

TRANSLUMINAL BALLOON ANGIOPLASTY IMPROVES BRAIN TISSUE OXYGENATION AND METABOLISM IN SEVERE VASOSPASM AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE: CASE REPORT

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OBJECTIVE AND IMPORTANCE: The effect of transluminal balloon angioplasty on cerebral biochemical monitoring during treatment of severe cerebral vasospasm after subarachnoid hemorrhage (SAH) was investigated.

CLINICAL PRESENTATION: In a 36-year-old man, an anterior communicating artery aneurysm caused an SAH (Hunt and Hess Grade IV, Fisher Grade III). After clipping, intraparenchymal monitoring (intracranial pressure, brain tissue oxygen tension [$P_{ti}O_2$], and microdialysis sampling of extracellular glucose, lactate, pyruvate, and glutamate) was initiated. Flow velocities obtained by transcranial Doppler sonography increased in the internal carotid artery (ICA)/middle cerebral artery bilaterally.

INTERVENTION: After a decrease of $P_{ti}O_2$ to less than 2 mm Hg and an increase of the lactate-to-pyruvate ratio to 44 in the territorial region of the left ICA, angiography demonstrated a 70 to 80% stenosis of the left ICA, which was dilated by a temporary occlusion balloon. This maneuver normalized the ICA diameter, $P_{ti}O_2$ increased immediately from 1.5 to 40 mm Hg, the lactate-to-pyruvate ratio decreased from 44 to 30, and extracellular glucose increased from 0.4 to 0.9 mmol/L. No major changes in glutamate or intracranial pressure were seen. In the clinical follow-up, the patient showed a good recovery 6 months after SAH.

CONCLUSION: Transluminal balloon angioplasty led to a continuous and effective resolution of cerebral vasospasm observed by sustained, improved cerebral biochemical parameters. Both $P_{ti}O_2$ and lactate-to-pyruvate ratio might provide an early diagnosis of severe cerebral vasospasm after SAH and continuous surveillance of threatened tissue regions after transluminal balloon angioplasty.

KEY WORDS: Brain tissue oxygen tension, Cerebral vasospasm, Microdialysis, Multimodal neuromonitoring, Subarachnoid hemorrhage, Transluminal balloon angioplasty

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Cerebral vasospasm (CVS), the most common complication after subarachnoid hemorrhage (SAH) (25, 31), was demonstrated angiographically in 23 to 70% of patients independent of surgical or endovascular treatment (4, 17, 20, 35). As a consequence of CVS, permanent ischemic neurological deficits occur in 20 to 36% of the patients (4, 20), and 7 to 20% die because of it (18, 20, 25). The correct diagnosis of CVS is crucial, but clinical follow-up in high-grade SAH (Hunt and Hess Grade IV-V) is imprecise, angiography does not allow us to estimate the risk of ischemia that might result from a detected stenosis, and transcranial Doppler studies have shown discrepancies between flow

velocities and CVS (7, 14, 29). Cerebral blood flow studies (xenon computed tomography [CT], single-photon emission CT) permit intermittent statements only (49), and techniques such as CT, three-dimensional CT, magnetic resonance imaging, and magnetic resonance angiography are still being evaluated (3, 16, 39, 43). Therefore, continuous measurements are currently under investigation for the reliable detection of cerebral ischemia, such as jugular vein oxygen saturation (jugular bulb oximetry) and local methods (laser Doppler flowmetry, thermodilution probes, brain tissue oxygen tension [$P_{ti}O_2$], and microdialysis). In this report, transluminal balloon angioplasty (TBA) that induced long-lasting,

dramatic cerebral metabolic improvement monitored with local tissue metabolites (microdialysis) and intraparenchymal $P_{ti}O_2$ measurement is described.

CASE REPORT

A 36-year-old man was admitted 2 days after the sudden onset of headache; it had resolved at first, but he experienced a repeated acute onset of severe headache after participating in a soccer game. An accompanying severe meningism with left-sided hemiparesis and impaired consciousness (Glasgow Coma Score [GCS], 11 [44]) was found. The initial cranial CT revealed an SAH of Fisher Grade III (15), and an SAH of Hunt and Hess Grade IV (22) was diagnosed. Cerebral angiography demonstrated an anterior communicating artery aneurysm, which was treated surgically by clipping the following day.

