Expect the Unexpected with Erector Spinae Plane Block in Spine Surgery - Plan for the Worst and Hope for the Best: An Anesthesiologist Perspective

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Abstract
Spine surgery is associated with multiple postoperative complications, ranging from simple nausea and vomiting to devastating complications leading to postoperative morbidity or mortality. The postoperative neurological impairment, especially in the neurologically intact patient, is a dreadful event that makes it difficult for the surgeon to perform technically challenging or high-risk spine surgeries. Preoperative or intraoperative factors that can influence the postoperative neurological status include nature and the severity of the pathology, comorbid conditions of the patient, preexisting neurological symptoms, multiple levels involved, complex surgery or instrumentation, surgical blood loss, neurological monitoring, hemodynamic parameters, polypharmacy, and total duration of the surgery.

In addition to several known contributing factors (fixation failure, epidural hematoma, spinal cord edema, and ischemia-reperfusion injury), the role of the erector spinae plane block (ESPB) has recently been cited as a potential cause of postoperative transient paralysis after spine surgery. ESPB is considered a simple and safe regional anesthesia technique that may have an advantage in success rate and analgesic efficacy when used as an adjunct to general anesthesia in spine surgeries. Despite varied patterns of the drug spread, ESPB has been showing promising results due to consistent involvement of dorsal rami that supply all pain generators of the spine surgeries.

The potential role of ESPB in causing postoperative transient neurological complications is a diagnosis of exclusion that requires thorough clinical assessment and step-by-step evaluation using imaging modalities. Before administering ESPB in spine surgery, essential knowledge includes anatomical and technical considerations, drug distribution patterns, safe and effective volumes/types of local anesthetics, and possible associated complications. This review article describes the possible roles of all factors that lead to postoperative neurological impairment and suggests some tips and tricks for using ESPB in spine surgeries to prevent or manage such serious complications appropriately.

Keywords: Transient paraplegia; Erector spinae plane block; ESP block complications; ESP block in spine surgery; Paraplegia due to RA

Abbreviations: RA: Regional anesthesia; GA: General anesthesia; ESPB: Erector spinae plane block; ERAS: Enhanced recovery after surgery; LA: Local anesthetics; CT: Computed tomography; MRI: Magnetic resonance imaging; ESM: Erector spinae muscles; TP: Transverse process; SMPB: Sacral multifidus plane block; RLB: Retrolaminar block

Introduction
The occurrence of perioperative complications may be inevitable, but their prevention and management are always a shared responsibility of all team members involved. Thorough evaluation of such complications will help develop strategies to prevent and manage the same in the future. A systematic and stepwise approach is warranted before categorizing it as a surgical or anesthetic complication. Several interventions have been introduced in the surgical and anesthetic techniques to improve patient safety and satisfaction. Application of regional anesthesia (RA) alone or as an adjunct to general anesthesia (GA) is one such advance that helps reduce many polypharmacy-related side effects or complications. If a particular complication-reduction modality is inherently causing complications, it requires a comprehensive understanding of the situation and its contributing factors.

An erector spinae plane block (ESPB), a safe and simple RA technique, has shown promising results as an adjunct to multimodal analgesia in various orthopedic, general, thoracic, abdominal, obstetrics, and spine surgeries. In addition to its superior postoperative analgesic profile in spine surgeries at various levels, ESPB reduces hospitalization costs and the possible side effects of extensive anesthetic use. Since opioids have been linked to tumor recurrence [1,2], ESPB also reduces the risk of spine tumour recurrences by significantly reducing its consumption. ESPB meets all criteria suitable for enhanced recovery after
surgery (ERAS) protocol [3] by facilitating early discharge and mobilization of patients. Being a novel RA technique, not many complications have been reported so far except for some anecdotal reports of bilateral quadriceps weakness, transient aphasia, or bilateral fascia lata syndrome. The exact cause of the postsurgical neurological impairment is multifactorial and multi-channel. The pathogenesis of spinal cord lesions after spine surgeries is usually mechanical (pressure) damage via extensive hematoma or edema, resulting in pressure on the spinal cord leading to ischemic damage [7]. An altered cerebrospinal fluid flow dynamic may also cause cord compression [8]. In either case, the ultimate pathogenic cause is a secondary cellular injury due to the disruption of ionic homeostasis, development of free radicals, lipid oxidation, and degeneration of the cytoskeleton [7]. T2 weighted MRI scans can help detect changes suggestive of misplaced implants, hematomas, edema, compressive lesions, white cord syndrome, or direct trauma to the spinal cord. Symptoms due to spinal cord edema typically occur at 48-72 hours post-surgery and may be relieved by anti-edema measures like fluid restriction [19].

