This chapter presents some of the known complications of regional anesthetics through illustrative case reports. Although the case reports are not those of actual patients, they are representative of real cases and incorporate features from actual reports or clinical experiences. Care has been taken to make certain that the depiction of presenting signs and symptoms are true to those actually reported in the literature. Reports are followed by relevant discussion, which includes our present understanding of underlying mechanisms and etiologic factors that are believed to contribute to the development of the complications. Diagnosis and management of complications are briefly covered as well. It is not possible to discuss all known regional anesthetic complications here. Therefore, five clinically important, well-recognized syndromes complicating routine regional anesthetics have been chosen. These should be appreciated and understood by all practitioners in this subspecialty.

Case 1: Permanent Cervical Spinal Cord Injury Following Interscalene Brachial Plexus Block Performed During General Anesthesia

A 17-year-old healthy adolescent girl with a diagnosis of recurrent shoulder dislocation presented for arthroscopic stabilization of her left shoulder. Following the administration of midazolam and propofol in divided doses, the patient was unrousable but breathing spontaneously. Her oxygen saturation (SpO2) was 100% by pulse oximetry. The left interscalene groove was palpated and a 5-cm insulated stimulating needle was inserted at the standard level corresponding to C6. The needle was advanced until a motor response (wrist flexion) was observed at a current amplitude of 0.41 mA, pulse duration of 0.1 ms. After negative aspiration, 40 mL 0.25% bupivacaine with 5 µg/mL epinephrine was injected in divided doses, with negative aspiration before each 5-mL aliquot. Within 20 seconds of the local anesthetic injection, the patient became apneic and her SpO2 decreased to 74%. Positive pressure ventilation with 100% oxygen returned her SpO2 to 100% and the patient was endotracheally intubated following intravenous propofol and vecuronium. After the surgical procedure and discontinuation of general anesthesia, the patient was unable to be extubated and was noted to have dilated pupils bilaterally. She was transferred to the postanesthetic care unit where she required mechanical ventilation for several hours. After extubation, it was recognized that she had a dense left-sided hemiparesis. She devel-
opened bowel and bladder dysfunction and a postdural puncture headache was noted the following day. Magnetic resonance imaging was performed which revealed syringomyelia of the left side of the lower cervical and upper thoracic spinal cord, extending from C6-7 to T1 (see Figure 22-1).

Discussion

This patient received an injection of local anesthetic into the substance of the cervical spinal cord. Intraneural injection of local anesthetic under pressure has been shown experimentally to spread along the longitudinal axis of nerves. Therefore, theoretically, injection into a proximal nerve root could spread directly into the spinal cord. However, Benumof reported four cases in which interscalene block, performed on patients during general anesthesia, resulted in permanent injury to the cervical spinal cord. Imaging studies of the cervical spinal cords of these patients showed varying degrees of vacuolization or a syrinx of the spinal cord. Benumof cited the pattern of injury and lack of damage to structures outside of the cervical spinal cord as providing convincing evidence that injection in each case was made directly into the substance of the spinal cord. Similar medicolegal cases have been reviewed by this author. In each case, the interscalene injection was performed during general anesthesia, which prevented the patient from being able to withdraw from the needle in the event of a painful paresthesia or painful injection. Although performance of interscalene block during general anesthesia is, at present, within the accepted standard of care, general anesthesia should be viewed as a relative contraindication to interscalene block. By contrast to the interscalene block by paresthesia, as originally described by Winnie, the ability to use a nerve stimulator to elicit an appropriate motor response eliminates the necessity for patient feedback or cooperation. Furthermore, profound sedation or general anesthesia can be administered without compromising the ability to elicit a

Figure 22-1. Scan of the T1-weighted sagittal section of the cervical spinal cord magnetic resonance imaging performed 9 months after interscalene block in a patient who received an interscalene block under general anesthesia. A syrinx or cavity is present in the central portion of the right half of the cervical spinal cord. (From Benumof, reprinted with permission.)
motor response to electrical stimulation. Finally, the length of the needle should be appropriate. The brachial plexus is very superficial at the interscalene groove. The plexus can be located and blocked in the majority of patients by using a 2.5-cm needle. Needles longer than 3.75 cm are not necessary for the performance of an interscalene block.

**Significant Neuropraxia Associated with Regional Anesthesia**

Significant neurologic injuries associated with regional anesthesia are indeed rare. Although incidents of significant neuropraxia are, in all likelihood, not underestimated by practicing anesthesiologists, they are certainly grossly underreported. Major neuropraxia usually follows departure from accepted technique, administration of brachial plexus block under general anesthesia, or failure to heed a patient's reaction or complaint of severe pain upon injection.

Several large-scale multicenter clinical investigations support the safety of regional anesthesia. In a recent excellent prospective investigation from France, Auroy et al. studied 103,730 regional anesthetics. Of 34 serious neurologic complications (radiculopathy, cauda equina syndrome, or paraplegia), 21 (62%) were associated with paresthesia or pain upon injection. Thirteen patients did not have associated paresthesia. Of these, 12 were spinal anesthetics, nine of which were with 5% hyperbaric lidocaine. One can generalize that for the most part these serious injuries were either paresthesia-associated or 5% intrathecal lidocaine-associated. Although brachial plexus blocks were not discussed separately, peripheral nerve blocks had an exceptionally low incidence of neurologic complications.

It has been shown, in animals, by Selander et al. that histologic evidence of nerve damage can result when nerves are pierced by needles or when local anesthetic is injected into the nerve. Short beveled needles cannot be counted on to prevent nerve injury, as was shown by Rice and McMahon in a rat model. These investigators found that rat sciatic nerve impaled by short-beveled needles had more frequent lesions that were more severe and took longer to heal than those induced by conventional long-beveled needles. In a study comparing short bevel to conventional beveled needles with regard to postblock neuropraxias, Selander et al. found no significant differences in minor neuropraxias. They reported that all neuropraxias resolved over a 6-month period.

