etiology,3 the father–son relationship in our report, coupled with the reported cases of familial occurrence in subependymoma, strengthens the evidence of a genetic influence in the etiology of these unusual tumors.1–3

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References

Sequestered Fourth Ventricle

TO THE EDITOR: I read with interest the recent article by Dr. Montes and colleagues (Montes JL, Clarke DB, Farmer JP: Stereotactic transtentorial hiatus ventriculoperitoneal shunting for the sequestered fourth ventricle. Technical note. J Neurosurg 80:759–761, April, 1994). The authors presented their experience in four patients with “the sequestered fourth ventricle,” which was successfully treated using the transtentorial hiatus ventriculoperitoneal stereotactic technique. They state that it is important to avoid a direct approach from the roof of the fourth ventricle that might risk damage to the underlying brain stem. In addition, they conclude that the stereotactic supratentorial hiatus approach is a safer, more effective treatment than the traditional method (that is, the transcerebellar approach). Their idea may be derived from their experience of brainstem tumor biopsy using the stereotactic supratentorial approach. I agree with the authors that fourth ventricular shunting is achieved by means of the stereotactic supratentorial route. This technique, however, seems to be safer and more effective only when used by the authors or other specialists, and it may be very complex (requiring stereotactic apparatus) and carry potential risk for epilepsy secondary to penetrating injury to the parietal cortex. In my experience1 and according to the literature,4 fourth ventricular shunting is easily performed through the transcerebellar route because of the enlarged fourth ventricle without using a stereotactic technique. Although no standard technique for fourth ventricular shunting has been adopted to date, I do not think that the stereotactic transtentorial approach is the most popular technique.

The development of an abnormally enlarged fourth ventricle has been referred to as double-compartment hydrocephalus,2,3 isolated fourth ventricle,5,7–9 trapped fourth ventricle,6,8,10 disproportionately large communicating fourth ventricle,9 or communicating fourth ventricular hydrocephalus.1 To my knowledge, the term “the sequestered fourth ventricle” has not been used to date; many readers may wonder what kind of pathological condition is applied to this terminology.

In conclusion, stereotactic transtentorial hiatus shunting does not seem to be accepted as a standard procedure because the transcerebellar approach is easily and safely performed without using a stereotactic technique.

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References

RESPONSE: We thank Dr. Aoki for his comments on our paper on stereotactic shunting for the isolated fourth ventricle.

We do not dispute the fact that fourth ventricular shunting can be easily performed through the transcerebellar route; however, our experience and that of many of our colleagues (known through personal communications) is that often enough, with the shrinking of the fourth ventricle, the tube gets embedded in the wall of the cyst or sometimes, with the re-expansion of the brain stem, it may occasionally penetrate it.

We advocate placing the tube following the long axis of the ventricle to make it a more precise, safe, and durable procedure. We consider the use of a stereotactic apparatus an improvement in the precision of the procedure, and stereotaxis is a standard surgical technique in most neurosurgical centers. Furthermore, we do not consider the potential for epilepsy a factor in the decision to choose this approach. Most of these patients have postmeningitic
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or posthemorrhagic hydrocephalus, and we sincerely doubt that the shunt trajectory in the parietal lobe would change the already present high risk of epilepsy associated with the primary event. Finally, the risk of a brainstem injury in the transcerebellar approach, from our perspective, outweighs any potential complication from the stereotactic transparietal approach.

We agree with Dr. Aoki that the term “sequestered fourth ventricle” has not been commonly used. Nevertheless, terms such as “encysted fourth ventricle” have been used before. The term “isolated,” which we use in our article, indicates the lack of communication between the fourth ventricle, the remaining ventricular system, and subarachnoid space. Because of this, we speak of a sequestered fourth ventricle, but if that raises confusion, we apologize.

We believe that the stereotactic approach for shunting the sequestered fourth ventricle is safe and more precise than the traditional method, and that it is an alternative in the management of this group of patients.

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Reference

Does CVP Influence ICP After Brain Injury?

