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9

Complications and liability related to regional and neuraxial anesthesia

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Regional anesthesia is responsible for approximately one-fifth of professional liability claims. The present investigation evaluated common and rare complications related to regional and neuraxial anesthesia, including postdural puncture headache, backache, transient neurological symptoms, inadvertent intrathecal injection, epidural hematoma and abscess, meningitis, arachnoiditis, postoperative urinary retention, local anesthetic systemic toxicity, and cardiac arrest. Regional anesthetic techniques are increasingly

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used in perioperative care of surgical patients for acute pain management and for chronic pain states. This manuscript also provides an overview and analysis of the existing literature and makes some recommendations in terms of strategies to prevent or minimize the potential patient injury, with a focus on those more commonly associated with patient injury and liability exposure. The role of ultrasound in preventing patient injury during regional anesthesia is also discussed.

Introduction

Regional and neuraxial blocks are techniques commonly used by all anesthesiologists regardless of subspecialties [1]. A working knowledge of the pros and cons of these blocks is paramount when developing a plan for a specific patient. Neuraxial blocks fall under the category of regional block and include spinal, epidural, sacral, and combined spinal and epidural blocks. Neuraxial blocks can provide surgical anesthesia as the sole anesthetic technique (regional anesthesia) or serve as a postoperative analgesic technique in combination with other anesthetic techniques. Regional anesthesia can be the sole anesthetic technique for all surgical procedures in lower abdomen or lower extremities, such as orthopedic procedures on lower extremities, female reproductive surgery (gynecological procedures or cesarean sections), or male urological surgery especially those involving the bladder or urinary tract (Table 1, [1]). Decisions about using general versus neuraxial anesthesia are based on a number of factors, which include patient's choice, surgeon's preference, anesthesiologist's choice, patient's comorbidities, and postoperative analgesia management. Of benefit, neuraxial anesthesia is associated with a decreased risk of deep venous thrombosis (DVT) and pulmonary embolism (PE). Even though

Table 1
Different types of blocks used in anesthesia practice [3,9].

Type of Upper Extremity Blockades	Interscalene Supraclavicular Infraclavicular Axially Intravenous Regional
Types of Lower Extremity Blockades	Lumbar Plexus Sciatic Femoral/abductor canal Lateral Femoral Cutaneous Obturator Popliteal and saphenous Ankle
Types of Head and Neck Blockades	Cervical Plexus Stellate Occipital Maxillary Mandible Retrobulbar
Types of Truncal Blockades	Intercostal Interpleural Paravertebral Transversus abdominis plane
Neuraxial	Spinal Epidural Caudal Combined epidural and spinal

almost all patients at risk are prophylactically managed with mechanical and pharmacological DVT/PE prevention, the benefit of neuraxial anesthesia must be still considered [2]. A potential drawback to neuraxial anesthesia is the time needed to perform it; neuraxial anesthesia may modestly increase intraoperative time and recovery room time for ambulatory procedures. A study unveiled that neuraxial anesthesia was associated with a 35-min increase in time to start surgery and a 9-min increase in induction time [3].

Recently, it was demonstrated that regional anesthesia reduced the incidence of postoperative pain, opioid consumption both in short term and long term, and other related side effects such as postoperative nausea and vomiting [4]. Possible mechanics of antiinflammatory effects from regional anesthesia include C-fiber block, reduced cytokine production, and sympathetic nerve blockade [5]. As postoperative pain is predominantly caused by tissue inflammation and C-fiber activation due to surgical injury, reduced cytokine production could potentially limit the inflammatory reactions after surgery and the severity of postoperative pain. Cytokines can also affect the development of postoperative hyperalgesia symptoms [4]. Martin and colleagues recently found out that postoperative nerve blocks, regardless of catheter placement and its location, can provide improved postoperative analgesia and fewer opioid-related side effects than predominantly opioid-based analgesic technique. Combined sciatic and femoral nerve block could reduce clinical inflammation (evaluated by local skin temperature and circumference of the knee) after major knee surgery compared with morphine analgesia [6].

