Focal Midbrain Tumors in Children

To the Editor: I totally agree with the conclusions of Vandertop et al. (2) concerning this distinct group of brainstem tumors. We have operated with good results on several children presenting with the same clinical and imaging features (no mortality and slight morbidity). In two of these young patients, postoperative magnetic resonance imaging disclosed incomplete removal and both underwent subsequent surgery with no difficulties and an excellent long-term outcome. It is clear that surgical treatment has to be considered with this group of tumors and that total removal is to be attempted because it is the only way to definitively cure these patients.

I regret that the authors’ references did not include the cooperative study that M. Jan, Y. Guegan, and I presented at the 39th Congress of the Société de Neurochirurgie de Langue Française (1), which was held in Ljubljana, Slovenia, in June 1989. In this series of more than 130 cases, issued from 28 French-speaking neurosurgical departments, 88 were neuroepithelial tumors and 12 were benign midbrain astrocytomas (pilocytic astrocytomas). In this study, we emphasized the distinctiveness of this group and its surgical curability.

I was all the more disappointed by this omission as I had personally offered a copy to Dr. Hoffman when I visited him in Toronto in 1989. I am concerned that certain authors--or reviewers--persist in thinking that there is only one medical literature worthy of notice, the one that is published in English periodicals. I am not quite sure that this behavior is really scientific.

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Focal Dystonia Secondary to Cavernous Angioma of the Basal Ganglia: Case Report and Review of the Literature

To the Editor: Lorenzana et al. (3) present a very interesting example of focal dystonia secondary to a cavernous angioma, involving the anterior third of the putamen, the anterior limb of the internal capsule, and adjacent hemispheric white matter. Based on the location of this lesion and their review of the literature, they conclude that disruption of the striatopallidothalamic projection to premotor cortex is the cause of symptomatic dystonia. Several lines of evidence suggest that it would be more appropriate to conclude that alteration of the normal functioning of pathways within and adjacent to the basal ganglia and internal capsule is the cause of dystonia in their patient.

A careful review of the literature reveals that virtually all striatal and thalamic lesions associated with symptomatic dystonia also extend into adjacent white matter, particularly the internal capsule (3-4). In addition, many cases of secondary dystonia have been associated with lesions of the brain stem and mesencephalon (1,2). Destruction or distortion of the internal capsule could have profound effects on brain stem motor structures, such as the inferior olive, cerebellum, red nucleus, pedunculopontine nucleus, and vestibular nuclei, by virtue of their direct and indirect cortical afferent inputs, which course through the internal capsule.

Precise anatomical descriptions of lesions associated with symptomatic dystonia, based on either high-quality magnetic resonance imaging or detailed postmortem pathological examination, are important contributions to a determination of the pathophysiology of this movement disorder.

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Management of Symptomatic Chronic Extra-Axial Fluid Collections in Pediatric Patients

To the Editor: We read with great interest the article by Litofsky et al.(1). In our opinion, the most important information in this article is the clear evidence of the advantage of a shunting procedure as the initial treatment for patients with chronic extra-axial fluid collections. It is also important to restrict the number of surgical procedures as much as
possible. However, we would like to raise some questions of interest.

The authors did not precisely state what type of shunting device was used and what the opening pressure(s) of the device(s) was. Were there any differences in outcome using different devices? What are the authors' recommendations?

In some of our patients, we have noted that the extra-axial fluid had a very high protein level, up to 900 mg/dL. In those patients, a standard low-pressure Hakim pediatric shunt failed because of occlusion and we have had to use a straight catheter instead. Even this has sometimes led to problems with occlusion of the catheter. What is the authors' experience in cases with a high protein level in the fluid? Were there any patients with extremely high protein levels? The protein level was not discussed by the authors, but according to the methods section, total protein was indeed assessed in most of the patients.

A. Tommy Bergenheim
Marwan I. Hariz
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In Reply: We appreciate the comments of Bergenheim and Hariz regarding our conclusion of the advantage of subdural peritoneal shunting as the initial treatment for children with chronic subdural fluid collections. We would like to answer their questions as follows.

In almost all the cases of subdural peritoneal shunting in our study, a ventricular catheter-reservoir was placed into the subdural space through a burr hole. The distal end of the reservoir was connected to a peritoneal catheter with a metal connector; no valve was used. Contrary to the situation when a ventriculoperitoneal shunt is placed, we are not concerned about overdrainage when no valve is used in subdural peritoneal shunting. The lack of a valve should help prevent obstruction of the system by proteinaceous material.

