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REVIEW

The Application of Erector Spinae Plane Block in Chronic Pain Management: From Anatomical Mechanisms to Clinical Innovations

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Abstract: Erector Spinae Plane Block (ESPB) is a fascial plane-based regional analgesia technique that involves injecting local anesthetics between the deep aspect of the erector spinae muscle and the transverse processes of the vertebrae. This blocks the dorsal rami of spinal nerves, thereby achieving multi-segmental analgesia. With the widespread adoption of ultrasound guidance, ESPB has gained extensive application in acute and chronic pain management due to its relative simplicity and high safety profile. However, its systematic evaluation and value positioning in chronic pain management remain to be clarified. This article aims to provide a comprehensive overview of ESPB, spanning from its anatomical basis to clinical innovations. It focuses on reviewing its mechanisms of action, clinical efficacy, technical advantages, and limitations in managing chronic pain (including cancer-related pain and neuropathic pain). Future research directions are also discussed to inform clinical practice and related studies.

Keywords: erector spinae plane block, chronic pain, anatomical mechanism, clinical application, technological innovation

Introduction

Global Burden of Disease (GBD) studies have established pain-related disorders as the predominant cause of years lived with disability (YLDs) worldwide.[1] In North America alone, chronic pain affects over 20% of the adult population,[2] significantly impairing quality of life and imposing substantial personal and socioeconomic burdens.[3,4] While pharmacotherapy remains a mainstay of treatment, its efficacy is often limited by suboptimal outcomes and significant adverse effects, which constrain its clinical utility. Consequently, the development of effective and minimally invasive interventional analgesic techniques has become a critical objective in pain medicine.

Regional nerve block techniques have gained increasing prominence in managing both acute and chronic pain due to their procedural simplicity and favorable safety profiles. Among these, the ESPB—a relatively novel truncal nerve block first described in 2016—has rapidly emerged as a focus of research and clinical interest. Its appeal stems from its unique anatomical targets and potential for broad analgesic coverage.[5] The clinical applications of ESPB have expanded considerably from initial use in thoracic neuropathic pain to include various chronic pain conditions, such as chronic post-surgical pain syndromes and cancer-related pain.[6] However, the widespread adoption of this technique is hindered by the lack of a systematic evidence synthesis and robust efficacy assessments. The current literature primarily consists of case reports and small-scale observational studies, which exhibit considerable heterogeneity in quality.[7–9] Furthermore, key questions regarding its mechanism of action, patterns of drug diffusion, and comparative effectiveness against established techniques like epidural or paravertebral blocks remain unresolved, resulting in a fragmented knowledge base.

This review aims to synthesize the current evidence on the mechanisms and clinical applications of ESPB in chronic pain management. We will analyze its anatomical and pharmacodynamic foundations and summarize its advancements in treating cancer pain, neuropathic pain, and other chronic disorders. The performance of ESPB will be compared with conventional techniques, such as epidural and paravertebral blocks. Finally, we will explore the potential of ESPB in

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preventing chronic post-surgical pain, evaluate its safety profile and associated complications, and suggest priorities for future research.

Pathophysiological Mechanisms of Chronic Pain

Chronic pain is defined as pain that persists or recurs for more than three months, wherein pain itself is the sole or primary complaint.[10] The transition from acute to chronic pain is a complex, multifactorial process, with sensitization of the peripheral and central nervous systems representing a key mechanism.[11–13] The release of chemical mediators (eg, ATP, prostaglandins, growth factors, cytokines, and neuropeptides) from peripheral nociceptors and inflammatory cells opens voltage-gated sodium channels. This lowers the firing threshold of dorsal root ganglion neurons, thereby inducing peripheral sensitization.[3] Persistent peripheral sensitization leads to a reduced pain threshold concurrent with massive neurotransmitter release in the spinal dorsal horn and dorsal root ganglia. This process further activates microglia and astrocytes, driving the progression from peripheral to central sensitization.[11]

