Recognition and Treatment of Hydrocephalus Following Spontaneous Subarachnoid Hemorrhage*

KENNETH SHULMAN, M.D., BOSTON F. MARTIN, M.D., NINA POPOFF, M.D.,
AND JOSEPH RANSOHOFF, M.D.

Departments of Neurological Surgery and Neuropathology, New York University School of Medicine,
New York, New York

Extraventricular obstructive hydrocephalus following spontaneous subarachnoid hemorrhage in adults has been the subject of previous reports; 2, 6, 8, 12, 14, 15, 22, 23 the ventricular dilatation is postulated to occur secondary to an adhesive arachnoïdal reaction caused by the bleeding and leading to an obstruction of the cerebrospinal-fluid pathways. The appearance of the hydrocephalus often is delayed, for fibrosis of the meninges is thought to require at least 10–14 days to develop following subarachnoid hemorrhage. 12 Such a pathological process has been produced in the meninges of young dogs by Bagley 5 after repeated injections of blood into the cisterna magna or over the cerebral hemispheres. A more recent study 16 and personal data obtained in adult dogs with experimental subarachnoid hemorrhage, however, demonstrated hydrocephalus in only a very small number of animals receiving blood into the chiasmatic cistern.

Moreover, it is well known that in many patients with subarachnoid hemorrhage of spontaneous, traumatic, or operative origin a progressive hydrocephalus does not develop. The exact factors leading to sufficient reaction in the meninges with resultant hydrocephalus have eluded precise definition, and no statistics are available on the incidence of this complication in a large series of adults. Relevant data are available, however, in infantile hydrocephalus. Laurence 14 has determined, either by air study or autopsy, that the site of obstruction of fluid occurred at the exit foramina of the fourth ventricle or within the basal cisterns in 48 per cent of cases studied. Indeed, bleeding at the time of birth or shortly thereafter was present clinically in three-fifths of such children, suggesting that contamination of the subarachnoid space by red blood cells played a role in causing the arachnoïdal block in 30 per cent of his children with hydrocephalus.

The present paper reports the occurrence of a significant degree of ventricular dilatation in adults secondary to spontaneous subarachnoid hemorrhage from aneurysms or arteriovenous malformation. The presence of hydrocephalus was suggested by the patient’s clinical course, and procedures diverting cerebrospinal fluid were undertaken to treat the condition. Based on this experience, it is our contention that this complication is oftentimes not recognized and remains untreated. If properly managed, a number of patients can be improved significantly.

Case Material

This report is based on 7 patients with aneurysms of the anterior portion of the circle of Willis and 1 patient with an arteriovenous anomaly within the right lateral ventricle. Six of these patients have been cared for by members of the Department of Neurological Surgery of the New York University Medical Center. The other 2 patients (J.T. and S.G.) have been treated by Dr. J. Lawrence Pool.† All patients presented typical signs and symptoms of spontaneous subarachnoid hemorrhage, i.e., sudden severe headache, stiff neck with nuchal pain, photophobia, and malaise. Lumbar puncture established the diagnosis of subarachnoid hemorrhage, and

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† Gratitude is expressed to Professor Pool for his kindness in permitting these 2 cases to be included in this review.
Angiography showed the vascular pathology. At varying intervals after admission, the patients underwent craniotomy with aneurysmal clipping, resection of the malformation or, in 1 case, simple ventricular drainage. Hydrocephalus was suspected when, after a satisfactory postoperative course, mental confusion and headache supervened. At this time, lumbar cerebrospinal-fluid pressure usually was elevated and absorption of phenolsulfonphthalein decreased. Pneumoencephalogram demonstrated hydrocephalus of the extraventricular obstructive type. A permanent shunting procedure was performed in 7 patients and ventricular drainage in the single patient who expired before definitive craniotomy could be performed. The second death in the series was attributed to cerebral infarction following ligation of the common carotid artery. The remaining patients all showed a significant clinical improvement after treatment of the hydrocephalus.

