Several studies have shown that a reduction in cerebral blood flow (CBF) occurs in adults with normal-pressure hydrocephalus and that an improvement in blood flow occurs post–shunt treatment. In neonates, semiquantitative studies using transcranial Doppler ultrasound have shown a decrease in the middle cerebral artery resistive index after shunt insertion, indicating a reduction in downstream resistance. Near-infrared spectroscopy has demonstrated an increase in CBF in infantile hemorrhagic hydrocephalus after CSF removal. Leliefeld et al. used an MRI-based technique to simultaneously measure cerebral inflow via the carotid and basilar arteries and brain parenchymal volume to obtain quantitative data on CBF. In a study of 15 infants from 1 day to 7 months of age, the mean CBF before shunting was 25 ml/100 g/min, which increased to 50 ml/100 g/min following treatment with a shunt. A doubling of CBF after shunting suggests a significant increase in vascular resistance before shunt placement as well as rapid improvement with a reduction in resistance post–shunt insertion. Given this rapid change in resistance, reversible compression of the blood vessels must occur somewhere along the vascular tree. Multiple sites of compression have been described in the literature, from the arterioles around the ventricles to the capillary bed to the venules and sinuses. The purpose of this study was to identify the site of reversible compression of the vascular tree in neonatal hydrocephalus.

Methods

The hospital ethics committee approved the study protocol; therefore, the study was performed in accordance with the 1964 Declaration of Helsinki.

Abbreviations used in this paper: CBF = cerebral blood flow; ICP = intracranial pressure; VENC = velocity encoding.
The radiology information system at a tertiary care referral hospital was retrospectively interrogated, and all children referred for MRI examinations, including MR venography and susceptibility weighted imaging, in the first 28 days of life in the period from April 2010 to April 2013 were selected for analysis. A chart review was performed to obtain clinical findings in this cohort.

All patients had undergone imaging on a 3-T superconducting magnet (Vario, Siemens). Standard sagittal T1-weighted and axial T2-weighted and FLAIR images as well as 2D time of flight MR venography sequences were obtained. The maximum dimension of the dominant transverse sinus was measured on the MR venogram for each child (Fig. 1 left). Obtaining an axial susceptibility weighted sequence is standard in neonates undergoing brain MRI to exclude cerebral hemorrhage, and this image was used to measure the subependymal veins. The maximum dimension of the subependymal vein was measured as the vein passed over the thalamus adjacent to the lateral ventricle on the susceptibility weighted study (Fig. 1 right). The ventricular size index, or Evans index, was measured as the maximal cross-sectional size of the frontal horns of the lateral ventricles compared with the size of the inner aspect of the intracranial cavity along the direction of the ventricular measurement and expressed as a percentage. Patients with hydrocephalus underwent MR flow quantification sequences, which have been described in detail elsewhere. In brief, two velocity-encoding (VENC) values were used: 40 and 150 cm/sec. The lower VENC value was selected to maximize the accuracy of the venous sinus measurements, and the higher one was used to maximize the arterial measurements. In those patients with a very low arterial inflow, the lower VENC value was used for the arterial measurement as well. The arterial plane of section was selected to intersect the vertical portion of the petrous internal carotid arteries and to pass through the clivus. The venous plane was selected to pass through the sagittal sinus 2 cm above the torcular herophili. Planar imaging and raw flow quantification data were archived on the hospital picture archiving system, and the raw data were available for reprocessing. Regions of interest were placed around the carotid arteries, basilar artery, and sagittal sinus in each patient. Care was taken to exclude aliasing by retrospectively manipulating the base lines of each resultant graph.

Adding the flow from the 3 arteries gave the total supratentorial blood inflow. To calculate CBF, brain volume was measured by manually tracing the parenchyma on contiguous 3-mm axial T2-weighted slices, and the resulting cross-sectional areas were multiplied by the slice thickness. Sagittal sinus outflow was obtained from the region of interest placed around this vessel. The percentage of arterial inflow represented by the sagittal sinus was calculated. The pulsatility index for each vessel was calculated as the peak velocity minus the minimum velocity divided by the average velocity.