Central sensitization itself is propelled by neuroglial activation and neuroinflammatory responses. Activated microglia and astrocytes release pro-inflammatory cytokines, chemokines, and neuropeptides that not only enhance the excitability of second-order spinal neurons but also interfere with descending inhibitory pathways, resulting in widespread pain.[11,12] Persistent neuroinflammation exacerbates neuronal responses to stimuli through "wind-up" phenomena, predisposing individuals to persistent pain long after the initial tissue injury has healed.[14,15] The upregulation of NMDA-type glutamate receptors and neuropeptide receptors induces rapid intracellular calcium elevation and amplifies pain signaling, ultimately manifesting as allodynia (pain from normally non-noxious stimuli).[15]

Neuroplasticity is critical for the maintenance of chronic pain. It refers to the ability of neurons and synapses to undergo morphological, functional, and adaptive changes. During pain transmission, neuroplasticity alters neuronal connectivity, synaptic efficiency, and the functional state of neural pathways, leading to pain exacerbation and persistence. While synaptic connections strengthen during acute pain, persistent noxious input induces adaptive structural and functional changes in the nervous system, heightening neuronal sensitivity.[13] This modulation involves presynaptic and postsynaptic signaling pathways, calcium flux, and multiple neurotransmitters.[13] Synaptic remodeling within the context of central inflammation also affects higher brain structures responsible for pain perception, emotion, and cognition, establishing chronic pain as a complex neuro-immuno-affective disorder.[1]

Psychosocial factors significantly contribute to chronic pain.[16] Modern evidence-based classifications emphasize the integral role of social and psychological components in its development, noting that pain and dysfunction result from a dynamic integration of biological, psychological, and social factors.[17,18] Genetics also influences susceptibility to chronic pain and variability in pain perception.[19] Familial studies show strong heritability for certain chronic pain disorders, with recent evidence implicating polymorphisms in the serotonergic, dopaminergic, and catecholaminergic systems.[20]

Chronic pain is associated with a reduced quality of life and shortened life expectancy. Although opioids remain essential for severe pain, long-term high-dose prescribing for chronic non-cancer pain carries significant risks of dependence and increased mortality. This underscores the urgent need for the development of effective opioid-sparing analgesic techniques for postoperative, traumatic, and chronic pain conditions.

Mechanisms of Erector Spinae Plane Block (ESPB)

Anatomical Basis

The successful implementation of the erector spinae plane block (ESPB) relies on a precise understanding of the relevant anatomy to ensure accurate local anesthetic deposition. The erector spinae muscle group, core to the posterior spine, comprises three components: the iliocostalis (most lateral), longissimus (intermediate), and spinalis (most medial). This group originates broadly from the sacral dorsum, lumbar spinous processes, and posterior iliac crest. The iliocostalis ascends to insert on rib angles, the longissimus attaches to transverse processes and adjacent ribs, and the spinalis terminates on spinous processes. Accessory muscle bundles further contribute to a complex anatomical landscape. This arrangement enables spinal extension, lateral flexion, and rotation, which are essential for posture and stability.

Ultrasound guidance clearly visualizes these muscular relationships and the key landmark of the transverse process, enhancing the block's accuracy and safety.[21]

Drug Diffusion Pathways and Extent

The efficacy of ESPB in chronic pain management depends critically on the distribution of local anesthetic within specific anatomical compartments.[22] Early cadaveric studies suggested diffusion was limited to the dorsal rami of spinal nerves, but subsequent research has confirmed extension into the paravertebral space, intervertebral foramina, and even the epidural space.[23,24] For instance, 12 of 16 published cadaver studies on thoracic ESPB reported dye penetration into the paravertebral space. In vivo radiological evidence further supports this, demonstrating multisegmental paravertebral and epidural spread.[25] Anatomically, the primary pathway for this diffusion is via perforations in the posterior thoracolumbar fascia and the intertransverse connective tissue complex, which are traversed by the dorsal ramus and its accompanying vessels.[26]

