CHAPTER 12

REGIONAL ANESTHESIA

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INTRODUCTION

Regional anesthesia has its greatest use in the surgical repair of extremity wounds, and during wartime, extremity wounds occur more frequently (see Figure 1-6, Chapter 1, Combat Trauma Overview) than any others. A regional anesthetic procedure can be done for (*a*) almost any isolated wound that does not cross the midline of the body, and (*b*) multiple wounds of a single extremity or both legs. A craniotomy can be done under local infiltration, although penetrating head wounds are rarely managed without general anesthesia. Intrathoracic and intraabdominal pain may be relieved quite well by regional block, and this technique should be a major part of preoperative and postoperative management. Surgery in these areas is best accomplished under general anesthesia owing to the need, in many cases, for control of the airway and ventilation. Not more than 10%[1] of all combat casualties will arrive in a state of shock at a medical treatment facility with surgical capabilities, thereby assuming hypovolemia and a significant risk for major conduction nerve block anesthesia.

Nerve blocks may be useful to provide preoperative analgesia, intraoperative anesthesia as the sole anesthetic or as a supplement to general anesthesia, or postoperative analgesia. Some nerve blocks are suited for all three of these uses, while others provide good analgesia but may not be acceptable for intraoperative anesthesia.

The physiological benefits of regional anesthesia are numerous (Exhibit 12-1), but *the most important advantage of regional anesthesia is that general anesthesia is avoided*, with its associated loss of airway reflexes, cardiac and respiratory depression, need for endotracheal intubation and mechanical ventilation, loss of patient cooperation, and residual anesthetic effects. Regional anesthesia is not without risk; however, nerve blocks are usually less invasive and affect fewer vital body systems than general anesthesia (Table 12-1).

EXHIBIT 12-1

PHYSIOLOGICAL BENEFITS OF REGIONAL ANESTHESIA

Analgesia

Clear sensorium

Deafferentation

Improved respiratory mechanics

Unimpaired ventilatory control

Hemodynamic stability

Improved regional blood flow

Blunted neurohumoral response to injury

Reduced metabolic effects of injury

Reduced urinary retention

Improved bowel function

Improved immune function

Reduced muscle spasm

BENEFITS OF NEURAL BLOCKADE

Any new therapy or new indication for an older technique must be evaluated in terms of the potential benefits to be gained versus the possible risks. The risks of these procedures include immediate and long-term complications arising from (*a*) needle and catheter placement, (*b*) toxicity from the local anesthetic agent itself, and (*c*) side effects of neural blockade (eg, hypotension). The risks associated with individual blocks are discussed in the sections describing each technique and the section on local anesthetic pharmacology.

The quality of analgesia provided by neural blockade is superior to that provided by other forms of pain relief, because local anesthetics may completely block the transmission of afferent and efferent nerve impulses.[2] Other forms of analgesia only modify the perception of pain by various mechanisms in the central nervous system (CNS) and the periphery. For example, salicylates and other nonsteroidal antiinflammatory drugs (NSAIDs) decrease afferent nerve activity after a noxious stimulus by desensitizing peripheral pain receptors (ie, nociceptors) and decreasing local inflammation.[3] Inflammatory mediators cause increased blood flow and capillary permeability (ie, the wheal and flare reaction) around the point of injury, which leads to edema and an increased perception of pain (ie, hyperalgesia).[4] Afferent impulses are subject to

TABLE 12-1

REGIONAL VERSUS GENERAL ANESTHESIA

Clinical Criteria	Regional	General
Sensorium	Lucid	Unconscious
Patient Positioning	Self-assist	Dead weight
Airway Reflexes	Intact	Lost
Respiratory Drive	Intact	Impaired
Circulatory Effects	Sympathectomy	Cardiac depression
Medications Administered	Few	Many
Postoperative Analgesia	Yes	No

modulation at the level of the spinal cord prior to transmission to higher centers. One site of modulation by several systems is the dorsal horn of the spinal cord. Endogenous and exogenous opioids, α-adrenergic agonists, serotonin, substance P, and descending inhibitory nerves have been shown[5] to affect neural activity in the dorsal horn. Inhibitory systems may be stimulated by administering epidural or intrathecal opioids, systemic opioids (increases descending inhibitory impulses), or α-adrenergic agents (clonidine).[5] (Also see Chapter 13, Perioperative Pain Management, and Figure 13-1.) None of these treatments are as effective as local anesthetics applied to peripheral nerves or injected into the epidural or intrathecal space because nerve impulses are completely blocked by local anesthetics and may provide surgical anesthesia. Studies[2,6,7] that compare nerve blocks using local anesthetics to systemic, epidural, or intrathecal opioids uniformly reveal that the analgesia provided by neural blockade is superior to that of the other forms.

Owing to their close proximity, somatic neural blockade with local anesthetics will often also block sympathetic afferent and efferent nerves. Selective sympathetic nerve block may also be accomplished, usually at a paravertebral location, in which a ganglion of the sympathetic chain is blocked. A transient condition equivalent in effect to a sympathectomy by either mechanism has several potentially beneficial effects. Decreased sympathetic outflow causes vasodilation and increased regional blood flow to the area innervated, which may decrease ischemic pain, promote healing, improve plastic surgical graft survival, and retard vascular graft thrombosis.[8,9] Blockade of sympathetic afferent fibers inhibits segmental spinal reflexes that may increase pain perception by local secretion of norepinephrine, substance P, and bradykinin, which increases nociceptor sensitivity. The vasodilation caused by sympathectomy may also decrease systemic blood pressure if an adequate intravascular volume is not maintained. A mild decrease in blood pressure may be desirable because myocardial work is reduced and wide variations in blood pressure, especially in response to manipulation or movement of the blocked area, may be prevented. Additionally, decreased intraoperative bleeding and perioperative blood replacement are reported benefits of neural sympathectomy, probably due to the combination of stable hemodynamics and dilation of venous capacitance vessels.[10] Perioperative thromboembolism is also reduced, not only for the above reasons but also because patients with effective analgesia tend to ambulate earlier.[11]

Modification of the neurohumoral response to surgery and trauma is the subject of much attention and effort.[12–14] The *stress response* is a protective reflex in which acute injury or stress triggers a tremendous sympathetic discharge, which then results in elevated plasma catecholamines, cortisol, glucose, antidiuretic hormone, and acute-phase proteins. These changes produce tachycardia, vasoconstriction, hypertension, increased oxygen consumption, negative nitrogen balance, and salt and water retention. In 1914, George W. Crile and William E. Lower coined the term “anoci-association” to describe the physiological changes associated with injury, and correctly predicted that the changes may be detrimental and retard the healing process.[15] Most efforts to attenuate the surgical stress response have been directed toward treating the consequences of the efferent sympathetic activity (eg, tachycardia, hypertension, increased myocardial oxygen consumption, and myocardial ischemia and infarction).[16–18] This practice may be expected to contribute to improved outcome and therefore has changed the way many anesthesiologists practice. Neural blockade, however, may go one step further by blocking somatic and sympathetic afferent activity, which serves to prevent the neurohumoral response to injury rather than to treat it after the fact. Outcome data are incomplete, but evidence[14] indicates that morbidity and mortality are reduced in high-risk surgical patients.

Neural blockade improves decreased respiratory function after trauma or operation.[19] Both lung volumes (ie, functional residual capacity [FRC] and forced expiratory volume in 1 second [FEV_1]) increase, in some cases to near baseline levels.[20] This improvement is greater than that achieved with intramuscular or epidural opioid analgesia, even if complete pain relief is obtained.[21] The mechanism of improvement is probably related not only to inhibition of segmental spinal reflexes that cause skeletal muscle spasm or splinting but also to superior pain relief. Improved respiratory mechanics support normal blood gases, pulmonary toilet, and decreased incidence of atelectasis and infection.

Nerve blocks using higher concentrations of local anesthetics (ie, lidocaine 1.5%–2.0% or bupivacaine 0.5%–0.75%) may produce motor neuron blockade and skeletal muscle relaxation. Dilute solutions of local anesthetics may block only sympathetic and sensory nerve fibers, and may spare motor nerves because of their larger size and greater myelinization. Skeletal muscle relaxation inhibits patient movement and ambulation, which may be distressing to the uninformed patient, but is desirable in certain conditions such as muscle spasm. The relaxation of muscle spasm by neural blockade also improves blood flow through the muscle tissue, which may permit metabolic products associated with muscle ischemia to wash out. This may break the pain–spasm–ischemic pain–continued spasm cycle and prevent sensitization of sensory receptors. Immobility of limbs for a short while after surgery may also prevent disruption of repair caused by excessive patient movement.

Properly administered neural blockade promotes a stable hemodynamic profile. Blockade of somatic and sympathetic afferent fibers attenuate increases in heart rate, blood pressure, and myocardial work in response to pain.[22–24] Nerve blocks may also prevent elevation of plasma epinephrine, norepinephrine, antidiuretic hormone, cortisol, and other chemical mediators, which minimizes secondary changes in hemodynamic parameters.[12] Circulatory changes in response to stimulation such as patient movement or procedures (eg, changing surgical dressings) may also be minimized. Indirectly, nerve blocks promote hemodynamic stability by enabling patients to be extubated soon after surgery and thus avoid the circulatory changes associated with prolonged intubation and mechanical ventilation. Although sympathetic blockade may cause systemic arterial hypotension, this does not usually occur unless a major conduction block (eg, spinal or epidural anesthesia) is used and extends to the midthoracic or higher level of the spine. Systemic arterial hypotension may be minimized by adequate intravascular volume loading prior to the block.

Neural blockade may also obviate the need for other analgesics, sedatives, and anesthetics. Alternative methods of analgesia may provide not only suboptimal analgesia but also incur the risks of the alternative therapy. Systemic opioids, for example, exhibit tremendous variation in interpatient analgesic response, especially when given intramuscularly on an intermittent or as-needed dosage schedule.[25] Systemic side effects of opioids include sedation, depression of ventilatory response to carbon dioxide, nausea, pruritus, and constipation.[26] Continuous or on-demand administration of opioids by mechanical patient-controlled analgesia pumps may minimize side effects.[26,27] The interpatient variability of opioids administered by the epidural or intrathecal route is also unpredictable.[28] Because a patient's response to a prescribed dose of opioid cannot be predicted, opioids should be administered with caution, regardless of route, and side effects treated promptly. The use of continued intubation and mechanical ventilation for analgesia after surgery risks complications caused by prolonged airway instrumentation and positive pressure ventilation.

LOCAL ANESTHETICS

Chemical Structures and Physicochemical Properties

Local anesthetics are chemical compounds that cause reversible blockade of nerve impulses. Commonly used local anesthetics are tertiary or secondary amines bound to an aromatic ring by either an amide or an ester chemical linkage. This distinction divides these drugs into two groups: aminoamides and aminoesters (Figure 12-1). These compounds exhibit both lipophilic and hydrophilic properties. The aromatic ring structure is the lipophilic portion

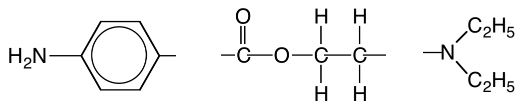
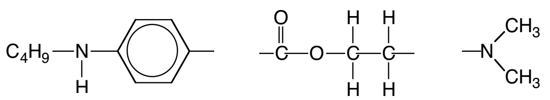
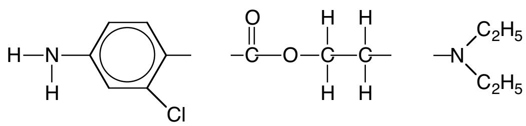
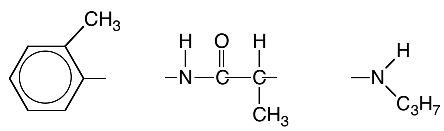
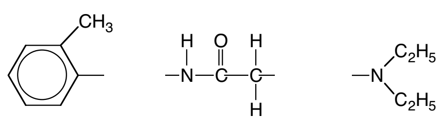
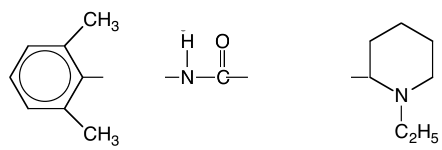
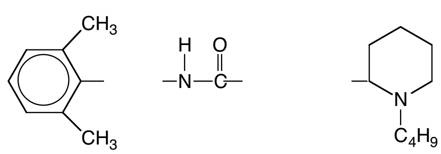
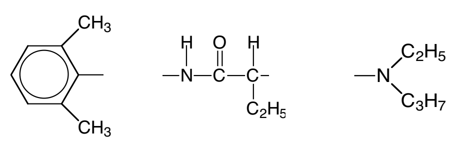
Agent	Aromatic Lipophilic	Intermediate Chain	Amine Hydrophilic	Molecular Weight (base)	pKa (25°C)	Partition Coefficient	% Protein Binding
Esters							
Procaine				236	8.9	0.02	5.8
Tetracaine				264	8.6	4.1	75.6
Chloroprocaine				271	8.7	0.14	—
Amides							
Prilocaine				220	7.7	0.9	~55
Lidocaine				234	7.7	2.9	64.3
Mepivacaine				246	7.6	0.8	77.5
Bupivacaine				288	8.1	27.5	95.6
Etidocaine				276	7.7	141	94

Fig. 12-1. Physicochemical properties of local anesthetics. Adapted with permission from Scott DB. T*echniques of Regional Anaesthesia*. Norwalk, Conn: Appleton & Lange; 1989: 16.

of the molecule. The amine structure is the hydrophilic portion. Ionization of the amine portion to a positively charged species enhances the hydrophilic properties of the molecule.[29]

Local anesthetics are weak bases with pKa values between 7.5 and 9.0. Their physicochemical properties largely determine their clinical anesthetic characteristics. Inherent potency of all anesthetic compounds, including local anesthetics, correlates well with each compound's lipid solubility. Chloroprocaine, for example, is poorly soluble in lipid and has a relatively low anesthetic potency (concentrations of 2%–3% are required for clinical use). Bupivacaine, on the other hand, is lipid soluble and has greater anesthetic potency (more dilute solutions, 0.25%–0.5%, are clinically effective). Onset of action correlates roughly with a local anesthetic's pKa. The pKa of any compound is the pH at which equal amounts of nonionized and ionized species exist in equilibrium. The amount of each form at any given time depends on the local pH. This is illustrated by the formula

$$pKa = pH + \log (Bh^{+}/B)$$

Generally, the higher the pKa of these compounds, the greater the ionization at a physiological pH of 7.4, and the slower the onset of neural blockade.[30] Duration of anesthetic action is dependent on the local anesthetic's affinity for protein. Anesthetics that are highly protein bound (eg, bupivacaine) in general have a longer duration of action than those that are not (eg, procaine).

Physiology of Neural Blockade

Impulses are propagated along nerve cell axons by progressive depolarization of the cell membrane. Depolarization occurs when sodium rapidly enters the cell through sodium channels. Local anesthetics are thought to physically block, or to induce conformational changes within, these sodium channels by reversibly binding to receptors on the intracellular side of the membrane. While the sodium channel is inactive, nerve impulse conduction cannot occur.[31]

The nonionized lipophilic form of local anesthetic most easily crosses the myelin sheath and nerve cell membrane, but it is the ionized hydrophilic form that attaches to the receptor in the cell's sodium channel to block nerve impulse conduction.[32] This is why a local anesthetic with a high pKa (the ionized form is predominant at pH 7.4 and crosses the nerve membranes poorly) generally has a long onset time.

