EMPORARY vessel occlusion during aneurysm surgery and intracranial mass lesions are examples of conditions in which cerebral metabolism is compromised as a result of decreased availability of glucose and O2 to the brain tissue.

Temporary occlusion of a parent vessel during aneurysm surgery is an accepted method of facilitating dissection and clipping of both ruptured and unruptured aneurysms.5,13,20 Many authors assert that this method reduces the risk of intraoperative bleeding from the aneurysm, which decreases the morbidity and mortality rates associated with the surgery.17 Although temporary occlusion is now frequently used, it is not without risk.21 The major problem contributing to the risk is a lack of knowledge about how well the brain tissue supplied by the temporarily occluded artery tolerates this interruption of O2 and glucose delivery. The safe temporary occlusion time, as described by many authors, varies from a few minutes to 1.5 hours.1,5,8 This wide range of safe occlusion times depends on several factors, such as which artery has been occluded, whether the aneurysm has ruptured, the age and preexisting vascular status of the patient, and which endpoints have been used to evaluate the safety of the procedure.21

Recently, new methods to assess oxygen tension (PO2), carbon dioxide tension (PCO2), and pH have become available for continuous measurement of these parameters in brain tissue and are becoming increasingly popular.25–27 A substrate (O2) and a product (CO2) of mitochondrial metabolism can be measured directly. We applied this method to understand more fully the effects of temporary vessel occlusion in both ruptured and unruptured aneurysms and to determine a safe occlusion time.

Intraoperative monitoring of substrate delivery during aneurysm and hematoma surgery: initial experience in 16 patients

EGON M. R. DOPPENBERG, M.D., JOE C. WATSON, M.D., WILLIAM C. BROADDUS, M.D., PH.D., KATHRYN L. HOLLOWAY, M.D., HAROLD F. YOUNG, M.D., AND ROSS BULLOCK, M.D., PH.D.

Division of Neurosurgery, Medical College of Virginia, Virginia Commonwealth University, Richmond, Virginia

The effects of proximal occlusion of the parent artery during aneurysm surgery in humans are not fully understood, although this method is widely used. The reduction in substrate that can be tolerated by normal and subarachnoid hemorrhage (SAH)–affected brain is unknown. Therefore, the authors measured brain oxygen tension (brain PO2), carbon dioxide tension (brain PCO2), pH, and hemoglobin oxygen (HbO2) saturation before and after temporary occlusion in 12 patients with aneurysms. The effect of removal of a traumatic intracranial hematoma on cerebral oxygenation was also studied in four severely head injured patients.

A multiparameter sensor was placed in the cortex of interest and locked by means of a specially designed skull bolt. The mean arterial blood pressure, inspired O2 fraction, and end-tidal PCO2 were analyzed. Brain PO2 and HbO2 saturation data were collected every 10 seconds. Descriptive and nonparametric analyses were used to analyze the data.

A wide range in baseline PO2 was seen, although a decrease from baseline in brain PO2 was found in all patients. During temporary occlusion, brain PO2 in patients with unruptured aneurysm (seven patients) dropped significantly, from 60 ± 31 to 27 ± 17 mm Hg (p < 0.05). In the SAH group (five patients), the brain PO2 dropped from 106 ± 74 to 87 ± 73 mm Hg (not significant). Removal of intracranial hematomas in four severely head injured patients resulted in a significant increase in brain PO2, from 13 ± 9 to 34 ± 13 mm Hg (p < 0.05).

The duration of safe temporary occlusion could not be determined from this group of patients, because none developed postoperative deterioration in their neurological status. However, the data indicate that this technique is useful to detect changes in substrate delivery during intraoperative maneuvers. This study also reemphasizes the need for emergency removal of intracranial hematomas to improve substrate delivery in severely head injured patients.

KEY WORDS • temporary arterial occlusion • aneurysm • intracranial hematoma • brain oxygen
been demonstrated directly in humans. We therefore measured \( \text{PO}_2 \), \( \text{PCO}_2 \), and \( \text{pH} \) in brain tissue during removal of intracranial hematomas in four severely head injured patients.