Postoperative neurological impairment after spine surgery in a neurologically intact patient is always daunting for the operating surgeon and the patient. Several common theories on neurological deterioration after decompressive spine surgeries include vascular compromise, hypotension, ischemia, direct trauma, or stretching of the neural elements. The major contributing factors of acute paraparesis following spine surgery include fixation failure, epidural hematoma, spinal cord edema, and ischemia-reperfusion injury [6].

Who is the culprit?

The occurrence and severity of ischemia-reperfusion injury correlate with tissue ischemia time, the extent of ischemic tissue, and the oxygen requirement of the affected tissue [20]. The presence of deep tendon and superficial reflexes may rule out the high degree of preoperative stenosis are considered poor prognostic factors [18].

Contributory factors

Neurons in the spinal cord are susceptible to ischemia and hypoxia. The mechanisms of spinal cord ischemia are multi-factorial and multi-channel. The pathogenesis of spinal cord lesions after spine surgeries is usually mechanical (pressure) damage via extensive hematoma or edema, resulting in pressure on the spinal cord leading to ischemic damage [7]. An altered cerebrospinal fluid flow dynamic may also cause cord compression [8]. In either case, the ultimate pathogenic cause is a secondary cellular injury due to the disruption of ionic homeostasis, development of free radicals, lipid oxidation, and degeneration of the cytoskeleton [7].

White cord syndrome, an imaging feature of spinal cord ischemia [9], is diagnosed as high intramedullary signal changes on sagittal T2 weighted MRI scans and is often seen in surgeries on the cervical spine. The spinal infarct is one of the leading causes of paraplegia or quadriplegia in patients with preexisting vascular pathologies (thrombosis) or embolic events during surgery [10]. The anterior spinal cord has a higher risk of ischemia due to fewer anterior spinal artery feeding vessels [10] than the highly vascular posterior spinal cord due to anastomotic pial vessels. The sparing of the posterior column leads to unchanged intraoperative somatosensory evoked potentials [11]. The ischemia-reperfusion injury occurs upon restoring the blood flow to previously ischemic tissues and organs. Increased inflammatory cytokines such as TNFα and IL1β may be considered vital indicators for evaluating decompression-associated spinal cord ischemia-reperfusion injury [12,13]. Its reported incidence is 2-5.7% following cervical and 14.5% following posterior thoracic decompression surgeries [14,15].

Transient paralysis is one such complication that manifests itself as a temporary (up to 72 hours) loss of sensations, movements, anal reflexes, and sphincter function below the affected spinal segments [16]. It can occur after vertebroplasty, laminectomy, or thoracic decompressive procedures [17,18]. The longer duration of symptoms, multiple compression sites, and the high degree of preoperative stenosis are considered poor prognostic factors [18].

Discussion

Postoperative neurological impairment after spine surgery in a neurologically intact patient is always daunting for the operating surgeon and the patient. Several common theories on neurological deterioration after decompressive spine surgeries include vascular compromise, hypotension, ischemia, direct trauma, or stretching of the neural elements. The major contributing factors of acute paraparesis following spine surgery include fixation failure, epidural hematoma, spinal cord edema, and ischemia-reperfusion injury [6].