Analysis of Data

A. Location of Aneurysm or Arteriovenous Malformation (Table 1).

Five aneurysms were on the anterior communicating artery and 2 were found on the intracranial internal carotid artery. The arteriovenous malformation was on the medial wall of the right lateral ventricle at the foramen of Monro. The location of the lesions in the present material supports previous data\(^5\) showing that aneurysms of the middle cerebral artery rarely cause hydrocephalus, and that aniomatous malformations that lead to ventricular dilatation occur within the ventricle.\(^12\)

B. Evidence of Multiple Episodes of Bleeding.

In 4 of the 8 patients, there was clear historical evidence of bleeding prior to the episode leading to hospitalization and diagnosis. This interval ranged from 3 weeks to 6 months in the aneurysmal group and was prolonged to 9 months in the malformation. Whether repeated small hemorrhages are more apt to produce the meningeal reaction is not clear.

<table>
<thead>
<tr>
<th>Patient, Age, Sex</th>
<th>Location of Lesion</th>
<th>History of Previous S.A.H.</th>
<th>Time from S.A.H. to Diagnostic Study</th>
<th>Ventricular Dilatation on Initial Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.G., 67 M</td>
<td>Ant. comm.</td>
<td>+</td>
<td>6 weeks</td>
<td>+</td>
</tr>
<tr>
<td>L.S., 54 F</td>
<td>Ant. comm.</td>
<td>+</td>
<td>3½ weeks</td>
<td>+</td>
</tr>
<tr>
<td>J.T., 30 M</td>
<td>Ant. comm.</td>
<td>-</td>
<td>9 weeks</td>
<td>?</td>
</tr>
<tr>
<td>S.G., 45 M</td>
<td>Ant. comm.</td>
<td>0</td>
<td>8 days</td>
<td>?</td>
</tr>
<tr>
<td>D.D., 47 F</td>
<td>Ant. comm.</td>
<td>0</td>
<td>10 days</td>
<td>?</td>
</tr>
<tr>
<td>C.O., 57 F</td>
<td>Post. comm.</td>
<td>0</td>
<td>8 weeks</td>
<td>?</td>
</tr>
<tr>
<td>L.H., 56 M</td>
<td>Int. carotid bif.</td>
<td>0</td>
<td>10 days</td>
<td>?</td>
</tr>
<tr>
<td>A.M., 52 F</td>
<td>A-V. rt. int. vent.</td>
<td>+</td>
<td>18 months</td>
<td>+</td>
</tr>
</tbody>
</table>

C. Interval from Onset of Symptoms to Angiographic Diagnosis.

The fibrotic reaction of the meninges that can follow the contamination of the subarachnoid space with blood is estimated to require 10–14 days to develop.\(^12\) Therefore, hydrocephalus may be seen on this basis if angiography is performed 2 weeks or more following the bleeding. In our cases, this interval ranged from 6 days to 9 weeks and in Case J.T. considerable hydrocephalus was demonstrated. Angiograms of the other patients showed some ventricular dilatation in all cases in which the films were available for review. The average time elapsing between the bleeding and the contrast study was 3½ weeks.

D. Amount of Bleeding as Determined by Initial Lumbar Puncture (Table 2).

There was no apparent correlation between the number of red blood cells on initial lumbar puncture and the subsequent development of hydrocephalus. It is perhaps the number of cells enmeshed in the pia-arachnoid rather than the number free in the cerebrospinal fluid that determines the degree of fibrosis that will result. Adams and Prawirohardjo\(^1\) demonstrated in dogs that 24 hours after red blood cells were injected into the cisterna magna less than 5 per cent of the total dose remained in the cerebrospinal fluid, while 20 per cent were recovered from the systemic blood, the remainder being enmeshed in the pia-arachnoid. Such data suggest that a second lumbar puncture after 24 hours should show only 5 per cent of the number of red blood cells found on the initial tap. The impression is gained, however, that
greater than 5 per cent is found in humans on repeated lumbar puncture suggesting that the phagocytic properties of human pia-arachnoid are less than those of the dog. In 5 of 6 patients in whom data were available, the protein in the cerebrospinal fluid was elevated as high as 350 mg. per cent. Whether an elevated protein can contribute to the development of hydrocephalus remains controversial.\(^7\)\(^-\)\(^11\) It seems possible that certain individuals bear a heightened allergic susceptibility and react more severely to the presence of blood in the subarachnoid space.\(^5\)