The extent and direction of diffusion are key determinants of block success. A cadaver study injecting contrast at the L4 level showed effective craniocaudal spread within the erector spinae plane, from L2 to beyond the sacrum.[22] This extensive spread along the fascial plane was corroborated by a comparative study at the L2 and L4 levels.[27] The depth of tissue penetration and the specific pathways taken are thus critical anatomical factors for efficacy. Several variables influence diffusion: for example, patient positioning affects spread due to gravity, with the prone position enhancing dispersion into the paravertebral and neural foraminal spaces.[28] The volume of injectate is another key factor, with larger volumes correlating with a greater diffusion area.[28]

Synthesized evidence from clinical, cadaveric, and mechanistic studies indicates that the primary mechanism of action is the direct physical spread of local anesthetic to neural structures within the fascial planes deep to the erector spinae muscle.[25,26] In practice, this spread is modulated by technical factors (eg, needle approach), injectate characteristics (volume, concentration), and the physicochemical properties of the anesthetic agent (eg, lipophilicity). A systematic investigation of these diffusion dynamics is therefore essential for both clinical application and technical refinement.

Analgesic Mechanisms

The precise analgesic mechanisms of ESPB are not fully elucidated. A recent review systematically summarized several potential mechanisms, including: 1) direct neural blockade and central inhibition via diffusion to the paravertebral or epidural space; 2) systemic analgesic effects mediated by absorption into the bloodstream; 3) immunomodulatory actions of local anesthetics; and 4) modulation of mechanosensation via the thoracolumbar fascia.[29] At the molecular level, analgesia primarily involves local anesthetics binding to voltage-gated sodium channels on neurons, inhibiting sodium influx and thereby preventing action potential propagation.[30]

The dorsal root ganglion (DRG) may be a key target due to its high susceptibility to nociceptive modulation.[31] However, the anesthetic concentration reaching the DRG via ESPB may be insufficient to cause a detectable sensory blockade on conventional testing. Meaningful analgesia may still occur through a selective blockade of C-fibers or partial sodium channel inhibition at systemic sub-anesthetic concentrations.[31] Future studies employing specialized nociceptive assessments are needed to clarify the DRG's role in ESPB-mediated analgesia for both acute and chronic pain.[31]

Clinical Applications of ESPB in Chronic Pain Management

Chronic pain pathogenesis involves multiple pathophysiological targets, including pathological alterations in the nervous system, inflammatory responses, and psychological factors, often accompanied by sensitization of central and peripheral nerves.[32] Patients with chronic pain exhibit persistent neuroinflammation, leading to hyperalgesia and pain perception from non-noxious stimuli.[33] Conventional pharmacotherapy often yields suboptimal outcomes in such cases, whereas ESPB effectively interrupts pain signal transmission, thereby alleviating symptoms. The mechanism of ESPB in chronic pain management is closely linked to its modulation of inflammatory responses. Studies indicate that ESPB significantly reduces levels of inflammatory cytokines (key mediators in chronic pain initiation and maintenance) in postoperative and chronic pain patients.[34] Additionally, ESPB mitigates pain by suppressing local and systemic inflammation while preventing functional impairment associated with chronic pain.

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Cancer-Related Pain

Cancer-related pain is a prevalent and severe symptom that substantially compromises patients' quality of life. Although oral analgesics and adjuvants manage 80% of cases, interventional pain management becomes crucial for patients with opioid tolerance or inadequate response.[35] Recent evidence highlights ESPB's potential in cancer pain management across diverse malignancies.[35] Sirohiya et al demonstrated significant pain reduction (mean NRS decrease: 4.2 points) following ESPB in patients with heterogeneous chronic cancer pain.[36] Other studies report efficacy in post-mastectomy pain syndrome (PMPS).[37,38] With advancing understanding of neural conduction mechanisms, ESPB shows unique advantages in managing thoracic/abdominal oncologic pain. Optimized pain control protocols can markedly improve quality of life in advanced cancer. However, current evidence supporting ESPB for cancer pain remains scarce, methodologically heterogeneous, and generally low-quality. Future work requires rigorous comparative studies, technical standardization, and larger cohorts to comprehensively evaluate efficacy and provide reliable clinical guidance.[6]