**Clinical Material and Methods**

These studies were approved by the Committee for Conduct of Human Research at the Virginia Commonwealth University. Informed consent was obtained from patients or their families.

**Patient Population**

In total, 16 patients were studied. Five patients suffered a subarachnoid hemorrhage (SAH) due to a ruptured aneurysm and underwent surgical clipping of the aneurysm. Seven patients underwent elective surgery for clipping of an unruptured aneurysm. The mean age for the 12 patients with aneurysms was 48 \( \pm \) 9 years. All patients with aneurysms received a bolus of 500 ml of mannitol immediately before the craniotomy and were maintained in a normothermic and normotensive state. Cerebrospinal fluid was drained via either a lumbar drain or a ventriculostomy catheter. An additional four patients underwent surgery for evacuation of an acute traumatic intracranial hematoma.

**Multiparametric Sensor**

A multiparameter, minimally invasive sensor (Paratrend 7; Biomedical Sensors, Malvern, PA) was used to obtain continuous measurements of brain tissue \( \text{PO}_2 \), \( \text{PO}_2 \) (brain \( \text{PO}_2 \)), brain tissue \( \text{PCO}_2 \), \( \text{PCO}_2 \) (brain \( \text{PCO}_2 \)), brain tissue \( \text{pH} \) (brain \( \text{pH} \)), and brain hemoglobin saturation (brain \( \text{HbO}_2 \)). The sensor, originally developed for radial artery applications, was supplied as a sterile, single-use, disposable unit. The device consisted of two modified optical fibers for \( \text{pH} \) and \( \text{CO}_2 \) measurements and a miniaturized Clark electrode for \( \text{O}_2 \) measurement. Before placement in the patient, the sensor was calibrated by using sterile precision gases bubbled in sequence through the tonometer with microprocessor control. The accuracy and precision of the sensor have been validated both in vitro and in vivo in our previous studies prior to intracranial use in humans.\(^{25–27}\) The sensor data were digitally transferred from the host computer to a Macintosh personal computer (Apple Computer, Inc., Cupertino, CA) at intervals of 10 seconds. The time of insertion of the probe depended on the procedure. In the four cases of traumatic intracranial hematoma, the sensor was placed as rapidly as possible by one surgeon, while a second surgeon prepared the scalp and bone for craniotomy. In all other cases the sensor was placed under direct vision in the region of interest after opening of the dura.

A specially designed stainless-steel Luer lock with a low-profile threaded bolt was screwed into a 3-mm skull hole that had been drilled adjacent to the craniotomy site (Midas Rex Pneumatic Tools, Fort Worth, TX). After puncturing the dura and pia using a blunt-tipped stainless-steel stylet, the sensor was inserted into the cortex and secured to the Luer lock of the bolt to achieve optimum fixation (Figs. 1 and 2). The time taken for sensor placement never exceeded 5 minutes.

**Location of the Sensor**

In patients undergoing aneurysm surgery, the sensor was placed in the cortex within the expected distribution area of the parent artery carrying the aneurysm. In patients with an acute intracranial hematoma, the probe was placed “blind” in tissue adjacent to the hematoma.
Intraoperative measurement of cerebral metabolites

Data Collection and Analysis

In patients with an aneurysm, data summated for the 5-minute period immediately prior to proximal occlusion were compared with the lowest measured value during temporary occlusion. When an artery was temporarily occluded more than once in a patient, only the first occlusion period was used in the data analysis.

In patients with a hematoma, the mean values during the first 5 minutes of the procedure were compared with the mean of the last 5 minutes after hematoma removal. Data obtained in the patient with the epidural hematoma were averaged over a 2-minute period because of the urgency of the procedure. Descriptive statistics and non-parametric tests (Spearman’s rank test, Mann–Whitney U-test) were used to analyze the data.

The following physiological parameters were measured: inspired oxygen fraction (FiO2), end-tidal (ET) PCO2, mean arterial blood pressure, and arterial blood gas levels (to validate the ETPCO2). These data were compared for the study periods and analyzed for significant changes.