Local anesthetic duration of action correlates with the degree of protein binding because the local anesthetic receptor is thought to be a protein in or near the sodium channel. Anesthetics with high affinity for protein will dissociate from the receptor slowly and therefore the effect will be prolonged. Once dissociated from the receptor, the drug is taken up by the bloodstream and metabolized in the plasma (ester hydrolysis) or the liver (ester hydrolysis and amide microsomal degradation).[33]

Clinical Use

Onset of local anesthetic activity in vivo is dependent on several factors in addition to the pKa of the drug and the tissue pH. The number of molecules available for participation in neural blockade is important; therefore, higher concentrations and larger doses tend to speed onset. The addition of vasoconstrictors to the local anesthetic solution decreases the rate of anesthetic uptake by the bloodstream and, in effect, increases the local concentration, which shortens onset time.[33] Systemic absorption of epinephrine or phenylephrine used for this purpose may produce undesirable hemodynamic changes in the compromised patient. The addition of bicarbonate to the drug solution increases local tissue pH, which increases the amount of easily diffusible nonionized drug present and speeds onset. Carbonation of local anesthetic solutions also speeds the onset of action because carbon dioxide rapidly crosses the cell membrane and lowers intracellular pH. This acidosis favors ionization of intracellular local anesthetic molecules, which increases the activity of the drug by enhancing receptor binding.

Local anesthetic activity and duration of action are also enhanced by adding vasoconstrictors to the solution.[29] The anatomical location of the anesthetic injection affects the onset and duration of the local anesthetic action because the vascularity of the tissue and the regional blood flow determine the rate of local anesthetic uptake by the blood.[31] The local anesthetic concentrations in plasma after injection of a standard dose in different anatomic locations is shown in Figure 12-2. The highest plasma concentration occurs after injection into the intercostal space.

Tachyphylaxis (ie, the development of a decreased effect from a given dose of drug after repeated administration) may be caused by a gradual exhaustion of local tissue-buffering capacity, which is, in turn, caused by repeated injections of the relatively acidic local anesthetic salt solutions. Continuous infusion of anesthetic solution (rather than intermittent injection) or using a different local anesthetic may decrease tachyphylaxis.

Properties of Individual Drugs

The clinical uses and appropriate doses of commonly used local anesthetics are listed in Table 12-2. Although local anesthetics share a common mechanism of action, individual drugs have different indications, side effects, and other properties, such as addiction potential. Important properties of individual drugs are noted below.

Bupivacaine has a high affinity for protein (95% of the drug is bound to protein in plasma) and may be relatively more cardiotoxic than other local anesthetics, because dissociation from cardiac sodium channels may be very slow.[29] Bupivacaine may

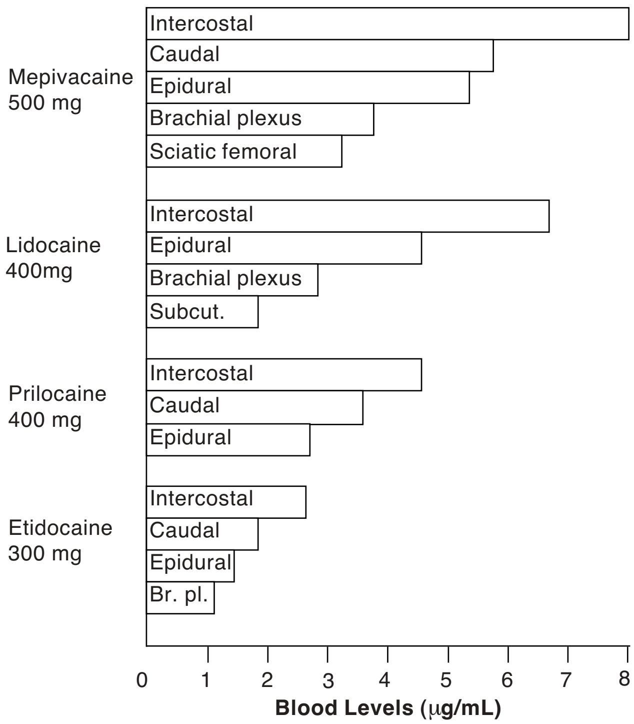


Fig. 12-2. Comparative peak blood levels of several local anesthetic agents. Reprinted with permission from Covino BG, Vassallo HG. *Local Anesthetics: Mechanism of Action and Clinical Use*. New York, NY: Grune and Stratton; 1976: 97. In: Carpenter RL, Mackey DC. Local anesthetics. In: Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*. Philadelphia, Pa: JP Lippincott; 1989: 384.

produce sensory blockade without motor blockade (ie, selective sensory block) at low concentrations.

Chloroprocaine is rapidly metabolized in plasma by pseudocholinesterases and therefore has a short duration of action and low toxicity potential. Patients with abnormal or decreased plasma cholinesterase may have a prolonged duration of action. Use of chloroprocaine may prolong the effect of other drugs metabolized by cholinesterases (eg, succinylcholine).

Cocaine is used exclusively for topical anesthesia of the nose and throat. It causes vasoconstriction, CNS stimulation, and is highly addictive.[30] Addition of a vasoconstrictor is both unnecessary and undesirable.

Etidocaine and lidocaine are structurally similar and have similar onsets of action. Etidocaine's duration of action is longer than lidocaine's and the degree of motor blockade is greater. Lidocaine has the largest number of clinical uses and may be used for topical, infiltration, plexus, epidural, and spinal anesthesia; and intravenous treatment of cardiac dysrhythmias.

Mepivacaine is not effective when used topically. It is also not desirable for use in obstetric anesthesia because neonatal metabolism of mepivacaine is five times slower than in the adult. Because U.S. military forces are used increasingly in operations other than war (OOTW, eg, peacekeeping missions), anesthetic procedures used with obstetric, neonatal, and pediatric patients are assuming greater relevance to military medicine.

Prilocaine may produce methemoglobinemia due to the accumulation of a metabolite, *o*-toluidine, which oxidizes hemoglobin to methemoglobin. This may occur after high doses of prilocaine, usually greater than 600 mg, have been administered. If methemoglobinemia occurs, it can be treated by administering reducing agents (eg, methylene blue 1 mg/kg).[29]

Adverse Reactions to Local Anesthetic

True allergy to local anesthetic drugs is extremely rare. When it occurs, it is usually due to hypersensitivity to a preservative (eg, *p*-aminobenzoic acid) or a metabolite of local anesthetics. Most reactions assumed to be allergic are actually due to

- systemic effects of toxic plasma concentrations of local anesthetic as a result of relative overdosage, or to intravascular injection of an otherwise appropriate dose;
- systemic epinephrine effects;
- a vasovagal reaction; or
- total spinal blockade (ie, high spinal causing cardiopulmonary collapse, which may mimic anaphylactic shock).

The cause of the adverse reaction must be determined quickly before appropriate treatment can be instituted.

Toxic plasma concentrations are manifest by CNS signs: first tinnitus, circumoral numbness, metallic taste, and then with increasing plasma concentration, tremulousness that may progress to seizure activity. A very small dose of local anesthetic may cause CNS signs if injected into an artery that perfuses the brain (eg, the vertebral artery). These CNS signs may be followed by cardiovascular effects, which require higher plasma concentrations. Direct depression of myocardial inotropy and chronotropy occur, in addition to systemic arterial vasodilation. Cardiac dysrhythmias and cardiovascular collapse may occur secondary to acidosis and hypoxia caused by seizure activity when local anesthetic plasma concentration is below cardiotoxic

TABLE 12-2

COMMONLY USED LOCAL ANESTHETICS: CLINICAL USES AND APPROPRIATE DOSES

Agent	Concentration (%)	Clinical Use	Onset	Usual Duration (h)	Recommended Maximum Single Dose (mg)	Comments	pH of Plain Solutions*
Amides							
Lidocaine	0.5–1.0	Infiltration	Fast	1.0–2.0	300	Most versatile agent.	6.5
	0.25–0.5	IV Regional			500 + Epi		
	1.0–1.5	Periph nerve blocks	Fast	1.0–3.0	500 + Epi		
	1.5–2.0	Epidural	Fast	1.0–2.0	500 + Epi		
	4	Topical	Mod	0.5–1.0	500 + Epi		
	5	Spinal	Fast	0.5–1.5	100		
Prilocaine	0.5–1.0	Infiltration	Fast	1.0–2.0	600	Least toxic amide agent.	4.5
	0.25–0.5	IV Regional			600	Methemoglobinemia usually occurs above 600 mg.	
	1.5–2.0	Periph nerve blocks	Fast	1.5–3.0	600		
	2.0–3.0	Epidural	Fast	1.0–3.0			
Mepivacaine	0.5–1.0	Infiltration	Fast	1.5–3.0	400 500 + Epi	Plain mepivacaine solution has longer duration than plain lidocaine solution. Useful when epinephrine is contra-indicated.	4.5
	1.0–1.5	Periph nerve blocks	Fast	2.0–3.0			
	1.5–2.0	Epidural	Fast	1.5–3.0			
	4.0	Spinal	Fast	1.0–1.5	100		
Bupivacaine	0.25	Infiltration	Fast	2.0–4.0	175 225 + Epi	Lower concentrations provide differential sensory/motor block. Ventricular arrhythmias and sudden cardiovascular collapse reported following rapid IV injection.	4.5
	0.25–0.5	Periph nerve blocks	Slow	4.0–12.0	225 + Epi		
	0.25–0.5	Obstetric epidural	Mod	2.0–4.0	225 + Epi		
	0.5–0.75	Surgical epidural	Mod	2.0–5.0	225 + Epi		
	0.5–0.75	Spinal	Fast	2.0–4.0	20		
Etidocaine	0.5	Infiltration	Fast	2.0–4.0	300 400 + Epi	Profound motor block useful for surgical but not for obstetric analgesia.	4.5
	0.5–1.0	Peripheral	Fast	3.0–12.0	400 + Epi		
	1.0–1.5	Surgical epidural	Fast	2.0–4.0	400 + Epi		
Dibucaine	0.25–0.5	Spinal (hyperbaric)	Fast	2.0–4.0	10	Recommended only for spinal and topical use.	——
	0.00067	Spinal (hypobaric)	Fast	2.0–4.0	10		
	1.0	Topical	Slow	0.5–1.0	50		
Esters							
Procaine	1.0	Infiltration	Fast	0.5–1.0	1,000	Used mainly for infiltration and differential spinal blocks. Allergic potential after repeated use.	5–6.5
	1.0–2.0	Periph nerve blocks	Slow	0.5–1.0	1,000		
	2.0	Epidural	Slow	0.5–1.0	1,000		
	10.0	Spinal	Mod	0.5–1.0	200		
Chloroprocaine	1.0	Infiltration	Fast	0.5–1.0	800 1,000 + Epi	Lowest systemic toxicity of all local anesthetics.	2.7–4
	2.0	Periph nerve block	Fast	0.5–1.0	1,000 + Epi	Intrathecal injection may be associated with sensory/motor deficits.	——
	2.0–3.0	Epidural	Fast	0.5–1.0	1,000 + Epi		
Tetracaine	0.5	Spinal	Fast	2.0–4.0	20	Use is limited primarily to spinal and topical anesthesia.	4.5–6.5
	2.0	Topical	Slow	0.5–1.0	20		
Cocaine	4.0–10.0	Topical	Slow	0.5–1.0	150	Topical use only. Addictive, causes vasoconstriction. CNS toxicity initially features marked excitation (fight or flight response). May cause cardiac arrhythmias owing to sympathetic stimulation.	——
Benzocaine	≤ 20	Topical	Slow	0.5–1.0	200	Useful only for topical anesthesia.	——

*Epinephrine-containing solutions have a pH 1 to 1.5 units lower than plain solutions.

Epi: epinephrine; IV: intravenous; Mod: moderate; Periph: peripheral; CNS: central nervous system

Adapted with permission from Covino BG. Clinical pharmacology of local anesthetic agents. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1988: 112–113.

levels. In general, treatment for toxic local anesthetic reactions is supportive until the drug is redistributed or metabolized, and the tissue levels decrease. The injection of local anesthetic should cease immediately, the airway be supported, and supplemental oxygen be administered. Tracheal intubation and pharmacological treatment of seizures is rarely required.

Intravascular absorption of epinephrine may cause tachycardia, hypertension, hypotension (a β-adrenergic effect), anxiety, restlessness, and a feeling of impending doom. These effects are often mistaken for local anesthetic toxicity. Treatment is supportive.

A vasovagal reaction may occur during needle placement or with injection of local anesthetic. Bradycardia, hypotension, sweating, and pallor precede fainting. Administration of atropine intravenously or intramuscularly and supportive measures are indicated.

True allergies to local anesthetics have been reported in a very small percentage of patients. These reactions are almost always due to hypersensitivity to the *p*-aminobenzoic acid metabolic derivatives of ester-type anesthetic drugs. Allergic reactions to amide-type drugs are extremely rare. Hypersensitivity to preservatives (eg, methylparaben) may also occur. Allergic reaction symptoms include urticaria and cutaneous flushing, wheezing, tachycardia, arterial hypotension, and cardiovascular collapse. Treatment consists of maintenance of a patent airway, ventilation with 100% oxygen, and administration of epinephrine that is titrated to maintain a normal blood pressure. Steroids and histamine blocking drugs may also be given.

REGIONAL ANESTHESIA FOR THORACIC AND ABDOMINAL SURGERY

Regional anesthesia for thoracic and abdominal surgery—except for superficial or lower-abdominal procedures—is usually used in combination with general anesthesia. Why should patients receive regional anesthesia if they are going to have a general anesthetic anyway? There are numerous advantages and disadvantages, and numerous and varied outcome studies have been performed. The advantages of combined general–regional anesthesia versus general anesthesia alone include the following:

- The general anesthetic requirement may be reduced, which may result in a reduction in dose-related general anesthetic effects, a faster emergence, earlier extubation, and shorter hospital stay.[14,34]
- The combined technique produces a smoother intraoperative course with more stable hemodynamic parameters and fewer changes of anesthetic depth required.[35,36]
- Noxious afferent input to the spinal cord may be minimized, which may reduce spinal cord sensitization, thereby decreasing or causing the development of permanent spinal cord sensitization, which may give rise to a chronic pain pathway. Thus the neurohumoral response to tissue injury is attenuated.[12]
- Postoperative analgesia may be provided by extending the nerve block technique after discontinuing general anesthesia.[34,35]
- The blockade of sympathetic nerve impulses to and from the spinal cord may be used to produce deliberate and controlled hypotension (to reduce bleeding) and may also provide analgesia for sympathetically mediated pain.[37]

The disadvantages of combined general–regional anesthesia versus general anesthesia alone include the following:

- The additional invasive procedure adds to the risk of anesthetic complications.[38]
- The nerve block procedure requires additional anesthetic preparation time. This is especially disadvantageous if the procedures are done in the operating room.
- The combination of major conduction nerve blocks and general anesthetic–induced myocardial depression may result in severe hypotension.[14,34]
- Some evidence[34] indicates that the combination of general and regional anesthesia is associated with a greater degree of intraoperative hypothermia than either technique used alone.
- There is a theoretical increased risk of intraoperative awareness if low concentrations of inhaled anesthetic agents are used.

Many outcome studies identify positive physiological effects of regional versus general anesthesia, including reduced intraoperative blood loss, improved postoperative respiratory function, maintenance of bowel motility, and more normal metabolic, immune, and hemostatic functions.[14,34,39,40] However, very few studies investigate whether these physiological changes (*a*) are clinically important or (*b*) affect patient outcome. Moreover, studies comparing the outcome of patients who undergo combination general–regional anesthesia versus general anesthesia only are fewer still.[14,36] Comparative studies have been hindered by a relative underutilization of local anesthetics in favor of opioids, inappropriate epidural catheter placement (eg, lumbar placement for thoracic or upper-abdominal surgery), and administration of lipophilic opioids at a spinal level (lumbar) distant from the site of action (thoracic).