**Should Regional Anesthesia Be Performed on the Anesthetized Patient?**

The above referenced large-scale multicenter clinical investigations support the safety of regional anesthesia; however, if we as anesthesiologists continue to “run red lights” by placing needles in close proximity to the nerves of unconscious patients, we are bound to have an occasional serious, albeit rare, “accident.” Regional anesthesia is frequently performed in patients who are under general anesthesia or heavy sedation. Spinal or epidural anesthesia does not require patient participation for a successful anesthetic. The advent of the nerve stimulator has allowed for high success rates of peripheral nerve or plexus blockade without absolute need of an awake patient who is able to report a paresthesia. The practice of performing regional blocks during general anesthesia has become an accepted standard of care for the pediatric patient. Until recently, there have been no compelling clinical data that absolutely support the hypothesis that regional anesthesia is unsafe in the heavily sedated or fully anesthetized patient. Nevertheless, conventional wisdom, backed by the clinical experiences of experts, has supported the contention that regional anesthesia in an unconscious or anesthetized patient is contraindicated.

Bromage wrote that “Sudden severe pain during placement of a spinal or epidural (or severe pain upon injection during peripheral nerve block) is a time-honored signal
that nerve tissue is endangered and that the needle should be promptly withdrawn
and then resited.\textsuperscript{13} It does not require more than common sense to see that this
crucial patient feedback is lost once the patient is rendered unconscious. That is, the
patient loses his only defense against your needle! Regional anesthetic complications
are in all probability highly underreported.\textsuperscript{15} Nevertheless, there are numerous case
reports that support the notion that regional anesthetic complications are frequently
associated with painful paresthesias or other sensations. These symptoms will cer-
tainly be missed in the unconscious patient. New data strongly question the routine
use of the nerve stimulator in the anesthetized patient.

Moore,\textsuperscript{14,15} another expert regional anesthesiologist, who also had clinical experi-
ence that spanned several decades, has written editorials in support of accepted par-
esthesia techniques of regional anesthesia. In one editorial, Moore wrote, “It is
unfortunately the case that using the nerve stimulator while attempting to locate a
nerve, particularly in unconscious patients, has not avoided neuropathy. We have
reviewed six medico-legal cases in which permanent brachial plexus neuropathy
occurred and in which the nerve stimulator was used.” In a different editorial,\textsuperscript{14} Moore
warned to stop injecting if, upon injection of 0.5–1 mL of local anesthetic, the patient
complains of cramp-like pain. Urmey has reviewed a case in which brachial plexus
block by nerve stimulator in an anesthetized patient was implicated in ipsilateral
phrenic paralysis.

Trentman et al.\textsuperscript{16} reported a case of brachial plexus neuropathy that followed an
attempt at subclavian vein catheterization. During multiple attempts, the patient
noticed the sudden onset of a “hot, electrical” sensation down her arm which resulted
in a “severe lower trunk (brachial) plexopathy” as documented by electromyography.
It is likely that if anesthetized, this patient may have had several more attempts and
potentially further nerve injury.

Barutell et al.\textsuperscript{17} reported on an obese patient who had an interscalene block admin-
istered with an 8.8-cm needle. The patient noted a sharp paresthesia in her right arm
when the needle was inserted and, at the same time, a “brisk jerk of the head
occurred.” The paresthesia worsened upon injection. Despite this warning, injection
was continued and the patient had a respiratory arrest after 8mL and pupils were
noted to be fixed and dilated. The patient subsequently was found to have a permanent
and complete denervation of C8 and T1 nerve roots. It is probable that, if injection
had been stopped, no permanent injury would have occurred.

Urmey and Stanton\textsuperscript{18} reported that they were unable to consistently elicit a motor
response following sensory paresthesia during interscalene block administration. These investigators designed a study to determine if nerve contact by a needle as evi-
denced by a clear sensory paresthesia was necessarily associated with a motor response
upon nerve stimulation up to 1.0 mA.

Twenty interscalene block patients were prospectively studied using the paresthesia
technique of Winnie with a 22-gauge, 3.8-cm (1-1/2”) needle. In 10 patients, a short-
beveled insulated needle was used (Stimex, B-D) and in the following 10 patients a
long-beveled noninsulated needle (B-D) was used. Immediately after the report of a
paresthesia and before local anesthetic injection, the nerve stimulator power was
turned on and amperage slowly increased from 0 to 1.0 mA maximal amperage. Pres-
ence and location of upper extremity motor response, if any, were recorded. After
this, 50mL mepivacaine 1.5% with epinephrine was injected. Interscalene block was
carefully evaluated by a single observer.

All 30 patients had easily elicited paresthesias, 22 to shoulder, six to arm, and two to
the hand. Only 9 of 30 patients (30%) had visible or reported motor response. All blocks
had good evidence of sensory and motor blockade and 26 were judged “excellent” by
blinded orthopedic surgeons. The remaining block was judged as “good.” No patient
required general anesthesia. This study showed that evidence of sensory response
(paresthesia), presumably caused by nerve contact, was not associated with ability to
elicit a motor response in 70% of patients, despite stimulation at milliamperage that exceeds the minimal accepted by most anesthetists. Conversely, this study provided evidence that, if patients are under general anesthesia, a lack of motor response does not guarantee that contact with a sensory nerve fascicle has not occurred.

This was the first clinical study that examined the relationships between a sensory response (paresthesia) and a motor response to electrical stimulation in peripheral nerve or plexus block. Subsequent to the original report of the above data, these findings were confirmed in the axilla in a study by Choyce et al.19 These investigators studied 72 patients during axillary block, using a similar protocol to that published by Urmey and Stanton. Paresthesia was associated with a motor response to electrical stimulation up to 0.5 mA, pulse duration of 0.1 ms in only 77% of the patients studied. Therefore, 23% lacked a motor response.

Mulroy and Mitchell20 reported four cases in which a mechanical paresthesia (“presumed nerve contact”) was obtained before motor response to electrical stimulation during brachial plexus block performance with a nerve stimulator.

All of these reports confirm what experienced clinicians believe, that is, that neither paresthesia nor use of a peripheral nerve stimulator completely rule out the possibility of the needle’s tip being intraneural.21 In fact, in all the devastating cases presented by Benumof using a nerve stimulator, minimal amperage was ≥0.81 mA.

Bollini et al.22 further researched the relationship between a paresthesia and a motor response to electrical stimulation in a study of 22 patients undergoing interscalene brachial plexus block. Interscalene block was performed with an insulated needle coupled to a peripheral nerve stimulator. A motor response was obtained at 0.5 mA in all patients. The nerve stimulator was then turned off and the needle further advanced in the same direction. A mechanical paresthesia was elicited in 21 of the 22 patients. The most likely explanation was that a motor response to electrical stimulation occurred with the needle’s tip at a small distance from the nerve, whereas mechanical paresthesia required nerve contact.

