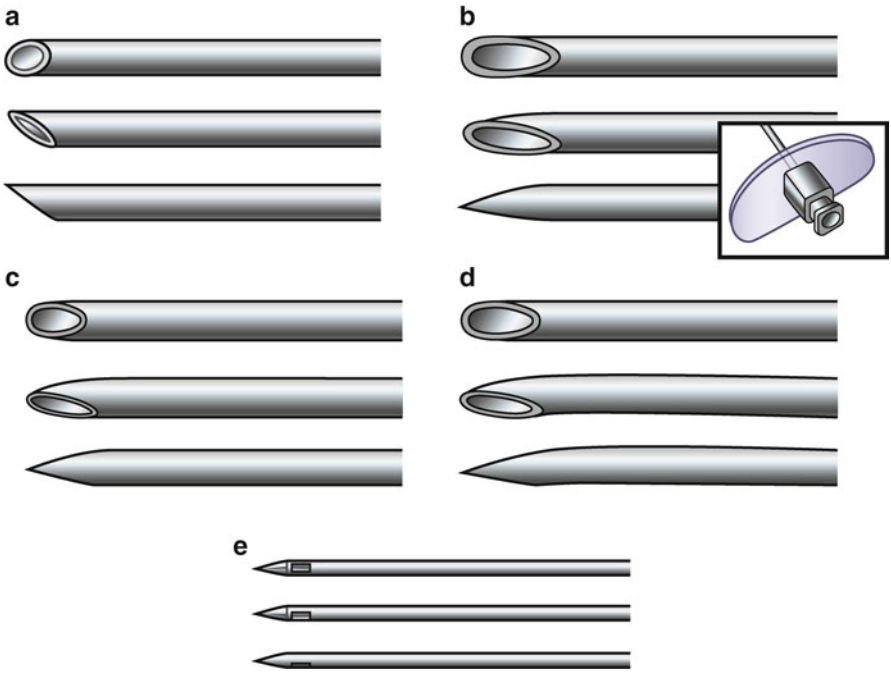

Fig. 12.7 Types of needles and their tips. Adapted from Regional Anesthesia and Analgesia, WB Saunders, Philadelphia

The hollow of the needle is occupied by a removable trochar, which does not protrude though the end of the needle. This is only removed once the needle is introduced through the skin and immediate soft tissues. Many anesthetists will feel for the "bite" of the ligamentum flavum before removing the trochar and connecting the loss of resistance syringe. The trochar is there to prevent a plug of skin or other tissue to block the end of the needle.

Another commonly seen epidural needle is those specifically designed for a needle through needle combined spinal–epidural technique. As well as spinal needle which will protrude through the tip of the Touhy needle, the Touhy needle has a slightly altered tip which will allow the smooth passage of the spinal needle though the end without the slight resistance which may occur if the technique is performed though a regular Touhy needle.

Epidural needles are available in various gauges. 16, 17, and 18 G are the most frequently used. The length of commonly used Touhy needle is 10 cm, but there is also a 15-cm needle when extra length is required. Pediatric sizes are also available. The greater caliber of epidural needles is important to allow the easy flow of saline required to detect a loss of resistance. However, it is also possible to use smaller caliber spinal needles to access the epidural space, but this is done in specific situations only. A spinal needle may be used to access the epidural space for X-ray

guided procedures, such as those performed for the management of chronic pain conditions. Spinal needles can also be used for caudal anesthesia in adults.

LOR Syringes

These syringes are Leur slip syringes with low plunger resistance allowing easy identifications of the sudden loss of resistance as the needle slips into the epidural space. These are of 10 ml capacity and made of PVC or PP and are to be either filled with saline or air prior to attaching to the epidural needle.

Epidural Catheters

These are made of either nylon or Teflon and are biology inert. These are transparent and 90 cm long. The distal tip is colored which will help to identify it during removal from the epidural space. The tip of the catheter may be closed or open; closed tip is claimed to reduce the risk of intravascular injection. There are side ports on the distal end of the catheter. The first 15 cm of the catheter has marking at 5 cm intervals. There is 1 cm marking from 5 to 15 cm of the distal end. The proximal open end is connected to the Leur lock, which in turn connects to the filter.

Epidural Filters

The pore size of these filters is 0.22 μm in size and helps to filter off viruses and bacteria. They also help to filter off foreign bodies such as glass particles (from ampoules).

Connectors

Connectors for the epidural catheters come in various designs, some are screw-in and others snap-up. The epidural kits also contain stabilizers that help to facilitate easy passage of catheters through the needle.

Aids to Identify Epidural Space

A variety of gadgets to detect the entry of tip of the needle into the epidural space, such as prefilled balloons, spring-loaded syringes, radiological imaging, or ultrasonography are available. But their routine use in practice is not substitute for technical skills that needs to be acquired with clinical practice. In exceptional

Table 12.2 Adapted from regional anaesthesia – the requisites in anesthesiology

Surgery/procedure	Level of catheter insertion
Procedure in the neck	C6–T1
Mastectomy	T6–T7
Thoracotomy	T4–T6
Upper abdominal	T6–T8
Lower abdominal	T10–T12
Lower limb	L2–L4
Labour/delivery	T10–S4
First stage	T10–L1
Second stage	S2–S4

circumstances, where the procedure is difficult due to extreme obesity or anatomical abnormality, adjuvants such as X-ray or ultrasound guidance could prove valuable.

Selection of the Site of Insertion

Two major factors dictate the level of epidural insertion. The first is the type/site of surgery or the purpose of the epidural (Table 12.2). Ideally to produce optimum analgesia, the epidural drugs should be injected into the spinal level corresponding to the dermatome, which coincides with the midpoint of surgical incision. When using a catheter, it is manipulated into the epidural space so that the tip of the catheter should roughly correspond to the midpoint of the surgical incision (not very accurate when radiological screening is not used).

The second factor is the patient factors such as the local area of insertion of the needle such as whether the spines are easily palpable or not, the interspinous space is capacious to accommodate the needle or not, the presence of infection, anatomical abnormalities of spine such as scoliosis, previous spinal surgery, etc. In obese individuals, the spines might be difficult to palpate, and in elderly, the spinous ligaments might be calcified and/or bones fused making identification and insertion difficult.

The experience and familiarity of the operator with the technique and availability of adjuvants such as ultrasound scanner or epidural space detecting devices also play a role. Sometimes proper positioning of patient might be difficult requiring modification of the site of insertion of epidural needle.

Insertion Technique

Epidurals may be theoretically inserted at any spinal level. In practice, however, it is most common to see thoracic, lumbar, and caudal epidurals. The technique is

Table 12.3 Technique of inserting an epidural

Preprocedure:
- Examine/assess the patient as for a general or spinal anaesthetic to identify any contra-indications
- Correct any correctable conditions, such as dehydration/cardiac failure
- Obtain consent for the procedure

Procedure:
- Institute ECG, blood pressure and oxygen saturation monitoring
- Prepare and draw up all drugs, including vasopressors
- Obtain large bore IV access and begin an infusion of colloid or crystalloid
- Sit or lie the patient fully flexed
- Use aseptic technique (gloves, gown, face mask, hat) and wear eye protection
- Prepare the skin with alcohol based antiseptic
- Identify the insertion level
- Drape the skin
- Infiltrate the skin with local anaesthetic (or give sedation or a general anaesthetic)
- Flush epidural catheter with saline
- Insert epidural needle through infiltrated area
- Use loss of resistance technique to identify epidural space
- Insert epidural catheter
- Secure at required depth
- Give a test dose of local anaesthetic

Post-procedure:
- Continue monitoring
- Give anaesthetic/analgesic by bolus or continuous infusion

Monitor block density and height

broadly similar for each. In all cases, an aseptic technique should be used, with scrupulous attention to prevention of infection. It should be carried out by a skilled practitioner, or under the supervision of one.

The technique of inserting an epidural may be summarized in Table 12.3.

Preprocedure

The patient should have an assessment done as though for a general anesthetic, particular attention being paid to any cardiovascular problems such as valvular lesions, restrictive cardiac disease, dehydration, or other conditions, which could prevent the patient from being able to increase his/her cardiac output when vasodilatation occurs as sympathetic blockade becomes established. Clinical examinations include systemic examination, assessment of the airway, as well as examination of spines and anatomical abnormalities.

Informed *consent* should be obtained for the procedure, including a description of the procedure of injection, management during the block, and recovery postprocedure. Advantages and disadvantages, and common and serious side effects should be discussed. The patient should be given the option to refuse, and alternatives should be discussed.

Procedure

Move the patient to an area with appropriate resuscitation and airway monitoring facilities readily available. Monitoring should be commenced as for other forms of anesthesia (ECG, SpO2, and blood pressure). Drugs to be used should be drawn up. These should include local anesthetic for skin infiltration, such as lidocaine 1 or 2%, the drugs to be injected via the epidural (as discussed later), and vasopressor agent(s) such as ephedrine, phenylephrine, or metaraminol.

A large bore (for example 16 G) IV access should be obtained and an intravenous fluid infusion commenced. While preloading patients in spinal or epidural anesthesia remains controversial, it would be prudent to ensure that it is possible to give drugs and fluids if required.

Sedation is used depending on the situation. When it is used, patient should be able to communicate and cooperate with the operator. This optimal sedation can improve patient comfort without losing cooperation.

The patient should be positioned either sitting or lying, with the spine fully flexed. Epidural can be performed in prone position as well. There are advantages and disadvantages to each position, and it will depend on patient and technical factors and operator preference which position is used.

In the lateral decubitus position, the patient may be more able to adopt the position required without the requirement for an assistant. If required, this allows a greater degree of sedation to be employed. The patient should lie with their back parallel with the edge of the bed/trolley. A pillow should be placed under the head to keep the spine level. The knees should be drawn up to the abdomen with thighs flexed, with the upper arm across the chest and the lower arm projecting 90° from the body. Ideally the patient should adopt a fetal position with the spine maximally flexed to open the spaces between the vertebrae. If necessary, the patient can be asked to increase the flexion by grasping the back of the head/neck and attempting to draw elbows and knees together. In obese patients in the lateral position, identification of the midline may be more difficult as the tissue midline is distorted by the subcutaneous adipose tissue being displaced by gravity.

In the sitting position, the patients should sit up at the edge of the bed, with their feet on a stool or other support. They should start with a straight back, with their chin on the chest arms hugging a pillow, or on a Mayo table or stand in front of them. It is important that they do not lean forward, but they should arch their back. The authors often ask patients to “arch your back like an angry cat” or to “pretend you are a slob and slouch.” Lateral rotation and flexion of the spine should be avoided, and an assistant may help to prevent this and to encourage the patients to keep their shoulders leveled [5].

The level at which the epidural is inserted should be identified. The landmarks that can be used to identify spinous processes and hence vertebral level have already been dealt with (Table 12.2). It should be borne in mind that the accuracy of the landmarks is known to be poor. Using an ultrasound scanner could be helpful in difficult situations.

The bed should be positioned at a height convenient for the operator to work at the insertion level. The midline should then be identified and if desired the location marked with an indelible skin marker. The operator should then adopt an aseptic technique, including surgical scrub, face mask, hat, and sterile gloves. There is controversy regarding the use of a face mask [6], however, wearing one has been shown to reduce the incidence of infection rates during central venous catheterization, and the author would continue to recommend their use. Eye protection should also be worn in case of inadvertent aerosolization of local anesthetic during the procedure.

The skin should be prepared with chlorhexidine gluconate or iodine in spirit, and a sterile fenestrated drape used to isolate the area where the epidural will be inserted. The applied disinfectant should remain in contact with the skin for the recommended duration (e.g., alcohol-based disinfectants should be left to dry on its own).

A skin weal should be raised using local anesthetic such as lidocaine 1 or 2%, and local infiltration performed to the supraspinous and interspinous ligaments. The needle used to infiltrate may be used as a "seeker" to identify the depth to the ligamentum flavum if the patient's body habitus is favorable and also to determine cephalad angulation required to pass between the spinous processes. It can also be used to identify the bony landmarks.

The operator should adapt a position that is convenient for them. Some operators will work from a standing position, while others work from a sitting position. Building up dexterity and adaptability from early years of training will prove valuable in later years of career.

Preparing the procedure tray before the procedure is a useful and rewarding habit to learn. The epidural catheter should be connected to the filter and be flushed with saline to ensure patency of the orifices at the distal end. The loss of resistance syringe should be tested to ensure free movement. If loss of resistance to saline is to be used to identify the epidural space, the syringe should be filled with 5–10 ml of saline.

The epidural needle is inserted with the stylet in situ, and bevel facing cephalad or caudad, perpendicular to the skin in vertical and horizontal planes. Common techniques used for holding and advancing the needle observed by the authors include holding the flanges between thumb and index finger of both hands and bracing the remaining fingers against the back to prevent too rapid advancement, and holding the needle with the thenar eminence at the hub, index and middle finger supporting the needle with thumb held parallel to the axis of the needle. The needle is then slowly advanced until increased resistance is met, representing the ligamentum flavum. The stylet should then be removed and the loss of resistance (LOR) syringe attached and the needle re-angled slightly cephalad.

For loss of resistance to saline (LORS), the nondominant hand is used to brace against the back (see Fig. 12.8), to stabilize the needle and prevent sudden rapid forward motion. The syringe is held in the dominant hand, and constant pressure is applied to the plunger of the syringe with the thumb as the needle is advanced. While the bevel of the needle is within the ligaments, there will be considerable resistance to pressure, but as the bevel exits the ligamentum flavum and enters the epidural space, there will be a sudden loss of that resistance, the contents of the

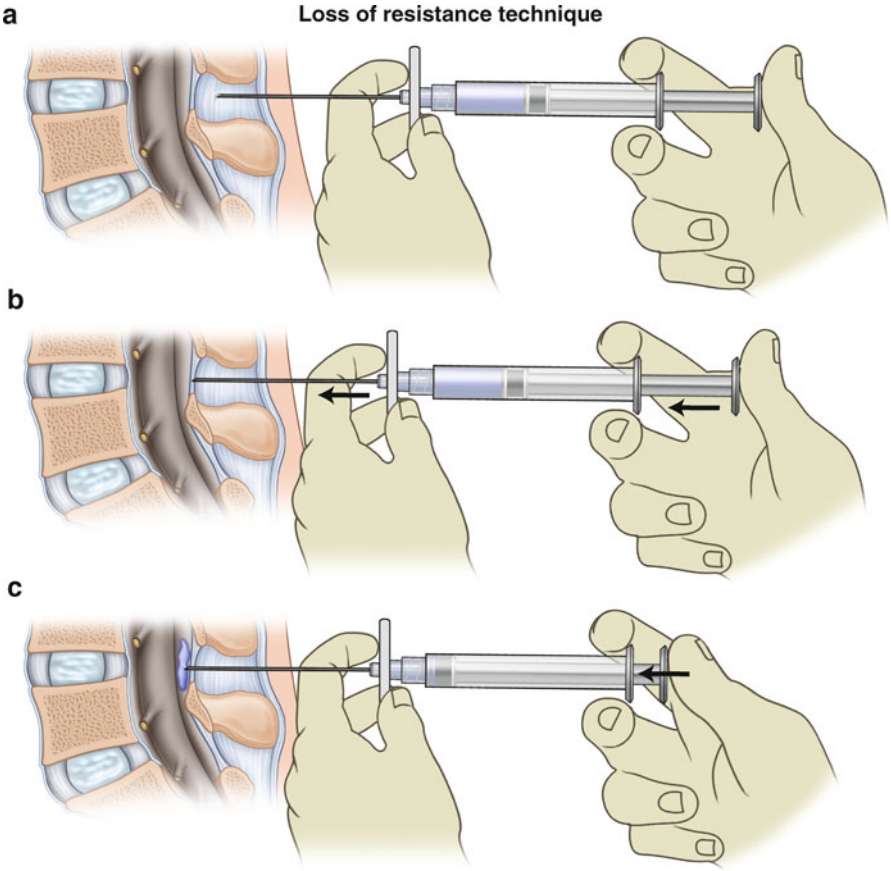

Fig. 12.8 Loss of resistance technique. Adapted from Visser L. Epidural Anesthesia

syringe will be discharged into the space, and the needle will cease in its forward motion. It is important to warn the patients that they will feel pressure in the back, but they should not feel pain. If they feel pain or discomfort, the operator should ask the patients whether they feel it to left or right, or in the midline, as it may be necessary to reevaluate the direction of insertion of the needle.

Alternatively a technique of intermittent advancement may be used, where the needle is advance 1–2 mm at a time using the same hold as described for the insertion of the needle, and the plunger of the needle is depressed intermittently to assess for loss of resistance. A small air bubble purposefully introduced into the LOR syringe can help to monitor the pressure inside the syringe. As long as the needle tip is inside the ligamentum flavum, the bubble can be observed to be compressible within the syringe by exerting pressure over the plunger.

Once LOR has occurred, the needle should be advance 1 mm further to ensure the opening is fully within the epidural space, and the syringe removed from the hub, ensuring the needle does not move. A small amount of fluid may now drain from the needle, but should stop after only a few drops. If not, then this may be CSF and dural puncture may have occurred. If fluid runs freely, then definite dural puncture has occurred, and there are several options available to the operator, which will be discussed below. If blood appears in the needle, then it should be withdrawn and either redirected or resited depending on assessment of the landmarks. In the absence of blood or CSF, the depth to which the needle has been inserted should be noted. The patient should be told to remain absolutely still to prevent displacement. If a single shot technique is to be used, injection should take place now.

If a catheter is to be inserted, the stabilizer should be placed in the needle hub, and the catheter advanced slowly into the epidural space so that 15–18 cm of catheter is within the needle and space, while the needle is stabilized. The patients should be warned that they may feel paraesthesia, burning, tingling, or electric shock, but that this should only last a moment.

Catheter advancement may be made easier if a 5–10 ml of saline is injected into the space once LOR has occurred, which may separate the tissues slightly to allow passage of the catheter. If the sensation persists after halting advancement, then it may be necessary to remove the catheter and resite the needle. Withdrawing the catheter through the needle could lead to shearing of the catheter on the bevel, and this is not recommended and should be avoided.

The needle is then withdrawn over the catheter, ensuring nondisplacement of the catheter. It is recommended that 4–5 cm of catheter be left in the epidural space [7], and the catheter should be withdrawn now to a depth of 5 cm + the depth of the space. For example, if the skin is at the 6-cm mark on the needle, the catheter should be withdrawn to the 11-cm mark. The filter and connector should be attached at the proximal end of the catheter using a sterile technique. The catheter should then be aspirated to check for CSF or blood. If blood is freely aspirated, then placement within a blood vessel must be assumed, and the catheter should be removed and a new attempt at insertion made.

If CSF is aspirated at any point during the procedure (either via the needle or the catheter), then the option of placing/leaving an intrathecal catheter may be considered. This may then be used to provide SPINAL anesthesia, with the relevant cautions applied. This technique is beyond the scope of this chapter. Alternatively, withdraw and resite. The patient should be warned about the possibility of PDPH, and a note of dural puncture should be made in the medical/anesthetic records.

Once the catheter is at the required depth, it should be secured. This may be done using proprietary securing devices or a clear transparent dressing. If the latter is used, one or two loose loops of the catheter should be made on the skin so that tension on the catheter will unravel the loops rather than displacing it. A further alternative includes tunnelling the catheter under the skin for a short distance before making the loops. The catheter is then brought over the patient's shoulder and secured in situ with a cloth tape such as Mepore® or Hypafix®.

A test dose of 3 ml of local anesthetic (with or without 1:200,000 epinephrine equivalent to 15 μm) may now be given and the patient asked to return to the supine position. Monitoring for signs of intravascular injection or intrathecal injection should occur (20% increase in heart rate, rise in blood pressure, spinal anesthesia). It should be borne in mind that sedation may reduce the reliability of the lidocaine only test in an awake patient [8]. If after 5–10 min no signs of either have been detected, then incremental injection of the desired analgesic/anesthetic drugs may occur. Continued observation for signs of systemic toxicity and catheter displacement into the dural space should be continued. Epidural block may take anything up to 20 min to become established.

Loss of resistance to air is another technique for identifying the epidural space. As above, the LOR syringe is attached to the needle hub when the needle is in the interspinous ligament of ligamentum flavum, but with 5–10 ml of air inside. The wings of the needle are gripped between the thumb and forefinger of both hands with the dorsa of the hand resting against the patients back, and the needle advanced 2 mm at a time. The plunger is gently pressed, and if there is resistance (colloquially termed "bounce"), the needle is very carefully advanced another 2 mm. As the needle enters the epidural space, a sudden give way or "click" may be felt." At this point air can be freely injected into the epidural space. The syringe is removed and the catheter threaded as above. Provided great care is taken in advancing the needle it should not pierce the dura. As this technique requires intermittent removal of one hand and testing for LOR, it is relatively slower but probably safer as there is less chance for the needle to overshoot and produce an accidental dural puncture.

A further possible technique is the "hanging drop," where a drop of saline is placed at the end of the needle once the stylet has been withdrawn. As the needle is advanced into the epidural space, the negative pressure that exists within the space withdraws the drop into the needle (due to the denting of the dural by the needle). This technique used to be popular for thoracic epidural injections.

Paramedian approach is an alternative approach to the epidural space, especially in the thoracic level and when the ligaments are calcified. The needle is inserted 1–2 cm lateral to the spinous process of the more cephalad vertebra. The needle is then advanced perpendicular to the skin until contact is made with the lamina or the pedicle of the vertebra. The needle should then be redirected cephalad 15–30° and medially 15–30° and the needle "walked off" the bone. A loss of resistance technique is then used to detect the epidural space.

Thoracic Epidural

The thoracic vertebral spinous processes are much more steeply angled and project further. The dura is more closely aligned to the ligamentum flavum, and the spinal cord may lie closer to the dura. The paramedian approach may be preferable at this level. The positioning of the patient and the technique of advancement remain substantially the same as for lumbar epidural, but with the proviso that the epidural needle should only be advanced 1 mm at most after LOR.

Table 12.4 The Bromage Scale (1965)

Degree of block	Bromage criteria	Score (%)
No block	Full flexion of knees and feet	0
Partial block	Just able to flex knees and full flexion of feet	33
Almost complete	Unable to flex knees, some foot flexion	66
Complete	Unable to move legs or feet	100

Caudal Approach

Patient is positioned either in the lateral decubitus or prone position. Sacral hiatus is then identified by palpation. The surface marking of sacral hiatus is that it lies at the apex of an equilateral triangle base of which is formed by the line joining the two postsuperior iliac spines. The hiatus lies in its apex with the sacral cornua on either side. A 22 (or 23)-G needle is then inserted at 45° angle with its bevel facing the operator into the ligament. Once the ligament is negotiated which is felt as a "pop," the needle is pushed down into a more acute angle and advanced further (2 cm in adults and about 1 cm in children) into the caudal canal (Fig 12.5). Care should be taken not to advance the needle too much into the caudal space as this will increase the risk of dural puncture. A canula over the needle can also be used; then the canula can be left in epidural space after withdrawing the needle. The anesthetic solution is injected after careful aspiration for CSF or blood.

Post-procedure

The patient should continue to be monitored after the anesthetic has been given for signs of local anesthetic toxicity. These include light-headedness, tinnitus, circumoral and tongue numbness and paraesthesiae, visual disturbances, muscular twitching, convulsions, unconsciousness, coma, respiratory arrest, and cardiovascular collapse.

Block height and density should be assessed at regular intervals. This can be done by using the Bromage scale and by assessing for loss of sensation to cold, touch, and pinprick. The dermatome level at which sensation is lost should be recorded (Table 12.4) [9, 10].

Troubleshooting

Unable to Identify Anatomy

In pregnant or obese patients, it may be difficult to identify the midline, especially in the lateral decubitus position. These patients should therefore be asked to adopt the sitting position where possible. It may be helpful, though not necessarily reliable, to ask the patients whether they feel that the operator's hand/needle is in the midline. Alternatively, it may be possible to use ultrasound to identify the midline and interspinous space.

Repeated Contact with Bone

"Position, position, position" is the most common reason for repeated contact. The patient should be asked to flex more, or position should be changed from lateral decubitus to sitting or vice versa. Other techniques are to reinsert the needle slightly away from the midline, withdrawing the needle to the subcutaneous tissue level and reposition the needle at a steeper angle, or inserting the needle close to lower border of the upper spine. In lateral decubitus position, there is a tendency for the soft tissues to sag under gravity leading to the midline of the back to move away from the spinous processes, and this can mislead the operator. This happens more frequently in obese individuals.

Unable to Thread the Catheter

The stabilizer that comes in most commercial packs should be used to aid in inserting the catheter, as it will prevent kinking in the relatively large hub of the needle. Slight rotation of the needle about the longitudinal axis may facilitate insertion. Sometimes, even after obtaining LOR to saline or air, the tip of the needle will be only half way though the ligamentum flavum. Then it will be difficult for the catheter tip to pass into the epidural space. Advancing the needle slightly in could solve the problem.

Fluid or Blood Returns Via the Needle or Catheter

Saline may return via the needle or catheter, but as described above, it should stop after a few seconds. If it does not, then dural puncture should be assumed. If flow does stop, then incremental doses of anesthetic should be given and the patient observed for signs of intrathecal block. This applies whether or not a catheter is used. Testing the fluid for glucose using an indicator strip of glucometer can distinguish between CSF and the saline.

If blood returns via the catheter, then it is likely in an epidural vein. If on withdrawing the catheter so that 2–3 cm remains within the space, blood flow stops, and blood cannot be aspirated via the catheter, then the catheter may be used cautiously, but with the provisos that all doses should be preceded by aspiration to check for blood, they should be given incrementally, and the patient should be monitored very closely for signs of toxicity. The catheter should be flushed with saline before a test dose is given.

Pain on Insertion

A brief sensation of electrical shock or paraesthesia on insertion of a catheter is common. If it persists, then the catheter is likely up against a nerve root and should be withdrawn a few millimeters until the sensation stops. If sufficient catheter remains within the space, then it may still be used. Otherwise it should be resited.

Unilateral Block

The precise cause for this could be difficult to infer. The cause could be that the tip of the catheter could have moved out of the epidural space through the intervertebral foramen (this happens when too much a length of the catheter is introduced into the epidural space). Connective tissue septa can theoretically prevent uniform distribution of the local anesthetic solution. This problem is managed by pulling out the catheter so that about 3–4 cm of it remains inside the epidural space and then giving another top-up or by turning the patient on the unblocked side before the top-up and keeping in this position for about 15 min. If this fails then the epidural will have to be resited.

Pharmacology of Epidural Blockade

Site of Action of Epidurally Administered Drugs

The precise nature of the fate of epidurally administered drugs is not fully understood. However, Hogan [11, 12] has demonstrated that the spread of solutions injected into the epidural space results to form a coat around the cylindrical dural sac while some of it pass through the foramina. There are four potential possibilities for these drugs to exert their observed effects. Once injected they can pass along the intervertebral foramina into the paravertebral space and exert its effects on the nerve roots and plexuses. Or they diffuse through the dura into the subarachnoid space. They can penetrate the dural cuffs of the spinal nerves and interfere with nerve conduction. Other possible pathway is by axonal transmission. Epidural veins also contribute to the systemic absorption of the drugs administered.

Drugs and Doses

Local Anesthetics

At one time or another almost all local anesthetic agents have been used for providing epidural anesthesia or analgesia, either alone or in combination with a variety of other drugs ranging from epinephrine to ketamine to opioids. All of the preparations used for neuraxial blockade should be preservative free so that the risk neurotoxicity is minimized.

Choice of drugs will depend on the procedure being performed and the indication for which the epidural has been inserted. For a short procedure, for example, a short acting local anesthetic such as prilocaine, chloroprocaine, or lidocaine would seem to be a sensible choice, whereas for a prolonged procedure, a drug such as

bupivacaine or ropivacaine would be better. When we discuss about duration of local anesthetic medication, it can be described in terms of "two-dermatome regression" and "complete resolution." The former is the time taken for the block to recede by two dermatomes from its maximum extent, while the latter is the time taken for the sensory block to wear off completely. This is influenced by dural surface area, volume of fat in the epidural space. Velocity of blood flow in the epidural space might also influence the duration of epidural anesthesia [13]. Table 12.5 shows the recommended dose and duration of action of commonly used local anesthetics [14].

Chloroprocaine

The onset is rapid within 6–12 min and has a duration of approximately 40–50 min. It can be used as an infusion so as to prolong the effect. It is available in 2 and 3% concentrations. The epidural dose is 15–25 ml [15]. Higher doses are associated with backache and reduced efficacy of adjuvants such as morphine and clonidine.

Lidocaine

Lidocaine is probably the most widely used local anesthetic. It is available in a range of concentrations including 1, 1.5, and 2%. It has a rapid onset of action, within 10–20 min. The dose is 10–20 ml of 1%, 10–15 ml of 1.5%, or 10–20 ml of 2% depending on site and desired block level. Without epinephrine it will give a duration of action from 1 to 2 h [16]. Tachyphylaxis limits its use for long-term use. It is a popular topping-up agent for caesarian sections either alone or in combination with epinephrine and sodium bicarbonate; the latter ensures rapid onset by altering the pH so that more of the unionized drug is available to penetrate the neural tissues. But this effect is not found to be consistent.

Mepivacaine

Mepivacaine comes as a 1, 1.5, or 2% concentration. It has an onset of action ranging from 3 to 20 min, with a duration of action of up to 2–2.5 h. Dose ranges are 15–30 ml 1%, 10–25 ml 1.5%, and 10–20 ml 2% [17–19].

Ropivacaine

This is single isomer with a moderately rapid onset of action. It is available as 0.2, 0.5, and 1% concentrations. For analgesia the 0.2% solution may be used, 10–20 ml at 30–60 min intervals, or at infusion rates of 6–14 ml/h. For surgery, 15–30 ml of 0.5% or 15–20 ml of 0.10% ropivacaine may be used. Onset of action is within 5–13 min and duration of action 3–5 h [20]. It does not have any significant motor sparing action as claimed.

Table 12.5 Recommended dose and duration of action of commonly used local anesthetics

Drug	Presentation	Onset of action (min)	Usual dose	Duration of action
Chloroprocaine	As a solution in 2% and 3% concentration	6–12	15–25 ml	40–50 min
Lidocaine	As solution of lidocaine (usually hydrochloride) with or without epinephrine, in concentrations of 1, 1.5 and 2%	10–20	10–20 ml (1%) 10–15 ml (1.5%) 10–20 ml (2%) Depending on site and desired level of block	Without epinephrine from 1 to 2 h With epinephrine may be considerably longer
Mepivacaine	As a solution in 1, 1.5 or 2% concentration.	3–20	15–30 ml (1%) 10–25 ml (1.5%) 10–20 ml (2%)	2–2.5 h
Ropivacaine	As a solution in 0.2, 0.5, and 1% concentration.	5–13	10–20 ml (0.2%) at 30–60 min intervals or infused at 4–14 ml/h 15–30 ml (0.5%) for surgery 15–20 ml (0.1%)	3–5 h
Bupivacaine (and Levobupivacaine, which is equipotent but with fewer cardiotoxic effects)	As a solution in 0.25, 0.5 or 0.75% in 5–10 ml vials or ampoules As a solution of 0.1% in infusion bags or syringes of 50–500 ml, often with fentanyl 2 μg/ml or 4 μg/ml for epidural infusion	5–20	10–20 ml of either concentration at 1–2 h intervals, or infustions of 5–15 ml/h of 0.1% solutions	Up to 2–2.5 h

Bupivacaine

Bupivacaine is available as 0.25 or 0.5 or 0.75% solutions in 5–10 ml vials or ampoules. It is also widely available as a 0.1% solution in large volume bags (100–250 ml or more), often with fentanyl 2 or 4 μg/ml for epidural infusion. Bolus doses are usually 10–20 ml of either concentration with repeat dosing at 2-h intervals, depending on desired block, and infusion rates from 5 to 15 ml/h for the 0.1% solutions [21, 22]. Levobupivacaine, the levo form, has fewer cardiotoxic side effects without loss of the anesthetic potency [23]. This levorotatory isomer of bupivacaine drug is not yet available in the USA.

Adjuvant Drugs

These are used to augment or supplement the effects of local anesthetics. Though various drugs have been used for this, the following are commonly used in clinical practice:

Opioids

Opioids are probably the most commonly used adjuvant drugs. Morphine, fentanyl, sufentanil, hydromorphone, and diamorphine have all been used as epidural adjuvants [24–26]. They prolong the duration of analgesia without any effect on the motor system. This effect is mediated by the opioid receptors in spinal cord. The recommended infusion regimen is shown in Tables 12.6 and 12.7.

Epinephrine

Epinephrine can be used as an additive to increase the depth and duration of block. It causes local vasoconstriction thereby decreasing the clearance of local anesthetic from the tissues. This effect means that often the concentration and dose of drug used can be reduced. The usual concentration used is 1:200,000 (5 μm/ml) [27]. Epinephrine prolongs duration of both sensory and motor blockade. This is significant in the case of short and intermediate duration anesthetic agents. In contrast this effect is not seen in the case of longer acting agents. Epinephrine can also exert its α2 adrenergic effect on the adrenergic receptors present in the spinal cord and reduce the transmission of nociceptive impulses. In addition to the neuraxial effects, epinephrine produces reduction in systemic vascular resistance as a result of its systemic absorption from the epidural space. This is the result of β2 stimulation of arterial adrenergic receptors. The resulting reduction in the mean arterial pressure can lead to the observed reflex tachycardia.

Table 12.6 Epidural opioids recommended dose as continuous infusion

Drug	Solution (mg/ml)/(%)	Bolus dose (mg)	Basal infusion (per h)	Breakthrough doses (mg)	Increments in breakthrough (mg)
Morphine	0.1/0.01	4–6	0.5–0.8 mg	0.2–0.3 every 10–15 min	0.1
Hydromorphone	0.05/0.005	0.8–1.5	0.15–0.3 mg	0.15–0.3 every 10–15 min	0.05
Fentanyl	0.010/0.001	0.0005–0.0015	0.0005–0.001 mg/kg	0.010–015 every 10–15 min	0.010
Sufentanyl	0.001/0.0001	0.0003–0.0007	0.0001–0.0002 mg/kg	0.005–0.007 every 10–15 min	0.005
Alfentanyl	0.25/0.125	0.01–015	0.10–0.018 mg/kg	0.25 every 10 min	0.25

Table 12.7 Epidural opoid-bupivaine combination administered as infusion

Drug combinations	Solution (%)	Basal infusion (ml/h)	Breakthrough doses, ml (intervel, min)	Increments in breakthrough (ml of the solution)
Morphine	0.01	6–8	1–2 (10–15)	1
Bupivacaine	0.05–0.1			
Hydromorphone	0.0025–0.005	6–8	1–3 (10–15)	1
Bupivacaine	0.05–0.1			
Fentanyl	0.001	0.1–0.15/kg	1–1.5 (10–15)	1
Bupivacaine	0.05–0.1			
Sufentanyl	0.0001	0.1–0.2/kg	1–1.5 (10–15)	1
bupivacaine	0.05–0.1			

Adapted from de Leon-Casasola OA, Lema MJ. Postoperative epidural opioid analgesia: what are the choices. Anesth Analg. 1996;83:867–875

Table 12.8 Epidural adjuvants

Drug	Dose	Effect
Epinephrine	1:200,000 (5 μg/ml)	Increases local vasoconstriction thereby decreasing the clearance of local anaesthetic from the tissues. This effect means that often the concentration and dose of drug used can be reduced
Clonidine	300–600 μg as a sole agent 75–150 μg in combination with local anaesthetic (intra- or post-operatively)	Selective alpha-2 adrenergic agonist Side effects include hypotension, bradycardia, and sedation, and operators should bear this in mind when choosing to use it It has been shown to reduce opioid use by 50% and prolong the analgesic effects of local anaesthetics by 100%
Ketamine	0.5–1 mg/h (with morphine) up to 0.25 mg/kg/h (with sufentanil). Single boluses up to 1 mg/kg have also been used In combination with local anaesthesia for caudals 0.5 mg/kg has been used	Potential side effects include dizziness, diplopia, dysphoria, dreams, hallucinations disorientation, strange sensations, light headedness, sleep difficulties, and confusion, though these are less common when ketamine is used epidurally

Clonidine

Clonidine is a selective alpha-2 adrenergic agonist, which has been used extensively in epidural and spinal anesthesia for many years. Its side effects include hypotension, bradycardia, and sedation, and operators should bear this in mind when choosing to use it. It has been used as a sole agent in a dose of 300-600 μg, and in conjunction with local anesthetics both intra- and postoperatively at doses of 75-150 μg. It has been shown to reduce opioid use by 50% and prolong the analgesic effects of local anesthetics by 100% [28].

Ketamine

Ketamine has been used in conjunction with local anesthetics and opioids for intra- and postoperative analgesia in doses ranging from 0.5–1 mg/h (with morphine) up to 0.25 mg/kg/h (with sufentanil). Single boluses up to 1 mg/kg have also been used. Potential side effects include dizziness, diplopia, dysphoria, dreams, hallucinations and disorientation, strange sensations, light-headedness, sleep difficulties, and confusion, though these are less common when ketamine is used epidurally (Table 12.8) [29].

Factors Affecting Spread of Drugs in the Epidural Space [30]

Patient Factors

These have minor effects on the epidural spread of local anesthetics.

Age

This is a minor factor affecting spread. With advancing age, intervertebral foraminal narrowing leads to higher spread of anesthetic. In younger individuals, part of the injected dose moves out through the foraminae.

Weight

Obesity increases the spread of injected drugs, and a possible explanation is that the situation is often similar to that seen in pregnancy as the raised intra-abdominal pressure decreases the volume of epidural space secondary to venous engorgement. So these patients require lesser doses (in comparison to their weight) for a given level of blockade. However, this is not a consistently observed effect as there is a lot of individual variability.

Height

Taller patients require higher dose than shorter ones, and then again this effect is not observed always.

Drug Factors

Total Dose

This is a *major* factor determining the spread of epidural injections. Higher doses will produce higher level of blockade. Yet again, it is impossible to accurately predict the level of blockade for a given dose.

Volume

Higher volume will produce greater spread of anesthetic. But this can lead to reduction in the concentration of the drug if the total dose is kept constant. This can lead to reduced intensity of the nerve conduction block. When the concentration is maintained, increasing the dose will increase the dermatomal spread though in a nonlinear fashion.

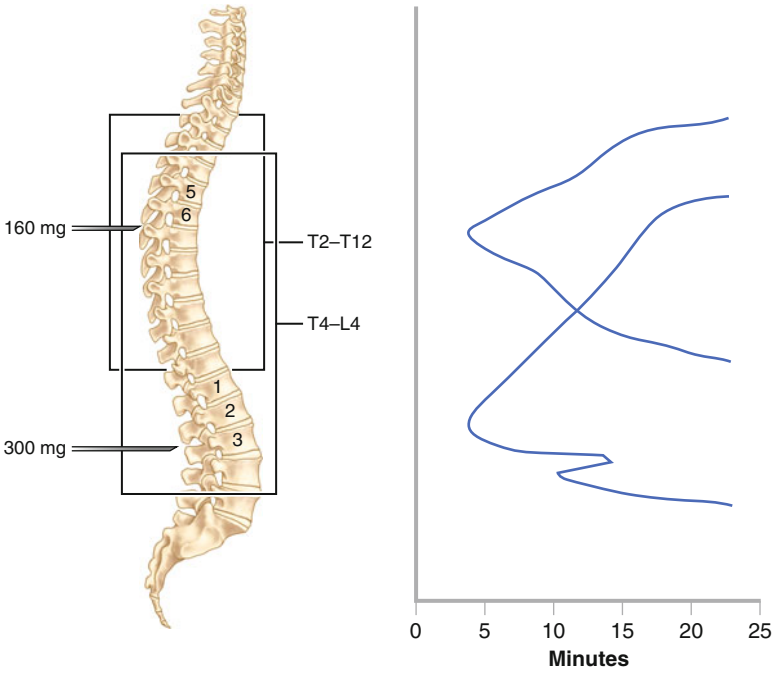

Fig. 12.9 Site of injection and spread of epidural drugs: Adapted from Mulroy MF. Regional anesthesia – an illustrated procedural guide. 3rd ed. Philadelphia, PA: Lippincott

Technical Factors

Position of Patient

The position of the patient during injection has a minimal effect.

Site of Injection

Site of injection is a *major* determinant of spread of epidural drugs. Thoracic injections require lower dose as the epidural space is narrow in this location, while caudal injections require higher volumes as the space is more. The effect is immediate and maximum at the site of injection, and the effect spreads cephalad and caudad over time (Fig. 12.9).

Type of Pharmacological Agent

Local anesthetics produce sensory, motor, and sympathetic blockade, while opiates produce analgesia without any of the above effects.

Complications of Epidural Neuraxial Blockade

Complications following epidural can be immediate or delayed (Table 12.9).

Immediate Complications

Trauma

Multiple attempts due to inexperienced operator, poor positioning, and lack of patient cooperation can lead to injury to ligaments and soft tissues. Trauma to spine and disk, though rare, is possible. This can be minimized with careful planning, proper positioning, developing rapport with patient, and a gentle careful technique. Identification of epidural space can be facilitated by using aids such as ultrasound.

Neurological Damage

Neurological damage, though rare, can occur during the procedure or as later sequelae. This may be traumatic or can be the result of inadvertent injection of neurotoxic medications. Performing epidural with the patient awake can minimize this complication. Though the damage is produced during the procedure, clinical presentation with persistence of sensory loss or abnormal sensation or as neuropathic pain might be delayed until the effect of epidural injection wears off. Confirmation of diagnosis is by detailed neurological examination and radiological imaging. Though annoying both for the patient and the physician, complete or near complete recovery often follows. In some instances, this could be permanent.

Bleeding

Bleeding can potentially occur from different tissue layers all along the path of the needle. If bleeding occurs through the needle, then it may have to be withdrawn, and the procedure should be attempted at a different level. Bleeding from catheter can be managed by withdrawing the catheter by a few millimeters and flushing it with saline, and this process is repeated until the catheter becomes clear (beware of catheter being completely pulled out of epidural space, or too little of it being inside. The latter runs the risk of delayed dislodgement)or by reinsertion at a different level. Initial injections of local anesthetic solutions then need to be administered through the catheter slowly and with frequent aspirations as a precaution.

Dural Tap

It presents usually as a dramatic gush of CSF through the Touhy needle or less frequently after the passage of epidural catheter. Sometimes this could remain concealed and later present as an inadvertent high spinal or a PDPH. There is a high incidence of PDPH (about 75%) following this complication. Management options include performing epidural in another level or converting it into a continuous spinal by inserting an intrathecal catheter (needs to be very careful when administering local anesthetic through this catheter as there is risk of accidental overdosing of local anesthetic).

Sympathetic Blockade

This is truly an excessive physiological effect of the extended epidural blockade.

Hypotension

Hypotension is secondary to vasodilatation and subsequent reduction in venous return. It usually has a slow in onset in comparison with spinal and is easily treated with fluid loading and/or vasopressors (ephedrine, metaraminol, or phenylephrine). This is exaggerated in presence of intravascular volume depletion secondary to dehydration or hemorrhage. Epidural epinephrine administered as an additive with local anesthetic can exaggerate this effect by its beta effect.

Bradycardia

Bradycardia is secondary to inhibition of sympathetic cardioaccelerator fibers (T1–T5) and reduced venous return. The treatment, in presence of hemodynamic compromise, is with vagolytic agents (atropine or glycopyrrolate). Ephedrine by virtue of its effect both on heart rate and blood pressure is yet another option.

Bronchospasm

Bronchospasm, though rare, is a possibility secondary to loss of bronchodilatory effect of the sympathetic system and the unopposed parasympathetic activity.

Hypotension can lead to an increase in pulmonary shunt and lead to *hypoxemia*, which is treated with supplementation of oxygen and by measures to increase blood pressure.

Local Anesthetic Toxicity

Accidental intravascular injection of local anesthetic drugs can lead to a range of side effects from mild symptoms like dizziness and circumoral tingling sensation to life-threatening circulatory collapse and convulsion. Management of this is in the line of airway, breathing, circulation (ABC), and organ support. Intralipid infusion [31] is claimed to be effective in reversing the effects of LA toxicity.

Total Spinal Anesthesia

This is the result of a large volume of local anesthetic injected accidentally into the subarachnoid space. Clinical manifestations include profound hypotension, respiratory insufficiency, and loss of consciousness. Management is resuscitative and in line with ABC and other supportive measures. GA may have to be induced and the patient need to be kept asleep until the local anesthetic wears out. Prevention is the best strategy in the management of this condition by giving a test dose before the full dose followed by testing motor functions of lower limbs. As this complication potentially can occur until the epidural is stopped and catheter is removed, it is also prudent to administer the local anesthetic after careful aspiration of the catheter for CSF every time a top-up is given. Often the local anesthetic solutions in the epidural space that are returning through the catheter can be confused with CSF. Testing for the presence of glucose in the CSF is helpful in this situation.

Subdural Injection

Subdural space lies between the dural and arachnoid layers and is a potential space that, unlike epidural space, extends into the cranial cavity. Relatively small volumes of local anesthetic solutions entering the subdural space following injection through a misplaced catheter can produce high levels of blockade. The incidence of these complications is very low [32] (less than 1/1,000 epidurals) and associated with slow onset of high block with a predominant sensory blockade and motor sparing. This may be associated with Horner's syndrome. Management is supportive and allows the block to wear off spontaneously.

Inadequate Block

It presents as unilateral or patchy block due to uneven distribution of the local anesthetic solution. Various reasons have been attributed as causative factors. These include epidural catheter tip positioned outside the space (when the epidural catheter is threaded too much into the epidural space, it is possible that it can exit through the intervertebral foramen), mechanical obstruction to spread of anesthetic solution (for sacral sparing), bands or septae separating the individual nerve roots, or the air

bubble introduced into the epidural space during the course of identification of the epidural space. This is managed by pulling out the catheter, increasing the concentration of local anesthetic solution, adding adjuvants such as a narcotic, or resiting the epidural.

Other immediate minor side effects are nausea, vomiting (treat hypotension, antiemetics), shivering (may respond to small doses of pethidine or ketamine), and Itching (commonly due to opioids treated with nalbuphine, naloxone, or antihistaminics).

Delayed Complications

Delayed complications include:

Post-dural-Puncture Headache

Onset is usually 24–48 h following the dural puncture and can last up to 10 days. This results from leakage of CSF and the consequent reduction in the CSF pressure. It presents as frontal or occipital headache with nuchal extension, which is characteristically worse on standing. Associated symptoms include nausea, tinnitus, hearing loss, photophobia, and diplopia.

Initial treatment is conservative with simple analgesics, rehydration (oral or i.v.), abdominal binding, and bed rest. Other medications that are found to be of benefit include caffeine, sumatriptan, and ACTH. If this fails, then an epidural blood patch is performed using the patient's own blood. During this one person draws patient's own blood aseptically, while another person performs the epidural injections of this freshly drawn blood. The dural rent is sealed off by the subsequent formation of a blood clot. Though, often this produces a dramatic relief of PDPH, this may have to be repeated if the headache returns.

Back Pain

Back pain is secondary to local trauma and is common and usually resolves with simple analgesics. Those due to soft tissue hematoma especially in the ligaments take a longer time (6–8 weeks) to resolve. Chronic back pain per se due to tissue (bone, disk, or ligament) injury during epidural injection is possible but very rare.

Epidural Hematoma

Incidence is 1/150,000 but a serious delayed complication of epidural neuraxial blockade. Preexisting coagulopathy is a risk factor. Pain may be the first presenting

symptom and followed by loss of neurological functions, which is a result of compression of the cord by the expanding hematoma inside the rigid spinal canal. Undue prolongation of residual motor block should raise the suspicion of this condition. Radiological imaging should be undertaken immediately to confirm or rule out hematoma. Management is early (within 6 h) surgical decompression as delay in intervention can cause permanent neurological damage.

Epidural Abscess

Epidural abscess is another rare but serious complication of epidural injection. Risk factors include systemic sepsis and history of intravenous drug abuse. Initially it presents with nonspecific symptoms such as fever and backache several days following epidural injection. There may be local tenderness over the spine and later progressing on to sensory loss and paraplegia. Laboratory findings may include elevated white cell count and raised ESR and CRP. Diagnosis is confirmed by MRI. Treatment is urgent surgical decompression.

Adhesive Arachnoiditis and Cauda Equina Syndrome

This complication has been reported [33] following epidural injections and is the end result of neurotoxicity of injectates (e.g., lignocaine, glass particles from ampoules). Clinical features are bowel and bladder disturbances, pain, paresthesia, and patchy sensory abnormalities affecting the perineal regions and lower limb. Chemical arachnoiditis due to glass is prevented by not using medications from glass ampoule or using a glass filter for drawing up the injectate. Treatment is conservative and recovery may be incomplete.

Retained Catheter

Excessive lengths of epidural catheter introduced into the epidural space can lead to knotting, and removal can be difficult or impossible. Leaving excessive length of catheter inside the epidural space is probably not a good practice. Terminal portion of catheter can break off and get lodged in the epidural space following an attempt to withdraw it through the needle. These catheters can be left in the epidural space without undue fear of tissue reaction as they are implantation tested. However, the patient should be warned of its presence in order to avoid future confusion with regard to the presence of a foreign body.

Table 12.9 Complications of epidural anesthesia

Immediate	Delayed
Trauma	Residual neurological damage
Dural puncture	Post dural puncture headache
Secondary effects of sympathetic blockade	Back pain
Inadvertent intravascular injection of drugs	Epidural hematoma
Total spinal	Epidural abbess
Sudural injection	Arachnoiditis and cauda equine syndrome
Incomplete block	Retained catheter
Shivering	
Nausea and vomiting	
Itching	

Clinical Pearls

Surface Anatomy

- The depth of the epidural space when inserting a needle is most shallow in the lumbar region deepest in the cervical region.
- As vertebral flexion is permitted in the cervical and lumbar regions, it is beneficial to make the patient maximally flex when attempting to access the space in these regions.
- The thoracic vertebrae permit mainly rotation only, so flexion makes little difference here.

Epidural Block

Successful administration of epidural block begins with giving detailed attention to patient selection and thoughtful planning of its execution. It starts with history taking including the anatomical location of the planned procedure, examination of the patient paying specific attention to the airway and spine. If indicated, full blood count and a clotting screen should also be requested to rule out contraindications for the procedure.

In History

Specifically Explore

- Symptoms of autonomic neuropathy.
- Hypovolemia.

Table 12.10 Epidural analgesia and drugs affecting hemostasis: current recommendations

Drug	When to stop
Aspirin	Continue
NSAID	Continue
Heparin	Stop before 4 h
LMWH	Stop before 12 h
Warfarin	Stop before 5 days (INR to be below 1.5)
Clopidogrel	Stop before 1 week (range 5–10 days)
Teicoplidine	Stop before 2 weeks
GIIa/III	Stop before 4 weeks

- Sepsis local or systemic.
- Coagulopathy – INR below 1.5 is acceptable.
- Medications affecting hemostasis may have to be stopped before performing neuraxial block. The current consensus [34] on these and neuraxial block are summarized in Table 12.10.

Examination

Along with systemic examination, proper assessment of the airway is extremely important in all regional techniques because of the potential for failure or complications. Loosing airway in a critical situation spells disaster. Spine should be evaluated to assess any abnormality and evidence of local infections, tattoos, etc.

During Procedure

- Be prepared to administer GA which means checking the equipment and preparing the necessary medications.
- Maintaining verbal contact with patient at all times as this serves to calm the patient and can give early warning of immediate/imminent complications. Ability to speak is a good sign of adequate respiration and cerebral perfusion.
- Proper positioning (in sitting or lateral) depending on the patient and the operators preferences.
- If difficulty is encountered in identifying the spinous processes, palpating the thoracic spines (which felt with relative ease even in obese individuals than the lumbar) downward helps to identify the lumbar interspaces in obese individuals.
- When performing epidural injections with the patient in a lateral position, the spine has a tendency to yield to gravity and can sag downward, leading to difficulty in keeping the needle in the midline. The needle used to infiltrate the local anesthetic can be used as a seeker.
- Accidental epidural venous puncture occurs; avoid administering heparin for 2 h and LMWH for 24 h.

- Accidental subdural injection can occur especially with multi-lumen epidural catheters. During insertion the tip can penetrate the dura into the subdural space, while the rest of the catheter remains in the epidural space. Rapid injections can force the LA into the subdural space, while slow injections lead to epidural spread. Subdural injections can manifest as patchy epidural block, high or total spinal. Rotating the Touhy needle inside the epidural space should be avoided (which can help in threading the epidural catheter) as this can produce dural tear with subsequent passage of catheter into the subdural space.
- Knowledge of anatomy is essential, and ability to appreciate the position of the vertebral column relative to the overlying soft tissue is desirable in order to perform epidural block with ease and speed.
- When using catheters, it is prudent to test their patency by flushing with normal saline. All connections need to be tested as well.
- From the point of reducing neurological complications, performing the epidural block with the patient awake is the safe option as the patient will be able to guide the operator in avoiding nerve damage.
- Full aseptic technique should be followed at all times including when topping up.
- Cather migration is possible, and aspirating the catheter for blood and CSF is mandatory before each top-up. Failure to do so can end in a catastrophe.

Multiple-Choice Questions

1. Regarding epidural analgesia the following statement is correct:
   (a) Reduces Itpostoperative mortality
   (b) There is a reduction in venous thrombosis
   (c) It increases catecholamine release
   (d) Can lead to metabolic dysfunction

2. Epidural space extends from:
   (a) Cranial cavity to sacral foramina
   (b) Foramen magnum to lower border of L2
   (c) Foramen magnum to sacrococcygeal ligament
   (d) Foramen magnum filum terminale

3. Epidural space is thinnest in:
   (a) Cervical region
   (b) Thoracic region
   (c) Lumbar region
   (d) Caudal region

4. Regarding surface markings, the following statement is NOT true:
   (a) Vertebra prominence corresponds to C8 vertebra
   (b) Inferior angle of the scapula corresponds to T7 vertebra
   (c) Superior aspect of iliac crest corresponds to L4 vertebra
   (d) Posterior superior iliac spine corresponds to S2 vertebra

5. When performing thoracic epidural injection:
   (a) It is always performed by a paramedian approach
   (b) Midline approach is impossible because of the acute angulation of the thoracic spinous processes
   (c) It is possible to perform it by inserting the needle in the midline at an acute angle
   (d) Motor blockade produced at this level can paralyze the diaphragm

6. When performing a caudal epidural injection, the structures through which the needle passes through are:
   (a) Skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, and ligamentum flavum
   (b) Skin, subcutaneous tissue, sacrococcygeal membrane and ligamentum flavum
   (c) Skin, subcutaneous tissue, interspinous ligament, and sacrococcygeal membrane
   (d) Skin subcutaneous tissue and sacrococcygel membrane

7. A 75-year-old man with a history of ischemic heart disease (IHD) is undergoing hip arthroplasty under epidural. His base line heart rate and mean arterial pressure (MAP) prior to anesthesia were 75/min and 100 mm of Hg, respectively. After administration of epidural you notice that his heart rate has dropped to 55/min and MAP to 50 mm of Hg. Choose the most logical intervention:
   (a) There is no need for any intervention as the reduction in heart rate has reduced the myocardial oxygen demand
   (b) Atropine is indicated for bradycardia
   (c) Hypotension need not be treated as it helps to reduce blood loss
   (d) Treat hypotension as the reduction in myocardial oxygen demand will be offset by reduced oxygen supply secondary to hypotension

8. All of the following are true regarding epidural blockade except:
   (a) A reduction in heart rate can be due to blockade of cardioaccelerator fibers
   (b) A reduction in heart rate can be due to baroreceptor-mediated reflex bradycardia
   (c) Reduction in blood pressure due to vasodilatation reduced venous return and reduced adrenal cortical secretions
   (d) Selective lumbar or thoracic blockade has the benefit of providing hemodynamic stability as a result of compensatory vasoconstriction in unblocked segments

9. Which of the following effect is NOT caused by epidural blockade?
   (a) Parasympathetic blockade leading to reduction in blood pressure and nausea
   (b) Small, contracted bowel
   (c) Increased upper GI motility
   (d) Increased GI secretions

10. Electrodes for spinal cord stimulation are place in:
    (a) Subarachnoid space
    (b) Subdural space
    (c) Intrathecal space
    (d) Epidural space

11. The following is an absolute contraindication for epidural blockade:
    (a) Coagulopathy
    (b) Patient refusal
    (c) A bleeding patient
    (d) A septic patient

12. You are performing an epidural catheter insertion for labor analgesia. The epidural space is identified at a depth of 6 cm. A catheter is then passed and on withdrawing the Touhy needle the marking at the skin corresponds to 15 cm. The length of catheter remaining inside the epidural space probably is:
    (a) 15 cm
    (b) 10 cm
    (c) 9 cm
    (d) 11 cm

13. The structures negotiated by a Touhy needle during the paramedian approach are:
    (a) Skin, subcutaneous tissue, paraspinous muscles, and ligamentum flavum
    (b) Skin, subcutaneous tissue, interspinous ligament, and ligamentum flavum
    (c) Skin, subcutaneous tissue, paraspinous muscles, and interspinous ligament
    (d) Skin, subcutaneous tissue, supraspinous ligament, and ligamentum flavum

14. When epinephrine is used as an additive in epidural anesthesia:
    (a) The dose of local anesthetic should be reduced to avoid drug toxicity
    (b) It increases the depth of neural blockade
    (c) There is a rapid systemic absorption of the drugs injected which is indicated by an increase in heart rate
    (d) A sudden increase in heart rate indicates successful epidural deposition of the drug

15. The following statement is NOT true regarding PDPH:
    (a) CSF pressure is low
    (b) The treatment of choice is immediate blood patch
    (c) Can present with cranial nerve symptoms
    (d) Its relation to posture is characteristic

Answers:

1. b – Epidural analgesia reduces the stress response to surgery by providing effective analgesia, which is beneficial for cardiovascular, respiratory, and metabolic functions. There is a reduction in postoperative hypercoagulable

state, which in turn reduces the incidence of deep vein thrombosis. Though there is a reduction in postoperative morbidity, there is no evidence for reduction in mortality after surgery.

2. c
3. a – It is 2 mm in thickness at the cervical region and 5–6 mm in the lumbar region.
4. a – There is no C8 vertebra though there is C8 nerve root.
5. c – Though paramedian approach is relatively easy to perform, a midline approach can also be used to administer thoracic epidural injection. Motor blockade at thoracic level does not affect diaphragmatic function as the innervation of diaphragm is by the phrenic nerve, which originates at cervical level (C3–C5).
6. d – There is no ligamentum flavum over the sacral hiatus as it fuses with the caudal lamina.
7. d – Though reduction in heart rate reduces myocardial oxygen demand, oxygen supply will be maintained only if MAP is maintained. In presence of IHD, it is safe not to allow the MAP to drop below 20% of the base line value.
8. c – Reduction in blood pressure is due to a reduction in adrenal *medullary* secretions.
9. a – Sympathetic blockade leads to unopposed vagal dominance.
10. d
11. b – All the others are relative contraindications.
12. c
13. a – Paramedian approach avoids both supraspinous and interspinous ligaments, and hence, it is easier by this approach in the elderly who may have calcified ligaments.
14. b – Epidural epinephrine produces local vasoconstriction and decreases systemic absorption of local anesthetic, and hence, a higher dose of local anesthetic can be used. A sudden increase in heart rate indicates the intravascular injection of the drug (into epidural veins).
15. b – First line of treatment for PDPH is bed rest, simple analgesics, and hydration. If this fails then epidural blood patch is indicated.

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13

Upper Extremity Nerve Blocks

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Contents

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Introduction

For more than a century, brachial plexus blockade has been an indispensable tool in the regional anesthesiologist's armamentarium. By providing surgical anesthesia and postoperative analgesia to the entire upper limb, it has been intimately linked to advances in orthopedic and ambulatory anesthesia. Furthermore, with the advent of ultrasonography, upper extremity blocks are being rediscovered under a new light. Every month, anesthesia journals report novel methods to anesthetize different parts of the brachial plexus. Navigating this plethora of studies can be a daunting task. This chapter aims to present a concise discussion of approaches and techniques for brachial plexus blockade based on available evidence.

Clinical Anatomy of the Brachial Plexus

The brachial plexus (Fig. 13.1) is derived from the anterior primary rami of the fifth, sixth, seventh, and eighth cervical nerves as well as the first thoracic nerve in about 75% of the individuals, with variable contributions from the fourth cervical nerve in 15–62% of cases ("prefixed" brachial plexus) and the second thoracic nerve in 16–73% of cases ("postfixed" brachial plexus).

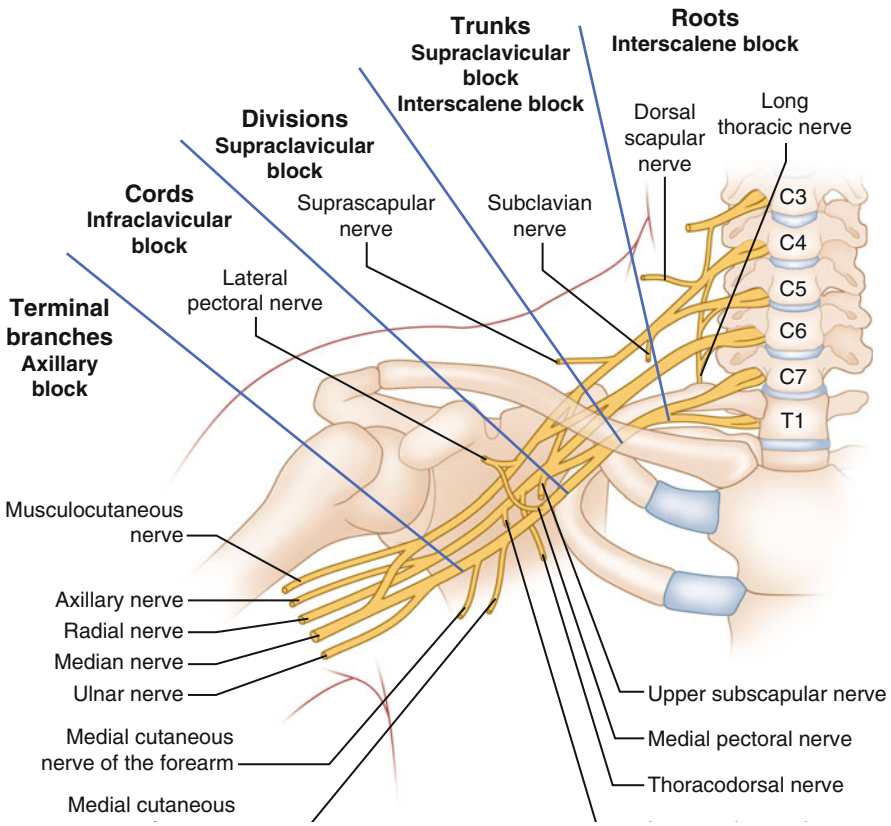

Fig. 13.1 Anatomy of the brachial plexus

The length of the roots, from foramina to trunk, varies between 30 mm (C8 and T1), 40 mm (C5), 50 mm (C6), and 60 mm (C7). The duramater and the epidural connective tissues in the vertebral canal follow the roots to form the perineurium and epineurium, respectively. The roots leave the intervertebral foramina and course between the anterior and middle scalene muscles in the posterior triangle of the neck. Before forming the three trunks (superior, inferior, and middle), the roots give rise to the following nerves:

(a) The long thoracic nerve (C5, C6, and C7), which innervates the anterior serratus muscle, either traverses the middle scalene muscle or exits between the posterior and the middle scalene muscles.
(b) The dorsal scapular nerve (C5), which innervates the rhomboid and levator scapulae muscles, exits behind the middle scalene muscle.
(c) Although the phrenic nerve stems from the C3, C4, and C5 nerves, in 20% of cases, it originates entirely from the roots of the brachial plexus.
(d) The C5, C6, C7, and C8 roots also provide innervation to the scalene and longus colli muscles.

Of the three trunks, the only one giving rise to peripheral branches is the superior trunk. The suprascapular nerve (C5 and C6), which supplies the supra- and infraspinatus muscles, and the nerve to the subclavius muscle (C5 and C6) both originate from the latter.

At the lateral edge of the first rib, each trunk separates into anterior and posterior divisions.

Subsequently, the divisions join to form three cords. The cords are termed lateral (formed from the anterior divisions of the superior and middle trunks, therefore C5 + C6 + C7), posterior (formed from all posterior divisions, C5 + C6 + C7 + C8 + T1), and medial (formed from the anterior divisions of the lower trunk, C8 + T1) based on their relationship with the axillary artery. The cords give rise to multiple side branches:

(a) The lateral pectoral nerve originates from the lateral cord.
(b) The medial pectoral nerve, the medial cutaneous nerve of the arm, and the medial cutaneous nerve of the forearm originate from the medial cord.
(c) The upper subscapular nerve, the lower subscapular nerve, and the thoracodorsal nerve originate from the posterior cord.

At the lateral border of the pectoralis minor muscle, the cords divide into terminal branches: the musculocutaneous nerve (lateral cord), axillary nerve (posterior cord), radial nerve (posterior cord), median nerve (lateral and medial cords), and ulnar nerve (medial cord).

Choosing the Right Approach

Surgery of the Shoulder, Clavicle, and Proximal Humerus

The clavicle and the (posterior) proximal humerus are innervated by the subclavian and suprascapular nerve, respectively (Fig. 13.2). Because they target the latter prior to their take-off from the superior trunk, the cervical paravertebral, interscalene, and supraclavicular approaches can be used.

Although some authors claim that the cervical paravertebral approach differs from its interscalene counterpart because the posterior, and not anterior, cervical roots are anesthetized [1], this remains ambiguous. In the largest randomized controlled trial ($n = 80$) to have compared the two blocks, no differences were found in terms of success rate, extent of the block, as well as onset and offset times [2]. To date, only one study has compared interscalene and supraclavicular blocks. Although block duration, patient satisfaction, postoperative pain scores, and analgesic requirements were similar, the supraclavicular approach resulted in fewer side effects (Horner's syndrome, recurrent laryngeal nerve palsy, and symptomatic diaphragmatic paralysis) [3].

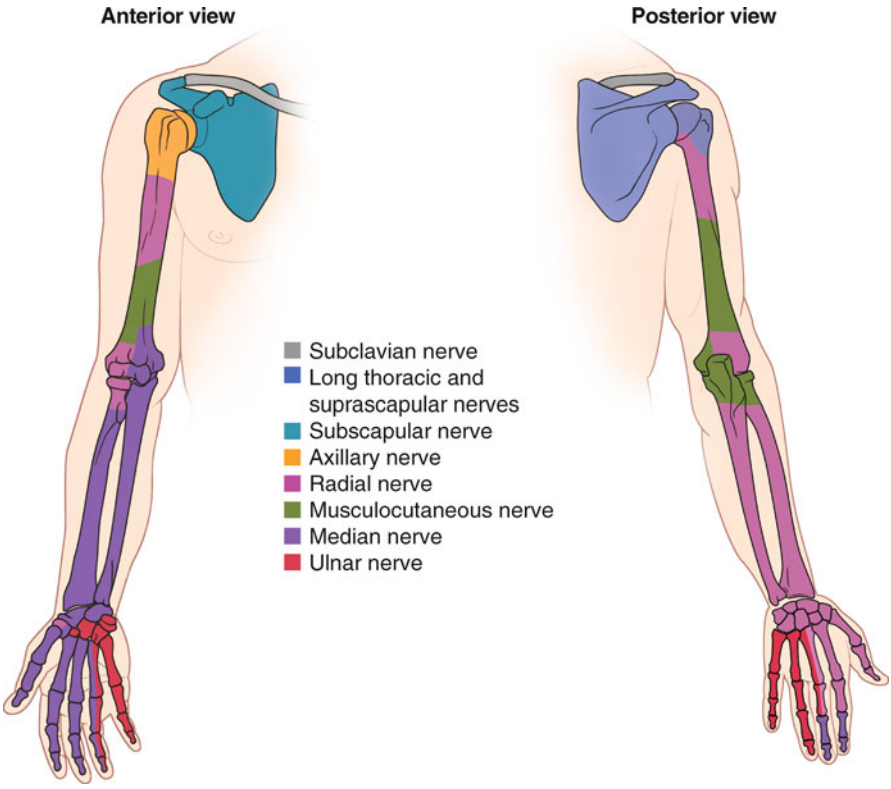

Fig. 13.2 Bony innervation of the upper extremity

Surgery of the Distal Humerus, Forearm, and Hand

The supraclavicular, infraclavicular, and axillary approaches can be used for surgical procedures involving the distal humerus, forearm, and hand. Humeral canal blocks should be reserved for surgery distal to the elbow.

When optimal techniques are utilized for each approach, the literature seems to suggest that supraclavicular, infraclavicular, axillary, and humeral canal blocks result in comparable success rates. As expected, approaches requiring a multiple-injection technique (axillary and humeral canal) can be associated with a longer performance time, more needle passes, or higher block-related pain scores [4–7].

Interscalene Brachial Plexus Block

The Evidence

The interscalene approach anesthetizes the brachial plexus at the level of the roots and trunks. Identification of the plexus in the interscalene groove can be achieved with elicitation of paresthesia, nerve stimulation, or ultrasonography.

To date, three trials have compared elicitation of paresthesia and neurostimulation with mixed results. In two studies, no differences were found [8, 9]. In contrast, the third trial recorded higher failure rates (10 vs. 0%) and postoperative pain scores in the paresthesia group [10].

Comparison of neurostimulation and ultrasonography has also yielded contradictory results. In one study, echoguidance improved the rate of surgical anesthesia (98.8% vs. 91.3%) as well as the onset and offset times [11]. In contrast, another trial observed no differences in performance time, surgical anesthesia, and postoperative neural deficits. However, patients in the ultrasound group required fewer passes [12].

The Techniques

Nerve Stimulation

The patient is supine with the head turned toward the contralateral side. At the level of the cricoid cartilage, posterior to the sternocleidomastoid muscle, the neck is palpated to identify the groove between the anterior and middle scalene muscles (Fig. 13.3).

The skin is infiltrated with local anesthesia. Because the plexus is very superficial, a small volume (<0.3 ml) should be used; otherwise, the evoked motor response may be abolished. A 2.5-cm block needle, connected to a nerve stimulator set at a current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is inserted in the interscalene groove. The needle is oriented in a slight caudad direction to avoid penetration of the intervertebral foramen. Typically, contraction of the deltoid, biceps, triceps, or pectoral muscles is seen. All four constitute acceptable evoked motor responses. If diaphragmatic contraction is encountered, the needle tip is close to the phrenic nerve (situated on the anterior scalene muscle) and thus should be redirected posteriorly. Conversely, if the needle is too posterior, stimulation of the dorsal scapular nerve and shoulder elevation (contraction of the rhomboid and levator scapulae muscles) will occur. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 20–30 ml of local anesthetic are injected.

Ultrasound Guidance

The patient is placed in a supine or semisitting position with the head turned toward the contralateral side. At the level of the cricoid cartilage, the neck is scanned with

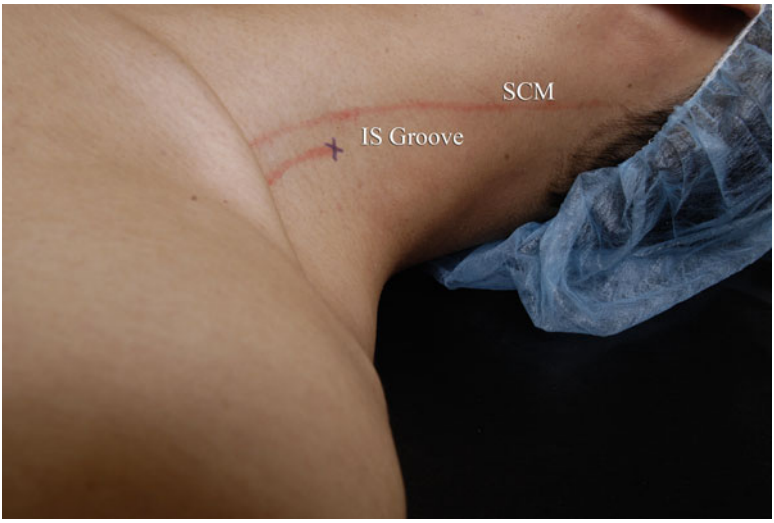

Fig. 13.3 Landmarks for interscalene brachial plexus block (*IS* interscalene, *SCM* sternocleidomastoid muscle, *X* puncture site)

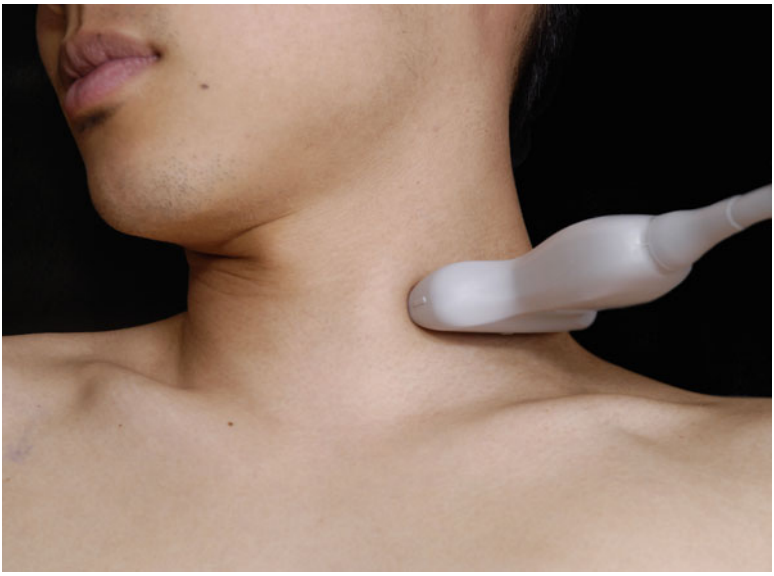

Fig. 13.4 Position of the ultrasound probe for interscalene brachial plexus block

a high-frequency probe (Fig. 13.4). The brachial plexus appears as a column of hypoechoic nodules. The exact nature of the latter (roots vs. trunks) remains controversial (Fig. 13.5). Using an in-plane technique and a lateral to medial direction, the skin and subcutaneous tissues are infiltrated with local anesthesia.

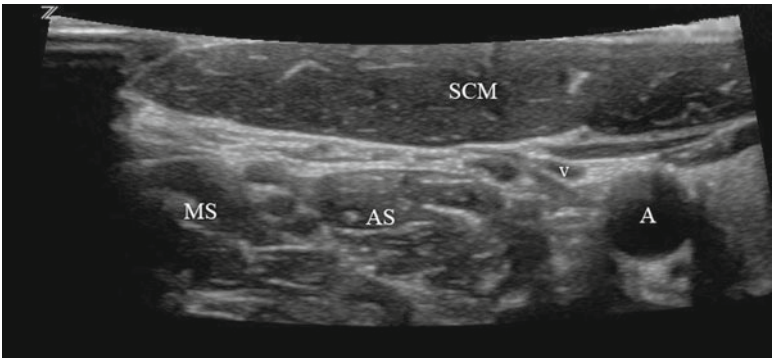

Fig. 13.5 Ultrasonographic appearance of the interscalene and cervical paravertebral brachial plexus (*A* carotid artery, *AS* anterior scalene muscle, *MS* middle scalene muscle, *SCM* sternocleidomastoid muscle, *V* internal jugular vein)

A 5-cm block needle is then inserted. Care must be taken to visualize the entire length of the needle during the advancement process. The classic target for this block is situated between the first and second or second and third nodules. However, an injection in the middle scalene muscle, next to the interscalene groove but without penetration of the latter, seems to provide a similar efficacy [13]. A volume of 20–30 ml of local anesthetic is commonly used.

Complications

Due to the proximity of the cervical sympathetic chain and the recurrent laryngeal nerve, Horner's syndrome and hoarseness can occur. With appropriate technique and equipment, some complications can be prevented: a slight caudad orientation of the needle will decrease the risk of dural cuff puncture and vertebral artery or neuraxial injection. Similarly, limiting the length of needle insertion can prevent the occurrence of a pneumothorax. The most vexing side effect remains the 100% incidence of ipsilateral hemidiaphragmatic paralysis caused by migration of local anesthetics to the C3–5 roots or the phrenic nerve [14]. Usually well tolerated by healthy subjects, it becomes a prohibitive risk in patients with pulmonary compromise. To date, no preventive measure has been found. For instance, a reduction in local anesthetic volume (from 45 to 20 ml) and digital pressure above the injection site did not reduce phrenic paralysis [15–18]. Although 10 ml of bupivacaine 0.25% was not associated with changes in pulmonary function in healthy volunteers [19], a recent study reported a 45% incidence of diaphragmatic paralysis despite limiting the dose of local anesthetic to 5 ml of ropivacaine 0.5% [20].

Cervical Paravertebral Brachial Plexus Block

The Evidence

The cervical paravertebral approach anesthetizes the brachial plexus at the level of the roots and proximal trunks. Identification of the plexus can be carried out with loss of resistance, nerve stimulation, or ultrasonography. To date, no study has compared these three modalities. Most clinicians seem to prefer the latter two.

The Techniques

Nerve Stimulation

The patient is placed in a sitting or lateral decubitus position with the surgical side uppermost. The neck is flexed to facilitate palpation of the C6 and C7 spinous processes. Three to four centimeters lateral to the latter, a paravertebral line is traced in a cephalocaudal axis. This often corresponds to the groove between the levator scapulae and trapezius muscles. The puncture site is located on the midpoint of this paravertebral line (Fig. 13.6).

The skin and subcutaneous tissues are infiltrated with local anesthesia. A 10-cm block needle, connected to a nerve stimulator set at a current of 1.5 mA, a pulse

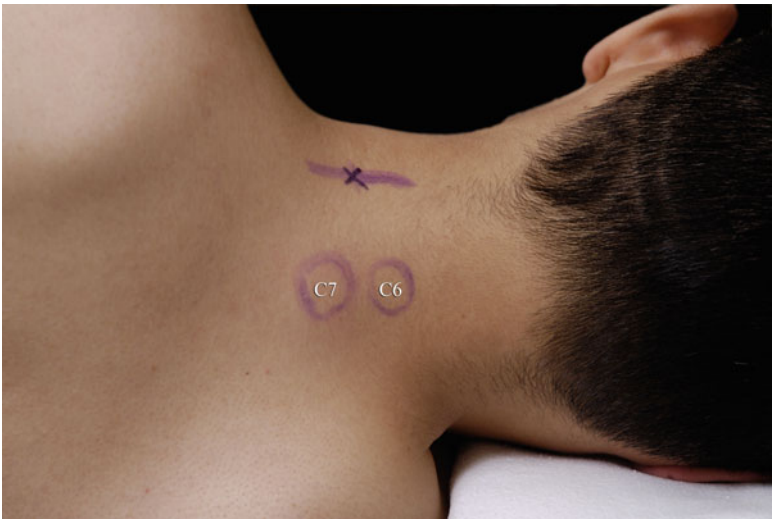

Fig. 13.6 Landmarks for cervical paravertebral brachial plexus block (*X* puncture site)

Fig. 13.7 Position of the ultrasound probe for cervical paravertebral brachial plexus block

width of 0.1 ms, and a frequency of 2 Hz, is inserted perpendicularly to the skin until contact with the pars intervertebralis or transverse process. It is then walked laterally off the bone and advanced until contraction of the deltoid, biceps, triceps, or pectoral muscles is seen. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 20–30 ml of local anesthetic are injected.

Ultrasonography

The patient is placed in a lateral decubitus position with the surgical side uppermost. At the level of the cricoid cartilage, the neck is scanned with a high-frequency probe to identify the carotid artery (Fig. 13.7). The probe is moved laterally until the interscalene groove can be identified. The brachial plexus appears as a column of hypoechoic nodules (Fig. 13.5). The puncture site for this block is situated in the groove between the levator scapulae and trapezius muscles. The skin and subcutaneous tissues are infiltrated with local anesthesia. Using an in-plane needle, a 10-cm block needle is directed toward the brachial plexus. Care must be taken to visualize the entire length of the needle during the advancement process. The target for this block can be the plexus itself (between the hypoechoic nodules) or the middle scalene muscle, next to the interscalene groove but without penetration of the latter. A volume of 20–30 ml of local anesthetic is commonly used.

Complications

Adverse events related to the cervical paravertebral approach are similar to those associated with interscalene blocks (Horner's syndrome, hoarseness, vascular breach, and hemidiaphragmatic paralysis). Two potential complications deserve special mention. Because the needle traverses the extensor muscles of the neck, muscular pain can be problematic; inserting the needle in the groove between the levator scapulae and trapezius muscles may decrease the incidence of neck pain. Neuraxial spread of local anesthetic agents can occur in up to 4% of cervical paravertebral blocks [21]. To minimize this risk, some authors recommend avoiding sharp needles, which can pierce the dural cuffs. The vertebral artery is situated anterior to the pars intervertebralis or articular column of the vertebrae. Therefore, the vertebral artery should be protected from puncture if the needle is introduced to contact bone and walked laterally off the latter.

Supraclavicular Brachial Plexus Block

The Evidence

The supraclavicular approach anesthetizes the brachial plexus at the level of the trunks and divisions. This block can be performed by elicitation of paresthesia, neurostimulation, or ultrasonography. The last two modalities are usually preferred. Although various surface landmarks have been described, the plumb bob technique is most commonly used [22]. For nerve stimulation, currents of 0.9 and 0.5 mA yield similar success rates, onsets, and durations of anesthesia [23]. For ultrasound guidance, the "eight ball, corner pocket" technique, whereby local anesthetic is injected at the intersection of the first rib and subclavian artery, seems to provide a reliable method to block the brachial plexus [24]. Compared to neurostimulation, ultrasonography results in a similar success rate coupled with a lower incidence of phrenic nerve blockade [25].

The Techniques

Nerve Stimulation

For the "plumb bob" technique, the patient is supine with the head turned toward the contralateral side. The head is raised to identify the insertion of the lateral border of the sternocleidomastoid on the clavicle. A 5-cm block needle is inserted at this point

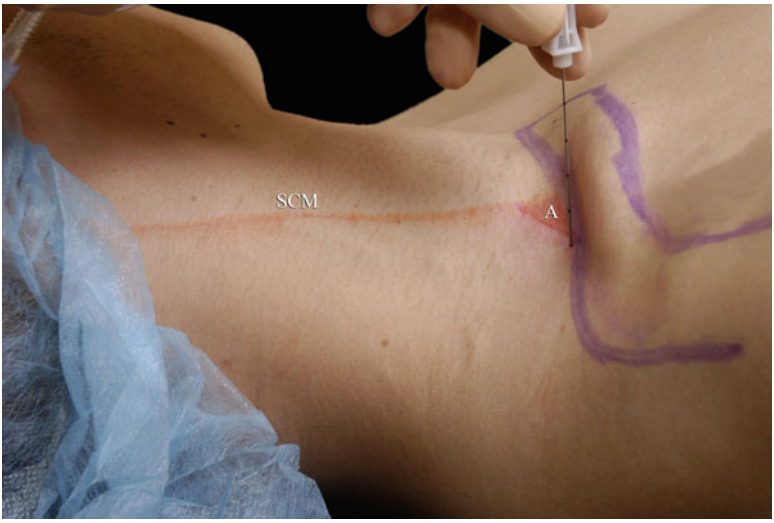

Fig. 13.8 Landmarks for supraclavicular brachial plexus block (*A* subclavian artery, *SCM* sternocleidomastoid muscle)

perpendicularly to the floor (Fig. 13.8). Failure to elicit an evoked motor response should be followed by redirection of the needle in a cephalad or caudad direction (in a parasagittal plane). Care is taken not to exceed an arc of 30°. After ensuring that the evoked motor response is still present at a current of 0.9 mA or lower, 30–40 ml of local anesthetic are commonly used.

Ultrasound Guidance

The patient is supine or semisitting with the head turned toward the contralateral side. Using a high-frequency probe, the supraclavicular area is scanned to identify a short-axis view of the subclavian artery (Fig. 13.9). The first rib can be seen under the vessel. Superolateral to the artery, a collection of hypoechoic structures (trunks and divisions of the brachial plexus) can be seen. The skin and subcutaneous tissues are infiltrated with local anesthesia. Using an in-plane technique and a lateral to medial direction, a 5-cm block needle is directed toward the "corner pocket," i.e., the intersection between the subclavian artery and the first rib (Fig. 13.10). Care must be taken to visualize the entire length of the needle during the advancement process. Using a mix of lidocaine and bupivacaine, Duggan et al. [26] reported the ED50 and ED95 of ultrasound-guided supraclavicular blocks to be 23 and 42 ml, respectively.

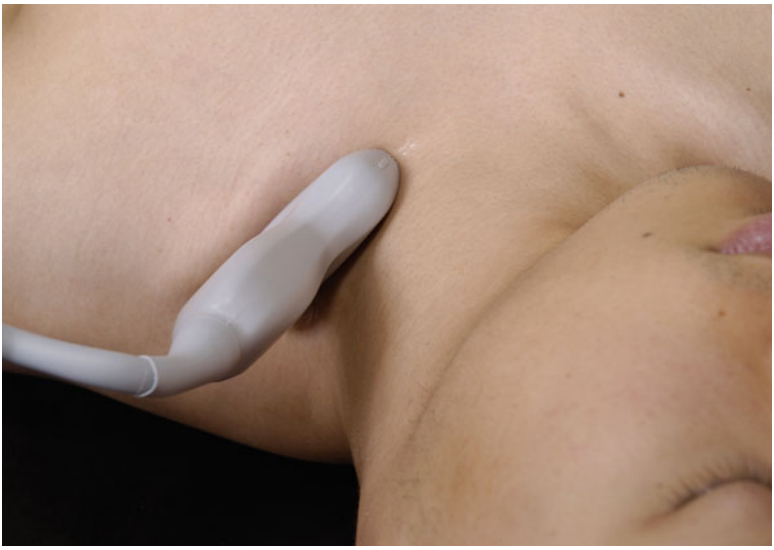

Fig. 13.9 Position of the ultrasound probe for supraclavicular brachial plexus block

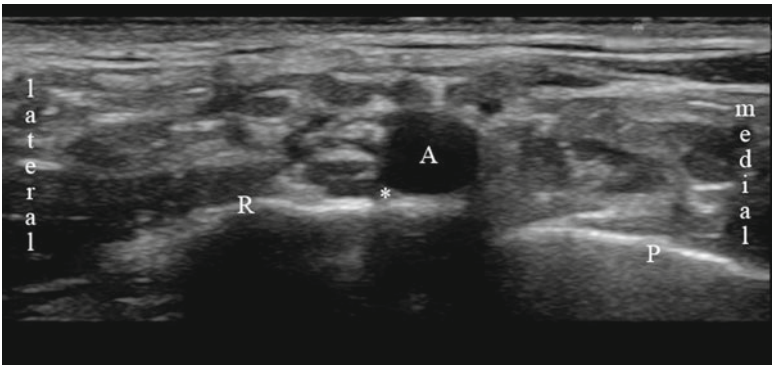

Fig. 13.10 Ultrasonographic appearance of the supraclavicular brachial plexus (*A* subclavian artery, *P* pleura, *R* first rib, *asterisk* indicates target)

Complications

Vascular puncture, recurrent laryngeal nerve paralysis, and Horner's syndrome can occur after supraclavicular blocks. The risk of pneumothorax can be as high as 6% if traditional techniques, which direct the needle in a cephalocaudal direction toward the lung, are used. Because phrenic nerve blockade can occur in 67% of cases, like its interscalene counterpart, this block is contraindicated in patients with pulmonary compromise [27].

Infraclavicular Brachial Plexus Block

The Evidence

The infraclavicular approach anesthetizes the brachial plexus at the level of its cords (lateral, posterior, and medial). This block can be performed with neurostimulation or ultrasonography. For neurostimulation-guided infraclavicular blocks, the available literature favors a double-injection technique (avoiding the musculocutaneous/median combination) or a single-injection technique aiming for radial-type stimulation [28]. The latter may be slightly superior because of a shorter performance time and better sensory blockade of the ulnar and radial nerves at 30 min [28]. For ultrasound-guided infraclavicular blocks, the optimal technique consists of a single-injection dorsal to the axillary artery [29, 30].

Comparison of nerve stimulation and ultrasonography has yielded mixed results. Two trials comparing single-stimulation infraclavicular block with single- or multiple-injection ultrasound-guided block found similar rates of surgical anesthesia and onset times [31, 32]. However, in another study, ultrasonography was associated with a higher rate of surgical anesthesia, a shorter performance time, and fewer paresthesia [33]. Although some practitioners routinely combine neurostimulation and ultrasonography, this practice seems to offer minimal benefits. Compared to ultrasound guidance alone, the combination of both modalities unnecessarily increased the performance time [34, 35] and led to a lower success rate [34].

The Techniques

Neurostimulation

Since the first description by Raj et al. [36], several sets of landmarks have been described for infraclavicular blocks. In North America, the most popular method is the pericoracoid technique [37]. With the patient supine, the arm to be blocked is adducted. A point 2 cm medial and 2 cm caudad to the tip of the coracoid process is identified [37] (Fig. 13.11). The skin and subcutaneous tissue are infiltrated with local anesthesia. A 5- to 10-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is inserted perpendicularly to the skin. Usually, elbow flexion (lateral cord stimulation) is encountered first. Using a parasagittal plane, the needle tip is redirected in a caudad direction in search of a radial-type response (extension of the forearm, wrist, or fingers). After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 30–40 ml of local anesthetic are deposited.

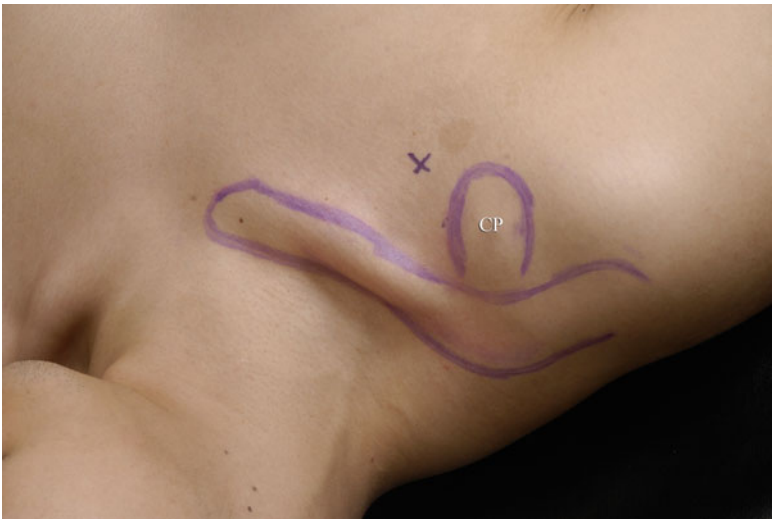

Fig. 13.11 Landmarks for infraclavicular brachial plexus block (*CP* coracoid process, *X* puncture site)

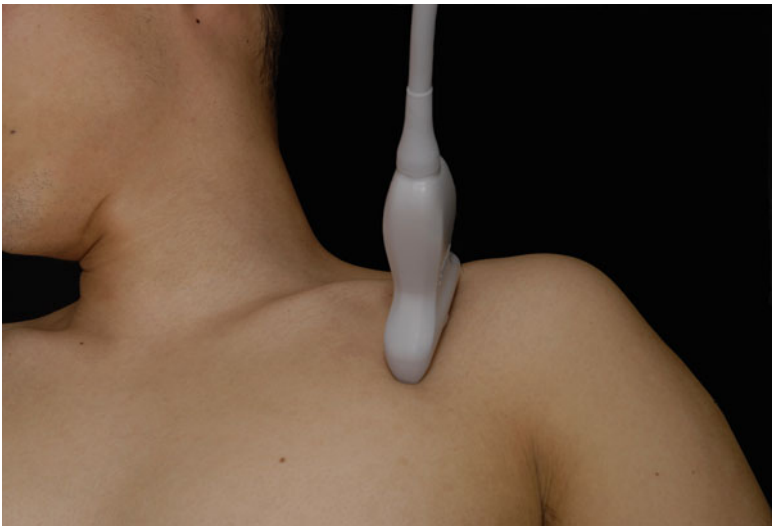

Fig. 13.12 Position of the ultrasound probe for infraclavicular brachial plexus block

Ultrasound Guidance

The patient is positioned supine. The arm is flexed so that the forearm and hand can rest comfortably on the torso. A high-frequency ultrasound probe is placed in the infraclavicular fossa, medial to the coracoid process, to obtain a short-axis view of the axillary vessels (Fig. 13.12). The axillary artery and vein can be found under

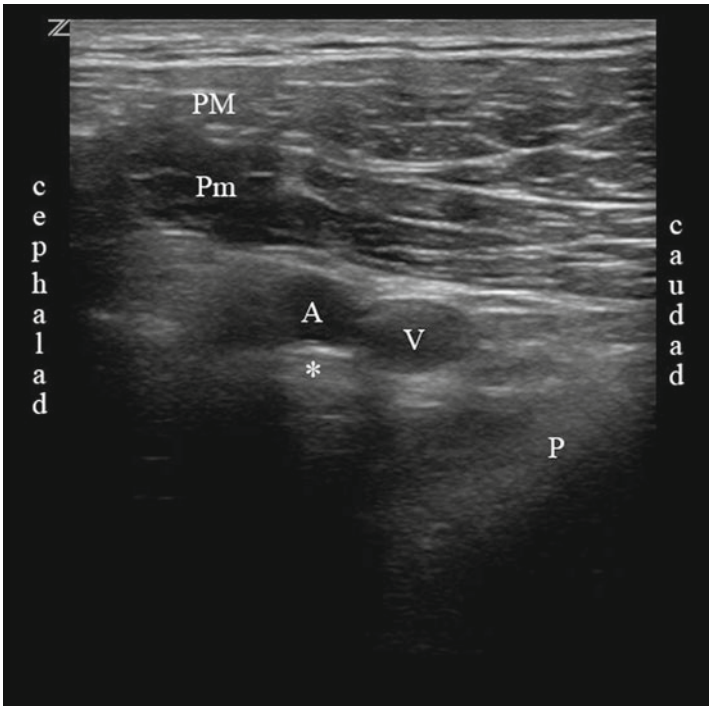

Fig. 13.13 Ultrasonographic appearance of the infraclavicular brachial plexus (*A* axillary artery, *P* pleura, *PM* pectoralis major muscle, *Pm* pectoralis minor muscle, *V* axillary vein, *asterisk* indicates target)

the pectoralis major and minor muscles. The pleura can sometimes be seen under the vessels (Fig. 13.13). Local anesthetic is used to infiltrate the skin, subcutaneous tissues, and pectoralis muscles. Using an in-plane technique and a cephalad to caudad direction, a 10-cm block needle is advanced until the tip lies just dorsal to the artery. Care must be taken to visualize the needle during the advancement process. Usually, a pop can be felt just before the needle assumes the correct position. Thirty to forty milliliters of local anesthetic agent are injected.

Complications

Vascular puncture can occur. Because of the depth of the vessels, external compression can be difficult to achieve. Thus, caution should be exercised in coagulopathic patients, and perhaps, a different approach should be considered. There have also been anecdotal reports of Horner's syndrome, phrenic paralysis [38], and pneumothorax [39] associated with infraclavicular blocks.

Axillary Brachial Plexus Block

The Evidence

The axillary approach anesthetizes the brachial plexus at the level of its four main terminal branches (musculocutaneous, median, radial, and ulnar nerves). Performing this block by fascial clicks, elicitation of paresthesia, transarterial injection, and single-nerve stimulation yields a similar success rate between 70 and 80% [40]. Thus, most practitioners prefer multiple-nerve stimulation and ultrasound guidance.

With neurostimulation, evidence suggests that a three-injection technique (in which the ulnar nerve is not located) provides a similar efficacy to the four-injection technique [41]. A triple-stimulation technique seeking the median, musculocutaneous, and radial nerves, as opposed to the median, musculocutaneous, and ulnar nerves, also seems to provide a higher success rate [42]. Furthermore, for the radial nerve, a distal motor response (wrist or finger extension) should be preferred to a proximal response (forearm extension) [43].

Compared to a multiple-stimulation technique, a higher success rate and shorter onset time have been reported with ultrasonography [44]. However, another trial found similar success rates [45].

The Techniques

Nerve Stimulation

The patient is positioned with the shoulder abducted and the elbow flexed. The axillary area is palpated to identify the axillary artery. In the axilla, the musculocutaneous and median nerves are most often situated above the artery, whereas the radial and ulnar nerves can be found below the latter. However, a great deal of anatomical variability can occur. For this block, two distinct puncture sites (above and below the artery) are required (Fig. 13.14). The skin is infiltrated with local anesthesia. Because the median nerve is very superficial, a small volume (<0.3 ml) should be used above the artery; otherwise, the evoked motor response may be abolished for the median nerve. A 5-CM block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is first inserted above the artery to locate the musculocutaneous nerve (elbow flexion). After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 ml of local anesthetic are deposited. Subsequently, the needle is repositioned to locate the median nerve (above the artery) and radial nerve (below the artery). Wrist/finger flexion is sought for the former, whereas wrist/finger extension is sought for the latter. For each of these two nerves, a local anesthetic volume of 10–14 ml can be used.

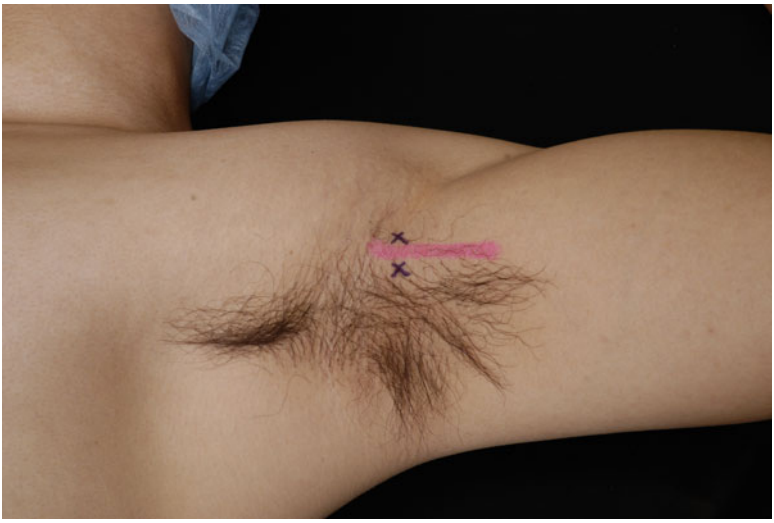

Fig. 13.14 Landmarks for axillary brachial plexus block (*X* puncture sites)

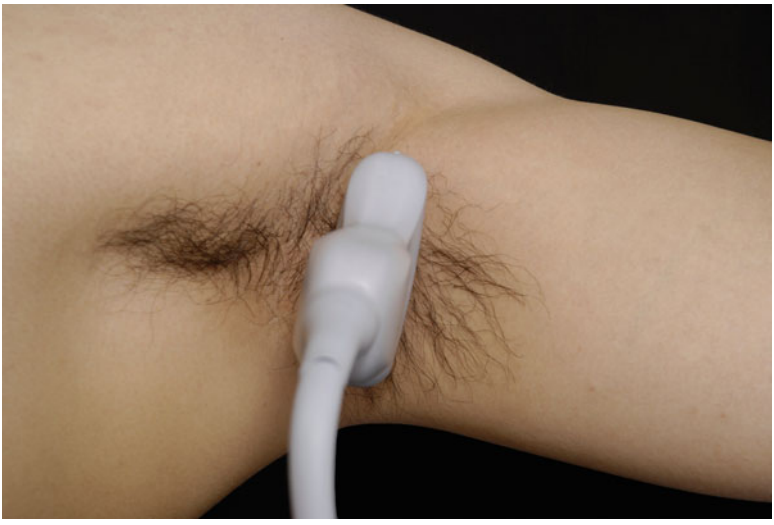

Fig. 13.15 Position of the ultrasound probe for axillary brachial plexus block

Ultrasound Guidance

The patient is positioned with the shoulder abducted and the elbow flexed. The axilla is scanned with a high-frequency linear ultrasound probe to identify a short-axis view of the axillary artery (Fig. 13.15). The musculocutaneous nerve, a hyperechoic structure,

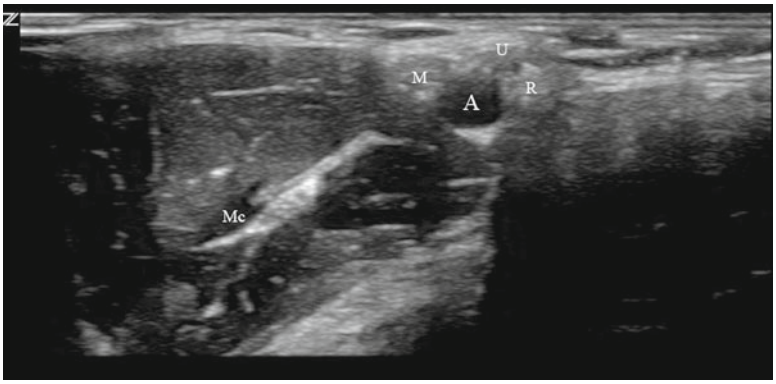

Fig. 13.16 Ultrasonographic appearance of the axillary brachial plexus (*A* axillary artery, *M* median nerve, *Mc* musculocutaneous nerve, *R* radial nerve, *U* ulnar nerve)

can be found anterior and lateral to the artery, in the belly of the coracobrachialis muscles, or in a plane between the coracobrachialis and biceps muscles (Fig. 13.16).

Using an in-plane technique, the skin and subcutaneous tissues are infiltrated with local anesthesia. A 5-cm block needle is then inserted. Care must be taken to visualize the entire length of the needle during the advancement process. The needle is first directed toward the musculocutaneous nerve. Five to seven milliliters of local anesthetic are injected. Subsequently, the needle is redirected toward each of the three remaining nerves: local anesthetic is injected until circumferential spread is achieved for each of them. Although a recent report has advocated a volume as low as 1 ml per nerve [46], in practice, volumes of 5–10 ml can be used. Alternatively, if all three nerves cannot be identified, a perivascular technique can be performed whereby multiple injections are carried out until the axillary artery is surrounded by local anesthetic (donut sign).

Complications

Transient numbness, vascular puncture, intravascular injection, bruising, and soreness at the injection site have been reported, but the overall safety margin for the block is very high.

Humeral Canal Block

The Evidence

Similar to the axillary approach, the humeral canal block anesthetizes the brachial plexus at the level of the terminal branches.

The Techniques

Nerve Stimulation

The patient is positioned with the shoulder abducted and the elbow flexed. Midway between the shoulder and elbow, the arm is palpated to identify the axillary artery. The musculocutaneous and median nerves are most often situated above the artery, whereas the radial and ulnar nerves can be found below the latter. However, the radial nerve can be difficult to find because it courses posterior to the humerus. For this block, two distinct puncture sites (above and below the artery) are required (Fig. 13.17). Because the median and ulnar nerves are very superficial, a small volume (<0.3 ml) is used; otherwise, the evoked motor responses could be abolished. A 5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is first inserted above the artery to locate the musculocutaneous (elbow flexion) and median (wrist/finger flexion) nerves. Subsequently, the needle is repositioned under the artery to locate the radial (wrist/finger extension) and ulnar (extension of the fourth/fifth fingers and ulnar deviation of the wrist) nerves. For the median and radial nerve, currents of 0.8 mA or lower and 0.6 mA or lower should be used, respectively. For the ulnar and musculocutaneous, a threshold of 0.7 mA or lower is recommended [47]. A volume of 5–7 ml of local anesthetic is deposited for each nerve.

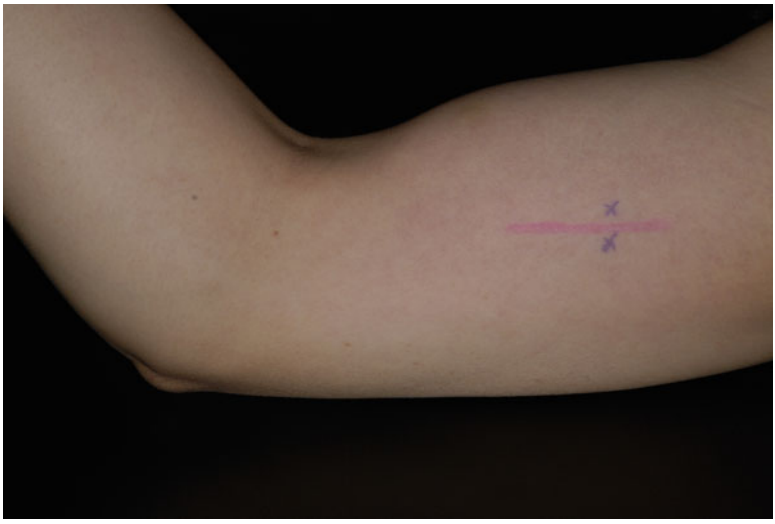

Fig. 13.17 Landmarks for humeral canal block (*X* puncture sites)

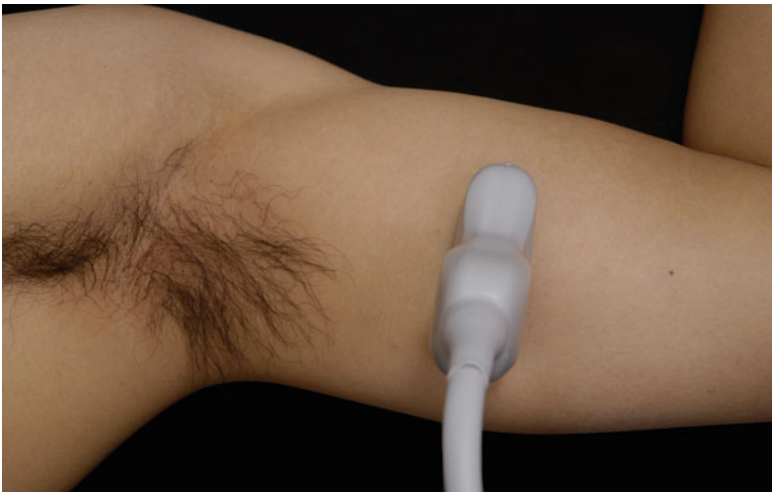

Fig. 13.18 Position of the ultrasound probe for humeral canal block

Ultrasound Guidance

The patient is positioned with the shoulder abducted and the elbow flexed. The arm is scanned with a high-frequency, linear ultrasound probe to identify a short-axis view of the axillary artery (Fig. 13.18). The musculocutaneous and median nerves are situated above the artery, whereas the radial and ulnar nerves can be located below the latter (Fig. 13.19). Using an in-plane technique and puncture sites above or below the artery, a 5-cm block needle is directed toward each of the four neural structures. Care must be taken to visualize the entire length of the needle during the advancement process. Local anesthetic is injected until circumferential spread is achieved for each nerve. Five to seven milliliters are commonly used per nerve.

Complications

Although vascular puncture, bruising, and soreness at the injection site can occur, the overall safety margin for the block is very high.

Supplemental Blocks

In the event of an incomplete brachial plexus block, missing nerves can be anesthetized in a more distal location.

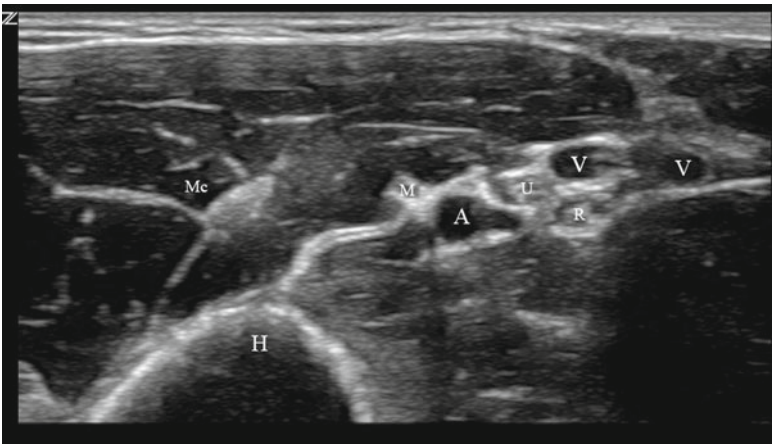

Fig. 13.19 Ultrasonographic appearance of nerves in the humeral canal (*A* brachial artery, *H* humerus, *M* median nerve, *Mc* musculocutaneous nerve, *R* radial nerve, *U* ulnar nerve, *V* brachial vein)

Suprascapular Nerve Block

The Evidence

The suprascapular nerve can be blocked with neurostimulation or ultrasonography. No randomized control trial has compared these two modalities. Cadaveric dissection suggests that ultrasonography targets the suprascapular nerve in the suprascapular fossa, whereas nerve stimulation contacts the nerve in the notch [48].

The Techniques

Neurostimulation

The patient is positioned sitting and leaning forward slightly. The spine of the scapula is identified and is traced. A vertical line passing through the tip of the scapula is also drawn. These two lines divide the scapula into four quadrants. A bisector is drawn for the superolateral quadrant. The puncture site is located 2–3 cm along this bisector (Fig. 13.20). A 5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is introduced at this point perpendicular to the skin. If the scapula is contacted, the needle is redirected superior and medially to enter the suprascapular notch. Abduction or external rotation of the arm is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 10 ml of local anesthetic are deposited.

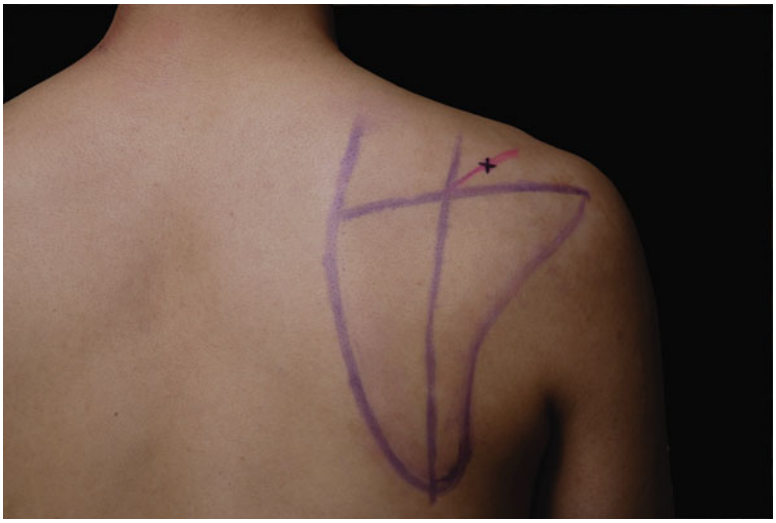

Fig. 13.20 Landmarks for suprascapular nerve block (*X* puncture site)

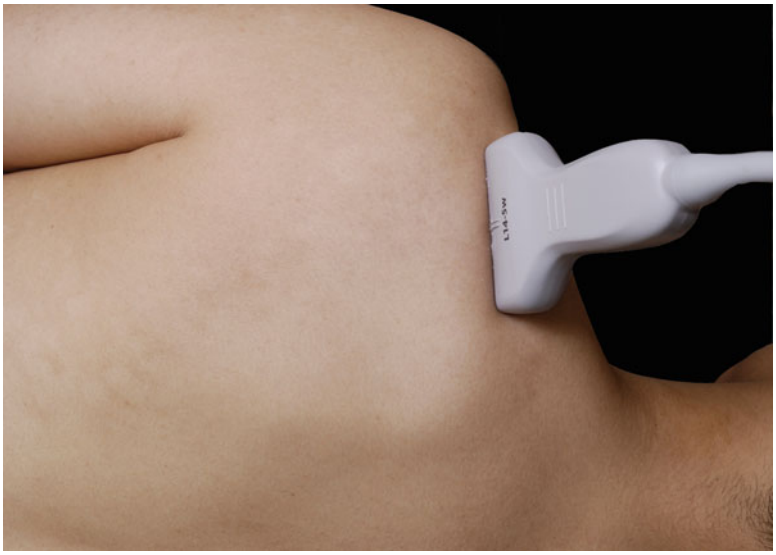

Fig. 13.21 Position of the ultrasound probe for suprascapular nerve block

Ultrasound Guidance

The patient is positioned in the lateral decubitus position so that the side to be blocked is nondependant (Fig. 13.21). Using a high-frequency, linear ultrasound probe, the area cephalad to the scapular spine is scanned to identify the suprascapular

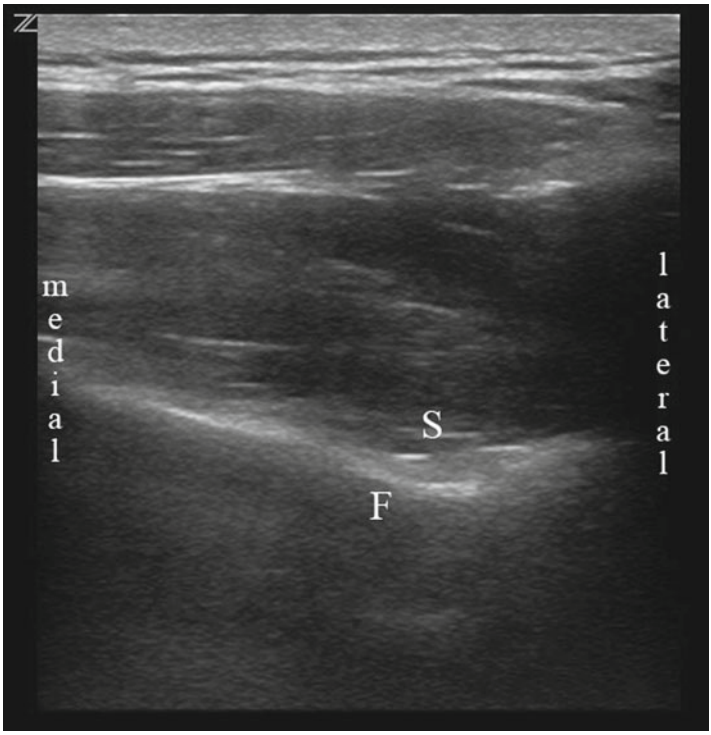

Fig. 13.22 Ultrasonographic appearance of the suprascapular fossa (*F* fossa, *S* supraspinatus muscular fascia)

fossa (Fig. 13.22). The skin and subcutaneous tissues are infiltrated with local anesthesia. Using an in-plane technique, a 10-cm block needle is advanced toward the suprascapular fossa. Care must be taken to visualize the needle during the advancement process. The entire length of the needle may not be visible due to the steep angle of advancement. However, its tip can be followed through tissue distortion. A volume of 10 ml of local anesthetics is deposited in the fossa.

Upper Extremity Distal Nerve Blocks (Radial, Median, and Ulnar Nerves)

The Evidence

The radial, median, and ulnar nerves can be blocked at the elbow or wrist. Blocks performed at the elbow offer more versatility because they can be used for forearm, wrist, and hand surgery. In contrast, blocks performed at the wrist can only be used for procedures involving the hand. To date, no study has compared elicitation of paresthesia, neurostimulation, and ultrasonography for elbow blocks.

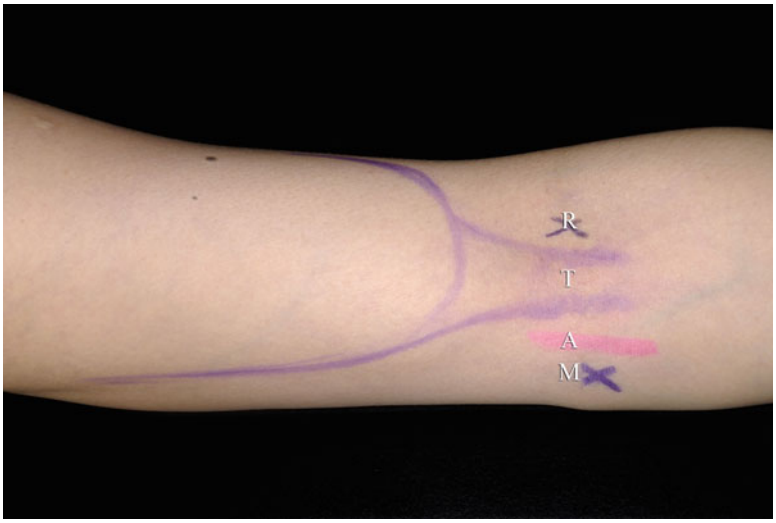

Fig. 13.23 Landmarks for supplemental median and radial nerve blocks at the elbow (*A* brachial artery, *M* median nerve, *R* radial nerve, *T* bicipital tendon)

The Techniques

At the Elbow

Neurostimulation

(a) Radial Nerve
The patient is supine with the upper extremity supinated and abducted. The radial nerve is located lateral to the bicipital tendon between the brachialis and brachioradialis muscles (Fig. 13.23). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is inserted to a depth of 1–2 cm. Wrist or finger extension is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 ml of local anesthetic are deposited.

(b) Median Nerve
The patient is supine with the upper extremity supinated and abducted. The median nerve is located just medial to the brachial artery (Fig. 13.23). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The needle is inserted medial to the brachial artery at a depth of 1–2 cm. Wrist or finger flexion is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 ml of local anesthetic are deposited.

