

Pain during injection of rocuronium bromide

We have noted marked local pain following intravenous injection of rocuronium bromide which has not, to our knowledge, been previously reported.

To attempt to achieve optimal conditions for tracheal intubation, we have given half of a standard induction dose of propofol, with lignocaine 20 mg, followed by rocuronium bromide $0.6 \text{ g} \cdot \text{kg}^{-1}$ followed immediately by the remaining propofol. In all of our patients there was marked discomfort when rocuronium was injected. This did not seem to be associated with any obvious local reaction and was entirely separate in timing and quality from discomfort caused by propofol.

Rocuronium bromide is a new nondepolarising muscle relaxant formulated with sodium acetate, sodium chloride and acetic acid to produce a solution of pH 4 [1]. The low pH is a possible cause of pain. Slight increases in plasma histamine levels have been observed following rocuronium administration, but no clinical signs of histamine release have been observed [1].

Pain has also been reported with vecuronium bromide [2]. Since most injections of muscle relaxant will be given to anaesthetised patients, this reaction is unlikely to be observed often, but will be seen with this anaesthetic technique and if the priming principle [3] or pretreatment before suxamethonium is performed.

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Continuous spinal anaesthesia and cauda equina syndrome

I wish to take issue with the recent 'Dear Doctor' letter from ASTRA (UK) (Dec 1994) disclaiming the use of their local anaesthetic solutions for continuous spinal anaesthesia (CSA) after several case reports of cauda equina syndrome following this technique [1]. I believe that this is a safe and extremely useful technique in the elderly and very sick patients, in trained hands. It is not the method that is the problem, but its incorrect use. I have been interested in, and continue to practise, this important technique since I worked with Alon Winnie in Chicago in 1986. There, continuous spinal anaesthesia (using 20 gauge catheters) has been used routinely for many years without complications.

Since 1987, there has been renewed use and interest in the technique, particularly with the development of microspinal catheters in 1990 [2]. However, these soon proved to be difficult to use and failed or inadequate blocks often occurred. In 1991, the first reports of cauda equina syndrome in patients following CSA were published [1]. The features common to these four patients were that initially they had inadequate blocks, and larger than normal doses of 5% heavy lignocaine or 1% amethocaine were used to obtain adequate anaesthesia. Following this, the FDA, in 1992, banned the use of microspinal catheters.

Studies using spinal column models have demonstrated that restricted spread of local anaesthetic will occur if a spinal catheter is placed caudally, if the drug is injected slowly and a hyperbaric solution is used. Rapid injection of local anaesthetic and cranially directed catheters give a uniform distribution and avoid this problem. Fast injection through microspinal catheters is difficult to achieve [3, 4]. A recent study of the position of spinal catheters has shown that they are more likely to be placed caudally if inserted with the patient placed in the lateral as opposed to the sitting position and if more than 3 cm of catheter are inserted into the subarachnoid space [5]. Finally, there is evidence from an animal model that solutions of 5% lignocaine and 0.5% amethocaine may be neurotoxic in certain circumstances [6].

I believe that there is now sufficient evidence to explain why cauda equina syndrome has occurred following continuous spinal analgesia and how to avoid it. The three most important factors appear to be: a restricted or failed block, caused by a caudally placed catheter and use of hyperbaric solutions; excess local anaesthetic; use of 5% lignocaine or 0.5% amethocaine, neither drug being available in the UK. I believe CSA is a safe and extremely useful technique provided the following recommendations

are heeded: no more than 3 cm of catheter are left in the subarachnoid space; hyperbaric solutions of either 5% lignocaine, or amethocaine 0.5 or 1.0% are avoided; plain bupivacaine (0.25–0.5%) is used; if the block is inadequate or fails do not keep giving further local anaesthetic. Not more than 15 mg of bupivacaine should be given to establish an adequate surgical block initially, (usually 5–10 mg is required, particularly for operations below the umbilicus). If smaller doses have been used, top-ups of up to $5 \text{ mg} \cdot \text{h}^{-1}$ bupivacaine can be given. If the block is still inadequate after 15 mg of bupivacaine have been given the technique should be abandoned; the sitting position is better for correct catheter placement.

In this hospital, we have used the technique, with 20 gauge catheters inserted through 18 gauge Tuohy needles made by Braun Medicals, successfully and without complications both for anaesthesia and postoperative pain relief [7]. For a number of years and on several occasions it has prevented patients from needing postoperative ventilation. It would be unfortunate if this extremely useful anaesthetic was abandoned due to its inappropriate application.

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