E. Findings at Craniotomy Relevant to Hydrocephalus.

In 3 patients, ventricular dilatation was recognized at the time of craniotomy. The exact findings are of some interest. In C. O. the anterior tip of the temporal lobe was resected in order to approach the aneurysm and in so doing, an enlarged temporal horn was encountered. In J. T., Dr. J. L. Pool used ventricular drainage in order to allow exposure of the aneurysm of the anterior communicating artery. In this patient, the observation was also made that there were dense adhesions of the arachnoid to the optic nerve. Patient S. G. was noted by Dr. D. Reynolds to have dense adhesions about the aneurysm of the anterior communicating artery which had rebled after clipping 7 weeks previously. In retracting the subfrontal area, a ballooned third ventricle was opened spontaneously. Gillingham\(^8\) has commented on the enlarged ventricles at the time of craniotomy and advocated drainage to facilitate retraction.

F. Time from Craniotomy to Symptoms of Hydrocephalus.

Symptoms of hydrocephalus became manifest 3–4 weeks after craniotomy. The lumbar-puncture pressure was elevated in all patients at the time hydrocephalus was suspected. The response to repeated lumbar puncture was not in itself useful in predicting whether a shunting procedure could be expected to produce improvement. Our usual experience was to note slight improvement after lumbar puncture and dramatic improvement after shunting.

G. Signs of Hydrocephalus (Table 3).

The most common symptoms and signs were headache, mental confusion and deepening state of consciousness. Pyramidal-tract signs were not prominent.

Although ventricular dilatation signifies a generalized increase in intracranial tension, only 1 of our patients manifested papille-

### Table 2

<table>
<thead>
<tr>
<th>Patient, Age, Sex</th>
<th>Admission Lumbar Puncture</th>
<th>Operation and Findings</th>
<th>Preoperative L.P. Pressures mm. H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.G., 67 F</td>
<td>RBC 5,880</td>
<td>Ventricular drainage</td>
<td>280-190-270</td>
</tr>
<tr>
<td>L.S., 54 F</td>
<td>RBC 47,000 Prot. 106 mg.%</td>
<td>Craniotomy</td>
<td>600-200-260-290</td>
</tr>
<tr>
<td>J.T., 39 M</td>
<td>RBC 40 Prot. 140 mg.%</td>
<td>Ventricular drainage—craniotomy: adhesions</td>
<td>250-300</td>
</tr>
<tr>
<td>S.G., 45 M</td>
<td>RBC 40 Prot. 89 mg.%</td>
<td>Craniotomy—craniotomy: 3rd ventriculostomy</td>
<td>180-120-220-180</td>
</tr>
<tr>
<td>D.D., 47 F</td>
<td>RBC 40 Prot. 89 mg.%</td>
<td>Craniotomy</td>
<td>180-120-220-180</td>
</tr>
<tr>
<td>C.O., 57 F</td>
<td>Prot. 30 mg.%</td>
<td>Craniotomy: dilated ventricles</td>
<td>210-180-160-140</td>
</tr>
<tr>
<td>L.H., 36 F</td>
<td>RBC 8,500 Prot. 350 mg.%</td>
<td>Craniotomy</td>
<td>210-280-240</td>
</tr>
<tr>
<td>A.M., 22 F</td>
<td>RBC 41,000 Prot. 315 mg.%</td>
<td>Craniotomy: dilated ventricles</td>
<td>180-300</td>
</tr>
</tbody>
</table>
dem. The lack of this finding has been commented upon by others.\textsuperscript{6,12} The operative observation in patient J.T. of perineurial arachnoidal fibrosis about the right optic nerve may explain the failure of papilledema to develop. Experimentally, Griffith \textit{et al.}\textsuperscript{9} were able to show that the bleeding of subarachnoid hemorrhage filled the perineurial space of the optic nerves, preventing the development of papilledema in response to experimental neoplasms. Bradford and Sharkey\textsuperscript{4} shunted blood from the femoral artery to the lumbar subarachnoid space in dogs. After death, blood was found within the perineurial sleeves of the optic nerve with diffusion into the areolar tissue behind the globe; however, papilledema did not develop in any of the animals and the nerve head was not found to be swollen at necropsy.