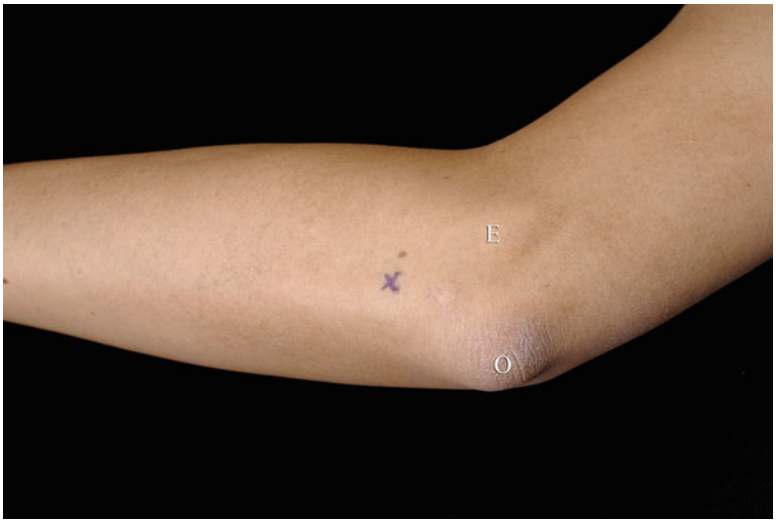

Fig. 13.24 Landmarks for supplemental ulnar nerve block at the elbow (*E* medial epicondyle, *O* olecranon, *X* puncture site)

(c) Ulnar Nerve
The patient is supine with the forearm flexed on the arm to locate the ulnar groove. The nerve is located in the groove between the medial epicondyle and the olecranon process (Fig. 13.24). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is inserted 1–3 cm proximal to a line joining the bony landmarks and directed along the longitudinal axis of the humerus. Ulnar deviation of the wrist and flexion of the little finger are sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 5–7 ml of the local anesthetic agent are deposited.

Ultrasound Guidance

(a) Radial Nerve
The patient is positioned supine with the upper extremity abducted. At the level of the elbow crease, a high-frequency, linear ultrasound probe is used (Fig. 13.25). The radial nerve appears as a hyperechoic crescent (Fig. 13.26). Using an in-plane technique, a 5-cm block needle is advanced toward the nerve. A volume of 5–7 ml of local anesthetic is deposited.

(b) Median Nerve
The patient is supine with the upper extremity abducted. At the level of the elbow crease, a high-frequency, linear ultrasound probe is used (Fig. 13.25). The median nerve is located medial to the brachial artery (Fig. 13.27). Using an in-plane technique, a 5-cm block needle is advanced toward the nerve. A volume of 5–7 ml of local anesthetic is deposited.

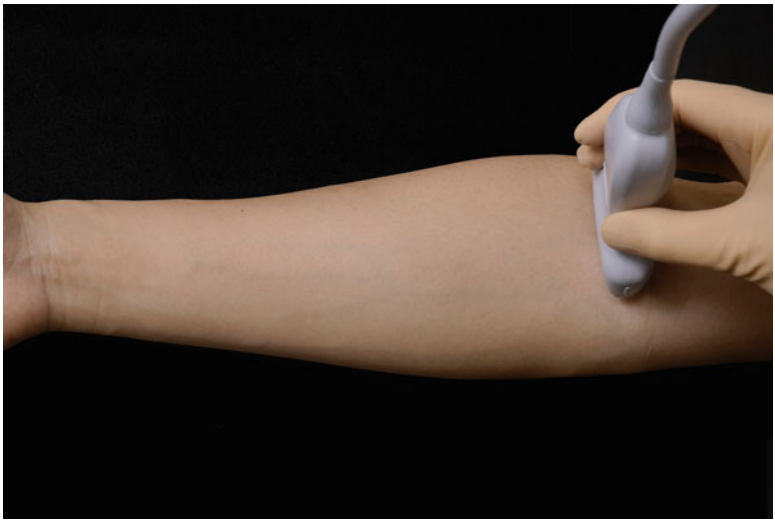

Fig. 13.25 Position of the ultrasound probe for supplemental median and radial nerve blocks at the elbow

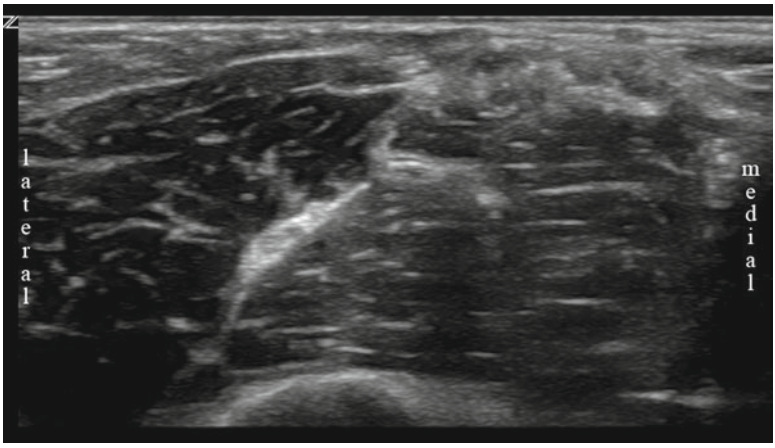

Fig. 13.26 Ultrasonographic appearance of the radial nerve at the elbow

(c) Ulnar Nerve
The patient is positioned supine. The elbow is flexed and the forearm internally rotated so that its radial aspect rests comfortably on the torso. A high-frequency, linear ultrasound probe is used to scan the proximal forearm (Fig. 13.28). The ulnar nerve appears as a hyperechoic structure (Fig. 13.29). Using an in-plane technique, a 5-cm block needle is advanced toward the nerve. A volume of 5–7 ml of local anesthetic is deposited.

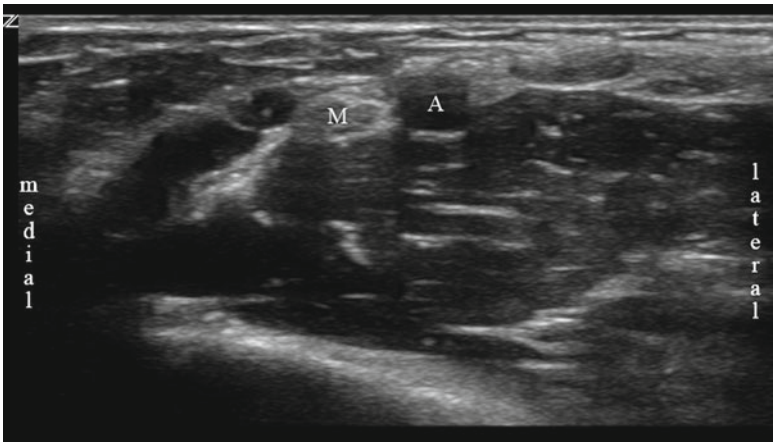

Fig. 13.27 Ultrasonographic appearance of the median nerve at the elbow (*A* brachial artery, *M* median nerve)

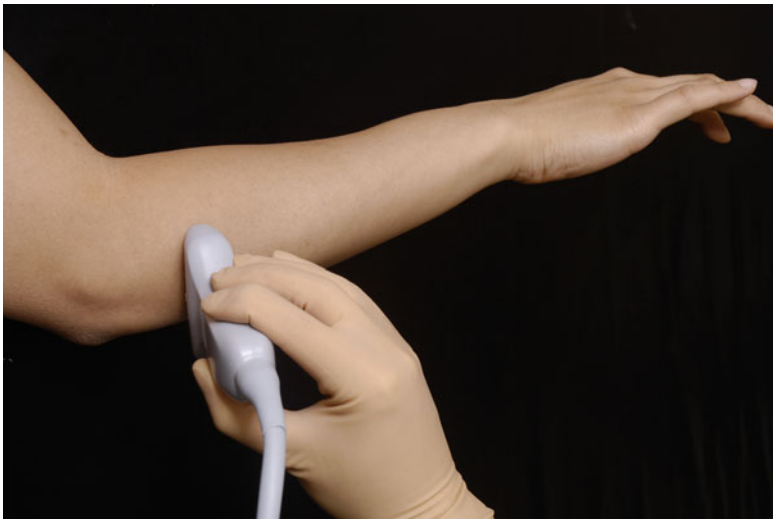

Fig. 13.28 Position of the ultrasound probe for supplemental ulnar nerve block at the elbow

At the Wrist

(a) Radial Nerve
The radial nerve can be blocked at the wrist without the use of neurostimulation or ultrasonography. A field block is performed by injecting 5–7 ml of local anesthetic subcutaneously in and around the anatomical "snuff box" (Fig. 13.30).
(b) Median and Ulnar Nerves

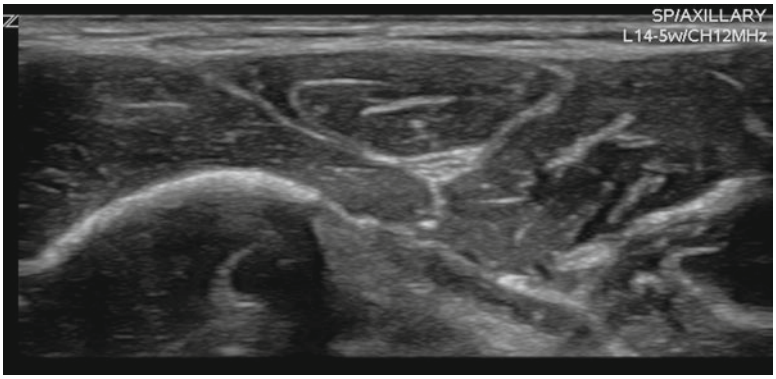

Fig. 13.29 Ultrasonographic appearance of the ulnar nerve at the elbow

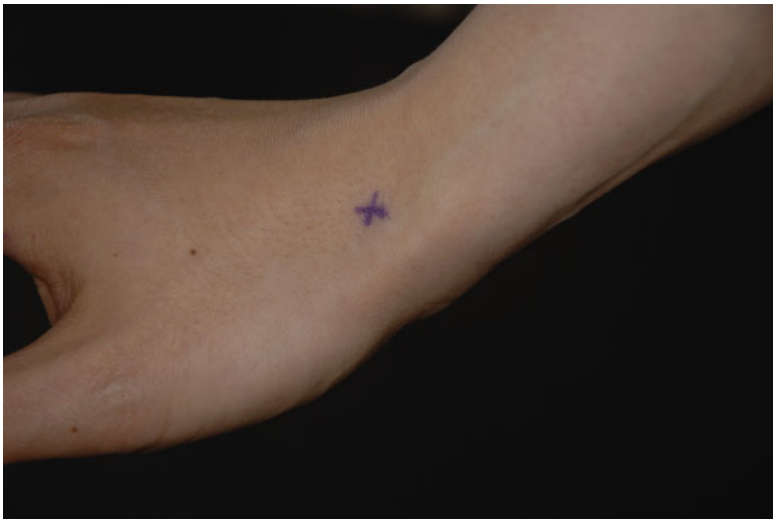

Fig. 13.30 Landmarks for supplemental radial nerve block at the wrist (*X* site of infiltration)

Neurostimulation

(a) Median
The median nerve is located between the tendons of the flexor palmaris longus and the flexor carpi radialis (Fig. 13.31). A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is introduced approximately 3 cm above the wrist crease. Thumb flexion is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 3–5 ml of local anesthetic are deposited.

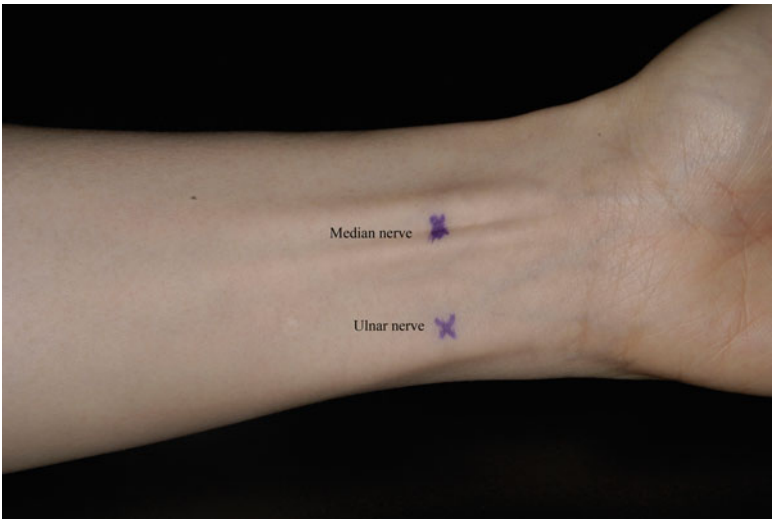

Fig. 13.31 Landmarks for supplemental median and ulnar nerve block at the wrist

(b) Ulnar
The ulnar nerve is located medial to the ulnar artery, below the tendon of the flexor carpi ulnaris muscle. A 2.5-cm block needle, connected to a nerve stimulator set at an initial current of 1.5 mA, a pulse width of 0.1 ms, and a frequency of 2 Hz, is commonly used. The block needle is introduced medial to the artery, 3 cm proximal to the wrist crease (Fig. 13.31). Flexion of the fifth finger is sought. After ensuring that the evoked motor response is still present at a current of 0.2–0.5 mA, 3–5 ml of local anesthetic are deposited.

Ultrasound Guidance

The patient is positioned supine with the upper extremity abducted. The distal forearm is scanned with a high-frequency, linear ultrasound probe (Fig. 13.32). The median nerve appears in the middle of the screen (Fig. 13.33). The ulnar nerve is medial to the ulnar artery (Fig. 13.34). Using an in-plane technique, a 2.5–5-cm block needle is advanced toward each nerve. A volume of 5–7 ml of local anesthetic is deposited for each nerve.

Digital Nerve Block

This block is performed with the hand in the prone position. A 2.5-cm block needle is introduced into the web space of the finger to be anesthetized: this corresponds to the proximal phalanx. A volume of 1–2 ml of local anesthetic is deposited on either side of the finger.

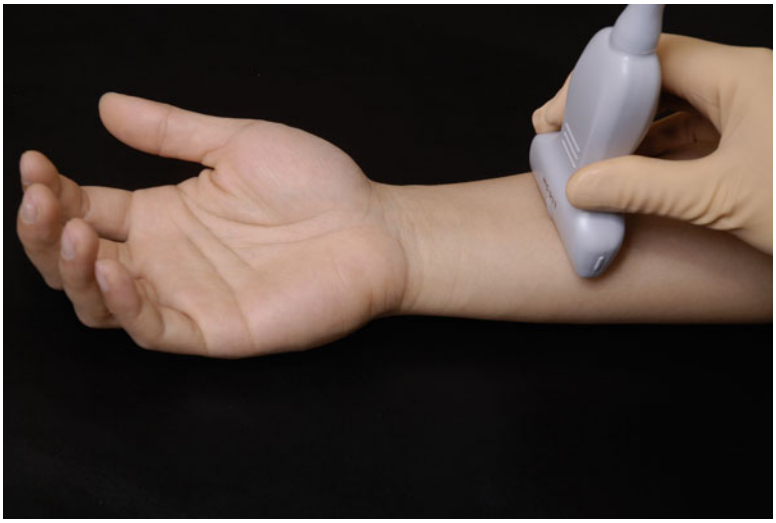

Fig. 13.32 Position of the ultrasound probe for supplemental median and ulnar nerve block at the elbow

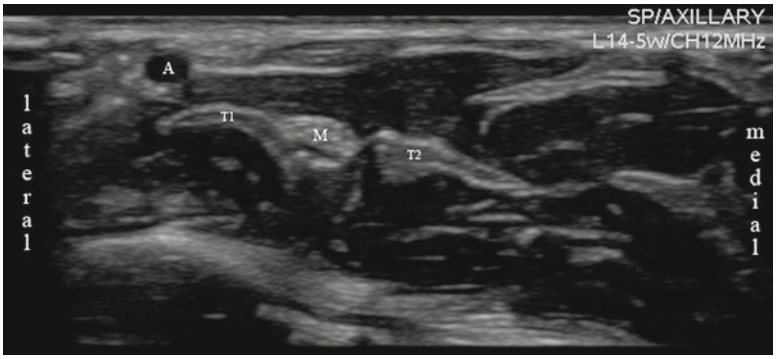

Fig. 13.33 Ultrasonographic appearance of the median nerve at the wrist (*A* radial artery, *M* median nerve, *T1* tendon of the flexor carpi radialis muscle, *T2* tendon of the palmaris longus muscle)

Complications

Most supplemental blocks possess a high safety profile. Vascular puncture (brachial, ulnar, or suprascapular arteries) can occur. For suprascapular nerve blockade, care must be taken not to advance the needle too far past the suprascapular notch: this can lead to a pneumothorax.

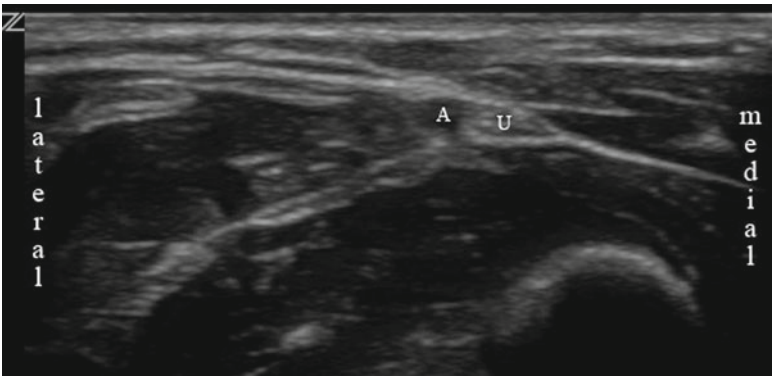

Fig. 13.34 Ultrasonographic appearance of the ulnar nerve at the wrist (*A* ulnar artery, *U* ulnar nerve)

Continuous Brachial Plexus Block

The Evidence

Continuous brachial plexus blockade can be achieved using a blind catheter, a technique whereby the block needle locates the plexus with neurostimulation, and the catheter is simply advanced 1–5 cm past the needle tip. Alternatively, a stimulating catheter can be used: after the obtention of a satisfactorily evoked motor response with the needle, the nerve stimulator is connected to the catheter to ensure that, during the latter's advancement, muscular contractions are preserved. Lastly, ultrasound guidance can also be used to confirm the proximity of needle and catheter to the brachial plexus.

To date, two trials have compared these different techniques. In the setting of shoulder surgery, blind and ultrasound-guided interscalene catheters resulted in similar pain scores and local anesthetic/opioid consumption postoperatively. However, ultrasonography yielded a slightly quicker performance time and lower block-related pain score [49]. The second study compared blind, stimulating, and ultrasound-guided infraclavicular catheters. Unfortunately, the results are difficult to interpret because of the differences in evoked motor responses between groups [50]. Although two trials have demonstrated that, compared to neurostimulation, ultrasound guidance resulted in a quicker performance time for interscalene and infraclavicular catheters, these studies did not rigorously assess pain control during the postoperative infusion of local anesthetic [51, 52].

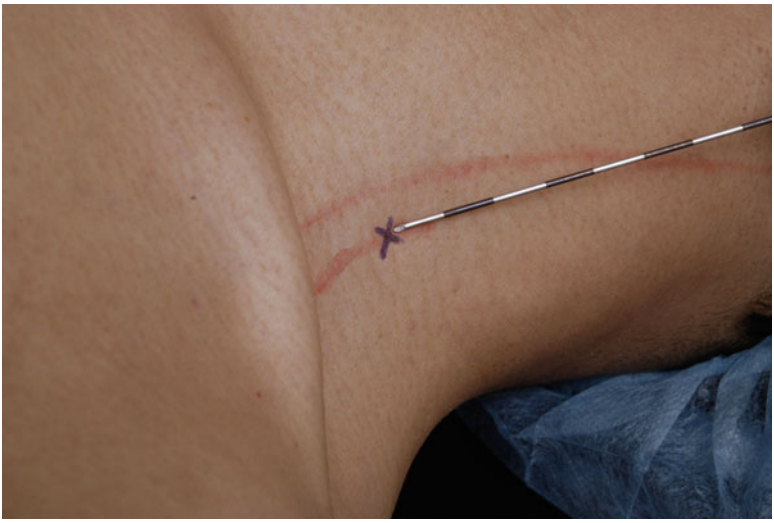

Fig. 13.35 Needle angulation for continuous neurostimulation-guided interscalene brachial plexus block

The Techniques

Blind and Stimulating Catheters

With landmarks similar to single-shot blocks, the block needle is used to locate the brachial plexus. Because neural structures are very superficial in the interscalene and axillary areas, the needle should be inserted with a flat angle to the skin to facilitate subsequent catheter advancement (Figs. 13.35 and 13.36). Although some authors dilate the perineural sheath with a small bolus (5–10 ml) of D5W prior to threading the catheter, this seems to provide minimal benefits [53]. For blind catheters, normal saline or local anesthetic can be used; for stimulating catheters, D5W will preserve the evoked motor response for catheter advancement. After a satisfactorily evoked motor response is obtained with the needle at 0.5 mA, the blind catheter is simply advanced 3–4 cm past the needle tip. A distance greater than 4 cm should be avoided to prevent catheter coiling [54]. If a stimulating catheter is used, the nerve stimulator is disconnected from the needle and connected to the catheter. During the advancement process (3–4 cm), care must be taken to ensure that the evoked motor response and stimulatory threshold do not change. The operator may need to withdraw the catheter into the needle and change the latter's bevel orientation or angulation to accomplish this. After the blind or stimulating catheter has been successfully inserted, the needle is carefully withdrawn over the catheter and the latter secured with adhesive dressings.

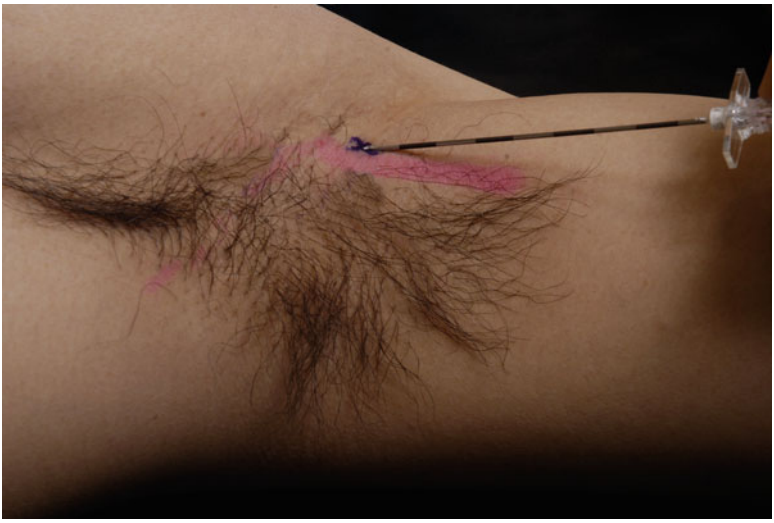

Fig. 13.36 Needle angulation for continuous neurostimulation-guided axillary brachial plexus block

Ultrasound Guidance

After the bolus of local anesthetic has been injected through the needle, the catheter is advanced 3–4 cm past the needle tip. Care must be taken to visualize in real time its passage into the perineural space (Fig. 13.37). This may require the help of an assistant, as a third hand is needed to hold the probe while the operator inserts the catheter through the needle. If the catheter cannot be seen with certainty, its tip can be identified with the injection of a few milliliters of local anesthetics or saline. Alternately, 1 ml of air can be used. This will produce an unmistakable hyperechoic shadow. Air should be used sparingly in order to preserve the quality of the image. After successful placement of the ultrasound-guided catheter, the needle is carefully withdrawn over the catheter and the latter secured with adhesive dressings.

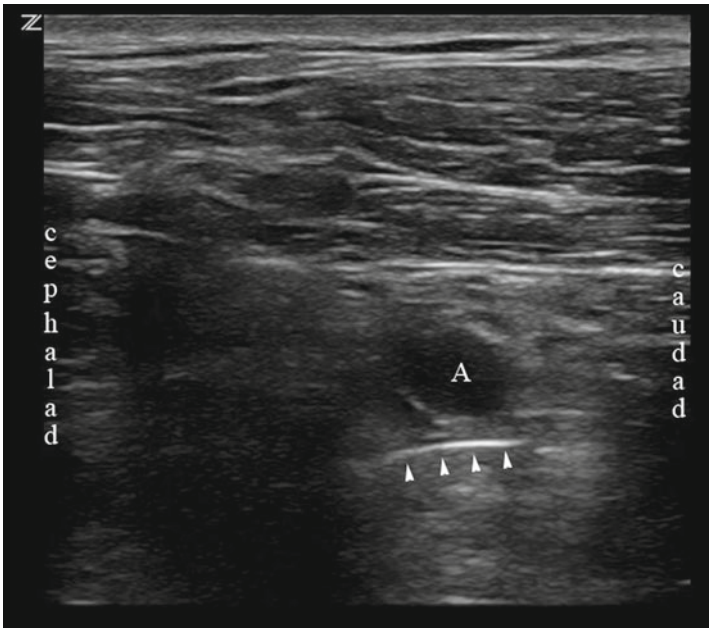

Fig. 13.37 Ultrasonographic appearance of a continuous infraclavicular catheter inserted with ultrasonography (*A* axillary artery, *arrows* indicate catheter)

Clinical Pearls

Clinical Anatomy of the Brachial Plexus

- Although most textbooks recommend selecting nerve blocks based on the cutaneous innervation of the surgical site (Fig. 13.38), knowledge of the osseous innervation (Fig. 13.2) is far more important as postoperative pain rarely stems from trauma to skin.
- The medial and lateral pectoral nerves (which innervate the pectoral muscles) arise from the medial and lateral cords, respectively. Thus, pectoral contraction is an acceptable evoked motor response when performing neurostimulation-guided interscalene or cervical paravertebral brachial plexus block.
- Pectoral contraction should not be accepted for neurostimulation-guided infraclavicular brachial plexus block since it is difficult to differentiate between electrolocation of the nerves and direct stimulation of the pectoral muscles.
- The suprascapular nerve originates from the superior trunk and supplies the posterior aspect of the shoulder. For surgical procedures involving the shoulder joint, it is important to block this nerve before its take-off from the superior trunk. This is best achieved with an interscalene, cervical paravertebral, or supraclavicular approach.

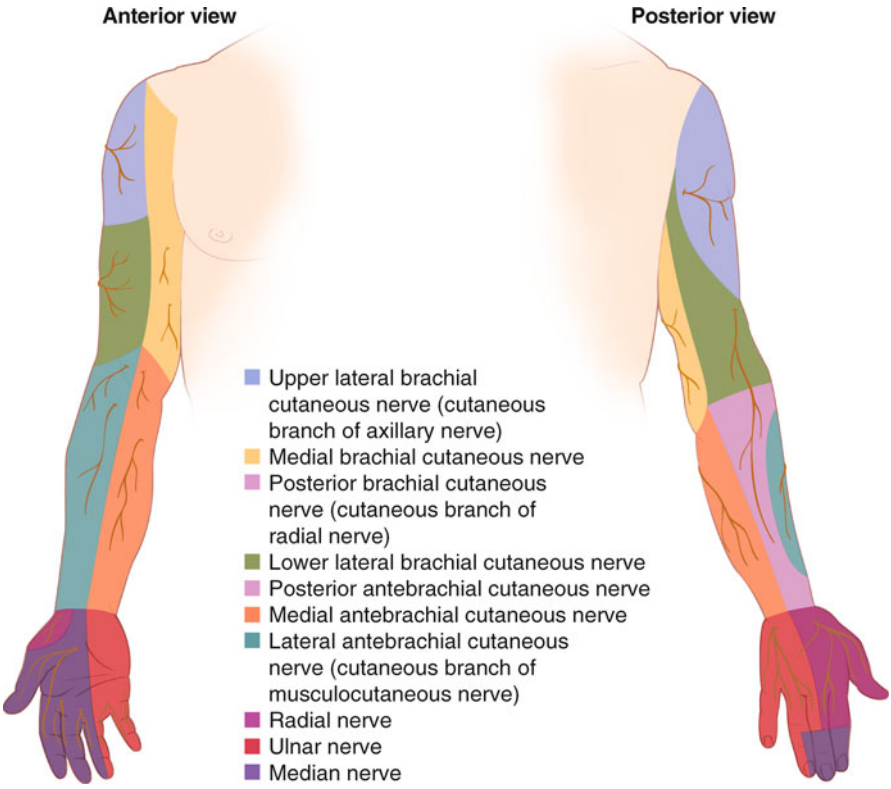

Fig. 13.38 Cutaneous innervation of the upper extremity

- The nerve to the subclavius muscle originates from the superior trunk and is responsible for the bony innervation of the clavicle. For procedures involving the clavicle, an interscalene, cervical paravertebral, or supraclavicular brachial plexus block will enable this nerve to be blocked proximal to its bifurcation.

Choosing the Right Approach

- For single-shot blocks, the cervical paravertebral, interscalene, and supraclavicular approaches can be used to anesthetize the brachial plexus. For continuous perineural catheters, interscalene and cervical paravertebral blocks offer an advantage over supraclavicular blocks because of the latter's proximity to the surgical site. Cervical paravertebral catheters provide an elegant option because they can be tunneled around the hairline and secured on the nonoperative shoulder.
- In light of the comparable efficacy, the selection between supraclavicular, infraclavicular, axillary, and humeral canal approaches should be dictated by potential

adverse events and patient characteristics. For instance, supraclavicular blocks, and their inherent risk of phrenic paralysis, should be avoided in patients with pulmonary compromise. Infraclavicular blocks may be technically difficult in subjects with ample pectoral muscles or breast tissue. Axillary and humeral canal blocks should be avoided in patients (with fractures) who cannot comfortably abduct the upper limb.

Interscalene Brachial Plexus Block

- To ensure that the interscalene groove has been properly identified, palpation of the latter above the clavicle should reveal the presence of an arterial pulsation (subclavian artery).
- If interscalene blocks are performed postoperatively with nerve stimulation, shoulder elevation (dorsal scapular nerve stimulation) can be mistaken for abduction (brachial plexus stimulation) because of the presence of slings and surgical dressings. Before injecting the local anesthetic, the operator should palpate the deltoid muscle and confirm the presence of abduction.
- If the brachial plexus cannot be identified with ultrasonography at the interscalene level, the supraclavicular area can be scanned to locate the subclavian artery. Typically, the brachial plexus (cluster of trunks and divisions) is situated superolateral to the latter. Next, the plexus is slowly traced back toward the cricoid cartilage until it becomes a column of hypoechoic nodules (roots or trunks).

Supraclavicular Brachial Plexus Block

- For neurostimulation, a distal evoked motor response (wrist or hand) seems to provide a better block.
- The risk of pneumothorax is decreased when this block is performed with ultrasound guidance because the entire length of the needle can be visualized.
- Ultrasound-guided injection into the hypoechoic cluster of trunks and divisions reliably anesthetizes the arm and forearm but may spare the hand.

Infraclavicular Brachial Plexus Block

- Magnetic resonance imaging reveals a great deal of anatomic variability in the location of the three cords around the axillary artery. For instance, despite its name, the medial cord is usually posterior (dorsal) to the artery [55].
- With the pericoracoid technique, in order to minimize the risk of pneumothorax, the needle should never be directed medially when searching for the appropriate evoked motor response.

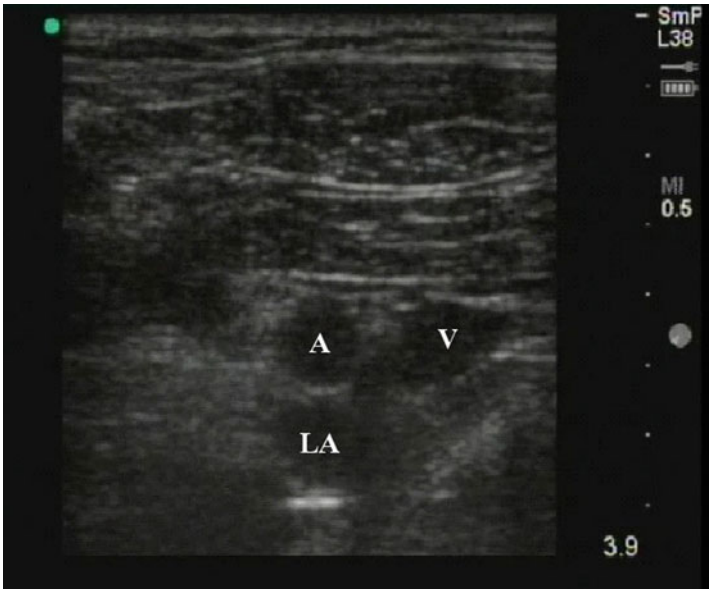

Fig. 13.39 The "Double Bubble" sign (A axillary artery, LA local anesthetic agent, V axillary vein)

- If the needle tip is correctly located with ultrasonography, injection of the first few milliliters of local anesthetic will give a picture resembling a "double bubble" [56]. The "superior bubble" represents the axillary artery in a short axis, and the "inferior bubble," the pool of local anesthetic (Fig. 13.39). As more local anesthetic is deposited, the inferior bubble will turn into a U shape, wrapping itself around the artery, and the latter will be gently pushed ventrally. If a "double bubble" fails to form, the needle tip may be too dorsal in relation to artery; thus, it should be repositioned to lie immediately adjacent to the vessel.
- Occasionally, two axillary veins (cephalad and caudad to the artery) can be present. In this situation, another approach should be considered to avoid vascular puncture.

Axillary Brachial Plexus Block

- If an evoked motor response cannot be obtained, the musculocutaneous nerve can be blocked by contacting the humerus and injecting local anesthetic as the needle is pulled back into the belly of the coracobrachialis muscle. Two or three passes (with different angulations) are needed.
- Care must be taken not to apply too much pressure with the ultrasound probe. This may lead to compression of superficial veins and unrecognized intravascular injection [57].

Humeral Canal Block

- For ultrasound-guided humeral canal blocks, if the radial nerve cannot be identified dorsal to the humerus, the probe can be moved proximally toward the axilla: the nerve will be located more superficially around the brachial artery.
- If the musculocutaneous, median, and ulnar nerves cannot be identified, they can be traced from the axilla downward. Alternatively, the median and ulnar nerves can be traced back from the elbow.

Supplemental Blocks

- At the elbow, for ulnar nerve block, local anesthetic should not be injected directly into the groove: high pressure in this tight fascial compartment can damage the nerve.
- Although many textbooks recommend supplementing brachial plexus blocks with an intercostobrachial nerve block (subcutaneous infiltration of the medial arm with 5–7 ml of local anesthetic) for tourniquet tolerance, this step is seldom necessary. Tourniquet-related pain stems from muscular compression and should be covered by the brachial plexus block. In contrast, the intercostobrachial nerve only provides sensory innervation to the skin.
- For digital nerve block, epinephrine must be avoided in the local anesthetic solution as this may produce ischemia of the fingertips.

Continuous Brachial Plexus Block

- The insertion of stimulating perineural catheters may require multiple attempts. A systematic approach is required to find the optimal combination of needle angulation and bevel orientation. The needle is first rotated 90° at a time to attempt catheter advancement. After the four quadrants have been unsuccessfully explored, the needle angulation is changed and the four quadrants tried again. These two steps (change of angulation and exploration of four quadrants) are repeated until the catheter can be inserted 3–4 cm past the needle tip with preservation of the evoked motor response.
- The optimal stimulatory threshold for perineural catheter placement has not been established. In their practice, the authors tolerate a threshold as high as 0.8 mA (pulse width = 0.1 ms).

Multiple-Choice Questions

1. All the following nerves originate from the brachial plexus EXCEPT:
   (a) Thoracodorsal nerve
   (b) Intercostobrachial nerve
   (c) Lateral pectoral nerve
   (d) Long thoracic nerve

2. All the following nerves originate from the C5 nerve root EXCEPT:
   (a) Suprascapular nerve
   (b) Dorsal scapular nerve
   (c) Phrenic nerve
   (d) Ulnar nerve

3. An injury to the posterior cord will lead to all the following deficits EXCEPT:
   (a) Decreased shoulder abduction
   (b) Decreased elbow extension
   (c) Decreased sensation of the lateral aspect of the shoulder
   (d) Decreased sensation over the medial aspect of the forearm

4. For clavicular surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Cervical paravertebral block
   (b) Supraclavicular block
   (c) Superficial cervical plexus block
   (d) None of the above

5. For shoulder surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Infraclavicular combined with suprascapular nerve blocks
   (b) Infraclavicular combined with superficial cervical plexus blocks
   (c) Cervical paravertebral block
   (d) None of the above

6. For elbow surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Infraclavicular block
   (b) Axillary block
   (c) Supraclavicular block
   (d) None of the above

7. For hand surgery, all the following blocks provide adequate postoperative analgesia EXCEPT:
   (a) Interscalene block
   (b) Humeral canal block
   (c) Supraclavicular block
   (d) None of the above

8. All the following are potential side effects of interscalene blocks EXCEPT:
   (a) Hoarseness
   (b) Exophthalmos
   (c) Myosis
   (d) Dyspnea

9. All the following are potential side effect of infraclavicular blocks EXCEPT:
   (a) Horner's syndrome
   (b) Dyspnea
   (c) Winged scapula
   (d) Perioral numbness

10. All the following evoked motor responses are acceptable for neurostimulation-guided interscalene blocks EXCEPT:
   (a) Shoulder elevation
   (b) Pectoral contraction
   (c) Finger flexion
   (d) Wrist extension

11. All the following evoked motor responses are acceptable for neurostimulation-guided infraclavicular blocks EXCEPT:
   (a) Pectoral contraction
   (b) Elbow extension
   (c) Wrist extension
   (d) Finger flexion

12. Which of the following evoked motor responses is considered suboptimal in the performance of neurostimulation-guided axillary blocks?
   (a) Elbow flexion
   (b) Elbow extension
   (c) Thumb opposition
   (d) Wrist extension

13. With ultrasonography, all the following structures are hyperechoic EXCEPT:
   (a) Musculocutaneous nerve
   (b) Lateral cord
   (c) Superior trunk
   (d) Median nerve in the forearm

14. With ultrasonography, all the following structures are hypoechoic EXCEPT:
   (a) Inferior trunk
   (b) Phrenic nerve
   (c) C7 root
   (d) Medial cord

15. With ultrasonography, all the following nerves can be anesthetized using a perivascular injection EXCEPT:
   (a) Interscalene brachial plexus
   (b) Axillary brachial plexus

(c) Median nerve at the elbow
(d) Ulnar nerve at the wrist

Answers:

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

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14

Peripheral Nerve Blocks for the Lower Extremity

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Contents

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Introduction

Certain steps must be taken before offering a peripheral nerve block to a patient:

- Surgeon and primary anesthesia care team aware and in agreement.
- Patient meets all criteria for surgery and anesthesia: NPO, cardiopulmonary status, etc.
- Patient evaluation is complete: laboratory values, EKG, and CXR.
- Paperwork is completed: patient identification, surgical consent, and laterality.
- Contraindications to block are not present: coagulopathy, refusal (see later section).
- Patient consents to block: risks, benefits, and options explained.

Preparation

- Monitors: blood pressure cuff and pulse oximeter.
- Supplemental oxygen: nasal cannula or face mask.
- Laterality verification (when applicable) by two independent parties.
- Pillows or blankets for positioning.
- Skin marker for laterality and landmarks.
- Drugs: emergency drugs (local anesthetic toxicity, hypotension) and sedative (optional).
- Sterile supplies: antiseptic soap, gloves, needle (see each section), and gauze.
- Local anesthetic: lidocaine to anesthetize skin, local anesthetic, or saline to be injected through block needle.
- Special equipment: nerve stimulator (set to 1.5 mA, 2 Hz, and 0.1 ms; may need to increase frequency to 0.3 ms in patients with neuropathy) and ultrasound (US).
- Catheters sterile supplies (when applicable): towels or drapes, dressing materials to secure the catheter, and sleeve for US probe.
- Nurse or trained assistant to help with documentation, monitoring, and emergencies.

Complications

1. Local anesthetic toxicity from intravascular injection: aspirate prior to the initial injection of local anesthetic and again after every 5 ml injected.
2. Nerve injury: this is much more likely due to a number of other phenomena than direct needle trauma. Differential diagnosis includes direct surgical trauma, ischemic injury from prolonged tourniquet application, and compressive injury from

improper patient positioning. Nerve injury during regional anesthesia can be permanent but usually resolves over weeks. It may result from compression by local hematoma formation, injury by intraneural injection, and needle-associated direct nerve trauma. Minimize this complication by avoiding paresthesias, pain with injection, injection with muscle stimulation at <0.2 mA, and injection with increased resistance.
3. Mask the onset of lower extremity compartment syndrome: lower extremity nerve blocks may be relatively contraindicated in patients with fractures of the tibia and fibula or elective orthopedic procedures of the tibia and fibula.
4. If placing a catheter, confirm with the patient's surgical team that a tourniquet will not be placed over the catheter during the operation. While much evidence is not available, concern exists that tourniquet placement over a peripheral nerve catheter may cause or be associated with an increased risk of nerve damage.

Contraindications

- Patient refusal (number one reason not to do a nerve block).
- Allergy to local anesthetics.
- Infection at the site of injection, sepsis, and generalized systemic infections (elevated temperature and white blood cell count).
- Coagulopathy (more concerning at noncompressible/deep block sites, e.g., lumbar plexus).
- History or diagnostic evaluation that would cause cancellation of surgical procedure.

Sciatic Nerve Block

Anatomy

The largest nerve of the human body, the sciatic nerve, provides motor and sensory innervation to the posterior thigh and lower leg. Sciatic nerve blockade is indicated for pain management associated with lower extremity surgery. Since the saphenous nerve, a branch of the femoral nerve, supplies the medial aspect of the lower leg, very few surgical procedures can be performed with the sciatic block alone, and it is usually combined with a lumbar plexus, femoral, or saphenous nerve block.

The sciatic nerve can be blocked from several different locations depending on the desired area of analgesia. We will discuss each of these in groups according to how the patient is positioned for the nerve block.

Indication

The sciatic nerve arises from the lumbosacral plexus (ventral rami of L4–5 and S1–3) and is actually two nerves in close apposition (tibial – medial – and common peroneal – lateral – nerves). In the pelvis, it is part of the sacral plexus with superior gluteal nerve, inferior gluteal nerve, and posterior femoral cutaneous nerve. The sciatic nerve leaves the pelvis though the greater sciatic foramen below the piriformis muscle, runs between muscle layers in the gluteal region (superficial to superior and inferior gemellus, quadratus femoris, and obturator internus muscles; deep to the gluteus maximus muscle), and continues distally toward the posterior thigh between the greater trochanter and ischial tuberosity.

Response to Nerve Stimulation

Successful stimulation of the sciatic nerve is identified by plantar flexion/inversion (tibial nerve) or dorsiflexion/eversion (common peroneal nerve). However, several studies have shown tibial stimulation to be associated with a more frequent success rate compared with peroneal stimulation with various approaches [1, 2].

Procedure

Needle: 10 cm insulated needle (15 cm – obese patients; 5 cm – prone popliteal approach).

Posterior Approaches

Place the patient in the lateral position with the nonoperative down, operative side up. Straighten the patient's dependent, nonoperative leg and flex the operative extremity as much as they are able at the hip and to a lesser extent at the knee. Flexing the knee at the hip flattens the gluteal muscles and brings the sciatic nerve into a more superficial position.

1. *Labat or "Classic" Approach*. First described in 1924, it has the advantage of blocking the posterior femoral cutaneous nerve [3]. (The parasacral approach does this as well).

   Landmarks

   - Posterior superior iliac spine (PSIS).
   - Greater trochanter.
   - Sacral hiatus.

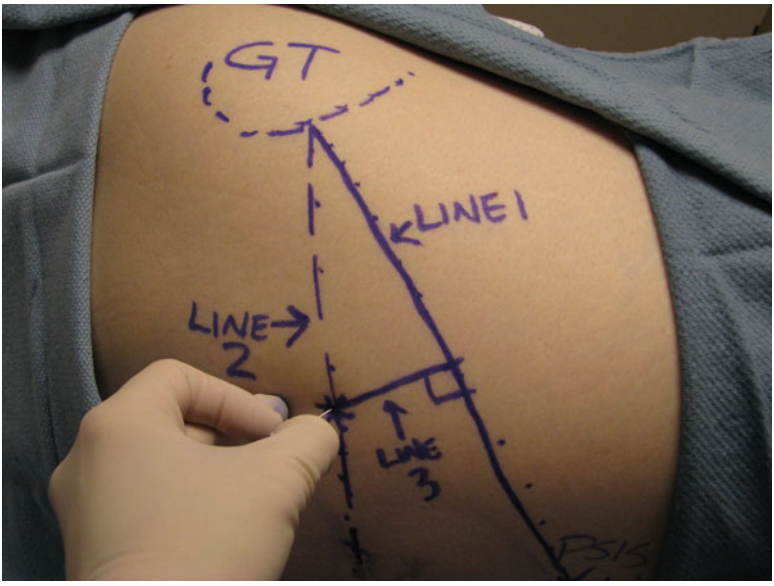

Fig. 14.1 Labat approach. The greater trochanter (GT), posterior superior iliac spine (PSIS), and sacral hiatus (SH) are identified. *Line 1* connects GT to PSIS. *Line 2* connects GT to SH. *Line 3* bisects and is perpendicular to *line 1* and is drawn caudad to intersect *line 2*. The needle is inserted where *line 3* intersects *line 2*

Draw a line between the greater trochanter and PSIS (line 1). Draw a second line from the sacral hiatus to the greater trochanter (line 2). Draw a perpendicular, caudad line from the midpoint of line 1 that intersects line 2. (This is line 3). The needle insertion points in where lines 2 and 3 intersect (see Fig. 14.1). Insert the needle perpendicular to all planes. Stimulation of the gluteus maximus muscle is often encountered just before the sciatic nerve stimulation.

Alternative
Instead of drawing line 2, you may draw a perpendicular and caudad line (equivalent to line 3) from the midpoint of line 1 that is 5 cm long. The needle is inserted at the end of this line perpendicular to all planes.

Tips

- Hamstring muscle stimulation: redirect the needle tip laterally.
- Piriformis muscle stimulation is very painful to the patient and is deep to the nerve.

2. *Parasacral Approach (Sacral Plexus Block)*. The most proximal approach to the sciatic nerve at the level of the sacral plexus, the nerves are targeted in the greater sciatic foramen. Stimulating the superior gluteal nerve (gluteus medius/minimus or tensor fascia lata), inferior gluteal (gluteus maximus) or sciatic (peroneal, tibial) nerve is acceptable at this level. The posterior femoral cutaneous nerve is

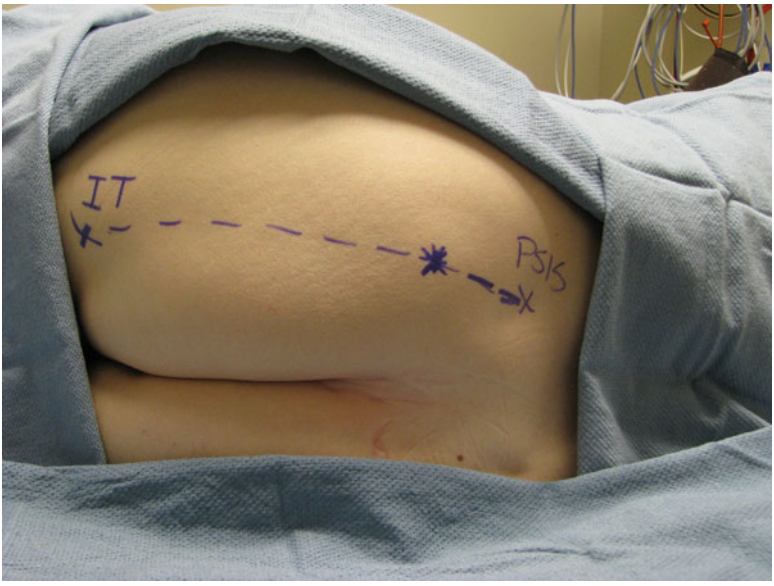

Fig. 14.2 Parasacral approach. Posterior superior iliac spine (PSIS) and ischial tuberosity (IT) are identified. A line is drawn between PSIS and IT. The needle insertion point is 7–8 cm caudal to the PSIS along this line

purely sensory. Superior gluteal nerve stimulation is preferred for patients undergoing hip surgery (innervates hip capsule). Unlike other sciatic nerve blocks, the parasacral approach also will block the obturator nerve [4].

Landmarks

- PSIS.
- Ischial tuberosity.
  Draw a line connecting the PSIS to the ischial tuberosity. Insert the needle 7–8 cm caudal to the PSIS along this line, perpendicular to all planes (see Fig. 14.2). The nerve is usually 6–8 cm below the skin.

  Ultrasound
  Scan the region caudal to the posterior superior iliac spine with a low-frequency probe and identify to the ischial bone and the lateral border of the sacrum. (These define the greater sciatic foramen). The sacral plexus appears as a hyperechoic bundle (see Fig. 14.3). Visualization may be improved by aiming at the greater trochanter.

Tips

- If os is contacted, "walk off" caudally and laterally until the needle advances through the sciatic foramen. If you continue to touch os, reexamine landmarks.
- The needle insertion point should be approximately at the level of the intergluteal cleft (see Fig. 14.2).

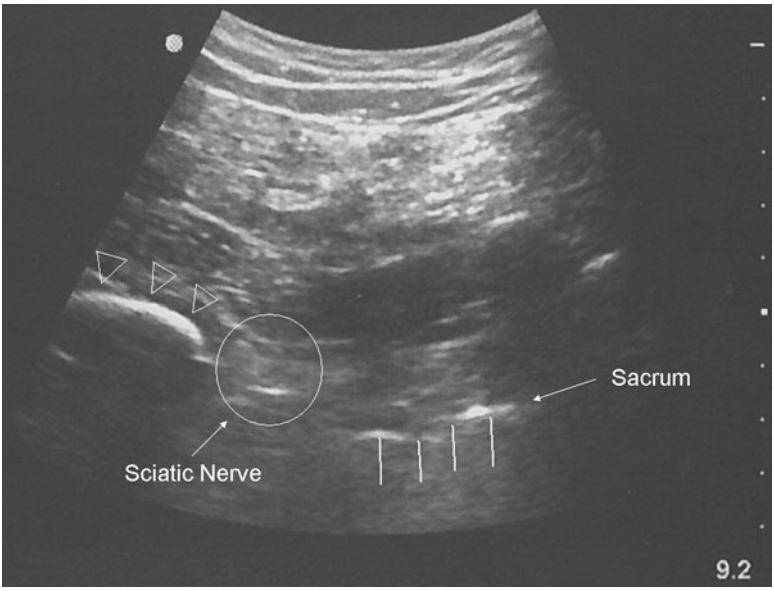

Fig. 14.3 Ultrasound-guided parasacral approach. *Arrow heads* identify the ischial bone (IB) and *straight lines* identify the sacrum. The sacral plexus is marked by a *circle*

3. *Mid-gluteal (Carlo Franco) Approach*.

   Landmarks

   - Gluteal Crease.
     Identify the midpoint of the gluteal crease. Draw a line perpendicular to the crease 10 cm in length. The insertion point is at the 10 cm mark, perpendicular to all planes.

4. *Gluteal and Subgluteal Approach*.

   Landmarks

   - Greater trochanter.
   - Ischial tuberosity.
     Gluteal: Draw a line connecting these landmarks and identify the midpoint of this line. Insert the needle perpendicular to all planes (see Fig. 14.4).

     Ultrasound
     If using an ultrasound, identify the greater trochanter and ischial tuberosity under ultrasound. The nerve will be located between these and may have a "flat" appearance (see Fig. 14.5). Insert the needle from the lateral aspect of the probe (near the greater trochanter).

     Subgluteal: Draw a line connecting these landmarks and, at the midpoint, draw a 3- to 5-cm perpendicular line in the caudad direction (see Fig. 14.4). Insert the needle perpendicular to all planes.

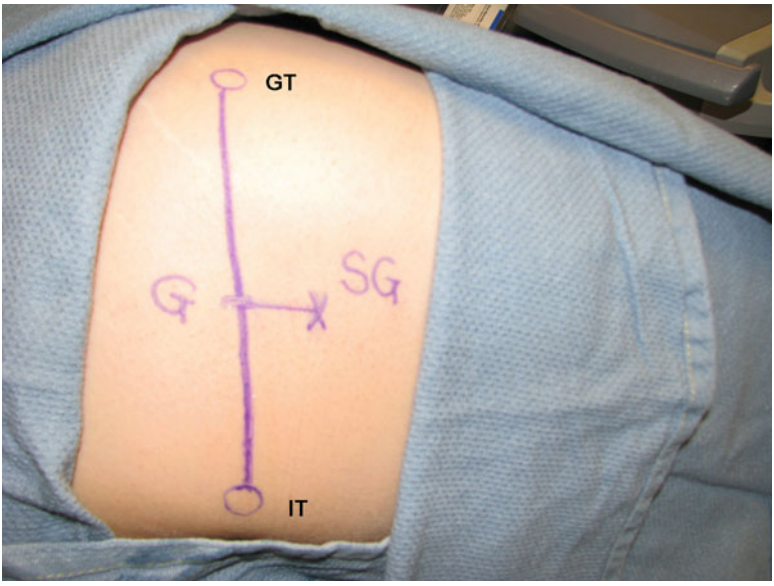

Fig. 14.4 Gluteal and subgluteal approach. The patient is positioned laterally (superior = lateral; inferior = medial). The *lateral circle* marks the greater trochanter (GT), and the *medial circle marks* the ischial tuberosity (IT). A line connects GT and IT. The midpoint of this line marks the insertion point for a gluteal (G) approach. Caudad 3–5 cm to the midpoint of the line is the insertion point for a subgluteal (SG) approach

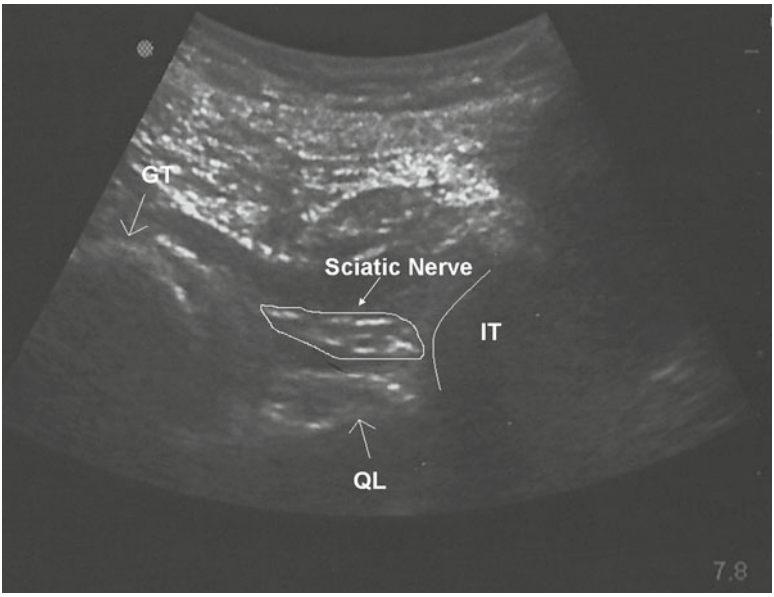

Fig. 14.5 Ultrasound-guided gluteal approach. The *arrow* on the left identifies the greater trochanter. The *arrow* at the bottom identifies the quadratus lumborum. The *curve line* on the right identifies the ischial tuberosity. The sciatic nerve is outlined by a *white line* and lies in the middle of the other structures

Tips

- Look or palpate for a longitudinal groove that runs along the posterior thigh starting at the bottom of the buttocks. This groove represents the origin of the semitendinosus from the ischial tuberosity and the biceps femoris long head. It is the path of the sciatic nerve.
- The subgluteal approach is beneficial due to the superficial position of the sciatic nerve. This approach will also not cause direct muscle stimulation of the gluteus maximus which may be painful for some patients.
- In the gluteal approach, stimulation of the gluteus maximus gives you an approximate depth of needle insertion. The sciatic nerve is 1–2 cm deeper than the gluteus maximus.

Supine Approaches

Use these approaches when it is not possible to move patients out of the supine position. Position the patient completely supine. A pillow may be placed behind the head if needed.

1. *Raj Approach*. Known as the lithotomy approach, the patient remains supine with the hip and knee flexed at 90° with the foot resting on a table or held by an assistant.

   Landmarks

   - Greater trochanter.
   - Ischial tuberosity.
     Draw a line connecting these landmarks and identify the midpoint of this line. Insert the needle perpendicular to all planes (see Fig. 14.6).

2. *Anterior Approach*. In this approach, the sciatic nerve is blocked just beyond the hip joint (posterior cutaneous nerve of the thigh will likely not be blocked). The depth of insertion is also commonly greater than the depth for a posterior approach, and a longer needle may be required. Externally rotate the patient's leg if possible [5, 6].

   Landmarks

   - Pubic tubercle (PT).
   - Anterior superior iliac spine (ASIS).
     Identify the ASIS and PT and connect with a line, which represents the inguinal ligament. Bisect the initial line and extend a perpendicular line 8 cm caudad (see Fig. 14.7). Insert the needle perpendicular to the skin in a slight lateral direction. The needle will likely strike close to the medial edge of the femur at approximately the level of the greater trochanter. Remove the needle slightly and redirect the tip more medially (needle change to a more vertical orientation) and advance the needle. Repeat this until the needle "walks off" the medial edge of the femur. This approach has a high risk of vascular puncture, and aspiration for intravascular placement is particularly important.

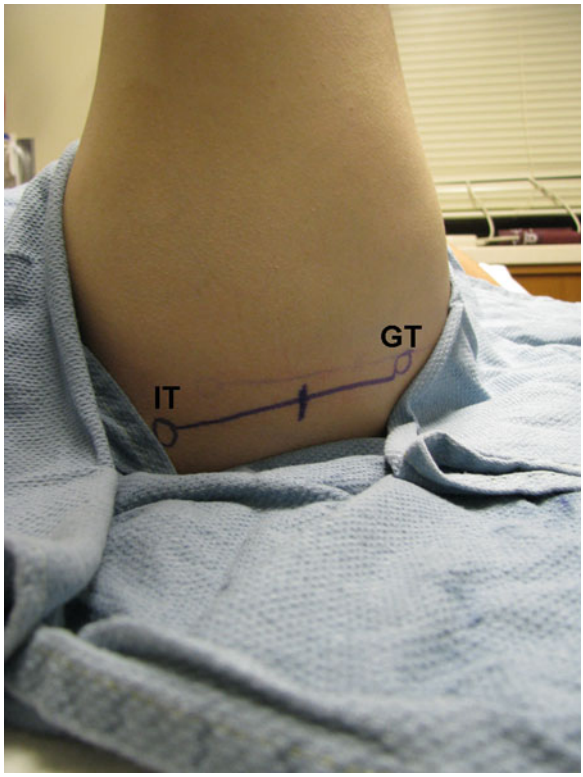

Fig. 14.6 Raj approach. The *lateral circle* marks the greater trochanter, and the *medial circle* marks the ischial tuberosity. The middle of the line connecting these points marks the needle insertion point

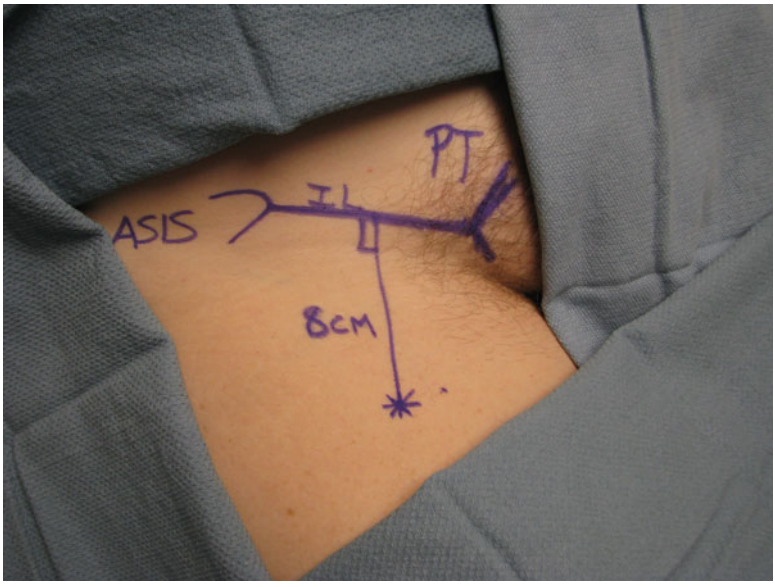

Fig. 14.7 Anterior approach. The pubic tubercle (PT) and anterior superior iliac spine (ASIS) are connected by a *line* representing the inguinal ligament. At the midpoint of this line, a perpendicular 8 cm line is drawn caudally. The insertion point is at the end of this line

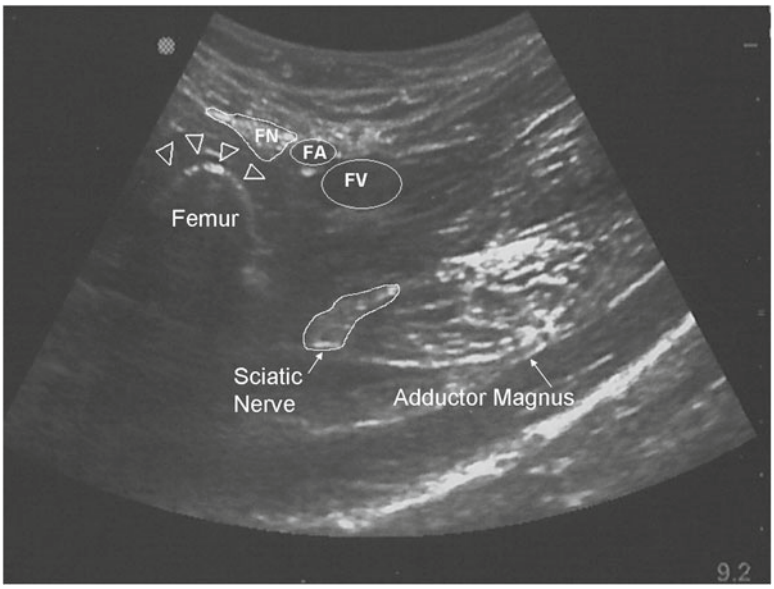

Fig. 14.8 Ultrasound-guided anterior-medial approach. The femur is marked by four *arrow heads*, while the femoral nerve and vessels are *outlined superficially*: nerve (FN), artery (FA), and vein (FV). The sciatic nerve is identified and outlined medial and dorsal to the femur and lateral to the adductor magnus (*left* is lateral and *right* is medial)

Ultrasound
Ultrasound may facilitate this block greatly since there is no need to palpate bony structures and allow visualization of femoral vessels. The nerve will be lateral to the femur and 2 cm dorsal. The needle may be inserted medial or lateral to the probe, but a medial insertion with help avoid puncture of the femoral vessels. The nerve will be located between the femur and the adductor magnus muscle (see Fig. 14.8).

Tips

- Once bone is identified, note this depth. The final depth of the sciatic nerve should be an additional 5 cm or less past this depth.
- Not successful: insert needle 1–2 cm medial to the original insertion site. (This allows the needle to pass the femur's medial edge at a greater angle and directs the tip posterior to the femur).

3. *Popliteal Lateral Approach*. ideal for surgeries of the lower leg, foot, and ankle, this approach preserves the function of the hamstrings. To help avoid an incomplete block, a larger volume of local anesthetic (30–40 ml) is often used, but volume may be decreased and success rate increased with US guidance or a two injection technique [7].

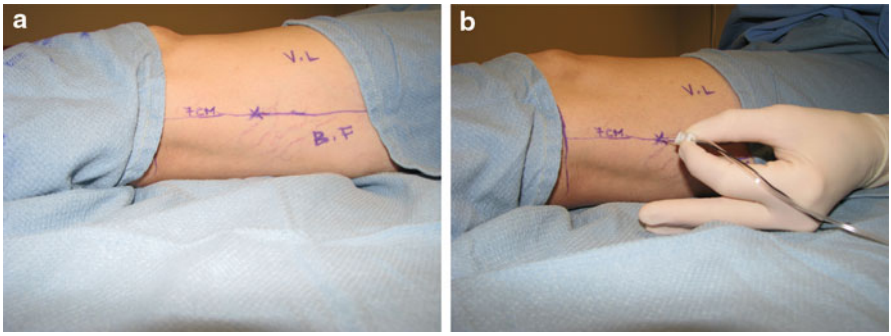

Fig. 14.9 Lateral popliteal approach. (**a**) The vastus lateralis (VL) and biceps femoris (BF) muscles are identified. (**b**) The popliteal crease is identified, and the needle insertion point is marked 7 cm cephalad

Landmarks

- Vastus lateralis muscle.
- Biceps femoris muscle.
- Head of fibula.
- Superior border of patella.

Identify the vastus lateralis and biceps femoris muscles (see Fig. 14.9a). Draw a line demarcating the space between the two muscles. Identify the popliteal crease. The insertion point is 7–10 cm cephalad to the popliteal crease along the original line (see Fig. 14.9b). A cephalad insertion point increases the likelihood that the peroneal and tibial components are in close proximity and is more likely to result in a successful block [6, 8, 9].

Alternative Approach.
Identify the head of the fibula. Draw a line cephalad, parallel to the floor/bed. Identify the superior border of the patella and draw a line perpendicular to the floor. The intersection of these lines marks the insertion point. Insert the needle perpendicular to the skin and parallel to the floor. First, the more lateral peroneal nerve will be stimulated. Note this depth of needle insertion and advance the needle until a tibial response is illicited after depositing two thirds of the anesthetic around the tibial nerve, withdraw the needle to the peroneal depth noted earlier, and deposit the remaining local anesthetic [10].

Ultrasound
Scan over the popliteal fossa and identify the popliteal artery (deep) and tibia nerve (superficial). Follow the nerve cephalad and slightly lateral until a second nerve (peroneal nerve) is observed to join the first. The nerve block should be performed where the two nerves are in close proximity (see Fig. 14.10) or appear to combine. The needle is inserted in the lateral thigh approximately 2 cm deep to the skin so that the needle is parallel to the probe.

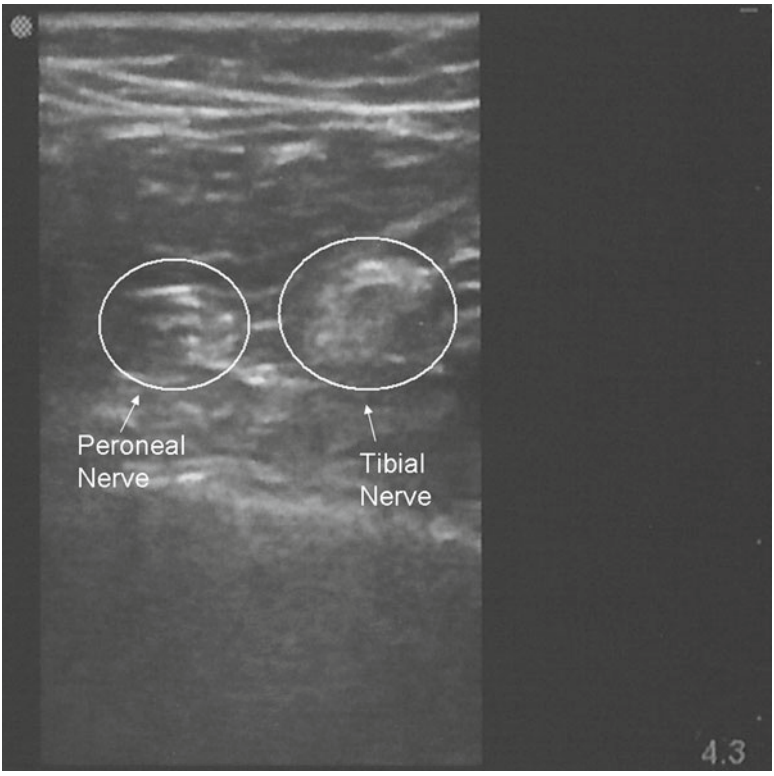

Fig. 14.10 Ultrasound-guided popliteal approach. The common peroneal nerve (lateral) and tibial nerve (medial) are identified in close approximation (*left* is lateral and *right* is medial)

Tips

- If the femoral shaft is contacted, withdraw the needle to the skin and redirect 30° posteriorly/dorsal to the initial insertion plane. The nerve should be 1–1.5 cm deeper than the distance to the femur.

Prone Approaches

When not contraindicated by fractures, the prone position allows full access to the target area. Place the patient prone with a folded blanket or pillow below the lower leg of the operative lower extremity. The operative lower extremity should be slightly bent at the knee, the lower leg supported, and the foot should rests freely above the bed.

1. *Popliteal Prone Approach.*

   Landmarks

   - Semimembranosus muscle (medial).
   - Biceps femoris muscle (lateral).

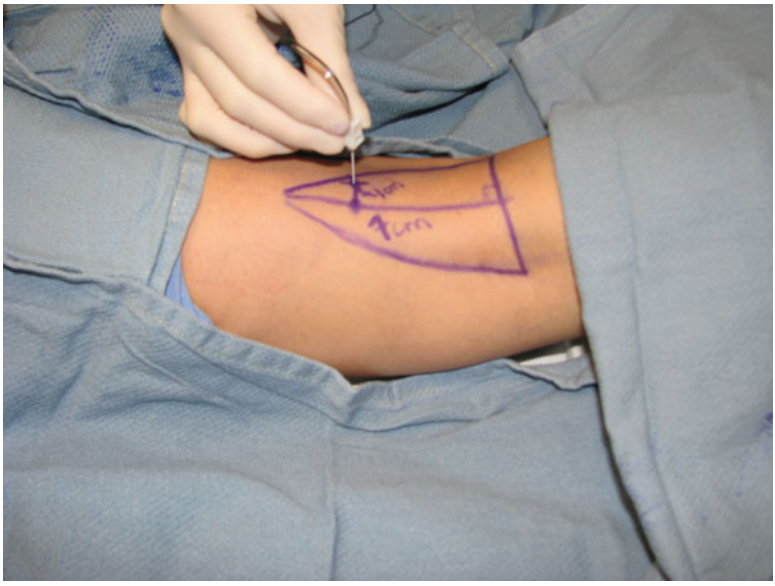

Fig. 14.11 Prone popliteal approach. Semimembranosus muscle (medial), biceps femoris muscle (lateral), and popliteal crease (inferior) are marked. The insertion point is 7 cm cephalad and 1 cm lateral

- Popliteal crease (inferior).
  Identify and mark the medial, lateral, and inferior borders of the popliteal triangle. Needle insertion should be 7–10 cm cephalad and 1 cm lateral to the midline of the popliteal crease (see Fig. 14.11). Insert the needle in a cephalad direction at a 45–60° angle to the skin.

  Ultrasound
  Start by scanning the patient at the popliteal crease and identify the popliteal artery and tibial nerve (superficial to the artery). Trace the tibial nerve proximally until the peroneal structures are visualized in close approximation (see Fig. 14.10). Insert the needle medial or lateral to the probe.

Tips

- Unable to visualize landmarks: have the patient bend the knee against resistance.
- No nerve stimulation with needle insertion: redirect the needle tip 1 cm lateral to the initial insertion site.
- Stimulation of biceps femoris muscle: redirect the needle tip slightly medial. (Note that more medial insertion increases the risk of inadvertent vascular puncture).

Lumbar Plexus

Introduction and Anatomy

The lumbar plexus originates from the ventral rami of L1–4 (variable contributions from T12 and L5), forms within the body of the psoas muscle, and supplies the lower abdomen and upper leg. It consists of six peripheral nerves: femoral, obturator, lateral femoral cutaneous, ilioinguinal, genitofemoral, and iliohypogastric nerves. Consequently, a lumbar plexus block consistently blocks the three nerves supplying the lower extremity (femoral, obturator, and lateral femoral cutaneous nerves). The lumbar plexus block remains controversial because of the deep location of the plexus within the psoas muscle and the potential for bleeding into the retroperitoneum, a noncompressible area. Contacting and identifying the L4 transverse process before entering the plexus is very important in a lumbar plexus bock. This landmark serves as a needle depth safety point that should prevent advancing the needle too deep into the retroperitoneum [11–14].

Procedure

Needle: 10 cm insulated needle (15 cm needle may be needed for obese patients).

Position: Place the patient to a lateral position (Sim's position) with the nonoperative down, operative side up.

Landmarks

- Posterior iliac crest.
- Spinous processes (midline).

Draw a line from the top of the posterior iliac crest to midline. This line (intercristal line) is positioned over the L4 transverse process. Mark 4–5 cm lateral to midline on the operative side (approximately as lateral as the PSIS from the midline) on the intercristal line (see Fig. 14.12).

Insert the block needle perpendicular to all planes with the bevel pointed superior or lateral and monitor for a quadriceps muscle twitch (patellar snap). This twitch indicates stimulation of the femoral nerve and proximity to the lumbar plexus. If the L4 transverse process is not contacted 5–6 cm deep to the skin and stimulation of the quadriceps has not been noted, make small adjustments cephalad or caudad in attempt to contact the transverse process. The needle may need to be advanced further in more obese patients. Once os has been contacted and the L4 transverse process identified, withdraw the needle 1–2 cm and redirect the tip 15° cephalad/caudal

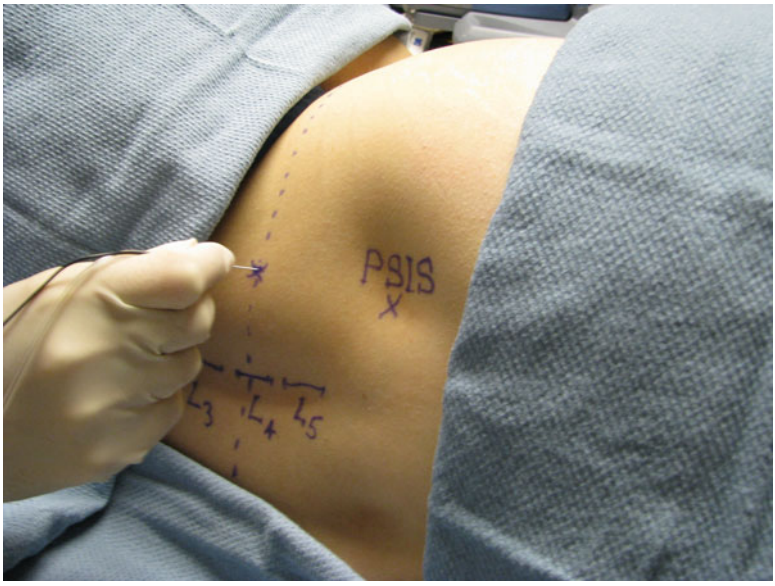

Fig. 14.12 Lumbar plexus nerve block. The intercristal line (*dashed line*) is identified using the posterior iliac crest. The needle insertion site is 4–5 cm from midline and is approximately as lateral as the patient's posterior superior iliac spine (PSIS)

with the goal of placing the tip just superior/inferior to the L4 transverse process. The plexus should be stimulated 2 cm or less beyond the transverse process. Make sure to check for CSF as well as blood when aspirating before injection [11–14].

Alternative: Loss of Resistance Technique

Contact the transverse process as above. Flush the needle with 1 ml (remove possible tissue plug) and attach loss of resistance (LOR) syringe. Advance needle checking for LOR. (This is more subtle than the typical LOR found with epidurals). This is a fascial plane block (inserts local anesthetic between the quadratus lumborum and psoas muscles) and is volume dependent. Consequently, catheters may need increased rates.

Tips

- Unable to find transverse process: remove needle and reinsert 4 cm lateral to midline but perpendicular to all planes. (Do not deviate the needle tip medially as this will increase the risk of epidural or subarachnoid block).
- No stimulation with repeated attempts: change to loss or resistance technique.
- Stimulation of hamstrings indicates stimulation of the sacral plexus (do not accept this twitch). Needle is too caudal and/or medial. Redirect lateral and/or

cephalad. (Injection with stimulation of the sciatic nerve may increase the risk of retrograde spread of the local anesthetic into the epidural space [12]).
- Stimulation of adductors indicates obturator nerve stimulation. The needle is likely too medial and should be directed laterally.
- Os is repeatedly encountered: needle is likely hitting the posterior pelvis and has been inserted too caudad at L5 level or lower. Remove needle, recheck landmarks (likely reinsert needle at more cephalad location).
- Always insert with needle tip lateral or cephalad (never medial). This will decrease the chance of diffusion into the epidural space and decrease the incidence of epidural catheter placement with continuous techniques.
- Place the dressing as far lateral as possible when placing a lumbar plexus catheter. This will leave the midline uncovered and allow for a subarachnoid block if so desired for surgical anesthesia.

Complications

Complications specific to lumbar plexus blocks include epidural injection or diffusion (most common complication), intrathecal injections, intravascular injection, and retroperitoneal bleeding (especially in patients on therapeutic anticoagulants).

Femoral Nerve Block

Introduction and Anatomy

Derived from L2–4, the femoral nerve is the largest branch of the lumbar plexus. After traveling through the psoas muscle, the nerve passes anterior to the iliopsoas, under the inguinal ligament, and becomes superficial in the anterior thigh deep to the fascia lata and fascia iliacus. Here, it is separated from the femoral artery and vein by the fascia iliacus.

Femoral nerve blockade provides analgesia to the anterior thigh, anterior knee and medial leg, and ankle and foot. A femoral nerve block has been referred to as the "3-in-1" block inferring that the femoral, lateral femoral cutaneous, and obturator nerves could all be blocked by one injection [15, 16]. However, studies have demonstrated that the obturator nerve is often missed with this technique [17–20].

Procedure

Needle: 5 cm insulated needle.

Position: position the patient supine. A pillow may be placed under the head, if needed. Retract the patient's pannus with tape as needed (do not cover the insertion site).

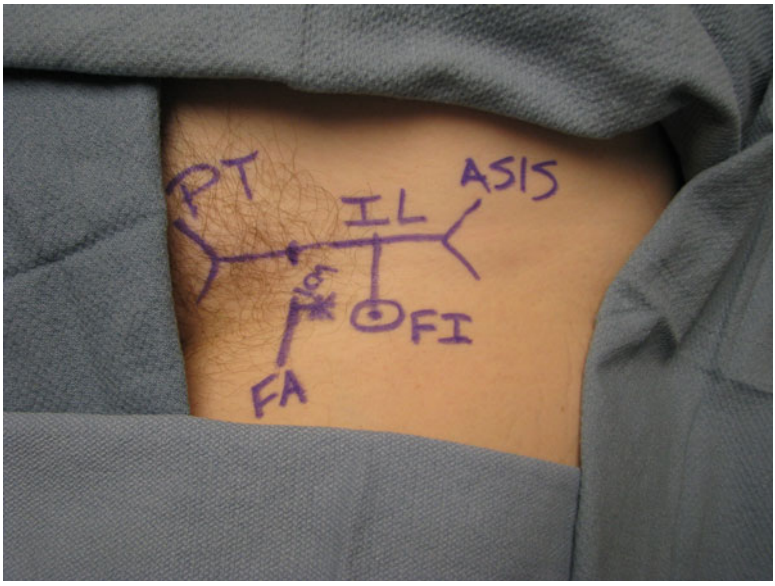

Fig. 14.13 Femoral nerve block. A *line* connects the pubic tubercle (PT) to the anterior superior iliac spine (ASIS) and identifies the inguinal ligament (IL). The femoral artery (FA) is palpated and marked. The needle insertion site is 1 cm lateral FA

Landmarks

- Inguinal ligament.
- Pubic tubercle.
- Anterior superior iliac spine.
- Femoral artery.

Identify the inguinal ligament by drawing a line from the pubic tubercle to the anterior superior iliac spine. Palpate the femoral artery just distal to the ligament (see Fig. 14.13). Insert the needle 1–1.5 cm lateral to the femoral artery. (Remember that the nerve is lateral to the artery with the mnemonic NAVELS: Nerve, Artery, Vein, Empty space, Lymphatics, and Symphysis). Insert the block needle in a cephalad direction at a 45–60° angle to the skin and monitor for an evoked response of the rectus femoris (patellar snap).

Ultrasound
Identify the femoral vessel and nerves (the femoral nerve is lateral to vessels; see Fig. 14.14). Clinicians may insert the needle for in-plane (preferred by author) or out-of-plane approach.

Tips

- Stimulation of the medial thigh occurs: redirect tip laterally. [Do not accept this evoked motor response (likely a superficial branch or direct vastus medialis or sartorius muscle stimulation)].

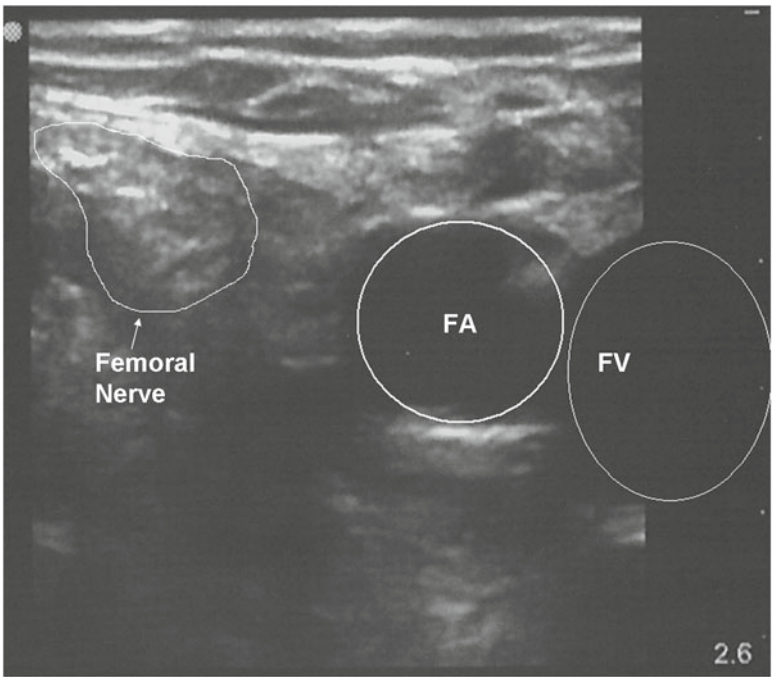

Fig. 14.14 Ultrasound-guided femoral nerve block. Lateral to medial, the femoral nerve (FN), artery (FA), and vein (FV) are identified (*left* is lateral and *right* is medial)

- Do not direct your tip medially; you will increase the risk of vascular puncture. If you think you are too lateral, reinsert the tip 0.5 cm medially but maintain the cephalad direction with insertion.
- You may note two "pops" with needle insertion. This is due to penetration of fascia lata (superficial) and fascia iliaca.

Alternative: Fascia Iliaca Block

This technique is useful for patients on anticoagulation, patients with a recent vascular study or cardiac catheterization in the groin of the operative side, or patients with intense pain negating nerve stimulation. Needle placement is far from the vessels and nerves are not stimulated.

Identify the above landmarks except the femoral artery. Divide line on inguinal ligament into thirds. At the lateral mark (between lateral and middle thirds), draw a 1- to 2-cm line perpendicular to the original line (Fig. 14.15). Insert the needle at the bottom of this second line perpendicular to all planes. Feel for two "pops": fascia lata and fascia iliacus. The neural structures are present in this plan. Insert local anesthetic.

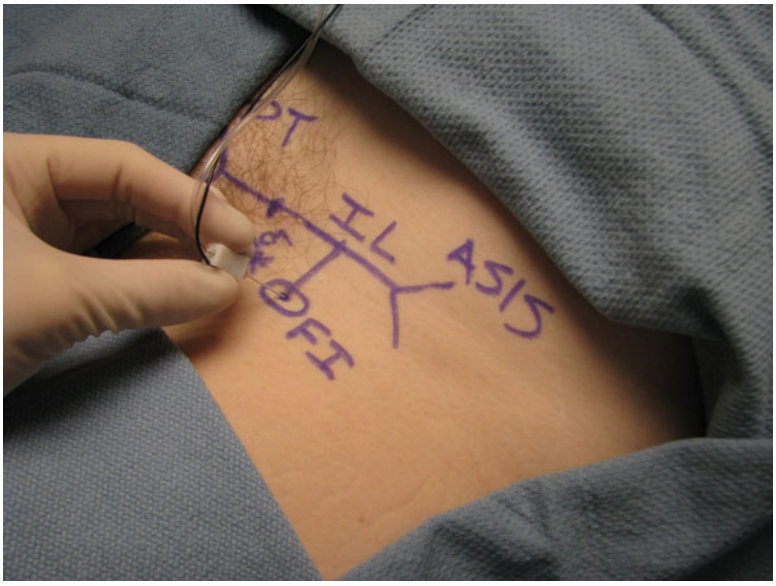

Fig. 14.15 Fascia iliaca block. The inguinal ligament is identified by drawing a line from the pubic tubercle to the anterior superior iliac spine. This line is divided into thirds. At the lateral mark (between lateral and middle thirds), a 1–2-cm line is drawn perpendicular to the original line. This marks the needle insertion site

Contraindications

A prosthetic femoral artery graft is a relative contraindication to femoral nerve block since it is difficult to know the orientation of the femoral nerve and native vessel to the palpable vascular graft. An ultrasound should be utilized in these patients in order to fully evaluate the altered vascular anatomy.

Questions

1. While performing a sciatic nerve block with nerve stimulation, you observe foot dorsiflexion. What part of the sciatic nerve are you stimulating?
2. Which sciatic nerve block(s) will also block the posterior femoral cutaneous nerve of the thigh?
3. Which sciatic nerve block will also block the obturator nerve?
4. While performing a popliteal nerve block from the lateral approach, you contact the femur. How should you redirect your needle?
5. What muscle contraction should be observed with stimulation of the lumbar plexus?

6. You are having difficulty placing a lumbar plexus block and change to a loss or resistance technique for a psoas compartment block. After you have found the transverse process, what should you do before attaching the loss of resistance syringe?
7. What nerve roots does the femoral nerve originate from?
8. Where is the femoral nerve anatomically located in relation to the femoral artery?
9. In a fascia iliaca block, what two fascial layers cause the two "pops"?
10. What is the number one contraindication to performing a nerve block?

Answers:

1. Common peroneal
2. Labat and parasacral
3. Parasacral
4. 30° Posterior/dorsal
5. Quadriceps (patellar "snap" due to femoral nerve stimulation)
6. Flush the needle with 1 ml of solution to clear any plug at the tip
7. Lumbar roots 2–4
8. Nerve is lateral to artery (NAVELS)
9. Fascia lata (superficial) and fascia iliaca (deep)
10. Patient refusal

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15

Regional Anesthetic Techniques for Foot Surgery

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Contents

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Introduction

Over the last decade, outpatient surgery has consistently gained in popularity by providing a significant reduction in the cost of hospitalization and the patient's length of stay. Foot and ankle surgery procedures are commonly performed in an outpatient setting [1]. Key issues in foot and ankle surgery include rising demand for outpatient procedures, managing postoperative pain and decreasing the use of opiates, and avoiding the side effects of general anesthesia in certain patient populations [2].

Foot and ankle surgeries produce moderate to severe postoperative pain that is sometimes difficult to control with oral pain medications alone [3]. Research has shown that regional anesthesia has been used successfully in foot and ankle surgeries to reduce postoperative pain [1], with one study reporting that regional anesthesia reduces perioperative opioid requirements [4]. Another study indicated that monitored intravenous sedation can be safely and effectively carried out together with regional anesthesia in foot surgeries. The article reported high patient satisfaction and reduction in postoperative pain using this combination [5]. Using monitored intravenous sedation instead of general anesthesia significantly reduces side effects, including nausea, vomiting, and throat discomfort. Intravenous sedation also reduces recovery time and avoids unwanted admission to the hospital [6]. The combination of regional anesthesia with monitored intravenous sedation also can be used in American Society of Anesthesiologists (ASA) 3 and 4 patients undergoing lower limb-preservation procedures without increasing their pulmonary or cardiac complications. This finding is significant because historically it has been assumed that ASA 3 and 4 patients needed to be under general anesthesia regardless of the surgical procedure due to the higher rate of complications associated with this patient population [7]. There are also other specific patient populations in which regional anesthesia may be a superior anesthetic technique. Patients with asthma, for example, benefit greatly from regional anesthesia because it avoids airway manipulation [1].

Despite the numerous benefits of regional anesthesia reported in recent studies, there is some anecdotal evidence that performing regional anesthesia increases operating room time and delays turnovers [2]. However, with judicious preoperative timing and planning as well as skillful regional anesthesia administration, delayed turnover can be minimized. This chapter will discuss several common regional anesthesia techniques in foot surgeries and offer clinical pearls.

Anesthetic Agents

Lidocaine (Xylocaine), 1 and 2%, and bupivacaine (Marcaine), 0.25 and 0.5%, are commonly used agents in foot and ankle surgery. Lidocaine has a faster onset of action than bupivacaine, but bupivacaine has a longer duration of action than lidocaine [8]. Many surgeons would also use 1:1 mixtures of lidocaine and bupivacaine to take advantage of the faster onset of lidocaine and longer duration of bupivacaine.

The maximum safe dosage of lidocaine without epinephrine in a normal, healthy adult is 4.5 mg/kg, which is approximately 300 mg of lidocaine in a 70-kg adult. With the addition of epinephrine, the maximum dosage increases to approximately 500 mg. The maximum safe dosage of bupivacaine without epinephrine is 2.5 mg/kg or approximately 175 mg in a 70-kg adult. With the addition of epinephrine, the maximum dose becomes 300 mg [5]. Epinephrine (1:100,000) is frequently added to lidocaine and Marcaine to utilize its vasoconstriction properties and enhance the effects of the anesthesia. However, it is generally not recommended to use local anesthetics with epinephrine when injecting circumferentially around the ankle joint or toes due to the risk of causing tissue ischemia.

Ankle Block

Introduction

This block essentially involves the blocking of five nerves that innervate the foot: posterior tibial, sural, superficial peroneal, saphenous, and deep peroneal nerves. These nerves are the terminal branches of principle nerve trunks that innervate the proximal leg. Each nerve needs to be blocked individually in order to achieve complete anesthesia.

Anatomy

Posterior Tibial Nerve

This nerve is one of the two branches of the sciatic nerve. It courses down the posterior leg along with the posterior tibial artery and vein. At the ankle joint, it runs just posterior to the medial malleolus and gives off the medial calcaneal branch before it dives plantar medially toward the sole of the foot and divides into the medial and plantar nerves (Fig. 15.1). This nerve provides sensory innervations to the medial heel as well as the entire sole of the foot (Fig. 15.4).

Sural Nerve

This nerve arises from both the posterior tibial and common peroneal nerves. It courses down the posterior lower leg with the small saphenous vein and gives off the lateral calcaneal branch before it curves posterior and inferior to the lateral malleolus; it then runs along the lateral side of the foot before it becomes one of the digital nerves of the fifth toe (Fig. 15.1). This nerve provides sensory innervations to the area of the lateral malleolus and lateral heel, as well as to the lateral side of the foot (Fig. 15.2).

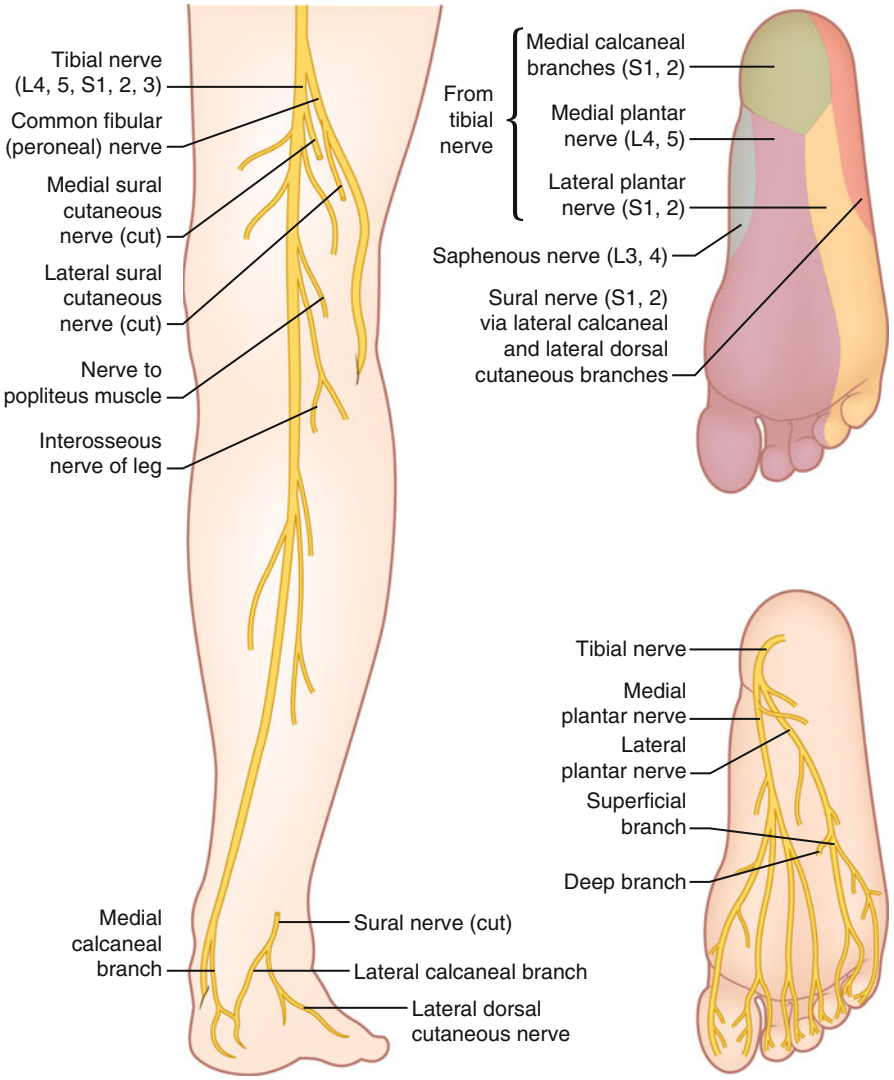

Fig. 15.1 Posterior and plantar sensory nerve distributions and innervations of the sole

Superficial Peroneal Nerve

This nerve arises from the common peroneal nerve as it wraps around the fibular head. It courses down the lateral compartment of the lower leg and pierces the deep fascia at the level of the ankle and divides into the medial and intermediate dorsal cutaneous nerves (Fig. 15.1). This nerve provides sensory innervation to the central dorsum area of the foot (Fig. 15.3).

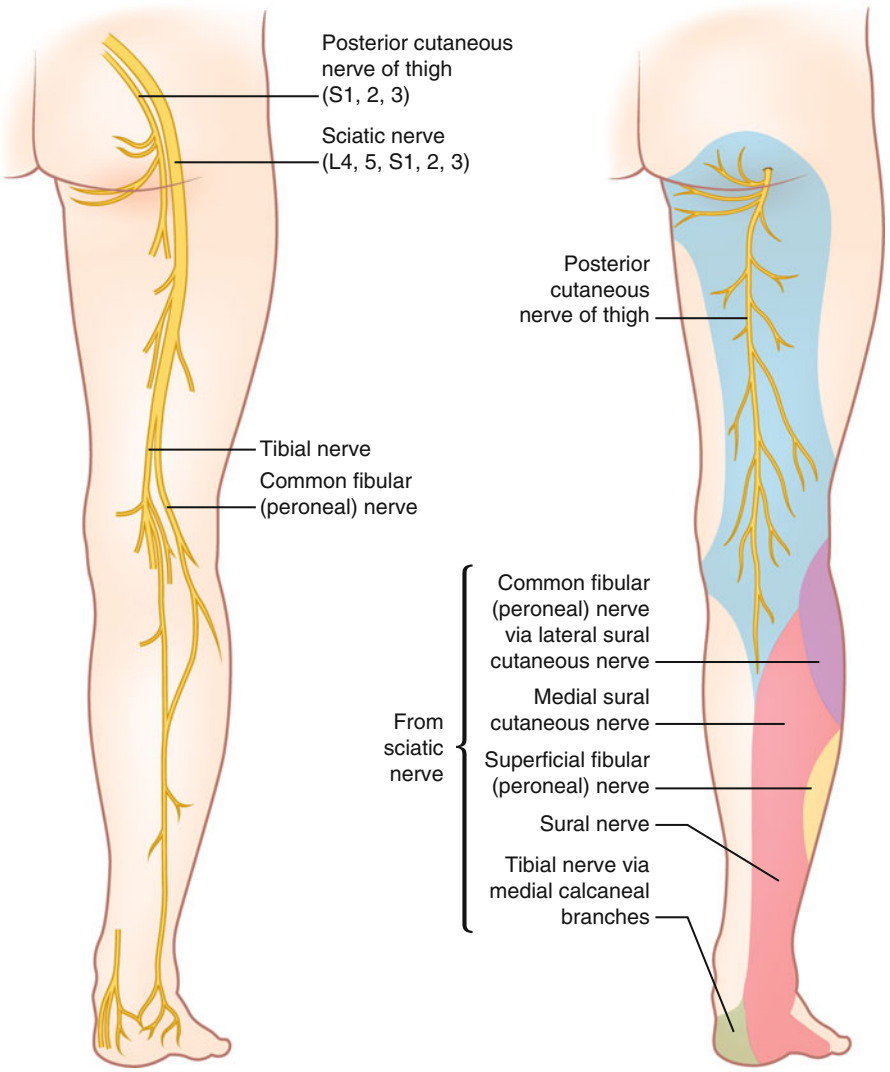

Fig. 15.2 Posterior leg sensory nerve distribution and innervations of heel

Saphenous Nerve

This nerve arises from the femoral nerve. It exits the adductor canal and courses down the medial side of the leg along with the great saphenous vein. At the level of the ankle, it runs anterior to the medial malleolus and ends at the medial side of the midfoot (Fig. 15.1). This nerve provides the sensory innervation of the medial malleolus (Fig. 15.4).

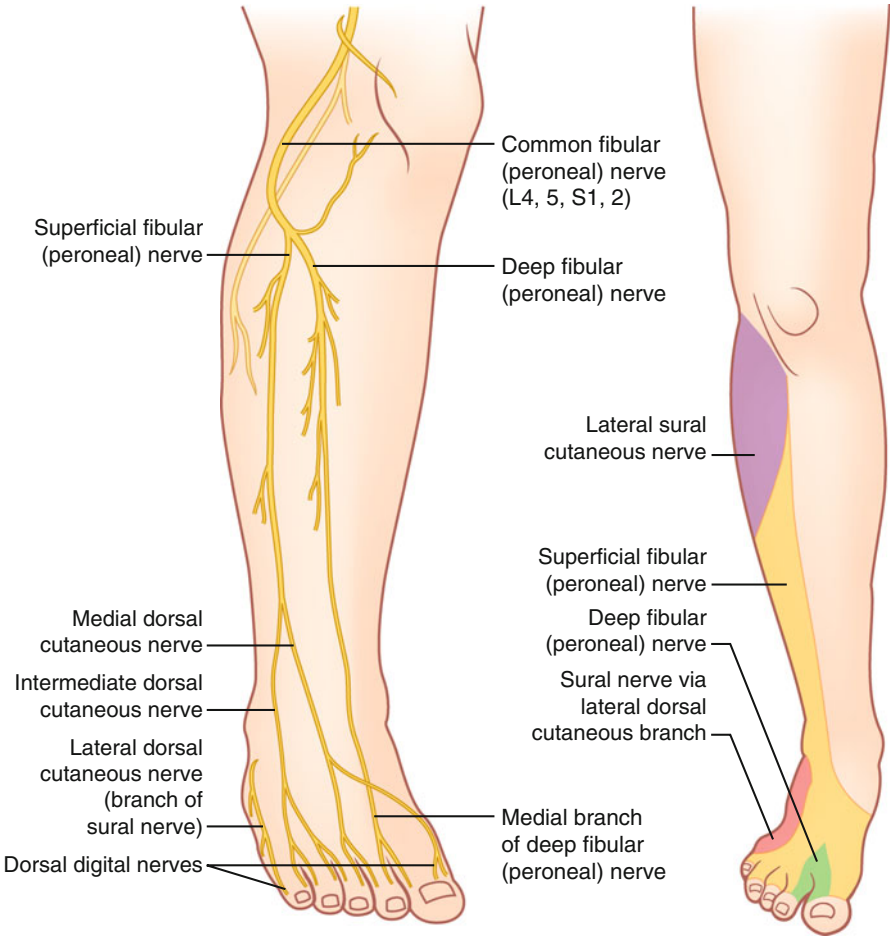

Fig. 15.3 Anterior leg sensory nerve distribution and innervations

Deep Peroneal Nerve

This nerve arises from the common peroneal nerve after it wraps around the fibular head. It courses deep down the anterior compartment of the lower leg and dorsum foot. This nerve provides mostly motor innervations of the anterior compartment muscles of the lower leg. The only sensory innervation the deep peroneal nerve provides is at the first interdigital space (Fig. 15.4).

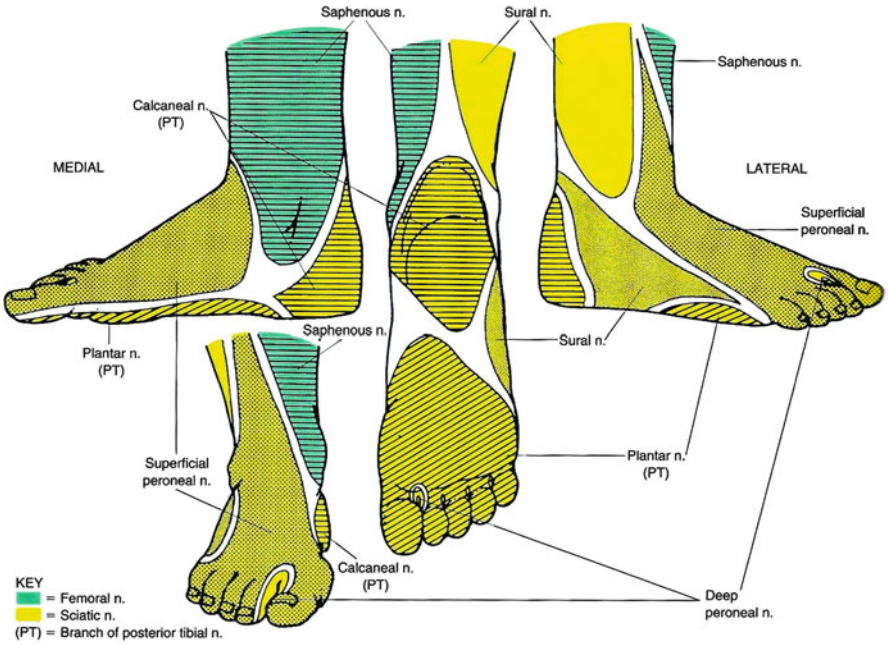

Fig. 15.4 Sensory nerve innervations of the foot and ankle with permission [1]

Indications

- All surgical procedures of the foot
- Providing postoperative pain relief in both adults and children
- Supplemental anesthesia to incomplete proximal regional nerve block, including popliteal or sciatic block

Procedure

Posterior Tibial Nerve Block

Positioning: Prone with pillow under the ankle

Landmarks:

- Medial malleolus
- Achilles tendon
- Posterior tibial artery

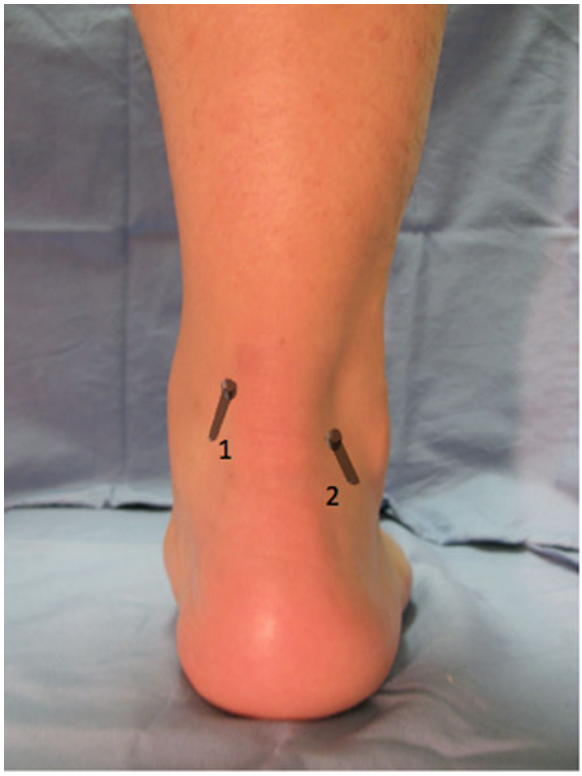

Fig. 15.5 Posterior view of the foot and ankle. *Arrow 1* shows the direction of needle insertion for anesthetizing the posterior tibial nerve. *Arrow 2* shows the direction of needle insertion for anesthetizing the sural nerve

Injection Technique: The point of needle insertion is approximately two fingerbreadths posterior to the medial malleolus and 1–1.5 cm anterior to the Achilles tendon. The pulse of the posterior tibial artery is palpated, and a 25-gauge, 1.5-in. -long needle is inserted just posterior to the palpated pulse angle toward the medial malleolus. Advance the needle until the posterior side of the medial malleolus is encountered. Withdraw the needle about 2–3 mm and aspirate to ensure the needle tip is not inside the artery. Inject 5–8 ml of local anesthetic in a single spot (Figs. 15.5 and 15.6).

Sural Nerve Block

Positioning: Supine with leg internally rotated

Landmarks:

- Lateral malleolus
- Achilles tendon

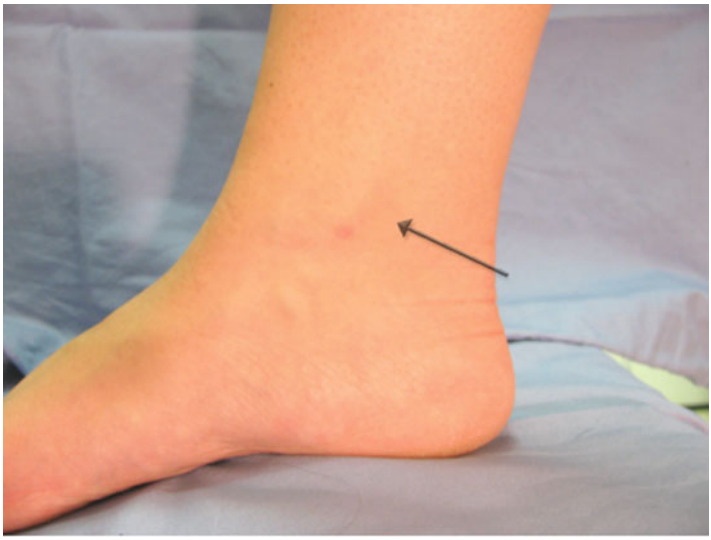

Fig. 15.6 The *arrow* shows the direction of the needle insertion in posterior tibial nerve block from the lateral view

Injection Technique: The point of needle insertion is just anterior to the Achilles tendon and 2–3 cm above the lateral malleolus. A 25-gauge, 1.5-in.-long needle is inserted, and a small wheal is raised after a negative aspiration test. Advance the needle toward the fibula and approximately 5 ml of local anesthetic is injected subcutaneously (Fig. 15.5).

Superficial Peroneal and Saphenous Nerve Blocks

Positioning: Supine with leg in neutral position

Landmarks:

- Medial and lateral malleoli

Injection Technique: Insert a 25-gauge, 1.5-in.-long needle at the anterior border of the lateral malleolus and 2–3 cm above the ankle joint. After a small wheal is raised and a negative aspiration test, advance the needle across the anterior ankle until the anterior border of the medial malleolus is reached. Inject 5–10 ml of local anesthetic subcutaneously while advancing the needle. The needle might need to be withdrawn and reinserted in order to reach across the entire area. Perform an aspiration test each time the needle is reinserted to ensure the local anesthetic is not being injected into a blood vessel (Fig. 15.7).

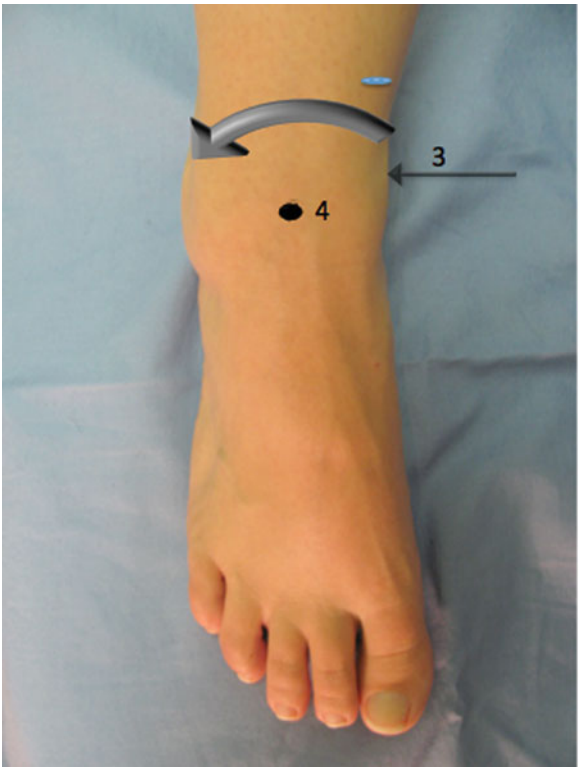

Fig. 15.7 Anterior view of the foot and ankle. *Arrow 3* shows the place of needle insertion in superficial peroneal nerve block. The *gray arrow* indicates the direction of needle advancement. *Arrow 4* shows the direction of needle insertion in deep peroneal nerve block. Note the needle is perpendicular to the coronal plane

Deep Peroneal Nerve Block

Positioning: Supine with the leg in neutral position

Landmarks:

- Dorsalis pedis artery
- Extensor hallucis longus tendon
- Medial and lateral malleoli

Injection Technique: At the level of the malleoli, identify the extensor hallucis longus tendon by dorsiflexing and plantarflexing the hallux. Palpate the pulse of the dorsalis pedis artery just lateral to the tendon. Insert a 25-gauge, 1.5-in.-long needle just lateral to the palpated artery pulse and raise a small wheal after a negative aspiration test. Advance the needle deep through the deep fascia and inject 2–3 ml of local anesthetic (Fig. 15.7).

Mayo Block

Introduction

Essentially, this is a field block for surgeries involving the first metatarsal phalangeal joints as well as any hallux-related surgeries. This technique allows the surgeon to use less local anesthetic while still achieving the desired level of anesthesia to perform the surgery. This field block can also be used in the fifth toe and ray surgeries and is termed "reverse Mayo block."

Anatomy

The hallux is innervated by four nerves: one on each side of the hallux dorsally as well as plantarly. The dorsal medial nerve is one of the terminal branches from the medial dorsal cutaneous nerve. The plantar nerves arise from the medial plantar nerve. The lateral dorsal nerve is the sensory branch of the deep peroneal nerve as it innervates the first interdigital space. The medial dorsal nerve arises from the medial dorsal cutaneous nerve (Figs. 15.1 and 15.3).

Indications

- All surgeries involving the first metatarsal phalangeal joints, and hallux, including bunionectomy, amputation, and joint arthrodesis.

Procedure

Landmarks:

- First metatarsal phalangeal joint
- First intermetatarsal space

Positioning: Supine

Injection Technique: Insert the needle medially proximal to the first metatarsal phalangeal joint and approximately two-thirds down the metatarsal shaft. Direct the needle laterally and inject local anesthetic as it crosses the first metatarsal dorsally. The needle is then withdrawn and redirected plantarly. First, advance the needle laterally and inject local anesthetic subcutaneously just below the skin.

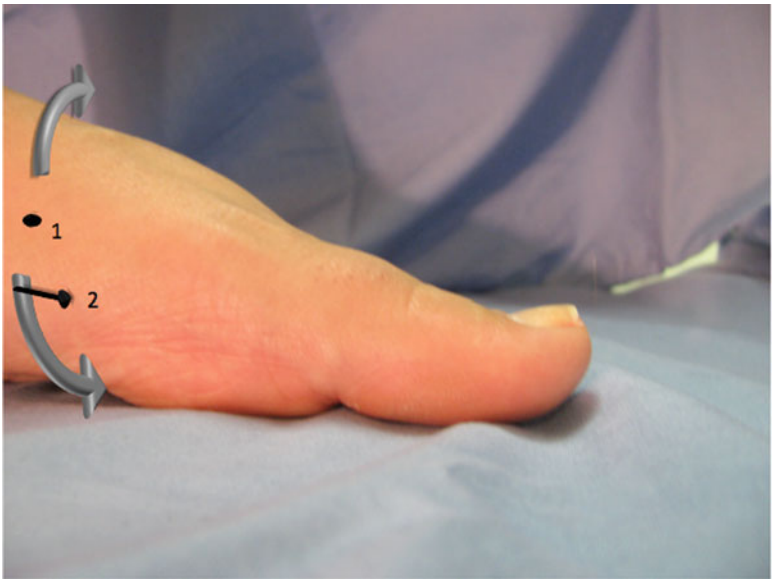

Fig. 15.8 Lateral view of the forefoot. *Arrow 1* shows the direction the needle insertion for anesthetizing the dorsal and plantar nerves around the first ray. Note the needle is perpendicular to the sagittal plane. The *gray arrows* show the direction of needle advancements. *Arrow 2* shows the needle insertion for anesthetizing the deep branch of the plantar nerve

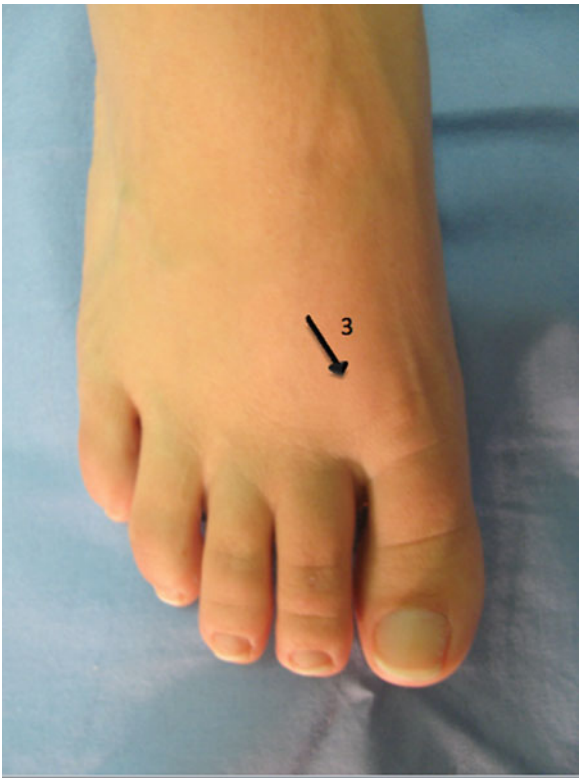

Fig. 15.9 AP view of the forefoot. *Arrow 3* shows the placement of needle for anesthetizing the sensory branch of the deep peroneal nerve

Second, withdraw the needle and then direct it deep just below the first metatarsal and inject local anesthetic as the needle advances laterally. Additional local anesthetic is then deposited distally at the first interdigital space (Figs. 15.8 and 15.9).

Digital Block

Introduction

This is a field block technique utilized when performing toe surgeries. This technique can be repeated to anesthetize multiple toes. This block can also be performed more proximally to include the metatarsals.

Anatomy

Each toe is innervated by four nerves: two dorsal proper digital nerves and two plantar proper digital nerves. Each pair of nerves courses along both sides of the toe (Figs. 15.1 and 15.3).

Indications

- All surgeries involving the toe, including amputation, arthroplasty, and arthrodesis
- Nail procedures

Procedure

Landmarks:

- Metatarsal phalangeal joint
- Metatarsal head and neck
- Proximal phalanx

Positioning: Supine

Injection Technique: Insert the needle at one side of the toe at the level just proximal to the metatarsal neck. Raise a wheal with 0.25–0.5 ml of local anesthetics. Advance the needle plantarly until just beneath the plantar skin and deposit 0.25–0.5 ml of local anesthetics. Repeat the technique on the other side of the digit to complete the block. For distal toe surgeries such as nail avulsion procedures, this block can be performed more distally at the level of the proximal phalanx. Using the same technique, local anesthetic is deposited on each side of the proximal phalanges at the midshaft (Fig. 15.10).

