

## CORRESPONDENCE

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## Subarachnoid Catheters and the Cauda Equina Syndrome: Hypotheses in Need of Trial

*To the Editor:*—The search for the etiology of cauda equina syndrome associated with subarachnoid catheters has focused on three areas of suspicion:

1. Maldistribution due to caudal subarachnoid pooling of spinal solutions, leading to osmotic damage.
2. Osmotic damage from hypertonic, glucose-containing solutions of 800 and 550 mosmol/kg for "heavy" lidocaine and bupivacaine, respectively.
3. Neurotoxicity from subarachnoid pooling of concentrated local anesthetic solutions.<sup>1,2</sup>

In the 3 yr since microcatheters were first associated with the cauda equina syndrome, it is remarkable that experimental inquiries have not included the potential for mechanical injuries to the meninges and the spinal cord itself.

The physical nature of microspinal catheters imposes two acknowledged handicaps: a high internal resistance to flow related to their small caliber, and a tendency to kink because of their thin walls. Each or both of these factors may frustrate attempts at standard safety maneuvers in verifying subarachnoid location by free aspiration of cerebrospinal fluid. A third factor, enhanced tissue penetrability related to their small cross-sectional area, has not been examined.

The flimsy arachnoid membrane is an important pharmacologic barrier<sup>3</sup> but a poor physical container and is easily separated from the tough dural tube that surrounds and supports it.<sup>4</sup> The underlying subdural space is readily entered from the outside by spinal or epidural needles and catheters<sup>5-7</sup> and from the inside through the delicate arachnoid membrane. We know that injections into the subdural space may strip the arachnoid away from substantial areas of the overlying dura, causing local ballooning of the subdural space and the possibility of localized ischemic damage to the meninges and neighboring neural elements.<sup>8</sup> We also know that subarachnoid-placed catheters are capable of penetrating the substance of the spinal cord, permitting injection of fluids or air directly into the cord with potentially catastrophic results.\* Whether this type of injury to the lumbar enlargement can mimic a "cauda equina syndrome" is currently unclear.

Based on this background information, it is reasonable to propose two related hypotheses that seem ripe for testing in the context of risks and benefits of continuous subarachnoid anesthesia:

**Hypothesis 1.** An unknown proportion of attempted subarachnoid catheterizations will end in the subdural space by one of two routes: 1) direct entry during insertion of the spinal needle or 2) entry into the subarachnoid space followed by retrograde penetration of the arachnoid on the opposite side of the thecal tube. Injections into the subdural space will be loculated and will distend the loculated region in proportion to the volume injected. Within such

intensely focused areas of maldistribution, hyperosmolar or neurotoxic concentrations of local anesthetic may be held in close juxtaposition to neighboring cord or spinal roots, causing localized neural injury. In addition, vascular elements spanning the distended loculus may be sufficiently stretched to suffer partial or complete occlusion,<sup>8</sup> leading to ischemic injury of the arachnoid.

**Hypothesis 2.** An unknown proportion of attempted subarachnoid catheterizations may result in immediate or delayed penetration of the spinal cord by the tip of the intrathecal catheter.

Both of these hypotheses need to be tested using appropriate animal and human tissue preparations before the risks of microspinal catheters can be seen in clear anatomic and pharmacologic perspective. Microcatheters are not unique in the nature of their anatomic risks. They differ from large subarachnoid catheters only in degree, by factors related to their small size. Microcatheters strengthened by ferromagnetic inlays may present greater risk because of their increased rigidity and penetrability and because of their energetic mobility in the presence of intense magnetic fields encountered during magnetic resonance imaging.<sup>9</sup>

Finally, contemplation of the first hypothesis may force reassessment of a false assumption underlying current practice and clinical research of single-shot subarachnoid anaesthesia. Research publications continue to be based on an inadequate single-space paradigm, despite radiologic evidence that the subdural space is entered partially or completely in 5-10% of attempted subarachnoid punctures.<sup>10,11</sup> Acknowledgment of this second space as a potential sink of variable size would do much to bring about a reevaluation of existing data on dose-spread relationships for both single-shot and continuous spinal anesthetics and to encourage the adoption of techniques designed to avoid inadvertent subdural entry.<sup>12</sup>

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## Severe Sepsis after Intravenous Injection of Contaminated Propofol

*To the Editor:*—Several cases of postoperative sepsis due to intravenous injection of contaminated propofol have recently been reported.<sup>1</sup> We describe a severe outbreak of sepsis that underscores the importance of aseptic techniques in the handling of this commonly used anesthetic.

During an 8-h period, four patients who had undergone various clean surgical procedures in the same operating room developed *Klebsiella pneumoniae* septicemia within a few hours after surgery. Three patients were admitted to intensive care with severe sepsis, two of them in our unit. These two patients developed acute respiratory distress syndrome, refractory septic shock, and multiple organ failure but finally recovered after aggressive supportive therapy. The other two patients also recovered.

Two anesthesiologists had been involved in the care of patients 1 and 2 and of patients 3 and 4, respectively.

The epidemiologic investigation showed that the septicemia was due to injection of contaminated propofol. The same *K. pneumoniae* strain was identified in a culture of the incriminated propofol and in blood cultures from the four patients. Cultures of unopened vials of propofol from the same lot were negative. The following sequence of events was reconstructed. The evening before surgery, a vial of 500 mg propofol was opened, and the next day (12 h later) the remaining contents of this vial (stored at room temperature) were used to induce anesthesia in patients 1 (at 9 AM) and 2 (at 11 AM). For the induction of anesthesia in patient 3 (at 2 PM), the remnants of the first vial of propofol were drawn into a syringe, to which was added propofol from a second vial. About 2 h later, patient 4 received propofol from the second vial, which had been contaminated by the syringe used for the third patient. Propofol was given only for induction as a bolus of 150 mg, except in the case of patient 4, who received two boluses of 50 mg for light sedation.

Patients 1-3 presented the most severe sepsis, because the time between contamination of the vial and induction led to infusion of a larger bacterial inoculum.

Other intravenous anesthetics contain preservatives or have low or high pH. By contrast, propofol, a nonpyrogenic, soybean oil-in-water emulsion, contains no preservative or antimicrobial agents. Various microorganisms grow extremely well in propofol.<sup>2,3</sup>

To avoid severe life-threatening complications due to bacterial

growth in contaminated propofol, anesthesiologists should be aware of the manufacturer's new recommendations for propofol use, which include cleaning of the outside of the ampule with alcohol immediately before opening,<sup>4</sup> preparation of propofol syringes under aseptic conditions immediately before the anesthesia procedure, and use of each vial, catheter, and syringe for only one patient.<sup>2</sup>

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