Role of ESPB

ESPB involves depositing the local anesthetic solution between the erector spinae muscles (ESM) and the transverse process (TP) under ultrasound guidance. The ESM consists of three muscles: iliopectalis, longissimus, and spinalis. They arise from and insert into various bony components of the vertebral column [22] and form a paraspinal column that extends from the sacrum to the base of the skull. It gradually tapers upwards in the paravertebral groove on either side of the spinous processes. The retinaculum (thoracolumbar fascia in the lumbar region) that envelops this muscular column also facilitates the LA spread to several thoracic and lumbar levels [23]. The diverse multilayered fascial arrangement deep to the ESM may cause the inconsistent LA spread, resulting in multisegmented sensory block mainly involving dorsal rami with sometimes ventral rami.
Table 1: Causative factors responsible for postoperative neurological compromise in spine surgery.

| Postoperative neurological compromise in spine surgery- causative factors |
|---|
| 1. Direct trauma to spinal cord/ nerves |
| • During surgery / postoperative compressive hematomas |
| • Misplaced implants |
| • Scars in the spinal canal -secondary pressure trauma |
| • Causes transient loss of sensation, movement, anal reflexes, and sphincter function below the plane of injury |
| • Such paralysis subsides within 72 hours |
| 2. Epidural hematoma |
| • Incidence approximately 1% postoperatively |
| • Most frequently with surgery of metastasis (5.9%) |
| • Common in patients with coagulopathy or multisegmented surgery |
| • Very rarely in anterior cervical spine surgery |
| • Symptoms emerge postoperatively within 1-2 h (cervical spine) or 5-6 h (lumbar spine) |
| 3. Spinal cord ischemia-reperfusion injury |
| • The sudden expansion of the compressed cord and gush of blood following decompressive surgery leads to disruption of blood-brain and blood-spinal cord barrier - causing acute neurological dysfunction |
| • Gradually progressive paralysis occurring within 6 h of surgery |
| • Rapidly progressing motor-to-sensory dysfunction from lower-to-upper extremities |
| • Completely or partially reversed with high-dose methylprednisolone, dehydration and neurotrophic drugs |
| 4. White cord syndrome |
| • Postoperative MRI feature of spinal cord ischemiareperfusion injury |
| • Mainly observed in cervical spine surgery |
| 5. Spinal cord infarcts |
| • Due to pre-existing vasculopathies (thrombosis) or embolic events during surgery |
| • Anterior cord > Posterior cord |
| 6. Hysterical paralysis |
| • Incidence between 0.3% and 3.8% |
| • Caused by psychological factors, mostly in female |
| • The clinical characteristics: |
| ➢ paraplegia, hemiparesis, and single paralysis |
| ➢ normal/ enhanced deep tendon reflex |
| ➢ no pathological reflexes or |
| ➢ other positive signs of the nervous system |
| • Differentiated from the pathological paraplegia by: |
| ➢ lack of symptoms like anal sphincter disorder, skin dystrophy due to bedsores, or muscle atrophy; |
| ➢ normal reflexes; and |
| ➢ associated mental disorders with intolerant and irritable patients |
| • Chronic back pain associated with mental stress-important cause of morbidity. |
| • The psychological trigger during repeated assessment of sensation in legs and feeling (palpation) by the surgeon also induce paraplegia. |
| 7. Local anesthetic drug |
| • Local anesthetic used during regional analgesia technique as an adjunct to general anesthesia. |
| • Used during infiltration before vertebroplasty or during paraspinal blocks. |
| • Paralysis/paraparesis: due to inconsistent spread of LA in neuraxis (intrathecal or epidural), or paravertebral spaces, or plexuses/spinal nerves around it. |
| • Duration of paraplegia: depends on the volume, type and concentration of LA as well as the spread pattern of drug. |
| • Onset and recovery follows a differential blockade pattern. |
This para neuraxial block, when given bilaterally in spine surgery, can be advantageous in success rate and analgesic efficacy [24]. The absence of risks such as hypotension, vascular spread, or pneumothorax makes ESPB relatively safer than epidural anesthesia or paravertebral block. Bilateral ESPB offers effective perioperative analgesia without influencing the hemodynamic parameters. It significantly reduces the perioperative opioid requirements in spine surgeries at various levels (cervical, thoracic, and lumbar, and sacral) [25-32]. Its outcome depends on the volume and concentration of LA used, drug spread, and the anesthesiologist's experience in selecting and locating the correct level of the TP.