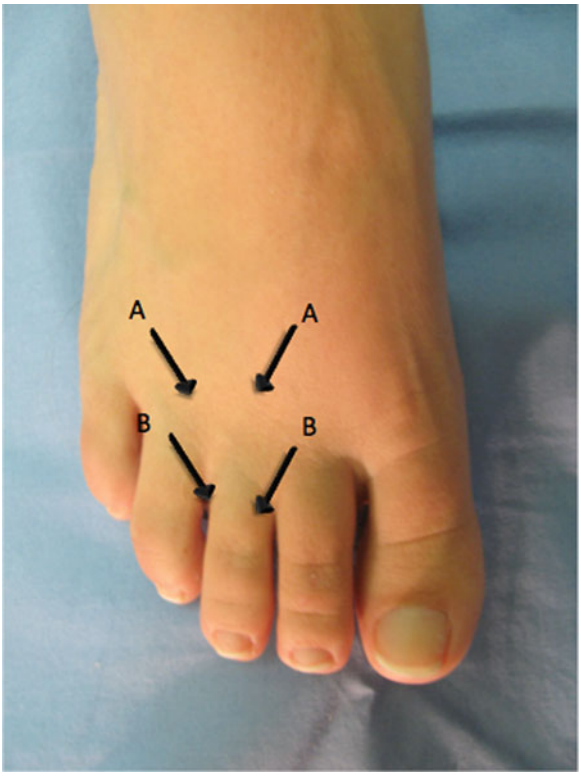

Fig. 15.10 AP view of the forefoot. *Arrows A* show the needle placement for anesthetizing the third ray. *Arrows B* show a more distal needle placement for anesthetizing only the third toe distal to the metatarsal phalangeal joint

Complications

Complications are rare in local foot and ankle anesthesia. Most allergic reactions are associated with methylparaben, a preservative in local anesthetics [8], although true allergy to local anesthetics can occur. Local toxicity occurs when large amounts of local anesthetics are injected directly into nerves or skeletal muscles; this could cause irreversible conduction loss and muscle necrosis, respectively. Systemic toxicity is also possible if sufficient amount of local anesthetic is injected intravenously or intra-arterially. Symptoms of systemic toxicity include tinnitus, convulsions, and cardiac dysrhythmias [9]. Necrosis or gangrene of the toes is another possible complication when excessive volume of local anesthetics is injected circumferentially around the toe. The risk of gangrene increases when epinephrine is added due to its vasoconstriction properties.

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16

Regional Anesthesia of Thorax and Abdomen

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Contents

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In this chapter, we will describe traditional and ultrasound paravertebral nerve block techniques, traditional intrapleural block by loss-of-resistance technique, ultrasound-guided transversus abdominis plane and rectus sheath block, and ultrasound-assisted ilioinguinal and iliohypogastric nerve blockade for postoperative pain control of thorax and abdomen. If a practitioner has mastered the paravertebral nerve block in lateral and paramedian approach, the intercostal nerve block is the redundant technique and may not be specifically useful. Therefore, we will not discuss it in this chapter.

Paravertebral Nerve Block

Introduction

Paravertebral nerve blocks (PVBNB) have been used as an analgesic modality for chest and abdomen since 1905 [1, 2]. PVBNB is a highly useful and versatile technique that could be employed to control pain after chest trauma, placement of chest tubes, breast surgery, herniorrhaphy, laparoscopic surgery, cholecystectomy, nephrectomy, soft tissue mass excision, and bone harvesting from the iliac crest (see Table 16.1). Furthermore, the technique is a valuable tool in treating acute and chronic pain in chest and abdomen (see Table 16.1). The contraindications are tumors in paravertebral space; current use of antiplatelet or anticoagulation medications such as clopidogrel and ticlopidine, and warfarin; severe coagulopathy; and allergy to local anesthetics (see Table 16.2).

Table 16.1 Indications

General surgery	*Urologic surgery*
Hernia–ventral, inguinal	Nephrectomy
Laparoscopic surgery	Adrenalectomy
Mastectomy	Ureteral surgery
Cholecystectomy	CystectomyAxillary dissection
Hepatic surgery	*Gynecologic surgery*
Pancreatectomy	Hysterectomy with abdominal debulking
	Oophorectomy
Colectomy/hemicolectomy	Node dissectionExploratory laparotomy
	Thoracic surgery
Plastic surgery	Esophagectomy
Breast reconstruction	Thoracotomy
Breast reduction	Visually assisted thoracoscopic surgery
Tissue flaps	Open heart surgeryMinimally invasive CABG
	Minimally invasive mitral valve replacement
Pain conditions	Sternal surgery
Herpetic neuralgiaRib fractures/thoracic traumaTraumatic liver capsule painIntercostal neuralgia	

Table 16.2 Contraindication

Absolute	Relative
Patient refusal	Tumor occupying PVS
Inability to get an informed consent	Kyphoscoliosis
Sepsis or infection at the site of needle placement	Previous thoracotomy
Coagulopathy	Previous radiation Rx to area
Allergy to LA	
Empyema	

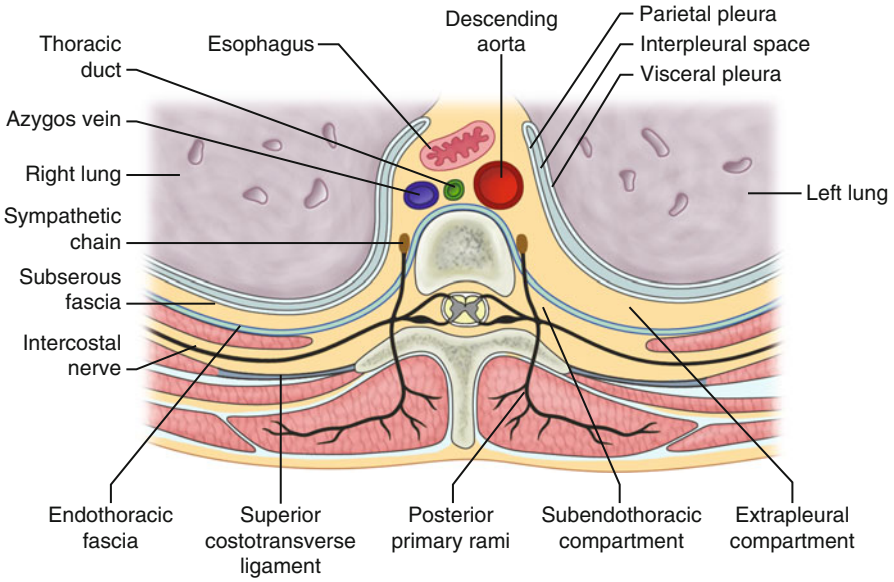

Fig. 16.1 Anatomy and boundaries of paravertebral space (adapted from ref. [2])

Anatomy

The paravertebral space forms a wedge shape and lies adjacent to vertebral bodies [2]. The boundaries of the space are as follows: anterolaterally, parietal pleural; posteriorly, costotransverse ligament (thoracic level); medially, the vertebral bodies and epidural space; and superiorly and inferiorly defined by the costotransverse articulation (see Figs. 16.1 and 16.2) [1, 2]. The space is divided by the endothoracic fascia into anterior (ventral) and posterior (dorsal) compartments.

Within the paravertebral space are the spinal intercostal nerve, its dorsal rami, anterior and posterior rami communicantes, and anteriorly, the sympathetic chain (Fig. 16.2). The intercostal nerve has no fascial sheath at this point and thus is easily penetrable by local anesthetics. Injection of local anesthetic in the space may result in ipsilateral motor, sensory, and sympathetic blockade.

The boundaries of the space are as follows: anterolaterally, parietal pleural; posteriorly, costotransverse ligament (thoracic level); medially, the vertebral bodies and epidural space; and superiorly and inferiorly defined by the costotransverse articulation. Please note the endothoracic fascia dividing the space into anterior and posterior compartments.

Some radiographic studies have demonstrated that injection of local anesthetic in the ventral compartment leads to a multisegmental longitudinal spread, whereas deposition to the dorsal space leads to the distribution in cloud-like appearance above and below the injection point [3–6]. The onset of the block is very slow and does not correlate with the volume and mass of injectate [6].

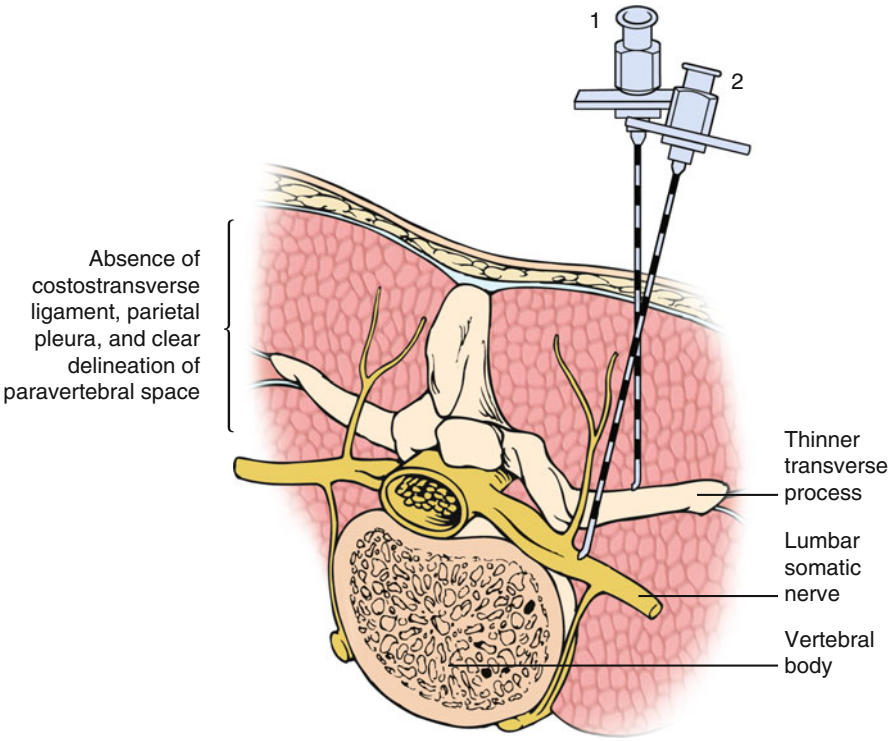

Fig. 16.2 Anatomy of the paravertebral space. *ETF,* endothoracic fascia; *SCL,* superior costotransverse ligament; *LCL,* lateral costotransverse ligament; *ITL,* interspinous costotransverse ligament (adapted from ref. [11])

Dermatomal Spread of Local Anesthetic After Injection

As for the spread of local anesthetic in the paravertebral space, Cheema et al. examined six patients injected with 10–15 cc 0.5% bupivacaine at T9–T10 and observed mean ipsilateral sensory block to pinprick five dermatomes (T6–L3) and sympathetic block of eight dermatomes (T5–L3) [4]. Saito et al. examined 16 volunteers injected with 22 cc 1% lidocaine and saline control at T11 level [5]. The researchers found 12 dermatomes blockade to somatic pinprick sensation (six rostral and six caudal), a minimum of six dermatomes sympathetic increase in temperature. In this study, the bilateral spread was not observed [5]. Karmakar et al. wrote a case report of an injection of 10 cc of local anesthetic at T8 level with the patient developing sensory changes and quadriceps muscle weakness, associated with T12 to L2 spread of local anesthetic [7]. The authors attributed these findings to possible communication between the lower PVT space and retroperitoneal space. The possible route is the transmission of local anesthetic bolus injection along endothoracic fascia to psoas compartment fascia.

Because of the absence of superior costotransverse ligament, much thinner transverse process, and absent paravertebral space, the performance of the block at the lumbar area is significantly different. We will describe the difference between the performance of the block at the thoracic and lumbar areas in the procedure section.

The Depth of Paravertebral Space

Chelly et al. retrospectively reviewed 559 records for 1,318 thoracic PVT blocks and determined that the depth of PVT space at T4–T8 is shallower (3.8–5.7 cm) and more variable [8]. At T9–T12 PVT space, the depth is at 5–6 cm and appeared to be less variable. The BMI has a significant influence on the depth from the skin to paravertebral space at these levels.

Complications of the Paravertebral Blockade

The complications from inexperienced placement of paravertebral block involve vessel puncture, hematoma, epidural spread (via the intervertebral foramina), intrathecal spread (via dural cuff), and pneumothorax [1, 2].

Procedure

Traditional Paravertebral Block Technique

Depending on the surgical procedure, the corresponding levels should be blocked (please see Table 16.3). For mastectomy, we perform T1–T6 single-shot paravertebral blocks. For breast biopsy, the injections are made at the level of biopsy as well as one level above and one level below.

For thoracotomies, visual-assisted thoracotomies, and minimally invasive mitral valve replacements, we place a paravertebral catheter at the T4 level unilaterally. For minimally invasive coronary artery bypass procedures, we place bilateral T4 catheters. For inguinal hernia repair, we perform T10 through T12 unilateral PVB injections. For prostatectomy and hysterectomy, we perform bilateral T10 through T12 injections. Recently, we have developed the technique for total hip replacement by performing an L2 paravertebral blockade. The indications for the block are summarized in Table 16.1.

Table 16.3 The level of the blocks

Segmental distribution	Anatomical area	Surgical procedure
T3, T4, T5	Upper chest	Thoracotomy Esophagectomy Mastectomy
T7, T8, T9	Subcostal, upper abdomen	Nephrectomy
		Adrenalectomy
		Ureteral surgery
		Cholecystectomy
		Liver resection
		Splenectomy
T8, T9, T10	Abdomen	Ex.Lap
T10, T11, T12	Lower Abdomen	Prostatectomy Hysterectomy Herniorrhaphy
L1, L2	Upper leg	Hip

Positioning

The patient should be positioned upright with the neck and back flexed and the shoulders pointing forward. The feet should be placed on a stool. However, the block can be performed in the lateral and prone position.

Landmarks

The spinous process for the level of block placement should be palpated and the most superior aspect of the corresponding spinous process marked.

If the needle is passed in caudal direction, the level at which spinous process is marked will correspond to the next numbered nerve root. For instance, the block done at C7 actually blocks the T1 nerve roots.

Technique

After marking the midline and spinous processes, the needle entry is marked 2.5 cm lateral (Fig. 16.3). These markings will overly costotransverse articulation in the thoracic area of the next vertebral body. However, in the lumbar area, the transverse process is at the same level as the spinous process.

Materials (See Fig. 16.4)

22-gauge, 8-cm Tuohy needle "finder"
18-gauge, 10-cm insulated needle with 10-cm extension tubing

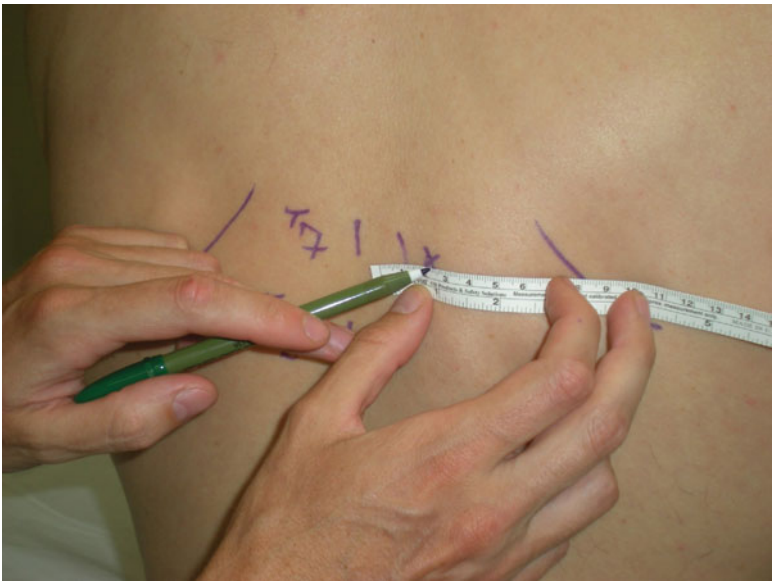

Fig. 16.3 The insertion point of the needle should be located 2.5 cm from midline

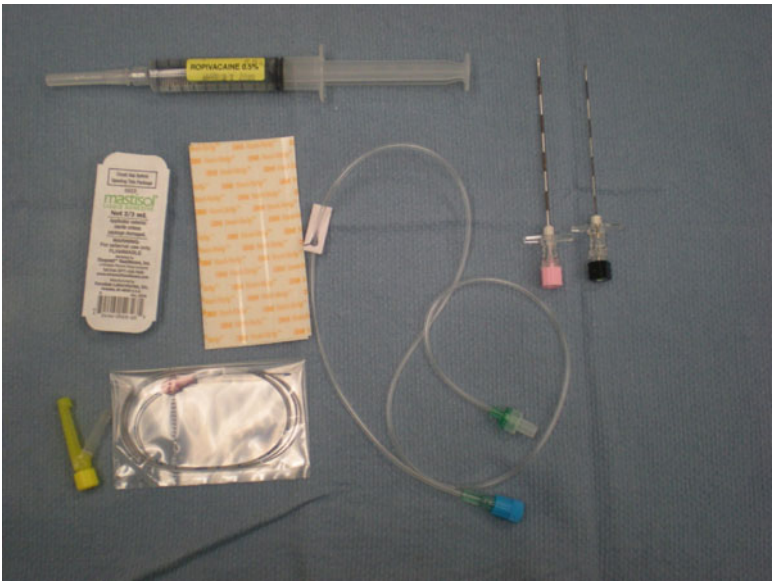

Fig. 16.4 Needles, 10-cm extension tubing, catheter, local anesthetic, and securing material

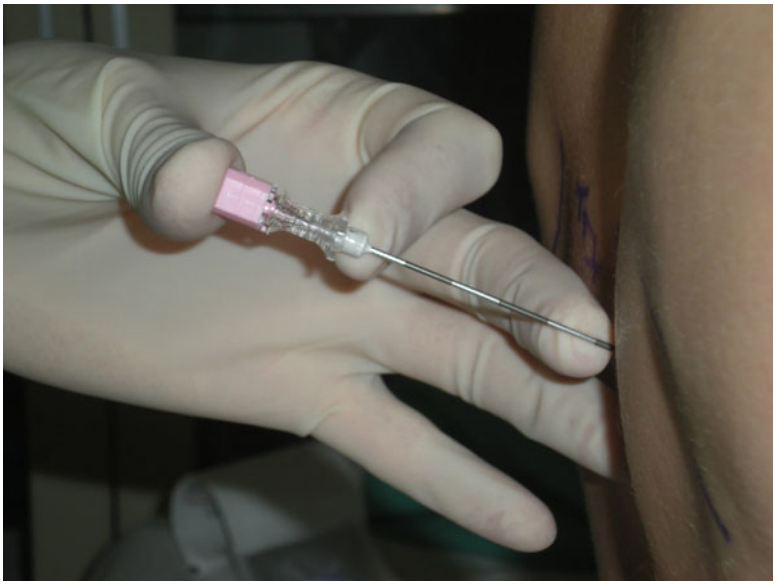

Fig. 16.5 After finding the depth to the transverse process, the fingers should be positioned 1 cm superficial on the needle and the needle angled 30–45° to the surface and advanced to the previously established distance

20-gauge, multiorifice nerve block catheter
3 M "Steri-Strips"
"Tegaderm"

After sterilizing the skin, place a wheal of 1% lidocaine with a 22-gauge 3-cm needle at each level to be blocked. The finder needle, a 22-gauge Tuohy, is inserted through the anesthetized skin until it contacts the transverse process (2–5 cm depth, depending on the body mass index of the patient).

If you cannot find the transverse process, attempt to redirect caudally or cranially until the transverse process is encountered. After identifying the depth to the transverse process with the finder needle, insert an 18-gauge Tuohy needle with the fingers serving as a backstop to prevent the needle to pass beyond the prior established depth. Then place your fingers 1 cm away from the established depth, withdraw the needle to subcutaneous tissue, and angle it to "walk off" the lower edge of the transverse process, advancing no more than 1 cm into the space (Fig. 16.5).

Often, you will appreciate a "pop" as you advance through the superior costotransverse ligament. Place a couple of drops of local anesthetic on the hub of the needle and ask the patient to inhale (Fig. 16.6).

If the fluid appears to be sucked into the needle, you have to withdraw the needle a couple of millimeters. If you detect the negative pressure on inhalation, it means that the needle is placed intrapleurally and needs to be withdrawn. Next, attach the extension tubing and with the help of an assistant inject 3–5 cc of local anesthetic.

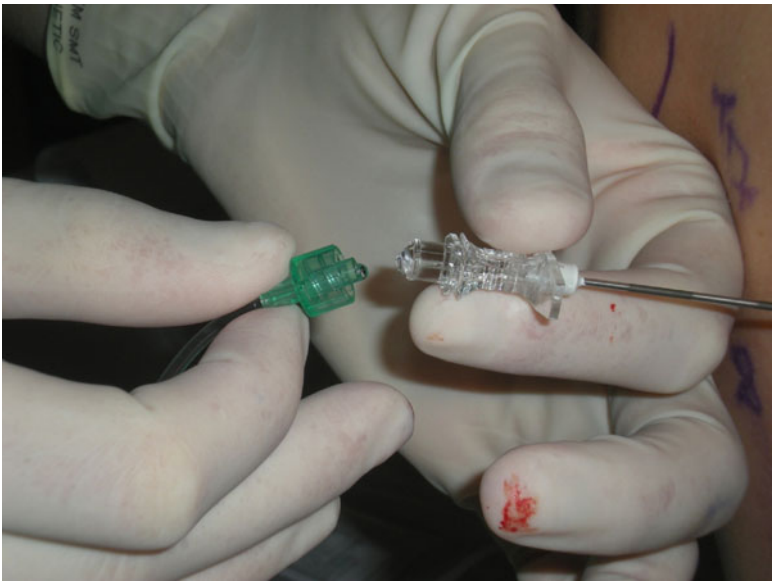

Fig. 16.6 "The bubble test." Place a small drop into the needle hub and ask the patient to inhale. If the bubble is sucked in, then the needle is placed intrapleurally and should be withdrawn

If the resistance to the injection is encountered, the needle should be redirected. After each redirection, the "negative pressure test" must be repeated.

Upon successful identification of the paravertebral space, inject 3–5 cc of local anesthetic. If you need to thread the catheter, it should be placed no more than 5 cm deeper than the encountered depth of paravertebral space (Fig. 16.7).

Because the spread of local anesthetic appears to be greater caudally, the catheter placement should be done at a more cranial position above the expected procedure. After the catheter is placed to the required depth, 10 cc of local anesthetic is administered through the catheter. The catheter is secured with Mastisol, 3 M "Steri-Strips," and sterile occlusive dressing (Fig. 16.8).

The performance of the paravertebral block at the lumbar area is basically similar. However, the needle should not be advanced greater than 0.5 cm from the transverse process. Also, it is much harder to find a transverse process at the lumbar area and may require several passes before one encounters a transverse process.

Local Anesthetic

At the University of Pittsburgh Medical Center, we use 0.5% ropivacaine 5 cc per each level for single PVBs. As for the infusions, we use 0.25% lidocaine for bilateral paravertebral catheters and 0.0625% bupivacaine for unilateral catheters.

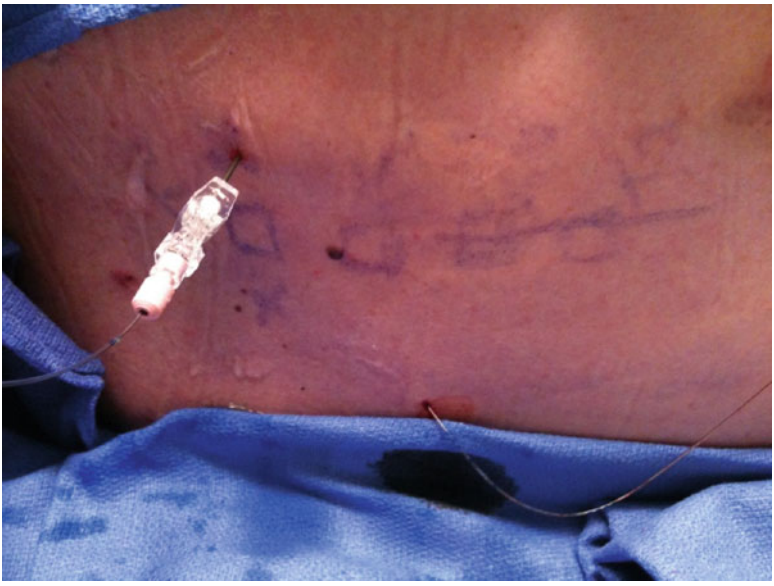

Fig. 16.7 Placement of the paravertebral catheter for bilateral rib fractures in right lateral decubitus position

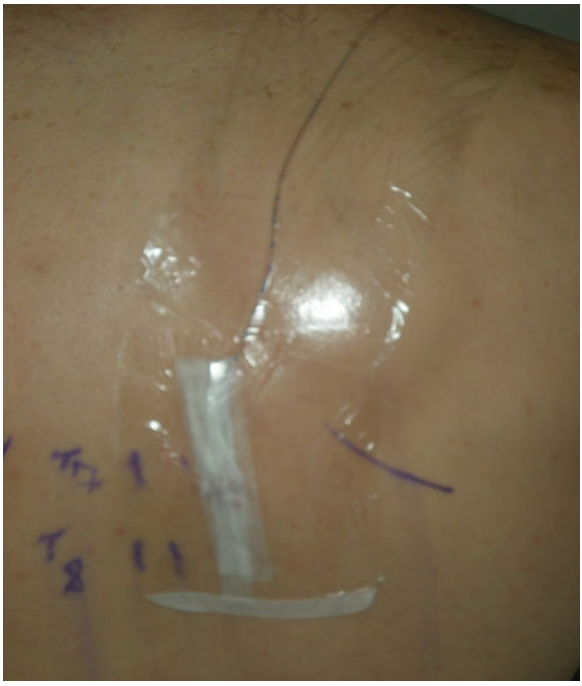

Fig. 16.8 Securing the catheter with Steri-Strips and dressing

Precautions and Fine Points

1. During the placement of the block, patients frequently develop a vasovagal response and become severely bradycardic and hypotensive accompanied with the loss of consciousness. Therefore, a syringe of an anticholinergic agent such as glycopyrrolate and a pressor agent (ephedrine) should be immediately available.
2. For single-shot PVBNB, inject through a 22-gauge needle.
3. Do not aim medially as it would lead to neuraxial blockade.
4. If the wall of the bone is encountered upon needle placement, the needle is placed too medially and is hitting vertebral laminae. Redirect the needle laterally.
5. The needle should be directed caudad of the transverse process as the distance between transverse process and pleura is greater inferiorly. The rib is usually located more cephalad, so the placement of the needle cranially may lead to intercostal placement of the catheter.
6. The needle position should always be identified with the hanging drop technique. With the intrapleural positioning, the drop will be pulled into the needle upon inspiration, and with the paraspinal muscle localization, the drop will remain stable or will bulge.
7. Do not inject a large volume of local anesthetic through the 18-gauge needle as it will lead to neuraxial blockade.
8. The insertion of the catheter is often met with resistance. If you encounter the resistance upon catheter placement through the Tuohy needle, slightly redirect the needle cephalad and attempt to place the catheter. If this maneuver is unsuccessful, direct the bevel of the needle laterally and reattempt the catheter placement. However, if all attempts to place the catheter are met with resistance, take the needle with inserted catheter out and place the needle at the next transverse process level. Never remove the catheter from the needle as it will shear the catheter.
9. Due to the delayed onset of neuraxial blockade, the patient should be continuously observed and monitored during and after the nerve block placement.

Intercostal Paravertebral Approach

Introduction/Indication

Recently, an intercostal approach to paravertebral space has been described [9]. The indications for the block placement are surgical procedures of the thorax and abdomen and are very similar to indications for traditional paravertebral nerve block. Also, this approach is quite useful when the placement of traditional paravertebral blockade is contraindicated such as if the patient is anticoagulated, thrombocytopenic, or coagulopathic or if there is a question of transverse process fracture and unstable spine [9, 10].

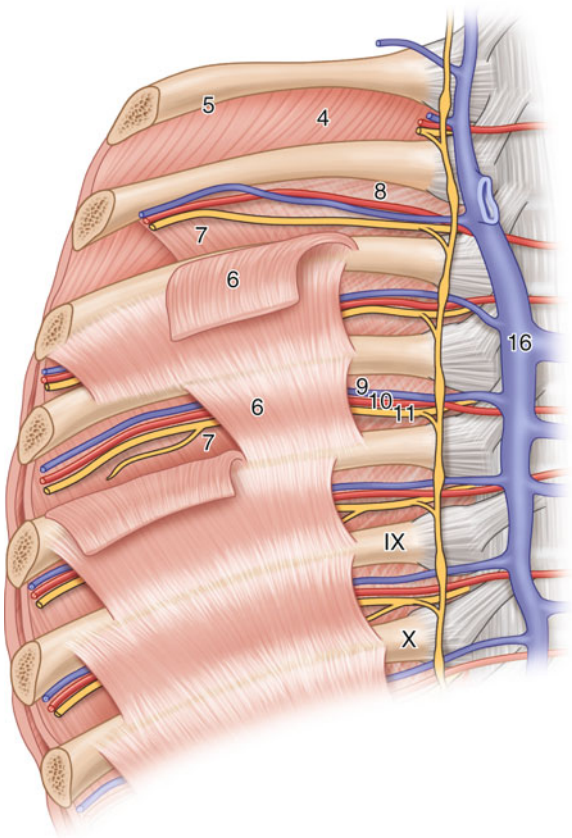

Fig. 16.9 Gross anatomy of thoracic paravertebral space via intercostal approach *4,* external intercostal muscles; *5,* costal groove; *6,* innermost intercostal muscles; *7,* internal intercostal muscle; *8,* internal intercostal membrane; *9,* intercostal vein; *10,* intercostal artery; *11,* intercostal nerve; *IX,* 9th rib; *X,* 10th rib; *16,* azygous vein (adapted from ref. [11])

Anatomy/Landmarks

The intercostal space is contiguous with paravertebral space. The intercostal nerve accompanied by artery and vein lies in the subcostal groove between the internal and innermost intercostal muscles (see Fig. 16.9) [11]. The T7 vertebral body should be identified by palpating the inferior border of the scapula. Then, the desired rib and level as well as insertion site should be marked on the lower portion of the rib 8 cm lateral to the midline.

Positioning

The patient assumes a sitting position with the legs resting on a chair. The patient is asked to assume "a bad posture" position bringing scapulae apart (see Fig. 16.7). The block could also be performed in lateral and prone position.

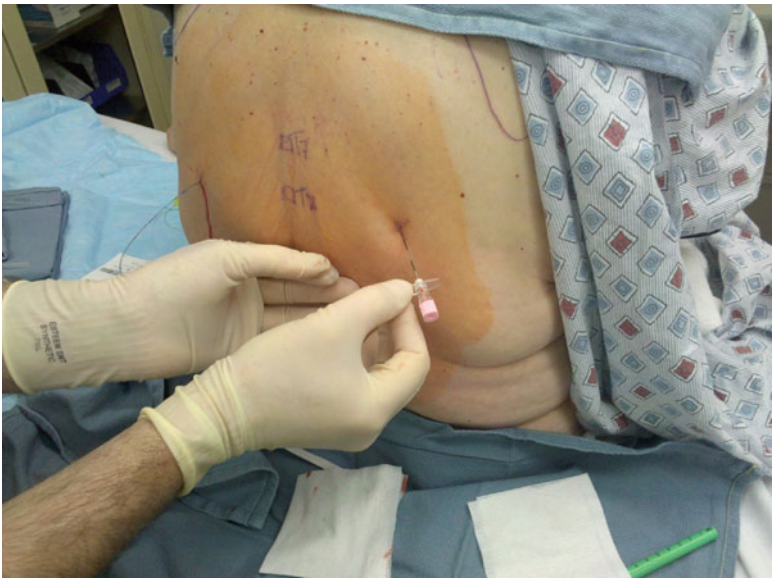

Fig. 16.10 Positioning and placement of the needle for the intercostal approach to the paravertebral space. The needle should be directed 45° cephalad and 60° medial to the sagittal plane and walked off the lower part of the rib

Needles and Catheters (See Fig. 16.4)

22-gauge, 8-cm Tuohy needle "finder"
18-gauge, 10-cm insulated needle with 10 cm extension tubing
20-gauge, multiorifice nerve block catheter

Procedure (See Fig. 16.10)

- The skin should be prepped and draped with aseptic technique. 1% Lidocaine is used to anesthetize the skin and periosteum under the insertion point.
- Place the 18-gauge Tuohy needle through the anesthetized insertion site and contact the rib. Redirect the needle 45° cephalad and 60° medial to the sagittal plane and walk off the lower part of the rib.
- After bony encounter, advance 5–6 mm and enter the intercostal neurovascular space.
- Perform the previously described negative pressure check after aspiration for heme or air.
- Inject 5 cc of local anesthetic through the needle and thread the catheter.
- Secure the catheter 5–8 cm past the needle tip.

- Bolus the catheter with additional 10 cc of local anesthetic.
- If needed, thread the catheter through the Tuohy needle and secure.

Precautions and Fine Points

1. The points are the same as for the traditional paravertebral nerve block placement.
2. Pneumothorax is very likely to occur in inexperienced hands. Do not advance the needle more than 5–6 mm under the lower border of the rib into the intercostal space.
3. The needle position should always be identified with the hanging drop technique. With the intrapleural positioning, the drop will be pulled into the needle upon inspiration, and with the intercostal muscle localization, the drop will remain stable or will bulge.
4. Again, do not inject the local anesthetic rapidly and in large amount through the 18-gauge Tuohy. The epidural spread may occur.
5. Also, due to the delayed onset of neuraxial blockade, the patient should be continuously observed and monitored during and after the nerve block placement.

Ultrasound-Guided Paravertebral Nerve Blockade (Paramedian or Classic Approach)

Introduction

Because of recent advances in ultrasonography equipment, the placement of nerve block needle in the paravertebral space can now be performed with a 40- to 60-cm curved array probe oscillating at 2–5 MHz (see Fig. 16.8). This block is particularly useful in obese and morbidly obese patients. The midline is easily identified by palpation or with ultrasound assistance, and the transverse and spinous processes and ribs are visualized with ultrasound guidance.

Equipment

We use a 40- to 60-cm curved array probe oscillating at 2–5 MHz. For smaller patients, an 11-cm curved array probe oscillating at 5–8 MHz can be used.

Needles

Please see Fig. 16.2 and description for traditional paravertebral nerve block.

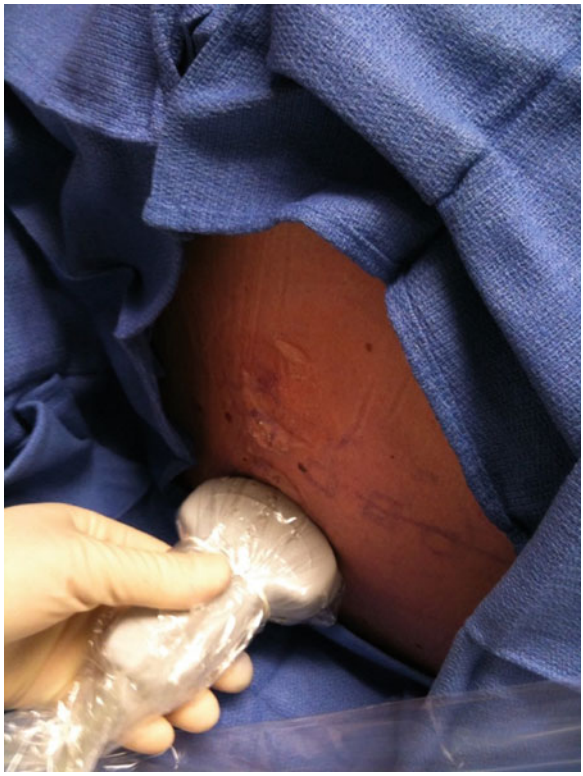

Fig. 16.11 The low-frequency transducer should be positioned 2.5 cm from midline in order to view the paravertebral space (The patient is positioned in lateral decubitus position)

Local Anesthetic

Again, we prefer to use 0.5% ropivacaine 5–15 cc per block.

Position

The patient could be positioned sitting up with shoulders relaxed and feet resting on a stool, or if the patient is unable to sit up, the lateral decubitus position could be employed.

Technique

The anatomic landmarks are identified and marked as for traditional paravertebral nerve block. The skin is prepped and draped in sterile fashion. Then, the region is scanned with a 40- to 60-cm sterile covered curved array probe oscillating at 2–5 MHz placed sagittally 2.5 cm to the spinous processes (Fig. 16.11).

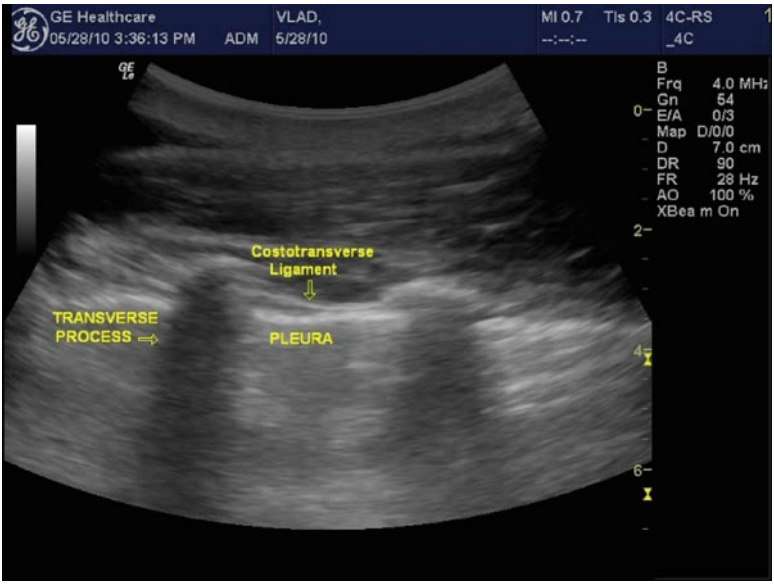

Fig. 16.12 The paramedian ultrasound view of the paravertebral space

The first hyperechoic structure identified is the superior costotransverse ligament, and the second hyperechoic structure is the pleura (Fig. 16.12).

Upon identification of clearly discernible ultrasonographic view of the paravertebral space, the skin is anesthetized with 1% lidocaine, attached to extension tubing, using a 25-gauge, 3.75-cm needle at the position for nerve block needle (Fig. 16.13).

Then, an 18-gauge Tuohy needle is attached to the extension tubing and directed in-plane cephalad or caudad to the transducer.

The needle is placed deep to the superficial costotransverse ligament about 0.5–1 cm but superficial to the endothoracic fascia. Because the visualization of endothoracic fascia is problematic with the current ultrasound technology, we recommend placement of the needle 0.5–1 cm deeper than the identified costotransverse ligament.

After the negative aspiration for blood or CSF, 3–5 cc of 0.5% ropivacaine is injected slowly. The slight tenting of the pleura should be observed (Fig. 16.14). Then, the multiport 20-gauge catheter is placed through the needle and left 4–5 cm beyond the tip of the needle. The needle is removed, and the catheter is fixed using Steri-Strips and covered with a transparent dressing (see Fig. 16.8). After repeating aspiration for blood and CSF, additional 10 cc of 0.5% ropivacaine is injected through the catheter.

The local anesthetic injected through the catheter can be observed under real-time ultrasonography to spread in the paravertebral space.

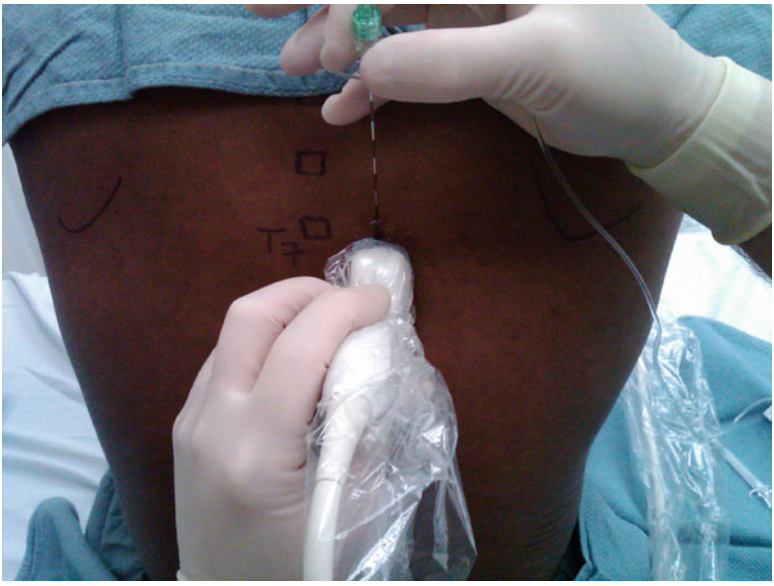

Fig. 16.13 The 18-gauge Tuohy needle is attached to the extension tubing and placed in-plane caudad or cephalad to the transducer

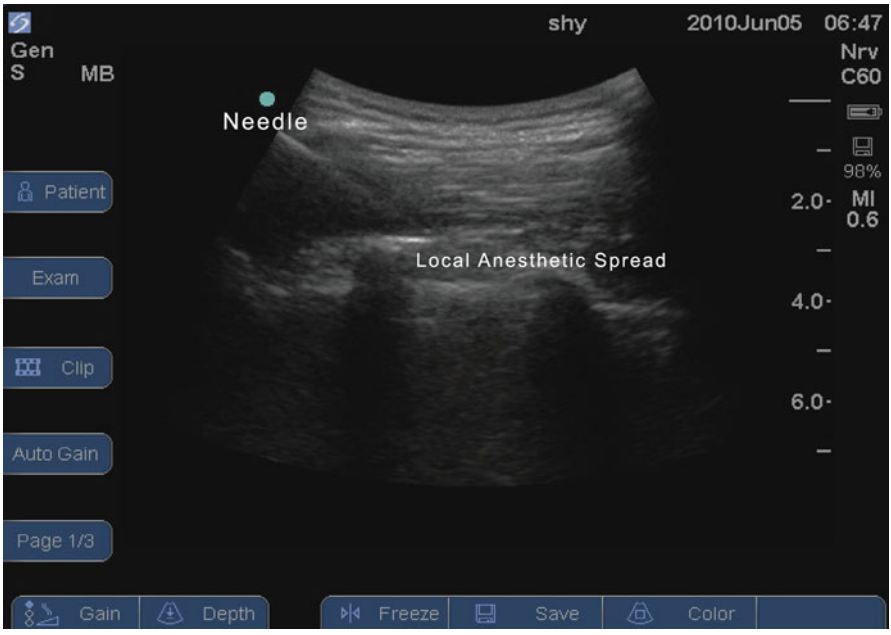

Fig. 16.14 Please note the needle positioned to the left of ultrasound image, the spread of local anesthetic in the paravertebral space, and tenting of the pleura

Precautions and Fine Points

1. Sometimes the catheter placement through the needle could be difficult. Try to readjust the needle in the horizontal direction and rotate the hub.
2. If you are unable to thread the catheter through the needle, always remove the needle with the catheter to prevent shearing the catheter.
3. The needle should never be introduced in lateromedial direction to minimize epidural or subarachnoid insertion.
4. If you visualize a sawtooth pattern of bony structures, the transducer is pointed medially, and you are observing vertebral laminae.
5. For higher thoracic levels, a 5- to 10-cm linear array transducer can be used due to the more superficial location of the paravertebral space.

Ultrasound-Assisted Intercostal Approach to Thoracic Paravertebral Space

Introduction and Indications

The intercostal space is contiguous with thoracic paravertebral space, and the neurovascular bundle located at the inferior edge of the rib becomes the thoracic spinal nerve roots and intercostal arteries (see Fig. 16.10). In the paravertebral space, the spinal nerve root is situated anterior to the transverse process and in close proximity to parietal pleura. This approach is less dependent on identification of superior costotransverse ligament, which is difficult to visualize in obese individuals. The lateral approach to paravertebral space affords the visualization of the space between external and internal muscle layers and between internal and inner intercostal muscles [12]. The deposition of local anesthetic into these spaces leads to spread of local anesthetic to the paravertebral space. The spread along dermatomes could be increased with larger volume of the injectate. The block is less painful to the patient because of avoidance of thick paraspinous muscles (Fig. 16.15).

Position

Prone or sitting upright.

Transducer

40- to 60-cm curved array probe oscillating at 2–5 MHz or 5–8 MHz 11-cm curved array transducer. For thinner patients, a 2.5-cm linear array transducer oscillating at 10–13 MHz is used.

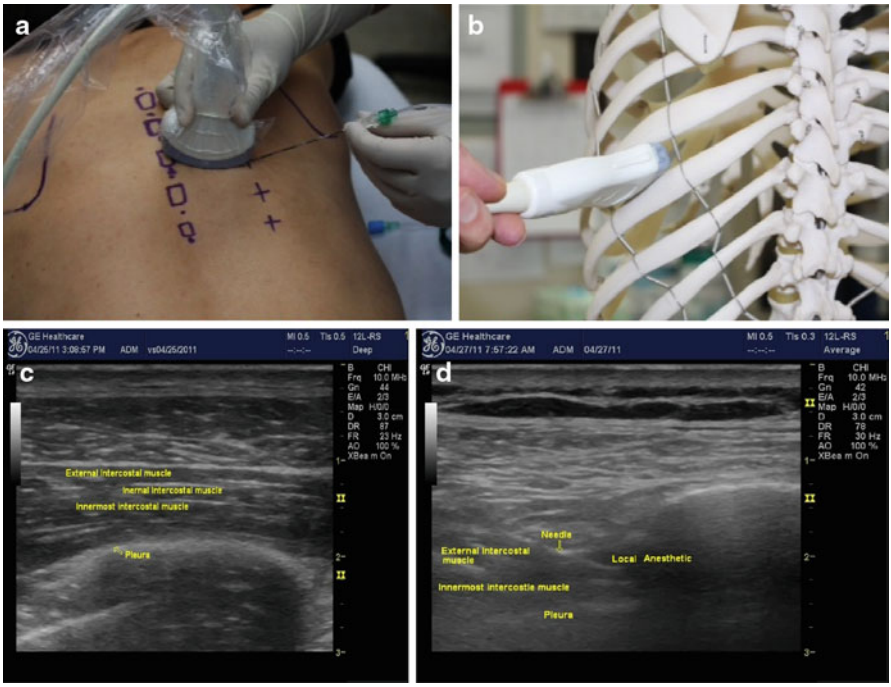

Fig. 16.15 The ultrasound anatomy of the abdominal wall at the anterior axillary line between the iliac crest and 12th rib

Needles and Materials (See Fig. 16.4)

Technique

The patient is positioned prone and/or sitting and the desired intercostal space palpated lateral to the paraspinous muscles (Fig. 16.16). The transducer is first placed longitudinally to the midline, and the ribs and pleura are visualized.

The transducer is turned horizontally along the long axis of the rib, and the rib is identified (Fig. 16.17). By toggling the transducer, the external intercostal muscle, internal intercostal membrane, and pleura are identified.

The puncture site is at the lateral side of the transducer (see Fig. 16.18). The needle is introduced in-plane to the transducer (Fig. 16.18a). As the needle is advanced past the rib, 2 to 3 cc of local anesthetic is injected (Fig. 16.18b). The plane between the external and internal intercostal muscles dissected by the local

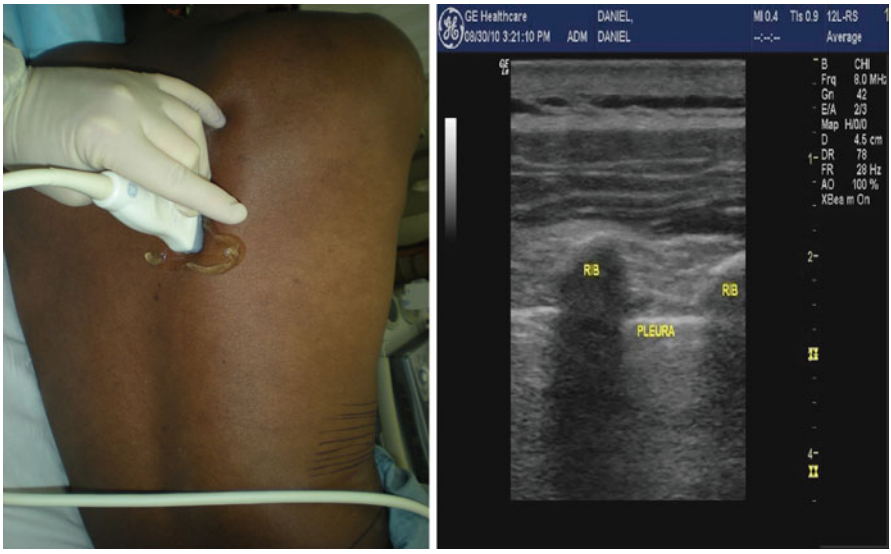

Fig. 16.16 Placement of the probe in vertical orientation and ultrasound image of the intercostal space

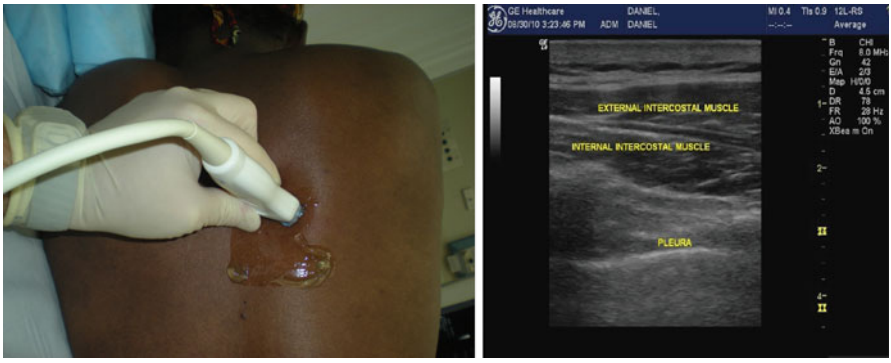

Fig. 16.17 By positioning and toggling the probe in the sagittal orientation, the intercostal approach to thoracic paravertebral space is visualized

anesthetic appears (Fig. 16.18c). The operator advances the needle 2–3 mm deeper, and 3–5 cc of local anesthetic is injected. The second tissue plane between the internal and inner intercostal muscles appears (Fig. 16.18d). Upon identification of this plane, 10–15 cc of local anesthetic is slowly deposited. If the continuous technique is desired, the catheter can be inserted 5–10 cm beyond the tip of the needle.

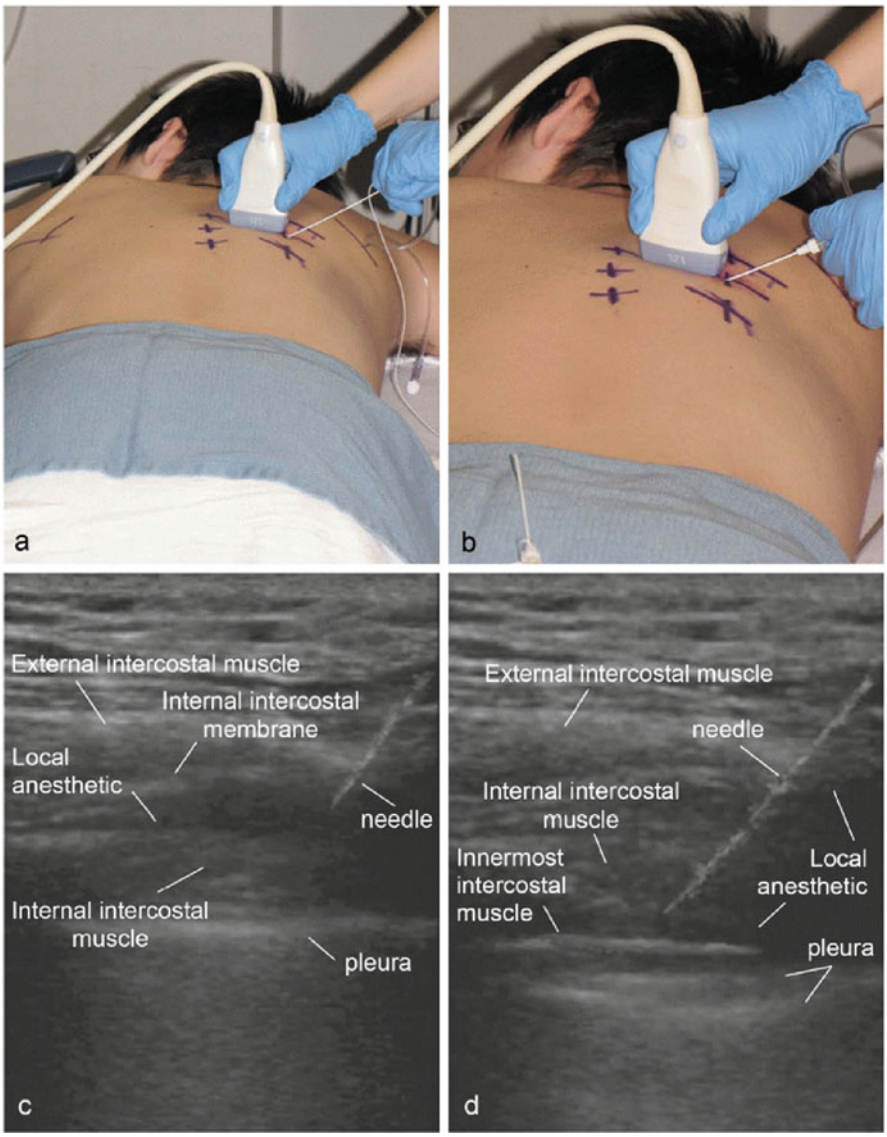

Fig. 16.18 Intercostal approach to the thoracic paravertebral space. (**a**) The transducer is positioned longitudinally below the rib. (**b**) The needle is introduced in lateral to medial direction below the rib. (**c**) Injection of local anesthetic and hydrodissection of the space between the external intercostal muscle and internal intercostal muscle. (**d**) The needle is introduced further with hydrodissection, opening the space between the internal intercostal muscle and innermost intercostal muscle (figure adapted from ref. [11])

Precautions and Fine Points

1. The time for sensory blockade is usually 30–40 min.
2. The pneumothorax is very likely in inexperienced hands.
3. The catheter can be placed intrapleurally if the needle is introduced into the pleural space. The local anesthetic infusion can then commence through the catheter in the intrapleural space.
4. Again, the operator should be prepared to treat vasovagal syncope or sympathectomy.

Continuous Interpleural Block

Position

The patient should be placed in lateral decubitus position with the nondependent arm displaced anteriorly and cephalad to rotate the scapula forward and provide exposure to the posterolateral chest wall.

Indications

This block is performed to provide analgesia after mastectomy, nephrectomy, and cholecystectomy; rib fractures; herpetic neuralgia; pancreatitis; and invasive tumors of the chest wall, flank, and retroperitoneum [13–15].

Needles and Catheters

We use an 18-gauge epidural needle and a 21-gauge catheter (also see Fig. 16.4).

Local Anesthetic

We place initial bolus of 20–30 cc 1% lidocaine and infuse 0.25% lidocaine at 6–8 cc/h.

Anatomic Landmarks

The intercostal space at seventh to eighth rib, the scapula, and the posterior axillary line (see Fig. 16.19).

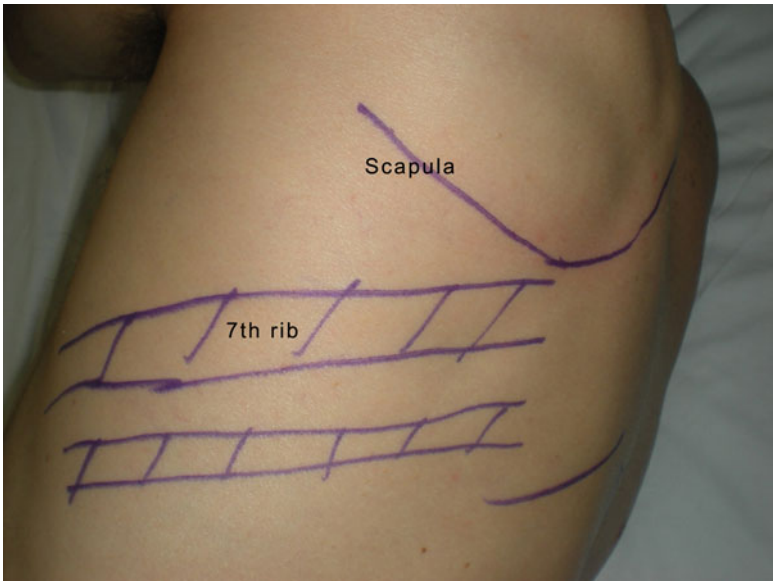

Fig. 16.19 The anatomic landmarks for the intrapleural block. Please note the scapula and seventh and eighth ribs with adjacent intercostal space

Technique

The needle puncture site is located at the seventh or eighth intercostal space, at the level of posterior axillary line, and at the superior border of the eighth rib at the previously marked level of scapular tip [13]. The needle is advanced in a vertical direction perpendicular to the chest wall. The needle is inserted 1 cm past the ribs, and the glass syringe with a plunger removed is attached to the needle (Fig. 16.20). The syringe is filled with saline. The needle and the syringe are slowly advanced while monitoring for a downward displacement of the fluid column under the influence of negative pleural pressure. Once the water column starts falling, the syringe is removed, and the catheter is advanced 6–10 cm into the pleural space. The needle is removed, and the catheter is secured with Steri-Strips (3 M, St. Paul, MN) and covered with a transparent dressing (Fig. 16.21).

Precautions and Fine Points

1. The block can be performed 8–10 cm lateral from the spine.
2. The placement of intrapleural block causes a small degree of pneumothorax (about 10%) [16]. Therefore, this block should only be performed in spontaneously ventilating patients and avoided in patients on positive pressure ventilation.

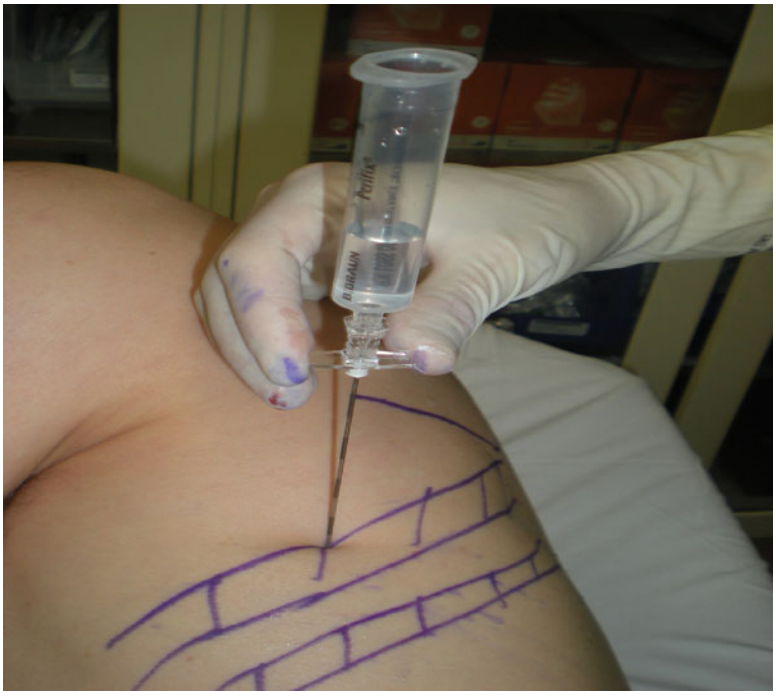

Fig. 16.20 The "falling column" technique for interpleural block

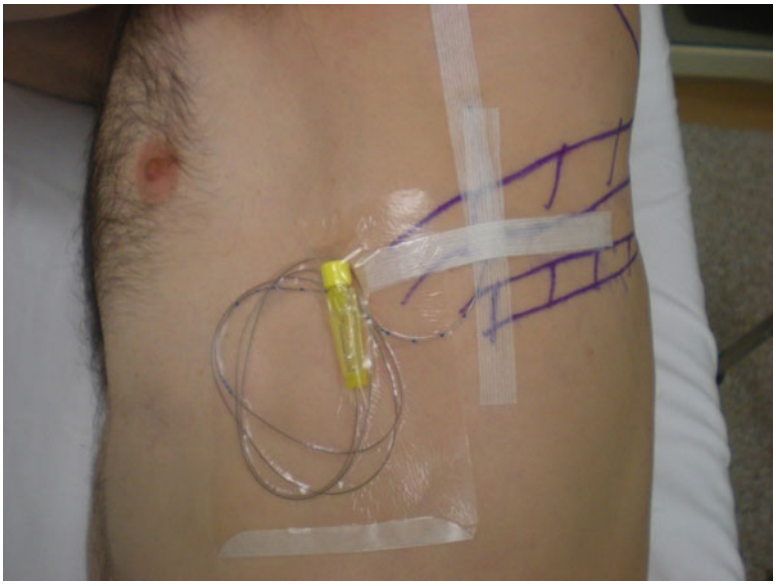

Fig. 16.21 The catheter is secured with occlusive material

3. The lung injury is reduced if the block is performed with a "falling column" technique and not with "loss-of-resistance" technique.
4. Because of the drug sequestration, drug loss through chest tubes, and uneven distribution inside of the pleural space, the intrapleural block has a questionable efficacy after thoracotomy.
5. Some analgesia post-rib fractures can be achieved by clamping the chest tube for 30–45 min following the bolus of local anesthetic.
6. The catheter can be placed intraperitoneally if a very low intercostal space is chosen.
7. If one encounters difficulty in threading the catheter such as resistance, this indicates that the catheter may have entered the lung parenchyma or the catheter encountered pleural adhesions. Any resistance to the catheter placement indicates improper position.
8. In order to reduce peak systemic levels of local anesthetic, epinephrine should be added.
9. The patient's positioning will determine site of the blockade. If the patient is placed supine, the intercostal nerves will be blocked. If the patient is placed in lateral decubitus position and the dependent site is being blocked, again the intercostal nerves on the dependent side will be anesthetized. If the patient is placed in Trendelenburg, the upper thoracic and cervical sympathetic blockade can occur, manifesting as Horner's syndrome [16].
10. This block is particularly useful in anticoagulated patients with multiple rib fractures already on ventilator.
11. In order to reduce cumulative local anesthetic toxicity, lidocaine may be the preferred agent for infusion. We recommend using 0.25% lidocaine infusions.

Transversus Abdominis Plane Block (TAP)

The transversus abdominis plane blocks have traditionally been performed with the blind technique through the angle of Petit. Recently, the ultrasound-guided TAP block has been developed with a goal of improved localization and deposition of the local anesthetic as well as improved accuracy [17, 18]. The TAP block initially described by O'Donnell placed the local anesthetic in the plane between the internal oblique and the transversus abdominis muscles [18]. The studies on the efficacy of the TAP block are limited. However, the research shows a great promise in postoperative pain control in prostatectomy, large- and small-bowel surgery, and cesarean section [18–20].

Anatomy

The abdominal wall is composed of three muscle layers: external oblique, internal oblique, and transversus abdominis (see Fig. 16.22). The abdominal anterolateral wall is innervated by the anterior rami of T7–L1 spinal nerves. The intercostal nerves

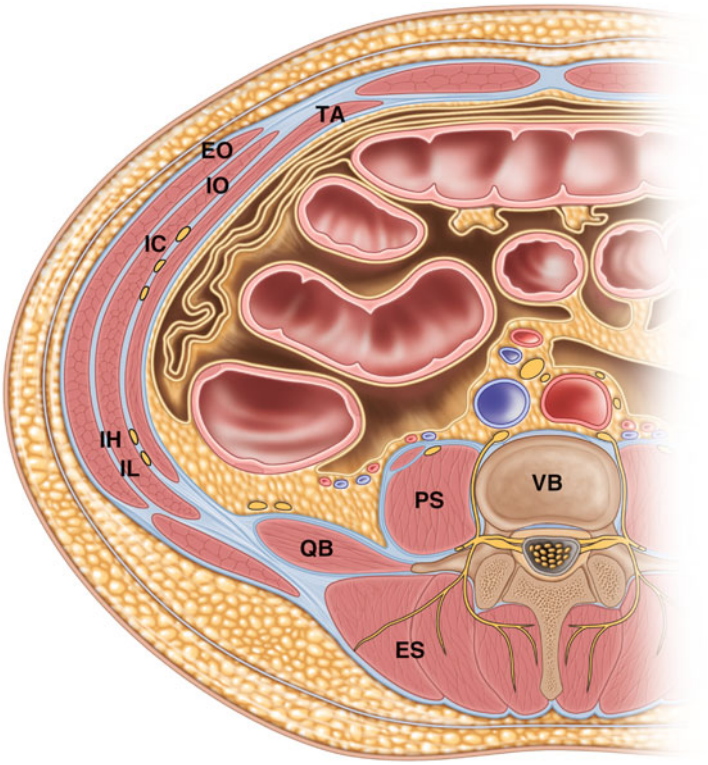

Fig. 16.22 The cross section of the abdominal wall. Please note that the nerves are not obvious on the ultrasound image and somewhat discrete between the plane of internal oblique and transversus abdominis muscles. *EO,* external oblique muscle; *IO,* internal oblique muscle; *TA,* transversus abdominis muscle; *IC,* intercostal nerves; *IL,* ilioinguinal nerve; *IH,* iliohypogastric nerve; *PS,* psoas; *ES,* erector spinae; *VB,* vertebral body; *QB,* quadratus lumborum (adapted from ref. [11])

(T7–T11), the subcostal nerve (T12), the iliohypogastric nerve, and the ilioinguinal nerve (L1) supply the lower portion of the abdominal wall. These nerves enter the abdominal wall between transversus abdominis and internal oblique.

Position

The patient is placed supine (see Fig. 16.23).

Needles

We use a 22-gauge 8-cm Tuohy needle. An 18-gauge Tuohy needle may be used for catheter placement.

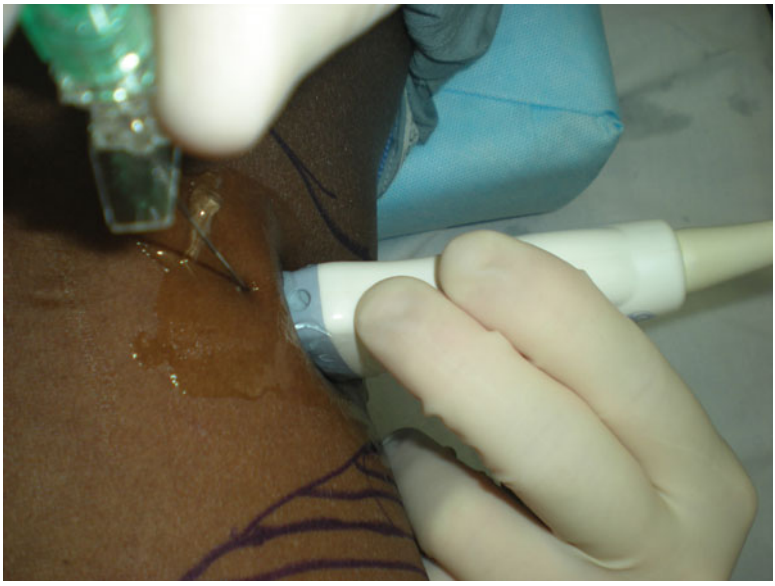

Fig. 16.23 Positioning of needle and probe for the TAP block. Note iliac crest caudad to the ultrasound probe

Local Anesthetic

For a rapid onset of block, 1% lidocaine 20 cc can be used. We usually use 20–30 cc of 0.5% ropivacaine.

Probe: 40–60-mm curved array at a frequency of 3–8 MHz

Technique

The procedure can be done preoperatively or postoperatively. Because the anatomy of the abdominal wall is intact preoperatively, we prefer to perform this block before the operation. This procedure must be done under extreme aseptic technique due to the possible penetration of the peritoneum.

The patient is positioned supine, and the probe is placed above the iliac crest at the anterior axillary line (Fig. 16.22). The muscle layers are identified on the ultrasound image (Fig. 16.23). Then, the needle is inserted in-plane.

The needle movement is carefully observed until the tip is positioned between the plane of transversus abdominis and internal oblique (Figs. 16.24 and 16.25).

The local anesthetic is injected, and the appearance of the plane between the internal oblique and transversus abdominis is observed (Fig. 16.25).

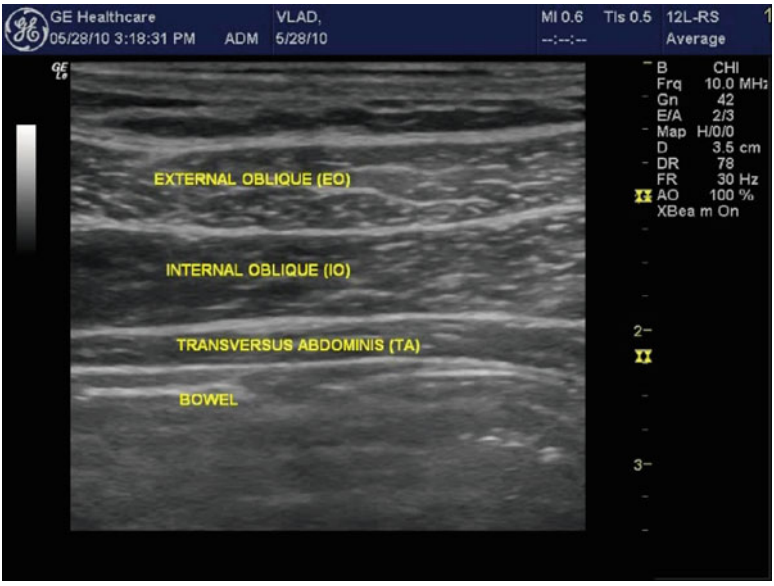

Fig. 16.24 The ultrasound anatomy of the abdominal wall at the anterior axillary line between the iliac crest and 12th rib

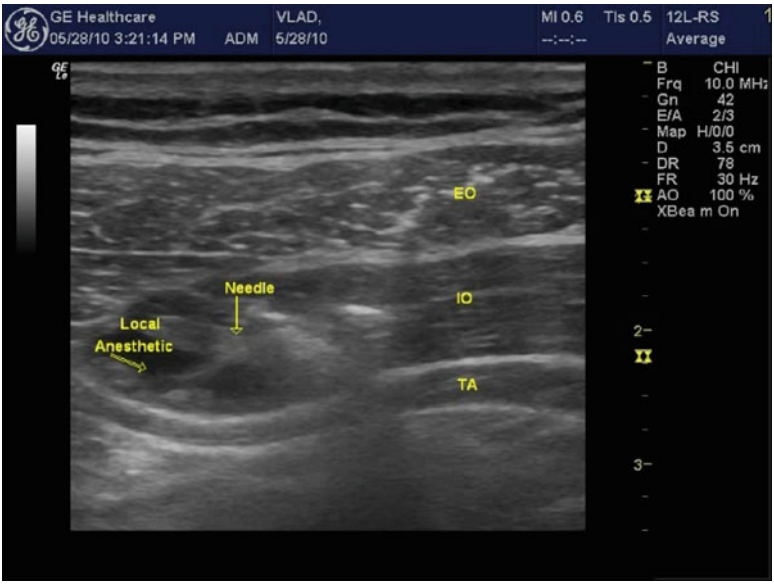

Fig. 16.25 The local anesthetic injection in the facial plane between internal oblique and transversus abdominis muscles. Note the lifting up of internal oblique muscle with local anesthetic injection. *EO* external oblique; *IO* internal oblique; *TA* transversus abdominis muscles

Precautions and Fine Points

- This block requires hydrodissection with normal saline of the plane between the internal oblique and transversus abdominis muscles prior to placing the local anesthetic between the internal oblique and transversus abdominis.
- The needle could easily penetrate into the peritoneal space, so be very careful with needle adjustments and depth.
- Never deposit the local anesthetic into the muscle tissue. The block will be ineffective.
- The plane between internal oblique and transversus abdominis will appear once the normal saline has been injected. After finding the correct plane, inject local anesthetic into the lacuna.

Rectus Abdominis (Rectus Sheath) Block

The rectus sheath block was introduced into practice in 1899 to achieve abdominal muscle relaxation and as an analgesic technique. In the current practice setting, the block appears to decrease opioid requirements after diagnostic and interventional laparoscopy. The rectus sheath blockade is primarily used for surgeries around the middle of abdomen [21, 22]. This technique could be used for percutaneous gastrostomy surgery and umbilical hernia and midline ventral hernia repairs [21, 22].

Anatomy

The anterior rami of T8–T12 spinal nerves innervate the umbilical area. The nerves are located in-plane between the internal oblique and transversus abdominis muscles (see Fig. 16.22). The posterior wall of the rectus sheath is formed by the tendon sheath of the transversus abdominis muscle. The anterior wall of the rectus sheath is formed by the tendons of the internal and external oblique muscles. The rectus abdominis muscle is enveloped by the anterior and posterior walls of the rectus sheath, beginning at the linea alba and conjoining in the midline of the abdomen into a single sheath. The intercostal nerves are located in the space posterior to the rectus abdominis muscle and posterior wall of the rectus sheath. After traveling under the rectus abdominis muscle, the nerves perforate the sheath neath the linea alba. The end branch of the nerves is the anterior cutaneous branch which arises just before the aponeurosis of rectus abdominis muscle. This nerve may travel anterior to the rectus muscle under the anterior rectus sheath or posterior to the rectus muscle and posterior rectus sheath. The anterior cutaneous branch innervates the midline of the abdomen. Because of the anatomical location, these end nerves can be anesthetized either anterior or posterior to the rectus abdominis muscle.

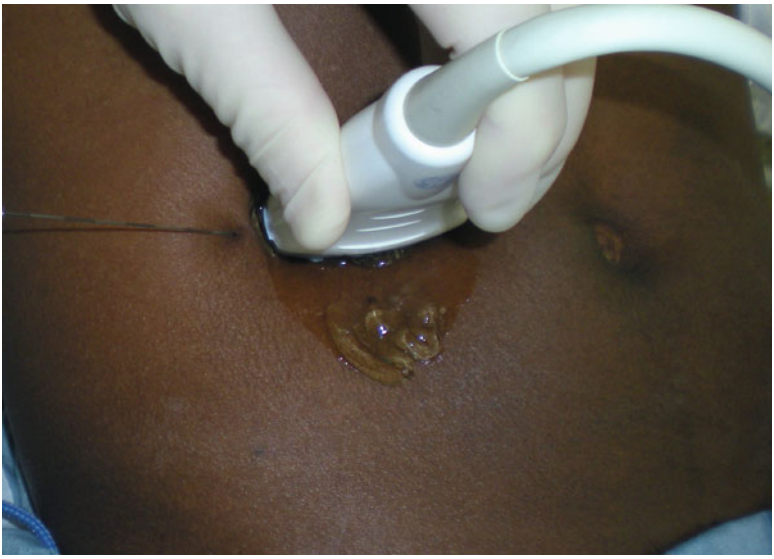

Fig. 16.26 The needle and probe position for the rectus sheath block

Patient Position

Supine (see Fig. 16.26).

Transducer

8–13 MHz 25- or 35-mm linear transducer.

Transducer Placement

Lateral to the midline in transverse plane.

Needle

24-gauge 2.5-cm needle or 22-gauge, 5-cm nerve block needle; 22-gauge, 8-cm Tuohy needle; 18-gauge Tuohy needle for continuous techniques.

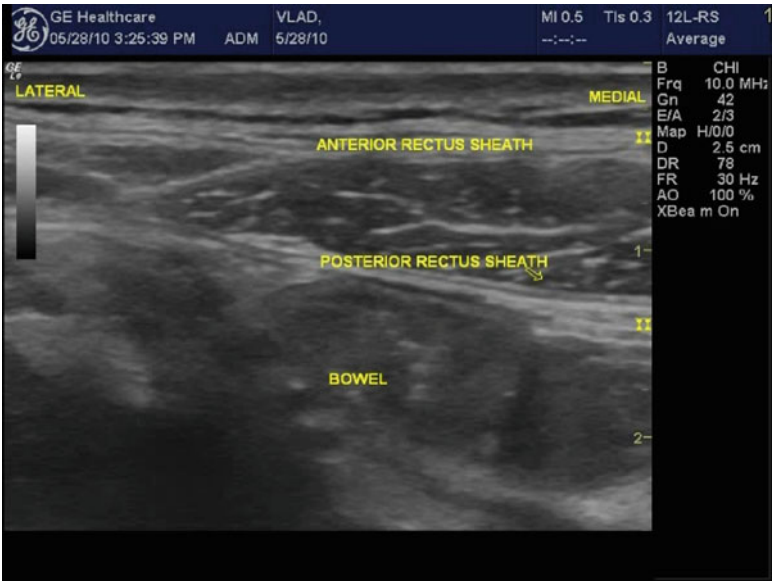

Fig. 16.27 Ultrasound image of rectus abdominis muscle. Note the close proximity of the peritoneum and bowel to the muscle

Local Anesthetic

20–30 cc of 0.2–0.5% ropivacaine depending on the size of the incision and patient's weight.

Technique

The skin over the umbilicus and rectus muscles is prepped and draped in sterile fashion. The ultrasound probe is placed in transverse orientation to the longitudinal rectus muscles and just above the umbilicus (Fig. 16.26). The probe locations should be 2–5 cm lateral to the midline.

The anterior and posterior borders of the rectus sheath are identified on ultrasound (see Fig. 16.27). The needle is inserted in-line and lateral to the probe. The needle is positioned near the lateral edge of the rectus sheath. The injection of local anesthetic should be done under the belly of the rectus sheath muscle and above the posterior rectus sheath. Another option is to place half the volume of the local anesthetic anterior to the rectus muscle and half the volume posterior to the rectus muscle. The procedure should be repeated on the other side of the abdomen as well. If the continuous technique is chosen, the catheter can be placed posterior to the rectus muscle.

Precautions and Fine Points

- Again, the utmost care should be taken so as not to penetrate the posterior rectus sheath and enter the peritoneal space with the needle.
- Local anesthetic should not be deposited into the muscle mass of the rectus. The blockade will be ineffective.

Ilioinguinal and Iliohypogastric Block

Anatomy and Indications

The blockade of ilioinguinal (IL) and iliohypogastric (IH) nerves is employed for analgesia during inguinal hernia repair, orchiopexy, and hydrocelectomy [23–25]. When employed for the aforementioned procedures, this block is just as effective as a caudal block with fewer complications. The IL-IH block cannot be used as the only anesthetic for the surgery because the ilioinguinal and iliohypogastric nerves do not cover visceral pain from peritoneal traction and manipulation of the spermatic cord. The ilioinguinal (L1) and iliohypogastric (T12 and L1) nerves are branches of the lumbar plexus and innervate the scrotum and inner thigh. The nerves are located in the space between the iliacus and psoas muscles. Distally, the nerves emerge from the transversus abdominis muscle near the anterior superior iliac spine (ASIS) and then travel between the transversus abdominis muscle and internal oblique muscle. There are very few complications due to this block and involve colonic and small-bowel perforation. The complications were reported before the ultrasound technology was used for this block. After the placement of the block below the inguinal ligament, one report cited a motor block of the quadriceps muscle. If the IL-IH block is performed blindly, the success rate of correct placement is only 14% [26]. With the use of ultrasound technology, the effective volume of local anesthetic can be reduced to 0.075 cc/kg [27].

Patient Position

Supine.

Transducer

25- or 35-mm linear probe oscillating at 13 MHz.

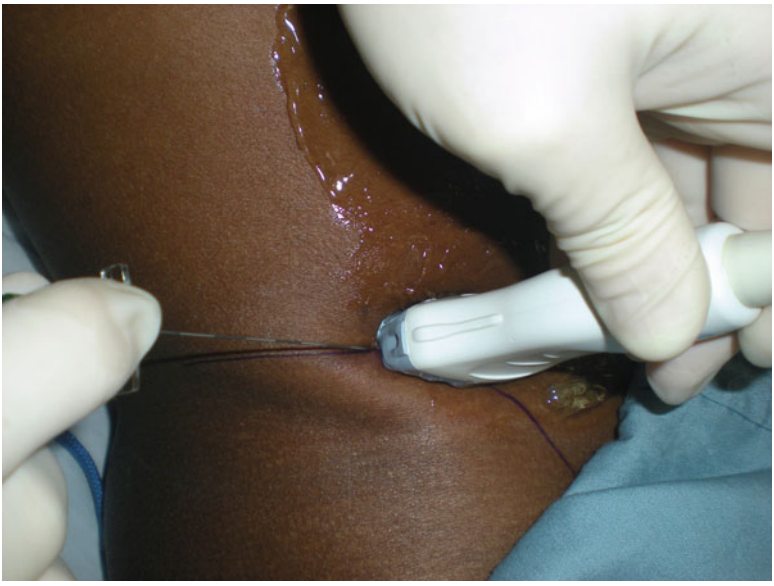

Fig. 16.28 The probe and needle position for the ilioinguinal (IL) and iliohypogastric (IH) nerve blocks. The transducer is positioned just above and lateral to the anterior superior iliac spine

Orientation of Transducer

Transverse oblique.

Needles

24-gauge, 2.5-cm or 22-gauge, 5-cm blunt or insulated needle, or 22-gauge 8-cm Tuohy needle.

Technique

The area lateral to the ASIS is sterile washed and draped. The ultrasound probe is placed immediately lateral and just above the ASIS (see Fig. 16.28). The needle is inserted in place to the transducer.

The ultrasound image of the transversus abdominis and internal oblique muscles is obtained. The plane where the IL and IH nerves are located is between the

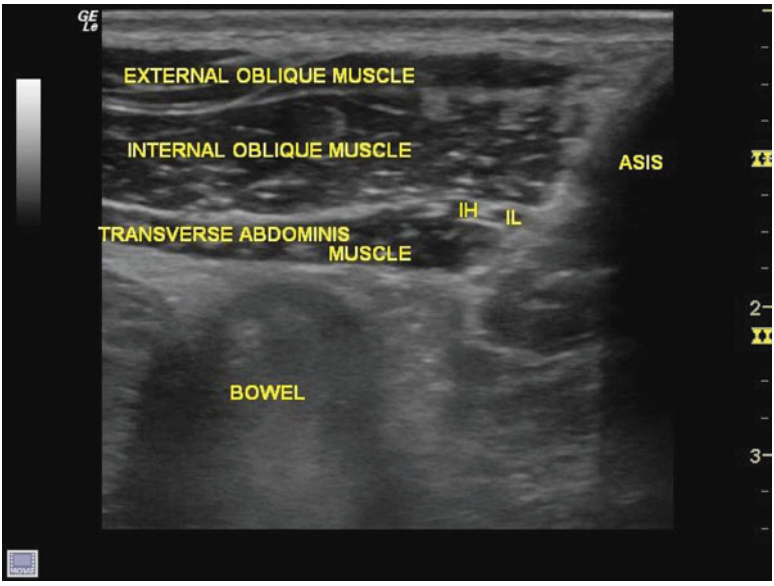

Fig. 16.29 Ultrasound image of iliohypogastric (IH) and ilioinguinal nerves. Please note the proximity of the bowel (*ASIS* anterior superior iliac spine)

transversus abdominis and internal oblique muscles (Fig. 16.29). Uncommonly, the IL and IH can be found between the plane of external oblique and internal oblique muscles. The needle is placed in-line at the lateral or medial end of the probe and directed toward the nerves.

The local anesthetic is positioned next to the nerves. If one cannot readily visualize the nerves, the local anesthetic can be deposited in the plane between the transversus abdominis and internal oblique muscles. Due to the anatomic variation, the nerves may be located between the external and internal oblique muscles. Therefore, one may inject local anesthetic in the plane between the external and internal oblique as well as the plane between the internal oblique and transversus abdominis muscles.

Precautions and Fine Points

- Again, the utmost care should be taken so as not to penetrate the posterior rectus sheath and enter the peritoneal space with the needle.
- Local anesthetic should not be deposited into the muscle mass of the rectus. The blockade will be ineffective.

Multiple-Choice Questions

1. After placing a paravertebral catheter for a thoracotomy, a test dose of local anesthetic is injected through it. During the injection of 1 mL, the patient suddenly begins to cough and is noted to have some bronchospasm which is relieved within a few minutes. Bronchodilators were ordered. The next step would be:
   (a) Inject more local anesthetic.
   (b) Send the patient for surgery and evaluate the catheter intraoperatively under direct vision.
   (c) Pull out the catheter. Order a chest X-ray.
   (d) Secure the catheter and further assess after surgery since time is important.

2. A 90-year-old patient with sleep apnea is scheduled for a scapulectomy. The surgeon informs you that the patient is to be anticoagulated 24 h later. The best method for managing the postoperative pain is:
   (a) C7–C8 epidural
   (b) C7 paravertebral catheter
   (c) Intraoperative ketamine, narcotics with a postoperative PCA
   (d) Postoperative PCA

3. You are called from the floor that the patient who had paravertebral single-shot block for prostatectomy earlier today complains about bilateal leg weakness. You will:
   (a) Order MRI.
   (b) Administer 1 mg of morphine.
   (c) Order lumbar spine X-ray.
   (d) Do nothing; it is common.
   (e) Check patient in 2 h. If weakness is still present, order neurology consult.

4. What is the content of the paravertebral space?
   (a) Intercostal (spinal) nerve
   (b) Rami communicantes
   (c) Dorsal ramus
   (d) Sympathetic chain
   (e) Intercostal vessels
   (f) All the above

5. What are the factors increasing the likelihood of epidural spread of PVNB?
   1. Needle position – proximity to the intervertebral foramen
   2. Bevel turned laterally
   3. Anatomy – lack of foraminal stenosis, lateral disc bulging, zygapophysial joint hypertrophy, or epidural fibrosis
   4. Low pressure gradients – small volume and low speed of injection

(a) 1, 2, 3
(b) 1, 3
(c) 2 ,4
(d) 4
(e) 1, 2, 3, 4

6. The psoas major does not form a caudal boundary and seal off the thoracic paravertebral space:
   (a) True
   (b) False

7. A linear relationship was established between paravertebral depth and thoracic level, with depth increasing at inferior thoracic levels with the exception of:
   (a) T1
   (b) T5
   (c) T7
   (d) T12
   (e) L2

8. The lumbar triangle of Petit is bounded posteriorly by:
   (a) The external oblique muscle
   (b) The internal oblique muscle
   (c) Iliac crest
   (d) Rectus abdominis muscle
   (e) The latissimus dorsi muscle

9. After placement of bilateral paravertebral block at T6 level, patient developed hypotension. Possible causes are all of the following, except:
   (a) Pneumothorax
   (b) Stimulation of sympathetic chain in the paravertebral space
   (c) Vagal reflex
   (d) Bilateral sympathetic blockade from epidural spread
   (e) Allergic reaction to local anesthetic

10. The insertion point for the lateral approach to paravertebral block:
    (a) 2.5 cm lateral to the spinal process
    (b) 5 cm lateral to the spinal process
    (c) 8 cm lateral to the midline
    (d) In mid-axillary line

11. For paravertebral nerve blocks, there is no difference between bupivacaine and lidocaine in terms of VAS at rest, morphine requirements, or postoperative pulmonary function:
    (a) True
    (b) False

12. The equipment necessary for continuous paravertebral block placement include all of the following, except:
    (a) No. 11 scalpel blade
    (b) 18-gauge Tuohy needle
    (c) Multiorifice catheter
    (d) Chlorhexidine preparation

13. To compare thoracic paravertebral and epidural blocks in patients undergoing thoracotomy, paravertebral nerve block:
    (a) Is comparable to thoracic epidural analgesia
    (b) Decreases VAS scores
    (c) Decreases morphine consumption
    (d) Increases PFTs

14. Paravertebral nerve block may be performed by the following technique, except:
    (a) Nerve stimulation
    (b) Paresthesia
    (c) Ultrasound
    (d) Loss of resistance

Answers:

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

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17

Head and Neck: Scalp, Ophthalmic, and Cervical Blocks

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Contents

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Even though head and neck blocks are among the easiest to perform due to constant and reliable landmarks, they are still infrequently used by anesthesiologists in the operating room. This is in part because general anesthesia offers a safe and easy alternative for most surgeries involving these anatomical areas. Nonetheless, neural blockade has become the mainstay of anesthetic techniques for (a) most ophthalmologic cases, (b) neurosurgical procedures or carotid endarterectomies where intraoperative neurological assessment is required, and (c) a safe alternative for patients with low functional reserve that would have a poor tolerance to general anesthesia. These blocks can also prove to be useful for the anesthesiologists themselves, in techniques for airway management in awake patients.

In this chapter, we will review blocks that are relevant to the anesthesiologist working in the operating room, focusing on acute and surgical pain management. Many other resources will provide you with information about blocks of the head and neck for chronic pain patients.

The Scalp Block/Block of the Head

The scalp block is not a block in itself but a combination of multiple nerve blocks, namely, the supraorbital, supratrochlear, greater auricular, auriculotemporal, and greater and lesser occipital nerve blocks.

Anatomy

The innervation of the scalp can be divided into anterior and posterior distributions: anteriorly, from branches of the trigeminal nerve (fifth cranial nerve), and, posteriorly, from branches of the cervical plexus and posterior spinal rami (Fig. 17.1).

Trigeminal Nerve Branches

All originating from the gasserian ganglion, the branches of the trigeminal nerve are the supraorbital and supratrochlear nerves (branches of V1 – traveling within the conus of the eye) innervating the forehead, and the zygomaticotemporal nerve (V2) and the auriculotemporal nerve (V3) innervating the temporal area, and, for the latter nerve, the anterior part of the ear.

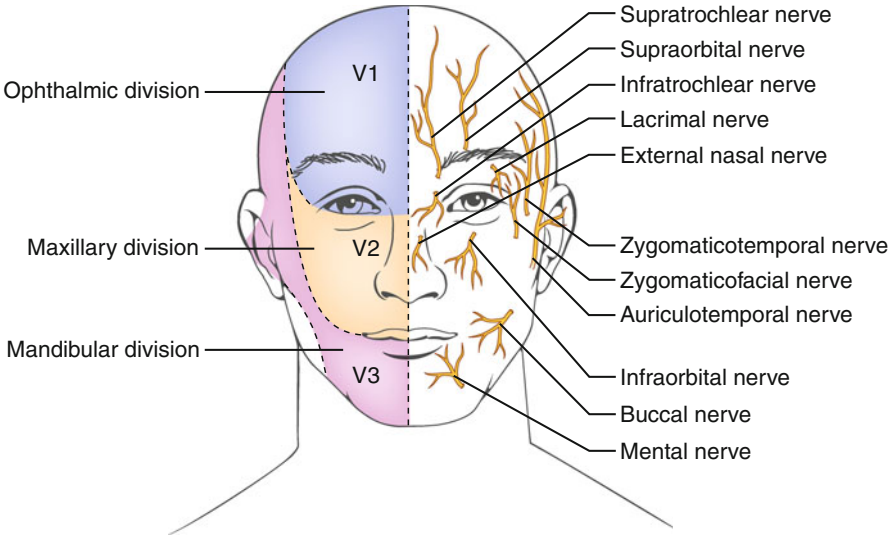

Fig. 17.1 Innervation territories of the scalp

Occipital Nerves

The greater auricular nerve and lesser occipital (from the superficial cervical plexus) and the greater occipital nerve (originating from the dorsal primary ramus of C2) are responsible for the sensory innervation of the posterior aspect of the head (Fig. 17.2). The greater occipital nerve innervation area covers the skin located between the nuchal line and the vertex of the skull, whereas the lesser occipital and greater auricular innervate the skin behind/below the ear and in front (over the parotid) of the ear, respectively.

Indications

A. Awake craniotomies [1]
B. Stereotactic biopsies
C. Attenuation/prevention of the sympathetic stimulation and stress response associated with Mayfield pins insertion [2, 3]
D. Postoperative analgesia for craniotomies [4, 5]
E. Plastic procedures of the scalp [6]

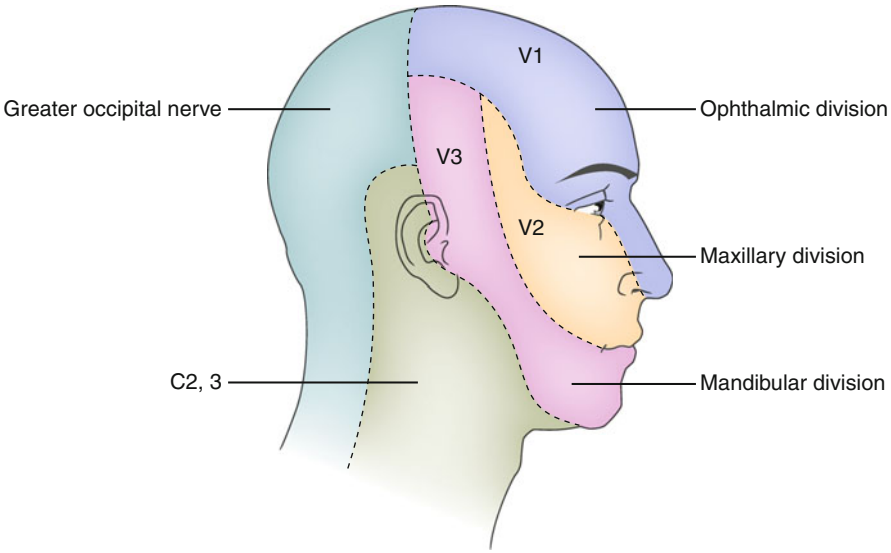

Fig. 17.2 Specific areas of innervation of the scalp

Local Anesthetics

Most local anesthetics are suitable for facial nerve blocks, and less concentrated local anesthetics will work well on these sensory nerves; 0.5% ropivacaine, 0.25% bupivacaine, or 1% lidocaine would be good choices. Epinephrine is a good adjunct to identify an inadvertent intravascular injection and to lower the possibility of local bleeding at the puncture site.

Techniques

As with all neural blockade techniques, the patient should be monitored, and appropriate resuscitative drugs and equipment should be readily available. A strict sterile technique is not always possible (for blocks performed in hair-covered areas), but the technique should be as aseptic as possible.

Supraorbital and Supratrochlear

1. After palpation of the supraorbital foramen, slightly medial to the plane of the centered pupil on the superior orbital rim (approximately 2–2.5 cm lateral to midline), a 25-G 30-mm needle is inserted above or below the eyebrow toward the foramen (but not entering it) (Fig. 17.3).

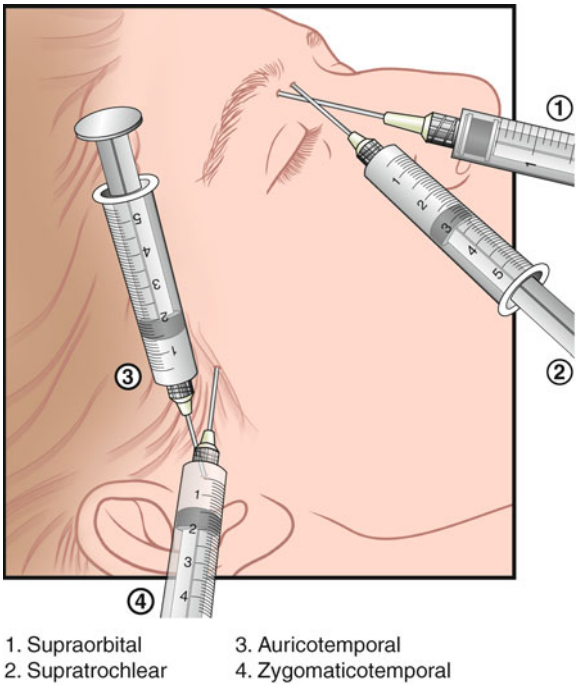

Fig. 17.3 Anterior scalp blocks

2. A subcutaneous infiltration of 2–3 mL of local anesthetic solution suffices, followed by local pressure to prevent hematoma formation.
3. The supratrochlear nerve can be specifically blocked at the intersection of the superior orbital rim and the nasal bridge, with the needle aiming at this intersection, pulled back slightly after bony contact, then injecting 1 mL of solution (Fig. 17.3). An alternative is to infiltrate local anesthetics subcutaneously from the supratrochlear notch to the nasal bridge.

Auriculotemporal

A 25-G needle is inserted just between the tragus and the superficial temporal artery, just on the posterior aspect of the zygomatic bone. 3–5 mL of anesthetic solution is injected (Fig. 17.3).

Zygomaticotemporal

A 25-G needle is inserted 2.5 cm anterior to the tragus, perpendicular to all planes, and injection of 3–5 mL of anesthetic solution is performed deep to the fascia and superficially as the needle is withdrawn (Fig. 17.3).

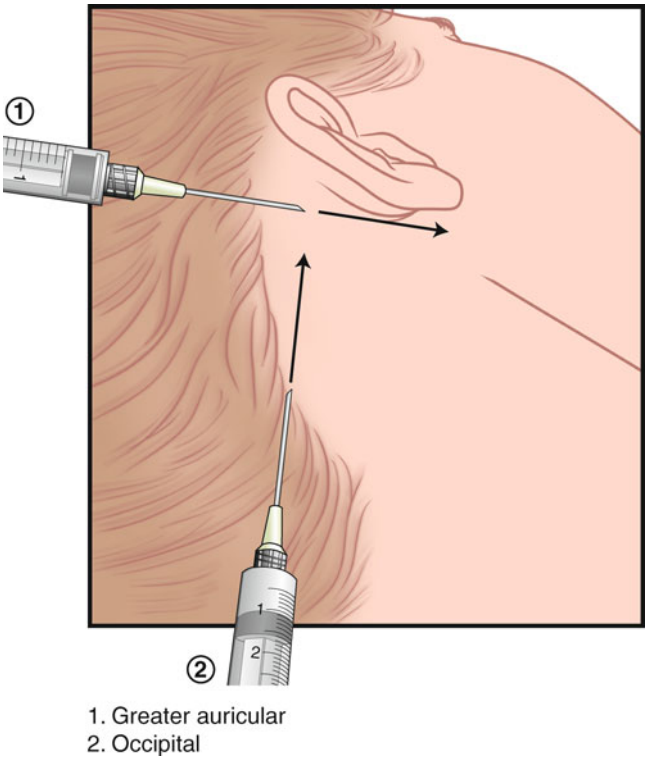

Fig. 17.4 Posterior scalp blocks

Greater Auricular

A 25-G needle is used to create a subcutaneous wheal of local anesthetics following the posterior aspect of the ear, over the mastoid process, with an approximate volume of 5 mL (Fig. 17.4).

Greater and Lesser Occipital Nerve

1. After palpation of the occipital artery, approximately midway between the occipital protuberance and the mastoid process, a bended 25-G needle is inserted lateral to the occipital artery toward the mastoid process.
2. A subcutaneous infiltration is made lateral to this point, following the nuchal line, using 3–5 mL of solution, after negative aspiration for blood, as the needle is withdrawn (Fig. 17.4).

Risks and Complications

All these blocks yield risks related to local anesthetics hypersensitivity, hematoma formation, and infection. The supratrochlear and supraorbital nerve blocks have an additional potential risk for intraneural injection. The clinician should recognize the risk of intravascular injection with the auriculotemporal and greater occipital nerve blocks.

Ophthalmologic Blocks

In many countries, the blocks of or around the eye are performed by the ophthalmologist. This section will focus on the two more relevant techniques for the anesthesiologist, the retrobulbar and peribulbar blocks. Those blocks are not suitable for open globe surgery, and the reader should note that less invasive surgical techniques used nowadays also permit topical anesthesia to be sufficient for some lens and anterior chamber procedures.

Anatomy

Like most structures of the head, branches from the trigeminal nerve give sensory innervation to the eye and adnexa. However, ophthalmologic surgery requires immobility as much as insensibility.

A. Motor innervation is supplied by the oculomotor nerve (CN III), trochlear nerve (CN IV), and abducens nerve (CN VI), as well as by the temporal division of the facial nerve (CN VII) to the orbicularis oculi muscle.
B. Sensory innervation is provided by branches emerging from the ophthalmic (V1) branch of the trigeminal nerve through the ciliary ganglion.
C. The branches of the ophthalmic division of the trigeminal and most of these previously mentioned nerves travel in the orbit behind the globe, which provides a space for local anesthetic deposition (Fig. 17.5). All the nerves can thus be blocked here except for the temporal division of the facial nerve. The main difference between the two discussed techniques is the site of injection of the local anesthetic solution, intraconal (inside the cone formed by the rectus muscles) for the retrobulbar or extraconal for peribulbar. The reader should note that the cone is anatomically incomplete and there is no anatomic structure linking the extrinsic ocular muscles together [7]. Clinically, the peribulbar block is considered safer [8] than the retrobulbar, since the structures at risk are intraconal. It also has the advantage of providing a complete akinesia of the eye adnexa, owing to direct diffusion to the eyelids. However, the success rate of the peribulbar block could be lower (this is controversial, many studies showing similar success rate [8, 9])

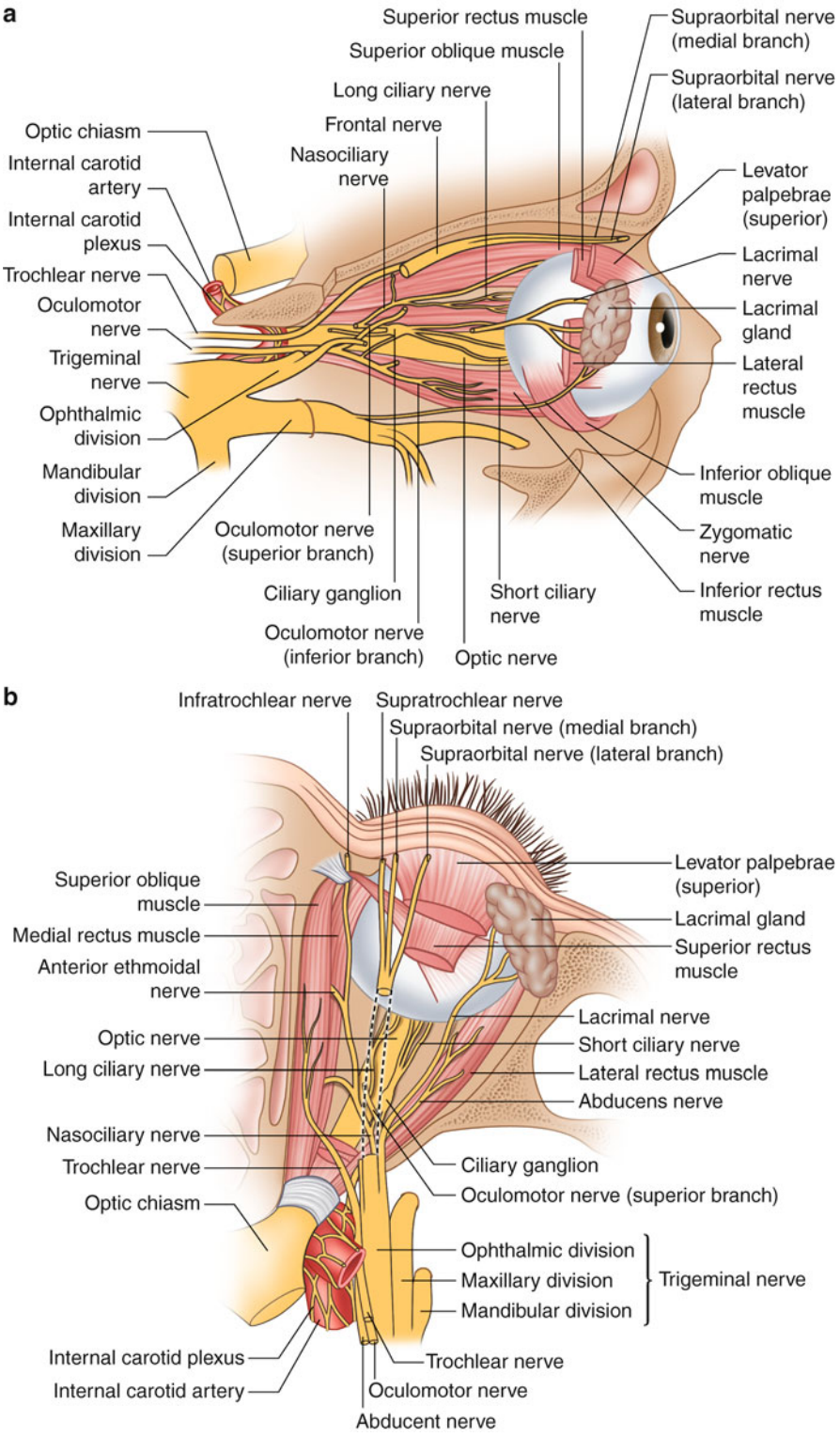

Fig. 17.5 Innervation of the eye and adnexia

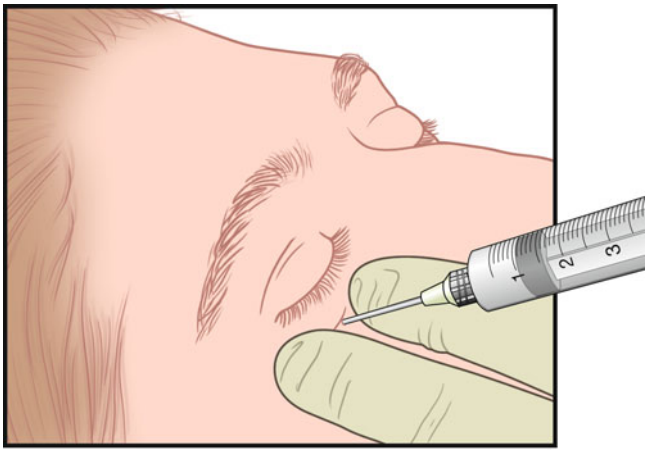

Fig. 17.6 Retrobulbar anesthesia

and necessitates a larger volume of solution compared to the retrobulbar block. The onset of the peribulbar block is also slower than that of the retrobulbar block due to the time needed for diffusion of the local anesthetic [10].

Local Anesthetics

A. For shorter cases, 2% lidocaine is used, and 0.5% bupivacaine or 0.5% ropivacaine can be used for longer procedures.
B. Use of adjuncts such as hyaluronidase, bicarbonates, and epinephrine is controversial and could be considered for selected cases.

Techniques

As with all blocks, patients should be monitored and resuscitation drugs and equipment should be ready for use. A strict aseptic technique should be used.

Retrobulbar

1. With the patient's gaze neutral or oriented inferonasally, thus pulling the optic nerve and meningeal sheath away from the injection site, a 25-G 30-mm short-bevel needle is inserted at the inferolateral side, transpalpebral, tangentially to the globe, until the equatorial line of the globe is reached (approximately 1.5–2 cm) (Fig. 17.6).

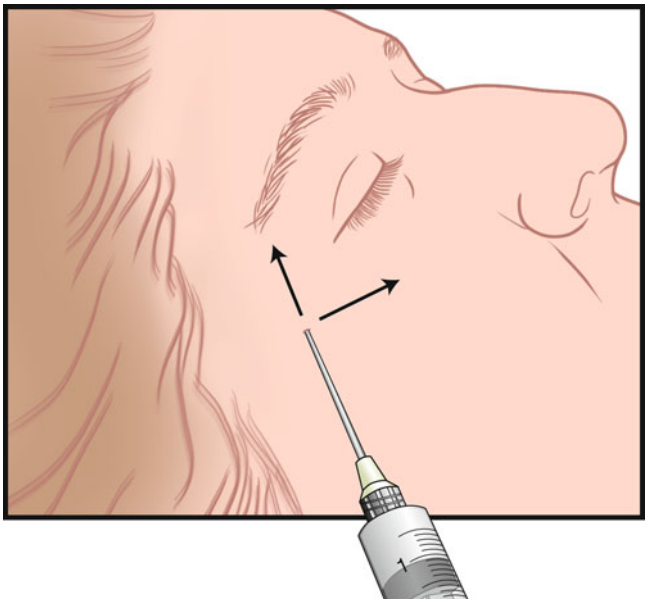

Fig. 17.7 Block of the temporal branch of the facial nerve (CN VII)

2. The needle is then redirected superiorly, toward the point situated just below the apex of the orbit and advanced for a maximum depth of 25–30 mm. The eye will rotate slightly inferiorly and will suddenly return to neutral position as the conus is entered. Keeping the bevel facing the sclera reduces the risk of globe perforation.
3. After a negative aspiration test, 3–4 mL of local anesthetic solution should be injected slowly and painlessly. There should be minimal resistance to injection.
4. After injection, intermittent mechanical pressure should be applied on the globe for 5–10 min to promote even distribution of local anesthetic.
5. For complete akinesia, this block needs supplementation of the temporal branch of the facial nerve to avoid orbicularis oculi movement during the surgical procedure. A 25-G needle is inserted 1 cm lateral to the lateral canthus. Then a subcutaneous infiltration is made along the inferior and the superior orbital border, with 2–3 mL of solution on each side (Fig. 17.7).

Peribulbar

1. A 25-G 30-mm short-bevel needle is inserted at the junction of the middle and the lateral thirds of the lower lid and directed to the equator of the globe (Fig. 17.8).
2. After a negative aspiration test, 5 mL is injected.
3. In most patients, this inferolateral injection alone produces satisfactory anesthesia and akinesia [11]. If not, a superonasal approach can be added. This is essentially

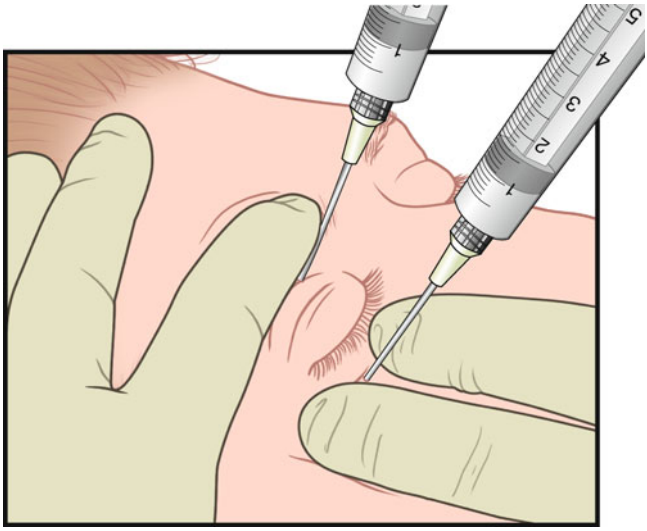

Fig. 17.8 Peribulbar anesthesia, inferolateral and superonasal approach

the same approach, but this time, the insertion site is at the intersection of the medial and middle thirds of the upper lid (Fig. 17.8). Note that the onset time for surgical block can be as long as 20 min [10].

Risks and Complications

A. Retrobulbar [9, 12]:

1. Trauma to adjacent structures
2. Globe perforation
3. Retrobulbar hemorrhage
4. Optic nerve injury
5. Oculocardiac reflex
6. Misplaced injections
7. Intra-arterial injection, with associated seizures
8. Subarachnoid injection

B. Peribulbar [8, 12]:

1. Trauma to adjacent structures
2. Globe perforation
3. Peribulbar hemorrhage
4. Central retinal artery occlusion
5. Oculocardiac reflex
6. Toxic injury to rectus muscles, with persistent paresis [13]

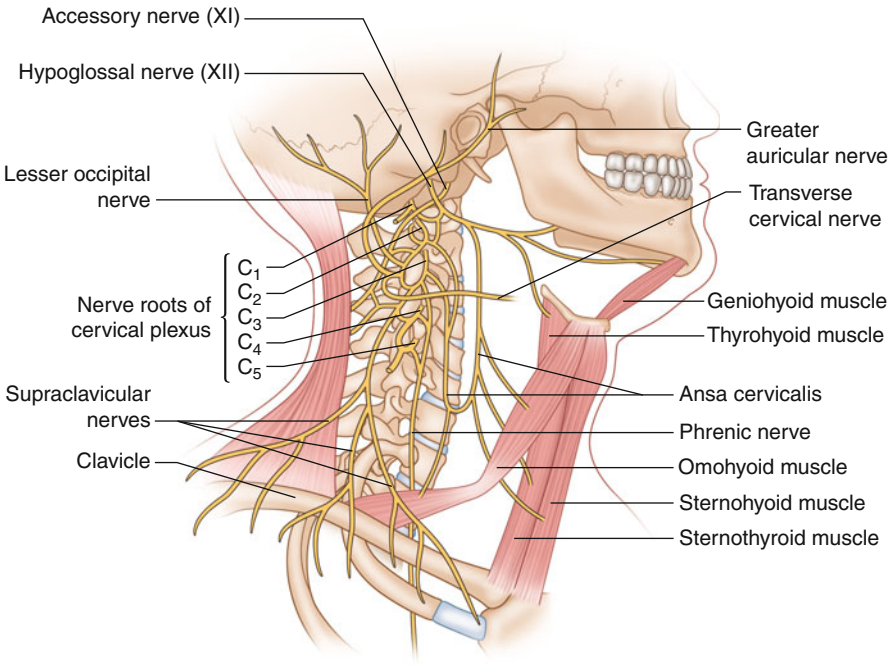

Fig. 17.9 Anatomy of the cervical plexus

Cervical Blocks

Cervical blocks are useful for anterior neck surgery, such as carotid endarterectomy, thyroidectomy, and lymph node biopsies. Superficial plexus block is the most frequently performed technique because of the ease of performance and low risk. Deep cervical plexus block has been less popular because some evidence has shown no added benefits to superficial plexus block for carotid and thyroid surgery, but a definitive increase in risk [13, 14]. Recently, some authors have advocated the use of a high interscalene block to obtain the same endpoints as with the formal deep cervical plexus block with one injection site and more complete block [15].

Anatomy

A. The cervical plexus originates from the anterior rami of C1–C4 spinal nerves, which emerge from the intervertebral foramen behind the vertebral artery. Superficial branches come out on the posterior aspect of the sternocleidomastoid to give sensory innervation to the skin via the lesser occipital, the great auricular, the transverse cervical, and the supraclavicular nerves.
B. The deep branches innervate deeper structures of the neck and participate in the constitution of the phrenic nerve (Fig. 17.9).

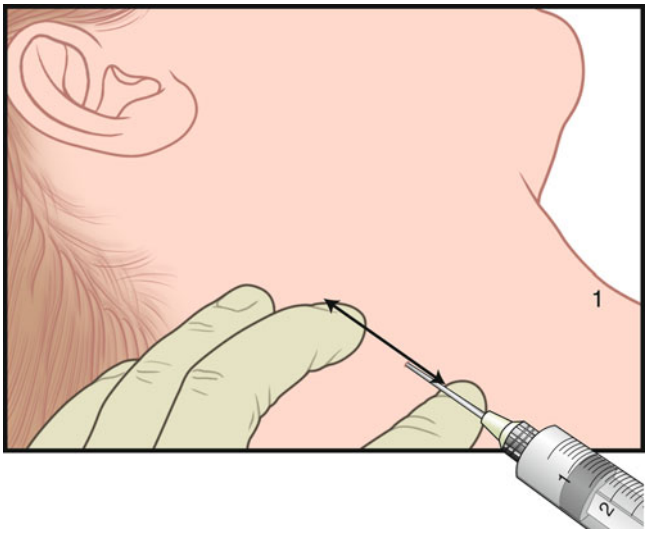

Fig. 17.10 Superficial cervical plexus block. A subcutaneous infiltration is made along the posterior border of the sternocleidomastoid muscle

Local Anesthetics

Good choices for the superficial cervical plexus block are 1% lidocaine or 0.25% bupivacaine, whereas 2% lidocaine, 0.5% ropivacaine, or 0.5% bupivacaine are more suitable for a deep cervical plexus block or for the high interscalene block. The solution used for the two latter blocks should contain at least 2.5 μg/mL of epinephrine in order to identify inadvertent intravenous injection (intra-arterial injection will be obvious with or without epinephrine).

Techniques

Superficial Plexus Block

The needle is inserted at the intersection of a line drawn horizontally from the cricoid cartilage and a vertical line drawn along the posterior border of the clavicular head of the sternocleidomastoid muscle. Five milliliter of local anesthetics is infiltrated along the middle third (Fig. 17.10).

Deep Plexus Block

This block is a cervical paravertebral block. Three injections have to be done on the transverse process of C2, C3, and C4.

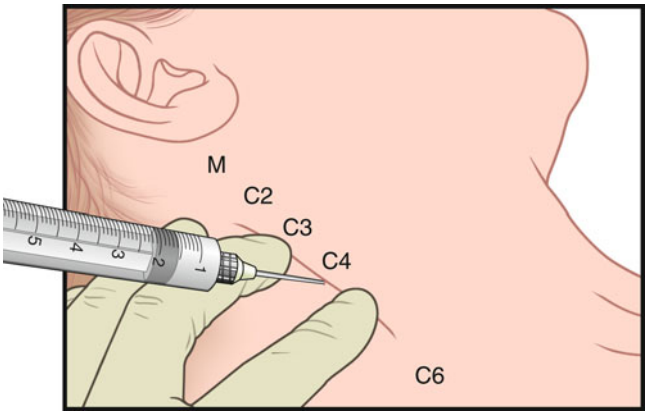

Fig. 17.11 Deep cervical plexus block

1. The insertion points are located on a vertical line drawn between the mastoid process and the C6 transverse process (Chassaignac's tubercle), which is in the same craniocaudal plane as the cricoid cartilage.
2. The lower border of the mandible is the approximate level of C4, which is marked on the line.
3. The space between the mastoid process and this point can now be divided into thirds, showing the insertion points for C2 and C3.
4. The 22-G needle is inserted in the posterio-medio-caudad direction until contact is made with the transverse process.
5. An aspiration test should be made to ensure that the vertebral artery or the subarachnoid space has not been punctured, and only then 3–4 mL of local anesthetic solution should be injected per level (Fig. 17.11).

High Interscalene

This approach provides the opportunity of using a single injection technique to block the cervical plexus (deep and superficial components).

