Spread of local anesthetics after erector spinae plane block: an MRI study in healthy volunteers

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ABSTRACT
Background  Erector spinae plane block (ESPB) is a truncal fascial block with a disputed mechanism and anatomical site of effect. This study aimed to perform a one-sided ESPB and use MRI to investigate the spread of the local anesthetic (LA) and the corresponding cutaneous loss of sensation to pinprick and cold.

Methods  Ten volunteers received a right-sided ESPB at the level of the seventh thoracic vertebra (Th7), consisting of 30 mL 2.5 mg/mL ropivacaine with 0.3 mL gadolinium. The primary outcome was the evaluation of the spread of LA on MRI 1-hour postblock. The secondary outcome was the loss of sensation to cold and pinprick 30–50 min after the block was performed.

Results  All volunteers had a spread of LA on MRI in the erector spinae muscles and to the intercostal space. 9/10 had spread to the paravertebral space and 8/10 had spread to the neural foramina. Four volunteers had spread to the epidural space. One volunteer had extensive epidural spread as well as contralateral epidural and foraminal spread. Four volunteers had a loss of sensation both posterior and anterior to the epidural space, the paravertebral space, and the neural foramina while the spread to the epidural space was inconsistent. We show that sensory loss is not indicative of the actual spread of local anesthetics, and can probably not confidently be used to assume clinical effect.

Conclusion  We found that LA consistently spreads to the intercostal space, the paravertebral space, and the neural foramina after an ESPB. Epidural spread was evident in four volunteers. Sensory testing 30–50 min after an ESPB shows highly variable results, and generally under-represents what could be expected from the visualized spread on MRI 60 min after block performance.

Trial registration number  NCT05012332.

INTRODUCTION
Erector spinae plane block (ESPB) is one in a line of new truncal fascial blocks that in contrast with traditional nerve blocks do not target specific peripheral nerves, but rather depend on indirect spread of local anesthetics (LA) to reach the intended neural structures to induce analgesia. Since its inception in 2016 by Forero et al.,1 the ESPB has received significant interest from the regional anesthesia community, based on possible indications ranging from cardiothoracic and abdominal to lower limb surgery.1

The ESPB needle targets the transverse process under ultrasound guidance at approximately the level of desired analgesic effect.2 LA is then injected and spreads in the potential space between the transverse process and the erector spinae muscles. For an ESPB to be effective in providing analgesia to either the anterolateral chest wall or the anterolateral abdominal wall, the LA must reach some or all of the following neural structures; the ventral rami of the spinal nerve, the dorsal root ganglion, the intercostal nerves or the epidural space.3 LA may reach these structures by rapid diffusion of the injected LA across the intertransverse connective tissue complex into the paravertebral space and then potentially the epidural space according to one hypothesis.2 Another hypothesis for analgesic effect suggested is the systemic effect of LA.3 The mechanisms behind the observed clinical effect of the ESPB remain controversial, and further investigations into the precise mechanisms of the block have been called for.1

WHAT IS ALREADY KNOWN ON THIS TOPIC
⇒ One previous MRI study and a few single case reports show that the erector spinae plane block (ESPB) spread to the erector spinae muscles, the dorsal rami, the intercostal space, and neural foramina. Clinical studies have demonstrated adequate analgesic effect of the ESPB in thoracic and some abdominal surgery procedures. Only one study has assessed the MRI spread as well as loss of cutaneous sensation simultaneously in live subjects. Hence, further studies are needed to support clinical decision-making regarding nerve blocks in the truncal area.

