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Delayed Subarachnoid Migration of an Epidural Arrow FlexTip Plus Catheter

To the Editor:—The published description of delayed subarachnoid migration by Jaeger and Madsen is beyond belief.[1] According to their report, the patient was found severely hypopneic approximately 28–32.5 h after commencement of an epidural infusion of 0.125% bupivacaine with 6 mg of hydromorphone per milliliter at 14 ml/h. During that infusion period, the patient would have received a cumulative epidural dose of 392–455 ml fluid, 490–599 mg bupivacaine, and 2,352–2,730 mg of hydromorphone—a dose of hydromorphone large enough to fell a full-grown white rhinoceros.

Assuming an overlooked printing error of "milligrams" instead of micrograms (a fact kindly corroborated by the senior author), the amended cumulative dose of hydromorphone would lie somewhere between 2.35–2.73 mg of epidural hydromorphone. In addition, a hole had been accidentally driven through the dura by a Tuohy needle of unstated caliber, leaving free access to the subarachnoid space. Regardless of the dural puncture, epidural hydromorphone undergoes rostral spread with repeated doses,[2] and sudden respiratory failure has been reported 4.5 h after a single bolus epidural injection of 1 mg hydromorphone.[3]

Therefore, in the presence of a waterlogged, opioid-rich epidural space and an open highway into the subarachnoid space *via* the accidental dural puncture hole, the subsequent respiratory collapse becomes highly predictable. There seems no logical reason to invoke some remote *deus ex machina* to explain the outcome, least of all a highly improbable suspect such as the remarkably soft and pliable Arrow FlexTip Plus epidural catheter.

Philip R. Bromage, M.B.B.S., F.F.A.R.C.S., F.R.C.P.(C)
Montgomery Center, Vermont

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Presumed Delayed Catheter Migration

To the Editor:—I read with interest and consternation Drs. Jaeger and Madsen's[1] correspondence on what they presumed to be delayed subarachnoid migration of an epidural Arrow FlexTip Plus catheter. There are several areas in this case report that concern me.

First and foremost is in regard to patient monitoring and appropriate documentation. It is worrisome that the only references to the patient's level of consciousness or motor function on the evening preceding the event are the following " . . . Sometime that evening the patient's husband recalled that she was more somnolent and seemed weaker . . ." and ". . . At 2:00 AM the next day, the nurse found the patient barely responsive . . .". Incomplete documentation at the time of the critical incident is also apparent, when no arterial blood gas is drawn, when fluid is aspirated from the epidural catheter but is not tested to determine its nature, and when the catheter is removed before any confirmation of its location. Thankfully, the patient was successfully treated.

Vigilance in the operating room should not be left at the door as we tread into the unfamiliar territory of intensive care units, "stepdowns," and hospital wards. It is therefore mandatory that any group of clinicians that sets itself up as an Acute Pain Service (APS) ensures that patients in its care are appropriately monitored and that evidence of monitoring be documented in the nursing record. Standing orders should be clear as to when to alert the APS, particularly with respect to level of consciousness and motor function, and the APS must be available to respond 24 h per day.

Second, the epidural infusion of bupivacaine 0.125% with hydromorphone 6 mg/ml, I trust, is a printing error. At 14 ml/h, this would result in 84 mg hydromorphone delivered! For our routine thoracic cases we use T4–T8 hydromorphone 0.05 mg/ml without local anesthetic, at rates of 2–5 ml/h (0.1–0.25 mg/h) to achieve dynamic pain control. When we do use thoracic local anesthetic/opioid admixtures, we use bupivacaine 0.1% with 0.005 mg/ml fentanyl at rates

of 6–12 ml/h. We have considered bupivacaine/hydromorphone admixtures, but there is no current literature to guide us in this regard. Based on other admixtures however, bupivacaine 0.1–0.125% with hydromorphone 0.010 mg/ml would seem to be appropriate.

Finally, with respect to their conclusion that the catheter migrated into the subarachnoid space, it is at least as plausible that the catheter remained in the epidural space but that the infusate found its way intrathecally *via* at least one rent in the dura. There was obvious technical difficulty in attempting to secure epidural catheter placement, before surgery, at T7–T8; successful placement did *not* occur, they state, despite "multiple" attempts. The fact that "doses of local anesthetic" failed to confirm placement of a catheter or that blood, cerebrospinal fluid, or paresthesia were not noted during these attempts does not rule out dural puncture at this level. Second, there was documentation that a dural puncture did occur at T11–T12 during attempts to secure epidural catheter placement after surgery. Eventually, a catheter appears to have been successfully passed at T10–T11. The fact that the Tuohy needle was "oriented cephalad," however, does not rule out catheter tip placement adjacent to the documented T11–T12 dural puncture and may have resulted in catheter placement close to an unrecognized dural puncture at T7–T8, particularly when it appears that an excessive amount of catheter was threaded into the epidural space.

Gordon O. Launcelott, M.D., F.R.C.P.C.
Director Acute Pain Service
Department of Anaesthesia
QE II Health Sciences Centre
Halifax, Nova Scotia, Canada

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Delayed Subarachnoid Migration of an Epidural Catheter

To the Editor: —Jaeger and Madsen recently presented a case of delayed subarachnoid migration of an epidural catheter.[1] Diagnosis was based on a late onset of phenomena suggestive for subarachnoid block (SAB), even though initially the block appeared to be epidural. Unfortunately, the diagnosis was not confirmed by analysis of aspirated fluid or radiographic determination of catheter position. There may, however, be another explanation for the described event.

The authors state that their first attempt at catheter placement at T11–T12 resulted in an obvious subarachnoid puncture. At the second, successful attempt at T10–T11, an Arrow FlexTip Plus catheter was inserted with the Tuohy needle bevel oriented cephalad. More than 24 h after establishing a normal epidural block, the patient was found with what appeared to be a high SAB caused by intrathecal local anesthetic (LA) and opioids. The authors attribute this to a delayed subarachnoid migration of the catheter. However, it is possible that the catheter was still in the epidural space. It was demonstrated for both lumbar[2] and thoracic[3,4] epidural anesthesia that roughly 50% of catheters inserted through a cephalad-oriented Tuohy needle are directed in caudad direction. The close proximity of the orifice (the Arrow FlexTip catheter is an end-hole catheter) to the preexistent dural puncture site may then have permitted large amounts of local anesthetic and opioid to enter the subarachnoid space and cause a SAB. After an epidural infusion at a rate of 14 ml/h for over 24 h, the few ml of clear fluid that could be aspirated from the catheter may have been either LA/opioid solution, cerebral spinal fluid, or a combination of both. It is unfortunate that the authors did not record the amount of centimeters that the catheter was advanced beyond the Tuohy needle tip. The stated 15 cm marking at the skin is not of any importance without knowledge of the skin–dura distance in this particular patient.

Although in this case there was no laboratory or radiographic evidence to support either the theory above or the authors' theory, epidural catheter insertion one level cephalad of an inadvertant dural puncture site may not always prevent epidural solution from entering the subarachnoid space. This was demonstrated in a case report by Van Zundert and Scott, in which a patient died after lumbar epidural administration of a large amount of LA, one level cephalad of an earlier dural puncture site.[5] Diagnosis was confirmed during autopsy by determination of high levels of LA in the cerebrospinal fluid and location of the catheter in the epidural space.

W. Anton Visser, M.D.
Clinical and Research Fellow
Department of Anaesthesia
University of British Columbia
910 W. 10th Avenue
Vancouver, British Columbia
Canada

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