Mechanisms of action of the erector spinae plane (ESP) block: a narrative review

Mécanismes d'action du bloc du plan des muscles érecteurs du rachis (*erector spinae*, ESP) : un compte rendu narratif

Ki Jinn Chin, MBBS (Hons), MMed, FRCPC · Kariem El-Boghdadly, MBBS, FRCA, MSc

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Abstract *The erector spinae plane (ESP) block is an emerging regional anesthetic technique with significant potential for clinical benefit. Nevertheless, its exact mechanism(s) of action has been much debated. We reviewed the available literature to explore the possible mechanisms of analgesia for the ESP block. These include neural blockade and central inhibition from direct spread of local anesthetic to the paravertebral or epidural space; analgesia mediated by elevated local anesthetic plasma concentrations due to systemic absorption; immunomodulatory effects of local anesthetics; and an effect mediated through the mechanosensory properties of thoracolumbar fascia. Based on evidence from clinical, human cadaveric, animal, and mechanistic laboratory studies, the most probable primary mechanism is a direct effect of local anesthetic via physical spread and diffusion to neural structures in the fascial plane deep to the erector spinae muscles and adjacent tissue compartments. Biological plausibility of this primary mechanism is confirmed by injectate spread to the ventral rami of spinal nerves (though quite variable) in most studies. There is consistent involvement of dorsal rami; epidural spread is*

a less commonly observed phenomenon. A systemic effect of local anesthetic is also plausible, but unlikely to be a major contributor to clinical analgesic efficacy. The evidence for significant analgesia due to other proposed mechanisms, such as fascia-mediated analgesia or lymphatic spread, are currently limited and thus remain speculative. Understanding the mechanisms of action could assist clinicians in further investigating and refining ESP block performance, with the ultimate goal of optimizing analgesic efficacy and improving postoperative patient outcomes.

Résumé *Le bloc du plan des muscles érecteurs du rachis (bloc ESP) est une technique émergente d'anesthésie régionale qui pourrait s'accompagner de bienfaits cliniques considérables. Néanmoins, ses mécanismes d'action exacts font l'objet de nombreux débats. Nous avons passé en revue la littérature disponible pour explorer les mécanismes possibles de l'analgesie par le bloc ESP. Il s'agit notamment du bloc nerveux et de l'inhibition centrale dus à la diffusion directe de l'anesthésique local à l'espace paravertébral ou péri-dural; de l'analgesie médiée par des concentrations plasmatiques élevées d'anesthésique local dues à son absorption systémique; des effets immunomodulateurs des anesthésiques locaux; et d'un effet médié par les propriétés mécanosensorielles du fascia thoraco-lombaire. Selon les données probantes tirées d'études cliniques, cadavériques, animales, et mécanistes de laboratoire, le mécanisme primaire le plus probable est un effet direct de l'anesthésique local par sa propagation et sa diffusion physique aux structures neuronales dans le plan fascial plus profond que les muscles érecteurs du rachis et aux compartiments tissulaires adjacents. La plausibilité biologique de ce mécanisme primaire est confirmée par*

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K. J. Chin, MBBS (Hons), MMed, FRCPC (✉)
Department of Anesthesiology and Pain Medicine, Toronto
Western Hospital, University of Toronto, Toronto, ON, Canada
e-mail: gasgenie@gmail.com

K. El-Boghdadly, MBBS, FRCA, MSc
Department of Anaesthesia and Perioperative Medicine, Guy's
and St Thomas' NHS Foundation Trust, London, UK

King's College London, London, UK

la propagation du produit injecté aux rameaux ventraux des nerfs rachidiens (bien que variable) dans la plupart des études. Les rameaux dorsaux sont constamment impliqués; la diffusion périurale est moins souvent observée. Un effet systémique de l'anesthésique local est également plausible, mais il est peu probable qu'il s'agisse d'un contributeur majeur à l'efficacité analgésique clinique. Les données probantes appuyant une analgésie significative due à d'autres mécanismes proposés, tels que l'analgésie médiée par les fascia ou la propagation lymphatique, sont actuellement limitées et demeurent de l'ordre de la spéculation. En comprenant les mécanismes d'action, les cliniciens pourraient approfondir leurs connaissances et améliorer la performance des blocs ESP, dans le but ultime d'optimiser leur efficacité analgésique et d'améliorer les devenir postopératoires des patients.

Keywords erector spinae plane block · regional anesthesia · local anesthetics

"When you have excluded the impossible, whatever remains, however improbable, must be the truth."

- Arthur Conan Doyle

Since its first description in 2016,¹ the erector spinae plane (ESP) block has attracted unprecedented attention and has stimulated an explosion of interest in fascial plane blocks in general. While fascial plane blocks are not new—the transversus abdominis plane (TAP) block was popularized more than a decade ago²—the ESP block is unique in its breadth of application. It has been utilized for acute and chronic pain conditions, not only of the torso but also of the upper and lower limbs.^{3–5} It has been used in settings where regional anesthesia has traditionally had a limited role, such as cardiac surgery⁶ and spine surgery.^{7,8} It has also been embraced by specialties outside of anesthesiology, including emergency medicine⁹ and prehospital trauma care.^{10,11}

Controversy has accompanied the wide attention the ESP block has garnered. This has principally centred around the two questions: “does it provide effective analgesia?” and if so, “how does it provide effective analgesia”? Answers to the first question have begun to emerge, with the publication of randomized-controlled trials and meta-analyses.^{12–14} The second question is equally important as it is crucial for optimizing block performance and the delivery of safe and effective analgesia. We therefore conducted this narrative review with the objectives of synthesizing the available evidence to support or refute the various proposed mechanisms of action for the ESP block.

The ESP block

The basic ESP block technique involves ultrasound-guided injection of a relatively large volume of local anesthetic (0.3–0.5 mL·kg^{−1}) into the fascial plane between the tips of the vertebral transverse processes and erector spinae muscle (Fig. 1). Local anesthetic spreads within this potential space over 3–6 vertebral levels in a cranio-caudal direction. Medial-lateral spread is usually confined to the boundaries of the erector spinae muscle, limited by its attachment to the angle of the ribs and the enveloping thoracolumbar fascia.^{15,16} Clinical evidence suggests that this results in somatic and visceral analgesia in the territory supplied by the congruent spinal nerves (Fig. 2).^{12,17–19} The mechanism of action originally proposed in the early descriptions of the ESP block was of local anesthetic spreading anteriorly from the plane of injection, through channels in the inter-transverse connective tissues, to the paravertebral space, where it could act on ventral rami and spinal nerve roots.¹ Nevertheless, this has been challenged by recent cadaveric studies^{15,20} and observations of inconsistent cutaneous sensory loss in clinical studies,²¹ raising the question of whether there are other mechanisms at work.

Sources of evidence

Direct evidence for the possible mechanisms of action of the ESP block comes from several sources, most of which have focused on investigating the physical spread of injected solution. Both human and animal cadaver models have been utilized in anatomical studies. Spread is most often determined by anatomical dissection or sectioning, but radiocontrast studies using either computed tomography (CT) or magnetic resonance (MR) imaging have also been employed. Mechanisms of action can also be inferred from results of clinical studies in human volunteers and patients, including physical spread by CT or MR imaging as well as physiologic effects of local anesthetic injection. Both modalities are equally important as the correlation between detectable physical spread and the clinical manifestations of neural blockade is imperfect.²² Each source of evidence has advantages and limitations, and these should be considered when interpreting the data.

Considerations in cadaveric studies

As of 1 June 2020, there have been 18 published studies of the ESP block in human cadavers and three studies in animal models (Table 1). The majority have involved unembalmed cadavers, also referred to as (thawed) fresh

Fig. 1 Conceptual illustration of local anesthetic spread following an erector spinae plane (ESP) block. Local anesthetic is injected into the fascial plane between erector spinae muscle (ESM) and the tip of the transverse process. A certain fraction penetrates anteriorly (block arrow 1) into the paravertebral space through channels in the intertransverse connective tissue complex (a collective term for the ligaments and other connective tissues that span adjacent transverse processes), and may also track through the intervertebral foramen into the epidural space. There is cranio-caudal spread along the fascial plane beneath erector spinae muscle (block arrow 2), usually over several vertebral levels. Posterior tracking into the erector spinae

muscle itself (block arrow 3) is common. Lateral spread (block arrow 4) tends to be limited to the lateral border of the thoracolumbar fascia (not shown) encasing the erector spinae muscle. (Illustration adapted and reproduced with the permission of Dr. Vicente Roques.) ACB = anterior cutaneous branch; CB = collateral branch; DR = dorsal ramus; ESM = erector spinae muscle; IN = intercostal nerve; LCB = lateral cutaneous branch; RC = rami communicantes; RMM = rhomboid major muscle; SG = sympathetic ganglion; TM = trapezius; muscle; VR = ventral ramus.