Postoperative ileus can be a significant postoperative morbidity and is one of the most common causes of delayed discharge from the hospital after abdominal surgery. The estimated economic impact of postoperative ileus can be as high as $750 million to $1 billion in the United States alone [7]. The consequences of delayed gastric emptying include increased incidence of postoperative nausea and vomiting, increased risk of aspiration, and delayed absorption of medications. Postoperative ileus is an important consideration in discussing the advantages of neuraxial anesthesia such as thoracic epidural anesthesia. The local anesthetics in epidural anesthesia can block afferent and efferent inhibitory reflexes, increase splanchnic blood flow, and likely have some anti-inflammatory effects. Epidural anesthetics offer an additional benefit of blocking the afferent stimuli, which trigger the endocrine metabolic stress response to surgical stimulation and, thus, inhibit the catabolic activity of hormones released during this process that can potentially induce postoperative ileus as well [8,9]. Regional anesthesia is also beneficial in decreasing opioid consumption and minimizing opioid-related decreased bowel function.

Although the direct cardiac and pulmonary effects of peripheral nerve block are negligible, neuraxial anesthesia may cause profound sympathetic blockade with subsequent cardiac output drop and potentially compromise peripheral perfusion (Table 2). The effect of neuraxial anesthesia on a patient's ventilation is largely determined based on the location of the block. Neuraxial blocks in the mid thoracic level have minimal respiratory effects in patients without preexisting pulmonary disease. Intercostal muscles may be weakened/paralyzed due to thoracic block; however, diaphragmatic

Table 2
Complications of nerve block and neuraxial anesthesia [7,9,15].

Complications of Nerve block	Complications of Neuraxial Anesthesia
Direct nerve damage	Hypotension
Hematoma	Bradycardia
Infection	Cauda Equina Syndrome
Drug Error	Epidural abscess or hematoma
Local anesthetic systemic toxicity	High or total spinal anesthesia
Hypotension	Subdural injection
Confusion	Nerve injury
Pruritus	Back pain
Allergic reaction	Postdural puncture headache
Myotoxicity	Infection
Secondary injury	Urinary retention
	Inadvertent epidural/intrathecal injection [15]

function remains intact. Thus, adequate ventilation can still be maintained. Also, resting tidal volume, forced expiratory volume, and forced vital capacity can all be adequate, whereas maximum inspiratory volume and vital capacity may be negligibly decreased. However, neuraxial blocks leading to a high spinal block can impair active exhalation and cough and, consequently, reduce peak expiratory flow, maximal minute ventilation, and expiratory reserve volume [10]. These consequences should be considered in patients with pre-existing issues of pulmonary secretions or obstructive pulmonary disease, as they actively depend on accessory muscles to maintain ventilation. Reduction or even avoidance of opioids and optimization of postoperative analgesia via neuraxial regional analgesia are favorable to postoperative pulmonary function. Individual risk assessment and physician skills are important perspectives to consider and obtain optimal outcome in abdominal and thoracic surgery.

As with any other medical intervention, complications from regional anesthesia or nerve block do arise and should always be considered when formulating anesthetic and analgesic plans for perioperative patients. The incidence of cardiac arrest when using regional anesthesia has been reported to be as high as 0.06%, and this complication frequently results in death or irreversible brain damage [11]. Additional high-severity complications related to regional anesthesia include cauda equina syndrome, epidural hematoma, and unintentional intravenous injections of local anesthetic. Less severe injuries such as postdural puncture headache, insufficient analgesia, transient paresthesias, and back pain are more frequent outcomes of regional anesthesia [12]. As stated earlier, neuraxial anesthesia can also lead to increased intraoperative time and postoperative recovery time.

Regional anesthesia is responsible for approximately one-fifth of professional liability claims. Although many of the complaints regarding regional anesthesia are transient and resolve over a short period of time, there is evidence to suggest that closed claims can be classified as permanent minor injuries. We recently published data suggesting that the highest number of claims are related to brachial plexus injuries associated with interscalene blocks performed for shoulder or rotator cuff repairs (review of 62 claims evaluated from 400 + academic and community institutions between 2006 and 2016) [13]. Furthermore, femoral and sciatic nerve blocks that resulted in lower extremity injuries were the most common nerve blocks associated with a result in payment, and the largest contributing factor to these injuries was cited as "technical knowledge/performance issues." There is additional evidence to suggest that approximately one-half of these claims are associated with obstetric and gynecological procedures [14]. Higher severity injuries continue to result from neuraxial anesthesia–related cardiac arrest and neuraxial hematomas due to coagulopathy-related hemorrhage. Although it is difficult to establish direct cause–effect relationships, the trends in these data sets are clear. The study of closed malpractice claims yields an opportunity to make effective changes from examining adverse events and the associated patient, physician, and technician factors that contributed to the event. This review will aim at discussing the common and rare complications of regional and neuraxial anesthesia and making suggestions regarding strategies to prevent patient injury. It will also discuss the blocks and techniques more commonly associated with injury.