H. \textit{Special Studies.}

Two patients were studied with intrathecal neutral phenolsulphonphthalein. If it can be demonstrated that renal function is normal with 70 per cent excretion of dye after 2 hours following intravenous administration, then the failure to excrete at least 40 per cent in 6 hours after intrathecal instillation implies a block of cerebrospinal-fluid absorption.\textsuperscript{13} In patient C.O. the amount excreted was 15 per cent and in D.D. less than 5 per cent was produced. The test was not done in the remaining patients.

I. \textit{Radiographic Studies.}

In 6 patients, pneumoencephalography was performed showing 2–3X enlargements of the entire ventricular system including the fourth ventricle (Fig. 1). The air filled the basal pathways up to the interpeduncular cistern. No air passed anterior to this point, nor did air enter the cisterna ambiens. In no patient was there filling of the cortical subarachnoid spaces. It has been our experience that this picture is diagnostic of an extra-ventricular block at the incisura.

A number of patients had postoperative angiograms to check the aneurysmal obliteration. Ventricular dilatation at this stage was suggested by an abnormally wide sweep of the thalamostriate veins.\textsuperscript{17}

J. \textit{Treatment of Hydrocephalus (Table 4).}

One patient, R.G., was managed by external ventricular drainage alone and died from rebleding before craniotomy. The remainder of the series had a shunting procedure. Once hydrocephalus had been established by air study such a procedure was instituted as soon as possible. A number of patients, stuporous prior to shunting, responded rapidly with marked improvement in their state of consciousness after the by-pass.

K. \textit{Pathology.}

Two of the 8 patients succumbed and the pertinent clinical and autopsy findings follow:

\textit{Case 1. R.G., female, aged 67.} Thirty-nine days prior to death, the patient collapsed and had bloody spinal fluid. After gradual improvement she deteriorated with increasing cerebrospinal-fluid pressure, but without evidence of rebleding. In addition to an aneurysm, ventricular dilatation was noted on angiography performed 4 weeks after the onset of symptoms. She was placed on

<table>
<thead>
<tr>
<th>Patient, Age, Sex</th>
<th>Headache</th>
<th>Confusion</th>
<th>Lethargy</th>
<th>Papilledema</th>
<th>Lumbar Pressure Elevated</th>
<th>Special Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.G., 67 F</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>L.S., 54 F</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>J.T., 59 M</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>(atrophy)</td>
<td></td>
</tr>
<tr>
<td>S.G., 45 M</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>D.D., 47 F</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>C.O., 57 F</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>L.H., 36 F</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>A.M., 22 F</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

\textsuperscript{17} PSP < 5\%
ventricular drainage, improved, then died suddenly with evidence of fresh bleeding.

The brain showed a diffuse recent subarachnoid hemorrhage distributed mainly over the brain stem and dorsolateral aspect of the cerebellum. The cisterna magna was markedly distended by fresh clotted blood. The leptomeninges showed a slight generalized thickening. A ruptured aneurysm, 0.5 cm in diameter, was present at the junction of the left anterior cerebral and anterior communicating arteries.

Coronal sections of the cerebrum revealed the entire ventricular system to be markedly dilated and filled with fresh clotted blood.

Microscopically (Fig. 2) the leptomeninges at the base of the brain showed fibrous thickening with evidence of recent and old subarachnoid hemorrhage. There was a large number of red blood cells, and numerous phagocytes containing hemosiderin, as well as some lymphocytes and plasma cells.

**Case 2.** C.O., female, aged 57. The patient complained of severe intermittent headache in the left supraorbital region and transient episodes of blurred vision on the same side for approximately 1 year prior to her last admission to the hospital. Six months before death, she was treated for a well documented subarachnoid hemorrhage which cleared, leaving her with a residual organic mental syndrome. A left carotid arteriogram performed some 4 months prior to death revealed an aneurysm of the left posterior communicating artery. Shortly thereafter, a left frontal craniotomy with left partial temporal lobectomy and clipping of the aneurysm was performed. The patient did well initially but, over the next 4 weeks, showed signs of increased intracranial pressure with elevated spinal-tap pressures. A pneumoencephalogram revealed moderate, generalized ventricular dilatation. Three months before death, another episode of subarachnoid hemorrhage occurred. A repeated left carotid arteriogram revealed the presence of the aneurysm at its original location. Occlusion of the left common carotid artery was followed by hemiplegia and aphasia. The patient expired approximately 5 months following her last admission to the hospital, and 17 months after the onset of clinical symptoms.

