Detecting intravascular injection during caudal anaesthesia in children

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EDITOR:
Caudal anaesthesia may be used as an adjunct to general anaesthesia to provide postoperative analgesia in infants. Since inadvertent intravascular injection can result in life-threatening cardiovascular complications, criteria have been defined for detecting intravascular injection. Besides careful aspiration before injection, an epinephrine containing test dose is commonly used [1]. An increase in amplitude of the T-wave on the electrocardiogram (ECG) by $\geq 25\%$ or an increase in heart rate (HR) of $\geq 10$ beats min$^{-1}$ reliably detected intravascular injection in simulated intravenous (i.v.) test dose experiments [1–3]. We present three cases to illustrate that multiple factors can influence the sensitivity of an epinephrine test dose in infants (Fig. 1).

Patient A, a 7-week-old male weighing 5.3 kg, was scheduled for inguinal hernia repair. Induction of anaesthesia with sevoflurane, cannulation with a 24-G needle and placement of a laryngeal mask, he was placed in the lateral position. After introduction of a 25-G caudal needle (Epican Paed (Braun, Melsungen, Germany), 30 mm, short bevel) into the caudal space and negative aspiration, a test dose of 1 mL of bupivacaine 1.25 mg mL$^{-1}$ containing epinephrine 2.5 $\mu$g mL$^{-1}$ was administered. No changes in T-wave or HR were seen after 20 s. During subsequent administration of the next 1 mL of the same solution, a rise in T-wave amplitude occurred followed by a rise in HR from 149 to 156 beats min$^{-1}$. Blood could now be aspirated through the caudal needle. The ECG changes resolved within 1 min. The caudal needle was repositioned and, after negative aspiration and a further test dose, a total of 7 mL of bupivacaine 1.25 mg mL$^{-1}$ with epinephrine was given without further sequelae. The surgery and recovery were uneventful.

Patient B, a 7-week-old female weighing 5.4 kg, was scheduled for ingunal hernia repair. Induction of anaesthesia was performed as described above. After insertion of a caudal needle and negative aspiration, 1 mL of bupivacaine 1.25 mg mL$^{-1}$ with epinephrine 2.5 $\mu$g mL$^{-1}$ was injected as a test dose. After 25 s, there was no significant change in T-wave morphology or HR, and injection was continued until, after another 2 mL of the same solution, an increase in T-wave amplitude occurred without a rise in HR. Repeated aspiration was positive for blood. After repositioning the needle and negative aspiration and test dose, 7 mL of bupivacaine with epinephrine was administered without complications. Surgery was performed and recovery was uneventful.

Figure 1. Electrocardiogram (ECG) and pulse oximetry record of patient B. Baseline ECG followed by T-wave changes after injecting 3.75 mg bupivacaine with 7.5 $\mu$g of epinephrine (paperspeed 12.5 mm s$^{-1}$).

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Patient C, a 13-week-old male weighing 7 kg, received general anaesthesia as for patient A to undergo inguinal hernia repair. A caudal block was performed with a 25-G caudal needle. Aspiration was negative for blood. A test dose of 1 mL bupivacaine 1.25 mg mL\(^{-1}\) with 2.5 \(\mu g\) mL\(^{-1}\) of epinephrine was injected. No changes occurred in T-wave amplitude or HR. During injection of the next 3 mL of the solution, a rise in T-wave amplitude and an increase in HR of 126–140 beats min\(^{-1}\) were observed. Aspiration revealed blood in the needle hub. After redirection of the needle, aspiration was again positive for blood. The caudal technique was abandoned and sufentanil was administered i.v. No further problems occurred during surgery.

Paediatric regional anaesthesia has become increasingly popular over the years. Most paediatric anaesthesiologists prefer performing regional anaesthesia under general anaesthesia [4]. Although complications are rare, there is concern about safety. Inadvertent intravascular injection of local anaesthetic can result in life-threatening cardiovascular or neurological events. The incidence of accidental intravascular placement of an epidural needle or catheter in children may be as frequent as 5.6%; aspiration alone without previous injection fails to detect up to 86% of intravascular placements [2]. Therefore, although its validity has been questioned, a test dose containing epinephrine is usually employed. Several studies have been performed to optimize the sensitivity and specificity of this test dose. In children, a peak HR increase of \(\geq 10\) beats min\(^{-1}\) is considered a reliable indicator of an intravascular injection, whereas systolic blood pressure changes yield conflicting results. Increase in T-wave amplitude of \(\geq 25\%\) has been described to be as reliable as HR changes and to occur earlier after injection of the test dose [1,3].

