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Bacteremia, Spinal Anesthesia, and Development of Meningitis

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Septic meningitis subsequent to subdural anesthesia has been reported only rarely in the past 25 years.^{1,2} Numerous series have reported thousands of spinal anesthetics free of this complication,^{3,4} and it is now thought to be completely avoidable by heeding contraindications and maintaining asepsis during lumbar puncture.

REPORT OF A CASE

A 60-year-old man was admitted for removal of kidney stones. Preoperative evaluation showed moderate obstructive pulmonary disease and chronic alcoholic intake. Balanced anesthesia using nitrous oxide-oxygen-meperidine-pancuronium was given for kidney stone removal, without complication. Postoperatively the patient had slight fever (temperatures 38.0-38.4 C) and leukocytosis (leukocyte counts 8,000-15,000) for three days, following which he had increasing amounts of hematuria, necessitating blood transfusions. On postoperative day 6 the patient was afebrile, with a leukocyte count of 12,000 (58 per cent polymorphonuclears), but because of continuing hematuria, he was taken to the operating room for emergency transurethral clot evacuation of the bladder. At that time gentamicin administration was started. An atraumatic lumbar puncture was performed with a 22-gauge needle from a disposable spinal kit (Sherwood Medical Instruments) following 1-minute iodophor preparation of the area. Cerebrospinal fluid was clear and free-flowing, and 10 mg tetracaine, 1 per cent, with 100 mg of 10 per cent dextrose, was injected. A satisfactory block was established. Twenty minutes later transfusion of a unit of whole blood was started, and another 20 minutes later, a shaking chill developed. The blood transfusion was discontinued after approximately 200 ml had been infused and diphenhydramine, 25 mg, was given despite the absence of cutaneous rash and change in vital signs. Shaking chills continued in the recovery room as the spinal block wore off. Hematologic tests revealed no cross reaction with the donor blood and no hemolysis. Culture of the donor blood and two consecutive cultures of the patient's blood showed no growth. Urinary culture was negative as well. At 0200 hours the next day, the temperature was 39.4 C and the blood pressure was 240/130 torr. Carbenicillin was added to gentamicin therapy despite the negative blood culture. Later that day the patient complained of headache and back pain, and appeared confused. Meningismus was not present. This clinical course continued for two days, after which time a lumbar puncture was performed. Opening pressure was 130 mm H₂O, with cloudy fluid containing 94 erythrocytes and 324 leukocytes/cu mm (80 per cent polymorphonuclears). Cerebrospinal fluid protein was 84 mg/100 ml and glucose was 44 mg/100 ml, with a serum glucose of 111 mg/100 ml. One colony of *Staphylococcus epidermidis* grew, but was considered likely to be a contaminant. At this time carbenicillin and gentamicin were discontinued and nafcillin and chlor-

amphenicol started. Repeat lumbar puncture three days later showed 119 leukocytes/cu mm with only 18 per cent polymorphonuclears, protein 81 mg/100 ml, glucose 50 mg/100 ml with serum glucose 100 mg/100 ml, and no growth on culture. A third lumbar puncture after another two days showed 1,400 leukocytes/cu mm with 30 per cent polymorphonuclears, glucose 24 mg/100 ml and protein 100 mg/100 ml. Cerebrospinal fluid culture grew streptococcus, group D, enterococci, so nafcillin was replaced by ampicillin. Chloramphenicol, which had been discontinued two days before, was reinstated, and the patient's condition started to improve.

DISCUSSION

Group D streptococci are unusual etiologic agents in meningitis, and it is important for therapy to distinguish between enterococcal and nonenterococcal organisms.⁵ In this case it is possible, but not likely, that the organisms were present in the central nervous system prior to spinal anesthesia. It is more likely that the enterococci entered the blood stream during the bladder irrigation and traversed the dura at the puncture site. This apparent complication of spinal anesthesia is very unusual considering the frequency of spinal blocks for urologic procedures.

Possible sources of contamination are fourfold: 1) break in sterile technique; 2) infection in the tract of injection; 3) contaminated instrument tray; 4) bacteremia. Urologic surgery is a common cause of bacteremia.⁶ Bacteremia, as reflected by increases in specific antibodies, can occur in as many as 59 per cent of urologic operations even though blood cultures are much less frequently positive.⁷

In the present case, the lumbar puncture may have created a site of diminished resistance or *locus minoris resistentiae*^{8,9} in the blood-brain barrier, allowing contamination. Epidural abscess may also occur in this manner. If this mechanism exists, then it is surprising how rarely bacterial meningitis is reported, especially in view of the very frequent utilization of spinal analgesia in urologic procedures. For the cardiac, diabetic, or otherwise compromised patient undergoing urologic procedures, regional anesthesia is often the preferred technique.

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Wrist Circumference Predicts the Risk of Radial-arterial Occlusion after Cannulation

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Thrombotic occlusion of the radial-artery frequently occurs following percutaneous cannulation for monitoring purposes. Although usually temporary and asymptomatic,¹ these thrombi occasionally result in serious vascular complications such as distal ischemia and gangrene of the hand,² or necrosis of the skin overlying a segment of occluded artery.³ Recently, we observed that measurement of wrist circumference prior to cannulation can be used as a noninvasive method for predicting the likelihood of development of subsequent radial-artery occlusion.

METHODS

The subjects of this study were 100 consecutive patients (60 male, 40 female, mean age 59 years \pm 5 SD) requiring direct arterial monitoring for major elective operations. During the preanesthetic interview, informed consent for the study was obtained and the patency of both radial and ulnar arteries was verified using Allen's test⁴ and a Doppler ultrasonic flow probe. Wrist circumference was measured to the nearest centimeter on the side to be cannulated, using a snugly applied tape measure at the bony prominence of the radial and ulnar styloids.

Just prior to operation radial-artery cannulation was performed with 18-gauge Teflon[®] Catheters[‡] as previously described.¹ Heparinized 0.9 per cent saline solution (2 units/ml) was infused continuously via an

Intraflow[§] system at a rate of 3 ml/hour, and all catheters were removed after 26 hours \pm 4 SD. Just before decannulation, arteriography was performed by injecting 3 ml contrast solution[¶] while a roentgenogram of the wrist and hand was performed. Two radiologists who did not know the clinical histories measured vessel lumen diameter to the nearest .25 mm and recorded the presence or absence of occlusive vascular lesions.

After decannulation, radial-artery function was evaluated daily using both Allen's test and Doppler examination as previously described¹ until the patients were discharged from the hospital. Statistical comparisons were performed using Yates' correction for chi-square test, Student's t test for nonpaired data, and linear correlation of two variables.

RESULTS

Occlusive radial-artery thrombi developed in 35 patients following percutaneous cannulation. Fifty-two patients had wrist circumferences measuring less than 18 cm, and these sustained a 47 per cent incidence of arterial occlusion. This was significantly different ($P < .05$) from the 21 per cent incidence of occlusion that occurred in the 48 patients with wrist circumferences of 18 cm or more. A negative linear relationship ($r = -.98$) was found between the incidence of post-cannulation radial-artery occlusion and the patients' wrist circumferences (fig. 1).

Confirming previous observations that cannulation causes more occlusive lesions in smaller radial arteries than in larger ones,⁵ we also found a negative linear relationship between the sizes of the cannulated vessels and the incidence of thrombosis (fig. 2).

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