1. The interscalene groove is palpated and marked, and the needle is inserted at the highest point of the interscalene groove usually coinciding with a horizontal line from the lower angle of the jaw (C4), with a posteromedial and slightly caudad angle (Fig. 17.12).
2. Levator scapulae movement as demonstrated by elevation and internal rotation of the scapula should be sought with the nerve stimulator.
3. A 30-mL volume should be used, and digital pressure should be applied caudally to the needle to promote cephalad diffusion. This block can also be ultrasound-guided [16].

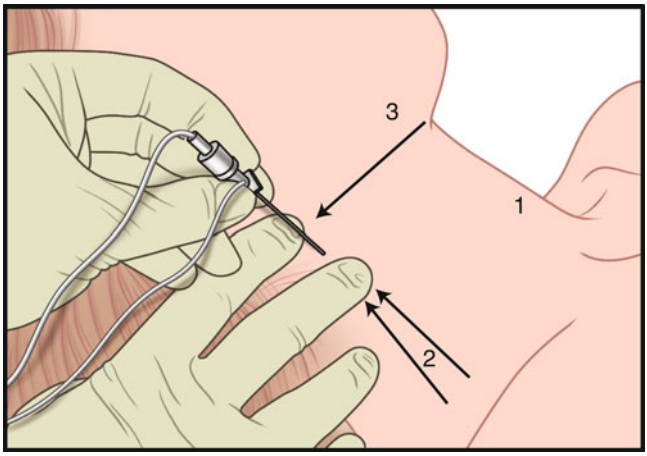

Fig. 17.12 High interscalene block

Risks and Complications

A. Superficial cervical plexus block is a low-risk procedure, the main risk being inadvertent intravascular injection.
B. Deep cervical plexus block and high interscalene:

1. Intravertebral injection, seizures
2. Intrathecal injection, total spinal anesthesia/brainstem anesthesia
3. Phrenic and recurrent laryngeal nerve block
4. Brachial plexus blockade
5. Hematoma

Airway Blocks

These blocks are particularly useful for the anesthesiologist performing an awake fiber-optic intubation and for tracheotomy and bronchoscopy. Blocks involve the laryngeal branches of the vagus nerve, namely, the superior laryngeal nerve and the recurrent laryngeal nerve.

Anatomy

Superior Laryngeal Nerve

The laryngeal surface of the epiglottis and the laryngeal inlet, down to the vocal folds, are innervated by the superior laryngeal nerve, which leaves the vagus trunk,

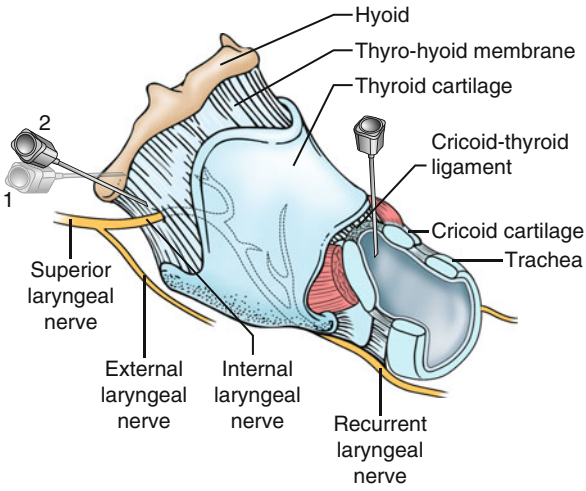

Fig. 17.13 Innervation of the larynx

crosses the greater cornu of the hyoid, then penetrates the thyrohyoid membrane. This nerve also has a motor component (external branch) innervating the cricothyroid muscle.

The Recurrent Laryngeal Nerve

The recurrent laryngeal nerve, as its name suggests, is a branch of the vagus nerve traveling lower down the thorax then ascending in the neck between the trachea and the esophagus. This nerve provides sensory innervation of the larynx below the vocal cords and the trachea, and motor innervation to all intrinsic muscles of the larynx, except the cricothyroid muscle (Fig. 17.13).

Glossopharyngeal Nerve

The other structures of the oropharynx and the ventral portion of the epiglottis are innervated by the glossopharyngeal nerve, which can be blocked easily by a number of noninvasive techniques including mouthwash/gargles with local anesthetics mixture or spraying of local anesthetics.

Local Anesthetics

The short duration and onset time of 2% lidocaine makes it perfect for most airway blocks, except for the gargles which are usually made with a 4% viscous lidocaine preparation.

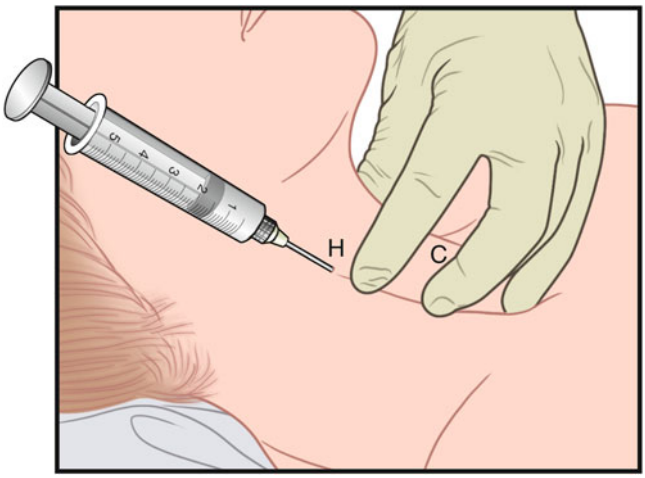

Fig. 17.14 Superior laryngeal nerve block. *H* hyoid cornu, *C* cricoid cartilage

Techniques

Superior Laryngeal Nerve

1. A 25-mm 25-G needle is inserted over the greater cornu of the hyoid bone and is then walked off the caudad aspect of it until the thyrohyoid membrane is pierced. The needle should not go deeper than the hyoid itself.
2. After aspiration, 3 mL of 1% lidocaine is injected.
3. To get precise landmarks, the hyoid should be slightly pushed toward the site to be blocked while the cricoid cartilage is held medial (Fig. 17.14).

Recurrent Laryngeal Nerve

Although this nerve can be blocked directly, it is easier to use a translaryngeal technique to approach.

1. A 20-G needle is introduced in the cricothyroid membrane until air is aspirated.
2. Then the local anesthetic solution (usually 3–5 mL of 2% or 4% lidocaine) is quickly injected and the needle immediately withdrawn (to avoid trauma as the patient coughs).

Risks and Complications

A. Systemic toxicity.
B. Aspiration of gastric content, since airway reflexes will be abolished.

Clinical Pearls

The Scalp Block/Block of the Head

- Scalp block is an effective means of preventing the elevation of blood pressure and associated elevation of intracranial pressure when Mayfield pins are inserted for head positioning [2, 3].
- Scalp blocks are painful, and appropriate sedation should be given to patients before proceeding.

Opthalmologic Block

- Oculocardiac reflex is most frequent in the pediatric and geriatric populations. Patients with preexisting high vagal tone, stressed, or on beta blockers, are also considered to be higher-risk patients.
- Highly myopic patient is at increased risk for globe perforation, so a discussion with the ophthalmologist before blockade can help define the relative risk for a given patient.

Cervical Blocks

- The superficial cervical plexus block alone is often enough for most cervical surgeries, provided that the surgeon infiltrates the deeper structures as he dissects through them [13, 14].
- The caudad orientation of the needle is the key to avoid penetration of the subarachnoid space and vertebral artery during deep cervical plexus and high interscalene block.
- For carotid endarterectomy surgery, the surgeon must additionally infiltrate directly around the carotid artery to block autonomic responses associated with manipulation of the carotid. At the present time, however, direct infiltration into the carotid sinus is not recommended for prevention of postoperative hemodynamic lability [17].
- If akinesia of the trapezius is indicated, the accessory nerve can be blocked with the same insertion point as for the superficial plexus block, with the needle penetrating just below the fascia, perpendicular to all planes.

Airway Block

- A mouthwash with local anesthetics is as effective as the glossopharyngeal block and better tolerated to produce anesthesia of the pharyngeal structures [18].

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18

Local Anesthesia of the Masticatory Region

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Contents

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Introduction

Local anesthetics are used in a wide range of clinical situations in dentistry. Some indications are to alleviate sensory input thus minimizing discomfort during treatment procedures, to treat inflammatory and chronic pain, and for diagnostic and prognostic purposes. Knowledge of the scope of anesthesia effects and specific anatomical factors associated with proper technique is essential for their safe and efficacious use in order to achieve intended goals.

The trigeminal nerve provides the vast majority of sensory innervation to the masticatory region. The cell bodies for the sensory fibers comprise the semilunar (Gasserian) ganglion, which lies in Meckel's cave in the inferior-medial aspect of the middle cranial fossae. Originating in the ganglion are the three major branches of nerve: the ophthalmic, maxillary, and mandibular divisions. Knowledge of the neural pathways of the maxillary and mandibular divisions is essential for effective deposition of local anesthetic agents.

Maxillary Nerve Anesthesia

The maxillary nerve is purely sensory. It passes through the foramen rotundum to enter the pterygopalatine fossa. Branches of the maxillary nerve supply sensory innervation to the hard (teeth and bone) and soft (mucosa and gingival) tissue of the upper jaw. The following nerve blocks are those primarily utilized in daily practice.

The Posterior Superior Alveolar Block

The area anesthetized by this block includes much of the posterior and lateral aspects of the tuberosity of the maxilla with its associated mucosa. Pulpal anesthesia for the maxillary second and third molars and the distal and palatal roots of the maxillary first molar is also achieved with this block. The placement of the needle for administration of the anesthetic is posterior and superior to the maxillary second molar. The needle is directed toward the distal aspect of the maxillary tuberosity where branches of the posterior superior nerve enter into the maxilla via their respective alveolar foramina (Fig. 18.1).

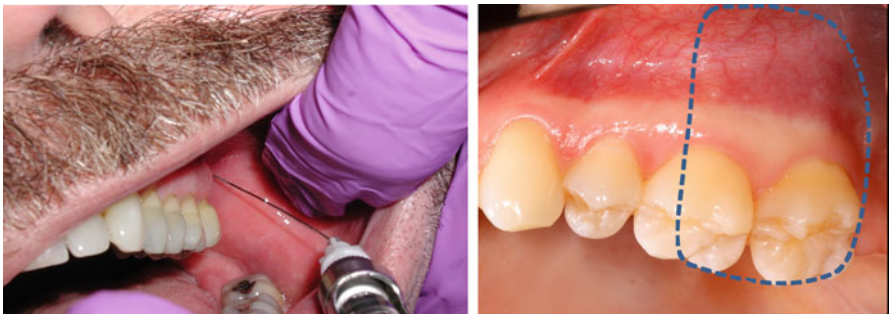

Fig. 18.1 Area anesthetized utilizing the posterior superior alveolar nerve block

The most common complication for anesthetic injections into the region involves needle trauma to the superior alveolar venous plexus or posterior superior artery, which results in hemorrhage, swelling, and hematoma formation. The hematoma may become visible externally on the face. Harn et al. describe a triangle of safety just superior to the maxillary second molar that is 99% free of significant vascularization [1], which entails reduced risk of vascular compromise. Harn reports decrease in hematoma formation with precautions. Precautions include placement of the needle with the bevel toward the periosteum and utilization of an aspirating syringe in order to avoid intravascular injection of anesthetic. It is important that needles be replaced if bone is bumped during an injection or when several injections are to be performed. The needle tip can become barbed or jagged which will increase the risk to neuronal or vascular tissues in the region. Another rare complication is transient diplopia or strabismus, which can be very disconcerting to the individuals involved [2]. No absolute explanation has been made for this type of complication. The distal placement of the anesthetic needle does also present a very small risk for needle breakage; however, the risk is much less than that of the inferior alveolar nerve block [3].

The Middle Superior Alveolar Block

The area anesthetized with this block includes lateral aspect of the maxillary alveolar process with its associated mucosa. The middle superior alveolar nerve provides innervation for the mesiobuccal root of the maxillary first molar, the second premolar, and likely the first premolar. The posterior superior alveolar nerve and middle superior alveolar nerves may form an anastomosis, which results in the superior dental plexus, which can then provide joint innervations to the maxillary posterior teeth. The middle superior alveolar nerve is contained in a thin “rib” of bone in the lateral aspect of the maxillary sinus. When performing this block, the needle is placed superior to the first maxillary molar anterior to the zygomatic process of the

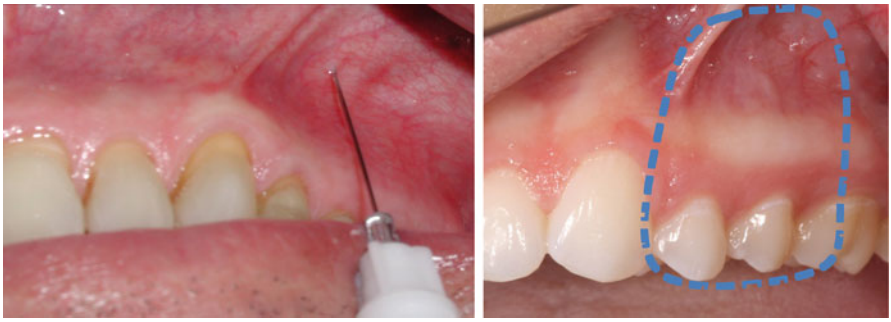

Fig. 18.2 Area anesthetized utilizing the middle superior alveolar nerve block

maxilla high in the buccal vestibule under the mucosa where approximately 1 ml of anesthetic is deposited. There are few reported adverse reactions associated with the middle superior alveolar nerve block. However, it must be remembered that all local anesthetics are both neurotoxic and myotoxic with small risks associated even with dental injections (Fig. 18.2).

The Anterior Superior Alveolar Block

The region anesthetized by this block includes the anterior aspect of the maxillary alveolar process with its associated mucosa and the innervation for the maxillary cuspid, lateral incisor, and central incisor. The anterior superior alveolar nerve runs in a thin "rib" of bone in the anterior wall of the maxillary sinus approximating the nasal labial fold externally. When performing this block, the needle is directed to the labial vestibule superior to the maxillary canine and lateral incisor. The injection is made submucosally with a deposit of approximately 1 ml of anesthetic. There have been few reported adverse reactions with the anterior superior alveolar block (Fig. 18.3).

Infraorbital Block

The region anesthetized by the infraorbital block is widespread, providing dermal, alveolar process, and dental anesthesia. The dermal region of anesthesia involves the upper lip, lateral aspect of the nose, and the region below the eye to the lateral canthus of the eye [4]. Intraorally, the anesthesia is provided to the alveolar process and the central incisor, lateral incisor, canine, and first premolar, second premolar, and usually, the buccal root of the first maxillary molar (likely through the anterior superior alveolar nerve and middle superior alveolar nerve) along with the adjacent alveolar

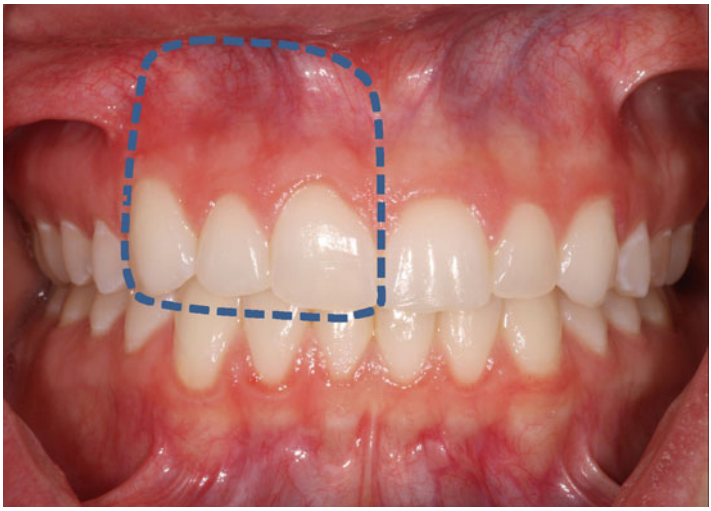

Fig. 18.3 Area anesthetized utilizing the anterior superior alveolar nerve block

process and mucosa. There have been reports that the infraorbital blocks provide profound pulpal anesthesia for second premolars, first premolars, and canines (but not central incisors and lateral incisors) for endodontic procedures [5].

The target zone for this injection is the infraorbital foramen, which can be externally palpated below the orbital rim (a depression). This orientation of the orbital rim makes the injection safe as a bony barrier between the infraorbital nerve and the contents of the orbit. The foramen is readily accessible via intraoral approach. The alveolar process is flattened in this region and provides reasonable access to the infraorbital foramen. A finger can be place extraorally palpating the infraorbital notch, which lies immediately superior to the foramen. The needle is directed to the buccal vestibule superior to the maxillary first premolar. Once the needle approximates the foramen, approximately 1 ml of anesthetic is deposited. While an extraoral approach is possible, the intraoral approach is more kind to the patient since the mucosa is readily anesthetized with 20% benzocaine and is much easier to penetrate. The intraoral approach also minimizes the chance of infraorbital bruising. No difference in efficacy for intraoral and extraoral approaches has been found [6]. There have been few reports of adverse events associated with this anesthetic block (Fig. 18.4).

Greater Palatine Block

The region anesthetized by the greater palatine block is the hard palate including the palatal process of the maxilla and the soft tissues overlying the bony plate beginning anteriorly at the level of the first premolar extending to the posterior aspect of the hard palate from the midline to the lingual marginal gingival surrounding the teeth.

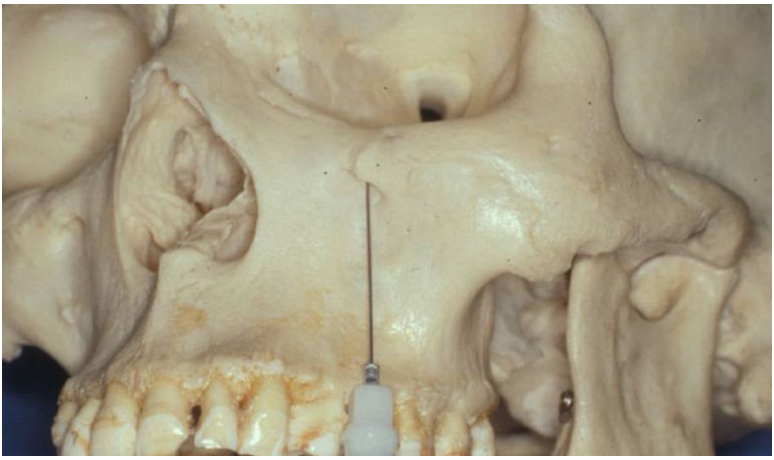

Fig. 18.4 Needle approximating the infraorbital foramen

The target area for this block is the greater palatine foramen located distal, medial, and superior to the maxillary second molar. This injection site is located clinically with digital palpation of the depression in the soft tissue formed by the foramen. The needle will likely need to be bent in a 45° angle approximately 1 in. from the tip [7] so that the tip of the needle can probe for the opening of the foramen which is 2–3 mm in diameter. The palatal tissue is very fibrous and tightly bound to the hard palate. Thus expression of anesthetic in this area can result in significant discomfort to the patient. Pre-injection application of topical benzocaine 20% may reduce discomfort associated with insertion of the needle; however, the topical anesthetic effect does not eliminate pain associated with injection of the initial bolus of local anesthetic [8]. Utilization of pressure and or cold [9] with pressure can alleviate much of this discomfort. The handle of a dental mirror can be pressed against the tissue firmly while the initiation of the injection is made. Once a few drops of local anesthetic are deposited, the mirror handle may be removed. Slow, gradual deposition of local anesthetic is recommended. One study concluded that the injection pressure below 306 mm of mercury [10] was effective in pain reduction. There are few reports of adverse effects from this injection (Fig. 18.5).

Anterior Middle Superior Alveolar Block

The region anesthetized includes the palate and associated soft tissue adjacent to the first and second premolars. The target zone is the palatal tissue approximating half of the distance between the midpalatal suture and the marginal gingiva of the first and second premolars. The needle is placed, and 0.5 ml of anesthetic is deposited

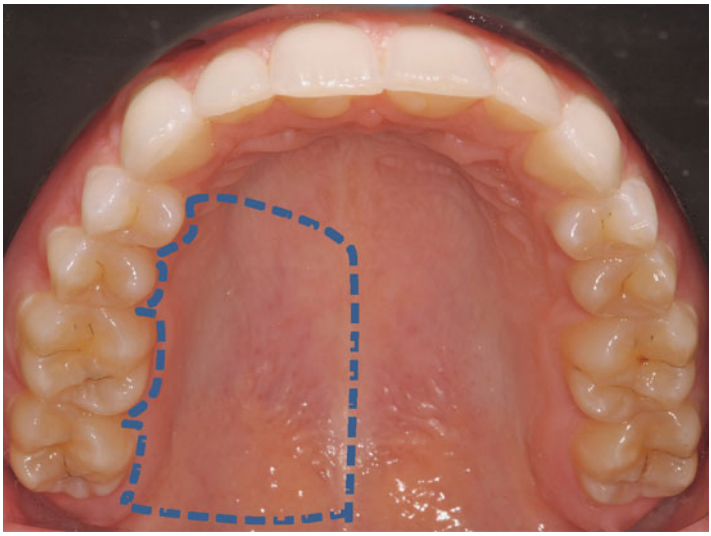

Fig. 18.5 Area anesthetized utilizing the greater palatine nerve block

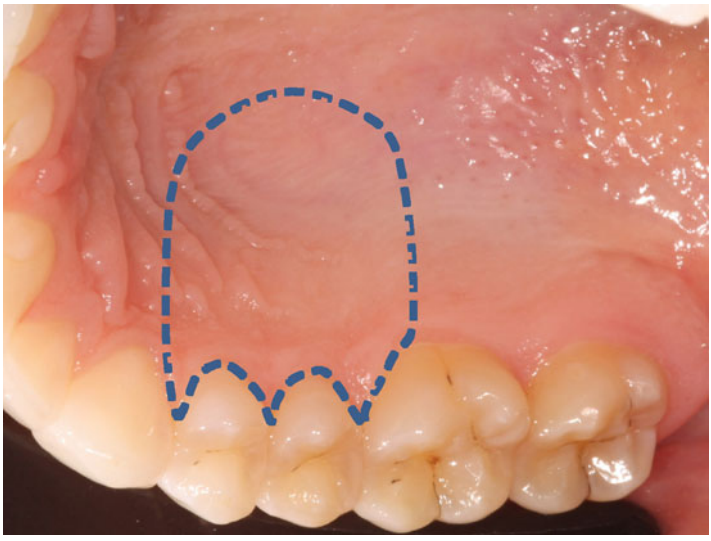

Fig. 18.6 Area anesthetized utilizing the anterior middle superior nerve block

into the very dense fibrous palatal tissue [11]. Often the tissue blanches indicating the extent of the anesthesia to the tissue. As with any palatal injections, pressure or cold anesthesia can help to alleviate some of the discomfort associated with this block. There are few reports of adverse effects from this anesthetic block (Fig. 18.6).

Maxillary Nerve Block (V_2)

The maxillary nerve (V_2) enters the pterygopalatine fossa through foramen rotundum. This region may be accessed by several approaches.

Greater Palatine Foramen Approach

Using the same technique for the greater palatine nerve block, the greater palatine foramen can be located. This provides access to the palatine canal through which the second division of the trigeminal nerve which lies within the pterygopalatine fossa may be anesthetized [12]. The maxillary nerve block effectively anesthetizes all branches of the maxillary nerve including the greater and lesser palatine, the posterior superior alveolar, middle superior alveolar, anterior superior alveolar, nasal palatine, and infraorbital nerves. This approach also provides anesthesia to the parasympathetic nerve fibers, which synapse in the sphenopalatine ganglion and even some sympathetic that travel through the region.

Once the greater palatine foramen is located, the needle (27 gauge long) is advanced within the palatine canal with a gentle probing with redirection-as-needed technique. The needle may need to be withdrawn slightly and redirected if progress up the canal is impeded. Once the needle has been advanced half to two third of its length, 0.5–1 ml of anesthetic is deposited slowly. Forceful advancement of the needle or injection of anesthetic against resistance is contraindicated due to the potential for unnecessary neural or vascular damage. The greater palatine artery travels adjacent to the greater palatine nerve in the greater palatine canal. Therefore, the risk of hematoma formation is present though very small. Any hematoma formation would be limited by the tightly restricted space of the greater palatine canal. One study reports that the risk of this event or other complications was very small including only one incidence of positive aspiration of blood during the injection [13]. Interestingly, otolaryngologists may utilize anesthesia in the greater palatine canal for the treatment of epistaxis with efficacy for reduction in bleeding with no associated serious complications [14]. The extraoral approach as described below can be utilized; however, the risk for complications is much greater for hematoma formation (maxillary artery) among other much more serious complications such as brainstem anesthesia and respiratory arrest (Fig. 18.7) [15].

Extraoral Approach Through the Pterygomaxillary Fissure

This approach utilizes the external lateral face in the region just below the zygomatic process of the maxilla in the region of the superficial masseter muscle as landmarks for initial needle placement. The patient then opens the mouth wide,

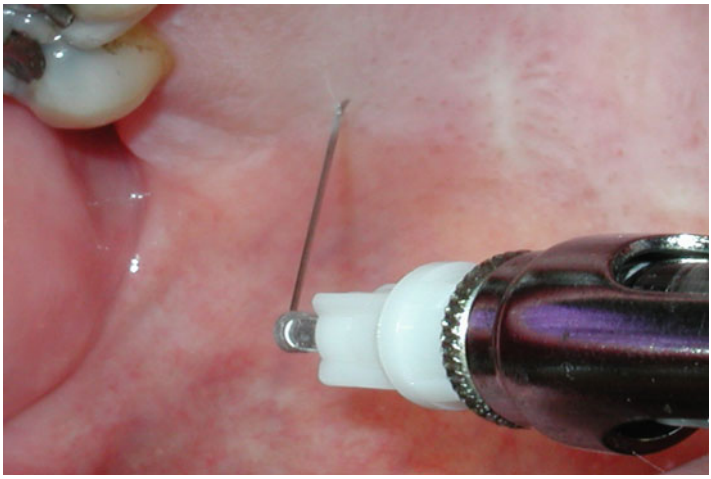

Fig. 18.7 Greater palatine foramen approach to maxillary nerve block

and the needle, inserted superior or just anterior to the coronoid process into the infratemporal fossa. The pterygomaxillary fissure lies deep, and at least a 27-gauge-long needle would need to be utilized. The posterior border of the maxilla would be tracked distally until the needle inserts just anterior to the lateral pterygoid plate of the sphenoid bone. The needle would be advanced almost to its full length. Stojcev reports that an angle of 60° to the sagittal plane and 10° to the horizontal plane help provide access to the pterygomaxillary fissure. The study also reports a success rate of 75% utilizing this approach [16]. Approximately 1.8 ml of anesthetic would be deposited in the region following careful aspiration. The risks of intravascular injection of anesthetic or trauma to vascular structures are much greater that attend this injection as compared to the greater palatine approach. The vascularity of the posterior superior alveolar region has been discussed previously. Inside the pterygomaxillary fissure is the very large maxillary artery, which is a terminal branch of the external carotid and provides a large exposure as it moves in a convoluted loop through this regions. The risks of hematoma and other vascular adverse events need to be assessed for this approach (Fig. 18.8).

Sphenopalatine Foramen Approach

There are different approaches to placement of local anesthetic in the pterygopalatine fossa. The specific approach may be influenced by the desired outcome: anesthesia required for dental treatment, differential diagnosis of orofacial pain conditions, or management of orofacial pain conditions. Importantly, the pterygopalatine ganglion is associated with the maxillary nerve and the transnasal approach attempts to

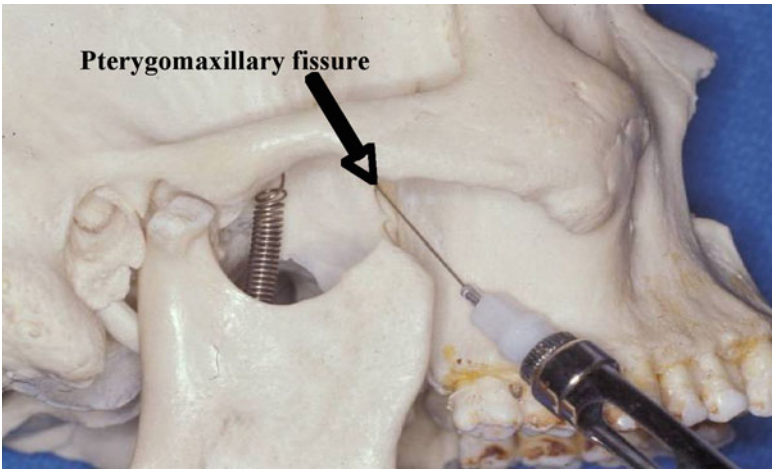

Fig. 18.8 Pterygomaxillary fissure approach to the sphenopalatine fossa for maxillary nerve block

anesthetize the ganglion in order to affect the parasympathetic afferents which synapse in the ganglion. The target is the sphenopalatine foramen in the nasal cavity posterior to the middle turbinate and the pterygopalatine ganglion, which lies lateral to the foramen. The nares are prepared first with 2% viscous lidocaine which provides both lubrication and topical anesthetic. Cotton-tipped applicators with appropriate anesthetic (lidocaine, tetracaine, cocaine 10%) are inserted through the nose to the desired location and left for 20–30 min. The patient may experience lacrimation, light-headedness along with a bitter taste and numbness in the back of the throat [17]. If cocaine is utilized, then cardiac monitoring and pulse oximetry are utilized for possible cardiac adverse effects.

Anesthetic Block Through Nares

An alternative to the application of cotton-tipped applicators is the utilization of lidocaine transnasal spray. While this approach may not achieve effective blockade to the entire maxillary division, it may serve to provide invaluable diagnostic and/or therapeutic effects. Concentrations of 4–8% lidocaine are sprayed unilaterally or bilaterally (2 sprays per side) utilizing a metered spray bottle into the nares [18]. The advantage to this technique is the ease of application and the rapidity of efficacy of the anesthetic (in certain cases within minutes). This technique has been utilized for V_2 pain associated with trigeminal neuralgia [19, 20] and neurovascular pain associated with different headache entities. The benefit of transnasal lidocaine for the management of migraine headaches has been suggested. One study shows the likely decrease of parasympathetic outflow through the pterygopalatine ganglion

with application of lidocaine [21]. Interestingly, there was significant pain relief but no effect on the peripheral allodynia due to central nervous system involvement. A case report demonstrated the ability of intranasal lidocaine 4% for the prevention of migraine following aura [22]. The benefits of the lidocaine occur primarily in the nasal cavity and pterygopalatine fossa with little systemic uptake as documented by Kanai (only 0.6 μg per ml^{-1} maximum plasma concentration). Studies report no significant adverse effects with only a small amount of burning associated with the lidocaine and the bitter taste from postnasal drip.

Nasopalatine Block: Incisive Canal

The nasopalatine block effectively provides anesthesia to the anterior portion of the hard palate with its associated mucosa from the first premolar to the incisive papilla bilaterally. The target zone for placement of anesthetic solution is the incisive papilla and the incisive canal above it in which the nasal palatine nerve enters into the palate. The needle is placed into the incisive papilla and directed superiorly several millimeters. Approximately 0.5 ml of anesthetic is deposited into the canal. The injection is painful and tactics such as providing pressure anesthesia as previously mentioned can be helpful. McArdle presents a technique for the deposition of a small amount of anesthetic in the facial gingival, which is less painful as compared to the palatal tissue. Then sequential small increments of anesthesia can be directed through the interdental papilla into the palatal tissues. Once the surface is anesthetized, then an injection into the incisive papilla is much less uncomfortable [23]. There are few reported adverse effects to this block, but patients will remember for a long time if it is very painful. Occasionally, the incisive papilla can be sore and slightly swollen as sequelae, which can be a problem if the patient's lower incisors occlude on the papilla (Fig. 18.9).

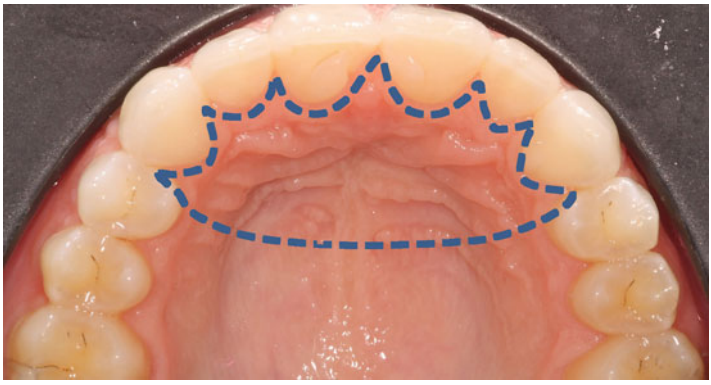

Fig. 18.9 Area anesthetized utilizing the nasopalatine nerve block

Mandibular Anesthetic Blockade

The mandibular nerve (third division of the trigeminal nerve) is a mixed nerve with two roots: a large sensory and a smaller motor root. Once branching off of the trigeminal ganglion, it reaches the infratemporal fossa via the foramen ovale. Branches of the mandibular nerve supply sensory innervation to the hard (teeth and bone) and soft (mucosa, gingiva, and tongue) tissues of the lower jaw and floor of the mouth. Motor branches to the muscles of mastication, the tensor tympani, and the tensor veli palatini muscles leave the trunk in the infratemporal fossa. The nerve gives off numerous sensory branches.

Inferior Alveolar Block

The region anesthetized includes all mandibular teeth to the midline, the body of the mandible, inferior portion of the ramus, buccal mucoperiosteum and mucus membranes anterior to the first molar, anterior two third of the tongue and floor of the mouth, and lingual mucus membranes with the associated periosteum to the midline. The target of this anesthetic block is the inferior alveolar nerve before it enters into the mandibular foramen and travels inside the body of the mandible inside the mandibular canal. The access to the inferior alveolar nerve is limited by the sphenomandibular ligament that attaches to the lingual and the medial aspect of the ramus of the mandible. To correctly navigate this small region, there are several external landmarks that will guide the needle into the correct location. The three-dimensional direction of the needle is determined by the plane of occlusion, the depth of the coronoid notch, and the pterygomandibular raphe. The anesthetic should be deposited following careful aspiration. At least 1–1.8 ml of anesthetic is utilized. When possible, it is best to avoid utilizing bilateral inferior alveolar blocks since a very broad area of the mandible would be anesthetized. The patient would have great difficulty in reporting unnecessary trauma during the procedure or prevention of self-inflicted actions such as lip biting following a procedure. The patient may have a perceived or actual difficulty in swallowing as well (Fig. 18.10).

The literature provides more case reports of adverse effects for the inferior alveolar block than any other oral anesthetic block. The most common complication is paresthesia which is almost universally associated with inferior alveolar block. In one study [24], 182 reports of paresthesia, all but two were associated with the inferior alveolar block. Of those, the lingual nerve was implicated at least twice as often as the inferior alveolar nerve. The complications for the inferior alveolar block include:

1. Electric shock. Electric shocks are most often experienced by direct contact with the lingual nerve during the injection because it is so close to the surface of the mucosa. The inferior alveolar nerve will respond in the same way, but it is much better protected. It has been shown that there is no correlation with the patient experiencing an electrical shock and neurologic damage to either nerve [25].

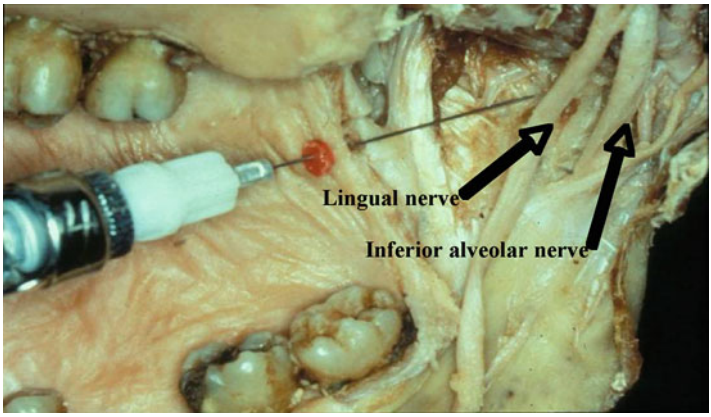

Fig. 18.10 Needle placement for inferior alveolar and lingual nerve blocks

2. Injection injuries to the lingual and inferior alveolar nerve. One study [26] reports that 42 of 54 injuries occurred to the lingual nerve and 12 of 54 to the inferior alveolar nerve. The lingual nerve injuries are much more incapacitating than the inferior alveolar nerves resulting in sensory loss to both feeling and taste (damage to the chorda tympani nerve as well). There is no clarity on whether these injuries will spontaneously improve. Krafft and Hickel report a high spontaneous resolution of 17 of 18 in 6 months. Hillerup reports the opposite. The etiology of the reason behind the injury is unclear. There are several rationales proposed.

   Direct trauma: This was the earliest proposed rationale. There is no strong evidence to support this. There is no evidence for the electric shock – direct trauma with correlation of paresthesias.

   Neurotoxicity: There is mounting evidence that this may be the most likely causative factor for nerve injury in respect to local anesthesia. Most of the injuries are associated with the local anesthetics with 4% concentration. Hillerup reports that 54% of nerve injuries were associated with articaine 4% following its utilization in Denmark (Hillerup 2006). In a 21-year retrospective study, Haas [27] reports on Canadian reports of paresthesias. In 1993, there were 14 cases of paresthesia of which articaine 4% was apparently implicated in 10 of 14, and prilocaine 4% was implicated in the other 4 out of 14 cases.

   Neural ischemia associated with neural toxicity: Perineural and endoneurial fibrotic changes are likely associated with high concentration of local anesthetic. This can likely result in endoneurial edema and injury to the nerve [28].

   Intravascular injections: The medial aspect of the ramus in the region is heavily vascularized with the inferior alveolar artery and other sources. An intra-arterial injection of local anesthetic with epinephrine concentrates the anesthetic in the peripheral tissues. This may be demonstrated by blanching of facial skin, intraoral

regions, and even eyes symptoms. These sequelae normally fade away within 60 min or less [29].

Diplopia: This transient response is also seen with inferior alveolar blocks as well as posterior superior alveolar blocks [30].

Broken needles: Although very rare, broken needles occur in the anesthetic block for the inferior alveolar nerve more than any other. In one study, 16 of 17 reports of broken needles occurred in this region [3].

Of all the anesthetic blocks, the inferior alveolar block has the most reports of inefficacy. Conservatively, inferior alveolar blocks fail at least 15% of the time [31]. Another study reports that 87% of subjects with an inferior alveolar block reported numb lips after 5 min [32]. Endodontic literature reports that pulpal anesthesia with teeth with irreversible pulpitis resulting from inferior alveolar blockade demonstrate much less than expected efficacy (36%) [33].

Why do inferior alveolar blocks demonstrate more unpredictability than other blocks? The reason likely involves the complexity of the anatomy and the difficulty in access. There are several common errors that can contribute to less desirable predictability.

A. Injection error: An approach that is too straight rather than from the contralateral premolar region. If the approach is from the region of the central incisors, the needle often contacts the ascending ramus of the mandible thereby stopping the injection before there is any access to the region of the lingula and the mandibular foramen. If the injection is more medial, the needle will be on the medial aspect of the sphenomandibular ligament which prevents infiltration of the anesthetic from reaching the inferior alveolar nerve. This may result in the deposition of anesthetic in the tail of the parotid gland that wraps around the distal aspect of the ramus often with the resultant taste to the patient and even some anesthesia of the facial nerve. The correction involves correction of the approach by directing the needle from the opposite premolar region so that the needle can reach the desired location.
B. Injection error: An approach that is too inferior. The needle needs to be directed ideally at a bisection of the depth of the coronoid notch and at the same inclination as the occlusal plane. If the needle is below this plane, the anesthetic will be deposited in a region that is below the mandibular foramen and below the sphenomandibular ligament without access to the inferior alveolar nerve. The correction involves raising of the needle to the correct plane.
C. Accessory innervations to the mandibular molar region: There have been documented other accessory innervations to the mandibular molar region including branches from the cervical plexus from C_2 and C_3, branches from the motor nerve to the mylohyoid muscle [34], and even additional branches from the inferior alveolar nerve entering the mandible [35]. The solution to possible accessory innervations to mandibular posterior teeth or even anterior teeth would be additional anesthesia likely with a buccal approach or even a lingual approach. Infiltrations in the proper depth and location will anesthetize addition innervations.

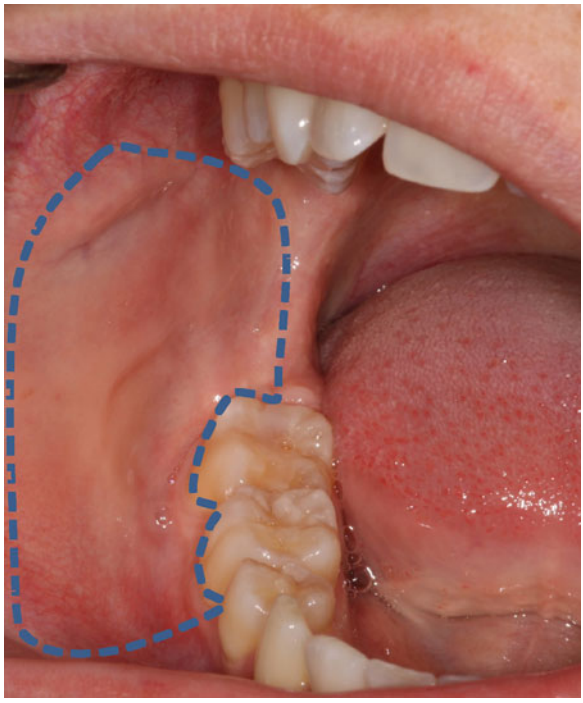

Fig. 18.11 Area anesthetized utilizing a long buccal nerve block

Buccal Block

The buccal block anesthetizes the region of the mucous membranes including gingiva and periosteum to the buccal of the mandibular molars. This injection is used adjunctively with the inferior alveolar block, which will not anesthetize the buccal tissues. The target is the buccal mucosa lateral to the maxillary second molar and lateral to the mandibular coronoid process. Approximately 0.5 ml of anesthetic is deposited under the mucosa. There are few reported adverse effects for this injection (Fig. 18.11).

Mental Block

The mental block anesthetizes the buccal mucous membranes anterior to the mental foramen to the midline. This usually involves the gingiva, the buccal mucosa adjacent to the mandibular first molar to the lower lip but may also include either the first or second premolar if sufficient anesthetic enters the mental foramen. The target is the mental nerve as it leaves its respective foramen. The mental foramen is most

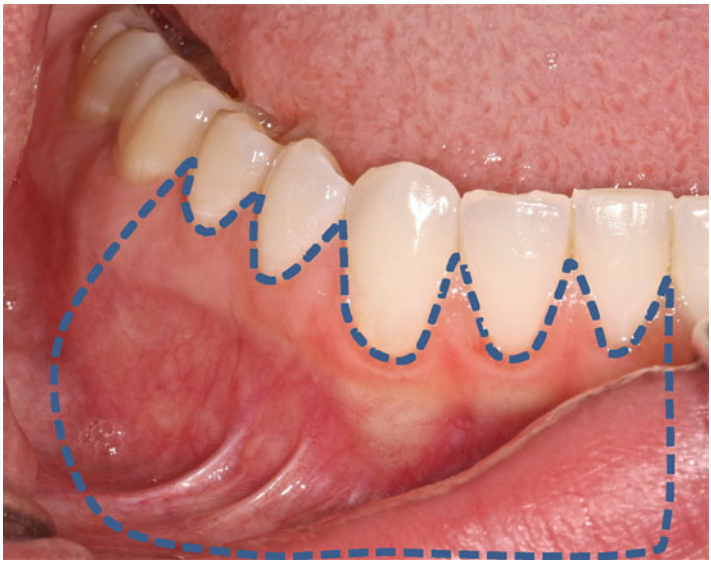

Fig. 18.12 Area anesthetized utilizing a mental nerve block

likely present on the buccal osseous surface of the mandible apical to the second premolar or between the apices of the first and second premolars. It may vary in position from the canine all the way to the mandibular first molar [36]. The needle is inserted in a posterior and inferior direction at the base of the mandibular vestibule and moved into position to the region of the periosteum over the foramen. Approximately 0.5 ml of anesthetic is deposited. Very few adverse effects are reported in the literature. There are suggestions for utilizing mental blocks as an alternative to the inferior alveolar block if the area of concern is anterior to the mental foramen since this block is much more readily accessible (Fig. 18.12) [37].

Incisive Block

The incisive block anesthetizes the mucous membranes including the gingiva and buccal mucosa of the vestibule and lip. The pulps of the premolars, canines, and incisors may also become anesthetized. The target is the buccal mucosa in the bottom of the vestibule in the apical region of the teeth or soft tissue that requires anesthetic. The needle is inserted and approximately 0.5 ml of anesthetic is deposited. There are few recorded adverse reactions to this injection. It has been reported that articaine 4% with epinephrine anesthetic provides superior anesthesia as compared to lidocaine 2% with epinephrine both in regard to duration and depth of anesthesia for the mental and incisive blocks (Fig. 18.13) [37].

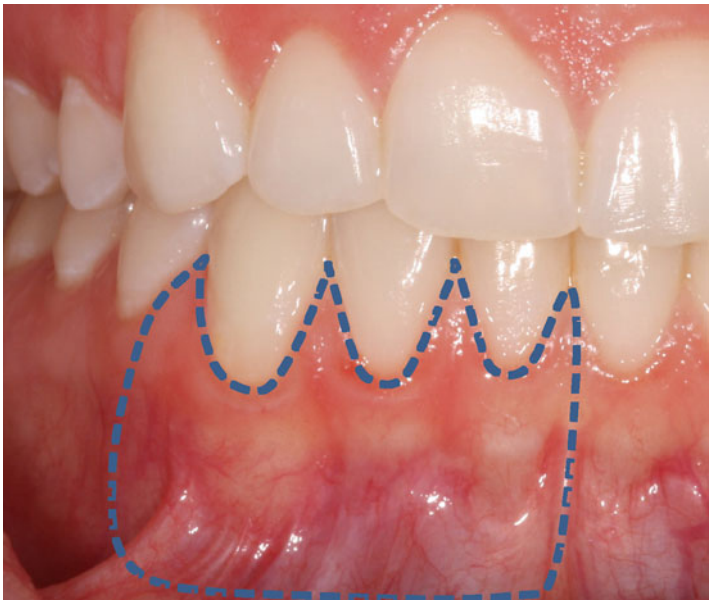

Fig. 18.13 Area anesthetized utilizing an incisive nerve block

Gow-Gates Mandibular Block

The area anesthetized by the Gow-Gates block is the entire region of the mandibular branch of the trigeminal nerve. It is essentially a V_3 division block, which includes the body and inferior portion of the mandible, teeth, buccal mucosa, anterior two third lingual mucosa and tongue, and preauricular region of the face associated with the auriculotemporal nerve. This block is utilized following failure of an inferior alveolar nerve block but has many applications of its own for both diagnostic anesthesia and dental anesthesia. The target for the needle is in a plane that is much higher in a superior direction to the occlusal plane and that of the inferior alveolar block. The region of injection is the anteriomedial aspect of the condyle. The mouth must be opened fully with the needle inserted but below the second molar with the approach from the opposite mandibular premolar region. The needle is inserted 25–28 mm [38], with check for aspiration, and 1.8 ml of anesthetic deposited.

There are a number of benefits for utilizing the Gow-Gates block as compared to the inferior alveolar block. The risk of positive aspiration or intravascular injection is greatly diminished. This is related to the decrease in vascularity of the region as compared to the medial aspect of the mandible. It is reported that once mastered, the Gow-Gates block has a risk of approximately 1.6% for aspiration of blood as compared to a range of 3.6–22% for the inferior alveolar block [38]. Gow-Gates (the author) reports a more predictable anesthetic technique as well with a success rate

of 98–100% as compared to 83.9–85.4% for the inferior alveolar block. Other reports vary and suggest that the efficacy of a Gow-Gates block is comparable to an inferior alveolar block for extractions and pulp testing [39]. So it seems likely that adverse effects in relationship to vascular compromise are lessened and possibly risks of paresthesia as well. There are few, if any, reports of paresthesia related to Gow-Gates blocks. The few reports of adverse effects are related to temporary vision defects [40].

Auriculotemporal Block

The utility for use of an auriculotemporal nerve block is diagnostic and procedure related. The block can serve as an aid in determining the degree of involvement of the temporomandibular joint (TMJ) in cases of orofacial pain. Since there is a significant convergence from the masseter muscle region and the TMJ, it not surprising that it is difficult to readily determine where the pain is coming from. The auriculotemporal block anesthetizes the auriculotemporal nerve, a branch of the mandibular nerve, which provides sensory innervation to the vast majority of the TMJ, the preauricular region, and the portion of the scalp superior to the helix of the ear. The target area for placement of the 27-gauge-short needle is a small concavity anterior to the base of the tragus as the mandible opens. The needle is angled at a 20° anterior angle matching the external ear canal to prevent inadvertent deposition of the anesthetic in that area. The needle is advanced until in some cases it gently contacts the posterior aspect of the mandibular ramus/condylar neck [41]. If this occurs, the patient is asked to open the mouth, thus allowing the needle to pass just behind the ramus to the medial side. Then, approximately 0.5–0.8 ml of anesthetic is deposited following aspiration (Fig. 18.14).

There are few reported risks associated with the auriculotemporal block. However, patient education prior to the block is important since the normal short-lived-associated factors induce unnecessary anxiety. Since the facial nerve is in close proximity to the injection target zone, it will be likely affected for the duration of the anesthetic resulting in temporary paresis of the muscles of facial expression subserved by the temporal, zygomatic, and buccal branches. Should the patient be unable to close their eyelid, this can be accomplished manually. A moistened 2×2 gauze can hold the lid closed.

Trigger-Point Injections

Local anesthetic is utilized in the treatment of myofascial pain in the head and neck region. The focal point of this injection is the “trigger point” identified within the painful muscle. This region of taut band is identified thorough musculoskeletal examination. The trigger point presents as a hard knot-like area in a muscle that

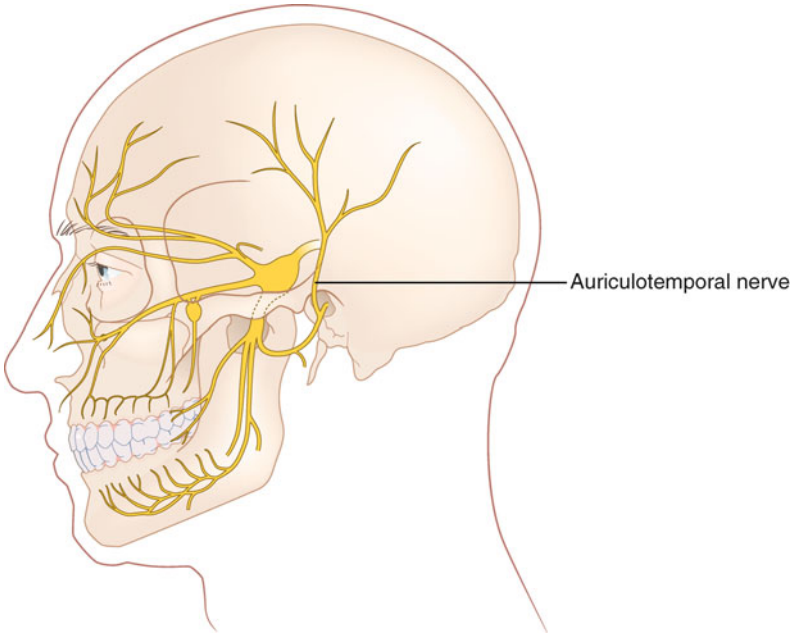

Fig. 18.14 Auriculotemporal nerve, adapted from *Atlas of Human Anatomy* 3 rd edition by Frank H. Netter, Icon Learning Systems Teterboro, New Jersey 2003

when palpated duplicates the patients' chief pain concern and is associated with pain not only in the local region but also at a distant site known as the "zone of reference". Typically, a 27-gauge-short needle is utilized. The muscle must be stabilized so that the trigger point does not move out of position as the needle is inserted through the skin or intraoral mucosa. As the trigger point is reached, the patient often will describe pain that duplicates their chief concern and the muscle itself may exhibit a "twitch" response. Approximately 0.3–0.5 ml of local anesthetic without vasoconstrictor is deposited. The needle is then redirected in the region of anesthetized tissue, and adjacent muscle fibers are also anesthetized. Following the injection, pressure is applied for 5 min in order to control bleeding. Cross frictional massage followed by gentle stretching with ice massage is recommended following injection so as to soften the taut band in the muscle. The patient is given postoperative instructions to limit vigorous utilization of the injected muscle(s). It must be strongly emphasized that trigger-point injections are part of an overall comprehensive approach to management of myofascial pain. For the masticatory system, this multidisciplinary approach always involves patient education, patient involvement with stretching and self-behavior modification techniques, regimens that incorporate improvements related to sleep hygiene, and often, the utilization of nutritional supplementation. Other aspects may include physical therapy, clinical psychology (habit modification/relaxation training), and pharmacotherapy.

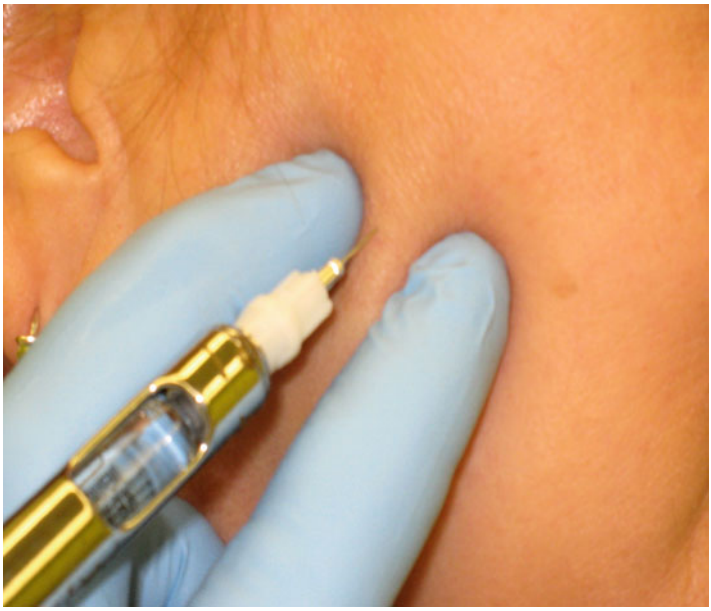

Fig. 18.15 Masseter muscle trigger-point injection. Note that the operator has isolated the taut band in order to stabilize the target zone within the muscle

The use of local anesthetics is ubiquitous in health care. There are adverse reactions, but they are "extraordinarily negligible" [42]. However, efficacy and comfort are predicated on a detailed knowledge of anatomy, physiology, neural pathways, and the pharmacodynamics and pharmacokinetics of the anesthetic solutions utilized (Fig. 18.15).

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19

Topical and Regional Anesthesia of the Airway

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Introduction

Despite recent advancements in video and digital laryngoscopes, awake intubation continues to be an important modality for anesthesiologists to secure the airway. Awake intubation (AI) can be performed for a variety of conditions that result in a difficult airway. Congenital anomalies involving the head and neck, tumors of the airway, morbid obesity, cervical spine pathology, and trauma associated with facial and cervical instability all make direct laryngoscopy challenging and can be indications for awake intubation.

On the other hand, contraindications to regional and topical anesthesia of the airway include patient refusal, coagulopathy, infection at the blockade site, allergy to local anesthetics, and a patient in whom the loss of protective airway reflexes would be unsafe.

There are many benefits to performing a fiber-optic intubation in an awake as opposed to an anesthetized or apneic patient. The awake patient can manipulate the tongue and swallow secretions, thereby clearing the airway. An awake patient can also breathe spontaneously, allowing the anesthesiologist more time to secure the airway. Furthermore, the patient can phonate, which can assist in visualization of the glottic opening and vocal cords. The largest hurdle to awake intubation is often patient cooperation. Excessive salivation, intact reflexes, and patient discomfort can lead to failure of AI. This can be overcome with topical and regional anesthetic techniques, often with the addition of sedation.

Relevant Airway Anatomy

The anatomy of the upper airway and in particular its pattern of sensory innervation of the component subdivisions of the upper airway must be considered when performing an awake oral or nasal intubation. It is important that the physician is able to locate the nerves based on anatomical landmarks in order to deliver adequate anesthesia to the patient.

The upper airway includes the nasal cavity, oral cavity, and the three subdivisions of the pharynx: the nasopharynx, the oral pharynx, and the laryngopharynx (hypopharynx). These subdivisions of the upper airway are innervated primarily by branches of the trigeminal (CN V), glossopharyngeal (CN IX), and vagus (CN X) cranial nerves, respectively (Fig. 19.1).

The trigeminal nerve (CN V) has three divisions, the ophthalmic division (CN V1), the maxillary (CN V2), and mandibular division (CN V3), each of which is involved in supplying sensory innervation to different parts of the upper airway.

The anterior aspect of the nasal cavity is supplied by the anterior ethmoidal nerve, a branch of the nasociliary nerve from the ophthalmic division of the trigeminal nerve (CN V1). It subdivides into lateral internal nasal branches to the anterior aspect of the lateral wall of the nasal cavity and medial internal nasal branches to anterior

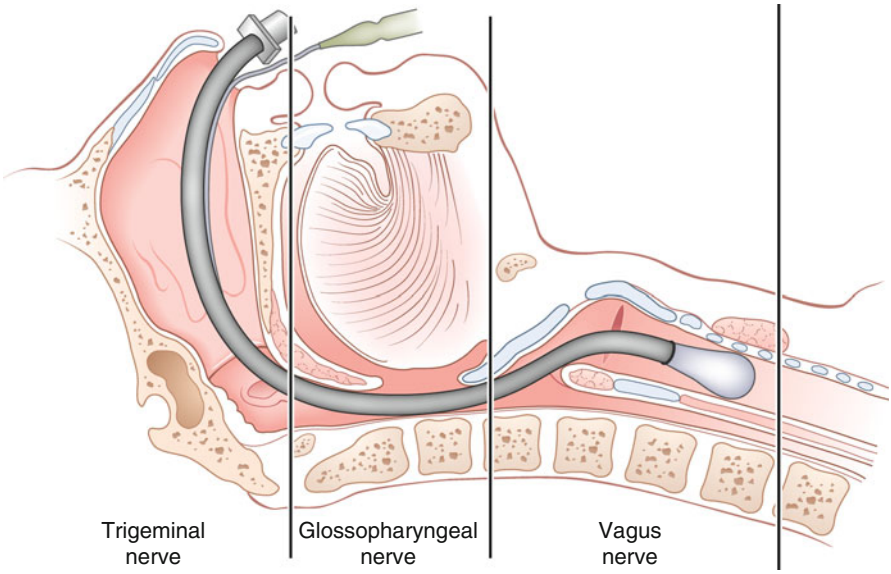

Fig. 19.1 Simplified innervation of airway structures. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

superior parts of the nasal septum. After giving off these branches, this nerve continues as the external nasal nerve which exits the nasal cavity between the nasal bone and nasal cartilages to provide sensory innervation to the skin on the external surface to the tip of the nose inferior to the nasal bones and superior part of the nares.

The infraorbital nerve, the terminal branch of the maxillary division of the trigeminal nerve (CN V2), gives rise to internal nasal branches which supply sensory innervation to the inferior part of the nares, the skin on the side of the nose, the nasal vestibule, and the anterior movable part of the nasal septum at which point it interdigitates with terminal branches of the anterior ethmoidal nerve. Branches of the anterior superior alveolar nerves from the infraorbital nerve supply the inferior part of the nasal septum, the floor of the nasal cavity near the anterior nasal spine, and the anterior one fourth of the lateral nasal wall up to the level of the opening of the maxillary sinus. The posterior three fourths of the lateral nasal wall, roof, and floor receive sensory innervation from the lateral posterior superior nasal nerves (to the mucous membrane covering the superior and middle concha) and posterior inferior nasal nerves which are branches coursing anteriorly from the greater palatine nerve as it courses inferiorly from the pterygopalatine ganglion. The pterygopalatine ganglion is located within the pterygopalatine fossa in line with the middle turbinate. The posterior and inferior parts of the nasal septum are supplied by the medial posterior superior nasal nerves, which branch from the pterygopalatine ganglion and enter the nasal cavity through the sphenopalatine foramen. One of the large medial posterior superior nasal nerves is called the nasopalatine nerve which courses from posterior superior to anterior inferior between the periosteum and the

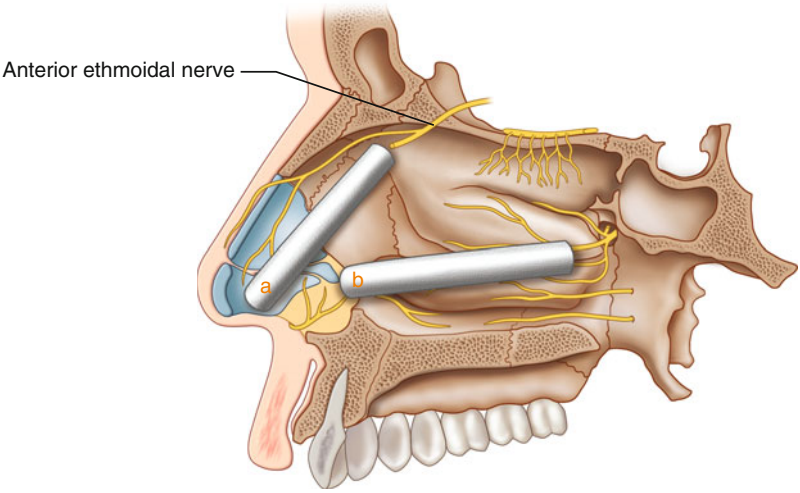

Fig. 19.2 Innervation of the right nasal cavity. (**a**) and (**b**) cotton pledgets can provide topical anesthesia to the nasal cavity. Adapted from Drake RL et al. *Gray's Anotomy for Students*, 2nd Ed, 2010

mucosa of the nasal septum. The terminal part of the nasopalatine nerve courses through the incisive canal in the anterior midline of the hard palate to provide sensory innervation to the most anterior part of the hard palate within the oral cavity. The nerve of the pterygoid canal (Vidian nerve) supplies the most posterior and superior part of the nasal septum (Fig. 19.2).

The nasal cavity communicates posteriorly with the nasopharynx by means of the posterior nasal choanae. Sensory innervation to the nasopharynx posterior to the opening of the pharyngotympanic tube is mediated primarily by the pharyngeal nerve, a branch from the pterygopalatine ganglion that courses anteriorly through the palatovaginal or pharyngeal canal to enter the posterior aspect of the nasopharynx.

The oral cavity receives its sensory innervations from branches of the trigeminal, facial, and glossopharyngeal nerves.

Sensory innervation of the tongue is provided by the lingual nerve, a branch of the mandibular division of the trigeminal nerve (CN V3) to the anterior two-thirds of the tongue and lingual branches of the glossopharyngeal nerve (CN IX) to its posterior one third. The glossopharyngeal nerve provides general sensory innervation of the posterior one-third of the tongue and also provides the sensation of taste from the same area.

The hard palate is innervated by the greater palatine nerve and nasopalatine nerve, both of which are branches of the maxillary division of the trigeminal nerve (CN V2). Its terminal fibers overlap the distribution of fibers from the nasopalatine nerve, which enters the oral cavity through the incisive canal and innervates the mucosa covering the most anterior part of the hard palate.

The soft palate receives its sensory innervation from the lesser palatine nerves. The lesser palatine nerves also descend through the greater palatine canal and course through the lesser palatine foramina on the posterior lateral aspect of the hard palate. They course posteriorly into the soft palate to innervate the mucosa of the soft palate, palatine tonsil, and uvula.

The oropharynx is continuous anteriorly with the oral cavity, is bounded superiorly by the soft palate, and extends inferiorly to the level of the upper border of the epiglottis. The oral cavity communicates with the oropharynx through the oropharyngeal isthmus, which is demarcated by the palatoglossal arch. The palatine tonsil and the palatopharyngeal arch (posterior tonsillar pillar) comprise its lateral boundary, while the pharyngeal aspect of the tongue is located anteriorly.

Cranial nerve VII (the facial nerve) and cranial nerve IX (the glossopharyngeal nerve) supply the sensory innervation to this part of the upper airway. The mucous membranes of the palate and nasopharynx, i.e., caudal parts of the oral cavity, have sensory afferent fibers that are in the facial nerve. The palatine tonsil not only receives sensory nerve branches from the lesser palatine branch of the maxillary division of the trigeminal nerve (CN V2), but also has sensory innervation from the glossopharyngeal nerve (CN IX), which is located just lateral to the palatine tonsil. The posterior tonsillar pillar or palatopharyngeal arch is innervated by a dense plexus formed by the pharyngeal branches of the cranial nerves IX and X. The pharyngeal mucosa from the inferior opening of the pharyngotympanic tube (Eustachian tube) to approximately the middle level of the aryepiglottic fold is supplied by glossopharyngeal sensory fibers. Sensory nerve fibers from the caudal pole of the palatine tonsil and root of the tongue have been reported to be very dense [1].

The glossopharyngeal nerve exits the jugular foramen anterior to the vagus and spinal accessory nerves and courses anteriorly and inferiorly between the internal carotid artery and the internal jugular vein posteromedial to the styloid process and its associated muscles. The nerve courses anteriorly between the internal carotid artery and external carotid artery, crosses the stylopharyngeus muscle, and enters the pharynx between the superior and middle constrictor muscles. After entering the pharynx, it branches to supply the posterior one-third of the tongue and the mucous membrane of the palatine tonsil and pharynx. Pharyngeal branches of the glossopharyngeal nerve unite at approximately the middle constrictor muscle to contribute the sensory component to the pharyngeal plexus. Tonsillar branches supply sensory branches to the mucous membrane over the tonsil and soft palate. In the posterior aspect of the oral cavity, the glossopharyngeal nerve is located just lateral to the palatine tonsil and posterior tonsillar pillar and medial to the lateral pharyngeal space. It also has an intimate anatomical relationship to the significant anastomosing vascular supply to the palatine tonsil.

The laryngopharynx (hypopharynx) extends from the superior border of the epiglottis and glossoepiglottic folds to the inferior border of the cricoid cartilage. The piriform fossa is a small paired depression that lies medial to the laminae of the thyroid cartilage and lateral to either side of the laryngeal aditus, separated from the cavity of the larynx by the aryepiglottic folds.

The sensory innervation of the larynx and the laryngopharynx is mediated by branches of the vagus nerve (CN X). The vagus nerve exits the jugular foramen and descends within the carotid sheath between the internal jugular vein and the internal carotid artery. After giving off branches, which form the motor component of the pharyngeal plexus, the vagus nerve, at approximately the level of the C1–C2 vertebra, gives rise to the superior laryngeal nerve. The superior laryngeal nerve arises from the inferior ganglion of the vagus and descends along the lateral aspect of the pharynx, initially posterior and subsequently medial to the internal carotid artery. The superior laryngeal nerve subsequently divides into a small external laryngeal nerve and a larger internal laryngeal nerve.

The external branch of the superior laryngeal nerve is motor to the cricothyroid and the cricopharyngeus muscles and does not participate in the sensory innervation of the larynx or hypopharynx. The external laryngeal nerve lies between the pretracheal fascia and the inferior pharyngeal constrictor muscle. The external and internal branches of the superior laryngeal nerve are the only nerves coursing from lateral to medial at this level.

The internal branch of the superior laryngeal nerve carries afferent sensory innervation from the mucosa of the larynx, extending from the laryngeal surface of the epiglottis and back of the tongue inferiorly to the level of the vocal folds. It is also responsible for sensory innervation of the piriform fossa. The sensory innervation to the hypopharyngeal wall is dense [2]. The internal branch courses anteriorly from its origin, crosses the greater cornu of the hyoid bone, and pierces the thyrohyoid membrane superior to the superior laryngeal branch of the superior thyroid artery. After perforating the thyrhyoid membrane, the internal laryngeal nerve arborizes to innervate the laryngeal mucosa from the upper surface of the vocal folds to the base of the tongue.

The trunk of the internal laryngeal nerve enters the larynx through the thyrohyoid membrane and subdivides into three major branches. The anterior (superior) "epiglottic branch" supplies the mucosa on lingual surface of the epiglottis and a small part of the anterior wall of the epiglottic vallecula. The middle "ventricular branch" supplies the aryepiglottic fold and courses caudally to innervate the mucosa of the ventricular fold. The posterior "postcricoid branch" courses directly to the piriform sinus and also sends a few small branches to the interarytenoid muscles.

The density and laterality of the sensory innervation of the airway vary. The posterior tonsillar pillars, the laryngeal surface of the epiglottis, and the postcricoid and arytenoid regions have a high density of sensory innervation. Other areas such as the posterior part of the hypopharynx were reported to have little or no identifiable sensory nerve supply.

The sensory innervation of the mucosa of the larynx below the level of the vocal folds is from the right and left recurrent laryngeal nerves which also supply motor innervation to all of the intrinsic muscles of the larynx except the cricothyroid muscle. As a result of embryologic development of the aortic arches, the right and left recurrent laryngeal nerves arise at different levels. The right recurrent laryngeal nerve arises anterior to the first part of the subclavian artery as the nerve winds

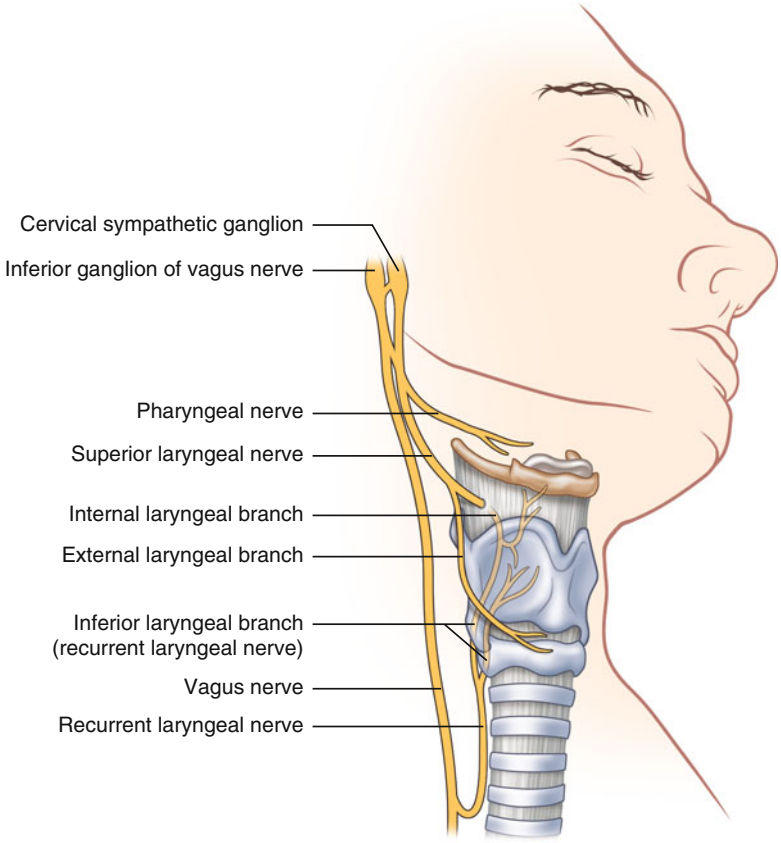

Fig. 19.3 Innervation of the larynx. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

around the vessel. The left recurrent laryngeal nerve arises on the left side of the arch of the aorta where it winds around this vessel posterior to the attachment of the ligamentum arteriosum.

The recurrent laryngeal nerve can be identified as it enters the larynx just posterior to the inferior cornu of the thyroid cartilage. In the lower portion of its course, the nerve can be palpated on the surface of the trachea. It courses superiorly in the groove between the trachea and the esophagus. Before entering the larynx, the nerve supplies branches for the cricopharyngeus muscle. Below the border of the inferior constrictor, the recurrent laryngeal nerve divides into an anterior branch, which is mainly motor (sometimes referred to as the inferior laryngeal nerve) and a posterior branch, which is mainly sensory. The sensory branch supplies the mucosa of the larynx inferior to the vocal cords and communicates with branches of the internal laryngeal nerve (Fig. 19.3).

Preparation

The first step in preparation for airway manipulation in a conscious patient is education. The anesthesiologist needs to explain to the patient the reasons behind the decision to perform an awake intubation, as well as the steps involved. The patient needs to understand that his or her cooperation is necessary to facilitate the process. A calm patient can be intubated much easier than a restless one.

The individual performing the fiber-optic intubation is the airway manager. He or she should also inform the other individuals involved with the intubation of the plan. This includes notifying a surgeon, who would be available to perform an emergent cricothyroidotomy, if necessary.

Lastly, appropriate monitors should be applied to the patient. A nasal canula can be used to supplement oxygenation during oral fiber-optic intubation. Likewise, a face mask placed inferiorly to cover the mouth and chin can be used to supplement oxygenation during nasal fiber-optic intubation [3]. Either method should allow end-tidal CO_2 monitoring. Oxygen saturation and blood pressure should also be monitored. Electrocardiogram leads should be attached, and intravenous access should be obtained.

Premedication

Antisialogogues should be administered early before airway instrumentation in an awake patient in order to minimize secretions. By doing so, antisialogogues improve not only the fiber-optic view but also the ability of local anesthetics to reach the airway mucosa. Intravenously administered glycopyrrolate is the preferred agent. Atropine i.v. or i.m. is an alternative. Intramuscular administration of atropine is favored over intravenous injection in order to avoid tachycardia and, less commonly, psychosis [4]. Scopolamine can also be given; however, it may cause delirium, especially in older patients [5].

Topical vasoconstrictors should also be administered before performing a nasotracheal intubation. This will help to decrease bleeding and open the airways, as topical anesthesia tends to reduce the caliber of a normal airway [6]. The nasal passages can be accessed for patency by asking the patient to note the air movement from each nostril after the contralateral side is compressed. Each nostril can also be examined fiber-optically to identify pathology as well as access the degree of patency [7]. After choosing the nasal passage, deposit at least two sprays of 1% phenylephrine or 0.05% oxymetazoline into each nostril [8].

Most patients experience some degree of anxiety associated with an awake intubation; therefore, administration of sedatives and hypnotics may be appropriate. However, it is important to remember that sedation cannot compensate for inadequate topical anesthesia and can be dangerous in a patient with a critical airway [9]. Short-acting and intravenous medications are ideal for this process.

Table 19.1 Commonly used medications for awake intubation

Medication	Dosage and route	Class	Side effects
Glycopyrrolate	3–6 μg/kg IV,IM	Antisialogogue	Slight tachycardia
Atropine	7–10 μg/kg IV,IM	Antisialogogue	Tachycardia, psycosis
Scopolamine	0.3–0.6 mg IV,IM,SC	Antisialogogue	Sedation, delirium
Midazolam	20–40 μg/kg IV, IM	Benzodiazepine	Delayed awakening
Fentanyl	1–2 μg/kg IV	Opioid	Respiratory depression
Dexmedetomidine	Loading dose: 1 μg/kg over 10 min Infusion: 0.2–0.7 μg/kg/h	α_2-Agonist	Bradycardia, hypotension
Propofol	0.25 mg/kg IV	Hypnotic	Cardiovascular depression
Ketamine	0.2–0.5 mg/kg	Hypnotic	Increased secretions

Midazolam and fentanyl are commonly used, as they are easy to titrate and reverse. Dexmedetomidine can be used and is suitable in this setting because it does not cause significant respiratory depression [10]. Propofol and ketamine can also be used as a last resort when comfortable sedation cannot be achieved (Table 19.1).

Approach to Anesthesia of the Airway

As the patient is being prepared and premedicated for an awake fiber-optic intubation, the process of anesthetizing the airway with topically applied local anesthetics may be started. This is a dynamic process that can also involve performing regional anesthesia on specific nerves. Because both noninvasive and invasive means of airway anesthesia are available, it is up to the anesthesiologist to decide which combination will work best for the individual patient. Various local anesthetics may be utilized; however, lidocaine has a better safety profile than other agents used for airway anesthesia [11]. Also excessive doses of local anesthetics can cause toxicity, and the total amount used in both topical and regional techniques must be considered. The route of administration of lidocaine determines the time to the peak concentration of the local anesthetic. Therefore, the route of endotracheal intubation and the specific technique of localization will determine which anatomic structures will need to be anesthetized first.

Topicalization of the Airway

Topicalization of the Nasal Cavity and Nasopharynx

The nasal cavity can be anesthetized in order to facilitate awake nasotracheal intubation. The nerves in this area lie just beneath the nasal mucosa, allowing them to be blocked by diffusion of topical anesthetic solution. Vasoconstrictors should be

applied to the nasal mucosa as early as possible with this technique. One method is with the use of cotton pledgets soaked in 2–4% lidocaine, as illustrated in Fig. 19.2. The pledgets (a) can be positioned anteriorly and superiorly along the middle turbinate in order to block the anterior ethmoidal nerve. After several minutes, a second applicator (b) can be positioned in a more inferior and posterior direction along the inferior turbinate in order to block the pterygopalatine ganglion. The pterygopalatine ganglion can also be blocked invasively using an oral approach [12]. Another means of anesthetizing the nasal cavity involves the insertion of nasal trumpets covered in 2% lidocaine jelly. The nasopharynx can then be anesthetized by placing the plastic sheath of a 20-gauge angiocatheter or a laryngotracheal mucosal atomizer through the nasal trumpet and then injecting 4 ml of 1% lidocaine. This frequently causes the patient to cough, which aids in the spread of local anesthetic to the more distal airways.

Topicalization of the Oral Cavity and Oral Pharynx

In most patients, topicalization of the mucosa of the oral cavity and oral pharynx is sufficient to allow AI. Local anesthetic can be sprayed onto the mucosa of the oral cavity by the anesthesiologist. One popular preparation is Cetacaine, which is a pressurized solution of benzocaine, tetracaine, and aminobenzoate, delivered in oily foam. However, care must be taken with this method, as overzealous use of any benzocaine-based product can result in methemoglobinemia. Furthermore, pressurized aerosol sprays contain preservatives that may cause a sore throat following the procedure [13].

Topicalization of the Airway with Aerosolized Local Anesthetic

One of the simplest and most comfortable means of anesthetizing the airway down to the trachea is through the use of aerosolized local anesthetic. It does, however, require patient cooperation, which can be difficult in select populations. With this technique, 5 ml of 4% lidocaine is nebulized with oxygen at 6 L/min via a face mask. One major disadvantage to using this approach is that the level of anesthesia achieved throughout the airway is highly variable [14]. Lack of dense airway anesthesia coupled with an intact gag reflex can make AI challenging.

Spray-As-You-Go Technique

The spray-as-you-go (SAYGO) technique is a noninvasive method of intermittently anesthetizing the airway. It takes more patience on the part of the anesthesiologist

and requires less resistance from the patient. This technique is especially useful as it allows the patient to maintain his or her airway reflexes as long as possible, thereby protecting a patient who is at greater risk for gastric aspiration. After proper premedication and application of the appropriate monitors, the patient's nose or mouth can be anesthetized, depending on the route of intubation. As the fiber-optic bronchoscope is then advanced into the pharynx, 4% lidocaine is then injected through the suction port and into the airway by one of two methods. The first method involves attaching a 5-ml syringe filled with the local anesthetic to the suction port; then injecting, in 0.2-ml increments, the least amount needed to anesthetize the area; then waiting approximately 1 min before proceeding distally in the airway; and repeating the maneuver. The second method utilizes an epidural catheter placed through the suction port of the fiber-optic bronchoscope in order to deliver the local anesthetic.

Regional Anesthesia of the Airway

Glossopharyngeal Nerve Block

In most patients, topicalization of the airway is sufficient to allow awake intubation. However, a pronounced gag reflex found in some patients leads to discomfort and subsequent failure of AI despite adequate topicalization. This occurs because the pressure receptors at the root of the tongue, which initiate the gag reflex, lie in the submucosa and are not consistently blocked by topical anesthesia [15]. A bilateral glossopharyngeal nerve block can be performed using either an intraoral or peristyloid approach in order to suppress the gag reflex. Aspiration before injection must be performed during this block, as both approaches involve deposition of local anesthetic in close proximity to the carotid artery.

The intraoral approach to the glossopharyngeal nerve block is performed in a patient who is able to open his or her mouth widely. As depicted in Fig. 19.3, after the patient's oral cavity is anesthetized and the mouth is opened, a number 3 Macintosh laryngoscope blade is used to assist with visualization of the palatine tonsil and the posterior tonsillar pillar. A 22-gauge or smaller spinal needle is then inserted 0.5 cm into the submucosa at the base of the posterior tonsillar pillar. Following a negative aspiration test, 2 ml of 1% lidocaine is then injected. The block is then repeated on the contralateral side. A variation of the intraoral approach is often performed by otolaryngologists and involves blocking the nerve from a posterior direction (Fig. 19.4) [16].

An alternative to the intraoral approach is the peristyloid approach to the glossopharyngeal nerve block. This technique is more appropriate for a patient who is unable to open his or her mouth wide enough for intraoral blockade. The patient is placed in a supine position, with the head in a neutral position. A line is then drawn to connect the mastoid process and the angle of the mandible, as shown in Fig. 19.5.

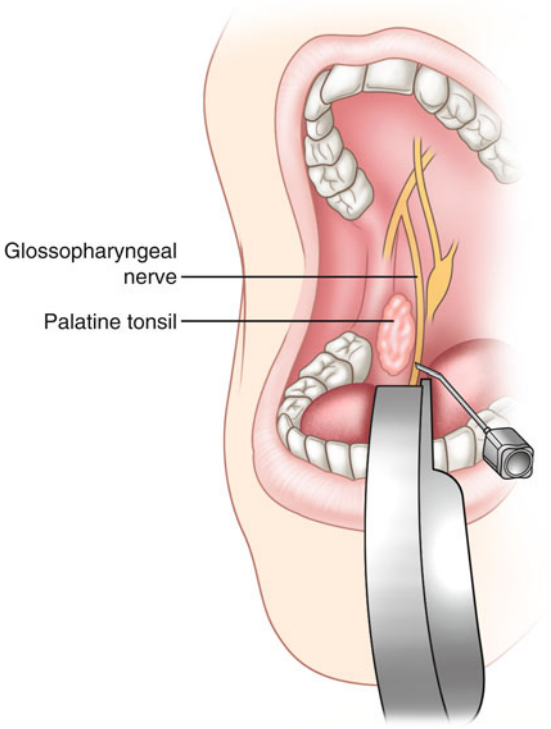

Fig. 19.4 Glossopharyngeal nerve block – intraoral approach. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

The styloid process is then palpated using deep pressure near the middle of this line. A short 22-gauge needle is then inserted and advanced until the styloid process is contacted. The needle is then withdrawn slightly and directed posteriorly off of the styloid process. Five milliliters of 1% lidocaine are injected after a negative aspiration test. The block is then repeated on the contralateral side.

A modified peristyloid approach can also be performed using an echogenic needle under ultrasound guidance. The ultrasound is used to identify the carotid artery and can decrease the risk of vascular injury and intravascular injection. This modification may improve the accuracy and safety of the glossopharyngeal nerve block [17].

Superior Laryngeal Nerve Block

During an AI, the superior laryngeal nerve is blocked bilaterally in order to facilitate passage of the endotracheal tube through the larynx. To block this nerve, the patient is placed in a supine or sitting position with the neck maximally extended. The neck region is prepped with an aseptic technique. The hyoid bone is displaced ipsilaterally.

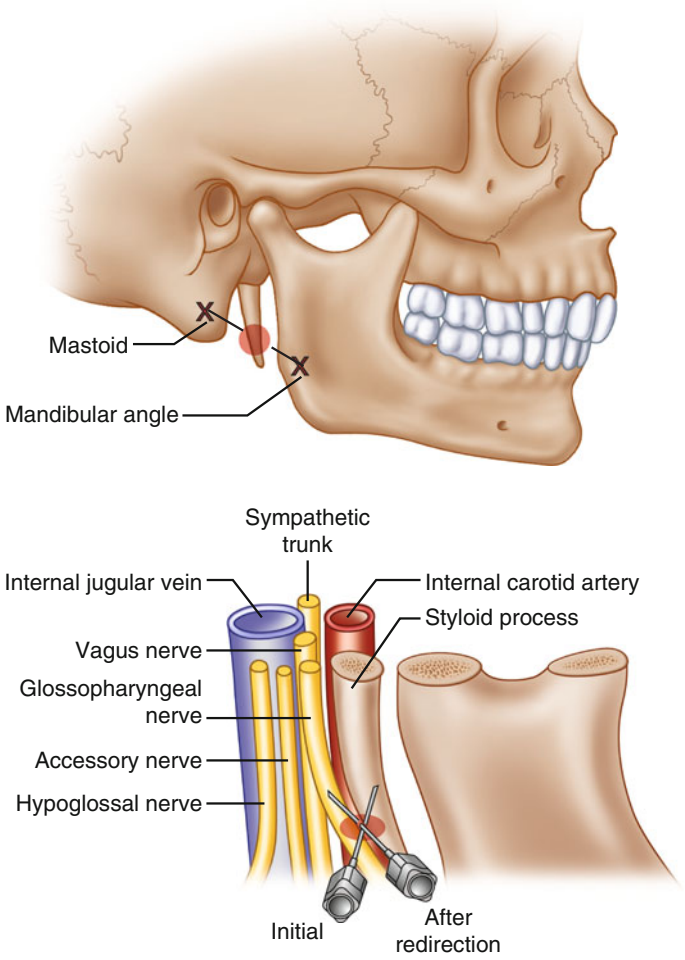

Fig. 19.5 Glossopharyngeal nerve block – peristyloid approach. Adapted from Brown DL. *Atlas of Regional Anesthesia* 3rd Ed, 2005

A 25-gauge needle is inserted and walked of the greater cornu inferiorly. The needle is then advanced 2–3 mm, thereby piercing the thyrohyoid membrane. Aspirate, and if no air or blood encountered, 2 ml of 1% lidocaine is then injected. If air is aspirated, the needle is probably in the airway; redirect and repeat the procedure. If blood is aspirated, the needle is intravascular; redirect and repeat the procedure (Fig. 19.6).

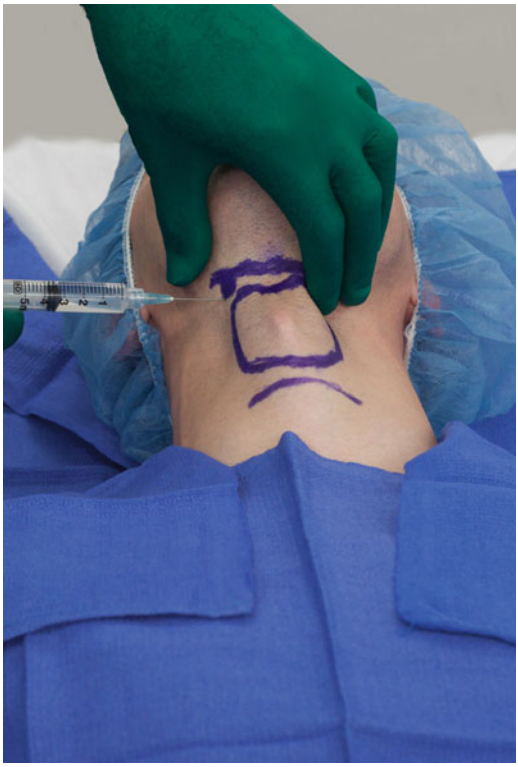

Fig. 19.6 Superior laryngeal nerve block

Recurrent Laryngeal Nerve Block

The transtracheal or translaryngeal block is performed in order to block the recurrent laryngeal nerve, thus properly anesthetizing the area below the vocal cords. This block prevents the patient from coughing as the endotracheal tube passes through the vocal cords, and it is also useful during an awake tracheostomy. The block is typically not performed on patients at risk for elevated intracranial pressure or intraocular pressure. To block this nerve, the patient cartilage is placed in a supine or sitting position. An aseptic technique is used to prep the neck. The thyroid cartilage and cricoid cartilage is palpated. The space between the two is the cricothyroid membrane. A 20-gauge angiocatheter is advanced midline in the neck through the cricothyroid membrane while aspirating for air. Once air is aspirated, the angiocatheter is advanced into the airway. The needle is removed and then the angiocatheter is again aspirated for air to confirm correct placement. At this point, 4 ml of 4% lidocaine is injected into the airway. The patient should cough, confirming placement of the local anesthetic in the airway. Only the sensory aspect of the recurrent laryngeal nerve is blocked (Figs. 19.7 and 19.8).

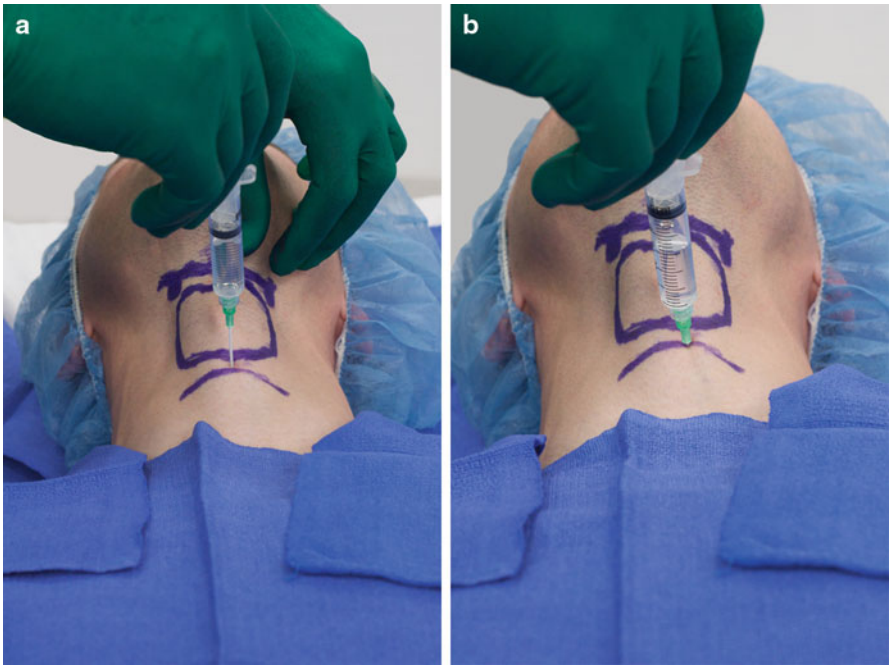

Fig. 19.7 Translaryngeal nerve block

Author's Approach

Education of the patient is paramount for a successful awake intubation. A cooperative patient is much easier to intubate than a combative one; therefore, the anesthesiologist must explain the steps of the procedure to the patient. After placement of the appropriate monitors, an antisialagogue is given to help minimize secretions. Light sedation is given to establish a level at which the patient is comfortable while at the same time responsive and breathing spontaneously. Topical anesthetic spray, aerosolized local anesthetics, or ointment can be used to help anesthetize the airway. Appropriate nerve blocks are then performed, as described above.

In a patient lying in the supine position, the tongue falls posteriorly into the back wall of the pharynx causing obstruction of the view with the fiber-optic bronchoscope; therefore, the head of the bed is raised. This forces the scope deeper into the back of the pharynx. Furthermore, in an upright patient, saliva pools low in the pharynx out of the field of view. The length of the scope is lubricated, and an appropriate size endotracheal tube is placed over it. A monitor is recommended as it allows everyone in the room to visualize what the airway manager is doing.

Face the patient and have an assistant pull the tongue forward with gauze or provide a jaw thrust [18]. An intubating airway is used to prevent the patient from biting on the scope. The scope is placed in the patient's mouth. The epiglottis and

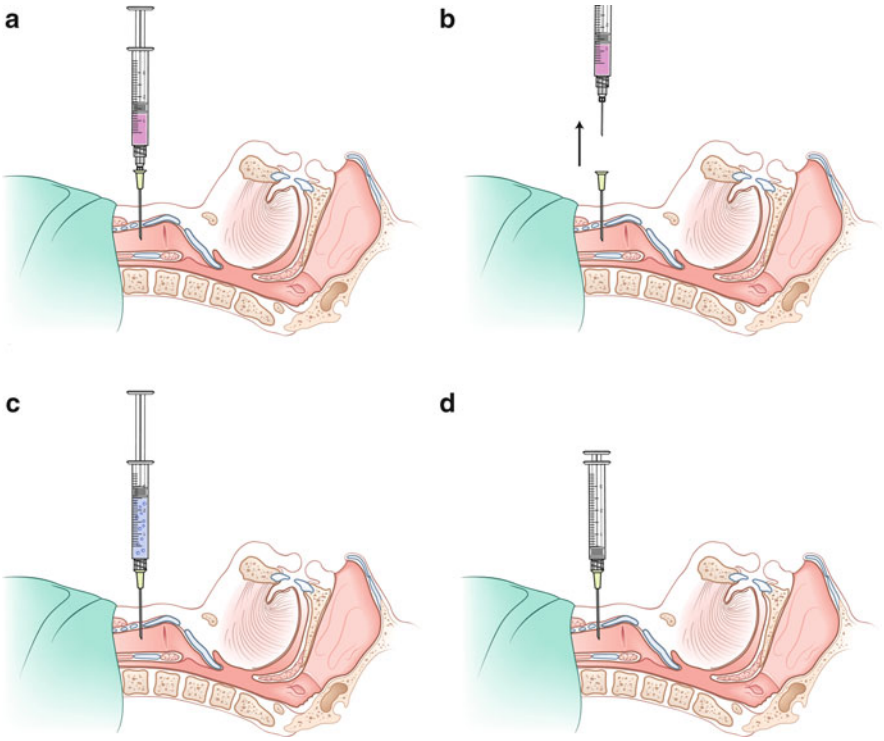

Fig. 19.8 Translaryngeal nerve block – midsagittal view of angiocatheter technique. Adapted from Hagberg CA. *Benumof's Airway Management* 2nd Ed, 2007. (**a**) Insertion of angiocatheter. (**b**) Removal of needle. (**c**) Confirmation of aspiration test. (**d**) Injection of local anesthetic

glottic opening should be visualized midline. Move the scope toward the glottic opening and pass it between the vocal cords. Once tracheal rings are visualized, advance the scope until the carina is observed and pass the endotracheal tube over the scope. Confirm visualization of the carina and then remove the scope. Inflate the cuff of the ETT and attach it to the breathing circuit. Check for end-tidal CO_2 and bilateral chest rise. Induction of the patient is appropriate at this time.

Multiple-Choice Questions

1. Which of the following nerves could be blocked in order to prevent a gag reflex?
   (a) Branches of the trigeminal nerve
   (b) Glossopharyngeal nerve
   (c) Superior laryngeal nerve
   (d) Recurrent laryngeal nerve

2. Care must be taken when performing a glossopharyngeal nerve block due to the potential for infiltration of the:
   (a) Carotid artery
   (b) Jugular vein
   (c) Thyroid cartilage
   (d) Vagus nerve

3. Bilateral blockade of the recurrent laryngeal nerve can result in:
   (a) Airway dilation
   (b) Airway obstruction
   (c) Pneumothorax
   (d) Hoarseness

4. Which of the following nerves must be blocked during an awake tracheostomy?
   (a) Trigeminal
   (b) Glossopharyngeal
   (c) Superior laryngeal
   (d) Recurrent laryngeal

5. The anterior ethmoidal nerve is a branch of:
   (a) Cranial nerve I (Olfactory)
   (b) Cranial nerve III (Orbital)
   (c) Cranial nerve V (Trigeminal)
   (d) Cranial nerve VII (Facial)

6. Which of the following muscles is innervated by the superior laryngeal nerve?
   (a) Cricoarytenoid
   (b) Cricothyroid
   (c) Thyroarytenoid
   (d) Vocalis

7. Which of the following local anesthetic used in topical anesthesia of the airway is associated with methemoglobinemia?
   (a) Benzocaine
   (b) Cocaine
   (c) Lidocaine
   (d) Tetracaine

8. Which of the following structures lies in close proximity to the internal branch of the superior laryngeal nerve as it pierces the thyrohyoid membrane?
   (a) Inferior thyroid artery
   (b) Lingual artery
   (c) Superior thyroid artery
   (d) Subclavian artery

9. Displacement of the hyoid bone in which of the following directions aids in the blockade of the superior laryngeal nerve:
   (a) Contralateral
   (b) Inferior
   (c) Ipsilateral
   (d) Superior
10. The left recurrent laryngeal nerve winds around which of the following structures:
   (a) Arch of the aorta
   (b) Left subclavian artery
   (c) Left subclavian vein
   (d) Left vertebral artery

Answers:

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

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Selective Regional Anesthesia Options in Surgical Subspecialties

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Iliohypogastric and Ilioinguinal Nerve Block

Introduction

These two nerves lie in close proximity to one another and are frequently blocked together with the same needle insertion. Dr. Harvey Cushing reported in the Annals of Surgery in 1900 that "almost all cases of hernia, with the possible exception of those in young children, could undoubtedly be subjected to the radical operation under local anesthesia" [1]. Today, because of infrequent use, some authors claim that the block is underutilized for herniorrhaphy [2]. Recently, Yilmazlar and colleagues compared the ilioinguinal/iliohypogastric nerve blocks to spinal anesthesia for inguinal herniorrhaphy. They found that patients receiving the ilioinguinal/iliohypogastric nerve blocks had shorter time-to-home readiness, quicker oral intake postsurgery, and no need for recovery room care [3].

Indications

This block is indicated as a treatment for acute and chronic pain involving the groin area, lower abdominal wall, and inguinal region. If used for herniorrhaphy, the hernia sac needs additional local infiltration because it contains peritoneum and visceral nerves. There is no specific contraindication for this block.

Anatomy

The iliohypogastric nerve may have a small contribution from T12, but primarily originates from L1. It travels around the body, starting posteriorly, and then heading laterally before emerging anteriorly. At the anterior superior iliac spine (ASIS), the iliohypogastric nerve pierces through the posterior portion of the transverse abdominal muscle and divides into lateral and anterior branches. The lateral branches penetrate both the internal and external oblique muscles and provide sensation to the skin of the posterior lateral gluteal region. The anterior branch penetrates through the internal oblique muscle approximately 2 cm medial to the anterior superior iliac spine and perforates the external oblique muscle, distributing sensory fibers to the skin of the abdomen above the pubis (Fig. 20.1).

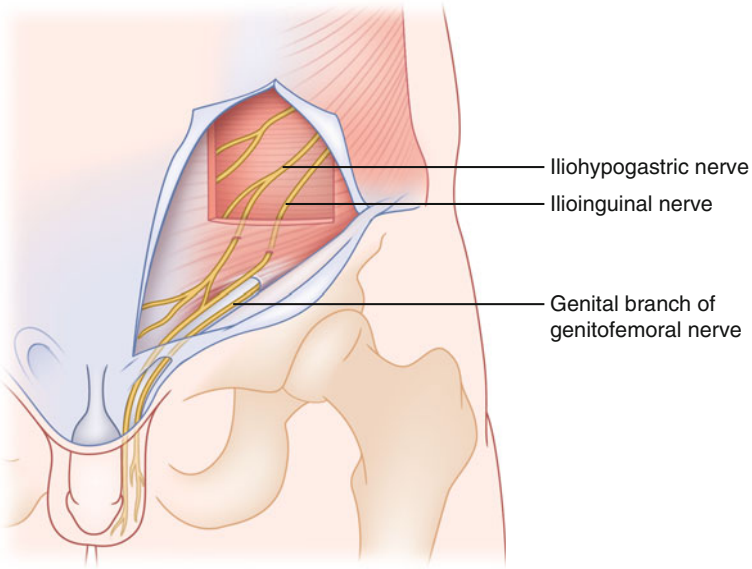

Fig. 20.1 Three nerves (Iliohypogastric nerve (IH), ilioinguinal nerve (IL), and genitofemoral nerve) starting from spinal cord to exiting inguinal canal

The smaller ilioinguinal nerve originates from L1. It emanates from the upper part of the lateral border of the psoas major muscle and courses caudad to the iliohypogastric nerve. It crosses obliquely and anteriorly to the quadratus lumborum and iliacus muscles and then perforates the transverse abdominis muscle near the anterior part of the iliac crest. In the anterior abdominal trunk, the nerve travels between the transverse abdominis and the internal oblique muscles (Fig. 20.1). It occasionally anastomoses with branches of the iliohypogastric nerve at the ASIS level. It pierces the internal oblique muscle and accompanies the spermatic cord through the inguinal ring into the inguinal canal. It provides skin sensation over the root of the penis, to the superior inner aspect of the thigh, to the upper part of the scrotum in males, and to the skin covering the mons pubis and lateral part of labia in females [4].

Technique

Ultrasound-Guided

After positioning the patient supine, locate and label the ASIS and the umbilical button. Next, draw a line between the ASIS and the umbilical button. A linear high-frequency (7–13 MHZ) ultrasound probe is used for this block. By adjusting the

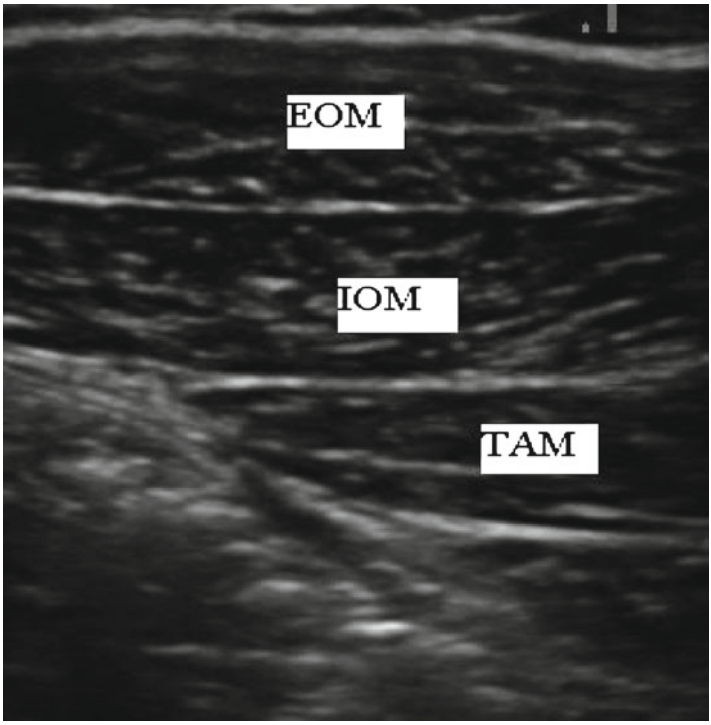

Fig. 20.2 Ultrasound image showing the layers of abdominal muscles. *EOM* the external oblique muscle, *IOM* the internal oblique muscle, *TAM* the transverse abdominal muscle

ultrasound setting (depth is usually better at 1–3 cm), the differential muscle layers can be visualized, and the nerves can sometimes be imaged. Unfortunately, the nerves are difficult to consistently identify. The ilioinguinal nerve is usually located in the plane between the transverse abdominal muscle and the internal oblique muscle above the ASIS, while the iliohypogastric nerve is usually located between the internal oblique muscle and the external oblique muscle. We typically use a 21-gauge or 23-gauge needle and inject locally throughout the needle's path with 5–8 ml of local anesthetic directly deposited to each nerve if possible. The total local anesthetic volume is 20–30 ml.

Ideally, one should identify and target the individual nerve; however, the nerves cannot always be identified. In this case, the reliable end points for the II/IH nerve blocks are the transverse abdominal/internal oblique muscle plane where the ilioinguinal nerve is reported to be found in 100% of cases [5], and the plane between the internal oblique muscle and the external oblique muscle, which contains the iliohypogastric nerve (Fig. 20.2).

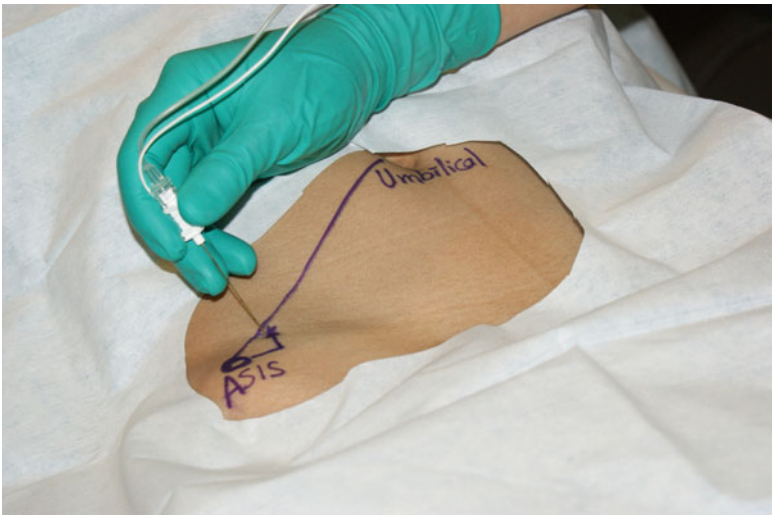

Fig. 20.3 Showing the ilioinguinal/iliohypogastric nerve block needle entry point: 3 cm medial and 3 cm above ASIS

Using Anatomical Landmarks

The patient is placed in a supine position. After marking the ASIS and drawing a line between the ASIS and the umbilical button, the patient's lower quadrant should be sterilely prepared. The injection site is located 3 cm medial to the ASIS and 3 cm above the ASIS (Fig. 20.3). As mentioned above, the key to adequate blockade is injecting sufficient local anesthetic into the two planes: the plane between the transverse abdominal muscle and the internal oblique muscle (the ilioinguinal nerve) and the plane between the internal oblique muscle and the external oblique muscle (the iliohypogastric nerve). Using a 21-gauge or 23-gauge needle, advance the needle at a right angle to the skin in all planes. A "click" is felt as the needle passes through the external oblique muscle. Before advancing further, deposit 8–10 ml of local anesthetic agent into this plane. Next, advance the needle until a second "click" is felt. This indicates that the needle has advanced through the internal oblique muscle. At this point, inject another 8–10 ml of local anesthetic agent. One should inject 8–10 ml into each plane and along the needle path. We limit our total dose to less than 40 ml.

Some anesthesiologists use two separate needle entry points for ilioinguinal and iliohypogastric blockade. To access and block the iliohypogastric nerve, a needle is directed 3 cm medial and 3 cm superior to the ASIS. Blockade of the ilioinguinal nerve can be accomplished by placing a needle 2 cm medial to the ASIS and 2 cm inferior to the entry point for the iliohypogastric nerve. Next, the needle is directed toward the pubic symphysis in a fan-like manner, piercing through the fascia of the

external oblique muscle and depositing local anesthetic along its path. Because the ilioinguinal and iliohypogastric nerves are located at different fascial planes among the three muscles (IO, EO, and TA), these blind techniques have a low success rate.

Anesthetic Agents

Our group typically uses 0.5% bupivacaine or 1% ropivacaine for surgical anesthesia and 0.25% bupivacaine or 0.5% ropivacaine for postoperative analgesia or chronic pain analgesia. Beaussier et al. reported that adding clonidine (75 μg) to local anesthetic (ropivacaine) can reduce motion pain but may increase the chance of orthostatic hypotension [6]. Popping and colleagues analyzed multiple studies and concluded that adding clonidine to intermediate- or long-acting local anesthetics for single-shot peripheral nerve or plexus blocks prolongs the duration of analgesia and motor blockade by about 2 h [7]. The increased risk of hypotension, fainting, and sedation may limit its usefulness. After studying three concentrations (0.125, 0.25, and 0.375%) of levobupivacaine, Disma et al. reported that 0.25% levobupivacaine provided satisfactory postoperative analgesia and fewest side effects [8].

Continuous ilioinguinal/iliohypogastric nerves block with ultrasound-guided placement of bilateral catheters has been reported [1]. This block provides postoperative analgesia for procedures using a Pfannenstiel incision. Also, it provides a good option for patients when epidural analgesia is contraindicated. The technique involves inserting an 18-gauge Tuohy epidural needle at the same entry point as single-shot block (3 cm medial and 3 cm above the ASIS). With ultrasound guidance, after penetrating the external oblique muscle and internal oblique muscle, a multilumen catheter is threaded through the Tuohy needle into the plane between the internal oblique muscle and the transverse abdominal muscle. The catheter should be directed medially about 3 in. This block should be performed bilaterally. Once placed, each catheter is connected to a continuous infusion of 0.5% ropivacaine or 0.2–0.25% bupivacaine set at a flow rate of 2 ml/h. This technique is very similar to the transversus abdominis plane block (TAP) but differs in two ways: (1) the needle entry for the ilioinguinal/iliohypogastric continuous block is more medial than the technique for the TAP block and (2) this technique aims for blockade of L1 and T12, while the TAP technique blocks sensory fibers from T10 to L1, or even higher. Gucev et al. placed continuous catheters into the plane between the internal oblique muscle and the transverse abdominal muscle. Using 0.2% ropivacaine with oral ibuprofen for postoperative analgesia after cesarean delivery resulted in lower pain scores postoperatively, found minimal use of supplemental opioids, and had no reports of nausea and vomiting [9]. This suggests that continuous ilioinguinal/iliohypogastric nerve blockade deserves further study, as a possible component of multimodal analgesia after cesarean delivery.

Complications

1. Hemodynamic changes are usually minimal because this block does not cause sympathetic blockade.
2. Local anesthetic toxicity is always a concern, but the possibility in this block is very small, even though this block involves multiple-point injections. The volume is small, and the blood circulation at the injection sites is less luxurious than the epidural or intercostal spaces. The total dose is significantly lower than the toxic dosage.
3. There are reports of small and large bowel perforations, so a blunt needle is recommended for this block. When inserting the needle, try to avoid being too deep or inserting without assurance of needle location. In most patients, the needle is inserted no more than 1.5 cm after passing through the external oblique muscle layer.
4. Subcutaneous hematoma can occur after this block.
5. Pelvic hematomas have been reported, as have bowel hematomas in pediatric patients [10].
6. Transient femoral anesthesia was reported to occur in about 3.5–7% of the patients who received ilioinguinal/iliohypogastric nerve block and occurs more frequently when the injection site is located lower than the ASIS and the needle tip is deep.

Genitofemoral Nerve Block

Introduction

The genitofemoral nerve block is used as treatment for chronic pain of the pelvis, the perineal area, and the upper thigh and can be combined with ilioinguinal/iliohypogastric nerve blocks for surgical procedures involving the groin area.

Indications

1. Performed with ilioinguinal and iliohypogastric nerve blocks for inguinal hernia repair, orchiopexy, and hydrocelectomy
2. As a nerve block supplementing femoral nerve block for long saphenous vein stripping
3. For the diagnosis of genitofemoral neuralgia
4. For chronic pain syndromes in the pelvic or groin areas, such as border nerve syndrome [11]

There are no specific contraindications for this block.

Anatomy

The genitofemoral nerve originates from the L1 and L2 ventral rami and is formed within the psoas major. The nerve, primarily sensory in function, contains a small motor component and descends obliquely, advancing through the psoas muscle to emerge at its abdominal surface near the medial border. There, the genitofemoral nerve divides into femoral and genital branches at various distances from the inguinal ligament. The femoral branch joins the femoral artery and travels underneath the inguinal ligament, penetrating the fascia lata. It supplies sensation to a small area of skin immediately below the inguinal ligament. The genital branch enters the inguinal canal through the deep ring and travels with the spermatic cord to supply the cremaster and dartos muscles and sends small terminal sensory fibers to the skin of the scrotum in males. It runs inside the inguinal canal with terminal fibers to the round ligament of the uterus and the skin of labium majus in females.

The Technique

Ultrasound-Guided Technique

For block of the genital branch, we will use the technique described by Peng. The probe is placed perpendicular to the inguinal ligament. Adjusting the probe position, usually cephalad, identify the femoral artery. It serves as a reference structure. Next, identify the internal ring and spermatic cord, which is oval or circular in shape and contains one or two arteries (the testicular artery and the artery to the vas deferens). The vas deferens is often seen as a thick tubular structure within the spermatic cord. The probe is then moved medially and caudally to a final location approximately 1 in. lateral to the pubic tubercle. An out-of-plane technique is usually used with the needle approaching the skin from the lateral aspect of the probe. Local anesthetic without epinephrine is used to avoid the possible vasoconstriction effect on the testicular artery. Because of the anatomical anomalies found with the location of the genital branch in the genitofemoral nerve, we suggest depositing 5 ml of local anesthetic inside and another 5 ml outside the spermatic cord to provide adequate blockade [12].

The femoral branch can usually be visualized with the ultrasound probe immediately lateral to the femoral artery. It sometimes appears attached to the femoral artery lateral wall. Block of the femoral nerve can also block this nerve.

Anatomical Landmark Technique

The femoral branch is blocked by locating the femoral artery pulse. After inserting a 25-G needle just lateral to the femoral artery pulse, deposit 5 ml of local anesthetic solution. Next, inject 5 ml of local anesthetic in a fan-like pattern along a 5–7 cm path inferior to femoral pulse.

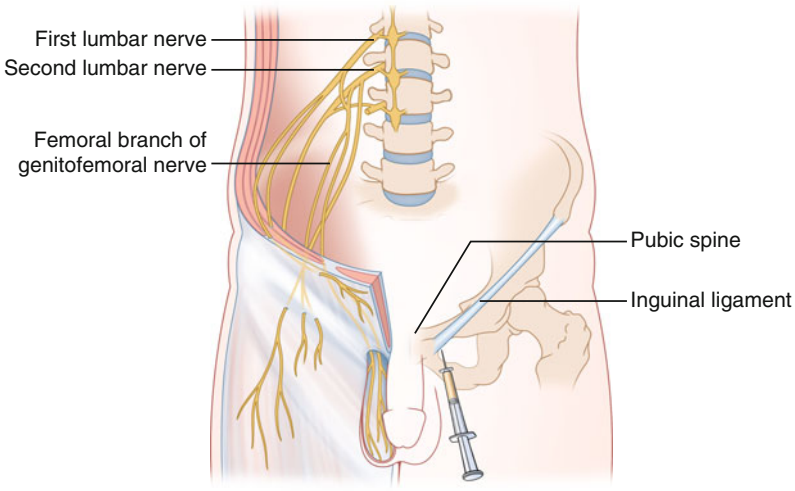

Fig. 20.4 Anatomic landmark of genital branch of genitofemoral nerve

The genital branch is blocked by identifying the pubic tubercle and inserting a 25-G needle 1 in. lateral to the pubic tubercle and below the inguinal ligament. A total local anesthetic volume of 10 ml, without epinephrine, is needed to achieve this block (Fig. 20.4).

Complications

1. Local pain.
2. Local anesthetic toxicity is always a concern, but with this block, risk is low because the total local anesthetic dose is significantly below the toxic dose. Also, blood flow is not as rich as in the epidural or intercostal spaces.
3. Subcutaneous hematoma can occur after this block.

Penile Block

For many years, the penile block has been widely used for circumcisions and other penile surgeries. However, the anatomy of the penile block still confuses anesthesiologists and contributes to variation of techniques.

Indications

1. Circumcision
2. Phimosis and paraphimosis reduction

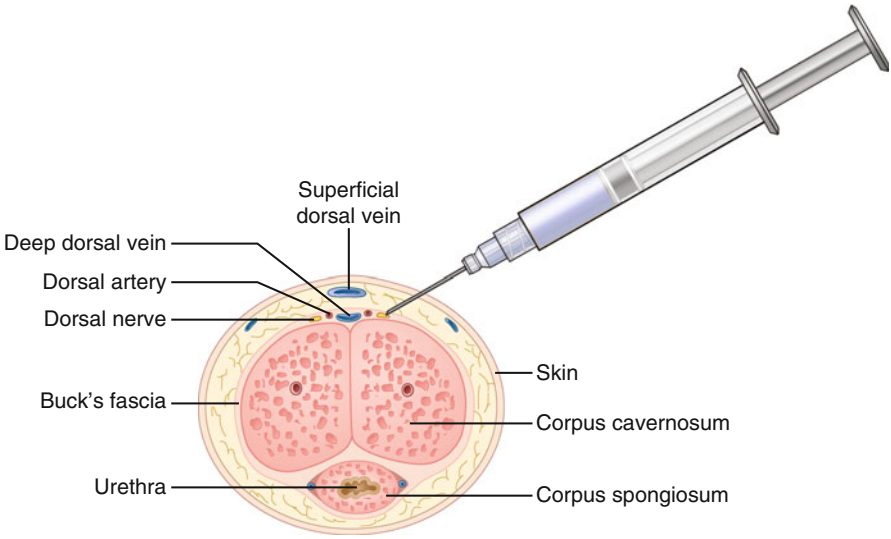

Fig. 20.5 Penile block

3. Dorsal penile skin surgery
4. Distal hypospadia repair
5. Postoperative analgesia in penile surgery

Contraindications

1. Suspected testicular torsion
2. Infection of the skin at the proposed injection site

Anatomy

The penile nerve is derived from the pudendal nerve (S2-4). The penile nerve divides into the right and left dorsal nerves of the penis and courses under the pubis symphysis. Next, it travels under Buck's fascia to supply sensory innervations to the penis. Both left and right penile nerves travel lateral to the penile arteries (Fig. 20.5).