Proximal Occlusion

Brief proximal occlusion was used during aneurysm surgery in all 12 patients. In 10 patients this was achieved by placement of a temporary clip on the parent artery, as close as possible to the aneurysm. In one patient undergoing elective surgery for clipping of a large ophthalmic aneurysm, the internal carotid artery (ICA) was occluded by inflation of a balloon catheter. In another patient, temporary occlusion was achieved by snaring the ICA using a Rimell tourniquet applied at the neck during dissection of a giant ophthalmic aneurysm.

Results

Measured Parameters

In all patients, stable measurements were obtained prior to the study period to avoid artifacts caused by probe insertion. In general, stability was reached after approximately 15 to 30 minutes and was judged by the presence of stable baseline values for 5 minutes. Changes were seen within 30 seconds after temporary clip application. No patient undergoing surgery for clipping of an aneurysm suffered a new neurological deficit postoperatively.

When comparing the data obtained during the periods studied, no significant differences were found in mean arterial blood pressure, FiO2, and ETPCO2 in patients undergoing surgery for an aneurysm. In the group with traumatic intracranial hematomas, the mean ETPCO2 increased during the procedure, from 23 ± 6 to 29 ± 8 mm Hg. This was because of normalization of the respiratory parameters after hematoma decompression.

Unruptured Aneurysms. In the seven patients undergoing elective surgery for an unruptured aneurysm, the mean temporary occlusion time (± standard deviation [SD]) was 6 ± 2.1 minutes (range 3.5–8.9 minutes). During temporary clip occlusion of the parent artery the brain PO2 dropped significantly, from 60 ± 31 to 27 ± 17 mm Hg (p < 0.05). The brain PCO2 increased from 46 ± 25 mm Hg (not significant), whereas the brain pH dropped significantly, from 7.2 ± 0.1 to 6.96 ± 0.29 (p < 0.05), and the brain HbO2 fell from 77 ± 18 to 33 ± 21% (p < 0.05). Table 1 shows the measured parameters before and during temporary occlusion and their percentage change.

A significant positive correlation was found when the baseline brain PO2 values were correlated with baseline PaO2 (r = 0.87, p < 0.05). No similar correlation could be found for PCO2, or pH (Table 2, Fig. 3).

Ruptured Aneurysms. All five patients underwent surgery within 12 hours after aneurysmal SAH. In this group of patients, the average temporary occlusion time was 6.2 ± 1.6 minutes (range 3.7–8 minutes). The mean brain PO2 dropped during temporary clip occlusion, from 106 ± 74 to 87 ± 73 mm Hg. The brain PCO2 increased from 60 ± 50 to 64 ± 51 mm Hg, whereas the brain pH dropped from 7.03 ± 0.47 to 6.95 ± 0.55. The brain HbO2 saturation fell from 76 ± 41 to 68 ± 40%. Table 3 shows the measured parameters before and during temporary occlusion and their percentage change, together with the Hunt and Hess grade12 for each patient. In this group the brain PO2, brain PCO2, and brain pH did not correlate with the same arterial parameters (Table 2).

### TABLE 1
Comparison of brain PO2, PCO2, pH, and HbO2 in seven patients before and during temporary intraoperative occlusion of a major parent vessel during dissection of unruptured aneurysms

