Chapter

17 SPINAL AND EPIDURAL ANESTHESIA

Kenneth Drasner and Merlin D. Larson

Collectively referred to as central neuraxial block, spinal anesthesia and epidural anesthesia represent a subcategory of regional or conduction anesthesia. In addition to their current widespread use in the operating room for surgical anesthesia and as an adjunct to general anesthesia, neuraxial techniques are effective means for controlling obstetric (see Chapter 33) and postoperative pain (see Chapter 40).

COMPARISON OF SPINAL AND EPIDURAL ANESTHESIA

Spinal anesthesia is accomplished by injecting local anesthetic solution into the cerebrospinal fluid (CSF) contained within the subarachnoid (intrathecal) space. In contrast, epidural anesthesia is achieved by injection of local anesthetic solution into the space that lies within the vertebral canal but outside or superficial to the dural sac. Caudal anesthesia represents a special type of epidural anesthesia in which local anesthetic solution is injected into the caudal epidural space through a needle introduced through the sacral hiatus. Although epidural anesthesia is routinely performed at various levels along the neuraxis, subarachnoid injections are limited to the lumbar region below the termination of the spinal cord.

When compared with epidural anesthesia, spinal anesthesia takes less time to perform, causes less discomfort during placement, requires less local anesthetic, and produces more intense sensory and motor block. In addition, correct placement of the needle in the subarachnoid space is confirmed by a clearly defined end point (appearance of CSF).

Advantages of epidural anesthesia include a decreased risk for post–dural puncture headache (assuming a negligible incidence of inadvertent dural puncture), a lower incidence of systemic hypotension, the ability to produce a segmental sensory block, and greater control over the intensity of sensory anesthesia and motor block achieved by adjustment of the local anesthetic concentration. The routine placement of catheters for epidural anesthesia imparts additional benefit by allowing titration of the block to the duration of surgery. Additionally, a catheter provides a means for long-term administration of local anesthetics or opioid-containing solutions (or both), which are highly effective for control of postoperative or obstetric pain.

Patients may remain completely awake during surgery performed under neuraxial block, but more commonly they are sedated with various combinations of intravenous drugs, including sedative-hypnotics, opioids, and anesthetics (propofol). Skeletal muscle relaxation can be profound in the presence of neuraxial anesthesia and this may obviate the need for neuromuscular blocking drugs. However, despite potential advantages, patients may be reluctant to accept neuraxial anesthesia for fear of permanent nerve damage. Although there does exist the rare possibility of neural injury as a result of neuraxial anesthesia, patient concerns generally far exceed the clinical reality and at times are based on undocumented and unfounded stories of paralysis.[1]

As with other regional techniques, central neuraxial techniques require an understanding of the underlying anatomy and physiologic principles.

ANATOMY

Vertebral Canal

The spinal cord and its nerve roots are contained within the vertebral (spinal) canal, a bony structure that extends from the foramen magnum to the sacral hiatus (Fig. 17-1).[2] On a lateral view the vertebral canal exhibits four curvatures, of which the thoracic convexity (kyphosis) and the lumbar concavity (lordosis) are of major importance to the distribution of local anesthetic solution in the subarachnoid space. In contrast, these curves have little effect on the spread of local anesthetic solutions in the epidural space.

In addition to structural support, the vertebral canal provides critical protection to vulnerable neural structures. Unfortunately, this bony canal also creates a barrier to an advancing spinal or epidural needle seeking to trespass this space. Successful neuraxial block is thus critically dependent on the anesthesia provider's appreciation of the anatomy of this structure.

ARCHITECTURE

The building blocks of the vertebral canal are the vertebrae, which are stacked to form the tubular column (Figs. 17-2 and 17-3; also see Fig. 17-1).[2,3] This complex architecture is best appreciated by examination of a skeleton or a three-dimensional model. Although the structure of the vertebrae varies considerably, depending on their location and function, each consists of an anterior vertebral body and a posterior arch. The posterior arch is created by fusion of the lateral cylindrical pedicles with the two flattened posterior laminae. A transverse process extends out laterally at each junction of the pedicle and laminae, whereas a single spinous process projects posteriorly from the junction of the two laminae. Each pedicle is notched on its superior and inferior surface, and when two adjacent vertebrae are articulated, these notches form the intervertebral foramina through which the spinal nerves emerge.

NOMENCLATURE AND FEATURES

Of the 24 true vertebrae, the first 7, located in the neck, are called cervical vertebrae, the next 12 are attached to the ribs and are called thoracic (or dorsal) vertebrae, and the remaining 5 are the lumbar vertebrae. Another five vertebrae, called false or fixed vertebrae, are fused to form the bony sacrum (Fig 17-4).[4] Thus, the sacrum and coccyx are distal extensions of the vertebral column, and the sacral canal is a continuation of the vertebral canal through the sacrum.

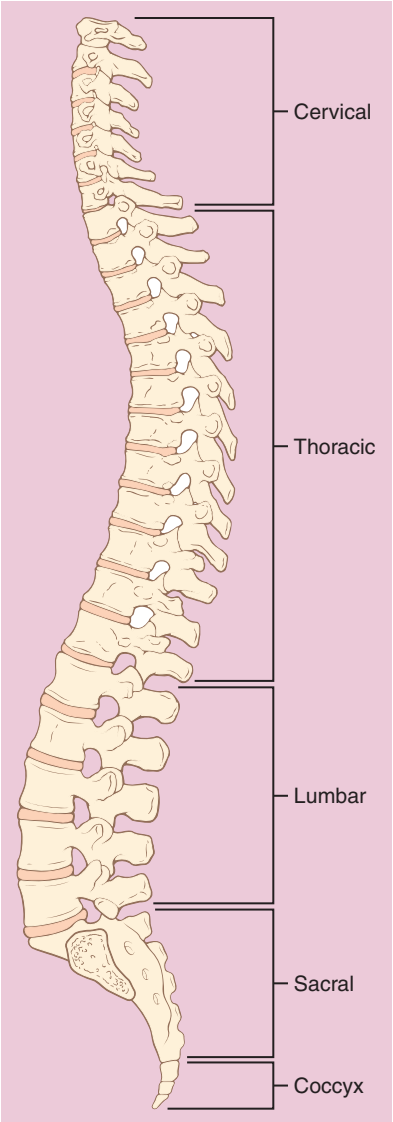

Figure 17-1 The vertebral column from a lateral view exhibits four curvatures. (From Covino BG, Scott DB, Lambert DH. Handbook of Spinal Anaesthesia and Analgesia. Philadelphia, WB Saunders, 1994, pp 12-24.)

The features of the midthoracic and lumbar vertebrae can ideally be represented by two articulated vertebrae (Fig. 17-5).[5] The nearly perpendicular orientation of the spinous process in the lumbar area and the downward angular orientation in the thoracic area define the angle required for placement and advancement of a needle intended to access the vertebral canal. The wide interlaminar space in the lumbar spine reflects the fact that the lamina occupies only about half the space between adjacent vertebrae. In contrast, the interlaminar space is just a few millimeters wide at the level of the thoracic vertebrae.

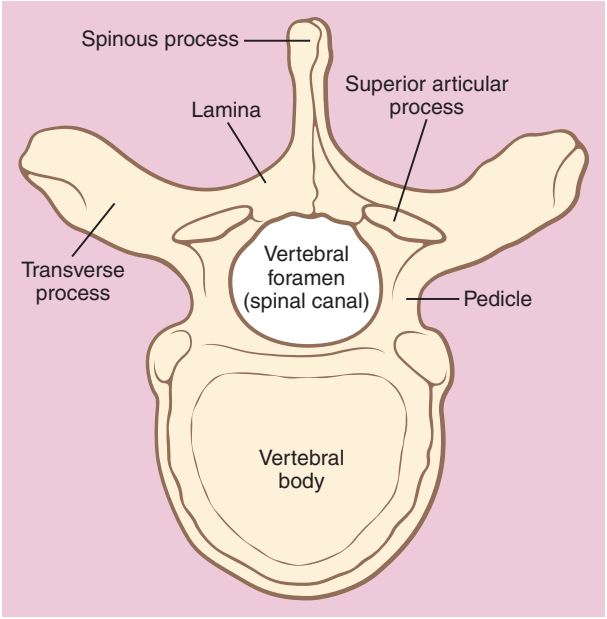

Figure 17-2 Typical thoracic vertebra. (From Covino BG, Scott DB, Lambert DH. Handbook of Spinal Anaesthesia and Analgesia. Philadelphia, WB Saunders, 1994.)

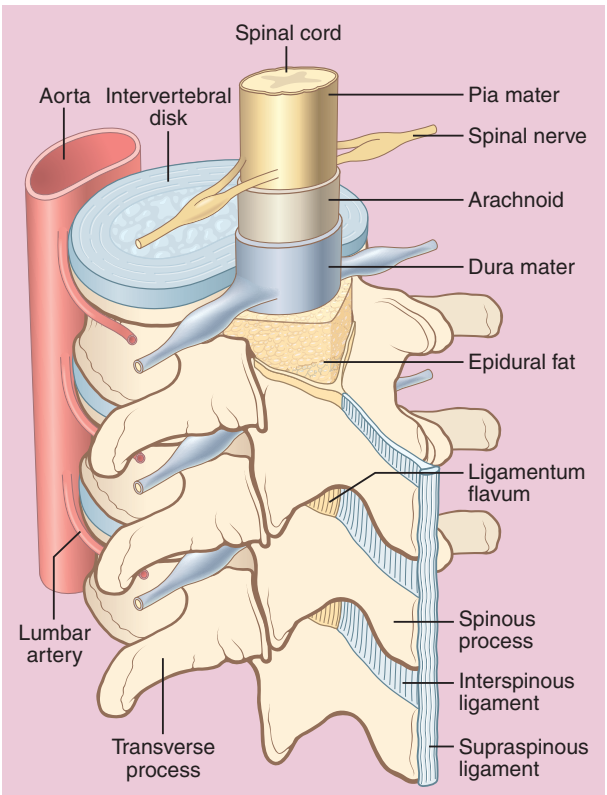

Figure 17-3 The spine in an oblique view. (From Afton-Bird G. Atlas of regional anesthesia. In Miller RD [ed]. Miller's Anesthesia. Philadelphia, Elsevier, 2005.)

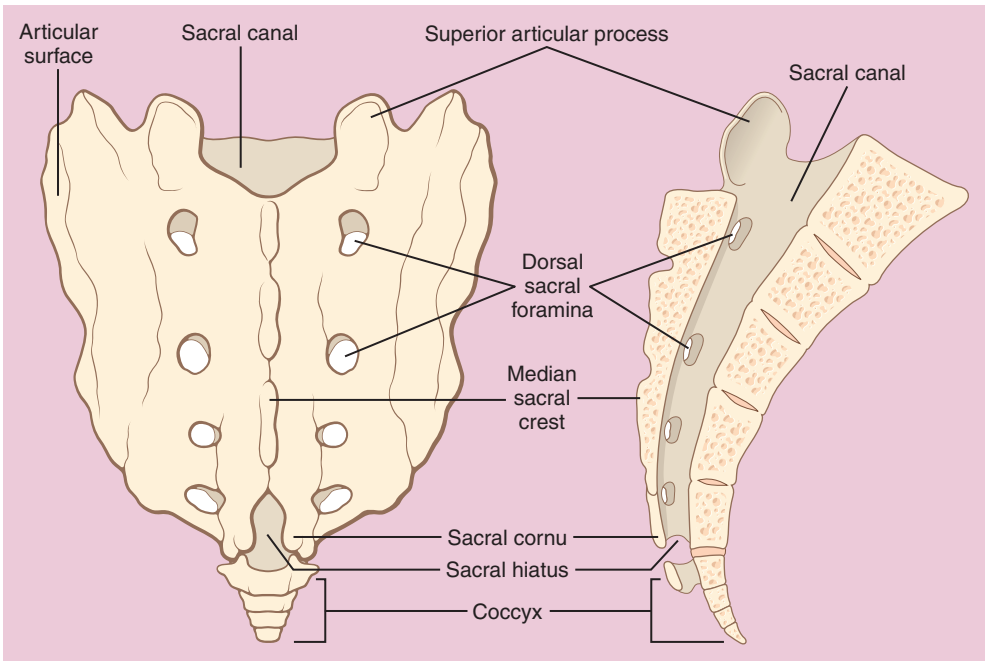

Figure 17-4 The sacrum in lateral and posterior view. (From Brown DL [ed]. Atlas of Regional Anesthesia. Philadelphia, WB Saunders, 1992.)

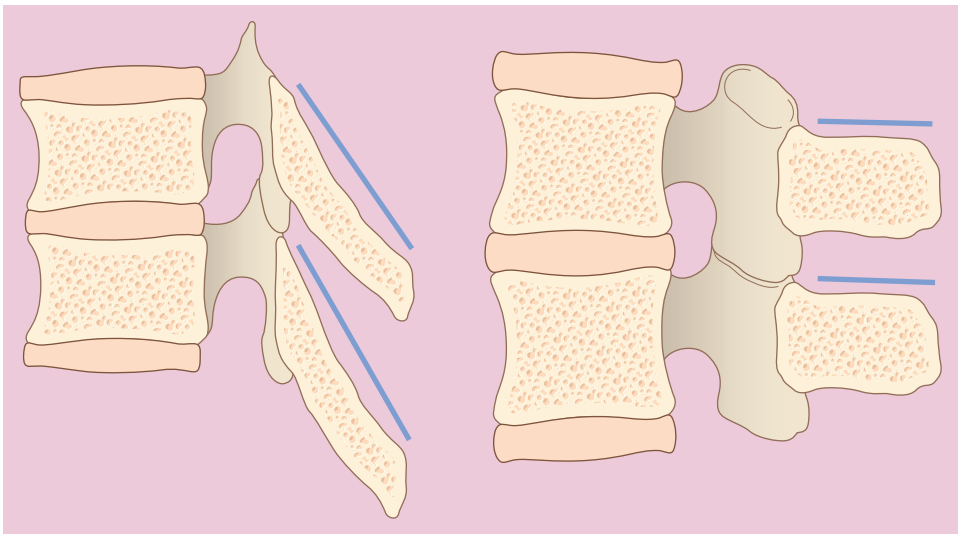

Figure 17-5 Lateral view of the thoracic and lumbar vertebrae. Note the sharp downward angulation of the thoracic spinous processes versus the nearly perpendicular angle that they assume in the lumbar vertebrae. (From Kardish K. Functional anatomy of central blockade in obstetrics. In Birnbach DJ, Gatt SP, Datta S [eds]. Textbook of Obstetric Anesthesia. Philadelphia, Churchill Livingstone, 2000, pp 121-156.)

SACRUM AND SACRAL HIATUS

The sacrum is a large curved wedge-shaped bone whose dorsal surface is convex and gives rise to the powerful sacrospinalis muscle. The opening between the unfused lamina of the fourth and fifth sacral vertebrae is called the sacral hiatus. There is considerable anatomic variability in the features of the dorsal surface of the sacrum. Indeed, the sacral hiatus is absent in nearly 8% of adult subjects, thereby preventing entry through the sacrococcygeal ligament into the sacral canal and performance of caudal anesthesia.

SURFACE LANDMARKS

Surface landmarks are used to identify specific spinal interspaces (Fig. 17-6).[4] The most important of these landmarks is a line drawn between the iliac crests. This line generally traverses the body of the L4 vertebra and is the principal landmark used to determine the level for insertion of a needle intended to produce spinal anesthesia. The C7 spinous process can be appreciated as a bony knob at the lower end of the neck. The T7-T8 interspace is identified by a line drawn between the lower limits of the scapulae and is often used to guide needle

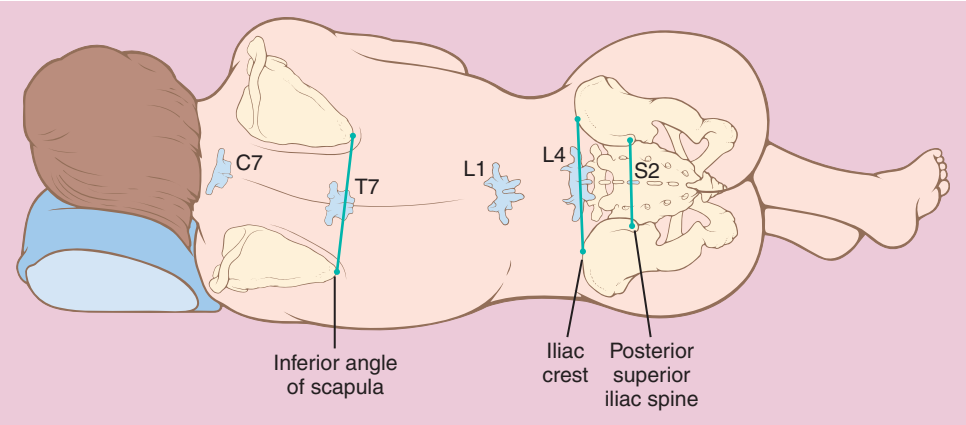

Figure 17-6 Surface landmarks are a guide to the vertebral level. (From Brown DL [ed]. Atlas of Regional Anesthesia. Philadelphia, WB Saunders, 1992.)

placement for passage of a catheter into the thoracic epidural space. The terminal portion of the twelfth rib intersects the L2 vertebral body, whereas the posterior iliac spines indicate the level of the S2 vertebral body, which is the most common caudal limit of the dural sac in adults. Other interspaces are identified by counting up or down along the spinous processes from these major landmarks.

