from the overzealous use of some of the more rigid implants available. For this reason we have found that Teflon provides the most effective decompression because of its pliability. If compression by the implant is the proposed cause of recurrence, why is the recurrence rate so low? Drs. Ryu and Yamamoto propose an interesting alternative technique for performing microvascular decompression by coapting the vascular loop to the dura with the use of fibrin glue. We wonder, however, whether fixation of the artery with fibrin glue will provide long-term decompression of the nerve. Intuitively it would seem that continuous arterial pulsation would, over time, detether the vascular loop from the dura mater and cause recurrent compression of the nerve.

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Vascularized Pedicle Graft

TO THE EDITOR: I read with interest the report by Jensen and colleagues (Jensen R, McCutcheon IE, DeMonte F: Postoperative swelling of pericranial pedicle graft producing intracranial mass effect. Report of two cases. *J Neurosurg 91:*124–127, July, 1999). I simply wanted to add a case of my own that was as dramatic as that reported by the authors. My case involved a young man with a large esthesioneuroblastoma that was gross-totally removed by a craniofacial bifrontal resection. The large defect in the frontal fossa was covered with the usual vascularized pericranial graft, which I applied in two layers, as is my custom. The first (inferior) layer was sutured to the dura posteriorly and all around the bone defect through multiple holes. The second layer of periosteum, which was bent over posteriorly, was applied as a more superior layer and sutured all around the dura to recover the dural defect that had already been covered with a free flap of cadaveric lyophilized dura. To avoid a cosmetic defect, the bifrontal bone flap was replaced rather tightly over the orbitonasal bone piece that had been removed and replaced a separate piece. The patient did very well for 2 days and then deteriorated abruptly to the point of unresponsiveness. A computerized tomography (CT) scan showed an ill-defined hemorrhagic mass in the subfrontal region. During immediate surgical reexploration, we found an extraordinarily thickened, boggy, congested pericranial graft, which we removed completely except for the piece covering the bone defect. The patient recovered well and did not have a cerebrospinal fluid leak, although we did treat him with lumbar drainage for 5 days.

In retrospect, the problem we encountered was undoubtedly due to the fact that we replaced the bone flap too tightly, thus constricting the vascularized pericranial graft and allowing arterial blood to flow continuously into the graft, but preventing adequate venous efflux, just as postulated by these authors. Interestingly, when I discussed this case at our complication conference, my colleague, Dr. Jacques Morcos, reminded me that one of my colleagues in Minnesota had encountered an identical case. I wish I had learned from that experience rather than having to wait for my own!

My letter is simply meant to point out that the authors' two cases are not unique and that perhaps this complication occurs with some frequency. As the authors remark, it should be included in the differential diagnosis of deterioration after skull base procedures in which vascularized pedicle grafts are used to cover skull base defects. A CT scan, which produces only thick axial slices, may not always be diagnostic of this condition because the planes of the slices may fail to demonstrate the true size of the subfrontal mass. Obviously, a magnetic resonance image with coronal and sagittal slices will demonstrate this mass very adequately, but these patients can deteriorate too abruptly to allow a magnetic resonance image. The problem can easily be prevented by not replacing the bone flap so tightly as to constrict the base of the vascularized flap.

The authors should be thanked for bringing this problem to neurosurgical attention.

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RESPONSE: We would like to thank Dr. Heros for his interest in our paper and for his contribution of a third case of this unusual complication. It surely needs to be considered as a cause of postoperative deterioration in a patient who has undergone cranial base surgery in which vascularized pedicle grafts are used to cover defects. It is, however, a relatively rare complication, occurring in less than 0.5% of all our pedicle pericranial graft cases. (Amazingly, both of our cases occurred within the same week.) Although assuring that the bone flap is not replaced in a fashion that constricts the pericranial flap is mandatory, it must be kept in mind that this complication can occur simply on the basis of venous torsion alone.

Dr. Heros makes an excellent point with respect to the diagnosis of this complication. Routine axial CT scans may underestimate the size of the compressing mass. We have taken to performing a somewhat modified form of CT scanning in which the gantry is tilted such that a modified coronal scan is obtained.

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Brain Tissue Oxygenation

TO THE EDITOR: We read with interest the paper by Menzel and coworkers (Menzel M, Doppenberg EMR, Zauner A, et al: Increased inspired oxygen concentration as a factor in improved brain tissue oxygenation and tissue

lactate levels after severe human head injury. *J Neurosurg 91:*1–10, July, 1999) and we would like to add some comments.

The authors suggest that the increase in brain tissue oxygen tension ($ptiO_2$) obtained by ventilation with pure O_2 could correspond to an effective increase in O_2 supply to the brain tissue itself. This improvement in $ptiO_2$ would cause a shift from anaerobic to aerobic glucose metabolism, as testified by the reduction in lactate concentration in the cerebral extracellular fluid (ECF). They therefore suggest that ventilation with 100% O_2 should be considered at least for the first 6 to 8 hours after any severe head injury. We are not convinced that the results fully support the main conclusions.