After surgery, nimodipine (1 mg/h) was given, and the patient was extubated 3 days after admission (GCS, 11). However, during the next 3 days, he slowly deteriorated, with signs of limb extension caused by pain stimuli (GCS, 7). Cranial CT revealed diffuse brain swelling with narrowing of the basal cisterns without clear signs of infarction. Therefore, he was reintubated and ventilated. After initiation of hypertensive, hypervolemic, hemodilution (“triple-H”) therapy, systolic arterial pressure was maintained at approximately 170 mm Hg with high doses of catecholamines (12 μ g/min epinephrine, 12 μ g/min norepinephrine, 7 μ g/kg/min dopamine) and plasma expander. Flow velocities measured by transcranial Doppler increased from 76 cm/s (Day 3) to 240 cm/s (Day 6) in the left M1 segment of the middle cerebral artery (M1-MCA) and from 53 to 200 cm/s in the right M1-MCA. Multimodal neuromonitoring (intracranial pressure [ICP], $P_{ti}O_2$, and microdialysis) was initiated (Table 1).

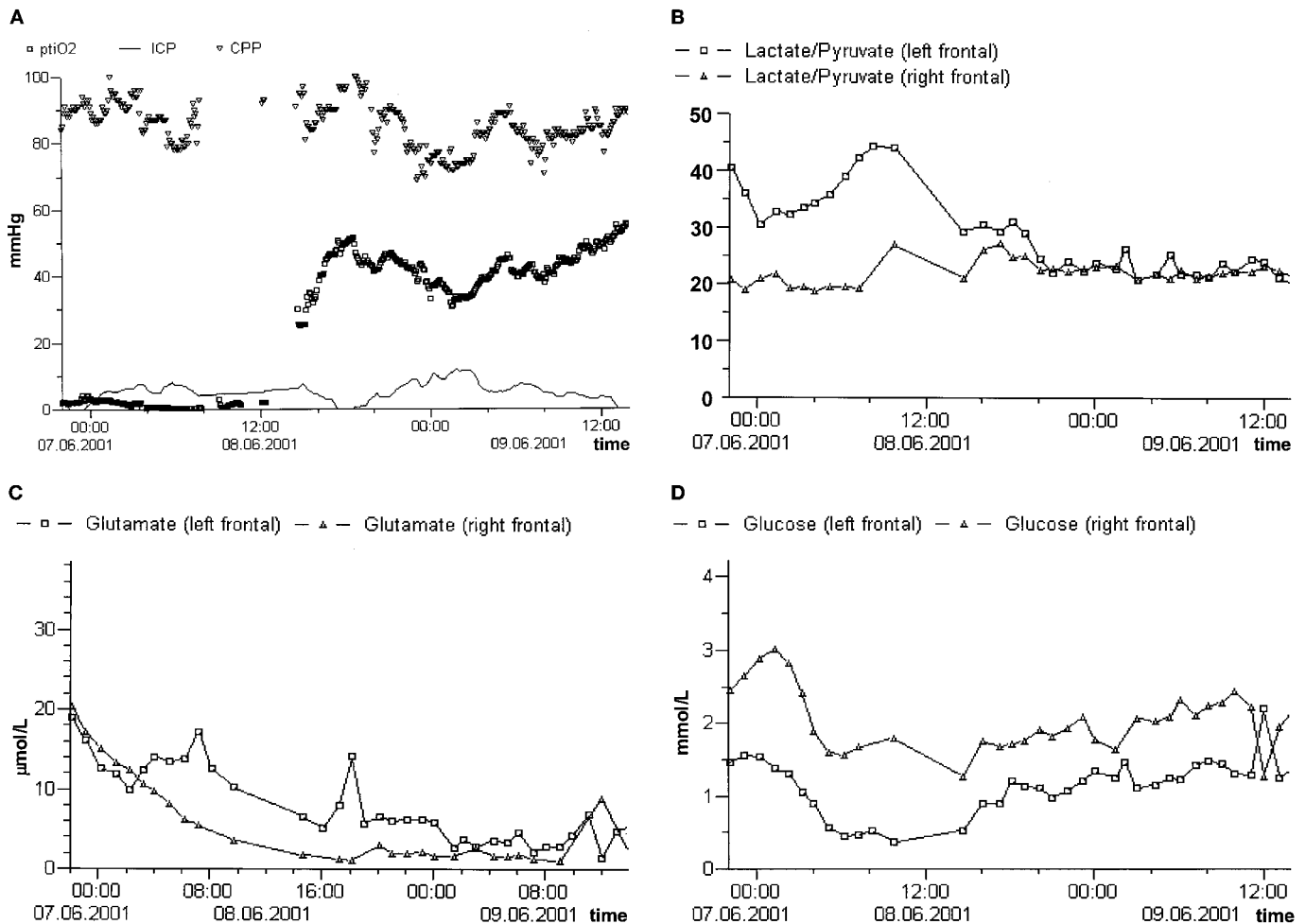


FIGURE 1. Thirty-six-hour interval of multimodal neuromonitoring (ICP, cerebral perfusion pressure, $P_{ti}O_2$, lactate, pyruvate, glutamate, glucose) representing the time before and after TBA (indicated on the scales as 12:00 noon on August 6, 2001). ICP and cerebral perfusion pressure remained nearly unchanged, $P_{ti}O_2$ recovered from 0 to more than 30 mm Hg (A), LPR decreased after TBA (B), and glutamate showed the typical bilateral decrease probably caused by local ischemia after probe application (C). Glucose (D) increased slightly after TBA.

TABLE 1. Location and specification of the applied probes used for multimodal neuromonitoring^a

Probe	Location	Specification
Intracranial pressure	Right frontal	Codman Microsensor; Codman & Shurtleff, Inc., Raynham, MA
P _{ti} O ₂	Left frontal	Licox; GMS, Kiel-Mielkendorf, Germany
