Secondary spread of caudal block as assessed by ultrasonography

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Editor’s key points
- Recent radiography-based studies failed to describe patterns of redistribution and secondary spread as a contribution to the final spread of caudal block in children.
- Using ultrasound, this study dynamically observed intraspinal distribution and cranial spread of caudally injected local anaesthetics.
- Horizontal intrasegmental redistribution and longitudinal cranial spread were identified as patterns of secondary spread.
- These patterns explain the difference between initial ultrasound-assessed cranial level and the final block level determined by cutaneous testing.

Background. Redistribution and secondary spread after the initial injection of local anaesthetics (LAs) are important factors that contribute to the final spread of caudal block in children. However, to date, these phenomena have yet not been studied in detail. Thus, the aim of this observational study was to define patterns of secondary spread and redistribution of a caudal block by means of real-time ultrasonography scanning and cutaneous testing.

Methods. Ultrasound assessment of LA spread within the caudal–epidural space and epidural pressure was followed during 15 min after initial injection (1.5 ml kg⁻¹, ropivacaine 0.2%) in 16 infants. At 15 min post-injection, cutaneous testing was also performed to assess the cranial dermatomal level of the block (at end-tidal sevoflurane 2.5%).

Results. The median ultrasound-assessed cranial spread was Th10 and Th8 at 0 and 15 min, respectively, and the sensory level at 15 min was Th4. The caudal injection was initially found to compress the terminal part of the dural sac, later followed by a partial re-expansion as epidural pressure was returning towards pre-injection values. An intrasegmental redistribution from the dorsal to the ventral compartment of the epidural space was also observed.

Conclusions. Two separate patterns of secondary spread of caudal block could be observed, being horizontal intrasegmental redistribution and longitudinal cranial spread. The observed bi-directional movement of cerebrospinal fluid (coined ‘the CSF rebound mechanism’) does explain a major part of the difference between the initial ultrasound-assessed cranial level and the final level determined by cutaneous testing.

Keywords: anaesthesia, paediatric; anaesthetic techniques, regional, caudal; equipment, ultrasound machines; measurement techniques, ultrasound

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Early publications on the spread of caudal block, generated by skin testing under concomitant light halothane anaesthesia, indicate a mid-thoracic level when injecting 1.0–1.5 ml kg⁻¹ of local anaesthetic (LA).1–4 However, more recent studies using radiographic visualization techniques fail to identify spread much above the thoraco-lumbar junction, despite using similar volumes of LAs.5–9

Except for analysing different dimensions concerning the spread of caudal block (cutaneous testing by skin pinch/pick vs radiographic determination of the most cranial level of the epidural injectate), the main difference between earlier and more recent studies is the timing of the assessments (cutaneous testing after ~20 min vs immediate imaging after injection). Thus, recent radiographic studies did not take secondary spread-redistribution into account.

The aim of the current observational study was to define the patterns of such secondary spread-redistribution by ultrasonographic visualization and to substantiate or refute our hypothesis that a bi-directional movement of cerebrospinal fluid (CSF) caused by the initial injection of LAs is an important mechanism responsible for secondary longitudinal cranial spread of the LA.

Methods

The study was approved by the Regional Ethics Review Board of Stockholm, Sweden (Nr 2009/1752-31/4). Written informed consent was obtained from the parents of all children.

Infants <6 months of age (ASA physical status I–III) undergoing elective inguinal hernia repair requiring caudal
anaesthesia for intra- and postoperative analgesia were eligible for inclusion in this prospective observational study.

Known contraindications to regional anaesthesia (e.g. coagulation disorders, allergy to amide LAs, or local infection at the planned injection site) or anatomical abnormalities of the lumbo-sacral spine were defined as exclusion criteria.

**Biometric data**

According to established clinical routine, the children were weighed on admission. Patient height was measured with the patient lying supine on the operating table after induction of anaesthesia, using a regular measuring tape.