Fig. 2 Simplified schematic illustration of the anatomy and course of a typical thoracic spinal nerve and its branches. Beyond the dorsal root ganglion (DRG), the spinal nerve root splits into a dorsal and ventral ramus. The dorsal ramus (DR) ascends through the thoracolumbar fascia (dotted line) and the erector spinae muscle (ESM) to the superficial tissues. The ventral ramus (VR) continues along the inner caudal surface of the rib as the intercostal nerve (IN), giving off collateral branches to muscles and bone along the way. The lateral cutaneous branch (LCB) arises close to the angle of the rib.

The IN terminates in the anterior cutaneous branch (ACB). (Illustration adapted and reproduced with the permission of Dr. Vicente Roques.) ACB = anterior cutaneous branch; CB = collateral branch; DR = dorsal ramus; ESM = erector spinae muscle; IN = intercostal nerve; LCB = lateral cutaneous branch; SG = sympathetic ganglion; VR = ventral ramus.

Table 1 Cadaveric studies of injectate spread in the erector spinae plane (ESP) block

Author/year	Subject population	ESP block	Injectate	Method of assessment	Other findings
Mid-thoracic ESP block = 10 studies					
Foreiro 2016 ¹	Un-embalmed human adult cadavers, <i>n</i> = 2.	Bilateral T5 ESP block, total <i>n</i> = 4.	20 mL of methylene blue solution. CT contrast dye added in 1 cadaver.	Dissection in layers, <i>n</i> = 2. CT imaging, <i>n</i> = 1.	First description of ESP block. Injection was superficial to ESM in 1 cadaver and deep to ESM in the other. Superficial injection did not penetrate anteriorly beyond the deep surface of ESM. Deep injection produced cranio-caudal spread deep to ESM from T1 to T8. Lateral spread beyond tips of TP and deep to intercostal muscles over 4–5 segments. Anterior spread deep to intercostal muscles and to “vicinity of ventral and dorsal rami”. Cranial-caudal spread deep to ESM from T1 to T6 in majority of injections. Dorsal rami involved in 70% of injections, but retrograde spread to DRG in only 10%. Anterior spread to ventral rami in 5%. Cranio-caudal spread deep to ESM over 9–14 segments. Lateral spread to intercostal spaces over 5–9 segments. Anterior spread to neural foramina over 2–3 segments, to epidural space over 2–5 segments. Spread to T4–T6 neural foramen and spinal nerves (3 segments) in all specimens. Spread extended to T3 in 20% and T7 in 40% of specimens. Intercostal spread was only seen superficial to the external fascia of external intercostal muscles. Dissection did not involve laminectomy to examine epidural space. Anterior chest wall and organs removed to expose ribs and spine. Video analysis of injection in real-time, followed by dissection of parietal pleura to expose PVS. Spread to 3–7 intercostal spaces (mean, 4.6). No dye was found in the intervertebral foramen or spinal canal. Anterior spread to PVS, ventral rami, sympathetic trunk, and communicating rami over 1–4 segments in 60% of specimens.
Ivanusic 2018 ¹⁵	Un-embalmed human adult cadavers, <i>n</i> = 10.	Bilateral T5 ESP block, total <i>n</i> = 20.	20 mL of methylene blue solution.	Dissection in layers, <i>n</i> = 10.	
Adhikary 2018 ⁴⁷	Un-embalmed human adult cadavers, <i>n</i> = 3.	Unilateral T5 ESP block, total <i>n</i> = 3.	20 mL of methylene blue and MRI contrast solution.	Dissection in layers, <i>n</i> = 3. MRI, <i>n</i> = 3.	
Yang 2018 ⁴⁸	Un-embalmed human adult cadavers, <i>n</i> = 10.	Unilateral T5 ESP block, total <i>n</i> = 10.	20 mL of latex and green ink solution.	Dissection in layers, <i>n</i> = 10.	
Vidal 2018 ⁵⁰	Un-embalmed human adult cadavers, <i>n</i> = 3.	T5 ESP block, <i>n</i> = 2. T4 ESP block, <i>n</i> = 5. T10 ESP block, <i>n</i> = 2.	20 mL of methylene blue solution.	Video analysis of injection in real-time, <i>n</i> = 3. Dissection in layers, <i>n</i> = 3.	
Nielsen 2019 ¹³⁹	Soft-embalmed human adult cadavers, <i>n</i> = 5.	Unilateral T5 ESP block, total <i>n</i> = 5.	20 mL of methylene blue solution.	Dissection in layers, <i>n</i> = 5.	

Table 1 continued

Author/year	Subject population	ESP block	Injectate	Method of assessment	Other findings
Diwan 2019 ¹⁴⁰	Un-embalmed human adult cadavers, $n = 5$.	Unilateral T4/5 ESP block, total $n = 5$.	20 mL of methylene blue solution.	Transverse sections at 5cm intervals, $n = 5$.	Letter to editor with limited reporting of results. Similar spread compared with paravertebral block on contralateral side.
Choi 2019 ¹²⁸	Un-embalmed human adult cadavers, $n = 7$.	Bilateral T5 ESP at T5 with 30mL and 10mL on either side, total $n = 14$.	30mL or 10mL of latex and green ink solution.	Thoracoscopic inspection of PVS, $n = 12$. Dissection in layers, $n = 14$.	Medial retrolaminar spread as well. Comparison of anterior spread with 30mL vs 10mL of injectate. With 10mL there was anterior spread only to the SCTL at T4–T6 but not PVS. With 30mL, there was greater cranio-caudal extent of spread and penetration to PVS at ≥ 1 level (median, 3). Lateral spread over external intercostal muscles and posterior TLF, but no penetration into intercostal spaces.
Govender 2020 ¹³⁰	Un-embalmed human neonate cadavers, $n = 2$.	Unilateral T5 or T8 ESP block on left side only, total $n = 2$.	0.2 (T8) and 0.5mL (T5) of methylene blue solution.	Dissection in layers, $n = 2$.	Anterior spread with T5 ESP to ventral and dorsal rami from T3 to T6 and with T8 ESP to T7–T11. Superficial spread of injectate in muscle layers was more extensive.
Shibata 2020 ²⁴	Soft-embalmed human adult cadavers, $n = 5$.	Unilateral T4 ESP block, $n = 5$.	20 mL of methylene blue solution.	Dissection in layers, $n = 5$.	Spread to superficial and deep planes of the ESM over 5–6 levels. Spread to dorsal rami in 100% from T2 to T6. No spread to ventral rami or PVS or intercostal space. Epidural space not examined.
Low thoracic ESP block = 6 studies					
Chin 2017 ⁴⁹	Un-embalmed human adult cadaver, $n = 1$.	Bilateral T7 ESP block, total $n = 2$.	20mL of CT contrast dye.	CT imaging, $n = 1$.	Cranio-caudal spread deep to ESM from T2 to L3 and C5–L2 on either side. Lateral spread to intercostal spaces at T10–T12.
Aponte 2019 ²⁰	Un-embalmed human adult cadavers, $n = 4$	Bilateral T7 ESP in 2 cadavers	20 mL of methylene blue and radiocontrast	CT imaging, $n = 4$. Dissection in layers, $n = 2$.	Cranio-caudal extension of spread in ESM from T1 to L3 on CT imaging.
		Unilateral T7 ESP in 2 cadavers		Axial cryosection, $n = 2$.	Lateral extension to costotransverse region up to scapular line on CT imaging.
		Total $n = 6$			No comment on involvement of neural structures apart from ventral and dorsal rami.

