RESPONSE: We appreciate Dr. Piek's thoughtful comments. Some of the issues he raises can be easily addressed, while others go beyond the scope or intent of our paper. We reported our initial experience with a device that can measure intracranial pressure (ICP) from the subdural space, subarachnoid space, ventricles, or the brain parenchyma itself. Since accurate measurement of ICP was at issue, we elected to compare this device to the gold standard of ICP measurement. Our device is accurate and correlates significantly with ICP measured by ventricular catheter (ventricular fluid pressure, VFP). Technical data about the transducer have recently been published, but were not available at the time we submitted the manuscript. A summary is provided now, in Table 1.

We do not routinely perform bacteriological analysis of implanted devices upon removal of the device. However, no infections were encountered that were attributable to the fiberoptic probe. We have now accumulated experience with over 200 patients in whom this device has been used and have not seen either an infectious or a hemorrhagic complication.

Clinical experience with BTP measurements in man is limited. However, studies in various animal models using mostly devices that incorporate wicks have indicated that BTP may be higher, lower, or essentially the same as VFP or pressures measured from the cisterna magna in uninjured anesthetized animals. However, many of these reports suggest that in the injured or "edematous" brain, BTP was higher than VFP. Our clinical studies with the fiberoptic probe were conducted in severely head-injured adults and children in whom ICP monitoring was clinically indicated. It is likely, therefore, that we were recording BTP from "injured" brain. Thus, the finding that BTP was slightly higher than VFP was not unexpected and seemed to be supported by the available experimental literature.

TABLE 1
Summary of technical data on the brain tissue pressure monitor

<table>
<thead>
<tr>
<th>Features of Device</th>
<th>Specifications</th>
</tr>
</thead>
<tbody>
<tr>
<td>catheter size</td>
<td>no. 4 French</td>
</tr>
<tr>
<td>transducer type</td>
<td>fiberoptic</td>
</tr>
<tr>
<td>frequency response (system)</td>
<td>-3 dB at 33 Hz</td>
</tr>
<tr>
<td>measurement range (system)</td>
<td>-10 to +250 mm Hg</td>
</tr>
<tr>
<td>zero drift (system)</td>
<td>maximum of 3 mm Hg/24 hrs</td>
</tr>
<tr>
<td>temperature range (system)</td>
<td>maximum of 3 mm Hg over temperature range of 22°C to 38°C</td>
</tr>
<tr>
<td>linearity and hysteresis (system)</td>
<td>± 2 mm Hg or better</td>
</tr>
<tr>
<td></td>
<td>± 6% of reading or better</td>
</tr>
<tr>
<td></td>
<td>± 7% of reading or better atmosphere</td>
</tr>
<tr>
<td></td>
<td>~700 to 1250 mm Hg</td>
</tr>
</tbody>
</table>

Finally, it is important to reemphasize that, to our knowledge, the clinical measurement of BTP has not been demonstrated to be of any value other than for providing knowledge of ICP. We use this fiberoptic device as one method to measure ICP. Clearly, much more information about the characteristics of BTP under various conditions is necessary, and therefore we look forward to Dr. Piek's forthcoming report.

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References


Staged Treatment of AVM’s

To THE EDITOR: Regarding the article by Spetzler, et al. (Spetzler RF, Martin NA, Carter LP, et al: Surgical management of large AVM’s by staged embolization and operative excision. J Neurosurg 67:17–28, July, 1987), the authors are certainly to be congratulated on their skill and innovative techniques. Catheterizing and embolizing a surface vessel associated with an arteriovenous malformation (AVM) is moderately difficult, but performing the same procedure on deep feeders,
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such as Spetzler, et al., describe with the posterior cerebral artery, is a technical masterpiece. However, I am pleased that they state in their last sentence that the staged or “stepwise throttling of very large AVM’s afforded by embolization combined with feeding artery ligation appears to minimize the risks” of normal perfusion pressure breakthrough (NPPB). The important word is “appears.”