To THE EDITOR: We read with great interest the article by Hariri, et al. (Hariri RJ, Firlick AD, Shepard SR, et al: Traumatic brain injury, hemorrhagic shock, and fluid resuscitation: effects on intracranial pressure and brain compliance. J Neurosurg 79:421–427, September, 1993). The authors conclude that reduction in brain compliance can occur secondary to increase in central venous pressure (CVP) following resuscitation from hemorrhagic shock and head injury. They also claim that this may worsen intracranial hypertension in patients with traumatic brain injury and hemorrhagic shock. We would like to comment on these conclusions.

We have previously reported that intravenous administration of a large volume (0.2 ml/gm) of isotonic solution given over an 18-hour period after closed head injury in rats did not significantly affect neurological outcome or brain tissue specific gravity (brain edema). Moreover, we have just concluded a study that showed no significant difference in neurological outcome between control rats (head injury and no intravenous fluid administration) and head-injured rats treated with 0.25 ml/gm 0.9% saline (three times their blood volume) intravenously over a 0.5-hour period, 1 hour after head injury. Although CVP was not measured, it must have been high in rats receiving three times their blood volume over 30 minutes. Todd has stated that 19 mm Hg of hydrostatic pressure (CVP) is equivalent to 1 mOsm/kg (unpublished data). The works of Battistella, et al.; Prough, et al.; Todd, et al.; Walsh, et al.; and Zornow, et al., cited in the paper, stress the importance of osmolality over oncotic pressure or CVP as the main factor influencing brain edema. Unfortunately, the authors did not refer to either osmolality, electrolyte concentration, or brain edema, although fluid resuscitation was established with lactated Ringer’s solution (250 mOsm/kg).

Figure 1 in the article by Hariri, et al., indicates a linear correlation between CVP and sagittal sinus pressure. Increased sagittal sinus pressure increases intracranial pressure (ICP) and almost by definition decreases brain compliance (Fig. 4 in Hariri, et al.). The finding that low sagittal sinus pressure is associated with low ICP is supported by the work of Feldman, et al., who showed in head-injured patients that head elevation from 0° to 30° significantly reduced ICP while cerebral perfusion pressure (CPP), cerebral blood flow, cerebral metabolic rate of oxygen, arteriovenous difference of lactate and several other physiological parameters did not change. Head elevation, by increasing the hydrostatic gradient between the head and the heart, improves the venous return from the head and reduces the sagittal sinus pressure. However, ICP alone does not predict outcome after head injury. There is substantial evidence that a major mechanism by which elevated ICP causes brain insult is the reduction of CPP. However, the authors did not calculate the CPP. Analyzing the data presented in the paper, we have calculated the difference between baseline ICP values and the values 3 hours after head injury. The ICP increase was similar in Groups I, II, III, IV (7, 9, 7, 8 mm Hg, respectively). At the same time, the mean arterial blood pressure (MAP) in Groups I and III (CVP < 6 mm Hg) was 85 mm Hg and 90 mm Hg, respectively, while in Groups II and IV (CVP > 6 mm Hg) the MAP was 95 mm Hg and 90 mm Hg, respectively. Thus the CPP in the high CVP groups was equal to or greater than in the low CVP groups. Further analysis of the text and figures shows that 6 hours after head injury, the MAP was 82 mm Hg, 92 mm Hg, 92 mm Hg, and 100 mm Hg, and ICP was 20 mm Hg, 24 mm Hg, 24 mm Hg, and 33 mm Hg in Groups I, II, III, and IV, respectively. Therefore, the CPP was the lowest, 62 mm Hg in Group I, 68 mm Hg in Group II, 68 mm Hg in Group III, and 67 mm Hg in Group IV. Although ICP increased the most in one of the high CVP groups (Group VI), CPP was compromised the most in one of the low CVP groups (Group I). As no information is available on the clinical end point following the experiment, we believe that no conclusions concerning the effect of high versus low CVP on outcome can be drawn from this experiment.

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