Table 1 continued

Author/year	Subject population	ESP block	Injectate	Method of assessment	Other findings
Dautzenberg 2019 ¹⁴¹	Un-embalmed human adult cadavers, $n = 11$.	Unilateral T2 ESP block on 1 side, and T8 ESP block on other side, total $n = 22$.	20 mL of methylene blue solution.	Dissection in layers, $n = 11$.	Cranio-caudal spread at T8 was consistent over many levels (9–20). Cranio-caudal spread at T2 was more variable over fewer levels (4–14). Lateral extent of spread and spread to epidural space was not assessed. Spread to PVS, ventral and dorsal rami in 36% of T2 injections, and 9% of T8 injections Spread to the ventral rami in 25–75% of injections at T9–T11. Epidural spread was inferred from dural staining. Spread to various paraspinal and intercostal muscles was assessed, but not specifically to intercostal space or nerves. Lateral spread to costotransverse junction in 83%; to posterior axillary line in 17%, on CT imaging. Caudal spread to L3 in 83%; L2 in 17% on CT imaging.
Altinpulluk 2019 ¹⁴²	Embalmed human adult cadavers, $n = 4$.	Bilateral T9 ESP blocks, total $n = 8$.	20 mL of methylene blue solution.	Dissection in layers, $n = 8$.	PVS spread in 17% on CT imaging. Faint staining of PVS at T10–11 in 50% on dissection. Dorsal rami involved at 4–5 levels below T10, and 3–7 levels above T10, on dissection. Faint staining of intercostal nerves at T9–12 in 50% and at T11 in 17% on dissection. No spread to epidural space. No staining of lumbar nerve roots or plexus. Cranio-caudal spread at T8 over 3 levels (T6–9). Cranio-caudal spread at T10 over 4 levels (T9–T12). Lateral spread at both levels into intercostal space. Anterior spread at both levels into PVS but not epidural space.
Elsharkawy 2019 ¹⁴³	Un-embalmed human adult cadavers, $n = 6$	Unilateral T10/11 ESP, total $n = 6$	30mL of 0.5% methylcellulose, India ink, and CT contrast solution	Dissection in layers, $n = 6$. CT imaging, $n = 6$	Anatomical staining classified as none/faint/deep. Deep staining of C5–C7 nerve roots in 100%, variable staining of C8 nerve root in 100%, of T1 nerve root in 50%. Deep staining of suprascapular, long thoracic, and dorsal scapular nerves in 100%. Variable staining of phrenic nerve in 30%.
Govender 2020 ¹⁴⁴	Un-embalmed human neonate cadaver, $n = 1$.	Unilateral T8 ESP block on 1 side and T10 ESP block on other side, total $n = 2$.	1mL/kg (2.3mL) of CT contrast dye.	CT imaging, $n = 1$.	
High thoracic / cervical ESP block = 1 study					
Elsharkawy 2020 ²⁵	Un-embalmed human adult cadavers, $n = 5$.	Unilateral C6 ESP block on 1 side and C7 ESP block on other side, total $n = 10$.	20mL of India ink solution.	Dissection in layers, $n = 10$.	

Table 1 continued

Author/year	Subject population	ESP block	Injectate	Method of assessment	Other findings
Lumbar ESP block = 1 study					
De Lara Gonzalez 2019 ¹⁴⁵	Un-embalmed human adult cadavers, <i>n</i> = 6.	Bilateral L4 ESP blocks, total <i>n</i> = 12.	20 mL of methylene blue and CT contrast dye.	CT imaging with 3D reconstruction, <i>n</i> = 6. Dissection in layers in layers, <i>n</i> = 2. Axial cryosection, <i>n</i> = 2. No dissection, <i>n</i> = 2.	2 cadavers not dissected because of “technical and operational problems”. Dorsal rami involved in 100%. Spread to anterior aspect of TPs in 75% and ventral rami involved in 17%. Cranio-caudal spread between L1–L2 and L4–L5. Lateral spread was confined to the erector spinae muscle by the TLF. Medial spread was limited to the articular processes.
Animal models of ESP block = 3 studies					
Ferreira 2019 ¹⁴⁶	Un-embalmed canine cadavers, <i>n</i> = 8.	Bilateral ESP block at T5 in caudal-cranial direction. Total ESP injections = 16.	0.5 mL/kg (low-volume) or 1.0 mL/kg (high-volume) of methylene blue solution.	Dissection in layers, <i>n</i> = 8.	More extensive cranial-caudal spread with high vs low-volume injections: mean (SD) of 8.4 (1.6) vs 6.4 (1.9) segments. Spread largely confined to transversospinalis and longissimus muscles. Dorsal rami stained in 100%; median of 2 levels (range, 1–4). Staining of PVS and spinal nerves in 12.5% (2 levels). Staining of ventral rami and intercostal nerves in 12.5%. No spread to epidural space, or sympathetic trunk.
Portela 2020 ¹⁴⁷	Un-embalmed canine cadavers, <i>n</i> = 9	Unilateral (<i>n</i> = 2) or bilateral single-injection ESP block at T9 (<i>N</i> = 4). Total ESP injections = 10.	0.3 mL/kg (low volume) or 0.6 mL/kg (high volume) coloured dye solution. Gadodiamide MRI contrast dye.	Pre- and post-injection MRI, <i>n</i> = 2. Axial cryosection, <i>n</i> = 2. Dissection in layers, <i>n</i> = 7.	No significant difference in cranial-caudal spread between low and high-volume injections - median of 4 segments (range 3–8). No anterior spread to ventral rami or PVS or epidural space or intercostal space. Lateral-medial spread confined by the TLF sheath.

Table 1 continued

Author/year	Subject population	ESP block	Injectate	Method of assessment	Other findings
Otero 2020 ¹¹²	Live pigs, $n = 6$.	Bilateral ESP block at T6 in both cranial-caudal and caudal-cranial directions. Total ESP injections = 12.	0.6 mL/kg of methylene blue solution.	Euthanized 20 min after block. Axial cryosection, $n = 2$. Dissection in layers, $n = 4$.	Cranial-caudal spread T5–T9 in most cases, median of 5 segments. Lateral spread limited by the attachments of TLF. Medial spread between LT and MF muscles. Spread to prevertebral thoracic lymph nodes. Dorsal rami stained in 100%. No spread to epidural space, PVS, ventral rami, or intercostal nerves.

CT = computed tomography; DRG = dorsal root ganglion; ESP = erector spinae plane; ESM = erector spinae muscle; LT = longissimus thoracis; MF = multifidus; MRI = magnetic resonance imaging; PVS = paravertebral space; SCTL = superior costotransverse ligament; SD = standard deviation; TLF = thoracolumbar fascia; TP = transverse process.

frozen cadavers. Cadaveric studies are popular in investigating the physical spread of ultrasound-guided dye injections and can inform as to which nerves may potentially be reached by local anesthetic solutions. Nevertheless, for several reasons, cadaveric injections are likely only a rough approximation of what occurs in live subjects. The biomechanical properties of cadaveric tissues are quite different from living ones and the injected solutions may not spread the same way through the various tissue planes. Generally speaking, embalmed cadaver tissue is less pliable, while at the other end of the spectrum, fresh frozen cadavers may suffer from diminished tissue integrity. It is unclear what the exact impact of cadaveric tissue architecture on injectate spread (both within and across fascial planes) might be, and there is no consensus as to what type of cadaver model is optimal for studying the physical spread of fluid injections.

There is similarly no consensus on the physical properties of the ideal injectate. An aqueous dye solution (e.g., methylene blue) is most commonly used but is sometimes criticized for a presumed propensity to spread too widely. Improper dissection technique may further contribute to spread of dye in patterns that would not otherwise occur in intact fascial spaces. On the other hand, more viscous injectates (e.g., with added latex or resin) may underestimate the spread. There is unfortunately no direct evidence to indicate which of these most accurately reflects conditions in live subjects. We are, however, of the opinion that physiologic spread may often be more extensive than commonly thought. Tissue planes and compartments in live subjects are subject to dynamic forces—muscles and fasciae tense, relax, and slide over each other; thoracic and abdominal intra-cavity pressures rise and fall, and may be transmitted to adjacent spaces such as the thoracic paravertebral spaces.²³ This contributes to a gradual spread of local anesthetic effect over time—i.e., clinical progression over hours rather than minutes has been observed.²⁴ A final consideration is that extent of dye spread is determined by macroscopic visual inspection of the results of dissection or imaging, and more subtle boundaries of spread may not be evident. In some cadaveric studies, a distinction is made between heavy or faint dye staining of nerves,²⁵ but it is unclear how this correlates with anesthetic effect in living subjects. As will be discussed, even very low concentrations of local anesthetic in the vicinity of a nerve may exert a physiologic effect.