**Anaesthetic protocol**

A peripheral venous cannula was already inserted on the general paediatric ward after prior EMLA cream application. General anaesthesia was thereafter induced by propofol (2–3 mg kg\(^{-1}\)), subsequently followed by insertion of a laryngeal mask airway to secure the airway. In patients undergoing laparoscopic surgery, tracheal intubation was performed, facilitated by either i.v. atropine (10 \(\mu\)g kg\(^{-1}\)) and 1 mg kg\(^{-1}\) of succinylcholine or 6% end-tidal sevoflurane. Succinylcholine was used purely for study reasons in order to allow later cutaneous testing. Anaesthesia was thereafter maintained by sevoflurane–oxygen–air (\(\text{FIO}_2 0.3\), end-tidal sevoflurane concentration 2.5%) with the patient breathing spontaneously. Standard monitoring devices [ECG, pulse oximetry, end-tidal \(\text{CO}_2\), and non-invasive arterial pressure (NIAP)] were used throughout the anaesthetic.

A separate staff anaesthetist, not involved in the study, was responsible for the general anaesthetic.

**Haemodynamic data**

Heart rate and non-invasive arterial pressure were recorded immediately before turning the patient to the left lateral decubitus position for performance of the caudal block (baseline) and also immediately after surgical skin incision (to determine block success/failure, see below).

**Caudal block procedure, epidural pressure monitoring, and ultrasound investigation**

After induction of anaesthesia, the child was positioned in the left lateral decubitus position. The lumbo-sacral junction was palpated and the spinous process of L5 was thereby identified. The lumbar spinal processes were thereafter palpated and counted in a rostral fashion to identify the L3, L4, and the Th12 spinal processes. The 12th rib was subsequently identified by ultrasound visualization and followed medially to further verify that the spinous process of Th12 had been correctly identified. The skin overlying the spinous process of L3, L4, and Th12 was then marked using a regular skin marking pen.

The ultrasound equipment used was a portable Sonosite M-Turbo software 3.4.1 (Sonosite Inc., Bothell, WA, USA) with a 38 mm wide 7–13 MHz linear array probe. An initial longitudinal paramedian ultrasonographic investigation was performed to identify the epidural space, the conus medularis, the cauda equina, and the termination of the dural sac.

At the level L3–4, the distance between skin and the dorsal dura and also the anterior–posterior (A–P) diameter of the dural sac (distance between the dorsal to ventral dura) were measured using the caliper function of the ultrasound machine.

After sterile preparation of the injection site, the sacral cornuae and sacro-coccygeal membrane were palpated. The caudal space was subsequently cannulated using a 24 G i.v. cannula (BD Neoflon\textsuperscript{TM}, Becton, Dickinson and Co., NJ, USA). An aspiration test was performed to exclude inadvertent intravascular placement, whereafter the caudal catheter was connected to the pressure transducer via a three-way stop-cock (BD Connecta\textsuperscript{TM}, Becton, Dickinson and Co.) and a baseline epidural pressure was obtained on the monitor (Infinity\textsuperscript{®}, Delta, Dräger GMBH, Lübeck, Germany). The LA solution was then manually injected at a rate of \(\sim 0.5\) ml s\(^{-1}\). A total volume of 1.5 ml kg\(^{-1}\) of 0.2% ropivacaine (Narop\textsuperscript{®}, AstraZeneca, Södertälje, Sweden) was used, representing the upper limit of safe dosage (3 mg kg\(^{-1}\)) as described by Bösenberg and colleagues.\textsuperscript{10}

Immediately after the completion of the injection, the three-way stop-cock was switched, so that the caudal–epidural space was directly connected to the pressure transducer and the epidural pressure was thereafter measured continuously. Post-injection epidural pressure values were noted in the study protocol immediately after injection, and 3, 6, 9, 12, and 15 min post-injection. Pressure values were only deemed reliable when pulse synchronous pressure variations could be seen on the concomitant pressure trace of the monitor.

During the injection, the ultrasound probe was maintained in the previously determined optimum paramedian position (see above) in order to visualize the cranial spread of the LA within the caudal–epidural space. The probe was moved cranially as needed in order to follow the advancement of the front of the LA bulk flow within the epidural space, as evidenced by an anterior displacement of the dorsal dura, resulting in a reduction in the A–P diameter of the dural sac (Fig. 1). After the injection of 1.5 ml kg\(^{-1}\) of LA, the front of the epidural LA was identified and was positioned in the middle of the ultrasound picture. A skin mark was made corresponding to the middle of the ultrasound probe and the level reached was later determined by counting the spinous processes from the previously indicated Th12 spinous process. If the ultrasonographic front of the LA was ‘hidden’ behind the bone shadow of the vertebral lamina, the front was approximated to the middle of the corresponding bone shadow.