The false negative tests in these patients are of concern and we can offer three possible explanations. Firstly, too little epinephrine could have reached the circulation. In order to detect an intravascular injection a threshold dose of epinephrine is required. Experiments in which the test dose was injected i.v. indicated that HR changes and T-wave changes are the most sensitive indicators if 0.5 \(\mu g\) kg\(^{-1}\) of epinephrine is administered [5]. Some authors even advocate a higher dose of 0.75 \(\mu g\) kg\(^{-1}\) [6]. It is therefore important to adjust the test dose to the body weight of the child. Although we used a dose of 0.47 and 0.46 \(\mu g\) kg\(^{-1}\), in patients A and B, respectively, it is possible that some of the dose was not injected directly i.v. Therefore, a reliable test dose may require a higher dose of epinephrine. In patient C, we used a dose of 0.36 \(\mu g\) kg\(^{-1}\) of epinephrine, which could be too low.

Secondly, we did not use atropine pretreatment. Atropine 10 \(\mu g\) kg\(^{-1}\) has been reported to improve the efficacy of the epinephrine-containing test dose by augmenting the HR response. These effects are probably due to the suppression of baroreflex function to hypertensive stimuli [7]. Furthermore, the T-wave change is different without atropine pretreatment [5]. Therefore, atropine is included in the protocol for children receiving regional anaesthesia with a combined local anaesthetic–epinephrine solution in many clinics.

The third and probably the most important reason for the failure of our test dose is that we did not wait long enough after injecting the solution. The indicators of systemic injection may be delayed for up to 60–90 s [1]. This suggests that one should wait for at least 90 s before administering the remaining local anaesthetic dose. Atropine pretreatment may lead to a faster haemodynamic response to i.v. epinephrine [3].

In all cases, initial aspiration was negative for blood. After injection of local anaesthetic solution and a positive T-wave response, repeated aspiration was positive in all three patients. This suggests that repeated aspiration after injection is more sensitive in predicting intravascular needle position. One could argue that routine repeated aspirations after a test dose during performance of a caudal block should be performed in all cases.

In conclusion, a false negative test dose can occur during a caudal block in infants. To obtain the highest sensitivity of detecting intravascular injection, we recommend atropine pretreatment, routine aspiration followed by administration of a test dose containing at least 0.5 mcg kg\(^{-1}\) of epinephrine, evaluation of HR and T-wave morphology for 90 s and administration of the local anaesthetic in small aliquots with repeated aspirations before each injection.

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Octaplas® is not equivalent to fresh frozen plasma in the treatment of acute angioedema

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EDITOR:

In the UK, when an anaesthetist prescribes the blood product ‘fresh frozen plasma’ (FFP), the current National Blood Service practice is to supply single-donor-unit plasma unless otherwise specified. However, since 2002 in the Republic of Ireland, the policy of the Irish Blood Transfusion Service (IBTS) has been to dispense the commercial product Octaplas® (Octapharma Ltd, Coventry, UK) in place of FFP whenever that is prescribed. Octaplas® is a solvent/detergent-treated pooled-donor plasma, which has been shown to be as effective as FFP in the replacement of clotting factors [1] and to lack the potential antigenicity found in single-donor FFP [2]. However, prior to its widespread introduction, concerns had already been expressed regarding the relative therapeutic equivalence of these two plasma products in certain clinical contexts [3]. IBTS routinely supplies Octaplas® rather than FFP when the latter is prescribed; because of this ‘at source’ substitution, most clinicians are unaware of the difference between the two products. We recently encountered one critically ill patient to whom both Octaplas® and FFP were administered and whose case highlights further limitations to Octaplas® use.

A 19-yr-old female was brought by ambulance to our Accident and Emergency Department with sudden onset of lip and tongue swelling, associated with progressive dyspnoea and stridor. Subcutaneous epinephrine had been administered without improvement. Ongoing stridor suggested imminent airway compromise and, following rapid sequence induction, the patient’s trachea was intubated. At direct laryngoscopy, glottic structures were noted to be swollen. She was subsequently transferred to the ICU for further management.

Information from the patient’s mother suggested a possible history of type III hereditary angioedema, a rare oestrogen-dependent X-linked dominant disease with a reported prevalence between 1 in 10 000 and 1 in 50 000 [4]. This diagnosis had been reached in another hospital near the patient’s home (150 km away), to which she had previously presented with recurrent episodes of generalized abdominal pain associated with acute lip and tongue swelling. She had never required intubation before. Although a diagnosis of hereditary angioedema was initially suspected, the patient’s serum C3, C4, C1-esterase inhibitor and CH100 levels were either supra-normal or within normal limits on testing, and more detailed enquiry into her family history revealed similarly affected male relatives. This informed a revised diagnosis of idiopathic relapsing angioedema, further supported by the predominance of airway structure involvement and resistance to standard treatments. Specifically, previous episodes had not responded to epinephrine, antihistamine or corticosteroids, but had resolved after intravenous infusion of FFP; accordingly, FFP was now prescribed. However, 2 U of Octaplas® were received from the Blood Bank, and were infused by ICU nursing staff. In contrast to previous episodes treated with FFP, Octaplas® produced no improvement in her condition.

Subsequent contact with the hospital where prior acute episodes had been managed provided the information that episodes do not respond to Octaplas® and require 2–4 units of single-donor FFP. This triggered concerns regarding the therapeutic equivalence of Octaplas® and FFP in this context.

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