Ligaments

The vertebral column is stabilized by several ligaments (Figs. 17-7 and 17-8).[2] Adjacent vertebral bodies are joined by anterior and posterior spinal ligaments, the latter forming the anterior border of the vertebral canal. The ligamentum flavum is composed of thick plates of elastic tissue that connect the lamina of adjacent vertebrae. The supraspinous ligament runs superficially along the spinous processes, which makes it the first ligament that a needle will traverse when using a midline approach to the vertebral canal.

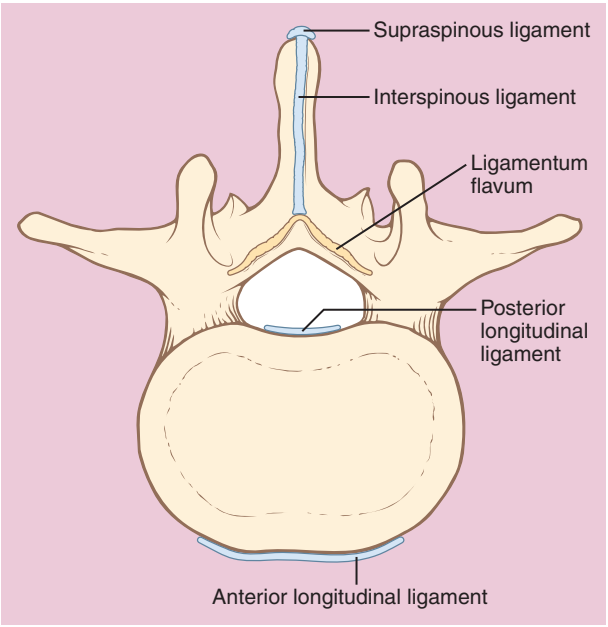

Figure 17-7 Cross section of a lumbar vertebra showing the attachment of the spinal ligaments. (From Covino BG, Scott DB, Lambert DH. Handbook of Spinal Anaesthesia and Analgesia. Philadelphia, WB Saunders, 1994, p 15.)

Spinal Cord

The spinal cord begins at the rostral border of the medulla and, in the fetus, extends the entire length of the vertebral canal. However, because of disproportionate growth of neural tissue and the vertebral canal, the spinal cord generally terminates around the third lumbar vertebra at birth and at the lower border of the first lumbar vertebra in adults. As a further consequence of this differential growth, the spinal nerves become progressively longer and more closely aligned with the longitudinal axis of the vertebral canal. Below the conus, the roots are oriented parallel to this axis and resemble a horse's tail, from which the name cauda equina is derived (Fig. 17-9).[6] The nerve roots of the cauda equina move relatively freely within the CSF, a fortunate arrangement that permits them to be displaced rather than pierced by an advancing needle.

Meninges

In addition to the CSF, the spinal cord is surrounded and protected by three layers of connective tissue known as the meninges.

DURA MATER

The outermost layer, the dura mater, originates at the foramen magnum as an extension of the inner (meningeal) layer of cranial dura and continues caudally to

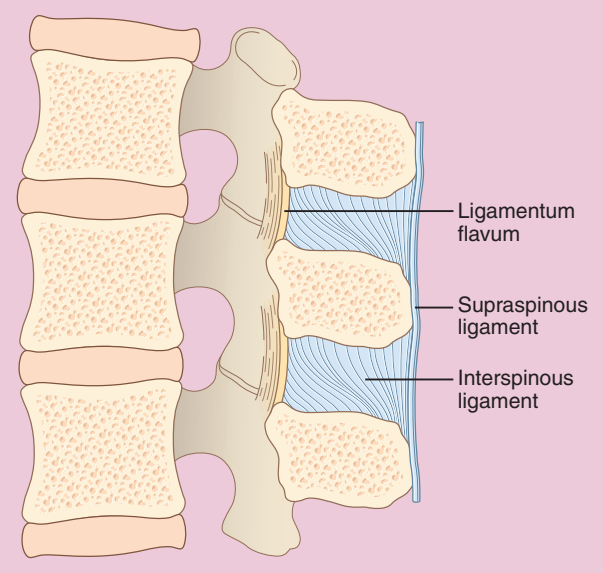

Figure 17-8 Sagittal section through adjacent lumbar vertebrae showing the attachment of the spinal ligaments. (From Covino BG, Scott DB, Lambert DH. Handbook of Spinal Anaesthesia and Analgesia. Philadelphia, WB Saunders, 1994, p 15.)

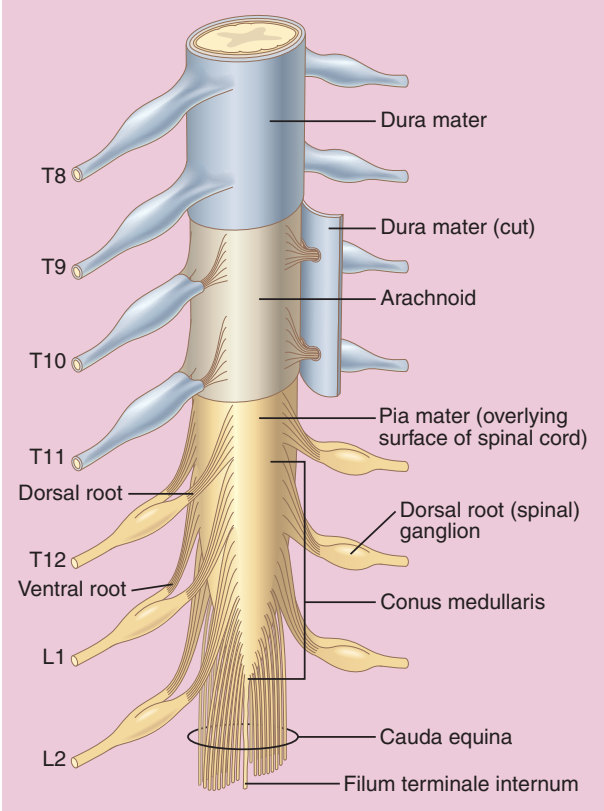

Figure 17-9 Terminal spinal cord and cauda equina. (From Bridenbaugh PO, Greene NM, Brull SJ. Spinal [subarachnoid] blockade. In Cousins MJ, Bridenbaugh PO [eds]. Neural Blockade in Clinical Anesthesia and Management of Pain. Philadelphia, Lippincott-Raven, 1998, pp 203-242.)

terminate between S1 and S4. It is a tough fibroelastic membrane that provides structural support and a fairly impenetrable barrier that normally prevents displacement of an epidural catheter into the fluid-filled subarachnoid space. Although cases of epidural catheter migration into the subarachnoid space occur clinically, it has been well established in cadaver studies that catheters cannot penetrate an intact dura.

ARACHNOID MEMBRANE

Closely adherent to the inner surface of the dura lies the arachnoid membrane. Though far more delicate than the dura, the arachnoid serves as the major pharmacologic barrier preventing movement of drug from the epidural to the subarachnoid space. Conceptually, the dura provides support and the arachnoid membrane imparts impermeability. Because the dura and arachnoid are closely adherent, a spinal needle that penetrates the dura will generally pass through the arachnoid membrane. However, "subdural" injections can occur in clinical practice and result in a "failed spinal" because of the relative impermeability of the arachnoid membrane.

PIA

The innermost layer of the spinal meninges, the pia is a highly vascular structure closely applied to the cord that forms the inner border of the subarachnoid space. Along the lateral surface between the dorsal and ventral roots, an extension of this membrane forms the denticulate ligament—a dense serrated longitudinal projection that provides lateral suspension through its attachment to the dura. As the spinal cord tapers to form the conus medullaris, the pia continues interiorly as a thin filament, the filum terminale. Distally, the filum terminale becomes enveloped by the dura at the caudal termination of the dural sac (generally around S2) and continues inferiorly to attach to the posterior wall of the coccyx.

Spinal Nerves

Along the dorsolateral and ventrolateral aspect of the spinal cord, rootlets emerge and coalesce to form the dorsal (afferent) and ventral (efferent) spinal nerve roots (Fig. 17-10).[2] Distal to the dorsal root ganglion, these nerve roots merge to form 31 pairs of spinal nerves (8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal). Because the sensory fibers traverse the posterior aspect of the subarachnoid space, they tend to lie dependent in a supine patient, thus making them particularly

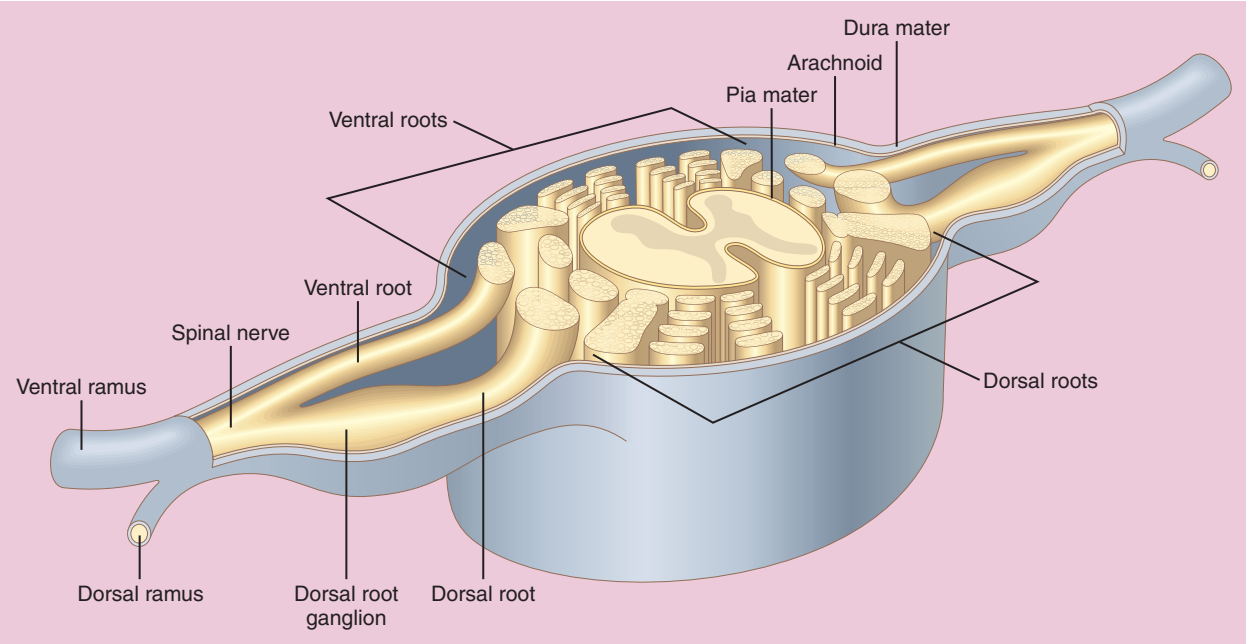

Figure 17-10 The spinal cord and nerve roots. (From Covino BG, Scott DB, Lambert DH. Handbook of Spinal Anaesthesia and Analgesia. Philadelphia, WB Saunders, 1994, p 19.)

vulnerable to hyperbaric (heavier than CSF) solutions containing local anesthetic.

As the nerves pass through the intervertebral foramen, they become encased by the dura, arachnoid, and pia, which form the epineurium, perineurium, and endoneurium, respectively. The dura becomes thinned as it traverses this area (often called the dural sleeve), thereby facilitating penetration of local anesthetic. The onset of epidural block by local anesthetics thus occurs by blockade of sodium ion conductance in this region. With time, epidural local anesthetics transfer into the subarachnoid space, and the nerve roots and spinal cord tracts are variably affected. This accounts for the observation that the onset of an epidural block spreads rostrally and caudally from the point of injection, but the pattern of recession is not a strict reversal of the onset.

PREGANGLIONIC SYMPATHETIC NERVE FIBERS

Preganglionic sympathetic nerve fibers originating in the intermediolateral gray columns of the thoracolumbar cord leave with the ventral nerve roots passing into the spinal nerve trunks (Fig. 17-11). They then leave the nerve via the white rami communicates and project to the paravertebral sympathetic ganglia or more distant sites (adrenal medulla, mesenteric and celiac plexus). After a cholinergic synapse (nicotinic) in the autonomic ganglia, the postsynaptic sympathetic nerve fibers join the spinal nerves via the gray rami communicantes and innervate diverse adrenergic effector sites.

CERVICAL NERVES

The first cervical nerve passes between the occipital bone and the posterior arch of the first cervical vertebra (atlas), and this relationship continues, with the seventh cervical nerve passing above the seventh cervical vertebra. However, because there are eight cervical nerves but only seven cervical vertebrae, the eighth cervical nerve passes between the seventh cervical vertebra and the first thoracic vertebra. Below this point, each spinal nerve passes through the inferior notch of the corresponding vertebra. For example, the T1 spinal nerve passes through the notch formed by the first and second thoracic vertebrae.

DERMATOME

The area of skin innervated by each spinal nerve is called a dermatome (Fig. 17-12).[7] Because the lower nerve roots descend before exiting the intervertebral foramen, the spinal cord terminations of the afferent fibers from each dermatome are more rostral than their corresponding vertebral level. For example, the sensory fibers from the L4 dermatome enter the spinal canal below the L4 vertebral body. However, primary afferent terminals for the L4 dermatome are located anterior to the T11-T12 interspace.

Subarachnoid Space

Between the arachnoid and the pia lies the subarachnoid space, which contains the CSF formed mainly by the choroid plexus of the lateral, third, and fourth ventricles.

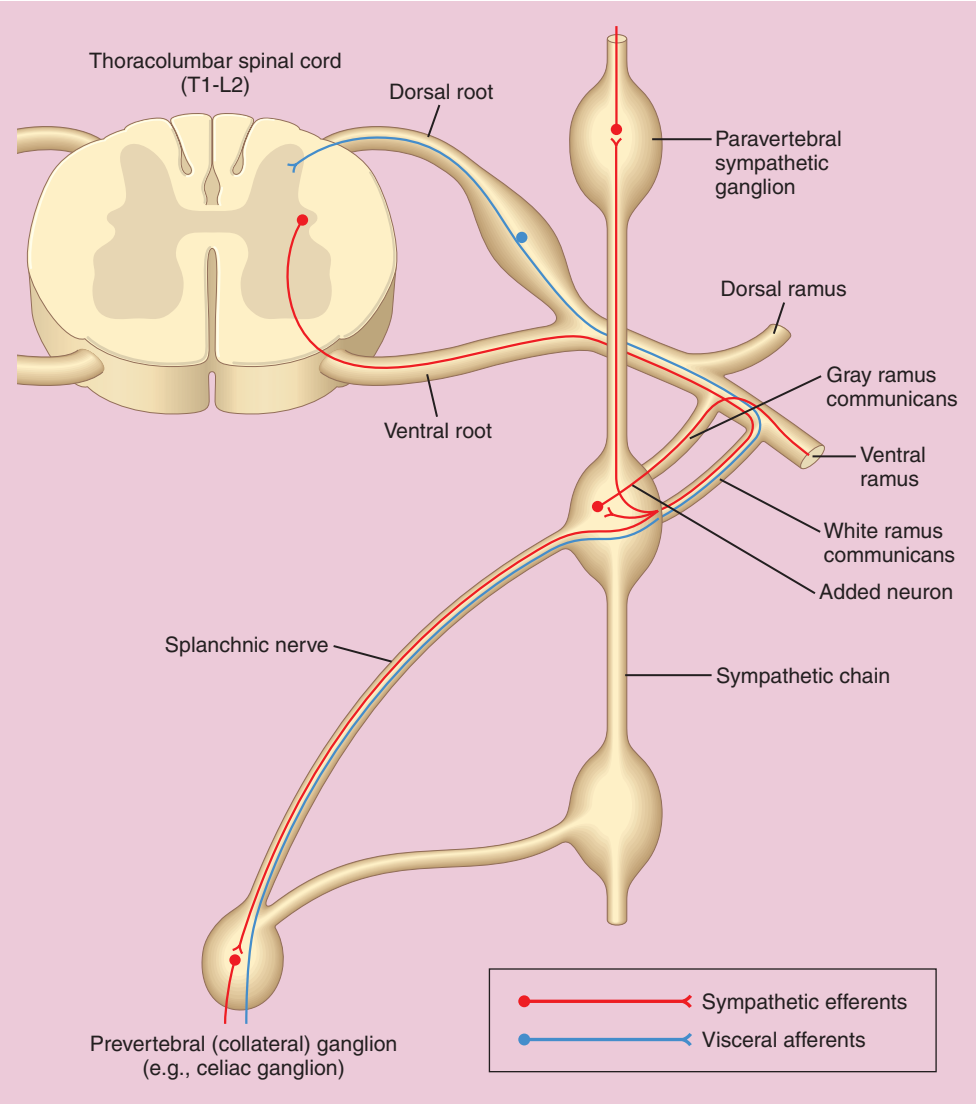

Figure 17-11 Cell bodies in the thoracolumbar portion of the spinal cord (T1-L2) give rise to the peripheral sympathetic nervous system. Efferent fibers travel in the ventral root and then via the white ramus communicans to paravertebral sympathetic ganglia or more distant sites such as the celiac ganglion. Afferent fibers travel via the white ramus communicans to join somatic nerves, which pass through the dorsal root to the spinal cord.