Directly after the caudal injection and the ultrasonographic visualization of the instant cranial spread of the LA, the ultrasound probe was again positioned at the L3–4 level to measure the compression of the dural sac (distance in millimetres from the skin to dorsal dura and A–P dural sac diameter) at this level. New measurements were performed at the L3–4 level every 3rd minute during 15 min after the injection of LAs.
We also noted if any redistribution of LA solution could be visualized to the ventral part of the epidural space. Owing to the contents present in the ventral epidural space, it is very difficult to make exact ultrasound measurement between the ventral part of the dural sac and the anterior wall of the spinal canal (vertebral bodies and intervertebral discs). Thus, the absence or presence of LAs in the ventral part of the epidural space was categorized as 0 (no fluid layer visible), + (minor layer of fluid layer identified), or ++ (clearly identifiable fluid layer anterior to the dural sac). However, a millimetre estimate of the ventral redistribution space was generated by subtracting the sum of the measured skin–dorsal dura distance and the dura–dura distance at 3, 6, 9, 12, and 15 min from the same value measured immediately after injection when both the ventral epidural space and the dura were maximally compressed by the injectate in the dorsal epidural space.

After 15 min, the spinal epidural space was again scanned in a longitudinal fashion to determine the most cranial level of LA spread visible by ultrasound at this time point.

After the completion of the ultrasound examination sequence, the child was again carefully placed in the supine position and the level of cutaneous analgesia was immediately tested by sequential skin pinching from the umbilical level in a cranial direction. A skin fold was firmly pinched between the thumb and the index finger for 5 s and any positive response (increased heart rate and/or NIAP >15% compared with baseline in association with skin incision, and (iii) intraoperative need for supplemental administration of fentanyl (1 μg kg⁻¹), as judged by the attending anaesthetist.

The cutaneous testing was, thus, carried out ~15–17 min after the initiation of the caudal block and was performed at an end-tidal sevoflurane concentration of 2.5% (0.75 MAC).

Skin incision was performed not earlier than 20 min after the caudal block. The caudal block was considered a failure if one or more of the following was noted: (i) movement of the limbs in association with skin incision, (ii) an increase in heart rate, NIAP, or both of >15% compared with baseline in association with skin incision, and (iii) intraoperative need for supplemental administration of fentanyl (1 μg kg⁻¹), as judged by the attending anaesthetist.

Statistics
The three main study parameters were maximal cranial segmental spread and skin–dural distance and dural sac diameter. The data in Figure 3 are expressed by mean values and 95% confidence intervals. The regression lines in Figures 2(a) and 4 were calculated by linear regression. Patient characteristics are given as median (range).

Results
A total of 16 male infants were enrolled in the study. The patients were 12 (4–17) weeks old at the time of surgery and were born at 35 gestational weeks (29–39). Patient...
weight and height were 5.25 kg (2.27–6.89) and 56 cm (45–63), respectively. All patients underwent elective inguinal hernia repair either by the laparoscopic (n=8) or traditional open (n=8) techniques.

The median level of cranial spread as assessed by ultrasound directly after the caudal block was Th10 (range Th12–Th9). Fifteen minutes after the caudal injection, the most cranial level had increased to a median level of Th8 (range Th11–Th6). In 10 out of 16 patients, a cutaneous segmental level of analgesia could be assessed ~15 min after the caudal block. The most cranial sensory level determined by cutaneous stimulation was in median Th4 (range Th10–Th3). Thus, in six patients, no response to the cutaneous stimulus could be obtained (Fig. 2a).