Conclusion

Regional anesthesia is extremely safe. As physicians, we have an obligation to do everything possible to minimize complications when we perform regional blocks. Without doubt, most nerve injuries following regional anesthesia go unreported. Nevertheless, many of the complications that have been reported were associated with sensory and/or motor response warnings. Oversedation, general anesthetics, and muscle relaxants obliterate to certain degrees such warnings, and therefore are not part of what constitutes an optimally safe scenario for regional blockade. It is true that heeding a single warning paresthesia may not always prevent nerve injury in the awake patient. However, the unconscious patient could theoretically have several such injuries that occur during search for a nerve or during intraneural injection without any valuable warning or ability to defend themselves against your needle.

Case 2: Respiratory Failure Following Interscalene Block

A 66-year-old man with a torn rotator cuff presented for right rotator cuff repair. His medical history was only remarkable for a 30-pack-year smoking history and chronic obstructive pulmonary disease. Preoperative pulmonary function testing showed a forced vital capacity (FVC) of 1.8 L (40% predicted). The surgeon called the anesthesiologist 2 days preoperatively to request regional anesthesia, in view of the patient’s pulmonary disease.

The anesthesiologist administered an interscalene block with 30 mL 0.5% bupivacaine with epinephrine. Approximately 7 minutes after the interscalene injection, the
patient became dyspneic and progressively cyanotic, requiring intubation. Attempts to wean and extubate the patient repeatedly failed. Only 12 hours after the interscalene block was the patient finally able to be extubated.

Discussion

Respiratory failure as a complication of interscalene block is very rare. However, respiratory failure can occur secondary to brachial plexus–related pneumothorax, inadvertent subarachnoid or epidural injection of local anesthetic, or as in this case, from diaphragmatic paresis. With proper technique, the incidence of pneumothorax or epidural/spinal anesthesia following interscalene block should be virtually nonexistent. However, diaphragmatic paresis with resultant respiratory impairment has been shown to occur with an incidence of 100%. In one study, this incidence was not reduced by decreasing anesthetic volume to 20 mL. Reductions in routine pulmonary function tests of 20%–40% can be expected within 15 minutes of interscalene block administration. These reductions persist 3–5 hours after mepivacaine interscalene block and at least 9 hours after bupivacaine interscalene block when epinephrine is added.

Anatomic Basis for Phrenic Nerve Paresis

Phrenic nerve paralysis has been reported to occur following supraclavicular block. Various incidences have been reported which may depend on the sensitivity of the diagnostic test used. Nevertheless, it is a frequent accompaniment of supraclavicular block, with reported incidences up to 80%. Urmey et al. reported a 100% incidence of ipsilateral hemidiaphragmatic paresis following interscalene block. This finding has been supported by numerous subsequent studies.

Two anatomic theories have been proposed to explain diaphragmatic paresis. Originally, phrenic paresis was attributed to spread of local anesthetic solution into the space between the anterior and middle scalene muscles. Presently, diaphragmatic paresis is believed to occur by local anesthetic action on the cervical nerve roots C3–C5, which form the phrenic nerve. The brachial plexus is characterized by free communication with the cervical plexus above it. Both plexuses are surrounded by the same fascial confines, the fascia arising from the anterior and middle scalene muscles. Local anesthetic labeled with radiographic contrast has been shown by Winnie et al. to spread freely into the cervical plexus following interscalene injection. Sensory studies by Urmey et al. support that sensory dermatomal levels of C3 and often C2 occur routinely. Thus, motor anesthesia of these same nerve roots can be expected as well and this is the most likely anatomic explanation for phrenic nerve paresis. Another fact that supports this concept is that the timing of sensory anesthesia of these cervical nerve roots coincides with the onset and maximal degree of diminution in pulmonary function associated with interscalene block.

The extremely rare complication of permanent phrenic nerve paralysis can occur from trauma to the phrenic nerve during exploration or injection. The phrenic nerve runs anterior to the interscalene groove. This complication has been reported following interscalene block.

Pulmonary Function Alterations

Consistent reductions in pulmonary function occur following interscalene block. Whereas reductions of 20%–40% in forced expiratory volume in 1 second (FEV₁) and FVC have been found in several studies, diminutions of more than 60% have been observed in isolated cases. These results indicate complete or near complete phrenic nerve paresis. The magnitude of the pulmonary function reductions are similar to those that have been reported following surgical phrenic nerve ablation or complete phrenic nerve paralysis of a pathologic etiology. In a study of direct
phrenic nerve infiltration with 1% mepivacaine in healthy volunteers, Gould and coworkers demonstrated a 27% reduction in vital capacity. The decreases in pulmonary function seem to be largely independent of local anesthetic volume or concentration. Doses of 20–28 mL of 0.75% bupivacaine were found by Pere to result in pulmonary function decreases of 20%–40%. These decreases in pulmonary function as well as altered diaphragmatic motility persisted for at least 24 hours when an infusion of 5–9 mL bupivacaine 0.125% was administered. A mean reduction in FVC of 32.0% ± 8.9% was found in a group of patients following a 20-mL injection of 1.5% mepivacaine for interscalene block.

Contraindications to Interscalene Block

Absolute contraindications to interscalene brachial plexus block include a history of contralateral pneumonectomy or preexisting contralateral hemidiaphragmatic paresis. Relative contraindications include severe chronic obstructive pulmonary disease or any neuromuscular disorder in which a 25% decrease in FVC would not be tolerated. A preblock FVC of 1 L or less is a contraindication to interscalene block. Conditions such as ankylosing spondylitis, in which rib cage motion is restricted, may also place a patient at increased risk of respiratory failure following the diaphragmatic paresis associated with this regional anesthetic.

Another relative contraindication involves patient positioning in the lateral position (with the functionally intact diaphragm down) or any positioning or strapping that will inhibit contralateral chest wall expansion. Respiratory failure has been reported following extubation in a patient who had surgery in the lateral position, with the unblocked side dependent, during combined interscalene block and general anesthesia.

Diagnosis of Hemidiaphragmatic Paresis and Treatment of Associated Respiratory Dysfunction

There are several methods of diagnosing hemidiaphragmatic paresis. These include fluoroscopy, double-exposure chest X-ray, and ultrasonography. The sensitivity of fluoroscopy has been challenged and this criticism also applies to the double-exposure radiographic technique. Ultrasonography of the zone of apposition of diaphragm to rib cage is simple and very sensitive. This technique, in addition to pulmonary function testing, may be used for preoperative evaluation of the patient if preexisting diaphragmatic disease is suspected.