WHAT THIS STUDY ADDS
⇒ In this study, we present data on MRI spread and loss of cutaneous sensation in relation to the ESPB. The data show a consistent spread of local anesthetics to the intercostal space, the paravertebral space, and the neural foramina while the spread to the epidural space was inconsistent. We show that sensory loss is not indicative of the actual spread of local anesthetics, and can probably not confidently be used to assume clinical effect.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY
⇒ This study substantiates the claim of clinical analgesic effect, not cutaneous testing, to be in focus when performing an ESPB, and adds evidence to the discussion of the mechanisms behind the ESPB.
This study aimed to investigate the spread of LA after a one-sided ESPB using MRI and compare the spread of LA against the cutaneous loss of sensation to pinprick and cold. This would then contribute to more knowledge pertaining to the mechanisms behind this relatively new fascial block.

METHODS
Participants and recruitment
Healthy volunteers working at the hospital were recruited after reading an invitation published on the hospital’s intranet front page. The inclusion criteria were age >18 years with no comorbidities. Exclusion criteria were: allergy to latex, gadolinium and LA, body mass index (BMI) >40, severe renal and/or hepatic disease, local infection at the site of injection, systemic infection, atrioventricular block 2–3, inability to understand written or spoken Norwegian, inability to cooperate, claustrophobia, pregnancy and metal implants not MRI-compatible.

As the volunteers made contact, they received an email sent by the study nurse. The mail included the patient information sheet and a consent form. If the volunteer fulfilled the eligibility criteria and consent was obtained, the volunteer was contacted by mail to set up an intervention date. A signed informed consent form was then obtained from all volunteers prior to the intervention. All volunteers were given compensation in the form of a fixed gift card of US$100 to cover any travel expenses.

Intervention
Every intervention was done under standard American Society of Anesthesiologists (ASA) monitoring and with intravenous access established. All volunteers were given the choice of procedural pain relief in the form of alfentanil 0.5–1 mg. The skin was prepared thrice with chlorhexidine 5 mg/mL with added phenol red. All interventions were performed in a sitting position under ultrasound guidance (Fujifilm, Sonosite, X-porte ultrasound system, Bothell, Washington, USA). A linear probe (Sonosite HFL50xp) was used if the depth of the transverse process was <4 cm and a curvilinear probe (Sonosite C60XP) if the depth was ≥4 cm. The probe was covered with a sterile probe cover. After identifying the transverse process underneath the Erector spinae muscles, the needle (Stimuplex Ultra 360, 20 G, 100 mm, Braun Medical AG, Melsungen, Germany) was inserted from cranial to caudal using an in-plane technique and a parasagittal view at the level of the seventh thoracic vertebra (Th7). The level of Th7 was determined and marked before the start of the intervention by counting the ribs cranially from the 12th rib using the curvilinear probe. All volunteers received a unilateral ESPB with 30 mL of 2.5 mg/mL ropivacaine and a total of 0.3 mL gadolinium (Clariscan 0.5 mmol/mL, GE Healthcare AS, Oslo, Norway). The time of the block was registered. All volunteers lay supine in a bed with a slightly raised head between the intervention and the testing. Any decrease in heart rate and blood pressure combined with clinical symptoms was treated with 0.5 mg atropine, which could be repeated in case of continuing symptoms.

MRI
All MRI examinations were performed with the same Siemens MAGNETOM Skyra 3T machine at Sykehuset Ostfold Kalnes. The Body 18 coil was combined with the spine 32 coil. T2-weighted sagittal images were acquired covering the spinal column from the level of C7 to L2. Isometric, fat-suppressed T1 SPACE sequences with a slice thickness of 0.9 mm and 3D reconstruction were performed. The T2-weighted images were mainly used for anatomical orientation. The distribution pattern of the gadolinium/ropivacaine mixture was evaluated using the T1 SPACE FS sequences. The spread was evaluated in the following regions: erector spinae muscle plane, paravertebral space, intercostal space, neural foramina, and epidural space. If the contrast signal was clear, the spread was marked down as positive. If the contrast signal was subtle or uncertain, the radiologist maintained conservative reporting and marked the spread as negative. All images were reviewed and analyzed by the same radiologist blinded to the cutaneous loss of sensation to pinprick and cold.

