the diagnosis. The disparity between the negative cerebrovascular angiograms obtained preoperatively in a patient who underwent surgical evacuation of a pontine hematoma caused by a venous angioma and the postoperative angiographic study showing the malformation was attributed to compression of the abnormal veins by the adjacent clot. This and other mechanisms may be responsible for the angiographic silence observed in many cases of ruptured venous angioma, as documented in our bibliography. To us, angiographic opacification of the abnormal veins is not a requisite for diagnosing venous angioma. This lesion can be defined as a vascular malformation consisting solely of veins histologically and without histological or angiographic evidence of arterial constituents, whether or not the veins are visualized angiographically.

Regarding the CT presentation of venous angiomas, it must be noted that the CT scan may be nonspecific or even negative. The CT scan overlooked venous angiomas in 13% to 27% of the cases in different studies, either because of the size of the angioma or for other reasons. Preliminary studies indicate that MR imaging is more sensitive and specific than CT scanning for the detection of occult angiomas, but its ability to distinguish between AVM's, cavernous angiomas, and venous angiomas remains to be determined.

In brief, most venous angiomas whether ruptured or not, have a typical angiographic appearance which permits a specific diagnosis to be reached. In some cases spontaneous hemorrhage renders venous angiomas angiographically occult and thus unrecognizable unless histological examination is performed. Although the majority of venous angiomas have a silent clinical course and should be left alone, a significant proportion cause hemorrhagic accidents which may demand surgical attention. Until the actual incidence, natural history, and clinical significance of venous angiomas are better defined, these lesions must be considered in the differential diagnosis of spontaneous intracranial hemorrhage in patients of any age.

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References

Management of Posttraumatic Brain Swelling

To THE EDITOR: The recent article by Lobato, et al., concludes that posttraumatic brain swelling is not amenable to surgical treatment (Lobato RD, Sarabia R, Cordobes F, et al: Posttraumatic cerebral hemispheric swelling. Analysis of 55 cases studied with computerized tomography. J Neurosurg 68:417–423, March, 1988). This finding is based upon their experience and that of an earlier study by Cooper, et al.1 Cooper and coworkers did qualify their conclusion by stating that there may be a benefit from hemicraniectomy in those patients who initially present without brain-stem dysfunction and who later deteriorate from hemispheric swelling. In general, hemicraniectomy has not found favor among those treating posttraumatic brain swelling. However, in a single case where we performed it the result was beneficial.

Our patient was a 14-year-old boy who was struck by a truck while riding a motorcycle. He sustained multiple trauma, a cardiorespiratory arrest at the scene, and a closed head injury producing coma and decerebration with preserved pupillary light reflexes. Intracranial pressure (ICP) monitoring with a subarachnoid bolt was implemented after initial care for pneumothorax, long bone injuries, and a ruptured spleen. Within 3 days there was some eye-opening and pursuit eye movement. The ICP remained normal until the 7th day, when it became elevated and was associated with pupillary changes. There was no improvement with optimal pulmonary support and diuretic agents. The initial computerized tomography (CT) scan (Fig. 1 left) demonstrated no unilateral mass effect. However, by Day 7 CT scanning showed a low-density brain swelling in the left hemisphere (Fig. 1 center). A hemicraniectomy was agreed to by the patient's family in a desperate attempt to preserve the boy's life. We had never previously resorted to this treatment, but it seemed a viable option at the time because of the unilaterality of the mass.
lesion, the patient’s youth, and the delayed but then rapidly changing neurological status since the traumatic event. Postoperatively, there was immediate improvement in pupillary and motor signs. A CT scan on the 14th postoperative day revealed resolution of the unilateral mass effect (Fig. 1 right). The patient survived and is left with a spastic right hemiparesis and motor dysphasia. He was able to finish high school, run, and play basketball, and is presently a hospital volunteer in transportation. His present neurological morbidity is clearly acceptable to the patient and his family, considering the alternative. We adhere to the tenet of others, that hemicraniectomy has little to add to the treatment of unilateral hemispheric brain swelling in a general application, but it could be considered in the rare case.

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Reference

RESPONSE: The interesting case reported by Dr. Sisco, et al., illustrates how hemicraniectomy may be life-saving in a patient who develops cerebral hemispheric swelling several days after head injury. We would resort to this surgical procedure if faced with a similar case; however, the clinical course in this particular patient is quite uncommon. Usually, cerebral hemispheric swelling is associated with large extra-axial hematomas and occurs immediately after trauma. Patients present with a very poor neurological status (82% of our patients scored 5 points or less on the Glasgow Coma Scale and 74% had unilateral or bilateral mydriasis on admission), suggesting the association of severe primary injury or secondary brain-stem damage at the time of diagnosis. Although we did not perform hemicraniectomy in any of our patients, we share the opinion that it must be considered in severely head-injured patients who do not show brain-stem dysfunction immediately after injury and who subsequently develop neurological deterioration with otherwise uncontrollable intracranial hypertension from cerebral hemispheric swelling.

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Periventricular Vessel Changes in Hydrocephalus:
Erratum

TO THE EDITOR: Two errors were introduced in the course of publication of our article (Del Bigio MR, Bruni JE: Changes in periventricular vasculature of rabbit brain following induction of hydrocephalus and after shunting. J Neurosurg 69:115–120, July, 1988). In the Materials and Methods section, in paragraph two on page 116 the perfusion pressure should be 120 cm H2O, not 200 cm H2O. Also, the order of References 6 and 7 was changed in the bibliography but not in the text, so that all the citations in the text referring to 6 should actually be 7 and vice versa.

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Fig. 1. Left: Computerized tomography (CT) scan on admission showing no unilateral mass effect. Center: Scan on Day 7 demonstrating an area of low density, indicating brain swelling in the left hemisphere. Right: Postoperative CT scan showing that the mass effect has resolved.

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