At autopsy, there was no gross evidence of subarachnoid hemorrhage. A small unruptured aneurysm was present at the junction of the internal carotid and posterior communicating arteries on the left. Multiple extensive areas of recent infarction, affecting the cortex and white matter as well as the basal ganglia, were present bilaterally. The ventricular system appeared slightly dilated and revealed an old granular ependymitis. Microscopically, the leptomeninges were infiltrated with some hemosiderin containing macrophages and revealed focal areas of fibroblastic proliferation.

**Discussion**

The location of an aneurysm or malformation seems to bear a relationship to the devel-

<table>
<thead>
<tr>
<th>Patient, Age, Sex</th>
<th>Treatment of Hydrocephalus</th>
<th>By-Pass Performed</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.G., 67 F</td>
<td>Ventricular drainage</td>
<td>6 weeks</td>
<td>Died</td>
</tr>
<tr>
<td>L.S., 54 F</td>
<td>Ventriculojugular</td>
<td>10 weeks</td>
<td>Improved; discharged</td>
</tr>
<tr>
<td>J.T., 39 M</td>
<td>Interventricular-lumbar peritoneal</td>
<td>12 weeks</td>
<td>Improved; discharged</td>
</tr>
<tr>
<td>S.G., 45 M</td>
<td>Ventriculojugular</td>
<td>8 weeks</td>
<td>Improved; discharged</td>
</tr>
<tr>
<td>D.D., 47 F</td>
<td>Ventriculopleural</td>
<td>8 weeks</td>
<td>Improved; discharged</td>
</tr>
<tr>
<td>C.O., 57 F</td>
<td>Ventriculopleural</td>
<td>12 weeks</td>
<td>Improved</td>
</tr>
<tr>
<td>L.H., 36 F</td>
<td>Ventriculopleural</td>
<td>6 weeks</td>
<td>Died</td>
</tr>
<tr>
<td>A.M., 22 F</td>
<td>Ventriculojugular</td>
<td>18 months</td>
<td>Improved; discharged</td>
</tr>
</tbody>
</table>
opment of hydrocephalus after subarachnoid hemorrhage. In previously reported examples of this complication there were 5 aneurysms of anterior communicating artery, 3 of basilar artery and 3 of the internal carotid artery. The present material adds 5 lesions of the anterior communicating and 2 of the internal carotid arteries. The absence of this complication after aneurysmal bleeding of the middle cerebral artery has already been noted. Aneurysms in this location comprise 25 per cent of our total cases yet we have not seen hydrocephalus in this group. A number of possible explanations can be offered. Aneurysms in this position are in intimate contact with the brain substance of the frontal and temporal lobes, and bleeding probably occurs into the cerebrum with less contamination of the cerebrospinal fluid and hence less tendency to cause meningeal fibrosis. Secondly, the direction of the cerebrospinal fluid flow within the Sylvian fissure is such that the fluid passes upward into the cortical subarachnoid space rather than into the basal cisterns, resulting in a focal fibrosis less likely to produce obstruction of the cerebrospinal fluid. The third possibility is that repeated episodes of small bleeding increase the likelihood of meningeal fibrosis and hydrocephalus. The absence of specific neurological signs following a small bleeding from aneurysms of the anterior communicating artery or internal carotid artery is less likely to lead to a definitive neuroradiological study in contrast to bleeding from an aneurysm of the middle cerebral artery which usually produces neurological deficit and earlier diagnosis.

