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REPLY

Dr. Kumar reports a valid method of repositioning an epidural catheter that has entered an epidural vessel. We apply this technique in our practice. We agree with Dr. Kumar on the need to prevent blood clot formation. However, in our case report, a clot formed in the catheter while we were preparing the flush synringe with sterile normal saline. In the event that a clot forms, the catheter may still be salvaged by dislodging the blood clot with reinsertion of the stylet wire.

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Epidural morphine injections for the treatment of postspinal headache

To the Editor:

Postspinal headache (PSH) was first documented by August Bier.[1] In 1898, he reported severe headache following subarchnoid injection of cocaine which was exacerbated by sitting or standing, and relieved by lying down. The incidence of PSH varies between 0.4 per cent[2] and 70 per cent.[3] The most accepted theory of PSH relates it to CSF hypovolaemia, produced by escape of CSF through the dural vent, which deprives the brain of its cushion and places tension on its pain-innervated anchoring structures.[4]

Tourtellotte *et al.*[5] listed 49 modalities of treatment for PSH, ranging, alphabetically, from an abdominal binder to irradiation of the skull. Even surgical repair of the dural hole was advocated in persistent cases.[6]

Currently, the most accepted mode of therapy for PSH was introduced by Gormley;[7] autologous blood is introduced into the epidural space in the vicinity of the presumed dural hole, resulting in effective and total relief of headache. However, failure of this mode of therapy was reported recently.[8,9]

We report six patients (28–70 yr old) with PSH in whom total relief of pain was obtained by epidural injections of morphine via an epidural catheter. Five of the patients developed PSH after surgery using combined spinal-epidural anaesthesia. The spinal injections were done through a 25-gauge needle. An epidural catheter was left in place for supplemental anaesthesia or postoperative analgesia. Epidural morphine 3.5–4.5 mg was injected postoperatively. On the first postoperative day the patients developed PSH. Another injection of epidural morphine, 3.5–4.5 mg, relieved the pain after 30 min, and the pain did not return. The sixth patient, 28 yr old, underwent an inadvertant dural puncture with a 19G needle used for the introduction of the 25G spinal needle. Thirty-six hours later she developed PSH. An epidural catheter was introduced into the L_{2-3} interspace, and morphine 3.5 mg was injected through it. After 45 min her headache was relieved. Twenty hours later she complained again of PSH. Another injection of morphine 3.5 mg through the epidural catheter resulted in disappearance of the headache after 45 min, without its return.

There have been several reports of the successful use of epidural morphine after inadverdent spinal tap to prevent post-spinal headache,[10,11] but this, to our knowledge, is the first report of the beneficial use of this modality after headaches have occurred. The availability of this technique may detract from the reservations of using spinal anaesthesia in young adults. Our experience also challenges the CSF leakage theory of PSH.

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Clinical, electrical and mechanical correlations during recovery from neuromuscular blockade with vecuronium

To the Editor:

The recent paper by Dupuis *et al.*[1] on the correlation between evoked EMG and MMG train-of-four (TOF) fade and respiratory mechanics following vecuronium represents a valuable addition and extension to the original work of Ali *et al.*[2] However, I am not sure that they have proved their conclusion that acceptable levels of TOF recovery differ depending on the monitoring technique. According to their data an EMG TOF ratio of 0.90 is equivalent to an MMG value of 0.72. (Incidentally their regression equation in Figure 3 is wrong. It should ready = 1.033x *minus* 20.605.) This is almost identical to the data we reported earlier[3] where the comparable MMG value would have been 0.73.

However, in both studies EMG data was obtained from the digiti minimi muscle while MMG data represented the adductor pollicis muscle. Dupuis *et al.* state that the issue of whether data obtained from thenar or hypothenar muscles are similar is beyond the scope of and presumably not relevant to their study. I must respectfully disagree. There is evidence that the digiti minimi are less sensitive to nondepolarizing neuromuscular blockers than the adductor pollicis muscle.[3] If the level of single twitch depression is less in the hypothenar muscle group than in the adductor pollicis is it not reasonable to suppose that TOF fade will also be less affected in the latter muscle? More recent work[4] suggests that when EMG and MMG data is recorded simultaneously from the adductor pollicis that the information obtained is interchangeable. Unless EMG and MMG recordings of TOF fade are both taken from the same muscle speculation concerning "acceptable" EMG TOF fade ratios should be taken with some skepticism. Hence, the authors' conclusions relative to the correlation of the EMG fade ratio and respiratory mechanics are valid only for situations where the test muscle was the digiti minimi.

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REPLY

We appreciate the interest of Dr. Kopman in our study. As mentioned by him and as described in our paper the electromyographic (EMG) response of the adductor digiti minimi muscle was monitored in the study. Thus, the conclusions of the study apply to the monitoring of that muscle. We can speculate on the differences between EMG monitoring of the hypothenar and thenar muscles but this remains beyond the scope of our investigation.

The relation between the EMG T_4/T_1 and the MMG T_4/T_1 is: $y = 1.033\ x - 20.605$ as is correctly *expressed in the results section and we apologize for the + in Figure 3.*

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