Applied Anatomy for Regional Anesthesia of the Chest and Abdomen

Somatic Nerves

The chest and abdominal wall are innervated by branches of thoracic spinal nerves. Surgical procedures of the chest and abdomen involve almost exclusively areas innervated by nine pairs of spinal nerves, T-4 through T-12. T-1 is part of the brachial plexus, T-2 and T-3 innervate the chest wall above the level of the nipples, where thoracic surgery is rarely performed, and the lumbar spinal nerves involve only the peripheral (inguinal) lower abdomen. Each spinal nerve has a small dorsal ramus that innervates paraspinal muscles and skin, and a large ventral ramus that becomes the intercostal nerve and innervates skin, muscle, and bone in the distribution of the corresponding rib.[41]

Sympathetic and Parasympathetic Nerves

Sympathetic nerves to and from the chest and abdomen synapse in or pass through the paired sympathetic chain ganglia located anterior and lateral to the vertebral bodies from T-1 to L-5. Sympathetic outflow from the spinal cord reaches the sympathetic chain via the white rami communicantes; sympathetic afferents from the chain travel via the gray rami communicantes to the spinal cord. The greater, lesser, and least splanchnic nerves are formed by fibers from the sympathetic chain and synapse in the mesenteric ganglia.[41]

Afferent and efferent parasympathetic innervation of the chest and abdomen is largely via the paired vagal nerves.[41]

Applied Physiology for Regional Anesthesia of the Chest and Abdomen

Respiratory Effects

Control of ventilation is not inhibited by regional anesthesia.[42] Some evidence[43] suggests that systemic local anesthetics actually increase the ventilatory response to carbon dioxide.

The mechanics of ventilation are often disturbed after thoracic and abdominal surgery owing to decreased chest-wall compliance and diaphragm dysfunction.[34] Regional anesthesia does not affect motor nerves to the diaphragm (eg, the phrenic nerve, the most important contribution to which is from C-4), may rarely weaken some intercostal muscles, and has been shown to reduce diaphragm dysfunction after upper abdominal surgery.[39]

Increased airway resistance secondary to reduced sympathetic efferent activity to the lungs is a theoretical concern, although it has not been reported as a problem in patients with pulmonary disease who are undergoing thoracotomy. In fact, thoracic epidural anesthesia has been used to treat status asthmaticus, presumably by blocking sympathetic afferent fibers from the airways.[21]

Circulatory Effects

Cardiac Effects. Bradycardia, junctional rhythm, or heart block may be caused by blocking of sympathetic nerves to the heart, although complete blockade may be necessary for these effects.[15] Coronary artery blood flow, in normal and stenotic vessels, and cardiac function may increase dramatically owing to the sympathetic nerve block associated with thoracic epidural anesthesia.[44,45]

Systemic and Pulmonary Artery Blood Pressure. A decrease in systemic blood pressure is often associated with thoracolumbar sympathetic nerve block, largely due to dilation of venous capacitance vessels and decreased venous return to the heart.[44] Systemic vascular resistance is also reduced and cardiac output may increase if preload is maintained.[44] The degree of hypotension is dependent on the number of spinal segments blocked. Local anesthetics administered through a thoracic epidural catheter may cause less hypotension than a lumbar epidural catheter because some sympathetic outflow may be spared, innervation of the adrenal glands may be intact, and the pumping action of the lower extremity muscles to augment venous return may be functional.

A decrease in pulmonary artery pressure may be associated with blockade of sympathetic nerves to the chest unless pulmonary hypertension is caused by a mechanical resistance (ie, vascular changes or mitral valvular stenosis).[44] In this situation, the increased impedance offered is not affected by sympathetic blockade.

Endocrine, Gastrointestinal, and Urinary Effects

There is currently no better way to inhibit the neurohumoral response to tissue injury than via afferent neural blockade. This benefit may not be completely realized in thoracoabdominal surgery, however, because complete afferent blockade is not commonly attained owing to vagal afferents and humeral mediators such as cytokines.[12]

The sympathetic nerve block that may accompany regional anesthesia promotes gastrointestinal function by creating a predominant parasympathetic nerve influence on the gut, which increases peristalsis and gut motility.[40] This beneficial effect of regional anesthesia is often augmented by a reduced opioid requirement for adequate analgesia, and therefore, fewer opioid-induced side effects. These effects may combine to reduce the incidence of postoperative ileus.[34,35]

Urinary retention after surgery may be caused by residual general anesthetic effect, opioids, neural blockade, and other drugs. Like gut function, the ability to void may be maintained by segmental (thoracic) nerve blockade and the avoidance of opioid analgesics.[34,35]

Locomotion

Patients who have received regional anesthesia for thoracic and abdominal surgery should be able to ambulate earlier than patients who did not receive regional anesthesia. Motor nerves to the lower extremity originate in lumbar and sacral spinal nerves, which may be spared by segmental thoracic spinal nerve block. The superior analgesia provided by regional anesthesia, without the sedation caused by systemic analgesics, also promotes early ambulation.[34,35]

Specific Regional Anesthetic Techniques

The intelligent use of regional anesthesia for thoracic and abdominal surgery requires that the anesthesiologist have a thorough knowledge of (*a*) the anatomy and physiology related to neural blockade, (*b*) the patient's medical and surgical history, and (*c*) the planned surgical procedure. Techniques that may be useful for thoracic and abdominal surgery include thoracic epidural anesthesia, spinal anesthesia, intercostal nerve block, interpleural nerve block, and incisional local anesthetic infiltration. At least one of these techniques is usually indicated, regardless of the patient's condition or the surgical procedure planned. Characteristics of each technique are summarized in Table 12-3. Complications of regional anesthesia are discussed later in this chapter.

TABLE 12-3

REGIONAL ANESTHETIC TECHNIQUES FOR THORACIC AND ABDOMINAL SURGERY

Technique	Somatic Sensory Nerve Block	Sympathetic Nerve Block	Lower Extremity Motor Block
Epidural Anesthesia			
Thoracic	+	+	–
Lumbar	+	+	+
Spinal Anesthesia	+	+	+
Intercostal Nerve Block*			
Single	+	–	–
Continuous	+	–/+	–
Interpleural Nerve Block*	+	–/+	–
Paravertebral Nerve Block*	+	–/+	–
Peripheral Nerve Block	+	–	–

*Unilateral anesthesia: (+) = effect; (–) = no effect

Epidural Anesthesia

Epidural anesthesia is the most versatile regional anesthetic technique available in terms of surgical application, control of extent and duration of nerve block, and use for postoperative analgesia. Epidural anesthesia can produce segmental sympathetic, sensory, and motor block for many intrathoracic or intraabdominal procedures, lower extremity or abdominal surgery, analgesia during labor and delivery, and treatment of medical conditions such as Raynaud's phenomenon or postherpetic neuralgia.

Despite the advantages, thoracic epidural anesthesia should not be attempted by anesthesiologists who are unfamiliar with the technique. As an alternative, lumbar epidural anesthesia may be used for thoracic and abdominal surgery if local anesthetics are used intraoperatively to provide an adequate level of anesthesia but are discontinued postoperatively. Because high volumes of local anesthetic are required, the sympathectomy may be extensive, and motor block of the lower extremities will result. Epidural opioids may be used subsequently to provide postoperative analgesia without motor block, but this adds the potential for opioid side effects.

Owing to the close proximity of the underlying spinal cord, thoracic epidural anesthesia should not be routinely performed in anesthetized patients. As with any regional anesthetic technique, if needle insertion is difficult, multiple attempts should not be pursued because the incidence of complications increases inordinately. Know when to abandon the procedure and use an alternative technique.

Epidural anesthesia is contraindicated in the presence of informed patient refusal, local or severe systemic infection, abnormal coagulation, unstable neurological deficits, uncorrected hypovolemia, and possibly immunosuppression.

Technique. Epidural anesthesia may be performed at any level of the spine. The epidural space is identified by a characteristic loss of resistance to depression of the syringe plunger on entering the space (ie, *loss of resistance* technique) or by the disappearance of a drop of sterile saline from the needle hub on entering the space owing to the subatmospheric pressure of the space (ie, *hanging drop* technique). A *single-shot* technique may be used, in which one dose of anesthetic is injected into the space, or a *continuous* (usually intermittent injection) technique may be used, in which a catheter is threaded into the space through the needle; then the needle is removed. In the lumbar spine, a local anesthetic volume of approximately 1.5 to 2.0 mL per spinal segment is required for anesthesia. Epidural catheters may be kept in place for 48 to 72 hours or longer if the benefits of analgesia outweigh the risk of infection.

If local anesthetics will be used postoperatively, the tip of the epidural catheter should be placed as closely as possible to the midpoint of the range of spinal (thoracic) segments to be blocked. This location allows the use of lower volumes of local anesthetics, minimizes the extent of sympathetic nerve block, and is unlikely to cause lower-extremity motor block. Suggested levels of epidural catheter placement for given surgical procedures are listed in Table 12-4.

Local Anesthetic Drug Dosages. Local anesthetics must come into contact with the target nerves to exert their effect. Epidural dosing of local anesthetics is, therefore, largely based on volume. In the epidural space, local anesthetics spread in all directions from the catheter tip, but preferentially along lines of least resistance. The epidural space is smaller in the thoracic than in the lumbar spine, and the volume of anesthetic must be adjusted accordingly. General rules of thumb for adults are given in Table 12-5. The concentration of local anesthetic affects the speed of onset of block, degree of motor block, and duration of block. Motor-blocking strengths may be used intraoperatively, but the lowest con-

TABLE 12-4

LOCATION OF EPIDURAL CATHETER INSERTION

Site of Surgery	Epidural Catheter	Dose of Local Anesthetic (mL)
Thoracic	Upper thoracic spine (T-4 –T-7)	4–6
Upper abdominal	Mid thoracic spine (T-8–T-9)	6–8
Lower abdominal	Lower thoracic spine (T-10–T-11)	8–10

TABLE 12-5

EPIDURAL LOCAL ANESTHETIC DOSAGE GUIDELINES

Position of Catheter Tip	Volume Required per Spinal Segment (mL)	Volume Required for T-4 Level (mL)
Upper thoracic (T-4–T-7)	1.0	1–6
Mid thoracic (T-8–T-9)	1.5	8–10
Lower thoracic (T-10–T-12)	1.75	12–18
Lumbar (L-1–L-5)	2.0	20–28

centration that provides pain relief should be used postoperatively.

Opioid analgesics are usually administered on a milligram-per-kilogram basis. Lipophilic drugs, such as fentanyl, act via a spinal mechanism if placed in the thoracic epidural space, and via systemic absorption if placed in the lumbar epidural space.[46,47] Hydrophilic drugs, such as morphine or hydromorphone, spread more readily through the epidural space before absorption, act via a spinal mechanism, and are effective in the lumbar or thoracic epidural space.[48,49] Increased respiratory surveillance may be considered if a hydrophilic opioid is used in the upper-thoracic epidural space. Opioid dosage guidelines are given in Table 12-6.

Complications. The complications of epidural anesthesia are similar to those associated with spinal anesthesia (Table 12-7). Possible trauma caused by needle insertion may be greater owing to the larger needle size, and the risk of local anesthetic toxicity is greater owing to the larger doses used. Inadvertent dural puncture carries a high likelihood of post–dural-puncture headache. A "total" spinal (ie, the entire spinal cord has been blocked, at least to some extent) may result from intrathecal injection of a local anesthetic dose appropriate for epidural administration. As with spinal anesthesia, sympathetic nerve block may cause systemic hypotension, especially if the patient is hypovolemic. Bradycardia may occur if high thoracic spinal levels are blocked (ie, if cardioaccelerator fibers are anesthetized). Subarachnoid and epidural abscesses have been reported[41] after epidural needle or catheter insertion.

Spinal Anesthesia

Spinal anesthesia has many advantages compared to epidural anesthesia: much smaller local anesthetic doses are required, systemic vascular absorption and risk of systemic toxicity are negligible, onset of block is much faster, and the resultant nerve block is more complete, including motor blockade. Disadvantages include the inability to produce a segmental band of anesthesia, a more extensive virtual sympathectomy with more pronounced blood pressure changes, and fewer postoperative analgesic options.[50]

TABLE 12-6

EPIDURAL OPIOID DOSAGE GUIDELINES FOR A HEALTHY, 70-KG, ADULT PATIENT

Drug	Initial Dose (mg)	Injectate Volume* (mL)	Infusion Rate (mg/h)
Morphine	3–5	10	0.2–0.3
Hydromorphone	0.5–1.0	10	60–150
Fentanyl	75–150	10	30–100
Sufentanil	25–50	10	5–15

*Prepare opioid dose in preservative-free saline

TABLE 12-7

SPINAL VERSUS EPIDURAL ANESTHESIA

Characteristic	Spinal	Epidural
Onset	Fast	Slow
Local anesthetic dose	Small	Large
Local anesthetic plasma concentration	Nil	Nil
Density of block	Very dense	Less dense (spotty)
Need for supplementation	Less likely	More likely
Indication of needle placement	Reliable*	Less reliable†
Post–dural-puncture headache	Low risk	Possible
Hypotension	More likely	Less likely
Continuous technique	Less common	Very common
Postoperative analgesia	Possible (Duramorph‡)	Common

*By cerebrospinal fluid
†By loss of resistance
‡Morphine; manufactured by Elkins-Sinn, Cherry Hill, NJ

Spinal anesthesia may be used alone for many lower-abdominal procedures or lower-extremity surgery and may be combined with general anesthesia for upper-abdominal surgery. Spinal anesthesia is rarely used for thoracic surgery.

Spinal anesthesia is contraindicated for patients with coagulation abnormalities, infection at the site of needle placement, severe systemic infection, uncorrected hypovolemia, unstable neurological deficits, and possibly immunosuppression.

Technique. The basic technique for lumbar puncture may be found in any standard anesthesiology textbook. The injection of a single dose of local anesthetic through the spinal needle, a single-shot technique, is sufficient for many surgical techniques. The sensory and motor blockade produced by such a technique depends largely on the local anesthetic dosage, baricity, and location and speed of injection. Isobaric solutions (bupivacaine 0.5%, lidocaine 2%, and tetracaine 1%) are gaining popularity because neural blockade is independent of patient position, fewer spinal segments are blocked, and there are smaller reductions in systemic blood pressure. A continuous catheter technique may be used (*a*) to produce a slow onset of neural blockade by fractionating the total anesthetic dose and (*b*) to prolong neural blockade for long surgical procedures. Isobaric local anesthetic solutions are also useful in continuous catheter techniques because there is no tendency for the local anesthetic to accumulate in dependent portions of the subarachnoid space and thus potential neural toxicity (cauda equina syndrome) may be avoided.

The use of spinal catheters has diminished after reports of associated cauda equina syndrome.[51,52] The mechanism is thought to be related to uneven distribution of hyperbaric local anesthetic solutions in the spinal fluid and high (toxic) concentrations near the sacral nerve roots. To minimize the risk of neurological injury, spinal catheters should be inserted no more than 2 cm into the subarachnoid space, recommended maximum local anesthetic dosages should not be exceeded, and use of isobaric solutions should be considered.

Complications. The most common complication associated with spinal anesthesia is hypotension, which may be controlled by adequate fluid prehydration and the use of vasopressors. Nausea and vomiting may be minimized by good blood pressure control and small doses of antiemetics. Post–dural-puncture headache (also called spinal headache) may occur after any dural puncture (eg, for spinal tap, myelogram, spinal anesthesia) and may be minimized by using small-gauge, noncutting needles. Transient and persistent neurological deficits may rarely occur. Spinal hematoma or abscess are extremely rare but possible complications of spinal anesthesia.

Practical Points. Injection of hyperbaric local anesthetic solutions into the L-2–L-3 or L-3–L-4 interspace usually results in a T-4 level block, owing to the slope of the spinal curvature from the lumbar lordosis to thoracic kyphosis. Injection into the L-4–L-5 or L-5–S-1 interspace may result in a somewhat lower spinal level, owing to collection of some of the local anesthetic in the sacral spinal space.[53]

Isobaric local anesthetic solutions (eg, lidocaine 2%, tetracaine 1%, bupivacaine 0.5%) may result in a lower "highest" level, a smaller range of highest levels, and smaller blood pressure changes, compared to hyperbaric solutions.[53] The resultant spinal level of anesthesia is independent of patient position at the time of injection. Relatively large doses may be required to attain a high thoracic level.

Postoperative analgesia may be provided by longer acting opioids (eg, morphine 6–20 h) without causing motor block, but additional respiratory monitoring may be required postoperatively.[54,55] Spinal anesthesia or analgesia should not be used if local anesthetic block or prolonged analgesia (> 24 h) is required postoperatively. A continuous epidural or intercostal catheter technique may be more appropriate in this setting.