Microdialysis	Bifrontal	Catheter (CMA 70) with membrane length 10 mm, diameter 0.52 mm, perfusion pump (CMA 106) rate 0.3 μl/min with fluid containing 147 mmol/L Na ⁺ , 4 mmol/L K ⁺ , 2.3 mmol/L Ca ⁺ , 156 mmol/l Cl ⁻ , pH 6, 290 mosm/kg, 1-hr collection interval, measuring glucose, lactate, pyruvate, and glutamate (measured by CMA 600; CMA Microdialysis, Solna, Sweden)

^a P_{ti}O₂, brain tissue oxygen tension.

INTERVENTION

Before TBA, the patient's P_{ti}O₂ level was low (Fig. 1A), the left frontal lactate-to-pyruvate-ratio (LPR) was increased (Fig. 1B), and the glucose level decreased (Fig. 1D) without an increase in glutamate (Fig. 1C). The proximal segments of the MCA revealed high flow velocities (left M1-MCA, 240 cm/s; right M1-MCA, 200 cm/s). Median and tibial nerve somatosensory evoked potential amplitudes were reduced bilaterally with left-sided (right cortical) emphasis. The cranial CT scan revealed no ischemic areas. Six days after the hemorrhage, cerebral angiography of both internal carotid arteries showed a 70 to 80% stenosis of the left ophthalmic C6 segment (5) caused by vasospasm (Fig. 2A). The left anterior cerebral artery (ACA) was severely stenosed as well. TBA using a temporary occlusion balloon (Sentry; Boston Scientific, Fremont, CA) resulted in a normalization of the C6 diameter of the left internal carotid artery (Fig. 2B). We did not attempt to reach the left ACA. After TBA, the P_{ti}O₂ increased 20-fold within 1 hour (Fig. 1A), the LPR decreased on the affected left side

during the following 6 hours (Fig. 1B), and glutamate remained nearly unchanged (Fig. 1C). Flow velocities in the left M1 segment remained high (240 cm/s). All parameters monitored 3 hours before and 3 hours after TBA are summarized in Table 2. During the next 5 days, the ICP, cerebral perfusion pressure, P_{ti}O₂, and microdialysis parameters remained stable, and flow velocities slowly decreased bilaterally. No infarction was visible in cranial CT and cranial magnetic resonance imaging. Three days after TBA, the amplitudes of the median nerve somatosensory evoked potentials recovered partially, whereas bilateral tibial nerve somatosensory evoked potentials remained without significant change in amplitudes

