

Minimum effective local anaesthetic dose for spinal anaesthesia

Editor—As a comparison of the dose of local anaesthetic required for spinal anaesthesia with either levobupivacaine or ropivacaine, the study by Sell and colleagues is both interesting and informative. However, I have a number of concerns about the paper that has been based on that study.

In the title, and subsequently, the authors describe plain solutions of both drugs as being ‘isobaric’ yet the density figures they quote in the paper are well below the normal range for CSF. These solutions are not isobaric with cerebral spinal fluid and they spread in a manner that is different from those that are truly isobaric, the primary difference being much wider variability in the total spread produced.

In the introduction, the authors state that the minimum local anaesthetic concentration (MLAC) concept has been established for spinal and epidural anaesthesia, but I know of no work validating this methodology for spinal anaesthesia. All the published papers relate to epidural injection and the validity of the approach in that block is somewhat controversial. It is claimed that the MLAC concept is equivalent to that of MAC for general anaesthetics, but even the validity of that concept has been seriously questioned.

Furthermore, one of the references used in support of a statement that the methodology provides ‘reliable estimates of MLAD using the up-and-down method’ is by authors who have concerns that the method is not reliable.

Sensory and motor block were assessed at 20 min only, but it is well recognized that it may take 30 min or longer for the effect of an intrathecal injection to become maximum.

In regard to the controversy over using MLAC figures to make potency comparisons between local anaesthetic drugs, I think that the results of this study are quite interesting. The difference in MLAD (ED50) was small and non-significant, although ropivacaine is commonly thought of as less potent than the other long-acting local anaesthetic drugs, and the total dose ED100 of both drugs required to complete surgery was close to 15 mg, the dose that most practitioners would consider appropriate for a spinal anaesthetic. In spite of this, the authors claim that their study did not allow them to evaluate the top end of the dose–response curve, but surely that is exactly what they did with their top-up injections to allow surgery?

The authors also claim that dose is important in determining the extent of spinal anaesthesia, but there is a wealth of literature indicating that this is not the case. The primary determinant of the spread of an intrathecal injection is the baricity of the solution. Another factor, relevant in this study, is that the use of a catheter may well have produced a different directional pattern of local anaesthetic spread compared with injection through a needle.

The final conclusion of the paper is irrelevant because ‘patient comfort’ was not studied. I am sorry to write in such a critical fashion about an interesting study.

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Editor—Thank you for giving us the opportunity to respond to Wildsmith’s criticisms of our paper.

We were unfortunately not able to measure the individual CSF densities, and thus we had to rely on previous documentation, such as Lui and colleagues and Hocking and Wildsmith. The latter review states that ‘...given the normal variation [of CSF], it is necessary that solutions that are to be predictably hypobaric or hyperbaric in all patients have baricities below 0.9990 or above 1.0101, respectively’. The reported densities of the local anaesthetics used by us lie within these limits and thus, we argue that the solutions used by us are essentially isobaric.

We present MLAC as a previously reported example of a similar approach to assess the effects of local anaesthetics in a set-up similar to that we used in our study (i.e. the up–down sequence). We do not argue that the MLAC concept would be equivalent to the MAC concept, which it certainly is not in our opinion. To those readers who want to critically review our methodology we have provided references for the up–down sequence, both the original Dixon paper as well as a recent report using more complex mathematical approaches in assessing the reliability of the method.

It is true that our study period probably did not allow enough time for the maximal effect of the initial dose to be achieved. However, we feel that readers will understand from our statement in the paper that we only looked at the first 20 min after drug administration and that this is a limitation. In addition, in order to be able to execute this study in a busy unit, we had certain time frames to meet—again a fact that is clearly stated in the report. However, we feel that few anaesthetists would wait for more than 20 min before administering a top-up dose via a spinal catheter, should that be required.