Neuropathic Pain

The application of the ESPB is expanding from acute postoperative pain to include chronic neuropathic conditions such as postherpetic neuralgia (PHN), offering a targeted interventional option for patients refractory to conventional pharmacotherapy. PHN, a neuropathic pain syndrome that follows varicella-zoster virus reactivation, manifests as persistent, severe pain within affected dermatomes. ESPB demonstrates particular utility in its management. Under ultrasound guidance, the injection of local anesthetic into the fascial plane deep to the erector spinae muscle effectively blocks the dorsal rami and sympathetic fibers, producing significant analgesia.[3] Supporting this, an observational study of 34 patients with herpes zoster reported rapid and substantial pain relief following ESPB (with NRS scores dropping from 9 to 1.5 immediately and sustained at 1 after three months),[39] suggesting the potential for durable neuromodulatory effects. As part of a multimodal PHN regimen, ESPB can reduce opioid consumption and improve quality of life.[3] Its mechanism is thought to involve the blockade of nociceptive signaling and suppression of sympathetic hyperactivity, thereby alleviating central sensitization.[37] Furthermore, synergistic effects are observed when ESPB is combined with neuropathic agents such as pregabalin.[35]

However, the current evidence base is limited to observational studies, lacking large-scale randomized controlled trials to validate its long-term (≥12 months) efficacy and safety. Future research should focus on optimizing technical parameters (eg, injectate concentration/volume, single versus multi-level injection sites) and identifying predictive biomarkers. With advancements in ultrasound visualization and protocol standardization, ESPB holds promise as a core component of multidisciplinary PHN management.

Applications in Other Chronic Pain Conditions

The ESPB is also applicable to radiculopathies across various spinal segments. Radiological studies of lumbar ESPB confirm that the injectate diffuses into the epidural space through the intervertebral foramina.[40] Furthermore, cadaveric and clinical case reports suggest that cervical and sacral ESPB may also lead to epidural spread, which can be bilateral and partially mimic the effects of an epidural injection.[41] Although the evidence is still largely anecdotal, the relative technical simplicity and favorable safety profile of ESPB justify its experimental use for chronic conditions such as headaches and shoulder pain.

A recent innovation is the sacral ESPB, a novel plane block targeting the posterior sacral nerve rami, proposed by Tulgar et al for managing sacral radiculopathies and gluteal pain syndromes.[3] Moreover, ESPB shows promise in treating refractory chronic pain. For instance, it has been shown to significantly alleviate symptoms and improve function in cases of intractable sciatica, and it has also demonstrated efficacy in managing post-rib fracture pain and cervical radicular pain.[42] Beyond therapeutic applications, studies indicate that ESPB not only provides effective acute pain control after thoracoabdominal surgery but may also reduce the incidence of chronic postsurgical pain. Supporting this, analysis of patients with chronic thoracic pain revealed sustained pain reduction (as measured by the NRS) for up to six months after the block,[24] suggesting a potential preventive effect. Clinically, ESPB has demonstrated therapeutic benefits across a diverse range of conditions, including acute pancreatitis, renal colic due to urinary calculi, and functional abdominal pain syndrome.

Comparison with Other Neural Blockade Techniques

Versus Epidural Block

Compared to conventional epidural block (EA), ESPB demonstrates broader clinical indications. EA primarily targets severe postoperative pain but carries risks of hematoma, infection, cerebrospinal fluid leakage, and hypotension. Comparative studies in traumatic rib fracture analgesia revealed no significant differences in total opioid consumption or pain scores between ESPB and EA groups, whereas EA required longer catheterization duration and hospital stays.[43] Post-mastectomy comparisons showed extended hospitalization with EA but comparable opioid usage.[44] ESPB induces fewer hemodynamic disturbances than EA, making it preferable for patients with severe comorbidities. Additionally, ESPB outperforms EA in analgesic efficacy, procedural safety, complication rates, and patient satisfaction.

