Periventricular rigidity in long-term shunt-treated hydrocephalus

SANDEEP SOOD, M.D., JALIYA LOKUKETAGODA, M.D., AND STEVEN D. HAM, D.O.

Department of Pediatric Neurosurgery, Children’s Hospital of Michigan, Wayne State University School of Medicine, Detroit, Michigan

The failure of ventricles to enlarge after acute shunt malfunction in long-term shunt-dependent patients has perplexed investigators for many years.1-10 These patients often become very sick due to high ICP and slit-like ventricles are frequently detected at presentation. It is generally believed that the failure of the ventricles to enlarge is related to decreased brain compliance in these patients; however, the findings of Shapiro, et al.,15 have indicated that the pressure-volume index in these patients who acutely deteriorate is normal. Engel, et al.,9 have argued that periventricular gliosis due to long-term shunt-dependency prevents enlargement of the ventricles and causes elevated ICP and acute deterioration. Such periventricular gliosis has been shown in shunt-treated experimental hydrocephalus models.11,12 There is, however, no direct evidence that periventricular gliosis can or does restrict ventricular enlargement at the time of shunt malfunction in experimental or clinical hydrocephalus.3 Moreover, at the time of a shunt malfunction following a recent shunt revision, enlarged dilated ventricles may develop in some patients.5-10 This finding and the ability to induce ventricular dilation in these patients by using a lumbar drain12 indicate that periventricular gliosis may not restrict ventricular enlargement. To test the hypothesis that the shunt-dependent patients experience periventricular rigidity, we evaluated the transmission of pressure from the ventricle across the periventricular region to the surrounding brain.

Clinical Material and Methods

Fifteen pediatric patients, nine boys and six girls with a mean age of 9.7 years (SD 5.8 years; range 2-18 years) who had been ventricular shunt-dependent for more than 1 year were studied at the time of shunt revision. The patients underwent initial shunt placement (ventriculoperitoneal in 13 and ventriculoatrial in two) for congenital hydrocephalus (seven patients) or posthemorrhagic hydrocephalus (six patients). In all patients shunts were equipped with a differential pressure valve—Medos Programmable Valve (Codman, Raynham, MA) or a low-pressure PS Medical Valve (Medtronic, Goleta, CA). In one patient a Gravity Compensating Accessory (NMT Medical, Inc., Atlanta, GA) for siphon protection was used in addition to the differential pressure valve.

Of the 15 patients who presented with symptoms of shunt malfunction, six were obtunded, three suffered from headache with vomiting, and the remaining six experienced severe headache. In all patients small ventricles were evident at presentation, and the mean frontooccipital horn ratio for these patients was 0.26 ± 0.01, (normal ratio 0.37).12

Institutional research board approval and informed consent for each patient were obtained. The study was conducted after patients were anesthetized using pentothal or propofol with 1 to 2% isoflurane monitored noninvasively.

At the time of shunt revision a ventricular catheter was removed, the ventricular catheter was again inserted by a three-way connector on the ventricular catheter sheath and connected to the same track by a small burr hole but away from the sheath of the ventricular catheter. The burr hole was 2 cm from the cortical surface and connected to the ventricular catheter sheath by a three-way connector. The transducer was zeroed to the level of the burr hole. The CSF was drained using an ICP monitor (Codman, Raynham, MA) to 2 cm from the cortical surface and connected to a strip recorder so that simultaneous intraventricular (IVP) and intraparenchymal (IPP) pressures could be monitored. A variable amount (2–10 ml) of injected fluid was required to get a larger volume to obtain an optimal waveform response.

Two sets of measurements were completed.

A: Computerized tomography scans obtained at the time of presentation (black arrows) and at the time of shunt revision (white arrows). Note the slit ventricles.

B: Intraventricular pressure increases 5 mm Hg after saline infusion, whereas IPP increases 2.4 mm Hg and 3.3 mm Hg.

Abbreviations used in this paper: ICP = intracranial pressure; IPP = intraparenchymal pressure; IVP = intraventricular pressure; SD = standard deviation.