The location of the bleeding arteriovenous malformations associated with hydrocephalus is within the ventricular system. In the present case it was located along the medial surface of the right lateral ventricle with drainage into the septal and thalamostriate veins. The block of cerebrospinal fluid was at the incisura as in the aneurysmal group rather than at the outlets of the fourth ventricle.
The slight to moderate increase in size of the ventricles on the initial angiogram suggests that in the early acute phase of subarachnoid hemorrhage there is an impedance of absorption of cerebrospinal fluid or increased production of cerebrospinal fluid. The presence of clotted blood or cells and cellular debris in the basal cisterns obstructing these channels has been observed at autopsy in patients dying within 24 hours after subarachnoid bleeding. Blockage by red blood cells may also occur more distal at the arachnoid villi. Welch and Pollay have shown experimentally that these structures permit the perfusion of liquids and particles into the venous system in monkeys. Simmons recovered red blood cells labeled with P-32 in the systemic circulation after their injection into the subarachnoid space of rabbits. With large injections of blood into the subarachnoid space of dogs (1 to 2 cc. per kg.) Bradford and Sharkey noted that the red blood cells blocked the avenues of escape of saline injected into the subarachnoid space. Therefore, it would seem that the major sites of drainage for endogenous particulate matter in the human may be overtaxed acutely in subarachnoid hemorrhage and the continued production of cerebrospinal fluid leads to an acute ventricular dilatation, which in most cases is transient. The mechanisms by which a progressive ventricular dilatation occurred in the 2 cases that came to postmortem examination are not entirely clear. After the transient, acute ventricular dilatation subsides, a chronic progressive hydrocephalus will develop in a number of patients. In this late or chronic phase we must consider the meningeal reaction to red blood cells, the products of their breakdown or abnormal metabolites, which may lead to fibrous thickening of the leptomeninges. Hammes found patchy areas of fibrosis of the leptomeninges and focal areas of adhesions between the pia and the arachnoid in a series of 53 adults who died at different intervals of time after subarachnoid hemorrhage. In none of these cases, however, was ventricular dilatation present, and the author attributed the absence of hydrocephalus to the focal nature of the fibrosis. In the 2 cases being considered, the degree and the distribution of leptomeningeal fibrosis differed in no way from that seen in individuals of comparable age but with no history of antecedent bleeding except for the presence of hemosiderin in the current material. Therefore, an exact pathological correlate of the clinically proven hydrocephalus was not found.

The choice of a shunting procedure in most of our cases has been the ventriculo-pleural anastomosis. This procedure is simple, requiring a minimal amount of surgery in the acutely ill patient. In some of the patients the shunting device developed by Pudenz has been used, allowing for a palpable pump in the subcutaneous tissue of the scalp so that the patency of the system can be tested easily. The tubing has stayed open and has not required revisions. There have been no infections. The patients have not complained of pain in the chest from the foreign body in the pleural cavity. If the hydrocephalus arrests, the ventriculo-pleural shunt tubing need not be removed for there are no late serious complications such as the bacteremia encountered with the ventriculojugal bypass.

Summary

Eight adult patients with hydrocephalus secondary to spontaneous subarachnoid hemorrhage have been presented with a discussion of diagnosis and treatment. A number of possible mechanisms for the development of ventricular dilatation in this condition have been advanced and pathological data have been presented in 2 cases.

References

4. Bradford, F. K., and Sharkey, P. C. Physio-
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logic effects from the introduction of blood and other substances into the subarachnoid space of dogs. J. Neurosurg., 1962, 19: 1017–1042.


Discussion

Dr. Eldon L. Foltz: I would like to congratulate Dr. Shulman and his co-authors on this paper resulting from careful observations on the clinical and radiologic status of patients following subarachnoid hemorrhage. The hydrocephalus that results from subarachnoid bleeding, whether temporary or progressive, is still unrecognized in many instances, primarily because the presence of hydrocephalus is not suspected. Since our report in 1956 (Foltz and Ward) we have found that 10 to 12 per cent of patients who have suffered subarachnoid bleeding from a source near the mid-line do show definite hydrocephalus which can be demonstrated radiographically. This hydrocephalus may not be progressive in nature, for it may be self-limited and not require definitive treatment. However, it is our feeling now that 5 to 7 per cent of these patients will show symptomatic or progressive hydrocephalus which requires a shunting procedure. A temporary shunting procedure will oftentimes improve symptoms dramatically. We still use the ventriculostomy shunt since it is a very simple shunt to terminate when we are certain that the hydrocephalus has resolved. Resolution of the basal arachnoiditis does occur in some instances, and we are convinced that most of these patients can have the shunt removed later.