Outcome measures
The primary outcome was the evaluation of the spread of LA on MRI 1-hour postblock. The secondary outcome was the loss of sensation to cold and pinprick 30–50 min after the block was performed.

The loss of sensation to pinprick was tested using an 18G short bevel needle, while the loss of sensation to cold was tested with ice cubes inside a latex glove. The area with reduced sensation was marked on the skin of the volunteer and photographed. The reduced sensation to pinprick was marked with a continuous line, while the reduced sensation to cold was marked with a dashed line. The testing of sensation was performed over a period of 15–20 min, and if the area of reduced sensation expanded within this time frame, the largest area was recorded. The volunteers were instructed to regularly check for the return of sensation and mark the time of the return of normal sensation. The time of cessation of the block was then reported to the researcher the next day by either a text message or email.

Analyses
The MRI images were interpreted descriptively, and the cranio-caudal spread of LA and gadolinium in the predetermined anatomical locations was reported.

STATA V.17.0 (StataCorp 2019. Stata Statistical Software: Release 17, StataCorp) was used as statistical software. Ordinal numerical data are presented as median (IQR data (25–75)).

RESULTS
Ten healthy volunteers were included. All included volunteers completed the study.

Table 1 presents volunteer characteristics.

Figure 1 presents the extent of the spread of LA with gadolinium, injected at the Th7 level as evident from the MRI series. The pertinent anatomical areas represented are the paravertebral column from the level of C7 to L2.
The average time reported was 396.5 min (6.6 hours). Two volunteers (volunteers 6 and 8) needed 0.5 mg atropine as they had bradycardia with accompanying hypotension after the block was performed. The symptoms resolved after one dose of atropine and neither needed a second dose.

Online supplemental figure 5 presents axial and sagittal MRI of all volunteers. Online supplemental figure 6 presents ultrasonic images of an ESPB.

**Table 2** Spread of LA/gadolinium

<table>
<thead>
<tr>
<th>Volunteer no</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Spread percentage</th>
<th>Cephalad spread (median)</th>
<th>Caudal spread (median)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercostal</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>100</td>
<td>3</td>
<td>2.5</td>
</tr>
<tr>
<td>Paravertebral</td>
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<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<td>Yes</td>
<td>Yes</td>
<td>90</td>
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<tr>
<td>Neural foramina</td>
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<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>80</td>
<td>2</td>
<td>0.5</td>
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<tr>
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<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
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<td>No</td>
<td>No</td>
<td>40</td>
<td>0</td>
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<tr>
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<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>100</td>
<td>5</td>
<td>4.5</td>
</tr>
</tbody>
</table>

LA, local anesthetic.

**DISCUSSION**

This study is one of few studies investigating the spread of LA as well as simultaneous cutaneous loss of sensation to pinprick and cold after ESPB in healthy volunteers. Our results showed consistent spread of LA to the dorsal rami of the spinal nerve, through the intertransverse connective tissue complex into the paravertebral space, and to the neural foramina when performing ESPB at the level of Th7. However, spread to the epidural space was seen in only four out of ten volunteers. There was great variability in the cutaneous loss of sensation.

Our study corresponds with many of the findings by Schwartzmann et al.5 Their study was an observational MRI study that evaluated the spread of LA in six patients with pain who received an ESPB of 30–35 mL at the level of Th10. Their results showed spread to the Erector spinae muscles, the dorsal rami, the intercostal space, and neural foramina in all six patients included. Schwartzmann et al.6 showed intercostal spread to a median of 9 levels (5–11), while we had a more limited spread with a median of 5.5 levels (5–7). Their median spread to the neural foramina was 3 (2–6), while it was 4 (2–5) in our study. Two of six patients had spread to the epidural space, and the authors concluded that spread to the epidural space was an unlikely mechanism with this volume of injectate (30–35 mL). We used the same volume and showed epidural spread in four of ten volunteers. In one volunteer, there was clear evidence of contralateral epidural spread at the level of Th6. There are single case reports of epidural spread after ESPB. Our study correlates with the findings of Schwartzmann et al6 who also demonstrated inconsistent spread to the epidural space. Despite this inconsistency, it is important to take into account the possibility of epidural spread when performing an ESPB.

