Ultrasound and regional blocks

Editor—A review in the BJA in 2010 suggested that advantages of using ultrasound guidance in regional anaesthesia included direct visualization of structures and local spread, detection of anatomical variants, reduction in local anaesthetic volume, painless blocks, and improved block performance and patient satisfaction.1 We feel that the following case demonstrates an additional benefit of ultrasound guidance in establishing the aetiology of neurological deficit post-surgery.

A 76-yr-old man was admitted for plating of his left proximal humeral shaft after non-union of a fracture he had sustained 2 months earlier. He was in general good health. At the preoperative visit, a single-shot interscalene brachial plexus block was discussed; it was explained that this would be performed before induction of anaesthesia, and complications such as nerve damage, pneumothorax, and failure were explained. The surgeons also included the risk of radial and axillary nerve damage in their consent form.

In the anaesthetic room, the patient adopted the semi-supine position and routine monitoring applied. An ultrasound-guided interscalene brachial plexus block was performed using a high-frequency linear probe and portable ultrasound machine (M-turbo, HFL 39 13-6 MHz, SonoSite Ltd, Bothell, WA, USA) and a 22 g/50 mm regional anaesthesia needle (UniPlex NanoLine, Pajunk, Germany). The trunks/divisions of the brachial plexus were located in the supraclavicular fossa and traced proximally in the neck, so that the roots of C5 and C6 were seen just below the level of the cricoid cartilage. The block needle was inserted, advanced, and (re)-directed to lie adjacent to each nerve in turn with the needle tip kept fully visible throughout the procedure. A total of 25 ml of 0.5% bupivacaine was injected and seen to spread circumferentially around each nerve. The injection was painless and performed without undue resistance. The block was performed by a second-year anaesthetic SHO under direct supervision of a consultant anaesthetist with a special interest in regional anaesthesia. General anaesthesia was then induced with i.v. fentanyl and propofol and maintained with sevoflurane with spontaneous ventilation via a laryngeal mask airway. The anaesthetic was subsequently uneventful other than some mild hypotension which was treated with i.v. fluids and intermittent boluses of metaraminol.

During surgery, the proximal humerus fracture was identified by the surgeons and an 8-hole Philos plate (Synthes, UK) was applied to the proximal humerus. The plate position was confirmed by image intensifier, and although ‘slightly off’ posteriorly and distally, it was deemed to be in a good proximal position. The operation note stated that ‘care was taken to achieve sub-periosteal dissection to avoid radial nerve injury’.

Immediate recovery was uneventful and the patient was very comfortable requiring no additional opiates. The next day the on-call anaesthetic registrar was asked to review the patient by the ward staff because of residual left hand weakness, despite a resolved proximal block. Sensory deficit on the dorsum of the hand was noted and a 2/5 motor deficit to the wrist and finger extension. The following day (second day post-surgery), the patient was seen by both the surgical team and the anaesthetic SHO who had performed the block. On both occasions, the same sensory and motor deficits were elicited, but the patient felt that there had been improvement in function.

On day 5, he was reviewed by the operative orthopaedic consultant. The patient demonstrated wrist and finger drop, and continuing numbness and paraesthesia in the radial nerve territory. He thought that direct surgical nerve injury was unlikely and the continued neurological symptoms were due to neuropraxia secondary to the block. This was disputed by the anaesthesia team who felt that the distribution of symptoms did not fit with a C5/6 injury and intraneural injection had not been observed. After additional physiotherapy, the patient was sent home with a splint.

One week after discharge, the patient was reviewed in the orthopaedic outpatient clinic. Although his sensation was improving, there was a continued motor deficit suggestive of radial nerve injury. An ultrasound scan of the humerus demonstrated impingement of the radial nerve by the Philos plate. Urgent re-fixation was performed the following day under general anaesthesia only. The distal tip of the plate was found to have tented the radial nerve, pulling it between the intermuscular septum. The nerve was still in continuity, it was released from the plate, removed from the intermuscular septum, and the plate re-fixed anteriorly.