Intercostal Nerve Block

Intercostal nerve blocks may provide better postoperative analgesia than conventional opioid therapy. When the chest is open, the intercostal nerves can be injected under direct vision. Intercostal block has no effect on visceral structures, so by itself it is inadequate for surgery.

Intercostal nerve block should not be used in the presence of local or severe systemic infection, coagulation abnormality, severe pulmonary disease, or obesity or other physical condition that may make rib palpation difficult.

Anatomy. There are 12 pairs of thoracic anterior primary rami: the upper 11 comprise the intercostal nerves and the 12th is termed the subcostal nerve.[56] The 3rd through 11th pairs are responsible for the innervation of the muscles of the intercostal spaces and of the anterior abdominal wall, and for the cutaneous supply of the skin of the medial aspect of the upper arm and of the anterior and lateral aspects of the trunk from the level of the angle of Louis to just above the groin. The 1st, 2nd, and 12th pairs differ from the others and must be considered separately when intercostal nerve block is desired:

- The 1st intercostal nerve sends a large contribution, which passes across the front of the neck of the first rub, lateral to the superior intercostal artery, to enter the composition of the brachial plexus.
- The 2nd intercostal nerve differs from the typical intercostal nerves in that its lateral cutaneous branch crosses the axilla to supply the skin over the medial aspect of the upper arm. This branch is called the intercostobrachial nerve.
- The 12th thoracic nerve runs along the lower border of the 12th rib below the subcostal vessels, passes behind the lateral arcuate ligament to run in front of quadratus lumborum behind the kidney and colon.[56]

A typical intercostal nerve has four significant branches. The first branch is the gray ramus communicans, which passes anteriorly to the sympathetic ganglion. The second branch arises as the posterior cutaneous branch. This nerve supplies skin and muscles in the paravertebral region. The third branch is the lateral cutaneous division, which arises just anterior to the midaxillary line. This branch sends subcutaneous fibers posteriorly and anteriorly to supply skin of much of the chest and abdominal wall. The fourth and terminal branch of an intercostal nerve is the anterior cutaneous branch,[41] which innervates the ventral side of the chest and the abdominal wall.

Each nerve is joined to its corresponding thoracic sympathetic ganglion by a white and grey ramus communicans.[56] Distal to the rami communicantes, the nerve trunk divides into the dorsal and the ventral branches. The dorsal branch supplies the skin of the back, the muscles of the back, and the periosteum of the vertebrae through its lateral and medial branches.[57]

The ventral branch follows the rib into the costal sulcus, into the dorsal thoracic region and between the two laminae of the intercostal muscles in the intercostal space, and into the lateral and ventral portions of the thorax. The ventral branches each give off a lateral cutaneous branch, first to fifth in the posterior axillary line and sixth to twelfth in the anterior axillary line.[57]

The lateral cutaneous branches of intercostal nerves 7 through 12 participate in sensory and motor innervation of the anterior abdominal wall (skin, muscles, parietal peritoneum).[57]

At the angle of the rib (6–8 cm from the spinous processes), the nerve comes to lie between the internal intercostal muscle and the intercostalis intimus muscle. The costal groove is broadest and deepest at this point. The nerve is accompanied by an intercostal vein and artery, which lie superior to the nerve in the inferior groove of each rib. The costal groove forms a sharp angle with the rib. At this point, the intercostal groove ceases to exist, the lateral cutaneous branch is given off, and the intercostal nerve lies more inferiorly and moves toward the center of the intercostal space.[41]

Technique. The intercostal nerve block may be performed at several possible sites along the course of the nerve, using the following equipment:

- short, beveled, 23- or 25-gauge needle, and
- 10-mL syringe.

The most common site is in the region of the angle of the ribs just lateral to the sacrospinalis group of muscles (Figure 12-3).[41]

Skin markings can be made at the lateral edge of the sacrospinalis muscle (6–8 cm from the midline). The line may be curved medially and superiorly to avoid the scapulae. Skin at the lower edge of the rib

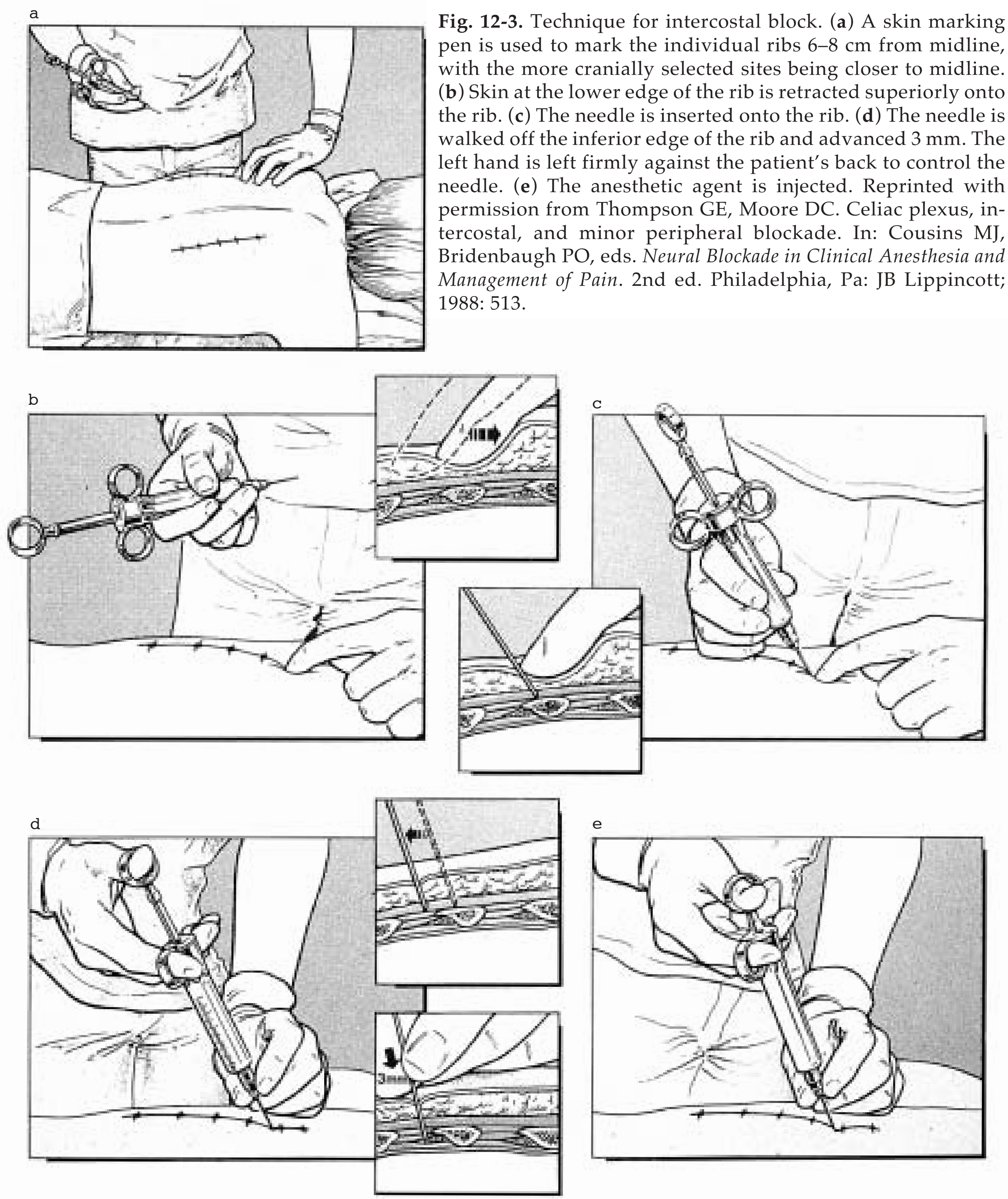


Fig. 12-3. Technique for intercostal block. (**a**) A skin marking pen is used to mark the individual ribs 6–8 cm from midline, with the more cranially selected sites being closer to midline. (**b**) Skin at the lower edge of the rib is retracted superiorly onto the rib. (**c**) The needle is inserted onto the rib. (**d**) The needle is walked off the inferior edge of the rib and advanced 3 mm. The left hand is left firmly against the patient's back to control the needle. (**e**) The anesthetic agent is injected. Reprinted with permission from Thompson GE, Moore DC. Celiac plexus, intercostal, and minor peripheral blockade. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade in Clinical Anesthesia and Management of Pain*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1988: 513.

is retracted superiorly onto the rib. The needle is inserted onto the rib then walked off the inferior edge and advanced 3 mm toward the sulcus of the rib.[41] Aspirate before injecting 3 to 5 mL per segment.[41,57] The patient should hold his breath during the injection.

Local Anesthetic Drug Dosage. Any local anesthetic used for peripheral nerve blocks may be used, such as 0.5% bupivacaine or 1% etidocaine, in the amount of 3 to 5 mL.[57]

Complications. Pneumothorax is the most feared complication.[41] Blood levels of anesthetic agent are higher after this block because of the proximity to vessels, which can lead to toxic concentrations of local anesthetics if the patient receives an overdose of drug. Hemothorax could occur if the intercostal vessels are damaged.

Practical Points. A catheter technique has many advantages compared to multiple single injections: a one-time-only risk of pneumothorax, the ability to provide prolonged continuous analgesia, and blockade of sympathetic nerve fibers.[58,59]

Since large volumes (20–30 mL/h) of local anesthetic solutions may be required, use of a drug with low toxicity potential should be considered. We have used 1% chloroprocaine with success, although tachyphylaxis may develop.

Interpleural Nerve Block

Injection of local anesthetics between the visceral and parietal pleurae provides somatic nerve block by diffusion to the intercostal nerves, and sympathetic nerve block by spread to the paravertebral area.[60,61] Interpleural nerve block may be used for unilateral anesthesia of the upper abdomen. This approach to surgical anesthesia is less useful for thoracic procedures, especially if the pleurae have been disrupted or if thoracostomy drainage tubes are used.[62,63]

The effects of interpleural nerve block are similar to those of continuous intercostal nerve block.[64] Compared with continuous intercostal nerve block, interpleural block is patient-position dependent, has a higher incidence of pneumothorax, requires a higher local anesthetic dose, and results in higher plasma concentration of local anesthetics.[65–67] These characteristics favor use of continuous intercostal nerve block for unilateral procedures of the upper abdomen and flank.

Interpleural nerve block should not be used in the presence of local or severe systemic infection, coagulation abnormality, severe pulmonary disease, or pleural abnormality.

Peripheral Nerve Blocks

Peripheral nerve blocks and incisional infiltration with local anesthetics provide local somatic anesthesia. These anesthetic techniques may be used as the sole anesthesia for extremely limited procedures, to supplement general anesthesia, or to provide several hours of postoperative analgesia.

Infiltration of the surgical wound with a local anesthetic drug is an extremely under-utilized technique. It does not require technical expertise, is safe, and can provide a "bridge" of analgesia between general anesthesia and postoperative analgesia.[68] Some evidence suggests that infiltration of the surgical site prior to incision provides preemptive analgesia (ie, it prevents spinal cord activation and hyperalgesia by blocking afferent nerves from the site of injury).[69]

Peripheral or infiltration blocks are contra-indicated in the presence of local infection or allergy to the local anesthetic.

SPECIFIC NERVE BLOCK TECHNIQUES

Regional anesthesia provides operative anesthesia and postoperative analgesia for selected casualties receiving surgery during wartime. Use of regional anesthesia in this casualty population may prevent some sequelae of general anesthesia (eg, nausea and vomiting) and should decrease labor requirements for patient care in the post operative recovery area. It is conceivable that inducing sympathetic blockade in selected cases (eg, after extremity injury on the battlefield) may decrease the incidence of phantom limb pain or reflex sympathetic dystrophy. This would be especially likely if the ultralong duration, microencapsulated local anesthetics prove useful in clinical practice, so that a one-time blockade procedure could induce a sympathectomy with several days' duration. Specific nerve block techniques are outlined below, separated by anatomical region.

Regional Anesthesia of the Head and Neck

Regional anesthesia of the head and neck has application during trigeminal or occipital nerve blocks for diagnostic or therapeutic applications for pain syndromes. Cervical plexus blockade is useful for surgical procedures on the neck and shoulder. Maxillary, mandibular, and ocular nerve blocks are used for procedures on the face.

Cervical Plexus Block

The cervical plexus is formed by loops between the ventral rami of the upper four cervical nerves. These emerge as four distinct nerves from the posterior border of the sternomastoid at approximately its midpoint. The first branch radiates upward and backward as the lesser occipital nerve to supply part of the posterior surface of the upper part of the ear and the skin behind the ear; the second branch runs upward and forward as the great auricular nerve, which supplies skin over the posterior surface of the ear and the anterior lower third of the ear, and the angle of the mandible; the third branch, the anterior cutaneous nerve of the neck, supplies the skin from the chin to the suprasternal notch; and the fourth branch, the supraclavicular nerve, supplies the skin over the inferior aspect of the neck and clavicle down as far as the area over the second rib. Laterally, these supraclavicular nerves supply the skin over the deltoid muscle and posteriorly as far as the spine of the scapula.[41]

The branches of the plexus are divided into superficial and deep. The superficial branches pierce the cervical fascia just posterior to the sternomastoid and supply the skin of the side of the face and neck. The deep branches contribute primarily efferent (ie, motor) fibers to the muscles of the neck and the phrenic nerve.

Indications. Cervical plexus blockade is useful for operations on the lateral or anterior neck such as carotid endarterectomy or thyroidectomy.[70] It is useful for treatment of pain syndromes such as reflex sympathetic dystrophy.

Anatomy. The dorsal rami supply the muscles of the back of the head and neck and innervate the skin from the vertex to the shoulders.[71] The ventral rami of the upper four cervical nerves form the cervical plexus. This plexus innervates the skin of the front of the neck, the shoulders, and the upper chest, together with the neck muscles. The plexus lies over the upper four cervical vertebrae, deep to the internal jugular vein and the sternomastoid muscle, and anterior to the scalenus medius and levator scapulae. The branches of the plexus divide into superficial and deep. The superficial branches supply the skin of the side of the face and neck. The deep branches contribute motor nerve fibers to the muscles of the neck and phrenic nerve.[72]

Technique. Both superficial and deep cervical plexus blockades can be accomplished using the following equipment:

- 5-cm, 22-gauge needles; and
- 3 or 4 mL of 1% lidocaine or its equivalent.[41]

All four branches of the superficial cervical plexus can be blocked by infiltration at the midpoint of the posterior border of the sternomastoid. Lidocaine, 1% or its equivalent, can be infiltrated at this area; 5 to 10 mL will be required. This will produce cutaneous analgesia of the anterior and lateral neck from the mandible to the clavicle.[41]

The deep cervical plexus blockade is a para-vertebral block of spinal nerves C-2 through C-4 as they emerge from the foramina in the cervical vertebrae (Figure 12-4). Each nerve lies in the sulcus in the transverse processes of these vertebrae.[41] A line is drawn from the mastoid process to Chassaignac's tubercle (C-6). This line crosses a line drawn laterally from the cricoid cartilage, which is at the level of C-4.

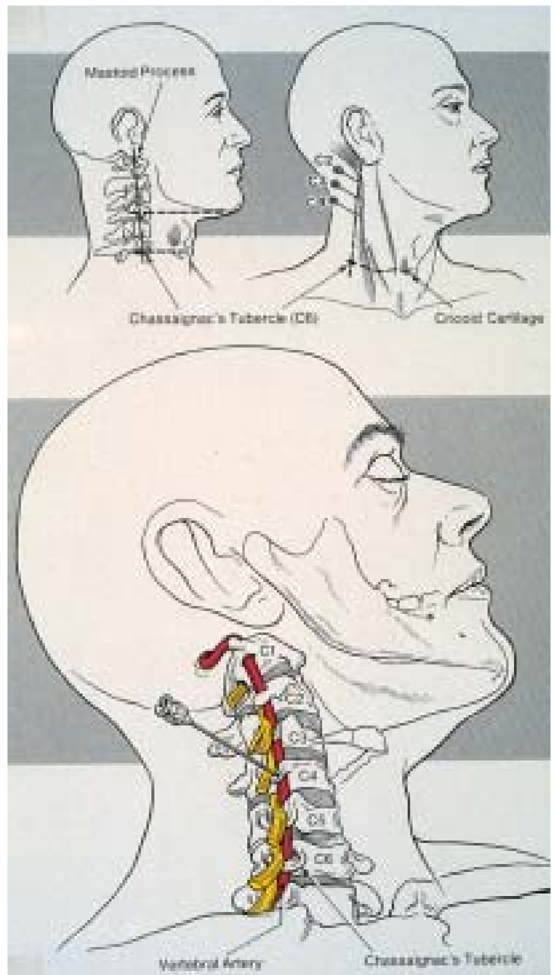


Fig. 12-4. When performing a deep cervical plexus block, note that caudad direction of the needle is essential to avoid penetration of an intervertebral foramen. Frequent aspiration is necessary to prevent injection into the vertebral artery. Reprinted with permission from Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade in Clinical Anesthesia and Management of Pain*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1988: 553.