We did not report our data concerning the protein content of the subdural fluid and its relationship to shunt malfunction because, as in many retrospective studies, the data were incomplete. To answer Bergenheim and Hariz's questions regarding protein, we reexamined the data. No subdural fluid protein data were available for 38 of the 75 shunted patients, including 4 who suffered a subdural peritoneal shunt malfunction. Of the other 37 patients, fluid protein values at the time of placement of the subdural peritoneal shunt were available. Thirty-two patients without shunt malfunctions had a mean protein level of 2245 ± 2052 mg/dL, and 5 patients with shunt malfunctions had a mean protein level of 1474 ± 1254 mg/dL. The difference here is not statistically significant by Student's t-test. Twenty-one of the 32 patients without shunt malfunction had protein levels over 1000 mg/dL. Some patients with very high protein levels, even more than 6000 mg/dL, did not have difficulties with shunt malfunction. Thus, subdural fluid protein does not appear to be related to shunt malfunction.

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J. Gordon McComb
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Deep Hypothermic Circulatory Arrest for the Management of Complex Anterior and Posterior Circulation Aneurysms

To The Editor: The recent article by Solomon et al. (1) has demonstrated nicely the usefulness of hypothermic circulatory arrest for complex aneurysms with modern cardiac bypass techniques. The morbidity and mortality in this group of patients from the cardiac surgeon's standpoint should be close to zero.

We have modified the reported technique in a small but perhaps significant way. Solomon, as well as most other authors reporting this technique in the recent literature, have performed most aneurysmal dissections in the fully arterialized state and have then gone to hypothermia and circulatory arrest for the "final aneurysmal dissection" and clipping. We have performed the majority of aneurysmal dissections with hypothermia and "low-flow" bypass (i.e., flow rates of 1 L/min) and have reserved total arrest for the final clipping, when necessary.

This modification is important in several ways. At low flow, the arterial tree, the parent vessel, and the neck and dome of the aneurysm are much more compliant and the dissection is dramatically easier than when fully arterialized. The likelihood of rupture itself is also lower because of the lower transmural pressure and the increased compliance of the vasculature. If aneurysmal rupture occurs during the dissection, the patient is already hypothermic, the pump can be immediately stopped, and the operation brought under immediate control. Finally, and perhaps most importantly, there is growing evidence that hypothermia and low flow are tolerated better than hypothermia and total circulatory arrest in terms of cerebral protection (2,4).

The last six complex giant intracranial aneurysms (three anterior circulation, three posterior circulation) treated at our institution were done with hypothermia, low flow, and circulatory arrest. The average duration of low flow was approximately 40 minutes, well within the acceptable limits (several hours) for flow of 1 L/min at 18°C. The average total arrest time was 8 minutes, again well within the published limit of 45 minutes at 18°C and significantly less than that of Solomon (22 min).

It should be noted historically that in Drake's report from 1963 (1) of 10 cases performed with hypothermia and circulatory arrest, the exposure of the aneurysm "was initiated under low rates of flow of 500-1000 cc. per min," once the core temperature reached 18°C. Because most of the dissection was done under low rates of flow, the period of total circulatory arrest was short. In 1963, it was thought that cardiopulmonary bypass and profound hypothermia added "enough additional hazard" so as not to be warranted in most cases.
We have our cardiovascular colleagues to thank for the dramatic strides made in the area of hypothermia and circulatory arrest in the last 3 decades. It is yet to be seen what the ideal technical strategy will be for its use with intracranial aneurysms.

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In Reply: We are pleased to discover that our article has stimulated other groups to investigate deep hypothermic circulatory arrest for the treatment of giant intracranial aneurysms. Kiwak's work with low-flow bypass is an interesting modification of this technique and seems to have potential application to many giant aneurysms.

Several comments seem to be in order. The reason we prefer to perform most of the dissection in the arterialized state is to avoid the necessity of dissecting while the patient is fully heparinized. The more dissection done during heparinization, the more problems with bleeding that can be expected. With low-flow bypass, it is still impossible to cut open the dome of the aneurysm. In almost all of our cases, it has been necessary to open the aneurysm to eliminate the mass and allow visualization of the opposite side of the aneurysm. Many cases have required aneurysmal endarterectomy and visualization of the lumen to perform proper clipping. We now have experience with 31 cases. In no instance has there been a complication referable to the time of arrest, which has ranged up to 60 minutes. There is no reason to suspect that 8 minutes of total arrest time will lead to fewer complications than experienced with a mean arrest time of 22 minutes.

Finally, although we certainly share Kiwak's enthusiasm for this technique, caution should be