Pulse oximetry is advocated for patients undergoing interscalene block or any brachial plexus injection above the clavicle. Supplemental oxygen is also recommended for such patients. Supplemental sedation should be carefully given and patients observed closely after the block. Equipment to assist or control ventilation should be immediately available, for the rare instances when it is necessary.

Dyspnea developing after interscalene block is rare in the otherwise healthy patient. In studies, we have found that pulmonary function changes occur early after completion of interscalene block and that these changes are essentially complete within 15 minutes. This is reassuring because maximal pulmonary alterations occur when the anesthesiologist is present and therefore fears of progression of dysfunction upon discharge to the floor or home are unfounded.

If a patient develops difficulty breathing following interscalene block, he or she should be reassured and closely observed. In this author’s experience, most patients complaining of dyspnea have more than adequate pulmonary function. Symptoms of dyspnea may be somewhat relieved by placing the patient in the upright or sitting position as tolerated. This position optimizes diaphragmatic geometry and utilizes favorable gravitational effects on the diaphragm to help expand the lungs, increasing functional residual capacity. The sitting position has been found to significantly increase FVC compared with the supine position in patients who underwent interscalene block.
Auscultation of the ipsilateral lung field often reveals diminished (or almost silent) breath sounds. One should be careful not to automatically assume that this implies pneumothorax. Pneumothorax is an extremely unusual complication of interscalene block if the block is performed correctly with a needle of proper length. Chest radiography may be obtained if pneumothorax is suspected. Finally, positive pressure ventilation may be needed in some patients. Assisted or controlled ventilation by face mask, laryngeal mask airway, or endotracheal intubation should be done if clinically indicated.

**Conclusion**

Hemidiaphragmatic paresis is an expected side effect of brachial plexus block performed above the clavicle. An understanding of the onset and duration of the associated pulmonary function changes is crucial to safe management, especially in patients with significant pulmonary disease.

**Case 3: Bradycardia During Spinal Anesthesia**

A 20-year-old female athlete was scheduled for elective knee arthroscopy. Medical history was notable only for the history of fainting. A spinal anesthetic with 60mg lidocaine 2% plain was administered at the L4-5 interspace. The patient elected to watch the video monitor and remained completely alert, refusing sedation. Thirty minutes after the spinal was injected, the patient complained of nausea. Her baseline heart rate of 50 bpm was now 28 bpm. Suddenly, the patient was asystolic. She quickly turned cyanotic despite positive pressure ventilation with 100% oxygen by mask.

**Discussion**

This patient experienced sudden bradycardia during spinal anesthesia. Bradycardia during spinal anesthesia is a potentially dangerous event. All anesthesiologists must have a strategy for identifying patients at risk and should institute immediate treatment when bradycardia occurs. Danger of sudden bradycardia progressing to cardiac arrest was clearly illustrated in a closed claims analysis of unexpected cardiac arrest during spinal anesthesia published by Caplan et al. in 1988. Unfortunately, although all 14 patients analyzed in this study were resuscitated intraoperatively, six of the 14 patients experienced severe neurologic deficits and subsequently died in the hospital. The remaining eight had serious neurologic sequelae. This analysis demonstrated that, although spinal anesthesia is widely regarded as very safe, sudden cardiac arrest may occur rarely. Despite the fact that all patients were healthy, difficult resuscitation with poor neurologic outcomes ensued. Thus, although rare, this complication may be unheralded and disastrous.

**Physiologic Factors in Sudden Bradycardia During Spinal or Epidural Anesthesia**

An imbalance between parasympathetic and sympathetic nervous systems has been proposed to occur in the setting of epidural or spinal anesthesia. With a significant sensory anesthetic level, the patient may be devoid of sympathetic output but have normal parasympathetic innervation. The normal response to sudden bradycardia, for example during fainting, is a reflex increase in sympathetic output. The patient with high-level spinal anesthesia may be unable to mount such a response. This also may explain the reports of difficult resuscitation in bradycardic patients who were otherwise healthy.

Asystole has been described during both spinal and epidural anesthesia. Inadequate filling pressures, decreased vascular resistance, a relatively empty ventricle, and an unaltered or heightened vagal output are often part of a physiologic scenario where
sudden bradycardia or asystole occurs. Caplan et al., in the closed claims analysis referred to above, cited the early use of intravenous epinephrine as a factor in those patients who had more successful resuscitation. Exogenously administered epinephrine acts to restore some of the sympathetic balance, inhibiting bradycardia, increasing cardiac filling pressures, and preserving cardiac output. It also acts directly on the cardiac conduction system with a chronotropic effect on the heart.

**Bezold-Jarisch Reflex**

Much attention has been focused on the Bezold-Jarisch reflex as the cause of sudden acute bradycardia during spinal or epidural anesthesia. The basis of this reflex is a decrease in stretch tension on mechanoreceptors located in the left ventricle. A suddenly empty left ventricle triggers this paradoxical reflex which results in increased parasympathetic activity. Sympathetic output is also inhibited. Anything that decreases left ventricular end-diastolic volume suddenly, such as spinal anesthesia, may trigger this reflex.

By contrast, bradycardia that is slow in onset, developing after administration of spinal anesthesia, has long been recognized and attributed to decreased activity of the cardioaccelerator nerves to the heart. This is a different phenomenon than the sudden bradycardia or asystole in the patient presented above. Complete sympathectomy of the heart itself only reduces heart rate by about 20%.

**Precipitating Factors in the Development of Sudden Bradycardia During Spinal or Epidural Anesthesia**

**Sedatives**

There are many theoretical and real factors that may be associated with the development of bradycardia during spinal or epidural anesthesia. Use of sedatives leading to hypoventilation and hypoxia during spinal anesthesia has been implicated as a possible contributing factor in sudden cardiac arrest in the patient with spinal anesthesia. However, this author has witnessed patients without any sedation experience sudden asystole.