The C-3 and C-2 nerve roots can be located by dividing the distance between the mastoid and horizontal line into thirds.[41] The C-5 nerve root lies midway between the C-6 line at Chassaignac's tubercle and C-4.

The needles are directed medially and caudally. Caudad direction is to avoid unintentionally entering the intervertebral foramen and producing a spinal block. The needle is advanced until the transverse process is contacted or paresthesias are obtained. Adequate anesthesia is provided by 3 to 4 mL of 1% lidocaine or the equivalent on each nerve. Deep cervical plexus block can often be obtained with injections at just one level with a larger volume, such as 6 to 8 mL.[41]

Complications. Complications of cervical plexus block include phrenic nerve block (a reason to avoid bilateral cervical plexus block), vertebral artery injection, epidural injection, subarachnoid injection, and recurrent laryngeal nerve block.[44]

Occipital Nerve Block

The skin over the posterior extensor muscles of the neck and occiput is supplied by the posterior rami of the cervical nerves.[41] The greater occipital nerve is the most prominent nerve supplying this area. It is sometimes blocked in the treatment of occipital tension headaches.

Anatomy. The greater occipital nerve can be blocked as it crosses the superior nuchal line. This line is identified by locating the greater occipital protuberance and the mastoid process. The greater occipital nerve crosses the superior nuchal line one third of the way between the external occipital protuberance and the mastoid process.[41]

Technique. Occipital nerve block is accomplished using a short needle (2–3 cm, 22- to 25-gauge) after a sterile skin preparation (Figure 12-5). The greater occipital nerve is located one third of the way between the greater occipital protuberance and the mastoid process.[41] It lies adjacent (medial) to the occipital artery, which can be palpated. Infiltration of 2 to 10 mL of 0.25% bupivacaine or its equivalent around the artery will provide a block of the greater occipital nerve.[57]

The lesser occipital nerve lies approximately 2.5 cm lateral to the greater occipital nerve. It can be blocked directly above and behind the mastoid process. This block is done by entering the skin vertically, directly at the exit point of the nerve behind the mastoid process. After definite bony contact has been established, 2 to 10 mL of local anesthetic is infiltrated (0.25% bupivacaine or its equivalent).

Complications. The occipital nerve block is associated with few complications, but inadvertent intravenous injection is possible.

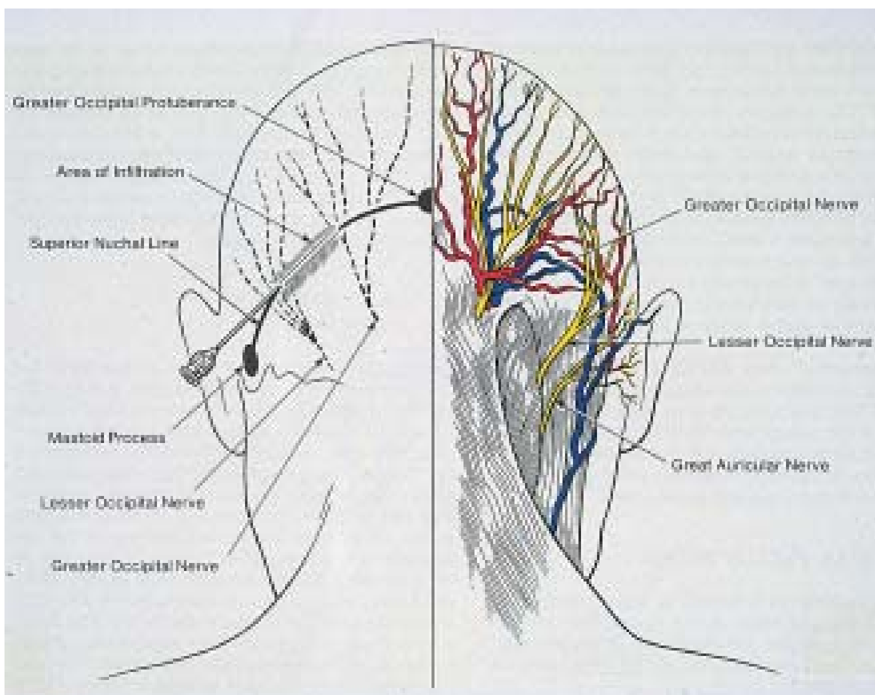


Fig. 12-5. In an occipital nerve block, the anesthetic agent is injected along an imaginary curved line extending between the mastoid process and the greater occipital protuberance. Reprinted with permission from Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade in Clinical Anesthesia and Management of Pain*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1988: 552.

Maxillary Nerve Block

Block of the maxillary nerve will anesthetize the skin and deep structures of the middle face including the nasal cavity, the maxillary bone and sinus, the upper teeth, and the upper part of the mouth and oral cavity. Indications for maxillary nerve block include trigeminal neuralgia of the second branch, pain of maxillary neoplasm, postoperative pain, atypical facial neuralgia, acute herpes zoster, and cluster headache.[57]

Anatomy. The maxillary nerve leaves the cranium through the foramen rotundum, crosses the pterygopalatine fossa, and enters the orbit through the inferior orbital fissure, where it becomes the infraorbital nerve.[50,71] Branches of the maxillary nerve include the posterior, middle, and anterior superior alveolar nerves.[57,72]

Technique and Local Anesthetic Drug Dosage. A 7-cm needle is inserted medially at the junction of the zygoma and the anterior edge of the ramus. The needle is inclined cephalad and posteriorly. The needle is advanced to the depth of the sphenoid bone.[57,72] A dose of 4 mL of 1% lidocaine or 0.25% bupivacaine is used to achieve anesthesia.[57]

Complications. The only reported complications are hemorrhage due to injury of the maxillary artery and subarachnoid injection.[57,72]

Mandibular Nerve Block

Block of the mandibular nerve will anesthetize the lower jaw and the skin and tissues of the lower face. The mandibular nerve, which is the largest branch of the trigeminal nerve, is sensory to the skin over the lower jaw, posterior face, lower lip, lower teeth and gums.[72] It supplies the motor fibers to the muscles of mastication.

Anatomy. The nerve divides into an anterior and a posterior trunk after leaving the foramen ovale. The sensory fibers of the anterior trunk run in the buccal nerve, which is distributed with branches of the facial nerve supplying the skin and mucous membrane on either side of the buccinator muscle. The posterior trunk divides into three branches: the auriculotemporal, the lingual, and the inferior alveolar nerves.[72] The auriculotemporal supplies the skin over the temporal region and external auditory meatus. The lingual supplies the mucous membrane of the floor of the mouth and anterior two thirds of the tongue, in addition to the lingual surface of the lower gums.[72] The inferior alveolar enters the mandibular foramen and runs in the mandibular canal supplying the lower teeth. At the mental foramen it gives off a lateral branch, the mental nerve, which supplies the skin over the anterior jaw and lower lip.[72]

Technique and Local Anesthetic Drug Dosage. To perform this block, a wheal is raised 0.5 cm below the zygomatic arch between the coronoid process and the condyle of the mandible. The needle is inserted at right angles to the skin until it contracts the pterygoid plate at a depth of 3 to 4 cm. It is then withdrawn a few millimeters and redirected 20° posteriorly until a paresthesia is elicited. Anesthetic drugs such as 5 mL of 2% lidocaine or 0.5% bupivacaine or their equivalent are used.[72]

Trigeminal Nerve Block

The trigeminal nerve, the largest of the cranial nerves, can be blocked for treatment of trigeminal neuralgia or postherpetic pain. The sensory supply to the face and anterior two thirds of the scalp is from the trigeminal nerve. It is formed by the union of a sensory and a motor root. This union is called the trigeminal, or Gasserian, ganglion. The trigeminal nerve supplies the skin of the face and scalp and the muscles of mastication via its three main branches: (1) the ophthalmic nerve, which leaves the cranium through the superior orbital fissure; (2) the maxillary nerve, which leaves through the foramen rotundum; and (3) the mandibular nerve, which leaves through the foramen ovale. All three nerves can be blocked with a single injection through the foramen ovale.[72]

Because the trigeminal nerve block is complex and possible complications include subarachnoid injection of local anesthetic at the base of the brain, we recommend that this block should *not* be performed in far-forward battlefield conditions. Radiographic capabilities to perform the block under radiographic control are required.

Ocular Nerves

Surgery of the eye can be done by numerous local techniques, including the following[72]:

- The cornea can be anesthetized by topical local anesthesia.
- The conjunctiva can be anesthetized by local infiltration.
- The globe can be anesthetized by retrobulbar injection.

- The nasociliary nerve supplies the skin and mucous membrane of the nose, cornea and conjunctiva; it can be blocked at the medial wall of the orbit.
- The supraorbital nerve supplies the upper eyelid and conjunctiva; it can be blocked at the supraorbital foramen.
- The infraorbital nerve supplies the skin of the ala of the nose, the lower eyelid, the cheek, and the upper lip; it can be blocked as it enters the face through the infraorbital foramen.

These procedures are not commonly done by most anesthetists, and it is recommended that they be performed only by personnel who are facile with local ophthalmic anesthesia.

Regional Anesthesia of the Airway

The nasal mucosa is innervated by fibers of the sphenopalatine ganglion via a branch of the middle division of the fifth cranial nerve. Branches of the fifth cranial nerve continue caudad to provide sensory innervation to the superior pharynx, uvula, and tonsils. Transmucosal topical application of local anesthetic can be used to anesthetize this region.[70]

Sensory innervation of the oral pharynx and supraglottic regions below the sphenopalatine fiber distribution is from branches of the glossopharyngeal nerve. These nerves lie laterally on each side of the pharynx in the region of the posterior tonsillar pillars. Topical anesthesia of these branches in the mouth and throat is an effective method of providing anesthesia to the submucosal branches of the glossopharyngeal nerve.[70]

The larynx is innervated by the superior laryngeal branch of the vagus nerve above the vocal cords. Below the vocal cords, the recurrent laryngeal nerve provides sensory innervation and supplies motor innervation for all but one intrinsic laryngeal muscle.[70]

Superior Laryngeal Nerve Block

The superior laryngeal branch of the vagus nerve is blocked as it sweeps around the inferior border of the greater cornu of the hyoid bone.[41] This can be palpated by pressing on the opposite greater cornu of the hyoid bone, which will displace the opposite cornu toward the side to be blocked.[41] A 2.5-cm, 25-gauge needle is walked from the inferior border of the greater cornu of the hyoid near its tip, and 3 mL of local anesthetic is infiltrated both superficially and deep to the thyrohyoid membrane (Figure 12-6). This procedure is repeated bilaterally. This block will produce anesthesia over the inferior aspect of the epiglottis and laryngeal inlet as far down as the vocal cords.[41]

Transtracheal Block

To produce anesthesia below the cords, a transtracheal puncture can be performed. A 20-gauge catheter is introduced through the cricothyroid membrane in the midline. After entry into the trachea is confirmed by aspiration of air, the catheter is advanced from the needle. Correct placement is confirmed again by aspiration of air, followed by rapid injection of 3 to 5 mL of local anesthetic, which produces a cough reflex. For this block, 2% to 4% lidocaine is commonly employed. Complications of the transtracheal block include the possibility of aspiration in certain patients due to the loss of the protective airway reflexes.

Nerve Blocks for Upper Extremity Surgery

Injuries to the upper extremities are common during wartime and may be amenable to regional anesthetic techniques during surgical repair. Regional anesthesia has the advantage of providing postoperative pain control and blockade of the sympathetic nervous system with selected blocks. Knowledge of the anatomy of the upper extremity is crucial for performance of regional anesthetic blockade.

The brachial plexus can be blocked at three locations[41]:

1. the interscalene approach, in which a needle is inserted in the interscalene groove at the level of the cricoid cartilage and advanced until the tubercle of C-6 is contacted or until a paresthesia is elicited;
2. the supraclavicular approach, in which the needle is inserted into the interscalene groove 1 cm or until a paresthesia is elicited; and
3. the axillary approach, which carries the least risk of pneumothorax.

The interscalene and supraclavicular approaches are more difficult to perform and therefore are less frequently used than the axillary approach.

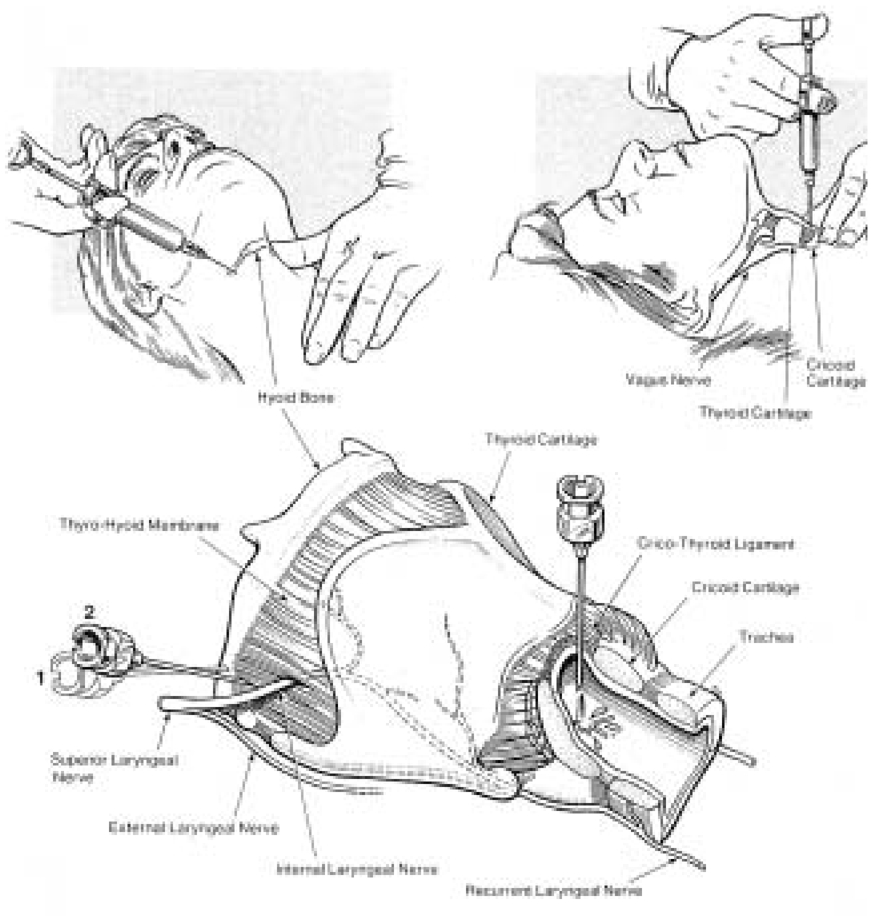


Fig. 12-6. Technique for anesthetizing the larynx and trachea such as might be used in performing bronchoscopy. The superior laryngeal nerve is blocked as it passes anterior and inferior to the posterior cornu of the hyoid bone. Anesthesia of the tracheal mucosa is obtained by injecting the agent directly through the space between the thyroid and cricoid cartilages. Reprinted with permission from Murphy TM. Somatic blockade of head and neck. In: Cousins MJ, Bridenbaugh PO. *Neural Blockade in Clinical Anesthesia and Management of Pain*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1988: 549

Brachial Plexus Block: The Axillary Approach

Innervation of the upper extremity is from nerve roots of C-5 through T-1, which form the brachial plexus. The median and musculocutaneous nerves lie above the axillary artery; the ulnar and radial nerves lie below and behind the artery.