Considerations in living subjects

Imaging studies of radiocontrast dye injections in living subjects provide the most compelling evidence for the impact of the physical spread of injectate and certainly

show what is possible. Generalization of study findings is, however, constrained by inter-individual variability and small study sample sizes (Table 2). There are also limitations to image interpretation; for example, it can be difficult to differentiate intramuscular (e.g., intercostal muscle) spread of contrast from the actual penetration of solution into the intercostal space (i.e., the plane between the innermost and internal intercostal muscles that contains the intercostal neurovascular bundle). Similarly, gadolinium magnetic resonance imaging (MRI) contrast in the prevertebral area may represent either physical spread from the paravertebral space or lymphatic uptake into prevertebral lymph nodes.²⁶ As with cadaveric studies, radiographic imaging only shows physical spread, and this often underestimates the extent of actual cutaneous sensory loss,²² which may again reflect the sensitivity of nerves to small undetectable amounts of local anesthetic.

Next to surgical anesthesia or analgesia, cutaneous sensory testing is arguably the most relevant method of assessing neural blockade in regional anesthesia. Nevertheless, even this is subject to imprecision. Different skin sensory testing modalities (e.g., light touch, pinprick, temperature) have different sensitivities to neural blockade, and can produce different patterns on testing (this is discussed in more detail below).^{27–29} Inter-individual and even intra-individual variation (i.e., different results are produced by the identical block technique performed at different times in the same individual) in neural sensitivity have been shown not just with fascial plane blocks (e.g., the TAP and ESP blocks)^{21,30–32} but also with discrete blocks of individual peripheral nerves in the upper and lower limbs.^{33–35} Together with normal anatomical variation in the anastomotic interconnections between nerves and the course they follow, this may explain the significantly different patterns of cutaneous sensory loss observed in clinical practice. Finally, it must be noted that cutaneous sensory testing only reflects blockade of terminal nerve branches in the skin. Pain often arises from deeper structures (e.g., muscle, bone, viscera) and analgesia of these structures may be achieved without obvious cutaneous sensory loss. To complicate matters further still, the presence of deep pain can modulate cutaneous sensation to produce either hypo- or hyperalgesia.³⁶

Proposed mechanisms of action for the ESP block

Physical spread of local anesthetic to the thoracic paravertebral space and associated neural structures

The spread of local anesthetic to the thoracic paravertebral space was originally proposed as the primary mechanism

of action of the ESP block.¹ This has since been challenged and remains controversial because of conflicting anatomical and physiologic evidence. Nevertheless, the weight of available evidence clearly shows that paravertebral spread can and does occur. Of the 16 cadaveric studies of thoracic ESP blocks published to date, 12 have found evidence of paravertebral dye penetration, even if only in a minority of specimens (Table 3). This is bolstered by radiological imaging in living subjects showing radiocontrast spread into the paravertebral and even epidural space across multiple levels.^{37–40} The posterior thoracolumbar fascia and inter-transverse connective tissue complex (a collective term for the ligaments, muscles, and other connective tissues that span adjacent transverse processes)⁴¹ is perforated by branches of the dorsal rami and accompanying blood vessels,^{42,43} and these are the most likely pathways for local anesthetic to track into the paravertebral space (Fig. 1). These channels probably do not allow for rapid bulk flow, but instead a gradual seepage of local anesthetic, as shown by the absence of visible spread into the paravertebral space on thoracoscopy *during* ESP block injection that was subsequently clinically effective.⁴⁴ This would also explain why the ESP block does not produce the pressure-like chest discomfort often associated with thoracic paravertebral blockade (attributed to rapid distension of the paravertebral space and pleural displacement). An even more important fact to recognize is that fascia is highly permeable to macromolecules, including local anesthetic drugs. The *macroscopic* appearance of fascia as a dense opaque layer is misleading; at the *microscopic* level, gaps in its largely acellular architecture of interlinked collagen fibres readily permit rapid diffusion.⁴⁵ This has been shown for various fasciae ranging from dura mater⁴⁵ to the epimysium of the transversus abdominis plane.⁴⁶ By contrast, deceptively fragile membranes such as the arachnoid mater and perineurium are much less permeable. They are composed of layers of tightly-apposed cells rather than collagen fibres; macromolecules cannot pass between the cells, only through them by a process of active transport or slow biphasic diffusion across the lipid cell membrane and aqueous intracellular milieu.

In keeping with this, imaging and dissection studies indicate that only a small fraction of injectate enters paravertebral and epidural spaces within the first 30–60 min (the usual interval in most studies), while the majority remains within the erector spinae muscle compartment.^{40,47,48} Penetration via diffusion into the paravertebral space may continue over a prolonged period, as evidenced by a report in which preoperative sensory loss over two dermatomes progressed to six dermatomes in the postoperative period.²⁴ Injectate

Table 2 Clinical and imaging studies of injectate spread after an erector spinae plane (ESP) block in patients

Author/year	Subject population	ESP block	Injectate	Method of assessment	Findings
Mid-thoracic ESP block = 4 studies					
Forero 2016 ¹	Chronic thoracic pain, $n = 1$.	Unilateral T5 ESP catheter inserted superficial to ESM.	20 mL 0.25% bupivacaine + 5 mL CT contrast dye.	Sensory assessment to pinprick. CT imaging of thorax.	Sentinel case of the ESP block— injection in superficial plane. Cranio-caudal spread from T1 to T11 over retrolaminar area on CT imaging; more detailed reporting not available. Pain scores reduced from 10/10 to 0/10. Sensory loss to pinprick over T2–T9. No visual evidence of physical spread into PVS with bolus injection through needle or catheter. Sensory loss to cold from T4–T7 over anterolateral thorax 2 hr after bolus. Pain scores at chest drain and incision sites 0/10 2hr after bolus; 5/10 at drain site after 16 hr of infusion. Plasma lidocaine concentrations $2.9 \mu\text{g}\cdot\text{mL}^{-1}$ 2hr after bolus, and 1.8 mg/mL during infusion. Paravertebral and intervertebral foraminal spread on CT imaging. Improved pain scores from 10 to 0/10 after 1st diagnostic injection. Improved pain scores from 8 to 2/10 after phenol injection. Cutaneous sensory loss from T5 to T12.
Visoiu 2019 ⁴⁴	Thoracoscopic blebectomy, $n = 1$	Unilateral T5 ESP catheter.	1) 20 mL 1.7% lidocaine + methylene blue injected through needle. 2) 11 mL 0.9% saline injected through catheter to assess spread and placement. 3) 8 mL/h 1% lidocaine infusion with 4 mL bolus every 4 hr.	Thoracoscopic intrathoracic visualization of paravertebral spaces. Sensory assessment to cold.	
Hernández-Porras 2020 ³⁸	Patient with thoracic pain from pleural metastases and pleural resection, $n = 1$.	Unilateral (1) T5 ESP block, and (2) T3 ESP block on 2 separate occasions.	(1) 20 mL 0.25% bupivacaine + 40mg methylprednisolone. (2) 20 mL phenol + CT contrast dye	Clinical observation. Cutaneous sensory testing modality unspecified. CT imaging at unspecified time after block.	
Diwan 2020 ³⁹	Elderly man with “visceral pain” from lung cancer, $n = 1$.	Unilateral T5 ESP block.	25 mL 0.25% bupivacaine + 3 mL iohexol CT contrast dye + 40 mg methylprednisolone.	Clinical observation. Cutaneous sensory testing modality unspecified. CT imaging 30 min after block	Paravertebral and epidural spread on CT imaging. Improved pain scores from 8 to 1/10. Dermatomal analgesia from T2 to T7.
Low thoracic ESP block = 6 studies					
Schwartzmann 2019 ³⁷	Chronic postsurgical abdominal pain, $n = 1$.	Unilateral T10 ESP.	29.7 mL 0.25% bupivacaine + 0.3 mL gadolinium MRI contrast dye.	Sensory assessment to ice and pinprick. MRI at 45 min and 90 min after block.	MRI showed spread to epidural space, paravertebral space, intervertebral foramina from T5 to T12. Spread to intercostal muscles at T6–T12, but spread to the intercostal space and nerves was not confirmed. Loss of cold and pinprick sensation over anterolateral torso from T6 to T12.
Elkoundi 2019 ⁷⁷	Patient with acute pancreatitis in the ED, $n = 1$	Bilateral T6 ESP blocks	15 mL 0.5% bupivacaine each side	Clinical observation. Cutaneous sensory loss to pinprick	Improved pain scores from 10 to 2/10. Cutaneous sensory loss to pinprick from T4–T10