The protocol did not dictate how the study drugs were to be administered after the initial dose and the first 20 min had passed. The supplemental doses were administered based on the clinical assessment by the attending anaesthetist. Thus, we feel that the supplemental doses were given in too random a situation to be used in the assessment of the dose–response curve. In addition, we wanted to particularly assess the MLAD region, as that dose can guide us initially in using as small doses as possible to patients susceptible to such adverse effects as hypotension and in whom supplemental doses can easily be given utilizing the spinal catheter.

In contrast to Wildsmith, we feel that determining the ED100 would not be very interesting. We assume that the local anaesthetic dose requirements more or less follow a normal distribution. Thus, determining ED100 and letting that guide us in administering local anaesthetics to patients, in whom adverse effects are to be avoided, could lead to over-shooting in dosing. This can easily be demonstrated if normal distribution and a dose–response curve are drawn on a single figure.

Baricity is a major determinant of intraspinal local anaesthetic spread. We should probably have stated this to provide a more comprehensive list of all factors affecting local anesthetic spread, although our study deals with essentially isobaric solutions. The fact that the catheter itself, particularly its tip position, may affect local anaesthetic distribution is stated in the paper.

We did not study ‘patient comfort’ as correctly pointed out by Wildsmith. Thus our conclusion is based only on the subjective

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865
Surgery to 6.5 g dl\(^{-1}\) of C0


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External iliac vein injury during total hip arthroplasty resulting in delayed shock

Editor—Vascular injuries related to total hip arthroplasty (THA) are very rare, but can cause limb loss or become life threatening.\(^1\)\(^2\) The occurrence of this has not been emphasized enough in anaesthesia literature. We present a case of external iliac vein injury during THA, which resulted in shock despite minimal bleeding during surgery. A 157 cm, 51 kg, 68-yr-old female with a 20 yr history of osteoarthritis underwent total hip arthroplasty under general anaesthesia. Her blood pressure suddenly dropped from 100–120/60–70 mm Hg to 60 mm Hg of systolic pressure during closure of the incision. The artificial tachycardia, ST depression of 0.25 mV on ECG were then followed by hypotension. Haemoglobin (Hb) concentration fell from 9.6 g dl\(^{-1}\) before surgery to 6.5 g dl\(^{-1}\) at THA completion in 30 min. Iatrogenic bleeding was doubtful as there had been little bleeding during the operation. Haemodynamic instability required fluid resuscitation including blood transfusion and vasopressor support. Vascular injury from drilling was suspected and the operation incision was closed immediately. Hb concentration fell to 3.7 g dl\(^{-1}\) 40 min after initial hypotension. A large retroperitoneal haematoma was found on abdominal echography. A laparotomy was undertaken 50 min after initial hypotension and revealed a 2.0 cm long laceration of the left external iliac vein. This laceration was repaired by vascular surgeons, and subsequent Hb concentration was 8.5 g dl\(^{-1}\). She was discharged 21 days after surgery without any further complications.

The external iliac vein appears to be more vulnerable than the artery because of its more medial position and the paucity of interposed tissue along the pelvic brim, which protects the artery.\(^1\)\(^2\) However, injury to the external iliac artery has been reported more frequently than the vein.\(^1\)\(^2\) This fact may indicate that venous injuries have been failed to be properly noticed. Hwang\(^3\) reported a case of a patient which resulted in shock 26 h after THA and the patient was found to have a 0.5 cm long laceration of the external iliac vein at laparotomy. This supports the conclusion that venous injury can lead to slow onset of shock, regardless of whether the laceration is large or small. Marked bleeding from the drill hole and profound hypotension are characteristic of intrapelvic arterial damage.\(^1\)\(^2\) However, venous damage that is of lower pressure may not present with the patient in a lateral position. As such, venous injury can be concealed and anaesthetists should pay close attention to the patient’s haemodynamics.

Vascular injuries occurring in major vessels require urgent surgical intervention. Early diagnosis and immediate control of any haemorrhage should prevent morbidity and mortality. This may involve interrupting THA, covering the incision with drapes, and returning the patient to a supine position.

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