Cadaver studies investigating the spread of LA after ESPB show conflicting results. The difference between the cadaver studies lies in the spread of LA or dye through the intertransverse connective tissue complex into the paravertebral space. There are studies showing none or very little spread,9–11 while other studies show staining of neural structures in the paravertebral space.12–15 The difference between using live patients, versus embalmed or fresh frozen cadavers is assumed to lie in the dynamic forces that live patients are subjected to.3 Examples of this are how muscles and fascia relax and slide over each other with movements, as well as the impact on the accentuated spread from elastic recoil of a distended fascial plane in live patients.3 Another important distinction between live patients and cadavers lies in the pressure...
differences created within the thoracic and abdominal cavity with breathing, which may transmit to the paravertebral space. This negative pressure gradient may accentuate the diffusion of LA into the lower pressurized paravertebral space.\(^5\)

All volunteers in our study were tested for loss of sensation to cold and pinprick. We found that there was great variability in the area affected. Four volunteers (number 1, 2, 6, and 9) had loss of sensation to both modalities beyond the anterior axillary line, while six volunteers had loss of sensation contained to the posterior trunk. Three studies have performed sensory testing on 6, 12, and 6 volunteers, respectively, after receiving an ESPB at Th4, Th5, and Th10.\(^5\) Byrne and Smith and Zhang et al.\(^16\)\(^\text{17}\) showed a lack of consistent sensory change beyond the posterior chest wall, while Schwartzmann et al.\(^5\) had loss of sensation on the anterior chest wall in all subjects. Neither Byrne and Smith\(^16\) nor Zhang et al.\(^17\) performed MRI, however, Byrne and Smith discuss the possibility of spread to the paravertebral area as a cause of the extended sensory loss in one participant.\(^16\)

Barrios et al.\(^18\) performed a similar study on 18 patients, where the cutaneous loss of sensation to cold and pinprick at the midclavicular line was mapped after an ESPB at the level of Th5-Th7. They showed consistent spread over a mean of 9 dermatomes (range 8–11) in the midclavicular line, which is in contrast to our findings as well as the studies by Byrne and Smith and Zhang et al.\(^16\)\(^\text{17}\) Lastly, Solvi et al.\(^19\) studied 28 patients who received a bilateral ESPB at the level of Th9, where they report the sensory loss to pinprick but not too cold. In this study, sensory block of the ventrolateral and ventromedial area was present in 69% and 55%, which is a larger proportion than the 40% in our study.

We found discrepancies between what could have been expected from the MRI images and the loss of cutaneous sensation to cold and pinprick. Some of the volunteers had a small area of sensory loss even though the spread on MRI was large (eg, volunteers 4 and 8), while others had a larger area of sensory loss than could be deduced from the MRI (volunteers 9 and 5). When comparing the MRI results with the cutaneous sensory testing it seems that the loss of sensation may be a combination of the dorsal rami analgesia and more compound analgesia arising from the affection of the ventral rami either in the intercostal space or the paravertebral space, the dorsal root ganglion at the neural foramina, or the epidural space.

We saw a clear tendency that the area of loss of sensation to cold was more extensive than the area of loss of sensation to pinprick in 7/10 volunteers. This corresponds with early findings after spinal hyperbaric tetracaine in 1958, where Greene found that the level of sensory loss to cold reached approximately 2 dermatomes higher than loss of pinprick sensation. Thus, different nerve fibers present a differential sensitivity to varying concentrations of LA.\(^3\) Ropivacaine displays a preferential blockade of C-fibers (transmits the sensation of cold and slow pain) before A-delta fibers (transmits pinprick). A low concentration of LA at the target site combined with the differential