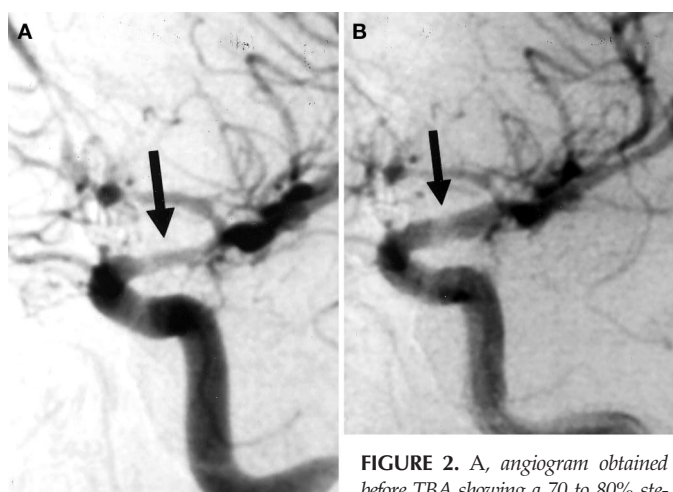


FIGURE 2. A, angiogram obtained before TBA showing a 70 to 80% stenosis (arrow) of the left ophthalmic C6 segment caused by vasospasm. The left ACA was also severely stenosed. B, angiogram showing that transluminal angioplasty resulted in a normalization of the C6 diameter (arrow). We did not attempt to reach the ACA.

TABLE 2. Monitoring data 3 hours before and 3 hours after transluminal balloon angioplasty of a 70 to 80% stenosis of the left C6 internal carotid artery segment^{a, b}

	3 h before TBA		3 h after TBA	
	Left	Right	Left	Right
ICP (mm Hg)	4		3	
CPP (mm Hg)	93		88	
P _{ti} O ₂ (mm Hg)	1.5		40.1	
Lactate (mmol/L)	1.9	2.6	1.6	1.9
Pyruvate (μmol/L)	43.7	95.8	51.0	70.8
Glucose (mmol/L)	0.4	1.8	0.9	1.7
Glutamate (μmol/L)	10.3	3.5	5.2	1.3
Lactate/pyruvate	44	27	30	27
Glucose/lactate	0.19	0.69	0.57	0.87

^a TBA, transluminal balloon angioplasty; ICP, intracranial pressure; CPP, cerebral perfusion pressure; P_{ti}O₂, brain tissue oxygen tension.

^b After TBA, ICP and CPP remained nearly unchanged. The P_{ti}O₂ sensor inserted in the affected blood supply region of the left internal carotid artery showed a greater than 20-fold increase. The lactate-to-pyruvate ratio decreased and glucose increased in the affected left hemisphere as an indication of a shift to more aerobic metabolism.

and latencies. Clinically, the patient sustained a combined sensorimotor aphasia and a slight right-sided hemiparesis (GCS, 12), which partially resolved during the hospital stay. After ventriculoatrial shunting for hydrocephalus, he left the clinic for rehabilitation with a Glasgow Outcome Score (24) of 3 (severely disabled, needed continuous assistance); during a 6-month period, he recovered to Glasgow Outcome Score 5 (good recovery with only minor mental deficits, returned to work as a farmer).

CONCLUSION

Angiography (23, 31, 48) and cerebral blood flow measurements (13, 23, 36) can be performed only at intervals; transcranial Doppler enables CVS to be suspected (1, 2, 9, 19, 30, 41) but does not reflect the risk of imminent ischemia (7, 14, 28, 29, 33, 34). This underlines the need for continuous monitoring techniques. This case report shows a recovery of metabolic parameters ($P_{\text{ti}}\text{O}_2$, microdialysis) after TBA, even when performed late, to resolve a severe CVS caused by a high-grade SAH. Similar results were described by Fandino et al. (12), who found an improvement of jugular vein oxygen saturation of more than 5% in 7 of 10 patients. This indicates that $P_{\text{ti}}\text{O}_2$ and microdialysis might be sensitive parameters to detect CVS, as has also been shown by other groups (40, 42, 45). It is of further interest that a low $P_{\text{ti}}\text{O}_2$ level during a period of 22 hours did not result in visible ischemia in the follow-up CT, because hypoxic values for $P_{\text{ti}}\text{O}_2$ are described in a range between 5 and 20 mm Hg (8, 27, 32, 46, 47). This low $P_{\text{ti}}\text{O}_2$ in the measured territory might not only be explained by reduced cerebral blood flow but might also be affected by oxygen consumption and altered intracellular-extracellular volume relation. Therefore, very low oxygen tensions in SAH should be interpreted carefully, and additional parameters should also be used to validate the causes of low $P_{\text{ti}}\text{O}_2$. Glutamate, as a promising candidate to indicate cerebral ischemia (6, 45, 50), showed no simultaneous increase with the LPR in this patient. One limitation for both $P_{\text{ti}}\text{O}_2$ and microdialysis is that they are measuring in a small territory, which limits our ability to draw conclusions about the whole brain tissue. Thus, especially in SAH, local vasospasm-induced narrowing of cerebral vessels might lead to ischemia of brain regions that are not monitored. How neuromonitoring can be integrated to decide on the timing and indications for TBA needs to be further clarified. Early angioplasty (<24 h after onset of symptoms) results in better functional outcome (11, 21), but the mortality rate ranges from 0 to 12% after TBA (10–12, 14, 26, 37, 38). In summary, the long-lasting improvement of oxygenation and shift to aerobic glycolysis are evidence of the success of TBA. Therefore, $P_{\text{ti}}\text{O}_2$ and microdialysis monitoring might allow us to keep the effect of TBA under surveillance.