1 J Neurosurg: Pediatrics / Volume 102 / March, 2005
Periventricular rigidity in shunt-treated hydrocephalus

![Image](image.png)

Results

Despite some loss of CSF during shunt revision, the mean IVP at presentation was high, measuring 24.1 mm Hg (range 10.7 mm Hg; range 15–54 mm Hg). In 11 patients, the mean IVP at presentation was greater than 15 mm Hg. The mean IVP was significantly higher in patients who were obtunded compared with those who experienced headache or vomiting (33.5 ± 10 mm Hg and 17.9 ± 4.9 mm Hg, respectively; p < 0.01).

The baseline IVP after drainage of CSF but prior to the infusion study was 9.7 ± 4.7 mm Hg and was significantly higher than the IVP of 7.5 ± 4.4 mm Hg (p < 0.001) (Fig. 1). When stratified for the site of the shunt, this difference was significant only in the nine patients with parietal shunts and not in those six patients who underwent frontal shunt revision (12.2 ± 4.2 mm Hg compared with 9.7 ± 4.2 mm Hg for parietal placement and 6.1 ± 2.6 mm Hg compared with 4.2 ± 2.1 mm Hg for frontal placement; p = 0.17; p < 0.001).

Analysis of variance revealed no significant relationship between the cause of hydrocephalus and ventricular size (p = 0.08), ICP at the time of shunt revision (p = 0.35), baseline IVP (p = 0.16), or IVP (p = 0.14).

Following the infusion of fluid into the ventricle, there was no significant difference between the increase in the IPP and IVP (3.6 ± 2.3 and 3.3 ± 2.1 mm Hg, p = 0.39 for the first set; 3.5 ± 1.1 and 3.2 ± 1.2 mm Hg, p = 0.12 for the second set of measurements) (Fig. 2). The difference between the mean increase in IVP and IPP was not significant even when stratified by etiological factors (p = 0.39 for posthemorrhagic hydrocephalus of prematurity and p = 0.6 for congenital hydrocephalus) or symptoms (Table 1).

The mean pressure-volume index calculated using the method described by Shulman and Marmarou was 24.1 ml (standard error of the mean 4.6 ml).

Discussion

Physical principles indicate that the compliance of a barrier separating two compartments determines the transmission of pressure from one to the other. A noncompliant or stiff barrier will not allow pressure transmission and, therefore, a gradient of pressure across the compartments will result. If a change in pressure occurs in one compartment, the amount of pressure change in the other compartment is lower if the barrier is stiff and noncompliant. These principles are explained in mathematical terms in the Appendix.

In this study the failure to demonstrate a significant difference in the change of pressure in the ventricles and the brain after infusion of fluid is evidence against the presence of a stiff ventricular wall. There is, however, strong evidence for the existence of periventricular gliosis that could result in a stiff ventricular wall. Microscopic examination of the ependymal wall in experimental hydrocephalus reveals severe ependymal damage and subependymal edema during the early stage of the evolution of hydrocephalus. A massive loss of cilia along the ependymal surface occurs, and within 3 hours of the induction of hydrocephalus by balloon occlusion of the fourth ventricle, a noticeable increase in the subependymal astroglial activity is evident. After 60 days an attempt at ependymal regeneration
is apparent, and chronic edema and reactive gliosis at the subependymal level begin. Similar changes have been observed in the ventricular wall adjacent to a ventricular catheter even in animals without hydrocephalus.5 Engel, et al.,4 have proposed that the shunt catheter itself promoted the induction of subependymal glial proliferation. Pressure necrosis occurs in the places where the catheter touches the ependymal wall, and a gradual demudulation of the cilia is evident. The inflammation of the periventricular tissue leads to gliosis and the outgrowth of tissue into the catheter.4 The most marked changes in the periventricular region, however, were found in experimental hydrocephalus that was treated with an effective shunt rather than an ineffective shunt;2 this occurred despite the normalization of water content and the extracellular space in animals treated with an effective shunt. It is believed that this phenomenon represents an ongoing process of tissue injury related to abnormal stresses created by shunt-induced negative pressure within the ventricle.2 From such dramatic periventricular gliosis and the resulting resistance to cannulation of the ventricle during shunt surgery it has been inferred that altered ventricular compliance is responsible for failure of the ventricles to dilate in shunt-dependent patients at the time of shunt malfunction. In this study there is no direct evidence of the existence of periventricular gliosis; however, the results provide compelling evidence that the ventricular wall is not stiff and is unlikely to restrict ventricular enlargement.

