Acute Hydrocephalus After Subarachnoid Hemorrhage

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This issue of Stroke includes an important discussion of acute hydrocephalus after subarachnoid hemorrhage (SAH), contributed by a group of investigators from The Netherlands1 who have made several important recent contributions to our understanding of the pathophysiology of SAH. The data of Hasan et al,1 derived from a careful study of 473 consecutive patients with SAH admitted within 72 hours after ictus, are interesting in several respects. They show that hydrocephalus, defined as a bicaudate index on computed tomogram (CT scan) exceeding the 95th percentile for age, occurs in approximately 20% of patients, which is in agreement with previous studies in which similar criteria were used.2-4 They also show that in approximately a third of these patients hydrocephalus is asymptomatic. More important, half of the patients with initial hydrocephalus and impairment of consciousness improve spontaneously within 24 hours. This finding confirms a previous report from the same group2 as well as another larger report from England3 but is in contrast to an earlier study by another group6 in which only one of nine patients with symptomatic acute hydrocephalus improved spontaneously.

An incidental finding of some interest is that Hasan et al1 found that hydrocephalus was just as frequent in their series of 46 patients who had negative angiograms and hence no identified etiology for their SAH. However, only one of these 46 patients was symptomatic, and there were no episodes of rebleeding or delayed ischemia, which confirms the generally held belief that such patients run a benign clinical course.

Improvement in consciousness after ventricular drainage occurred in 78% of the 32 patients treated by either external drainage (24 patients) or by an internal shunt (eight patients). This is in agreement with one report in which 18 of 21 such patients improved rapidly7 and another report in which such improvement occurred in seven of 11 patients.6

The disappointing aspect of the data of Hasan et al,1 however, is that ventriculitis developed in 50% of their patients treated with external drainage, and rebleeding occurred in 43% of all patients drained either externally or by internal shunt. Certainly, the high incidence of these two serious complications would support a very conservative approach toward ventricular drainage; however, some comments are in order before accepting these data as conclusive support for such a conservative approach. Hasan et al do not describe their technique for external drainage. It appears that precautions such as tunneling the ventricular catheter for several inches in the subcutaneous tissue,8 not leaving the catheter in place for more than 5 days, avoiding irrigation of the system, and maintaining a closed drainage system9 can reduce the rate of infection markedly. In addition, Hasan et al1 did not use prophylactic antibiotics. In one report, the use of prophylactic antibiotics reduced the risk of infection to 9% compared to 27% in a similar group of patients who did not receive antibiotics.10

Analyzing the problem of rebleeding after ventricular drainage is complicated. In an excellent study of continuous intracranial pressure (ICP) monitoring after SAH, Nornes11 showed that therapeutic maneuvers such as cerebral spinal fluid drainage and mannitol infusion, which lead to an abrupt decrease in ICP, may cause rebleeding after SAH. This has been confirmed by other observers,12,13 and was also noted by the authors in their previous report.2 In their current report, Hasan et al1 recorded rebleeding in 43% of their patients with acute hydrocephalus subjected to ventricular drainage, compared with 15% of hydrocephalic patients who were not drained and with 19% of patients without hydrocephalus; these differences were significant (p=0.044 and p=0.019, respectively). Hasan et al1 state that no factor other than the drainage itself could have accounted for these differences in rebleeding rates. However, there is no doubt that the first group of patients (those with hydrocephalus and drainage) were in worse neurologic condition than the other two groups since Hasan et al1 used either deterioration or a persistently decreased level of consciousness as an indicator for drainage. It is possible that the most important factor responsible for the higher rebleeding rate was, in fact, this difference in neurologic grade, which reflects to a large degree the
severity of the initial hemorrhage. The fact that patients in worse initial condition have a higher incidence of rebleeding was noted more than 20 years ago and was recently confirmed in a large cooperative study in which the rebleeding rate was 25% in patients in poor condition and 9.2% in good-grade patients.

Nevertheless, caution in reducing ICP too much or too abruptly remains in order. By keeping ICP >15 mm Hg, one group recorded a rebleeding rate of only 16% after ventricular drainage. In another study, in which drainage was allowed only when the ICP exceeded 25 mm Hg, the rebleeding rate was 17%; all rebleeding episodes occurred in patients in Grades III and IV, suggesting again that poor-grade patients have a higher incidence of rebleeding. Since Hasan and his colleagues apparently tried to keep ICP within the "safe" range of 15-25 mm Hg, it is not clear why their incidence of rebleeding was higher than in these other studies, but since they were very conservative in their indications for ventricular drainage, their drained patients were in poor neurologic condition and would therefore be expected to have a higher incidence of rebleeding.

I have found it convenient to classify hydrocephalus after SAH into three patterns. One group of patients develops delayed hydrocephalus days or weeks after SAH, usually after their aneurysm is clipped. These patients either fail to improve as expected or deteriorate after an initial improvement, with symptoms typical of the syndrome of "normal-pressure hydrocephalus," which, in fact, is what they have since at lumbar puncture ICP is usually in the high-normal range. Internal shunting is indicated in these patients, and generally they do very well afterward.

The second group develops mild to moderate ventricular enlargement in association with a slight worsening of the sensorium and increased headache during the first few days after SAH. I prefer to be conservative with this group and to treat them with corticosteroids, small doses of mannitol, and occasionally a cautious spinal tap to reduce ICP only moderately, as has been suggested by others. This conservative approach is preferred to avoid rebleeding if the aneurysm has not been clipped and to avoid a permanent shunt, which most of these patients will not require.

A permanent internal shunt is considered only if such conservative measures fail to result in significant improvement after several days or if the patient deteriorates in spite of such measures.

Patients in the third group present in poor neurologic condition immediately after SAH and have significant ventriculomegaly on their initial CT scan. They have true "acute" hydrocephalus and are the subject of the report in this issue of Stroke by Hasan and colleagues. These authors prefer not to place a ventricular drain in these patients immediately because some will improve spontaneously and because the authors encountered a high rate of infection and rebleeding in drained patients. Hasan et al recommend delaying ventricular drainage for at least 24 hours in the majority of these patients and then considering drainage only in those who fail to improve or deteriorate.

An alternative approach that I advocate is to perform immediate external ventricular drainage in all these patients if there is no other obvious explanation (such as massive intracerebral hemorrhage) for the decreased level of consciousness. Also, in contrast to the approach of Hasan et al, I would drain patients with massive intraventricular hemorrhage, most often using a different catheter for each lateral ventricle. In my experience, an occasional patient can be salvaged with this maneuver. By taking the appropriate technical precautions and by using prophylactic antibiotics as discussed above, the incidence of infection and rebleeding should not be unduly high. With early surgery, when appropriate, the rebleeding rate should be reduced even further. Provided that the rate of these complications can be kept acceptably low, little is lost with this approach in patients who might have improved spontaneously. Their drain can be safely removed in a few days after first testing it to see if occlusion results in deterioration. In my opinion, much is to be gained by early ventricular drainage in the other patients whose symptoms are primarily due to acute hydrocephalus and who do not do well without drainage. A delay of one or several days in relieving the usually very high ICP in these patients could not be other than deleterious and would add to the other injurious pathophysiologic effects of SAH.

References


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