**Neurocardiogenic Bradycardia**

Lack of adequate sedation in an overly anxious patient may contribute to development of sudden bradycardia in the patient during spinal or epidural anesthesia. The single most important identifiable predisposing factor for bradycardia during regional anesthesia in this author's practice has been a history of fainting or syncope. More than one patient with a history of fainting has been observed to develop sudden asystole during spinal or epidural anesthesia. This author has observed an unsedated patient who upon removal of surgical drapes and viewing the surgical dressing, at the end of his procedure, suddenly become asystolic and cyanotic, requiring cardiorespiratory resuscitation. Frerichs et al. have described a similar psychogenic cardiac arrest during epidural anesthesia for knee arthroscopy in a young athletic patient. Increased vagal tone in young or athletic patients may contribute to this syndrome. Often bradycardia is preceded by complaints of nausea, dizziness, or anxiety. Diaphoresis, yawning, or sighing are sometimes noted as well.

In the study by Carpenter et al., factors concluded to be associated with side effects of spinal anesthesia included peak block height, use of hyperbaric preparations of local anesthetics, administration of spinal anesthesia above the L3-4 interspace, and use of procaine. Patients receiving β-blockers had a significantly higher incidence of bradycardia. However, it is this author's opinion, based on experience, that this represents a different phenomenon and that these patients are at no increased risk of development of sudden severe bradycardia. Indeed β-blockers may be protective in patients prone to syncope because they have been used to decrease the incidence of bradycardia and syncope in patients during tilt-table testing.
Tarkkila and Isola\textsuperscript{54} analyzed the predictive value of several variables with regard to hypotension, bradycardia, and nausea. They found that anesthetic sensory levels above T6 and age younger than 50 years were associated with bradycardia during spinal anesthesia.

Cardiac arrest during neuraxial block occurs at a rate of 1.3–18 per 10,000.\textsuperscript{55} In a retrospective analysis at the Mayo Clinic, Kopp et al.\textsuperscript{55} recently examined the frequency of cardiac arrest during neuraxial anesthesia and examined the factors associated with survival. They found an incidence of cardiac arrest of 1.8 per 10,000 patients. Interestingly, spinal anesthesia was associated with an incidence of 2.9 compared with 0.9 per 10,000 for epidural anesthesia. In 46\% of the patients who arrested, the cardiac arrest was associated with a specific surgical event, such as the cementing of an orthopedic prosthesis. Importantly, survival of the arrest was significantly and favorably affected by rapid resuscitation (9 ± 20 versus 34 ± 12 minutes, $P < .001$).

### Treatment of Bradycardia during Spinal or Epidural Anesthesia

The predominant form of bradycardia that occurs during spinal or epidural anesthesia is nonthreatening, slow in onset, is not associated with major hemodynamic changes, and is easy to treat. Atropine or ephedrine will result in heart rate increases in most patients with this form of bradycardia. Bradycardia is often made worse when phenylephrine or any isolated $\alpha$-agonist is used to treat hypotension.\textsuperscript{56}

Bradycardia that is more precipitous or severe should be treated immediately. A direct-acting $\beta$-agonist is necessary and usually effective if administered early, before cardiac output decreases substantially. As discussed above, Caplan et al.\textsuperscript{47} alluded to the importance of early administration of intravenous epinephrine in bradycardia cardiac arrest. Sharrock et al.\textsuperscript{56} have also discussed use of epinephrine to treat or prevent bradycardia. In a study comparing epinephrine to phenylephrine during epidural anesthesia, epinephrine resulted in higher cardiac output, stroke volume, cardiac index, and a more effectively maintained heart rate.\textsuperscript{57}

Asystole occurring during spinal or epidural anesthesia must be treated immediately. One must keep in mind that this event is primarily a circulatory phenomenon and therefore treatment should be aimed at restoring cardiac function. This is not always a simple task in the setting of a high sympathetic block. Precordial thump-pacing has been used in several patients by this author with concomitant administration of intravenous epinephrine (8 $\mu$g is a good starting dose) and atropine (0.8–1.0 mg) with excellent and dramatic effect. Precordial thump-pacing has been reported in case reports of resuscitation following asystole during spinal anesthesia.\textsuperscript{49,58} If sudden asystole occurs during spinal or epidural anesthesia, every second that passes without treatment is crucial. Prolonged asystole diminishes cardiac output and leads to cyanosis in these patients very quickly. Decreased cardiac output rapidly results in an inability to effectively treat with intravenous pharmacologic support. Precordial thump-pacing often results in 1:1 capture and helps to maintain an effective cardiac output allowing definitive pharmacologic support. What begins as a pure circulatory event can quickly become a respiratory problem as well. Oxygen and positive pressure ventilation should be quickly added in the cyanotic patient or if a decrease in oxygen saturation occurs. Availability of transcutaneous pacing or transvenous pacing equipment can also be helpful in some situations. Finally, in rare patients in whom treatment is not quickly instituted, conventional cardiopulmonary resuscitation and advanced life-support measures are needed.

The importance of immediate availability and institution of pharmacologic therapy cannot be overemphasized. At the author’s institution, epinephrine is routinely diluted to 4 $\mu$g/mL and drawn up in a syringe for immediate availability. Atropine, ephedrine, and isoproterenol should also be available. Finally, as when performing any major regional anesthetic, equipment for airway management and positive pressure ventilation should be at hand.
Conclusion

Sudden and severe bradycardia or asystole have been associated with routine spinal or epidural anesthesia. Prospective, controlled studies are needed to identify variables placing patients at added risk. These include clinical, demographic, and anesthetic variables. It seems that younger patients with high vagal tone, patients with a history of syncope or fainting, and patients with higher sensory levels of spinal or epidural anesthesia may be at added risk. However, this complication can occur with any patient. Treatment, initially directed at the cardiovascular system, must be instituted immediately upon onset of symptoms or as soon as the occurrence of bradycardia or asystole is recognized. Even a short delay in providing proper therapy may result in a resuscitation with poor outcome.

Case 4: Epidural Hematoma

A 68-year-old woman without significant medical history presented for an outpatient knee arthroscopy and debridement. The anesthesiologist discussed spinal anesthesia with the patient, who accepted. A 25-gauge pencil-point needle was used. The anesthesiologist attempted the spinal needle insertion at L4-5, L3-4, and L2-3. After marked technical difficulty, successful subarachnoid access and injection was done at L2-3. The surgery and intraoperative anesthetic course were uneventful. However, 1 hour postoperatively, the patient complained of chest pain and she was noted to be in atrial fibrillation. An angiogram revealed a pulmonary embolism. The medical consultant recommended admission of the patient and anticoagulation with heparin. That evening the patient complained of back and leg pain and was started on patient-controlled analgesia with morphine. This resulted in some relief, but the following morning, a motor and sensory neural deficit was discovered in both legs. Emergency magnetic resonance imaging of the lumbar spine revealed an epidural hematoma at L5 extending to L1.

