Spinal Anesthesia in the Febrile Patient

Anesthesiologists have long considered sepsis to be a relative contraindication to the administration of spinal anesthesia. The needle might introduce infected blood into the subarachnoid or epidural space and cause meningitis or epidural abscesses. Some have speculated that performance of dural puncture alters the protection afforded by the blood–brain barrier and allows infection by an unknown mechanism.1

In support of these concerns, several authors have observed the development of meningitis after performance of dural puncture in bacteremic laboratory animals.2-4 Furthermore, there are several published cases of patients who underwent either diagnostic dural puncture or spinal anesthesia during confirmed or presumed bacteremia and who subsequently developed meningitis.5-9 However, physicians often perform diagnostic lumbar puncture in patients with fever and/or bacteremia of unknown origin. If dural puncture during bacteremia results in meningitis, one would expect that unequivocal clinical data should exist. However, clinical studies are few and conflicting and are limited primarily to pediatric patients at high risk for meningitis.10-15

Wegeforth and Latham10 performed diagnostic lumbar puncture in 93 patients who were suspected of having meningitis. The diagnosis was confirmed in 38 patients. Of the remaining 55 patients with normal cerebrospinal fluid, 6 were bacteremic at the time of lumbar puncture. Of those 6 patients, 5 subsequently developed meningitis. It was implied, but not stated, that meningitis did not occur in the remaining patients who had both sterile blood cultures and normal cerebrospinal fluid. These lumbar punctures were performed during an epidemic of meningitis, and one wonders whether some or all of these patients would have developed meningitis without antecedent lumbar puncture.

Teale et al.15 reviewed the records of 277 children with bacteremia. They noted that meningitis occurred in 7 of 46 (15%) children who had normal cerebrospinal fluid obtained during bacteremia, compared with only 2 of 231 (1%) children who did not undergo lumbar puncture (P < 0.001). All cases of meningitis after lumbar puncture occurred in infants less than 1 yr of age. Among bacteremic infants who underwent lumbar puncture, only 2 of 17 infants who received antibiotic therapy developed meningitis, compared with 5 of 6 untreated infants (P = 0.003). (It was implied, but not stated, that these infants received antibiotic therapy after lumbar puncture.)

In contrast, Smith et al.15 reported diagnostic lumbar puncture in 11 preterm neonates with sepsis at 9–22 days after delivery. None of the infants developed meningitis. Pray1 noted that 8 of 30 (27%) children with pneumococemia developed meningitis after normal lumbar puncture, compared with 86 of 386 (22%) similar children who did not undergo lumbar puncture (P = not significant). Similarly, Eng and Seligman12 observed that the incidence of meningitis after lumbar puncture did not significantly differ from the incidence of spontaneous meningitis in bacteremic patients. Shapiro et al.14 concluded:

The development of bacterial meningitis in children with occult bacteremia is strongly associated with the species of bacteria that causes the infection, but not with a lumbar puncture . . . . Children with high-density bacteremia may appear to be more severely ill than children who have bacteremia with lower concentrations of bacteria, and therefore may be more likely to undergo a lumbar puncture.14

Teale et al.15 acknowledged that clinical judgment might have enabled their pediatricians to perform lumbar
puncture in children in whom meningitis was developing before the cerebrospinal fluid was diagnostic. Thus, selection bias clouds our interpretation of these retrospective epidemiologic studies.

Some anesthesiologists have cited anecdotal cases of meningitis as the basis for recommending that we avoid spinal anesthesia not only in patients with existing infection, but also in patients at risk for transient, intraoperative bacteremia. However, few data suggest that spinal anesthesia before or during bacteremia is a risk factor for meningitis in adults. Dripps and Vandam prospectively studied 8,460 patients who received 10,098 spinal anesthetics between 1948 and 1951, and they reported no cases of central nervous system infection. Likewise, Phillips et al. reported their experience with 10,440 patients who received spinal anesthesia between 1964 and 1966. None of these patients experienced central nervous system infection. These authors did not state how many patients were febrile during administration of spinal anesthesia. However, a substantial number of these patients underwent obstetric or urologic procedures, and it is likely that some patients had bacteremia after, if not during, dural puncture. Similarly, two retrospective reviews of 27,000 and 505,000 obstetric patients who received epidural anesthesia included no cases of meningitis and only two cases of epidural space infection. This safety record is remarkable, given the frequency with which parturients develop chorioamnionitis, fever, and/or bacteremia during labor.

