McAlpine concerning histologic changes in transient paralysis. Instead of paraphrasing, let us this time quote the original source. "The effects of stretching a peripheral nerve beyond the limit of physiologic elasticity . . . [produce] damage to epineurial vessels, with resultant patches of ischemic changes in nerve fibers." Granted these observations were made on somatic fibers, but we theorize a milder form of this same stretch injury may explain the transient postoperative sympathetic dysfunction observed in our patient.

According to Dr. Vandam, our "gross diagram of a torso" did not illustrate the anterior relation of the cervical sympathetic trunk to cervical transverse processes. However, we felt it adequate to illustrate the concept of misalignment of the thoracic and cervical vertebrae (which was its primary purpose). Figure 1 illustrates the cervical sympathetic chain in greater detail, further demonstrating how it may be stretched from inadequate head support in a patient placed in the lateral position.

In answer to raised questions, intraoperative blood pressures were monitored via a right radial artery catheter and a left arm blood pressure cuff. Both pressures were approximately equal and unchanged in the lateral position. Blood pressure remained within 20 per cent of control throughout the entire anesthetic. When Horner's syndrome was observed postoperatively, there were no signs or symptoms to suggest a brachial plexus insult. No carotid bruits were auscultated, although occult disease may have existed. Chest x-ray did not demonstrate evidence of Pancoast tumor or cervical rib but did reveal lumbar and thoracic spine arthritis. It was on this basis that we assumed cervical vertebral arthritis and/or spurring may have existed.

Although stretch of the cervical sympathetic chain is unusual, in this case, we are still unable to conjure any other diagnosis to better explain the findings. The need for strict attention to spinal alignment in patients placed in the lateral position remains essential.

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Further Considerations Regarding the Components of an Effective Test Dose Prior to Epidural Block

To the Editor:—In the recent clinical report by Moore and Batra, it was stated that the purpose of the study was to determine the components of a "single" test dose of a local anesthetic solution which, within two minutes from its injection, would produce clinical evidence that the needle has penetrated either a blood vessel or the dura. In the report, it was shown that the addition of epinephrine 1:200,000 to 3 ml of local anesthetic produces a clinically detectable transient tachycardia if injected into a vein. However, the effect of a subarachnoid injection of 3 ml of local anesthetic was not studied. Three ml of 3 per cent chloroprocaine (90 mg) should produce an immediate block, as also should 3 ml of 0.75 per cent bupivacaine (22.5 mg) but the level of anesthesia might reach the upper thoracic dermatomes. However, neither 3 ml of 1.5 per cent mepivacaine nor

REFERENCES

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3 ml of 1.5 per cent lidocaine (45 mg) might produce definite signs of spinal anesthesia within two minutes. Mepivacaine in 1.5 per cent concentration is apparently ineffective as a spinal anesthetic whereas higher concentrations are effective. Similarly, lidocaine normally is used in a concentration of at least 5 per cent for intrathecal use and therefore, 1.5 per cent might not produce significant spinal anesthesia within two minutes.

Thus, it would seem that 3 ml of 0.75 per cent bupivacaine or 3 per cent chloroprocaine both with epinephrine 1:200,000 should provide clinical evidence of either an intravascular or intrathecal injection within two minutes. But 3 ml of 1.5 per cent lidocaine or mepivacaine both with epinephrine 1:200,000 could provide evidence of an intravascular but possibly not an intrathecal injection.

Perhaps the intrathecal injection of small volumes of these local anesthetic solutions should be specifically studied.

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In reply—Morison’s two primary concerns are: 1) doses of 90 mg of chloroprocaine and 22.5 mg of bupivacaine “might” result in high or total subarachnoid block; and 2) 45 mg of either lidocaine or mepivacaine “might” not produce significant spinal anesthesia in two minutes.

Whether high or total spinal block results from the unintentional injection of a local anesthetic drug depends on many factors other than dosage, e.g., baricity of the solution, position of the patient, intra-abdominal pressure (obesity, pregnancy, etc), height, and so forth. Regardless of these, 90 mg of chloroprocaine injected into the subarachnoid space is unlikely to result in high or total spinal block. From all indications, a 3-ml dose of 0.75 per cent hyperbaric bupivacaine (22.5 mg in 8.25 per cent or 8.0 per cent dextrose, specific gravity approximately 1.035) would do so. However, 0.75 per cent bupivacaine used for epidural block is isobaric (specific gravity, 1.007) and up to 4 ml of a 0.5 per cent isobaric solution (20 mg, specific gravity 1.006) has been used safely for spinal anesthesia. Morison and co-workers have injected 3 ml of a 0.75 per cent isobaric solution of bupivacaine unintentionally into the subarachnoid space while attempting intermittent epidural block in two parturients with no untoward sequelae, although analgesia to T1 and T4 ensued. To date, we have not injected 3 ml of 0.75 per cent isobaric bupivacaine subarachnoidally, so that comments on its possible effects would only be conjecture. In summary, even if a test dose containing 22.5 mg of bupivacaine did result in high or total spinal anesthesia, it would be: 1) an expected possible result of a test dose; 2) in all probability immediately recognized; and 3) treated correctly, as in the previously noted cases. Conversely, if a high or total spinal block occurs when not anticipated from a large dose—10 ml (75 mg) or more used to establish the required analgesia for an operative procedure—the outcome may be catastrophic. Furthermore, treating a high or total spinal block resulting from 22.5 mg, as compared to 75 mg or more, would seem to be a more desirable situation.

Secondly, Morison questions whether 3 ml of 1.5 per cent lidocaine or mepivacaine will produce significant spinal anesthesia in two minutes. A dose of 45 mg of either drug, whether it is in 3 ml (1.5 per cent solution) or in 0.9 ml (5 per cent solution) will consistently produce clinical evidence of spinal analgesia within two minutes (excluding technical errors). However, it may or may not produce analgesia adequate for a surgical procedure (e.g., an intra-abdominal operation). Even reference 6 cited by Morison states that onset of anesthesia with lidocaine is almost immediate. Furthermore, a 2-ml ampule of 1.5 per cent lidocaine (30 mg) in 7.5 per cent dextrose is available for spinal anesthesia for vaginal deliveries, and it is effective. Although mepivacaine is no longer available for spinal anesthesia, our experiences as well as a careful review of mepivacaine for spinal block support the effectiveness of 45 mg in producing clinical evidence of spinal anesthesia within two minutes.

Finally, what really is needed is a single-dose ampule

D. H. MORISON, M.B., CH.B., F.R.C.P.(C)
Associate Professor of Anaesthesia
McMaster University
Hamilton, Ontario L8N 3Z5

REFERENCES

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