Discussion

Many patients who present for elective or emergency surgery are receiving or will receive drugs that alter coagulation for treatment of coexisting diseases. This is important and something that all practicing regional anesthesiologists must have a strategy for handling. However, what makes this issue a growing concern is the fact that many of the surgical procedures for which we routinely administer regional anesthetics are associated with postoperative thromboembolic events, and our patients typically receive drugs to help prevent these events. Orthopedic, obstetric, urologic, and many general surgical procedures are associated with high incidences of deep venous thrombosis. We are now aware that pulmonary embolism is the leading cause of perioperative mortality in patients who have undergone elective total hip arthroplasty. Because of this, we are becoming more aggressive and successful in reducing the incidence of postoperative thromboembolic events through the use of perioperative, prophylactic anticoagulant and antiplatelet drugs.

One of our most potent means of reducing postoperative deep venous thrombosis (DVT) and pulmonary embolism is the use of epidural or spinal anesthesia. This has been clearly demonstrated in orthopedic patients undergoing repair of fractures or joint-replacement surgery. Regional anesthesia can reduce the incidence of DVT by approximately 50%. Numerous pharmacologic techniques have also been used and combined with regional anesthesia in attempts to further this reduction in DVT. These include antiplatelet drugs, warfarin, and heparin. Most recently, fragments of the heparin molecule called low-molecular-weight heparins (LMWHs) have been produced and are being used in a more widespread manner.
It is important for us to understand the risk to our patients of using such anticoagulant regimens if they are also to receive axial regional anesthetics. The problem is that the scientific literature presently cannot answer the question. In fact, the incidence of spinal or epidural hematoma associated with regional anesthesia has been estimated as less than 1 in 10,000. Thus, to get an adequate power for appropriate statistical analysis, it is necessary to study hundreds of thousands of patients in order to estimate the risk of our practice. This has added importance following the reports of development of hematomas secondary to withdrawal of epidural catheters. Therefore, we may not be safe even in the postoperative period with epidural analgesic infusions.

Some authors have cited articles as being definitive studies that show or argue that there is no heightened risk of epidural or spinal hematoma with concomitant anticoagulant use. One must be careful in making conclusions based on available scientific publications. One example is the claim made by Odoom and Sih from their study of regional anesthesia and perioperative warfarin use. They performed a retrospective review of only 1000 patients. There may have been underreporting of serious complications. In the published report, they stated, “It is concluded that, provided adequate precautions are taken, epidural anesthesia can be safely used in patients receiving anticoagulant therapy.” This irresponsible conclusion is simply unfounded based on this study of grossly inadequate power to answer such a question. This is a potentially devastating complication and practitioners can be falsely reassured by such publications.

Are Anticoagulants Associated with an Increased Risk of Spinal or Epidural Hematoma During Regional Anesthesia?

Association of the development of spinal or epidural hematoma with anticoagulant use has been based on either theory or sporadic case reports. In one review of case reports, Mayumi and Dohi identified 13 cases of spinal hematoma (Table 22-1). Of these case reports, five were associated with the use of heparin. In one case report, the patient had been given an antiplatelet drug. This article may be misleading, however, because there may have been selective reporting of cases.

In a comprehensive review of the literature by Vandermeulen et al., the experience from 1906 to 1994 was analyzed. Forty-two of 61 patients with reported spinal or epidural hematoma had hemostatic abnormalities. Placement of either the needle or catheter was cited as being difficult in 25% and bloody in 25%. Thus, of the 61 reported cases, 53 (87%) had some associated abnormality.

It is important to recognize that regional anesthesia is a technical specialty. Technique seems to be a factor in many of the reported cases of spinal or epidural hematoma. One typical case was detailed in Mayumi and Dohi’s review. A 70-year-old woman had a toe amputation. Ticlopidine (an antiplatelet drug) was started 10 days before surgery. Several laboratory indices of coagulation, including bleeding time, were within normal limits. However, several attempts were made to perform a lumbar spinal anesthetic, and postoperatively a spinal hematoma was identified at the T10 level.

Rao and El-Etr performed a study that is frequently quoted in the anesthesia literature. During the period from 1973 to 1978, 3164 epidurals and 847 continuous spinal anesthetics were studied. Fifty to sixty minutes after placement of the epidural or spinal, 5000 units of heparin were administered to each patient every 3 minutes to achieve therapeutic anticoagulation, defined by an activated clotting time that was twice the normal value. There were no epidural or spinal hematomas identified.

Horlocker and Wedel published a retrospective analysis of 188 patients who received postoperative warfarin after epidural catheters were in place. No incidents of spinal hematoma were identified. But this study was retrospective and only a small number of patient charts were reviewed.
Because of the difficulty of studying sufficient patient numbers to identify a risk of spinal hematoma, in the subsequent study, Horlocker et al.\(^6\) instead looked at the incidence of minor hemorrhagic complications (blood in needle or catheter). They found no documented spinal hematomas or serious neurologic sequelae. There was, however, blood noted in the catheter or needle in 22% of patients. Preoperative antiplatelet therapy did not increase the incidence of bloody needles or catheters. Although the term “minor hemorrhagic complication” was coined by these authors, I am not certain if these occurrences can be correctly termed “complications.” The meaning of their findings is unclear because the appearance of blood in the needle or catheter is a predictable event and has never been linked to serious sequelae.