The increase in ultrasound-assessed cranial level between 0 and 15 min was 2 segments (median value; range: 0–4.5). The corresponding value was 5.5 segments (median value; range: 2–7.5) when the level of cutaneous analgesia at 15 min was compared with the initial level determined by ultrasound immediately after the caudal injection. Despite the discrepancy as outlined above, a correlation was found between the ultrasound-assessed level at 15 min and the cutaneous-assessed level at 15 min (r-value 0.6) (Fig. 2b).

The median A–P diameter of the dural sac at the level of L3–L4 was 9.8 (range 7.2–11.6) mm before the block and was seen to be reduced to only 5.6 (range 3.0–6.8) mm immediately after the caudal injection. After 15 min, a partial re-expansion of the dural sac was observed in all patients (median 6.8 mm; range 5.8–8.5 mm) (Fig. 3a).

The skin to dorsal dura distance at the L3–4 level was 8.6 (median; range 6.1–12.5), 13.2 (9.1–17.2), and 11.6 (7.9–14.6) mm at baseline, immediately after injection, and 15 min post-injection, respectively.

During the first 3–6 min after injection, a re-distribution of the LA to the ventral compartment in front of the dura was observed (Fig. 3b). At the end of the observation period, a clear layer of fluid was seen in front of the ventral dura in all patients. The estimated median value of this fluid layer at 15 min was 1.1 mm.
Discussion

Despite being maybe the most frequently performed paediatric regional anaesthetic technique, the relationship between injected volumes vs cranial spread and also mechanisms for secondary cranial spread after caudal block remain unclear. Real-time ultrasound imaging now allows a unique opportunity to dynamically observe intraspinal distribution and cranial spread of caudally injected LAs. Owing to the lack of ossification of the vertebral column in small babies, ultrasound imaging is of very high quality in this age group.\(^\text{12}\) This discrepancy between an ‘immediate snapshot’ ultrasonographic picture and cutaneous testing 15–20 min after the initiation of the block is most likely explained by secondary cranial spread of LAs within the spinal canal. Various mechanisms for this to happen have previously been discussed, but until now, the dynamic sequence of redistribution and secondary cranial spread has not been studied and no defined mechanisms for secondary cranial spread have been verified.

Based on observations made in our previous studies and on pilot findings, we hypothesized that movements of CSF may be an important mechanism for secondary cranial spread. Thus, this study was conducted to observe the components of secondary spread after caudal block in infants, thereby verifying or refuting our hypothesis.

Before commenting on the study results, we would like to address the issue of cutaneous testing and caudal block. In early publications on the cranial level of caudal block, the methodology of cutaneous testing is poorly described and effectiveness has been discussed, but until now, the dynamic sequence of re-expansion of the dural sac when CSF is moving caudally due to reversal of the relative spinal-to-intracranial pressure gradient, thereby forcing the epidurally located LA to move to a more cranial level (c). The upper panel shows transcranial Doppler measurements (cerebral blood flow velocity), using a transtemporal window, in an adult: before (a), immediately on completion of injection (b), and after (c) a relatively rapid lumbar epidural injection (25 ml).\(^\text{15}\) Note the reduced peak velocity and the disappearance of diastolic flow immediately after the injection, providing indirect evidence of transiently increased intracranial pressure due to rostral movement of CSF caused by the epidural injection. The Doppler curves are reproduced by kind permission by A.M. Bombardieri.

All 16 blocks were judged to be clinically successful according to the criteria outlined in the Methods section.

Fig 5 Schematic drawing showing the spinal canal with the spinal cord (grey), dural sac with CSF (white), and the epidural space (yellow): before (a), immediately after (b), and ~15 min after (c) a high-volume (1.5 ml kg\(^{-1}\)) caudal block. The arrows indicate a rostral movement of CSF caused by the compression of the distal part of the dural sac by the injected LA (blue) (a). This is later followed by re-expansion of the distal part of the dural sac when CSF is moving caudally due to reversal of the relative spinal-to-intracranial pressure gradient, thereby forcing the epidurally located LA to move to a more cranial level (c). The upper panel shows transcranial Doppler measurements (cerebral blood flow velocity), using a transtemporal window, in an adult: before (a), immediately on completion of injection (b), and after (c) a relatively rapid lumbar epidural injection (25 ml).\(^\text{15}\) Note the reduced peak velocity and the disappearance of diastolic flow immediately after the injection, providing indirect evidence of transiently increased intracranial pressure due to rostral movement of CSF caused by the epidural injection. The Doppler curves are reproduced by kind permission by A.M. Bombardieri.
be estimated to $\approx 1.3$ ml, which corresponds to $\approx 16\%$ of the injected volume of LA (see Appendix).