The exact mechanism of action of the ESPB block and pattern of the drug spread is still unclear. It has been suggested to anesthetize the spinal nerves by passing through the costotransverse foramen of Cruveilhier, accompanying the dorsal ramus and artery to the paravertebral space [33]. The deposited drug can spread in any direction, such as craniocaudal, anterior-posterior, and lateral-medial planes to reach the paravertebral area, intervertebral foramina, and epidural space, or sympathetic chain [34-38]. Fluoroscopic, CT, and MR imaging in living subjects have similarly confirmed the injectate tracking to the paravertebral area, intervertebral foramina, and epidural space following lumbar ESPB [39-42]. There is also a possibility of LA diffusion through the microscopic gaps in the mostly acellular architecture of interlinked collagen fibers of the fascia covering the erector spinae muscle [43].

**ESPB at various spine levels**

The anatomical differences at the various spine levels can cause varied drug spread and ultimately affect the outcomes of ESPB. Cervical ESPB is technically challenging due to the difficulty in identifying the tips of the cervical transverse processes due to their shorter length. It is mainly given at the C6 or C7 vertebral level. The probe needs to be kept anterolaterally rather than posteriorly to see the cervical TPs [44]. It may not be safe due to its proximity to the neuraxis (shorter transverse processes) and the possibility of bilateral phrenic nerve involvement [45-48].

Thoracic ESPB at the upper vertebral levels (T2 or T3) can be preferred in cervical spine surgery by inserting the needle from caudal-to-cranial direction to achieve the desired LA spread and avoid technical difficulties and complications associated with cervical ESPB. Thoracic ESPB can provide multilevel analgesia even with the small volumes of LA due to rigid boundaries of the thoracic paravertebral spaces that facilitate drug spread at several levels involving ventral and dorsal rami. Lower thoracic level ESPB is mainly performed for lumbar spine surgeries by inserting the needle from cranial-to-caudal direction to achieve the desired LA spread and avoid technical difficulties associated with lumbar ESPB [49,50].

The lumbar ESPB can also be technically challenging due to the increased thickness of the ESMs with their tendinous attachment to the TPs [51,52] and increased corresponding depth of the intermuscular plane in the lumbar region. The psoas muscle is also closely adherent to the vertebral bodies and the anterior surface of the TPs. The anterior drug spread to include ventral rami may be compromised due to the lack of clear boundaries of lumbar paravertebral spaces [53]. There is a communication through the fat-filled plane between the ESM and TP with the fat-filled psoas compartment containing lumbar nerve roots and plexuses. The spread of LA to the epidural space is possible through this communication [54]. The compressed lamina and the ligaments of the lumbar spine favor LA spread more into the epidural space [55, 56]. Thus, the lumbar ESPB may result in either lumbar plexus block or epidural anesthesia. The resultant weakness in the quadriceps or lower extremity muscles depends on the LA concentration and volume used in ESPB.

Sacral ESPB is mainly described for gender reassignment surgery or perineal surgery [57-61]. Its application for lower lumbar or sacral spine surgery is yet to be determined. The sacral multifidus plane block (SMPB), one of the variants of the paraspinous block, involves the deposition of LA in the plane under the multifidus muscle and bony area between the median and intermediate crests of the sacrum. The possible mechanism of action of SMPB includes blocking the dorsal ramus and medial cluneal nerves directly by LA deposition and ventral rami by anterior LA spread through dorsal and ventral sacral foramina. The SMPB may also block the pudendal nerve (S2-S4), lumbosacral plexus, and sciatic nerve via the anterior and cranial LA spread [61, 62].

**The role of LA**

The possible role of LA used in ESPB in causing postoperative neurological compromise depends on its inadvertent spread into either the epidural or subarachnoid space. It can be determined based on the occurrence and recovery pattern of the neurological symptoms. Distal-to-proximal and motor-before-sensory recovery patterns are the hallmarks of the differential blockade of the LA [23]. Inadvertent spread of LA into the subarachnoid space can lead to severe hypotension and bradycardia, resulting in unstable intraoperative hemodynamics. The consequences of the epidural spread depend on the density of LA around the spinal nerves, which could be compromised in a subsequent surgical dissection affecting the potentiality of the epidural space.