Unexpected complications can result from hydrocephalus following subarachnoid bleeding and I should like to report on a long-term follow-up in 1 of the cases we reported on in our paper in 1956. This patient was a young man, 26 years of age, who had suffered subarachnoid bleeding from a large aneurysm of the left anterior cerebral artery. The aneurysm was operated on intracranially and a large tantalum clip was placed successfully across the base of the aneurysm, thereby obliterating the neck. The arteriogram demonstrated, as you can see on the reproduction, that the aneurysm did arise from the left anterior cerebral artery and that, in addition, there was ballooning of the anterior cerebral arteries which on a progressive arteriogram, had progressed, indicative of progressive enlargement of the ventricular system. The pneumoencephalogram to support this
opinion demonstrated a communicating type of hydrocephalus, but because the pressure was low, operative treatment was deferred. Three weeks later a second pneumoencephalogram was performed and these two studies in comparison showed continued enlargement of the ventricular system from the progressing communicating hydrocephalus. The aqueduct is well demonstrated, showing ventricular enlargement with good filling of the fourth ventricle, aqueduct and the entire third ventricle, as well. Intraventricular pressure at the time of the air study was normal, yet the ventricles were enlarging, and the clinical status of the patient had deteriorated remarkably. In spite of a low pressure, a ventriculomastoid shunt was performed and his clinical status improved dramatically within 48 hours.

For the ensuing 9 years the patient did well with an occasional episode of obstruction of the shunt which then cleared up spontaneously. He died 9 years after the subarachnoid hemorrhage from a cardiac infarct. A complete postmortem examination showed two points of great interest to us: (1) The aneurysm of the left anterior cerebral artery was no longer present, and the clip that had been placed across the neck of the aneurysm was lying free in the subarachnoid space at the base of the brain. There was no evidence of a scarred mass representing the previous aneurysm, and close inspection and examination of the artery itself showed no defect in the arterial wall. (2) There was persisting scarring in the basal arachnoidal areas from the previous subarachnoid bleeding, but the most important thing, and the most surprising part, was the presence of complete aqueductal stenosis. Whereas the aqueduct had been well illustrated in the pneumoencephalogram at the time of the original procedure of shunting the cerebrospinal fluid, at the time of his death 9 years later, complete obstruction of the aqueduct was present with no evidence of cerebrospinal fluid passing through the aqueduct.

The postmortem pictures demonstrated this very well and crosses sections of the mesencephalon showed that the acquired aqueductal stenosis was characterized by multiple branchings of the aqueduct shown as multiple rosette formations, forking of the aqueduct, and complete obliteration of any continuity of aqueduct from third ventricle to fourth ventricle.

The conclusion from a case such as this might be that in the presence of a shunting procedure which takes spinal fluid and shunts it outside the head, the aqueduct is no longer needed for the "circulation" of the spinal fluid and undergoes stenosis and obliteration. The pathological and histological findings in this instance are recognized as an acquired type of aqueductal stenosis, and yet there are many of the characteristics in this case that are completely compatible with the so-called "congenital" aqueductal stenosis. A further report on this type of observation is intended by us.

I enjoyed Dr. Shulman's paper immensely. I feel it contributes further to the thesis that there is more hydrocephalus following subarachnoid bleeding than has been recognized by many in the past.

Dr. LESTER A. MOUNT: I would like to compliment the authors and to thank them for emphasizing one of the problems in the treatment of patients with subarachnoid hemorrhage. The authors, together with Dr. Foltz, have really left little to say.

Although leptomeningeal fibrosis does not occur before 10 days, clots within the ventricular system may block any of the foramina before 10 days, with resultant increase in intracranial pressure in the development of hydrocephalus.

Recognition of the presence of hydrocephalus is the important thing so the treatment can be instituted. Headache, mental confusion and decreasing state of consciousness are the usual symptoms of its presence, but papilledema occurs late or it may not occur at all. Suspecting hydrocephalus, one can then verify its presence and proceed with therapy.

Dr. JOHN E. SCARFF: The essayists should be complimented for directing our attention to a fact established long ago by Walter Dandy, that blood introduced into the subarachnoid cisterns of laboratory animals will produce hydrocephalus. We clinicians have, over the years, failed to give this observation its proper importance in the treatment of severe cranio- cerebral injuries.