Technique. The axillary approach to a brachial plexus block is performed using the following equipment:

- 23-gauge, 1½-in., short, beveled needle;
- extension tubing; and
- 20-mL syringes.

The patient is in the supine position with the arm abducted and elbow flexed (Figure 12-7). The arm is externally rotated, so that the hand lies alongside the patient's head. The axilla is prepared and draped for surgery. The axillary artery is located as high as is practical in the axilla as it courses in the groove between the coracobrachialis and triceps muscles. The needle is advanced through the artery, which is deliberately entered. The needle is advanced until aspiration confirms it has passed just posterior to (ie, through) the artery, at which time one half of the local anesthetic solution is injected. The needle is then withdrawn until aspiration confirms it is just anterior to the artery; then the other half of the solution is injected.

The musculocutaneous nerve is blocked by infiltration of 5 mL of anesthetic into the coracobrachial muscle. Median cutaneous and intercostobrachial nerves can be blocked by subcutaneous infiltration of 10 mL of anesthetic solution in a half-ring around the medial aspect of the arm.

Local Anesthetic Drug Dosages. To achieve nerve blockade, the anesthesiologist should inject 30 to 40 mL of local anesthetic solution (1% lidocaine, 0.25% bupivacaine, 1.5% mepivacaine); in children, 10 to 20 mL should be injected.

Complications. When the axillary approach is used, complications are rare but include neuropathy, hematoma, and intravascular injection.

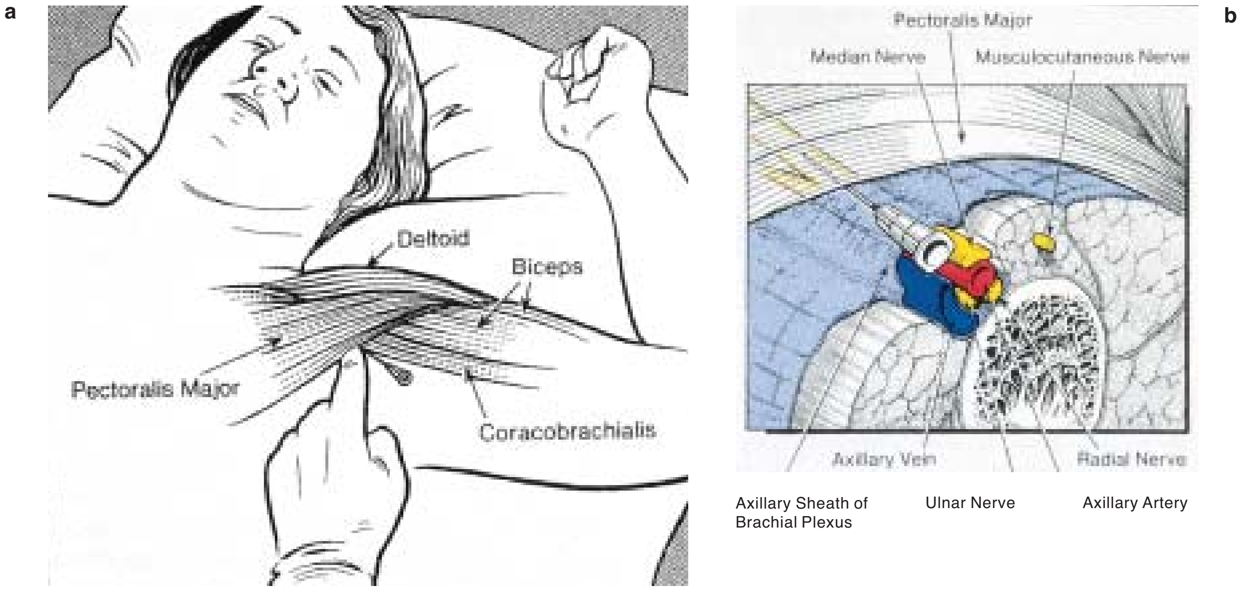


Fig. 12-7. The axillary approach to producing brachial plexus blockade. (**a**) The edge of the pectoralis major muscle and the axillary artery are the anatomical landmarks. (**b**) The anesthetic agent is injected deep to the axillary sheath of the brachial plexus. Reprinted with permission from Bridenbaugh LD. The upper extremity: somatic blockage. In: Cousins MJ, Bridenbaugh PO, eds. *Neural Blockade in Clinical Anesthesia and Management of Pain*. 2nd ed. Philadelphia, Pa: JB Lippincott; 1988: 401.

Brachial Plexus Block: The Interscalene Approach

The brachial plexus runs between the anterior and middle scalene muscles in the neck, where their fascia forms a perivascular sheath. The perivascular sheath is reached during this approach to a brachial plexus block by inserting a needle into the sheath at the sixth cervical vertebra, which corresponds to the level of the cricoid cartilage.

Technique. The patient is in the supine position, with the head rotated away from the side to be blocked (Figure 12-8). The groove between the anterior and middle scalene muscles is palpated behind the sternocleidomastoid muscle. The anterior scalene is immediately behind the sternomastoid, and a groove is palpable between the anterior and the middle scalene muscles.

A 3- to 4-cm needle is inserted perpendicular to the skin at the level of the cricoid cartilage (C-6) in the interscalene groove. The needle is slowly advanced until a paresthesia felt below the shoulder is elicited or a transverse spinous process is felt. If bone is contacted, the needle can be walked laterally along the transverse process until a paresthesia is elicited. A nerve stimulator with insulated needle may be used, instead of the anesthesiologist's relying on the casualty's perception of paresthesia. When it is in close proximity to the plexus, the nerve stimulator should elicit muscle twitching in the arm or hand.[72]

With an assistant using frequent aspiration, 30 to 40 mL of 1.5% mepivacaine (or its equivalent) is injected in fractionated doses. The patient's perception of pain while the anesthetic is being injected may indicate a direct intraneural injection, in which case the needle should be withdrawn about 1 mm and the injection should be repeated.

Complications. Complications of interscalene brachial plexus block include spinal or epidural blockade by inadvertent penetration of the epidural or dural spaces or entry via an intervertebral foramen, intraneural injection, and intraarterial or venous injection.

Brachial Plexus Block: The Supraclavicular Approach

The supraclavicular approach to brachial plexus block involves injecting local anesthetic into the perivascular sheath that surrounds the plexus at the level of the clavicle. When the brachial plexus passes between the clavicle and the first rib, it is joined by the subclavian artery, which runs deep to the anterior scalene muscle. The three trunks (superior, middle, and inferior) of the brachial plexus lie

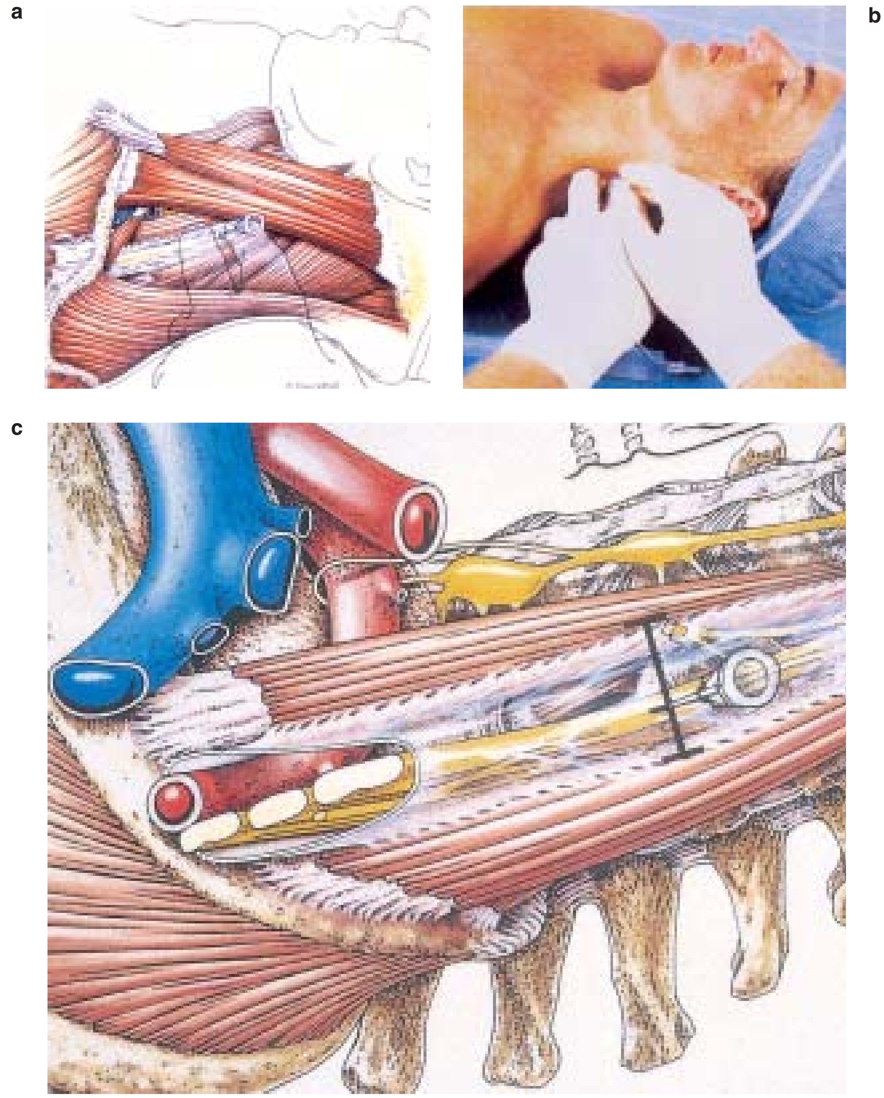


Fig. 12-8. (**a**) A needle is being placed in the perivascular sheath surrounding the brachial plexus. The anesthesiologist is palpating the interscalene groove to facilitate needle placement. (**b**) A flexible cannula is used during injection to ensure that the needle remains immobile after correct placement has been achieved. (**c**) The anatomy of the brachial plexus and its perivascular sheath at the C-6 level is seen in detail. Reprinted with permission from Scott DB. *Techniques of Regional Anaesthesia.* East Norwalk, Conn: Appleton & Lange; 1989: 93. Drawings by Paol Buckhöj.

in a vertical plane lateral to the subclavian artery.[72]

Technique. The patient is in the supine position, with the arm on the side to be injected pulled downward to depress the clavicle and shoulder (Figure 12-9). The subclavian artery can be palpated above the midportion of the clavicle. The interscalene groove is palpated posterior to the sternocleidomastoid muscle.

A 4-cm-long needle is inserted lateral to the subclavian artery and directed vertically and caudally. It is advanced slowly until the patient feels a paresthesia below the shoulder. If the needle contacts the first rib, the needle is withdrawn and the process is repeated as close as possible to the artery. A nerve stimulator can be used, and muscle movement in the arm or hand would indicate correct placement.[72]

When proper needle placement is obtained, fractionated doses of 1.5% mepivacaine (or its equivalent) are injected, using a flexible catheter, to a total dose of 44 mL.

Complications. Complications of this technique include arterial puncture, intraneural injection, pneumothorax, and spinal or epidural blockade.

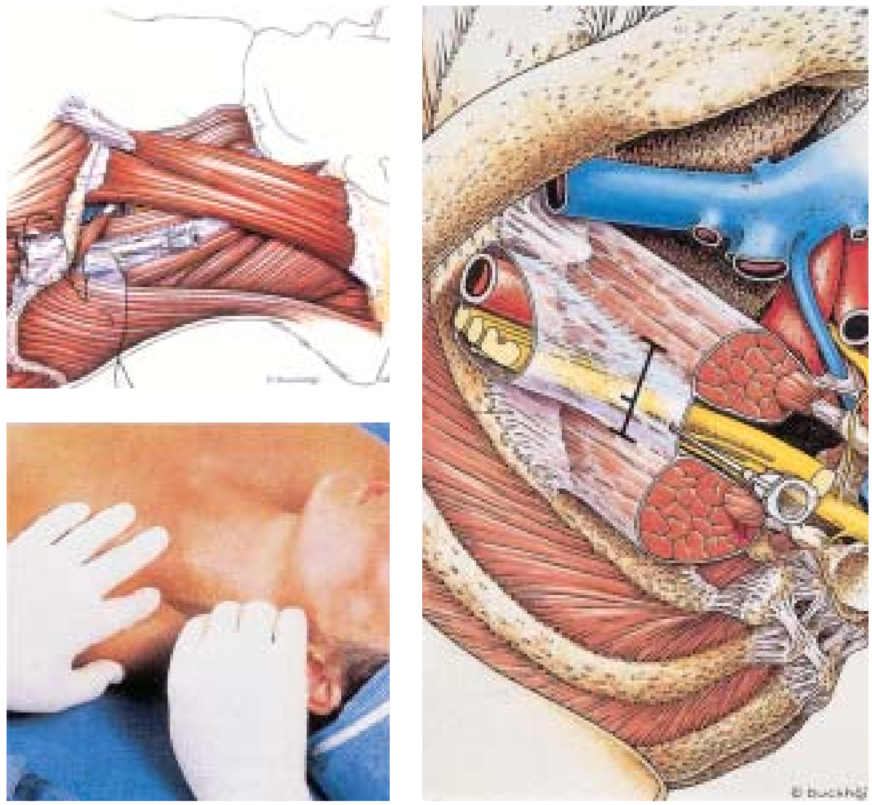


Fig. 12-9. (**a**) Needle placement into the perivascular sheath surrounding the brachial plexus and the subclavian artery at the level of the clavicle. (**b**) Palpation of the interscalene groove at the midportion of the clavicle and identification of the subclavian artery. (**c**) The three trunks of the brachial plexus and their relationship to the clavicle and interscalene muscles. Reprinted with permission from Scott DB. *Techniques of Regional Anaesthesia.* East Norwalk, Conn: Appleton & Lange; 1989: 99. Drawings by Paol Buckhöj.

Intravenous Regional Anesthesia: Bier's Method

Bier's method of achieving intravenous regional anesthesia (ie, the Bier block) allows analgesia and muscle relaxation in an extremity. It is performed by intravenously administering a local anesthetic distal to a tourniquet that provides complete vascular occlusion. We do *not* recommend the use of intravenous regional anesthesia for surgical procedures of the lower extremity. The high volumes of local anesthetic required to produce an adequate nerve block risk local anesthetic systemic toxicity and therefore make this technique a less desirable choice than specific nerve blocks.[73] Because the procedure is not infrequently used on the lower extremity, however, it is discussed here for the sake of completeness.

Technique. An intravenous catheter is placed in the distal extremity. A plastic extension tubing from the catheter is attached to a 50-mL syringe. A pneumatic double tourniquet is placed proximal to the operative site. The extremity is exsanguinated by the application of an Esmarch bandage or by elevation. The bandage is applied distal to proximal. The tourniquet is inflated after exsanguination and before removal of the bandage. The proximal pneumatic tourniquet is inflated above systolic pressure. Pulses are checked after inflation to confirm occlusion of circulation (usually 250–300 mm Hg in the arm and 350–400 mm Hg in the leg).

The local anesthetic solution is then injected. For short procedures, the intravenous catheter is removed at this point; for longer procedures, the cannula is left in place and the local anesthetic solution is reinjected after 90 minutes. The area anesthetized includes the distal extremity up to the area of the proximal tourniquet. If tourniquet discomfort begins, the distal tourniquet can be inflated, as this area is already anesthetized and the proximal tourniquet is deflated. Tourniquet pain is usually the limiting factor for success of this technique.

If surgery lasts longer than 40 minutes, the tourniquet can be deflated as a single maneuver. Between 20 and 40 minutes, the cuff can be deflated, reinflated immediately, and finally deflated after a minute to reduce the sudden absorption of anesthetic into the systemic circulation. The tourniquet is left inflated for at least 20 minutes.[70]

A separate intravenous site for injection of resuscitation drugs is needed for this block, as well as ready availability of all needed equipment.