Technique

Although many variations in blockade technique exist, the most common approach targets the two dorsal penile nerves for local anesthetic injection as well as local deposition circumferentially subcutaneously. Recent studies indicate that to achieve

adequate foreskin analgesia, supplementing dorsal nerve blocks with ventral subcutaneous infiltration just proximal to the incision line will improve surgical anesthesia and avoid inconsistency [13]. Metzelder found that the penile block for hypospadia repair in children works better than caudal anesthesia (significantly less impaired micturition) [14].

Ultrasound-Guided Penile Dorsal Nerve Block

Sandeman described this ultrasound-guided penile block in children under general anesthesia. They used real-time scanning to guide bilateral injections of local anesthetics into the subpubic space, deep to Scarpa's fascia either side of the midline fundiform ligament. Scanning can confirm that the local anesthetic has spread to contact the deep fascia on each side. A subcutaneous wheel of local anesthetic along the penoscrotal junction completes the block.

Complications

1. Inadequate block is common.
2. Hematoma occurs.
3. Penile ischemia is very rare.

Clinical Pearls

Iliohypogastric and Ilioinguinal Nerve Block

- Sedate the patient before proceeding with the block.
- The reliable end point for the inexperienced practitioner using ultrasound guidance for block of the ilioinguinal and iliohypogastric nerves is the plane between the transverse abdominal and the internal oblique muscles. The nerves are located in this plane in almost 100% of cases [5].
- One should feel resistance while moving through muscle tissue and a loss of this resistance when exiting the muscle. A blunt needle will make the loss of resistance more appreciable.
- Regardless of technique, if the nerves are not easily identifiable, target the anatomic plane where the nerves lie to deposit the local anesthetic.
- If you locate the target nerves, try to keep them in the middle of the ultrasound image.

- Pay attention to needle insertion depth. Do not insert the needle too deep and avoid getting into peritoneal cavity. This will reduce the incidence of bowel perforation.
- Adding a genitofemoral nerve block to the ilioinguinal/iliohypogastric nerve block may not offer any extra benefit to pediatric patients undergoing hernia repair [14].

Genitofemoral Nerve Block

- Successful injection of the genitofemoral branches requires appropriate volume, typically requiring 10 ml or more of local anesthetic solution.
- A multidirection infiltration will help the adequacy of the block.
- Just use plain local anesthetics; do not mix with epinephrine.
- Sterile preparation is important because the area is a breeding ground for pathogens.

Penile Block

- If possible, try to feel the pulse of the penile artery. The needle insertion site is less important because of the skin mobility; inject lateral to the pulse. The superficial dorsal vein can serve as a landmark for midline. Deposit local anesthetics under Buck's fascia where the penile nerves travel.
- Because the superficial and deep dorsal veins are both located at the dorsal midline, try to avoid a straight-down midline approach. This will significantly minimize the occurrence of hematoma.
- Penile ischemia can be prevented by avoiding puncture of the penile arteries, avoiding a larger than necessary volume of local anesthetic, and avoiding hematoma formation.

Multiple Choice Questions

1. The primary nerve root supplying the ilioinguinal and iliohypogastric nerves is:
   (a) L3
   (b) L2
   (c) L1
   (d) T12

2. All the following are advantages of ilioinguinal and iliohypogastric blocks compared to spinal except:
   (a) Quicker postoperative discharge
   (b) Faster postoperative oral intake
   (c) Less need for recovery room
   (d) Less postoperative surgical complications

3. The ilioinguinal nerve supplies sensation to all the following areas except:
   (a) Skin covering the base of the penis
   (b) Skin covering the upper scrotum
   (c) Skin covering the mons pubis
   (d) Skin covering the posterior aspect of the upper thigh

4. The ilioinguinal and iliohypogastric nerves are commonly located between the:
   (a) Transverse abdominal muscle and internal oblique
   (b) Internal oblique and external oblique
   (c) Transverse abdominal and rectus sheath
   (d) Rectus sheath and aponeurosis of external oblique

5. Continuous ilioinguinal and iliohypogastric blocks for Pfannenstiel incisions:
   (a) Place catheter unilateral
   (b) Place bilateral catheters between transverse abdominal and internal oblique
   (c) Place bilateral catheters between external and internal oblique
   (d) Place bilateral catheters between internal and external oblique aiming medially

6. Complications to the ilioinguinal/iliohypogastric block include all except:
   (a) Hemodynamic instability
   (b) Bowel perforation
   (c) Subcutaneous hematoma
   (d) Pelvic hematoma

7. Indications for genitofemoral nerve block include all except:
   (a) Supplemental block for hernia surgery
   (b) Aid in diagnosis of genitofemoral neuralgia
   (c) Treatment of some chronic pelvic pain syndromes
   (d) Primary block for orchiopexy surgery

8. The genitofemoral nerve originates from:
   (a) Dorsal rami of T12 and L1
   (b) Dorsal rami of L1 and L2
   (c) Ventral rami of L1 and L2
   (d) Ventral rami of T12 and L1

9. The genital branch of the genitofemoral nerve:
   (a) Enters the inguinal through the deep ring
   (b) Travels with the spermatic cord
   (c) Supplies the cremaster and dartos muscles
   (d) All of the above

10. Anatomical landmarks used for ultrasound block of the genital branch include all except:
    (a) ASIS
    (b) Umbilical button
    (c) Inguinal ligament
    (d) Quadriceps muscle

11. Clinical pearls for genitofemoral block include all except:
    (a) Use epinephrine mixed with local anesthetic
    (b) Use multidirectional infiltration
    (c) Sterile preparation extremely important
    (d) Use local anesthetic without epinephrine

12. Indications for penile block include:
    (a) Cystoscopy
    (b) Retrograde urethrogram
    (c) Circumcision and distal hypospadia repair
    (d) Testicular torsion

13. Contraindications for penile block:
    (a) Dorsal penile skin surgery
    (b) Postoperative analgesia for penile surgery
    (c) Penile skin infection
    (d) Phimosis surgery

14. All of the following concerning the penile nerve are true except:
    (a) Derived from S2-4
    (b) Courses under the pubic symphysis
    (c) Travels under Buck's fascia
    (d) Blocked with local anesthetic containing epinephrine

15. Complications from penile block:
    (a) Inadequate block
    (b) Penile ischemia is common
    (c) Hematomas are rare
    (d) Local anesthetic toxicity from large volume doses

Answers:

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

10. d
11. a
12. c
13. c
14. d
15. b

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Regional Anesthesia for Chronic Disease States

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CNS Disorders

Historically, the use of regional anesthetic techniques in patients with preexisting central nervous system (CNS) disorders has been considered relatively contraindicated. Probably, the most conservative legal approach in these patients is to avoid regional anesthesia. The recommendations of Vandam and Dripps in 1956 were to avoid spinal anesthesia in patients with preexisting CNS disorders, and these recommendations have greatly influenced the clinical management of these patients for the last several decades. The cause of postoperative neurological disorders is multifactorial and is usually difficult to evaluate because of the many patients and surgical and anesthetic risk factors that may play a role, Table 21.1 [1]. Therefore, the abundance of contributing factors makes it extremely difficult for clinicians and investigators alike to reliably isolate the effect of anesthetic technique on neurologic outcome.

However, high-risk patients, including those with significant cardiopulmonary disease, may benefit medically from regional anesthesia and analgesia. The decision to proceed with regional anesthesia in these patients should be made on a case-by-case basis. Meticulous regional anesthetic technique should be observed to minimize further neurologic injury.

Increased ICP

Dural puncture is not recommended in patients with clinical or radiological signs of increased intracranial pressure, such as a patient with primary and metastatic brain tumors. CT or MRI evidences of high ICP are cerebral edema, lateral shift of the midline structures, and obliteration of the fourth ventricle. The associated leakage of cerebrospinal fluid (CSF) following dural puncture decreases CSF pressure and may produce transtentorail and cerebellar herniation. Epidural and caudal anesthesia are also contraindicated in patients with increased intracranial pressure because of the risk of accidental dural puncture and because the intracranial pressure may be further increased by injection of local anesthetic solution into the epidural space [1].

Table 21.1 Contributing factors to deterioration in pre-existing neurological status

- Extremes of age/body habitus
- Surgical trauma
- Tourniquet inflation pressures/length of time for inflation
- Prolonged/difficult labor or normal vaginal delivery can result in a host of neurological deficits
- Improper patient positioning
- Anesthetic technique

Intracranial Aneurysms and Arteriovenous Malformations

Patients with preexisting uncorrected and vascular lesions, such as saccular aneurysms or arteriovenous malformations, are at increased risk of neurological compromise during spinal or epidural anesthesia. Alterations in intracranial pressure and mean arterial pressure associated with neuraxial block may result in subarachnoid hemorrhage or cerebral infarction. Neuraxial anesthesia in patients with a previous ischemic stroke is considered safe. Cerebral perfusion pressure should be maintained to prevent further ischemic damage [1].

Vascular malformations in the spinal cord, subdural, and epidural space are associated with congenital disease such as in Von Hippel–Lindau and Klippel–Trenaunay syndrome. Pregnancy, labor, and expulsion may augment the bleeding risk. Magnetic resonance imaging may allow verification of the possible safety of a neuraxial block. Neuraxial anesthesia should be avoided when such an examination is not available [2].

Seizure Disorders

Majority of seizures occurring in the perioperative period in patients with a preexisting seizure disorder are likely related to the patient's underlying condition, and that regional anesthesia in these patients is not contraindicated. Furthermore, because the likelihood of a postoperative seizure is increased in patients with a recent seizure, it is essential to be prepared to treat seizure activity, regardless of the anesthetic and analgesic technique [3]. On the other hand, recent onset of seizure may represent pathologic intracranial conditions such as neoplasm, trauma, infection, or stroke. So in these situations, extra precaution should be taken due to primary pathological conditions causing seizure disorders.

Chronic Preexisting Central or Peripheral Nerve Conditions

Patients with a preexisting neurological condition may be at increased risk for regional anesthesia-related nerve injury on the basis of the "double crush," which hypothesizes that nerve fibers that are already compromised are also more vulnerable to injury at another site (Fig. 21.1) [4].

Multiple Sclerosis

Multiple sclerosis is an acquired central nervous system disease characterized by multiple sites of demyelination in the brain and spinal cord. Multiple sclerosis does not affect the peripheral nervous system. Demyelination of axons results in a slowing

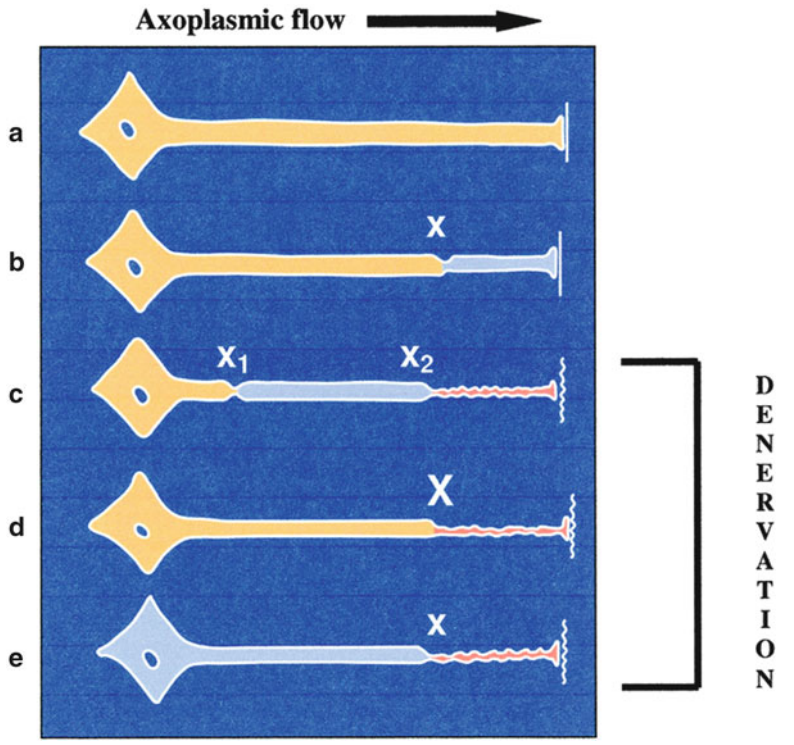

Fig. 21.1 The double-crush phenomenon. Axoplasmic flow is indicated by the *degree of shading*. Complete loss of axoplasmic flow results in denervation (**c–e**). (**a**) Normal neuron. (**b**) Mild neuronal injury at a single site (x) is insufficient to cause denervation distal to the insult. (**c**) Mild neuronal injury at two separate sites (x_1 and x_2) may cause distal denervation (i.e., double crush). (**d**) Severe neuronal injury at a single site (x) may also cause distal denervation. (**e**) Axon with a diffuse, preexisting underlying neurologic disease process (toxic, metabolic, and ischemic) may have impaired axonal flow throughout the neuron, which may or may not be symptomatic but predisposes the axon to distal denervation after a single minor neural insult at x (i.e., double crush)

of sensory and motor conduction, which leads to widely variable clinical signs and symptoms specific to the sites of demyelination. Epidural and, more often, spinal anesthesia have been implicated in the relapse of multiple sclerosis, although the evidence is not strong [1]. Patients with multiple sclerosis may have exacerbations of their symptoms over time, which may occur in an unpredictable fashion. Anesthesiologists have been cautious because of concern that there could be an exacerbation of the disease due to stress, fatigue, changes in temperature, and infection in perioperative period or due to natural course of disease, which could occur and even act as a coincidence. The mechanism by which spinal anesthesia may exacerbate multiple sclerosis is presumed to be direct local anesthetic toxicity. Epidural anesthesia has been recommended in preference to spinal anesthesia because the

concentration of local anesthetic in the white matter of the spinal cord after epidural administration is one fourth of spinal anesthesia. A dilute solution of local anesthetic with spinal or epidural anesthesia is also advised [1]. Because multiple sclerosis is a disorder of the CNS, peripheral nerve blocks do not affect neurological function and are considered appropriate anesthetic techniques. The largest series of neuraxial anesthesia in patients with preexisting CNS conditions involved 139 patients [4]. Postpolio syndrome and multiple sclerosis were the most common CNS disorders in this study. There were no patients with new or worsening postoperative neurological deficits compared with preoperative findings.

Amyotrophic Lateral Sclerosis

ALS is a degenerative disease with clinical features of both upper and lower motor neuron lesions with variability depending on the muscle groups involved. The clinical features of ALS involve progressive muscular atrophy with weakness and fasciculations of skeletal muscles. Bulbar muscle weakness often predominates with an associated risk of aspiration. Autonomic nervous system dysfunction is common with the associated risk of exaggerated hemodynamic responses during anesthesia. Epidural anesthesia has been successfully used in patients with ALS. However, a high epidural or spinal block can affect intercostal muscle function with detrimental effects in patients with minimal ventilatory reserve [5].

Chronic Spinal Cord Injury

Spinal cord injury results from trauma, bleeding, or a tumoral process. Possible consequences are bone decalcification, muscle spasms, pressure sores, deep venous thrombosis, thermoregulation problems, urological and renal complications, infections, metabolic disturbances, cardiovascular problems, and respiratory insufficiency. Lower extremity or abdominal surgery and also delivery may theoretically be performed without anesthesia if there is complete sensory loss at the operative site. However, depending on the level of cord transection, especially with lesions above T7, autonomic hyperreflexia may occur after skin trauma, distention, or examination of hollow viscus. Subsequent hypertensive crisis may result in headache, flushing, pupillary dilatation, convulsions, and intracranial bleeding. Prevention and early treatment of autonomic hyperreflexia is critical. Regional anesthesia is preferred to general anesthesia to prevent autonomic dysreflexia. A titratable technique using a combined spinal–epidural (CSE), continuous spinal or epidural should be considered rather than a single injection. Potential problems may be difficulty in placement, difficulty in control or examination of block level, and a potential increased risk of hypotension [2].

Previous Spine Surgeries

Previous spinal surgery has been considered to represent a relative contraindication to the use of regional anesthesia. This group of patient may vary from a simple lumbar laminectomy to a more extensive posterior spinal fusion and bone graft and placement of Harrington rod. Several postoperative anatomic changes make needle or catheter placement more difficult and complicated. Adhesions within or obliteration of the epidural space may affect the spread of epidural local anesthetic, producing an incomplete or "patchy" block, and may also increase the incidence of dural puncture. Thus, historically, it was concluded that epidural anesthesia may be successfully performed in patients who have had previous spinal surgery, but successful catheter placement may be possible on the first attempt in only 50% of patients, even by an experienced anesthesiologist. Although adequate epidural anesthesia is eventually produced in 40–95% of patients, there appears to be a higher incidence of traumatic needle placement, unintentional dural puncture, and unsuccessful epidural needle or catheter placement, especially if spinal fusion extends to between L-5 and S-1 [1]. In case of an extensive lumbar surgery and use of thoracic epidural for postoperative abdominal pain, it should be considered that epidural adhesions may hinder spread of local anesthetic to lumbar levels, so total volume required to produce adequate analgesia may be lower than normal.

Spinal Stenosis

Postoperative neurologic complications may be more likely or more severe in patients with preexisting severe spinal stenosis or other obstructive spinal canal pathology. In patients with known severe spinal stenosis or mass lesions within the spinal canal, a careful risk-to-benefit assessment of regional anesthesia to alternative perioperative anesthesia and analgesia techniques should be considered. In these patients, high local anesthetic volume neuraxial techniques (i.e., epidural anesthesia) may be associated with a higher risk of progressive mass effect when compared with low-volume techniques (i.e., spinal anesthesia) [6].

Peripheral Neurological Deficit

Patients with diseased or previously injured nerves (e.g., diabetes mellitus, severe peripheral vascular disease, or chemotherapy) may theoretically be at increased risk for block-related nerve injury. Although isolated case reports have described new or progressive neurologic deficits after regional anesthetic techniques in patients with multiple sclerosis or previous exposure to chemotherapy, clinical experience can neither refute nor confirm these concerns. Based on limited animal data, consideration may be given to avoiding more potent local anesthetics, reducing local anesthetic

Table 21.2 Mechanism of neurological injury directly related to regional anesthesia

- Direct needle- or catheter-induced trauma
- Paresthesia techniques
- The needle-bevel configuration, short/ long bevel
- Prolonged exposure to high concentrations of local anesthetic solutions
- Neural ischemia may occur as a result of systemic or local vascular insufficiency

dose and/or concentration, and avoiding or limiting vasoconstrictive additives in these patients [6]. There are no animal or human data to support the superiority of one nerve localization technique paresthesia, nerve stimulation, and ultrasound over another with regards to reducing the likelihood of nerve injury (Table 21.2).

Guillain–Barré Syndrome

Guillain–Barré syndrome also involves the peripheral nervous system. Painful distal extremity paresthesias are common, and autonomic dysfunction occurs in a significant number of patients. Guillain–Barré usually resolves spontaneously over weeks to months, but approximately 20% of patients will have residual neurologic deficits. Regional anesthesia is generally avoided in acute demyelinating phase of the disease. However, epidural narcotics have been used without complication in the acute phase of the disease in an attempt to control painful paresthesias. Epidural anesthesia has been used in parturients with some residual effects from an episode of Guillain–Barré in the past without adverse effects [5].

VP Shunt

VP shunt poses another challenge for neuraxial block, although formal contraindication for neuraxial block does not exist in the literature. It is common neurosurgical practice to perform lumbar puncture in patients with valveless shunts, as routine for fever investigation. There is a case report of successful spinal anesthesia for C/S in a patient with VPS without any neurologic changes secondary to the technique used [7]. Another series of five infants with VPS received spinal anesthesia for elective abdominal and perineal surgery. There was no report of complication due to spinal anesthesia [8].

Cardiovascular Disorders

The perioperative management, not just anesthetic technique, will dictate the outcome. Adherence to properly drawn clinical protocols can positively influence outcome. If regional anesthesia is selected, strict control of blood pressure and heart rate during the perioperative period is required [9].

Table 21.3 Effect of regional anesthesia on myocardial oxygen supply and demand

- Epidural anesthesia is associated with lower catecholamine levels.
- Prevent sympathetically mediated decrease in myocardial oxygen supply (vasoconstriction and hypercoagulable state and thrombosis).
- Selective blockade of cardiac sympathetic innervation (T1–T5) can increases blood flow to ischemic regions of myocardium.
- Improvement in regional distribution of myocardial blood flow by increasing the endocardial to epicardial blood flow ratio, although total coronary blood flow remains unchanged.
- Increased the luminal diameter of stenotic epicardial coronary arteries without changing the diameter of non stenotic segments.

Ischemic Heart Disease

A large percentage of patients who undergo surgery in the United States have risk factors for or have known coronary artery disease, and cardiac morbidity is the primary cause of death after anesthesia and surgery. Regional anesthesia has favorable effects on ischemic heart (Table 21.3) [10]. Some of these effects depend on dermatomal level of regional block and some do not. The choice of anesthesia is best left to the discretion of the anesthesia care team, which will consider the need for postoperative ventilation; cardiovascular effects, including myocardial depression; sympathetic blockade; and dermatomal level of the procedure. No one technique demonstrates a consistent advantage.

Chronic Heart Failure

The decision to use peripheral nerve block – neuraxial anesthesia as main anesthetic or adjunct to anesthesia or epidural for postoperative pain management – in patients with chronic heart failure depends on multiple factors, and generalization of recommendations is not possible. Yaeger et al. [11] reported that only 1 of 28 patients (3.6%) receiving epidural anesthesia (and "light levels of general anesthesia") and postoperative epidural analgesia developed CHF versus 10 of 25 patients (40%) given general anesthesia and postoperative parenteral narcotic analgesia. Although other studies have concluded that the choice of anesthetic techniques does not significantly influence cardiac morbidity and overall mortality.

Valvular Heart Disease

Understanding of the pathophysiology of each valvular heart disease and the physiologic perturbations of neuraxial anesthesia is extremely important in management of such patients. Use of invasive monitoring should be considered in severe cases.

Of the various valvular heart disease states, moderate and severe aortic stenosis (AS) is generally considered a contraindication to neuraxial anesthesia. The main reason is a risk of sudden and potentially profound decrease in systemic vascular resistance, which may precipitate life-threatening compromise in coronary perfusion. However, current evidence in the literature lacks the scientific validity provided by randomized clinical trials. The best information available are a few anecdotal observations that neuraxial anesthesia has been administered successfully in patients with significant AS while no contradictory evidence was found (i.e., adverse outcomes with neuraxial blockade in the same patient population). Benefits of regional anesthesia may outweigh the risks when the appropriate technique (continuous spinal or epidural) is selected and carefully conducted [12].

Pulmonary Hypertension

Primary pulmonary hypertension is a serious disease associated with high mortality during surgery and anesthesia. Literature on these patients is mostly limited to obstetrics case reports. Epidural anesthesia or combined spinal–epidural anesthesia has been used with great success. Spinal anesthesia should be avoided due to rapid hemodynamic changes.

Pulmonary Disease

Postoperative pulmonary complications are as frequent and clinically important as cardiac complications in terms of morbidity, mortality, and length of stay. Factors associated with an increased likelihood of developing pulmonary complications include preexisting chronic lung disease, abnormal results of pulmonary function tests, age >60 years, upper abdominal or thoracic surgery, smoking, obesity, and total anesthesia time > 3 h. Anesthetics disrupt central regulation of breathing and result in uncoordinated neural messaging. Due to resulting hypoventilation plus positional dependence, regional atelectasis occurs shortly after induction. It persists postoperatively and is compounded by ongoing disruption of respiratory muscles, limited respiratory excursion due to pain, and disruption of neurally mediated diaphragmatic functions after manipulation of abdominal viscera. Neuraxial blockade (either spinal or epidural anesthesia) blocks a constellation of stress responses to surgery (neuroendocrine, cytokine, and pain threshold) and may improve recovery and prevent complications. Postoperative epidural analgesia may reduce respiratory muscle dysfunction and pain-related hypoventilation. Intraoperative neuraxial blockade, either alone or in combination with general anesthesia, may prevent postoperative pulmonary complications, but the evidence is conflicting. Several metaanalyses (which included small unblinded studies) suggest that epidural anesthesia may reduce pulmonary risk, but recent large randomized trials do not confirm benefit [13].

Postoperative epidural analgesia may further reduce postoperative pulmonary complications. Good evidence suggests that lung expansion therapy (for example, incentive spirometry, deep breathing exercises, and continuous positive airway pressure) reduces postoperative pulmonary risk after abdominal surgery. Success of lung expansion therapy depends mostly of good level of analgesia.

Hepatic and Biliary Tract Disease

Delayed metabolism of local anesthetics and coagulopathic conditions are the main factors affecting regional anesthesia in patients with hepatic insufficiency. The pharmacokinetics of the majority of local anesthetics is affected by a poorly functioning liver associated with alterations in circulation and body fluids. All amide local anesthetics have hepatic metabolism, and less than 10% are excreted unchanged in the urine. When repeated doses or continuous infusions are used (epidural infusion or perineural catheter infusion), the accumulation of local anesthetics and their metabolites needs to be considered, and doses should be reduced accordingly. It should be kept in mind that patients with severe liver dysfunction may also have other diseases (such as nephropathy and cardiac disease), which may be even more important indications to reduce the dose of a drug. Conversely, in mild hepatic dysfunction related to alcoholism, there seems to be almost no alteration in the clearance of lidocaine.

In patients with hepatic dysfunction, single-dose blocks usually can be performed safely with normal doses of the local anesthetics. However, patients with severe liver disease often have renal dysfunction, which also requires dose reduction. The doses for repeat blocks within a short time period (<5 half-lives) and the doses for continuous infusion blocks need to be reduced markedly (10–50%) in patients with liver dysfunction mainly because of a significantly reduced clearance and accumulation of the local anesthetic and its metabolites in the blood [14].

Coagulopathy in Hepatic Insufficiency

Acute and chronic liver diseases are associated with coagulation disorders due to multiple factors (Table 21.4). This can cause spinal hematoma (in neuraxial block) or bleeding in deep tissue (in plexus block).

Table 21.4 Coagulopathy associated with liver diseases

• Decreased synthesis of clotting and inhibitor factors
• Decreased clearance of activated factors
• Quantitative and qualitative platelet defect and vitamin K deficiency
• Thrombocytopenia

Liver is the site of synthesis of all of the coagulation factors except vWF. Prolongation of prothrombin time (PT) and international normalized ratio (INR) are indicators of severity of liver damage. Thrombocytopenia is seen in acute hepatitis with and without liver failure and is a common feature in chronic advanced liver disease. Splenomegaly due to portal hypertension is considered the main cause of low platelet count in cirrhosis [15].

Renal Disease

Pharmacokinetics of local anesthetics in renal insufficiency is affected by decreased clearance of lidocaine, bupivacaine, and ropivacaine and decreased clearance of ropivacaine metabolites: 2,6-pipecoloxylidide (PPX) and 3-OH-ropivacaine (Table 21.5).

1. No change in elimination half-life of local anesthetics
2. Rapid rise in plasma concentration of local anesthetics following brachial plexus block (due to the hyperdynamic circulation)
3. Increase in acute phase protein, AAG (offers protection against local anesthetic toxicity)

It is recommended to reduce (by 10–20%) the dose of local anesthetics due to enhanced absorption following single-dose injection. In case of administration of repeat doses within the time span of less than 5 half-lives of performing continuous techniques, repeat doses should also be reduced (by 10–20%) due to the risk of local anesthetic toxicity or its metabolites [14].

Increased bleeding tendency in chronic renal insufficiency should be considered before regional anesthetic. Factors associated with hemostasis abnormality in uremic patients include the following:

1. Thrombocytopenia is a common finding in uremic patients. Platelet count is rarely below 80,000/mm^3. Suggested causes for deceased numbers of circulating platelets: platelet consumption, inadequate production, compliment activation during hemodialysis, heparin-induced thrombocytopenia (when heparin in used as an anticoagulation regimen in hemodialysis).
2. Platelet dysfunction – in terminal renal insufficiency, cyclic adenosine monophosphate (cAMP) is elevated, and hemodialysis partially corrects this abnormality.
3. Platelet–vessel wall interaction due to substances in uremic blood that interacts with $\alpha 2b\beta 3$ receptor.

Table 21.5 Factors affecting regional anesthesia in patients with renal dysfunction

• Clearance of local anesthetics
• Alteration in their hemostasis system
• Presence of uremic neuropathy
• Co-existing metabolic acidosis (may decrease the seizure threshold for local anesthetics)

Dialysis improves platelet abnormalities and reduces the risk of hemorrhage although hemodialysis can contribute to the bleeding through the platelet activation induced by the interaction between blood and the artificial surfaces, and also, the anticoagulation used during hemodialysis might transiently enhance bleeding diathesis [16].

Coagulopathy and Thrombocytopenia

Patients with alteration in their coagulation status are at increased risk of bleeding-related complications following regional anesthesia. The coagulopathy could be related to systemic illness (e.g., hepatic failure) or due to use of medications that alters the coagulation system (e.g., warfarin).

Neuraxial block and peripheral nerve blocks are not recommended in patients with coagulation disorder. American Society of Regional Anesthesia (ASRA) has published Practice Advisory for "Regional Anesthesia in the Patient Receiving Antithrombotic or Thrombolytic Therapy". The summary of the recommendations is shown in Table 21.6 [17].

The minimum platelet count below which it is safe to place a regional anesthetic is unknown. Bromage has recommended not placing an epidural anesthetic in any patient whose platelet count is 100,000/mm^3. Other authors do not define a minimum platelet count but are similarly cautious. Based on the results of a survey by Beilin et al., most anesthesiologists (66% of those in academic practice and 55% of those in private practice) will place an epidural anesthetic when the platelet count is between 80,000 and 100,000/mm^3. In a study by Beilin on 15,919 women presenting for labor and delivery, 30 received an epidural while platelet count was 69,000–98,000/mm^3, and in 22 women, epidural catheter was placed when the count was >100,000/mm^3 but subsequently decreased to 58,000–99,000/mm^3. There was no documentation of any neurologic complications in the medical records [18].

Van Veen recommends that platelet count of 80,000/mm^3 and higher is safe for placing/removing an epidural or spinal anesthetic, provided that platelet count is

Table 21.6 Summery of third ASRA recommendation for regional anesthesia in patients receiving antithrombotic or thrombolytic therapy

Thrombolytic therapy	No data available
Heparin IV	Removal of catheter 2–4 h after last dose
LMWH (thromboprophylaxis dose)	Placement/removal 10–12 h after last dose
LMWH (therapeutic dose)	Placement/removal 24 h after last dose
Warfarin	Placement; normal INR/removal; INR <1.5
Antiplatelet medications:	
Ticlopidine	Discontinuation for 14 days
Clopidogrel	Discontinuation for 7 days
Abciximab (G2b/3a inhibitors)	Discontinuation for 24–48 h
Eptifibatide (G2b/3a inhibitors)	Discontinuation for 4–8 h
Thrombin inhibitors	No data available
Fondaparinux	Neuraxial block not recommended

stable, the PT and PTT are not prolonged, and the patient is not on an antiplatelet drug or anticoagulant. It is possible that lower platelet counts also may be safe, but there is insufficient published evidence to make recommendations for lower levels at this stage. For patients with platelet counts of 50,000–80,000/mm^3 requiring epidural or spinal anesthesia, an individual decision based on risks and benefits should be made [19].

Although the most significant hemorrhagic complication of regional anesthesia is spinal hematoma, the associated risk after deep plexus block (retroperitoneal hematoma following lumbar plexus block in anticoagulated patients) and peripheral nerve block in patients with coagulopathy is undefined. It is recommended that deep peripheral blocks be managed similar to neuraxial blocks [17].

Regional Anesthesia in the Immunocompromised Patient

Patients who have altered immune status because of diabetes, neoplasm, immunosuppression after solid organ transplantation, and chronic infection with human immunodeficiency virus (HIV) or herpes simplex virus (HSV) are at greater risk of developing certain complications after regional anesthesia [20].

The frequency of serious CNS infections such as arachnoiditis, meningitis, and abscess after spinal or epidural anesthesia is considered to be extremely low in patients with normal immune function. Peripheral nerve block and continuous catheter techniques mostly cause bacterial localization and local inflammation. However, incidence of more serious side effects like abscess formation and necrotizing fasciitis is unknown. The attenuated inflammatory response of an immunocompromised patient may diminish the clinical sign and symptoms often associated with infection and results in a delay in diagnosis and treatment. Although regional anesthesia is not contraindicated in these patients, certain consideration is necessary (Table 21.7).

Acute Compartment Syndrome

Thick layers of fascia separate different groups of muscles in upper and lower extremities. These confined and nonexpendable spaces or compartments contain muscles, nerves, and blood vessels. Compartment syndrome may happen after a

Table 21.7 Some highlights of ASRA practice advisory for the prevention, diagnosis, and management of infectious complications associated with neuraxial techniques [21]

- Consider alternatives to neuraxial techniques for patients at high risk.
- Consider administering preprocedure antibiotic therapy.
- Consider removing unwitnessed accidentally disconnected catheters.
- Catheters should not remain in situ longer than clinically necessary.

simple trauma but mostly happens after a crush injury of the limbs. Surgery or a tight cast is possible but less common reasons for compartment syndrome. Permanent injury to the muscle and nerves could happen if the diagnosis is delayed. This is more common when injured person is unconscious or heavily sedated and cannot complain of the pain. Although the importance of pain in the diagnosis of compartment syndrome is controversial, virtually all analgesic modalities have been linked to a delayed diagnosis of compartment syndrome. Dense local anesthetic blocks can influence the assessment of pain and movement, making the diagnosis of compartment syndrome difficult without invasive pressure monitoring. Use of epidural anesthesia with dilute concentrations of local anesthetics to avoid motor and dense sensory blocks seems warranted. Whatever the mode of analgesia used, a high index of clinical suspicion, ongoing assessment of patients, and compartment pressure measurement are essential for early diagnosis [22].

Elderly Patient Considerations

Neuraxial block in the elderly might be anatomically more challenging due to loss of disk spaces, osteophytes, and change in consistency of ligaments. Age is also a major determinant of duration of complete motor and sensory blockade with peripheral nerve block, perhaps reflecting increased sensitivity to conduction failure from local anesthetic agents in peripheral nerves in the elderly population [23].

Aortic sclerosis is common in the elderly, and it could be associated with hemodynamically significant obstruction of left ventricular outflow. Severe aortic stenosis is considered as an independent, important risk factor for patients undergoing general anesthesia for noncardiac surgery. Although these patients will benefit from regional anesthesia, single-shot spinal anesthesia is generally considered unsafe in patients with severe aortic stenosis. Another comorbidity that is more common in elderly patients who require shoulder or upper extremity blocks and deserves attention is pulmonary compromise. Spread of local anesthetic to phrenic nerve causes ipsilateral hemidiaphragmatic paresis and could possibly lead to respiratory failure. Phrenic nerve block is very common with interscalene block and happens 40–60% of the time in supraclavicular block. In a patient with severe pulmonary compromise infraclavicular, axillary or paravertebral posterior approach to brachial plexus should be used. Ultrasound-guided low-volume interscalene block may decrease the incidence of phrenic nerve block.

Clinical Pearls

A good preoperative evaluation is critical, and patients should also be informed about perioperative implications of anesthesia, surgery and stress, and risk versus benefit of regional anesthesia. In difficult and complex cases with multiple comorbidities, decision should be made on an individual basis. A thorough history and

physical with special attention to neurologic exam is very helpful in differential diagnosis of postprocedure neurological deficit. In case of presence of a preexisting severe neurological deficit, proper documentation by a third party is necessary. In certain preexisting conditions, paresthesias, epinephrine, and high concentrations of local anesthetics should be avoided if a regional anesthetic is administered. Anesthesiologists should not automatically take all responsibility in cases of progressive or new deficits after the procedure. Finally, it is important to be aware of standard recommendations and guidelines related to regional anesthesia and patient comorbidities.

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22

Intravenous Regional Anesthesia

Lindsey Vokach-Brodsky

Contents

Introduction

Intravenous regional anesthesia (IVRA) was first described in 1908 by Bier, who used procaine injected intravenously between two forearm tourniquets [1]. Holmes is credited with reintroducing the technique in 1963 [2]. More than 100 years after the original description, with some modifications, Bier's technique is still used in modern anesthesia practice.

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The technique is reliable and simple to perform. It requires no special expertise. Historically, the success rate of IVRA, around 95%, compared favorably with brachial plexus block and also avoided the associated risks of pneumothorax, neuraxial spread of local anesthetic, and arterial puncture [3, 4]. Large studies have demonstrated an impressive safety record [5, 6].

Mechanism of Action

Bier postulated two mechanisms of action. Direct action of local anesthetic on nerve endings is followed by a slower onset of nerve block caused by spread of local anesthetic to the nerves via vasa nervorum. This explanation is still accepted today.

Technique

- Select an appropriately sized double tourniquet. Check tourniquet function.
- Establish IV access in the nonoperative limb.
- Place a small-bore IV cannula in a distal vein in the operative hand.
- Attach standard monitors and give supplemental oxygen.
- Place tourniquet on the arm over protective padding (e.g., Webril).
- Elevate the limb and apply the Esmarch bandage.
- Inflate first the distal tourniquet, then inflate the proximal tourniquet. Deflate the distal tourniquet. Tourniquet pressure should be set to 100 mmHg over systolic pressure.
- Remove the Esmarch bandage, then check for absence of the radial pulse and that the limb remains pale.
- Inject the local anesthetic over 30 s. The patient should be warned of a possible burning or cold sensation, which is short-lived. The cannula is then removed, and pressure is applied to the insertion site with a sterile pad while the arm is prepped for surgery.
- It is normal for the arm to appear mottled. Surgical anesthesia is achieved after about 10 min.
- After 30 min, the patient may complain of tourniquet pain. At this time, the distal tourniquet cuff is inflated followed by deflation of the proximal cuff, which will relieve tourniquet pain for up to another 20 min.
- The tourniquet must remain inflated for a minimum of 30 min after injection. After the tourniquet is released, the patient must be closely observed for signs or symptoms of local anesthetic toxicity.
- Reinflating the tourniquet several times for 1 min after a 10-s interval of deflation has been shown to reduce the rate of rise of systemic concentration of the local anesthetic but not to reduce its magnitude [7].

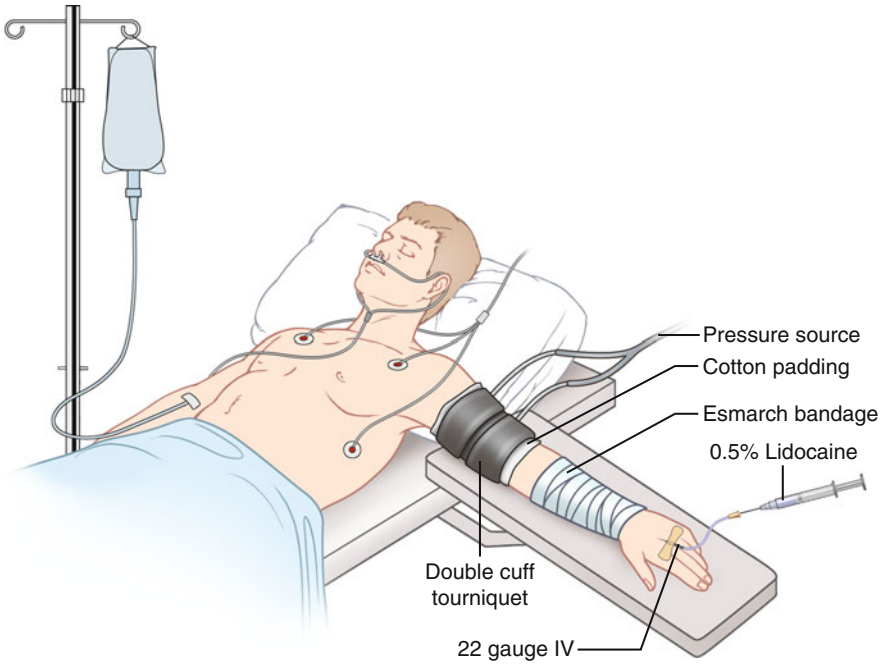

Fig. 22.1 Intravenous regional block (Bier block) of the upper extremity

- The numbness will wear off within 10 min of tourniquet release. Local infiltration of the surgical site at the close of surgery increases patient comfort (Fig. 22.1).

Forearm Tourniquet

Use of a tourniquet on the forearm reduces the volume of anesthetic required and enhances the safety of the procedure. Twenty-five milliliters is commonly used. Concerns about the effectiveness of forearm tourniquets have not been substantiated [8]. The bulky nature of the tourniquet may impede surgical access.

IVRA for the Lower Limb

While IVRA can be used successfully for anesthesia of the lower limb [9], higher volumes of local anesthetic are required, and the thigh tourniquet can cause the patient considerable discomfort. Placement of the tourniquet below the knee is associated with increased leakage under the tourniquet [9], and the common peroneal nerve is susceptible to injury at this site. IVRA using an ankle tourniquet has also been described for surgery on the foot; however, incomplete anesthesia may occur [10].

Choice of Local Anesthetic

Many different local anesthetics have been used for IVRA (see Table 22.1). The most commonly used are lidocaine and prilocaine. While other agents have been used successfully, the potential for the sudden release of a large volume of local anesthetic into the systemic circulation makes the use of the more potent agents inadvisable. A number of deaths were reported in the 1980s associated with IVRA using bupivacaine [18]. The FDA subsequently withdrew approval for this use of bupivacaine.

Preservative-free local anesthetic is preferable. Preservatives have been associated with venous irritation and allergic reactions [21].

Additives

Many additives to local anesthetics have been tried with the aim of improving anesthesia, ameliorating tourniquet pain, and providing postoperative pain relief. Clinically, significant improvements have been achieved with NSAIDs, in particular ketorolac, and with α-agonists such as clonidine and dexmeditomidine [22, 23].

Table 22.1 Local anesthetics used in IVRA

Drug	Dose	Comment	References
Lidocaine	3 mg/kg 40 ml 0.5%	Most commonly used in USA. Cardiac arrest and death has been reported following IVRA	[12, 13, 26]
Prilocaine	3 mg/kg 40 ml 0.5%	Less systemic toxicity than lidocaine. No reports of cardiac arrest Methemoglobinemia may occur with doses over 600 mg	[11–13]
Mepivacaine	5 mg/kg	Reported better intraoperative conditions than lidocaine	[14]
Ropivacaine	40 ml 0.2–0.25% (25 ml 0.375% with forearm tourniquet)	Longer postoperative analgesia than lidocaine.	[15–17]
Bupivacaine	Not recommended	5 deaths reported following IVRA	[18]
Chloroprocaine	40 ml 0.5%	Urticaria/venous irritation Solutions containing preservative have been associated with thrombophlebitis	[19, 20]
Articaine	40 ml 0.5%	Rapid onset, rapid metabolism Skin rashes/urticaria common Not available preservative free in USA	[12]

Muscle relaxants have been used successfully to improve motor block [24]. Additives can be associated with increased side effects, such as postoperative nausea and vomiting, hypotension, and sedation [22].

IRVA and CRPS

IVRA with guanethidine or bretylium has been used in the treatment of complex regional pain syndromes. However, a recent review of randomized controlled trials failed to show any lasting benefit for this treatment modality [25].

Contraindications

Many of the contraindications to IVRA relate to the use of a tourniquet (Table 22.2).

Complications

Complications of IVRA have recently been reviewed [26]. Systemic local anesthetic toxicity may lead to seizures, cardiac arrest, and death. While toxicity may be due to accidental or inappropriately early release of the tourniquet, seizures occurring after tourniquet time of 1 h and cardiac arrest after tourniquet time of 30 min have also been reported. No cases of cardiac arrest have been reported following prilocaine IVRA (Table 22.3).

Table 22.2 Contraindications to IVRA

Absolute	Relative
Patient refusal	Cellulitis
True allergy to local anesthetics	Sickle cell disease
	Paget's disease of bone
	Arteriovenous fistula
	Compound fracture/vascular injury

Table 22.3 Complications of IVRA

Major	Minor
Local anesthetic toxicity, including seizures, cardiac arrest and death	Skin discoloration
Nerve injury	Petechiae, urticaria
Compartment syndrome, amputation	Pain on injection
	Thrombophlebitis

Clinical Pearls

- IVRA is unsuitable for surgery of more than 1 h duration, due to tourniquet pain.
- It is best suited for superficial surgery of the forearm and hand which is not associated with severe postoperative pain.
- Use of a forearm tourniquet reduces the total dose of local anesthetic.
- Careful exsanguination is required to give a "dry" surgical field.
- Addition of ketorolac improves the quality of anesthesia.

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Regional Anesthesia and Trauma

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Contents

Trauma is a major cause of mortality and morbidity worldwide, and pain is the most common symptom reported by patients entering the Emergency Department [1]. Each year, more than 100,000 deaths in the USA and about 8% of all deaths worldwide are caused by traumatic injuries [2]. Trauma is also a leading cause of death in persons younger than 30 years [3]. An estimated 5.3 million people in the United States have long-term disabilities resulting from traumatic brain injuries and another 200,000 from spinal cord injuries [3].

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Among all the treatment modalities for trauma patients, pain management has become the core intervention because improved pain management has not only led to increased comfort in trauma patients, but has also been shown to reduce morbidity and improve long-term outcomes [4, 5]. Conversely, inadequate pain control leads to drastic clinical consequences, such as thromboembolic and pulmonary complications, lengthy hospital stay, and development of posttraumatic stress disorder [6–8]. Since trauma patients usually experience significantly more stress than patients with elective surgery, trauma patients tend to have increased morbidity as a result of stress-induced higher myocardial oxygen consumption if pain is not adequately controlled [9]. It has also been shown that the persistence of severe, uncontrolled pain can lead to series of anatomic and physiologic changes in the nervous system [10]. These neuroplastic changes underlie the development of chronic, disabling neuropathic pain. For example, one study [11] reported that inadequate pain control resulted in chronic pain syndromes in 69% of patients with spinal cord injuries.

Unfortunately, multiple studies have reported that trauma-related pain is still inadequately controlled [12]. A recent study by Whipple et al. [13] assessed adequacy of pain treatment in 17 patients with multiple trauma injuries. While 95% of staff and 81% of nurses reported adequate analgesia, 74% of patients rated their pain as moderate to severe. Lack of recognition of pain and its related symptoms, limited acknowledgment of various pain management approaches, excessive concern about narcotics-induced hemodynamic instability, respiratory depression, and addiction all contribute to the inadequacy of pain management in trauma patients.

Therefore, pain management in trauma patients still remains a challenge to clinical practitioners. Plus, the need to preserve the hemodynamic stability, the respiratory function, and patients' level of consciousness in this patient population further complicates the challenge.

Multimodal analgesia has been increasingly used to manage pain in trauma patients [14]. This wide range of measures includes regional anesthesia procedures, opioids, NSAIDs, NMDA receptor blockers, anticonvulsants, antidepressants, and α2-agonists. Although each modality has its own strength and weakness, regional anesthesia, e.g., peripheral nerve blocks, stands out as an important technique especially in the perioperative setting because many traumatic injuries eventually require surgical interventions [3]. Regional anesthesia can become the first choice of analgesia in patients with isolated orthopedic injuries and burning injuries because this technique avoids many adverse side effects associated with systemic opioids, such as nausea/vomiting, pruritus, urinary retention, hypotension, and respiratory depression.

Even though evidence that shows improvement of outcomes by regional anesthesia in trauma patients is still lacking, it is generally agreeable that adequate analgesia via regional anesthesia reduces incidence of intubation and postoperative morbidity related to traumatic injuries, resulting in positive outcomes [15].

Any regional anesthesia techniques applicable in the elective surgery patient are potentially useful in the trauma patient. Nevertheless, the challenges to manage both pain and other trauma-related complications simultaneously require clinicians to take into account all possible risks and benefits of this technique in order for an optimum patient care to be achieved.

Peripheral Nerve Blocks for Pain Management in Trauma to the Extremities

Peripheral nerve blocks (PNBs) provide rapid and effective analgesia with less opioid-related side effects, such as nausea/vomiting, pruritis, urinary retention, constipation, sedation, and respiratory depression [16].

Depending on the site of injury and the planned operative procedure, peripheral nerve blocks should only be in a designated clinical context.

Before placing the block, it is very important to perform a neurological exam of the patient, documenting sensory and/or motor impairments [3]. A preexisting neurological injury does not represent per se an absolute contraindication to peripheral nerve blocks, but it is important to document it both for medicolegal reasons and for considerations in developing a clinical plan of treatment. Continuous peripheral nerve blocks have been shown to decrease pain scores, increase joint range of motion, and decrease hospital stay and rehabilitation times compared to intravenous patient-controlled analgesia [17]. They also produce fewer side effects compared with epidural analgesia [18].

Continuous infusion of local anesthetics through implanted catheters has often been found necessary in order to manage trauma-induced pain because single injections do not usually provide long enough pain coverage [3]. Ideally, regional techniques should initially be used to diminish the inflammatory response caused by tissue injury and then continue as long as the painful insults persist [19]. This "preventive" strategy, as opposed to preemptive analgesia which, by definition, only covers the earliest phase of the inflammatory insult, has been postulated to be more beneficial in terms of preventing chronic pain syndromes, although clear evidence is still lacking. In this perspective, multiple sequential catheters are sometimes indicated in order to provide optimal long-lasting analgesia [20]. This strategy has been successfully applied in military medical care, especially for soldiers wounded in combat [14].

Since trauma can occur at multiple sites, nerve blocks at multiple sites are often necessary in order to effectively reduce the amount of IV opioids required. It has been demonstrated that trauma patients may safely benefit from multiple simultaneous continuous peripheral nerve catheter infusions to treat multiple injuries [16].

Both peripheral nerve stimulation and ultrasound may be used to guide needle placement for peripheral nerve localization: neither technique has been proven to be superior to the other in terms of block success, although ultrasound may potentially decrease the time and number of attempts to complete a block [21]. Moreover, eliciting an evoked motor response across a fractured site may cause increased pain, and ultrasound brings a certain advantage in this setting [3]. In cases of traumatic nerve injuries, ultrasound has obvious indications and benefits [22].

Upper Extremity Trauma

Patients frequently presenting to the Emergency Department (ED) with upper extremity injuries such as fractures, dislocations, lacerations, and burns often require immediate pain relief provided by peripheral nerve block. Brachial plexus block

usually provides adequate analgesia for upper extremity injuries. Depending on the injury sites, different approaches can be used, for example, interscalene, supraclavicular, or axillary blocks should provide effective pain relief for injuries at mid-distal arm, elbow, forearm, and/or hand [23]. Alternatively, various blocks at forearm should deliver adequate analgesia for hand or wrist injuries.

These nerve block approaches, however, are sometimes associated with risks of having various complications. For example, interscalene nerve block, which is often indicated for anesthesia and/or analgesia in patients with shoulder injuries, can cause Horner's syndrome that obscures the neurological assessment of the patients' consciousness level [3]. Accidental phrenic nerve block can result in an impairment of the ipsilateral diaphragmatic function [24]. The interscalene approach can also present an increased risk of infection should tracheostomy be performed or internal jugular vein catheter be implanted [3]. It is also known that both supraclavicular and infraclavicular nerve blocks are associated with pneumothorax [25]. Among these different approaches, axillary nerve block is probably the least desirable because it requires the largest scale of movement of the injured upper extremity and catheter positioning, and maintenance becomes difficult under the arm [3].

Use of ultrasound in upper extremity nerve blocks has improved the accuracy of needle insertion and catheter placement. The advantage of ultrasound-guided nerve block becomes obvious when it is hard to locate skin landmarks due to either excessive adipose tissues or anatomic distortions caused by neck injuries. It is noteworthy that the presence of C-collar is not a contraindication to performing upper extremity nerve blocks. Once cervical traumatic injuries are ruled out by proper imaging tests, C-collar can be removed and an ultrasound-guided nerve block can be performed.

Sympathetectomy that follows regional anesthesia of the upper extremity is often beneficial for revascularization, reimplantation, or in any other cases where blood flow is compromised [26]. Before performing the block, the risks and benefits should be discussed with the surgeon. Every effort should be made to avoid radial compartment syndrome. If necessary, a short-acting local anesthetic may be preferred and can be used reliably for surgical anesthesia.

Lower Extremity Trauma

Regional anesthesia at lower extremity usually includes lumbar plexus block and sacral plexus block at different sites. These nerve block procedures have been proved superior over morphine PCA in providing analgesia in patients with lower extremity trauma [27, 28]. They are also considered safer with less complications comparing with epidural block [3].

Lumbar plexus can be blocked using either anterior or posterior approaches. Although anterior approaches at the level of inguinal ligament (3-in-1 or fascia iliaca blocks) have obvious advantages with not having to put the patients at the lateral position, blocks at this level do not effectively cover all the branches of lumbar plexus. As a result, larger volumes of local anesthetics are often needed to provide

adequate analgesia [29]. Instead, posterior approaches (psoas compartment block) usually provide excellent analgesia [30] with relatively small dose of local anesthetics because adequate coverage of all lumbar plexus branches can be achieved at the level of psoas muscle compartment. Various approaches to sacral plexus block have also been used to provide analgesia to where sciatic nerve is distributed.

Depending on the injury sites, either lumbar plexus or sacral plexus or both have to be used to warrant adequate analgesia. For example, acetabular or femoral neck injury may only require a lumbar plexus block, whereas knee/patellar injuries and ankle injuries require both femoral and sciatic nerves to be blocked [3].

Complications from continuous lower extremity nerve blocks are rare, although minor events like local inflammation and vascular puncture may be common [31]. The incidence of infection associated to nerve blocks is poorly defined in literature, and even if the rate of catheter tip contamination results between 23 and 57%, the incidence of clinical local infections is only 0–3% [3].

Among these complications, it is worth noting that psoas compartment block could lead to epidural and/or intrathecal injection of local anesthetics either due to catheter displacement or local anesthetic spread resulting in bilateral block and hypotension [30]. The risk of neuraxial block may be lowered by avoiding medial direction of the needle when psoas compartment block is performed.

Acute Compartment Syndrome

In lower extremity musculoskeletal trauma, acute compartment syndrome is a potentially devastating complication, whose incidence has been previously described as 7.3 per 100,000 in men and 0.7 per 100,000 in women [32]. The most common cause of acute compartment syndrome is usually fracture (69%) with tibial fracture being the most common injury; soft tissue injury without fractures is the most common cause (23%) with 10% of these occurring in patients taking anticoagulants or with a bleeding disorder [33]. Pain out of proportion to the injury, aggravated by passive stretching of muscle groups in the corresponding compartment, is one of the earliest and most sensitive clinical signs of compartment syndrome, even though it can be diminished or absent in an established compartment syndrome [34]. Anesthetic techniques have been reported to contribute to delay of the diagnosis [35]. Patients receiving epidural analgesia with local anesthetics and opioids have been reported to have a fourfold increased risk of neurologic complications than patients receiving systemic narcotics [36]: epidural analgesia with local anesthetics and opioids is therefore not recommended in at-risk patients. However, in the trauma patient, the absence of pain in a compartment syndrome is often caused by superimposed central or peripheral neural deficit, and pressure or firmness in the compartment remains the earliest and sometimes the only objective finding of early compartment syndrome [33]. Various methods of measuring tissue pressure have been described [37], and their application is recommended any time the clinical picture may be borderline or the patient examination can be ambiguous. In this scenario, a

peripheral nerve block is not absolutely contraindicated, and each specific case should be addressed and discussed with the trauma team.

Peripheral Nerve Blocks for the Management of Chest Trauma

Rib fractures are the most common thoracic injuries with an incidence ranging from 10% to almost 30% in patients after trauma. Mortality rate of patients with rib fractures range from 5.8% (single rib fracture) to 34.4% (multiple rib fractures) with an overall rate of 10%. Pain associated with rib fractures usually impairs pulmonary function and increases pulmonary morbidity. Therefore, appropriate pain management in a timely manner should be a core intervention in managing these patients. Various techniques have been used to manage pain in patients with rib fractures. These include systemic opioids, intercostal nerve blocks, epidural analgesia, and intrapleural and paravertebral nerve blocks. Clear superiority of one technique over the others in terms of efficacy and safety has not been demonstrated in the literature.

Intercostal Nerve Block

Both multiple single-shot injections with local anesthetics above and below the fracture site and continuous intercostal infusions have been shown to be successful in relieving pain caused by rib fractures. However, the exact mechanism underlying the intercostal analgesia is still unknown. Anesthetics are supposed to spread to the paravertebral space, epidural space, or a combination of both. An early case report showed paravertebral spread of local anesthetic after 20 ml of 0.5% bupivacaine was injected into the intercostal space. The same mechanism was confirmed by Mowbray et al., who followed the spread of intercostal injection of 20 ml of bupivacaine and methylene blue through a catheter at thoracotomy in the paravertebral space. Indeed, a recent study verified a paravertebral catheter placement through the intercostal space. Therefore, it is possible that the major component of segmental block during intercostal catheterization may be secondary to paravertebral spread.

Intrapleural Nerve Block

There are many reports of the successful use of unilateral and bilateral interpleural blockade in patients with multiple rib fractures. This technique produces multisegmental intercostal nerve blockade by gravity-dependent retrograde diffusion of the local anesthetics to reach the intercostal nerve. A few studies have compared the interpleural nerve block with epidural block, paravertebral nerve block, and conventional opioids for analgesic efficacy in chest wall trauma with contrasting results.

Some reasons for the conflicting results include catheter position, presence of hemothorax, location of fractured ribs, characteristics of local anesthetics, loss of local anesthetic through chest tubes, and dilution in pleural effusion. Among these reasons, it is interesting to notice that the location of rib fractures may affect the analgesic efficacy by interpleural nerve block. It appears that interpleural nerve block is most useful in clinical settings such as lateral or posterior rib fractures in the healthy chest cavity.

Epidural Nerve Block

Many studies have shown that thoracic epidural nerve block with local anesthetics, opioids, or a combination of both produces dramatic analgesia in patients with multiple rib fractures. Pulmonary function such as functional residual capacity, forced vital capacity, airway resistance, maximal inspiratory force, and maximal tidal volume is also reported improved by epidural analgesia. Although evidence that epidural nerve block improves subjective pain score and a variety of pulmonary functions in rib fracture patients is abundant and compelling, there is limited evidence that epidural nerve block improves outcomes. In a meta-analysis by Carrier et al., evaluating seven randomized controlled studies (232 patients), epidural analgesia did not demonstrate significant benefits related to mortality, ICU length of stay, hospital length of stay, or duration of mechanical ventilation compared to other analgesia modalities, including opioid PCA or IV/IM opioid boluses and interpleural nerve blocks. Moreover, hypotension proved to be more frequent in patients receiving epidural analgesia. Thus, the evidence does not support the strength of the recent clinical practice guidelines on pain management in blunt thoracic trauma laid out by the East Association for the Surgery of Trauma (EAST), which stated that epidural analgesia may improve clinically significant outcomes in this population (Grade B recommendation) and that it should be considered the preferred analgesic modality (Grade A recommendation). In addition, in patients with mechanical ventilation and sedation, epidural analgesia is usually relatively contraindicated because of the patients' altered level of consciousness. Therefore, considering the potential for rare but major adverse events of epidural nerve block, clinically significant benefits other than better pain control need to be demonstrated to endorse the use of epidural nerve block as a standard of care in adult patients with traumatic rib fractures.

Paravertebral Nerve Block

Paravertebral nerve block is a regional anesthetic technique in which a single injection of anesthetic or a continuous infusion is delivered to the thoracic paravertebral space, producing a unilateral, multilevel, somatic, and sympathetic block. Since it is simple to perform, is associated with a low incidence of complications, requires no

additional nursing surveillance, and has few absolute contraindications, paravertebral nerve block has recently been used to control pain in a variety of conditions involving the chest and abdomen. Evidence is also accumulating in support of this modality in patients with trauma, such as rib fractures: single injection of 0.5% bupivacaine into the thoracic paravertebral space led to significant improvement of pain scores and vital capacity in patients suffering from blunt or penetrating thoracic trauma; continuous paravertebral anesthetic (0.5% bupivacaine at 0.1–0.2 ml/kg/h for 4 days) in 15 patients with isolated unilateral rib fractures also provided significant improvements in analogue pain scores, vital capacity, peak expiratory flow rate, oxygen saturation (SaO_2), and O_2 index (PaO_2/FiO_2 ratio). Compared with epidural nerve block, paravertebral nerve blocks have been shown to produce comparable pain relief and similar improvements in respiratory function in patients with unilateral fractured ribs, although epidural was complicated by a higher incidence of hypotension. A downside of paravertebral nerve blocks is that fewer practitioners are familiar with the technique, and large clinical trials are still lacking in the trauma population. Nevertheless, an increasing availability of data in the literature supports their efficacy in other clinical scenarios such as thoracotomies, which share a common pain mechanism with rib fractures, that is, intercostal nerve damage. Preoperative paravertebral nerve blocks have been demonstrated to significantly lower postoperative pain scores and better preserve postoperative lung function, measured by forced vital capacity, when compared to epidural analgesia. Moreover, a few review papers reported that paravertebral nerve block provided at least equally effective analgesia to epidural with less side effects, such as urinary retention, nausea/vomiting, and hypotension. Moreover, while epidural technique is contraindicated in the setting of coagulopathy due to the risk of hematoma and subsequent cord compression, the margin of safety is much higher with a paravertebral block and the more distensible paravertebral space.

Clinical Pearls

- Improved pain management has been shown to reduce morbidity and improve long-term outcomes.
- Multimodal analgesia has been increasingly used to manage pain in trauma patients; this wide range of measures includes regional anesthesia procedures.
- Peripheral nerve blocks (PNBs) provide rapid and effective analgesia with less opioid-related side effects, such as nausea/vomiting, pruritis, urinary retention, constipation, sedation, and respiratory depression.
- Brachial plexus block usually provides adequate analgesia for upper extremity injuries.
- Use of ultrasound in upper extremity nerve blocks has improved the accuracy of needle insertion and catheter placement. Its advantages become especially obvious when it is hard to locate skin landmarks.

- Regional anesthesia at lower extremity usually includes lumbar plexus block and sacral plexus block at different sites.
- The posterior approaches to the lumbar plexus usually provide excellent analgesia with relatively small dose of local anesthetics; various approaches to sacral plexus block have also been used to provide analgesia to where sciatic nerve is distributed.
- Pain associated with rib fractures usually impairs pulmonary function and increases pulmonary morbidity: appropriate pain management in a timely manner should be a core intervention in managing these patients.
- Pulmonary function such as functional residual capacity, forced vital capacity, airway resistance, maximal inspiratory force, and maximal tidal volume is reported improved by epidural analgesia in patients with chest trauma.
- Paravertebral nerve blocks have been shown to produce comparable pain relief and similar improvements in respiratory function than epidural analgesia in patients with unilateral fractured ribs, although epidural was complicated by a higher incidence of hypotension.
- Paravertebral nerve blocks provide at least equally effective analgesia to epidural with less side effects, such as urinary retention, nausea/vomiting, and hypotension in patients with chest trauma.

Multiple-Choice Questions

1. How many deaths in the USA are caused by traumatic injuries every year?
   (a) 100,000
   (b) 500,000
   (c) 1,000,000
   (d) 250,000
   (e) 25,000

2. Which percentage of deaths is caused by traumatic injuries worldwide?
   (a) 0.1%
   (b) 8%
   (c) 25%
   (d) 0.25%
   (e) 12%

3. In which percentage of patient's inadequate pain control resulted in chronic pain syndromes after spinal cord injuries?
   (a) 20%
   (b) 30%
   (c) 40%
   (d) 50%
   (e) 70%

4. In the recent study by Whipple et al. [13] about adequacy of pain treatment in patients with multiple trauma injuries, which percentage of patients rated pain as moderate to severe?
   (a) 24%
   (b) 10%
   (c) 0.2%
   (d) 74%
   (e) 99%

5. Which upper extremity block can have Horner's syndrome as a complication?
   (a) Axillary block
   (b) Infraclavicular block
   (c) Supraclavicular block
   (d) Interscalene block
   (e) Ulnar block at the elbow

6. In lower extremity musculoskeletal trauma, acute compartment syndrome is a potentially devastating complication, whose incidence has been previously described as:
   (a) 7.3 per 100,000 in men and 0.7 per 100,000 in women
   (b) 0.7 per 100,000 in women and 7.3 per 100,000 in men
   (c) 30 per 100,000 in men and women
   (d) 0.5 per 100,000 in men and 0.01 per 100,000 in women
   (e) 70 per 100,000 in men and women

7. The most common cause of acute compartment syndrome is usually:
   (a) Burn injury
   (b) Soft tissue injury
   (c) Fracture
   (d) Crush injury
   (e) Tissue edema

8. The most common fracture that can be complicated by compartment syndrome is:
   (a) Humerus fracture
   (b) Scaphoid fracture
   (c) Tibial fracture
   (d) Femur fracture
   (e) Scapular fracture

9. A predisposing factor for compartment syndrome in soft tissue injuries is:
   (a) Regional anesthesia
   (b) Hypertension
   (c) Anticoagulants or bleeding disorders
   (d) Hypotension
   (e) Vascular diseases

10. One of the earliest and most sensitive clinical signs of compartment syndrome is:
    (a) Pain out of proportion
    (b) Motor and sensory block
    (c) Paresthesia
    (d) Absence of pain
    (e) Pallor

11. Mortality rate of patients with single rib fractures is around:
    (a) 1%
    (b) 10%
    (c) 6%
    (d) 50%
    (e) 0.1%

12. Mortality rate of patients with multiple rib fractures is around:
    (a) 90%
    (b) 80%
    (c) 70%
    (d) 35%
    (e) 25%

13. The East Association for the Surgery of Trauma (EAST) stated that one of the following may improve clinically significant outcomes in this population (Grade B recommendation) and that it should be considered the preferred analgesic modality (Grade A recommendation):
    (a) Intrapleural block
    (b) Epidural block
    (c) Intercostal block
    (d) Paravertebral block
    (e) Morphine PCA

14. When compared to epidural, paravertebral nerve blocks have been demonstrated to cause less:
    (a) Hypotension and urinary retention
    (b) Failed block
    (c) Compartment syndrome
    (d) Foot drop
    (e) Infections

15. Which of the following have been demonstrated to provide comparable analgesia?
    (a) Intercostal and intrapleural blocks
    (b) Intercostal and paravertebral blocks
    (c) Epidural and intrapleural blocks
    (d) Paravertebral and epidural blocks
    (e) Paravertebral and intrapleural blocks

Answers:

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

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24

Postoperative Pain Management

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Contents

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Introduction

Postoperative pain remains a significant problem for patients undergoing ambulatory and in-house surgery and is frequently undermanaged. Ineffective pain control can result in negative outcomes such as prolonged rehabilitation, delayed wound healing, cardiovascular complications, and development of chronic pain. Consequently, physicians involved in postoperative pain management should develop individualized strategies based on patient characteristics and surgical procedures. Ideally, a specific plan for postoperative pain management is developed prior to surgery and includes the patient and physician and caregivers from several medical disciplines. A multimodal approach utilizes agents from different drug classes and various techniques and allows targeting different receptors and pain pathways. In addition, such an approach avoids side effects frequently associated with monotherapy. Over the last decade, regional anesthesia techniques and especially peripheral nerve blocks have emerged as an important component of such a multimodal approach. The ability of peripheral nerve blocks to provide effective and tailored pain control in combination with a favorable side-effect profile has resulted in outcome improvements, especially after major orthopedic surgery. This chapter will review the different tools available to the pain management physician and his team and illustrate their individual strengths and indications.

Single and Continuous Nerve Blocks

Indications

Peripheral nerve blocks (PNBs) offer various clinical postoperative advantages over opioid monotherapy, such as improved pain control, reduced side effect, improved patient satisfaction, shortened PACU stay or PACU bypass, improved physical therapy, and earlier hospital discharge. These benefits can contribute to substantial cost savings for patients, institutions, and even entire health-care systems [1, 2]. Before discussing

the particular indications of single-shot and continuous peripheral nerve blocks (CPNBs), the clinician must consider the following absolute contraindications:

1. Patient refusal
2. Infection at the injection/catheter placement site
3. Documented allergy to local anesthetic

Patients who present a clear indication for neural blockade include [3]:

1. Orthopedic surgeries:
   (a) Single-shot: All ORIFs, debridements, and upper or lower extremity soft tissue procedures
   (b) Continuous: All joint replacement surgeries, especially those requiring post-operative physical therapy; limb amputations; and any patient requiring multiple limb procedures over a short time period, such as serial debridements
2. Thoracic surgeries:
   (a) Single-shot: VATS and chest wall debridements
   (b) Continuous: Mastectomies and thoracotomies
3. Peripheral vascular surgeries:
   (a) Single-shot: A–V grafts, vein harvesting, and vascular reanastamosis or repairs
   (b) Continuous: Sympathectomy for thrombotic limb ischemia
4. Trauma patients:
   (a) Single-shot: Closed reduction of joint dislocations
   (b) Continuous: Rib fractures, traumatic amputations, or crushed limb injuries

Techniques

The appropriate technical selection must consider several key factors in addition to the patient's comorbidities and type of surgery. Many otherwise successful nerve blocks may fail to provide adequate anesthetic conditions when the duration and site of surgery, the need for tourniquet application, or complementary intraoperative sedation are neglected [2]. The following table (Table 24.1) provides a general guideline for the most common surgical procedures and appropriate nerve block selection.

Anesthetic Infusions and Adjuncts

The choice of local anesthetic for both single-shot PNBs and catheter infusions should be based on the following factors:

1. Duration of surgery
2. Patient disposition: ambulatory vs. inpatient

Table 24.1 Site/surgery-specific neural blockade

Surgical procedure	Nerve block technique	Notes
ENT surgery		
Carotid endarterectomy Radical neck dissection (postop pain) Cervical lymph node biopsy	Deep + superficial cervical plexus block or interscalene block + superficial cervical plexus	Intraoperative supplementation by the surgeon is often necessary to block neuronal innervation to the carotid artery from the glossopharyngeal nerve
Upper limb surgery		
Shoulder arthroscopy or arthroplasty, rotator cuff repair, humeral neck fracture	Interscalene	Often spares the deltopectoral groove and posterior arthroscopic port incision. Supplement with superficial cervical plexus block
Mid-arm and elbow procedures, forearm surgery	Supraclavicular, infraclavicular, axillary nerve blocks	Supraclavicular block may exhibit limited or slow ulnar nerve coverage. Intercostobrachial nerve (T2) must be supplemented to block tourniquet pain
Hand or wrist surgery	Axillary or infraclavicular nerve blocks	
Chest/abdomen surgery		
Thoracotomy, vats, mastectomy, chest wall procedures	Intercostal, paravertebral nerve blocks	Follow ASRA guidelines for anticoagulated patients
Rib fractures, flail chest	Paravertebral nerve block	
Abdominal incisions, cesarean section (postop pain)	Transversus abdominis plane nerve block, rectus sheath block	Requires bilateral blocks for midline incisions
Lower limb surgery		
Hip	Lumbar plexus	Follow ASRA guidelines in anticoagulated patients
Knee, patella, femur, thigh procedures	Lumbar plexus, femoral nerve block	Posterior knee will be spared due to sciatic innervations
Amputations	Combined femoral and sciatic nerve blocks	Blocks should be placed preoperatively to reduce incidence of phantom limb pain
tibia, fibula, ankle, foot procedures	Sciatic nerve block	Femoral (saphenous) nerve block supplementation required if the medial aspect of the lower leg is involved

3. Postoperative physical therapy
4. Time of block placement: preop vs. postop
5. Combination with general anesthesia

Table 24.2 Local anesthetic pharmacodynamics

Local anesthetic	Onset (min)	Anesthesia (h)	Analgesia (h)
3% 2-Chloroprocaine	5–10	1.5	2
1.5% Mepivacaine	5–15	2.4–4	3–6
2% Lidocaine	5–15	3–6	5–8
0.5% Ropivacaine	15–25	6–8	8–16
0.25% Bupivacaine	20–30	3–4	4–10
0.5% Bupivacaine	15–25	8–10	16–18

Table 24.3 Continuous peripheral nerve block infusions

Ropivacaine 0.2% infusions	Nerve block technique	Rate (mL/h)
Brachial plexus	Interscalene	6–8
	Supraclavicular/infraclavicular/axillary	8–10
Paraneuraxial	Paravertebral	3–6
Lumbar plexus	Lumbar plexus	8–12
	Femoral/fascia iliaca	8–12
Sacral plexus	Sciatic (all techniques)	8–12

In general practice, the most common agents selected for neural blockade are mepivacaine, ropivacaine, and bupivacaine. Our recommended injectates are as follows (Table 24.2):

Short procedure, preoperative PNB – 1:1 mixture of 1.5% mepivacaine and 0.5% ropivacaine. This combination offers good intraoperative anesthetic conditions for 2–3 h while providing adequate postoperative analgesia for 8–12 h.

Long procedure, preoperative PNB – 0.5% ropivacaine. For most procedures lasting more than 3 h, general anesthesia should be combined with the regional technique. This agent offers adequate intraoperative and postoperative analgesia for 10–16 h.

Postoperative PNB – for ambulatory patients, we often select 0.2% or 0.35% ropivacaine for its selective blockade of sensory transmission and motor sparing. This agent, therefore, minimizes the patient's inability to mobilize the operative limb at home. For inpatients, a higher concentration, such as 0.5%, can be utilized due to the availability of nursing staff who can assist the patient with ambulation.

CPNB – while any local anesthetic agent can be selected for an infusion as long as the appropriate concentration and rate are selected to avoid toxicity, the objective of a continuous PNB is often to extend analgesia (rather than anesthesia) in the postoperative period. Ropivacaine's selective blockade of sensory transmission and motor sparing at low concentrations are ideal characteristics for continuous nerve block infusions. Depending on the block technique, we recommend ropivacaine 0.2% at the following infusion rates if a regimen with only a basal infusion is chosen (Table 24.3).

Local Anesthetic Adjuvants for PNB

Prolongation of peripheral neural blockade is best accomplished by placement of a continuous nerve block catheter. However, certain agents have been studied that may offer some degree of analgesic prolongation for cases requiring <24-h coverage [4]:

Epinephrine	Prolongs medium-acting agent anesthesia by 60% and speeds onset
Dose	1:200,000 dilution, 5 mcg/mL
Agents	Medium-acting – lidocaine, mepivacaine; not for use with ropivacaine
Modality	Single-shot only; not supported for use in CPNBs
Toxicity	Tachycardia, hypertension, and neuronal ischemia; avoid in patients with diabetes or peripheral vascular disease who may have preexisting neuropathy
Clonidine	Off-label use. Prolongs medium-acting agent analgesia by 100%. Speeds onset in areas of localized infection. No benefits for tourniquet pain
Dose	0.1–0.5 mcg/kg up to 150 mcg total
Agents	Medium-acting – lidocaine, mepivacaine
Modality	Single-shot only
Toxicity	Hypotension, bradycardia, and sedation (rare if dose < 1.5mcg/kg)

All other adjuvants, including ketamine, neostigmine, tramadol, dexamethasone, and opioids, are not supported by the literature for direct combination with local anesthetic solutions during neural blockade injections.

Postoperative Management of PNB and CPNB

Single-Shot Nerve Blocks

In the immediate postoperative period, a number of precautions must be taken in order to minimize and avoid injury in those patients who have received a peripheral nerve block. All patients who have received a block should be readily identifiable by all clinical support staff. In addition to the procedure note, we recommend the routine use of a disposable identification bracelet, placed on the same limb as the patient's medical ID band. The bracelet should clearly state "nerve block, regional," or similar, in large type and, preferably, on a colored background. This will allow for rapid identification of patients who require additional precautions in the PACU, hospital floor, and at home. The patient should be routinely assessed for [5]:

1. Pain score and presence of breakthrough pain
2. Motor and sensory block
3. Blocked limb protection – i.e., padding, splinting, etc.

4. Signs of infection or hematoma at the catheter site
5. Hemodynamic stability – for paraneuraxial blocks
6. Antithrombotic therapy (ASRA guidelines)

Continuous Nerve Blocks

Management of patients with CPNBs requires daily evaluation of their catheter/pump system as well as various physiologic and physical parameters. The clinician must focus on the pain score reported by the patient in order to make any necessary adjustments to the anesthetic infusion. Prior to implementing any changes to the infusion, however, the patient should be evaluated for:

1. Stable vital signs
2. A catheter site that is clean, dry, and intact
3. A catheter-pump circuit that is unobstructed
4. Catheter depth that corresponds to the placement record
5. Pain pump that is filled and operational

In the presence of hemodynamic instability, paraneuraxial blocks can exacerbate hypotension and should be bolused very carefully and with low concentration of the local anesthetic agent. Significant hypotension may warrant withholding the catheter infusion until symptoms are treated or improved. Any catheter site that is painful, erythematous, warm, or pustulent should prompt immediate removal of the catheter due to concerns of infection.

To determine that a patient's breakthrough pain is not the result of a malfunctioning catheter-pump system, the clinician should inspect the tubing for kinks, disconnections, and a functioning reservoir. To evaluate the catheter for proper placement or presence of a distal obstruction, a small bolus (5 mL) of a local anesthetic (such as 1% lidocaine) can be slowly injected after careful aspiration for blood or CSF (paraneuraxial catheters). If the patient is reporting pain relief associated with the local anesthetic bolus, the infusion rate should be increased by 20%. If there is no improvement after the bolus injection, the catheter can be withdrawn 1–2 cm and rebolused. Inability to bolus the catheter due to obstruction or failure to establish any sensory level warrants removal of the catheter.

Most patients report significantly decreased analgesic requirements by the third postoperative day, at which time the catheter is often removed and the patient is transitioned to PO pain medication. It is not unusual, however, to use a CPNB for 5–7 days and as long as 35 days (reported by the US Military). The timing of catheter removal should be customized to each individual patient, taking into consideration the type of surgery, physical therapy, concomitant anticoagulation, risk of infection, and patient satisfaction. Pain pump systems are designed to be tamper-proof and safely removed by patients at home. Disposition, therefore, should not impact on the timing of discontinuation. If a patient is discharged with a pain pump, daily follow-up should be maintained either by follow-up visits or via telephone.

Table 24.4 Nerve block complications and their avoidance

Complication	Blocks involved	Strategy
Infection	All	Use strict sterile technique
Vascular puncture	All	Avoid multiple needle passes. Caution with anticoagulated patients. Use ultrasound color-flow if available. Maintain compression for 5 min if puncture occurs
Hematoma	All	Avoid multiple needle passes. Use ultrasound guidance to visualize needle path and adjacent vessels. Exert caution in anticoagulated patients. Monitor patient after block for signs of bleeding
Last	All	Aspirate prior to each injection. Inject in 5 cc boluses between aspirations. Avoid highly vascularized injection sites. LA mixtures have additive toxicities. Remain within the recommended drug dosage
Nerve injury	All	Avoid block performance under deep sedation/general anesthesia. Avoid forceful injections. Don't overcome resistance when injecting. Consider combining different nerve block modalities such as injection pressure monitoring, ultrasound, and nerve stimulation
Total spinal/epidural spread	Interscalene, deep cervical, supraclavicular, paravertebral, lumbar plexus	Avoid forceful injections. Inject slowly. Aspirate for CSF prior to injections. Minimize drug volume

Complication Prevention

Complications related to neural blockade are rare but not completely avoidable (Table 24.4). For instance, the incidence of nerve injury associated with peripheral nerve blocks varies depending on the definition between 0.02% up to 10%. While more serious injury such as permanent loss of motor function is on the lower end of this range, neurologic symptoms such as tingling or residual areas of numbness occur much more frequently. Intravascular injections of local anesthetics resulting in seizures are reported in 1:1,000 cases. In order to prevent the occurrence of complications, a number of basic technical steps can be adopted regardless of the technique employed:

1. Aspirate prior to any injection.
2. Inject in small 5 cc boluses and continue to aspirate between injections.
3. Inject slowly.
4. Do not overcome resistance during the injection.
5. Do not inject if the pressure is greater than 20 psi (requires injection pressure monitoring).

These simple maneuvers are designed for rapid and early identification of intraneural (intrafascicular) or intravascular needle-tip placement. The presence of any substance (blood, CSF, air, urine) or resistance upon injection warrants immediate withdrawal and repositioning of the needle. In order to respond rapidly and effectively to an accidental intravascular injection or local anesthetic systemic toxicity, the patient should be monitored using ASA standards during any nerve block procedure. We recommend the following protocol:

1. Oxygen via face mask
2. ECG, pulse oximetry, and noninvasive blood pressure monitoring
3. A running IV
4. Resuscitation and intubation equipment/drugs nearby

In addition to the ASA standard monitors and resuscitation equipment, certain essential drugs should also be immediately available whenever placing a nerve block in order to rapidly treat central nervous system and cardiovascular toxicity:

1. Midazolam – premedicate with 1–2 mg IV; if seizure develops, give 5 mg IV push.
2. Propofol – only use of midazolam is not available; large doses could exacerbate cardiovascular collapse and obtundation; if seizure develops, give 15–30 mg IV push.
3. Intralipid 20% emulsion – if signs of cardiotoxicity develop, give 1.5 mL/kg IV push. Note: Intralipid is most effective against bupivacaine-induced cardiotoxicity [6–8].

In the event of local anesthetic systemic toxicity (LAST), the following guidelines should be followed [9]:

1. *Get help*.
2. *Initial focus*.
   (a) Airway management: ventilate with 100% oxygen.
   (b) Seizure suppression: benzodiazepines are preferred.
   (c) Basic and advanced cardiac life support (BLS/ACLS) may require prolonged effort.
   (d) Epinephrine dose should be *decreased* to 100–200 mcg IV push boluses as part of ACLS protocol due to its arrythmogenic potential in LAST victims.
3. *Infuse* 20% lipid emulsion (values in parenthesis are for a 70-kg patient).
   (a) Bolus 1.5 mL/kg (lean body mass) intravenously over 1 min (~100 mL).
   (b) Continuous infusion at 0.25 mL/kg/min (~18 mL/min; adjust by roller clamp).
   (c) Repeat bolus once or twice for persistent cardiovascular collapse.
   (d) Double the infusion rate to 0.5 mL/kg/min if blood pressure remains low.
   (e) Continue infusion for at least 10 min after attaining circulatory stability.
   (f) Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 min.

4. *Avoid* vasopressin, calcium channel blockers, β-blockers, or local anesthetic.
5. *Alert* the nearest facility having cardiopulmonary bypass capability.
6. *Avoid propofol* in patients having signs of cardiovascular instability.

Epidurals

Indications

Any major surgical procedure below T1, management of acute postoperative pain for thoracic procedures, management of acute traumatic pain due to bone fractures, labor analgesia, cesarean section, and prevention of phantom limb pain postamputation.

Contraindications

- Patient refusal
- Infection at the injection site
- Hemodynamically significant hypovolemia or untreated bacteremia/sepsis
- Increased intracranial pressure
- Anticoagulation not compatible with epidural catheter placement (ASRA guidelines)

Benefits (Evidence-Based)

- Reduced autonomic hyperactivity
- Reduced cardiovascular stress
- Improved postoperative pulmonary function
- Improved postoperative gastrointestinal motility
- Reduced hypercoagulability

Patient Preparation

Prior to any neuraxial block, all patients should be placed on ASA-standard monitors as well as O_2 via face mask. A working peripheral IV should be present and a small 500 cc bolus infusion of isotonic fluids should be administered prior to the procedure for intravascular compensation of the sympathectomy-associated hypotension that may occur. Resuscitation equipment and drugs, in particular

epinephrine and ephedrine, should be readily available to manage any cardiovascular complications.

Epidural Injection Site Selection

The location of the injection/catheter is critical to successful epidural analgesia and patient safety. Although it is possible to overcome placement issues by infusing larger doses of local anesthetic, this practice places the patient at undue risk for hypotension, cardiovascular collapse, and potentially high neural blockade requiring intubation. It is preferable, therefore, to place the single-shot injection or catheter at a vertebral level that approximates the center of the surgical incision. This will minimize the dose and infusion rate required to achieve satisfactory analgesia and any potential side effects.

Technique: Single-Shot Versus Catheter Versus CSE

Regardless of the technique used, epidural placement is optimal if done before surgery. Patient positioning, intraoperative hemodynamic stability, and postoperative analgesia are superior if the neuraxial block is placed and established prior to surgical incision.

Special Situation: Epidurals and General Anesthesia

If combined with general anesthesia, catheters placed preoperatively should be tested before induction while the patient is awake and able to report symptoms and anesthetic level. Ideally, the epidural infusion should be used intraoperatively. If, however, epidural analgesia is withheld until the postoperative phase, a normal saline solution should be infused during surgery at 2–3 mL/h to maintain catheter patency.

Epidural Infusions

The following parameters must be decided:

- Basal rate (mL/h)
- Bolus dose (mL)

- Bolus interval (min)
- Max dose (mL/h)

Regardless of the local anesthetic agent used, epidural infusion rates should be calculated using a systematic approach: basal rate dose (BRD) = levels required (L) × 1.5 mL per level × distribution factor (df). The distribution factor (df) depends on the location of the epidural [10–12]:

$$\mathrm{BRD} = \mathrm{L} \times 1.5\mathrm{mL} \times (\mathrm{df}).k$$

1. One milliliter of LA produces 1–1.5 dermatomes of analgesia – obese and term/near term pregnancy patients have reduced epidural spaces and should prompt a 1 mL LA = 1 dermatome conversion.
2. In the lumbar region, 2/3 of the injectate travels cephalad while 1/3 travels caudad. This is a result of the negative intrathoracic pressure generated during spontaneous ventilation. In an intubated patient, however, positive pressure ventilation counteracts cephalad extension, and spread may occur equally in the cephalad and caudal directions.
3. In the thoracic region, location is critical. In a spontaneously ventilating patient, local anesthetic spread is greatest toward the midthoracic region:
   (a) High thoracic injections (C7–T2) result in preferential caudal spread.
   (b) Midthoracic injections (T3–T5) result in equally caudal and cephalad spread.
   (c) Low thoracic injections (T6–T12) result in preferential cephalad spread.
4. In the thoracic region, positive pressure ventilation reverses the above pattern so that epidural injections move preferentially away from the thorax.

Example: Calculate the infusion rate for an L4–L5 epidural used to provide analgesia for an abdominal surgery with an incision from the pubic symphysis (L1) to the umbilicus (T10).

Dermatomes from catheter site to cephalad edge of incision = L4 to T10 = 5
LA volume required to reach T10 = 1.5 mL/level × 5 levels = 7.5 mL
LA distribution factor toward T10 = 2/3, hence 7.5 = 2/3 (bolus) → (bolus) = 7.5 × 1.5 = 11.25 mL

Hence, 11.25 mL/h would be used as the basal rate, and 25–50% of this volume could be used as a bolus dose for breakthrough pain: approximately settings – basal rate = 12 mL/h, bolus = 3 mL, interval = 15 min, total = 24 mL/h max.

If patient-controlled epidural analgesia (PCEA) is used, the bolus dose is typically 25–50% of the basal rate, administered every 10–15 min. The infusion should be adjusted after several hours based on the number of bolus doses demanded (Table 24.5). If patient need exceeds the number of boluses per hour, the basal rate should be increased by 50% of the hourly bolus dose. Conversely, the basal rate may be left unchanged while the frequency or dosage of the bolus is increased. It is recommended that only one parameter be modified at a time to ensure patient safety and simplify evaluation of any medication changes.

Table 24.5 PCEA (patient-controlled epidural) infusion dosing table

Location	Basal rate (mL/h)	Bolus dose (mL)	Interval (min)	Total (mL/h)
Midthorax	4	1	15	8
Low thorax	6	2	15	14
Lumbar	12	4	15	28
Lumbar (labor)	6	6	10–15	42–30

Table 24.6 Local anesthetics for epidural blockade

Drug	Concentration (%)	Onset (min)	Duration plain/ +epinephrine (min)	Adjuvants
2-Chloroprocaine	3	10–15	45–60/60–90	Avoid w/ narcotics
Lidocaine	2	10–15	80–120/120–180	
Mepivacaine	1	15	90–160/160–200	
	2	15	Same	
Bupivacaine	0.25	15–20	160–220/180+	Avoid w/ DepoDur
	0.375–0.5			Avoid alkalinization
				No prolongation w/ epi
Etidocaine	1	15–20	120–200/150+	
Ropivacaine	0.5	15–20	140–180/150+	Avoid alkalinization
	0.6–0.75			No prolongation w/ epi
Levobupivacaine	0.5	15–20	160–220/180+	No prolongation w/ epi

Local Anesthetics for Epidurals

The choice of local anesthetic for epidural blockade should be based on the following (Table 24.6) [13–16]:

1. Required speed of onset – elective vs. urgent/emergent situation
2. Required length of blockade – short vs. long procedures
3. Required intensity of blockade – anesthesia vs. analgesia
4. Use of adjuvants (i.e., narcotics, epinephrine, etc.)

Epidural/Spinal Anesthetic Complications

Epidural Hematoma

Incidence = 1:168,000 (after epidural) 1:220,000 (after spinal) [17].
Symptoms: localized back pain, may radiate; sensory and/or motor deficits in the lower extremities; and urinary/rectal incontinence.
Diagnosis: spinal MRI (STAT) is the gold standard; spine CT also acceptable.
Treatment: surgical decompression and evacuation (emergently); neurosurgical consult should be sought as soon as diagnostic imaging is ordered.
Risk factors: anticoagulation, coagulopathy, traumatic needle placement, and multiple needle attempts.

Epidural Abscess

Incidence = 1:10,000 [18].
Symptoms: Localized back pain, may radiate; sensory and/or motor deficits in the lower extremities; and urinary/rectal incontinence. Symptoms are progressive and usually in the setting of fever, leukocytosis, and elevated ESR (unlike an epidural hematoma).
Diagnosis: spinal MRI (stat) is the gold standard; spine CT also acceptable.
Treatment: surgical decompression and evacuation (emergently); neurosurgical consult should be sought as soon as diagnostic imaging is ordered; IV antibiotics may be an option in the absence of neurologic sequelae.
Risk factors: IV drug abuse, concomitant nonspinal infections, and neurosurgical procedures.

Postdural Puncture Headache

Symptoms: positional headache, often relieved by recumbency; may be bilateral, frontal, or occipital; may radiate to neck; and described as throbbing and continuous; diplopia and tinnitus may be present; nausea and vomiting.
Onset: 12–72 h following a neuraxial procedure. If symptoms are reported earlier, pneumocephalus should be suspected.
Diagnosis: clinical signs/symptoms.
Treatment: caffeine 500 mg IV in 1 L over 2–3 h, may repeat once. Tramadol 50 mg PO q4h prn has shown 75% success in a recent pilot study involving PDPH patients [19]. Generous IV and PO fluids are encouraged during the treatment phase. If conservative management fails, an epidural blood patch using 10–20 mL of sterile autologous blood will yield 90–95% symptomatic relief. In those patients who fail the first blood patch, a second dose will yield 95% efficacy.

Narcotic Analgesia

Patient-Controlled Analgesia

Narcotic analgesia for postoperative pain management often relies on empiric dosing schedules that are dependent on a variety of patient clinical parameters: age, weight, previous narcotic exposure, concurrent medications, clinical status, and pain severity [20]. Caution should be exercised with elderly, patients with renal failure, and those who are suspected to suffer from obstructive sleep apnea (OSA). Frequent patient monitoring and clinical judgment are imperative for the safe implementation of these agents (Table 24.7).

Table 24.7

Morphine PCA dosing table (opioid naïve patients)					
Bolus (mg)	0.5	1	1	2	2
Interval (min)	6	10	6	15	10
Max boluses/HR	10	6	10	4	6
Hydromorphone PCA dosing table (opioid naïve patients)					
Bolus (mg)	0.2	0.2	0.4	0.3	0.4
Interval (min)	10	6	10	6	6
Max boluses/h	6	10	6	10	10
Fentanyl PCA dosing table (opioid naïve patients)					
Bolus (mg)	0.01	0.02	0.02	0.04	0.04
Interval (min)	6	10	6	10	6
Max boluses/h	10	6	10	6	10
Remifentanil PCA dosing table (opioid naïve patients)					
Bolus (mcg/kg)	0.25	0.25	0.4	0.4	0.5
Interval (min)	2	1	3	2	2
Max boluses/h	30	60	20	30	30

Opioid Dosing Conversion: Step-by-Step

- Total all oral or transdermal opioids currently taken by patient in a 24-h period.
- Multiply the amount of each drug by its bioavailability.
- Convert each bioavailable dose to IV morphine equivalents (Table 24.8).
- Add any parenteral opioids currently taken as IV morphine equivalents.
- Total all IV morphine equivalents.
- Reduce the new total by 30%.
- Select new opioid and convert dose from IV morphine equivalent (Table 24.8).

Neuraxial Opioids

The injection of opioids in the epidural or subarachnoid space as the sole analgesic agent or as an adjuvant to local anesthetics takes advantage of their direct action on the dorsal horn of the spinal cord, specifically within the substantia gelatinosa. The benefits afforded by this direct access to nociceptive receptors include:

1. Reduced dosing requirements
2. Decreased systemic side effects
3. Increased visceral analgesia for abdominal and thoracic procedures

In terms of onset and duration, opioid activity within the neuraxis is primarily determined by the lipid solubility of each medication. Hydrophilic (or poorly lipid soluble) narcotics, such as morphine, tend to exhibit a slower onset of action and

Table 24.8 Dosing and conversion chart for opioid analgesics

	Equianalgesic oral dose	Equianalgesic IV dose	Starting dose adults > 50 kg (oral)	Starting dose adults > 50 kg (IV)	Bioavailability	Starting dose adults < 50 kg (IV)
Opioid agonists						
Morphine	30 mg q 3–4 h	10 mg q 3–4 h	15–30 mg q 3–4 h	10 mg q 3–4 h	0.3	0.1 mg/kg q 3–4 h
Codeine	130 mg q 3–4 h	75 mg q 3–4 h	60 mg q 3–4 h	60 mg q 2 h IM or SQ	0.3	Not recommended
Fentanyl		0.1				
Hydromorphone	7.5 mg q 3–4 h	1.5 mg q 3–4 h	6 mg q 3–4 h	1.5 mg q 3–4 h	0.6	0.015 mg/kg q 3–4 h
Hydrocodone	30 mg q 3–4 h	Not available	10 mg q 3–4 h	Not available		Not available
Levorphanol	4 mg q 6–8 h	2 mg q 6–8 h	4 mg q 6–8 h	0.04 mg/kg q 6–8 h		0.02 mg/kg q 6–8 h
Meperidine	300 mg q 2–3 h	75 mg q 3 h	Not recommended	100 mg q 3 h	0.3	0.75 mg/kg q 2–3 h
Methadone (Acute)	20 mg q 6–8 h	10 mg q 6–8 h	20 mg q 6–8 h	10 mg q 6–8 h	1	0.1 mg/kg q 6–8 h
Oxycodone	20 mg q 3–4 h	Not available	10 mg q 3–4 h	Not available	0.8	Not available
Oxymorphone	Not available	1 mg q 3–4 h	Not available	1 mg q 3–4 h		Not recommended
Opioid agonist-antagonist and partial agonist						
Buprenorphine	Not available	0.3–0.4 mg q 6–8 h	Not available	0.4 mg q 6–8 h		0.004 mg/kg q 6–8 h
Butorphanol	Not available	2 mg q 3–4 h	Not available	2 mg q 3–4 h		Not recommended
Nalbuphine	Not available	10 mg q 3–4 h	Not available	10 mg q 3–4 h		0.1 mg/kg q 3–4 h
Pentazocine	150 mg q 3–4 h	60 mg q 3–4 h	50 mg q 4–6 h	Not recommended		Not recommended

Table 24.9 Neuraxial opioid side effects and treatment

Side effect	Frequency (%)	Treatment
Respiratory depression	<0.2	Naloxone bolus 0.2–2 mg IV
		Naloxone infusion 5 mcg/kg/h IV
Pruritus	20–60	Nalbuphine 3 mg IV
		Benadryl 25 mg IV
Nausea	6–50	Zofran 4 mg IV
		Check for hypotension: give IV fluid bolus PRN
Urinary retention	4–40	Catheterization

greater duration. Hydrophobic (or highly lipid soluble) narcotics, such as fentanyl and sufentanil, demonstrate much faster onset of action and shorter duration:

1. Morphine (intrathecal dose = 0.25 mg; epidural dose = 1–5 mg):
   (a) Poor lipid solubility (i.e., hydrophilic)
   (b) Slow onset of action = 60 min
   (c) Long duration = up to 24 h
   (d) Extensive cephalad spread = wide analgesic band
   (e) May produce delayed respiratory depression, and patients should be monitored for 24 h postinjection
2. Fentanyl (intrathecal dose = 10–15 mcg; epidural dose = 75–100)
   Sufentanil (intrathecal dose = 2–3 mcg; epidural dose = 10–15 mcg):
   (a) High lipid solubility (i.e., hydrophobic)
   (b) Fast onset of action = 6 min
   (c) Short duration = 2–6 h (sufentanil > fentanyl)
   (d) Limited cephalad spread = narrow analgesic band
3. Hydromorphone (intrathecal dose = 40–60 mcg; epidural dose = 0.25–1 mg):
   (a) Intermediate lipid solubility
   (b) Intermediate onset of action = 10–20 min
   (c) Intermediate duration = 8–12 h
   (d) Intermediate cephalad spread = intermediate analgesic band

The cephalad spread of neuraxial opioids within the CSF is related to their degree of water solubility. In turn, this spread determines both the size of the analgesic band as well as the potential for serious side effects such as delayed respiratory depression (see Table 24.9).

Continuous Epidural Infusions

Step 1: Test dose:

Prior to initializing or restarting an epidural infusion, all catheters should be tested for intravascular or intrathecal migration. A 3 cc test dose using lidocaine 1.5% with

1:200,000 epinephrine should be bolused through the catheter after negative aspiration for blood or CSF.

Positive test dose = IV or intrathecal catheter migration:

1. HR increase of 20%
2. BP increase of 20 mmHg
3. High anesthetic level (high spinal)

Step 2: Infusion.

Avoidance and Treatment of Neuraxial Opioid Side Effects

Monitoring: All patients who receive neuraxial opioids should be monitored for signs of respiratory depression.

1. May occur 6–18 h after initial injection.
2. Intrathecal morphine presents the greatest risk, and patient monitoring should be maintained for a minimum of 24 h.
3. Intrathecal fentanyl rarely produces respiratory depression after 2 h.

Risk factors:

1. Advanced age
2. Obstructive sleep apnea
3. Intrathoracic procedures
4. Concomitant systemic opioids or sedatives

Non-opioid Analgesia

Nonnarcotic agents are an integral component of multimodal analgesia and reduce the opioid requirements of patients while providing both a preemptive and complementary analgesic mechanism of action. This drug group is composed of various classes, including COX-2 inhibitors, NMDA agonists, acetaminophen, alpha-2 adrenergic agonists, and neuromodulators. In the postoperative management of acute pain, these agents offer an analgesic alternative that is devoid of the negative gastrointestinal, sedative, and respiratory side effects related to narcotics. Optimal postoperative pain management with these compounds, however, begins with their administration preoperatively, an analgesic strategy termed "preemptive analgesia."

NSAIDs (Table 24.10)

Mechanism: This drug class blocks the synthesis of prostaglandins by inhibiting cyclooxygenase (COX) types I and II, thereby reducing inflammatory mediators of

Table 24.10

NSAIDs	Dose (mg)	Route
Ketorolac	15–30	SQ/IV
Diclofenac	50–100	PO/IM/IV
Ibuprofen	300–800	PO
Indomethacin	25–50	PO/PR/IM
Naproxen	250–500	PO
Celecoxib	100–200	PO
Rofecoxib	25–50	PO

Table 24.11

COX-2 drug	Route	Onset (min)	Duration (h)	COX2/COX1 activity	Side effect
Celecoxib	PO	30–50	4–8	8	Sulfa allergy
Rofecoxib (withdrawn)	PO	30–50	12–24	35	Leg edema, hypertension
Parecoxib	IM/IV	10–15	6–12	–	
Valdecoxib	PO	30–40	6–12	30	
Etoricoxib	PO	20–30	>24	106	

the acute pain response at the peripheral and central nervous system. They are characterized by anti-inflammatory, antipyretic, and analgesic activity.

Evidence-based analgesia: Numerous studies suggest that ketorolac and diclofenac offer the greatest analgesic equivalency to narcotics, when used as part of a multimodal analgesic regimen. By reducing the incidence of opioid-induced PONV, sedation, and respiratory depression via decreasing the narcotic requirements of the postoperative patient, these agents also facilitate earlier discharge.

Adverse effects: Despite the numerous benefits of the NSAID drug class, they have the potential to cause gastrointestinal mucosal damage, renal tubular dysfunction, and inhibit platelet activity. As a result, these agents should be used with caution in patients with gastric ulcers, renal insufficiency, extremes of age, or actively bleeding. Furthermore, controversy exists regarding the inhibitory effect of ketorolac on osteoblastic activity and the impact on postoperative bone healing following orthopedic surgery. Although only animal data support this finding, ketorolac regimens should be minimized to 72 h of continuous parenteral use while monitoring renal function.

Timing: Maximal benefit is achieved by preoperative administration followed by postoperative continuation through discharge and at home.

COX-2 Inhibitors (Table 24.11)

Mechanism: Selective inhibition of COX-2.

Evidence-based analgesia: Celecoxib 400 mg PO, rofecoxib 50 mg PO, and valdecoxib 40 mg PO demonstrate a 40–50% reduction in narcotic requirements for postoperative pain management.

Adverse effects: COX-inhibitor activity on bone growth is dose-dependent and reversible; therefore, these agents should be used for no more than 3–5 days in the early postoperative period for orthopedic procedures. Due to their selective inhibition of COX-2, these agents minimize bleeding, renal tubular damage, and gastrointestinal damage compared to the nonselective NSAIDs.

Timing: Maximal benefit is achieved by preoperative administration followed by postoperative continuation for 3–5 days.

Acetaminophen

Mechanism: Selective inhibition of COX-2.

Evidence-based analgesia: Acetaminophen 35 mg/kg is equivalent to ketorolac 1 mg/kg IV. In adults, acetaminophen 2 g PO is equivalent to celecoxib 200 mg.

Adverse effects: Acetaminophen is the safest non-opioid analgesic of the NSAID class. It has minimal effects on postoperative bleeding or gastrointestinal mucosal damage, as well as no contraindications in the patient with preexisting cardiac disease. Overdosage, however, may result in hepatotoxicity and agranulocytosis.

Timing: In adults, acetaminophen 1 g q4h as an adjuvant to patient-controlled analgesia (PCA) morphine has been shown to improve pain relief after major orthopedic procedures. In children, an initial preoperative dose of 30–40 mg/kg followed by a maintenance dose of 15–20 mg/kg q6–8 h during the early postoperative period is recommended.

NMDA Antagonists: Ketamine

Mechanism: Primarily, antagonism of the NMDA receptor, but also has muscarinic, voltage-sensitive calcium channel, and opioid mu receptor antagonist properties. Limited local anesthetic activity is also demonstrated.

Evidence-based analgesia: Ketamine 0.1–0.2 mg/kg as an adjuvant intraoperative bolus dose has opioid-sparing effects with reduced incidence of adverse events. For sedation, a combination of ketamine 4–18 mg/kg/min with propofol 30–90 mg/kg/min greatly reduces the respiratory depression associated with the more commonly used sedative-narcotic regimens. In the postoperative period, a 1:1 ratio morphine-ketamine PCA with a lockout interval of 8 min has shown positive results for analgesia after major orthopedic procedures.

Timing: In the chronic pain patient, a preoperative or adjuvant induction dose of ketamine 50 mg IV has been shown to improve postoperative pain scores via suspected central NMDA receptor antagonism. Otherwise, most analgesic regimens with ketamine are limited to the intraoperative setting as small adjuvant bolus doses or in combination with sedatives.

Neuraxial use: Although studies are limited [21], ketamine has demonstrated enhanced analgesia when 0.25 mg/kg is administered epidurally in combination with ropivacaine for total knee arthroplasty. Small epidural doses of 20–30 mg have also been shown to improve epidural morphine-induced analgesia after major abdominal surgery. Intrathecal ketamine (1 mg) has demonstrated a 50% reduction in the intrathecal morphine dose requirement while maintaining equivalent analgesia in cancer patients. All neuraxial administrations of ketamine should be limited to a few days due to the risk of subpial vacuolar myelopathy when used as a continuous infusion in doses > 5 mg/day intrathecally.

Adverse effects: Include hypertension, diplopia and nystagmus, dysphoria, dizziness, nausea, sialorrhea, and cardiac arrhythmias. Premedication with midazolam and glycopyrrolate reduces the incidence of neurologic side effects and hypersalivation. Patients should also be carefully screened for preexisting psychiatric conditions or history of drug abuse due to their predisposition for ketamine-induced psychosis.

Alpha-2 Adrenergic Agonists: Clonidine and Dexmedetomidine

Mechanism: Modulation of central alpha-2 receptors.

Evidence-based analgesia: Both clonidine and dexmedetomidine reduce opioid requirements while potentiating central analgesic activity. As discussed earlier, clonidine (up to 100 mcg) can greatly prolong the duration of medium-acting local anesthetics when used for peripheral neural blockade. Centrally, epidural (25–50 mcg/hr) and intrathecal clonidine (75 mcg) improve postoperative analgesia when used as an adjuvant to the local anesthetic infusion with or without morphine. Premedication with oral clonidine has been shown to reduce the postoperative morphine-PCA requirements by up to 50% after radical prostatectomy.

Dexmedetomidine also reduces postoperative pain and opioid analgesic requirements. When administered intraoperatively as 1 mg/kg followed by 0.4 mg/kg/h infusion, a 66% reduction in PCA morphine requirements has been demonstrated after major surgery. In patients managed with fentanyl PCA for postoperative analgesia, dexmedetomidine demonstrates synergy when given as an infusion, thereby enhancing analgesia while reducing respiratory depression.

Timing: Clonidine should be used in the preoperative or intraoperative setting, while dexmedetomidine can be administered throughout the perioperative period.

Adverse effects: Include sedation, bradycardia, and hypotension.

Neuromodulators: Gabapentin and Pregabalin

Mechanism: Anticonvulsants with structural analogy to GABA. Exact mechanism is unknown.

Evidence-based analgesia: Gabapentin (700–1200 mg) PO has been used to treat chronic neuropathic pain in escalating doses up to a maximum of 2,400 mg PO per day. Preoperative gabapentin (1.2 g) has also been shown to significantly reduce opioid analgesic requirements by up to 66% without increasing side effects in patients undergoing arthroscopic knee surgery. Pregabalin is another related compound which has been studied for the treatment of neuropathic pain. Due to its greater potency, a dose of 75–100 mg PO per day, up to twice daily dosing, has demonstrated analgesic effects for chronic neuropathies of herpetic and diabetic origin.

Timing: Preoperative and postoperative use is supported by the literature. Dosing prior to bedtime is recommended due to the sedative effects associated with these compounds.

Adverse effects: Include (in order of frequency) dizziness, drowsiness, peripheral edema, myalgias and myoclonus, as well as first-degree heart block and hypotension.

Clinical Pearls

Epidurals

- Blood patches can be safely administered in an HIV-positive patient since the virus is neurotropic and already present in the CSF. Blood patches, however, are contraindicated in patients who have any hematogenous neoplasm such as lymphoma or leukemia since the injection of autologous blood could seed the neuraxis with neoplastic cells. Any potential diagnosis of PDPH should be made after ruling out meningitis.

Patient-Controlled Analgesia

- Avoid PCA basal rate infusions in opioid-naïve patients, OSA, and renal failure.
- Titrate doses slowly. Use smaller boluses initially and review narcotic consumption per hour to adjust PCA settings. Establish a basal rate only after hourly bolus dose use has been evaluated (Table 24.7).
- Fentanyl and remifentanil demonstrate the lowest incidence of nausea, vomiting, constipation, and respiratory depression.

- When dosing narcotics based on weight, use ideal body mass for lipophilic narcotics (fentanyl, sufentanil) and total body mass for hydrophilic compounds (morphine, hydromorphone).
- PCA use should be complemented by long-acting analgesics to establish a baseline level of analgesia. This can be achieved with NSAIDs, nerve blocks, epidurals, or other narcotics

Neuraxial Opioids

- In patients who are beta-blocked, look for an increase in systolic blood pressure. In the laboring obstetric patient, administer the test dose between contractions to discriminate any increase in BP from uterine pain.
- IV naloxone is the narcotic antagonist of choice. In the setting of respiratory depression, however, a single IV dose is not effective due to naloxone's shorter duration of action than the neuraxial opioid. Respiratory depression can recur as early as 20 min following a dose of naloxone. Patients, therefore, should be transferred to a monitored unit, their epidural/intrathecal infusions withheld, and a naloxone infusion started at 5 mcg/kg/h (this dose will reverse respiratory depression with minimal analgesic antagonism).

Multiple-Choice Questions

1. What fraction of the local anesthetic bolus will travel cephalad in a lumbar epidural catheter placed at the L4–L5 interspace in a spontaneously ventilating patient?
   (a) ½
   (b) 1/3
   (c) 2/3
   (d) ¾

2. How soon after a patient receives a prophylactic dose of Lovenox is it considered safe to place an epidural?
   (a) 2 h
   (b) 24 h
   (c) 12 h
   (d) No need to wait

3. What is the maximum INR that is considered safe for epidural placement?
   (a) 1
   (b) 2
   (c) 1.5
   (d) <1.5

4. The most important factor in determining anesthetic level after an epidural bolus is:
   (a) Concentration of local anesthetic
   (b) Volume of local anesthetic
   (c) Total dose of anesthetic
   (d) Patient position

5. The most important factor in determining the anesthetic level after a single-shot spinal is:
   (a) Volume of local anesthetic
   (b) Concentration of local anesthetic
   (c) Total dose of anesthetic
   (d) Baricity of local anesthetic

6. What nerve is typically spared after a single-shot interscalene block?
   (a) Median nerve
   (b) Musculocutaneous nerve
   (c) Radial nerve
   (d) Ulnar nerve

7. What steps are taken to avoid intravascular injection during nerve blockade?
   (a) Frequent aspiration prior to injection
   (b) Slow injection
   (c) Avoidance of injections when resistance is encountered
   (d) Adding epinephrine to the local anesthetic

8. What nerve must be blocked separately to ensure complete neural blockade during an axillary block?
   (a) Median nerve
   (b) Ulnar nerve
   (c) Radial nerve
   (d) Musculocutaneous nerve

9. A patient received an axillary single-shot nerve block for hand surgery and is complaining of tourniquet pain in the operative arm. Which nerve was missed?
   (a) Intercostobrachial
   (b) Musculocutaneous
   (c) Supraclavicular
   (d) Ulnar

(a) The Bezold–Jarisch reflex is associated with which nerve block?
   a. Axillary
   b. Infraclavicular
   c. Interscalene
   d. Interscalene in the sitting position

10. Which local anesthetic has the potential for greatest cardiotoxicity if dosage is held equal?
    (a) Lidocaine
    (b) Mepivacaine
    (c) Ropivacaine
    (d) Bupivacaine
11. Which opioid has the lowest incidence of respiratory depression when used for IV PCA?
    (a) Fentanyl
    (b) Meperidine
    (c) Hydromorphone
    (d) Morphine
12. When switching from IV to PO dosing, what conversion factor is required for hydromorphone?
    (a) 2×
    (b) 3×
    (c) 4×
    (d) 5×
13. Which non-opioid adjuvant exhibits synergy when coadministered with a fentanyl PCA?
    (a) Aspirin
    (b) Acetaminophen
    (c) Dexmedetomidine
    (d) Clonidine
14. Ketamine can be administered through which routes?
    (a) IV
    (b) IM
    (c) Epidural
    (d) All of the above

Answers:

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

12. a
13. d
14. c
15. d

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25

Sympathetic Blockade

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Blockade of the sympathetic nervous system is a key component of a pain management practitioner's skill set. These blocks can be used for diagnostic and therapeutic purposes and are generally indicated when pharmacological therapy is partially effective or ineffective in alleviating a patient's chronic pain. Detailed knowledge of the relevant anatomy is key as this will theoretically improve efficacy and minimize complications.

Indications and Contraindications

Specific indications will be listed with the individual blocks. Absolute and relative contraindications are listed in Table 25.1.

Table 25.1 Absolute (A) and relative (R) contraindications

Patient refusal – A
Local infection and Sepsis – A
Allergy to medications used – A and R
Coagulopathy – R
Anticoagulant therapy – R
History of facial trauma (SPG) – R
Pre-existing neurological deficits – R
History of previous surgery in the region of the block – R
Altered mental status – R

Table 25.2 Indications for sphenopalatine ganglion block

Sphenopalatine neuralgia
Trigeminal neuralgia
Migraine headaches
Cluster headaches
Post-traumatic headaches
Persistent idiopathic facial pain
Cancer of the tongue and floor of the mouth
Herpes zoster ophthalmicus

Evidence

The evidence for each block will be briefly reviewed. The grade of recommendation is based on Guyatt et al.'s [1] evidence-based medicine guidelines.

Sphenopalatine Ganglion Block

Indications

The indications for blockade of the sphenopalatine ganglion are listed in Table 25.2.

Anatomy

- The sphenopalatine ganglion (SPG) resides in the pterygopalatine fossa. The SPG is bordered medially by the palatine bone, cephalad by the sphenopalatine sinus, anteriorly by the posterior wall of the maxillary sinus, and posteriorly by the medial pterygoid plate.
- The pterygomaxillary fissure allows passage of a needle into the fossa, while the pterygopalatine foramen is located medial to the ganglion and is just posterior to

the middle turbinate. The fossa is approximately 1-cm wide and 2-cm high and resembles a V-shaped vase on a lateral fluoroscopic image. A large venous plexus overlies the fossa. Foramen rotundum and the pterygoid canal are located on the superolateral and inferomedial aspect of the fossa, respectively. The maxillary artery resides in the fossa.

- The ganglion is "suspended" from the maxillary nerve by the pterygopalatine nerves and is medial to the maxillary division of the trigeminal nerve. Posteriorly the ganglion is connected to the Vidian nerve which is formed by the deep petrosal (sympathetic from the upper thoracic spinal cord) and greater petrosal (parasympathetic from the superior salivatory nucleus) nerves.
- The ganglion has efferent branches and forms the superior posterior lateral nasal and pharyngeal nerves. Caudally, the greater and lesser palatine nerves exit the ganglion. Sensory fibers arise from the maxillary nerve, pass through the SPG, and innervate the upper teeth, nasal membranes, soft palate, and some parts of the pharynx. A small number of motor nerves are believed to travel with the sensory trunks.

Procedure

There are multiple techniques for blockade of the SPG. This chapter will focus on two techniques: the intranasal approach and the infrazygomatic approach.

Intranasal Approach

The intranasal SPG block can be safely performed in an office setting with appropriate monitoring. The location of the SPG in relation to the middle turbinate as well as the lateral nasal mucosa allows absorption of local anesthetic from one or two cotton-tipped applicators inserted into the naris. The local anesthetic of choice is 4% cocaine secondary to its inherent vasoconstrictor property. If this is not available or there is a contraindication to using cocaine, 1–2% lidocaine, 0.25–0.50% bupivacaine, or 0.2–0.5% ropivacaine can be used. If these are chosen, the practitioner can pretreat the nares with Neo-Synephrine to produce vasoconstriction.

- Place the patient in the supine position. Obtain baseline vital signs.
- Estimate the depth of insertion. Measure the distance from the opening of the naris to the mandibular notch beneath the zygoma. Place a mark corresponding to this depth on the shaft of the cotton-tipped applicator. Soak the applicators in the local anesthetic for several minutes.
- Slowly insert the applicator into the naris and advance in a line parallel to the zygoma with the tip angled laterally. Do not advance the applicator in a cephalad direction. This may cause epistaxis. The end point should be the depth marked on the applicator.

- Place a second applicator into the naris using the same technique, except advance it approximately 0.5–1.0 cm deeper and superior to the first. If resistance is encountered, slightly withdraw and redirect the applicator. The second applicator is not an absolute necessity. The nares of some patients may not accommodate it.
- Leave the applicator(s) in for 30–45 min. Signs of a successful block of the SPG include ipsilateral tearing, conjunctival injection, and nasal congestion.
- If the SPG is a pain generator or transmitter, analgesia should be apparent. If after 20–30 min there are no signs of a block or the patient has not received any pain relief, additional local anesthetic may be needed and can be trickled down the shaft of the applicator.
- Remove the cotton-tipped applicators after 45 min even if there are no signs of a block or analgesia. If there are no signs of a block or analgesia, the SPG may be too deep, i.e., too lateral, to be blocked by this technique or is not involved in the transmission of pain. Regardless, the infrazygomatic approach should be performed to rule out both of the aforementioned scenarios.

Infrazygomatic Approach

This approach to SPG blockade is technically challenging. Fluoroscopic guidance is highly recommended as this will anecdotally improve the success of the block, the speed at which it is performed, and decrease potential complications. Noninvasive monitors should be used. Light sedation with midazolam and fentanyl can be used, but on occasion, deeper sedation may be necessary for radiofrequency lesioning. Deep sedation is not necessary for pulsed radiofrequency (PRF).