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**Figure 2** MRI of a right-sided erector spinae plane block performed at the level of Th7. (A) Axial view at the Th6 level demonstrating contrast spread to the paravertebral space, neural foramina, and the epidural space (yellow arrow). (B) Axial view at the Th6 level demonstrating the spread of contrast to the contralateral epidural space and neural foramina (yellow arrow). (C) Sagittal view of the spinal canal demonstrating contrast to the epidural space (yellow arrows) from the level of Th6 to Th8.
sensitivity of the nerves may explain why in many cases there is a clear analgesic effect of the ESPB but at the same time normal sensation in the area. A higher concentration of LA in this study could potentially have produced a more profound block with a larger area of loss of sensation.

However, in daily clinical practice, the use of an ESPB often demands the use of bilateral blocks and a repeated dosing schedule in catheters of appropriate volumes of LA to ensure adequate spread. As to avoid toxic doses, the use of lower concentrations of LA is often necessary, and for this reason, we chose to use ropivacaine 2.5 mg/mL.

Another point to consider is that we tested the block after 30 min and during the next 20 min. During this time frame, we observed that the area of cutaneous loss of sensation to cold and pinprick expanded while testing. Zhang *et al* found that the range of sensory loss was maximal at 1 hour. Hence, further studies should test the block after 1 hour, and await performing the MRI until 2 hours postblock.

In our opinion, it is not possible to deduce the actual spread from the sensory testing, other than to say that if the sensory testing shows anterior trunk wall affection, epidural spread is more likely to have occurred. This is based on that volunteers 1, 2, and 6 had a loss of sensation anterior to the midaxillary line and had evidence of epidural spread on MRI. However, volunteer 4 does not follow this pattern, nor does volunteer 9. Volunteer 6 had an epidural spread while the reported sensory loss was contained to a small area on the posterior trunk wall, and volunteer 9 had extensive spread but did not have a detectable epidural spread.

Contrast uptake from various vascular structures, mostly venous, posed some diagnostic difficulties but were identified according to known anatomical localization and typical MRI appearance. Fat suppression was inhomogeneous in some patients with higher BMI, but the sequences were adequate for the required evaluations. Fat suppression techniques suppress the signal intensity of fat tissue and facilitate the easier evaluation of contrast spread and enhancement on T1-weighted sequences. The main diagnostic difficulty was the evaluation of epidural spread. In a few patients, spread to the epidural space was obvious and certain. In others, there was a subtle increase in signal intensity in expected areas of spread. Future studies might profit from performing a non-contrast fat-suppressed T1 uptake, before the injection of the agents.
gadolinium/ropivacaine mixture, in addition to the sequences performed in our study.

A strength of this study is that we have included ten volunteers as recommended to show a “modicum of insight on the prevalence of LA spread to the paravertebral space”; all injections were performed by the same anesthesiologist experienced with the technique. A limitation of the study is that the block duration was based on self-reporting, and as such is subject to bias. Figure 4 must, therefore, be read with caution.

CONCLUSION
In this study, we show that LA consistently spreads to the dorsal rami of the spinal nerve, through the intertransverse connective tissue complex into the paravertebral space, and to the neural foramina after an ESBP. Spread to the epidural space was only seen in four out of ten volunteers. Hence, the analgesic effect of the ESBP is more likely to originate from the above-mentioned neural structures rather than from any epidural spread. Sensory skin testing 30–50 min after an ESBP shows highly variable results and generally underestimates what could be expected from the visualized spread on MRI.

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Contributors MS stands as guarantor for the overall content. MS, NZ, JR, JSV and A-CLL conceived and designed the study, MS performed the blocks, NZ reviewed and analyzed the MRI results. All authors participated in the manuscript writing and reviewed all references. MS, NZ, JR, JSV and A-CLL approved the final manuscript.

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Competing interests None declared.

Patient consent for publication Not applicable.

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Provenance and peer review Not commissioned; externally peer reviewed.

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