The pressure across the partition between the ventricle and the brain (the periventricular region) is initially normal. Compensatory responses of patients with long-term symp-

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>ICP at Shunt Revision</th>
<th>Pressure Change After Saline Infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(mm Hg)</td>
<td>IPP (mm Hg)</td>
</tr>
<tr>
<td>P value</td>
<td></td>
<td>IVP (mm Hg)</td>
</tr>
<tr>
<td></td>
<td>(mean ± SD)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>headache/vomiting (9)</td>
<td>17.9 ± 2.4</td>
<td>4.62 ± 2.4</td>
</tr>
<tr>
<td>obstruction (6)</td>
<td>33.5</td>
<td>21.8 ± 5.0</td>
</tr>
</tbody>
</table>

* All pressures are in mm Hg (mean ± SD).
† Significant at a level of p < 0.01.
Periventricular rigidity in shunt-treated hydrocephalus

A compensatory venous congestion may result in patients with long-term shunt-dependency due to the uncoupling of the CSF–venous Starling relationship. It is possibly the increase in venous distensibility, which leads to a rapid collapse of the veins due to even a slight change in transmural pressure at the time of shunt malfunction. This results in venous congestion and a rise in brain parenchymal pressure, which prevent ventricular enlargement.

Conclusions

Although there is morphological evidence for the presence of periventricular gliosis in some long-term shunt-dependent patients, the findings of our study indicate that the periventricular wall does not restrict enlargement of the ventricles. A complex interaction between the altered venous compliance, venous distensibility, and a change in blood flow to the brain related to prolonged CSF overdrainage may be among the factors responsible for the failure of ventricles to enlarge.

Appendix

If the initial pressure in the ventricle is $P_{vi}$ and that in the brain is $P_{bi}$ and the compliance of the ventricle and the brain are defined as $C_v$ and $C_b$, respectively, then, after injection of volume $(v)$ into the ventricle, the final pressure in the ventricle is $P_{vf}$ and in the brain $P_{bf}$, then the following relationships are obtained:

$$C_v = \frac{\Delta v}{P_{vf} - P_{vi}}$$

$$C_b = \frac{\Delta v}{P_{bf} - P_{bi}}$$

The pressure across the partition between the ventricle and the brain (the periventricular region) is initially $\Delta p_{vi}$ and after infusion of fluid in the ventricle is $\Delta p_{vf}$, hence:

$$\Delta p_{vf} = P_{bi} - P_{vi}$$

$$\Delta p_{bf} = P_{bf} - P_{bi}$$

The relationship between the changes in the pressure across the periventricular region $(\Delta p)$ for a change in volume $(\Delta v)$ represents the compliance of the periventricular region $(C_p)$. Hence:

$$\Delta p = \Delta p_{vf} - \Delta p_{bi} = P_{bf} - P_{vf} - (P_{bi} - P_{vi}) = P_{bf} - P_{bi} - (P_{vf} - P_{vi}) = \Delta Pf - \Delta Pbf = P_{bf} - P_{vi}$$

$$C_v = \frac{\Delta p}{\Delta v} = \frac{\Delta Pbf}{\Delta v}$$

The stiffness or the compliance of the periventricular region $(C_p)$ can hence be estimated by difference in the change in pressure within the brain compared with that in the ventricle $(\Delta Pf - \Delta Pvf)$ after infusion of fluid into the ventricle. A greater difference in pressure would indicate that the periventricular region is stiffer and vice versa.

References


Manuscript received April 26, 2004. Accepted in final form August 11, 2004.

Address reprint requests to: Sandeep Sood, M.D., Wayne State University School of Medicine, Children Hospital of Michigan, 3901 Beaubien, Detroit, Michigan 48201. email: ssood@med.wayne.edu.