Because the spinal and cranial arachnoid spaces are continuous, cranial nerves can be blocked by local anesthetics migrating into the CSF above the foramen magnum.

Epidural Space

The epidural space lies between the dura and the wall of the vertebral canal, an irregular column of fat, lymphatics, and blood vessels. It is bounded cranially by the foramen magnum, caudally by the sacrococcygeal ligament, anteriorly by the posterior longitudinal ligament, laterally by the vertebral pedicles, and posteriorly by both the ligamentum flavum and vertebral lamina. The epidural space is not a closed space but communicates with the paravertebral spaces by way of the intervertebral foramina. The depth of the epidural space is maximal (about 6 mm) in the midline at L2 and is 4 to 5 mm in the midthoracic region. It is minimal where the lumbar and cervical enlargements of the spinal cord (T9-T12 and C3-T2, respectively) encroach on the epidural space, with roughly 3 mm left between the ligamentum flavum and the dura. There are subcompartments in the epidural space at each vertebral level, but injected fluid generally communicates freely throughout the space from the rostral limit at the foramen magnum to the sacral hiatus caudally. There is controversy regarding the existence and clinical significance of a connective tissue band (plica mediana dorsalis) extending from the dura mater to the ligamentum flavum and hence dividing the posterior epidural space into two compartments. Anatomic studies have suggested the presence of this structure and have led to the speculation that this tissue band may be

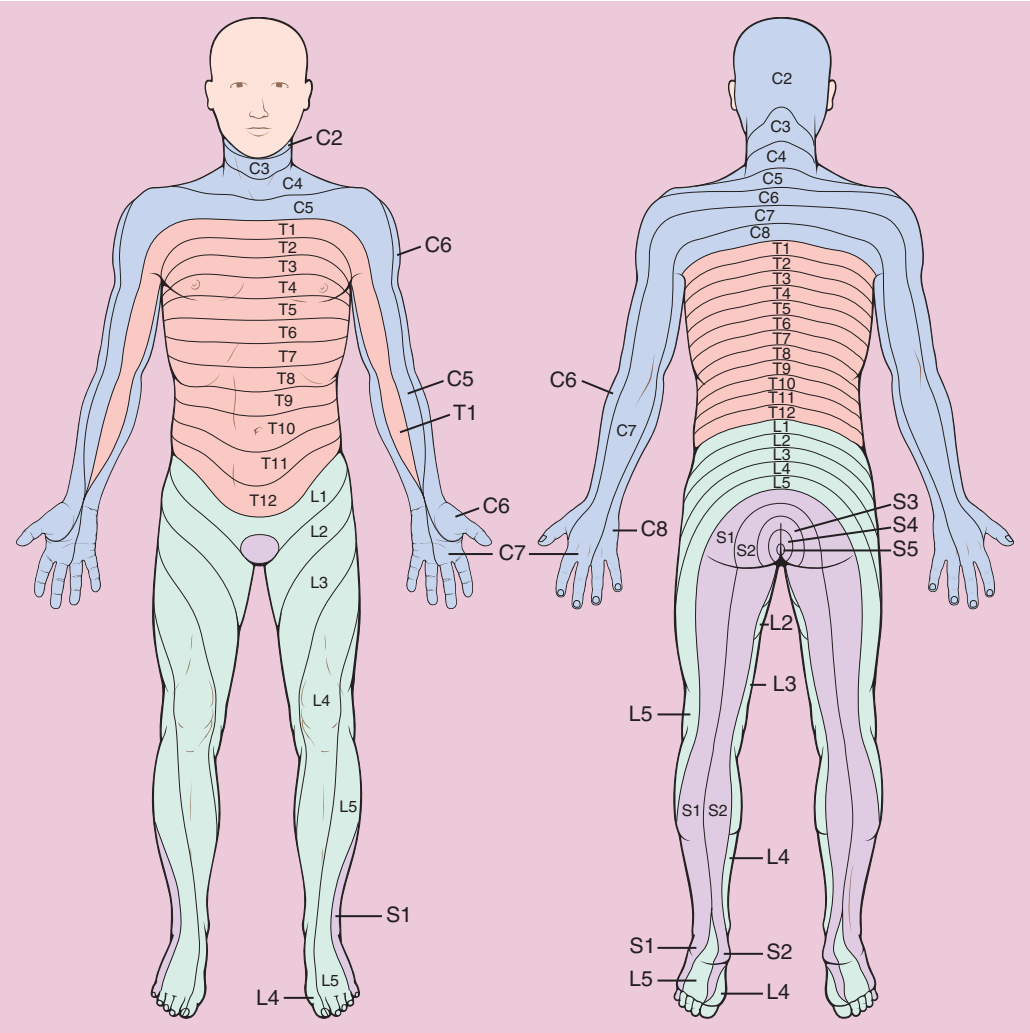

Figure 17-12 Areas of sensory innervation by spinal nerves. Note that the thoracic nerves innervate the thorax and abdomen and the lumbar and sacral nerves innervate the leg. (Modified from Veering BT, Cousins MJ. Epidural neural blockade. In Cousins MJ, Bridenbaugh PO, Carr DB, Horlocker TT [eds]. Neural Blockade in Clinical Anesthesia and Management of Pain. Philadelphia, Lippincott-Raven, 2009, pp 241-295.)

responsible for the occasional difficulty threading a catheter into the epidural space or the unexplained occurrence of a unilateral sensory block. Nevertheless, others are unable to confirm the presence of this structure.[8]

Blood Vessels

ARTERIAL

The blood supply of the spinal cord arises from a single anterior and two paired posterior spinal arteries (Fig. 17-13).[2] The posterior spinal arteries emerge from the cranial vault and supply the dorsal (sensory) portion of the spinal cord. Because they are paired and have rich collateral anastomotic links from the subclavian and intercostal arteries, this area of the spinal cord is relatively protected from ischemic damage. This is not the case with the single anterior spinal artery that originates from the vertebral artery and supplies the ventral (motor) portion of the spinal cord. The anterior spinal artery receives branches from the intercostal and iliac arteries, but these branches are variable in number and location. The largest anastomotic link, the radicularis magna (artery of Adamkiewicz), arises from the aorta in the lower thoracic or upper lumbar region.

Artery of Adamkiewicz

The vessel is highly variable but, most commonly, is on the left and enters the vertebral canal through the L1 intervertebral foramen. The artery of Adamkiewicz is

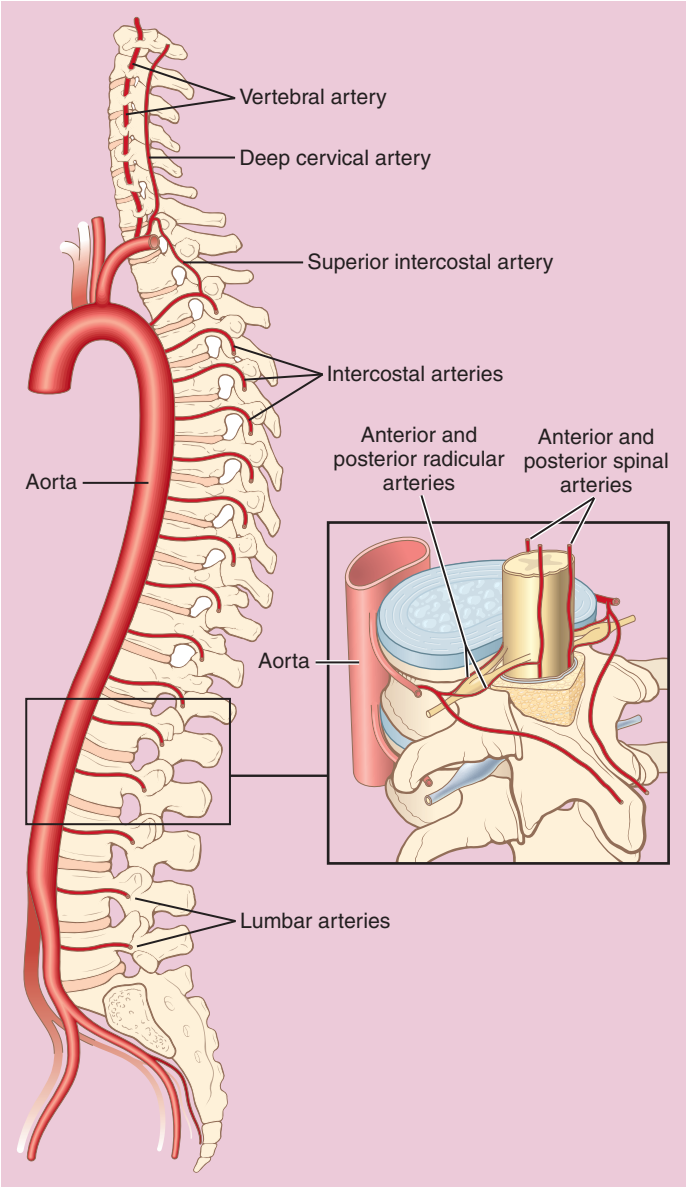

Figure 17-13 Arterial blood supply to the spinal cord. (Modified from Covino BG, Scott DB, Lambert DH. Handbook of Spinal Anaesthesia and Analgesia. Philadelphia, WB Saunders 1994, p 24.)

critical to the blood supply of the lower two thirds of the spinal cord, and damage to this artery during surgery on the aorta (aortic aneurysm resection) or by a stray epidural needle will produce characteristic bilateral lower extremity motor loss (anterior spinal artery syndrome).

VENOUS

The internal vertebral venous plexus drains the contents of the vertebral canal. These veins are prominent in the lateral epidural space and ultimately empty into the azygos venous system. The azygos vein enters the chest, arches over the right lung, and then empties into the superior vena cava. The internal vertebral venous plexus communicates above with the basilar sinuses of the brain and below with the pelvic connections to the inferior vena cava.

The anatomy of the venous plexus assumes additional importance in patients with increased intra-abdominal pressure or those with tumors or masses that compress the vena cava. In these patients, blood is diverted from the inferior vena cava and engorges veins in the epidural space, which increases the likelihood of accidental vascular cannulation during attempted epidural anesthesia. In addition, because the vertebral veins are enlarged, the effective volume of the epidural space is reduced, thereby resulting in greater longitudinal spread of injected local anesthetic solutions.

PREOPERATIVE PREPARATION

Preoperative preparation for regional anesthesia does not differ from that for general anesthesia (see Chapter 13). However, as with any regional anesthetic, a discussion with the patient regarding the specific benefits and potential complications should precede the block. Relevant complications include (1) those that are rare but serious, including nerve damage, bleeding, and infection, and (2) those that are common but of relatively minor consequence, such as post–dural puncture headache. There are no common serious complications (if there were, these techniques would not be used in clinical practice), and the infrequent minor problems are not of sufficient concern to warrant specific discussion. The possibility of a failed block should be discussed, and the patient should be reassured that in such circumstances, alternative anesthetic techniques will be provided to ensure their comfort.

Indications for Spinal Anesthesia

Spinal anesthesia is generally used for surgical procedures involving the lower abdominal area, perineum, and lower extremities. Although the technique can also be used for upper abdominal surgery, most consider it preferable to administer a general anesthetic to ensure patient comfort. In addition, the extensive block required for upper abdominal surgery and the nature of these procedures may have a negative impact on ventilation and oxygenation.

Indications for Epidural Anesthesia

Epidural anesthesia, like spinal anesthesia, is often used as the primary anesthetic for surgeries involving the abdomen or lower extremities. However, because of its segmental nature, anesthesia provided by lumbar epidural anesthesia may be suboptimal for procedures involving the lower sacral roots. Epidural anesthesia is also frequently used as a supplement to general anesthesia, particularly for thoracic and upper abdominal procedures. In such cases, significant benefit derives from the ability to provide continuous epidural anesthesia postoperatively to facilitate effective treatment of postoperative pain, and numerous studies confirm the superiority of epidural techniques compared to parenteral opioids.[9,10] Similarly, continuous epidural anesthesia is very effective and widely used for the control of labor pain. Cervical epidural anesthesia is very rarely used for operative surgery, but injections of solutions of corticosteroids and local anesthetics into the cervical epidural space are sometimes used to treat chronic pain.

Absolute and Relative Contraindications to Neuraxial Anesthesia

Absolute contraindications to neuraxial anesthetic techniques include patient refusal, infection at the site of planned needle puncture, elevated intracranial pressure, and bleeding diathesis. Patients should never be encouraged against their wishes to accept a regional anesthetic technique.

INFECTION

A Practice Advisory published by a task force of the American Society of Anesthesiologists addresses the issues related to infectious complications associated with neuraxial techniques, and can be reviewed for guidance.[11] Importantly, bacteremia does not necessarily mitigate against performance of a regional anesthetic technique. Although concern that an epidural abscess or meningitis might result from the introduction of infected blood during the procedure, clinical experience suggests that the risk is small and can be weighed against the potential benefit. In such cases, there is evidence to suggest that institution of appropriate antibiotic therapy before the block may decrease the risk for infection.[11]

PREEXISTING NEUROLOGIC DISEASE

The significance of any preexisting neurologic disease should be considered relative to its underlying pathophysiology. For example, patients with multiple sclerosis experience exacerbations and remissions of symptoms reflecting demyelination of peripheral nerves. Local anesthetic toxicity, when it occurs, can be associated with similar histopathology.[12] Although neuraxial anesthetic techniques have been viewed as acceptable for patients with multiple sclerosis, in the absence of compelling benefit, neuraxial techniques would be best avoided in these patients.

Chronic back pain does not represent a contraindication to neuraxial anesthetic techniques, although they may be avoided because patients may perceive a relationship between postoperative exacerbation of pain and the block, even though they are not causally related.

CARDIAC DISEASE

Patients with mitral stenosis, idiopathic hypertrophic subaortic stenosis, and aortic stenosis are intolerant of acute decreases in systemic vascular resistance. Thus, though not a contraindication, neuraxial block should be used cautiously in such cases.

ABNORMAL COAGULATION

The decision to use a neuraxial block in patients with abnormal coagulation, either endogenous or produced by the administration of anticoagulants, must be based on a risk-benefit assessment and include discussion with the patient and the surgical team. Guidelines developed

by the American Society of Regional Anesthesia and Pain Medicine (www.asra.com) are updated periodically based on evolving literature and changes in clinical practice, and can thus provide valuable guidance in the management of these patients.

SPINAL ANESTHESIA

An intravenous infusion is started before performance of the anesthetic, and all of the equipment, drugs, and monitors normally present for a general anesthetic are also required for neuraxial anesthesia. Supplemental oxygen is commonly administered. Although accurate end-tidal carbon dioxide monitoring may not always be feasible, a capnograph is often used to monitor breathing. This can be accomplished with specially designed nasal cannulae, or equipment can be easily improvised to permit sampling from nasal cannulae or face masks.

To decrease the discomfort associated with needle insertions, inclusion of an opioid in the preoperative medication should be considered. However, in selected patients premedication can be withheld, provided that there is adequate attention to infiltration of the skin and subcutaneous tissues with local anesthetic solution.

Sterile technique with hat, mask, and gloves is mandatory, and in modern practice, the required equipment is obtained from prepackaged sterile kits. Antiseptic preparation of the skin is performed, but contact with gloves and needles should be avoided because of the potential neurotoxicity of these antiseptic solutions.

Patient Positioning (Also See Chapter 19)

Spinal anesthesia can be performed with the patient in the lateral decubitus, sitting, or less commonly, the prone position. To the extent possible, the spine should be flexed by having the patient bend at the waist and bring the chin toward the chest, which will optimize the interspinous space and the interlaminar foramen.

LATERAL POSITION

The lateral decubitus position is more comfortable and more suitable for the ill or frail. It also enables the anesthesia provider to safely provide greater levels of sedation.

SITTING POSITION

The sitting position encourages flexion and facilitates recognition of the midline, which may be of increased importance in an obese patient. Because lumbar CSF is elevated in this position, the dural sac is distended, thus providing a larger target for the spinal needle. This higher pressure also facilitates recognition of the needle tip within the subarachnoid space, as heralded by the free flow of CSF. When combined with a hyperbaric anesthetic, the sitting position favors a caudal distribution, the resultant anesthesia commonly being referred to as a "saddle block." However, in addition to being poorly suited for a heavily sedated patient, vasovagal syncope can occur.

PRONE POSITION

The prone position is rarely used except for perineal procedures performed in the "jackknife" position. Performance of spinal anesthesia in this position is more challenging because of the limited flexion, the contracted dural sac, and the low CSF pressure, which generally requires aspiration with the plunger of the syringe to achieve backflow of CSF through the spinal needle.