### Low-molecular-weight Heparin

LMWHs are being used on an increasing basis for surgical thromboprophylaxis. The increasing popularity can be attributed to increased bioavailability of the drugs compared with conventional unfractionated heparin. The drug preparations are also characterized by longer half-lives and less antiplatelet activity. According to a Danish

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**Table 22-1. Spinal Subarachnoid Hematoma after Lumbar Puncture: A Review of Case Reports**

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age of patient (years)</th>
<th>Site of puncture</th>
<th>Underlying disease</th>
<th>Etiologic factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Courtin(^6)</td>
<td>20</td>
<td>?</td>
<td>Syphilis</td>
<td>?</td>
</tr>
<tr>
<td>Hammes(^6)</td>
<td>34</td>
<td>L3-4</td>
<td>Meningitis</td>
<td>Multiple puncture</td>
</tr>
<tr>
<td>King and Glas(^6)</td>
<td>63</td>
<td>?</td>
<td>Cirrhosis, diabetes mellitus, hypertension</td>
<td>?</td>
</tr>
<tr>
<td>Joosten et al.(^6)</td>
<td>74</td>
<td>L4-5</td>
<td>?</td>
<td>Anticoagulant therapy</td>
</tr>
<tr>
<td>Rengachary and Murphy(^5)</td>
<td>64</td>
<td>L1-2</td>
<td>Fractured femur</td>
<td>?</td>
</tr>
<tr>
<td>Kirkpatrick and Goodman(^5)</td>
<td>56</td>
<td>L3-4</td>
<td>T12 compression fracture</td>
<td>?</td>
</tr>
<tr>
<td>Sadjadpour(^7)</td>
<td>61</td>
<td>?</td>
<td>Pulmonary embolism, TIA</td>
<td>Heparin</td>
</tr>
<tr>
<td>Collmann and Rimpau(^7)</td>
<td>36</td>
<td>?</td>
<td>Chronic renal failure</td>
<td>Heparin, difficult lumbar puncture</td>
</tr>
<tr>
<td>Diaz et al.(^7)</td>
<td>55</td>
<td>?</td>
<td>Cerebrospinal fluid blockade</td>
<td>Heparin</td>
</tr>
<tr>
<td>Masdeu et al.(^7)</td>
<td>61</td>
<td>L4-5</td>
<td>Leukemia</td>
<td>Thrombocytopenia Multiple lumbar punctures</td>
</tr>
<tr>
<td>29</td>
<td>?</td>
<td>Diabetes mellitus, frontal subdural hematoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brem et al.(^7)</td>
<td>81</td>
<td>?</td>
<td>TIA</td>
<td>Heparin</td>
</tr>
<tr>
<td>63</td>
<td>?</td>
<td>TIA</td>
<td>Heparin</td>
<td></td>
</tr>
</tbody>
</table>

*Source:* Reproduced from Mayumi and Dohi.\(^6\) with permission from Lippincott Williams & Wilkins. TIA, transient ischemic attack.
survey, 60% of anesthetic departments in Denmark used spinal or epidural anesthesia with LMWH. A 10-year European experience with LMWH has identified six cases of spinal hematomas. Recently, LMWH was approved in the United States. Based on efficacy studies, higher doses than those used routinely in Europe were approved by the Food and Drug Administration. This has resulted in a longer active drug half-life which is more likely to extend the drug activity to the time of postoperative epidural catheter removal. These may be the reasons that in the first 18 months of the United States experience, the same number of spinal hematomas, six, as were reported in Europe were identified in a mere 18 months. This translates to a very significant approximate incidence of spinal hematoma of 0.1%.

According to the most recent guidelines developed at the ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation, patients receiving preoperative LMWH thromboprophylaxis should have needle placement deferred for at least 10–12 hours after the last dose. Higher-dose regimens of LMWH (e.g., enoxaparin 1 mg/kg daily) require delays of at least 24 hours before needle placement.

**Conclusion**

Regional anesthesia is deliberately used in patients at risk for postoperative deep venous thrombosis for its demonstrated benefits. Increasingly, these same patients are receiving concomitant drugs with anticoagulant effects. Although it has not been and may never be definitively demonstrated that these patients are at an increased risk for serious spinal hematoma, there is considerable evidence in isolated case reports of an association of spinal hematoma and anticoagulant use. There is a theoretical and case report–based link of anticoagulant use with spinal or epidural hematoma. Trauma during epidural needle, spinal needle, or epidural or spinal catheter insertion has been associated with hematoma formation. In all likelihood, technical difficulty in block placement places patients at an increased risk of this serious complication.

If necessary to administer spinal or epidural anesthesia to patients receiving heparin, warfarin, or LMWH, careful monitoring of prothrombin time and partial thromboplastin time should be done. Care in placement and removal of the epidural catheter should be taken. If symptoms or signs of epidural hematoma occur, aggressive diagnostic and therapeutic measures must be taken without delay. A delay of a few hours can lead to irreversible neurologic deficit.

**Case 5: High Epidural/Total Spinal**

A 54-year-old man was scheduled for femoropopliteal bypass grafting. His medical history was notable for hypertension and chronic obstructive pulmonary disease attributed to a 50-pack-year smoking history. Because of his chronic obstructive pulmonary disease, an epidural anesthetic was chosen. Epidural anesthesia was administered at the L3-4 interspace with 0.5% bupivacaine with added epinephrine. Fifteen milliliters of the local anesthetic was injected through the 17-gauge epidural needle with the patient in the lateral decubitus position. Upon turning the patient supine, an additional 10 mL was injected through the catheter in 5-mL aliquots. Within 1 minute, the patient had difficulty speaking and the SpO₂ decreased to 82%. The anesthesiologist found that he could aspirate fluid freely from the epidural catheter. He intubated the patient but was unable to wean the patient from the ventilator until the following day.

**Discussion**

High epidural or spinal anesthesia results in significant changes in cardiovascular and pulmonary physiology. It is predominately through effects on these two physiologic systems that high spinal or epidural anesthesia can pose such an immediate threat to
patient well-being, capable of causing rapid morbidity or mortality if not recognized or treated expeditiously. The cardiovascular effects include precipitous decreases in blood pressure, heart rate, and cardiac output. The effects on heart rate secondary to block of cardioaccelerator nerves or rapid vasodilation causing reflex bradycardia are discussed above under Case 3. Pulmonary changes are dependent on the level of spinal or epidural anesthesia. Changes increase in magnitude as anesthetic level progresses from abdominal and thoracic respiratory muscle paralysis to diaphragmatic paralysis. With total spinal or massive epidural anesthesia, function of the medullary respiratory control center may be blocked, leading to respiratory failure.

**Pulmonary Effects of High Spinal Anesthesia**

With more cephalad levels of epidural or spinal anesthesia, the chest wall muscles (rib cage and abdominal) are blocked – in extreme cases leaving the diaphragm to work alone. This approximates the situation in the quadriplegic patient. Under routine epidural anesthesia, the main muscle of respiration, the diaphragm, remains intact and therefore pulmonary function is little changed in most studies. This is in contrast to other regional anesthetics, for example, interscalene block and interpleural block, that affect the diaphragm by phrenic nerve paralysis and may have more profound effects on pulmonary function and chest wall mechanics. Nevertheless, rib cage muscular contraction and diaphragmatic contraction coordinate during normal breathing to move the rib cage in a homogeneous manner. If this coordination is interrupted, the characteristics of normal breathing will change.