Results

Ninety-two children underwent MRI examination of the brain during the study period. In 41 patients, however, a susceptibility weighted study was not performed because of either patient movement or time constraints. An MR venogram was obtained in 14 of the remaining 51 patients. Three boys among these 14 cases were found to have clinical and standard imaging findings consistent with hydrocephalus. The remaining 11 age-matched neonates, 6 males and 5 females, underwent MRI for conditions unrelated to head enlargement or elevated intracranial pressure (ICP) and served as controls. One of these controls was excluded because of significantly raised central venous pressure associated with congenital cardiac defects (this patient was subsequently found to have dilated subependymal veins).

Illustrative Cases

Case 1

At an antenatal ultrasound, this boy was noted to have growth retardation. He was born at 35 weeks’ gestation via Cesarean section as a result of distress seen on continuous cardiotocography during labor. Apgar scores were poor and the boy was resuscitated. His head circumference was 30 cm, which was between the 10th and 50th percentiles. He was placed in the neonatal intensive care unit for respiratory support (continuous positive airway pressure) for 5 days and developed hyaline membrane disease. An initial head ultrasound was normal (Fig. 2A). Two weeks later, a follow-up head ultrasound showed bilateral Grade I subependymal hemorrhages but no obvious parenchymal or ventricular extension. Despite the minor nature of the hemorrhages, there was prominent ventriculomegaly (Fig. 2B). The fontanels were full, and the head circumference had increased to the 75th percentile. An MRI examination was performed 2 days later, confirming the ventriculomegaly and subependymal hemorrhages (Fig. 2C); the subependymal veins were prominent. The aqueduct was patent. An MR venogram revealed the collapse of large segments of the sagittal sinus as well as both sigmoid sinuses and the jugular bulbs (Fig. 2D). Magnetic resonance imaging quantification of blood flow revealed an arterial inflow of 145 ml/min with a pulsatility index of 1.5. The brain volume was 215 cm³, making CBF 67 ml/100 g/min.
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Sagittal sinus flow was 25 ml/min and completely non-pulsatile; the cross-sectional area of the sinus was 0.40 cm$^2$. The sagittal sinus was returning 17% of the arterial inflow. The patient was treated conservatively and initially fared well, with a stable head circumference and non-bulging fontanels. At 2 months of age, however, the boy’s head circumference began to increase significantly, and he had bulging fontanels; therefore, a shunt was inserted, although follow-up MRI was not performed.

Case 2

This boy was born full term (38 weeks) via normal vaginal delivery. His mother had gestational hypertension (preeclampsia), and he was small with a head circumference at the 7th percentile. Initial Apgar scores were low but improved at 5 minutes, and there was no need for resuscitation. He was noted to be lethargic with vomiting and poor feeding from birth. Four days after birth, his head circumference was at the 17th percentile and continued to increase significantly, and he had bulging fontanels; therefore, a shunt was inserted, although follow-up MRI was not performed.

Sagittal sinus flow returned 40 ml/min or 50% of the arterial inflow and was nonpulsatile. The cross-sectional area of the sinus was 0.12 cm$^2$. Because of the rapid increase in the patient’s head size and the amount of blood in the ventricles, a shunt tube was inserted. Follow-up MRI 7 days after the initial scanning (4 days postoperatively) showed a reduction in ventricular size (Fig. 3B). Before shunt insertion, the subependymal veins had been prominent and the sagittal sinus had shown large segments of its length collapsed along with the collapse of the sigmoid sinuses and jugular bulbs bilaterally (Fig. 3C). After shunt placement, the sagittal sinus, transverse sinuses, sigmoid sinuses, and jugular bulbs returned to normal size (Fig. 3D). Follow-up MRI performed when the boy was 3 months of age showed a head circumference at the 14th percentile. The ventricles were prominent with some ex vacuo dilatation due to the loss of white matter. An MR venogram was normal. Magnetic resonance flow quantification showed an arterial inflow of 310 ml/min with a pulsatility index of 1.2. Brain volume was 550 cm$^3$, making CBF 56 ml/100 g/min. Sagittal sinus flow was 150 ml/min or 48% of the arterial inflow with a pulsatility index of 0.7. The cross-sectional area of the sagittal sinus was 0.21 cm$^2$.