On the basis of the physiology of O_2 transport and delivery, O_2 supply is the product of arterial blood O_2 content and blood flow (cerebral blood flow [CBF] in this case). The increase in the O_2 dissolved fraction caused by arterial hyperoxia has a limited impact on O_2 content, which is due mainly to the fraction bound to hemoglobin. Raising PaO_2 from 120 to 440 mm Hg leads to an increase in the O_2 dissolved fraction of 0.84 ml/dl (being $0.003 \times PaO_2$), which is almost negligible. The second factor affecting O_2 delivery is CBF, which is not easily measured at bedside. However, it has been reported that arterial hyperoxia may cause cerebral vasoconstriction, hence a decrease in CBF. It follows that O_2 brain delivery can hardly be increased substantially by manipulating the inspired O_2 fraction. We could speculate that even a small increase in O_2 content may make a difference in cases of severe tissue hypoxia. However, we must differentiate between O_2 tension, which responds impressively to changes in inspired O_2 fraction, and O_2 content and delivery, which change by approximately 3% in response to hyperoxia, but are the key factors in mitochondrial function.

Menzel, et al., interpret the reduction in ECF lactate as indicating an improvement in Krebs cycle function following hyperoxia. Extracellular fluid lactate alone, however, is not always a reliable index of cerebral aerobic or anaerobic metabolism.[1] Lactate production depends on substrate availability, that is, ECF glucose. In cerebral ischemia, reducing or raising the substrate supply by varying the glucose concentration reportedly alters lactate concentration.[2]

Of the two groups described, patients with hyperoxia had significantly lower ECF glucose and ECF lactate values than normoxic patients. It is thus not clear whether the decrease in ECF lactate is due to an improvement in aerobic glucose metabolism or simply to lower availability of glucose. Perhaps the ratio between lactate and pyruvate should be used rather than lactate alone. In addition, the fluctuation in ECF lactate may correspond to fluctuations in blood lactate that were not reported.

The study by Menzel, et al., was well designed, but we believe that the results are not really strong enough to support the conclusion that arterial hyperoxia substantially improves O_2 delivery and O_2 utilization in head injury. The authors are certainly right in pointing out that further validation of the study is needed.

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RESPONSE: We are pleased that our article has stimulated interest and discussion. Specifically, we would like to thank Drs. Rossi and Stocchetti for their scholarly comments on our data. They correctly point out that increasing inspired O_2 content will increase tissue O_2 tension but have minimal effect on increasing tissue O_2 content. We were careful to emphasize this fact in our article. Nevertheless, our data are clear: increasing inspired O_2 content resulted in a reproducible and consistent reduction in dialysate lactate in our patients.

We have documented this same phenomenon in animal studies. We have also shown that an increase in dialysate lactate is present in over two thirds of severely head injured patients, and it is frequently elevated even when CBF is normal, suggesting a nonischemic causation.[2] Moreover, there is a close correlation between increase in dialysate glutamate and dialysate lactate in these severely head injured patients.[1] We therefore speculate that our clinical data support the laboratory studies of Pellerin, et al.,[3] who have shown that glutamatergic activation of glycolytic metabolism by astrocytes produces lactate in large quantities, which is then made available via specific neuronal transporter systems for neurons to use as a substrate for metabolism.

There are several lines of evidence supporting the hypothesis that mitochondrial metabolism is qualitatively and quantitatively impaired after traumatic brain injury. This impairment may be mediated by opening of the mitochondrial pore transition. We have attempted to rationalize the data presented in our article by hypothesizing that increased O_2 tension rather than O_2 content provides a more profound O_2 gradient at the mitochondrion, which may enhance mitochondrial function and thus allow lactate to be more rapidly consumed, leading to a reduction in dialysate lactate as seen in our patients.

To test this hypothesis, we used the "cartesian diver" microrespirometry method to measure O_2 consumption (an indicator of mitochondrial Krebs cycle metabolism) in the rat brain after fluid-percussion injury. A 25 to 35% reduction in O_2 consumption was seen 6 hours after fluid-percussion injury. When fluid percussion–injured rats were given 80% inspired O_2 together with an increase in lactate, a substantial increase in O_2 consumption was seen (unpublished data). This suggests that augmentation of substrate delivery by increasing the O_2 tension in the tissue, together with a high lactate content as found in human severe head injury, will result in better mitochondrial function.

An important issue raised by Drs. Rossi and Stochetti is whether mitochondrial function will be changed more by changes in O_2 tension or O_2 content. We are unaware of any study in the literature that clarifies this point.

We would strongly contest the statement made in the penultimate paragraph of Drs. Rossi and Stochetti's letter.