In this issue of ANESTHESIOLOGY, Carp and Bailey report their observations of the association between meningitis and dural puncture in bacteremic rats. Briefly, 12 of 40 animals that underwent cisternal puncture during Escherichia coli bacteremia subsequently developed meningitis. Bacteremic animals not undergoing dural puncture did not develop meningitis, and dural puncture in the absence of bacteremia also did not result in infection. These results augment those from earlier laboratory studies in at least three ways. First, in the present study, the bacteremia was similar in magnitude to that which occurs clinically during the early phase of sepsis. Second, only animals with a circulating bacterial count of ≥ 50 CFU/ml at the time of dural puncture developed meningitis. Third, Carp and Bailey included a fourth group of bacteremic animals who received an intraperitoneal dose of gentamicin 15 min before performance of cisternal puncture. None of these 30 animals developed meningitis.

Carp and Bailey appropriately stated that one should not apply their observations to the performance of epidural anesthesia, which may incur a greater likelihood of venous injury and which typically includes the introduction of an indwelling foreign body. (However, epidural anesthesia usually does not result in violation of the meningeal barrier to infection.) The authors also acknowledged several other limitations in the application of their study to clinical practice. First, although E. coli is a common cause of bacteremia, it is an uncommon cause of meningitis. Second, the relative size of the dural tear produced by a 26-G needle in rats is greater than that which occurs in humans. Third, anesthesiologists perform spinal anesthesia at a lumbar interspace, in contrast to the cisternal site of dural puncture in the present study. Fourth, anesthesiologists inject a solution of local anesthetic, which is perhaps bacteriostatic and which may provide some protection against subsequent infection. I would add two additional limitations. First, the authors knew the identity of the bacterium (i.e., E. coli), and also they knew that it was susceptible to gentamicin. Second, in contrast to the transient, low-grade bacteremia encountered in many obstetric and urologic patients, animals in the present study likely had hemodynamic and metabolic derangements characteristic of early sepsis.

Nonetheless, epidemiologic data and the present study together provide helpful guidelines for anesthesiologists who give spinal anesthesia in patients at risk for bacteremia. First, one need not avoid spinal anesthesia in patients at risk for transient, low-grade, intraoperative bacteremia after dural puncture. Second, appropriate antibiotic therapy before anesthesia may lessen the risk of meningitis or epidural abscess in patients with established infection. Surgeons and obstetricians often begin antibiotic therapy before surgery or delivery in febrile patients. Heretofore, some obstetricians were reluctant to give antibiotics before delivery in patients with chorioamnionitis. However, several studies have recently suggested that antepartum antibiotic therapy reduces the risk of maternal and neonatal morbidity in patients with chorioamnionitis. I acknowledge that there are published cases of meningitis and epidural abscess after spinal anesthesia, despite preoperative administration of antibiotics. Nonetheless, I give spinal or epidural anesthesia to selected patients with evidence of systemic infection, provided that appropriate antibiotic therapy has begun, and the patient has shown a positive response to therapy (e.g., a decline in temperature).

We do not give regional anesthesia in the absence of other relevant information. Rather, we provide care for febrile patients who require anesthesia for labor, delivery, or emergency surgery. When one considers the risks of infection with regional anesthesia, one should ask: What are the alternatives? What are the consequences of withholding regional anesthesia in a febrile patient? For example, what is the greater risk in a febrile parturient: meningitis or epidural abscess after spinal or epidural anesthesia, or failed intubation and aspiration during general anesthesia?

Choice of anesthesia remains problematic in those cases when surgeons defer antibiotic therapy until they have...
obtained culture specimens at surgery. (It would have been interesting if Carp and Bailey had included a fifth group of bacteremic animals who received gentamicin 15 min after dural puncture.) If the anesthesiologist believes that spinal anesthesia is the anesthetic technique of choice, there may be some cases when he or she should insist that antibiotics are given before anesthesia. Otherwise, it seems prudent to avoid spinal or epidural anesthesia in untreated patients with overt evidence of sepsis.

Finally, in the rare cases when central nervous system infection occurs after regional anesthesia, one should not assume a cause-and-effect relationship between the anesthetic and the infection. Most cases of meningitis and epidural abscess occur spontaneously. Eng and Seligman stated: “Even if an appropriate temporal sequence . . . is documented . . ., one cannot differentiate spontaneous meningitis from lumbar puncture-induced meningitis in the individual patient.”

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References

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