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Parent Artery</th>
<th>Before Occlusion</th>
<th>During Occlusion (%) change</th>
<th>Percent</th>
<th>Before Occlusion</th>
<th>During Occlusion (%) change</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>mm Hg</td>
<td>PCO2</td>
<td>pH</td>
<td>HbO2</td>
<td>mm Hg</td>
<td>PCO2</td>
</tr>
<tr>
<td>1</td>
<td>MCA</td>
<td>25</td>
<td>39</td>
<td>7.21</td>
<td>96</td>
<td>44 (68)</td>
<td>7.21</td>
</tr>
<tr>
<td>2</td>
<td>ACoA</td>
<td>32</td>
<td>41</td>
<td>7.19</td>
<td>58</td>
<td>8 (75)</td>
<td>66 (61)</td>
</tr>
<tr>
<td>3</td>
<td>MCA</td>
<td>45</td>
<td>56</td>
<td>7.22</td>
<td>73</td>
<td>21 (53)</td>
<td>73 (30)</td>
</tr>
<tr>
<td>4</td>
<td>MCA</td>
<td>49</td>
<td>39</td>
<td>7.24</td>
<td>76</td>
<td>24 (51)</td>
<td>40 (3)</td>
</tr>
<tr>
<td>5</td>
<td>MCA</td>
<td>67</td>
<td>48</td>
<td>7.24</td>
<td>90</td>
<td>50 (25)</td>
<td>100 (108)</td>
</tr>
<tr>
<td>6</td>
<td>OA</td>
<td>98</td>
<td>43</td>
<td>7.27</td>
<td>97</td>
<td>34 (65)</td>
<td>49 (14)</td>
</tr>
<tr>
<td>7</td>
<td>MCA</td>
<td>104</td>
<td>54</td>
<td>7.04</td>
<td>96</td>
<td>44 (58)</td>
<td>95 (76)</td>
</tr>
<tr>
<td>mean ± SD</td>
<td></td>
<td>60 ± 31</td>
<td>46 ± 7</td>
<td>7.2 ± 0.1</td>
<td>77 ± 18</td>
<td>27 ± 17</td>
<td>66 ± 25</td>
</tr>
</tbody>
</table>

* ACoA = anterior communicating artery; OA = ophthalmic artery.
† p < 0.05.
Reperfusion Time. The time was measured between removal of the temporary clip until brain PO2 reached its baseline value. The mean recovery time for the entire group (12 patients) was 6.6 ± 2.8 minutes (range 1.8–9.8 minutes). No significant difference was found between recovery times in patients with SAH and those with unruptured aneurysms. Also, no correlation was found between the duration of temporary clip occlusion and the recovery time.

Intracranial Hematomas. Table 4 shows the mean ± SD of the measured parameters for the period immediately before craniotomy compared with the measurements obtained at the end of the procedure. The initial mean brain PO2 was extremely low in the group with hematomas in situ: 13 ± 9 mm Hg. It increased significantly, to 34 ± 13 mm Hg, with hematoma removal (p < 0.05). The brain PCO2 increased from 47 ± 5 to 49 ± 3 mm Hg. The brain pH increased from 7.10 ± 0.29 to 7.16 ± 0.04, and the brain HbO2 saturation increased from 23 ± 22 to 58 ± 11%.

Illustrative Cases

Case 7 (Unruptured Aneurysm)

This 64-year-old woman was admitted for elective clipping of an unruptured 8-mm aneurysm arising from the right middle cerebral artery (MCA). She had suffered an SAH caused by rupture of a left anterior communicating artery (ACoA) aneurysm 6 months before. After a pterional craniotomy was performed, the multiparameter sensor was placed at the junction of the frontal lobe and the superior temporal gyrus and secured via the bolt. Proximal control was achieved by means of a temporary clip placed on the MCA during the final stages of dissection around the aneurysm. The temporary clip was applied a total of four times because of dense adhesions between MCA branches and the aneurysm. Figure 4 shows the changes in brain PO2, brain PCO2, and brain pH caused by temporary occlusion. Baseline brain PO2 for this patient was 104 mm Hg. During the first MCA occlusion period, the brain PO2 dropped to 44 mm Hg (58% reduction). At the same time brain PCO2 increased 76%, from 54 to 95 mm Hg, whereas the brain pH dropped from 7.04 to 6.68. Brain HbO2 was 96% immediately before MCA occlusion and 47% after 5 minutes of occlusion.
Monitoring of the brain gas levels and pH guided the clip occlusion, so as to avoid subthreshold values, and allowed us to wait for sufficient recovery of substrate delivery before replacing the temporary clip. After aneurysm clipping, the patient recovered with no neurological deficit.

