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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.

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2. Bromage PR, Camporesi E, Leslie J: Epidural narcotics in volunteers: Sensitivity to pain and to carbon dioxide. Pain 1980; 9:145–60

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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