Anesthetic Drug Dosages and Complications. The duration of action of the local anesthetic agent is limited by tourniquet time rather than the agent. The amount of drug given depends on the size of the extremity. Average doses are 40 to 50 mL of 0.5% lidocaine for an arm, and 100 mL of 0.25% lidocaine for a leg. Agents should not be mixed with vasoconstrictors. Local anesthetic drug toxicity can be seen if the tourniquet fails or is relieved too quickly. Nerve damage secondary to tourniquet application can be seen. Tourniquet time should not exceed 2 hours.

Relative Contraindications. Relative contraindications to the Bier block include distal infection of the extremity, ischemia of the involved extremity, heart block, and seizure disorder.

Sole-Anesthetic Nerve Blocks for Lower Extremity Surgery

Neural blockade of the lower extremity may be useful to provide preoperative analgesia, intraoperative anesthesia as the sole anesthetic or as a supplement to general anesthesia, and postoperative analgesia.[73] Some nerve blocks are suited for all three of these uses, while others provide good analgesia but may not be acceptable for intraoperative anesthesia.

As has been described earlier in this chapter, lower extremity anesthesia can be obtained by spinal or epidural techniques. Additional nerve blocks that may be useful for preoperative analgesia, intraoperative general anesthesia supplementation, postoperative analgesia, or as the sole anesthetic are described in this section. Combined peripheral nerve block techniques are indicated if spinal or epidural anesthesia are undesirable.[73] Advantages of a combined peripheral nerve block technique include the blockade of only one leg, smaller hemodynamic changes owing to the limited sympathectomy, and avoidance of major conduction anesthesia. The major disadvantage of the technique is the need for at least two needle insertions to block separately the peripheral nerves formed by the lumbar plexus and those formed by the sacral plexus. Individual techniques are discussed in the following section.

Lumbar Plexus Block

Blockade of the lumbar plexus provides unilateral analgesia in the distribution of the femoral, lateral femoral cutaneous, and obturator nerves. Patients with trauma to or surgical procedures of the hip or thigh may therefore benefit from this block. Lumbar plexus block is contraindicated in

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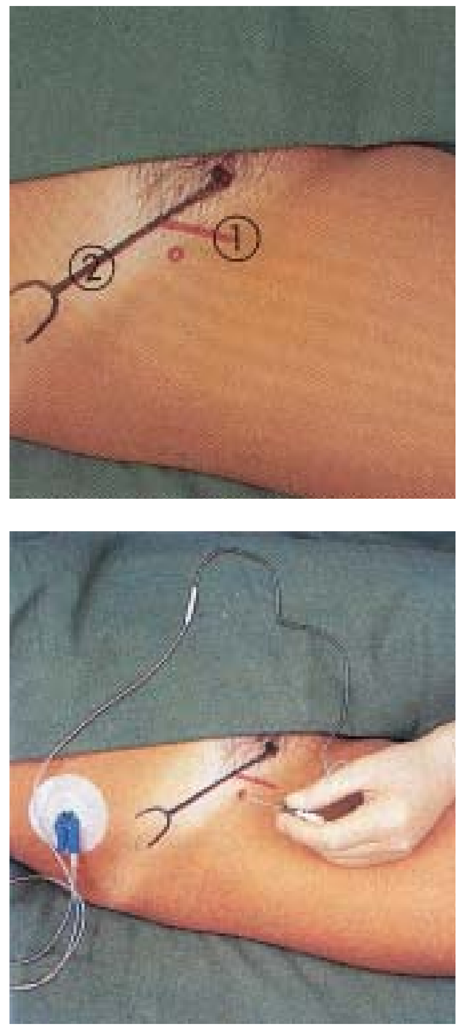

Fig. 12-10. The 3-in-1 lumbar plexus blockade. (**a**) The anatomical landmarks are the inguinal ligament (2) as it extends between the anterior superior iliac spine and pubic tubercle and the femoral artery (1). (**b**) The needle is inserted 1 to 1.5 cm laterally to the femoral artery and advanced in a cranial direction. Continuous infusion of anesthetic agent through a catheter is desirable. Reprinted with permission from Astra Chemicals GmbH, Zenz M, Hoerster W, Niesel HC, Kreuscher H, eds; DeKornfeld TJ, trans. *Regional Anesthesia*. 2nd ed. St. Louis, Mo: Mosby–Year Book; 1990: 109, 111.

the presence of local infection, coagulation abnormality, immunosuppression, and perhaps neurological deficits involving the ipsilateral limb. Two techniques have been described: the Winnie "3-in-1" paravascular approach from the femoral canal,[74] and the lumbar paravertebral approach to the lumbar plexus blockade.[73]

Winnie 3-in-1 Paravascular Approach. The Winnie 3-in-1 blockade can be performed with the patient in the supine position (Figure 12-10). This technique utilizes the fascial plane that the femoral nerve travels in as it crosses the pelvis. A large quantity of local anesthetic is injected in this plane so that it will spread upward into the pelvis and anesthetize the obturator and lateral femoral cutaneous nerves where they travel in conjunction with the femoral nerve. Paresthesias are usually necessary for this approach, as it is essential to have the needle exactly in the plane of the nerve. This block is performed by inserting the needle in a cephalad manner alongside the femoral artery angled at about 45° so that it passes under the inguinal ligament. After obtaining a paresthesia, 40 mL of anesthetic solution is injected incrementally.[70] During the injection, the anesthesiologist's index finger is moved to a point just distal to the needle, where firm pressure is applied to prevent retrograde flow of the injected local anesthetic.

Lumbar Paravertebral Approach. The lumbar paravertebral approach to the lumbar blockade may be used if the patient can be turned, because this approach is more often successful in blocking the obturator nerve (Figure 12-11). For continuous blockade, a Tuohy needle is inserted 3 cm lateral to the midline at the level of the L-3–L-4 interspace and is advanced until the transverse process of

b

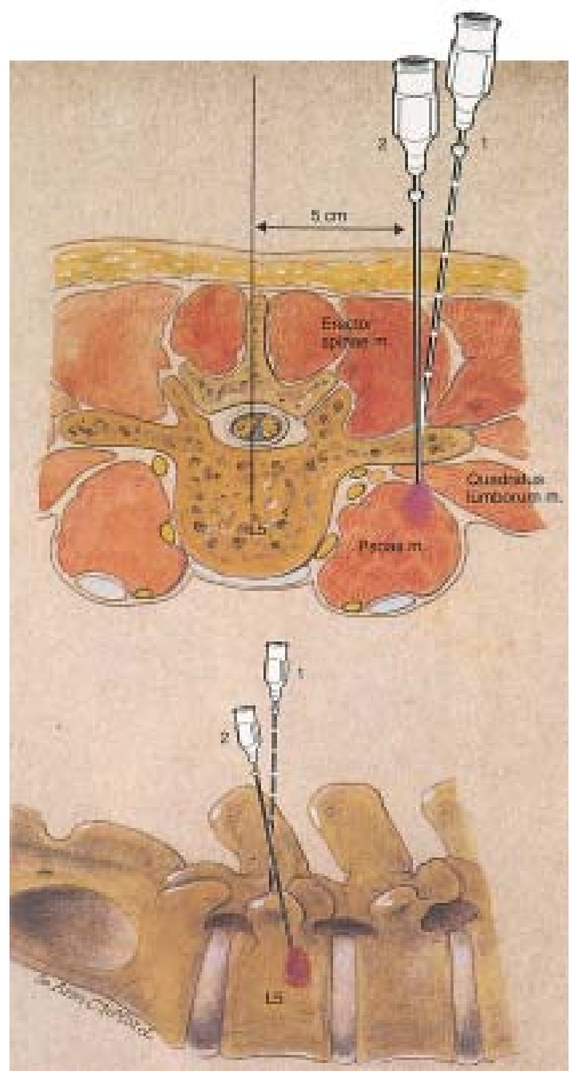

Fig. 12-11. Lumbar plexus block at the psoas compartment. (**a**) The iliac crests are used to determine the spinous process of the 4th lumbar vertebra. (**b**) A 15-cm long needle is inserted at a point 5 cm lateral to midline and 3 cm caudad to the 4th lumbar spinous process. The needle is advanced until the lateral process of the 5th lumbar vertebra is reached (needle point 1). The needle is then advanced in a cranial direction superior to the vertebral process and an injection made (needle point 2). Reprinted with permission from Brown DL. *Atlas of Regional Anesthesia*. Philadelphia, Pa: WB Saunders; 1992: 77, 78.

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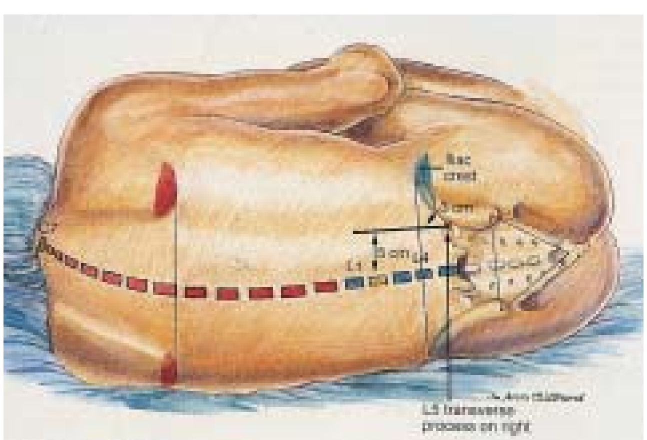

L-4 is contacted. The needle is then walked superiorly and laterally into the substance of the quadratus lumborum muscle, and is then advanced, using the loss-of-resistance technique, into the fascial plane between the quadratus lumborum and psoas muscles. The lumbar plexus lies within this psoas compartment. An epidural catheter may be passed into the compartment. Injection of 0.5 mL/kg of local anesthetic solution should provide a reliable block. The concentration of local anesthetic used depends on the degree of motor block desired. Continuous infusions of local anesthetic at 0.25 mL/kg/h may be used to continue the block.[73]

Femoral Nerve Block

Surgical use of femoral nerve block includes operations of the anterior portion of the thigh, both superficial and deep (Figure 12-12).[41] As with sciatic nerve block, the femoral nerve block is usually part of the combined block approach, incorporating not only sciatic but also lateral femoral cutaneous and obturator nerves.

The femoral nerve (which is composed of fibers arising from the spinal segments L-2–L-4) proceeds from the lumbar plexus in the groove between the psoas major and iliac muscles, where it enters the thigh by passing deep to the inguinal ligament. The femoral nerve lies anterior to the iliopsoas muscle and slightly lateral to the femoral artery.

Technique. The femoral nerve block may be accomplished using the following equipment:

- 1½-in., 22-gauge needle; and
- 20-mL syringe.

Palpate the femoral artery just below the inguinal ligament, and advance the needle toward the lateral border of the artery.[41] If a paresthesia is not obtained, redirect the needle to fan medial to lateral until a paresthesia is obtained. Then stabilize the needle and, after negative aspiration, inject 10 to 15 mL of local anesthetic solution. If a paresthesia is not obtained, the nerve can be blocked by injecting in a fanwise manner, medial to lateral, from the lateral border of the femoral artery.

Local Anesthetic Drug Dosages and Complications. Any anesthetic agent suitable for peripheral nerve blockade, 10 to 20 mL, is suitable. Hematoma at the site is possible because of the proximity to a major artery and vein. Residual nerve involvement is possible but rare, causing a dysesthesia or paresis.

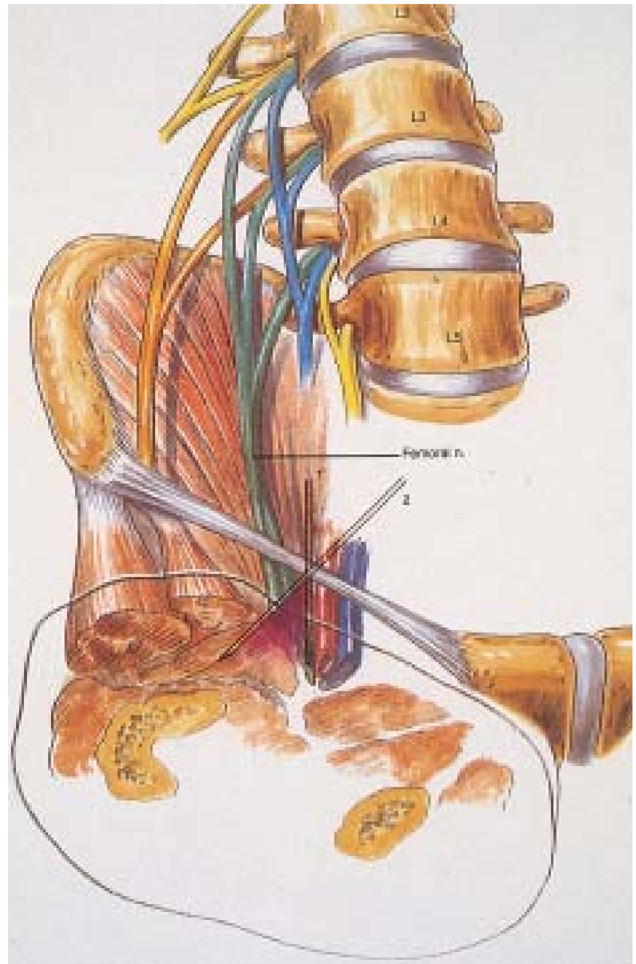

Fig. 12-12. Anatomy of the femoral nerve block. The inguinal ligament and the femoral artery are the landmarks. Injection is made lateral to the artery (2) after a femoral nerve paresthesia is produced (1). Reprinted with permission from Brown DL. *Atlas of Regional Anesthesia*. Philadelphia, Pa: WB Saunders; 1992: 95.

Sciatic Nerve Block

Sciatic nerve block is usually combined with femoral, obturator, or lateral femoral cutaneous nerve block to provide complete anesthesia of the lower extremity.[73] The sciatic nerve supplies sensory innervation to the posterior thigh and the entire leg and foot from just below the knee. The nerve (from spinal segments L-4–S-3) arises from the sacral plexus and passes from the pelvis through the sacrosciatic foramen beneath the lower margin of the piriformis muscle and between the tuberosity of the ischium and the greater trochanter of the femur (Figure 12-13).[41] The sciatic nerve becomes superficial at the lower border of the gluteus maximus muscle. From there, it courses down the posterior aspect of the thigh to the popliteal fossa, where it divides into the tibial and common peroneal nerves.[73]

Technique. The sciatic nerve block is performed with the following equipment:

- 21- or 22-gauge needle, 10 to 20 cm long; or
- 22-gauge, Teflon-coated (polytetrafluoroethylene, manufactured by Du Pont Polymers, Wilmington, Del.) stimulator needle with nerve stimulator; and
- 10-mL syringe.

The patient is relaxed and lying on the side that is not to be blocked. The upper leg is flexed at the hip and at the knee so that the heel rests on the knee of the lower, extended leg. The landmarks are connected with a line (greater trochanter, superior–posterior iliac spine). From the middle of this line, a line is drawn inferiorly at right angles and intersects a line drawn between the tip of the coccyx and the greater trochanter at about 3 cm. This point is marked for needle insertion. A 22-gauge, 15-cm long needle is introduced through a skin wheal at right angles to the skin and is advanced in the direction of the sacrosciatic foramen, through the gluteus maximus. Paresthesias, which may extend to the sole of the foot, prove the correct position of the needle. Electrical stimulation with a needle stimulator facilitates placement. After aspiration, 20 to 30 mL of the local anesthetic is injected.