Selection of Interspace

Several factors influence the selection of the interspace to be used for spinal anesthesia. The most obvious is the specific anatomy of the patient's spine and the likelihood that a needle can be successfully passed into the subarachnoid space. A second and often underappreciated consideration is that the interspace selected for spinal anesthesia has considerable impact on the distribution of anesthetic within the subarachnoid space. This, in turn, will affect the success or failure of the technique. For example, the likelihood of a "failed spinal" increases as interspaces that are more caudal are used, with up to a 7% incidence occurring when the L4-L5 interspace is selected.[13]

Although more rostral interspaces are associated with higher success rates, this benefit must be balanced against the potential for traumatic injury to the spinal cord, keeping in mind that the caudal limitation of the spinal cord in an adult usually lies between the L1 and L2 vertebrae. For this reason, spinal anesthesia is not ordinarily performed above the L2-L3 interspace. Nevertheless, some risk remains because the spinal cord extends to the third lumbar vertebra in approximately 2% of adults. Furthermore, the use of a conceptual line across the iliac crests to identify the body of the L4 vertebra often results in selection of an interspace that is one or more levels higher than believed.[14]

Spinal Needles

A variety of needles are available for spinal anesthesia and are generally classified by their size (most commonly 22 to 25 gauge) and the shape of their tip (Fig. 17-14).[15] The two basic designs of spinal needles are (1) an open-ended (beveled or cutting) needle and (2) a closed tapered-tip pencil-point needle with a side port. The incidence of post–dural puncture headache varies directly with the size of the needle. The pressure is lower when a pencil-point (Whitacre or Sprotte) rather than a beveled-tip (Quincke) needle is used. Consequently, a 24- or 25-gauge pencil-point needle is usually selected when spinal anesthesia is performed on younger patients, in whom post–dural puncture headache is more likely to develop. The design of the

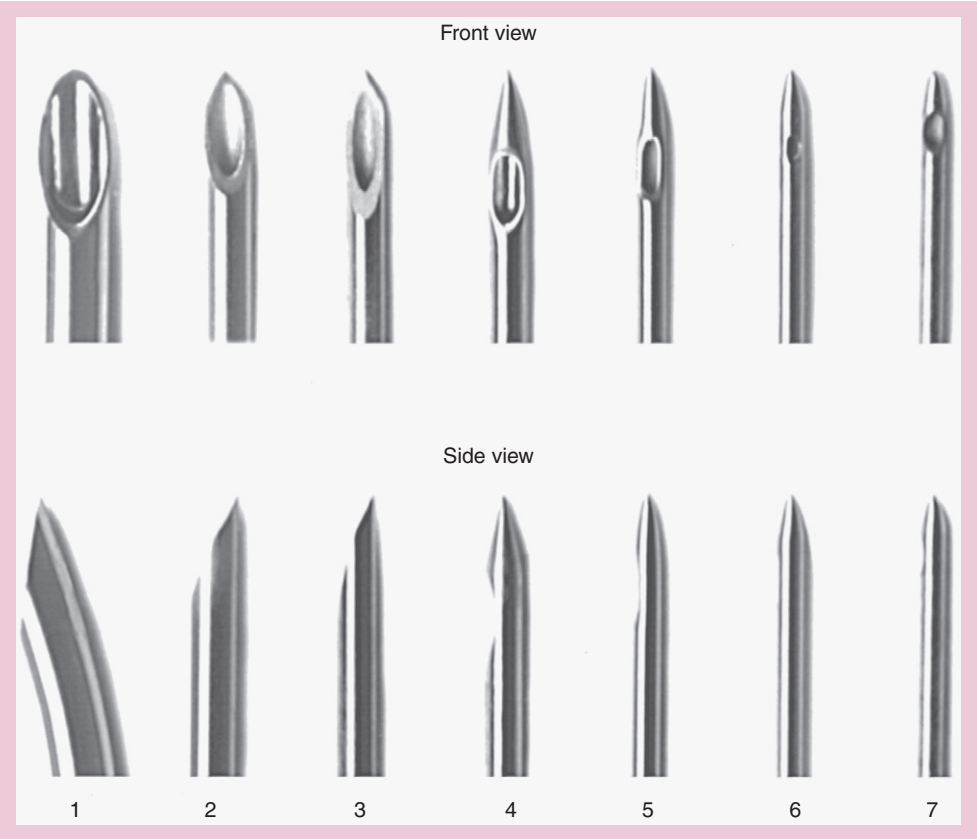

Figure 17-14 Comparative needle configuration for (1) 18-gauge Tuohy, (2) 20-gauge Quincke, (3) 22-gauge Quincke, (4) 24-gauge Sprotte, (5) 25-gauge Polymedic, (6) 25-gauge Whitacre, and (7) 26-gauge Gertie Marx. (From Schneider MC, Schmid M. Postdural puncture headache. In Birnbach DJ, Gatt SP, Datta S [eds]. Textbook of Obstetric Anesthesia. Philadelphia, Churchill Livingstone, 2000, pp 487-503.)

tip also affects the "feel" of the needle because a pencil-point needle requires more force to insert than a beveled-tip needle does but provides better tactile feel of the various tissues encountered as the needle is advanced.

Approach

Local anesthetic solution is infiltrated to anesthetize the skin and subcutaneous tissue at the anticipated site of cutaneous needle entry, which will be determined by the approach (midline or paramedian) to the subarachnoid space. The midline approach is technically easier, and the needle passes through less sensitive structures, thus requiring less local anesthetic infiltration to ensure patient comfort. However, the paramedian approach is better suited to challenging circumstances when there is narrowing of the interspace or difficulty in flexion of the spine. This can be readily appreciated by examination of a skeleton, which shows that the interlaminar space is largest when viewed from a slightly caudad and lateral vantage point.

MIDLINE TECHNIQUE

When using the midline approach, the needle is inserted at the top margin of the lower spinous process of the selected interspace. This point is generally easily identified by visual inspection and palpation. However, palpation of the spinous process and even identification of the midline become progressively more challenging with increasing obesity. In such circumstances, the patient should be as about perception of the needle to be midline or off to one side and adjust based on this feedback. After passage through the skin, the needle is progressively advanced with a slight cephalad orientation. Even in the lumbar area where the spinous processes are relatively straight, the interlaminar space is slightly cephalad to the interspinous space. Small needles tend to deflect or bend during insertion. Consequently, 24-gauge or smaller needles should be passed through a larger-gauge introducer needle placed in the interspinous ligament, which serves to guide and stabilize their path. This approach is particularly important when a beveled needle is used because the angle of the bevel displaces the needle from its path and causes it to veer in a direction opposite the bevel as it is being advanced.

As the spinal needle progresses toward the subarachnoid space, it passes through the skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, and the epidural space to reach and pierce the dura/arachnoid. The dural fibers appear to be largely oriented along the longitudinal axis of the

dural sac. Thus, orienting the bevel of a cutting needle parallel to this axis tends to spread rather than cut the fibers, which may reduce the risk for post–dural puncture headache.

PARAMEDIAN TECHNIQUE

The point of cutaneous needle insertion for the paramedian technique is typically 1 cm lateral to the midline but varies in the rostral-caudal plane according to the patient's anatomy and the anesthesia provider's preference. Success depends on an appreciation of the anatomy and appropriate angulation of the needle and not on the precise location of needle insertion. The most common error is to underestimate the distance to the subarachnoid space and direct the needle too medially, with resultant passage across the midline. With the paramedian technique, the needle bypasses the supraspinous and interspinous ligaments, and the ligamentum flavum will be the first resistance encountered.

TAYLOR APPROACH

The Taylor approach (first described by urologist John A. Taylor) describes the paramedian technique to access the L5-S1 interspace (Fig. 17-15). Though generally the widest interspace, it is often inaccessible from the midline because of the acute downward orientation of the L5 spinous process. The spinal needle is passed from a point 1 cm caudad and 1 cm medial to the posterior superior iliac spine and advanced cephalad at a 55-degree angle with a medial orientation based on the width of the sacrum. The Taylor approach is technically challenging but very useful because it is minimally dependent on patient flexion for successful passage of the needle into the subarachnoid space.

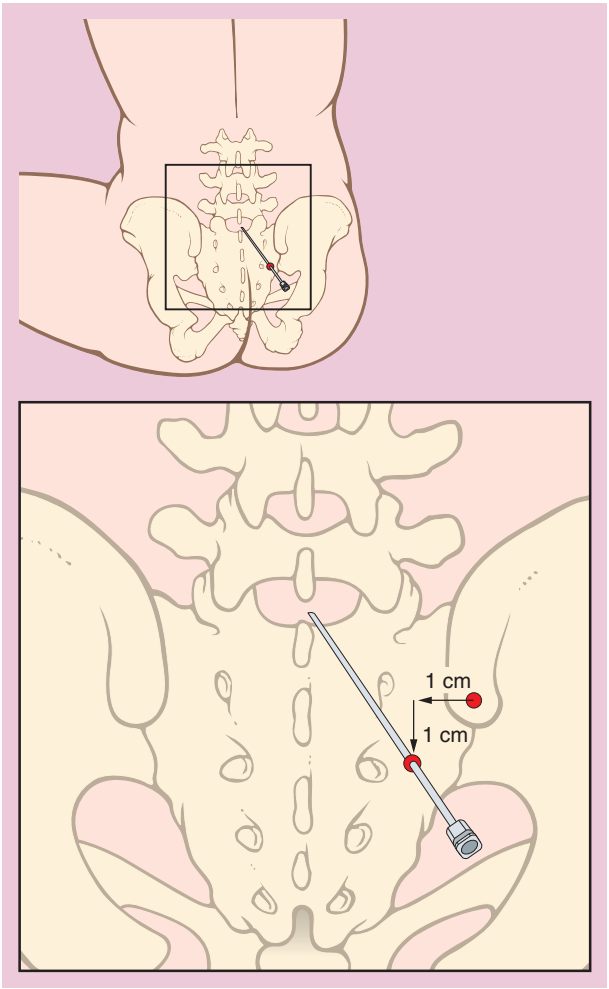

Figure 17-15 The L5-S1 paramedian (Taylor) approach. (From Brown DL [ed]. Atlas of Regional Anesthesia. Philadelphia, WB Saunders, 1992.)

Anesthetic Injection

After penetration of the dura by the spinal needle (can often be felt by the anesthesia provider's fingers as a rather distinct pop), the needle is further advanced a short distance to ensure that the bevel or side port rests entirely within the subarachnoid space. Free flow of CSF from the hub of the needle confirms correct placement of the distal end of the spinal needle. Occasionally, blood-tinged CSF initially appears at the hub of the needle. If clear CSF is subsequently seen, the spinal anesthetic can be completed. Conversely, if blood-tinged CSF continues to flow, the needle should be removed and reinserted at a different interspace. Should blood-tinged CSF still persist, the attempt to induce spinal anesthesia should be terminated. Similarly, spinal anesthesia should never be administered in the presence of a paresthesia. The occurrence of a paresthesia during needle placement mandates withdrawal of the needle.

If using a pencil-point needle, the side port can be positioned to encourage the desired distribution of local anesthetic solution within the subarachnoid space, which is generally cephalad. However, the orientation of the bevel of a cutting needle has no effect on the trajectory of the anesthetic stream emerging from the tip. The needle can be secured by holding the hub between the thumb and index finger, with the dorsum of the anesthesia provider's hand resting against the patient's back; the syringe is then attached to the needle, and CSF is again aspirated to reconfirm placement. One should ensure that CSF can be easily withdrawn and flows freely into the syringe. With the syringe firmly attached to the needle to prevent loss of local anesthetic solution, the contents of the syringe are delivered into the subarachnoid space over approximately a 3 to 5 seconds. Aspiration plus reinjection of a small quantity of CSF again at the conclusion of the injection confirms the needle position and verifies subarachnoid delivery of the local anesthetic

solution. Finally, the needle and syringe are withdrawn as a single unit and the antiseptic is wiped from the patient's back. The patient is then placed in a position that will encourage the desired distribution of local anesthetic solution or is positioned for surgery.

Level and Duration

The distribution of local anesthetic solution in CSF is influenced principally by (1) the baricity of the solution, (2) the contour of the spinal canal, and (3) the position of the patient in the first few minutes after injection of local anesthetic solution into the subarachnoid space. Assuming that an appropriate dose is selected, the duration of spinal anesthesia depends on the drug selected and the presence or absence of a vasoconstrictor (epinephrine or phenylephrine) in the local anesthetic solution (Table 17-1). During recovery, anesthesia regresses from the highest dermatome in a caudad direction.

BARICITY AND PATIENT POSITION

Local anesthetic solutions are classified as hypobaric, isobaric, and hyperbaric based on their density relative to the density of CSF. Baricity is an important consideration because it predicts the direction that local anesthetic solution will move after injection into CSF. Consequently, by selecting a local anesthetic solution of appropriate density relative to the position of the patient and the contour of the subarachnoid space, the anesthesia provider seeks to control both the direction and the extent of local anesthetic movement in the subarachnoid space and the resultant distribution of anesthesia.

Hyperbaric Solutions

The most commonly selected local anesthetic solutions for spinal anesthesia are hyperbaric (achieved by the addition of glucose [dextrose]), and their principal advantage is the ability to achieve greater cephalad spread of anesthesia. Commercially available hyperbaric local anesthetic solutions include 0.75% bupivacaine with 8.25% glucose and 5% lidocaine with 7.5% glucose. Tetracaine is formulated as a 1% plain solution and is most often used as a 0.5% solution with 5% glucose, which is achieved by dilution of the anesthetic with an equal volume of 10% glucose. However, the drug is also available in an ampoule containing Niphanoid crystals (20 mg), which is generally mixed with 2 mL of sterile water to produce a 1% solution.

The contour of the vertebral canal is critical to the subarachnoid distribution of hyperbaric local anesthetic solutions. For example, in the supine horizontal position, the patient's thoracic and sacral kyphosis will be dependent relative to the peak created by the lumbar lordosis (see Fig. 17-1).[2] Anesthetic delivered cephalad to this peak will thus move toward the thoracic kyphosis, which is normally around T6-T8. Placing the patient in a head-down (Trendelenburg) position will further accentuate this cephalad spread of local anesthetic solution and help ensure an adequate level of spinal anesthesia for abdominal surgery.

Hyperbaric local anesthetic solutions can also be administered caudad to the lumbosacral peak to encourage restricted sacral anesthesia (referred to as a saddle block reflecting sensory anesthesia of the area that would be in contact with a saddle). The spinal is performed with the patient seated.

Sometimes the impact of the lumbosacral lordosis should be minimized. In such cases, a pillow can be placed under the patient's knees to flatten this curve. Even more effective, the patient can be maintained in the lateral position, which will effectively eliminate the influence of the lumbosacral curvature on the distribution of local anesthetic solution in the subarachnoid space. Movement of hyperbaric local anesthetic solution will now be directly influenced by the patient's position on the operating table (the Trendelenburg position promoting cephalad spread and the reverse Trendelenburg position encouraging a restricted block).

Hypobaric Solutions

Hypobaric local anesthetic solutions find limited use in clinical practice and are generally reserved for patients

Table 17-1 Local Anesthetics Used for Spinal Anesthesia

		Dose (mg)			Duration (min)	
Drug	**Concentration (%)**	***T10***	***T4***	***Time to Onset (min)***	***Plain***	***Epinephrine (0.2 mg)***
Lidocaine	5*	40-50	60-75	2-4	45-75	NR
Tetracaine	0.5	8-10	12-15	4-6	60-120	120-180
Bupivacaine	0.5-0.75	8-10	12-15	4-6	60-120	NR
Ropivacaine	0.5-0.75	10-14	15-20	4-6	60-90	NR
Chloroprocaine	2-3	40-50	60	2-4	30-60	NR

*Must be diluted to 2.5% or less before administration.
NR, not recommended.

undergoing perineal procedures in the "prone jackknife" position or undergoing hip arthroplasty where anesthetic can "float up" to the nondependent operative site. A common technique is to use a 0.1% solution (1 mg/mL) of tetracaine by diluting the commercial 10% solution with sterile water. However, although these solutions have been used in clinical practice for many years, they are extremely hypotonic, and alternative solutions prepared with third- or half-normal saline will still permit gravitational control but will present far less osmotic stress to neural tissue.

Isobaric Solutions

Isobaric local anesthetic solutions undergo limited spread in the subarachnoid space, which may be considered an advantage or disadvantage depending on the clinical circumstances. A potential advantage of isobaric local anesthetic solutions is a more profound motor block and more prolonged duration of action than that achieved with equivalent hyperbaric local anesthetic solutions. Because the distribution of local anesthetic solutions is not affected by gravity, spinal anesthesia can be performed without concern that the resultant block might be influenced by patient position. Commercially prepared "epidural" anesthetic solutions, which are generally formulated in saline, are commonly used for isobaric spinal anesthesia. However, although these solutions are considered isobaric, they actually behave clinically as though they were slightly hyperbaric, due to the effect of their low temperature relative to the cerebrospinal fluid.