Table 2 continued

Author/year	Subject population	ESP block	Injectate	Method of assessment	Findings
Aydin 2019 ⁷⁸	Patients with renal colic in the ED.	Unilateral T8 ESP block	20–30 mL 0.25% bupivacaine 2% lidocaine mixture.	Clinical observation. Cutaneous sensory loss to cold.	Improved pain scores from 8–10/10 to 0–2/10. Cutaneous sensory loss to cold from T2 to L2 in 1 patient.
Schwartzmann 2020 ⁴⁰	Chronic abdominal-pelvic pain, $n = 6$.	Unilateral T10 ESP.	29.7 mL 0.25% bupivacaine + 0.3 mL gadolinium + 5 mL 1% triamcinolone.	Sensory assessment to ice and pinprick at 20 min after block. Pain scores at 30 min after block. T1-weighted MRI at 60 min after block.	MRI showed spread to 1) epidural space in 33% of patients, over 10 and 2 levels, 2) neural foramina (and by definition the PVS) in 100% of patients over a median [IQR] (range) of 3 [2–6] (2–10), 3) intercostal space and nerve in 100% of patients over 9 [5–11] (3–11) levels. The intercostal spread was 5–8 cm lateral, not reaching the angle of the rib. Pain scores reduced in 100% of patients from 8–10/10 to 0/10. Sensory loss over a variable area of the anterior and posterior torso in 100% of patients.
Mantuani 2020 ¹⁹	Young man with acute pancreatitis in the ED, $n = 1$.	Bilateral T7 ESP blocks.	10 mL NS + 15 mL 0.5% bupivacaine + epinephrine 5 $\mu\text{g}\cdot\text{mL}^{-1}$ each side.	Clinical observation.	Improved pain scores from 10 to 0/10 with no abdominal tenderness.
Pak 2020 ⁸¹	Elderly lady undergoing major open abdominal surgery, $n = 1$.	Bilateral T12 ESP catheters.	Loading dose of 10 mL 0.25% ropivacaine. Continuous infusion of 8 $\text{mL}\cdot\text{hr}^{-1}$ 0.2% ropivacaine.	Clinical observation.	Good analgesia with ESP blocks. Postoperative hypotension and quadriceps weakness noted with infusion, which resolved with catheter removal.
High thoracic ESP block = 2 studies					
Sullivan 2019 ⁷⁹	Patient for radical mastectomy + axillary dissection, $n = 1$.	Unilateral T3 ESP block.	20 mL 0.5% ropivacaine + epinephrine.	Clinical observation. Cutaneous sensory testing modality unspecified.	Unilateral facial erythema and upper limb diaphoresis lasting 12 hr (Harlequin syndrome). Hypotension lasting 4 hr. Cutaneous sensory loss from T2 to T5.
Bang 2020 ⁴	Patients with CRPS of the upper limb, $n = 2$.	Unilateral T3/T4 ESP catheter.	Loading dose of 10 mL 0.5% lidocaine. Continuous infusion of 4 $\text{mL}\cdot\text{hr}^{-1}$ 0.187% ropivacaine.	Clinical observation.	Reduction in pain scores, frequency of breakthrough pain, opioid use. Improvement in abnormal cold sensations in upper limb.
Lumbar ESP block = 5 studies					
Tulgar 2018 ⁹⁰	Hip and proximal femur surgery, $n = 1$.	Unilateral L4 ESP block.	20 mL 0.5% bupivacaine + 10 mL 2% lidocaine + 10 mL 30% iohexol.	CT imaging 90 min after block.	CT imaging showed spread (1) posterior to TPs from T12 to S1, (2) to neural foramina at L1–2, (3) around L4–5 spinal nerves, (4) into psoas muscle at L3, (5) around obturator and femoral nerve.
Chung 2018 ¹⁴⁸	CRPS of lower limb, $n = 1$.	Unilateral L4 ESP catheter.	5 mL 2% lidocaine + 15 mL radiocontrast bolus injection. Infusion of 4 $\text{mL}\cdot\text{hr}^{-1}$ 0.187% ropivacaine for 12 days.	Fluoroscopy.	Contrast spread observed from L2 to S1, but not involving psoas muscle. Pain score reduced from 7–8/10 to 3/10. Loss of cold sensation over L2–S1.

Table 2 continued

Author/year	Subject population	ESP block	Injectate	Method of assessment	Findings
Mantuani 2019 ¹⁸	Young man with acute appendicitis in the ED.	Unilateral L1 ESP block.	10 mL NS + 20 mL 1% lidocaine + epinephrine 5 $\mu\text{g}\cdot\text{mL}^{-1}$.	Clinical observation. Cutaneous sensory loss to cold.	Improved pain scores from 7 to 0/10. Resolution of guarding and rebound tenderness. Cutaneous sensory loss to cold from T10 to L2.
Elkoundi 2019 ⁸⁰	Patient with CRPS of foot and ankle.	Unilateral L4 ESP block.	30 mL 0.5% bupivacaine + 2% lidocaine.	Clinical observation.	Painless priapism 1 hr after block, lasting 3 hr, attributed to sympathetic blockade and unopposed parasympathetic stimulation. Improved pain scores from 10 to 0–2/10 and resolution of CRPS symptoms
Celik 2019 ⁸⁸ Ahiskalioglu 2020 ⁸⁹	Unilateral leg and back pain from lumbar disc herniation, $n = 1$.	Unilateral L4 ESP block.	20 mL bupivacaine + 10 mL lidocaine + 8.6 mL normal saline + 1 mL methylprednisolone + 0.6 mL gadobutrol.	MRI at 45 and 60 min after block.	Duplicate publication of same case. MRI showed spread to epidural spaces at L2–L5, intervertebral foramina at L1–L5, cranio-caudal spread in ESM from L1 to S4. Pain scores reduced from 9/10 to 0/10.

CRPS = complex regional pain syndrome; CT = computed tomography; DRG = dorsal root ganglion; ED = emergency department; ESP = erector spinae plane; ESM = erector spinae muscle; IQR = interquartile range; MRI = magnetic resonance imaging; PVS = paravertebral space; TLF = thoracolumbar fascia; TP = transverse process.

spread to the intercostal space (which is contiguous with the paravertebral space)²³ at multiple levels has also been reported in some studies,^{1,49,50} and may be an additional contributing mechanism for blockade of the ventral rami.

The original study by Forero *et al.*¹ reported extensive cutaneous sensory loss over the entire hemithorax consistent with blockade of dorsal and ventral rami of spinal nerves. In clinical studies that evaluated for sensory loss to cold or pinprick within 20–40 min of a preoperative mid-thoracic ESP block, this was not detectable in 2.4% (7/290) of patients.^{51–57} These observations of subtle or absent cutaneous sensory loss have called into question blockade of the ventral rami within the paravertebral space as the underlying mechanism of analgesia in ESP blocks.

There are several possible explanations for the inconsistent pattern of cutaneous blockade seen with ESP blocks. The first is overt block failure, due to sequestration of local anesthetic within the muscle or impaired diffusion into the paravertebral space, as may happen if the correct plane is not targeted.⁵⁸ The sonographically visible fascial plane between erector spinae muscle and transverse processes is structurally complex, consisting of closely apposed epimysium, thoracolumbar fascia (itself a multilamellar structure),¹⁶ inter-transverse ligaments, and periosteum. Precise placement of the needle tip in the desired site can therefore sometimes be a challenge, one that is further compounded by the fact that accurate needle-beam alignment and needle tip visualization in ultrasound-guided regional anesthesia is a complex skill.^{59,60} Although an oft-cited advantage of the ESP block is its easily recognizable injection endpoint of linear spread between the deep fascia of erector spinae muscle and transverse processes, this can sometimes be mimicked by an intramuscular injection, and technical modifications such as a transverse view have been suggested to help differentiate between them.⁶¹

The second explanation is the imprecision of cutaneous sensory testing and its imperfect correlation with analgesia, which is in turn related to the concept of *differential neural blockade*. Various cutaneous sensory testing modalities are subserved by different nerve fibre types—light touch, pinprick, and temperature by A-beta, A-delta, and C-fibres, respectively.²⁷ These fibres exhibit differing sensitivities to local anesthetic conduction blockade. If local anesthetics are applied at sufficiently low concentrations, this will produce differential blockade of these fibres with regard to latency, intensity, and duration.^{28,29,62}