The concentration of LA, which determines the mass of the drug, also affects the efficacy of any block. The deliberate use of LA in low concentrations can result in a preferred motor-sparing analgesic effect of such high-volume blocks [63, 64]. Bupivacaine and ropivacaine are the most commonly used LAs for bilateral ESPB. Both LA agents consistently display preferential blockade of C-fibres (slow pain) > A-delta fibers (fast pain) > A-beta fibers (touch/pressure) in both preclinical and clinical studies [64-66]. With the increasing concentration, these agents may result in loss of proprioception and loss of motor function. Lipid solubility and higher pKa of LA facilitate intraneural diffusion and ion channel...
blockade. Ropivacaine exhibits a relative motor-sparing effect due to its lower lipid solubility than bupivacaine [67]. Twenty milliliters of 0.375% ropivacaine is recommended for each side of the bilateral ESPB in adults [68, 69].

**Technical aspects of ESPB**

**Table 2: Tips and Tricks of using Erector Spinae Plane Block in spine surgeries.**

| Tips and tricks of using erector spinae plane block in spine surgeries |
|---------------------------------------------------------------|
| **1. Sterility:**                                             |
| • All blocks should be given under strict aseptic precautions. |
| • Always better if given under antibiotic coverage.          |
| **2. Ultrasound probes:**                                    |
| • **Linear high frequency probe:** For cervical, dorsal, sacral spine levels due to superficial location of transverse process |
| • **Curvilinear low frequency probe:** For scanning of all levels (cervical, thoracic and lumbar) for better visualization of the typical "trident sign" of transverse processes and tracing of the extent of drug spread under ESM. |
| **3. Local anesthetics:**                                    |
| • Diluted concentration of LA (ropivacaine 0.1- 0.2% or anawin 0.125%-0.375%) are recommended as this is a volume-dependent interfascial plane block, given mainly for analgesia purpose |
| • LA agents having motor-sparing effect (like ropivacaine) are used |
| **4. Selection of Appropriate level:**                        |
| • It depends on the innervation of all structures (osteotomes, myotomes and dermatomes) of the surgical field. |
| • After determining extent of spinal segment by considering innervations, midpoint of all the segments is selected. |
| • It is always better to give block after the extent of the incision is marked by the operating surgeon under C-arm guidance |
| **5. Ergonomics:**                                           |
| • Standing on the ipsilateral side (same side of the block to be given) with the ultrasound machine kept on the opposite side exactly in front of the regional anesthetist. |
| • Ultrasound machine, block site, and regional anesthetist should be one line. |
| • Changing side of both anesthetist and the ultrasound machine for opposite side injection |
| **6. Timing:**                                               |
| • For effective analgesia, block should be given at least 20 minutes before skin incision |
| **6. Landmarks:**                                           |
| • Identification of transverse process is difficult in cervical spine due to very short length. |
| • Identification of transverse processes in the thoracic region is easier by differentiating the appearance of ribs (round contoured) from the transverse process (squared contoured). |
| • Technically easier in thoracic region as compared to the cervical and lumbar region. |
| • Due to absence of ribs in lumbar region, identification of transverse processes is easier but technically difficult due to deeper location of target structure. |
| • It is always better to select upper thoracic level for cervical spine surgery or lower thoracic level for lumbar surgeries for the patient with difficult anatomy like obesity. |
| **7. Avoid:**                                               |
| • Patients having severe myelopathic symptoms. |
| • Patients having neurological deficit or any kind of neurological compromise. |
| • In severely stenotic multisegmented decompressive surgeries. |
| • Patient having compromised single or multiple level nerve roots. |
| • Block in such situation will lead to "double crush phenomenon" due to increased susceptibility of nerve injury in pre-existing neural compromise conditions. |
| • Patient having risk factors for spinal infarct like pre-existing vascular diseases or possibility of embolic events during surgery. |
| • Block can be avoided in infected conditions/traumatized vertebral segments |
| **8. Prevention:**                                          |
| • Intraoperative electrophysiological monitoring is recommended as a useful tool to aid in early diagnosis and avoidance of neurological deterioration in a high-risk patients with multilevel decompression surgeries or surgeries in severe myelopathy. |
| • High dose steroids, neurotrophic drugs or dehydration helps to reverse most of the condition causing surgical neurological compromise. |