- Place the patient in the supine position and sterilely prep and drape the appropriate side of the face.
- Palpate the mandibular notch and anesthetize the skin. If the notch is not palpable, identify the notch on a lateral fluoroscopic view.
- Identify the pterygopalatine fossa (appears as a "V") on the lateral image and superimpose the right and left fossae (Fig. 25.1a). This is accomplished by manipulating the C-arm or the head. The block can be performed with a 3.5-in., 22-gauge, short-bevel needle with the distal tip bent at a 30° angle away from the notch of the stylet, or with a curved, blunt, 10-cm, 20- or 22-gauge needle. The technique description will reflect the use of a blunt needle.
- Anesthetize the skin and insert a 1.25-in., 16- or 18-gauge angiocatheter through the skin and advance until it is just medial to the ramus of the mandible. This can be checked on an AP image.
- Pass the block needle through the angiocatheter and advance it medial, anterior, and slightly cephalad. Obtain a lateral image to check the direction of the needle. Your target is the upper midportion of the pterygopalatine fossa (Fig. 25.1b).
- Get an AP view and advance the needle toward the middle turbinate, stopping when the tip is adjacent to the palatine bone (Fig. 25.1c). If resistance is encountered

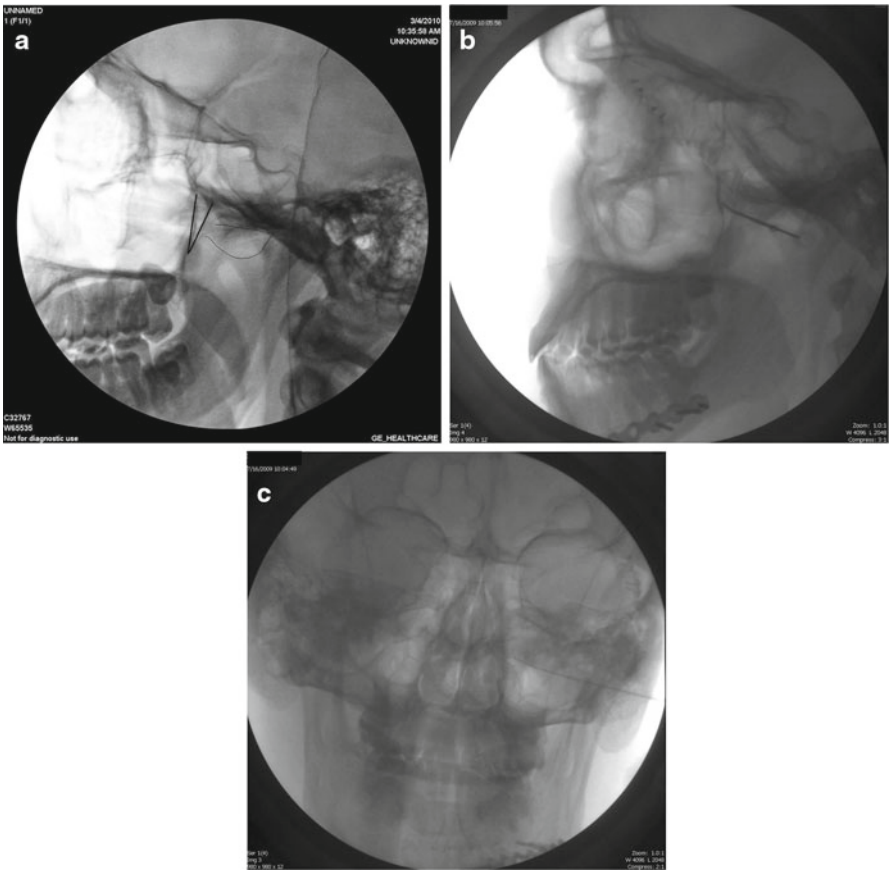

Fig. 25.1 (**a**) Lateral view for sphenopalatine ganglion block. The *dark lines* that form a vertical "V" indicate the pterygomaxillary fissure. The *lighter*, *slightly horizontal*, and *curved lines* outline the mandibular notch. (**b**) Lateral view. The tip of the needle is in the pterygopalatine fossa. (**c**) AP view. The tip of the needle is adjacent to the middle turbinate. The needle would not advance further than this point secondary to the facial trauma. The patient had bilateral face pain

at any point, withdraw and redirect the needle. Given the small size of the fossa, frequent AP and lateral images are may be required to redirect the needle.

- Once in the fossa, inject 0.5–1.0 ml nonionic, water-soluble contrast and observe for intravascular spread and/or intranasal placement of the needle.
- Once correct placement has been confirmed, inject 2 ml of local anesthetic (1–2% lidocaine, 0.25–0.50% bupivacaine, or 0.2–0.5% ropivacaine), with or without steroids.

Radiofrequency Thermocoagulation and Pulsed Radiofrequency

After a successful diagnostic block, two therapeutic choices are available: conventional radiofrequency thermocoagulation (RFTC) and pulsed electromagnetic field radiofrequency (P-EMF). An insulated RF needle with a 3- or 5-mm active tip is placed using the infrazygomatic approach.

- Once in place, sensory stimulation is performed at 50 Hz up to 1 V. If the tip of the needle is adjacent to the SPG, the patient should perceive a paresthesia at the root of the nose at less than 0.3 V. If the paresthesia is felt in the hard palate, the needle should be redirected cephalad and medial. A paresthesia in the upper teeth indicates stimulation of the maxillary nerve, and the needle should be more caudal and medial. Motor stimulation is not necessary.
- After appropriate sensory stimulation, RFTC can be performed at 67–80°C for 90 s times two cycles. Before lesioning, 2–3 ml of local anesthetic (1–2% lidocaine, 0.25–0.50% bupivacaine, or 0.2–0.5% ropivacaine) should be injected. To avoid inadvertent lesioning of other nerves around the SPG, a 3-mm active tip is a better choice.
- For P-EMF, the size of the active tip is not important as the electromagnetic field is projected from the tip of the needle and not from the shaft. With P-EMF lesioning, two to four 120-s lesions are performed at 45 V. Local anesthetic is not required for P-EMF.
- The choice of whether to do a RFTC or a P-EMF lesion after a successful block is up to the discretion of the pain management practitioner. The authors prefer P-EMF as it is a non-neurodestructive procedure.

Complications

Complications include bruising, bleeding, infection, damage to nerves, proptosis from retrobulbar hematoma, dysesthesias, paresthesias, and/or numbness from RFTC. Bradycardia (“Konen” reflex) has been described during RFTC and P-EMF and can be prevented with pretreatment with atropine or glycopyrolate [2].

Evidence

Day published an article on the current evidence for sympathetic blocks [3]. For the sphenopalatine ganglion block, ten articles (six case reports, three case series, and one retrospective review) were mentioned for the indications listed in Table 25.2. All were graded as 1C: strong recommendation and low-quality or very low-quality evidence. A case series on RFTC of the sphenopalatine ganglion for chronic cluster headaches by Narouze et al. also receives a 1C recommendation [4].

Stellate Ganglion Block

Indications

Indications for blockade of the stellate ganglion are listed in Table 25.3 [5–7].

Anatomy

- The stellate ganglion (SG) is the most inferior ganglion of the cervical sympathetic chain. It is most often formed when the inferior cervical ganglion fuses with the first thoracic ganglion forming an oval, dumbbell, or inverted "L" mass 2.5-cm long, 1-cm wide, and 0.5-cm thick [8–10]. In approximately 20% of the population the two ganglia remain separated [9].
- The SG is located in the posterior region of the superior thorax 5 mm anterolateral to bony structures [8, 11]. It is positioned anterior to and between the base of the seventh cervical transverse process and the neck of the first rib. In cases where fusion is absent, the inferior cervical portion is positioned anterior to the tubercle of the seventh cervical vertebra, while the first thoracic ganglion lies directly superior to the neck of rib number one [9].
- It is medial to the scalene muscles, anterolateral to the lateral border of the longus coli muscle, and lateral to the esophagus, trachea, and recurrent laryngeal nerve [8–10].
- The vertebral artery lies anterior to the ganglion as it originates from the subclavian artery [8]. Additional vascular landmarks include the common carotid artery which is located medially and the inferior thyroid artery which is anterior and lateral [8, 9].

Table 25.3 Indications for stellate ganglion block

Complex regional pain syndromes types I and II
Sympathetic maintained pain
Phantom limb pain
Postherpetic neuralgia
Angina pectoris
Arterial insufficiency
Hyperhidrosis
Meniere's disease
Chronic facial pain
Cervical pain
Atypical vascular type headaches
Hot flashes
Frostbite
Post traumatic stress disorder

- The postganglionic fibers exit the ganglion through the gray rami communicantes and provide sympathetic efferent impulses to the following [10]:
  - Cardiac plexus located posterior to the aortic arch.
  - Vascular smooth muscle of the subclavian arteries, vertebral arteries, and brachiocephalic trunk.
  - Skin sweat glands and erector pili muscles.

Procedure

There are several techniques for blockade of the stellate ganglion. This section will focus on two techniques: the anterior paratracheal approach at C6 and C7. Correct needle placement can be attained at each approach by different methods, palpating specific landmarks or using fluoroscopy. The authors prefer fluoroscopy as the landmarks are not easily palpable, and it provides a safer and more accurate method of performing these injections.

Patient Position

Place the patient in a supine position with the head flat on the table without a pillow and folded sheet under the shoulders for a slight neck extension. Allow a slight jaw opening to relax the skin and musculature tension over the targeted area in the neck. Sterilely prep and drape the neck region from the base of the chin to the upper sternum.

Anterior C6 Approach

- Palpating the anterior neck, identify the cricoid cartilage and palpate laterally with firm pressure until the C6 tubercle (Chassaignac's) is identified or identify using fluoroscopy.
- A 22-gauge, 1.5-in. B-bevel needle is advanced while the structures underneath are gently displaced posteriorly and lateral. Alternatively a 22- or 25-gauge, 3.5-in. Quincke needle can be used.
- The needle is advanced in a perpendicular plane until bony contact is made at the C6 transverse process.
- Once bony contact is attained, the needle is slightly withdrawn 0.5 cm to remove it from the longus coli muscle or periosteum (Fig. 25.2a).
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the C6 vertebral body (Fig. 25.2b). Failure of contrast spread caudad or cephalad typically suggests injection of contrast into the longus coli

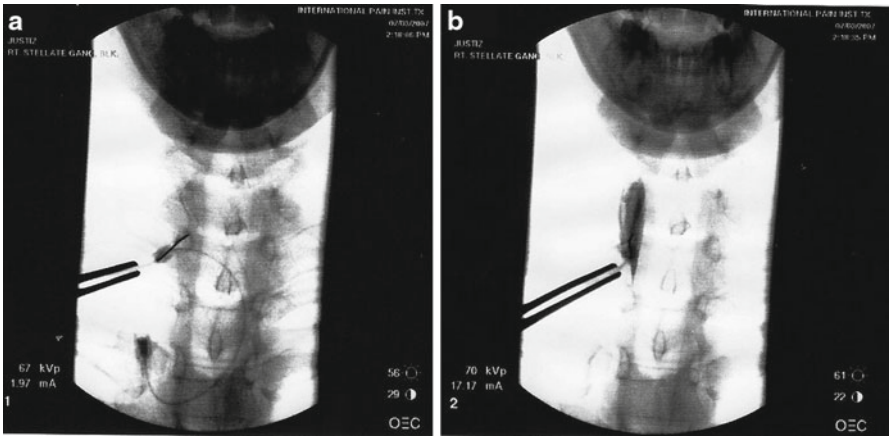

Fig. 25.2 (**a**) AP view of stellate ganglion block at the level of C_6. A Kelly clamp is holding a T-connector with extension tubing. (**b**) Contrast is spreading linearly cephalad and caudad

muscle, and instantaneous dissipation of contrast agent indicates intravascular placement. If this occurs, the needle needs to be repositioned.
- Obtain an AP and lateral image. The final position should be with the needle tip touching the C6 transverse process in both views.
- Once appropriate position is confirmed, inject 10 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration with or without steroids.
- A successful block is indicated by an increase in temperature of 1.5–3°C.
- Alternatively, this procedure can be performed blindly in the same steps as described above without the addition of radiocontrast agent.

Anterior C7 Approach

- Once the C6 tubercle (Chassaignac's) is identified, move one finger breadth inferiorly to locate your target, the C7 tubercle, or identify using fluoroscopy.
- Under fluoroscopy in an AP view, the junction of the C7 transverse process with the vertebral body is identified as the target.
- A 22-gauge, 1.5-in. B-bevel needle is advanced while the structures underneath are gently displaced posteriorly and lateral. Alternatively a 22- or 25-gauge, 3.5-in. Quincke needle can be used.
- The needle is advanced in a perpendicular plane until bony contact is made at the junction of the C7 transverse process and vertebral body.
- Skin is infiltrated with local anesthetic, and the needle is advanced in an AP view toward the junction of the C7 transverse process and the vertebral body while pushing the structures downward and laterally.

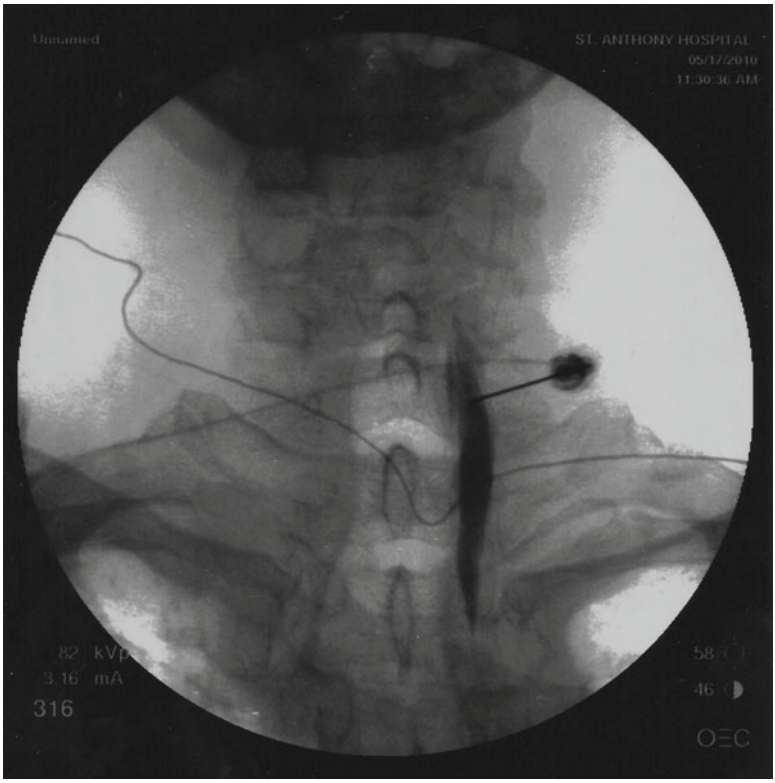

Fig. 25.3 Stellate ganglion block with contrast spread at the level of C_7. The contrast is extending caudally to the T_2 level

- The needle is advanced until bony contact is made.
- Correct needle position is confirmed with fluoroscopy in both the AP and lateral views with the needle tip at the junction of the C7 transverse process and the vertebral body.
- Once bony contact is attained the needle is slightly withdrawn 0.5 cm to remove it from the longus coli muscle or periosteum.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the C7–T1 plane in a superior and inferior direction (Fig. 25.3). Failure of contrast spread caudad or cephalad typically suggests injection of contrast into the longus coli muscle, and instantaneous dissipation of contrast agent indicates intravascular placement. If this occurs, the needle needs to be repositioned.

- Obtain an AP and lateral image. The final position should be with the needle tip touching the junction of the C7 transverse process and vertebral body in both views.
- Once appropriate position is confirmed, inject 5 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration with or without steroids.
- Alternatively, this procedure can be performed blindly in the same steps as described above without the addition of radiocontrast agent.

Radiofrequency Thermocoagulation [12]

For RFTC of the stellate ganglion, place the needles as in the C7 approach except for the following changes:

- Once the target is identified a 20- or 22-gauge, 5-mm active tip radiofrequency probe is advanced until bony contact is made at the junction of the transverse process and the vertebral body.
- Correct needle placement is confirmed by AP and lateral views followed by radiocontrast dye injection.
- This is followed by sensory and motor stimulation trials to ensure that the phrenic nerve and recurrent laryngeal nerve are not lesioned.
- Sensory stimulation is performed at 50 Hz and 0.9 V, while motor stimulation is done at 2 Hz and 2.0 V.
- During motor stimulation, the patient is asked to vocalize the vowels “aaa” and “eee” to ensure that the vocal cord function is not affected.
- This is followed by injection of 0.5 ml of local anesthetic for lesioning. Ropivacaine is preferred by the authors since it provides a greater sensory block than motor block. After waiting 2–3 min, radiofrequency current is applied for 60–90 s at 80–90° for one cycle.
- The needle is then redirected more medially in the same plane, and the same process is performed there for one cycle.
- Following the second lesion, the needle is again redirected medially and superiorly toward the upper junction of the transverse process and the vertebral body, and the final lesion is performed with the same parameters as before.

Chemical Neurolysis

The administration of a neurolytic agent can be performed via any approach desired. We will describe administration via the anterior C6 and C7 approaches.

- Utilizing the anterior approach at C6 or C7, the operator will perform the procedure in the same fashion.
- Once the needle is correctly placed, the operator should inject 2–3 ml of nonionic, water-soluble contrast under live fluoroscopy. This is to ensure that the

spread of the dye is around the vertebra and not intravascular, intrathecal, epidural, or along the longus coli muscle.
- If the dye spread is satisfactory, then a solution containing a mixture of local anesthetic, phenol, and steroid is injected. The total volume of 5 ml should consist of 2.5 ml of 6% phenol in saline, 1 ml of 40-mg triamcinolone, and 1.5 ml of 0.5% ropivacaine (the total 5-ml dose contains a final mixture of 3% phenol) [13].
- After the injection, the patient remains supine with the head elevated slightly for approximately 30 min to prevent complications.
- Patients should be advised of potential complications such as permanent Horner's syndrome or even recurrent laryngeal nerve paralysis.
- It has been advocated that a local anesthetic block prior to the neurolysis may help prevent these complications by observing the patient for 15–30 min, and if the patient develops the Horner's syndrome, the neurolytic block should not be performed. However, even the local anesthetic block cannot predict the outcome of the neurolytic injection. The patient should always be forewarned of the possibility of these complications before stellate ganglion block using the vertebral body approach.

Complications

The proximity of major vascular and neural structures can lead to potential complications as the needle is inserted and the medicines are injected. Complications include the block of neural structures such as the brachial plexus, vagus nerve, recurrent laryngeal nerve, and phrenic nerve. Intravascular injection leading to seizures and loss of consciousness is possible. Pneumothorax is another potential complication that can occur. Horner's syndrome (ptosis, myosis, and facial anhidrosis or enophthalmos) is another potential complication [14]. In reality, it is more of an expected side effect that occurs with blockade of the upper cervical sympathetics and will resolve within a couple of hours unless neurolysis is performed.

Evidence

According to Day, 11 articles were reviewed on the stellate ganglion block [3]. Ten articles (four case reports, five case series, and one retrospective review) received a 1C recommendation. The 11th article received a 1B recommendation: strong recommendation, moderate-quality evidence. A recent randomized controlled trial by Salviggio et al. [15] comparing stellate ganglion blocks versus oral medication for facial pain receives a grade of 1B.

T_2 and T_3 Sympathetic Block

Indications

Indications for blockade of the second and third sympathetic ganglia are listed in Table 25.4.

Anatomy

- The second thoracic (T_2) ganglion is commonly found at the head of the second rib near the costovertebral junction. It may also be slightly lateral within the intercostal space at the level of the intervertebral space near the upper border of the third rib [16, 17].
  - The majority of the ganglion is posterior to the parietal pleura; on the left side, it is within close approximation to the aortic arch [16].
- The third thoracic (T_3) ganglion is 17–20 mm dorsal to the ventral surface of the T_3 vertebral body and 2 mm rostral of vertebral bodies near the head of the third rib [18].
- The Kuntz nerve runs 2.3–15.7 mm lateral to the main body of the T_2 sympathetic ganglion [17]. Additional sympathetic connections are formed by the intrathoracic rami and rami communicantes found lateral, posterolateral, or posteromedial to the second and third ventral rami [19].
- Postganglionic fibers leave the sympathetic ganglion and enter the brachial plexus to provide sympathetic innervations to the distal regions of the upper extremity [19].

Procedure

This description will reflect the use of an introducer cannula and blunt, curved block needle. A sharp needle can be used as an alternative without the introducer.

Table 25.4 Indications for T_2 and T_3 sympathetic block

Complex regional pain syndrome type 1
Complex regional pain syndrome type 2
Sympathetically mediated pain of thorax, chest wall, thoracic and upper abdominal viscera
Hyperhidrosis
Ischemic pain
Herpes zoster
Postherpetic neuralgia

- Place the patient in the prone position. Attach appropriate monitors.
- Sterilely prep and drape the upper thoracic region from the base of the neck to the inferior aspect of the scapula.
- Identify the spinous process of the T_2 and angle the C-arm in the cephalocaudal (image intensifier cephalad) direction to square the superior and inferior end plates. This opens up the rib space to allow visualization of the lateral aspect of the vertebral body below the transverse process of T_2. Mark the skin over the spinous process.
- Oblique the C-arm to the appropriate side approximately 10–15°. The skin entry site is just lateral to the shadow of the bottom half of the vertebral body below the transverse process of T_2 and must not be greater than 4 cm lateral to the spinous process of T_2 viewed in the AP plane. This will theoretically decrease the chance of pneumothorax.
- Using a coaxial technique, anesthetize the skin and insert the introducer cannula. Using spot fluoroscopic images, advance the introducer until it is engaged in tissue.
- Obtain a lateral fluoroscopic image to check the depth of the introducer. Advance the introducer until it is just posterior to the T_2–T_3 foramen. Additional local anesthetic may be required during advancement of the introducer, but do not inject any more once the introducer reaches the foramen. Local anesthetic at this level may anesthetize the nerve root, and if a sharp needle is used, the patient may not respond should the needle pierce the nerve root.
- Return to the oblique image and check the direction of the needle. Maintaining coaxial technique with the curved tip pointing medially, advance the needle past the foramen until periosteum is touched (Fig. 25.4a).
- Recheck the depth of the needle with a lateral image. Rotate the needle tip cephalad and advance the needle. On a lateral image, the final needle tip position should be at the midpoint of the T_2 vertebral body in the cephalocaudal direction and at the junction of the middle and posterior thirds of the T_2 vertebral body in the anteroposterior direction (Fig. 25.4b).
- Obtain an AP image. The final position should be with the needle tip touching an imaginary line drawn through the midpoint of the T_2 pedicle shadow.
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the T_2 vertebral body and should not change position with respiration (Fig. 25.4c). If the location of the contrast ascends and descends with respiration, the needle tip is lateral and needs to be repositioned more medially.
- Once appropriate position is confirmed, inject 3–5 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary, but is not contraindicated.
- If there is caudal spread to T_3 ganglion, inject an additional 3–5 ml of the local anesthetic mixture. This will block the T_3 ganglion.
- If there is no caudal spread to T_3, block the T_3 ganglion using the same technique.

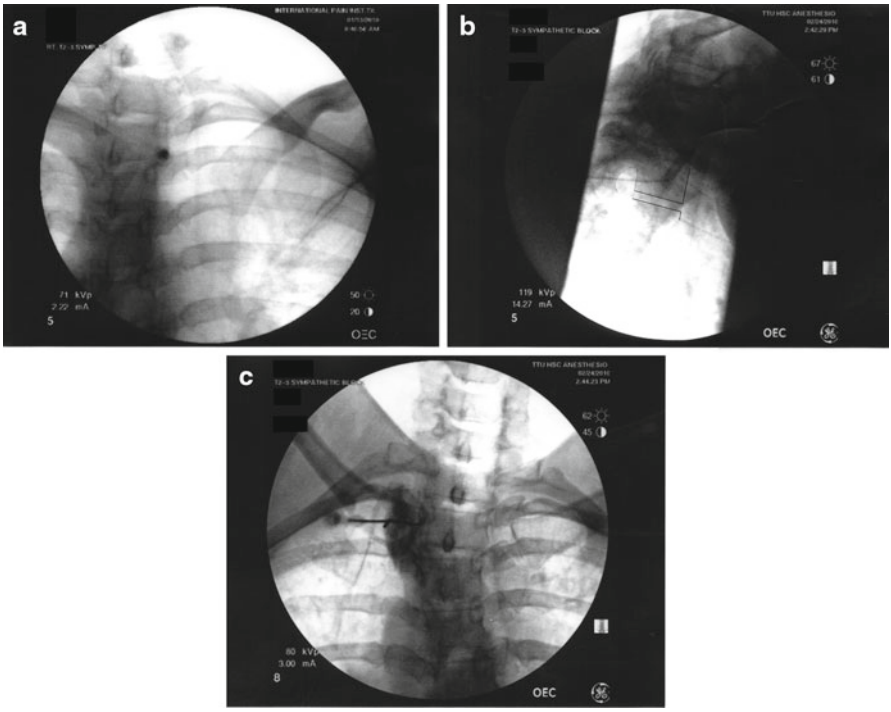

Fig. 25.4 (**a**) Oblique view for T_2 sympathetic block. This is a coaxial view with the needle adjacent to the inferior aspect of the T_2 vertebral body. (**b**) Lateral view with the needle tip at the midportion of the T_2 vertebral body. The vertebral bodies and the T_{2-3} foramen are outlined. (**c**) AP view of a thoracic sympathetic block. The needle tip is at the medial aspect of the pedicle shadow of T_2. Contrast is spreading linearly

Radiofrequency Thermocoagulation

For RFTC of the T_2 and T_3 ganglion, place the needles as above except for the following changes:

- Using a 10-mm active tip, the final needle tip position in the anteroposterior direction should be at the junction of the anterior and middle third of the vertebral body. This allows the active portion of the needle to span the middle third of the vertebral body. This will avoid potential lesioning of the ventral root of T_2 and T_3. The position in the cephalocaudal direction is the same as the block.
- Inject contrast under live fluoroscopy and observe for appropriate spread.
- Once the needle is in correct position, perform sensory (50 Hz up to 0.9 V) and motor (2 Hz up to 2 V) stimulation. No root stimulation should be perceived. The patient may feel a pressure deep in the chest with sensory stimulation. This is normal.

- If root stimulation is felt at a low voltage, the needle needs to be advanced 1–2 mm. Repeat sensory stimulation.
- After appropriate stimulation, inject 3 ml of the local anesthetic solution. Steroid can be included in the mixture to decrease the incidence of neuritis. Higher volumes may spread caudally and block the T_3 ganglion which will make stimulation at that level difficult. Wait 2–3 min and lesion at 80°C for 90 s. With a curved needle, two lesions are made, one in the cephalomedial direction and one in the caudomedial direction.

Complications

Complications include bruising, bleeding, infection, damage to nerves/spinal cord, neuraxial injection, and pneumothorax. This block should never be performed on a patient with a contralateral pneumothorax or pneumonectomy.

Evidence

There is a paucity of evidence for this block. Two articles for the percutaneous approach received 1C recommendations [3].

Splanchnic Nerves and Celiac Plexus Block

Indications

Indications for blockade of the splanchnic nerves and celiac plexus are listed in Table 25.5.

Contraindications

These procedures should not be performed on patients with partial or complete obstruction, or perforation of the small or large bowels. Blockade of the sympathetics will allow unopposed parasympathetic outflow, which will increase peristalsis and potentially convert a bowel obstruction into a perforation.

Table 25.5 Indications for splanchnic nerve and celiac plexus block

Cancer-related pain from the stomach to mid transverse colon, including the gall bladder, pancreas, spleen, testicle, kidney, and upper ureter
Non-malignant pain from the stomach to mid transverse colon, including the gall bladder, pancreas, spleen, testicle, kidney, and upper ureter

Anatomy

Splanchnic Nerves

- The *greater splanchnic nerve* is the most rostral, originating around the level of the fifth through ninth thoracic vertebrae. It originates from the four roots of the thoracic sympathetic ganglia, descends obliquely, pierces the crus of the diaphragm at a 90° angle, and joins to the celiac plexus [20–22].
- The *lesser splanchnic nerve* is formed by the rami of the ninth through 11th thoracic sympathetic ganglia. After exiting the ganglia, it travels inferomedial to the vertebral bodies at this level, passes through the crus of the diaphragm, and terminates at the celiac plexus [20, 22].
- The *least splanchnic nerve* is the most caudal, originating from the 12th thoracic sympathetic ganglion. It travels medially across the vertebral body, passes through the crus of the diaphragm, and terminates at the celiac plexus. Interestingly, this nerve was found to be absent in 43% of dissections, bilaterally [20, 22].
- Individually, each of these nerves contains preganglionic visceral sympathetic efferent neurons from the thoracic sympathetic chain plus postganglionic visceral afferent neurons that provide pain sensation from the upper abdominal organs to the central nervous system [21, 22].

Celiac Plexus

- This large network of nerves, including a right and left ganglia, is a region where pre- and postganglionic neurons from the sympathetic, parasympathetic, and visceral sensory divisions synapse [21, 22].
- This large star- or oval-shaped plexus is 0.5–4.5 cm in diameter and spans a region from the 12th thoracic vertebra disc space to the middle vertebral body of the second lumbar vertebra [22].
- The main body lays anterolateral to the aorta but also surrounds the celiac artery and superior mesenteric artery [22].
- Afferent fibers concerned with nociception pass diffusely through the celiac plexus and represent the main target of celiac plexus blockade.

Procedure

The techniques for blockade and neurolysis of the splanchnic nerves and the celiac plexus will be described separately. Pretreat the patient with 500 ml of normal saline or lactated Ringer's solution to decrease the incidence of post-procedure postural hypotension from dilation of the abdominal vasculature.

Splanchnic Nerve Block

This is a retrocrural block and involves blocking the greater, lesser, and least splanchnic nerves at the level of the T_{11} vertebra. Depending on the spread of the contrast caudally, a supplemental block may be needed at the level of the T_{12} vertebra in order to block the least splanchnic nerve.

- Identify the T_{11} vertebra and square the superior and inferior endplates with a caudocephalad tilt of the C-arm.
- Identify the spinous process in the midline and mark.
- Oblique the C-arm to the appropriate side approximately 10–15°. The skin entry site is just lateral to the shadow of the bottom half of the vertebral body below the transverse process of T_{11} and must not be greater than 4 cm lateral to the spinous process of T_{11} viewed in the AP plane. This will theoretically decrease the chance of pneumothorax.
- Using a coaxial technique, anesthetize the skin and insert the introducer cannula. Using spot fluoroscopic images, advance the introducer until it is engaged in tissue.
- Obtain a lateral fluoroscopic image to check the depth of the introducer. Advance the introducer until it is just posterior to the T_{11}–T_{12} foramen. Additional local anesthetic may be required during advancement of the introducer, but do not inject any more once the introducer reaches the foramen. Local anesthetic at this level may anesthetize the nerve root, and if a sharp needle is used, the patient may not respond should the needle pierce the nerve root.
- Return to the oblique image and check the direction of the needle. Maintaining coaxial technique with the curved tip pointing medially, advance the needle past the foramen until periosteum is touched (Fig. 25.5a).
- Recheck the depth of the needle with a lateral image. Rotate the needle tip cephalad and advance the needle. On a lateral image, the final needle tip position should be at the midpoint of the T_{11} vertebral body in the cephalocaudal direction and at the junction of the anterior and middle third of the T_{11} vertebral body in the anteroposterior direction (Fig. 25.5b).
- Obtain an AP image. The final needle tip position should be medial to the medial aspect of the T_{11} pedicle shadow (Fig. 25.5c).
- After negative aspiration for air, blood, and CSF, inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the T_{11} vertebral body and should not change position with respiration (Fig. 25.5d, e). If the location of the contrast ascends and descends with respiration, the needle tip is lateral and needs to be repositioned more medially.
- Once appropriate position is confirmed, inject 5 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.
- If there is caudal spread to T_{12} vertebral body, inject an additional 5 ml of the local anesthetic mixture. This will block the least splanchnic nerve.
- If there is no caudal spread to T_{12} vertebral body, block the least splanchnic nerve at T_{12} vertebral body using the same technique.

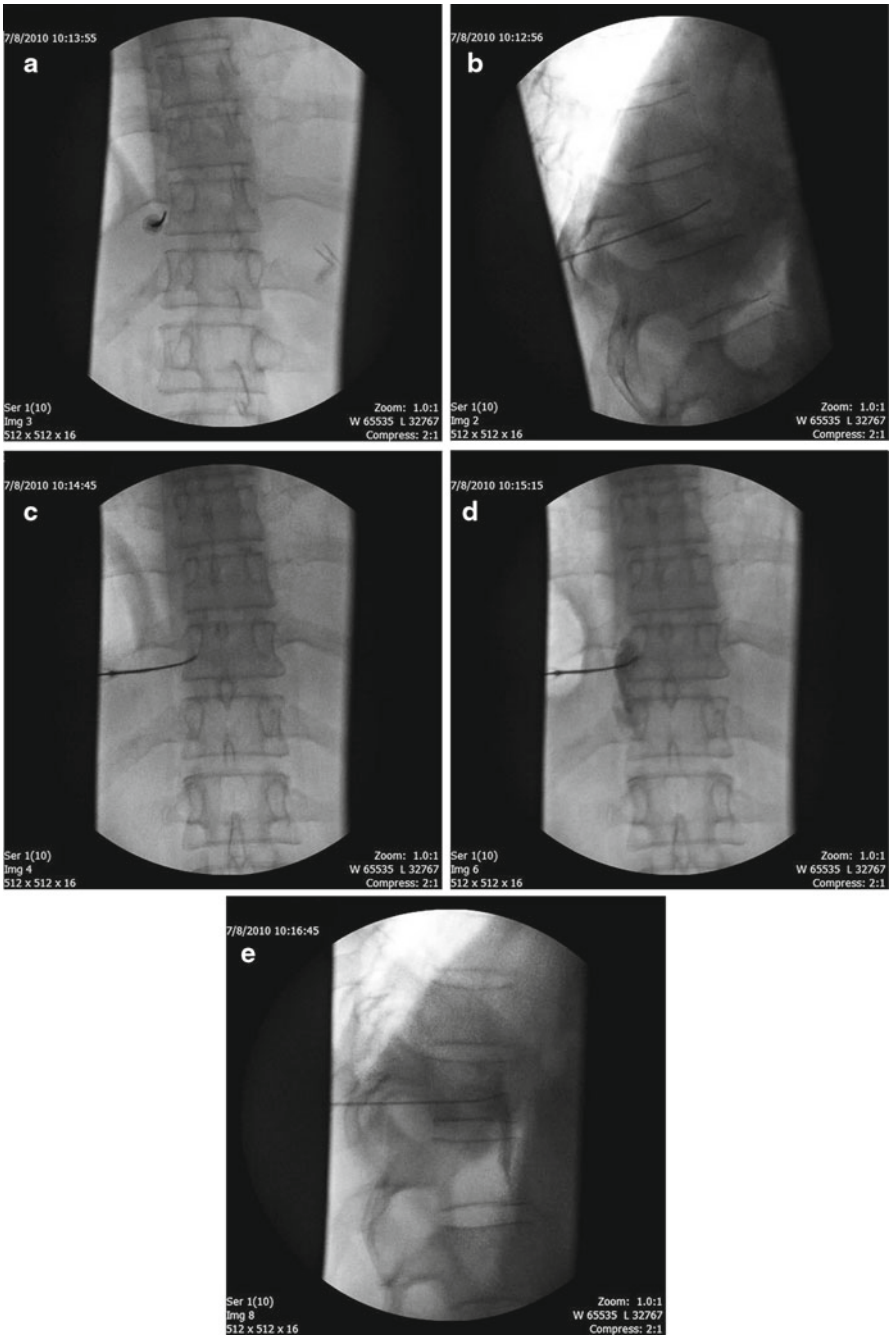

Fig. 25.5 (**a**) Oblique view for splanchnic nerve block. This is a coaxial view with the needle adjacent to the inferior aspect of the T_{11} vertebral body. (**b**) Lateral view with the needle tip at the junction of the anterior and middle thirds of the T_{11} vertebral body. (**c**) AP view at the T_{11} vertebral body. (**d**) AP view with contrast overlying the T_{11} vertebral body with some spread to T_{12}. (**e**) Lateral view at T_{11}. Contrast can be seen along the anterolateral aspect of the T_{11} vertebral body with linear spread to T_{12}

Radiofrequency Thermocoagulation

For RFTC of the greater, lesser, and least splanchnic nerves, place the needles at T_{11} and T_{12} except for the following changes:

- Using a 10-mm active tip, the final needle tip position in the anteroposterior direction should be across the junction of the anterior and middle third of the vertebral body. This will avoid potential lesioning of the ventral root of T_{11} and T_{12}. The position in the cephalocaudal direction is the same as the block.
- Inject contrast under live fluoroscopy and observe for appropriate spread.
- Once the needle is in correct position, perform sensory (50 Hz up to 0.9 V) and motor (2 Hz up to 2 V) stimulation. No root stimulation should be perceived. The patient may feel a pressure, deep in the abdomen with sensory stimulation. This is normal.
- If root stimulation is felt at a low voltage, the needle needs to be advanced 1–2 mm. Repeat sensory stimulation.
- After appropriate stimulation, inject 5 ml of the local anesthetic solution. Steroid can be included in the mixture to decrease the incidence of neuritis. Higher volumes may spread caudally and block the least splanchnic nerve which will make stimulation at that level difficult. Wait 2–3 min and lesion at 80°C for 90 s. With a curved needle, two lesions are made, one in the cephalomedial direction and one in the caudomedial direction.

Celiac Plexus Block

There are several approaches used to block the celiac plexus. Prone approaches include retrocrural, anterocrural, and transaortic. The retrocrural and anterocrural approaches require bilateral needles, while the transaortic approach requires only one needle. There is also an anterior approach using CT guidance, which will not be described.

Retrocrural Approach

- Place the patient in the prone position. After sterile prep and drape, identify the tip of the 12th rib on an AP fluoroscopic image and raise a skin wheal with local anesthetic.
- Insert the introducer cannula and angle toward the lower third of the L_1 vertebral body. If a sharp needle is used, this step is omitted.
- A 15-cm, 22- or 20-gauge curved, blunt block needle is inserted and advanced until the lower third of the L_1 vertebral body is touched. Check the position on a lateral image to make sure the needle is not entering the L_1–L_2 foramen. If not, inject 2–3 ml of local anesthetic.

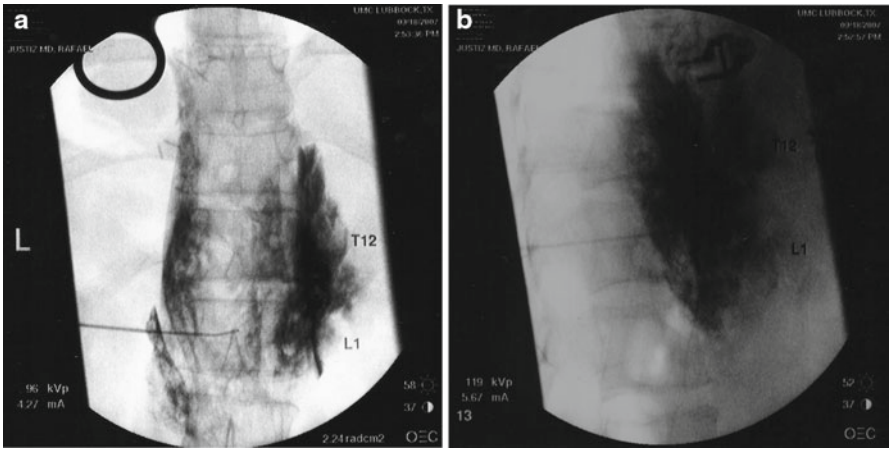

Fig. 25.6 (**a**) AP view of bilateral celiac plexus block. Contrast is spreading from T_{11} to L_1. (**b**) Lateral view with contrast anterior to the vertebral bodies

- Withdraw the needle slightly and using a steepened angle, advance along the lateral border of the L_1 vertebral body until the needle slips off of the anterior border.
- Using the same technique, insert another needle on the opposite side.
- On a lateral image, advance the right needle 2 cm and the left needle 1 cm past the anterior border of the L_1 vertebral body until aortic pulsations are felt.
- Check the needle position on an AP image. The right needle should be more toward the midline than the left needle as it is more anterior.
- After negative aspiration for blood, urine, and cerebrospinal fluid, inject 2–3 ml of a nonionic, water-soluble contrast through each needle. On the AP image, the contrast should be off of midline over the L_1 vertebral body (Fig. 25.6a). The contrast should spread in a linear fashion along the L1 vertebral body on a lateral image (Fig. 25.6b).
- Using 1–2% lidocaine, 0.2% ropivacaine, or 0.25% bupivacaine, inject a 2-ml test dose through each needle to rule out intravascular or intrathecal injection.
- After a negative test dose, 10–15 ml of the aforementioned local anesthetic is injected through each needle.

Transaortic Approach

This is a single needle approach from the left side. It is best to use a B-bevel needle such as a 20- or 22-gauge Chiba.

- Place the left-sided needle 1 cm past the L_1 vertebral body until aortic pulsations are felt as described in the retrocrural approach.
- Remove the stylet of the needle and swiftly advance it through the posterior wall, lumen, and anterior wall of the aorta. The needle tip will be in the preaortic area

where the celiac plexus lies. To decrease bleeding, a loss of resistance syringe filled with 5 ml of preservative-free normal saline can be attached to the needle and advanced using loss of resistance technique until the preaortic area is entered.
- Check needle tip position with AP and lateral fluoroscopic images. Inject 3–5 ml of nonionic, water-soluble contrast after negative aspiration. It should fill the periaortic area and should not spread retrocrural.
- Once needle tip position is confirmed, inject 10–15 ml of the aforementioned local anesthetics after negative aspiration for blood.

Neurolytic Celiac Plexus Block

After a positive diagnostic block with local anesthetic, a neurolytic block can be performed. Phenol in 6–10% concentrations and anhydrous alcohol in 50–100% concentrations have been used. A total volume of 30–40 ml (15–20 ml on each side) is common for a two-needle technique. The transaortic approach only requires 15 ml of either neurolytic. The injection of alcohol is painful and requires the injection of 5–10 ml of 0.2% ropivacaine or 0.25% bupivacaine prior to its injection. Prior to removal of the needle/s, 2–3 ml of preservative-free normal saline should be used to flush the needle/s. Failure to flush the needle/s could result in tissue sloughing along the tract of the needle.

Side Effects and Complications

The difference between a side effect of the procedure and a complication of the procedure must be explained to the patient beforehand. Side effects secondary to sympathetic blockade include hypotension from dilation of the abdominal vasculature and diarrhea from unopposed parasympathetic outflow. Complications include backache, bruising, bleeding (superficial and retroperitoneal), infection, damage to nerves/spinal cord, neuraxial injection, paralysis, pneumothorax, and tissue sloughing from the use of neurolytics.

Evidence

The evidence is strong for use of the splanchnic nerve/celiac plexus block/neurolysis for cancer-related pain. Fifteen articles were identified consisting of: one case report; six case series; two retrospective reviews; two prospective, randomized, controlled studies; one randomized, double-blinded study; one prospective, randomized, double-blinded, controlled study; one prospective, randomized, single-blinded, controlled study; and two meta-analysis [3]. The case report, case series, and retrospective reviews received 1C recommendations while the rest of the articles received 1B recommendations.

Lumbar Sympathetic Block

Indications

Indications for a lumbar sympathetic block are listed in Table 25.6 [23].

Anatomy

- Preganglionic afferent neurons synapse to autonomic afferent fibers which innervate the lower extremity and give off visceral branches to the lumbar splanchnic nerves.
- There is considerable variation in size, number, and position on the vertebral bodies of the ganglia. On average this chain contains 3–4 ganglia and are usually located between the second and fourth lumbar vertebra [24–26].
- The ganglia are cylindrical or elliptical in shape and are intertwined within the fascicles of the medial border of the psoas major muscle [27–29].
- In the horizontal plane, the ganglia reside 0–0.5 cm posterior to the anterior border and 1.8–3.0 cm laterally from the center of the third lumbar vertebra [27].
- On the right side of the vertebral column, the chain lies posterior to the inferior vena cava, while on the left side, it is posterior to the para-aortic nodes. As the chain continues caudally, it passes posterior to the common iliac artery [29].

Procedure

Traditionally, two approaches have been described for the lumbar sympathetic block: the classic approach described by Kappis and Mandal and the lateral approach described by Mandal and Reed. With the advent of fluoroscopy, there is now a new

Table 25.6 Indications for lumbar sympathetic block

Complex regional pain syndromes types I and II
Sympathetic maintained pain
Phantom limb pain
Diabetic gangrene
Phlegmasia
Arterial insufficiency
Hyperhidrosis
Alba Dolens
Erythromelalgia
Acrocyanosis
Intractable urogenital pain
Trenchfoot
Frostbite

so-called modern approach. Secondary to its safety and ease of performing, we will describe the modern (oblique-view) approach. This description will reflect the use of an introducer cannula and blunt, curved block needle. A sharp needle can be used as an alternative without the introducer.

Modern Approach

- Place the patient in the prone position. Attach appropriate monitors.
- Sterilely prep and drape the lumbar region.
- Identify the vertebral body of interest in an AP view and square off the vertebral end plates. For a single needle approach, L_3 is usually the target, but if the pain is located in the lower leg or foot, L_4 or sometimes L_5 may need to be the targeted level.
- Oblique the C-arm to the appropriate side approximately until the transverse process is situated just lateral to the vertebral body.
- Once the target is identified, a small skin wheal with local anesthetic is raised.
- Using a coaxial technique, anesthetize the skin and insert the introducer cannula. Using spot fluoroscopic images, advance the introducer until it is engaged in tissue.
- Obtain a lateral fluoroscopic image to check the depth of the introducer. Advance the introducer until it is just posterior to the vertebral body foramen. Additional local anesthetic may be required during advancement of the introducer.
- Return to the oblique image and place the blunt, curved block needle. Maintaining coaxial technique with the curved tip pointing medially, advance the needle past the foramen until periosteum is touched.
- Recheck the depth of the needle with a lateral image. Rotate the needle tip medial or lateral and advance the needle. On a lateral image, the final needle tip position should be at the anterior vertebral body.
- Obtain an AP image. The final position should be with the needle tip touching an imaginary line drawn through the medial aspect of the pedicle shadow at that level.
- After negative aspiration for air, blood, urine, and CSF, inject 2–5 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a linear fashion along the vertebral body (Figs. 25.7a and 25.8a). On lateral view, the solution should spread caudad and cephalad anterior to the vertebral body (Figs. 25.7b and 25.8b). On an AP image, the contrast should appear to spread medially, cephalad, and caudad directions while hugging the vertebral body. If the needle tip is too lateral, contrast may spread into the origins of the psoas muscle and the needle will need to be repositioned more medially.
- Once appropriate position is confirmed, inject 10–20 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration [30]. Steroid is not necessary, but is not contraindicated.

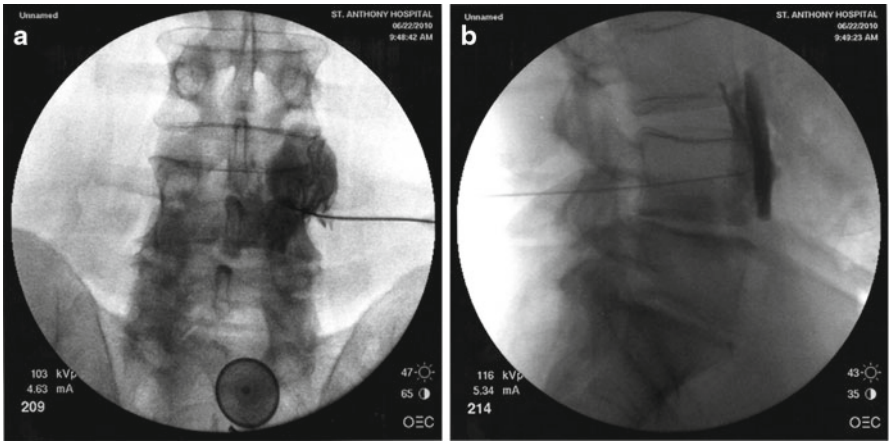

Fig. 25.7 (**a**) AP view of a lumbar sympathetic block at L_4. The tip of the needle is at medial aspect of the L_4 pedicle shadow. (**b**) Lateral view. The contrast is spreading linearly. There appears to be a small amount of contrast spread in the psoas muscle at the level of the $L_{3–4}$ disc

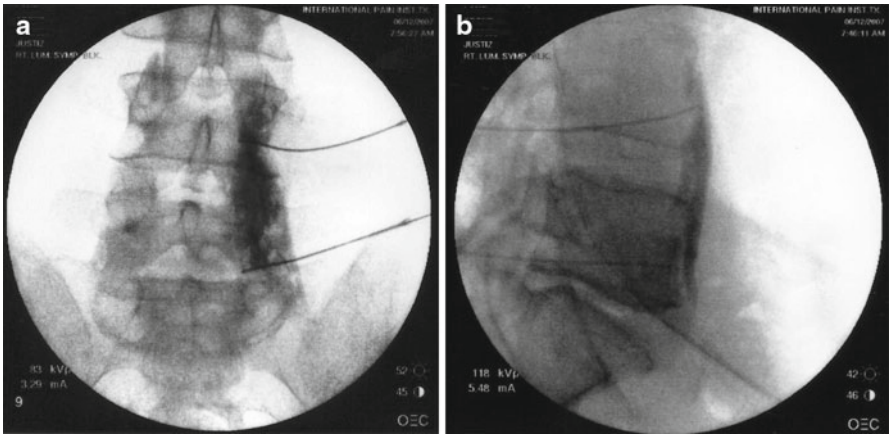

Fig. 25.8 (**a**) AP view of a two-level lumbar sympathetic block at L_4 and L_5. (**b**) Lateral view

Radiofrequency Thermocoagulation

For RFTC of the lumbar sympathetic chain, place the needles as described above using the modern approach except for the following changes. The needle position will vary at each independent vertebral level with the location of the appropriate ganglions. Lesion will be performed at the inferior one third of the L_2 vertebral body, upper one third of the L_3 vertebral body, and middle of the L_4 vertebral body [26, 27, 31–33].

- Using a 10-mm active tip on a curved, blunt electrode, the final needle tip position in the lateral fluoroscopic view should be at the anterior portion of each of the

respective vertebral bodies intended to be blocked. In an AP image, the needle tips should lie on an imaginary line at or near the medial aspect of the pedicle.
- Inject 2 ml of contrast under live fluoroscopy and observe for appropriate spread.
- Once the needle is in correct position, perform sensory (50 Hz up to 1.0 V) and motor (2 Hz up to 2 V) stimulation.
- With sensory stimulation, the patient may feel a pressure or discomfort in the lumbar region with 0.2–0.5 V; this is normal. If paresthesia is elicited in the groin region, the needle must be repositioned as it is situated too close to the genitofemoral nerve.
- Motor stimulation should not elicit any motor response in the lower extremities.
- 1–2 ml of local anesthetic (lidocaine 2%) is given; wait 2–3 min before lesioning.
- With a curved needle, two lesions are made, one in the cephalomedial direction and one in the caudomedial direction. Rotate the electrode tip cephalad and medial direction and lesion for 60–90 s at 80–90° for one cycle. Then, rotate the electrode tip caudal and medial direction and perform another cycle for 60–90 s at 80–90°.
- Lesions should be performed at the inferior one third of the L_2 vertebral body, the superior one third of the L_3 vertebral body, and the middle of the L_4 level vertebral body.

Neurolytic Lumbar Sympathetic Plexus Block

Chemical neurolysis can be accomplished in a similar fashion to the local anesthetic block with same-needle placement technique. Needle placement is confirmed with nonionic, water-soluble contrast prior to injection of the neurolytic. This is followed by 2–3 ml of 6–12% phenol per level when using multiple-needle technique or a larger volume of 15–20 ml through a single needle. Prior to removing the needle/s, 2–3 ml of preservative-free normal saline should be used to flush the needle/s to prevent tracking of the neurolytic agent [34, 35].

Complications

Like many procedures performed, a host of complications can occur. With proper and safe technique, many of these complications can be avoided. The most common complications will be post-procedural discomfort in the lumbar region for 3–5 days, retrograde ejaculation (bilateral sympathectomy), genitofemoral neuralgia [36], kidney or ureteral damage [37], and intravascular and subarachnoid injections.

Evidence

Continuing with Day's evidence-based article, 11 papers were graded. Four case reports and five case series earned 1C recommendations [3]. The remaining two articles, one a prospective randomized trial and the other a prospective randomized controlled trial, received 1B recommendations.

Superior Hypogastric Plexus Block

Indications

Indications for a superior hypogastric plexus block are listed in Table 25.7.

Anatomy

- These plexuses contain efferent pre- and postganglionic sympathetic, preganglionic parasympathetic, and afferent visceral sensory nerve fibers.
- The *superior hypogastric plexus* is embedded in connective tissue anterior to the midbody of the fifth lumbar vertebra and sacral promontory, positioned anterior to the aortic bifurcation, left common iliac vein, and medial sacral vessels [38]. It is in close proximity to the roof of the sigmoid colon mesentery with the attachment point of the mesocolon left of the plexus [39]. The plexus then branches to form the left and right hypogastric nerves. Postganglionic sympathetic and parasympathetic fibers innervate the pelvic organs as they exit the superior plexus.
- The *inferior hypogastric plexus* forms a triangular structure with the following landmarks: (1) the cephalad edge runs parallel to the hypogastric artery, (2) the caudal edge stretches from the fourth sacral root to the ureter entry point at the broad ligament, and (3) the dorsal edge runs along the ventral surface of the sacrum close to the S_2–S_4 nerve roots [40]. Postganglionic sympathetic, parasympathetic, and visceral sensory nerves provide innervations to the bladder, prostate, penis, uterus, vagina, and rectum [40, 41].

Procedure

As with the other sympathetic blocks, there are several approaches for blocking the superior hypogastric plexus: traditional, medial, and transdiscal. The trans-sacral foramina approach actually blocks the inferior hypogastric plexus and will not be described. The hypogastric plexus block needs to be performed with fluoroscopic or CT guidance. Only fluoroscopic techniques will be described in this chapter. A 15-cm, 20- or 22-gauge, curved, sharp or blunt needle is used. The patient is placed in the prone position with pillows placed under the lower abdomen to reverse the lumbar lordosis.

Table 25.7 Indications for superior hypogastric plexus block

Cancer related pain in the bladder, vagina, penis, rectum, anus, and perineum
Pain related to non-malignant conditions such as endometriosis, pelvic adhesions, pelvic inflammation, interstitial cystitis, irritable bowel syndrome, proctalgia fugax, vulvodynia

Traditional Two-Needle Approach

- Sterilely prep and drape the lower lumbar and sacral region.
- Identify the L_4–L_5 interspace and tilt the C-arm in the cephalocaudad direction to square the inferior end plate of L_4 and the superior endplate of L_5.
- Raise a skin wheal with local anesthetic approximately 5–7 cm lateral to the L_4–L_5 interspace.
- Insert an introducer cannula through the skin wheal, angling 30–45° medially and caudally. If using a sharp needle, this step is omitted.
- Insert the block needle through the cannula and advance toward the inferior, anterolateral aspect of the L_5 vertebral body. Check the depth of the needle with a lateral image.
- Adjust the angle of the needle until the tip walks off the anterior edge of the L_5–S_1 interspace. The transverse process of L_5 may sometimes be encountered and requires the initial angle of the needle to be steeper.
- On an AP image, the needle tip should be medial to an imaginary line drawn through the medial aspect of the lumbar pedicle shadows and extending caudally through the sacrum.
- Repeat the procedure on the opposite side using the same technique.
- Under continuous lateral fluoroscopy and after negative aspiration for blood and CSF, inject 2 ml of nonionic, water-soluble contrast through each needle. The contrast should spread caudally in a curvilinear fashion over the anterior aspect of the L_5–S_1 disc and sacral promontory. The AP view should show contrast over the upper portion of the sacrum extending caudally.
- The block is performed with 8–10 ml of 1–2% lidocaine or 0.2% ropivacaine or 0.25% bupivacaine.

Medial Two-Needle Approach

This approach is very similar to blockade of the L5 sympathetic ganglion, except the target is the inferior aspect of the L5 vertebral body at the L_5–S_1 disc. This technique can be used if the practitioner attempts the traditional approach and has difficulty maneuvering past the L_5 transverse process.

- Square the inferior end plate of L_5 and the superior aspect of the sacrum.
- Oblique the C-arm ipsilaterally, stopping just before the shadow of the inferior, lateral aspect of the L_5 vertebral body overlaps the iliac crest.
- Raise a skin wheal with local anesthetic over the inferior, lateral aspect of L_5.
- Insert the introducer cannula in a coaxial fashion and check the depth with a lateral image.
- Insert the curved, blunt block needle through the introducer.
- Return to the oblique view and advance the block needle checking its direction with spot images.
- Once bone is touched, turn the tip caudally and advance the needle on a lateral image.

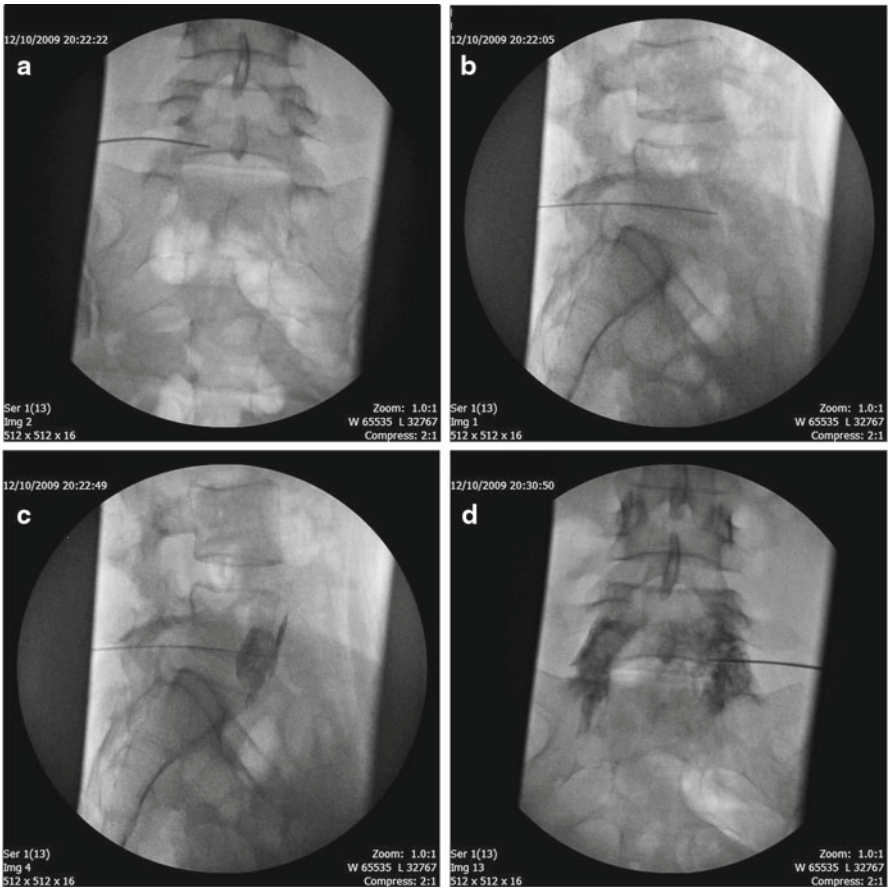

Fig. 25.9 (**a**) AP view of a medial approach to the hypogastric plexus. The needle is at the caudal aspect of L_5. (**b**) Lateral view. The tip of the needle is just anterior to the L5 vertebral body. (**c**) Contrast is spreading cephalad and caudal over the sacral promontory. (**d**) AP view with contrast spreading caudally

- As the needle is advanced, turn the tip medially to confirm that the needle is still on bone. Advance the needle until the tip is just past the inferior, anterolateral edge of the L_5 vertebral body (Fig. 25.9a, b).
- Repeat the procedure on the opposite side using the same technique.
- Under continuous lateral fluoroscopy and after negative aspiration for blood and CSF, inject 2–3 ml of nonionic, water-soluble contrast through each needle. The contrast should spread caudally in a curvilinear fashion over the L_5–S_1 disc and sacral promontory (Fig. 25.9c). The AP view should show contrast over the upper portion of the sacrum extending caudally (Fig. 25.9d).
- The block is performed with 8–10 ml of 1–2% lidocaine or 0.2% ropivacaine or 0.25% bupivacaine.

Transdiscal Approach

This is a one-sided (left) approach. A double-needle technique is used.

- Square the inferior end plate of L_5 and the superior aspect of the sacrum with a cephalocaudal tilt of the C-arm.
- Oblique the C-arm toward the left until an inverted triangle is created with the shadows of the superior end plate of L5, the lateral aspect of the superior articular process of the sacrum, and the iliac crest.
- Raise a skin wheal over the shadow of the lateral aspect of the superior articular process of the sacrum.
- Insert the introducer cannula in a coaxial fashion at the midpoint of the lateral aspect of the superior articular process of the sacrum.
- Insert the curved, sharp or blunt block needle through the introducer cannula and advance in a coaxial fashion toward the disc using spot images.
- Check a lateral image and advance until the tip of the needle is posterior to the L_5–S_1 foramen.
- Return to the oblique view and check to make sure the needle is coaxial. If not, withdraw the needle slightly and redirect medial.
- On a lateral view, advance the needle into and through the disc until the needle tip just exits the anterior portion of the disc.
- Check an AP image. The needle tip should be in the same position as described in the aforementioned techniques.
- Inject contrast and observe for proper spread. The block is performed with 8–10 ml of 1–2% lidocaine or 0.2% ropivacaine or 0.25% bupivacaine.
- As the needle is withdrawn, inject antibiotic (cephazolin is common) into the disc in order to avoid discitis.

Neurolytic Hypogastric Plexus Block

Chemical neurolysis can be accomplished with 5–8 ml of 6–10% phenol or 50–100% anhydrous alcohol. Confirm proper needle placement with nonionic, water-soluble contrast prior to the injection of either neurolytic. Prior to removal of the needle/s, 2–3 ml of preservative-free normal saline should be used to flush the needle/s. Failure to flush the needle/s could result in tissue sloughing along the tract of the needle.

Complications

The proximity of the iliac vessels increases the potential for vessel puncture or intravascular injection. Other complications include backache, bruising, bleeding (superficial and retroperitoneal), infection, damage to nerve roots, neuraxial injection, paralysis, discitis, rectal puncture, and tissue sloughing from the use of neurolytics.

Evidence

Six articles were identified for pelvic cancer pain [3]. One case report and five case series were graded as 1C, and a prospective randomized trial earned the grade of 1B. There were three case reports and one case series for noncancer-related pelvic pain and all were graded as 1C.

Ganglion Impar Block (Ganglion of Walther)

Indications

The indications for blockade of the ganglion impar block are listed in Table 25.8 [42–46].

Anatomy

- The ganglion impar receives preganglionic fibers from the sacral portion of the sympathetic trunk while postganglionic fibers carry sympathetic outflow to the perineum via the pudendal nerve [47]. Additional innervations by the parasympathetic and visceral sensory nervous systems have been reported.
- It is a single midline structure located anterior to the level of the sacrococcygeal junction, or in some cases, it can be more caudal at the midpoint of the coccyx [47–49].
- The ganglion can be oval, irregular, triangular, or rectangular with a mean length of 0.7–4.4 mm depending on the shape [48].
- In a cadaveric study with an $n = 50$, 18% of ganglia were located at the sacrococcygeal junction, while 46% of the ganglia were located from 20 to 30 mm from the tip of the coccyx [48].

Table 25.8 Indications for ganglion impar block

Perineal pain
Pain secondary to endometriosis
Phantom limb pain
Complex regional pain syndromes types I and II
Proctalgia fugax
Radiation enteritis
Rectal pain
Pelvic pain
Genital pain

Procedure

There are several techniques for blockade of the ganglion impar. This section will focus on two techniques with various approaches: the lateral and prone.

Lateral Technique [50]

- Patient is placed in the lateral decubitus with knees flexed toward the chest.
- C-Arm is placed in an AP direction to the table and rotated accordingly until the sacrum is visualized in a true lateral view. The target will be located at the fifth sacral vertebrae just cephalad to the sacrococcygeal junction.
- A 22-gauge, 3.5-in. spinal needle with a small bend distally is advanced toward the sacral-coccyx junction midline and toward the sacrum to avoid the posterior rectal wall.
- AP view is taken to ensure the needle tip is midline. After negative aspiration for blood, inject 2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a cephalad and caudal fashion along the S_5 vertebral body in the precoccygeal space.
- Once appropriate position is confirmed, inject 4–6 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.
- This technique is useful when there is calcification of the sacrococcygeal ligament.

Prone Technique

Trans-Sacrococcygeal Junction Approach [51, 52]

- Patient is placed in the prone position, and an AP view is used to visualize the sacrococcygeal junction.
- A 22-gauge, 3.5-in. spinal needle is advanced in an AP view in coaxial fashion through the sacrococcygeal ligament junction (Fig. 25.10a).
- A lateral view is obtained and the needle position should lie just past the sacrococcygeal ligament (Fig. 25.10b).
- After negative aspiration for blood, inject 2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a cephalad and caudal fashion along the S_5 vertebral body in the precoccygeal space (Fig. 25.10c, d).
- Once appropriate position is confirmed, inject 4–6 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.

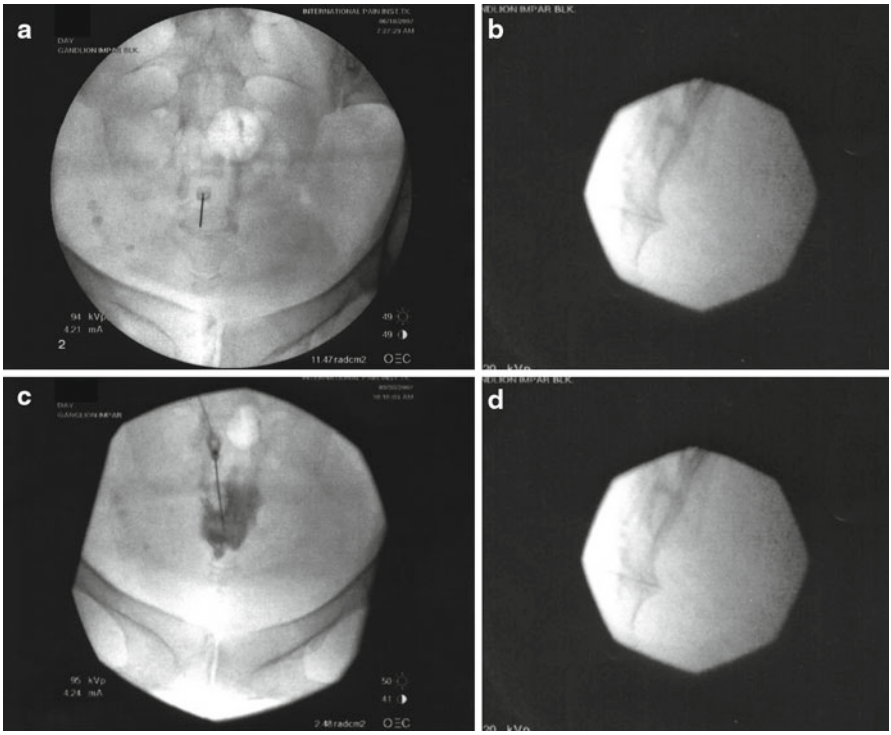

Fig. 25.10 (**a**) AP view of transacrococcygeal ganglion impar block. The needle is passing through the sacrococcygeal junction. (**b**) Lateral view with the tip of the needle just past the sacrococcygeal junction. (**c**) AP view with contrast. (**d**) Lateral view with contrast. The contrast is spreading just anterior the ventral surface of the sacrum

Paramedian Approach [53, 54]

- Patient is placed in the prone position, and an AP view is used to visualize the sacrococcygeal junction and the coccyx.
- A 22-gauge, 3.5-in. spinal needle with a small bend (20–30°) distally or multiple bends is placed inferior to the sacrococcygeal junction just lateral and inferior to the junction of the transverse process and body of coccyx in an AP view [55].
- Once bony contact is made, the needle is rotated laterally and advanced slightly until it slips off the coccygeal body.
- A lateral view is then taken, and the needle tip should lie on the posterior one third of the vertebral body. The needle is then rotated in a caudal and medial direction and advanced until the tip is just anterior to the coccyx. At this point, the needle should lie within the precoccygeal space.
- Another AP image is taken to ensure the needle tip is positioned near midline. After negative aspiration for blood, inject 2 ml of nonionic, water-soluble contrast under live fluoroscopy. Appropriate spread should be in a cephalad and caudal fashion along the S_5 vertebral body in the precoccygeal space.

- Once appropriate position is confirmed, inject 4–6 ml of local anesthetic (1:1 mixture of 1–2% lidocaine and 0.2% ropivacaine or 0.25% bupivacaine) after negative aspiration. Steroid is not necessary.
- This technique is useful when there is calcification of the sacrococcygeal ligament.

Radiofrequency Thermocoagulation and Pulsed Radiofrequency

For radiofrequency thermocoagulation (RFTC) [56] and pulsed electromagnetic field radiofrequency (P-EMF) [57] of the ganglion impar, any of the aforementioned techniques can be performed and modified with the use of a 20- or 22-gauge, 5-mm active tip radiofrequency probe. The trans-sacrococcygeal approach, lateral approach, paramedian approach, or a combination of two can be used. For simplicity, we will describe the trans-sacrococcygeal approach.

- Once the target is identified a 22-gauge, 5-mm active tip radiofrequency probe is advanced in an AP view in coaxial fashion through the sacrococcygeal junction.
- Correct needle placement is confirmed by AP and lateral views followed by radiocontrast dye injection.
- This is followed by sensory and motor stimulation trials. Sensory stimulation is performed at 50 Hz and 1.0 V, while motor stimulation is done at 2 Hz and 3.0 V.
- This is followed by injection of 1.0 ml of local anesthetic for lesioning. After waiting 2–3 min, radiofrequency current is applied for 60–90 s at 80–90° for 2–3 cycles. With each cycle, the needle tip should be rotated 90°.
- For P-EMF, the size of the active tip is not important as the electromagnetic field is projected from the tip of the needle and not from the shaft. With P-EMF lesioning, two to four 120-s lesions are performed at 45 V. Local anesthetic is not required for P-EMF.

Neurolytic Ganglion Impar Block

The administration of a neurolytic agent can be performed via any approach desired. We will describe administration via the trans-sacrococcygeal approach.

- Utilizing the trans-sacrococcygeal approach, the operator will perform the procedure in the same fashion.
- Once the needle is correctly placed, the operator should inject 1–2 ml of nonionic, water-soluble contrast under live fluoroscopy. This is to ensure that the spread of the dye is around the desired area.
- If the dye spread is satisfactory, then a solution containing a mixture of local anesthetic, phenol, and steroid is injected. The total volume of 5 ml should consist of 2.5 ml of 6% phenol in saline, 1 ml of 40-mg triamcinolone, and 1.5 ml of 0.5% ropivacaine or 0.5% bupivacaine. The total 5-ml dose contains a final mixture of 3% phenol [13]. Alternatively, similar amounts of 6–10% phenol or anhydrous alcohol can be used.

Complications

The proximity of the ganglion impar to the adjacent structures can lead to potential complications as the needle is inserted. The rectum lies just anterior to the precoccygeal space, and inadvertent puncture can lead to perforation and fistula formation. Other possible complications include epidural spread of agent, neurolytic injection into nerve roots or rectum, neuritis, cauda equina syndrome, tracking of contaminants back through the needle, and infection.

Evidence

Two case reports for cancer-related pain were graded as 1C [3]. One case report and one prospective case series for nonmalignant pain were also graded as 1C. In 2008, Agarwal et al. [47] published a case series of 43 patients who received CT-guided blocks and chemical neurolysis of the ganglion impar for both malignant and nonmalignant pain. It is graded as 1C. In the article, 12 additional ganglion impar blockade articles were reviewed which included the four mentioned in Day's paper. All were case reports and case series and were graded as 1C evidence.

Conclusion

Sympathetic blocks can be useful tools in the management of chronic malignant and nonmalignant pain. Neurolytic procedures can provide longer relief when the diagnostic blocks have been successful.

The majority of the available evidence is case series and case reports. The charge to current and future pain physicians is to implement well-designed clinical studies that support the need and use of these blocks.

Clinical Pearls

The practitioner should proceed with the block once an appropriate diagnosis is made, and the patient is an acceptable candidate. In certain patients, coagulation parameters should be checked. Knowledge of the anatomy around the targeted ganglia/ganglion is key. Proper needle placement is paramount to increase the success of the block and to decrease the incidence of untoward events. This includes observing for appropriate contrast spread and the absence of intravascular or neuraxial spread.

Multiple-Choice Questions

1. Sensory stimulation for location of the sphenopalatine ganglion will produce a paresthesia in which location?
   (a) The hard palate
   (b) The angle of the mandible
   (c) The upper teeth
   (d) The root of the nose
2. On the lateral fluoroscopic view, the target for needle placement for a sphenopalatine ganglion block is:
   (a) Inferior orbital fissure
   (b) Foramen ovale
   (c) Upper midportion of the pterygopalatine fossa
   (d) Lateral pterygoid plate
3. Which of the following is not a component of a Horner's syndrome?
   (a) Myosis
   (b) Mydriasis
   (c) Enophthalmos
   (d) Facial anhidrosis
4. A successful sympathetic block should raise the skin temperature by what amount?
   (a) 1°F
   (b) 1°C
   (c) 1.5–3.0°F
   (d) 1.5–3.0°C
5. The potential for which complication of a thoracic sympathetic block increases if the skin entry point is greater than 4 cm lateral to the spinous process of the targeted vertebral level?
   (a) Pneumothorax
   (b) Chylothorax
   (c) Arterial injection
   (d) Intraneural injection
6. While injecting contrast to confirm needle position for a thoracic sympathetic block, it is noted that the contrast ascends and descends with respiration. What does this indicate about the position of the needle tip?
   (a) The needle tip is too medial
   (b) The needle tip is too cephalad
   (c) The needle tip is too caudal
   (d) The needle tip is too lateral

7. The least splanchnic nerve is located at what vertebral body level?
   (a) T8
   (b) T10
   (c) T12
   (d) L_1
8. While performing sensory stimulation for a splanchnic nerve radiofrequency thermocoagulation, the patient complains of a pressure sensation deep in the abdomen. What is the appropriate action to this response?
   (a) Withdraw the needle a few millimeters and restimulate
   (b) Inject local anesthetic and commence lesioning
   (c) Advance the needle a few millimeters and restimulate
   (d) Abort the procedure
9. Which of the following is not required when using phenol for a neurolytic block?
   (a) Injection of local anesthetic prior to the injection of the phenol
   (b) Flushing the needle with normal saline prior to removal
   (c) Previous positive diagnostic block
   (d) A cancer pain related diagnosis only
10. While performing a radiofrequency thermocoagulation of the lumbar sympathetics at the L3 vertebral body level, the patient complains of pain in the groin. What is the appropriate next step?
    (a) Stop lesioning, inject more local anesthetic, and complete the RFTC after a couple of minutes
    (b) Ask the patient if he/she can tolerate the rest of the lesioning without stopping
    (c) Stop lesioning, reposition the needle, restimulate sensory, and if no stimulation in the groin, inject local anesthetic and resume lesioning after a couple of minutes
    (d) Stop lesioning and go on to the next level.
11. Which of the following is a side effect rather than a complication of a celiac plexus block?
    (a) Retroperitoneal hematoma
    (b) Diarrhea
    (c) Hematuria
    (d) Hypertension
12. What type of nerve fiber is not found in the superior hypogastric plexus?
    (a) Preganglionic sympathetics
    (b) Postganglionic sympathetics
    (c) Preganglionic parasympathetics
    (d) Postganglionic parasympathetics

13. Which of the following describes the appropriate contrast spread on a lateral fluoroscopic image for a superior hypogastric plexus block?
    (a) Linearly on the anterior superior aspect of the L_5 vertebra
    (b) Curvilinear over the anterior aspect of the L_5–S_1 disc and sacral promontory
    (c) Linearly on the anterior sacrum at the level of the anterior S_1 foramen
    (d) Linearly over the anterior surface of the lower sacrum
14. Although variable in location, the ganglion impar is more commonly located:
    (a) Caudal to the sacrococcygeal junction
    (b) Cephalad to the sacrococcygeal junction
    (c) At the sacrococcygeal junction
    (d) At the tip of the coccyx
15. For a patient with a fused sacrococcygeal junction, which two techniques can be used to block the ganglion impar?
    (a) Medial and lateral techniques
    (b) Lateral and caudal techniques
    (c) Caudal and paramedian techniques
    (d) Lateral and paramedian techniques

Answers:

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

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Part V
Special Populations

26

Regional Anesthesia for Chronic Pain

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Introduction

Sympathetic blockade has become a mainstay of therapy in the treatment of a variety of cancer-related and chronic pain conditions. The advent of ultrasound-, fluoroscopy-, endoscopy-, and CT-guided techniques have brought this important area of pain control to the regional anesthesiologist and pain management specialist. Imaging has allowed precise placement of needles to deliver local anesthetics, steroids, and neurolytic substances to block various sympathetic ganglia. With these techniques, pain relief can potentially be achieved for a variety of cancer and non-cancer pain conditions.

Autonomic Nervous System

The autonomic nervous system is composed of both the sympathetic and parasympathetic nervous systems, which provide opposing actions to one another. The autonomic nervous system is primarily responsible for a variety of homeostatic mechanisms in the body. These are paramount in maintaining organ perfusion, function, and metabolism. Specific areas of action include maintaining vascular tone, cardiac conduction and inotropy, pulmonary bronchodilation and bronchoconstriction, smooth muscle tone, and the transmission of pain.

Elements of the autonomic system can be found at various levels of the spinal cord. The parasympathetic nervous system is composed of cranial nerves arising from the brainstem and the sacral portion of the spinal cord, and it is termed the "craniosacral" portion of the autonomic nervous system. The sympathetic nervous system is composed of neural fibers arising from the thoracic and lumbar areas of the spinal cord, and it is termed the "thoracolumbar" portion of the spinal cord. It is the sympathetic nervous system that is of interest to the pain specialist, as it is not only important for homeostatic function of the body, but it also acts as a conduit for afferent nociceptive impulses from the periphery and major organs (Fig. 26.1).

The cell bodies of the sympathetic nervous system are found in the intermediolateral column of the thoracolumbar portion of the spinal cord. The primary ventral ramus carries the preganglionic sympathetic fibers as it exits the neural foramen. White rami communicantes allow the sympathetic axons to exit the ventral ramus and enter the paravertebral sympathetic ganglia and chain, which are located at the anterolateral portion of the vertebral body. In the sympathetic ganglia, preganglionic fibers synapse with postganglionic neural cells. Postganglionic fibers either travel through the gray rami communicantes to the original ventral ramus to peripheral sites, or they travel directly to the organs they affect. A variety of painful conditions can be successfully treated with blockade of the sympathetic chain at various levels including the stellate ganglia, celiac plexus, lumbar sympathetic chain, and superior hypogastric plexus.

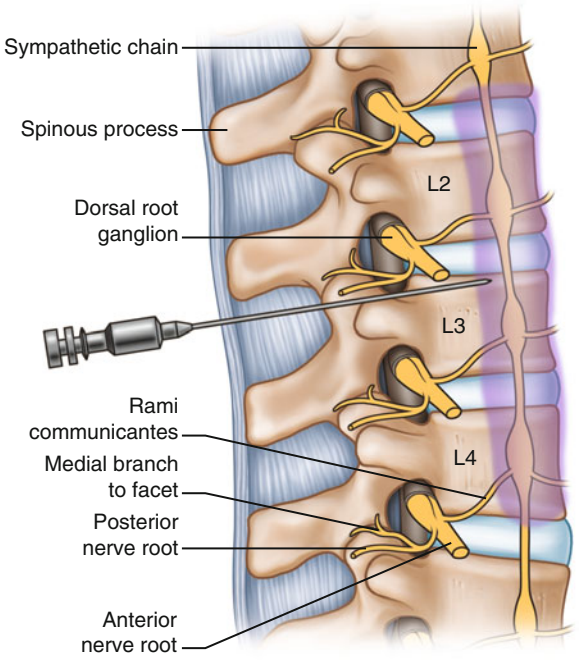

Fig. 26.1 Sympathetic chain and various ganglia

Chronic Pain States

CRPS I, CRPS II

Complex regional pain syndrome (CRPS) is a broad diagnosis based on different signs and symptoms. Pain is a presenting symptom in the vast majority of cases of CRPS, with the remaining diagnosis based primarily on history and physical exam. CRPS I (formerly known as reflex sympathetic dystrophy) describes a variety of painful conditions following an insult to an extremity that appears in a regional distribution with a distal predominance of abnormal findings. In CRPS I, a broad range of minor or major injuries to a limb precede the onset of symptoms. CRPS II (formerly known as causalgia) may potentially develop after a peripheral nerve injury. The presence of vasomotor changes (temperature changes, sweating abnormalities, edema, and vascular changes) suggests that sympathetic dysfunction plays a role in many aspects of the disease; this is termed sympathetic mediated pain.

CRPS I (Reflex Sympathetic Dystrophy)

1. The presence of an initiating noxious event, or a cause of immobilization.
2. Continuing pain, allodynia, or hyperalgesia with which the pain is disproportionate to any inciting event.

3. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor activity in the region of the pain.
4. This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

CPRS II (Causalgia)

1. The presence of continuing pain, allodynia, or hyperalgesia after a nerve injury, not necessarily limited to the distribution of the injured nerve.
2. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor activity in the region of the pain.
3. This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

While, primarily, the diagnosis of CRPS is based on clinical criteria, there are several diagnostic tests, with varying sensitivities and specificities, that can be used to support the findings. These tests include plain radiography, bone scan, quantitative sensory testing, temperature differences, MRI, and even skin biopsy.

The proposed mechanisms for CRPS involve a variety of physiologic and pathophysiologic factors. Theories involve possible changes to peripheral fibers, causing usual painless stimuli to elicit a painful response via low-threshold mechanoreceptors, resulting in allodynia; axonal injury may further potentiate this mechanism. In addition, communication between the somatic and sympathetic nervous system may result in abnormal sympathetic activity at the site of injury, causing alterations in skin blood flow, temperature regulation, sweating, as well as trophic changes. Furthermore, the somatic sensory nervous system may be more sensitive to circulating catecholamines. Finally, local inflammation and immobility of the affected area are not only proposed causes of CRPS, but can also potentiate injury and may predispose to the development of chronic changes. Currently, there is some evidence that immune cell-mediated inflammation and autoimmune responses are involved in the pathogenesis of CRPS.

The management of CRPS is multifactorial. Many treatments have been proposed, but few have withstood rigorous scientific investigation. The mainstay of therapy should focus on early intervention, combined with functional rehabilitation of the extremity. If the patient does not respond rapidly to conservative therapy using NSAIDs and functional rehabilitation, early consultation with a pain specialist should be considered. A variety of therapies can be offered, including neuropathic medications (i.e., tricyclic antidepressants, gabapentin, pregabalin, etc.), sympathetic ganglion blocks for sympathetically maintained pain, intravenous regional sympatholysis, spinal cord stimulation, or even irreversible sympathectomy in selected cases. Patients with acute CRPS should receive sympathetic interventions as soon as possible in order to achieve the highest degree of pain relief and to facilitate physical therapy. In children, CRPS is generally rare; symptoms tend to resolve without invasive intervention.

In patients with CRPS (types I and II), blockade of the sympathetic system (stellate ganglion, lumbar sympathetic chain) can provide profound pain relief when combined with other complimentary methods (physical therapy, neuropathic medications, etc.). The pain component that is relieved by specific sympatholytic procedures is considered sympathetically maintained pain. The positive or negative effect of a sympathetic blockade is not essential for the diagnosis of CRPS; it is, though, the only approach that is used to classify the pain as sympathetically maintained. If pain persists despite sympathetic blockade, the term sympathetic independent pain is used.

Visceral and Cancer Pain

The pain of cancer, inflammation, and solid viscus distension can be excruciating to many patients. Classically, these patients tend to be terminal cancer patients, for which tumor-induced visceral pain can be very significant. The generation of visceral and cancer pain involves a complex interplay of local organ and tissue manifestations, the somatic nervous system, and the sympathetic nervous system. Together, this interplay results in transmission of a variety of pain stimuli from solid organs. Due to the diffuse, nonspecific nature that visceral pain (especially cancer visceral pain) can produce, it is essential to understand the basic etiology and mechanisms of pain transmission prior to the institution of therapy.

The characteristics of visceral pain tend to differ from the characteristics of somatic pain. Visceral pain tends to be more diffuse and nonspecific in nature, making the exact pain difficult to localize. In addition, much of the visceral pain can be referred to cutaneous structures, further impeding localization. Some stimuli that lead to visceral pain can be similar to those that cause somatic pain; these include inflammation and ischemia. Stimuli unique for visceral pain include smooth muscle spasm and hollow-organ distension.

Visceral afferent fibers tend to arise on or in close relationship to the innervated organs. While the afferent fibers themselves convey sensory information, these fibers tend to pass with efferent autonomic fibers of the sympathetic and parasympathetic nervous systems; these afferents tend to carry visceral nociceptive information from the organ of interest to the central nervous system. Visceral afferent fibers, while passing with sympathetic fibers, tend to have cell bodies in the dorsal root ganglia, and terminate in the dorsal horn of the spinal cord, similar in nature to cutaneous nociceptive fibers. Two important differences in visceral versus nociceptive fibers are (1) visceral fibers tend to have thresholds for stimulation that respond only to noxious stimulation, and (2) the number of visceral afferent fibers tends to comprise a smaller proportion as compared to cutaneous afferents.

The relationship of visceral afferent fibers to the autonomic nervous system tends to make autonomic blockade (i.e., celiac plexus block, superior hypogastric plexus block) an attractive option to treat visceral malignant and nonmalignant pain.

Neuropathic Pain

Neuropathic pain occurs due to pathology in the peripheral nerves themselves and are classified as either mononeuropathy, polyneuropathy, mononeuritis multiplex, or autonomic neuropathy. The most common form is (symmetrical) peripheral polyneuropathy, which generally affects the feet and legs since they are the longest nerves from the CNS and are therefore more prone to injury. The form of neuropathy may also be classified on the part of the nerve involved (axonal degradation versus a demyelinating lesion) or based on the size of predominant fiber that is involved (large fiber versus small fiber peripheral neuropathy). If the underlying cause of a neuropathy cannot be identified, it is designated idiopathic. Examples of neuropathic conditions include complex regional pain syndrome, diabetic peripheral neuropathy, postherpetic neuralgia and phantom limb pain.

Neuropathy may be associated with numerous symptoms throughout the body, depending on the number and type of nerves involved; these symptoms include motor loss, sensory changes (paresthesias, numbness, etc.), and autonomic changes. Loss of muscle tone may be seen due to denervation atrophy. Fasciculations may also be seen, generally about 5 weeks following lower extremity denervation. Sensory symptoms include "negative" changes such as loss of sensation and "positive" changes such as tingling or pain. Uncontrolled diabetics, for example, often have a symmetric polyneuropathy described as a sensation in a "stocking and glove" distribution which feels like pins and needles. Loss of balance and coordination may also occur due to injury to the nerves involved in proprioception. Autonomic neuropathy leads to symptoms such as abnormal blood pressure, heart rate, sexual dysfunction, constipation, bladder control, and sweating [1].

Cranial Nerve and Cervicogenic Pain

Trigeminal neuralgia (TN), also known as tic douloureux, is a neuropathic disorder characterized by episodes of intense facial pain due to aberrant signals from one of the three branches of the trigeminal nerve [ophthalmic (V1), maxillary (V2), or mandibular (V3) branches]. It is estimated that 1 in 15,000 people suffer from trigeminal neuralgia. Symptoms generally begin after the age of 50 and are more common in females [2]. Various treatment modalities exist for trigeminal neuralgia, including neuropathic agents such as carbamazepine, nerve blocks such as gasserian ganglion and sphenopalatine ganglion blocks, radiofrequency rhizotomy of these ganglia, microvascular decompression of the trigeminal nerve from the superior cerebellar artery, and gamma knife radiation [3]. Occipital neuralgia is another painful condition due to pathology affecting the occipital nerve, which gets its distribution via the C2 and C3 nerves to form the greater and lesser occipital nerve (therefore, it is not technically a cranial nerve even though it affects the posterior portion of the cranium). Some experts advocate that cervicogenic headaches and

occipital neuralgia may be adequately treated with blockade of the "third occipital nerve" at the C2/C3 facet joint itself [4]. The occipital nerves can be the target for nerve blocks and possible rhizotomy. Other causes of cranial nerve pain include herpes zoster ophthalmicus and various forms of headaches which manifest as pain in a cranial nerve distribution (cluster headache, etc.).

Herpes Zoster Pain

After the varicella-zoster-virus-mediated chickenpox has resolved, often during childhood, the virus can remain latent in the dorsal root ganglia where it can reemerge later in life as herpes zoster, or shingles. Herpes zoster is a disease characterized by a transient rash in a dermatomal distribution that is usually painful. The term postherpetic neuralgia (PHN) is used if the pain persists after the rash has resolved. Older individuals and immunocompromised individuals are generally the ones that are at significant risk for reactivation of herpes zoster and the subsequent development of PHN. Studies have shown that peripheral and central demyelination in conjunction with neuron destruction may be involved. Both the vaccine against VZV (Varivax) and the newly released vaccine against herpes zoster (Zostavax) may lead to substantial reductions in morbidity from herpes zoster and PHN in the future [5]. Multiple medications are often utilized in reducing the pain associated with PHN, including antidepressants, neuropathic agents, opioids, NMDA receptor antagonists, topical lidocaine, and capsaicin. Intrathecal corticosteroids may play a role in treating PHN, but this is only based on level B evidence and needs to be further studied. Sympathetic blockade and spinal cord stimulation may also play a role in treating the pain of herpes zoster or PHN [6].

Phantom Limb Pain and Stump Pain

Limb amputation can be associated with a myriad of symptoms, including phantom limb sensation, phantom limb pain, and stump pain. Phantom limb pain is the phenomenon of experiencing pain in an extremity after that extremity has been removed, either primarily (as in trauma) or secondarily (as in surgical amputations). Patients report experiencing a wide range of pain characteristics including burning, cramping, and tingling as well as lancinating electrical shocks, itching, stabbing, throbbing, and even a feeling of "pins and needles" [7]. Although phantom limb pain is experienced in both upper and lower limb amputees, it tends to be localized distally. The onset of phantom limb pain occurs within the first 24 h for about half of all patients and within a week for another 25% [8]. Both central and peripheral nervous system mechanisms have been proposed for phantom limb pain, and some experts suggest that phantom pain is a combination of both. There are varying treatment modalities with different levels of evidence as far as efficacy for phantom limb pain.

These include treatments which have level 1 evidence such as opioids and gabapentin; level 2 evidence such as amitriptyline, tramadol, tricyclic antidepressants, calcitonin, TENS units, mirror therapy, ketamine, and memantine; and level 3 evidence such as carbamazepine, mirtazapine, perioperative epidural infusions, clonidine, mexilitene, and acupuncture [9, 10]. Stump pain, on the other hand, is the experience of pain at the site of the amputation itself and is often due to a neuroma which has formed at the incision site during the healing process. Stump pain, also called residual limb pain, is often treated with a combination of neuropathic medications, local anesthetic creams, and opioids; occasionally, more invasive modalities such as neuroma resection and muscle reimplantation are also utilized [11].

Review of Blocks

Stellate Ganglion Block

Introduction

The stellate ganglion is, essentially, a fusion of the superior thoracic sympathetic ganglion and the inferior cervical sympathetic ganglion. This fusion is present in about 80% of the population. The stellate ganglion is oval in shape and is about 2–3 cm long and 0.5–1 cm wide. Cell bodies for the sympathetic fibers that supply the head, neck, and upper extremity arise from T1 to T8, sometimes including T9. Preganglionic fibers travel to the sympathetic chain and travel cephalad to synapse in the inferior, intermediate, or superior cervical ganglia. Postganglionic fibers either travel along the gray rami communicantes to join the ventral rami comprising the cervical and brachial plexus, while the remaining postganglionic fibers travel from the ganglia directly to the head, neck, and upper extremity as perivascular structures. Some sympathetic fibers bypass these ganglia completely and course with the vertebral artery; thus, blockade of these ganglia can sometimes produce inconsistent and incomplete sympathetic blockade to structures in the head and neck, rendering this block of little value in such cases. The stellate ganglia are separated by loose connective tissue, allowing local anesthetic spread to superior and inferior sympathetic structures; this also allows local anesthetic spread to nonrelated structures, such as the brachial plexus.

The stellate ganglion block has been used successfully to treat a variety of sympathetically maintained syndromes of the upper extremity. It has long been one of the cornerstones of therapy (along with physical therapy and neuropathic medications) for complex regional pain syndrome (types I and II) of the upper extremity. Stellate ganglion block has also been used for a variety of upper extremity neuropathic pain syndromes, including postherpetic neuralgia and phantom limb pain. In addition, stellate ganglion blocks have been used successfully to treat syndromes of vascular insufficiency, including embolic disease, Raynaud's disease, vasospasm, and even angina pectoris.

Block Technique

A variety of fluoroscopy- and ultrasound-guided techniques operate on the premise of localizing the stellate ganglion via anatomical landmarks. This text will first describe blockade of the stellate ganglion using pure anatomic techniques, followed by options for fluoroscopy and ultrasound guidance, depending on operator preference.

Prior to performance of the block, it is mandatory that monitoring, IV supplies, airway equipment, and resuscitative drugs are immediately available. In addition, it is important that personnel trained in dealing with any immediate complications of the blockade are readily available.

The anterior approach is generally used to access the stellate ganglion.

1. The patient is placed supine, with a roll placed underneath the patient's shoulders; this facilitates extension of the patient's neck.
2. The level of C6 is identified, which generally corresponds to the cricoid cartilage.
3. Next, the skin is prepped with an antiseptic solution.
4. Two fingers are placed on the cricoid cartilage, and gently retracted laterally, allowing the carotid artery to be retracted and allowing the palpation of the anterior tubercle of C6 (Chassaignac's tubercle).
5. A skin wheal is placed over the tubercle, and a small needle (23–25 gauge, 2-in.) is advanced in a perpendicular fashion to contact the tubercle.
6. Once on the tubercle, the needle is withdrawn 2 mm.
7. Tubing is connected to the needle, the syringe is steadied, and aspiration is carried out.
8. A test dose of 1 cc of local anesthetic solution should be injected while watching for complications of intravascular or intrathecal injection.
9. Local anesthetic (generally 8–10 cc of 0.25% bupivacaine) is given slowly with frequent aspiration, and attention paid to keeping the needle in a stable position.

Signs of successful blockade include clinical recognition of the presence of Horner's syndrome (miosis, ptosis, enopthalmos, conjunctival injection, and hemianhydrosis). However, this does not always indicate complete sympathetic blockade of the upper extremity. Assessment of sympathetic blockade can be done clinically by examination of the extremity for venodilation, vasodilation, and warmth (a temperature increase of 1–3°C is typically seen). Alternative methods include measuring skin resistance (sympathogalvanic response), Doppler flow measurements, microneurography, and the sweat test.

Fluoroscopy-/Ultrasound-Guided Approach

Depending on operator comfort and preference, a fluoroscopy- or ultrasound-guided approach can also be used for confirmation of needle placement and local anesthetic spread, with appropriate needle localization and placement on Chassaignac's tubercle with direct visualization. While performing the fluoroscopic technique, contrast

dye can be used to confirm appropriate placement prior to local anesthetic injection. The various radiologic techniques used for needle placement are beyond the scope of this review book.

Complications

Complications of the stellate ganglion block include mechanical, infectious, bleeding, and pharmacologic ones. Mechanical complications constitute direct nerve and visceral injury during insertion/manipulation of the needle. These include brachial plexus injury, tracheal injury, esophageal injury, pneumothorax, hemothorax, and chylothorax. Bleeding complications are generally caused from a vascular injury with the needle, resulting in local hematoma or more significant perivascular bleeding. Infectious complications include local abscess, cellulitis, or osteomyelitis. Pharmacologic complications include blockade of the recurrent laryngeal nerve (resulting in hoarseness); blockade of the phrenic nerve (resulting in respiratory dysfunction); brachial plexus blockade (resulting in upper extremity weakness); vertebral artery injection (resulting in seizures); or possible epidural/intrathecal injection (resulting in a high spinal block).

Celiac Plexus Block

Introduction

The celiac plexus is located in the retroperitoneum at the T12–L1 level, and surrounds major vascular structures, including the abdominal aorta and branching arteries. It is a diffuse network of nerve fibers, composed of both sympathetic fibers from the anterolateral horn of the spinal cord from T5 to T12 (greater, lesser, and least splanchnic nerves) and parasympathetic fibers from the vagus nerve. Autonomic innervation is supplied to major gastrointestinal organs, such as the liver, gallbladder, pancreas, stomach, small bowel, and ascending and transverse colon. Nociceptive impulses from the abdominal viscera travel with the sympathetic nerves.

Local anesthetic and neurolytic blockade of the celiac plexus have been used for both malignant and chronic abdominal visceral pain. Blocks with variable success rate have been used for management of acute or chronic inflammatory pain (i.e., pancreatitis). Neurolytic celiac plexus blocks have most commonly been used for management of malignant intra-abdominal pain, particularly pancreatic cancer-related pain. The block can achieve dramatic pain relief and eliminate the need for high-dose opioid therapy (and its inherent side effects) in the management of end-of-life malignant cancer pain. In one meta-analysis study of pain relief for cancer pain following celiac plexus block, good to excellent pain relief was reported in 89% of patients 2 weeks after the neurolytic celiac plexus blockade was performed. The study also revealed there was partial to complete pain relief in 90% of patients 3 months post block, and in 70–90% of patients until death, even beyond the 3-month timeline.

Block Technique

Due to the presence of major vascular and neural structures encountered during block placement, it is recommended that an imaging modality (most commonly fluoroscopy- or CT-guided technique) be chosen to confirm appropriate needle placement. Three approaches to the treatment of visceral intra-abdominal malignancy pain include the retrocrural approach to block the celiac plexus (classic approach), the anterocrural approach to block the celiac plexus (transaortic approach), or a block of the splanchnic nerves (not described here, but involves advancement to the anterolateral portion of T12–L1).

1. An imaging modality should be chosen, emphasizing operator preference and comfort; generally, fluoroscopy is chosen for the interventional pain physician.
2. The patient should be placed in prone position, and the skin prepped with an antiseptic solution. The area should be draped and sterile technique utilized.
3. Radiographic guidance should verify the location of the 12th rib and the L1 vertebral body.
4. An entry point is chosen 5–7 cm left of the midline and 1–2 cm below the inferior margin of the 12th rib; a local anesthetic wheal is made at the entry point.
5. Using radiographic guidance, a 22-gauge spinal needle is advanced to the anterolateral margin of the L1 vertebral body.
6. A second needle is advanced on the right side using the same approach; the needles are passed no further than 0.5 cm anterior to the anterior border of L1.
7. For the anterocrural approach, the original (left-sided) needle is advanced 2–3 cm beyond the vertebral body while continuously aspirating; when blood is encountered (indicating likely intra-aortic placement), the needle should be advanced until there is negative aspiration, placing of the needle anterior to the aorta.
8. Needle position can be verified using a small amount of radiocontrast media; this will not only confirm proper needle placement, but will also rule out intravascular needle placement.
9. For the retrocrural approach, 20–25 cc of solution are used per side; for the anterocrural technique, only 8–10 cc of solution are required.
10. Local anesthetic is chosen for temporary blockade (e.g., 0.25% bupivacaine), while alcohol or phenol are chosen for neurolytic blockade. The need for higher volumes precludes the use of phenol in the retrocrural approach.

Complications

The adverse effects of celiac plexus blockade can be divided into expected physiologic side effects and complications. Expected physiologic side effects include diarrhea, abdominal cramping, and hypotension. These effects are generally transient and are due to sympathetic blockade. In a meta-analysis study, the most common adverse effects were local pain (38%), diarrhea (44%), and hypotension (38%). In addition, the side effects vary with the approach chosen. Hypotension is more common with the retrocrural technique, while diarrhea is more common with the anterocrural technique.

Complications of celiac plexus block include injury to adjacent structures [kidney injury (resulting in hematuria), lung injury (resulting in pneumothorax), vascular injury (resulting in aortic dissection or retroperitoneal hemorrhage)], intravascular injection, and paraplegia. Hemorrhage can be caused by bleeding into the retroperitoneum or bleeding into abdominal viscera. Damage to vascular structures, although rare, has been reported, including dissection of the abdominal aorta. It is recommended that the transaortic technique be avoided in patients with aortic pathology or atherosclerosis. Intravascular injection can occur with local anesthetic or neurolytic substances. Local anesthetic levels can reach high enough levels to cause toxicity with the high volumes required for the retrocrural approach to celiac plexus blockade. Intravascular injection of phenol can cause symptoms similar to local anesthetic toxicity.

The most feared complication of celiac plexus neurolysis is paraplegia. This is thought to occur because of spasm or necrosis of lumbar segmental arteries that perfuse the spinal cord. In many patients, the Artery of Adamkiewicz (with a variable level of appearance) is the dominant blood supply to the anterior two-thirds of the spinal cord; a spasm can cause complete paralysis. Other factors are likely responsible as well, such as direct vascular injury and possible retrograde spread of neurolytic agent to the spinal cord.

Lumbar Sympathetic Block

Introduction

Lumbar sympathetic block (LSB) is a modality available to aid in the inhibition of sympathetically mediated pain in the lower extremities (just as stellate ganglion block is used for such pain in the upper extremities). The LSB can be used to both help in the diagnosis and treatment of sympathetically maintained pain in conditions such as CRPS type I and type II as well as phantom limb pain. The lumbar sympathetic chain consists of three to five ganglia which lie anterior to the L2, L3, and L4 vertebral bodies. It is located anterior to the psoas muscle and posterior to the vena cava on the right side and to the aorta on the left. Based on anatomic studies, the number of rami which connect to each ganglia varies between zero and six, with the vast majority having just one ramus [1].

Block Technique

1. The patient is generally placed in prone position with a pillow under the abdomen to decrease the lumbar lordosis.
2. Using fluoroscopic guidance, the spinous process of L2, L3, and L4 are identified and marked.
3. The fluoroscope is then rotated obliquely toward the side to be blocked, visualizing the transverse process overlying that particular lumbar segment.

4. The area of proposed needle entry is then infiltrated with local anesthetic, and then, a 10-cm 20-gauge needle is inserted and advanced until it comes in contact with the transverse process. The needle depth is noted and then withdrawn slightly, angling caudad, and walked inferiorly off the transverse process. It should be avoided to enter the skin more than 7 cm lateral from the midline and inadvertently contact visceral structures during needle advancement (kidney, liver, spleen, etc.).
5. A slight medial angulation is used to contact the vertebral body at that level . This distance should also be noted. Then the fluoroscope should be rotated lateral.
6. The needle is advanced anteriorly to walk off that body (the tip thereby remaining close to the vertebra) until its anterolateral border is reached.
7. Injection of contrast solution at this point should demonstrate linear spread in a cephalocaudad direction.
8. This technique can either be performed at three individual levels (L2, L3, and L4; L3 with 5 mL of 0.25% bupivacaine at each level) or simply at L3 (with 15 mL of 0.25% bupivacaine).

To determine efficacy, temperature probes should be placed on both extremities prior to proceeding. After a successful block, an approximate increase in temperature of 3°C should be noted on the blocked side. It has been demonstrated that the distal lower extremity ipsilateral to the LSB had the greatest magnitude (8.7°C ± 0.8°C) and rate (1.1°C ± 0.2°C/min) of temperature change. It has been also shown that the great toe temperature was within 3°C of core temperature within 35 min after LSB. The patient should initiate physical therapy at this time to perform range of motion and strengthening exercises since the pain involved in performing such exercises should be decreased after a successful block has been performed (Fig. 26.2).

Superior Hypogastric Plexus Block

Introduction

The superior hypogastric block is useful for the treatment of pelvic pain, which is either nonmalignant or malignant in nature. The superior hypogastric plexus is situated in the retroperitoneum, bilaterally extending from the lower third of the fifth lumbar vertebral body to the upper third of the first sacral vertebral body. Therefore, the target for needle insertion is anterior to the body of the L5 vertebra at the L5–S1 junction. The plexus contains both postganglionic sympathetic fibers and afferent pain fibers. The plexus gives rise to the innervation of the rectum, bladder, perineum, vulva, vagina, prostate, and uterus. Pain originating from any of these pelvic structures could theoretically be treated by blocking this plexus.

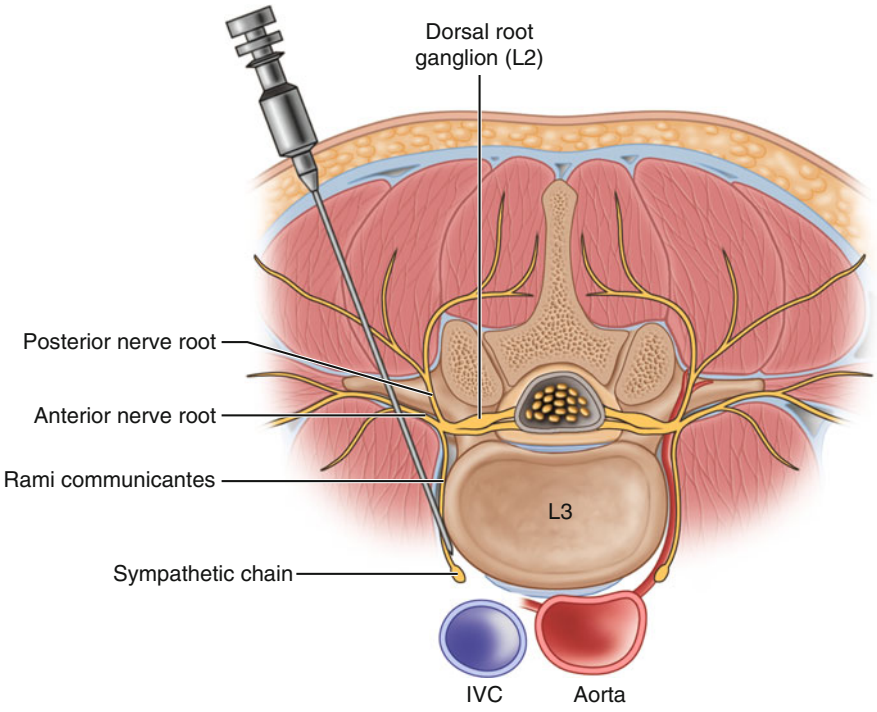

Fig. 26.2 Lumbar sympathetic block, axial diagram

It has been demonstrated that this block is effective in reducing pain scores in 70% of patients with pelvic pain associated with cancer; the majority of which in their particular study had cervical cancer.

Block Technique

1. The block can be performed under CT or fluoroscopic guidance, with the goal being to place the tip of the needles anterior to the L5–S1 junction. The technical difficulty of this block arises from the fact that the iliac crest oftentimes blocks adequate access to needle advancement in a purely oblique view, thereby necessitating a fairly lateral and slightly cephalad approach.
2. Once the needles are in position bilaterally, a lateral view should reveal appropriate contrast spread in a smooth posterior contour corresponding to the anterior psoas fascia [4].
3. This is subsequently followed by injection of 5–10 mL of local anesthetic (e.g., 0.25% bupivacaine) via both needles for the prognostic block and an equal volume of neurolytic agent (either alcohol or phenol) for the subsequent therapeutic block.

Conclusion

Various regional modalities exist to treat pain associated with chronic painful conditions including sympathetic-mediated pain, cancer pain, and nonmalignant visceral pain. A firm understanding of the autonomic nervous system, spinal anatomy, and radiographic visualization is required to perform these blocks successfully.

Clinical Pearls

- The autonomic nervous system is primarily responsible for a variety of homeostatic mechanisms in the body; these functions are important in maintaining organ perfusion, function, and metabolism.
- It is the sympathetic nervous system that is of interest to the pain specialist, as it is not only important for homeostatic function of the body, but also acts as a conduit for afferent nociceptive impulses from the periphery and major organs.
- CRPS I (formerly known as reflex sympathetic dystrophy) describes a variety of painful conditions following an insult to an extremity that appears in a regional distribution with a distal predominance of abnormal findings; CRPS II (formerly known as causalgia) may potentially develop after a peripheral nerve injury.
- In patients with CRPS (types I and II), blockade of the sympathetic system (stellate ganglion, lumbar sympathetic chain) can provide profound pain relief when combined with other complimentary methods.
- The generation of visceral and cancer pain involves a complex interplay of local organ and tissue manifestations, the somatic nervous system, and the sympathetic nervous system.
- Neuropathy may be associated with numerous symptoms throughout the body including motor loss, sensory changes, and autonomic changes.
- Herpes zoster is a disease characterized by a transient rash in a dermatomal distribution that is usually painful. The term postherpetic neuralgia (PHN) is used if the pain persists after the rash has resolved. Older individuals and immunocompromised individuals are at significant risk for reactivation of herpes zoster and the subsequent development of PHN.
- Phantom limb pain is the phenomenon of experiencing pain in an extremity after that extremity has been removed, either primarily (as in trauma) or secondarily (as in surgical amputations). Patients report experiencing a wide range of pain characteristics including burning, cramping, tingling, and feelings of electrical shocks.
- The stellate ganglion block has been used successfully to treat a variety of sympathetically maintained syndromes of the upper extremity. It has long been one of the cornerstones of therapy (along with physical therapy and neuropathic medications) for complex regional pain syndrome (types I and II) of the upper extremity.

- Local anesthetic and neurolytic blockade of the celiac plexus has been used for both malignant and chronic abdominal visceral pain. Blocks with variable success rates have been used for management of acute or chronic inflammatory pain (i.e., pancreatitis).
- Lumbar sympathetic block (LSB) is a modality available to aid in the inhibition of sympathetically mediated pain in the lower extremities (just as stellate ganglion block is used for such pain in the upper extremities). LSB can be used to both help in the diagnosis and treatment of sympathetically maintained pain in conditions such as CRPS type I and type II, as well as phantom limb pain.
- The superior hypogastric block is useful for the treatment of pelvic pain, which is either nonmalignant or malignant in nature. The superior hypogastric plexus gives rise to the innervation of the rectum, bladder, perineum, vulva, vagina, prostate, and uterus.

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Regional Anesthetic Techniques for the Pediatric Patient

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Introduction

In the hospital setting, effective treatment of acute pain in children is a high priority as clinical studies have shown pediatric patients experience pain from medical illnesses, during therapeutic and diagnostic procedures, and from trauma and surgery [1–3]. Although opiates can be safely administered to children, the elimination half-life in newborns is observed to be longer, and clearance is decreased when compared to older children and adults [4]. The optimal plasma concentration for effective analgesia is highly variable, requiring careful titration to obtain the desired level of analgesia while minimizing side effects [5–7]. Moreover, these observed differences are particularly pronounced in the preterm neonate as opiates, such as morphine, are less protein bound which allows greater penetration into the immature blood–brain barrier and increases the risk for respiratory depression [7–10].

Regional anesthesia, with or without general anesthesia, offers multiple benefits compared with general anesthesia alone. These benefits include reductions in morbidity and mortality [11–17], superior postoperative analgesia [18–21], and enhanced cost-effectiveness [22–25]. There has been an increase in the development and use of regional anesthetic techniques for the pediatric patient since the original publications of the 1950s [26–29]. However, these techniques continue to be less applied in the pediatric patient due to concerns about neurologic complications, operator inexperience, and proper equipment [30–32]. Many of these concerns were addressed in a sentinel article published in 1996, in which a prospective study of greater than 24,000 pediatric blocks were conducted, of which 89% were performed under sedation or general anesthesia, with an incidence of 0.9/1,000 complications, and with no deaths nor long-term sequelae [33]. Moreover, in a 5-year study recently conducted in Great Britain and Ireland, over 10,000 pediatric epidurals were performed with no correlation of neurologic injury with the use of general anesthesia prior to placement, and with only one child suffering from residual effects 12 months following surgery (~1:10,000) [34]. When properly performed, regional anesthesia is a safe, clinical practice with risk profiles similar to general anesthesia [33–35]. In a recent study containing pooled data from 165 studies, Dolin and colleagues compared the benefits of conventional pain management with PCA versus conduction blockade and found that the mean incidences of moderate to severe pain in pediatric patients treated with PCA were 36% and 10%, whereas patients with continuous epidural analgesia had much lower incidences of moderate (21%) to severe (8%) pain [36].

Ultrasonography

Certainly, any clinical technique has an incidence of failure. Neurovascular anatomy is highly variable, and nerve localization through the use of electrical current stimulation techniques provides little to no information in the proper placement of the stimulation needle and/or subsequent spread of local anesthetics. Therefore, percutaneous techniques utilizing surface anatomy and projection, even in the best of

hands, are fraught with failure. The development of high-resolution portable ultrasound (US), analysis of anatomic relationships, and, most importantly, observation in the spread of administered local anesthetics have made this modality feasible for pediatric regional anesthesia [37–40]. To develop skill in the use of US, one should attend an US-guided regional anesthesia course, begin with simple blocks, then progress to the more complicated procedures as experience develops [41].

Local Anesthetic Blocks

The technical expertise required in delivering this type of care and concerns about neurologic complications and proper equipment are reasons why regional anesthetic techniques are not used in the pediatric population [30–32, 41, 42]. In children, most regional techniques will require general anesthesia. With regard to selection of local anesthetics, the delivery site and the metabolic maturity of the child are important considerations [43–45]. The introduction of the local anesthetics, levobupivacaine and ropivacaine, has similar pharmacokinetic profiles when compared to racemic bupivacaine, is observed to have less cardiac toxicity [46–49], and hence may be beneficial in children [50]. However, local anesthetic toxicity is rare in children, but seizures, transient neuropathic symptoms, dysrhythmias, and cardiovascular collapse have been reported [51].

Topical Analgesia

As with adults, topical anesthesia is used to anesthetize the skin by local infiltration before intravenous catheter insertion or other minor procedures [52]. Likewise, local anesthetic infiltration is also employed to provide postoperative analgesia for incisional pain. Dosing guidelines are comparable to those for adults [53].

Early studies in pediatric patients employed a mixture of tetracaine, adrenalin (epinephrine), and cocaine (TAC) for the repair of minor skin lacerations in the emergency room setting [54–58]. In a large series in pediatric patients, this novel form of anesthesia resulted in quicker surgical repair times, markedly improved patient acceptance, and wound complication rates were not significantly different when compared to lidocaine infiltration [58]. Subsequent studies confirmed these findings [55, 56].

A eutectic mixture of local anesthetics (EMLA) cream was developed in the 1980s and contains 2.5% lidocaine and 2.5% prilocaine [59]. The mixture results in an oil–water emulsification with a total local anesthetic concentration of 5% and has the ability to anesthetize intact skin to a depth of 5 mm [60, 61]. Recommended application is 45 min to 1 h before the invasive procedure. An occlusive dressing is usually applied over the application site. Because of its potential for systemic toxicity, EMLA cream should not be in prolonged contact with mucous membranes or with traumatized skin [62–64]. Common uses include anesthesia for venipuncture, neonatal circumcision, and laser ablation of port-wine stains [65–70].

Another local anesthetic cream with a shorter onset of action (~30 min), ELA-Max, is also available and is composed of 4% liposomal lidocaine [71]. One study by Eichenfield and others observed comparable efficacy between ELA-Max at 30 min and EMLA cream applied 60 min before the procedure [72]. ELA-Max may also decrease the incidence of methemoglobinemia as it does not contain prilocaine [51].

Application of local anesthetics to mucous membranes may decrease discomfort during nasotracheal intubation, nasogastric tube insertion, and bronchoscopy [73–77]. This application may be accomplished by a number of methods including direct spray, nebulization, or ointment or jelly application [78–81].