Case 14 (Epidural and Acute Subdural Hematoma)

This 26-year-old man, who had been involved in a high-speed motor vehicle accident, was brought to the emergency room of the Medical College of Virginia with a Glasgow Coma Scale score of 4 after being intubated in the field. An emergency computerized tomography scan revealed a large left epidural hematoma (EDH) and a subdural hematoma (SDH) with massive shift. During the computerized tomography scan, both pupils became fixed and dilated. The patient was taken to the operating room for emergency craniotomy. Prior to opening the dura, the multiparameter sensor was placed in the frontal opercular field, which makes its current configuration less user-friendly in the operating room. Because of the flexibility of the probe, we experienced kinking of the sensor during insertion in one patient. This caused a sensor malfunction because of permanent damage to the fiberoptic system and necessitated a new sensor.

Because of the specially designed bolt, it is possible to leave the probe in place postoperatively to detect early effects of vasospasm after SAH and to monitor the efficacy of pharmacological interventions designed to enhance substrate delivery or to reduce vasospasm. However, these data need to be validated by other techniques such as Doppler flow measurements and CBF.

**Logistical Factors and Reliability**

Despite the fact that the sensor is supplied sterile, its calibration is a nonsterile procedure, unless appropriate precautions are taken. The sensor itself remains sterile in the calibration chamber; however, the remainder of the unit must be handled using sterile precautions. It must be wrapped in a sterile fashion for passage into the operative field, which makes its current configuration less user-friendly in the operating room. Because of the flexibility of the probe, we experienced kinking of the sensor during insertion in one patient. This caused a sensor malfunction because of permanent damage to the fiberoptic system and necessitated a new sensor.

Because of the specially designed bolt, it is possible to leave the probe in place postoperatively to detect early effects of vasospasm after SAH and to monitor the efficacy of pharmacological interventions designed to enhance substrate delivery or to reduce vasospasm. However, these data need to be validated by other techniques such as Doppler flow measurements and CBF.

**Aneurysms: Baseline Values**

A marked difference in baseline values was seen between the patients with ruptured and unruptured aneurysms. Surprisingly, for the patients with SAH, brain PO$_2$ was much higher (mean 106 compared with 60 mm Hg). These differences indicate that hyperemia was probably present in the SAH group, and this agrees with previous studies. However, the higher PaO$_2$ in the SAH group could also contribute to this difference. Previously, we

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**Table 4**

Comparison of brain PO$_2$, PCO$_2$, pH, and HbO$_2$ before and after removal of traumatic hematoma

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Type of Hematoma</th>
<th>Before Craniotomy</th>
<th>During Craniotomy</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>mm Hg</td>
<td>mm Hg</td>
<td>mm Hg</td>
</tr>
<tr>
<td>13</td>
<td>ICH</td>
<td>5 ± 1</td>
<td>53 ± 2</td>
<td>6.77 ± 0.00</td>
</tr>
<tr>
<td>14</td>
<td>EDH + ASDH</td>
<td>8 ± 1</td>
<td>44 ± 1</td>
<td>NA</td>
</tr>
<tr>
<td>15</td>
<td>ASDH</td>
<td>24 ± 0</td>
<td>49 ± 2</td>
<td>7.21 ± 0.01</td>
</tr>
<tr>
<td>16</td>
<td>ASDH</td>
<td>16 ± 0</td>
<td>42 ± 0</td>
<td>7.33 ± 0.01</td>
</tr>
<tr>
<td>mean ± SD</td>
<td></td>
<td>13 ± 9</td>
<td>47 ± 5</td>
<td>7.10 ± 0.29</td>
</tr>
</tbody>
</table>

* All values are presented as the mean ± SD. Abbreviations: ASDH = acute SDH; ICH = intracranial hematoma; NA = not available.
† p < 0.05.

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**Discussion**

We recently reported our experience with the Paratrend 7 O$_2$, CO$_2$, pH, and temperature continuous sensor in 24 patients with severe head injury. The sensor was inserted and fixed with a specially designed bolt beside a ventriculostomy catheter and a microdialysis probe. These studies demonstrated that the sensors were stable for up to 5 days and that brain PO$_2$ values were correlated closely with intracranial pressure, cerebral perfusion pressure, cerebral blood flow (CBF), dialysate glucose, and outcome. When the brain PO$_2$ was consistently below 25 mm Hg, the outcome was always death or vegetative survival. Similarly, in a validation study in which the feline model of MCA occlusion was used, the sensor was highly sensitive to ischemia; brain PO$_2$ values fell within infarcted tissue to approximately 15 to 20 mm Hg.