In general, smaller myelinated fibres, such as A-delta nociceptors that transmit “fast” or “first pain”⁶³ (e.g., pinprick), are more susceptible to conduction blockade than larger myelinated A-beta and A-alpha fibres, which

Table 3 Anatomical spread of injectate in human cadaveric studies of the erector spine plane block

Author/Year	ESP injections	Epidural space	Paravertebral space	Ventral rami	Dorsal rami	Sympathetic chain	Intercostal space
Thoracic ESP block (16 studies)							
Aponte 2019 ²⁰	T7 (n=6)	No	No	No	Yes (n=4)	NA	NA
Forero 2016 ¹	T5 (n=4)	No	No	Yes (n=1)	Yes (n=2)	No	Yes (n=2)
Ivanusic 2018 ¹⁵	T5 (n=20)	No	No	Yes (n=1)	Yes (n=14)	No	No
Chin 2017 ⁴⁹	T7 (n=2)	No	No	NA	NA	NA	Yes (n=2)
Shibata 2020 ²⁴	T4 (n=5)	NA	No	No	Yes (n=5)	NA	No
Vidal 2018 ⁵⁰	T4/5/10 (n=9)	No	Yes (n=9)	NA	NA	NA	Yes (n=9)
Nielsen 2019 ¹³⁹	T5 (n=5)	No	Yes (n=3)	Yes (n=3)	Yes (n=3)	NA	NA
Elsharkawy 2019 ¹⁴³	T10–11 (n=6)	No	Yes (n=3)	Yes (n=3)	Yes (n=6)	NA	Yes (n=4)
Govender 2020 ¹⁴⁴	T8 (n=1) T10 (n=1)	No	Yes (n=2)	Yes (n=2)	Yes (n=2)	NA	Yes (n=2)
Yang 2018 ⁴⁸	T5 (n=10)	NA	Yes (n=10)	Yes (n=10)	Yes (n=10)	Yes (n=10)	No
Dautzenberg 2019 ¹⁴¹	T2 (n=11) T8 (n=11)	NA	Yes (n=5)	Yes (n=5)	Yes (n=5)	NA	NA
Choi 2019 ¹²⁸	T5 (n=14)	Yes (n=1)	Yes (n=7)	Yes (n=7)	Yes (n=14)	Yes (n=1)	No
Altinpulluk 2019 ¹⁴²	T9 (n=8)	Yes (n=5)	Yes (n=6)	Yes (n=6)	Yes (n=8)	NA	NA
Adhikary 2018 ⁴⁷	T5 (n=3)	Yes (n=3)	Yes (n=3)	Yes (n=3)	Yes (n=3)	NA	Yes (n=3)
Diwan 2019 ¹⁴⁰	T4–5 (n=5)	Yes (n=5)	Yes (n=5)	Yes (n=5)	Yes (n=5)	Yes (n=2)	Yes (n=5)
Govender 2020 ¹³⁰	T5 (n=1) T8 (n=1)	Yes (n=2)	Yes (n=2)	Yes (n=2)	Yes (n=2)	NA	NA
Lumbar ESP block (1 study)							
De Lara Gonzalez 2019 ¹⁴⁵	L4 (n=12)	No	NA	Yes (n=2)	Yes (n=12)	NA	NA
Cervical ESP block (1 study)							
Author/year	ESP injections	Epidural space	Spinal nerve roots	Phrenic nerve	Suprascapular nerve	Long thoracic nerve	Dorsal scapular nerve
Elsharkawy 2020 ²⁵	C6 (n=5) C7 (n=5)	No	Yes (n=10)	Yes (n=3)	Yes (n=10)	Yes (n=10)	Yes (n=10)

ESP = erector spinae plane; NA = not assessed

are responsible for mechanosensation and proprioception, respectively. The smallest unmyelinated C-fibres responsible for “slow” or “second pain”⁶³ and temperature sensation, have a less predictable response. Some studies suggest that they do not follow the “size principle” and are less susceptible to blockade than A-fibres are.⁶⁴ Nevertheless, this phenomenon is only observed with lidocaine. Bupivacaine and ropivacaine (the local anesthetics most commonly used in ESP blocks) consistently display preferential blockade of C-fibres vs A-delta fibres vs A-beta fibres (in that order) in both

preclinical and clinical studies.^{28,62,65} This is attributed to the higher pKa and lipid solubility of bupivacaine and ropivacaine compared with lidocaine, which facilitates intraneural diffusion and ion channel blockade. This also explains the relative motor-sparing exhibited by ropivacaine, which is slightly less lipid-soluble than bupivacaine is.⁶⁶ C-fibres also have greater susceptibility to use-dependent conduction blockade, which may enhance differential block in pain states.⁶⁵

The clinical effect of neural conduction block is thus a complex continuum, rather than an “all or none”

phenomenon, which is dependent upon the fibre composition of the target nerve and the mass of local anesthetic acting on it. In general, increasing concentrations of bupivacaine or ropivacaine around a nerve will produce a progressive loss of sensory function, starting first with the perception of “slow pain” and heat/cold, followed by “fast pain”, touch or pressure, proprioception, and finally loss of motor function as well.

There are several ways this may manifest in clinical practice. It is most commonly seen during the onset phase of any regional anesthetic. As an increasing mass of local anesthetic diffuses towards and accumulates around the nerve fibres, there is gradual progression towards a more “dense” sensory block with a classic sequential loss of pinprick sensation followed by pressure sensation.⁶⁷ It is also evident in selective or “motor-sparing” blocks where pain sensation is inhibited without significant motor weakness or complete sensory loss. This is usually produced by the deliberate use of local anesthetic at very low concentrations^{29,62}—ambulatory labour epidural analgesia with bupivacaine or ropivacaine⁶⁸ is a prime example—but a similar phenomenon may result from injection of local anesthetic some distance away from target nerves.⁶⁹ The small fraction of local anesthetic reaching the paravertebral-epidural space in the ESP block may well represent the same principle at work.

A third consideration is that cutaneous innervation is highly variable and more complex than depicted in most anatomy textbooks. There are numerous sensory connections between peripheral nerves that blur the boundaries described in conventional dermatomal maps of cutaneous sensory innervation,⁷⁰ and any given patch of skin receives multi-segmental innervation. There is also contralateral crossover innervation of the territory in the midline of the anterior torso.^{70,71} This may serve to reconcile the preservation of parasternal cutaneous sensation observed after *unilateral* thoracic ESP block^{72,73} with the reported efficacy of *bilateral* ESP blocks in median sternotomy.^{74–76}

Finally, the testing interval may also be a determining factor in apparent cutaneous sensory loss, as sensory block often continues to progress in intensity and extent beyond the customary 30–45 min used in most studies.²⁴

Other nerve targets involved in physical spread of local anesthetic in thoracic ESP block

Lateral cutaneous branches

It has been suggested that the clinical effects of the ESP block are primarily due to isolated blockade of the lateral cutaneous branches rather than a more proximal action on the ventral rami and intercostal nerves. This is based on

one cadaveric study in which injectate spread was confined to the plane superficial to the ribs and intercostal muscles and did not penetrate the paravertebral or intercostal space to any meaningful extent.¹⁵ Nevertheless, there are several reasons why this explanation may be inadequate. The vast majority of studies show that the injectate does not spread beyond the lateral boundary of the erector spinae muscle, and does not reach the lateral cutaneous branches which arise at the angle of the ribs and only emerge into this plane close to the posterior axillary line. More importantly, there is ample clinical evidence that the ESP block produces physiologic responses due to neural blockade at paraneuraxial sites. These include analgesia in purely visceral pain syndromes (acute appendicitis,¹⁸ pancreatitis,^{19,77} renal colic),⁷⁸ complex regional pain syndrome,^{3,4} sympathetically mediated phenomena (Harlequin syndrome,⁷⁹ priapism,⁸⁰ hypotension,⁸¹ and motor blockade.^{82,83} Relief of pain emanating from deeper bony and muscular structures must also imply a site of action at the ventral rami rather than at cutaneous branches alone.