Isobaric spinal anesthesia is particularly well suited for perineal or lower extremity procedures, as well as surgery involving the lower part of the trunk (hip arthroplasty, inguinal hernia repair). Although spread of local anesthetic solution may be limited caudally, subarachnoid injection does not produce segmental anesthesia because the nerves innervating more caudad structures are vulnerable to block as they pass through the region of high local anesthetic concentration.

Adjuvants

VASOCONSTRICTORS

Vasoconstrictors are frequently added to local anesthetic solutions to increase the duration of spinal anesthesia. This is most commonly achieved by the addition of epinephrine (0.1 to 0.2 mg, which is 0.1 to 0.2 mL of a 1:1000 solution) or phenylephrine (2 to 5 mg, which is 0.2 to 0.5 mL of a 1% solution). Increased duration of spinal anesthesia probably results from a reduction in spinal cord blood flow, which decreases loss of local anesthetic from the perfused areas and thus increases the duration of exposure to local anesthetic. However, with epinephrine, its α_2-adrenergic analgesic activity may contribute to the anesthetic.

The effect of vasoconstrictors is not equivalent for all local anesthetics, with tetracaine-induced spinal anesthesia exhibiting the longest prolongation. The differences in duration are partly due to the differing effects of local anesthetics on spinal cord blood flow. Tetracaine produces intense vasodilatation; the effect of lidocaine is more modest, whereas bupivacaine actually decreases both spinal cord and dural blood flow.

The addition of vasoconstrictors to local anesthetic solutions containing lidocaine has been questioned because of reports of nerve injury attributed to spinal lidocaine, and epinephrine may increase lidocaine neurotoxicity.[16] Epinephrine has been associated with significant "flulike" side effects when coadministered with spinal chloroprocaine,[17] whereas adding epinephrine or phenylephrine to tetracaine is associated with an increased risk for transient neurologic symptoms.[18]

OPIOIDS AND OTHER ANALGESIC DRUGS

Opioids may be added to local anesthetic solutions to enhance surgical anesthesia and provide postoperative analgesia. This effect is mediated at the dorsal horn of the spinal cord, where opioids mimic the effect of endogenous enkephalins. Commonly, fentanyl (25 μg) is used for short surgical procedures, and its administration does not preclude discharge home on the same day. The use of morphine (0.1 to 0.5 mg) can provide effective control of postoperative pain for roughly 24 hours, but it necessitates in-hospital monitoring for respiratory depression. Clonidine, an α_2-adrenergic drug, is not as effective as opioids, and its addition to the local anesthetic solution augments the sympatholytic and hypotensive effects of the local anesthetics.[19]

Choice of Local Anesthetic

Although there are differences among the local anesthetics with respect to the relative intensity of sensory and motor block, selection is based largely on the duration of action and potential adverse side effects.

LIDOCAINE FOR SHORT-DURATION SPINAL ANESTHESIA

For decades, lidocaine was the most commonly used short-acting local anesthetic for spinal anesthesia. It has a duration of action of 60 to 90 minutes, and it produces excellent sensory anesthesia and a fairly profound motor block. These features, in conjunction with a favorable recovery profile, make lidocaine particularly well suited for brief surgical procedures, particularly in the ambulatory setting (also see Chapter 37).

Neurotoxicity

Unfortunately, despite a long history of apparent safe use, subsequent reports of major and minor complications associated with spinal lidocaine have tarnished its reputation

and jeopardize its continued clinical use. Initial reports of permanent neurologic deficits were restricted to its use for continuous spinal anesthesia, where extremely high doses were administered.[20] However, other reports suggest that injury may occur even with the administration of a dose historically recommended for single-injection spinal anesthesia.[21,22] These injuries have led to suggested modifications in practice that include a reduction in the lidocaine dose from 100 mg to 60 to 75 mg and dilution of the commercial formulation of 5% lidocaine with an equal volume of saline or CSF before subarachnoid injection.[22]

Transient Neurologic Symptoms

Lidocaine has been linked to the development of transient neurologic symptoms (pain or dysesthesia in the back, buttocks, and lower extremities) in as many as a third of patients receiving lidocaine for spinal anesthesia.[23] Factors that increase the risk for transient neurologic symptoms include patient positioning (lithotomy, knee arthroscopy) (see Chapter 19) and outpatient status (see Chapter 37).[18,24]

ALTERNATIVE LOCAL ANESTHETICS FOR SHORT-DURATION SPINAL ANESTHESIA

The etiology of transient neurologic symptoms is not established, but their occurrence has reinforced dissatisfaction with lidocaine and generated interest in alternative local anesthetics for short-duration spinal anesthesia. Mepivacaine probably has an incidence of transient neurologic symptoms less than lidocaine and may offer some benefit. Although procaine has a very short duration of action, the incidence of nausea is relatively frequent, and yet the incidence of transient neurologic symptoms is probably only marginally better.

Chloroprocaine

Chloroprocaine can be used as a spinal anesthetic.[25] Although this local anesthetic was linked to neurologic injuries in the 1980s, these injuries were due either to excessively large epidural doses that were accidentally injected into the subarachnoid space or to the preservative contained in the commercial anesthetic solution.[26] In any event, recent studies of low-dose (40 to 60 mg) preservative-free chloroprocaine suggest that chloroprocaine can produce excellent short-duration spinal anesthesia with little, if any, risk for transient neurologic symptoms.[17,27,28] The addition of epinephrine to chloroprocaine solutions for spinal anesthesia has side effects, and vasoconstrictors should not be used to prolong chloroprocaine spinal anesthesia. In contrast, both fentanyl and clonidine provide the expected enhancement of chloroprocaine spinal anesthesia without apparent side effects.

LONG-DURATION SPINAL ANESTHESIA

Bupivacaine and tetracaine are the local anesthetics most frequently used for long-duration spinal anesthesia. Although ropivacaine has been used as a spinal anesthetic, the advantages over bupivacaine are not obvious. Spinal bupivacaine is available as a 0.75% solution with 8.25% glucose for hyperbaric anesthesia. Tetracaine is prepared as 1% plain solution, which can be diluted with glucose, saline, or water to produce a hyperbaric, isobaric, or hypobaric solution, respectively. The recommended doses (5 to 20 mg) and durations of action (90 to 120 minutes) of bupivacaine and tetracaine are similar. However, bupivacaine produces slightly more intense sensory anesthesia (as evidenced by a less frequent incidence of tourniquet pain), whereas motor block with tetracaine is slightly more pronounced. The duration of tetracaine spinal anesthesia is more variable and more profoundly affected by the addition of a vasoconstrictor. Consequently, tetracaine remains the most useful spinal anesthetic in circumstances in which a prolonged block is sought. Unfortunately, the inclusion of a vasoconstrictor with tetracaine results in a significant incidence of transient neurologic symptoms.[18]

Documentation of Anesthesia

Within 30 to 60 seconds after subarachnoid injection of local anesthetic solutions, the developing level of spinal anesthesia should be determined. The desired level of spinal anesthesia is dependent on the type of surgery (Table 17-2). Nerve fibers that transmit cold sensation (C and A delta) are among the first to be blocked. Thus, an early indication of the level of a spinal anesthetic can be obtained by evaluating the patient's ability to discriminate temperature changes as produced by "wetting" the skin with an alcohol sponge. In the area blocked by the spinal anesthetic, the alcohol produces a warm or neutral sensation rather than the cold perceived in the unblocked areas. The level of sympathetic nervous system anesthesia usually exceeds the level of sensory block, which in turn exceeds the level of motor block. The level of sensory anesthesia is often evaluated by the patient's ability to discriminate sharpness as produced by a needle.

Table 17-2 Sensory Level Anesthesia Necessary for Surgical Procedures

Sensory Level	Type of Surgery
S2-S5	Hemorrhoidectomy
L2-L3 (knee)	Foot surgery
L1-L3 (inguinal ligament)	Lower extremity surgery
T10 (umbilicus)	Hip surgery Transurethral resection of the prostate Vaginal delivery
T6-T7 (xiphoid process)	Lower abdominal surgery Appendectomy
T4 (nipple)	Upper abdominal surgery Cesarean section

Table 17-3 Levels and Significance of Sensory Block

Cutaneous Level	Segmental Level	Significance
Fifth digit	C8	All cardioaccelerator fibers blocked
Inner aspect of the arm and forearm	T1-T2	Some degree of cardioaccelerator block
Apex of the axilla	T3	Easily determined landmark
Nipple	T4-T5	Possibility of cardioaccelerator block
Tip of the xiphoid	T7	Splanchnic fibers (T5-L1) may be blocked
Umbilicus	T10	Sympathetic nervous system block limited to the legs
Inguinal ligament	T12	No sympathetic nervous system block
Outer aspect of the foot	S1	Confirms block of the most difficult root to anesthetize

Skeletal muscle strength is tested by asking the patient to dorsiflex the foot (S1-S2), raise the knees (L2-L3), or tense the abdominal rectus muscles (T6-T12). The first 5 to 10 minutes after the administration of hyperbaric or hypobaric local anesthetic solutions is the most critical time for adjusting the level of anesthesia (Table 17-3). The first 5 to 10 minutes are also critical for assessing cardiovascular responses (systemic arterial blood pressure and heart rate) to the evolving spinal anesthesia. Delayed bradycardia and cardiac asystole mandate continuous vigilance beyond early attainment of anesthesia.[29]

Continuous Spinal Anesthesia

Inserting a catheter into the subarachnoid space increases the utility of spinal anesthesia by permitting repeated drug administration as necessary to maintain the level and duration of sensory and motor block (Table 17-4). Anesthesia can be provided for prolonged operations without delaying recovery. An added benefit is the possibility of using lower doses of anesthetic. With the single-injection technique, relatively high doses must be administered to all patients to ensure successful anesthesia in a large percentage of cases. With a catheter in place, smaller doses can be titrated to the patient's response.

TECHNIQUE

After inserting the needle and obtaining free flow of CSF, the catheter is advanced through the needle into the subarachnoid space. Care must be exercised to limit the catheter insertion distance to 2 to 4 cm because unlike placement in the epidural space, further advancement of a subarachnoid catheter runs the risk of impaling the spinal cord. The use of large-bore epidural needles and catheters introduces a significant risk for post–dural puncture headache.

Table 17-4 Continuous Spinal Anesthesia: Guidelines for Anesthetic Administration

- Insert the catheter just far enough to confirm and maintain placement.
- Use the lowest effective local anesthetic concentration.
- Place a limit on the dose of local anesthetic to be used.
- Administer a test dose and assess the extent of any sensory and motor block.
- If maldistribution is suspected, use maneuvers to increase the spread of local anesthetic (change the patient's position, alter the lumbosacral curvature, switch to a solution with a different baricity).
- If well-distributed sensory anesthesia is not achieved before the dose limit is reached, abandon the technique.

Adapted from Rigler ML, Drasner K, Krejcie TC, et al. Cauda equina syndrome after continuous spinal anesthesia. Anesth Analg 1991;72:275-281.

Microcatheters

Microcatheters (27 gauge and smaller) for continuous spinal anesthesia were withdrawn from clinical practice after reports of cauda equina syndrome associated with their use.[20] The injury associated with the use of microcatheters likely resulted from the combination of maldistribution and repetitive injection of local anesthetic solution. It appears that pooling of local anesthetic solution in the dependent sacral sac produced a restricted block that was inadequate for surgery. In response to inadequate anesthesia, injections were repeated and ultimately achieved adequate sensory anesthesia, but not before neurotoxic concentrations were reached in the caudal region of the subarachnoid space.[30] The microcatheter may have contributed to this problem because the long narrow-bore tubing creates resistance to injection and thereby results in a low flow rate that can encourage a restricted distribution. However, removal of microcatheters from clinical practice has not eliminated the risk. The problem of maldistribution is not restricted to microcatheters, and the same injuries have occurred with larger "epidural" catheters and other local anesthetics.[20]

Failed Spinal Anesthesia and Repetitive Subarachnoid Injections

Spinal anesthesia is not uniformly successful, and failure may derive from technical considerations such as an inability to identify the subarachnoid space or failure to inject all or part of the local anesthetic solution into the

subarachnoid space. A second and generally underappreciated cause of failure is local anesthetic maldistribution. In support of this mechanism is the correlation of success rate with the vertebral interspace, the more caudad interspaces being more prone to failure.[13] This issue becomes important when considering whether to repeat a "failed" spinal and, if so, the dose of anesthetic that should be used for the second injection. In the past, a "full dose" was given. However, if failure derives from maldistribution of the local anesthetic solution, this strategy may introduce a risk of injury.[31] In essence, an overdose of local anesthesia in the subarachnoid space could occur. If a spinal anesthetic is to be repeated, one should assume that the first injection was delivered in the subarachnoid space as intended, and the combination of the two doses should not exceed that considered reasonable as a single injection for spinal anesthesia.

Physiology

Spinal anesthesia interrupts sensory, motor, and sympathetic nervous system innervation. Local anesthetic solutions injected into the subarachnoid space produce a conduction block of small-diameter, unmyelinated (sympathetic) fibers before interrupting conduction in larger myelinated (sensory and motor) fibers. The sympathetic nervous system block typically exceeds the somatic sensory block by two dermatomes. This estimate may be conservative, with sympathetic nervous system block sometimes exceeding somatic sensory block by as many as six dermatomes, which explains why systemic hypotension may accompany even low sensory levels of spinal anesthesia.

Spinal anesthesia has little, if any, effect on resting alveolar ventilation (i.e., analysis of arterial blood gases unchanged), but high levels of motor anesthesia that produce paralysis of abdominal and intercostal muscles can decrease the ability to cough and expel secretions. Additionally, patients may complain of difficulty breathing (dyspnea) despite adequate ventilation because of inadequate sensation of breathing from loss of proprioception in the abdominal and thoracic muscles.

Spinal anesthesia above T5 inhibits sympathetic nervous system innervation to the gastrointestinal tract, and the resulting unopposed parasympathetic nervous system activity results in contracted intestines and relaxed sphincters. The ureters are contracted, and the ureterovesical orifice is relaxed. Block of afferent impulses from the surgical site by spinal anesthesia is consistent with the absence of an adrenocortical response to painful stimulation. Decreased bleeding during regional anesthesia and certain types of surgery (hip surgery, transurethral resection of the prostate) may reflect a decrease in systemic blood pressure, as well as a reduction in peripheral venous pressure, whereas increased blood flow to the lower extremities after sympathetic nervous system block appears to be a major factor in the decreased incidence of thromboembolic complications after hip surgery. There does not appear to be any difference in the perioperative mortality rate between regional anesthesia and general anesthesia administered to relatively healthy patients scheduled for elective surgery.

Side Effects and Complications

Side effects associated with spinal anesthesia can usually be predicted from the physiologic effects of the block. Persistent neurologic complications are rare and can be minimized by an appreciation of the factors that can contribute to injury.

NEUROLOGIC COMPLICATIONS

Neurologic complications after spinal anesthesia may result from trauma, either directly provoked by a needle or catheter or indirectly by compression from hematoma or abscess. The occurrence of a paresthesia can, on occasion, be associated with postoperative neurologic findings, which generally resolve. Such injuries will occur with greater frequency and will be more profound if injection of anesthetic is made in the presence of a paresthesia, thus emphasizing the importance of avoiding local anesthetic injection should a paresthesia be present or halting injection should this occur during administration. Local anesthetics, if administered in sufficient quantity (particularly within restricted areas of the subarachnoid space), can induce permanent injury.[20,31] Transient neurologic symptoms are a common occurrence after the subarachnoid administration of certain local anesthetics, particularly lidocaine.[18,23,24] Despite the designation as "neurologic," the cause and significance of this self-limited condition remain to be determined.

HYPOTENSION

Hypotension (systolic arterial blood pressure <90 mm Hg) occurs in about a third of patients receiving spinal anesthesia.[32] Hypotension results from a sympathetic nervous system block that (1) decreases venous return to the heart and decreases cardiac output or (2) decreases systemic vascular resistance.

Modest hypotension (e.g., <20 mm Hg) is probably due to decreases in systemic vascular resistance, whereas more intense hypotension (>20 mm Hg) probably is the result of decreases in venous return and cardiac output. The degree of hypotension often parallels the sensory level of spinal anesthesia and the intravascular fluid volume status of the patient. Indeed, the magnitude of hypotension produced by spinal anesthesia will be more with coexisting hypovolemia.

Treatment

Spinal anesthesia-induced hypotension is treated physiologically by restoration of venous return to increase cardiac output. In this regard, the internal autotransfusion produced

by a modest head-down position (5 to 10 degrees) will facilitate venous return without greatly exaggerating cephalad spread of the spinal anesthetic. Adequate intravenous hydration before the institution of spinal anesthesia is important for minimizing the effects of venodilation from sympathetic nervous system block. However, excessive amounts of intravenously administered fluids may be detrimental, particularly in a patient with limited cardiac function or ischemic heart disease, in whom excessive intravascular volume or hemodilution may be poorly tolerated.