Case 3

At the routine 18-week antenatal ultrasound, this boy was noted to have ventriculomegaly (Fig. 4A). He was...
The patient was treated conservatively. At the follow-up when the boy was 6 months of age, his head was tracking along the 97th percentile and he was clinically well with soft fontanels. An MRI study at that time showed prominent ventricles but small subependymal veins (Fig. 4E). An MR venography study showed an improved size of the distal sagittal, transverse, and sigmoid sinuses as well as the jugular bulbs (Fig. 4F). Flow quantification revealed a greatly improved arterial inflow of 540 ml/min with a pulsatility index of 1.1. Brain volume was 610 cm$^3$, making CBF 88 ml/100 g/min. Sagittal sinus flow was 310 ml/min or 57% of the arterial inflow with a pulsatility index of 0.4. The sinus cross-sectional area was 0.39 cm$^2$.

**Controls**

Indications for MRI in the control children were seizure and/or apnea in 4, suspected viral meningitis and/or cerebritis in 3, parenchymal hemorrhage without ventricular extension in 2, and reduced muscle tone in 1. The average gestational age at birth for this group was full term (39 ± 3 weeks). Clinically, fontanels in these patients were not distended, and head circumferences were within the normal range (34.4 ± 2.3 cm). Standard imaging showed no evidence of hydrocephalus or raised ICP. Magnetic resonance venography studies demonstrated no evidence of outflow stenosis or anomaly. Birth weight, age at MRI examination, and measurements of the veins are summarized in Table 1. On average, the subependymal veins were twice as prominent and the transverse sinuses were half as big in the patients with hydrocephalus, compared with those in controls, with no overlap in measurements between the groups.

**Discussion**

The effect of shunt insertion in neonatal hydrocephalus is the doubling of CBF in many patients. The study by Leliefeld et al. showed that preoperative CBF was lower at 17 ml/100 g/min in the 6 patients in the first 28 days of life than in the older cohort, in whom CBF averaged 25 ml/100 g/min. To put this into perspective, the threshold of CBF below which neuronal function is impaired and tissue is at risk for infarction is around 22 ml/100 g/min in the primate model, and the ischemic penumbra in adult humans is estimated to be 20 ml/100 g/min in the primate model, and the ischemic penumbra in adult humans is estimated to be 20 ml/100 g/min. The threshold for tissue loss in neonates is not defined; however, the metabolic load of the growing brain is unlikely to decrease the blood flow required to ward off tissue damage in the younger cohort. Indeed, studies of infantile hydrocephalus in animals suggest that impaired cerebral hemodynamics and oxidative metabolism play a significant role in the pathophysiology of brain injury in this disorder. Neonates with hydrocephalus have had elevated brain levels of lactate at MR spectroscopy, indicating anaerobic glycolysis and impaired metabolism. Thus, the low CBF associated with hydrocephalus in neonates is likely to be hemodynamically significant. Given the rapid restoration in blood flow after CSF diversion, a significant reduction in vascular resistance probably occurs. Blood flow is nor-
mally regulated by the arterioles. In neonatal hydrocephalus, a significant increase in CBF following a reduction in ICP is generally considered to indicate impaired autoregulation due to microvessel compression. This would indicate that the reversible elevation in resistance is not caused by arteriolar spasm. Indeed, in a group of neonates with hydrocephalus, those with adequate cerebrovascular reserve did well with conservative treatment, whereas those with poor reserve required an operation. The good shunt responders regained autoregulation.

Among the 3 hydrocephalus cases in the present study, the patient in Case 1 had a normal CBF of 67 ml/100 g/min and initially fared well (presumably the cerebrovascular reserve was adequate in this patient), whereas the patients in the other 2 cases had low CBF (27 and 23 ml/100 g/min). One of these latter patients required treatment, and the other was treated conservatively but suffered white matter volume loss.