Dorsal rami of the spinal nerves

Much of the early attention given to the ESP block was focused on analgesia in the distribution of the ventral rami of the spinal nerves. Nevertheless, it is unequivocally clear that local anesthetic spread associated with the ESP block will also block branches of the dorsal rami as they ascend through the plane of injection and this has been repeatedly shown in many studies (Table 3). These branches innervate the spine and paravertebral tissues and account for the analgesic efficacy of the ESP block in surgery on the spine and back.^{8,84}

Physical spread of local anesthetic in lumbar and cervical ESP blocks

Physical local anesthetic spread at lumbar vertebral levels

The concept of injecting between the transverse processes and the overlying erector spinae muscle has been extrapolated to the lumbar spine but there are significant differences in its anatomy compared with that of the thoracic spine. First, the erector spinae muscles are larger and thicker, they have tendinous attachments to the lumbar transverse processes,⁸⁵ and the plane between the two is thus not as readily hydrodissected. Second, the psoas muscle is closely adherent to the vertebral bodies and the anterior surface of the transverse processes; there is no paravertebral space comparable with that of the thoracic spine. Lumbar nerve roots also emerge from the intervertebral foramina in close proximity to the anterior surface of the transverse processes before splitting into

dorsal and ventral rami.⁸⁶ As in the thoracic spine, the dorsal rami penetrate posteriorly into the erector spinae muscle,⁸⁶ whereas the ventral rami run anteriorly into the psoas muscle and unite to form the lumbar plexus within a psoas muscle compartment. The nerves travel within interconnected fatty intramuscular compartments, which provide a potential route for injectate spread following a lumbar ESP block. In particular, the fat-filled plane between the erector spinae muscle and transverse process is continuous with the fat-filled psoas compartment that contains the lumbar nerve root and plexus, and also communicates with the epidural space (Fig. 3). These principles were borne out in a cadaver study of ESP injections at the level of the fourth lumbar (L4) vertebrae, which showed spread to the anterior aspect of the transverse processes and posteromedial border of the psoas muscle, with staining of the L3 and L4 spinal nerves in 75% and 17% of specimens, respectively.⁸⁷ Cranio-caudal spread was confined to the L1–L5 levels.⁸⁷ Fluoroscopic, CT, and MR imaging in living subjects have similarly confirmed that the injectate tracks to the paravertebral area, intervertebral foraminae, and epidural space following lumbar ESP block,^{3,88–90} and these findings correlate with those of studies reporting clinical analgesia of the proximal lower limb.^{91,92}

Physical local anesthetic spread at high thoracic and cervical vertebral levels

The ESP block has also been used to manage painful conditions of the upper limb, including degenerative shoulder disease,⁹³ complex regional pain syndrome,⁴ burn injuries,⁹⁴ and forequarter amputations.^{5,95} In these instances, ESP blocks are performed at the high thoracic level (T1–T3) or the low cervical level (C6–C7). The cervical components of the erector spinae muscle (semispinalis, longissimus, and iliocostalis cervicis) extend from the thoracic spine to insert on the C2–C6 transverse processes, and CT imaging in a living subject has shown a pathway for spread of injectate from T2 into the vicinity of the brachial plexus roots.⁹³ Similarly, cadaveric injection at the C6 and C7 level consistently produced staining of the C5–C8 nerve roots, as well as the suprascapular, dorsal scapular, and long thoracic nerves that innervate the shoulder girdle.²⁵

Systemic effect of local anesthetic injected into fascial planes

Fascial plane blocks such as the ESP block are characterized by the injection of large volumes of local anesthetic at doses close to maximum recommended limits,

Fig. 3 (A). Transverse magnetic resonance imaging (MRI) scan through the L3 vertebra. A fat-filled fascial compartment (arrows) is visible between the transverse process (TP) and the overlying erector spinae muscle (composed); this is the target plane in the lumbar erector spinae plane (ESP) block. The branches of the dorsal rami ascend posteriorly in the intermuscular planes between iliocostalis (IC), longissimus thoracis (LT) and multifidus (MF) muscles (which make up the erector spinae muscle); these planes are continuous with this compartment. The roots of the lumbar plexus are visible in the fat-filled layer between psoas major muscle (PM) and the vertebral body (dashed arrows). (B). Transverse MRI scan through the L3–4 intervertebral disc, caudad to the L3 articular process, TP and vertebral body (overlaid as a dot-dashed line). Note that the fat-filled compartment (solid arrows) between the erector spinae muscle and TP illustrated in (A) is continuous with the fat-filled psoas compartment that contains the lumbar nerve root and plexus (dashed arrows), and also communicates with the epidural space. This is the pathway for physical spread of local anesthetic in the erector spinae plane (ESP) block. (Image reproduced with permission from KJ Chin Medicine Professional Corporation.) AP = articular process; IC = iliocostalis; LT = longissimus thoracis; MF = multifidus; PM = psoas major; QL = quadratus lumborum; SP = spinous process; VB = vertebral body.

and it has been suggested that this produces plasma concentrations that may have systemic analgesic effects.⁷³

How do systemic local anesthetics produce analgesia?

There is good evidence for the analgesic benefit of intravenous (IV) lidocaine infusions in managing acute pain.^{96–98} The analgesic mechanisms are incompletely

understood, but are thought to involve neural and non-neural sites of action.⁹⁸

Neural effects include both central and peripheral effects. Lidocaine at clinically relevant plasma concentrations ($1\text{--}5\text{ }\mu\text{g}\cdot\text{mL}^{-1}$) acts on the spinal cord to inhibit excitatory activity of wide dynamic range neurons in the dorsal horn that are involved in central sensitization, as well as on the dorsal root ganglion to inhibit nociceptive transmission.^{99–101} Systemic lidocaine also acts directly on peripheral nerve endings to inhibit action potential propagation from A-delta and C-fibre nociceptors,¹⁰² although this is less significant than the central neuraxial effect.¹⁰³ Much higher lidocaine concentrations ($> 80\text{ }\mu\text{g}\cdot\text{mL}^{-1}$) are required to block action potential propagation in larger peripheral nerve fibres,¹⁰⁴ which would explain why sensory and motor block are not evident during IV lidocaine infusion, and similarly, why they are not always apparent following ESP and other fascial plane blocks.

Lidocaine also inhibits several different elements of the inflammatory pathway, including leukocyte adhesion to blood vessel endothelium, migration of immune cells into injured tissues, priming of neutrophils, and the release of inflammatory mediators.¹⁰⁵ These mediators are responsible for activating peripheral nociceptors, and thus the anti-inflammatory effect of IV lidocaine may account for its efficacy in treating conditions such as renal colic and critical limb ischemia where inflammation is a significant contributor to acute pain.⁹⁷ This mechanism may also partly explain the observed analgesic effect of the ESP block in acute appendicitis¹⁸ and pancreatitis.^{19,77}

Do ESP blocks achieve clinically significant local anesthetic plasma concentrations?

Plasma concentrations of $2\text{--}3\text{ }\mu\text{g}\cdot\text{mL}^{-1}$ are achieved 15–45 min after a single-injection paravertebral block with $5\text{ mg}\cdot\text{kg}^{-1}$ of lidocaine,¹⁰⁶ which is within the range of therapeutic IV lidocaine dosing regimens. There is currently no specific data on plasma lidocaine concentrations after ESP blocks. Nevertheless, one study comparing plasma levobupivacaine concentrations following either a continuous ESP or paravertebral block showed that while the time-dependent profile was similar, levobupivacaine concentrations were consistently 9–36% lower in the ESP block group.⁷³ Thus, while ESP blocks might result in effective local anesthetic plasma concentrations, we postulate that these are likely to be towards the lower end of the therapeutic range.

It should also be noted that plasma lidocaine concentrations decrease to $1\text{ }\mu\text{g}\cdot\text{mL}^{-1}$ or less by three hours after a single-injection paravertebral block.¹⁰⁷ Any systemic effect may therefore be less significant in single-

injection ESP blocks than continuous blocks, and probably cannot account for the prolonged postoperative analgesia reported in clinical studies.¹⁰⁸

A final consideration in extrapolating findings from studies of IV lidocaine to ESP and other fascial plane blocks is that the injectate in these blocks is almost always ropivacaine, bupivacaine, or levobupivacaine. Although most authorities agree that all local anesthetics are expected to share the same systemic properties of action,^{105,109,110} laboratory studies have found that pure S-enantiomers such as ropivacaine and levobupivacaine have less potent anti-inflammatory and anti-microbial properties.^{105,111} The clinical significance of this remains unclear.

In summary, while it is plausible that a systemic local anesthetic effect may contribute to analgesia in ESP blocks, this is by no means conclusive and requires further investigation.