Versus Other Regional Blockade Techniques

Compared to other regional analgesic techniques, the erector spinae plane block (ESPB) demonstrates a favorable safety profile, with a low incidence of severe adverse events reported in the literature. Its clinical advantages across various scenarios are supported by multiple randomized controlled trials (RCTs).[45–52]

For instance, in minimally invasive thoracic surgery, ESPB has been shown to provide better 24-hour recovery quality, lower complication rates, and superior analgesia compared to the serratus anterior plane block (SAPB).[45] Similarly, following laparoscopic nephrectomy, ESPB was more effective than quadratus lumborum block in reducing 24-hour opioid consumption and pain scores.[46] In pediatric patients undergoing inguinal herniorrhaphy, ESPB offered superior analgesia to caudal block,[47] and in open hepatectomy, it led to a significant reduction in 24-hour morphine use compared to subcostal transversus abdominis plane block.[48]

ESPB has also demonstrated non-inferiority to several established techniques. It provides analgesia comparable to intercostal nerve block in thoracoscopic surgery,[49–51] and while it may have a shorter analgesic duration than pectoral nerve blocks in post-mastectomy pain, it remains non-inferior to paravertebral block, with a comparable safety profile.[52]

In summary, the accumulating evidence positions ESPB not only as a reliable analgesic technique but also as one with an expanding spectrum of clinical indications.

Advantages and Limitations

ESPB serves as both an effective analgesic technique and a key component of multimodal analgesia strategies. It effectively controls pain while significantly reducing opioid requirements, facilitating enhanced recovery after surgery (ERAS).[53] Despite its unique potential in chronic pain management, ESPB has limitations: studies indicate inferior efficacy to EA in post-renal transplant patients;[54] its ability to block lower extremity nerve roots remains unconfirmed, limiting clinical utility in this region;[22] and it may provide inadequate analgesia for severe or complex pain conditions.[55,56] While excelling in acute pain management, its efficacy in chronic pain warrants further validation.[57] For patients with multifocal or complex pain, standalone ESPB may be insufficient. Consequently, despite its clinical value, ESPB's effectiveness and safety for lower extremity/distal applications require more robust evidence.[22] Clinical practice should tailor individualized pain management plans considering patient-specific factors and technical constraints.

Preventive Strategies for Chronic Post-Surgical Pain

Chronic postsurgical pain (CPSP) is a prevalent and complex syndrome that severely impairs the quality of life after surgery. Its pathogenesis involves multifactorial interactions, including preoperative pain, the extent of surgical trauma, and the quality of postoperative analgesic management. Consequently, establishing systematic prevention strategies is of significant clinical importance for reducing the incidence of CPSP.[58]

Effective prevention begins with preoperative management, which is critical for risk assessment and early intervention. Preexisting chronic pain or anxiety are known to significantly increase CPSP risk.[59] Multidimensional evaluations—

encompassing pain history, psychological status, and functional capacity—enable the creation of personalized perioperative analgesia protocols, thereby enhancing the precision of postoperative pain control.

The choice of intraoperative analgesic technique is equally pivotal. Regional nerve blocks, particularly the ESPB, are demonstrating increasing value. By effectively blocking nociceptive transmission from thoracoabdominal surgical sites, ESPB mitigates acute surgical pain and may thereby reduce central sensitization and the formation of pain memory, potentially preventing the development of CPSP.[60]

The implementation of postoperative multimodal analgesia strategies has been shown to significantly lower the incidence of CPSP.[59] These protocols typically combine regional techniques like ESPB with non-opioid analgesics and adjuvants, achieving synergistic analgesia while minimizing opioid-related adverse effects. Patient education and self-management strategies further contribute to comprehensive prevention.

In summary, preventing CPSP requires an integrated approach spanning the entire perioperative period, from preoperative risk stratification and patient education to intraoperative precision analgesia and postoperative multimodal management. As a safe and technically straightforward block, ESPB plays a valuable role in this strategy by significantly reducing acute postoperative pain and opioid consumption, with the potential to lower the incidence and severity of CPSP in specific procedures.[53,61] Future high-quality randomized controlled trials are warranted to definitively establish the efficacy of ESPB for CPSP prevention across diverse surgical populations and to inform standardized clinical pathways.

