COMMUNICATING HYDROCEPHALUS FROM SUBARACHNOID BLEEDING*

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The relationship of subarachnoid bleeding to subsequent hydrocephalus was first suggested by Bagley almost thirty years ago. From clinical and experimental studies of subarachnoid bleeding, he concluded absorption of some blood always takes place, but this is associated with meningeal thickening which may cause a block in the channels through which the fluid is returned to the blood stream. His experimental studies in dogs showed that ventricular enlargement did occur in 30 per cent of puppies after blood was injected into the cisterna magna. In both dog and man, he demonstrated meningeal thickening wherever blood came in contact with the meninges, even in the basal cisterns. He did not actually demonstrate a communicating hydrocephalus, though he did show that intermittent spinal fluid drainage produced clinical improvement.

Although hydrocephalus following subarachnoid bleeding has been described previously, a progressive obstruction in the cerebrospinal fluid pathways of the cisterns at the base of the brain which results in severe crippling neurological deficits, and impending death, has not been widely recognized. Strauss et al. found 4 “incidental” cases of hydrocephalus without ventricular obstruction at necropsy in their first 13 cases of subarachnoid hemorrhage from aneurysms. Sweet reported 2 cases in 1941 following aneurysmal rupture, neither recognized antemortem, and reviewed 6 other similar cases found in the literature. Strain and Perlmutter were the first to designate the point of obstruction in a similar case and successfully treated their patient. Such a hydrocephalus can also follow subarachnoid bleeding from trauma, for Penfield and Cone pointed out in 1943 that “mild degrees of hydrocephalus may be produced by extensive bleeding into the subarachnoid spaces, which tends to block absorption of cerebrospinal fluid for some time after the accident.”

Communicating hydrocephalus as a cause of progressing neurological deficits after subarachnoid hemorrhage appears to occur more frequently than the literature would indicate. During the past 3 years, 10 such cases in our series have been recognized because of progressive neurological deficits. A larger group showed mild ventricular enlargement not associated with significant neurological dysfunction. These 10 cases fall into three

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groups based on the etiology of the subarachnoid hemorrhage, namely 1) operative hemorrhage, 2) rupture of intracranial aneurysm and 3) head trauma. Brief clinical summaries of the cases in each group will be presented in sufficient detail to a) demonstrate the characteristic variability of signs and symptoms produced by this type of communicating hydrocephalus; b) show the need for pneumoencephalography at an early date in establishing the diagnosis; c) emphasize that profound and confusing neurological deficits associated with the hydrocephalus may be completely reversed by early treatment.

CASE REPORTS

I. SUBARACHNOID BLEEDING AT OPERATION

Case 1. M.D., a 60-year-old white female. Diagnosis: Intracranial neurofibroma left vagus nerve.


History. Hoarseness, and weakness of left shoulder and neck for 6 months with increasing left occipital headache. Weakness and inaccuracy of left hand for 3 months.

Examination. Paralysis of left vocal cord, paresis and atrophy of left shoulder and muscles of the neck, bilateral nystagmus, weakness of left tongue, hyperactive reflexes of upper extremity on the left with ataxia, and an equivocal right Babinski sign were present. Spinal fluid pressure was 150 mm.; protein was 290 mg. per cent. Roentgenograms of skull were normal.

Operation, Jan. 27, 1954. Ventriculography (Fig. 1A) and suboccipital craniectomy with resection of a neurofibroma of the left 10th nerve. At operation, done in the face-down position, a large artery in the tumor capsule bled profusely before it was clipped and blood spurted into and filled the basal cisterns anterior to the brain stem.

Course. Postoperatively, an aseptic meningitis was present for 10 days, secondary to break-down of the blood clot in the cisterns. The cerebrospinal fluid was xanthochromic and contained 2500 white blood cells; protein was 1400 mg. per cent. She improved and was asymptomatic from the 11th day until 6 weeks postoperative.

On Mar. 2, 1954, cerebrospinal fluid pressure was 100 mm. The fluid contained no cells; protein was 128 mg. per cent. On Mar. 6, 1954 there was onset of severe vertigo, nausea, and vomiting associated with the slightest motion of the head. She became completely incapacitated and lost 16 pounds in 10 days because of vomiting and inability to eat. High doses of Dramamine were ineffective.

On Mar. 16, 1954, pneumoencephalography (Fig. 1B) showed definite communicating hydrocephalus with enlarged ventricles as compared to the pre-operative ventriculogram. Spinal fluid pressure was 290 mm.

On Mar. 17, 1954, a right external ventriculomastoidostomy was performed with rapid and complete relief of vestibular irritability.

2nd Admission, Aug. 16, 1954. She had remained asymptomatic. Admission was to determine if the shunt could be terminated.

Examination. Paralysis of the left vocal cord and paresis of the left neck and shoulder persisted. The ventriculomastoidostomy was functioning well.

Course. On Aug. 22, 1954, pneumoencephalography (Fig. 1C) showed return of