More recently, we and other groups have used the sensor in the operating room. In contrast to other groups, who have reported the use of the Paratrend 7 as a monitoring adjunct in cerebrovascular surgery, we have evaluated the ability of this sensor to determine the effect of temporary arterial occlusion during aneurysm surgery. We have evaluated the hypothesis that the sensor would help to determine the duration of safe parent vessel occlusion. We also had the opportunity to evaluate the sensor in four patients with large traumatic hematomas, before and after craniotomy. This study yielded both expected and unexpected findings.

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reported the strong positive correlation between brain PO$_2$ and CBF in severely head injured patients. Unfortunately, CBF measurements were not made preoperatively in this study. Because the brain gas tension measurements were made within 12 hours post-SAH and before vasospasm developed, we speculate that these high brain PO$_2$ values may represent postischemic reperfusion hyperemia and the consequent episode of high intracranial pressure.$^{14,15}$

Several authors have measured the cerebral metabolic rate for oxygen (CMRO$_2$) and O$_2$ extraction fraction post-SAH, with different results and interpretations.$^{5,18}$ Carpenter, et al.,$^4$ using positron emission tomography scanning, found a depressed CMRO$_2$ together with reduced CBF after SAH. Extrapolating from these studies would suggest that higher brain PO$_2$ post-SAH can best be explained by a reduction in CMRO$_2$ that is relatively larger than the reduction in CBF. However, differences in techniques, measurements obtained during craniotomy compared with those in a closed skull, different measured endpoints, the relatively small numbers of patients in both studies, and the difference in time of measurement after rupture of the aneurysm all limit true comparison among these studies.

Brain PCO$_2$ was higher and brain pH was lower in our SAH group when compared with the unruptured aneurysm group, but the differences were not significant. This is likely because of the heterogeneity of the baseline values and our small number of patients. It is important to note that brain HbO$_2$ was lower in the SAH group, suggesting increased CMRO$_2$, as would be expected after an ischemic episode. We speculate that our findings, made within 12 hours after the initial bleed, together with the high levels of brain PCO$_2$ caused by increased metabolic CO$_2$ production, are the result of an attempt by the brain to restore ionic homeostasis after ischemia.$^{4,6,9,18,22,24}$

During arterial occlusion, brain PO$_2$ and O$_2$ saturation fell far less in the SAH group than in patients with unruptured aneurysms. Again, this agrees with the conditions of preexisting hyperemia and a better O$_2$ reserve in the SAH group, although it is well known that patients with SAH are more vulnerable to ischemia than those with unruptured aneurysms, possibly because of derangements in neuronal and astrocyte biochemistry or vasoregulatory function.$^{4,19}$ Clearly, more patients need to be studied before any definitive conclusions can be drawn regarding this matter.
Intraoperative measurement of cerebral metabolites

TABLE 5
Comparison of normal values in brain with values after arterial occlusion during aneurysm surgery in a feline model and in humans

<table>
<thead>
<tr>
<th>Group Studied</th>
<th>Before Occlusion</th>
<th>During Occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>cats (MCA occlusion)*</td>
<td>43 ± 11</td>
<td>25 ± 7†</td>
</tr>
<tr>
<td>present study (7 patients)‡</td>
<td>60 ± 31</td>
<td>27 ± 17</td>
</tr>
<tr>
<td>present study, MCA only (5 patients)</td>
<td>58 ± 30</td>
<td>30 ± 18</td>
</tr>
</tbody>
</table>

* FiO₂ = 30%.
† After 30 minutes of permanent occlusion.
‡ FiO₂ = 45 to 50%.

Aneurysms: Temporary Occlusion

Comparing the mean baseline values for all three parameters in the five patients with unruptured MCA aneurysms to those obtained in our previously published animal studies shows a close resemblance (Table 5). In the feline model of permanent MCA occlusion, data were obtained immediately before and 30 minutes after occlusion. During this period, brain PO₂ fell from 43 to 25 mm Hg. The lower baseline value for brain PO₂ in the feline model can be explained by the lower FiO₂, which led to a lower PaCO₂ (30% compared with 45–50%).