In terms of their effects on respiration, epidural and spinal anesthesia can be considered similar entities. However, with lower doses or weaker local anesthetics (e.g., for obstetric analgesia), an epidural may result in a differential block with motor nerve function relatively preserved. Indeed, one study by Freund et al. found reductions in expiratory reserve volume of 48% with spinal compared with only 21% with epidural anesthesia to similar anesthetic sensory levels.

**Pulmonary Function**

Conventional pulmonary function tests are relatively insensitive in detecting changes in the respiratory system caused by epidural anesthesia. Most studies to date suggest that routine spinal and epidural anesthesia have little effect on pulmonary function tests. Urmey and McDonald confirmed this in a recent study on 30 patients during high-dose (25–30mL lidocaine 2%) lumbar epidural anesthesia. Despite the large local anesthetic doses that resulted in a mean sensory level to pinprick of T-5.6, the mean decrease in FVC was only 176mL (P < .05) and peak expiratory flow rate decreased by just 0.34L/second (P < .05). Although these reductions were statistically significant, they are hardly of any clinical importance. This is attributable to the fact that routine pulmonary function tests are much more dependent on lung mechanics than expiratory muscle activity. Indeed, FEV is a clinically useful measurement in diagnosing lung pathology because of this dependence on the lung. The FEV is therefore highly reproducible despite variations in expiratory effort. Conversely, FEV is a relatively insensitive means of assessing changes in expiratory effort when this effort is compromised by epidural anesthesia.

This is supported by recent studies. Sundberg et al. found that high thoracic epidural anesthesia that blocked T-1 through T-5 had little effect on FVC which decreased by about 300mL. FEV had a similar diminution of only 200mL. Takasaki and Takahashi found similar small reductions in respiratory function with cervical and thoracic epidural anesthesia.

**Expiratory Muscle Compromise and Diminished Cough**

By contrast to the subtle changes that occur in pulmonary function tests, cough strength is markedly diminished by epidural anesthesia. We recently found that
patients receiving 0.75% bupivacaine lumbar epidural anesthesia had an approximate 50% reduction in peak intrathoracic pressure during maximal cough within 20 minutes of epidural injection (Figure 22-2). This is similar to the findings of Egbert et al. who found a 53% reduction in intraabdominal pressure change with cough during spinal anesthesia. This occurs because of thoracoabdominal muscle paralysis during epidural anesthesia. Normal contraction of these muscles causes a rapid increase in intrathoracic pressure that produces acute turbulent airflow in the lung. Therefore, the ability to cough effectively may be compromised by epidural anesthesia.

**Ventilatory Control**

Normal ventilation is altered very little by epidural anesthesia. Only small changes occur in tidal volume, respiratory rate, minute ventilation, or arterial \( P_{\text{CO}_2} \). Transient decreases in respiratory rate immediately after epidural injection have been reported. These transient changes may be attributable to increases in cerebrospinal fluid pressure.

Steinbrook et al. studied \( \text{CO}_2 \) response in unpremedicated patients during spinal anesthesia. They found a small increase in the ventilatory response to \( \text{CO}_2 \). Anxiety, chest wall afferent neural blockade, and sedation all may contribute to altered minute ventilation during routine epidural anesthesia. Interruption of the medullary respiratory control center may occur during massive epidural or total spinal anesthesia. This will lead to rapidly progressive respiratory failure requiring immediate intervention and positive pressure ventilation.

**Cardiovascular Effects**

Effects of high spinal or epidural anesthesia on the cardiovascular system can be largely attributed to interruption of preganglionic sympathetic nerves. This predictably results in arterial and venous vasodilation which decreases preload and afterload to the heart. Degree of systematic hypotension is dependent on the level of anesthesia; with more cephalad levels, increasing sympathetic denervation occurs. Change in blood pressure may also depend on preanesthetic volume status and supplemental drugs. Very early studies demonstrated that neuroaxial blockade resulted in diminutions in arterial blood pressure, cardiac output, stroke volume, and total peripheral resistance in volunteers.
Carpenter et al.\textsuperscript{51} prospectively studied 952 patients undergoing spinal anesthesia. The incidences of hypotension (defined as systolic blood pressure less than 90 mm Hg) and bradycardia (heart rate less than 50 beats per minute) with routine spinal anesthesia were investigated. These investigators found a 33\% incidence of hypotension and a 13\% incidence of bradycardia. These incidences were for a group of patients with a mean spinal anesthetic sensory level of T5 \( \pm \) 3. With higher levels or total spinal anesthesia, hypotension and bradycardia would be more prevalent.

The effect of massive epidural or total spinal is to cause venous and arterial vaso-dilation. The result is a decrease in total peripheral resistance as well as cardiac preload. Without pharmacologic support, a significant decrease in stroke volume occurs. Cardiac output decreases despite the reduction in afterload. Sharrock and colleagues\textsuperscript{56} characterized some of the changes in cardiovascular physiology in patients with epidural anesthesia levels at or above T4. Heart rate, mean arterial pressure, pulmonary artery diastolic pressure, cardiac index, stroke volume, and systemic vascular resistance all decreased in a statistically and clinically significant manner.

**Conclusion**

Without vasopressor therapy, massive epidural or total spinal anesthesia will inevitably lead to circulatory collapse and cardiac arrest. It is therefore mandatory to diagnose the complications early and treat them aggressively. Combination \( \alpha \)- and \( \beta \)-agonists are recommended. Pure \( \alpha \)-agonists such as phenylephrine may cause further decreases in cardiac output\textsuperscript{56} and may precipitate or worsen bradycardia. It is important to maintain preload at preanesthetic levels in these patients. Patient positioning can attenuate the effects of venodilation because of sympathectomy. Patients with conditions that inhibit effective venous return such as intraabdominal masses or pregnancy are more susceptible to large decreases in venous return during spinal or epidural anesthesia. Likewise, these patients may be very difficult to treat or resuscitate. Proper positioning, including lateral uterine displacement in the parturient, may be crucial in managing these patients.

**References**


30. Urmey W, Gloeggler P. Effects of bupivacaine 0.5% compared with mepivacaine 1.5% used for interscalene brachial plexus block [abstract]. Reg Anesth 1992;17:13.