Excluding the arterioles, the remainder of the vascular tree consists of a series of passive collapsible tubes. In order for blood to flow, the pressure within each segment must be higher than that in the segment immediately downstream. Thus, the arterioles are at a higher pressure than the capillaries, which are at a higher pressure than the venules and the sinuses. The pressure within the CSF of the ventricles, the interstitial spaces of the brain, and the subarachnoid spaces over the brain has been shown to be identical in communicating and noncommunicating hydrocephalus, with no static or pulse pressure gradient being detectable between compartments. Given that vascular collapse develops because of an elevated pressure gradient from the interstitial space to the lumen of the vessel and that this gradient will always be at its greatest where the intraluminal pressure is at its lowest, vascular collapse in hydrocephalus will always occur at the site of the most distal collapsible segment (that is, somewhere in the venous segments).

**Susceptibility Weighted MRI**

The susceptibility weighted MRI study is sensitive to paramagnetic substances such as deoxygenated hemoglobin and was originally developed to look for blood products in the brain parenchyma. It became obvious that this imaging study was also very sensitive to deoxygenated hemoglobin within the small veins. Thus, susceptibility weighted scans have been used to measure the size of the medullary veins in multiple sclerosis and in neonatal hypoxic-ischemic injury. In the latter condition, the increased prominence of the medullary veins has been associated with a worsening outcome as well as both dilation of the veins and an increase in deoxygenated hemoglobin. In the current study, the subependymal veins were twice as prominent in acute hydrocephalus as in the normal state, and although some of this increase may be attributable to an increase in deoxygenated hemoglobin, the veins were probably dilated as well. In children with hypoxic-ischemic injury—where there was an increase in deoxygenated hemoglobin but no increase in venous pressure—the medullary veins were more prominent than the subependymal veins, which were less consistently present in general and were prominent only when the medullary veins were moderately to extremely prominent. In the current study, the subependymal veins were always more prominent than the medullary veins. If the subependymal veins are larger than normal in hydrocephalus, then the pressure within them must be elevated over and above the elevated CSF pressure, and this indicates that the flow-limiting resistance must be further downstream than these segments—that is, within the sinuses.

### TABLE 1: Summary of characteristics in 13 patients who underwent MRI studies

<table>
<thead>
<tr>
<th>Group</th>
<th>Birth Weight (g)</th>
<th>Age at MRI (days)</th>
<th>Ventricular Size Index (%)</th>
<th>Subependymal Vein Size (mm)</th>
<th>Transverse Sinus Size (mm)</th>
</tr>
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<tbody>
<tr>
<td>control</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>1</td>
<td>3780</td>
<td>3</td>
<td>35</td>
<td>0.8</td>
<td>5.6</td>
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<tr>
<td>2</td>
<td>2940</td>
<td>9</td>
<td>31</td>
<td>0.5</td>
<td>5.2</td>
</tr>
<tr>
<td>3</td>
<td>3760</td>
<td>8</td>
<td>32</td>
<td>0.8</td>
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<tr>
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</tr>
<tr>
<td>5</td>
<td>3050</td>
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<td>27</td>
<td>0.7</td>
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</tr>
<tr>
<td>6</td>
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<td>28</td>
<td>0.7</td>
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<tr>
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<td>1920</td>
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<td>30</td>
<td>0.5</td>
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<tr>
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<td>28</td>
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<td>0.9</td>
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<tr>
<td>9</td>
<td>3000</td>
<td>2</td>
<td>29</td>
<td>1.0</td>
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</tr>
<tr>
<td>10</td>
<td>2500</td>
<td>22</td>
<td>31</td>
<td>0.6</td>
<td>5.2</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>2964 ± 580</td>
<td>12 ± 10</td>
<td>32 ± 3</td>
<td>0.7 ± 0.2</td>
<td>6.2 ± 1.4</td>
</tr>
<tr>
<td>hydrocephalus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1820</td>
<td>26</td>
<td>58</td>
<td>2.7</td>
<td>2.2</td>
</tr>
<tr>
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<td>59</td>
<td>1.3</td>
<td>2.7</td>
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<tr>
<td>3</td>
<td>2300</td>
<td>7</td>
<td>47</td>
<td>1.8</td>
<td>3.1</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>2273 ± 440</td>
<td>14 ± 10</td>
<td>54 ± 7</td>
<td>1.9 ± 0.7</td>
<td>2.7 ± 0.4</td>
</tr>
</tbody>
</table>
clusion of the sagittal sinus in cats leads to a 20% increase in the diameter of the cortical veins despite a significant elevation in ICP.30

