The authors are certainly to be congratulated on this piece of work, and I hope it will aid in tipping the balance away from the recommendation of general anesthesia during chymopapain injection. It is always very pleasing to see one's early stand, especially one so hotly criticized, eventually confirmed, and so elegantly.

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References


Pseudoappendicitis of Neurosurgical Origin

To The Editor: Yount, et al., have reported four cases of cerebrospinal fluid ascites as a result of ventriculoperitoneal shunting (Young RA, Glazier MC, Mealey J Jr, et al: Cerebrospinal fluid ascites complicating ventriculoperitoneal shunting. Report of four cases. J Neurosurg 61:180-183, July, 1984). We wish to add a further rare complication of this procedure.

This 42-year-old woman had a large brain tumor, mainly in the right middle fossa. Computerized tomography scans revealed a hypodense area indicating a cystic space-occupying mass, and a diagnosis of epidermoid tumor was made. (This was confirmed by a successful operation 3 years later.) At her first presentation, the patient refused operative removal of the mass; therefore, cyst-peritoneal drainage with a Hakim valve was undertaken to reduce the intracranial pressure. Three weeks later, she came to the surgical department with complaints of fever, vomiting, abdominal tenderness, and a typical McBurney's sign. An appendectomy was performed; the vermiform process was found to be normal, but cholesterol derivatives of the shunt fluid had caused a localized peritonitis.

This possible complication should be borne in mind when caring for patients with peritoneal shunt devices.

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Treatment of Shunt Infections

To The Editor: We enjoyed the recent report by Frame and McLaurin (Frame PT, McLaurin RL: Treatment of shunt infections with intrashunt plus oral antibiotic therapy. J Neurosurg 60:354-360, February, 1984), since it suggests a more pragmatic alternative in managing patients with the difficult problem of cerebrospinal fluid (CSF) shunt infections. However, two practical issues need clarification before adopting the methods suggested.

First, a few words regarding the direct adaptation of the methodology for determining "serum" inhibitory and bactericidal titers for use with CSF as suggested by the authors and also as recommended in a recent review in the Journal. Unlike the measurement of serum bactericidal levels, no standard laboratory methodology exists for similar assays of antibiotic activity in CSF, as we recently discovered during the management of a child with prolonged Pseudomonas meningitis. Since shunt infections are caused by a variety of bacteria, several different antibiotics have been used for intraventricular injection. Frame and McLaurin used vancomycin, kanamycin, gentamicin, ampicillin, methicillin, nafcillin, and cephalothin in their series. For the assay of gentamicin activity in the CSF, we have learned that a concentration of 0.05% NaCl is required in the diluent used for the serial twofold dilutions of CSF. Similar problems may well be encountered while assaying other antibiotics. Hence, a critical appraisal and standardization of laboratory methodology is required before recommending routine determination of an antibiotic activity in the CSF.

Second, although of the nine patients in Frame and McLaurin's series whose CSF was assayed for bactericidal and inhibitory titers, two had externalization of the distal ends of the shunts, there was no indication whether the antibacterial assay was performed before or after the shunt externalization. Another significant variable that may affect the antibiotic concentration in the ventricular fluid is prevention of the "washout phenomenon" by clamping the catheter distal to the site of injection for 2 to 4 hours after instillation of the antibiotic.

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References


Response: Kaul, et al., are correct in that the diluent for antibiotic standards used in measuring cerebrospinal fluid (CSF) concentrations of antibiotics must reflect the physicochemical milieu of the CSF because the unknown value is compared to the standards no matter what technique is used for the assay. However, inhibitory or bactericidal titers in body fluid are a "laboratory
artifact” which does not necessarily reflect only the concentration of antibiotics, and which is not compared to external standards for the value derived from the test. It is appropriate to use a diluent for the serial dilutions which is reasonably close to the fluid being assayed. Kaul, et al., correctly point out that there is very little standardization of this procedure, and each laboratory tends to have its own technique. The most common recommendation for serum inhibitory titers is that the specimen be serially diluted with serum. Since the protein content of the serum is approximately 200 times greater than that of CSF, we have elected to perform the serial dilutions in supplemented Mueller-Hinton broth, which contains no protein.

The more difficult question to be answered is whether the “laboratory artifact” of a serially diluted inhibitory or bactericidal titer accurately reflects therapeutic efficacy. This information can only be obtained by comparing the titer to clinical outcome. Assays were performed repeatedly on our patients both before and after shunt externalization. In an attempt to consolidate data, we reported in our paper the highest titer obtained in these patients. For those patients who had externalization, the titers were occasionally lower and sometimes higher after the externalization procedure. We could see no consistent relationship that would reflect the “washout phenomenon.”

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