Effects of injected local anesthetic on the spinal cord

Even if plasma local anesthetic concentrations achieved with ESP blockade are not adequate to exert a significant effect, the mechanism of action involved may still be relevant to ESP block. As discussed earlier, it is reasonable to assume that a small mass of local anesthetic penetrates into the paravertebral space, intervertebral foramina, and epidural space. Epidural spread in particular has been documented on MR or CT imaging in live subjects following ESP blocks.^{37,39,40,88} While this central neuraxial spread may not always be in physically detectable amounts, it may nevertheless be sufficient to produce therapeutic local anesthetic concentrations in the milieu surrounding the spinal nerve roots, dorsal root ganglia, or dorsal horn of the spinal cord, thus inhibiting nociceptive transmission and central sensitization in the same way that plasma-borne lidocaine is believed to act. These low neuraxial concentrations of local anesthetic would not, however, be expected to produce the same clinical effects (with regard to both quality of anesthesia and sympathectomy) as an appropriately dosed epidural anesthetic, where a much larger mass of drug is deposited directly around the neuraxis.

Immunomodulatory analgesic effect

An immunomodulatory mechanism of ESP blocks was recently postulated in a porcine study where dye spread to paraspinal lymph nodes, but not the paravertebral space, was observed.¹¹² The lymphatic system is an important circulatory system for endogenous and exogenous macromolecules that is increasingly being explored as a route for targeted drug therapies.¹¹³ Bidirectional

interaction between nociceptor neurons and the immune system is also a well-established phenomenon.^{114,115} It is therefore intriguing to consider if the delivery of local anesthetic via lymphatic channels to resident lymphocytes in lymph nodes might contribute to an immune-mediated, anti-inflammatory analgesic effect. Although much of the attention in immune-mediated peripheral nociception is focused on innate immune cells such as neutrophils and mast cells, T-lymphocytes also release cytokines (e.g., interleukin-5, interleukin-17, interferon gamma) that similarly activate peripheral nociceptors.¹¹⁴ Furthermore, T-lymphocytes have a role in central sensitization, participating in crosstalk with microglia, astrocytes, and oligodendrocytes to modulate synapses between nociceptor neurones and second-order interneurons in the dorsal horn.¹¹⁴ Any analgesic response mediated by the adaptive immune system would, however, likely be delayed compared with direct local anesthetic inhibition of neural transmission, and this would limit its contribution to acute postsurgical analgesia. For now, a lymphatic-based immunomodulatory mechanism of action for the ESP block, while not entirely improbable, remains more speculative than evidence-based.

Analgesic effect mediated by fascial innervation

Another speculative mechanism for fascial plane blocks relates to a direct action of local anesthetic on the fascia itself.¹¹⁶ There is no specific evidence for this, but proponents point to the rich innervation of the thoracolumbar fascia, particularly by sympathetic neurons, and the relationship between fascial mechanoreceptors, chronic back pain, and vasomotor reflexes.¹¹⁶ It is possible that local anesthetic may block nociceptors in the erector spinae muscle and thoracolumbar fascia and contribute to the efficacy of the ESP block in treating acute and chronic back pain.^{7,84,117} Nevertheless, it is unclear how blocking these fascial targets could modulate pain from remote locations innervated by anatomically distinct nerves.

Another theory is based on a functional concept of fascia as a whole-body matrix of connective tissue that links not only muscles but also other organs and body systems.¹¹⁸ It is postulated that fascial stimulation by physical therapy or acupuncture needles may trigger modulation of cellular processes not only in surrounding tissue but also in distant sites through connecting fascial planes.^{119,120} The precise nature of these interactions remains vague, but their existence is often invoked to explain the therapeutic basis for acupuncture, itself a controversial treatment modality.^{121,122} Nevertheless, preclinical animal studies of electroacupuncture indicate that it is capable of blocking inflammatory, neuropathic, and visceral pain, and that this

is neurally mediated through peripheral, spinal, and supraspinal mechanisms involving the release of endogenous opioids, serotonin, norepinephrine, and other neurotransmitters.¹²³ It is intriguing to note that ESP injection sites at the tips of the vertebral transverse processes correspond almost exactly to acupuncture points on the bladder meridian.¹²⁴ Nevertheless, this may be purely coincidental, as painful conditions traditionally linked to the bladder meridian are limited to the lower abdomen, spine, and leg. Acupuncture needles also rarely penetrate beyond skin, subcutaneous tissue, and the superficial fascia.¹²⁰ Most importantly, the analgesic effect of acupuncture is abolished by local anesthetic injection.^{125,126} Overall, it is therefore unlikely that ESP block analgesia is mediated by any direct effect on the thoracolumbar fascia.

Summary and implications for clinical practice

Based on the current evidence base, direct spread and action of local anesthetic on neural targets is the most plausible fundamental mechanism of analgesia provided by the ESP block. A systemic effect of elevated local anesthetic plasma concentrations is less likely to be a significant contributor, especially for single-injection blocks. Other more esoteric explanations related to fascia-mediated or lymphatic mechanisms remain speculative at this time.

The likely neural targets involved in direct spread are 1) nerves passing within the ESP or the erector spinae muscle (e.g., branches of the dorsal rami), and 2) nerves in compartments that are contiguous with the ESP via channels created by perforating neurovascular structures or intermuscular planes (e.g., spinal nerve roots, ventral rami, brachial plexus). This is consistent with the observed clinical effects of ESP blockade—i.e., somatic analgesia of both cutaneous as well as deeper musculoskeletal tissues, visceral analgesia, and manifestations of sympathetic blockade. Only a small fraction of injected local anesthetic finds its way into the paravertebral and epidural space (and does so in a time-dependent manner), but there is nevertheless good preclinical evidence that the resulting low concentration around neural targets in these compartments exerts selective yet significant effects on nociceptive transmission and processing.

The active mass of local anesthetic will vary with technical performance, volume injected, speed of injection, dynamic variation in intra-compartmental pressures, and tissue permeability, among other factors. These variables account for the range of experimental and clinical results that have been reported, and are the Achilles' heel of studies interpreting ESP blocks. It is probably unreasonable

to expect fascial plane blocks such as the ESP block to behave like other regional anesthesia techniques, given the inherent lack of direct visualization and injection around actual targets of interest (e.g., intercostal nerves, and paravertebral and epidural space). Nevertheless, moving forward the focus should be on clinical studies that seek to determine not only if the ESP block provides effective analgesia but also how to improve its success rate and consistency of effect. These should incorporate robust methods of sensory assessment, including modalities that are more objective or nociceptive-specific (e.g., thermal quantitative sensory testing)¹²⁷ and, most importantly, meaningful patient-centred outcomes.

Our current understanding of the ESP block invites several considerations regarding its performance. Spread into the paravertebral space is most likely at the level of injection as well as 1–2 levels higher and lower¹²⁸; thus, the targeted transverse process should be congruent with the spinal nerve(s) innervating the area of desired effect. Physical spread is related to injected volume, and the evidence indicates that the optimal volume in adult patients is greater than 10 mL, with 20–30 mL most commonly used.^{48,128} There is less data in the pediatric population, but 0.2–0.3 mL·kg⁻¹ is generally recommended.^{129,130} For the same reason, intermittent boluses (programmed or patient-controlled) may be preferred over continuous infusion-only regimens in continuous ESP blockade, an advantage that has been reported in continuous paravertebral blockade.^{131–133} Local anesthetic concentration, which in turn determines mass of drug, is another factor that has been linked to efficacy¹³⁴ but also requires further investigation. Finally, technique modifications involving injection into the inter-transverse tissue complex located deep (i.e., anterior) to the fascial layer investing the deep surface of the erector spinae muscle may also promote spread into the paravertebral space.^{24,135,136} At the same time, it must be recognized that any strategies (e.g., deeper injection, larger injection volumes) designed to increase paravertebral spread also increase the risk of adverse effects such as hypotension and motor blockade.^{81–83} Furthermore, maximum recommended doses must be observed when calculating concentration and total injected volume of local anesthetic, as local anesthetic systemic toxicity has been reported with the ESP block.^{137,138} One of the chief attractions of the ESP block has been its perceived favourable risk-benefit ratio, and this should not be compromised going forward.

In conclusion, the ESP block is a promising technique that has a growing evidence base to support its use in clinical practice. While we have not definitively excluded other mechanistic theories, the most probable action of any significance is via blockade of neural targets. This understanding will assist clinicians in investigating and

refining performance of the ESP block, with the ultimate goal of optimizing analgesic efficacy and improving postoperative patient outcomes.

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