Complications associated with neuraxial anesthesia

As shown in Table 2, regional anesthesia and neuraxial blocks can potentially cause some complications.

Post-dural puncture headache (PDPH)

PDPH is the most common complication after therapeutic and/or diagnostic lumbar puncture. The intracranial pressure (ICP) drops when the production of CSF is inadequate to replace the CSF leakage due to the lumbar puncture [16,17]. There are evidences showing that PDPH is associated with the decrease in ICP, leading to compensatory meningeal venous dilation [18,19] and downward displacement of the brain [20], as well as alterations in craniospinal compliance [16]. Young and pregnant women (18–50 years old) are mainly susceptible to PDPH [21,22]. PDPH may also be related to previous headaches and a low body mass index [21,23]. Procedure-related risk factors have also been revealed, including larger needle size, needle orientation perpendicular to dural fibers, and insufficient experience of the operator [23–26]. PDPH primarily occurs within 72 h after a dural puncture and typically presents as frontal and occipital headache that is dull or throbbing, which is worsened in an upright position and alleviated in supine position; however, the opposite to the traditional postural headache can also occur [27]. Mild-to-moderate PDPH can be relieved by bed rest, hydration, caffeine, and NSAIDs, whereas debilitating PDPH will require an epidural blood patch [28]. Epidural blood patch likely works by two mechanisms: by sealing the dural hole via blood coagulation and clot formation and by squeezing the subarachnoid space, leading to immediate increase in ICP [28,29].

Recently, Cohen et al. also reported using topical sphenopalatine ganglion block to treat PDPH, and they found that patients experienced a quicker headache relief without new complications from sphenopalatine ganglion block, which is believed to be safe, inexpensive, and well tolerated [30].

Backache

Localized point tenderness of the back is a relatively common complaint after neuraxial anesthesia. Although most backaches relieve within several days and do not lead to long-term back pain [31], it was cited as a reason by some patients who refused spinal anesthesia in future surgeries [32]. Traumatic injuries to the ligaments, fascia, bone, or nerve roots during the procedure are believed to cause back pain. The relaxation of paraspinal muscles due to the anesthetic effects can flatten the normal spinal curvature and stretch the paraspinal ligaments and joint capsules. These alterations, especially in the lithotomy position, predispose the patient to experience some postoperative back pain [33]. Various risk factors include preexisting backache, high body mass index, lithotomy position, multiple puncture attempts, and prolonged surgery time [34,35]. Mild back pain is usually self-limited, or controlled with NSAIDs, whereas severe back pain warrants more investigations to rule out the possibility of epidural hematoma or abscess, which may necessitate surgical intervention [36].

Transient neurological symptoms (TNS)

Schneider et al. described TNS after "an uncomplicated spinal anesthesia" in 1994. TNS usually presents as pain and/or dysesthesia originating from buttock area and radiating to the posterior thigh [37]. These symptoms typically start within 2–24 h after spinal anesthesia and last for several days. It can be relieved by ambulation and/or NSAID. The pathogenesis of TNS is not fully understood; previous studies have shown that the onset of TNS is associated with the use of high concentration (5%) lidocaine, significantly less likely by other local anesthetic agents [38]. Risk factors of TNS may include obesity and lithotomy positions [39].

Inadvertent epidural/intrathecal injection

Accidental injection of inappropriate amount or concentration of local anesthetic agents epidurally, subdurally, or intrathecally may cause high or total spinal anesthesia [15]. This may occur a few minutes after injection. Symptoms may include bradycardia, respiratory depression, hypotension, dysphagia, and dysarthria. In severe settings, patients may lose consciousness [40]. Studies on obstetric population reported an approximate incidence of total spinal anesthesia that was 1 out of 3000 cases [41]. When total spinal anesthesia occurs, a patient should be placed on Trendelenburg position, given IV fluids, and administered vasopressor/inotropic drugs to support systemic circulation. Cerebrospinal lavage can also be applied as a rescue strategy [15]. Sometimes mechanical ventilation may be necessary in some patients. Various strategies have been developed to prevent and minimize the incidence of inadvertent intrathecal administration of agents [15].