After the operation, there was no immediate improvement in neurological function. The patient was reviewed at 4 months and demonstrated only limited improvement in wrist extension. He was referred for tendon transfer but by the time of his next appointment, 7 months after the original procedure, the neurological deficit had nearly resolved with only minimal weakness of digital extension remaining.

Nerve damage after regional anaesthesia has been attributed to direct mechanical trauma by the needle or nerve compression from haematoma. However, direct injections into the epineurium have been reported with no adverse effects and conversely nerve function can be disrupted without apparent trauma or ischaemic compression.2 The timing of onset of nerve injury is variable with some presenting weeks after a single-shot technique.2 Because nerve injury after regional anaesthesia is a rare adverse event, it would be difficult to demonstrate that the use of ultrasound-guided blocks is safer than those performed with nerve stimulation, so only indirect evidence exists for its better safety in this regard. Unintentional neuronal injection is probably reduced by using ultrasound,3 although no study has directly compared the techniques, and a recent publication in the BJA demonstrated that ultrasound guidance reduced the number of needle passes and decreased the amount of local anaesthetic required for a successful block.3

We feel that this case demonstrates another advantage of using ultrasound guidance. By visualizing the structures, needle placement, and local anaesthetic spread, we were able not only to say that direct nerve trauma or pressure necrosis were unlikely but also that the pattern of injury was...
not consistent with C5 and 6 root damage alone. In these cases, a surgical aetiology should be sought.

Declaration of interest
None declared.

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Delayed subdural haematoma complicated by abducens nerve palsy and cortical vein thrombosis after obstetric epidural anaesthesia

Editor—A healthy 28-yr-old primigravida received epidural analgesia for labour pain (EpiLong Tuohy 18 G×90 mm, Pajunk GmbH, Geisingen, Germany). An inadvertent dural puncture occurred at L3–4. The needle was pulled back and a peridural catheter inserted, which provided good analgesia for an un-eventful delivery. A few hours later, the patient complained of posture-sensitive headache. Post-dural puncture headache (PDPH) was diagnosed. Theophylline (1×200 mg day⁻¹), paracetamol (4×1 g day⁻¹), and fluids were prescribed. On day 7, diplopia developed. Magnetic resonance imaging (MRI) showed bilateral subdural haematoma and diffuse meningeal swelling consistent with intracerebral hypotension (Fig. 1). On day 11, an autologous sterile blood patch (20 ml) was performed on the level of the initial epidural puncture site. The headache improved markedly and both headache and diplopia had improved by day 22. An MRI on day 17 showed that the haematoma had decreased, but an isolated thrombosis of a cortical vein (30 mm length) was detected. Treatment with low-molecular-weight heparin was started.

Bleeding history and Prothrombin A variant, factor V Leiden, MTHFR C677T Mutation, Protein C+S, FVII–XII, and VW-factor were all normal. The patient was discharged home on day 19. Three months later diplopia had receded completely and on a follow-up MRI, 10 months later, neither haematoma nor thrombosis was detectable.

After inadvertent dural puncture, the incidence of PDPH is up to 70%.¹ The leakage of cerebrospinal fluid (CSF) causes PDPH. After CSF hypotension, traction on intracranial pain-sensitive structures may result.² Symptoms often resolve within a few days spontaneously or when treated with analgesics and bed rest.³ More than 85% of PDPH will resolve within 6 weeks.²

Our patient presented with PDPH, which was unresponsive to conservative therapy. A blood patch was only considered when clinical findings and MRI suggested intracerebral hypotension resulting in subdural haematoma and abducens nerve palsy with diplopia. The loss of CSF may shift the brain caudally and cause traction and tearing of subdural veins, resulting in a subdural haematoma.⁴ Nerve palsy as a result of subdural haematoma has been reported and the abducens nerve is often affected. This can be explained by its long route from the pons through the petrous bone and dura, making it vulnerable when the brain is displaced caudally.⁵ Conservative treatment is

Fig 1 Bilateral subdural haematoma paramedian of the sagittal sinus and in the infratentorial region, bilateral meningeal swelling; MRI was done 8 days after inadvertent dural puncture.