Magnetic Resonance Venography

The 2D time of flight MR venography sequence used in the present study depicts flow because inflowing blood traveling perpendicular to the plane of the slice retains signal, while static and slow flow spins within the slice lose signal as a result of saturation effects. Turbulent flow also reduces signal because of dephasing.1 Rollins et al. studied 38 normal children younger than 2 years old (mean age 11 months) and found that 58% had a dominant transverse sinus on one side and 42% had co-dominant sinuses.35 Sixty-three percent of nondominant sinuses showed a flow gap, but no such gaps were seen in a dominant sinus. The sigmoid sinuses and jugular veins were smaller in caliber than the transverse sinuses but were always well demonstrated. In neonates, overall flow is lower than in older children, and more artificial flow gaps can be expected to occur. Among the 10 control patients in the present study, 3 were found to have co-dominant transverse sinuses and 7 had a dominant sinus. The dominant transverse sinus was always well demonstrated in the controls, but in 4 children some flow gaps were seen in the distal sagittal and sigmoid sinus regions. Presumably, the loss of signal in these segments is attributable to spin dephasing from turbulent flow. Because the dominant transverse sinus was always well demonstrated in the control patients, this segment was selected for measurement. Patients in the 3 hydrocephalus cases showed transverse sinuses that were half the size of those in controls, with the sinuses regaining a normal size after follow-up in 2 cases. Thus, hemodynamically significant stenosis seems to be in the sinus vasculature.

Flow Quantification

Normal arterial inflow varies greatly in the first few months of life because the brain is enlarging and the amount of blood per 100 g of tissue is also increasing. The expected brain weight for a normal newborn is 380 g, but by 6 months of age it is 640 g, and by 1 year of age it is 970 g.30 The average CBF measured using SPECT 133Xe does not vary as much; it is 50 ml/100 g/min at birth, rising to 57 ml/100 g/min at 6 months and 59 ml/100 g/min at 1 year.8 Thus, it is important to calculate the CBF rather than just look at the arterial inflow in the neonatal period. The method used in the present study is identical to the one suggested by Leliefeld et al.30 In 2 of the hydrocephalus cases, the CBF was very low initially (27 and 23 ml/100 g/min, similar to Leliefeld and colleagues’ findings). However, in 1 case, CBF was normal at 67 ml/100 g/min. Thus, although common, a low CBF is not a universal finding in neonatal hydrocephalus. Despite its normal CBF value, Case 1 still showed morphological evidence of venous compression. Ischemia due to venous collapse must require the ICP to limit cerebral perfusion pressure as well as to compress the veins. Perfusion pressure is the mean arterial pressure minus the ICP or the venous outflow pressure, depending on which is larger. In newborns, mean arterial pressure is heavily dependent on birth weight and gestational age. The cases in the current cohort would be expected to have a mean arterial pressure of approximately 40 mm Hg.17 The mean arterial pressure increases steadily during the 1st month of life,12 and so at some stage the CBF would be expected to improve in neonatal hydrocephalus, regardless of whether the child was treated, because the perfusion pressure would increase with the mean arterial pressure. Thus, the CBF measurement could have some early prognostic value but would become less sensitive to the effects of hydrocephalus over time. It is possible that the venous pressure elevation may also be modulated by the development of collateral venous flow over time. The better the collateral flow, the less likely that venous stenosis will be able to limit CBF.