Regional Anesthetic Blocks

Head and Neck Blocks

Blockade of the great auricular nerve acts as an opioid-sparing technique for tympanomastoidectomy and otoplasty [82]. The nerve arises from the superior cervical plexus (C_2, C_3) and provides sensory innervation to the later occipital region and medial auricle. It ascends superficial to the posterior belly of the clavicular head of the sternocleidomastoid muscle (Fig. 27.1). Local anesthetic is injected along this

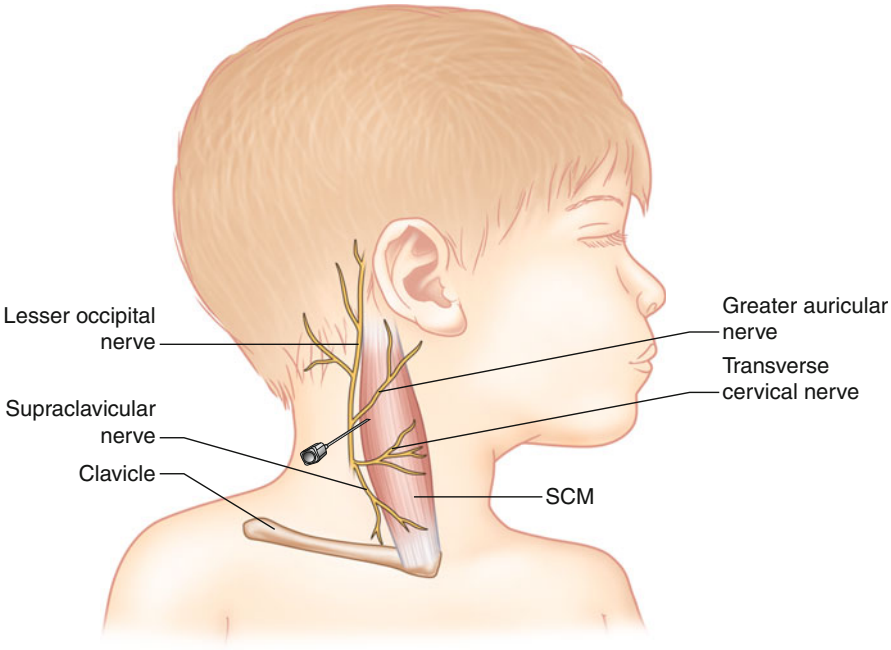

Fig. 27.1 Great aurical nerve block

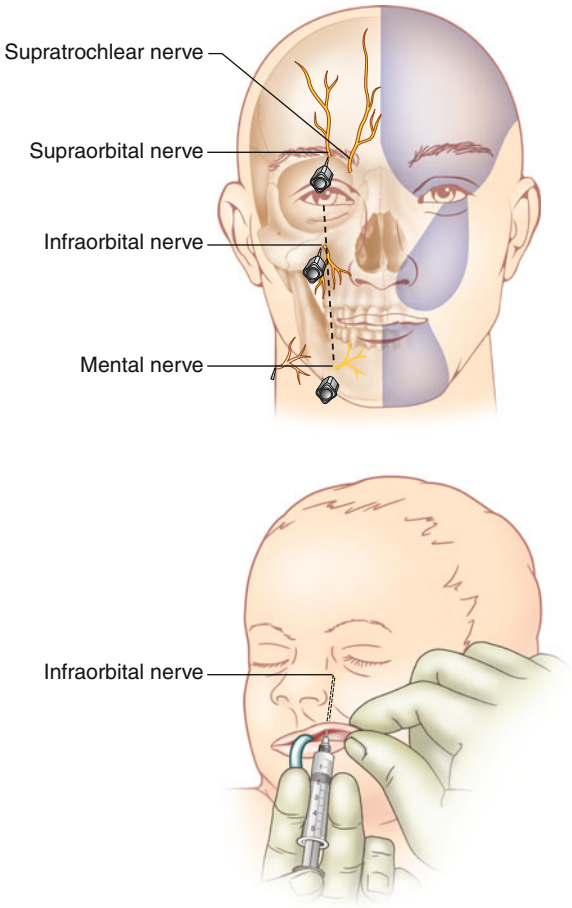

Fig. 27.2 Extraoral nerve block and intraoral nerve block

region at the level of the cricoid cartilage. Complications include intravascular injection of the carotid artery or internal jugular vein and phrenic nerve block resulting in Horner's syndrome [83, 84].

Effective pain relief for cleft lip repair as well as for sinus surgery, rhinoplasty, and nasal septal reconstruction can be provided by an infraorbital nerve block. The sensory nerve is derived from the second maxillary division of the trigeminal nerve and exits the skull through the foramen rotundum before passing through the infraorbital foramen. It then divides into four branches – internal and external nasal, superior labial, and inferior palpebral. These branches innervate the skin of the upper lip, lower eyelid and cheek, and lateral nose. Two field blocks, extraoral and intraoral, can block the nerve (Fig. 27.2). The external approach involves locating the infraorbital foramen approximately 0.5 cm inferior to the lower orbital margin. A 27-gauge needle is then inserted until bone is contacted. The needle is then withdrawn slightly, and a small amount of local anesthetic (0.25–0.5 ml) is injected. The intraoral

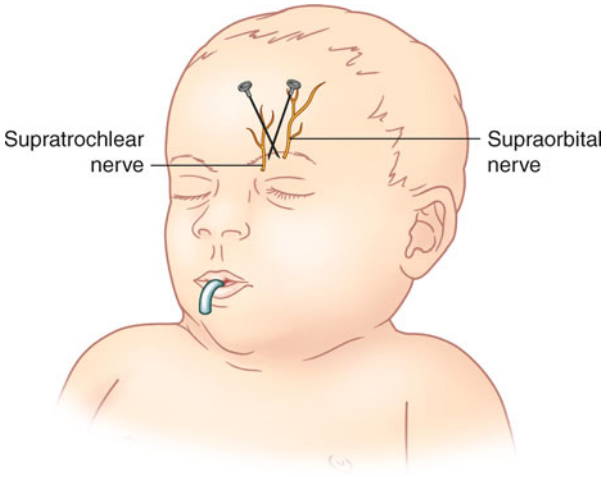

Fig. 27.3 Supraorbital and supratrochlear nerve block

approach starts with the same landmark by palpating the infraorbital foramen with the nondominant hand and keeping a finger there. The upper lip is then lifted and a 25–27-gauge needle is used to inject 0.5–1.5 ml of local anesthetic along the inner surface of the lip along the maxillary premolar toward the infraorbital foramen. Other than swelling around the eyelid, which can be reduced by pressure over the injection site for several minutes, complications of this block are rare.

Indications for supraorbital and supratrochlear nerve blocks include procedures on the scalp and forehead such as frontal craniotomies, ventriculoperitoneal shunt revisions, excision of skin lesions, and laser therapy for hemangiomas (Fig. 27.3). The nerves are branches of the ophthalmic division of the trigeminal nerve and supply the skin of the forehead and conjunctiva of the upper eyelid. The supraorbital nerve is found in the upper margin of the orbit at the supraorbital notch, and the supratrochlear nerve is in close proximity just medially. After palpating the supraorbital notch, a 27-gauge needle is inserted superior to the notch until bone is contacted. Local anesthetic (1 ml) is injected after slight withdrawal and negative aspiration for blood. The needle is withdrawn and directed slightly medially before injecting another 1 ml of local anesthetic. Hematomas and periorbital edema are common complications [85, 86] but can be minimized by applying pressure for approximately 5 min.

Brachial Plexus Block

Although there are several approaches to the brachial plexus in children, the axillary approach has been the most commonly used technique in the approach to the brachial plexus [87, 88]. The brachial plexus arises from the cervical nerve roots (C_5–T_1). Brachial plexus blocks are easy to perform in children due to less adipose

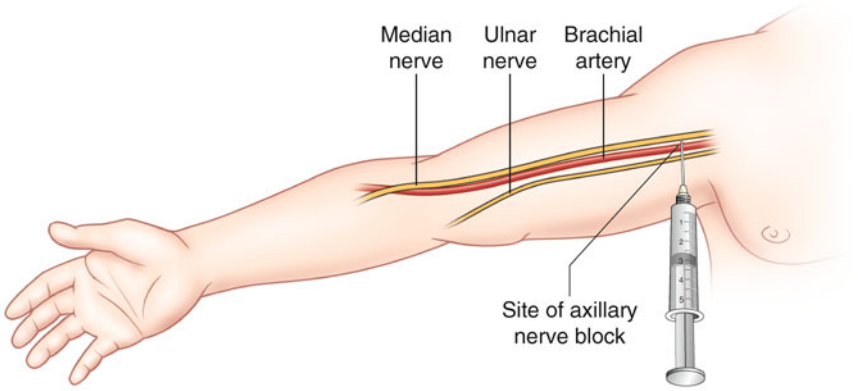

Fig. 27.4 Axillary block

tissue when compared to adults and the axillary artery is easier to palpate and isolate [89–91]. The arm is abducted to a 90° angle in relation to the chest wall. The artery is palpated and fixed in the axilla, and the 22-gauge, short-bevel, 2-in. needle allows accurate placement in and through the axillary artery (Fig. 27.4). With continuous aspiration, blood from the artery is obtained, the needle is further advanced until no further blood is withdrawn, and one half of the local anesthetic can be injected into the sheath. As the needle is withdrawn, again blood is aspirated until no further blood can be withdrawn, and the remaining half of the local anesthetic can be injected. The recommended dose of local anesthetic is 1 ml/kg of either 0.25% bupivacaine or 0.2% ropivacaine [53]. Vigilant aspiration should be performed to minimize intravascular injection. An additional circumferential subcutaneous cuff block for the intercostobrachial nerve to minimize tourniquet pain is also recommended.

The use of the nerve stimulator can assist the operator in advancing the 22-gauge, short-bevel, 2-in. needle into the sheath of the brachial plexus superior to the axillary artery. Once a twitch is elicited, local anesthetic solution can be injected into the sheath. Again, a ring of local anesthetic should be subcutaneously injected in a ring around the arm to block the intercostobrachial nerve to provide tourniquet-related pain relief. Recent case reports and a small series have described utilizing ultrasound in visualizing the interscalene approach to the brachial plexus [92–95].

Paravertebral Block

With the ability to target specific dermatomes, single-sided paravertebral blockade is indicated for patients undergoing renal surgery, thoracotomy, unilateral abdominal procedures such as cholecystectomy, and even inguinal surgery [96, 97]. The bilateral approach expands its use to procedures that cross or involve the midline. Lönnqvist and others demonstrated continuous paravertebral blockade to be superior to continuous epidural blockade in reducing morphine requirements in children

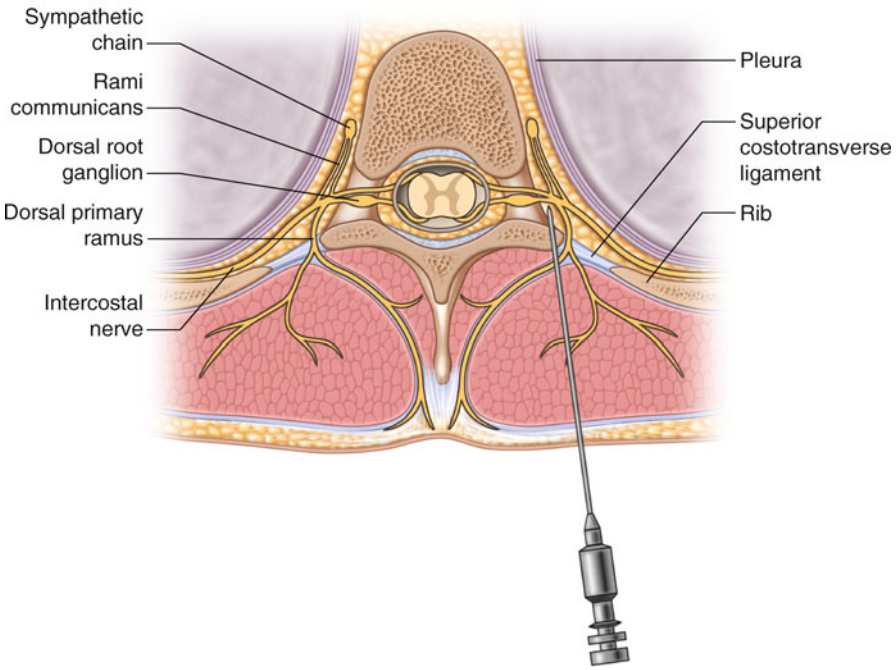

Fig. 27.5 Paravertebral block

undergoing renal surgery [96, 97]. A more recent study by Berta and others demonstrated single-shot paravertebral block to have a median duration of 600 min in pediatric patients undergoing major renal surgery ([98].

A wedge-shaped area, the paravertebral space, is bound anteriorly by the parietal pleural, posteriorly by the superior costotransverse ligament, laterally by the posterior intercostal membrane, and medially by the vertebra (Fig. 27.5). The space contains the spinal root as it emerges from the intervertebral foramina, its dorsal and ventral rami, and the sympathetic chain. Blockade may involve several dermatomes and can produce sensory, sympathetic, and motor blockade. In the pediatric population, the block is usually performed under general anesthesia with the patient in the lateral position. After establishing the midline, the point of lateral approach is estimated by measuring the distance between spinous processes. The needle is inserted perpendicular to skin until it contacts the transverse process. It is then slightly retraced and directed caudal to walk off the process. In adults, the needle is then advanced 1 cm deeper than the transverse process, while in children, the space is usually more superficial. Further confirmation may be obtained by a loss of resistance technique similar to epidural placement. A "pop" may be felt as the needle penetrates the paravertebral space. At this point, a drop of sterile fluid is placed at the needle hub, and the patient is given a deep breath to rule out intrapleural placement. A 22-gauge blunt needle is then used to inject 0.5 ml/kg of local anesthetic for unilateral blockade.

Ropivacaine 0.2% or bupivacaine 0.25% is typically used. A Tuohy needle is used to thread a catheter for continuous techniques. Typical infusion rates are 0.25 ml/kg/h in children and 0.2 ml/kg/h in infants of 0.1–0.125% local anesthetic.

The proximity of this block to the epidural space leads to the possibility of inadvertent epidural or spinal blockade resulting in hypotension or rarely a "high spinal" [99, 100]. Other complications include vascular or pleural puncture and pneumothorax [101, 102]. A 10.7% failure rate in adults and 6.2% in children was demonstrated in one series of 367 patients by Lönnqvist and others [103]. However, the use of bilateral paravertebral technique doubled the likelihood of advertent vascular puncture (9 vs. 5%) and an eightfold increase in pleural puncture and pneumothorax (3 vs. 0.4%), when compared with unilateral blocks [104].

Transversus Abdominis Plane Block

As a landmark-based technique, the transversus abdominis plane block (TAP) has provided excellent analgesia in adults undergoing lower abdominal surgery including hernia repair, appendectomy, abdominal hysterectomy, and cesarean section [105–108]. Application to the pediatric population, in which landmarks are difficult or impossible to palpate, has been eased by the use of ultrasound [109, 110]. The TAP block is especially useful in cases where neuraxial blockade is contraindicated [110]. It may substitute for ilioinguinal/iliohypogastric block and can also provide analgesia for more superior abdominal incisions from laparotomy or laparoscopy. Incisional pain can be well controlled, but the block is ineffective for visceral pain [109–111].

The anterolateral abdominal wall is innervated by the anterior rami of T_7–L_1 and includes the ilioinguinal, iliohypogastric, intercostal, and subcostal nerves (Fig. 27.6) [112]. These nerves travel in the intercostal space before entering the abdominal wall between the internal oblique and transversus abdominis muscle. This plane serves as the target for the TAP block. The landmark technique involves locating the lumbar triangle of Petit. The base of the triangle lies on the highest point of the iliac crest, and the apex is at the costal margin. Anterior and posterior borders include the external oblique muscle and latissimus dorsi muscle, respectively. A blunt, 22-gauge, 2-in. needle is inserted in this location and passes through the external oblique, then internal oblique (Fig. 27.6). After these two "pops" are appreciated, local anesthetic is injected with obvious care not to exceed toxic levels. A bilateral block may be used for midline incisions or procedures involving both sides [108, 110, 113, 114].

Aside from real-time visualization, ultrasound offers a distinct advantage for this block in the pediatric population as the triangle of Petit is difficult to ascertain in children and loss of resistance through less developed internal and external oblique muscles can be difficult to appreciate [112, 115]. Placement of the probe in the transverse plane above the iliac crest usually provides excellent visualization of the

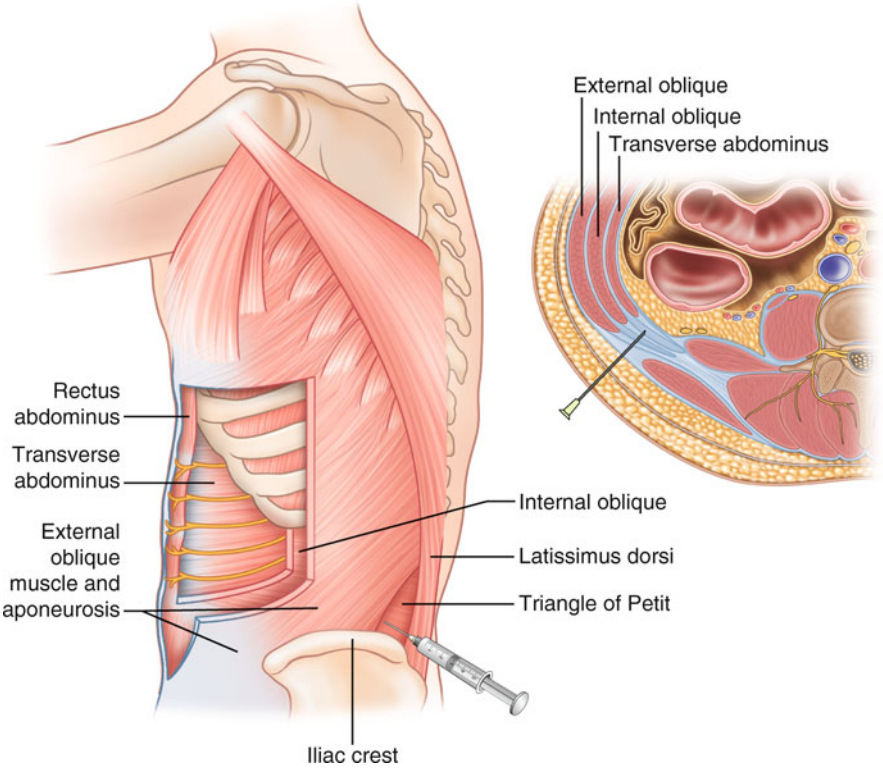

Fig. 27.6 TAP block

external and internal obliques, transversus abdominis, and peritoneum, though the probe may need to be directed more medially in some patients. Local anesthetic is deposited as the needle tip is visualized deep to the internal oblique. Spread within the plane confirms accurate placement. A Tuohy needle is used to place a continuous catheter 2–3 cm beyond the needle tip if prolonged analgesia is required.

Complications are similar to those associated with ilioinguinal blockade including peritoneal perforation and femoral nerve palsy [108, 113, 116]. The catheter-based technique has a theoretical increased risk of infection. There are no reported complications with the ultrasound-guided technique [108, 113].

Ilioinguinal/Iliohypogastric Block

Analgesia for inguinal hernia repair, hydrocelectomy, and orchiopexy is provided by an ilioinguinal/iliohypogastric (ILIH) block [117–119]. Originating from the lumbar plexus, the ilioinguinal and iliohypogastric nerves pass superficial to the

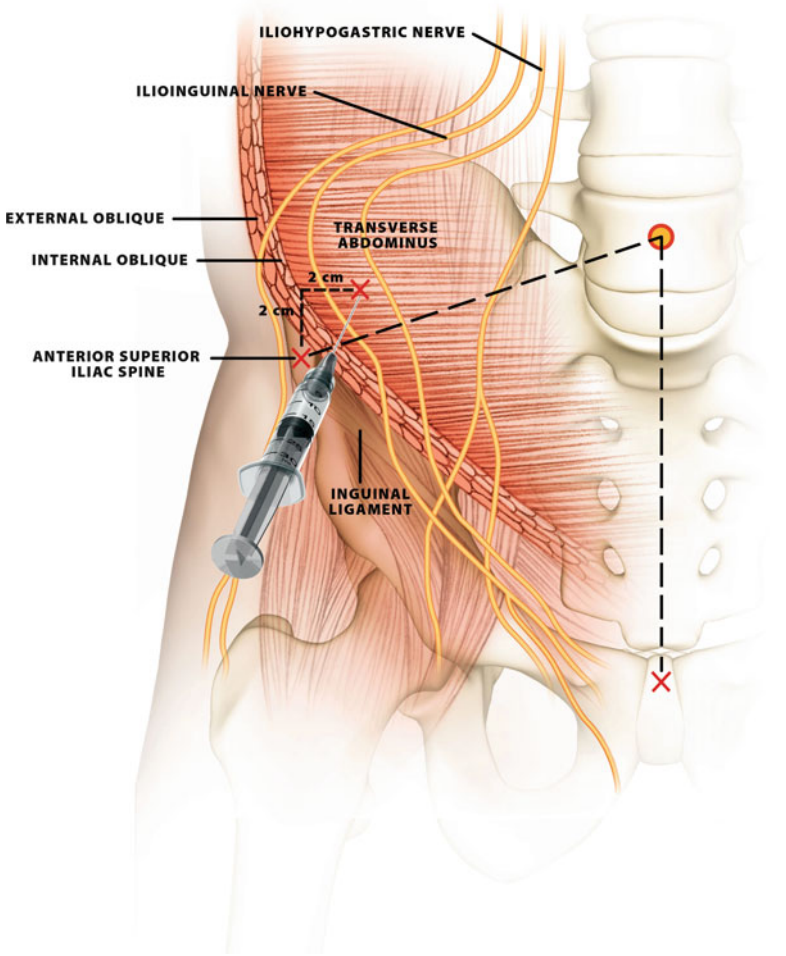

Fig. 27.7 Ilioinguinal/iliohypogastric block [145]

transversus abdominis near the anterior superior iliac spine (Fig. 27.7). These nerves can be blocked at this site before separating. The iliohypogastric nerve supplies skin over the lower anterior abdominal wall, while the ilioinguinal supplies skin over the scrotum or labium majus.

A blunt 22–25-gauge needle is inserted 1 cm superior and 1 cm medial to the anterior superior iliac spine (ASIS) (Fig. 27.7). A field block is then performed directing the needle parallel to the muscle wall in the direction of the ASIS. The needle is withdrawn while injecting anesthetic and redirected toward the inguinal ligament with care not to puncture the ligament. Penetration of the oblique muscles results in a characteristic "pop" after while local anesthetic is again injected. The

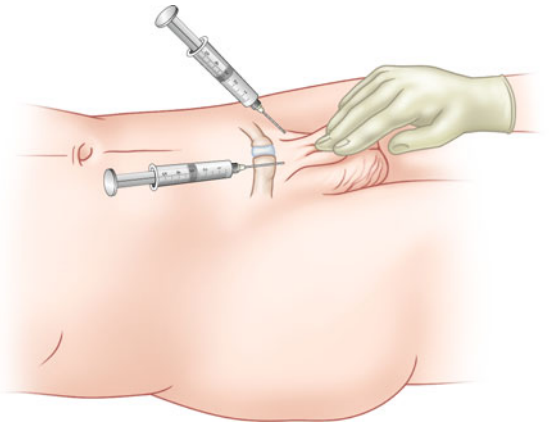

Fig. 27.8 Penile nerve block

block can also be performed postsurgically by the surgeon under direct vision. Bupivacaine 0.25% or ropivacaine 0.2 or 0.5% are typically used.

Ultrasound guidance involves direct visualization of the nerve or nerves by placement of the probe just medial to the superior aspect of the ASIS. An out-of-plane technique is typically employed as the nerve's proximity to the ASIS can make the in-plane technique challenging [120]. At this location, the nerves are typically less than 1 cm deep and run between the internal oblique and transversus abdominis muscle.

Serious complications are rare and include small-bowel or colonic perforation [121]. Transient femoral blockade resulting in motor weakness of the quadriceps can occur in up to 5% of patients if local anesthetic tracks inferior to the inguinal ligament [122].

Penile Nerve Block

Arising from the sacral plexus, innervation of the distal two thirds of the penis is supplied by branches of the pudendal nerve known as dorsal nerves. The nerves are surrounded by Buck's fascia and are near dorsal vessels (Fig. 27.8). Various techniques exist for anesthetizing these nerves for intraoperative and postoperative pain secondary to circumcision and uncomplicated hypospadias repair. They include application of topical cream, subcutaneous ring block, dorsal nerve block, and suprapubic nerve block [66, 123–125]. Numerous studies have shown the ring block to be more effective than the other techniques [123, 125].

Application of topical cream is the simplest method and has been employed because of its ability to penetrate intact foreskin [125]. As absorption may be increased through mucous membranes, care must be taken to use the minimum amount necessary. Subcutaneous ring block involves placing a skin wheal of local

anesthetic circumferentially around the base of the penis [26]. Injection of local anesthetic to the penis bilaterally at the symphysis pubis is known as the dorsal penile block. With downward traction of the penis, a 25-gauge needle is directed medially and caudally until Buck's fascia is penetrated at 10:30 and 1:30 and a characteristic "pop" is felt. Frequent aspiration is necessary due to the close proximity of the dorsal vessels at this location [126–129].

Most sources recommend the avoidance of epinephrine with these blocks as vasoconstriction can theoretically result in necrosis. A volume of 0.1 ml/kg of bupivacaine 0.25 to 0.5% or ropivacaine 0.2% is typically used and provides approximately 4–6 h of analgesia. Complications include hematoma formation resulting in necrosis, intravascular injection, and tissue edema affecting surgical conditions [118, 130, 131]. A recent study examined the role of US and found improved efficacy in the block [132].

Caudal Block

Although regional block needles are used in the performance of the pediatric caudal block, a number of studies advocate the use of styleted, short-beveled, 22-gauge needles [133–135], as the styleted needle may reduce the risk of introduction of a dermal plug into the caudal space [134]. The approach to the caudal canal is dependent upon proper angle of the needle as parallel insertion to the sacrum is required through the sacrococcygeal membrane [Fig. 27.9]. Final needle placement is dependent upon a "pop" as the blunt needle pierces the sacrococcygeal membrane. Aspiration should be performed prior to injection of the local anesthetic solution. A test dose including epinephrine (0.5 mcg/kg) helps identify that the needle is not in the intravascular or intraosseous space. During injection, the lack of subcutaneous swelling is a helpful sign of proper needle placement. Relaxation of the anal sphincter also predicts successful blockade [136].

Extension of the Caudal Catheter into the Lumbar or Thoracic Regions

Caudal catheters were used in the past in adults but lost their popularity with the development of lumbar and thoracic approaches to the epidural space [137]. However, there has been a recent resurgence in caudal catheter epidural in neonates and in infants as they can be used to facilitate the surgical anesthetic and be a component of a postoperative analgesia regimen. The caudal canal in neonates can allow access to the lumbar and thoracic segments with minimal resistance in passage of the catheter [137–141]. However, in older patients, the addition of fibrous and fatty tissue and, moreover, the development of septal membranes in the epidural space can impede caudal catheter advancement [142, 143].

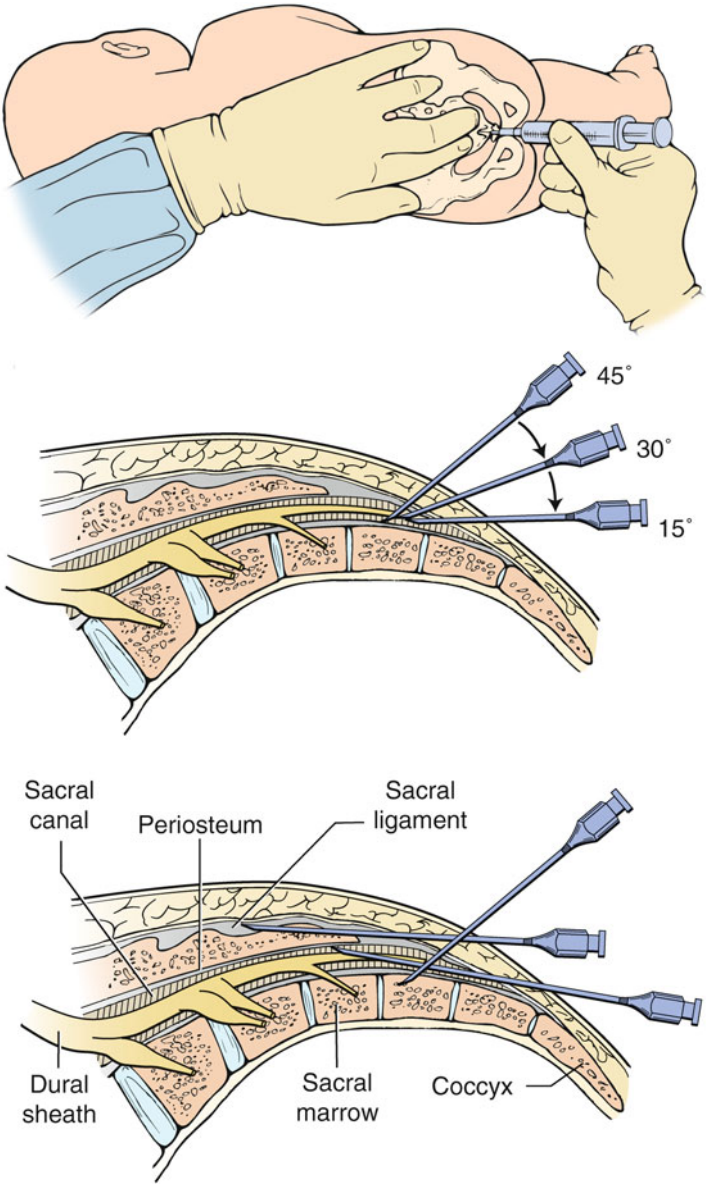

Fig. 27.9 Caudal block

Summary

The benefits of regional analgesia in the management of postoperative pain are recognized. Despite many reported advantages, the use of peripheral nerve blocks in perioperative care in children is underutilized. Although these regional techniques

are safe, they are not without risk [51, 144]. It is hopeful that the use of ultrasonography should decrease some of these risks [41]. Regional anesthesia can be an important component to multimodal analgesia. Certainly, the role of the parents regarding postoperative instructions is important to transition the analgesic regimen as the block wanes. However, these postoperative analgesia instructions should not be significantly different than what is currently employed for pediatric patients following general anesthesia. In any perioperative plan of care, the risks and benefits of any technique lie with the skill and experience of the caregiver. Nevertheless, regional anesthesia is an effective method of providing postoperative analgesia to the pediatric patient.

Multiple-Choice Questions

1. For a 1 month old undergoing bilateral hernia repair, the best choice for postoperative pain control is:
   (a) Hydromorphone 0.1 mg/kg given by slow intravenous push
   (b) Meperidine 1 ml given intramuscularly
   (c) Penile nerve block performed by the surgeon
   (d) Ilioinguinal/iliohypogastric block

2. Complications of an ilioinguinal/iliohypogastric block include:
   (a) Small-bowel perforation
   (b) Necrosis of the penis
   (c) Paralysis and weakness of hamstring muscles
   (d) Urinary retention

3. The dose of local anesthetics in infants is:
   (a) Higher than in adults because of increase in VDSS
   (b) Lower than in adults because of deficiency of alpha glycoprotein
   (c) Same as in adults
   (d) Unrelated to liver and kidney function

4. Ultrasonography has been shown to:
   (a) Definitely increase the incidence of complications in regional anesthesia
   (b) Appears to be of benefit in the success of performance of certain blocks
   (c) Should be used whenever regional anesthesia is contemplated in pediatric patients
   (d) Can cause problems with regional anesthesia because of “anesthesiologist distraction” similar to that seen with TEE

5. A 10 year old is involved in a bicycle accident that results in a broken humerus. He is healthy and has no prior medical history.
   (a) The best way to provide analgesia is to provide the patient with a PCA.
   (b) Performing a parascalene block on this patient after general anesthesia is induced is contraindicated.

(c) A parascalene approach to an interscalene block will provide excellent analgesia in the postoperative period.
(d) A musculocutaneous block is needed to complete an interscalene block.

6. The benefit of adding epinephrine to the local anesthetic solution includes all of the reasons below EXCEPT:
   (a) The tachycardia produced by intravascular epinephrine is advantageous in pediatric patients because their cardiac output is rate limited.
   (b) Epinephrine will prolong the action of the local anesthetic.
   (c) Epinephrine will reveal intravascular placement of the local anesthetic.
   (d) Epinephrine is relatively contraindicated in a penile block.

7. A 6-year-old girl is in the ER with a superficial scratch on her face. The plastic surgeon says he has to put a few sutures in to improve healing. She last ate a large meal 2 h ago.
   (a) The ideal anesthetic is to sedate her and perform a supratrochlear block.
   (b) Use TAC to provide local anesthesia for the skin on her face.
   (c) TAC is contraindicated when the skin is not intact.
   (d) Do a TAP block.

8. Eutectic mixtures of local anesthetics are:
   (a) Only used to "numb up the skin" prior to IV starts.
   (b) Should not be used on the face.
   (c) Can be used without fear of toxicity.
   (d) Commercially available mixtures of lidocaine and prilocaine, and lidocaine and tetracaine have been used successfully in pediatric patients.

9. A 2-year-old patient is scheduled to undergo resection of a Wilm's tumor. An optimal anesthetic would include which of the following regional techniques for postoperative pain management:
   (a) Caudal block with the addition of clonidine
   (b) Continuous lumbar epidural block with an infusion of lidocaine
   (c) Paravertebral block
   (d) Intercostal block

10. Complications of paravertebral blocks include all of the following:
    (a) High spinal
    (b) Pneumothorax
    (c) Epidural block
    (d) Puncture of large bowel

11. For a patient weighing 12.5 kg, the dose of local anesthetic most appropriate for a paravertebral block is:
    (a) Bupivacaine 0.25% 0.5 ml/kg
    (b) Levobupivacaine 0.5 mg/kg
    (c) Ropivacaine 0.5% 0.5 ml/kg
    (d) Lidocaine 2% 3 mg/kg

12. TAP block:
    (a) Is so called because a "pop" is felt as the needle traverses the external oblique muscle.
    (b) It is an easy block to perform on newborns because landmarks are easy to palpate.
    (c) Is excellent for relieving visceral pain.
    (d) Can be performed bilaterally to relieve the pain of midline incisions.

13. The following statements are true about the TAP block EXCEPT:
    (a) Is best performed in the lateral position.
    (b) Targets the posterior rami of T_7–L_1.
    (c) The nerves targeted by the TAP block travel in a plane between the internal oblique and the transversus abdominis muscles.
    (d) Ultrasonography is useful in locating the triangle of Petit.

14. The following statements are false about the ilioinguinal/iliohypogastric block EXCEPT:
    (a) Targets nerves that pass deep to the transversus abdominis muscle near the anterior superior iliac spine.
    (b) The ilioinguinal nerve supplies the lower anterior abdominal wall.
    (c) This block cannot be performed by the surgeon.
    (d) Is indicated for patients undergoing hydrocelectomy.

15. Maximum dose for:
    (a) Bupivacaine without epinephrine is 2 mg/kg.
    (b) Bupivacaine with epinephrine is 3.5 mg/kg.
    (c) Lidocaine is 7 mg/kg with epinephrine.
    (d) 2-Chlorprocaine with epinephrine is 10 mg/kg.

Answers:

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

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28

Obstetric Anesthesiology

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Contents

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A.D. Kaye et al. (eds.), *Essentials of Regional Anesthesia*,
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Obstetric anesthesiology gained noble notoriety when Prince Albert requested Dr. John Snow to provide analgesia for Queen Victoria during the birth of Prince Leopold. This unique beginning is reflected by the continuing uncommon nature of the practice [1].

No hard guidelines can be established to govern the conduct of obstetric anesthesia because each case is truly distinct and is best served by tailored response to the individual circumstances presented. Answers are often unclear or drift and put the capability of the anesthesia provider in focus because the outcomes for two patients are at risk. Providers of obstetric anesthesiology commonly use regional anesthesia techniques to provide care for their patients. This represents a growing trend based on the belief that there is an increased safety profile and maternal satisfaction associated with using regional anesthesia for the delivery process [2]. The classic technical skills required are mastered through repetition and training although the use of ultrasound in the labor suite can be anticipated to become more prevalent in order to facilitate catheter placement [3]. Historically, the suggested learning curve for proficiency is 60 regional anesthetic placements [4, 5].

The remarkable and persistent myth of painless childbirth has been the natural history still propagated despite the cumulative evidence to the contrary. Consequently, patient anxiety about accepting regional anesthesia remains challenging because of complex familial, personal or cultural factors frequently inconsistent with values or perception of the provider. How to lower these anxieties lacks a simple answer. Impressions made by the provider during first communications can relieve apprehensions and create a mutual understanding that is a functional anxiolytic without pharmacologic administration. The decision to use or decline regional anesthesia will always be an individual patient preference involving more complex variables than can be succinctly summarized. The usual elements of informed consent must always be respected, but the patient in overwhelming labor pain is frequently the patient who has reconsidered an earlier decision to decline regional anesthesia.

It seems inconceivable that any laboring patient would wish to suffer pain approximating that of distal amputation of a finger as depicted by the pain rating

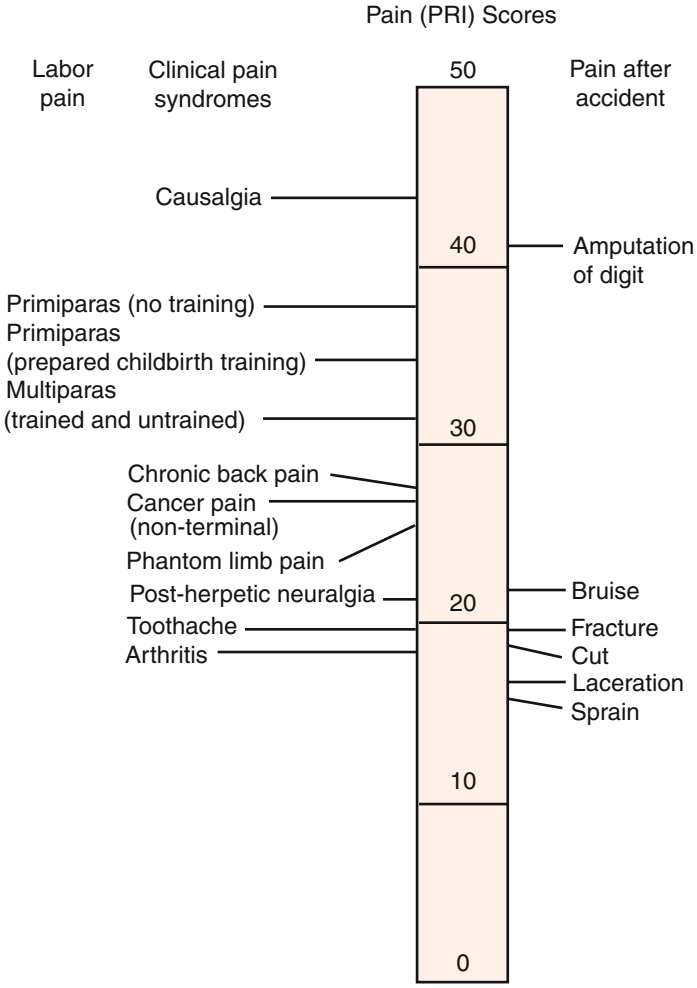

Fig. 28.1 Pain rating index

index in Fig. 28.1 [6]. Indeed the American College of Obstetricians and Gynecologists has issued position statements reflecting support for provision of pain relief during labor that requires only a patient request [7, 8]. Pain has personal meaning to each patient and once their tolerance has been met or their threshold exceeded, then the option for regional anesthesia should be quickly offered. To not do so has ethical implications, and current obstetric anesthesia practices have evolved to make available regional anesthesia earlier and later than has been the historical custom. Active management of labor by the obstetrical team makes the need for waiting until prescribed cervical dilation has been met a moot point. If the anesthesia provider is comfortable and capable, then late intervention also has no limits relative to dilation or station.

Communication is essential to providing safe care for mother and fetus. Changes in the obstetric population and obstetric practice have increased the number of high risk patients likely to be encountered. If the risk of childbirth is increased, then the need to continually examine practice habits becomes important to assure a good outcome for mother and fetus. The ASA guidelines for obstetric anesthesia should always guide practice conduct [9]. Verbatim, the guidelines state "The choice of analgesic technique depends on the medical status of the patient, progress of labor, and resources at the facility. When sufficient resources (e.g., anesthesia and nursing staff) are available, neuraxial catheter techniques should be one of the analgesic options offered."

Physiologic Changes of Pregnancy

Increased maternal metabolic rate, cardiac modifications, and especially circulatory volume changes which mediate the hemodynamic response to neuraxial anesthesia are the focus of most obstetric anesthesia providers because the likelihood of these changes directly impacting the mother or fetus is common. Obviously, all organ systems are impacted by the pregnant state. Total body water change (6.5–8.5 L gain at term) is impressive and is a significant hypervolemic adaptation of pregnancy. Proper respect paid to the changes known to occur in each system with appropriate risk determinations, have made the danger of unintended morbidity a rare event. Multiple consensus summaries of important physiologic and anatomic changes are available for review [10–13].

Cardiovascular Changes During Pregnancy

- Systolic and diastolic blood pressure decrease until midpregnancy with a return to prepregnancy values by term. Lowest blood pressures occur at 16–20 weeks.
- Decreased systemic vascular resistance 20%. All vascular beds effected especially uterine which increases from 50 mL/min at 10 weeks to 500 mL/min at term. Increased progesterone is implicated as a smooth muscle relaxant.
- Increased intravascular volume by 25–40%. ECHO reveals 10% increased end diastolic volume.
- Increased heart rate by 15–20% and cardiac output by 30–50% which is due to stroke volume and is position dependent.
- Increased total blood volume by 25% and plasma volume by 40–45% which is reflected in increased SV.
- Aortocaval compression with supine position. CO decreases 14% when patient is supine rather than tilted.
- Autotransfusion with uterine contraction can also add a 300–500-mL bolus to the circulating volume which could add another 10% to cardiac output at the time of measurement.

Pulmonary Changes During Pregnancy

- Elevated diaphragm.
- Increased upper airway edema and friability.
- Decreased functional residual capacity 20%.
- Increased minute ventilation 40–50%.
- Partially compensated respiratory alkalosis, pCO2 27–32 mmHg, pH 7.40–7.45.
- Depleted bicarbonate, 17–22 mEq implying limited buffering capacity.
- Oxygen consumption is increased 20% which creates a tendency for rapid maternal and fetal hypoxia and maternal rapid desaturation during supine position and during intubation.

Hematologic/Laboratory Changes During Pregnancy

Physiology

- Physiologic anemia, hemoglobin 10–12 mg/dL. Indeed red cell mass is increased but plasma volume is increased much more dramatically which is commonly called dilutional anemia.
- Leukocytosis, WBC count 12,000–25,000.
- Slight decrease in platelet count.
- Fibrinogen doubled at term.
- Pregnancy is a hypercoagulable state with increases in most procoagulant factors and a decrease in some of the natural inhibitors. Factors I, VII, VIII, and XII increase. (Thromboembolic disease is the #1 cause for maternal death in the USA making obstetricians very much quick to utilize anti clotting drugs to manage at risk patients.)
- BUN and creatinine decrease as a result of increased glomerular filtration rate.

Gastrointestinal/Endocrine Changes During Pregnancy

- Decreased gastric motility and emptying representing an increased risk of aspiration due to progesterone effects. Gastrin secretion increases and motilin secretion decreases.
- Bowel displaced cephalad in third trimester.
- Normal pregnant woman will remain euthyroid.
- Pregnancy conveys resistance to insulin due to human placental lactogen.
- Neuraxial analgesia effectively mitigates many of the physiologic changes that can be detrimental to labor. If pain or stress causes maternal hyperventilation,

then hypocarbia results with a decrease in uterine blood flow. Hyperventilation also causes a left shift in the maternal oxyhemoglobin dissociation curve decreasing the transfer of oxygen across the placenta [14]. Neuraxial analgesia results in a decrease in maternal oxygen consumption [15].

– Cardiac output increases less in labor with neuraxial analgesia and the decrease in systemic vascular resistance is usually beneficial to preeclamptic parturients and fetuses [16, 17]. Neuraxial analgesia blunts the stress response during labor. The normal increase in maternal circulating norepinephrine and epinephrine levels decrease after neuraxial analgesia [18, 19].
– Neuraxial analgesia may have a beneficial effect on fetal heart rate patterns and be advantageous for the fetus with marginal uteroplacental circulation [20]. Neuraxial analgesia is associated with lower maternal, fetal, and neonatal lactic acid levels [21].

Neuraxial Analgesia and the Progress of Labor

Neuraxial labor analgesia remains a controversial subject regarding the potential to slow the progress of labor and resultant delivery. Investigations have concluded that neuraxial analgesia actually improves dysfunctional labor [22]. Recently, there has been a focal concern that neuraxial labor analgesia may prolong labor and increase the rate of operative delivery. Some observational studies have loosely associated neuraxial analgesia with prolonged labor and higher rates of instrument and cesarean delivery. There is no clear causal link to any of these findings. Controlling for the variable of early painful labor suggests that independent of neuraxial analgesia, parturients with early pain have a higher incidence of dystocia that would require instrumental deliveries [23]. Cesarean delivery rates differ markedly. Higher cesarean rates are only partially explained by patient characteristics but are greatly influenced by nonmedical factors such as provider density, private insurance, the capacity of the local health care system, and malpractice pressure. Areas with higher usage rates perform the intervention in medically less appropriate populations – that is, relatively healthier births – and do not see improvements in maternal or neonatal mortality [24].

The best available evidence has to be interpreted that neuraxial analgesia given when needed has no significant impact on cesarean section or instrument delivery. A meta-analysis concluded that neuraxial analgesia does not increase the risk of cesarean delivery [25].As shown by Wong et al. the provision of early analgesia via CSE will decrease total labor time compared to narcotic analgesia [26]. The beta-2 effect of epinephrine is to act as a known myometrial relaxant that could prolong labor and negatively impact the fetus due to increased oxygen consumption if maternal stress or pain responsible for raising epinephrine levels is not attenuated.

Long term, the likelihood of developing postpartum depression is reduced when adequate analgesia is provided intrapartum [27]. Within the 2009 health care reform package is a concentration on the complications of postpartum depression that will be emphasized as a forward focus for prevention. Cognitive function is also apparently impaired when a woman suffers through labor with inadequate analgesia [28].

Numerous articles have reported on the psychological, cultural, and emotional components of pain. Juhan described the individual's attitude toward pain as the main determinant in the regulation of stress hormones [29]. The idea that sensory experience is shaped by one's attitude and beliefs has become widely accepted [30]. The attitudes of those communicating with the individual who is experiencing pain contribute to the individual's regulation of that pain. Social support, a sense of control and empowerment, and planning are all vital determinants of an individual's experience of pain especially evident when a woman is in labor [31, 32].

Labor Pain Pathways

Labor pain is transmitted from low thoracic, high lumbar, and low sacral nerve roots which are segmentally involved as labor stages change (Fig. 28.2).

First stage labor pains are related to the physical process of cervical dilation and stretching associated with uterine contractions. By definition, this occurs with the onset of labor and culminates when the cervix is completely dilated. This pain is a nociceptive visceral pain mediated through nerve roots T10–L1. By convention, classic Friedman curve analysis depicting the progress of labor the graph is divided into latent phase and active phases of labor. Active phase is subdivided into acceleration, maximum slope, and deceleration. Dysfunctional labor patterns or falling off the curve represent a patient more likely to require frequent anesthetic intervention. Uterine pain fibers combine in the inferior hypogastric plexus and Frankenhauser's ganglia and can be blocked by a paracervical regional technique if the patient is in first stage labor. The lumbar paravertebral sympathetic chain is also

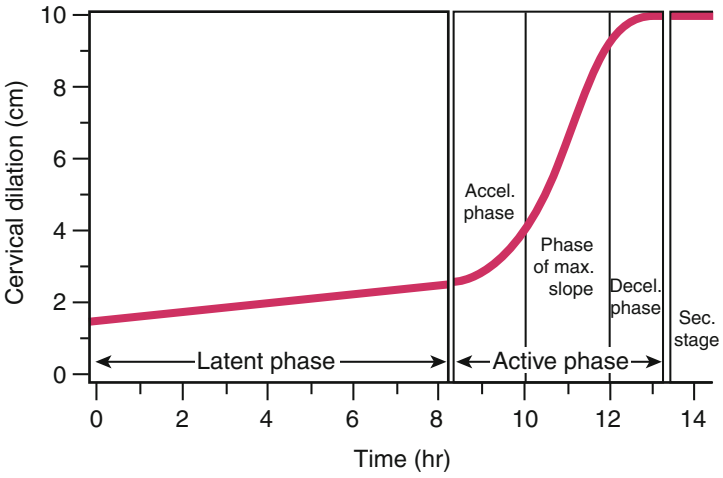

Fig. 28.2 Labor phases

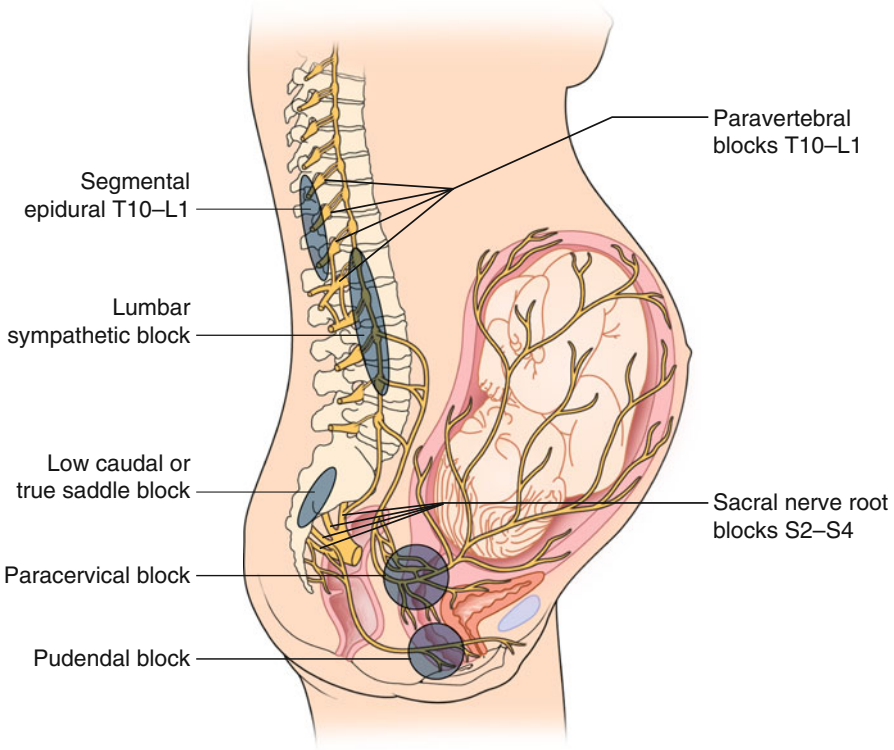

Fig. 28.3 Obstetric block anatomy

part of this process and conducts sympathetic efferent nociception. The T10–L1 dorsal nerve roots and their connection to the spinothalamic tract are the spinal cord destination for these first-stage nociceptive impulses which is also potentially an analgesic target for paravertebral block.

Second stage of labor is dominantly a somatic pain pathway conducted via the pudendal nerve with contributions from the S2–S4 roots. Second-stage pain most likely also continues with visceral pain associated with continued uterine contractions and is defined as commencing with complete dilation and terminating with delivery. An alternative block to relieve second stage labor pain is a pudendal block (Fig. 28.3).

Physiology of Labor Pain

By convention there are four major steps involved in the production of a pain reaction (1) transduction, (2) transmission, (3) perception, and (4) modulation. Transduction refers to the peripheral afferent conversion of a stimulus into an action

potential. The action potential is then transmitted via C fibers and A-delta fibers along the length of the neurons to cells in the spinal cord, specifically the dorsal horn tract of the spinal cord in Rexed's laminae.

Regional anesthesia functionally targets transduction and transmission when utilizing local anesthetic agents and the addition of narcotic or adjuvants like alpha or serotonin receptor agents can additionally target modulation in the descending pathways where amplification or dampening of the pain signal is thought to occur. Perception is an intricate multifaceted conscious awareness that is based in very complex interactions of expectations, individual responses to pain or its relief, and communication of these experiences.

Drug Response in Pregnancy

It is commonly recognized that pregnancy impacts dose requirements to provide equivalent anesthesia and analgesia compared to the nonpregnant state due to anatomic and physiologic changes. Increased levels of progesterone and anatomic compression of the epidural space by venous engorgement are reasons long known for less local anesthetic being needed to provide equianalgesic coverage of targeted dermatomes compared to the nonpregnant state. Progesterone has been characterized as a brain anesthetic acting more like a sedative than a gestational hormone and has the potential to be additive with many anesthetic processes [33]. Altered volumes of distribution, changes in protein binding, and increased hepatic and renal blood flow certainly change drug pharmacokinetic behavior in the pregnant state and require fine tuning the doses administered to achieve the expected result.

Drug diffusion into the CSF from the epidural space may be exaggerated by the increased pressure due to the fetal growth occupying the abdominal cavity. Cephalad spread can be accentuated due to increased lumbar lordosis. Venous engorgement increases the risk for intravascular injection or more rapid uptake to the CNS, heart, or liver.

Highly protein-bound drugs function differently due to the oncotic pressure decrease that is a usual physiologic change of pregnancy resulting in an increased free fraction of circulating drug.

Maternal endorphin and enkephalin levels are also higher in pregnancy, which provide natural pain relief and decrease drug levels needed to provide analgesia in the nonpregnant state.

Altered drug response is more crucial to the fetus because the uteroplacental perfusion is subject to hypotensive perturbation that can have profound fetal effects. Maternal, fetal, and placental issues along with drug pharmacology influence the passage of drugs from maternal circulation to the fetus. Uterine blood flow, molecular size of the drug, ionization, lipophilicity of the drug, pKa of the drug, and maternal/fetal pH are the controllable factors influencing how much drug the baby encounters. Ion trapping as predicted by an acidotic fetal environment is

the principal contingency to be aware of when choosing drugs to administer in large quantities such as for an impending cesarean section [34].

Lipophilicity determines the behavior and duration of narcotics administered in the epidural space, and there are functionally no changes when pregnant other than repeating that there is a decreased dose requirement to provide obstetric analgesia equivalent to the nonpregnant state [35]. Amide local anesthetics are metabolized by the liver and excreted by the kidney and have little pharmacologic change in pregnancy. Ester local anesthetics are degraded by pseudocholinesterases, which are decreased during pregnancy but not to a sufficient extent to influence the choice of local anesthetic.

Anesthesia for Labor

Epidurals or combined spinal-epidurals (CSE) offer the best solution for a labor expected to end with vaginal delivery. A probable duration of 12 h in labor is reasonable although highly variable. Currently utilized local anesthetic and narcotic solutions permit neuraxial techniques to achieve a controllable segmental block of sympathetic and sensory nerves with relative motor neural-function sparing. Solutions injected into the epidural space travel cephalad and caudad in the path of least resistance to provide analgesia to the dermatomes of interest [36]. The risk of incompletely or not blocking a painful area is very real and could persist despite all rescue maneuvers and should be discussed prominently in the informed consent process.

The lateral path of local anesthetic spreads through intervertebral foramina to the dural cuff. The local anesthetic can spread further through the dural cuff via arachnoid villa to the CSF. The foramen magnum and sacral foramina represent the cephalad and caudad limits of epidural local anesthetic spread. Epidural block occurs at mixed spinal nerves, dorsal root ganglia, and the spinal cord [37].

Epidural or CSE are usually placed at the L2–L3, L3–L4 or L4–L5 interspace which represents a midpoint between the targeted dermatomes for first-stage and second-stage labor relief. These interspaces are usually identified using the intercristal or Tuffier's line to identify the L4 vertebral body and palpating above or below for the most favorable interspace placement. Successful identification is presumptive but remains the best available technique unless ultrasound is available. Unfortunately, when misidentification occurs, the trend is to miss at a higher space than appreciated which could lead to serious morbidity [38–41].

A rule of thumb is that 1–2 mL of epidural local anesthetic per segment to be blocked will be needed to establish the desired level of conduction analgesia. Modifiers such as body habitus or medical status will change the dosing for each individual. Morbid obesity has been shown to cause a dramatic minimum local anesthetic concentration change requiring approximately 40% less infusate to achieve desired anesthesia. Obesity usually results in a 2-dermatomal higher spread from an equal injection volume in a nonobese patient [42]. Divided incremental

dosing should always be practiced but is especially emphasized in the obese patient. Obesity is a risk escalator for all morbidities in the pregnant patient.

Absolute and relative contraindications to epidural placement include patient refusal, infection at the site, uncorrected hypovolemia or coagulopathy, significant thrombocytopenia (<75,000), severe aortic or mitral stenosis, local anesthetic allergy, increased ICP and neurologic disease such as those which are demyelinating and likely to be adversely effected by local anesthetics.

Low-dose local anesthetic in combination with low-dose narcotic solutions provides the most comfort and satisfaction to the patient especially when a PCEA mode is utilized. Bupivacaine and ropivacaine have been the local anesthetics of choice because they have intermediate duration and are principally sensory in their clinical effect. Fentanyl or sufentanil are the narcotic additives with the most clinical use. Minimum local anesthetic concentrations necessary to maintain analgesia in labor have been established as 0.11% for ropivacaine and 0.067% for bupivacaine [43].

CSE can be argued to be the single best choice for labor analgesia since Wong has shown that shorter labor, better analgesia, and no change in operative delivery exist even with early neuraxial analgesia [26]. Earlier work had also suggested that combined spinal-epidural was associated with a 1 cm/h more rapid cervical dilation in nulliparous women [44]. Camian covered the validity of the published studies in detail and provided careful explanation to account for confounding findings in his review of regional anesthesia and analgesia for labor and delivery [45]. This information continues to be dynamic and as such will not have universal acceptance or interpretation. The clear consensus is that neuraxial anesthesia is not detrimental to labor but is more likely beneficial to safe outcomes and satisfaction.

The optimal recipe for labor analgesia has not been agreed upon and multiple combinations of local anesthetic and narcotic have enjoyed success in the labor suite. Cost and perceived difference in the incidence of side effects are likely to drive the choice of solutions used by any particular practice.

Low-range dosing with either ropivacaine or bupivacaine at 0.625% combined with fentanyl 2–3 mcg/mL or sufentanil 0.3–0.4 mcg/mL have had widespread favorable results as a continuous or PCEA infusion. I utilize a 0.1% ropivacaine plus 2 mcg/mL hydromorphone for anticipated prolonged deliveries and have received very positive patient feedback on its analgesic effect with no evidence of fetal depression.

A systematic review by Halpern and Carvalho concluded that background infusions and larger bolus settings for PCEA resulted in better analgesia. High-volume, dilute local anesthetic solutions of ropivacaine or bupivacaine were the most successful strategy and met more clinical goals related to maternal satisfaction, lack of motor block, and the need for clinician rescue intervention [46].

Saddle block for impending delivery is a technique to be considered when delivery is imminent no more than 90 min from placement. Low-dose hyperbaric bupivacaine can provide second-stage labor analgesia utilizing a small spinal needle rather than risking a dural puncture with a larger epidural needle in a patient likely to be a moving target while attempting regional anesthesia. L4–L5 or L5–S1 via a Taylor approach are interspace targets to consider. Saddle block can also be used to provide

immediate analgesia for a parturient unable to cooperate with positioning requests due to her labor pain but is unlikely to deliver before the spinal anesthetic wears off. After the spinal has achieved its desired analgesic effect, then the patient may be positioned more comfortably for epidural placement intended to last the duration of labor. Saddle-block dosing is also employed for cerclage placement for the patient with an incompetent cervix. Dosing for labor analgesia is 2.5–5 mg of bupivacaine with fentanyl 5–10 mcg or sufentanil 2.5–5 mcg added. Successful analgesic dosing requirement is likely to be on the lower end for either local anesthetic or narcotic.

Motor blocking is not desired but is a minimal detriment to pushing in labor since successful pushing is done more by diaphragmatic force than abdominal muscle contraction force [47]. In fact, parturients with spinal cord injury who are unable to sense contractions or are unable to push voluntarily deliver vaginally without difficulty. It does seem that despite its long history in the labor suite that coached maternal expulsive efforts do little to speed delivery. Nonetheless, motor block should be avoided if possible because it can increase the length of the second stage of labor.

Anesthesia for Cesarean Section

Cesarean delivery accounts for at least 31% of the births in the USA and is likely to become a higher percentage in the future due to the litigious risk perceived by obstetricians [48]. An emerging trend is that of the primary elective cesarean delivery for maternal preference independent of maternal or fetal medical need. Is this because the perceived risk of obstetric anesthesia and operative delivery by the patient is so low? ACOG has issued a qualified acceptance of this preference: "Acknowledgement of the importance of patient autonomy and increased patient access to information has prompted more patient-generated requests for surgical interventions not necessarily recommended by their physicians. Decision making in obstetrics and gynecology should be guided by the ethical principles of respect for patient autonomy, beneficence, nonmaleficence, justice, and veracity. Each physician should exercise judgment when determining whether information presented to the patient is adequate. When working with a patient to make decisions about surgery, it is important for obstetricians and gynecologists to take a broad view of the consequences of surgical treatment and to acknowledge the lack of firm evidence for the benefit of one approach over another when evidence is limited" [49].

The trend of whether to offer spinal or epidural for elective cesarean delivery is continuing to favor the use of spinal anesthesia. Many anesthesia providers think that spinal is a simpler technique, allows for rapid administration and onset of surgical block, reduces systemic toxicity and has an increased density of block which provides better comfort to the patient [50]. A survey of anesthesia providers for cesarean delivery in the USA revealed an 85% preference to administer a spinal, 11% choosing combined spinal-epidural, and 4% choosing epidural [51]. Confidence that there will be no complications and that your colleague obstetrician

can complete the operation within the time offered by one shot spinal are obviously key to choosing this pathway.

One meta-analysis suggests that fetal blood gas results favor epidural and general over spinal anesthesia [52]. When hypotension is controlled and phenylephrine is utilized as the vasopressor of choice then the fetal blood gas is more favorable when utilizing a spinal anesthetic technique [53]. Phenylephrine appears to be the vasopressor of choice to treat hypotension for surgical anesthesia since it has the most demonstrable benefit to fetal oxygenation. From our own lab in the dual perfused, single-isolated cotyledon, human placental model, exposure of the maternal circulation to ephedrine and phenylephrine caused an increase in fetal arterial perfusion pressure, whereas exposure to norepinephrine, epinephrine, and methoxamine did not. The pharmacodynamic mechanisms underlying these differences have yet to be explained. Thus, the clinical implications of the findings are as yet unclear [54]. The best management strategy is clearly to maintain maternal normotension utilizing whatever vasopressor is most effective [55].

Top-up dosing of in situ epidurals for failed trials of labor and general anesthetics for emergency cesarean delivery when medical contraindications to neuraxial anesthesia are present will cover the great majority of techniques needed in the operating room for nonscheduled cesarean operative cases.

Case urgency may be the driving force for choosing a technique, but in even the most extreme emergencies, the fall back should always be to that technique which the provider is most comfortable performing. Taking care of the mother and optimizing her physiology is what is best for the fetus and constitutes fetal resuscitation that may buy time in the operating room for more orderly provision of surgical anesthesia. The advocated 30-min declaration to delivery is a soft guideline that is often not achievable, but is a widely accepted community goal despite the lack of clinical evidence showing better fetal outcomes [56]. On a case-by-case basis, all the currently available data should be interpreted with caution and each declared urgent or emergent delivery accomplished as expeditiously as can be safely done [57]. Approximately 1% or less of cesarean deliveries will be crash sections.

Abnormal fetal heart rate tracings will be the number one cause for rapid transport to the operating room. Placental abruption, cord prolapse, preeclampsia, placenta previa and failed instrument delivery are probable causes for the urgent declaration [58].

There are many recognized combinations of local anesthetic/opioid for spinal anesthesia to cover cesarean delivery. Unless the provider mistakenly uses an exceptionally large dose of local anesthetic, total spinal is a rare complication (1:10,000), which is modifiable by utilizing hyperbaric preparations to reduce this risk when performing in an emergent setting.

Factors known to impact or suggested to influence the height of the block achieved after injection include volume of cerebrospinal fluid in the lumbosacral region, vertebral column length, baricity of the solution, volume of the solution, and speed of injection. Patient height and weight have never been strongly linked to influencing the spread of the local anesthetic to the heights desired [59].

Even if a T6 sensory tested level is documented, patients still have a significant risk of visceral pain, which interestingly represents the greatest obstetric patient fear in a survey of preferences to control in order to optimize their surgical experience [60]. Pruritus, PONV, and other variables ranked much lower than fear of pain. Patients reported a willingness to accept a pain VAS score of 5 in the study design. Surgical anesthesia should not be compromised due to concerns about hypotension and its impact on fetal perfusion. Hypotension is easily treatable and maternal pain that needs rescue may then represent a greater threat to fetal compromise than would have the hypotension immediately prior to delivery.

The spinal drug of choice is usually hyperbaric bupivacaine in doses between 7.5 and 15 mg. However, in mothers who receive less than 10 mg, there is a 71% risk of intraoperative pain [61]. Bupivacaine administered at doses less than 12.5 mg has not been found to abolish visceral pain [62]. The ED95 providing effective spinal block of women undergoing elective cesarean section has been calculated at 0.06 mg hyperbaric bupivacaine/cm height [63]. Given that the usual approach will be to combine a local anesthetic with narcotics for spinal cesarean delivery, the ED50 of 7.6 mg hyperbaric bupivacaine and the ED95 of 11.2 mg hyperbaric bupivacaine should be kept in mind, and the ED95 dosing plus narcotic chosen to more reliably provide comfort when the peritoneum is incised, the bladder flap is made, or the uterus is exteriorized. An important clinical investigation advising against the use of doses of intrathecal bupivacaine less than the ED95 has proven to be a good practice strategy to implement. An exception to this guideline would be if the spinal dosing is part of a CSE technique that would allow for extending the block [64].

Continuous spinal anesthesia is a method to provide dense titratable anesthesia for cesarean delivery. Currently the only method for providing continuous spinal anesthesia is limited to using an epidural needle to identify the intrathecal space and then threading a catheter through that needle to provide spinal anesthesia. This carries an enhanced risk of spinal headache from using a large-gauge needle to place the catheter. An ongoing government clinical trial (NCT00990574) with an over the needle Wiley catheter is being investigated at Stanford. The stated principal outcomes investigation is to assess hypotension occurrence and vasopressor need to treat comparing the continuous Wiley spinal catheter to single shot spinals at equal dosing. Headache incidence will also be evaluated. From a practical clinical perspective, a continuous spinal technique would seem to have many advantages over either single-shot spinal or epidural anesthesia. Continuous spinal would advantageously allow the development of a denser block compared to epidural, and also be incrementally dosable which represents an advantage over single-shot spinal dosing. Potentially, it would offer the opportunity to provide multiple doses for postoperative analgesia and utilize lower doses to minimize side effects after each dosing.

Epidural or CSE techniques possibly offer the advantage of less hypotension, which could make them a better choice for urgent operative delivery if maternal-fetal compromise allows time for their placement. Avoiding maternal hypotension greater than 20% of presenting baseline is a reasonable goal. Choosing opioids to add to the intrathecal dose can reduce local anesthetic hypotension but add their

own side effect complications. Narcotic-induced side effects include pruritus, nausea, vomiting, and respiratory depression. Even with low dosing of narcotics it is likely that the more minor side effects associated with intrathecal narcotics will be present. Respiratory depression in clinically relevant dosing is extremely unlikely. Severity of side effects may be dose dependent, but their occurrence seems to be a patient-dependent phenomenon [65]. Clinically relevant intrathecal dosing for morphine is 50–200 mcg, fentanyl range is 10–25 mcg, and sufentanil is 2.5–5 mcg. Increasing narcotic dosing does not improve analgesia nor significantly extend its duration, rather it is more likely to potentiate side effects.

Top up dosing of an indwelling epidural catheter for cesarean section can be accomplished rapidly with reported time requirement in the range of 3–14 min independent of prior sensory levels established for trial of labor. Proven epidurals can be rapidly dosed incrementally with little fear for toxicity or respiratory compromise. Lidocaine and chloroprocaine are the favored agents for fast onset, but bupivacaine, ropivacaine, and combinations of agents have all been reported to have successfully topped up an existing epidural catheter to surgical levels quickly [66–69]. Currently, the expected failure to achieve adequate surgical anesthesia with a labor epidural catheter is less than 5%, but that would mean approximately 1:20 parturients going for urgent cesarean delivery from labor could be expected to need conversion to general anesthesia or deep MAC supplementation [70]. This emphasizes the need to continually prove the analgesic efficacy of a labor catheter and to have multidisciplinary communication if there is perceived escalating risk of fetal compromise that would require urgent operative delivery. Delay in instituting anesthetic care and failure to communicate were standout reasons for obstetric anesthesiologist closed-claim liabilities [71].

Epidural catheter alone for elective cesarean section is an excellent option in the morbidly obese or massively morbidly obese patient because the sitting can be made higher in the lumbar spine where bony landmarks are more easily palpated and can be dosed slowly to mitigate the risks associated with neuraxial anesthesia in obese patients.

Combined spinal-epidural is an excellent choice for nonurgent and selected urgent presentations for cesarean delivery. Small spinal needles passing through a larger epidural needle that functions as a long introducer make this a popular choice for identifying the target rapidly in an obese patient. The rapid benefits of spinal anesthesia with the security of an epidural catheter and low risk for postoperative spinal headache represent the rationale most often given for choosing this anesthetic method. However, the currently accepted expectation for failure to obtain cerebrospinal fluid when attempting a CSE approximates 10%. Known causes for this phenomenon include inadequate needle length to penetrate the dura and lateral epidural space identification making it hard to position the port of the spinal needle in the subarachnoid space. There will also be a time lag to be able to prove that the epidural catheter can functionally extend the block until the initial spinal dosing has worn off (Table 28.1).

Table 28.1 Tabular guide for which technique to consider utilizing

Epidural	CSE	Spinal
Routine labor with anticipated imminent delivery	Urgent delivery vaginal delivery	Advanced dilation or saddle block station
Morbidly obese pt for C/S	Scoliosis	Routine C/S
Multiple prior C/S, therefore anticipated long case	Harrington rods or other spinal surgery	Continuous catheter for morbid obesity
Special considerations: L2–L3 catheter sitting for early or disproportionate pain which suggests possible C/S for dystocia	Prior failed epidural analgesia for trial of labor	Impending instrument delivery; supplemental for existing block
Site L3–L4 for more optimal development of comfort for first and second stage labor		

Adjuvant and Alternative OB Blocks

Paracervical block is essentially reserved for obstetricians and is unlikely to be performed due to the high incidence of fetal compromise reported even when the block is performed well. Paracervical block is an excellent intervention reserved for procedures like a dilation and curettage when there is a fetal demise. A field infiltration with local anesthetic via a spinal needle at the 4–5 o'clock and 7–8 o'clock positions where the cervix joins the vagina is all that is required. Appreciation of the correct tissue plane is difficult even when there is no presenting fetal head. Multiple reports of bad outcomes due to local anesthetic toxicity in a delivering fetus make this a very rare choice for analgesic intervention. This is not a block likely to be encountered and can only provide limited analgesia for the first stage of labor [72].

Pudendal block is also more likely to be performed by an obstetrician since the most successful block technique requires working within the vaginal cavity. This block would provide analgesia for the second stage of labor, but the usual clinical conditions of a patient unable to cooperate to facilitate the procedure make its use very rare. Palpation of the ischial spines and passing a spinal needle posterior to the bony process while requesting the writhing patient to be still make this a truly tough field block to accomplish. Vascular injury or injections with resultant local anesthetic toxicity are the most common complications noted in performing this block [73]. Transperineal approach to the space is possible but adjacent vascular structures are again a high risk to injure or inject and the success is low such that the risk-benefit ratio is unacceptable to offer to the patient for comfort. Saddle block anesthesia is much easier to perform and far more likely to be successful.

Paravertebral blocks do not have much utility since they are painful to perform and can only treat first-stage labor pain. The potential to place a catheter in the paravertebral space could increase their potential analgesic potential and reduce the number of needle sticks necessary to cover the T10–L1 dermatomes. Identification of the paravertebral space is done by walking a needle off the transverse process of the selected vertebral segment (usually L2), injecting local

anesthetic just anterior to the medial attachment of the psoas muscle bathing the exiting spinal nerve and the nearby sympathetic chain. Catheters can be placed in this anatomic space and a typical epidural solution infused to provide labor analgesia. The probability and predictability of success with this technique is less than that of an epidural and probably has little use as an alternative to epidural placement for anatomic abnormalities that might make an epidural placement difficult. Known common complications with this technique include painful placement and hypotension. Rare complications such as high spinal and retroperitoneal hematoma are known to have occurred [74].

Utilization of regional anesthetic techniques other than central neuraxial approach to provide comfort to parturients is gaining interest. Since the 2001 description of a TAP block technique by Rafi and succeeding descriptions of "how to" approaches for doing a TAP block [75, 76], multiple reports analyzing the utility of doing said block have populated the literature [77–79]. As might be expected, there exists disagreement as to how successful and therefore applicable the technique can be, but it seems to be gaining popularity because of how simple the block is to perform. TAP block functions like a distal intercostal block (ICB). The history of ICB in providing post operative analgesia for the abdomen is long [80, 81]. For each potential case, the question of how to perform the block and what to utilize when performing the block will be an individual decision. The greatest potential complication is the risk of local anesthetic toxicity whether it is systemic overdosing or unintended intravascular injection [82–84].

As the body of evidence mounts, it has become clear that TAP blocks can indeed be an excellent adjuvant for postoperative analgesic provision. Spinal and epidural anesthesia will remain the gold standards for providing obstetric anesthesia and analgesia, but having command of the ability to provide further analgesia or an alternative to traditional methods is useful when the need arises.

TAP blocks are the most intriguing alternative regional anesthetic technique being utilized for postcesarean analgesia or operative bring backs for wound complications in the lower abdomen which is the usual obstetric approach to the hysterotomy required for Cesarean delivery.

The interest in utilizing truncal blocks to provide prolonged analgesia postcesarean delivery has become a focal topic of interest among obstetric anesthesia providers. The potential to be a long-acting analgesic adjuvant absent side effects like pruritus, nausea, hypotension, or fetal drug accumulation make the block attractive. The list for known side effects related to using neuraxial anesthesia to provide postoperative analgesia is extensive and especially common when long-acting duramorph is utilized in the intraspinal space.

The TAP block can be performed more predictably with ultrasound, which would obviously be the safest and most predictable way to administer the block. Utilizing TAP blocks to target upper thoracoabdominal surgeries for postanalgesia are reported [85]. The TAP block can be performed without ultrasound and has a proven, repeatable success rate once the learning curve and adequate numbers have been performed using the double fascial click palpation technique.

The block will have limited application since the anticipated dermatomal distribution for the block seems to be T10 to L1 when utilizing perceived safe volumes of local anesthetic of choice [86, 87]. Further it has to be emphasized that a TAP block will be limited to somatic relief only and not address visceral pain typical in postoperative obstetric cases. A specific investigation into postcesarean analgesia with the TAP block did yield promising results [79].

The TAP block has proven success in gynecologic, urologic and bowel surgery, which involves an incision in the anterior abdominal wall in the dermatomes suggested to be covered by the block.

The triangle of Petit or the lumbar triangle is the anatomic identifier that marks the entry point for the needle to access the fascial plane between the internal oblique muscle and the transversus abdominis muscle. The superior aspect of the iliac crest serves as the origin for the latissimus dorsi muscle that marks the posterior-lateral border of the triangle while the external oblique aponeurosis inserts on the anterior half of the iliac crest marking the anterior-medial border of the triangle. The clinical appreciation of the true cephalad extent of the iliac crest is important and often more difficult to appreciate than expected when a large pannus is present that hides the correct insertion point without ultrasound guidance. The anterior superior iliac spine is often more easily palpable and could be a confounder for trying to identify the appropriate needle entry point. If the morphology of the patient is a challenge, it may be easier to identify the latissimus dorsi muscle and walk the palpating hand down to the iliac crest by taking advantage of the ability to identify and isolate the latissimus muscle. Take advantage of the insertion of the latissimus on the humerus and have the patient extend or medially rotate the humerus to isolate and definitively identify the latissimus dorsi connection to the iliac crest.

The needle of choice will be a blunt bevel whether using ultrasound-guided assistance or the double-click technique. Standard epidural 17-gauge or 18-gauge Tuohy needles with centimeter markings or a B bevel regional needle typically a 22-gauge needle are acceptable. Flexing the OR table while the patient is supine will make the iliac crest more prominent. Airplaning the table away from the intended block side will also make the target zone more appreciable. When using the double-pop technique, the needle is advanced perpendicular to the coronal plane encountering a more subtle external oblique pop then a more resistant internal oblique pop which is the stopping point for needle advancement and local anesthetic injection after aspiration. The anticipated resistance to injection should be similar to that when injecting an epidural catheter, if there is more resistance to injection than anticipated the probability is that the needle has advanced into the transversus abdominis muscle. Withdraw slightly, aspirate and repeat the injection trial. If the needle is truly in the intended plane, then the resistance will be very minimal to injection.

Utilizing ultrasound, the needle entry point will not be as important because the three muscles of interest are so clear sonographically. Orienting the probe to obtain in plane real-time images documenting needle advancement can be easily accomplished by placing the probe transversely in the Triangle of Petit. Appreciation of the fascial pops as the needle advances serves as a double reassurance when performing the block.

My preference for local anesthetic is 20 ml of ropivacaine 0.5% with 2 mg dexamethasone in each hemiabdomen. If an epinephrine-containing local anesthetic solution is desired for nonvascular confirmation is desired, then an easy approach is to dilute equal volumes of 2% lidocaine with 1:200,000 epinephrine commonly available to most obstetric anesthesia providers with the ropivacaine. This creates a 1:400,000 epinephrine combined ropivacaine-lidocaine solution. Adding fresh epinephrine is the best solution, but the addition of 2% lidocaine with epinephrine to ropivacaine or bupivacaine is a quick and easy pharmacologic solution available to almost all practitioners.

The biggest concern for performing a TAP block especially a bilateral TAP block will be safe dosing of the local anesthetic injectate. The potential for complication in a pregnant patient is heightened due to systemic vascular engorgement, which could result in accidental intravenous injection and the typical local anesthetic toxicity manifestations. The concept of test dosing as is done in epidural placement with an epinephrine-containing solution is a good practice habit that could detect an intravascular sitting before a full-dose single-shot injection is performed.

Cumulative maximum safe dosing for single shot injections is an easy calculation and can be done by classic methods that then allow the practitioner to choose agent, concentration, and volume. It can be expected that a 15–20-mL injection will be needed for each injection to achieve the dermatomal coverage targeted. The costal margin, the iliac crest, and the lateral border of the rectus will be the anatomical sites limiting spread. An excellent anatomic review by Rozen and colleagues found that only T9–L1 nerves were found in the midaxillary line below the costal margin to the inguinal ligament. Circumferential and cephalad spread are approximately equal and cannot be overcome with volume since the plane is permeable and leaky into spaces adjacent, therefore missing the targeted nerves to be blocked.

Indeed, because ventral rami and segmental nerves branch extensively and communicate widely with adjacent nerves is why a single shot TAP block is effectively a plexus block that is easily accomplished.

Complications

The mother and her fetus typically represent 120 life years when complications are litigated. Familiarity with the potential for harm and how to mitigate those consequences is the next best thing to preventing them.

Approximately 2.4 million epidurals per year are performed to provide labor analgesia in the USA. Accurate risk estimates for hematoma can be stated as 1 in 168,000; deep epidural infection, 1 in 145,000; persistent neurologic injury, 1 in 240,000; and transient neurologic injury, 1 in 6,700. Meta-analysis shows these risk incidences have improved compared to earlier reports because the data available has improved and can be more accurately quoted to the patient [88].

High Block

High block is an iatrogenic event that requires early recognition and rapid management to protect the patient. A clinical setting in which this is likely to occur is the patient with a functioning epidural who has failed a trial of labor and has gone to the operating room for cesarean delivery. Despite large volume dosing of local anesthetic, the epidural block will not rise to a sufficient level for surgical anesthesia. The determination to expedite the case and perform a spinal to provide a denser and higher block is made. What determines the dermatomal height achieved after spinal injection is inconclusive, but there is good evidence to suggest that the volume of lumbosacral CSF is a determinant of how high an intrathecal injection will rise, and in this case, the volume is diminished due to epidural fluid mass effect. By virtue of this compression, the likelihood of high spinal is increased even when using reduced dosing or usual spinal dosing. Mass effect plus the fact that sodium channels are occupied from epidural infusion that has crossed into the CSF make it very easy to overdose and get a high spinal in this setting [89]. The ED50 and ED95 of intrathecal isobaric bupivacaine are 7.25 and 13.0 mg [90], which can serve as guides for dosing or if the provider follows the recommendations of Norris [91] to not modify the dose of bupivacaine and to use a full 15-mg dose then the risk for high spinal is probably increased. It is an unsolved dilemma as to how much dosing is required in a patient with an inadequate but demonstrable level whether it was from epidural or intrathecal cause.

Patient awareness of ascending block precedes dramatic changes in blood pressure, heart rate, EKG rhythm or pulse oximetry which is often expressed by the patient as a sense of impending doom or fear. This may represent medullary hypoperfusion. Aggressively treat hypotension and hypoxemia and assess block height continuously while being ready to secure the airway by intubation. For hyperbaric solutions, the anatomic hindrance to continued cephalad spread is the T5 or T6 point of maximum kyphosis. If this barrier has been breached, then the cephalad spread is rapid and the consequences are overwhelming. Airway compromise and hypotension should be the focal points to guide treatment.

Sedation to provide anxiolysis is likely to further compromise an already at risk airway, so it is advisable to be very judicious in choosing this pathway if a patient has not yet lost their airway. Checking hand grip and changes in phonation are methods to assess the height of the spinal block. Be cautious when administering benzodiazepines to a weak patient who has not yet crossed the threshold for intubation. Do not obtund the patient unless inducing the patient for intubation. High spinals have been shown to decrease the sensitivity to midazolam meaning much less goes much further towards creating respiratory embarrassment [92].

Pulmonary Aspiration

The risk of pulmonary aspiration is a constant for any pregnant patient especially those who present emergently for stat cesarean delivery requiring general anesthesia. The risk of aspiration during high spinal is also very real and less so for

laboring patients or patients presenting for nonurgent cesarean delivery. Even if pharmacologic prophylaxis may not have sufficient time to be optimized in such a setting, it should be insisted upon being given. Protecting the mother protects the fetus at risk and any step needed to optimize maternal safety cannot be compromised. Administration of nonparticulate antacid, metoclopramide and an H2 blocker would be the preferred regimen for a patient known to be at high risk for aspiration. An assistant skilled at providing cricoid pressure should be a part of the team preparedness for such an emergency. Difficult intubations also increase the risk for aspiration. Extubation represents another risk point for aspiration that can be mitigated by using an OG or NG tube to suction the stomach.

Hypotension

Hypotension is rarely a significant medical risk to the parturient unless she has a medical comorbidity that cannot be compromised by hypotension such as valvular heart disease. The sympathectomy that characterizes successful regional anesthetic placement can be anticipated and appropriate interventions taken to reduce the degree of hypotension resultant. These measures can include preloading or more appropriately coloading with fluids as the block is established and aggressively using vasoactive drugs to lessen the hypotensive effect. Phenylephrine is now accepted for routine obstetric intervention and should be the vasopressor of choice for blocks below the T10 level [93]. Ephedrine is still useful and is probably a clinically better drug to treat hypotension as the block progresses cephalad above T10. Maternal heart rate may influence choice between phenylephrine and ephedrine. Drops in maternal MAP represent a greater risk to the fetus because the driving force for uteroplacental perfusion is maternal MAP. A good clinical strategy is to identify the fetus at potential risk and intervene aggressively if decelerations are noted after neuraxial placement that represents a change from FHR baseline. If hypotension is absent, but fetal decelerations are significant, then the suspicion should be that a hypertonic uterus is responsible. This is common after a CSE technique when rapid analgesia has resulted from intrathecal narcotic injection, but the maternal baseline pressure has not changed. Tocolytic treatment with terbutaline or nitroglycerin rather than urgent transport to the operating room should effectively remedy the deceleration problem.

Local Anesthetic Toxicity

A consensus statement has been issued by the ASRA practice advisory on local anesthetic systemic toxicity, which summarizes the diagnosis and treatment of this complication [94]. Classic signs and symptoms of local anesthetic toxicity include but are not limited to auditory changes, circumoral numbness, metallic taste, and agitation, which can lead to seizures. Local anesthetic CNS toxicity occurs before

Table 28.2 Local anesthetic drug information

Drug	Lidocaine	Prilocaine	Bupivacaine	Levobupivacaine	Ropivacaine
Description	Amide	Amide	Amide	Amide	Amide
Relative potency	2	2	8	8	6
Onset	5–10 min	5–10 min	10–15 min	10–15 min	10–15 min
Duration without epinephrine	1–2 h	1–2 h	3–12 h	3–12 h	3–12 h
Duration with epinephrine	2–4 h	2–4 h	4–12 h	4–12 h	4–12 h
Max dose without epinephrine	3 mg/kg	6 mg/kg	2 mg/kg	2.5 mg/kg[a]	3 mg/kg[a]
Max dose with epinephrine	7 mg/kg	9 mg/kg	2.5 mg/kg	3 mg/kg[a]	4 mg/kg[a]

[a]Indicates probable safe maximum dose (insufficient data)

cardiac toxicity, which manifests as arrhythmia and is a function of potency. No single intervention has been identified which eliminates the risk of local anesthetic toxicity, but early detection of intravascular dosing remains the focus. Minimizing the amount of local anesthetic needed to achieve the desired clinical effect is emphasized. Atypical presentations of local anesthetic toxicity occur in approximately 40% of reported cases. Provider vigilance to variability is crucial. Incremental dosing is always the correct dosing and adding an intravascular marker is another step to reduce the risk of complication that should be routinely used.

Treatment of local anesthetic toxicity should focus on preventing airway compromise and hypoxemia which potentiate the complication. Compared to lidocaine, bupivacaine is more dysrhythmic and more resistant to resuscitation. For small-dose inadvertent intravascular injection, the fact that pregnancy increases hepatic blood flow and increases amide clearance is beneficial. Benzodiazepines are the drugs of choice for halting seizures; if a benzodiazepine is not available then propofol or thiopental can be used. Early use of 20% lipid emulsion accelerates removal of bupivacaine from the circulation [95]. Recommended bolus dosing is 1 mL/kg repeated up to three times at 3-min intervals, and then titrate to effect with an expected dosing of 0.25 mL/kg/min. If these measures fail, then CPR should be begun and having cardiopulmonary bypass should be made available. Given enough time, the metabolism of the local anesthetic will occur, but aggressive life support will be needed to buy that time (Table 28.2).

Chloroprocaine is not included above because of the safety profile associated with esters. Esters have low toxic potential but are much more likely to generate an allergic reaction because of their linkage and the release of a PABA-like molecule after hydrolysis. Esters are not of themselves immunogenic, but the metabolites of esters are much more potentially allergenic than are the metabolites of amide-linked local anesthetic. The metabolites of many drugs are truly the culprits causing adverse reactions or unwanted side effects that all anesthesia providers' fear, and this is indeed the cause for reported ester local anesthetic allergic potential.

Spinal Headache

Postdural puncture headache is a risk that has a long and continued history in regional anesthesia. Usual risk quotations for epidural and spinal approaches resulting in a headache are 1–2%. It has been established that the dura is not a watertight meningeal layer and is therefore not truly the violated layer responsible for CSF leak causing a headache. More appropriately, it can be referred to as a meningeal puncture headache because the arachnoid layer must be breached to cause the headache [96, 97]. Unfortunately iatrogenic headaches from accidental dural puncture represent the most frequent temporary claim for litigated injury, which is why it should rank high on the list for risks in the informed consent process.

Why CSF loss generates a headache is controversial and probably multifactorial. Loss of intracranial support creating brain sag and the compensatory vasodilatation by cerebral vessels are thought to be the cause for cephalgia. The clinical picture is that the headache is postural, generally occurring 12–48 h after an epidural or spinal, and is a bilateral headache that can be frontal, occipital, or both. A headache that is exacerbated with 15 min of assuming an upright position and improved within 15 min of lying down is a meningeal puncture headache. Of the meningeal puncture headaches, 67% are severe, 23% are moderate, and 11% are mild [98].

If doubt exists about the cause for a postdelivery headache after regional anesthesia, then more extensive workup including radiologic evaluation or neurological consultation to rule out potential serious etiologies like dural venous sinus thrombosis, subdural hematoma, subarachnoid hemorrhage, or preeclampsia must be done before further treatment.

Treatment options include conservative management and therapies focused on supportive comfort including utilizing pharmacology approaches that yield more success when using models for treating vascular or migraine headaches. The definitive treatment for meningeal puncture headache is still a blood patch and should be offered as soon as conservative management fails to provide patient relief. When a wet tap happens, the option of placing an intrathecal catheter and leaving it in situ for 24 h has been shown to at least reduce the severity of headache. This adds an increased risk of infection and is not a universally accepted clinical practice. Minidose duramorph through the indwelling catheter before removal has been used which may only delay the onset of headache but has analgesic effect. Dosing with preservative free saline has also been tried with an upper limit of 20 mL or stopping if the patient complains of paresthesia, tinnitus, or uncomfortable pressure in her head.

Neurologic Complication

Neurologic complications are usually short and transient but can be more sinister and difficult to diagnose when persistent. Circumspect technique and vigilant adherence to meticulous preparation will reduce the chance of complication, but will not abolish it. Serious neurologic complications from regional anesthesia techniques

have a lower incidence in the obstetric population than in the general population. Major complications are rare and can be divided into damage caused by the needle, damage caused by the catheter, or complication caused by technique.

The estimated frequency for direct nerve damage is 1–10,000 to 1–30,000 and can be caused by needle or catheter placement. There is nothing to offer as treatment and the duration of time needed to appreciate improvement is 1–6 months. Direct trauma to nervous tissue may occur at the level of the spinal cord, nerve root, or peripheral nerve. Paresthesia or pain during sitting or injection should be respected and the process stopped immediately. Two-thirds of anesthesia-related neurological complications are associated with either paresthesia or pain during injection.

The complication of hemorrhagic complication is estimated to be a frequency greater than 1:150,000 for epidural placement and 1:220,000 or greater for spinal placement. Neurologic dysfunction that results can be catastrophic requiring immediate recognition and treatment to avoid permanent paraplegia. The cause can be needle or catheter placement and has been suggested to happen more often with epidural catheter removal than with placement. Anticoagulation therapy increases the risk of hematoma as does the progressive thrombocytopenia typical in preeclampsia, eclampsia or HELLP patients. The symptoms of epidural hematoma are bilateral leg weakness, urinary incontinence, and loss of rectal sphincter tone. These severe neurologic deficits may be preceded by sharp pain in the back or legs. Prolonged motor paralysis without regression warrants workup. Stat MRI is the radiologic exam of choice to identify potential epidural hematoma with neurosurgical notification of the potential problem as soon as possible. Symptomatic epidural hematoma must be decompressed surgically as rapidly as possible to facilitate recovery. ASRA issued its Third Consensus Statement that serves as a model to guide practice standards if regional anesthesia is perceived to be a risk due to antithrombotic or thrombolytic therapy. Understanding that this is a collection of observations and experiences of many experts, and then if any particular drug or class of drugs is encountered in a parturient, the recommended waiting times for placement and safest time to remove a catheter should be sought in this reference [99].

Epidural abscess is very rare and usually due to hematogenous seeding of the epidural space. Obstetric anesthesia providers should use hat, mask, and gown to decrease the risk of inoculating the patient. Oropharyngeal secretions are known to be potential sources for particularly bad infections. Presentation is usually within days of placement but commonly occur about 1 week out because that amount of time must pass to allow for pressure of the developing abscess to be sensed. Symptoms include fever, malaise, headache, and back pain. Palpation of the site or adjacent paraspinal areas will reveal focal pain. White blood cell count will be elevated. Pyrexia is usually present. Neurologic deficits will progress if the spinal cord is compressed and may manifest as lower extremity pain, weakness, bowel and bladder dysfunction, and paraplegia. Surgical intervention and a long course of appropriate antibiotics represent the best treatment [100, 101].

The spinal cord, nerve roots, or peripheral nerves are vulnerable to needle or catheter injury. Most anesthetic related neurological complications have the harbinger of paresthesia or pain upon placement or injection [102]. Resultant deficits are usually

detectable within 24–48 h and spinal anesthesia is three times more likely to result in neurologic injury or radiculopathic injury compared to epidural anesthesia. Spinal administration has an added risk of resultant neurologic injury without paresthetic warning being noted [103, 104]. Single nerve root neuropathy is rare approximating 1:10,000 incidence and more likely to resolve completely and more quickly than spinal cord, plexus or polyneuropathy injuries [105, 106].

Epidural catheters rarely may break or shear, but should be left indwelling unless increasing neurologic symptoms are expressed or a compromise in daily activities occurs. The epidural space should accommodate the presence of a benign catheter with less risk for complication than would be suffered by the patient if surgical retrieval were made.

Parturients who do not receive regional anesthesia frequently experience compression nerve injury. The reported incidence of permanent neurological deficits is as high as 1:2,100 deliveries [107]. Factors that increase risk are prolonged labor, maternal positioning during delivery, fetal presentation, fetal size, and instrument delivery. The lumbosacral plexus is most likely to be involved especially the lateral femoral cutaneous, femoral, and obturator nerves. The peroneal nerve is vulnerable to injury if prolonged or poorly positioned legs in stirrups occur during delivery. These nerve injuries are probably ischemic in origin and will usually be transient but may persist for as long as 6 weeks postdelivery [108].

Postpartum Back Pain

Backache postdelivery is common and is independent of whether an epidural was chosen for analgesia. One third to one half of women will experience back pain more pronounced after delivery than before becoming pregnant. Epidural analgesia has been clearly shown not to be a contributing risk. Suggested causes for long-term backache postpartum include change in pelvic tilt, ligamentous relaxation causing spinal anatomy changes due to the release of the hormone relaxin at delivery, musculoskeletal injury or stretch not appreciable due to analgesia or excitement of delivery and more. It is now clear that the use of epidural analgesia is not a direct cause of postpartum backache nor does it modify the risk of developing backache [109, 110]. Can an obstetric epidural cause adhesive arachnoiditis has been asked and functionally answered in the negative. Adhesive arachnoiditis is particularly painful and debilitating and an extremely rare potential complication of obstetrical epidurals and the infusions commonly run in them [111].

High Risk Anesthetic Patients

The three leading causes of maternal mortality are hemorrhage, thromboembolic disease and preeclampsia. Maternal mortality has recently increased across the globe although anesthetic deaths are declining in prevalence having fallen to the sixth

leading cause for maternal deaths [112, 113]. General anesthesia and associated airway loss complications represent the greatest threat to life for anesthetic-caused maternal death. Being able to predict, prepare, and handle the difficult airway in obstetrics cannot be overemphasized [114].

Worldwide, the quotation is that one parturient per minute dies which is true by actuarial analysis of reported data. Life threatening maternal etiologies can be expected to be encountered in any obstetric anesthesia practice whether in a small community or large tertiary care hospital. Hypertensive diseases of pregnancy are increasing in frequency and because of high risk medical management those afflicted are successfully carrying pregnancy to further gestations compatible with viable delivery feasible at approximately 24 weeks of gestation.

Hypertensive Disorders of Pregnancy

The overall incidence of hypertensive disorders in pregnancy approximates 6%. Eclampsia is reported at 1–2 per 1,000 deliveries. Women with preeclampsia and eclampsia have a 3- to 25-fold increased risk of severe complications, such as abruptio placentae, thrombocytopenia, disseminated intravascular coagulation, pulmonary edema, and aspiration pneumonia. More than half of women with preeclampsia and eclampsia require cesarean delivery. African-American women not only have a higher incidence of hypertensive disorders in pregnancy but also tended to have a greater risk for most severe complications. Preeclamptic and eclamptic women younger than 20 years or older than 35 years have substantially higher morbidity [115].

Pregnancy is a thrombogenic condition and pulmonary embolism tends to be considered a later pregnancy problem. However, symptoms suggestive of pulmonary embolism need to be taken seriously and treated and investigated at any stage of pregnancy. The potential for thromboembolic complications has introduced the relatively common usage of low-molecular-weight heparins for thromboprophylaxis, which carries the potential to be a strong contraindication to using neuraxial anesthesia if the last dose administered falls within the recognized time frame for unacceptable intraspinal bleeding.

Hypertensive disorders of pregnancy have defied modeling that can be agreed upon as representing disease course. Terminal end points being the most severe disease manifestation are usually recognized as acute fatty liver syndrome and eclampsia, but whether these endpoints are on a continuum including preeclampsia as a common pathway is debatable.

Preeclampsia morbidity and mortality is related to systemic endothelial dysfunction; vasospasm and small-vessel thrombosis leading to tissue and organ ischemia. Possible organ involvement includes CNS events such as seizures, strokes, or hemorrhage; renal tubular necrosis; hepatic coagulopathies; and placental abruption in the mother. Each of these complications can precipitate a request for urgent delivery (Table 28.3).

Table 28.3 Probable predictors for OB requested urgent delivery

Preeclampsia
Blood pressure: 140 mmHg or higher systolic or 90 mmHg or higher diastolic after 20 weeks of gestation in a woman with previously normal blood pressure
Proteinuria: 0.3 g or more of protein in a 24-h urine collection (usually corresponds with 1+ or greater on a urine dipstick test)
Severe preeclampsia
Blood pressure: 160 mmHg or higher systolic or 110 mmHg or higher diastolic on two occasions at least 6 h apart in a woman on bed rest
Proteinuria: 5 g or more of protein in a 24-h urine collection or 3+ or greater on urine dipstick testing of two random urine samples collected at least 4 h apart
Other features: Oliguria (less than 500 ml of urine in 24 h), cerebral or visual disturbances, pulmonary edema or cyanosis, epigastric or right upper quadrant pain, impaired liver function, thrombocytopenia, intrauterine growth restriction

Eclampsia is defined as a seizure in a patient with preeclampsia and carries added risk for respiratory compromise and all the known complications of an intracranial bleed. After airway control has been established, then pharmacologic treatment of the cause can be instituted. Magnesium is the treatment of choice for preeclamptics or eclamptics. The special pharmacology of magnesium as it relates to anesthetic drug choices must always be kept in mind along with its impact on renal physiology. The results of the MAGPIE study demonstrated that magnesium is the drug of choice to treat hypertensive disorders of pregnancy, and obstetric anesthesia providers should expect it to be on board when consulted for anesthetic management of a preeclamptic [116].

Preeclampsia in the USA is treated with prophylactic magnesium sulfate to prevent the escalation to eclampsia. Magnesium can potentiate maternal hypotension after the initiation of neuraxial anesthesia that is difficult to treat because magnesium attenuates the response to vasopressors. Dose reduction for depolarizing and nondepolarizing neuromuscular is wise in a patient on magnesium therapy because there will be a delayed recovery of muscular strength to acceptable levels for extubation if a general anesthetic is needed. Calcium chloride is the drug of choice for magnesium toxicity.

A good strategy for the anesthetic management of preeclampsia is to intervene early. The disease progression can rapidly devolve into an escalating high-risk anesthetic encounter. The patient usually has intravascular hypovolemia despite obvious third-space overload expressed as independent edema in all tissues. A progressive coagulopathy must be ruled out before approaching the patient to discuss risks and benefits of neuraxial anesthesia. A comprehensive review of the history of the present illness and its systemic manifestations is crucial to being able to provide safe anesthesia care.

If lab values are acceptable and appropriate intravascular resuscitation can be made then neuraxial anesthesia is the technique of choice whether the obstetric management plan is to attempt vaginal delivery or the plan is for cesarean delivery.

The number of platelets needed to be reassured about placing a neuraxial catheter in a preeclampsia/eclamptic patient is debatable. If the disease is mild, our absolute

acceptable number is 75,000 platelets, but at least 10% of all preeclamptics will fall below 100,000 platelets which seems to have a more formal acceptance threshold. Anesthetic judgment about offering neuraxial anesthesia to a parturient with less than 75,000 platelets is justifiable when the clinical picture supports that assessment. How functional those platelets are is as much of an issue as the absolute number. Thromboelastographic analysis or platelet function analysis may be needed if the clinical history suggests a bleeding trend despite the absolute platelet numeric count being acceptable. A platelet count of 50,000 seems to be the absolute lowest limit acceptable to consider providing neuraxial anesthesia.

If there is a clinical picture suggestive of HELLP, then my practice is to place an epidural catheter several hours before the patient requires analgesia. Any patient with HELLP will be delivered expeditiously, and to place a catheter before the platelet count is unacceptably low allows for the option of neuraxial analgesia for a trial of labor. In parturients with low platelet counts, a platelet count should be determined before removing the epidural catheter.

Neuraxial anesthesia is sometimes requested by obstetricians for blood pressure control, which should not be the primary reason to place an epidural, but rather to reap the secondary gain associated with good pain relief. Neuraxial analgesia does blunt the exaggerated hypertensive response to labor in the preeclamptic population, which results in better maternal blood pressures. In the interest of the fetus, epidural analgesia may improve intervillous blood flow and decrease the likelihood of urgent cesarean delivery for monitored nonreassuring fetal tracing. If an urgent cesarean delivery is needed, it is better to have a functioning in situ epidural to provide the anesthesia for operative delivery than to go through the increased risks of general anesthesia and a possible difficult intubation in a stress setting.

I consider spinal anesthesia to be the preferred method for operative anesthesia whether elective, urgent, or emergent in the preeclamptic patient. As noted in the section on hypotension for complications and under cesarean anesthetic conduct, it has been found that patients with preeclampsia have less severe spinal anesthesia-induced hypotension compared to general anesthesia. It had always been felt that sudden sympathectomy could compromise either the parturient or fetus, but multiple studies cited earlier have dispelled this belief and shown that using low-dose vasopressors creates a hemodynamically smooth course for cesarean delivery after spinal anesthesia. The increased risk associated with an indwelling catheter in a potentially coagulopathic patient might need to be endured if a long cesarean section is anticipated that requires a combined spinal-epidural technique to allow for extending the block duration. Fetal outcomes are favorable for spinal anesthesia delivery in the preeclamptic.

Maternal Hemorrhage

Maternal hemorrhage is the most preventable cause for maternal mortality [117]. Rapid anesthetic response is important to outcomes for the parturient and the fetus. Clinical scenarios with potential extreme urgency include placenta previa, placental

abruption, cesarean hysterectomy from abnormal placentation, and postpartum hemorrhage. Maternal and fetal status needs rapid assessment for frank bleeding or when bleeding is suspected but not obvious. If a patient is suspected of having a high risk for hemorrhage early enough to convene a joint care conference to possibly include nursing, obstetrics, blood bank, interventional radiology, and anesthesiology, that should be undertaken to explore the options for management and to understand the decision process guiding patient care among each specialty.

Placenta Previa

For nonurgent antenatal bleeding associated with placenta previa most anesthesia providers will consider neuraxial anesthesia if the risk for cesarean hysterectomy is not obviously increased. Controversy exists regarding the appropriate type of anesthesia for patients at high risk for cesarean hysterectomy. If the risk for placenta accreta, increta, or percreta is increased markedly because of abnormal implantation or prior uterine surgery, then controlled intubation is favored by many who prefer not to risk converting neuraxial anesthesia to general anesthesia. For a patient with multiple prior cesarean sections, the presumption that a placenta accreta will be encountered should always be a part of the anesthetic plan and appropriate contingencies to deal with the complication made. Large bore IV access and blood bank readiness to administer products are mandatory before commencing operative delivery.

Cesarean section for placenta previa diagnosed preoperatively with appropriate resuscitation prior to entering the operating room represents a good opportunity to provide neuraxial anesthesia absent any other risk factors. Large volumes of blood can be hidden in a partial abruption. The potential for maternal hypotension that compromises the uteroplacental flow will still exist, but is treatable and in my opinion is not a contraindication to the use of neuraxial anesthesia.

A cesarean hysterectomy can be anticipated to lose between 2,500 and 4,500 mL of blood, and the difficulty for surgeons trying to secure vascular pedicles is increased due to uterine size. A spontaneously breathing patient makes anatomical structure identification more difficult for the surgeon especially where the uterine artery dives under the ureter. A strong case can be made that it is best to secure the airway before the surgery begins because airway edema markedly worsens during large-volume resuscitation. Trying to secure an airway is also more difficult with an open abdomen and potential significant hypotension. Tenable arguments against the use of neuraxial anesthesia include the risk of severe maternal hypotension, patient discomfort associated with intraperitoneal manipulation and traction, and patient discomfort associated with a prolonged surgical procedure. In the circumstance of placenta previa and acute hemorrhage, general anesthesia is the best choice [118].

Having interventional radiologists place uterine artery catheters prior to cesarean delivery can mitigate blood loss. If blood loss continues postoperatively, then uterine artery catheters can be used to embolize the uterus and hopefully prevent postpartum hemorrhage and hysterectomy [119, 120]. The uterus has collateral

artery blood supplies that are branches of the ovarian and rectal artery system, which means that all bleeding is not controlled by securing the uterine artery supply.

Placental Abruption

The anesthetic considerations in patients with placental abruption are similar to those with placenta previa. The incidence of placental abruption is probably about 1 in 80 deliveries but is mostly encountered as mild or moderate without extreme risk. The incidence of abruption that impacts anesthetic management is approximately 1 in 150 deliveries. Of abruptions, 90% will have no fetal distress evident. However, large volumes of blood can be hidden in a partial abruption. An additional consideration is the possible presence of disseminated intravascular coagulation (DIC) triggered by the abruption. Neuraxial anesthesia is contraindicated in the presence of DIC. Patients who are hemodynamically stable without ongoing hemorrhage and without a coagulopathy are candidates for neuraxial analgesia/anesthesia. Vaginal delivery is possible for most cases of abruption. General anesthesia is indicated for acute hemorrhage or in the presence of DIC. Cesarean delivery for placental abruption can result in massive blood loss and transfusion products should be immediately available.

Postpartum Hemorrhage

The anesthetic considerations in postpartum hemorrhage are similarly focused on achieving hemodynamic stability and assessing maternal blood volume issues. Postpartum hemorrhage is often underestimated by simple under accounting or may also be anatomically hidden in the retroperitoneal gutters or in the broad ligament from delivery trauma. If an epidural is still present from delivery, then extension of epidural analgesia or initiation of neuraxial anesthesia is appropriate. However, in the face of hypovolemia and hemodynamic instability, general anesthesia is the anesthetic of choice.

Retained placenta and uterine atony are other commonly encountered causes for postpartum hemorrhage. Pharmacologic treatment using oxytocin, methylergonovine, prostaglandin F2 alpha, and misoprostol are used to try and target the myometrium to contract sufficiently to stop hypotonic bleeds while retained placenta requires manual extraction. Nitroglycerin or extension of epidural block facilitates cervical dilation and extraction. If operative removal is needed, then extension of neuraxial anesthesia of provision of deep MAC anesthesia is preferred. Inhalation agents are profound uterine relaxants and can increase bleeding after extraction and should be avoided. A hypotonic or atonic uterus has a rich blood supply and the amount of blood lost can accumulate rapidly. The uterine perfusion at delivery is 500–700 mL/min, and the risk for uterine atony complication is 2–5% of all deliveries [121].

VBAC

Uterine rupture is a potential risk for all parturients electing a VBAC trial who decline a repeat cesarean delivery. The risk for hemorrhage depends on the manifestation of the "rupture." True rupture requires emergent intervention on behalf of the mother and fetus while dehiscence is an urgent event with less blood than might be anticipated since the scar dehiscence should be in the relatively avascular lower uterine segment. It is strongly advised that the parturient trying to achieve VBAC should have an epidural for the labor. Epidurals will not mask the pain of uterine rupture and can be used to facilitate cesarean delivery urgently as needed avoiding the risk of general anesthesia [122].

Massive Transfusion

Obstetric hemorrhage can be profound with rapidly developing shock despite the physiologic adaptations of pregnancy that provide the parturient with more red blood cells and circulating volume. These adaptations may actually delay detection of impending collapse. We have massive transfusion guidelines that we institute when faced with extreme obstetrical hemorrhage. The following is a short synopsis of our guiding principles as developed by the Vanderbilt surgical trauma service [123, 124]:

1. The initial dose will consist of:
   (a) 6 RBCs: If trauma units are used; Rh pos for males and females with expected age >50; Rh neg for females with expected age <50.
   (b) 4 FFP: If trauma units are used, select AB FFP.
   (c) 1 Platelet dose.
   Products are sent together as *complete* doses as described above:
   (d) Only the number and type of products as outlined in this protocol can be issued.
   (e) Requests for additional numbers or type of product must be preapproved by the Blood Bank resident or BB attending.
   (f) RBCs, FFP, and platelets must be issued *together* for each cycle. RBC and platelets will not be sent without the FFP.
   (g) Exception: If FFPs are not thawed and ready at the initiation of the Massive Transfusion Protocol; RBCs if requested, cryoprecipitate can be issued.
   (h) Given the high ratio of plasma infused for each cycle, cryoprecipitate is not necessary. If MTP is started late in the resuscitation and the clinical team feels that fibrinogen may have been low from the beginning, then cryo may be considered.
2. An Emergency Release form is issued with any uncrossmatched units.
3. RBCs and FFP are packed in a cooler with ice:
   (a) Platelets are placed in a plastic ziplock bag, labeled with "Do Not Place Platelets in Cooler" sticker.

4. When the cycle is ready, the patient location is called to notify the staff that the cooler is ready for pick up, and asked if the MTP is to continue.
   (a) If an OR room telephone line is busy, it is permissible to call the OR Board to tell them the cooler is ready, but the *OR room must be called to ask if the MTP is to continue*.
5. If the protocol is to continue, additional coolers will be supplied as soon as all products in the cycle are ready. This is approximately every 30 min.
6. The 2nd and subsequent doses will consist of:
   (a) 6 RBCs
   (b) 4 FFP
   (c) 1 Platelet dose
7. When each dose is ready, the patient location is called to notify them that the cooler is ready. At this time, Blood Bank asks if the protocol is to continue.
8. This process is continued until the attending surgeon or anesthesiologist tells the Blood Bank to discontinue.

Lagniappe

Obstetric anesthesia is a subspecialty full of unpredictability and challenge. Nothing is normal no matter what it may look like on the surface, and the ability to always remain vigilant is Sisyphean. Fortunately patient outcomes are usually happy as the process of childbirth is a much-anticipated event that is more appreciated when the parturient is made comfortable. Interactions and feedback with obstetricians and nursing are vital to be kept in the loop and contribute to patient safety. Because the ambient environment of obstetrics obeys the second law of thermodynamics, there is always an entropic outbreak about to happen. Remaining calm and rational in the flight to the OR for emergent delivery is sometimes viewed as the anesthesiologist just not having the right appreciation or perspective for the events that triggered the chaos. The subspecialty is indeed the most gratifying when the patient recognizes us for making her comfortable, keeping her and the baby safe and being able to control all the variables making her anxious about the whole process. How to conduct a case is always a matter of confidence in your ability and is as much an art as a science as can exit in anesthesia. The pressure to do something for medicolegal issues should be resisted if it conflicts with what you think is the right management plan.

I use remifentanil liberally for procedure placement like arterial lines, epidurals, and spinals. From my experience with fetal surgery and our in utero monitoring, I know that a baby dislikes having its umbilical cord compromised much more than it dislikes anesthetic drugs like narcotics or benzodiazepines. Babies wake up about the same time as their mother after our general anesthetic/epidural/narcotic

administration for in utero repair. Inform the neonatal team of drugs administered and that team will know how to handle the consequences to the baby. It is all about multicollaborative teamwork and sharing information.

The possibility of developing chronic pain after a routine cesarean delivery is real, so I choose multimodal paths to achieve anesthesia and analgesia. I routinely use clonidine in spinals although I use doses of 0.2 mcg/kg/it or less. I use ketamine and propofol as an adjunct to cover patchy epidural blocks or to sedate the patient uncomfortable with being awake for cesarean surgery. My usual induction drug sequence is to give 100 mg lidocaine IVP, then remifentanil 2 mcg/kg IVP, then 1 mg/kg propofol which has ketamine mixed at 2.5 mg/ml and succinylcholine 1 mg/kg. I have had no reported recall using this recipe and require little vasopressor support. For inductions or preceding spinal placement, I also add 400 mcg phenylephrine/L to my crystalloid and infuse it rapidly to coload and mitigate hypotension. Match the hatch is a fishing phrase that translates well to any individual obstetric anesthesia practice because the obstetric anesthesia provider has the onus of doing his job to fit the obstetrician's ability and expectations. Fortunately we have a wide armamentarium of drugs to achieve this goal.

The only constant in the field is change and it behooves the anesthesia provider to know more about the practice of obstetrics than does an obstetrician feel compelled to understand our specialty. Much is expected of obstetric anesthesia providers and we have met the challenge for the most part and have advanced the safety of mother and baby mostly through regional anesthetic use. Ultrasound will be common in the near future especially if the sonography improves to allow for real-time imaging when performing neuraxial techniques.

Clinical Pearls

- Early pain may be a predictor for likely C/S, therefore consider placing epidural catheter higher rather than lower to facilitate conversion to surgical anesthesia.

Anesthesia for Labor

- When continuous fetal heart rate tracing is being performed the L&D nurses usually place the bands holding the toco and fetal heart monitors over the iliac crests which is usually visible along the patient back roughly equivalent to Tuffier's line.
- Data from trials to assess risk for cesarean delivery cannot control for the factor thought to be most responsible for operative rates which is obstetrician preference.

- Maintain analgesia because tachyphylaxis or tolerance is possible when more rescue dosing is needed due to inadequate analgesia especially with bupivacaine.
- Hyperbaric bupivacaine 2.5–5 mg can be utilized to cover sacral nerves when utilizing CSE, spinal or continuous IT catheter. Sacral sparing is often an unwanted epidural fact.
- Opioids help relieve persistent perineal pain and hot spots of missed segments; sufentanil has the least dermatomal spread and hydromorphone the most dermatomal spread for narcotics that are lipophilic.

Anesthesia for Cesarean Section

- Phenylephrine is my drug of choice for hypotension caused by sympathectomy below T10, from T10 to T5 ephedrine will have more effective pressor action.
- The only epidural catheter worth keeping is one that can be predicted to be dosed rapidly to section anesthesia. Redosing more than three times in an hour is an indicator of a catheter that should be replaced.

Adjuvant and Alternative OB Blocks

- Although no longer frequently used, both the paracervical and pudendal block served as tools to differentially map the course of labor.

Hypotension

- Time to a clinically appreciable pressor action of phenylephrine is approximately 1 min, while ephedrine clinical onset is approximately 2 min.
- The Institute for Safe Medication Practices has placed oxytocin on the High Alert list due to the potential of oxytocin to cause profound hypotension.

Neurologic Complication

- The course of labor and childbirth delivery is more likely to cause nerve injury than is anesthesia conduct.

Multiple-Choice Questions

1. A G4P3003 presents for 3rd repeat C/S. BMI is 45, and pt has comorbidities of gestational DM not requiring insulin. VS are BP 145/90, P 98, and SpO2 is 96% on RA. Hct is 32 and blood glu is 145. Last C/S took 2 h to complete due to adhesions encountered. Best choice for anesthesia?
   (a) Spinal
   (b) Combined spinal-epidural
   (c) Epidural
   (d) General anesthesia

2. Which of the following is an inappropriate rescue drug for high spinal?
   (a) Vasopressin 2 U IVP
   (b) Phenylephrine 200 mcg IVP
   (c) Midazolam 2 mg
   (d) Ephedrine 10 mg

3. A parturient presents for VBAC at 37 weeks, now G3P2002 with both prior deliveries by C/S due to CPD. U/S reveals anterior placenta. VS are normal and pt has no significant medical history except for gestational nausea and backache. Pt wishes to have natural labor. Which of the following is not true?
   (a) Pt is low risk for abnormal placental implantation.
   (b) Early neuraxial anesthesia placement is preferred.
   (c) Pt needs a type and screen in the blood bank.
   (d) Pt is high risk for abnormal placental implantation.

4. Preeclampsia:
   (a) Occurs before 20-week gestation.
   (b) Can occur after delivery.
   (c) Is a contraindication to neuraxial anesthesia.
   (d) Has a mild, moderate and severe form.

5. Spinal block height:
   (a) Is strongly predicted by pt height.
   (b) Is reduced in obese patients'.
   (c) Is best predicted by volume of lumbosacral CSF.
   (d) Is adequate if a T8 level is achieved for C/S.

6. Pregnancy:
   (a) Is a hypocoagulable state.
   (b) Is a hypercoagulable state.
   (c) Has higher baseline maternal BP to feed the fetus.
   (d) Offers no challenges to the anesthesia provider.

7. Neuraxial anesthesia:
   (a) Slows labor.
   (b) Increases the risk for C/S.
   (c) Causes maternal backache postpartum.
   (d) Decreases maternal stress catecholamine levels.

8. Urgent C/S will be performed most frequently:
   (a) For a VBAC patient
   (b) Fetal heart tracing abnormalities
   (c) Placental abruption
   (d) Eclampsia

9. Spinal headache after wet tap:
   (a) Occurs immediately.
   (b) Is rarely severe.
   (c) Is exacerbated by lying down.
   (d) The definitive treatment is blood patch.

10. Magnesium sulfate therapy:
    (a) Is a relative contraindication to neuraxial anesthesia.
    (b) Potentiates neuromuscular blockade.
    (c) Is algolic.
    (d) Requires higher dosing of local anesthesia to achieve labor analgesia.