Epidural hematoma

It is very rare for neuraxial anesthesia to cause epidural hematoma. Puncture of vessels in the venous plexus may cause bleeding into the enclosed epidural space with or without pre-existing coagulopathy. The accumulation of blood in epidural space may result in increased direct pressure on spinal cord or cauda equina and increased risk of ischemia or infarction. Spinal or epidural hematoma can be silent for various amount of time before sudden onset of symptoms such as back pain, lower extremity weakness, and bladder and bowl dysfunction [42]. According to a retrospective study, the incidence of neuraxial anesthesia–induced epidural hematoma is roughly 1 out of 18,000 for epidural anesthesia and 1 out of 158,000 for spinal anesthesia [43]. Risk factors include pre-existing coagulopathy, renal failure, anticoagulation therapy, old age, female, osteoporosis, spine abnormalities, and larger needle size [44]. Epidural hematoma may lead to permanent neurological deficits; therefore, it is imperative to closely monitor patients with indwelling epidural catheter placement and above-mentioned risk factors. If epidural hematoma is suspected, emergency diagnostic investigations should be employed as soon as possible. A surgical decompression given within 8 h after the onset of symptoms may largely reduce the chance of permanent neurological impairment [36].

Epidural abscess

Epidural abscess is also very rare but a serious complication with potentially debilitating permanent neurological sequelae, or even fatal outcome. The incidence rate varies in different studies, and mortality rates are estimated to be 5%–16% worldwide [45]. Bacteria and other pathogen can enter the epidural space and may spread longitudinally, resulting in inflammation and spinal damage via direct pressure, thrombosis, obstruction of blood supply, or bacterial toxins. The initial signs of spinal epidural abscess may be nonspecific, as fever and/or malaise as well as the classic triad of fever, spinal pain, and neurological deficits may not be seen especially in the early stage [46]. Clinical manifestations may progress into localized severe back pain, motor weakness, paresthesia, and paralysis [47]. The major culprit of epidural abscess is staphylococci, followed by gram-negative bacilli and streptococci. Abscesses were also found to be caused by anaerobes and *Mycobacterium tuberculosis* [45]. Routine laboratory findings are not reliable methods of diagnosis, whereas MRI is a much more sensitive tool to determine the location and size of the infection [48,49]. Management of spinal epidural abscess usually includes elimination or removal of the abscess and eradication of causative organisms. This can be achieved by aspiration, drainage, and surgical resection. Empirical antibiotics, including vancomycin and cephalosporin, should be given as soon as the diagnosis is established. Once the causative organism is identified, subsequent sensitive antibiotics are usually provided for 4–6 weeks [50,51].

Meningitis

Meningitis is a very rare but potentially life-threatening complication of neuraxial anesthesia. Meningitis can occur as a result of contaminated equipment or inadequate aseptic technique. Meningitis typically presents with symptoms that can include fever, headache, neck stiffness, nausea, vomiting, and photophobia [26]. The incidence of meningitis following neuraxial anesthesia is estimated to be around 1 in 100,000 neuraxial procedures. However, some retrospective studies have found it to be as high as 1 in 2500 [52]. The most common etiology for neuraxial anesthesia–related meningitis is contamination of the puncture site. This can occur following transmission of aerosolized commensal, contamination with skin flora from the patient or physician, or hematogenous spread from infection of another site. Aerosolization from a physician's breath, despite wearing a face mask, and droplet contamination from other personnel in the room without a face mask during administration of neuraxial anesthesia are proposed mechanisms of infection. This mechanism seems to be verified by matching DNA fragments of organisms from the patient's CSF with what was isolated from the operator's nasopharyngeal swabs [53]. Viable organisms that make up skin flora may still be present in hair follicles even following sterile skin preparation with disinfectants. This provides a pathway to contaminate the epidural space during insertion of an epidural catheter [54]. Strict compliance to aseptic technique should be emphasized during neuraxial anesthetic procedures to minimize the risk of life-threatening complications such as meningitis. Exogenous infectious sources can also be the cause of meningitis in patients undergoing neuraxial anesthesia; for example, the improper storage of donated medical supplies following the tsunami in Sri Lanka in 2004. These supplies were stored at high temperatures and humidity, and despite aseptic techniques, cases of chemical, bacterial, viral, and fungal meningitis were reported after treatment with subarachnoid, epidural, and other combined neuraxial techniques. Packaging was inadequately retaining its sterility under these conditions. Thus, it is crucial to evaluate supplies and storage conditions of all supplies for invasive procedures to prevent devastating complications such as meningitis [52].