Our own experience has been that complete removal in one stage has eliminated the so-called “NPPB.” This is a very convenient term with no real physiological basis. This phenomenon does not happen anywhere else in the body if a shunt is obliterated; in fact, circulation is always improved after obliterating the diverting features of a shunt. I suppose the most dramatic example is that of a ductus arteriosus. The sequence of events considered to be the result of NPPB occurs in other forms of intracranial surgery also. We recognize that these events may be due to 1) excessive retraction, 2) a long period of retraction or occlusion of too large or too many arteries supplying normal tissue, or 3) too large or too many veins draining normal tissue.

In our own series, we were astonished to find how often there was residual AVM when we thought that the lesion was completely removed. By using intraoperative serial angiography, we were no longer fooled in such cases, since any residual AVM was immediately evident, as was its exact location. It should be stressed that single-film intraoperative angiography can often be misleading in patients with AVM’s, whereas a series of three or more films at 1-second intervals will demonstrate flow through to the venous stage and reveal the presence of a residual fistula.

I would also seriously question the advantage of adding a barbiturate to the already injured brain.

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Reference

RESPONSE: Dr. Parkinson’s thoughtful letter raises several points that deserve a reply. He states that, in his own experience, single-step obliteration of arteriovenous malformations (AVM’s) “has eliminated the so-called ‘NPPB’” (normal perfusion pressure breakthrough). It is important to remember that I have repeatedly stated in this article and previously that the risk of NPPB is small, that it occurs infrequently, and in my experience is limited to large and high-flow AVM’s that demonstrate poor normal circulation on angiography and that frequently present clinically with progressive or fluctuating deficits. In reviewing Dr. Parkinson’s own series of 100 AVM’s, I note that the only patient with a true high-flow AVM pictured angiographically (although it was small) died of undetermined cause after the lesion was obliterated “quickly and simply.” Of 10 surgical deaths, three were due to postoperative intracerebral hematoma. We are not given any information as to whether residual AVM was present in these patients — particularly pertinent since a strong argument is made for intraoperative angiography — or whether any of these patients might indeed have had NPPB.

Dr. Parkinson states that NPPB “is a very convenient term with no real physiological basis.” That is simply not true. It reminds me of the arguments 15 years ago about whether vasospasm was a true clinical entity, a time when very knowledgeable and superb neurosurgeons flatly stated that they never saw it. There is no complexity to NPPB. With the brain making up 2% of body weight yet receiving 15% of cardiac output, utilizing 20% of inspired oxygen, and basically being dependent on the oxidative metabolism of glucose, the sensitivity of the brain to vascular insults is not surprising.

It has been clearly demonstrated that, in patients with hypertension, the autoregulatory control curve is shifted to the right, and undoubtedly, in chronically hypertensive patients, the curve is shifted to the left. The brain’s homeostatic mechanisms adjust to provide normal flows based on metabolic demand. Obviously, any patient’s blood pressure can be raised past the upper boundary of autoregulatory control with subsequent edema and hemorrhage, as occurs in a hypertensive crisis.

Also established is that areas around large AVM’s are perfused with very low pressure because of the steal through low-resistance AVM vessels. There can be no doubt that when pressures in feeding vessels supplying surrounding brain are less than 25% of systemic pressure and that when this perfusion is further compromised by high venous pressures (again, from the shunt through the AVM), the poorly perfused brain is relatively ischemic. Blood flow measurements by either xenon-133 (Xe) or Xe-enhanced computerized tomography have shown ischemic areas around high-flow AVM’s, autoregulatory control in vessels perfusing a large AVM has been shown to be lost, and direct arterial and venous pressure measurements have uniquely demonstrated low arterial perfusion pressures and high venous resistance pressures, leading to the unequivocal conclusion that around a high-flow AVM there is relatively ischemic brain tissue. It has also been demonstrated that the sudden obliteration of these high-flow shunts, whether by surgery or aggressive embolization, has resulted in surrounding edema or hemorrhagic breakthrough. Rather than being nonphysiological, NPPB is based on everything we know that controls the fine-tuned mechanism which generally keeps blood flow constant throughout a range of blood pressure variations while accommodating to changes in local flow to provide varying metabolic needs.

Dr. Parkinson notes that similar findings occur in