11. Paracervical blocks:
    (a) Are easier and safer to perform than neuraxial anesthesia.
    (b) Are safer for the fetus.
    (c) Are limited to first stage of labor relief.
    (d) Are limited to second stage of labor relief.

12. Combined spinal-epidural anesthesia:
    (a) Has been associated with shorter labors.
    (b) Doubles the risk for postpartum headache.
    (c) Should only be performed when a parturient wants a walking epidural.
    (d) Causes profound hypotension.

13. A patient is seen the day after a prolonged vaginal delivery with epidural analgesia. She complains of numbness only in the lateral femoral cutaneous nerve distribution. Which of the following is true?
    (a) The epidural could not have caused this because it is only a sensory nerve.
    (b) The effect is probably permanent if it does not resolve within 2 weeks.
    (c) The obstetric team probably caused it by compressing the nerve under her inguinal ligament during delivery.
    (d) No follow-up is necessary.

14. Pt with epidural catheter now s/p SVD with partial abruption but continues to ooze despite 30 U of oxytocin. History of gestational PIH but not preeclamptic.
    (a) Administer methergine
    (b) Draw DIC labs
    (c) Order trauma blood from the blood bank
    (d) Administer 10 more units of oxytocin.
15. The most likely complication for neuraxial anesthesia is:
    (a) Wet tap
    (b) Transient neural injury
    (c) Inadequate analgesia or failed block
    (d) Infection

Answers:

1. The answer is c because the expected duration could exceed single shot spinal dosing and an epidural is not proven to work in the CSE technique since it cannot be tested after spinal dosing. General anesthesia is least desirable.
2. c
3. The answer is a. pt is 24–40% chance for accreta and therefore large volume blood loss.
4. The answer is b. As noted, it is in the differential diagnosis for postpartum hypertensive headache.
5. c
6. b
7. d
8. b
9. d
10. b
11. c
12. a
13. c
14. The answer is b because that is the probable cause for continued bleeding. Trauma blood is inappropriate without blood bank current cross match due to the possibility of changed profile associated with fetal-maternal blood mixing with abruption. Methergine is contraindicated with hypertensive disease of pregnancy.
15. c

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29

Regional Anesthesia for Outpatient Surgery

Joshua E. Smith

Contents

Introduction

Outpatient surgery has seen a tremendous increase in volume in recent years. With this increase in volume, an increase in painful, invasive surgery has occurred. A result of this change has been poor outpatient pain control in this surgical setting [1, 2]. This has presented anesthesiologists with a challenging environment, in particular with regard to pain management. Of great value is regional anesthesia.

The use of regional anesthesia is effective in treating postoperative pain for extremity surgery, including both upper and lower extremities [3]. Additionally, the use of blocks can decrease operating cost for a surgical suite due to the decreased amount of anesthesia-dedicated time [5]. Also, the use of continuous catheter techniques has been utilized successfully in both academic and, more importantly, private practice settings [6]. Furthermore, in literature, there has been a demonstrated improvement in recovery following peripheral nerve blocks [7]. Finally, several advantages with the use of regional blocks have been demonstrated with regard to

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Table 29.1 Essential needs for a practice implementing an outpatient regional service

1.	Start small. Focus on one or two surgeons' patients. This allows for a practice to begin interrogating their system for not only placement and perioperative management of blocks, but also night and weekend coverage as well as follow up
2.	Patient selection is important initially. Patients who have the cognitive ability to understand the goal of the block, as well as manage a catheter will increase satisfaction, which will help with 'buy-in' from surgeons and ancillary staff
3.	Communicate with patients about their expectations for the blocks. Impress on them that the block is only one aspect of their pain regimen
4.	It is helpful to have the block area organized. The goal with a dedicated block area is to expedite patient care in a safe manner. Coordination of preoperative nursing needs and block placement allows for more time for block placement
5.	A dedicated block nurse is very helpful when starting a block program. Ideally, this/these personnel are able to perform nursing documentation, execute timeouts, sedate patients and help with the actual block placement
6.	Working with scrub nurses to develop ways to drape patients without removing indwelling catheters is important to prevent suboptimal block duration
7.	Follow up with patients is valuable in that it allows for detection of gaps in the system utilized, as well as the efficacy of blocks
8.	Ensuring that patients have a way to get in touch with an on call physician is of obvious importance

time to discharge, secondary to decreased systemic narcotic use, decreased nausea, and decreased respiratory depression [8, 9].

This chapter will discuss the implementation of outpatient regional anesthesia, utilizing both "single-shot" techniques, as well as continuous catheter techniques. Table 29.1 summarizes essential needs for a practice implementing an outpatient regional service.

In daily clinical practice, it is necessary to provide a quick, safe, and effective block for each patient. Furthermore, it is very important to maintain open lines of communication between the anesthesia team, surgeon, and patient – in particular, with regard to expectations for the surgeon and patient. Letting each party know what the goal of the block is – frequently to decrease and not eliminate pain – will result in increased patient satisfaction.

Furthermore, it is extremely important to follow up with patients after they have left the perioperative area. This can be accomplished with daily phone calls – either for the duration of a continuous nerve block or until resolution of a single shot. These phone calls allow for the anesthesiologist to make further adjustments to that patient's regimen, to discover gaps in their own system, and to allow for detection of possible complications from blocks.

Each of the above stipulations involves an increase in the amount of work and energy used, but the benefit is patients with lower pain scores, shorter PACU stays, and higher satisfaction with the perioperative experience.

For both single-shot and continuous techniques, a number of topics will be covered. These include patient selection, patient education, block selection, medication selection, postoperative care, and follow-up.

Single-Shot Techniques

Patient selection is of obvious importance with all regional anesthesia. First, the patient has to consent to the block. Second, they need to understand how the block is to be performed and what they can expect from a standpoint of nerve stimulation, skin infiltration, etc.

Depending on the site of the surgery, an appropriate block needs to be chosen. Prior to placement of the block, it is important to discuss with the patient, and any present caregiver, what can be expected with the block. They need to understand that as the extremity begins to regain sensation, it will be necessary to begin analgesics, as their exposure to the surgical pain will increase with time as the block resolves. This will frequently necessitate the use of pain medication prior to the patient going to sleep that evening. If the patient fails to anticipate this, their pain level upon awakening will be quite high, and their satisfaction with the block will be decreased [11].

Also, a patient education form can be used. This form can reinforce what they can expect with the block, the need for preemptive analgesia, and have a way for them to contact the anesthesiologist if there is a problem. Additionally, it is helpful to inform them of the need to go to the emergency room if they experience shortness of breath following an upper extremity nerve block (secondary to pneumothorax or phrenic nerve paresis). An example of this sheet is included at the end of the chapter.

Additionally, it is of importance to instruct the patient to not weight bear on the treated limb in order to avoid falls. Also, protecting the affected extremity is paramount, as it is possible that they will be unable to sense either pressure or temperature changes until resolution of the block. Finally, proprioception will be decreased, and the absence of this sensation can predispose to injury [11].

Certain blocks may not be appropriate for some patients. As an example, selection of a femoral nerve block may or may not be suitable. Patients could have significant quadriceps weakness after discharge and could suffer a fall if they do not have a caregiver that can help them ambulate. In patients for whom a femoral nerve block is chosen, it is of great importance to impress upon them and their caregiver that they will have weakness and need to have a great deal of assistance in ambulating until the block has resolved.

Also, selection of an interscalene block in the setting of pulmonary disease may not be indicated, as the risk of phrenic nerve paresis exists with this block, which could result in the need for supplemental oxygen. This can prolong the time to PACU discharge or necessitate an inpatient stay for supplemental oxygen. This obviously is not the objective with outpatient surgery.

Not only proper selection of the type of block, but also of the medication to be used. Longer-acting local anesthetics, such as bupivacaine or ropivacaine, have been frequently used for postoperative pain control. Additionally, the use of adjuvant agents, such as epinephrine, clonidine, and dexamethasone, has been described to increase block duration [12–14].

Postoperatively, it is important to evaluate the patient's pain level and augment their analgesic regimen with systemic medications as needed. Occasionally, additional blocks may need to be performed, particularly saphenous blocks in foot

and ankle surgery to supplement a popliteal block. Following the achievement of a satisfactory pain score, the patient may then be discharged to a same-day surgery unit for subsequent discharge home.

Follow-up should occur until resolution of the block. Questions about the duration of numbness and motor block should be asked. Also, time to first oral analgesic can be helpful for determining efficacy of block. It is valuable to find out the reason for the first oral analgesic, as patients may take them due to surgical pain, block site pain, or they may be simply taking them as directed. Following the resolution of the block, it is insightful to ask their satisfaction scores, as this can help determine which techniques are working well in a given practice.

Continuous Block Techniques

Patient selection is very important for these blocks [10]. It is necessary to have a patient who can understand the block and follow instructions after they go home. In the event that a patient cannot understand instructions, it may be beneficial to avoid the placement of a catheter. The author feels that you have to be able to trust the patient, and absence of this trust is an indication for a single-shot technique or no block at all.

Patient education again is very important. Discussion with both the patient and any caregiver is very beneficial, especially with the caregiver, as the patient may not be able to visualize the catheter insertion site, especially with interscalene or posterior popliteal approaches. Care of the catheter insertion site and the dressing is very important, as well as care for the infusion device. Finally, removal of the catheter at the conclusion of the infusion is of vital importance, and the patient and caregiver need to know what to expect and how to remove the catheter. Again, an example of a patient education form for continuous techniques is included at the end of the chapter.

Block selection is vital as with single-shot techniques. A distinguishing characteristic for continuous techniques is the need for the insertion site and dressing to be well away from the surgical field. For example, it can be difficult to secure an interscalene catheter properly and still allow the surgeon the exposure they need for the procedure. Use of a benzoin type of preparation can increase the adherence of the dressing to the skin. Also, interaction with the surgeon and scrub nurses can be very beneficial, to develop methods to prevent dislodging the catheter during removal of the surgical drapes.

Drug selection can be of less importance versus a single-shot technique. Less importance is placed on the need for longer-acting local anesthetics, as the patient will have an infusion postoperatively. However, the use of less cardiotoxic local anesthetics is very important, as the risk of an unwitnessed cardiac arrest following discharge is present, however unlikely it may be. The medicolegal ramifications of this alone could warrant the use of more expensive ropivacaine versus less expensive (soon ropivacaine will be generic). A bolus of local anesthetic after placement of the catheter, with a small amount of epinephrine as a test dose, may be used to demonstrate the lack of immediate intravascular absorption.

Postoperatively, it is again important to assess the patient's pain level. Additionally, boluses of local anesthetic can be considered in this setting. Also, examination of the catheter insertion site and the integrity of the dressing is important as there may be need to reinforce the dressing to avoid inadvertent catheter removal.

During follow-up, it is important to again assess the efficacy of the block. In a patient who is receiving less analgesia than expected, it may be necessary to increase the rate of infusion if an adjustable pump is being used. Obviously, this is where trusting a patient's judgment comes into play. Also, it is important for the patient or a caregiver to assess the appearance of the insertion site. Presence of erythema can be the first sign of infection, and removal of the catheter may be warranted, as well as starting antimicrobial therapy. Finally, following removal of the catheter, it is important for the patient to verify that the tip is intact so that no foreign body is left in situ.

Complications

The most frequent complication of outpatient peripheral nerve blocks is inadequate analgesia. Options for treating this include increasing the rate of infusion in the instance of a continuous block or use of opioids or other pain relievers. If none of these measures work, the final option is to instruct the patient to present to an emergency department for pain control.

Another complication unique to outpatient blocks is local infection which can frequently be prevented by observing aseptic technique during block placement, limiting the duration of infusion, tunneling the catheter, and the use of prophylactic antibiotics. Furthermore, avoidance of the femoral and axillary continuous blocks is helpful [15]. Finally, the other major complication of continuous peripheral nerve blocks is dislodgement of the catheter. The best way to prevent this is adequate securing of the dressing and patient education to prevent excessive wear to the dressing (Tables 29.2 and 29.3).

Table 29.2 Instructions for nerve blocks

You have received a peripheral nerve block. This is an injection of medication next to a nerve that will decrease the amount of pain you have after surgery. This is information about your nerve block
The medicine will wear off over the next day, and you will begin having pain. Begin taking your pain medicine when you start to have pain from your surgical site. The nerve block is only one part of your pain therapy
If within 2 days you do not have sensation and/or strength in that area, we would like for you to call us, so that we will know this, and be able to help you
If you had arm surgery, and begin having shortness of breath at home, go immediately to the emergency room
If you need help immediately, call hospital paging at (555)555-1234 and ask the operator to page the Anesthesia doctor who is on call
The doctor on call WILL NOT prescribe, call in, or refill any narcotic/pain medications

Table 29.3 Instructions for nerve block catheter

You are being sent home with a continuous infusion of medicine through a catheter that is lying near nerves that supply sensation to your surgical site. Please read the following instructions carefully regarding the care and removal of this catheter
Begin taking your pain medicine when you start to have pain from your surgical site. The nerve block is only one part of your pain therapy
The rate of the medicine infusion is dialed to _ on your pump dial. DO NOT change this rate
If you had surgery on an arm or shoulder and your catheter is located in your neck or shoulder area, and you experience any trouble breathing, use the white clamp between the pump and the dial to clamp the tubing, and call the Anesthesia doctor who is on call (contact information below)
DO NOT inject anything into the catheter or tubing. This may result in severe limb injury or loss.
You should remove the catheter when the pain pump is empty. To remove:
1. Clamp the pump with the white clamp between the pump and the dial
2. Leave it clamped for 2 h
3. Peel off all adhesive parts, gently pull the white catheter out, and dispose of the entire apparatus (including the On-Q medicine ball)
The catheter should come out easily and you should not experience pain during removal. If you meet any resistance, turn your head or bend your knee and try again. If you still have pain or resistance, leave catheter in place and call the Anesthesia doctor on call (contact information below). Once the catheter is out, examine the end - there should be a small (3–5 mm) metal tip. If not, call the Anesthesia doctor who is on call and save the catheter for examination
If at any point the dressing breaks down and the catheter becomes exposed to air, remove the catheter. If you have redness or pus that develops around the catheter site, or if you develop a fever greater than 101 degrees Fahrenheit, call the Anesthesia doctor who is on call
If you need help immediately, call hospital paging at (555)555-1234 and ask the operator to page the Anesthesia doctor who is on call
The doctor on call WILL NOT prescribe, call in, or refill any narcotic/pain medications

Clinical Pearls

- Pump selection is important for continuous blocks for very obvious reasons. Ideal characteristics of a pump is one that is simple to operate, allows for multiple infusion rates, is small (but with a large reservoir), and has a very low failure rate. Simplicity of operation allows for ease of communication between the anesthesiologist and patient over the phone regarding the state of the pump. Multiple infusion rates allow for flexibility in dosing but also have a downside in that the patient may alter the pump rate without the direction of the anesthesiologist. Small size allows ease of movement and ambulation while the pump is connected, while a large reservoir allows for a longer infusion time.
- Drug selection for the pumps can be influenced by several factors. For the author, the simpler the system, the less likely that complications can occur. I choose to limit the drug to only a local anesthetic, and it is always the same drug at the same concentration. This allows for few errors, particularly on block days that are busy, and also when a patient calls about their block.

- Selection of a single-shot versus a continuous catheter technique can be important for reasons other than desire for a longer block or patient compliance. In the case of a patient with pulmonary disease, it may be wise to place an interscalene catheter rather than performing either a single shot or no block at all. The advantage of the catheter technique is that the patient's block can be incrementally bolused, titrating to effect. The smaller volume of local anesthetic is less likely to cause phrenic nerve paresis, and also, the presence of the block can decrease the amount of opioid that is needed in the postoperative setting, which can reduce the chances of complications from systemic narcotic.

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Part VI
Additional Topics

30

Outcome Studies and Infection Control in Regional Anesthesia

Joel Barton • Stuart A. Grant

Contents

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Introduction

Regional anesthesia began half a century after ether was first used for general anesthesia. Each branch of anesthesia has waxed and waned in popularity since their introduction, but now, with the advent of evidence-based medicine, true comparisons of outcome following general or regional anesthesia should be available.

Regional anesthesia holds a certain promise as our population continues to age and acquire comorbid conditions and may represent a gentler mode of providing care for patients who may not tolerate the insult of general anesthesia.

Regional Anesthesia has many theoretically beneficial effects both intraoperatively and in the postoperative period although studies have alternatively demonstrated or refuted these effects. The majority of evidence pertains to neuraxial regional anesthesia, though there are increasing numbers of studies examining peripheral nerve blockade. The outcome data on regional anesthesia is difficult to generalize because of many variables: insertion site, medication delivered, duration of use, congruency to surgical site and risks, and complications of a given surgical procedure. For this reason, when data is discussed, the entire perioperative environment must be considered.

When looking at outcomes, there are many disparate outcomes that are measured. Traditional outcomes include morbidity and mortality, cardiovascular, pulmonary, and GI endpoints. Alternative outcomes measured include overall opioid use, length of stay, quality of life, and patient satisfaction. Traditional outcomes such as myocardial infarction, pneumonia, and mortality have decreased through innovation and best clinical practices to the point that large randomized controlled trials for each type of surgery are needed to prove a significant difference, making the measurement of such outcomes more challenging. Opioid use is easily tracked and compared, though it is dependent on the assumption that fewer opioids are always better. Length of stay and cost analysis are among the outcome measurements that emphasize the relative stresses different types of anesthesia place on our health care system. As health care increasingly emphasizes a patient-centered approach, other outcome measurements, such as quality of life, quality of recovery, and patient satisfaction deserve attention as well.

Intraoperative Effects of Regional Anesthesia

It is well established that surgery is a pro-inflammatory event [1, 2], inciting an increase in cytokines and a hypercoagulable state intra- and postoperatively [2–4]. The surgical stress response is divided into the endocrine, metabolic and inflammatory pathways, although, in reality, there are a myriad of interactions among the three. Neuraxial anesthesia has been shown to blunt the stress response and should therefore theoretically improve morbidity and mortality associated with these pathways (Fig. 30.1).

The neuroendocrine response to stress has been well described by Charmandari et al. [5]. The perioperative period is characterized by increased epinephrine, cortisol, and inflammatory mediators, all a function of the neuroendocrine response to surgical injury. This, coupled with other elements of the perioperative period such as inhaled anesthetics, decreased level of activity, and glucocorticoid therapy lead to hyperglycemia. Hyperglycemia and the stress response lead to decreased immune function, increased oxidative stress, endothelial dysfunction, a procoagulant state, fluid shifts, electrolyte fluxes, and increased inflammatory mediators and mitogens. For the patient, the consequences can be delayed wound healing, increased infection, potential end-organ dysfunction, and delayed recovery [6]. This has been a driving force in the investigation of a multimodal approach to blunt these responses, of which regional anesthesia plays a key role.

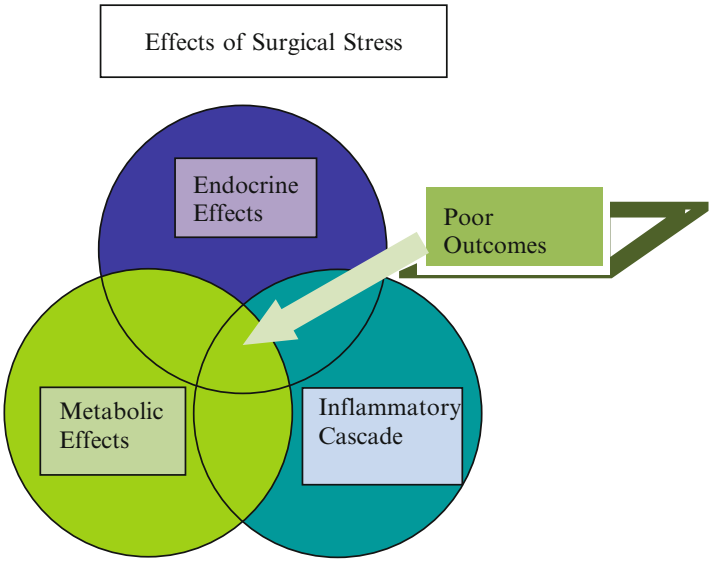

Fig. 30.1 Effects of surgical stress

Table 30.1

	General	Regional
Immunosuppression	↓	↔
Natural killer cell function	↓	↔
Catecholamine levels	↑	↔
ACTH	↑	↑/↔
Cortisol	↑	↑/↔

Blood Loss and Transfusion Requirements

Intraoperative blood loss during total hip and total knee replacement was reviewed in a meta-analysis in 2010 that included 880 patients in 12 studies [7]. Results were mixed, with five studies showing no significant difference in blood loss between the regional anesthesia and general anesthesia. Of the remaining seven studies in the meta-analysis, five showed reduced blood loss with regional anesthesia and another two studies showed increased blood loss with regional anesthesia.

When looking strictly at total hip arthroplasty, the pooled data from ten of the studies demonstrated a statistically significant decrease in blood loss with regional anesthesia. In the meta-analysis, two trials were excluded from the regional anesthesia arm for significant reductions in blood pressure. Given that hypotension is often a desired effect of regional anesthesia, it is reasonable to include the results of these studies in clinical decision making for a particular patient. In addition, five of the studies in this meta-analysis included data on intraoperative transfusion. Overall, the incidence of transfusion was reduced by more than half with the use of regional anesthesia for total hip replacement.

Cancer Recurrence and Regional Anesthesia

Surgical stress places the body in an immunocompromised state, affecting the ability of natural killer (NK) cells to function. In addition, animal studies have shown a dose-related effect by opioids that suppresses NK cells [8]. NK cells are the primary defense we have against cancer. NK cells recognize and kill tumor cells in the body (Table 30.1).

It has long been a concern that surgery itself promotes the growth of dormant metastases and accelerates the growth of previously slow-growing masses, with opioids for postoperative pain control only exacerbating the problem. Regional anesthesia has been shown to decrease the stress response to surgery and preserves more immunologic function. Regional anesthesia causes a smaller reduction of NK cell function than general anesthesia [9]. The mechanism of this regional anesthesia-mediated preservation of NK function is the blockade of afferent transmission of noxious stimuli to the central nervous system and the blockade of efferent activation

of the sympathetic nervous system, thereby both diminishing the body's response to surgery and decreasing the amount of anesthesia necessary for surgery. Spinal anesthesia added to halothane general anesthesia significantly decreased the surgical promotion of lung metastases in rats [10]. Wada et al. demonstrated that spinal anesthesia added to sevoflurane general anesthesia decreased the surgical promotion of liver metastases in a murine model with the surgical insult being laparotomy. They demonstrated that the tumoricidal function of liver mononuclear cells and the improved T1–T2 helper cell ratio were instrumental in the better outcomes [11]. The first human study of a possible influence of regional anesthesia on long-term cancer outcomes was published by Exadaktylos et al. [12]. This was a retrospective study of 129 patients receiving mastectomy and axillary node dissection with an average follow-up of 32 months. Fifty patients received general anesthesia and a paravertebral block, while 79 patients received general anesthesia and postoperative morphine analgesia. On follow-up, recurrence and metastasis-free survival was 94 and 77% at 3 years for the paravertebral and morphine analgesia groups, respectively. This was a retrospective study, with inherent weaknesses, and the methodology of the study has been criticized for its poor bias control. In 2008, Christopherson et al. published an association between type of anesthesia and survival in colon cancer patients, with patients who had epidural analgesia 4.65 times less likely to have died by 18 months than those who did not have an epidural [13]. In 2010, a retrospective review of 669 patients receiving surgery for colon cancer was divided into an epidural arm and a general anesthesia with parenteral narcotic arm. It was found that there was only a significant difference in cancer recurrence in patients greater than 64 years of age, but not in younger patients, based on the use of regional anesthesia. It is postulated that the risk of recurrence in the face of regional anesthesia may be related to tumor type as well [14]. These data highlight the need for prospective studies to demonstrate causality. Given that cancer is the second leading cause of death in developed countries, the possible impact of this information demands a timely pursuit of an answer.

Postoperative Effects of Regional Anesthesia

Mortality

Short-term mortality is positively affected by regional anesthesia and postoperative epidural pain control, and this improvement has been demonstrated in various surgical interventions [15]. One Medicare claims database analysis demonstrated an improved survival using regional anesthesia with an odds ratio of 0.52 for mortality at day 7 and 0.74 at postoperative day 30, with significant 95% CI and *P* values [16]. The analysis was from years 1997 to 2001, and it would be beneficial, given the change in approach to the perioperative care of the patient in the last decade, to see if this mortality benefit was sustained. Currently, no such study has been done.

Long-term mortality is an all-encompassing outcome and therefore difficult to attribute to a single factor in the majority of surgical procedures. Surgical mortality has been decreased by a multimodal approach to modulating the body's response to surgical stress, an approach to this problem spearheaded by Kehlet and co-workers for the last decade that has resulted in improved patient outcomes [2, 17, 18]. This comprehensive accelerated recovery pathway is more important than any single factor (such as regional anesthesia) within the pathway. Given the relative rarity of mortality in many surgical procedures, it is difficult to determine the effect of one aspect of a multimodal approach. One way to compensate for this is to select a single operation and use this as a benchmark of the effect of regional anesthesia on long-term mortality. This benchmarking has been done using the example of hip surgery in the elderly. There are obvious difficulties with this assumption, the first being that the body of an elderly person with a fractured hip is responding the same to stress as a patient who is responding to the pain of a surgical incision. Also, it is incorrect to compare an elderly patient who is undergoing an elective hip arthroplasty with a patient who has suffered a hip fracture. Although studies from 1986 and 1987 [19, 20] demonstrated a short-term benefit without a long-term benefit to regional anesthesia in patients with fractured hips, a Cochrane Database review in 2004 [21] found insufficient evidence to demonstrate differences in mortality due to anesthetic choice, and an Australian study published in 2005 [22] found that the choice of anesthetic had minimal impact on mortality following hip fracture surgery. This loss of difference in more recent studies is perhaps due to the multimodal approach now taken to care for hip fracture patients.

Cardiovascular Outcomes

Postoperative myocardial infarction (PMI) is a leading cause of postoperative morbidity, and so, investigation of methods of reducing postoperative myocardial infarction will not only have great benefit for individual patients but also for the health care system as a whole. Neuraxial analgesia decreases cardiovascular and metabolic stress induced by surgery [23, 24], diminishes postoperative hypercoagulability [25], improves the patient's respiratory function, and blunts sympathetically induced coronary vasospasm [25, 26].

A review published in the *BMJ* in 2000 involving 9,559 patients in 141 trials looked at multiple postoperative outcomes. A total of 104 myocardial infarctions were reported, with approximately one third fewer events in the patients with neuraxial anesthesia [27]. The next year, a meta-analysis was published looking specifically at the risk of postoperative myocardial infarction [28]. A total of 1,173 patients in 17 studies were included. This meta-analysis concluded that at least 24 h of postoperative epidural analgesia reduced the rate of postoperative myocardial infarction by 40% in patients with a thoracic epidural. It further suggested that high-risk cardiac patients should have postoperative epidural analgesia but calls for further studies for a more certain determination. Lumbar epidural analgesia did not have as great a benefit on cardiac morbidity as thoracic epidural analgesia.

Caveats to these results include: The results are only significant using a fixed-effects model and not the random effects model of analysis, and as the model becomes more conservative, the studies lack the power to show significance. Also, when one of the more criticized studies, Yeager et al. [29], is not included in the analysis, the data does not have statistical significance.

There have been many studies concerning regional anesthesia and the incidence of deep vein thrombosis (DVT), of which the older studies [4, 30–32] showed a statistically and clinically significant benefit to regional anesthesia. Since the advent of a multimodal approach to the care of orthopedic patients and standardization of DVT prophylaxis, there is no significant difference in the incidence of DVT postoperatively, regardless of the method of anesthesia selected. The most recent meta-analysis on this reviewed ten studies reporting incidence of DVT and eight reporting incidence of pulmonary embolism [7]. Provided the patient receives DVT prophylaxis, there exists no significant difference in the incidence of deep vein thrombosis or pulmonary embolism based on the choice of anesthesia. It may be beneficial to consider a regional anesthetic technique in a patient at high risk for a thromboembolic event in order to maximize all factors for its prevention, but there is no data to support this decision.

The effect of neuraxial anesthesia on hypercoagulability combined with the vasodilation from sympatholysis is more notable in vascular surgery. This was demonstrated in the PIRAT study of the early 1990s [33]. In this study, it was demonstrated that neuraxial anesthesia resulted in a significantly decreased number of graft failures due to thrombosis. Fibrinogen, plasminogen activator inhibitor-1, and D-dimer were followed in the study, and the regional anesthetic appeared to prevent the postoperative inhibition of fibrinolysis, resulting in fewer graft failures.

Pulmonary Outcomes

Surgery results in a multifactorial etiology of pulmonary complications, with the incidence of postoperative pulmonary complications varying widely depending on the surgical intervention. Surgery may disrupt normal respiratory activity, and anesthesia has an impact on respiratory responses to acid–base changes and hypoxia postoperatively depending on the medications given. The mechanisms of respiratory impairment include a reflex inhibition of the phrenic nerve, surgical dressings affecting the mechanics of respiration, and uncontrolled pain may result in a change of respiratory mechanics [34] (Table 30.2).

Table 30.2

	General	Regional
Pneumonia	↔/↑	↔/↓
Respiratory failure	↑	↓
Respiratory function	↓	↑
Pain score	↑	↓

Recent guidelines from the American College of Physicians [35] underscore the continued significance of postoperative pulmonary complications. In fact, cardiac and pulmonary complications have an equal incidence postoperatively and may have the same increased risk of mortality and increase in length of stay for non-cardiac procedures.

It has been demonstrated that epidural analgesia provides superior analgesia to intravenous patient-controlled analgesia (PCA), and epidurals improve respiratory function [36]. While the optimum benefit on respiratory function of different infusion solutions and adjuvants is undetermined, it has been shown that thoracic epidurals with a 0.25% bupivacaine infusion do not impair ventilatory mechanics, inspiratory respiratory muscle strength, or airway flow, even if the patient has severe chronic obstructive pulmonary disease [34, 36, 37].

The largest meta-analysis ($n = 9{,}559$) comparing neuraxial blockade with general anesthesia was published in 2000 and found that neuraxial blockade in mixed surgical procedures demonstrated a significantly decreased risk of pneumonia (3.1 versus 6%, OR 0.61 with 95% CI 0.48–0.76) [27]. This finding is in agreement with an earlier meta-analysis examining pulmonary complications following thoracic epidurals [38]. There is a great degree of variance in the incidence of pulmonary complications based on the surgery considered. When considering specific surgical interventions, thoracic epidural analgesia in the setting of coronary artery bypass surgery was shown to decrease the incidence of postoperative pulmonary complications from 30.3 to 17.2% [39]. Open abdominal aortic surgery with a thoracic epidural for analgesia also demonstrated a significantly reduced risk of respiratory failure, but a relative risk reduction in pneumonia postoperatively that was not statistically significant. In meta-analyses specific to mixed abdominal, total hip, and knee replacement surgeries, there were insufficient postoperative pulmonary complications to perform an analysis [40, 41]. VACS [42] was a large randomized controlled trials done through the Veterans Affairs Administration, and MASTER [43] was a large randomized controlled trial based in 26 hospitals across six countries. Both of these found a significant decrease in respiratory failure in high-risk patients, but no significant decrease in respiratory failure when all patients receiving thoracic epidurals were included. The high-risk patient findings were the result of subgroup analysis, which is a weakness of these studies. Currently, there is sufficient evidence in the literature to support the use of thoracic epidural analgesia for pulmonary risk reduction in the case of high-risk patients, especially for open abdominal aortic surgery or coronary artery bypass grafting.

Gastrointestinal Outcomes

The use of fast-track protocol in gastrointestinal surgery and a multimodal approach to the perioperative care of the patient is a change in the practice of medicine which includes the use of an epidural corresponding to the site of surgical intervention, both for intraoperative anesthesia and postoperative analgesia. Patients undergoing

Table 30.3

	General	Regional
Length of stay	↔	↔
Postoperative ileus	↑	↓
Hypotension	↓	↑
Visual analog pain scale	↑	↓
Postoperative nausea and vomiting	↔	↔

major abdominal surgery including colon resection have superior analgesia and decreased postoperative ileus with a thoracic epidural. However, most of these studies are in the setting of a fast-track protocol that includes early and advanced feeding, early removal of drains and catheters, and enforced mobilization. The role of thoracic epidural outside of a fast-track protocol is less well defined (Table 30.3).

A recent meta-analysis [44] of 16 randomized controlled trials of thoracic epidurals versus general anesthesia and systemic opioid analgesia for colorectal surgeries focused on length of stay with secondary outcomes of postoperative pain control, duration of postoperative ileus, incidence of anastomotic leak, incidence of postoperative nausea and vomiting, pruritis, sedation and respiratory depression, incidence of cardiac and pulmonary complications, as well as hypotension and motor blockade. When data was not available in the published article, the authors were contacted. Only two of the included trials were laparoscopic; the rest were open colorectal surgery. All but two trials done after 2000 had patients enrolled in a fast-track program. Ultimately, it was found that thoracic epidural analgesia does not significantly diminish length of hospital stay, although it does diminish duration of postoperative ileus by an average of 36 h and provides significantly better visual analog pain scales. Patients with a thoracic epidural showed increased incidence of pruritis 21 versus 5%, and urinary retention 10 versus 1% for epidural analgesia and systemic analgesia, respectively, but showed no significant difference in degree of sedation or postoperative nausea and vomiting. There were no significant differences in motor blockade, anastomotic leakage, or cardiopulmonary events. There was an increase in the incidence of hypotension with epidurals, although there was no comment on the clinical significance of this finding. A review of the literature in 2007 in Colorectal Disease [45] showed similar findings. In addition, this review showed that epidural analgesia resulted in improved blood gasses, oxygen saturation, and pulmonary function tests. These results were only clinically significant in the setting of high-risk patients, where there was a statistically significant decrease in the postoperative time on mechanical ventilation as well as a diminished incidence of respiratory failure in the high-risk population. The increased risk of hypotension was noted in this review as well, although none of the patients required more than intravenous fluid for management and no adverse clinical sequelae were noted.

Although there is a clear role for thoracic epidural anesthesia and analgesia in large open abdominal cases, the data regarding the use of a thoracic epidural in laparoscopic abdominal surgery is less clear, with positive findings on pain relief, dietary intake, and length of stay that have not always proven reproducible, perhaps due

to decreased invasiveness and surgical stress response compared to open procedures. Taqi et al. [46] compared systemic opioids to thoracic epidural analgesia with local anesthetic and fentanyl using 50 consecutive patients undergoing laparoscopic colon resection with a standard non-accelerated perioperative care plan. It was demonstrated that there was a significant difference in reduction of postoperative ileus of 1–2 days, as well as a quicker return to full diet and better pain control. However, there was no significant difference in readiness to discharge or length of stay. A prospective observational study published by Zingg et al. [47] in 2009 enrolled 76 patients to compare the effects of general anesthesia with systemic opioids and thoracic epidural analgesia using a combination of local anesthetic and opioid on postoperative pain control and ileus. The thoracic epidural group required fewer analgesics, had a mean opioid use of 12 mg of morphine compared to 103 mg of morphine, had significantly lower visual analog pain scale and time to gastrointestinal recovery of 2.96 versus 3.81 days ($P=0.025$). This correlates well with open procedures that have shown decreased opioid use and decreased ileus. While the study did not find a significant difference in nasogastric tube reinsertion or postoperative vomiting, the investigators noted that this may have been due to liberal metoclopramide use in the systemic opioid group. Clear benefits of a thoracic epidural extended as far out as postoperative day 7 in this study, which did follow a multimodal approach to perioperative care though no specific fast-track regimen was followed. No differences in surgical and anesthetic morbidity or mortality were noted between the two arms of the study. Weaknesses of the study include the fact that the patients in this study were a subset of a larger study, and there was no standardized postoperative opioid regimen.

Postoperative nausea and vomiting is also of significant concern when considering anesthetic technique. PONV can result in delayed discharge, unplanned admissions, and diminished patient satisfaction. A review published in Anesthesiology in 2003 offers a comprehensive discussion of PONV and regional anesthesia. The majority of the literature regarding PONV is in the setting of general anesthesia. When comparing regional to general anesthesia, the preponderance, though not all of the literature in this review supports the belief that regional anesthesia has a lower incidence of PONV than general anesthesia [48]. A 2009 meta-analysis of orthopedic procedures demonstrated a significant decrease in postoperative nausea and vomiting in pooled data, with an odds ratio of 0.27 and a 95% CO of 0.29–0.80 [7].

Rehabilitation and Length of Stay

The pharmacodynamics of regional anesthesia are variable and depend on whether there is use of a single shot or continuous catheter blockade, the concentration of the local anesthetic used, and the presence of adjuvants such as opioids. All of this affects the motor block that may be seen after a peripheral nerve block. Motor block must be considered when choosing the type of block and local anesthetic solution. Motor block may affect time to rehabilitation, frequency of complications, and time

to recovery. Recently, it was demonstrated that a weakness in the muscles around the knee can reduce stability during rotations and direction changes several hours after surgery for patients with femoral or femoral–sciatic nerve blocks and can limit the quality of postoperative rehabilitation [49]. However, the improved pain control seen with regional anesthesia relative to general anesthesia [50] may improve the time to physical therapy, permitting the patient to have earlier mobilization. This has been seen both in orthopedic surgery [51] and in colon resection [46]. A recent meta-analysis of studies comparing peripheral nerve blocks to neuraxial techniques reviewed 8 non-blinded studies and demonstrated no significant difference in rehabilitation indices between neuraxial anesthesia and peripheral nerve block, but a lower incidence of motor blockade in the peripheral nerve block patients [52].

In addition to length of stay for the original procedure, the incidence of readmission must also be considered. In the case of complex knee surgery, if the procedure was performed under general anesthesia without a regional technique, a review of 1,200 cases showed a tenfold greater risk of readmission. Just considering femoral blockade and combined femoral and sciatic blockade against general anesthesia without a regional technique, the risk of unplanned readmission decreased by 2.5 times [53].

Postoperative Pain Relief

Postoperative pain control is an important outcome, not only for patient-centered reasons, but also because of its impact on length of stay, time to beginning rehabilitation, and recovery of function, as well as minimization of atelectasis and pneumonia in thoracic procedures. Through randomized controlled trials and meta-analysis of RCTs, it has been shown that continuous epidural analgesia and continuous peripheral nerve catheters provide superior postoperative analgesia than intravenous patient-controlled analgesia (PCA). In addition, single injection peripheral nerve blocks are limited by the duration of the local anesthetic infused, lasting from 10 to 24 h at most [54–56]. These results were shown in many different types of surgery, although the predominance of the data is in orthopedic surgery. These benefits have been demonstrated in studies of continuous nerve catheters in the hospital setting [57] as well as at home. This superior analgesia resulted in an earlier time to walking or movement and an improved side-effect profile. When considering epidural anesthesia versus peripheral nerve blockade, a meta-analysis published in 2008 found that the level of pain relief, measured by visual analog pain scales, was equivalent, although in two of three studies, patients with continuous peripheral nerve catheters rated their satisfaction with the anesthesia higher, and all had less hypotension, pruritis, urinary retention, and did not incur the risk of central nervous system complications [52].

Regional anesthesia does have several caveats to consider with these results. First, the surgical site must be concordant with the area of analgesia. If the incision or area of surgical trauma extends beyond the dermatomes covered by the epidural or peripheral nerve catheter, then it will obviously be less than fully functional for

analgesia. In addition to catheter/surgical site congruency, the choice of local anesthetic or opioid, the duration of the infusion, and a multimodal approach to pain control are critical elements to optimizing postoperative pain control.

Chronic Pain

Chronic pain is a potential consequence of surgery that has long-lasting implications, severely affecting the quality of life of the patient. Chronic pain is not an uncommon problem, with an estimated 11–47% of patients suffering postsurgical chronic pain depending on the surgical procedure. This results in up to a quarter of the patients visiting chronic pain clinics reporting postsurgical chronic pain as their chief complaint [9]. The intensity of postoperative pain seems to affect how the central nervous system remodels itself in response to the surgical insult, and therefore, it is assumed that blunting this acute pain would decrease central sensitization and would affect the incidence of postsurgical chronic pain. This hypothesis is, as of yet, unproven and has mixed data behind it.

In the field of chronic pain, it has been well established that the degree of pain suffered, as well as the duration, has significant bearing on whether or not a patient will develop a chronic pain syndrome following their procedure. What is not yet as clear is if regional anesthesia can improve upon general anesthesia's incidence of chronic pain. Currently, the data are mixed. For example, in thoracic surgery, there is no clear data that states whether epidurals or paravertebral blocks are preferable from a long-term outcome standpoint, primarily due to a lack of long-term data on patients who had a paravertebral block. Paravertebral blocks have a better side-effect profile than thoracic epidural analgesia with equal control of immediate postoperative pain [58, 59]. Although chronic pain in thoracic surgery has several possible etiologies, using regional techniques for analgesia are thought to minimize chronic post-thoracotomy pain [60]. There is no consensus on the time of initiation of thoracic epidural analgesia (TEA) in thoracotomy in terms of prevention of chronic pain. Several studies have demonstrated a benefit to initiation of TEA prior to surgery [61, 62], but this benefit was not seen in a subsequent meta-analysis [63]. In a mixed surgical population, despite evidence of better at home pain control and mobility in patients discharged to home with peripheral nerve catheters, there are no reliable data showing a significant difference in chronic pain compared to patients with general anesthesia and intravenous and oral pain control [64]. The primary predictor of chronic pain syndrome is the degree of postoperative pain control and regional anesthesia decreases postoperative pain, yet regional techniques risk nerve damage with the development of a subsequent chronic pain syndrome. Further study with long-term follow-up data is required to provide satisfactory evidence for an answer to this question.

Lois and De Kock reviewed three types of postsurgical pain in 2008 [9]. They were phantom limb pain, thoracotomy pain, and pain after abdominal surgery. The incidence of phantom limb pain is reported to be 30% to 81%. There are a

series of case reports, and a cross-sectional survey in the literature as well as several low-powered randomized prospective studies. In addition to the small number of patients, the interventions, both surgical and anesthetic, are wide ranged, without the degree of standardization required to make them sufficiently comparable to pool the data. In 1988, Bach et al. published a prospective study of 25 patients undergoing lower-limb amputation, found that three pain-free postoperative days of epidural morphine and/or bupivacaine resulted in a decreased incidence of phantom limb pain at 1-year follow-up [65]. In 1995, an interesting preliminary report of 23 patients comparing three groups, one with preoperative epidural continued postoperatively, one with only intraoperative and postoperative epidural, and one with general anesthesia and systemic analgesia which demonstrated a significant difference in the presence and severity of phantom limb pain between the preoperative epidural and the general anesthesia group at 1 year after surgery [66]. Other studies demonstrated that, although postoperative analgesia was better in the immediate postoperative period, this benefit was gone by the first week postoperatively, with no difference in phantom limb pain [67]. There is also no study showing conclusive evidence that a peripheral nerve block or perineural intraoperative nerve blockade would reduce the incidence of phantom limb pain [9, 68].

A randomized double-blind trial of the effectiveness of adding epidural ketamine to bupivacaine found no significant difference with the use of ketamine as an adjuvant in an epidural for the prevention of phantom limb pain [68]. There is a case study of clonidine used as a perineural adjuvant in a patient who had phantom limb pain after a below the knee amputation who was getting an above the knee revision on the same extremity. Although the patient remained free of pain at 1-year follow-up, this is a case report and no conclusive evidence exists that adjuvants to local anesthetics are of significance in the prevention of phantom limb pain.

Post-thoracotomy pain is another postsurgical chronic pain syndrome with a high incidence. More than half of all patients who have a thoracotomy have post-thoracotomy pain to some degree. In 1999, a randomized single-blind study of 70 patients reported that the initiation of epidural block before tissue injury as opposed to following tissue injury intraoperatively reduced the incidence of post-thoracotomy pain by half at 6 months [61]. Although this finding has been borne out by other, later studies [9, 62], there was no consensus in the data. A meta-analysis done in 2005 pooled the data of six studies, with a total of 458 patients and found that, although preemptive thoracic epidural was better for control of acute pain, it had no effect on the incidence of chronic pain compared to postoperative induced epidural analgesia [63]. Paravertebral blocks are being used increasingly as an alternative to epidurals, being as effective for the management of acute pain and having a more favorable side-effect profile. A recent review of the available data by Conlon and coworkers in 2008 found no studies that looked at the effect of paravertebral blocks on the incidence of chronic pain and no studies that compare paravertebral blocks to epidural analgesia with chronic pain as a reported outcome. Ultimately, based on the available data, it was their conclusion that thoracic epidural was the optimal mode of analgesia for the minimization of chronic post-thoracotomy pain [69].

Abdominal surgery is also a source of chronic pain, although there is less data on the role of regional anesthesia in the development of chronic postsurgical abdominal pain. General abdominal, obstetric, and gynecological surgeries have been investigated regarding the modulation of chronic pain by the use of a neuraxial technique. In the case of general abdominal surgery, Lavand'homme et al. [70, 71] compared general anesthesia to patients who had neuraxial blockade. The study found a 23% incidence of chronic pain at 1 year after surgery in the patients who had general anesthesia. The patients who had received neuraxial analgesia demonstrated a 1% incidence of chronic pain at 1 year. It was their hypothesis that the preoperative blockade of nociceptive inputs reduced central sensitization resulting in the dramatic difference in the incidence of postoperative pain. In the case of gynecological surgery, Brandsborg et al. [72] published the results of a nationwide questionnaire that found that spinal anesthesia was associated with less chronic pain at 6-month post-hysterectomy, whereas epidural anesthesia was not associated with a decrease in chronic pain. It was their contention that the spinal blockade was denser and therefore more protective than the epidural blockade. Given that this was a questionnaire, there are many inherent weaknesses to the data. Nevertheless, a similar finding and possible explanation was advanced for the protective effect of spinal blockade on chronic pain in Cesarean section [73].

Postoperative Cognitive Decline

Postoperative cognitive decline (POCD) spans a wide spectrum of characteristics and severity, with impairment in cognitive function, memory, and consciousness being the three primary areas assessed. Impairment in cognitive function is assessed by the ability of the patient to perform simple mental tasks when asked to do so. Impairment is self explanatory, while impairment in consciousness is frequently seen as postoperative delirium. Risk for postoperative cognitive decline is exacerbated by increasing age, medical comorbidities, preexisting cognitive dysfunction, and type of surgery. Patients at higher risk for immediate postoperative cognitive dysfunction also demonstrate an increased risk of long-term cognitive dysfunction. Postoperative cognitive dysfunction, while an important outcome itself, is also a predictor of poor patient outcomes, increased mortality, and increased length of stay [15].

There is a surprisingly high incidence of postoperative cognitive dysfunction (POCD), especially in high-risk patients. A large international multicenter trial of approximately 1,200 patients over the age of 60 found 25.8% of patients had POCD 1 week after surgery, and 9.9% after 3 months, compared to 3.4% at 1 week and 2.8% at 3 months for nonsurgical controls [74]. While the percentage of postoperative cognitive dysfunction in our patient population is unlikely to change, the population as a whole is aging and a greater number of older patients with multiple comorbidities are presenting for surgery, increasing the overall burden to our health care system that POCD presents. In 1986, acute confusional states were estimated

conservatively to cost Medicare at least $2 billion, and in 1999, estimates of the cost of inpatient delirium to the health care system were $4 billion, with an increasing inpatient days by 17.5 million annually [75].

Risk factors for postoperative cognitive dysfunction following noncardiac surgery have been well elucidated and are divided into preoperative, intraoperative, and postoperative factors. Cardiac bypass presents its own cognitive dysfunction and is outside of the purview of this discussion. Of the preoperative factors, age features most prominently. The patient over 70 years old is 3.4 times as likely to have postoperative cognitive dysfunction as the patient under 70 years old (15 versus 4%) after undergoing matched noncardiac surgery. Poor baseline cognitive status, either due electrolyte imbalance or pharmacologically adjusted through alcohol or other means, is also a preoperative risk factor. Intraoperative risk factors include the specific surgical intervention especially orthopedic surgery, aortic aneurysm repair and cardiac procedures, and duration of surgery and anesthesia. The effects of profound sustained hypotension, hypoglycemia, anemia, and hypoxia on POCD are not certain. Postoperative use of meperidine, benzodiazepines, or anticholinergic medications is associated with increased POCD. Other postoperative risk factors include infection, respiratory complication, and increased postoperative pain. [15].

Given these risk factors, one would expect that regional anesthesia would be protective against POCD. This has been shown not to be true. In 2003, the International Study of Postoperative Cognitive Dysfunction (ISPOCD) [76] randomized 438 patients at 12 different institutions to regional or general anesthesia for a range of noncardiac procedures and found no significant difference in the incidence of POCD at 3 months. The next year, a systematic review of 24 trials [77] that considered POCD as an outcome found that the choice of regional or general anesthesia had no bearing on the incidence of POCD. One of the methodological issues with this review was that the postoperative analgesic regimen was not standardized across studies. With improvements in peripheral nerve catheters and ultrasound technology, this is becoming a promising area for clinical impact. In an observational study in 2005 [78], elderly patients undergoing major lower extremity joint replacement who had continuous peripheral nerve catheters for postoperative analgesia had a more than 58% decrease in postoperative delirium. Although there was another systematic review of postoperative analgesia and its effect on outcomes in 2007 [79], it was determined that more definitive research was needed on this aspect of regional anesthesia. The improved postoperative analgesia [57, 80] as well as the improved sleep and decreased postoperative fatigue [80] with peripheral nerve catheters are expected to be major contributors to a decrease in postoperative cognitive decline.

Patient Satisfaction

While clinical outcomes remain important, patient-oriented outcomes have taken on an increasing prominence both in the literature and in the impact of daily practice. Patient-oriented outcomes include postoperative pain control, quality of life, and

patient satisfaction. Ultimately, a good deal of patient satisfaction is dependent on the management of the expectations of the patient and conveying your genuine concern for them as their healthcare provider.

Patient satisfaction influences the interaction of society and the individual with the health care community and is also used as a benchmark of service and for marketing. Whether or not patient satisfaction is a true indicator of quality of care remains controversial [81] and is beyond the scope of this chapter. Regardless of validity or reproducibility, patient satisfaction is an acknowledged endpoint of outcomes research, which is ultimately designed as a patient-centered assessment.

A review article looking at patient satisfaction with regard to regional anesthesia was published by Wu and colleagues in 2001 [66]. Patient satisfaction is multidimensional, involving sociodemographic, cognitive, and affective elements [82, 83]. This makes patient satisfaction very hard to standardize. Many different theories have been advanced, but few have been sufficiently tested and validated. Many patient satisfaction theories are modifications of customer satisfaction and marketing theories, and may be classified into three broad categories: intrapatient comparisons, patient–provider comparisons, and interpatient comparisons.

There are several methodological issues with the measurement of patient satisfaction. Many patient surveys have not undergone psychometric construction to evaluate such a multivariate outcome as patient satisfaction. In addition, many surveys are unable to discriminate between portions of care the patient found to be satisfactory and those the patient found unsatisfactory. Many surveys lack reliability and validity, and bias may be introduced in many aspects of the survey process itself.

Bearing in mind the limitations expressed above, in 2007, a systematic review of the literature comparing postoperative regimens found that postoperative regional analgesia, particularly with local anesthetics resulted in significantly lower visual analog pain scores, and yet, there was a paucity of data on patient satisfaction and few validated instruments to reliably measure satisfaction. Even with improved pain control with a regional technique, improved satisfaction scores were not always seen [79]. In a 2008 review of the role of peripheral nerve blocks in ambulatory surgery, patient satisfaction was greater with perineural local anesthetic when compared to both placebo and PCA narcotic groups [54]. The determination of the true effect of regional anesthesia on patient satisfaction will require large multicenter RCTs with validated instruments for measuring satisfaction and strict methodological controls.

Neurologic Complications

Neurologic complications of peripheral nerve blockade are a major concern to both patients and providers. The instance of nerve lesion varies based on the site of blockade. In a review of 32 studies [84], the rate of neuropathy after spinal and epidural anesthesia was 3.78:10,000 and 2.19:10,000, respectively, with permanent

neurologic injury rates of 0–4.2:10,000 and 0–7.6:10,000 for spinal and epidural anesthesia, respectively. The rates of neuropathy after interscalene brachial plexus block, axillary brachial plexus block, and femoral nerve block were 2.84:100 for interscalene, 1.48:100 for axillary, and 0.34:100 for femoral. There was only one permanent neuropathy reported in 16 studies of neurologic complications of peripheral nerve block. In 1997 [85] and 2002 [86], Auroy et al. reported the incidence of nerve lesions in 21,278 and 50,223 peripheral nerve blocks, with a combined incidence of 0.02% in each study. In the 2002 study, the incidence of nerve injury in blocks with adverse neurologic sequelae ranged from 0.03% in femoral blockade to 0.31% in popliteal blocks. Of the nerve injuries seen, seven persisted for greater than 7 months. In 2005, a multicenter prospective trial published by Capdevila et al. [87] involving 1,416 patients with endpoints including neurologic damage and infection found an overall incidence of 0.21%, with three lesions during femoral nerve block, one of whom had pain on injection and two who had the block done under general anesthesia. To complicate the picture, the risk of femoral nerve injury in total hip arthroplasty is cited as 0.1–0.4%. Deficits from these lesions all resolved, although the longest duration injury took 10 months to return to baseline. Though the incidence of neural complication was low, making the determination of independent risk factors for neurologic injury difficult, intensive care unit hospitalization was positively associated with nerve injury, and the use of bupivacaine was associated with increased paresthesia and dysesthesia.

Block Site Infectious Complications

Like many other outcome measurements, there are many variables to infectious complications including patient history, duration of peripheral nerve catheter, site of blockade, and infection control precautions taken. In a study of 700 patients with interscalene catheters placed for upper extremity surgery, six patients showed clinical evidence of infection, one at 3 days after surgery, four at 4 days, and one at 5 days [88]. The catheters were removed and cultured, with three showing coagulase-negative *Staphylococcus*, one colonized with *Staphylococcus aureus* and two that grew no bacteria in culture. All catheters were scanned by ultrasound to look for a fluid collection in the setting of clinical infection. Of the six, five had no fluid collection, and all five were treated with antibiotics and had no further complications. The one patient with evidence of a fluid collection had this surgically drained, an antibiotic course administered, and there was no further complication. In summary, out of 700 patients with an interscalene catheter, 0.8% demonstrated clinical evidence of infection, with only 0.1% requiring surgical drainage of a fluid collection, and no long-term sequelae were found related to infection at 1, 3, or 6 months follow-up [88]. In a prospective trial by Capdevila that involved a range of peripheral nerve catheters, the insertion technique was a standardized aseptic technique with cap, mask, sterile gown and gloves, and sterile draping of the surrounding area. There were 256 interscalene catheters, 126 axillary catheters, 20 lumbar plexus

catheters, 683 femoral catheters, 94 fascia iliaca catheters, 32 sciatic catheters, 167 popliteal catheters, and 32 cubital or median nerve catheters in the study. In their 969 catheters, 278 or 28.7% had positive bacterial colonization on testing. The most common organism was coagulase-negative *Staphylococcus*, and 242 of the colonized catheters were single-organism cultures. Only 3% of all patients demonstrated clinical signs of inflammation, and of these, only 44% had positive cultures. Of catheters that had no clinical sign of inflammation, 18.6% were culture positive. The one major infectious adverse event reported was a *S. aureus* psoas muscle abscess and cellulitis in a diabetic woman with a femoral catheter for total knee replacement. This corresponds to other case reports of adverse infectious events, the majority of which were *S. aureus* infections in diabetics. Additional risk factors for local inflammation or infection in this study were found to be postoperative monitoring in the intensive care, catheter duration longer than 48 h, male sex, and lack of prophylactic antibiotics [87].

Data on infection rates in neuraxial techniques are similarly broad in range, with a study of 170,000 epidural and 550,000 spinal anesthetics between 1987 and 1993, citing a rate of 1.1 infections to the spine or central nervous system per 100,000 blocks, whereas a 1-year national survey published in 1999 listed the risk of epidural abscess to be 1 in 930 blocks with the risk of persistent neurologic deficit to be 1 in 4,343 blocks. While there remains a wide reported range of both infectious and neurologic complications with a multiplicity if variables among studies making studies less than completely comparable with one another, there is agreement in the severity of these complications to individual patients and the importance of striving to minimize these unfortunate occurrences.

Infection Control

Infection control should be a focus of the anesthesia provider from the planning of the block until after the patient removes the catheter at home. Given the variability in how regional anesthesia was being performed and the potential for devastating complications, as well as the many clinical unknowns surrounding regional anesthesia and infectious complications, the American Society of Regional Anesthesia recognized the need for a consensus statement on infection control and the importance of aseptic technique. For this reason, a practice advisory panel on the infectious complications associated with regional anesthesia and pain medicine was convened at the 2004 Annual Spring Meeting. Articles resulting from this panel were subject first to internal review and then to external peer review and ultimately published in Regional Anesthesia and Pain Medicine in 2006 and may also be found on the ASRA website [89–92]. This provides a peer-reviewed set of guidelines for aseptic technique during regional procedures, regional anesthesia in the setting of an immunocompromised patient, and regional anesthesia in the infected or febrile patient. The articles and source material for these recommendations will be reviewed below. The article on infection risks in chronic pain treatments is beyond the scope of this chapter and will be omitted from this review.

Regional Anesthesia and Aseptic Technique [89]

Infection sources related to the patients' health such as immunosuppression, trauma, malignancy, or pregnancy are classified as intrinsic sources, whereas skin invasion through a needle tract, contaminated needles, syringes, catheter hubs, or breaches in sterile technique are extrinsic sources. A survey of Australian obstetric anesthesiologists indicated that there was a broad range of what was considered essential for adherence to strict aseptic technique [93]. Given the broad range of methodologies listed in the literature, one may assume that this variance of opinion exists in more than Australia. The consensus statement provides an overview of hand washing, jewelry, gowns, gloves, masks, bacterial filters, and antiseptic solutions.

Based on extrapolation from surgical data, it is recommended that an alcohol-based antimicrobial scrub for hand washing be used prior to a regional procedure, although there are no randomized controlled studies of hand washing and regional anesthesia, nor are there likely to be in the future. Bacteria counts are higher on the hands of physicians who do not remove their rings, increasing the probability of nosocomial infection, and the necessity of removing of wristwatches is a view held by many infection control experts. The UK has initiated a program called "Bare Below the Elbow" for all physicians to limit nosocomial infections. Data on its effectiveness is forthcoming.

There are no studies of microcontamination after use of sterile gloves in a sterile procedure. Sterile gloves however, never negate the need for hand washing. Between nonsterile vinyl and latex gloves, vinyl gloves were almost nine times as likely to have leaks after use than latex gloves, with microcontamination on the hands of 13% of the health care providers tested before and after nonsterile procedures [94]. This is material evidence that bacteria traveled through leaks in the gloves, or that there was perforation of the gloves during the procedure.

The issue of wearing masks as a method of infection control has been controversial, with some studies stating that it may increase surgical infection with a posited mechanism of action being friction against the face resulting in scaling of epidermal tissue into the sterile field. The methodology of the study resulting in these data was widely criticized, and a more rigorous study resulted in data indicating no difference in surgical infection rate with or without the use of surgical masks. Nonetheless, there are case reports of a cluster of streptococcal meningitis from patients who had spinal anesthesia from a provider who was being treated for recurrent tonsillitis, did not wear a mask and spoke throughout the procedures [95], and a reported case of epidural abscess with a strain of *S. aureus* shown to be from the strain colonizing the nose of the healthcare provider who placed the epidural [96]. Although the use of masks for the prevention of infection remains a contested issue, there is a very clear role for the use of masks as protective gear to prevent patient body fluids from coming in contact with healthcare providers, making the question of wearing a mask for infection control academic. There are insufficient data regarding the use of gowns during regional block to make a recommendation, but, like masks, the gown may provide an important piece of protective gear for the healthcare provider. Ultimately, the degree of severity of infectious complications would argue for an enhanced

sensitivity to aseptic technique despite the lack of conclusive evidence from randomized controlled trials, which would have ethical issues in their methodology.

Hub contamination and bacterial filters are also to be considered with regional techniques. Micropore filters are designed to filter bacteria out that may exist in the infusing solution as well as prevent foreign material from gaining access to the epidural space, but there have been documented epidural abscesses in the presence of antibacterial filters [89]. There are several possible mechanisms for this occurrence. There may be hematogenous spread of bacteria from a distant source, the bacteria may migrate along the tract outside the catheter, the filter may have diminished function after a period of time, or there may be direct contamination of the hub while changing the filter. Given that it has been shown that contamination of the hub of a catheter has a direct correlation with the number of filter changes and that some filters retain antimicrobial potency for up to 60 days, it is recommended that no filters be used for a catheter that has an intended duration of use of less than a week, and catheters to be retained long-term have limited sterile bacterial filter changes. This is significant because approximately half of all catheter colonization is due to hub contamination. It has been shown that an unconnected catheter with a static column of fluid will not have bacterial transmigration at a site 20 cm from the hub, so if one were to find a disconnected catheter with a stable meniscus, and the disconnect is within the last 8 h, the catheter 20 cm from the hub could be soaked in iodine for 3 m and cut sterilely to reconnect the existing catheter. If the meniscus has regressed 5 cm or more from the hub, or if the disconnect time is uncertain or greater than 8 h, it is recommended that the catheter be removed as soon as possible [89]. The study on which this guideline is based used a fentanyl solution and not a local anesthetic as the fluid medium to measure bacterial spread, nor did it examine the effect local anesthetic injection had after bacterial contamination [97].

Any antiseptic solution used as a prep for regional anesthesia procedures must be broad spectrum, with fast onset, long duration, minimal toxic skin effects, and not be inactivated by biological fluids. The majority of the literature reviews povidone-iodine and chlorhexidine gluconate.

Chlorhexidine is effective against Gram-positive and Gram-negative bacteria and yeast, alters cell wall permeability, precipitates cell membrane and cytoplasm components and adheres to the stratum corneum for prolonged effect. The addition of isopropyl alcohol potentiates its bactericidal effects. It remains effective in the presence of blood and other body fluids, produces few skin reactions and has few pathogens resistant to it [89]. It is currently FDA approved for surgical skin preparation. There is insufficient testing for acquisition of an FDA approval for preparation for a regional anesthesia procedure. Currently there are no reports of neurologic or central nervous system adverse events due to chlorhexidine used as a skin antiseptic preparation.

Povidone-iodine is also effective against most Gram-positive and Gram-negative bacteria, though its mechanism is dependent on its continued release of iodine, which disrupts protein synthesis. Because of its mechanism, povidone-iodine requires several minutes for maximum effectiveness. Release of iodine is accelerated by isopropyl alcohol, but it is inactivated with organic material such as blood or pus.

Some patients may have acute skin or systemic reactions to iodine, and certain strains of *S. aureus* have developed resistance to it. It is currently FDA approved for preparation of the surgical site and does not have an FDA indication for preparation of a site for a regional anesthesia procedure due to lack of clinical data.

In head to head comparisons of povidone-iodine and chlorhexidine with alcohol solutions, chlorhexidine has proven more effective in all [98–105] but one [106] study, although there is evidence that the two solutions may be used sequentially with greater benefit than chlorhexidine alone [107]. The solution which seems to have the broadest spectrum of bactericidal activity was 0.5% chlorhexidine with 80% ethanol, and the authors of the Advisory Panel suggested that this solution may be the most effective way to maintain asepsis for a prolonged period of time to diminish the risk of bacterial colonization [89]. The application of either chlorhexidine or iodine may be done by spray or via scrub without a significant difference in the efficacy of antimicrobial activity, though the spray application is faster [108, 109]. Of significance, if the spray technique is used, it has been shown that the nozzles of multi-use chlorhexidine spray bottles do not become colonized even after a median follow-up of 5 months, in contradistinction to multi-use iodine bottles [89, 109, 110]. Chlorhexidine dressings have been shown to significantly reduce the number of epidural catheters colonized on removal and reduce the overall bacterial count by a factor of 100 compared to nonmedicated dressings [111, 112]. These findings may also be extrapolated to peripheral nerve catheter dressings, with the understanding that some dressings may see more movement and be in an area which is inherently more conducive to bacterial growth, such as over a femoral nerve catheter.

Regional Anesthesia in the Immunocompromised Patient [91]

Regional anesthesia is also complicated by the patient whose immune system is compromised, as his or her susceptibility to infection is increased, and both the frequency and severity of infection are increased; however, it has been shown that regional anesthesia diminishes the suppression of immune function caused by surgical stress [113]. Horlocker and Wedel published the findings of the Practice Advisory Panel on the Infectious Complications regarding regional anesthesia in these patients [91].

The primary barrier to infection, the skin will be breeched by both surgery and the regional anesthesia, enforcing the importance of aseptic technique. Both cellular and humorally mediated immunity are suppressed for several days after surgery. Neuraxial anesthesia has been shown to significantly diminish the surgical stress response, although it must be continued in the postoperative period. Therefore, the patient population that would have a significant benefit in preserving what remained of their immune system also represents a greater risk of meningitis, epidural abscess, and site infection. In 2002, there was a limited study comparing peripheral and neuraxial techniques, and their effect on surgical stress. This showed that epidural was superior to peripheral nerve blocks for suppression of stress hormones, although

pain scores were equivalent [114]. Therefore, the techniques that may limit risks of epidural abscess or meningitis may also be less effective at preserving the remnant of immune response that remains in the immunosuppressed patient. The consequences of meningitis and epidural abscess are sufficiently dire that great precautions are merited to avoid these sequelae. Untreated, bacterial meningitis has a 100% mortality rate, and even with appropriate and timely antibiotic therapy mortality remains at 30%. Epidural abscesses are primarily bacteria, although fungal and mycobacterial abscesses may present. Immunosuppression is an independent risk factor for the formation of epidural abscess. Complete recovery is reported in less than 40% of cases and most often when surgical intervention is undertaken in less than 36 h.

Central nervous system infections due to neuraxial anesthesia is rare, although older studies reported a smaller incidence, 1.1 per 100,000 in a Finnish study [115] as opposed to a later study which reported the incidence of epidural abscess to be 1:1,930 catheters [116]. In this study, eight of nine epidural abscesses were in immunocompromised patients with an extended duration of epidural catheterization. *S. aureus* was isolated in two-thirds of these cases. A 10-year study published in 2004 demonstrated a lower incidence of epidural abscess and meningitis [117]. This was a retrospective study from Sweden reviewing 1,260,000 spinal anesthetics and 450,000 epidural anesthetics, including 200,000 labor epidurals and reported 42 infectious complications, 13 of which were epidural abscesses. Epidural abscesses occurred only in the epidural group, whereas there was perforation of the dura in 25 of 29 meningitis cases, for a total incidence of meningitis in central neuraxial procedures of 1:53,000. Interestingly, they found that epidural abscess was related to the immune function of the patient, but the patients who had meningitis after spinal blockade were not immunodeficient. All positive cultures in the epidural abscess patients contained *S. aureus*, whereas 11 of the 12 positive cultures in the meningitis patients contained alpha-hemolytic streptococci and only one had *S. aureus*. Although the incidence of neurologic complication has increased, it is not certain if this is a true increase, which may reflect change in patient population and patient risk factors, or if it is a reporting bias.

Although it has been shown that immunocompromised patients are at greater risk for CNS infection with neuraxial blockade, there is little data on the exact incidence of complications within a given immunocompromised population. A review of 1,620 pediatric patients demonstrated one report of a *Candida tropicalis* epidural collection in a child with metastatic cancer with complete resolution of neurologic symptoms after surgical decompression [118]. An older study of 350 cancer and HIV patients with long-term tunneled epidural catheterization demonstrated one infection per 1,702 catheter days, with infections being either deep track or epidural, or both, with skin flora as the most common cultured flora. All were treated with catheter removal and antibiotics. None required surgical intervention, and 15 of the 19 had replacement of the catheter upon resolution of the infection. It was the conclusion of these two studies that long-term epidural catheterization in immunocompromised patients is safe as long as there is a heightened clinical suspicion and

surveillance for the possibility of infection. This increased suspicion must stretch beyond the immediate post-procedure period in immunocompromised patients as evinced by a case report of epidural abscess in an elderly patient who had epidural steroids as well as two epidural catheters for shingles and presented 3 weeks later with an epidural abscess covering T5–T9 [119].

Two separate viral infections must be considered in terms of neuraxial anesthetic management. There is a theoretical concern in herpes simplex virus type 2 (HSV-2) of introduction into the central nervous system resulting in aseptic meningitis as the presenting clinical picture. Multiple studies have been done, primarily in the obstetric population where the majority of patients had recurrent HSV-2, and there remains insufficient data to state the risk of CNS infection due to neuraxial anesthesia during a primary infection. Epidural and spinal anesthesia in patients with HSV-2 recurrences appears to be safe [91, 120–122].

Human immunodeficiency virus presents a different set of concerns. Since CNS involvement occurs during the first few weeks of infection and 90% of HIV patients have neuropathic abnormalities on autopsy [123], the introduction of the virus into the CNS is a moot point in HIV. The concern is that there are many different factors which may contribute to neuropathy: the virus itself, opportunistic infections, retroviral medications, an increased risk of worsening of neurologic deficits in the perioperative period due to regional anesthesia, the surgical intervention, and surgical positioning. The many factors contributing to neuropathy provide a confusing picture, decreasing the ability to divine the specific cause of a neurologic deficit. There are studies of HIV-positive parturients who were of relatively stable immune status and early in the disease process who had safe spinal or epidural anesthesia [124–126] and of a small sample of HIV-infected men who had epidural blood patches without sequelae that could be directly attributed to the blood patch [127]. Blood patches in HIV-positive patients have been debated without definitive resolution in the literature [128, 129]. Those opposing blood patching in the presence of HIV stated that there was a subset of HIV patients with minimal CNS involvement, and the introduction of virus into the CNS with a blood patch also introduces risk of accelerating CNS involvement, and that conservative treatment should be considered [129]. Those arguing for the safety of epidural blood patch in HIV-infected patients point out that 100% of the patients have CSF positive for HIV if there are four samples taken over 6 months and that subarachnoid block is used for anesthesia in parturients which carries the same theoretical risk of introduction of blood bearing a viral load into the CSF [128].

The etiology of immunosuppression in a patient brings up the possibility of secondary effects with complications occurring primarily in two categories: hemorrhagic and neurologic. With immune system compromise comes opportunistic infection, and the most common disorder of hemostasis during an infection is thrombocytopenia, although one must also be wary for disseminated intravascular coagulation. In addition, some anti-infectious agents will result in thrombocytopenia through bone marrow suppression or further immune system compromise. If patients have evidence of petechiae or purpura, coagulation studies and a platelet

count should be obtained. Currently, given the risk of significant infectious and hemorrhagic complications, there is no role for neuraxial anesthesia in the patient with an active untreated infection unless extraordinary circumstances may mitigate the significant risks. In the face of cancer, there are both hemorrhagic and thromboembolic risks, as well as considerations for certain types of cancer. Solid tumors predispose patients to thromboembolic events, and hemorrhagic complications occur more frequently with acute leukemia although there is a spectrum of events with any tumor. Ninety percent of patients with metastatic disease demonstrate laboratory evidence of DIC, although a much smaller percentage have clinical evidence of the dysregulation of their hemostasis [130]. Patients in acute DIC are far more likely to have a hemorrhagic complication, while those patients with a fulminating chronic DIC are far more likely to have a prothrombotic complication. Given the far higher thrombotic risk during the perioperative period for patients with cancer, aggressive thromboprophylaxis is required and is important to consider when discussing possible anesthetic management plans. The most common cause of bleeding for cancer patients is thrombocytopenia, which may be due to decreased production, sequestration, or increased destruction, both by the malignant process and its treatment regimen. If a patient has recently had chemotherapy or has a myoproliferative process, a targeted evaluation of their hemostasis is warranted. Specifically, in the presence of circulating leukemic cells, neuraxial techniques should be avoided. Dural puncture in acute lymphoblastic leukemia patients may seed the CNS with blast cells, significantly worsening the probable outcome.

Patients who are immunocompromised often have a preexisting neurologic deficit either due to their disease, the treatment for their disease, or both. Any further insult, in the form of needle trauma, local anesthetic toxicity, or ischemia due to blood vessel spasm has a synergistic effect, either exacerbating old neurologic damage or creating new neurologic deficits. If considering a spinal or epidural anesthetic, a review of recent radiographs is necessary to rule out vertebral metastases at the desired level of entry, and a clinical exam is required to rule out spinal cord compression. Peripheral neuropathy is a common complication of chemotherapy. It is seen in 100% of people who take vincristine, 85% of those who have at least 300 mg/m^2 of cisplatin, 60% of patients who have taken at least 250 mg/m^2 of paclitaxel, and is common in several other chemotherapeutic agents [91].

The incidence of infection in the setting of peripheral nerve block has not been widely studied, and that is also true within the subset of immunocompromised patients. Several infectious complications of peripheral nerve block were cited earlier in the chapter. The complications in the literature range from colonization of the catheter and local redness [87] to necrotizing fasciitis after a single shot axillary block [131]. It is significant to note that the majority of reported peripheral nerve block complications involved immunocompromised patients, most often with diabetes, and were frequently, though not always, associated also with the presence of an indwelling catheter; the duration of the catheter is significant. Although the data are limited, it seems that the risk factors for continuous peripheral nerve block with an indwelling catheter are similar to the risk factors surrounding infection of an epidural.

Regional Anesthesia in the Febrile or Infected Patient [90]

Although dural puncture has been considered a risk for development of meningitis, central neuraxial infections are very rare, with a series of 65,000 spinal anesthetics yielding only three cases of meningitis [132], a review of 50,000 epidurals yielding no CNS infection, and a multicenter prospective study of 40,640 spinal and 30,413 epidural anesthetics yielding no infectious complications [85]. Moen et al. reviewed 1.26 million spinals and 450,000 epidurals done in Sweden between 1990 and 1999 and found 29 cases of meningitis and 13 epidural abscess cases [117]. Given the low incidence of these complications, very large data sets are required to extract meaningful information. The limited data available suggests that a patient with bacteremia receiving an epidural or spinal anesthetic is at greater risk for CNS infection.

Initial clinical and laboratory investigations were conducted in 1919, Wegeforth and Latham reported their results of lumbar punctures in military personnel during a meningitis epidemic in the *American Journal of Medical Science*. Six of the 93 lumbar punctures done in patients who did not have meningitis at the time of the procedure did have septicemia and five of those six subsequently developed meningitis, although there was a notable lack of controls to make this more than an association. An experimental animal model of meningitis after dural puncture in the setting of septicemia was also published by Weed et al. in *JAMA* that year as well. A replicate of this animal model was published in 1992, with 12 of the 40 rats subjected to cisternal puncture after *Escherichia coli* bacteremia developing meningitis [133]. Data from studies in humans examining meningitis has been mixed however. Studies with no significant difference in the incidence of meningitis [134], those with an association and no clear causal relationship [135], and those with a significantly different incidence in meningitis [136] have been published with weaknesses associated with each study. Most reports of meningitis after dural puncture are related to either a break in sterile procedure or due to unusual or nosocomial organisms [90]. Epidural anesthesia may also result in meningitis without epidural abscess. Two parturients demonstrate this in the literature [137], one patient with an area of cellulitis at the insertion site and meningitis from *Streptococcus faecalis,* and the other patient with CSF, urine, blood, and vaginal cultures positive for *Staphylococcus uberis*. The first patient's most likely cause was cellulitis at the insertion site, though other causes could not be excluded, and it was posited that the second parturient acquired meningitis from hematogenous seeding from the vagina during delivery.

As mentioned previously, epidural abscesses are a very rare though serious CNS complication of lumbar puncture. The overall incidence of epidural abscess of all causes is cited as 0.2–1.2 cases per 10,000 in tertiary care hospitals [138], although that citation was from 1975. There are only case reports and retrospective reviews, and the incidence of epidural abscess varies widely from none in 9,232 epidural anesthetic cases [139] and 2 in 13,000 cases [140] to 9 in 17,372, or 0.05% [116]. In one study, there was a significant difference in the incidence of epidural abscess between the university (1:5,661) and non-university (1:796) setting. Additional common factors in these patients included increased catheter duration, immunocompromised patients with chronic disease, and perioperative antithrombotic

therapy, most often low-molecular weight heparin. Another patient population expected to incur additional risk of epidural abscess would be parturients with chorioamnionitis, 8% of whom are bacteremic. This however is not seen in the literature. Two retrospective studies [141, 142] identified 319 and 531 women respectively with chorioamnionitis. All of these women received epidural analgesia for labor, with only 43 and 123 patients respectively receiving pre-epidural antibiotics. In the second study, fully one third of the patients received no antibiotics during their entire labor. None of these women had an epidural abscess. In fact, in a 4-year review of 505,000 labor epidurals, one review found only one epidural abscess [143]. It is proposed that, although pregnancy is a relatively immunosuppressed state, there is a short duration for the epidural catheter, and the patients generally have fewer comorbidities to predispose them to opportunistic infection.

Ultimately, the decision of whether to perform a regional anesthesia technique on a febrile patient must be made based on evaluation of that patient. The experimental and epidemiological data does suggest an association between dural puncture during bacteremia and meningitis, although this association is primarily from the pediatric population, which has a much higher incidence of meningitis. Animal models of dural puncture during untreated bacteremia frequently had bacterial counts far higher than are clinically relevant. Nevertheless, given the possible association, expert opinion would advise not performing neuraxial anesthesia in a floridly bacteremic patient without some very persuasive extenuating circumstances. If a patient however has received appropriate antibiotics to treat systemic infection and demonstrated an appropriate response, all data suggests that it is safe to perform spinal anesthesia provided there is antibiotic dosing prior to dural puncture. Epidural anesthesia data under the same circumstances is reassuring, but limited and no recommendation for epidural anesthesia under these circumstances is made by the Advisory Panel. A summary of the recommendations of the Practice Advisory Panel on the Infectious Complications for regional anesthesia procedures is provided below [89–91]. Please note that the recommendations and the data surrounding the infection risks in chronic pain treatments are omitted from this review and summary.

Recommendations of ASRA Practice Advisory Panel

Grade A

1. Thorough hand washing prior to any regional anesthesia procedure is required. Alcohol-based antiseptic solutions provide the maximal antimicrobial protection.
2. Sterile surgical gloves should be used in addition to hand washing, both as a barrier to infection and as protection for the provider from blood-borne pathogens.
3. Alcohol-based chlorhexidine solutions significantly reduce the probability of site and catheter colonization and have the most rapid and potent bacteriocidal activity. Therefore, these should be the antiseptic of choice before any regional anesthesia procedure.

4. Current data suggest that patients with systemic infection who have been given appropriate antibiotic therapy and have shown a positive response to therapy such as a decrease in fever may safely undergo spinal anesthesia although an indwelling epidural or intrathecal catheter in this setting remains controversial.

Grade B

1. Higher bacterial counts have been identified on health care workers who do not remove jewelry before hand washing. It is recommended that all jewelry be removed before washing hands in preparation for a regional anesthesia procedure.
2. Surgical masks reduce the likelihood of contamination from bacteria in the clinician's upper airways and, though they have not been found to significantly reduce the incidence of infectious complications in regional anesthesia, remain an important barrier against blood-borne pathogens as recommended by the Occupational Safety and Health Administration.
3. The use of bacterial filters is not supported with epidural or perineural catheters whose short duration is limited to a period of days of use.
4. The attenuated inflammatory response within the immunocompromised patient may diminish clinical signs and symptoms associated with infection, and the range of potential range of pathogens is expanded, including atypical and opportunistic microorganisms. Early consultation with infectious disease is advised if there are suspicions of infection in order to initiate early and effective therapy.
5. Delays in diagnosis and treatment of central nervous system infections result in worse neurologic outcomes and increased mortality.
6. The risk of epidural abscess increases with the duration of epidural catheterization.
7. Central neuronal block has been shown to be safe in patients with recurrent HSV infection, although intrathecal and epidural opioids have been associated with exacerbations of HSV-1.
8. Central neuraxial infections such as arachnoiditis, meningitis, and abscess after spinal or epidural anesthesia are rare.
9. Available data suggests that spinal anesthesia may be safely performed in patients at risk for low-grade transient bacteremia after dural puncture.
10. A delay of even a few hours in diagnosis and treatment of major CNS infection may significantly worsen neurologic outcome.

Grade C

1. The decision to perform a regional anesthetic technique must be made on an individual basis considering the anesthetic alternatives, the benefits of regional anesthesia, and the risks of CNS infection as well as the risk of hemorrhagic or neurologic complications.
2. Insufficient data exists regarding the safety of neuraxial anesthesia in the face of primary HSV-2 infection, but reports exist of viremia, fever, and meningitis, suggesting a conservative approach.
3. There are little data on neuraxial and peripheral techniques in HIV-infected patients, but what data exist suggests that these techniques, including

epidural blood patch, may be performed safely, although one must consider any preexisting neurologic pathology.
4. The decision to perform a regional anesthetic technique must be made on an individual basis, with consideration of alternate anesthetic options, the relative benefit of regional anesthesia, and the relative risks, including CNS infection.
5. Although results are conflicting, expert opinion is that, with the exception of extraordinary circumstances, neuraxial anesthesia should not be performed in patients with untreated systemic infection.

Insufficient Data

1. There is insufficient data to recommend the duration and method of hand washing (standard hand washing versus full surgical scrub).
2. Although there are data from the ICU that the use of gowns makes no impact on infection rates, there are insufficient data to make recommendations on the use of gowns for regional anesthesia.
3. Although epidural catheters should be removed in the presence of local erythema and/or discharge, there are no satisfactory data to suggest that infection at a remote site or the absence of antibiotic therapy is a risk factor for infection.

Conclusions

In conclusion, the choice of regional anesthesia is still an individual one tailored to the comorbidities and desires of each patient, balanced with a risk profile specific to the case. Some of the theoretical benefits posited by the physiologic effect of regional anesthesia have been shown to be true, while others do not seem to have the expected clinical effect. There are distinct pulmonary benefits, especially in thoracic and large open abdominal procedures. These benefits are less apparent in minimally invasive procedures. Decreases in postoperative myocardial infarction can be seen for patients undergoing cardiac or vascular procedures, but only if they are in a high-risk category for myocardial infarction and if neuraxial analgesia is continued at least 24 h postoperatively. There is ample evidence that epidurals improve time to first ambulation, time to reach physical therapy goals, and time to discharge in Orthopedics. Epidurals also decrease time to return of bowel function and reduce length of stay in colorectal surgery.

Clinical Pearls

Blood Loss and Transfusion Requirements

- Regional anesthesia decreases blood loss in total hip replacement by the mechanism of systemic blood pressure control.

Cancer Recurrence and Regional Anesthesia

- Regional anesthesia decreases postoperative immunosuppression and prevents intraoperative catecholamine and stress hormone surges. This preserves natural killer cell function, which has been shown to improve long-term survival in several different cancers. Controlled trial results are pending with the possibility of a major impact on the survival rates of many cancers.

Mortality

- The biggest effect on long-term mortality has been the multimodal approach to patient care with a comprehensive enhanced recovery pathway, of which regional anesthesia is only one aspect.

Cardiovascular Outcomes

- Thoracic epidurals may reduce postoperative myocardial infarction, although some studies demonstrating this benefit were less rigorous. The previous benefit of decreased DVTs with neuraxial analgesia has been obviated by thrombosis prophylaxis. Regional anesthesia does seem to have some advantages in vascular surgery, through sympatholysis, vasodilation, and decreased fibrinolysis. Regional anesthesia results in fewer graft failures than with general anesthesia.

Pulmonary Outcomes

- Regional anesthesia does not impair respiratory mechanics, in fact, it improves them. Thoracic epidurals have been shown to decrease respiratory failure and pneumonia in high-risk patients.

Gastrointestinal Outcomes

- Regional anesthesia as part of a multimodal approach to patient care has been shown to provide superior pain control, decreased opiate requirements, and reduced duration of postoperative ileus. Reduced PONV is supported by some studies, though the findings are not uniform in the literature.

Rehabilitation and Length of Stay

- Regional anesthesia permits earlier mobilization, but the possibility of motor block and residual weakness must also be managed. Peripheral nerve blocks may have an advantage in decreasing postoperative motor block. Regional anesthesia is also associated with a decreased rate of readmission in complex knee surgery.

Postoperative Pain Relief

- Pain control in continuous peripheral nerve catheters and epidurals is equivalent, but patient satisfaction and side-effect profile are better in continuous peripheral nerve catheters. Peripheral nerve catheters can be run in patients discharged home. Both provide superior analgesia compared to PCA.

Chronic Pain

- Although the argument that regional anesthesia should decrease chronic pain is theoretically persuasive, there are mixed data as to whether this is seen clinically.

Postoperative Cognitive Decline

- Regional anesthesia and general anesthesia have an equivalent incidence of POCD. Emerging data indicates that there may be a decrease in POCD with postoperative regional analgesia for pain control.

Patient-Oriented Outcomes

- Patient-oriented outcomes are important considerations when planning an anesthetic. Patient satisfaction is improved with regional anesthesia and analgesia compared to placebo or with regional anesthesia compared to general anesthesia and PCA.

Neurologic Complications

- The incidence of neurologic complications is very low but differs by block. Permanent nerve injury is far less common than transient neurologic symptoms.
- ICU patients are at increased risk for nerve injury.

Infectious Complications

- Many continuous peripheral nerve catheters are colonized with bacteria, and yet have no active signs of infection. Diabetes may present an increased risk for infectious complications, as does lack of prophylactic antibiotics, catheter duration >48 h, male sex, and ICU monitoring.

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31

Regional Anesthesiology Education

Jonathan C. Beathe

Contents

Expertise in the practice of regional anesthesia is not achieved with technical skill alone. Although the technically adept can adequately deliver local anesthetic adjacent to target nerves, it is the regional anesthesiologist who combines such applied anatomy with the practice of perioperative medicine. The nontechnical aspects of the practice of regional anesthesia cannot be ignored: clinical judgment, patient selection and communication, complication management and avoidance, a wide breadth of knowledge of physiology and pharmacology, and the formulation of an appropriate care plan with contingencies, are examples. A discussion of regional anesthesia education should first acknowledge that the discipline, at its core, is the practice of medicine. Maintaining this perspective, this chapter aims to provide a pertinent overview addressing past, present, and future challenges and directions within this exciting, rapidly evolving field.

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History

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

Evolution of Practice

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

Multiple surveys have demonstrated how regional anesthesia training experience has evolved. For instance, training programs in 1980 reported the use of a regional anesthetic in 21.3% of cases. However, the discrepancies between training programs (2.8–55.7% of total delivered anesthetics) led to concern that some residents would not gain adequate experience to meet the growing demand to provide regional anesthetics [7]. When the popularity of epidural anesthesia and resident exposure to pain consultations expanded in the 1980s, survey data reflected the shift. In 1990, the reported use of regional anesthetics increased to 29.8% [8]. Unfortunately, wide variation in the use of regional anesthesia between training programs remained unchanged. The disparities in the number of regional anesthetics performed between residency training programs improved over the course of the next 10 years; however, the overall use of regional anesthesia by residents in the year 2000 appeared to

plateau at 30.2% [9]. Since then, it is unlikely that the overall utilization of regional anesthesia by resident training programs has increased markedly. Consider 2006 data from a prominent tertiary teaching hospital: even when selecting for surgical procedures amenable to a regional anesthetic, only 36.5% of such patients are provided regional anesthesia [10].

As of July 1, 2008, the latest ACGME Program Requirements for Graduate Medical Education in Anesthesiology state the minimum regional anesthesia clinical experience that should be obtained by each resident: 40 epidural, 40 spinal, and 40 peripheral nerve blocks [5]. Each resident should also have "clinical experience with interventional pain procedures." Evidence suggests that most residents meet or exceed these standards, with the exception of peripheral nerve block anesthesia. A 1999 survey discovered that 50% of graduating clinical anesthesia (CA) year three residents, although confident in performing neuraxial anesthesia, lacked adequate experience with many commonly performed peripheral nerve blocks [11]. These findings are consistent with a subsequent report of the year 2000, when approximately 40% of residents had inadequate exposure to these techniques [9]. This is not surprising, considering that a minimum number for individual peripheral nerve block techniques is notably absent from ACGME requirements.

Simply expanding such requirements may not be a solution, as debate continues regarding what constitutes adequate experience and proficiency in regional anesthetic procedures. Consideration of the number of cases alone does not necessarily reflect resident mastery of the regional anesthetic technique. Studies that address this topic also vary greatly in methodology and lack standardization. Existing studies do, however, provide insight into what level of experience may be necessary to achieve a specific endpoint of success. In one of the earliest investigations of trainee "learning curves" in regional anesthesia, Kopacz and colleagues demonstrated that significant improvement in success rate occurs after 20 spinal and 25 epidural anesthetics [12]. They also reported that a 90% success rate was not achieved until after performing 45 spinal and 60 epidural anesthetics. In a subsequent analysis of resident training at a Swiss institution using a standardized self-evaluation questionnaire, different results were demonstrated. Although a rapid improvement of success was also observed during the first 20 attempts, 71 attempts at spinal anesthesia were required to reach a success rate of 90% [13]. The study described epidural anesthesia as the most difficult task, with a success rate of only 80% after 90 attempts (Fig. 31.1). With the axillary approach to brachial plexus blockade, only a 70% success rate was achieved after 20 cases (Fig. 31.2). The first assessment of the number of attempts necessary for a resident to achieve proficiency in interscalene anesthesia demonstrated that 87.5% report "autonomous success" after 15 cases [14]. Of note, only 50% of residents were able to perform interscalene anesthesia autonomously after seven to nine previous block attempts. These studies, despite their limitations, suggest that the current RRC requirement that mandates experience with 40 *unspecified* peripheral nerve blocks is likely inadequate to ensure proficiency with various *specific* peripheral nerve blocks.

With the addition of emerging technology and novel techniques to approach traditional peripheral nerve blocks, additional variables are added to the rational

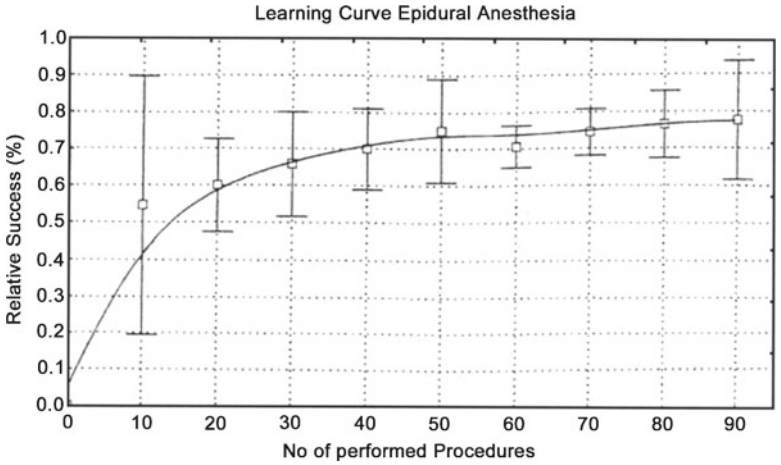

Fig. 31.1 Learning curve – epidural anesthesia

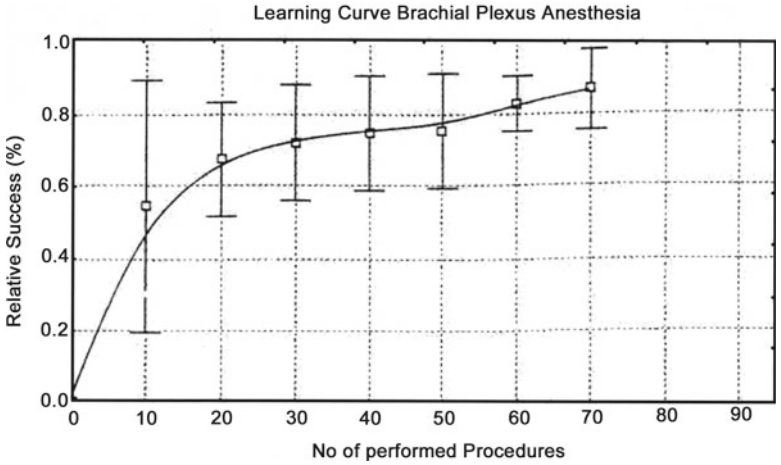

Fig. 31.2 Learning curve – brachial plexus anesthesia

determination of training requirements. For example, the manual skill required in ultrasound-guided regional anesthesia (UGRA) of the brachial plexus is not directly applicable to the safe performance of interscalene blockade utilizing the paresthesia technique. Sites and colleagues, in a study characterizing novice behavior associated with learning UGRA, explore the entirely new set of skills involved in UGRA [15]. By using video analyses of 520 nerve blocks performed by anesthesia residents, a multitude of errors such as "needle not visualized during advancement" were assessed. They observed that by the 60th block, the trainee was still committing

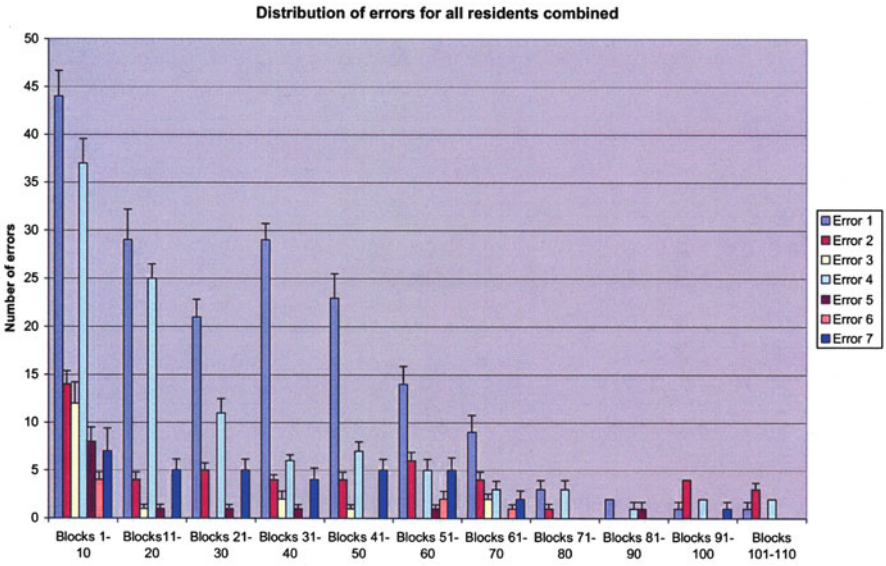

Fig. 31.3 Distribution of errors for all residents combined

an average of 2.8 errors per procedure (Fig. 31.3). It is a reasonable hypothesis that the necessary clinical exposure associated with proficiency is related to not only the specific peripheral nerve block, but also to the method used to accomplish the technique. Few studies investigate the influence of ultrasound and related imaging technology upon learning curves for peripheral nerve blocks. Early data suggest, however, that residents inexperienced in UGRA can rapidly master basic ultrasound skills in a simulated interventional procedure [16]. Despite incomplete science, it is clear that emerging technology will continue to play a role in anesthesiology training. Such influence should be considered in future assessment of the adequacy of resident education. For example, a resident primarily exposed to UGRA during residency may be less inclined to perform an appropriate RA technique by another means if ultrasound technology is unavailable in their subsequent practice. Questions like this abound, but it is evident that resident exposure to regional anesthesia – particularly in the realm of peripheral nerve blockade – can be improved.

Overcoming Obstacles and Expanding Practice

When attempting to improve the regional anesthesia exposure of residents, it is helpful to understand the potential obstacles that exist to expanding practice. As observed by Hanna and colleagues in a prospective observational study in 2009,

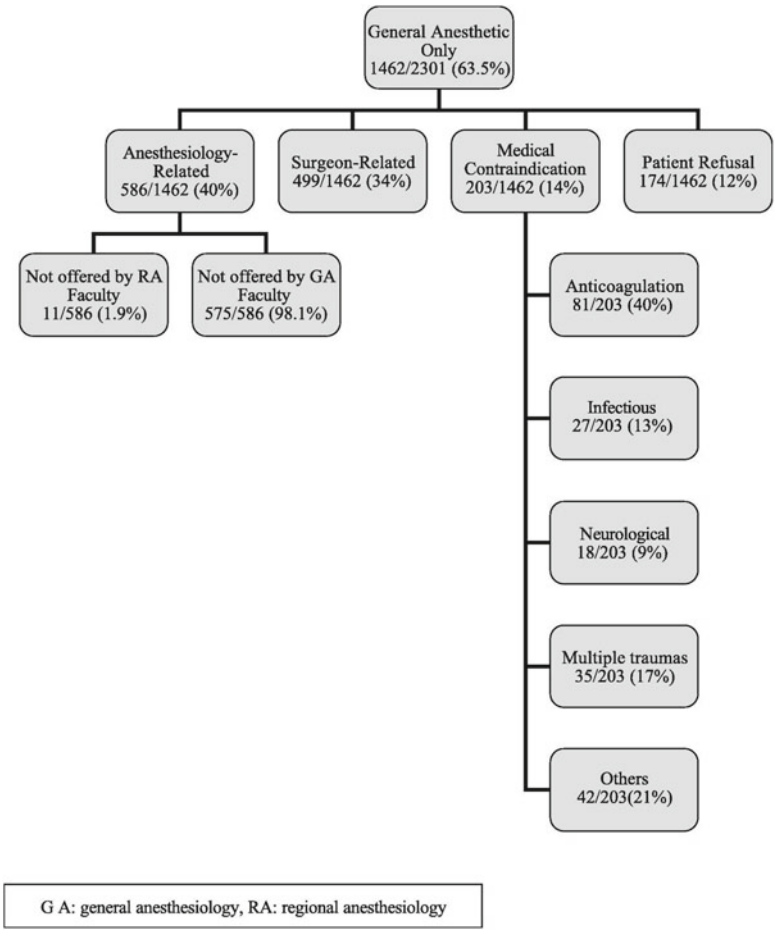

Fig. 31.4 When selecting for surgical procedures amenable to a regional anesthetic, many patients still receive general anesthesia(63.5%). This is primarily attributable (40%)to anesthesiologyrelated factors, such as a lack of regional anesthesia experience

a multitude of factors shape resident experience in regional anesthesia [10]. In over 2,000 cases amenable to a regional anesthetic, the frequency and reasons for not performing such a technique were investigated. Surprisingly, they found that anesthesiology-related factors – not the surgeon, patient, or medical reasons – were the primary factors for not performing a regional anesthetic. They also observed, predictably, that designated regional anesthesia faculty performed regional anesthesia more often (68% of cases). In over 98% of the cases in which regional anesthesia was not performed, despite being an appropriate anesthetic selection, staff members not designated as "regional anesthesia faculty" were involved (Fig. 31.4). These findings support the assertion that the process of increasing resident exposure to

regional anesthesia is facilitated by the presence of dedicated, trained faculty. Even with the addition of regional anesthesia experts to faculty rosters, the specialty faces additional challenges to improving the resident experience. The apprenticeship model of education remains the predominant teaching style in residency training programs, despite having significant limitations. Problems with inconsistency in the quality of learning experiences need to be addressed. Additional challenges include the development of improved methods of trainee evaluation, and expanding curriculum development to achieve consistency in both technical and clinical achievement.

Curriculum Development

As most physicians lack formal training in education, the apprenticeship model of education remains the predominant teaching style in regional anesthesia. Unfortunately, progress in education methodology has not kept pace with the significant advances in medical technology and standards of care. Recently, educators in regional anesthesia have identified ways to improve upon existing models of teaching. Initial efforts to improve education have focused on increasing the numbers of peripheral nerve blocks performed by trainees. Martin and colleagues, outlining the use of a CA-3 resident in the preoperative area to perform regional anesthesia techniques, have described such an educational model [17]. This relatively simple modality significantly increased resident exposure to regional anesthesia (Fig. 31.5). In support of these findings, it has been demonstrated that residency

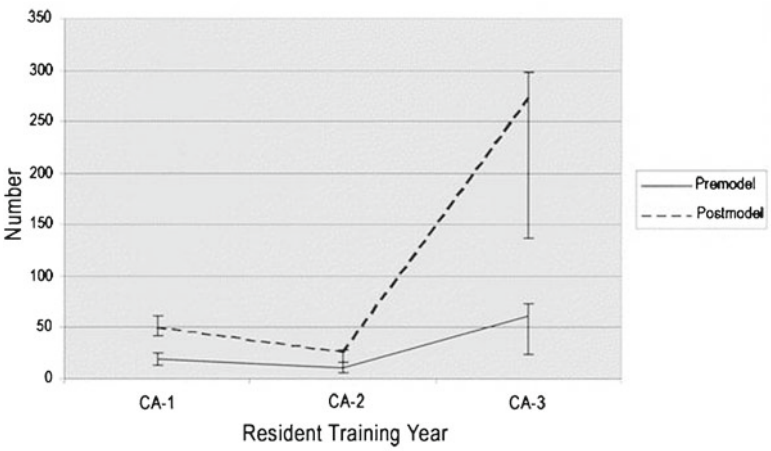

Fig. 31.5 An organized regional anesthesia rotation increases resident exposure to nerve blocks

Table 31.1 Clinical expectations during the regional anesthesia rotation (from [19])

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

programs that include a specific peripheral nerve block rotation expose their trainees to a greater number of peripheral nerve block techniques [18]. More recently, the focus of curricula has expanded to complement traditional patient care experiences with novel educational activities. In their sentinel article, Smith and colleagues at the Mayo Clinic have described in detail their institution's approach to regional anesthesia education [19]. As stated, the primary educational objectives of their curriculum include (1) standardizing educational content, (2) quality care and patient safety, and (3) resident evaluation and improvement. Some highlights of this "learner-centered" approach include a comprehensive "preclinical" educational program for UGRA, a list of criteria expected of residents during patient care, and CA-2 or CA-3 residents functioning in the role of a teacher of CA-1 residents during the 8-week rotation in regional anesthesia. Placing a senior resident in the role of educator is supported by evidence of potential benefit to academic performance and perceptions of clinical competency [20]. Clinical expectations during the regional anesthesia rotation and the performance checklist for UGRA proficiency are reproduced in Tables 31.1 and 31.2. Their work is a significant contribution

Table 31.2 Performance checklist for ultrasound-guided regional anesthesia proficiency (from [19])

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

to advancing the quality of regional anesthesia training and can be adapted to accommodate the needs of various institutions. However, work is needed to demonstrate the validity of this approach to improving resident education.

Additional challenges in curriculum development were created with the introduction and rapid evolution of UGRA. Questions surrounding standards of care, assessment of competency, and the definition of expert performance naturally follow the emergence of new technology. Efforts to define the scope of practice of UGRA have been summarized by the ASRA and the European Society of Regional

Anaesthesia and Pain Therapy (ESRA) Joint Committee Recommendations for Education and Training in UGRA [21]. This collaborative effort is an example of the profession accepting responsibility for self-regulation and practice improvement, while recommending that UGRA privileges be based at the individual institution level. The stated goals of the document are to (1) define and structure the common tasks used when performing an ultrasound-guided nerve block, (2) articulate the core competencies and skill sets associated with UGRA, (3) suggest a training process for both established practitioners and residents, and (4) recommend the establishment of a quality improvement (QI) process for UGRA. The skill sets that the ASRA-ESRA Joint Committee has associated with proficiency are reproduced in Table 31.3. The document provides an additional framework to enhance existing UGRA curricula, both for residency training and postgraduate pathways. It also further establishes the representative tasks that define the practice of UGRA, which aids the development of novel educational tools such as simulator-based instruction.

As curricula continue to be optimized, the utilization of the multitude of emerging educational resources will also expand. Simulator training has already established an important role in medical education. For example, laparoscopic simulator training leads to reliably fewer errors during actual surgery [22]. Just as military pilots are more likely to respond appropriately to rare emergency situations after related simulator training [23], anesthesiologists may handle emergencies more effectively if they have been previously exposed to the crisis situation in simulated form [24]. Indeed, the expansion of simulation education in anesthesia training is likely to benefit both practitioner and patient. Examples of emerging educational resources with potential to optimize curricula are listed in Table 31.4.

Fellowship Training

Regional anesthesia practice is not unlike most professions in that considerable time is required to establish expert performance. Evidence also exists that superior medical treatments are linked to more extensive training and specialization in associated medical fields [25]. As such, fellowship training in regional anesthesia should be considered a means to excel beyond basic competencies and become an expert in the field. Formal regional anesthesia fellowships emerged in the early 1980s, and subspecialty training offered by regional anesthesia fellowships has grown substantially to include 36 institutions to date (Table 31.5). However, with the absence of uniform standards, early programs did vary in duration, organization, and objectives. It was not until October 2003, after collaboration with the directors of several regional anesthesia fellowship programs, that the first Guidelines for Regional Anesthesia Fellowship Training were developed [26].

Table 31.3 Skill sets associated with proficiency (from [21])

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

Table 31.4 Emerging educational resources

Simulation Education
Challenging clinical scenarios in both leadership and supportive roles
Management of regional anesthetic emergencies
Multimedia Resources
Performance of virtual regional anesthesia techniques
Interactive presentation of relevant anatomy
Three-dimensional animation [32]
Procedural training videos
Video analysis to teach and evaluate skills [33]
E-learning
Web-based learning modules
Online problem-based learning
Standardization of curriculum
Online coursework assessments

The guidelines recommend the necessary components of subspecialty fellowship training, emphasizing the clinical foundation of regional anesthesia, educational curricula, and opportunity for academic achievement. This document was reviewed in 2006 and again in early 2009. Publication of revised guidelines is anticipated. Although they extend beyond clinical considerations, an important goal of the published guidelines is to provide a framework to progress beyond basic proficiency and achieve focused clinical expertise. With rapidly emerging technologies and new procedures, it becomes more difficult for physicians to safely integrate the latest regional anesthesia techniques into their practice. As mentioned, evidence exists that residency training alone is not adequate to achieve mastery in many regional anesthesia techniques, particularly peripheral nerve blockade. This deficiency likely translates to fewer acute pain management options for the patients of our graduating residents. In this regard, additional fellowship training can broaden the pain management strategies used by practitioners by providing the necessary expertise. An additional argument for fellowship training is to provide an expansion upon the limited experience that residency "block room" regional anesthesia training provides. The intraoperative portion of a regional anesthetic is not to be underestimated, as myriad challenges, complications, and emergencies occur during this time. Fellowship training provides further experience to anticipate, recognize, and appropriately treat the patient in a timely fashion when such scenarios present themselves. Over the course of a fellowship, trainees also have the opportunity to achieve advanced exposure to regional techniques on patients with significant comorbidities such as morbid obesity, severe scoliosis, ankylosing spondylitis, or significant cardiopulmonary disease. Following regional anesthesia fellowship, trainees are empowered to apply regional anesthesia techniques to

Table 31.5 Regional anesthesia fellowship programs from the ASRA Website

Program	Location
University of California at Sand Diego	San Diego, CA
University of California – Irvine	Orange, CA
David Geffen School of Medicine-University of California at Los Angeles	Los Angeles, CA
Stanford University Hospital	Stanford, CA
Walter Reed Army Medical Center	Washington, DC
Jackson Memorial Medical Center-University of Miami	Miami, Fl
University of Florida College of Medicine	Gainesville, FL
Mayo School of Graduate Medical Education- Mayo Clinic Florida	Jacksonville, FL
The Andrews-Paulos Research and Education Institute	Gulf Breeze, FL
University of Illinois at Chicago	Chicago, IL
University of Iowa	Iowa City, Iowa
Ochsner Medical Center	New Orleans, LA
Johns Hopkins Medical Center-Johns Hopkins University	Baltimore, MD
Brigham and Women's Hospital	Boston, MA
Mayo Clinic, Mayo School of Graduate Medicine	Rochester, MN
Dartmouth-Hitchcock Medical Center	Lebanon, NH
University of New Mexico	Albuquerque, NM
Columbia University/NYPH Medical Center	New York, NY
Hospital for Special Surgery – Weill Medical College Cornell University	New York, NY
Mount Sinai Medical Center	New York, NY
St. Luke's-Roosevelt Hospital Center-Columbia University	New York, NY
Upstate Medical University	Syracuse, NY
Duke University Medical Center	Durham, NC
Wake Forest Medical Center	Winston-Salem, NC
Oregon Health & Science University	Portland, OR
Hershey Medical Center – Penn State Health Science	Hershey, PA
University of Pittsburgh Medical Center	Pittsburgh, PA
Vanderbilt University Medical Center	Nashville, TN
Virginia Mason Medical Center	Seattle, WA
University of Wisconsin Hospital and Clinics	Madison, WI
McGill University	Montreal, Qc, Canada
University of Toronto – Saint Michael's Hospital	Toronto, ON, Canada
University of Toronto – Sunnybrook Health Science Centre	Toronto, ON, Canada
University of Toronto – Toronto Western Hospital	Toronto, ON, Canada
University of Ottawa	Ottawa, ON, Canada
University of Alberta-Hospital University of Alberta	Edmonton, Alberta, Canada

clinical situations that are not traditionally endorsed. For instance, with appropriate execution and patient selection, neuraxial blockade can be a reasonable anesthetic option for hip surgery in the setting of aortic stenosis [27]. In order to ensure a

safe outcome, advanced techniques such as hypotensive epidural anesthesia [28] also require a level of expertise that fellowship training provides. Although clinical considerations are arguably the foundation of fellowship training, didactic components are not of lesser value. Particularly, as regional practice expands in an era of regulatory oversight and evolving rules and measures, it will be increasingly important to inject our specialty with research initiatives that further validate and justify our practice. Fellowship training is a platform that can be increasingly utilized to meet this end by providing important exposure to academic pursuits. With increasing interest and expansion of regional anesthesia technique and practice, fellowship training is an indispensable means to provide valued experts in the field.

The Practicing Anesthesiologist

Although fellowship training provides the definitive opportunity to achieve expertise in regional anesthesia, it is not always practical for physicians in the midst of their careers to dedicate a full year to formal training. Advances in science and technology have and will continue to challenge physicians in every discipline to stay current with modern practice and procedures. Parallels can be drawn between the practicing anesthesiologist seeking to advance his/her regional anesthesia skills and the experienced surgeon who would like to introduce a modern yet unfamiliar laparoscopic technique into his/her practice [29]. Similar questions are raised regarding the safety and efficacy of the new modality and if specific minimal educational requirements should be met [30]. Just as surgeons are faced with the daunting responsibility of ensuring the safe introduction of new procedures into practice, expert regional anesthesiologists must also adequately provide the educational opportunities required of lifelong learning and the development of new technical skills. This may take the form of the fellowship graduate arriving to a practice unfamiliar with UGRA, providing new skills and information to otherwise more experienced physicians. As fellowship graduates alone cannot meet this educational demand, a multitude of opportunities have developed to provide regional anesthesia exposure to the practicing anesthesiologist (Table 31.6). Although such opportunities provide a valuable service, extensive work remains to adequately define and assess competency in regional anesthesia. As we already know, limited training is associated with higher complication rates. Consider laparoscopic surgical data, in which the rate of complications associated with the clinical learning curve can be decreased by additional education [31]. Future efforts to accomplish the difficult task of competency assessment will allow us to further optimize patient safety in regional anesthesia.

Table 31.6 Regional anesthesia continuing education programs

Annual ASA Meeting
Annual Spring ASRA Meeting (focused on Regional Anesthesia)
Annual Fall ASRA Meeting (focused on Pain Medicine)
ASRA Excellence in Regional Anesthesia Workshops Regional Workshops
Northwestern University's Feinberg Pavilion - Chicago, Illinois
Duke University - Durham, North Carolina
ASRA Ultrasound for Pain Medicine Workshops - Regional Locations:Rush University Medical Center, The Cleveland Clinic
Annual International Anesthesia Research Society Meeting
Annual HSS Regional Anesthesia Symposium – "Controversies and Fundamentals in Regional Anesthesia"
Annual NYSORA Meetings in Asia, Europe and America
Annual International Anesthesia Research Society Meeting
Ultrasound for Regional Anesthesia (ISURA) 2010, Toronto, Canada
Ultrasound Guided Regional Anesthesia Preceptorship, Duke University Medical Center, Durham, NC
UltraSound Guided Regional Anesthesia and Vascular Access, Northwest Anesthesia Seminars, Various Locations
Carolina Refresher Lectures: Care of the Surgical Patient 2010, Kiawah, SC
Dannemiller Anesthesiology Review Course 2010, Chicago, IL
"In Celebration of Patient Safety" Florida Society of Anesthesiologists (FSA) 2010 Annual Meeting, Palm Beach, FL
Ninth Biannual Hands on Ultrasound Guided Regional Anesthesia Workshop, Houston, TX
Hawaii Anesthesiology Update 2010, Maui, HI
Texas Society of Anesthesiologists 2010 Annual Meeting, San Antonio, TX
First International Congress of Regional Anesthesia and Pain Interventions
Fourth Annual Regional Anesthesia in Children Conference, Seattle, WA
Anaesthesia in the Office-Based Setting, Boston, MA
Anesthesia Camp Laguna Beach, Laguna Beach, CA
Ontario Anesthesia Meeting, Toronto, Canada
Regional Anesthesia Study Center of Iowa (RASCI) Workshop, Iowa City, IA
Introductory Ultrasound Workshop, Toronto, Canada
Advances in Physiology and Pharmacology in Anesthesia and Critical Care, White Sulphur Springs, WV
Illinois Society of Anesthesiologists Midwest Anesthesiology Conference (MAC), Chicago, IL
21st Annual University of California-Davis Anesthesiology Update, Monterey, CA
Pediatric Anesthesia Conference: New and Challenging Cases, Austin, TX
Survey of Current Issues in Surgical Anesthesia, Naples, FL
Advanced Ultrasound Workshop, Toronto, Canada
New York State Society of Anesthesiologists' 64th PostGraduate Assembly in Anesthesiology (PGA), New York, NY

Multiple-Choice Questions

1. This physician helped teach the founder of the modern specialty of anesthesiology and became the first president of the original American Society of Regional Anesthesia in 1923:
   (a) Emery Rovenstine
   (b) Gaston Labat
   (c) Victor Pauchet
   (d) Carl Koller

2. Between the year 1980 and 2000, the reported use of regional anesthetics by training programs increased by approximately what percent?
   (a) 5%
   (b) 10%
   (c) 15%
   (d) 20%

3. Considering the use of regional anesthesia (RA), wide discrepancies existed between training programs in the 1980s. Approximately what range of RA case percentages were observed between low RA volume and high RA volume programs during this time?
   (a) <5–55%
   (b) 10–60%
   (c) 15–45%
   (d) 20–45%

4. By the year 2000, the overall use of regional anesthesia techniques by residents in training increased to approximately what percent of total case volume?
   (a) 25%
   (b) 30%
   (c) 35%
   (d) 40%

5. As of July 1, 2008, the ACGME Program Requirements for Graduate Medical Education in Anesthesiology state the following minimum number of epidural, spinal, and peripheral nerve blocks to be performed by each resident:
   (a) 40
   (b) 50
   (c) 60
   (d) 80

6. In the year 1999, approximately what percent of graduating anesthesia residents reported a lack of adequate experience with many common peripheral nerve blocks?
   (a) 10%
   (b) 25%
   (c) 35%
   (d) 50%

7. In early investigations of trainee "learning curves" in regional anesthesia, approximately what range of experience level was required to achieve a 90% success rate with spinal anesthesia?
   (a) 45–70 cases
   (b) 40–55 cases
   (c) 30–45 cases
   (d) 50–60 cases

8. After 60 ultrasound-guided nerve blocks performed by trainees, what is the approximate average number of errors committed per procedure?
   (a) 1
   (b) 3
   (c) 5
   (d) 7

9. Designated regional anesthesia faculty are observed to select a regional anesthetic technique for approximately what percentage of cases?
   (a) 25%
   (b) 50%
   (c) 65%
   (d) 30%

10. What has been observed to be the primary reason for not performing a regional anesthetic in clinical settings amenable to such a technique?
    (a) Surgeon preference
    (b) Patient refusal
    (c) Anesthesiology-related factors
    (d) Medical contraindications

11. Significant improvement in success rates of *spinal* anesthesia are observed after approximately what level of experience is achieved?
    (a) 10 cases
    (b) 15 cases
    (c) 20 cases
    (d) 25 cases

12. Significant improvement in success rates of *epidural* anesthesia are observed after approximately what level of experience is achieved?
    (a) 10 cases
    (b) 15 cases
    (c) 20 cases
    (d) 25 cases

13. After the experience of 90 cases is achieved, what is the approximate observed success rate of epidural anesthesia?
    (a) 50%
    (b) 70%
    (c) 80%
    (d) 94%

14. After the experience of 20 cases is achieved, what is the approximate observed success rate of axillary approach brachial plexus anesthesia?
    (a) 50%
    (b) 60%
    (c) 70%
    (d) 80%

15. The Accreditation Council for Graduate Medical Education (ACGME) did not formally recognize a minimum number of regional blocks as a requirement of training until:
    (a) 1980
    (b) 1996
    (c) 1976
    (d) 1970

Answers:

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

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