Since the spinal canal can be regarded as a rigid tubular structure, the re-expansion of the terminal part of the dural sac must force epidural fluids in a rostral direction. If the approximated rebound volume of CSF ($1.3$ ml) is inserted into the Takasaki and colleagues equation\textsuperscript{7} for estimating the cranial level of caudal block, this would result in an increased cranial level of approximately four segments in our patient population (median patient weight 5.25 kg). This line of reasoning is supported both by the ultrasound-assessed increase in the cranial level of two segments and by the cranial level determined by skin pinch at 15 min (5.5 segments more cranial compared with the initial ultrasound assessment). The reason for the difference between ultrasound assessment at 15 min and the level determined by skin pinch at the same time point is most likely multifactorial. However, the limited ability of ultrasound to visualize very thin layers of LAs may at least partially explain this difference between ultrasound-assessed cranial level at 15 min and the level determined by cutaneous testing. In this context, it may be worth noting that over time and depending on assessed dimension (ultrasound or cutaneous testing), the volume per segment requirement regression lines gradually approach the volume per segment requirement according to Takasaki and colleagues (Fig. 4).

The CSF rebound mechanism for secondary longitudinal spread in the epidural space is similar, but the reverse, to what has been described in the setting of the combined spinal–epidural technique described in adults.\textsuperscript{13} This seminal paper describes how an epidural injection of saline increases the cranial level of the initial spinal block by a compression of the dural sac. Cranial shift of CSF after epidural injection in adults has also been verified by magnetic resonance imaging.\textsuperscript{14} Recent findings using transcranial Doppler have also been able to demonstrate that an epidural injection of LAs in adults will briefly but substantially raise the intracranial pressure to such an extent that the mean cerebral blood flow velocity was reduced by 63%.\textsuperscript{15} This observation does in our opinion provide further support for the presence and importance of the CSF shifts that are associated with epidural volume injections.

Thus, in the context of caudal block, the CSF rebound will, contrary to the situation when a combined spinal–epidural technique is used, instead compress the epidural pool of LAs, thereby forcing the epidurally located LA in a cranial direction (Fig. 5). Although the issue of secondary longitudinal spread may be multifactorial (e.g. potential secondary subarachnoid diffusion), we believe that the CSF rebound represents an important mechanism behind this phenomenon.

In conclusion, the present study observed two separate patterns of secondary spread of caudal block, horizontal intrasegmental redistribution, and longitudinal cranial spread. Furthermore, the initial injection of LA causes an almost complete compression of the terminal part of the dural sac, thereby forcing CSF cranially. As the epidural pressure returns towards normal, a rebound of CSF will partly re-expand the dural sac. This re-expansion of the dural sac will then in turn force the epidurally deposited LA in a cranial direction that will increase the maximum height of the caudal block. We believe that this CSF rebound mechanism causing secondary longitudinal spread does explain the sizable difference between the initial ultrasound-assessed cranial level and the level calculated according to earlier published equations.

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**Declaration of interest**

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**Appendix: Calculation for estimation of the CSF rebound volume**

The dural sac is approximated to have the form of a cylinder. The volume of a cylinder $V = \pi \times r^2 \times L$, where $r$ is the radius (or diameter/2) and $L$ the length of the cylinder. The cylinder length was estimated to be 108 mm, which is the median value for the distance from Th 10 to the termination of the dural sac in a 5.25 kg infant. The median value for the diameter of the dural sac at the L3–4 level immediately after the caudal–epidural injection was 5.6 mm and after 15 min, 6.8 mm. Dural sac volume immediately after epidural injection would then be $\approx 2.6$ ml (2659 mm$^3$) and after 15 min, 3.9 ml (3920 mm$^3$). The difference in volume at the time of epidural injection and after 15 min (1.3 ml) would be the re-expansion volume or the CSF rebound volume.

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