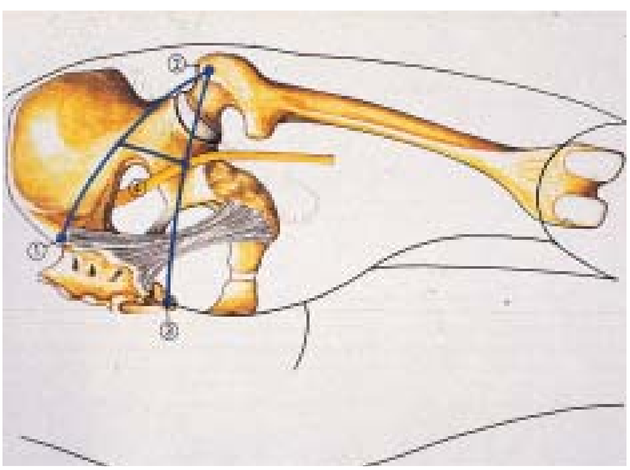

Fig. 12-13. The sciatic nerve block. A line is drawn between the posterior superior iliac spine and the greater trochanter. Then a line is dropped from its midpoint at right angles, which intersects a line drawn between the tip of the coccyx and the greater trochanter at about 3 cm. 1: posterior-superior iliac spine; 2: greater trochanter muscle; 3: tip of coccyx; 4: sciatic nerve. Reprinted with permission from Astra Chemicals GmbH, Zenz M, Hoerster W, Hiesel HC, Kreuscher H, eds; DeKornfeld TJ, trans. *Regional Anesthesia*. 2nd ed. St. Louis, Mo: Mosby–Year Book; 1990: 116, 117.

Local Anesthetic Drug Dosages and Complications. The sciatic nerve block can be achieved with 20 to 30 mL of 1.5% lidocaine or 0.5% bupivacaine. No significant complications secondary to this block have been documented. Residual dysesthesias have been reported but are usually self-remitting.[41]

Sciatic Nerve Block at the Popliteal Fossa

Sciatic nerve block from the popliteal fossa approach will provide unilateral anesthesia for procedures below the level of the knee, with the exception of the saphenous nerve distribution, which is a continuation of the femoral nerve, originating from the lumbar plexus (Figure 12-14).[41,73] The popliteal approach is especially amenable to a catheter technique.

Technique. Sciatic nerve block from the popliteal fossa approach can be accomplished using the following equipment:

- 18-gauge, 2½-in. catheter; and
- 20-gauge epidural catheter.

Insert the catheter over a blunt stylet 10 cm above the popliteal crease in the midline at an angle of approximately 30° to the skin. The needle is advanced in an anterior and cephalad direction until a fascial "pop" is felt. The stylet is then withdrawn and 20 mL of local anesthetic solution is injected through the catheter in incremental doses. A 20-gauge epidural catheter is then threaded through the short catheter; the short catheter is removed; and the epidural catheter is fixed in place using a sterile, transparent dressing. The sciatic nerve is anesthetized at its bifurcation to the common peroneal and tibial nerves.

Contraindications. Sciatic nerve block from the popliteal fossa approach is contraindicated in the presence of local or systemic infection, coagulation abnormality, immunosuppression, and unstable neurological deficits.[41]

Complications. The sciatic nerve block may be complicated by needle trauma, infection, and inadequate nerve block.

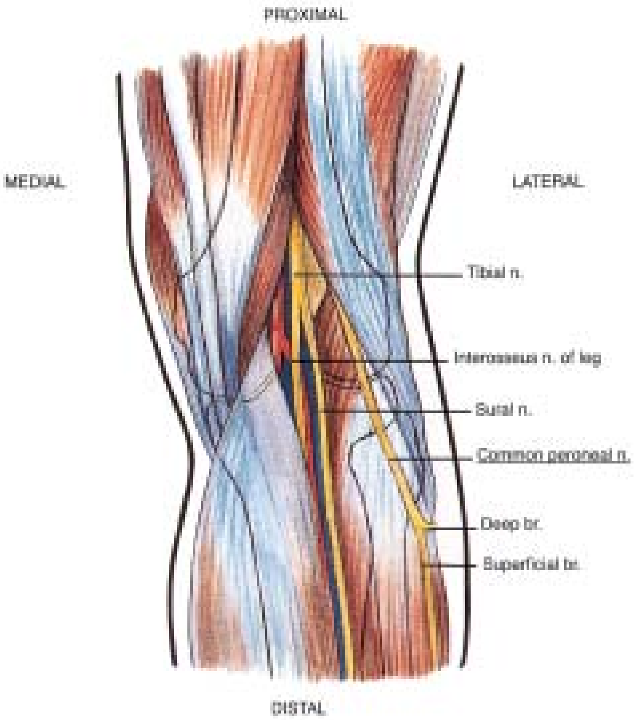

Fig. 12-14. Sciatic nerve block at the popliteal fossa. The injection is made in the posterior midline about 10 cm cranial to the transverse crease in the skin of the popliteal fossa. Reprinted with permission from Brown DL. *Atlas of Regional Anesthesia*. Philadelphia, Pa: WB Saunders; 1992: 112.

Lateral Femoral Cutaneous Nerve Block

The lateral femoral cutaneous nerve supplies the skin over the anterolateral aspect of the thigh as low as the knee. A posterior branch pierces the fascia lata and passes backward to supply the skin on the lateral side of the thigh. One of the terminal anterior branches forms part of the patellar plexus and must also be blocked for operations on the knee.

The lateral femoral cutaneous nerve (L-2–L-3) emerges at the lateral border of the psoas muscle at a level lower than the ilioinguinal nerve.[41] It passes obliquely under the iliac fascia and across the iliac muscle to enter the thigh deep to the inguinal ligament at a point 1 to 2 cm medial to the anterior superior iliac spine.

Technique. Lateral femoral cutaneous nerve block can be accomplished using the following equipment:

- 22-gauge, 1½ in. needle; and
- 10-mL syringe.

The patient is placed in the supine position. After palpation of the anterior superior iliac spine, a skin wheal is placed 2 to 3 cm inferior and 2 to 3 cm medial to it.[41] A 3- to 4-cm needle with syringe attached is inserted perpendicular to the skin (Figure 12-15). After the needle passes through the skin, the anesthesiologist feels firm fascia lata, followed by a sudden release as the needle passes through.

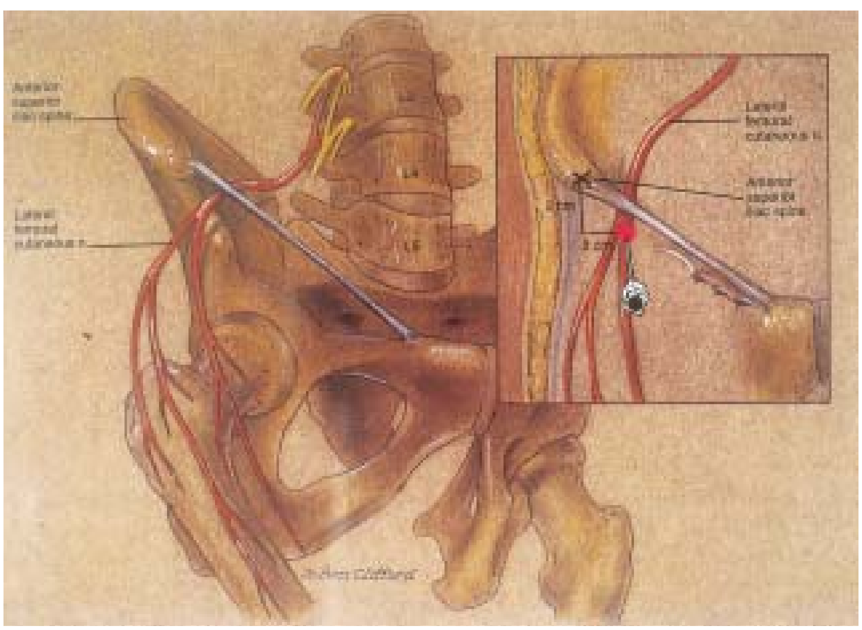

Fig. 12-15. The lateral femoral cutaneous nerve block as it passes under the inguinal ligament at a point about 2 to 3 cm inferior and 2 to 3 cm medial to the anterior superior iliac crest. Reprinted with permission from Brown DL. *Atlas of Regional Anesthesia*. Philadelphia, Pa: WB Saunders; 1992: 100.

Ten milliliters of a local anesthetic solution can be deposited fanwise as the needle is moved upward and downward, depositing solution both above and below the fascia.

An alternative technique is to direct the needle through the skin wheal in a slightly lateral and cephalad direction to strike the iliac bone just medial and inferior to the anterior superior iliac spine where the nerve emerges. Ten milliliters of local anesthetic is deposited in a medial fanwise fashion at this point.

Local Anesthetic Dosage and Complications. Any anesthetic agent suitable for peripheral nerve block can be used, in the amount of 10 mL. With the exception of remotely possible dysesthesia or hypoesthesia, there are no known risks with this nerve block.[41]

Ankle Block

The ankle block provides anesthesia for the foot by blocking five nerves at the level of the malleoli: the posterior tibial, sural, superficial peroneal, deep peroneal, and saphenous nerves.[41] The latter three innervate the dorsum of the foot (Figure 12-16).

The tibial nerve (composed of fibers from L-4–S-3), the larger of the two branches of the sciatic nerve, reaches the distal part of the leg from the medial side of the Achilles tendon, where it lies behind the posterior tibial artery.[41] The medial branch supplies the medial two thirds of the sole and plantar portion of the medial three and one half toes, up to the nail. The lateral branch supplies the lateral one third of the sole and plantar portion of the lateral one and one half toes.

The sural nerve is a cutaneous nerve that arises through the union of a branch from the tibial nerve and one from the common peroneal nerve. It proceeds subcutaneously along the short saphenous vein behind and below the lateral malleolus to supply the lower posterolateral surface of the leg, the lateral side of the foot, and the lateral part of the fifth toe.

The superficial peroneal nerve (composed of fibers from L-4–S-2) perforates the deep fascia on the anterior aspect of the distal two thirds of the leg and runs subcutaneously to supply the dorsum of the foot and toes, except for the contiguous surfaces of the great and second toes.

The deep peroneal nerve (composed of fibers from L-4–S-2) courses down the anterior aspect of the interosseous membrane of the leg and continues midway between the malleoli onto the dorsum of the foot. It innervates the short extensors of the toes as well as the skin on the adjacent areas of the first and second toes. The anterior tibial artery lies medial to the nerve, as does the tendon of the extensor hallucis longus muscle.

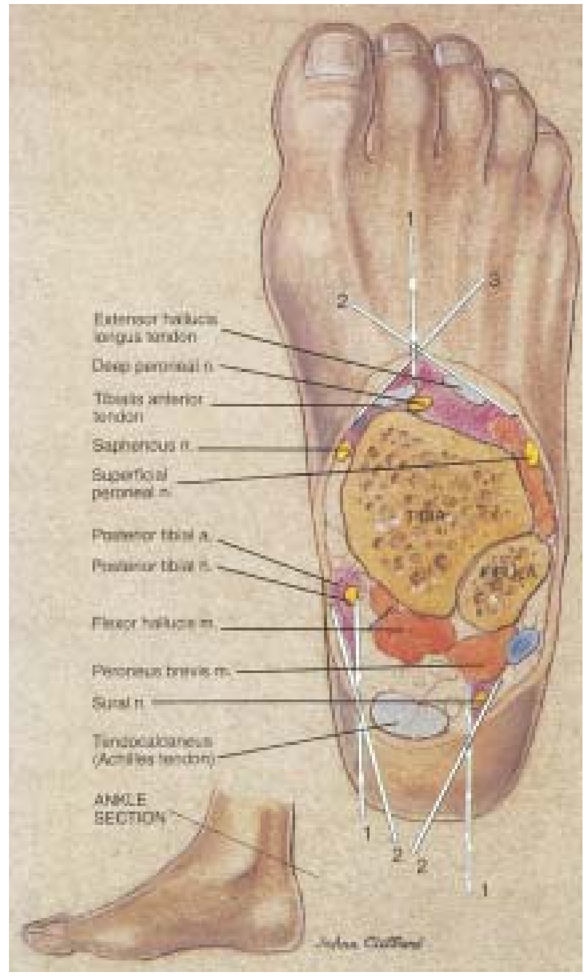

Fig. 12-16. Complete anesthesia of the foot can be obtained by blocking five nerves as they pass caudad to the ankle. The posterior tibial nerve is blocked near the posterior tibial artery as it passes the medial malleolus. The deep peroneal nerve is blocked near the dorsalis pedis artery. The same anterior needle site on the dorsum of the foot is used to infiltrate the tissues around the saphenous and superficial peroneal nerves. The sural nerve is blocked near the lateral malleolus. Needles labeled 1, 2, and 3 show sequence of infiltration. Reprinted with permission from Brown DL. *Atlas of Regional Anesthesia*. Philadelphia, Pa: WB Saunders; 1992: 119.

The saphenous nerve is the sensory terminal branch of the femoral nerve. It becomes subcutaneous at the lateral side of the knee joint. It follows the great saphenous vein to the medial malleolus and supplies the cutaneous area over the medial side of the lower leg anterior to the medial malleolus and the medial part of the foot.[41]

Technique. The ankle block can be accomplished using the following equipment:

- 22- to 25-gauge, 1½ in. needle; and
- 10-mL syringe.

The technique is modified according to the nerve to be blocked.

Posterior Tibial Nerve. The needle is introduced just posterior to the posterior tibial artery and is advanced until a paresthesia to the sole of the foot is elicited, at which point 5 mL of local anesthetic solution is injected.[41]

Sural Nerve. The needle is advanced into the groove between the lateral malleolus and the calcaneus, where 5 mL of local anesthetic solution is injected.

Saphenous Nerve. Infiltration of 5 mL of local anesthetic solution in the area where the saphenous vein passes anterior to the medial malleolus blocks this nerve.

Deep Peroneal Nerve. This is the major nerve to the dorsum of the foot, and it is blocked by placement of 5 mL of local anesthetic solution just lateral to the anterior tibial artery.

Superficial Peroneal Branches. A subcutaneous ridge of anesthetic solution (5–10 mL) is placed between the anterior tibial artery and the lateral malleolus.

Local Anesthetic Drug Dosages and Complications. Any anesthetic agents suitable for peripheral nerve block can be used, in the amounts specified above. Epinephrine-containing solutions are not used because of possible end-artery vasoconstriction and subsequent tissue ischemia. No major complications have been reported.[41]

SUMMARY

The techniques for regional anesthesia find potential application in several phases of combat casualty care: analgesia in the preoperative phase, intraoperative use as the sole anesthetic or as a supplement to general anesthesia, and analgesia in the postoperative phase. The most important benefit of regional anesthesia is that the deleterious effects of general anesthesia, such as loss of airway reflexes and respiratory depression requiring prolonged observation, are avoided. Furthermore, the quality of analgesia provided by regional blockade is superior to other forms of pain relief because local anesthetics completely block afferent and efferent nerve impulses.

Blockade of afferent impulses may have benefits that go beyond simple analgesia; the neurohormonal response to injury is itself blunted. The chemical agents used in regional anesthetics consist of a hydrophilic secondary or tertiary amine group bound to a lipophilic aromatic ring joined by either an amide or an ester linkage. The propensity for the hydrophilic group to ionize, as measured by the pKa, is an important determinate of potency; the higher the pKa, the greater the ionization and the longer the duration of action. This relationship arises from the fact that although passage of the anesthetic agent through the lipid myelin sheath of a nerve fiber depends upon lipid solubility, analgesia is caused by alterations in the structure of the proteins that constitute the sodium channels of the nerve axon. This latter effect depends on the binding of the ionized form of the hydrophilic group with receptor proteins.

Regional anesthetic techniques are determined by knowledge of anatomy and the nature of the planned surgery. Since the extremities are the most common site of war wounds, regional techniques would appear to be especially applicable. The epidural and spinal techniques are the most versatile regional procedures. They can be used for extensive abdominal and lower extremity operations, although they are contraindicated in the presence of severe systemic infection or uncorrected hypovolemia. Nerve blocks for specific anatomical regions, which with an experienced provider might find application in combat casualty care, are the brachial plexus block for injuries of the upper extremity and the sciatic and femoral nerve blocks for injuries of the legs. Intravenous regional anesthesia using the Bier method is especially useful in treating isolated injuries of the hand or forearm. Perhaps the most common technique using local anesthetics is also the simplest: local infiltration of the wounds, which is especially useful when a few, small, soft-tissue wounds require treatment.

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