Similar trends were seen in all patients when comparing brain PO₂, brain PCO₂, and brain pH before and after temporary occlusion of the parent vessel. Brain PO₂ dropped, brain PCO₂ increased, and brain pH fell. However, the baseline values measured before temporary occlusion showed wide variability among patients in both the ruptured and unruptured aneurysm groups. This occurred despite the fact that data collection was initiated after stable measurements were obtained. Several factors could contribute to this. In the healthy brain with normal cardiopulmonary status an increase in FiO₂ leads to an increase in PaO₂, followed by an increase in brain PO₂, a mild increase in brain PCO₂, and a mild drop in brain pH. The fact that brain PO₂ correlates well with PaO₂ in the unruptured aneurysms (near-normal brain tissue) supports this hypothesis. The lack of correlation between PaO₂ and brain PO₂ in the ruptured aneurysm group suggests loss of O₂ vasoregulation in the injured brain. The mean time for the brain PO₂ to return to baseline (levels measured before temporary occlusion) was 6 minutes. The maximum period measured was almost 10 minutes. A safe reperfusion time depends not only on the resupply of O₂, but also on the preconditioning status of the tissue. However, there was no correlation between reperfusion time and status (ruptured compared with unruptured aneurysm) or duration of temporary occlusion. This is probably because of the relatively small numbers of patients in both groups.

Effect of Vascular Anatom

The heterogeneous responses to temporary arterial occlusion in the four measured parameters may be due in part to the integrity of the collateral circulation for delivery of substrate to the acutely substrate deprived tissue. In general, the largest changes were found when the occluded vessel was an end artery such as the MCA. Also, the interpatient anatomical variability in the distribution area of the parent vessel may play an important role. The pattern of immediate redistribution of blood flow after occlusion of a major artery is determined by the anatomy and physiological status of the cerebral vasculature. Even if the sensor could be placed in exactly the same cortical area, we speculate that occluding a major vessel would most likely result in different redistribution patterns for blood flow and therefore different substrate supply (O₂) and metabolic product washout (CO₂) in different patients.

Duration of Vessel Occlusion

Unfortunately, no safe time limits for temporary occlusion can be determined from these data, because no patient developed a postoperative neurological deficit and we did not increase the duration of occlusion beyond 10 minutes. In previous studies in our group of patients with severe head injury we found that patients with a brain PO₂ below 25 mm Hg over a prolonged period of hours or days all had a poor outcome. In all of the 12 patients with aneurysms currently reported, the baseline brain PO₂ was 25 mm Hg or higher. However, during temporary occlusion, five patients experienced a drop in brain PO₂ to below 25 mm Hg for brief periods, but none of these patients displayed a worsening postoperative neurological deficit. It thus appears that the duration (and/or the density) of substrate delivery reduction during occlusion was not sufficient to produce neurological deficits. However, it is important to note that in two patients, brain PO₂ fell below 10 mm Hg for as long as 3 to 4 minutes, yet no deficits were seen, possibly because of protective effects of anesthesia.

Intracranial Hematomas

In this study we have shown the devastating effects of space-occupying traumatic intracranial hematomas on...
substrate delivery and the beneficial effect of removal of these lesions. These results agree with established principles and findings in which other methods such as CBF measurement have been used.1,21

A clear improvement in substrate delivery to brain tissue was found in all four patients after hematoma removal. The striking increase in mean brain PCO₂, as seen in these patients, may be due in part to an increase in ETPCO₂ (Case 16, Table 4) caused by changes in ventilator settings (decreased ventilation rate). Clearly, however, this may also reflect increased metabolism and CO₂ generation. A clear improvement in brain PO₂ was seen in all four patients. The mean brain PO₂ increased by almost 250% during surgery. These findings strongly support the emergency management of severely head injured patients by removal of traumatic intracranial hematomas as soon as possible after diagnosis.

References