A number of years ago when I was caring for cranio-cerebral injuries at Bellevue Hospital, I became aware of one subgroup of such patients who did not appear too seriously ill at the time of their admission, but began to "go bad" 8-12 hours after their injury; and from then on got progressively worse and died 24 to 48 hours after their injury, with all the symptoms and signs of acute severe increase of intracranial pressure. These patients usually showed large amounts of blood in the cerebrospinal fluid obtained upon lumbar tap at the time of admission. Trephinations made over the two cerebral hemispheres in search of surface hematomata failed to reveal any localized massive hematoma which could be evacuated. Autopsy showed the basal cisterns and the sulci leading up from the basal cisterns over the cerebral hemispheres to be filled with clotted blood, which of course had prevented the cerebrospinal fluid from reaching the areas over the cerebral cortex whence the cerebrospinal fluid could have been absorbed. Death of these patients, it is now obvious, had been caused by acute, severe, communicating hydrocephalus.

All patients with severe cranio-cerebral injuries, I believe, should have a cautious diagnostic lumbar puncture performed immediately upon admission to determine the relative amount of blood in the cerebrospinal fluid. Those patients who show heavy concentrations of blood in the cerebrospinal fluid should, in my opinion, have some form of ventricular drainage established promptly which could as soon as surgically feasible, be converted into some type of semipermanent shunt, such as a ventriculopleural shunt. This would take care of the excess cerebrospinal fluid which cannot be absorbed, and prevent development of acute communicating hydrocephalus, until the blood clots in the subarachnoid cisterns have liquefied and the red corpuscles in the capillary subarachnoid spaces within the cerebral sulci have hemolized and been removed by natural processes.

In brief, we would do well, I believe, to apply the suggestions which the essayists have made for the treatment of postoperative subarachnoid hemorrhage to post-traumatic subarachnoid hemorrhage. If this were done routinely I believe that a number of patients, who otherwise would be lost with severe cranio-cerebral injuries, might be saved.

Dr. RICHARD E. STRAIN: I want to compliment the authors for reemphasizing the necessity, at times, of surgically treating hydrocephalus developing after subarachnoid hemorrhage.
In 1954 before this Society, I reported that surgery for progressive hydrocephalus secondary to subarachnoid hemorrhage might be lifesaving in cases of aneurysmal removal in patients in whom a progressive downhill course developed after improvement following operation for their aneurysm. Strauss, Globus and Ginsberg in their classical study of subarachnoid hemorrhage, reported in 1934, had noted the presence of hydrocephalus in 4 of their first 13 cases studied. Bagley, in 1928, had produced such hydrocephalus experimentally by introducing blood into the subarachnoid space.

[Slide] My first patient had a large aneurysm of the anterior cerebral-anterior communicating arteries which was removed, after resecting the tip of the left frontal lobe. Marked hydrocephalus was found when the frontal lobe was resected.

[Slide] It was necessary in this removal to clip both anterior cerebral arteries. After reelevation of the flap resulted in normal findings when progressive lethargy developed the 2nd postoperative day, repeated lumbar punctures disclosed pressure of 440 mm. of H2O with considerable clinical improvement after each puncture. A lumbar subarachnoid peritoneal shunt 5 days after the initial operation was done with prompt (within hours) improvement. This patient has been followed approximately 10 years now, has driven a car and been productive as a bookkeeper.

Eight months after operation, he fell in a bathtub, fracturing L3 vertebra. This was followed by a severe left sciatica, made worse by pressing on his subcutaneous polyethylene tube. So 3½ years after operation the tube was clamped, without symptoms, for a week and then removed with relief of the sciatica. Various intelligence tests in 1957, 4 years after operation, done at the University of Miami, were normal.

The second patient was operated upon 6 months after the initial case. Multiple aneurysms had produced subarachnoid bleeding followed by lethargy and coma.

In both of these instances I think surgery for the progressive hydrocephalus following subarachnoid bleeding appeared lifesaving.

Foltz and Ward subsequently verified this in 1956, as have Kibler, Couch and Crompton from Queen Square in 1961. Schneider and McKissock have had isolated cases.

I want to congratulate the authors for their reemphasis on this aspect of surgical therapy.

DR. KENNETH SHULMAN: I thank all the discussers. We, too, would like to put in as innocuous a shunt as possible and which perhaps, if not needed any longer, would not do any harm. For this reason we are using the ventriculopleural shunt, feeling it remains in place and does not constantly challenge the patient with severe complications of ventriculo-atrial shunts.