Sympathomimetics with positive inotropic and venoconstrictor effects, such as ephedrine (5 to 10 mg IV), are often chosen as first-line drugs to maintain perfusion pressure during the first few minutes after the institution of spinal anesthesia. Phenylephrine (50 to 100 μg IV) and other sympathomimetics that increase systemic vascular resistance may decrease cardiac output and do not specifically correct the decreased venous return contributing to the spinal anesthesia–induced hypotension. Nevertheless, anesthesia providers have long used phenylephrine successfully to treat hypotension associated with spinal anesthesia. Furthermore, phenylephrine is of particular value when administration of ephedrine is associated with significant increases in heart rate. In the past, phenylephrine was contraindicated in parturients because of possible detrimental effects on uterine blood flow, which has not been confirmed (see Chapter 33).[33] In the rare instance when hypotension does not promptly respond to ephedrine or phenylephrine, epinephrine should be given to avoid progression to profound hypotension or even cardiac arrest.

BRADYCARDIA AND ASYSTOLE

The heart rate does not change significantly in most patients during spinal anesthesia. However, in an estimated 10% to 15% of patients, significant bradycardia occurs. As with hypotension, the risk for bradycardia increases with increasing sensory levels of anesthesia. Speculated mechanisms for this bradycardia include block of cardioaccelerator fibers originating from T1 through T4 and decreased venous return (Bezold-Jarisch reflex).

Although bradycardia is usually of modest (<20 bpm) severity and promptly responsive to atropine or ephedrine, precipitous bradycardia and asystole can happen in the absence of any preceding event (Fig. 17-16).[29,34] This catastrophic event can probably be prevented through maintenance of preload and reversal of bradycardia by aggressive stepwise escalation of treatment (ephedrine, 5 to 50 mg IV; atropine, 0.4 to 1.0 mg IV; epinephrine, 0.05 to 0.25 mg IV), whereas the development of profound bradycardia or asystole mandates immediate treatment with full resuscitative doses of epinephrine (1.0 mg IV).

POST-DURAL PUNCTURE HEADACHE

Post–dural puncture headache is a direct consequence of the puncture hole in the dura, which results in loss of CSF

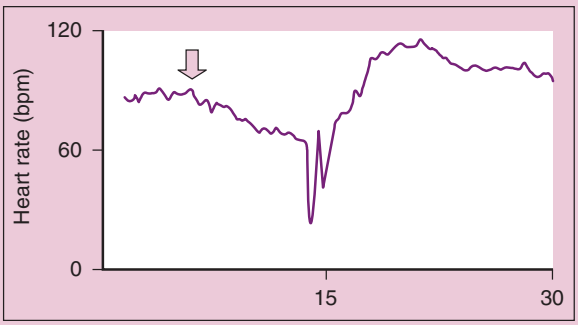

Figure 17-16 Recording of heart rate over time from a single patient who received a spinal anesthetic at the arrow, followed by a subsequent gradual decrease in heart rate that culminated in precipitous bradycardia that was unresponsive to atropine and ephedrine. Immediate institution of external cardiac compressions was promptly followed by sinus rhythm (85 beats/min). The precipitous bradycardia occurred while the patient was conversing with the anesthesia provider and in the presence of normal vital signs (oxygen saturation, 98%; systemic blood pressure, 120/50 mm Hg; heart rate, 68 beats/min). (From Mackey DC, Carpenter RL, Thompson GE, et al. Bradycardia and asystole during spinal anesthesia: A report of three cases with morbidity. Anesthesiology 1989;70:866-868, used with permission.)

at a rate exceeding production. Loss of CSF causes downward displacement of the brain and resultant stretch on sensitive supporting structures (Fig. 17-17).[35] Pain also results from distention of the blood vessels, which must compensate for the loss of CSF because of the fixed volume of the skull.

Manifestations

The pain associated with post–dural puncture headache generally begins 12 to 48 hours after transgression of the dura, but can occur immediately even up to several months after the event. The characteristic feature of post–dural puncture headache is its postural component: it appears or intensifies with sitting or standing and is partially or completely relieved by recumbency. This feature is so distinctive that it is difficult to consider the diagnosis in its absence. Post–dural puncture headache is typically occipital or frontal (or both) and is usually described as dull or throbbing. Associated symptoms such as nausea, vomiting, anorexia, and malaise are common. Ocular disturbances, manifested as diplopia, blurred vision, photophobia, or "spots," may occur and are believed to result from stretch of the cranial nerves, most commonly cranial nerve VI, as the brain descends because of the loss of CSF. Although symptomatic hearing loss is unusual, formal auditory testing will routinely reveal abnormalities.

Though generally a transient problem, loss of CSF may rarely result in significant morbidity because caudal displacement of the brain can result in tearing of

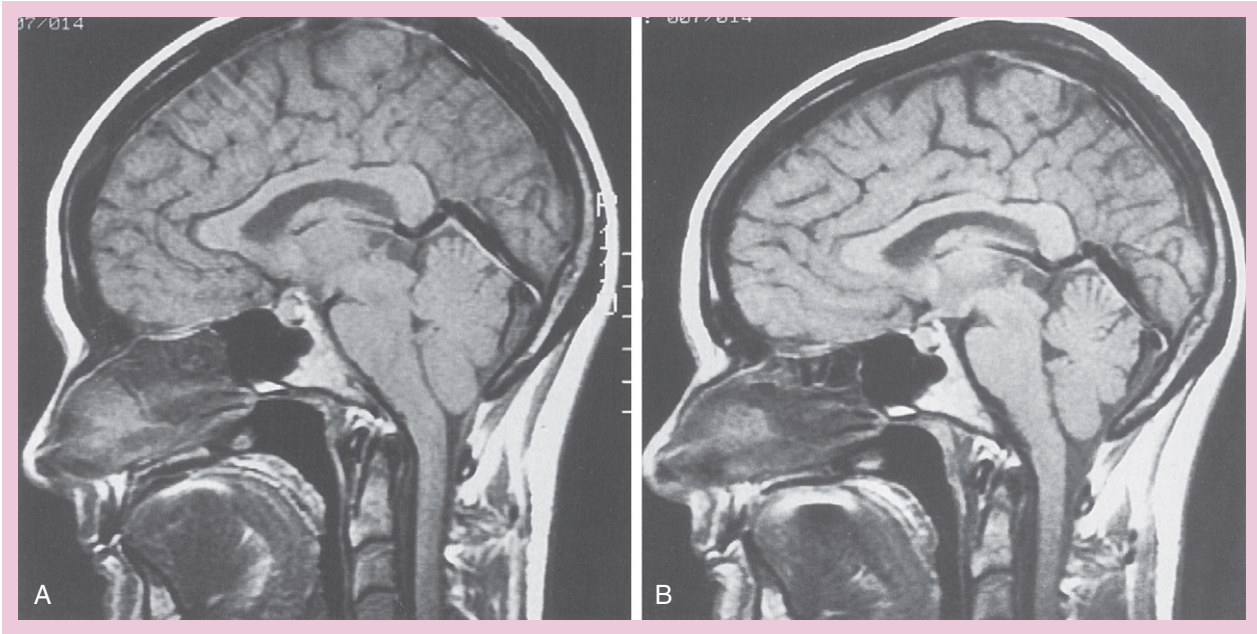

Figure 17-17 Anatomy of a "low-pressure" headache. **A,** A T1-weighted sagittal magnetic resonance image demonstrates a "ptotic brain" manifested as tonsillar herniation below the foramen magnum, forward displacement of the pons, absence of the suprasellar cistern, kinking of the chiasm, and fullness of the pituitary gland. **B,** A comparable image of the same patient after an epidural blood patch and resolution of the symptoms demonstrates normal anatomy. (From Drasner K, Swisher JL. In Brown DL [ed]. Regional Anesthesia and Analgesia. Philadelphia, WB Saunders, 1996.)

bridging veins with the development of a subdural hematoma. Concern should be raised if post–dural puncture headache is progressive or refractory or loses its postural component.

Risk Factors

Age is one of the most important factors affecting the incidence of post–dural puncture headache. Children are at low risk, but after puberty, risk increases substantially and then slowly declines with advancing age. Females are at increased risk even in the absence of pregnancy.[36] A previous history of post–dural puncture headache places a patient at increased risk for the development of this complication after a subsequent spinal anesthetic.

The incidence of post–dural puncture headache varies directly with the diameter of the needle that has pierced the dura. However, the benefit of a smaller needle with respect to post–dural puncture headache must be balanced against the technical challenge that it may impose. The common use of 24- and 25-gauge needles represents a balance between these two considerations. The shape of the hole created by the needle also has an impact on loss of CSF; this has led to the development of "pencil-point" needle tips, which appear to spread the dural and arachnoid fibers and produce less tear and a smaller hole for a given diameter needle.

Treatment

Initial treatment of post–dural puncture headache is usually conservative and consists of bed rest, intravenous fluids, analgesics, and possibly caffeine (500 mg IV). More definitively, a blood patch can be performed, in which 15 to 20 mL of the patient's blood, aseptically obtained, is injected into the epidural space. The injection should be made near or preferably below the site of initial puncture because there is preferential cephalad spread. The patient should remain supine for at least 2 hours and relief should be immediate. The immediate effect is related to the volume effect of the injected blood, whereas long-term relief is thought to occur from sealing or "patching" of the dural tear.

HIGH SPINAL ANESTHESIA

Systemic hypotension frequently accompanies high spinal anesthesia, and patients will become nauseated and agitated. Total spinal anesthesia is the term applied to excessive sensory and motor anesthesia associated with loss of consciousness. Apnea and loss of consciousness are often attributed to ischemic paralysis of the medullary ventilatory centers because of profound hypotension and associated decreases in cerebral blood flow. However, loss of consciousness may also be the direct consequence of local anesthetic effect above the foramen

magnum inasmuch as patients may lose or fail to regain consciousness despite restoration of systemic arterial blood pressure. Lesser degrees of excessive spinal anesthesia may warrant conversion to a general anesthetic because of patient distress, ventilatory failure, or risk of aspiration. Total spinal anesthesia is typically manifested soon after injection of the local anesthetic solution into the subarachnoid space.

Treatment of high or total spinal anesthesia consists of maintenance of the airway and ventilation, as well as support of the circulation with sympathomimetics and intravenous fluid administration. Patients are placed in a head-down position to facilitate venous return. An attempt to limit the cephalad spread of local anesthetic solution in CSF by placing patients in a head-up position is not recommended because this position will encourage venous pooling, as well as potentially jeopardizing cerebral blood flow, which may contribute to medullary ischemia. Tracheal intubation is usually warranted and is mandated for patients at risk of aspiration of gastric contents (e.g., pregnant women). Sometimes, thiopental or propofol should be given before tracheal intubation if consciousness is retained and cardiovascular status is acceptable.

NAUSEA

Nausea occurring after the initiation of spinal anesthesia must alert the anesthesia provider to the possibility of systemic hypotension sufficient to produce cerebral ischemia. In such cases, treatment of hypotension with a sympathomimetic drug should eliminate the nausea. Alternatively, nausea may occur because of a predominance of parasympathetic activity as a result of selective block of sympathetic nervous system innervation to the gastrointestinal tract. Similar to bradycardia, the incidence of nausea and vomiting parallels the sensory level of spinal anesthesia.

URINARY RETENTION

Because spinal anesthesia interferes with innervation of the bladder, administration of large amounts of intravenous fluids can cause bladder distention, which may require catheter drainage. For this reason, excessive administration of intravenous fluids should be avoided in patients undergoing minor surgery with spinal anesthesia. However, adequate intravascular fluid replacement must be administered to maintain effective preload and reduce the degree of hypotension and possible progression to bradycardia and asystole.[29] Inclusion of epinephrine in the local anesthetic solution may be associated with a prolonged time to voiding.

BACKACHE

Minor, short-lived back pain frequently follows spinal anesthesia and is more likely with multiple attempts at correct advancement of the spinal needle. Backache may also be related to the position required for surgery. Ligament strain may occur when anesthetic-induced sensory block and skeletal muscle relaxation permit the patient to be placed in a position that would normally be uncomfortable or unobtainable. Backache can be confused with transient neurologic symptoms.

HYPOVENTILATION

Decreases in vital capacity can occur if the motor block extends into the upper thoracic and cervical dermatomes. Loss of proprioception from the intercostal musculature can produce dyspnea. Exaggerated hypoventilation may accompany the intravenous administration of drugs intended to produce a sleeplike state during spinal anesthesia. Constant vigilance of ventilatory status is enhanced by monitors, which must include pulse oximetry and may include capnography.

EPIDURAL ANESTHESIA

Epidural anesthesia, like spinal anesthesia, can be instituted with the patient in the sitting or lateral decubitus position, whereas the prone position is generally selected for caudal blocks. As with subarachnoid injection, the sitting position facilitates placement by encouraging flexion and aiding in recognition of the midline. However, the lateral position is associated with a lower incidence of venous cannulation.[37] Patients typically receive drugs to produce sedation, except when epidural catheters are placed in pregnant women.

Timing of Catheter Placement

Controversy exists regarding the wisdom of placing lumbar epidural catheters after induction of general anesthesia. Although there is concern that an inability to elicit a patient response might increase the risk for neural injury, a retrospective review challenges this assertion.[38] Nonetheless, many anesthesia providers believe that lumbar epidural anesthesia and catheter placement are best performed in a communicative patient.[39] Performance of thoracic epidural anesthesia in an anesthetized patient should be avoided.[40] However, the same considerations do not apply to pediatric anesthesia, for which a conscious patient would probably impart no benefit but instead add substantial risk (see Chapter 34). Consequently, it is standard practice to place caudal, lumbar, and even thoracic epidural catheters in children after induction of general anesthesia.

Epidural Needles

The most commonly used epidural needle (Tuohy needle) was originally designed and first used for continuous spinal anesthesia and only later adapted for epidural use. The modern Tuohy needle has a blunt tip so that it might rest against the dura without penetration, but the tip has

retained its gentle curve, which serves to guide the catheter's exit obliquely from the needle. Other epidural needles represent modifications of this basic design. For example, the Weiss needle has prominent wings to help stabilize the anesthesia provider's grip, and the Crawford needle has a straighter tip that may be better suited to the steep approach of a midline thoracic epidural or to passage of a catheter into the sacral canal.

Epidural Catheters

Like needles, catheter designs vary (Fig. 17-18).[41] For example, some catheters have an inner stainless steel wire coil to impart flexibility and prevent kinking. This characteristic makes them less likely to (1) pierce an epidural vessel,[37] (2) find false passage into a fascial plane, and (3) be advanced out of the epidural space through the intervertebral foramen. However, their flexibility also makes them more difficult to thread into the epidural space. The tip of the catheter may be open or have a closed "bullet" tip with proximal ports. Bullet-tipped or multiorifice catheters tend to produce more uniform distribution of local anesthetic solution, but they have the disadvantage of requiring greater insertion depth to ensure complete delivery of local anesthetic solution into the epidural space.

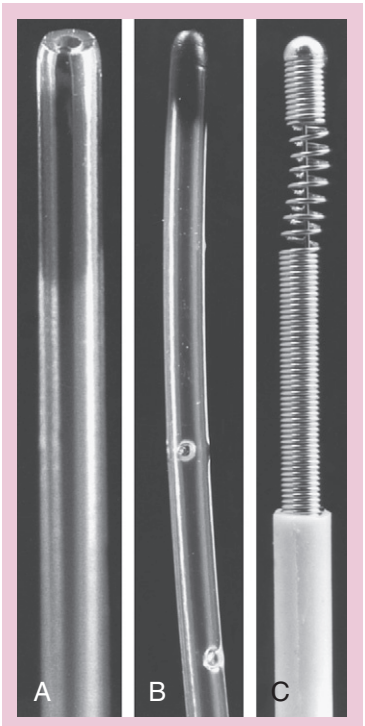

Figure 17-18 **A** to **C**, Epidural catheters may have an open end or several openings proximal to the tip. (From Brown DL. Spinal, epidural and caudal anesthesia. In Miller RD [ed]. Miller's Anesthesia. Philadelphia, Elsevier, 2010, pp 1611-1638.)

Epidural Kit

As with spinal anesthesia, epidural anesthesia is performed with equipment obtained from prepackaged sterile epidural kits. Most epidural kits contain a 17- or 18-gauge needle, which permits passage of a 19- or 20-gauge catheter, respectively. Both the needles and the catheters have calibrated markings so that the anesthesia provider can determine the depth of insertion from the skin, as well as the distance that the catheter has advanced past the tip of the needle.

Each epidural kit has one or two needles for infiltration of the skin and for probing the intervertebral space before insertion of the larger epidural needle. The length of this "finder needle" is usually 3.8 cm, which is sufficient to reach the subarachnoid space in some patients, although the depth of the epidural space is generally 4 to 6 cm. The distance to the epidural space will be affected by body weight and by the angulation of the needle (insertion depth is obviously greater with marked cephalic angulation).