Arachnoiditis

Arachnoiditis is the inflammatory reaction and edema of the arachnoid layer of the meningeal sac following an injurious event leading to arachnoid fibrosis, adhesions, and scarring. Arachnoiditis may present in various forms, most commonly as transient nerve root irritation, conus medullaris, and cauda equina syndrome, which may manifest as lower back pain, dysesthesia, and numbness. Arachnoiditis may also be associated with radiculitis, fibrosis, clumped nerve roots, dural sac malformations, syringomyelia, and pseudomeningocele [55]. Arachnoiditis can also result from epidural abscesses, traumatic punctures, use of local anesthetics, and various other substances accidently injected into the spinal canal. Another possible etiology of arachnoiditis is the use of high doses or concentrations of anesthetics and the prolonged contact of these substances with nerve roots, spinal cord, and leptomeninges. Arachnoiditis can also result from chemical inflammation of the reactive vessels that make up the arachnoid layer of the meningeal sac.

Cardiac arrest

The incidence of cardiac arrest as a complication of neuraxial anesthesia has been shown to vary by different methods of investigations and statistics. The incidence of cardiac arrest was found to be higher in patients who received spinal anesthesia when compared to epidural anesthesia, and in those cardiac arrested patients who underwent a neuraxial technique, the anesthetic agents contributed to 54% of the arrests [56]. However, the frequency of cardiac arrest due to neuraxial anesthesia in general is still very low, ranging from 1.5 to 6.4 per 10,000 cases [55]. A common factor involved in the precipitation of cardiac arrest while using neuraxial anesthesia is the development of bradycardia and hypotension. The proposed mechanism likely involves the indirect effects of neuraxial anesthesia on the heart due to increased vagal tone. Neuraxial blocks can cause a decrease in preload that can favor the parasympathetic nervous system, causing bradycardia [55]. Myocardial reflexes are also believed to result in cardiac arrest, specifically by causing bradycardia. Some of these proposed reflexes include the collapse of low-pressure baroreceptors in the right atria and vena cava and the paradoxical Bezold-Jarisch response that causes bradycardia when mechanoreceptors in the inferoposterior wall of the left ventricle are stimulated [57]. Vasodilatation mediated by hypoxemia and hypercarbiacan also result in decreased atrial filling. Vasodilatation and unopposed vagal tone can lead to severe bradycardia and hypotension, which precipitate a cardiac arrest. Respiratory insufficiency can also exacerbate this effect. The combination of factors including higher vagal tone, hypercarbia, hypoxia, and myocardial reflexes can coalesce to precipitate cardiac arrest in patients undergoing regional anesthesia [58].

Postoperative urinary retention (POUR)

POUR is defined as the inability to void after surgery in the presence of a full bladder. This definition encompasses those patients who have an overdistended bladder on ultrasound, even in the absence of clinical symptoms. The traditional symptoms of POUR, such as lower abdominal pain and discomfort, can usually be concealed by neuraxial anesthesia [59]. Bladder anesthesia occurs after blocking afferent signaling from the bladder to the micturition center in the brain. Detrusor strength can return to normal level in 1–3.5 h after ambulation. However, sensory blockade can potentially last for 7–8 h after a spinal injection of local anesthetic agent [59]. The incidence of POUR following neuraxial anesthesia was significantly higher in comparison to general anesthesia, whereas no significant difference was found in POUR incidence after epidural anesthesia compared to systemic analgesia [59].

Local anesthetic systemic toxicity (LAST)

LAST is one of the major concerns when utilizing regional anesthesia/nerve blocks in anesthesia practice. Local anesthetics act by blocking voltage-gated sodium channels in peripheral nerve to block signal transduction. Systemic accumulation of these agents over a short period of time can potentially cause systemic toxicity that can damage the vital organ functions [55]. All local anesthetics have been shown to cause neurological and cardiovascular toxicities in high doses, secondary to unintentional venous injection or accidental overdose. Neurological toxicity can manifest as numbness of the tongue and perioral area initially, evolving into seizures, respiratory failure, and even coma and death. Cardiovascular toxicity usually occurs after clinical presentation of neurological toxicity. LAST-related cardiotoxicity usually cause bradycardia, ventricular arrythmias, or even asystole. These complications are treated by maintaining adequate ventilation and oxygenation, intravenously administering intralipid to reverse the local anesthetic toxicity, using benzodiazepines or other intravenous agents to control seizures, and employing cardiopulmonary resuscitation measures to support cardiovascular stability and systemic circulation. Some patient may have arrhythmias refractory to routine treatment [60]. Caution must be taken when administering local anesthetics, for either peripheral nerve blocks or neuraxial anesthesia, to avoid systemic toxicities from overdose or accidental intravenous administration.