Significant venous stenosis will limit the amount of flow through an affected vessel, increasing pressure. In time, collateral vessels will open to carry blood flow around the obstruction. An obstructed sinus would be expected to carry less of the arterial inflow, with a larger percentage of the inflow passing via the collaterals. Thus, the percentage of arterial inflow carried by the sagittal sinus would decrease as collateral veins opened. The percentage of venous return for a group of control subjects in the 1st year of life was 52% ± 8%, and this value did not vary greatly with age or head circumference.6 This value was almost identical to the 51% ± 8% found in 12 controls with a mean age of 8 ± 5 years in another study;7 suggesting the percentage of venous return varies little throughout childhood. In external hydrocephalus and in older children with communicating hydrocephalus, there was evidence of venous collapse; venous return was 37% ± 8% and 38% ± 12%, respectively,6 indicating collateral flow does occur in older age groups with hydrocephalus. Patients in Cases 2 and 3 in the present hydrocephalus cohort had an initial venous return in the normal range (50% and 45%, respectively), and the percentage of venous return did not change with improvement in the main venous channels at follow-up. This finding suggests that there was no significant collateral venous flow available in these children, which may have contributed to the limitation of CBF. In Case 1, in which there was normal CBF, the venous return was very low at 17%, suggesting that the venous collapse was hemodynamically significant to at least the level of the venous sinus flow. The difference between the 17% return found and the 50% expected suggests that collateral flow and the development of collateral flow may have moderated the venous pressures and maintained perfusion pressure and CBF in this case.

A final observation about flow regards its pulsatility. Arterial pulsation is significantly increased in neonatal hydrocephalus.9,39 The arterial pulsatility indices calculated from the MRI-demonstrated flow in this cohort revealed elevated pulsation similar to that in the Doppler data in the literature. Venous sinus pulsation occurs from compression of the cortical veins by CSF.4 There was no sinus flow pulsation at all in the initial flow studies for the 3 hydrocephalus cases despite the elevated arterial pulsation. The sagittal sinus pulsatility index obtained using transvaginal Doppler ultrasound in the last trimester showed
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a normal value of 0.39, indicating normal sinus flow is pulsatile even before birth. Complete loss of sagittal sinus pulsation has been found using this Doppler technique in 3 cases of hydrocephalus and 1 case of craniosynostosis.23 Thus, the results of MRI flow studies of the sinuses in the present cases are identical to the Doppler findings. Similarly, dampening of the venous pulsation is noted to occur in adults with idiopathic intracranial hypertension in which the elevation in venous pressure limits the ability of CSF pressure to compress the cortical veins.2 Thus, dampening of the venous pulsation in the present cohort is another indication of the elevation in venous pressure. Venous pulsation returned to normal in the 2 cases in which there was improvement in venous outflow at follow-up, suggesting that a drop in venous pressure had occurred.

The current study consists of only 3 cases, which may limit the generalizability of the findings; however, the literature indicates that there is a hemodynamically significant, reversible outflow stenosis occurring somewhere in the vascular tree in most neonates. From first principles we can predict that stenosis should occur in the collapsible segment of the vascular tree, which has the lowest intraluminal pressure, that is, within the veins. From this we can deduce that the 3 present cases are probably representative of many infants with hydrocephalus in this age group. One may ask, “Is flow-limiting stenosis the cause or the result of hydrocephalus?” The answer appears to be that compression of the veins from CSF pressure causes venous stenosis. Once initiated, however, an elevation in venous pressure will limit CSF absorption and tend to raise CSF pressure. A positive feedback loop initiated in such a manner can potentiate the CSF absorption abnormality. Interestingly, the cause of hydrocephalus in the patient in Case 3 would normally be ascribed to congenital aqueduct occlusion, and a third ventriculostomy would be expected to be curative. Further research is required to determine whether there is any prognostic value to the venous findings in neonatal hydrocephalus.

Conclusions

There is a limitation of the CBF in many neonates with hydrocephalus, suggesting a flow-limiting stenosis of the vascular tree. A review of susceptibility weighted MRI, MR venography, and flow data suggested that venous sinus collapse causes this flow-limiting stenosis. The reduction in CBF may be exaggerated in neonates because of their lower cerebral perfusion pressure and lack of venous collaterals.

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Disclosure

The author reports no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Address correspondence to: Grant Bateman, M.B.B.S., M.D., Department of Medical Imaging, John Hunter Hospital, Locked Bag 1, Newcastle Region Mail Centre, Newcastle, NSW 2310, Australia. email: grant.bateman@hnehealth.nsw.gov.au.