Technique

LUMBAR AND LOW THORACIC EPIDURAL

The technique for a lumbar epidural and low thoracic epidural is similar because the anatomic features of the spine are similar at these vertebral levels. Both midline and paramedian approaches can be used successfully, but the midline is more popular. Advantages of the midline approach include (1) simpler anatomy because there is no need to determine the appropriate lateral orientation of the needle and (2) passage of the needle through less sensitive structures and less probability of contacting facet joints or large spinal nerves that innervate the leg. However, the paramedian approach is better suited when challenging circumstances such as hypertrophied bony spurs, spinal abnormalities, or failure to adequately flex create obstacles to needle advancement. Some anesthesia providers also prefer to use a paramedian approach based on the unproven concept that needle passage through the interspinous ligament increases the risk for postoperative backache.

THORACIC EPIDURAL

In contrast to procedures performed in the lumbar area, thoracic epidural anesthesia is generally accomplished through a paramedian approach. In this region the spinous processes are angulated and closely approximated, which makes it difficult to avoid bony obstruction when approaching from the midline. Identification of the lamina is the initial step. If the spinous process can be identified, a skin wheal is raised 0.5 to 1 cm off midline at the caudad tip of the spinous process. The finder needle is then directed at a right angle to the skin and the needle advanced until the lamina is contacted. Local anesthetic

is deposited, the needle is withdrawn, and the longer epidural needle is positioned and advanced in a similar manner. After the lamina is contacted, the needle is repeatedly retracted and advanced in a slightly more medial and cephalad direction until it fails to make bony contact at the depth anticipated by previous insertions. If contact with bone continues to occur, the needle is retracted and positioned at a slightly different angle and the process repeated. If success is not obtained, the process is repeated at an insertion site that is slightly (about 1 cm) cephalad or caudad. The midline approach to the thoracic epidural space is similar to that described for lumbar epidurals except that the needle must be advanced cephalad at a more acute angle to pass between the steep down-sloping spinous processes (see Fig. 17-5).[5]

Identification of the Epidural Space

Firm engagement of the needle tip in the ligamentum flavum is the most critical step in identification of the epidural space when using either a paramedian or midline approach.

LOSS-OF-RESISTANCE TECHNIQUE

With the loss-of-resistance technique, a syringe containing saline, air, or both is attached to the needle, and the needle is slowly advanced while assessing resistance to injection (Fig. 17-19).[3] One method is to use a syringe containing 2 to 3 mL of saline with a small air bubble (0.1 to 0.3 mL). If the needle is properly seated in the ligamentum flavum, it will be difficult to inject the saline or the air bubble, and the plunger of the syringe will "spring back" to its original position. If the air bubble cannot be compressed without injecting the saline, the needle is most likely not in the ligamentum flavum. In this case, the needle tip may still be in the interspinous ligament, or it may be off the midline in the paraspinous muscles. After proper positioning in the ligamentum flavum, the needle is advanced while continuous pressure is exerted on the plunger of the syringe. An abrupt loss of resistance to injection signals passage through the ligamentum flavum and into the epidural space, at which point the contents of the syringe are delivered. An often-cited advantage of this method is that the dura tends to be forced away from the advancing needle by the saline ejected from the syringe. The introduction of fluid into the epidural space prior to catheter insertion also serves to reduce the incidence of epidural vein cannulation.[37]

HANGING-DROP TECHNIQUE

The "hanging-drop" technique is an alternative method for identifying the epidural space. With this technique, a small drop of saline is placed at the hub of the epidural needle. As the needle passes through the ligamentum flavum into the epidural space, the saline drop is retracted into the needle by the negative pressure in the epidural

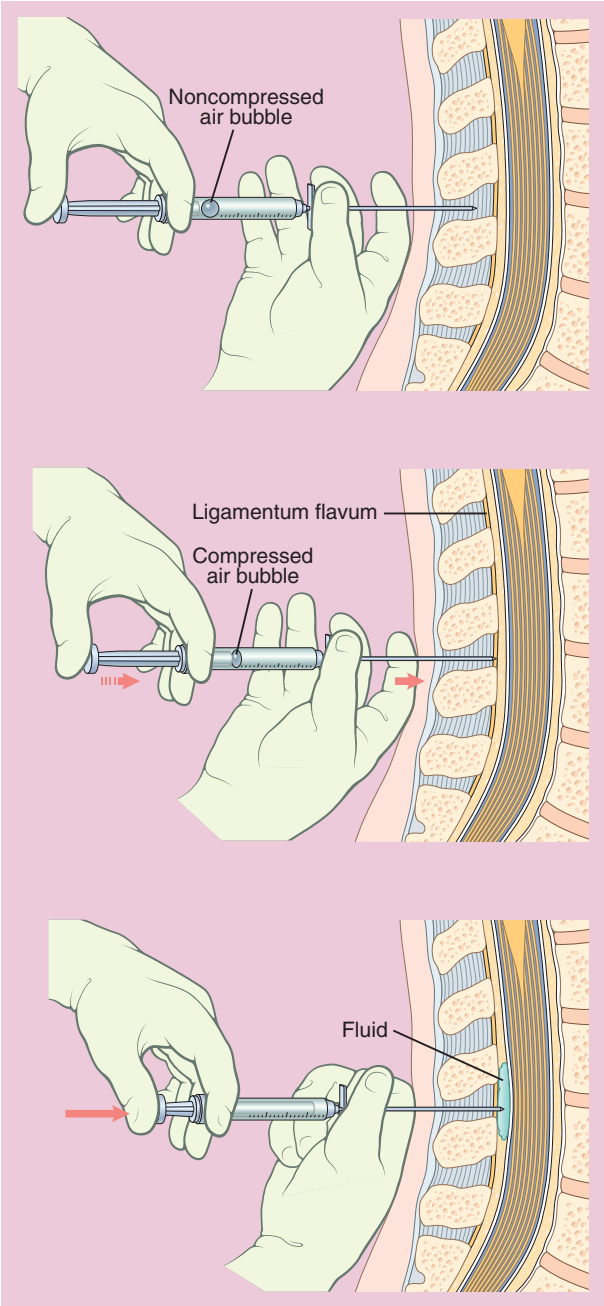

Figure 17-19 Loss-of-resistance technique. The needle is inserted into the ligamentum flavum, and a syringe containing saline and an air bubble is attached to the hub. After compression of the air bubble is obtained by applying pressure on the syringe plunger, the needle is carefully advanced until its entry into the epidural space is confirmed by the characteristic loss of resistance to syringe plunger pressure, and the fluid enters the space easily. (From Afton-Bird G. Atlas of regional anesthesia. In Miller RD [ed]. Miller's Anesthesia. Philadelphia, Elsevier, 2005.)

space. Interestingly, the hanging-drop technique can be used in the lumbar region despite the lack of negative pressure in the lumbar epidural space. In this region, the needle pushing the dura away from the ligamentum flavum creates negative pressure. Accordingly, this technique is likely to be associated with a higher incidence of accidental dura penetration (wet taps).

Administration of Local Anesthetic

As with spinal anesthesia, epidural anesthesia can be performed by injection of local anesthetic solution through the needle (single shot) or more commonly, through a catheter threaded into and maintained in the epidural space (continuous).

SINGLE-SHOT EPIDURAL ANESTHESIA

The advantage of the single-injection technique is its simplicity, and the distribution of local anesthetic solution tends to be more uniform than when administered through an indwelling catheter. Achievement of anesthesia with the single-injection technique begins with administration of a test dose of local anesthetic solution (e.g., 3 mL of 1.5% lidocaine with 1:200,000 epinephrine). Failure of the test dose to produce sensory and motor anesthesia is assessed after 3 minutes to rule out accidental subarachnoid injection (spinal anesthesia has a more rapid onset of sensory and motor block). If epinephrine has been included in the test dose, the heart rate is carefully monitored to detect an increase that may signal accidental intravascular injection. The local anesthetic solution is then injected in fractionated doses (e.g., multiple injections of 5-mL aliquots) over a 1- to 3-minute period at an appropriate volume and concentration (dose) for the planned surgical procedure. Intermittent dosing is critical because a negative test result does not conclusively rule out intravascular placement. Moreover, the needle's position may have changed during or between injections.

CONTINUOUS EPIDURAL ANESTHESIA

With the continuous epidural technique, a catheter is advanced 3 to 5 cm beyond the tip of the needle positioned in the epidural space. Further advancement increases the risk that the catheter might enter an epidural vein, exit an intervertebral foramen, or wrap around a nerve root. The epidural needle is withdrawn over the catheter, with care taken to not move the catheter. No attempt should be made to withdraw a catheter back through the needle because shearing (transection) of the catheter might result, with retention of the transected tip of the catheter in the epidural space. The catheter is taped to the patient's back, and an empty 3-mL syringe is attached to the distal end of the catheter. Negative pressure is applied to the syringe, and failure to aspirate CSF or blood helps rule out accidental subarachnoid or intravascular placement. After negative aspiration and a negative test dose, epidural anesthesia is initiated by the administration of local anesthetic solution in fractionated doses (multiple injections of 5-mL aliquots). It is important to reconfirm negative aspiration of CSF or blood from the catheter before any subsequent dose of local anesthetic is administered. Documentation of the level of sympathetic nervous block and sensory anesthesia is determined as described for spinal anesthesia (see the earlier section "Documentation of Anesthesia").

CAUDAL ANESTHESIA

Caudal anesthesia in an adult is performed with the patient in either the prone or the lateral position. The sacral area is prepared and draped and the sacral cornu (typically 3 to 5 cm above the coccyx) identified by the anesthesia provider's palpating fingers. The depression between the cornu is the sacral hiatus, and a skin wheal is raised. The needle is introduced perpendicular to the skin through the sacrococcygeal ligament (generally felt as a rather distinct pop) and advanced until the sacrum is contacted. The needle is then slightly withdrawn, the angle is reduced, and the needle is advanced about 2 cm into the epidural caudal canal (Fig. 17-20).[3] Confirmation that the needle is properly positioned can be obtained by rapidly injecting 5 mL of air or saline through the needle

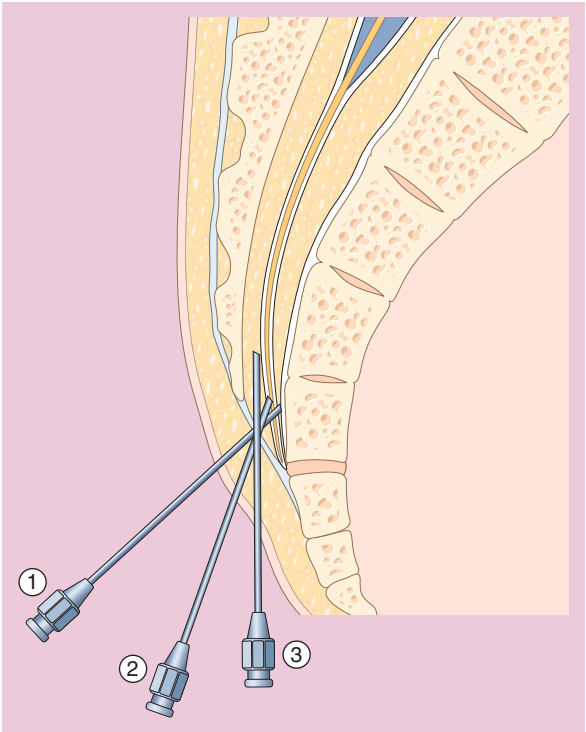

Figure 17-20 Caudal anesthesia: (1) skin penetration at a 60- to 90-degree angle, (2) redirection of the needle, and (3) slight penetration (1 to 2 mm) within the spinal canal. (From Afton-Bird G. Atlas of regional anesthesia. In Miller RD [ed]. Miller's Anesthesia. Philadelphia, Elsevier, 2005.)

while palpating the skin directly covering the caudal canal. Subcutaneous crepitus or midline swelling indicates that the needle is positioned posterior to the bony sacrum and requires replacement.

Although infection is rare, the nearness of this approach to the rectum mandates particular attention to sterile technique. Subarachnoid injection may occur if the needle is advanced too far cephalad in the sacral canal, or it may result from anatomic variation (the dural sac extends beyond S2 in approximately 10% of individuals). Anatomic variation may also hinder success inasmuch as the sacral hiatus is absent in nearly 10% of patients.

In contrast to adults, location of the sacral hiatus and performance of caudal anesthesia are technically easy in children. After induction of general anesthesia, the child is placed in the lateral position and a needle or catheter is advanced into the sacral canal. A long-acting local anesthetic will limit general anesthetic requirements and produce effective postoperative analgesia for procedures involving the perineum or lower lumbar dermatomes.

Level of Anesthesia

The principal factors affecting the spread of epidural anesthesia are dose (volume multiplied by concentration) and site of injection. However, administration of an equivalent dose (mass) at lower concentration may foster greater spread, particularly with lower concentrations of local anesthetic. Cephalad-to-caudad extension of epidural anesthesia depends on the site of administration of the local anesthetic solution into the epidural space. Lumbar epidural injections produce preferential cephalad spread because of negative intrathoracic pressure transmitted to the epidural space, whereas resistance to caudad spread of local anesthetic solution is created by narrowing of the space at the lumbosacral junction. In contrast, thoracic injections tend to produce symmetrical anesthesia and result in greater dermatomal spread for a given dose of local anesthetic. This latter effect results, at least in part, from the comparatively smaller volume of the thoracic epidural space, which is smaller in the thoracic region. The site of placement of the local anesthetic solution in the epidural space also defines the area of peak anesthetic effect, which decreases with increasing distance from the injection site.

Spread of anesthesia may vary directly with age and inversely with height, although these effects are likely to be overshadowed by interpatient variability. Achievement of anesthesia is not equivalent at all dermatomes. For example, anesthesia in the L5-S1 distribution may be relatively spared, an effect believed to result from the large diameter of these nerve roots. In contrast to spinal anesthesia, the baricity of local anesthetic solutions does not influence the level of epidural anesthesia. Likewise, patient position during performance of an epidural block is less important, but the dependent portion of the body may still manifest more intense anesthesia than the nondependent side. This effect is most noticeable in a pregnant woman who has remained in a specific lateral position for a prolonged period during labor.

Duration of Anesthesia

The duration of epidural anesthesia, as with spinal anesthesia, is principally affected by the choice of local anesthetic and whether a vasoconstrictor drug is added to the local anesthetic solution. Because achievement of epidural anesthesia is delayed relative to spinal anesthesia, onset time is an additional consideration in selection of the local anesthetic. In this regard, the local anesthetics most commonly selected for epidural anesthesia are (1) chloroprocaine (rapid onset and short duration), (2) lidocaine (intermediate onset and duration), and (3) bupivacaine, levobupivacaine, and ropivacaine (slow onset and prolonged duration of action) (Table 17-5). Tetracaine and procaine have long latency times, which makes them unsuitable for epidural use. Levobupivacaine is clinically similar to bupivacaine, whereas ropivacaine is less potent and often used at higher concentrations.

Adjuvants

EPINEPHRINE

The addition of epinephrine (generally 1:200,000; 5 μg/mL) to local anesthetic solutions decreases vascular absorption of the local anesthetic from the epidural space, thus

Table 17-5 Local Anesthetics Used for Epidural Anesthesia

Drug	Concentration (%)	Time to Onset (min)	Duration (min)	
			Plain	Epinephrine (1:200,000)
Chloroprocaine	2-3	5-10	45-60	60-90
Lidocaine	1-2	10-15	60-120	90-180
Bupivacaine	0.25-0.5	15-20	120-200	150-240
Ropivacaine	0.25-1.0	10-20	120-180	150-200

maintaining effective anesthetic concentrations at the nerve roots for more prolonged periods. Decreased vascular absorption also serves to limit systemic uptake and reduce the risk for systemic toxicity from the local anesthetic. These effects are far more pronounced when epinephrine is coadministered with chloroprocaine or lidocaine than with bupivacaine. The inclusion of epinephrine also serves as a marker of intravascular injection that may occur with cannulation of an epidural vein. The use of an intravascular marker has been classified as a level IIa recommendation (level of evidence B) in a recent practice advisory published by the American Society of Regional Anesthesia.[42]

OPIOIDS

Similar to spinal anesthesia, opioids are often administered with epidural local anesthetic solutions to enhance surgical anesthesia and to provide postoperative pain control. However, in contrast to spinal administration, lipid solubility of the opioid is a critical factor in determining the selection and appropriate use of epidural opioids. For example, morphine, which is relatively hydrophilic, spreads rostrally within the CSF and can produce effective analgesia for thoracic surgery, even when administered into the lumbar epidural space. In contrast, a lipophilic opioid such as fentanyl is rapidly absorbed into the systemic circulation and exhibits little rostral spread. Consequently, the lipophilic opioids demonstrate limited selective spinal activity when administered in the lumbar epidural region because their site of action, the dorsal horn of the spinal cord, rests several segments rostral to the site of administration.

SODIUM BICARBONATE

Local anesthetic effect requires transfer across the nerve membrane. Because local anesthetics are weak bases, they exist largely in the ionic form in commercial preparations. Adding sodium bicarbonate to the solution favors the nonionized form of the local anesthetic and promotes more rapid onset of epidural anesthesia. Most commonly, 1 mL of 8.4% sodium bicarbonate is added to 10 mL of a solution containing lidocaine or chloroprocaine. Alkalinization of a bupivacaine solution is not recommended because this local anesthetic precipitates at alkaline pH.