Nerve injury

The incidence of intraneural injection in regional anesthesia is higher than anticipated previously [51]. Intraneural injection and its significance are the hot topics of current debates. Some small-sample-sized clinical studies suggested that intraneural injection may not necessarily be directly responsible for the nerve injury, instead the inflammatory responses secondary to nerve irritation may potentially contribute more to the observed perioperative nerve injury [51]. This finding seems to be validated by some animal studies; Kapur et al. reported that intraneural injections did not always lead to nerve injury in canine model. High injection pressure during intraneural injection may likely be indicative of intrafascicular injection and may, therefore, predict the development of neurological injury [61]. Ultrasound guidance has not yet been proven to reduce the incidence of nerve injury during peripheral nerve blockade [61]. Increased utilization of peripheral nerve block over the last decade did not seem to be associated with an increase in perioperative nerve injury [62]. Avoiding high injection pressure during intrafascicular injection is likely to minimize the likelihood of nerve injury.

Chronic regional pain syndrome (CRPS)

One of the pathophysiological mechanisms for the development of CRPS is direct nerve injury [63]. Peripheral nerve blocks, even guided by ultrasound imaging technique, may cause somewhat direct nerve injury by intraneural or intrafascicular injections [61]. Obviously, these two entities seem to have some overlapping zones for causal relationship.

Prevention of patient injury from peripheral nerve blocks and neuraxial anesthesia should be prioritized while attempting to achieve the desired results of regional anesthesia. Maintaining aseptic technique during the induction of regional anesthesia requires anesthesia providers and anesthesiologist assistants to wash their hands using an aqueous aseptic solution as well as wear a gown, cap, face mask, and sterile gloves. Skin preparation of the puncture site should include disinfection of the skin with chlorhexidine or iodine tincture, followed by a second disinfection after placement of a sterile drape [13]. All supplies utilized in regional anesthetic procedures should be properly stored to avoid contamination. Aseptic technique and proper storage of materials can reduce the risk of meningitis when providing regional anesthesia. Avoidance of intraneural/intrafascicular injection of local anesthetic agent(s) is imperative in minimizing nerve injury. Proper dosing and administration of anesthetics as well as monitoring for the development of bradycardia help reduce the risk of toxicity that can lead to neurological and cardiovascular complications.

Summary

As the current opioid crisis is increasingly becoming a sociopolitical issue in the United States, reduction of using opioid medications perioperatively is highly emphasized. Therefore, multimodal analgesia by adopting strategies utilizing nonopioid analgesics, nonpharmacological measures, peripheral nerve blocks, and neuraxial anesthesia is steadily incorporated into clinical practice in many medical specialties. Nerve block and neuraxial anesthesia are associated with some complications, which may lead to some medicolegal liability exposures for practitioners. These complications may include postdural puncture headache, backache, transient neurological symptoms, total spinal anesthesia, spinal or epidural hematoma, epidural abscess, meningitis, arachnoiditis, cardiac arrest, urinary retention, and local anesthetic toxicity. The strategies to reduce practitioner's liability exposure from the regional anesthesia–associated complications should include strict aseptic technique when performing regional anesthesia procedure, avoid intraneural/intrafascicular injection of local anesthetic agent(s), and make the best efforts to avoid accidental overdose and inadvertent injection to undesired locations.

Practice points

- Neuraxial blocks fall under the category of regional block and include spinal, epidural, sacral, and combined spinal and epidural blocks.
- Neuraxial blocks can provide surgical anesthesia as the sole anesthetic technique (regional anesthesia) or serve as a postoperative analgesic technique in combination with other anesthetic techniques.
- Decisions about using general versus neuraxial anesthesia are based on a number of factors, which include patient's choice, surgeon's preference, anesthesiologist's choice, patient's comorbidities, and postoperative analgesia management.
- A potential drawback to neuraxial anesthesia is the time needed to perform it; neuraxial anesthesia may modestly increase intraoperative time and recovery room time for ambulatory procedures.

Research agenda

- Multimodal analgesia by adopting strategies utilizing nonopioid analgesics, nonpharmacological measures, peripheral nerve blocks, and neuraxial anesthesia is steadily incorporated into clinical practice in many medical specialties.
- Nerve block and neuraxial anesthesia are associated with some complications, which may lead to some medicolegal liability exposures for practitioners.
- The strategies to reduce practitioner's liability exposure from the regional anesthesia-associated complications should include strict aseptic technique when performing regional anesthesia procedure, avoid intraneural/intrafascicular injection of local anesthetic agent(s), and make the best efforts to avoid accidental overdose and inadvertent injection to undesired locations.

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