They state "it is thus not clear whether the decrease in ECF lactate is due to an improvement in anaerobic glucose metabolism or simply to lower availability of glucose." Glucose in plasma and glucose in dialysate did not significantly differ between our two groups of patients, and as outlined earlier, there is strong evidence to suggest that lactate utilization by neurons for aerobic metabolism occurs independent of the glucose content in tissue, via mechanisms we have previously outlined. For this reason, we agree that the lactate/pyruvate ratio is clearly of importance in determining tissue ischemia.

Finally, we agree with Drs. Rossi and Stochetti that our results are provocative and demand further studies for their verification. We stated this repeatedly in our article. Potentially, however, the implications of these studies are important: augmentation of inspired O_2 fraction is a simple measure, and our data suggest that it may possibly benefit the injured brain. Studies are ongoing in our institution to validate these early observations, and we invite Drs. Rossi and Stochetti to assist in this endeavor in their own institution.

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Endoscopy for Cysts

TO THE EDITOR: The report by Dr. Hayashi and coworkers (Hayashi N, Endo S, Tsukamoto E, et al: Endoscopic ventriculocystocisternostomy of a quadrigeminal cistern arachnoid cyst. Case report. *J Neurosurg 90:* 1125–1128, June, 1999) demonstrates the suitability of the endoscopic precoronal/transforaminal/transventricular approach for arachnoid cysts bulging into the posterior third ventricle. The authors' proposal, however, is not novel to us: in our 4-year experience with neuroendoscopy we have already used it twice for quadrigeminal plate (one case) and supracerebellar (one case) cysts. Exploration of the posterior third ventricle is easily and routinely performed by means of our steerable/flexible endoscope (the same model used by our Japanese colleagues) and we therefore found it natural to choose such a route to treat those lesions. In 80 neuroendoscopic procedures performed to date we have always used the same 4-mm diameter instrument and found it extremely versatile: its maneuverability is such that we have sometimes been able, by means of a ventriculostomy, to reach as far as the first cervical roots. On the other hand, other authors have advocated the apparently more straightforward occipital burr hole approach using either a pure or endoscope-controlled microsurgical technique.[1,2]

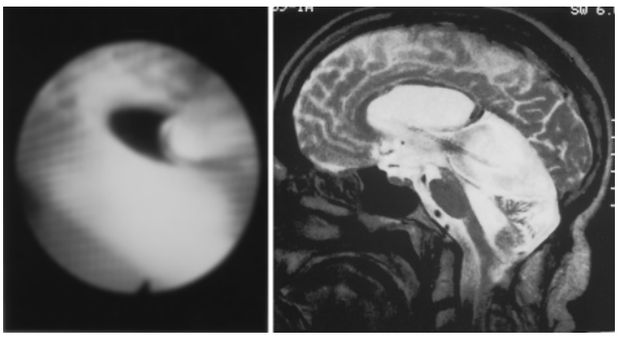

FIG. 1. *Left:* Endoscopic view. The apparently patent ventriculocystostomy is actually occluded by a transparent membrane, which is bluntly perforated with a Fogarty catheter. *Right:* Postoperative T_2-weighted midsagittal MR images demonstrating good stent positioning. The heterogeneous signal from the cyst content suggests flow within the cavity.

Our first case conforms closely to that reported by Hayashi, et al.: a 31-year-old woman presented with intracranial hypertension and Parinaud's syndrome. The quadrigeminal cyst was punctured and a ventriculostomy was performed. At 26-month follow up both clinical and imaging results are satisfactory.

The second case has a much longer and more complicated clinical history. A 49-year-old woman suffering from ataxia and intermittent headaches had undergone suboccipital craniotomy for a large supracerebellar arachnoid cyst 8 years earlier, with no benefit. Precoronal endoscopic exploration and ventriculostomy were successfully performed, although scarring from previous open surgery hindered fenestration of the opposite wall. The patient improved only transiently and control magnetic resonance (MR) imaging disclosed a functioning ventriculostomy but an unchanged cyst. Two years later a reexploration confirmed patency of the ventriculostomy, whereas the ventriculocystostomy was occluded by a transparent membrane (Fig. 1 *left*), which was again perforated. A stent was placed to ensure a permanent communication. Control MR imaging disclosed good stent positioning and cerebrospinal fluid flow within the cavity (Fig. 1 *right*). Although no significant changes in size were detected, the patient's clinical picture improved steadily.

Endoscopic surgery for arachnoid cysts is an appealing treatment option, but results are still controversial. Although a high success rate is reported for intraventricular, posterior fossa, and suprasellar cysts, only half of patients harboring arachnoid cysts improve.[1] Reclosure has been repeatedly described[1,2] and observed by us as well. We believe that it may be a consequence of cyst shrinkage; therefore, double perforation into ventricles and cisterns whenever feasible, along with more widespread use of internal stenting,[2] could promote further improvement in the already promising results reported to date.

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