Failed Epidural Anesthesia

Failed epidural anesthesia may occur when local anesthetic solution is not delivered into the epidural space or because spread of the local anesthetic solution is inadequate to cover the relevant dermatomes. A false loss of resistance can occur in the interspinous ligament before entry into the ligamentum flavum or as the needle passes through fascial planes. For example, the paramedian approach in the thoracic region requires the needle to pass through the latissimus dorsi and trapezius muscles as they insert onto the thoracic vertebrae. It is conceivable that a needle passing through these fascial coverings could transmit a feeling of loss of resistance to the fingers of the anesthesia provider. In some cases, failure results from advancement of the catheter through an intervertebral foramen, which generally gives rise to a limited unilateral block. Fortunately, these blocks can often be salvaged by retracting the catheter a few centimeters. Opinion varies on the presence of a midline barrier to diffusion of local anesthetics.

If epidural anesthesia is nearly adequate and there are concerns that additional local anesthetic would create a risk for systemic toxicity, small doses of chloroprocaine, which are rapidly hydrolyzed in plasma, may provide adequate extension to permit surgery.[43] At other times, failure of epidural anesthesia may be managed by replacement of the epidural catheter or abandonment of the technique in favor of a general or spinal anesthetic. However, subarachnoid injection after a failed epidural produces unpredictable and often excessive spinal anesthesia.[44] This effect probably results primarily from compression of the dural sac by the volume of anesthetic solution in the epidural space.

Physiology

The major site of action of local anesthetic solutions placed in the epidural space appears to be the spinal nerve roots, where the dura is relatively thin. A spinal nerve root site of action is consistent with the often-observed delayed onset or absence of anesthesia in the S1-S2 region, presumably reflecting the covering of these nerve roots with connective tissue. To a lesser extent, anesthesia results from diffusion of local anesthetic solutions from the epidural space into the subarachnoid space.

Because the epidural space ends at the foramen magnum, the cranial nerves cannot be blocked by epidural injection of local anesthetics. Even with very high sensory blocks there are areas of sensation that will be unaffected by local epidural anesthetics because they are innervated by afferent fibers in the cranial nerves, though it is possible to completely block the motor breathing apparatus by high epidural anesthesia despite loss of consciousness because the phrenic nerve, which innervates the diaphragm, arises from C3 to C5. The oculomotor nerve contains the pupilloconstrictor fibers that induce miosis after opioid administration. Preservation of this response may provide a potential clue to distinguish high epidural anesthesia from total spinal anesthesia in that the latter may induce pupillary dilatation and loss of the light reflex even in the presence of opioids.[45]

SYMPATHETIC NERVOUS SYSTEM BLOCK

As with spinal anesthesia, the most important physiologic alteration produced by an epidural block is sympathetic nervous system block leading to pooling of blood in the

large capacitance venous system of the visceral compartment. The result is a reduction in preload and a decrease in cardiac output and systemic blood pressure. As the sympathetic nervous system block extends into the higher T1 through T4 spinal nerves, there is interruption of the cardioaccelerator fibers that control myocardial contractility and heart rate. Because of the sympatholytic effects of epidural anesthesia, patients with low blood volume or other causes of reduced venous return such as pregnancy, ascites, or vena cava obstruction are prone to exaggerated decreases in systemic blood pressure. Additionally, parasympathetic nervous system innervation of the heart is not impaired. Vagal reflexes can therefore produce significant bradycardia and even sinus arrest during epidural anesthesia.

In contrast to spinal anesthesia, the onset of sympathetic nervous system block produced by epidural anesthesia is generally slower, and the likelihood of abrupt hypotension is less. β-Agonist effects from the systemic absorption of epinephrine in the local anesthetic solution produce sufficient vasodilation to accentuate systemic blood pressure decreases when compared with those produced by local anesthetics alone.

Opinions vary regarding whether sympathetic nervous system block from epidural anesthesia is advantageous or deleterious in a normovolemic patient. Sympathetic nervous system denervation of the bowel increases mucosal blood flow and peristalsis, which may hasten the return of bowel function.[46] Thoracic epidural anesthesia with selective block of cardiac sympathetic fibers favorably alters myocardial oxygen supply, reduces cardiac ischemic events, decreases myocardial infarct size after coronary artery occlusion, and improves functional recovery from myocardial stunning in experimental animals.[47] Surgical bleeding is less for some procedures during the hypotension produced by epidural anesthesia. Disadvantages of sympathectomy include loss of the body's compensatory mechanisms in response to surgical bleeding and the risk for stroke, spinal cord ischemia, or myocardial infarction if systemic blood pressure is persistently or dangerously low. Additionally, compression of the dural sac by the large volume of epidural fluid may increase pressure in the subarachnoid space and elevate the systemic blood pressure required for adequate perfusion of the spinal cord.

MOTOR BLOCK

Motor block results in difficulty with ambulation after lumbar epidural anesthesia. This complication can impede recovery by restricting the patient to bed rest. Diaphragm function is unaffected by epidural anesthesia unless the motor block rises into the upper cervical nerve roots. With surgery in the thorax and upper abdominal region, epidural anesthesia has favorable effects on respiratory function because it prevents pain-induced splinting and permits uninhibited coughing and deep breathing.

OUTCOME

Surgery is associated with increased catabolism that results in loss of muscle protein and negative nitrogen balance. Adequate epidural anesthesia can prevent this catabolic response after surgical procedures on the lower abdominal region and lower extremity. More critically, there is evidence to suggest a reduction in morbidity when epidural analgesia is utilized for thoracic or major abdominal procedures. For example, a meta-analysis identified a less frequent incidence of cardiovascular and gastrointestinal complications, a shortened period of mechanical ventilation, and a reduction in the incidence of renal insufficiency with postoperative epidural analgesia versus systemic opioid analgesia for abdominal aortic surgery.[10]

Side Effects and Complications

Side effects of epidural anesthesia resemble those described for spinal anesthesia, with the added risks of accidental dural puncture, accidental subarachnoid injection, and local anesthetic systemic toxicity, the latter attributable to the high doses of local anesthetic required for the epidural anesthetic. Additional potential complications include epidural hematoma and epidural abscess, particularly in patients with preexisting coagulopathy or infection.

EPIDURAL HEMATOMA

Although potentially attributed to bleeding from vascular trauma during placement of the epidural needle or catheter (or both), it is recognized that both epidural hematoma and epidural abscess may occur spontaneously. If an epidural hematoma is suspected, urgent performance of magnetic resonance imaging is needed because recovery of motor function correlates inversely with the time until surgical decompression of the epidural hematoma.[48]

ACCIDENTAL DURAL PUNCTURE ("WET TAP") AND HEADACHE

Theoretically, epidural anesthesia, which avoids dural puncture, should circumvent the problem of post–dural puncture headache. Unfortunately, inadvertent trespass of the dura does occur, with the incidence greatly affected by the experience of the anesthesia provider. Moreover, if a "wet tap" does occur during attempted performance of epidural anesthesia, the risk for post–dural puncture headache is far greater than after deliberate dural puncture with a small pencil-point needle. Post–dural puncture headache that occurs in the absence of a recognized dural puncture probably reflects the fact that the CSF leak may be too small to be detectable through the epidural needle. When fluid appears at the hub of the epidural needle, it may be difficult to distinguish between CSF or saline used in the syringe to determine loss of resistance. One method to determine the source of fluid is to allow some of it to drip on the anesthesia provider's forearm. The saline, having been administered at room temperature, will be cool

and thus easily distinguished from warm CSF. However, concern for infections such as human immunodeficiency virus, which is concentrated in CSF, has largely relegated this technique to historical interest.

Management

Accidental dural puncture may be managed by converting to single-injection or continuous spinal anesthesia, or epidural anesthesia can be attempted at a different lumbar interspace. Placement of an epidural catheter at another interspace, or passage of a subarachnoid catheter, may decrease the risk for post–dural puncture headache.

SYSTEMIC HYPOTENSION

As with spinal anesthesia, systemic hypotension parallels the degree of sympathetic nervous system block. However, because the onset of sympathetic nervous system block is slower, excessive decreases in systemic blood pressure do not usually accompany epidural anesthesia administered to normovolemic patients. Treatment of hypotension is as described for spinal anesthesia.

SYSTEMIC ABSORPTION AND INTRAVASCULAR INJECTION

The large doses of local anesthetics required for epidural anesthesia plus the presence of numerous venous plexuses in the epidural space increase the likelihood of substantial systemic absorption of local anesthetic. Nevertheless, the resulting blood concentrations of local anesthetics are rarely sufficient to produce systemic toxicity, especially if epinephrine is added to the local anesthetic solution. However, accidental intravascular injection of local anesthetic will produce high blood levels and predictable toxicity ranging from mild central nervous system symptoms (restlessness, slurred speech, tinnitus) to loss of consciousness, seizures, and cardiovascular collapse. Cardiac toxicity is of particular concern with the use of bupivacaine and related anesthetic compounds (see Chapter 11).

ACCIDENTAL SUBARACHNOID INJECTION

Accidental subarachnoid injection of the large volumes of local anesthetic solution used for epidural anesthesia may produce rapid progression to total spinal anesthesia. Immediate treatment is focused on supporting ventilation and restoring or maintaining hemodynamics. However, in contrast to an excessive block produced during spinal anesthesia, the large epidural doses of local anesthetics injected into the subarachnoid space can result in permanent neurologic deficits because of the neurotoxic effects of these agents. In the past, such concerns were limited to chloroprocaine. However, reports of injury have established the potential for neurotoxic injury with the subarachnoid administration of epidural doses of lidocaine and probably any local anesthetic.[49] Consequently, consideration should be given to irrigation of the subarachnoid space with saline, particularly if CSF can be readily aspirated from the misplaced catheter. This maneuver may circumvent or minimize neurologic injury.

Although readily diagnosed in an awake patient, subarachnoid injection may go unrecognized when epidural anesthesia is used in conjunction with a general anesthetic. An unexpected dilated nonreactive pupil after local anesthetic injection into an epidural catheter may indicate migration of the catheter into the subarachnoid space.

SUBDURAL INJECTION

The subdural space is difficult to enter deliberately because the arachnoid is generally closely adherent to the overlying dura. The rare occurrence of subdural injection is difficult to detect because CSF cannot be aspirated through the catheter and the usual test dose is negative. Subdural injection of a local anesthetic solution can produce an unusual block characterized by patchy sensory anesthesia and often unilateral dominance. Subdural placement of an epidural catheter is dangerous because the catheter can abruptly pierce the thin arachnoid membrane and thereby enter the subarachnoid space.

NEURAL INJURY

Neural injury after an epidural anesthetic is very rare but seems to be more likely if a paresthesia occurs during performance of this technique. The development of paresthesia as a result of the advancing epidural needle reflects stimulation of a nerve root and is a signal to the anesthesia provider that the needle is not in the midline and needs to be redirected. As with spinal anesthesia, injection of local anesthetic solution in the presence of a paresthesia is contraindicated because nerve damage may be induced or enhanced by the injection. Nevertheless, occurrence of paresthesia is an inherent risk of epidural anesthesia, and neurologic changes attributed to the development of a paresthesia reflect injury that is almost always transient.

COMBINED SPINAL-EPIDURAL ANESTHESIA

Combined spinal-epidural anesthesia is a technique in which a spinal anesthetic and an epidural catheter are placed concurrently. This approach combines the rapid onset and intense sensory anesthesia of a spinal anesthetic with the ability to supplement and extend the duration of the block afforded by an epidural catheter. The technique is commonly used in obstetric anesthesia (see Chapter 33).

Technique

Combined spinal-epidural anesthesia is most commonly performed by placing a needle in the epidural space, followed by passage of a small spinal needle into the subarachnoid space through the lumen of the epidural

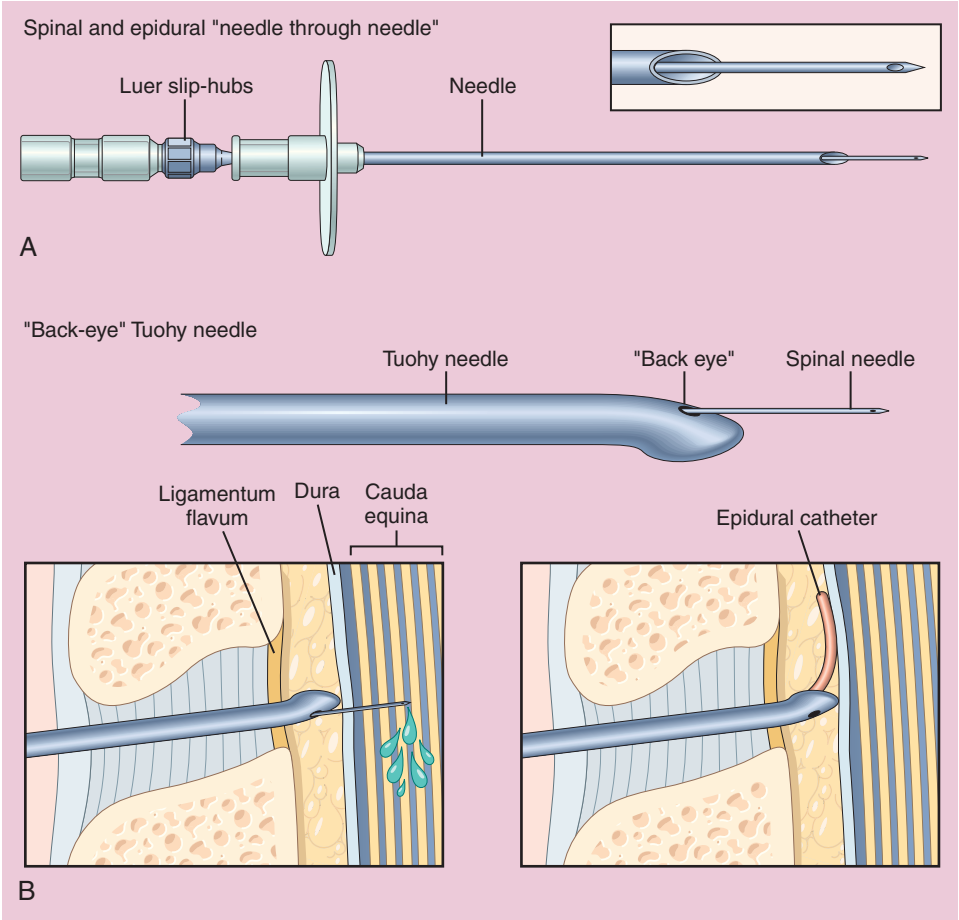

Figure 17-21 **A,** A spinal needle and epidural needle are used for the combined spinal-epidural technique. **B,** Tuohy needle with a "back eye" that permits placement of the spinal needle directly into the subarachnoid space (*left panel*) and subsequent threading of the epidural catheter into the epidural space after removal of the spinal needle. (Modified from Veering BT, Cousins MJ. Epidural neural blockade. In Cousins MJ, Bridenbaugh PO, Carr DB, Horlocker TT [eds]. Neural Blockade in Clinical Anesthesia and Management of Pain. Philadelphia, Lippincott-Raven, 2009, pp 241-295.)

needle. After injection of the local anesthetic solution, the spinal needle is removed and a catheter is threaded into the epidural space through the epidural needle. Although standard spinal and epidural equipment may be used, there are commercially available needles specifically designed for combined spinal-epidural anesthesia (Fig. 17-21).[6]

An undocumented concern associated with combined spinal-epidural anesthesia is that the meningeal puncture site may permit high concentrations of subsequently administered epidural local anesthetics to enter the subarachnoid space or facilitate passage of the epidural catheter through the dura.

COMBINED EPIDURAL-GENERAL ANESTHESIA

Advantages of epidural block during general anesthesia include less need for opioids, pain-free emergence from anesthesia, and block of the stress response that is nearly complete for most surgical procedures performed below the umbilicus. Various modifications of the combined epidural-general anesthetic are used, but if the administration of general anesthesia is not altered by limiting the use of volatile anesthetics and opioids, there is little advantage to the technique. Combined epidural-general anesthesia requires strict attention to fluid management and blood pressure. Sympathomimetics with α-adrenergic activity, such as phenylephrine, dopamine, or epinephrine, can be used to counteract the consequences of afterload reduction, especially in patients at risk for stroke or myocardial ischemia. Excessive intravenous fluid administration to treat hypotension is discouraged because it is often not effective and can lead to intravascular fluid overload as the block recedes.

QUESTIONS OF THE DAY

1. What is the origin of the arterial blood supply to the spinal cord?
2. When inserting a spinal anesthetic, what structures are traversed by the needle during a midline approach? During a paramedian approach?

3. What are the manifestations of "transient neurologic symptoms" after spinal anesthesia? What are the risk factors?
4. What is the usual mechanism of hypotension after spinal anesthesia?
5. What factors determine the spread of local anesthesia delivered in the epidural space?
6. What differences in technique must be used during midthoracic epidural placement compared to lumbar placement?

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