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.

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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.

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In Reply:—I thank Drs. Bromage, Launcelott, and Visser for taking the time to raise issues about this case that we were unable to include in our original letter because of word limitations. First, both are correct that the concentration of hydromorphone in the epidural infusate was 6 *micrograms* per milliliter and not milligrams as erroneously published. The size of the Tuohy needle in the Arrow epidural kit is 17 gauge.

Drs. Bromage and Launcelott assert that it is possible that all, or at least a significant fraction, of the epidural infusate (1.25 mg bupivacaine and 6 μg hydromorphone per ml and given at a rate of 14 ml/h) passed through the dural rent created during placement one vertebral interspace below the final epidural insertion at T10–11. I could not refute that notion without assaying cerebral spinal fluid. However, on clinical grounds, one would expect to see a significant and persistent cephalad migration of paralysis or at least noticeable paresis. Serial evaluations by the Anesthesia Pain Service and the nursing staff failed to reveal any extraordinary abnormalities in this patient who was ambulatory and receiving chest physiotherapy. Specifically, her neurologic examination at 5:00 PM the first postoperative day (30 h post-insertion) revealed a decreased sensation to cold but intact crude touch from T4 to L1 and intact motor. Reportedly, the patient was able to feed herself dinner without difficulty. After the incident, the patient's husband offered his impression that perhaps later that evening she appeared more somnolent and weaker, however, not sufficiently to be alarming. We had no other documentation of her condition before her nurse's discovery at 2:00 AM the next morning. I am compelled to assume that the epidural catheter was functioning as expected until sometime later that evening when there was a relatively abrupt transition to a subarachnoid block, with a dense motor block of lower and upper extremities with a sensorium responsive only to intense stimulation.

Despite her dismal diagnosis of mesothelioma, the patient was relatively robust and should have been capable of normal absorption and metabolism of bupivacaine and hydromorphone. Accumulation of drug can occur and must be monitored clinically, which is the purpose of APS rounds and vigilant observation as per our nursing protocol. Nonetheless, I do not believe that it is an appropriate comparison to equate the effects of 2.35–2.73 mg of hydromorphone given as an infusion over approximately 30 h with Dr. Bromage's experience with a single 1-mg epidural bolus. Finally, if a large portion of an epidural infusate or the migration of a catheter can occur through a previous accidental dural puncture, then perhaps we ought to reconsider the wisdom of the widespread practice of placing epidural catheters a level of two above such sites.

With regards to Dr. Launcelott's concerns regarding the quality of our care for this patient, I would like to state that our APS system provides the close and responsive service that he recommends. Our APS rounds *bis in die* include the completion of a standard daily progress note, which details drug dose and technique, catheter site assessment, patient sensory and motor levels, side effects, and patient satisfaction with the therapy. The nurses are instructed to contact the APS liberally for consultation (available 24 h) regarding changes in the patient's state or comfort level, and standing orders exist for the cessation of epidural infusions and the administration of naloxone should events such as the one we present occur. These steps were followed as our case illustrates. In addition, I echo the concerns of Dr. Launcelott regarding the monitoring of patients using epidural analgesia outside the operating suite. With the present economic pressures to explore new avenues by which to ply our trade outside of the operating room, anesthesiologists are finding themselves using drugs and techniques under conditions that are frequently suboptimal as compared with those in a conventional OR. It is neither feasible to place all patients in intensive care units for the sole purpose of postoperative pain management nor is it possible for the anesthesiologist to remain continuously at the bedside. Attempts at using pulse oximetry on a large scale on the wards often lead to non-compliance because of restriction to patient movement, frequent false alarms, and inattentiveness by nursing staff because of current workloads. Because I suspect that the patient may ultimately be our best "monitor," more time spent educating the patient and family as to the need for continuous self-assessment and recognition of early warning signs of undesired levels of weakness or sedation before the placement of epidural catheters could prove useful.

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