(ESM: Erector spinae muscles, LA: Local anesthetics).
Unexpected outcomes like a neurological compromise can be correlated with possible technical errors while administrating ESPB. The first technical aspect is identifying the correct landmark under ultrasound depending on the surgical extent and the desired level of the block. It may further depend on the sonoanatomy quality and the experience of the anesthetist. Sometimes misidentifying the lamina as the tip of the TP can lead to the retrolaminar block (RLB), another variant of the paraspinal block. In RLB, the needle insertion is slightly medial, targeting the lamina of the vertebra instead of the tip of the TP. It works via diffusion of LA into the paravertebral space through the soft tissue gaps between adjacent vertebrae [70]. Both RLB and ESPB were consistently associated with the posterior spread of injectate to the back muscles and fascial layers [37].

Fluoroscopic-guided ESPB can lead to RLB due to the inability to see the tip of TP dearly like under ultrasound, resulting in deposition of the LA solution over the lamina. The proximity of the RLB to the neuraxis can lead to a high probability of epidural spread, which carries the risk of motor weakness. The second important aspect is the ergonomics associated with bilateral ESPB. Administering the bilateral ESPB by standing on only one side of the patient may result in deviation from the ideal needle trajectory on one side compared to the other. Therefore, technical considerations should focus on stabilizing the needle by one person, injecting LA by another person, and performing such bilateral blocks while standing on either side.

The third important aspect includes technical modifications such as keeping an ultrasound probe in a transverse view to help differentiate intramuscular drug spread from the effective linear drug spread between ESM and TP [71]. The fourth aspect is finding alternatives that involve dorsal rami consistently without causing drug spread to other unwanted areas. The thoracolumbar interfascial plane block is one such alternative that targets only the dorsal rami of the spinal nerve. Thus, it can provide more focused dermatomal coverage of the back required for thoracic and lumbar spine surgeries [72, 73]. However, its efficacy in spine surgeries is yet to be determined. We have suggested some tips and tricks for using ESPB in spine surgeries (Table 2), keeping all technical aspects in mind.

Conclusion

Postoperative neurological impairment following spine surgery is a serious concern for the operating surgeon and the patient. The role of ESPB in causing such complications is the diagnosis of exclusion made after a thorough evaluation of clinical symptoms and radiological studies. For that, understanding of various mechanisms involved in ESPB leading to neurological impairment is essential. It should encourage the anesthetists to take extreme precautions while administering this novel block, considering the anatomical differences at various spine levels. Surgeons should anticipate and explain the possibility of neurological deterioration while explaining the risks and benefits of the proposed surgical intervention. Intraoperatively, real-time neurophysiological monitoring is recommended as a useful tool to avoid further neurological deterioration, especially in extensive and multilevel surgeries or in high-risk and neurologically compromised patients.

After identifying or diagnosing such complications, intensive care and regular checking of spinal function are of great importance, along with simultaneous radiological workups to rule out various causative factors. Once paralysis occurs, early diagnosis and early intervention are essential in restoring spinal function. Despite the rare possibility of such complications, ESPB is still a promising option for ensuring effective perioperative analgesia in spine surgeries. It helps reduce postoperative morbidity by keeping the hemodynamic parameters stable and significantly reducing intraoperative blood loss. It can also avoid postoperative complications that lead to delay in mobility and discharge by significantly reducing the need for opioids and polypharmacy. However, further studies are needed to determine the safe concentration and volume of the LA solution used in ESPB, the exact surgery-specific vertebral level to cover desired surgical innervations, and the accurate LA deposition site to prevent spread to undesired areas.

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