Complications and Safety

Despite ESPB's promise in chronic pain management, its risks require thorough evaluation. Efficacy and safety profiles vary across populations. Technical implementation carries risks of local infection, hematoma, and nerve injury.[62] Anesthetic overdose or misuse may cause systemic toxicity.[63] Thus, patient-specific pain pathophysiology must be assessed before ESPB adoption. Further exploration of efficacy, complication risks, and technical refinements is essential for optimizing chronic pain management.

Future Research Directions

ESPB demonstrates significant clinical value in acute/chronic pain, evolving from neuropathic pain treatment to a key component of multidisciplinary pain management.[37] While showing positive outcomes, its safety/efficacy requires validation through high-quality RCTs.[64,65] Future studies should compare ESPB with traditional regional techniques and evaluate long-term outcomes to establish its role in chronic pain.

Adjuvant-enhanced strategies and novel drug carriers represent emerging research foci. Studies indicate that adjuvants (eg, nalbuphine, dexmedetomidine, dexamethasone) combined with ropivacaine in multilevel paravertebral blocks enhance sensory blockade duration and postoperative analgesia versus ropivacaine alone.[66,67] These adjuvants prolong peripheral nerve block duration and improve analgesia quality.[68–72] Clinical evidence confirms that ropivacaine-adjuvant combinations reduce postoperative VAS scores, patient-controlled analgesia demand, and CPSP incidence.[73]

ESPB studies on local anesthetic-adjuvant combinations are increasing. Current evidence focuses on dexmedetomidine-ropivacaine, showing superior analgesia to monotherapy.[69,71,73–79] Limited studies explore nalbuphine or dexamethasone with ropivacaine.[70,73–75,78] Most surgical applications involve thoracic/abdominal procedures, with adjuvant combinations outperforming standard regimens. Novel adjuvants like low-molecular-weight dextran[80] and clonidine[81] have also been explored in ESPB.

Beyond chemical adjuvants, novel carriers like liposomal bupivacaine offer new directions. This sustained-release formulation has been trialed in transversus abdominis plane blocks,[82–86] brachial plexus blocks,[83,87–92] and other fascial plane/peripheral nerve blocks.[93–96] Limited evidence suggests prolonged analgesia in some regional blocks, though superiority over conventional bupivacaine remains unconfirmed in TAP/brachial plexus blocks.[97] Thus, liposomal bupivacaine's clinical value requires further high-quality RCTs.

Current ESPB research on drug carriers remains scarce. Future work should evaluate local anesthetic-adjuvant efficacy in chronic pain patients and directly compare combinations via large-scale RCTs to identify optimal regimens.

Adjuvant strategies and novel carriers represent innovative approaches that expand clinical options and enhance chronic pain management.

Conclusion

ESPB, an emerging fascial plane block, demonstrates growing value in chronic pain management. By precisely blocking dorsal rami of thoracic spinal nerves, it effectively suppresses pathological pain signaling, showing favorable efficacy and safety across chronic pain conditions. Compared to paravertebral or epidural analgesia, ESPB offers superficial placement, lower complication risks, and better patient acceptance. Ultrasound guidance further enhances its precision and individualization.

Current evidence supports ESPB's efficacy in neuropathic pain, CPSP, and cancer-related pain, with multidisciplinary collaboration facilitating technical refinement. However, widespread adoption faces challenges due to heterogeneous evidence, necessitating further high-quality validation.

Future research should focus on: standardizing image-anatomy-guided protocols; developing sustained-release local anesthetics to prolong blockade; and establishing multidimensional outcome assessments (pain intensity, functional recovery, quality of life). Through technical standardization and evidence accumulation, ESPB may become integral to multimodal chronic pain management frameworks.

Disclosure

Shengfang Du reports a patent Operating room cervical spine support mattress pending to Shengfang Du. The authors report no other conflicts of interest in this work.

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