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COMMENTS

The indications for transluminal balloon angioplasty (TBA) continue to be debated. Most important, the discussion focuses on the reasons for poor outcome and failure to reverse ischemic deficits after TBA. In this case report, the authors have carefully studied a poor-grade patient with severe cerebral ischemia after aneurysmal subarachnoid hemorrhage (SAH). The patient's metabolic parameters showed significant deterioration in brain tissue oxygen tension (P_{ti}O₂) and the development of anaerobic glycolysis. Angioplasty was successful in rapidly reversing and normalizing P_{ti}O₂ and promoting aerobic glycolysis. This type of metabolic analysis indicates that TBA can be effective if instituted before irreversible brain injury occurs. The suggestion is that monitoring metabolism in high-risk patients might identify at an early stage patients who are candidates for therapeutic intervention.

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The notable aspect of this case report is the chemical change in the brain found by performing microdialysis after TBA of cerebral vasospasm (CVS). An improvement was seen; the patient's P_{ti}O₂ increased, and the lactate-to-pyruvate ratio improved. Cause-and-effect relationships are intuitive and convincing. Unfortunately, in this case, there was no clear correlation with the patient's clinical condition, which did not change in the hours or days after treatment. It might be argued that irreversible infarction precluding the eventual recovery seen was staved off, but even the authors did not advance this conjecture. The major limitation of the microdialysis monitoring technique is its highly restricted sampling site and size, which the authors do emphasize. This case report adds further support for performing timely TBA in patients with severe CVS to reverse cerebral ischemia (although the support the article provides would have been greater if the patient had

improved promptly), but the role of the demanding technique of microdialysis in the routine management of patients with ruptured cerebral aneurysms remains quite unclear.

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In this report, the authors describe changes in several parameters measured by invasive and noninvasive means after performing TBA of the left internal carotid artery for symptomatic CVS. These parameters included $P_{\text{t}}\text{O}_2$, glucose, lactate, pyruvate, and glutamate levels in addition to intracranial pressure and arterial flow velocities. $P_{\text{t}}\text{O}_2$ was measured with a Licox GMS probe (GMS, Kiel-Mielkendorf, Germany) placed in the left frontal lobe. The $P_{\text{t}}\text{O}_2$ level changed from 1.5 to 40 mm Hg after angioplasty was performed in the left internal carotid artery for CVS. The glucose, glutamate, lactate, and pyruvate levels were measured with a CMA 70 probe (CMA Microdialysis, Solna, Sweden) placed in both frontal lobes. After angioplasty was performed, the patient's glucose level changed from 0.4 to 1.8 mmol/L and the lactate-to-pyruvate ratio changed from 44 to 27, but the glutamate level did not change. The patient's intracranial pressure did not change after angioplasty. Transcranial Doppler sonography showed no change in the 240-cm/s blood flow velocity of the left M1 segment after angioplasty, but it did show an increase in the left M2 segment blood flow velocity from 66 to 220 cm/s after angioplasty. The authors explain that $P_{\text{t}}\text{O}_2$ values lower than 5 mm Hg suggest ischemia, that high (i.e., >50) lactate-to-pyruvate ratios indicate a conversion from aerobic to anaerobic metabolism, and that increased glutamate levels are seen after tissue is infarcted. They think that their report is the first of changes in tissue metabolic parameters in CVS after angioplasty, but they note that Fandino et al. (1) reported similar findings in a patient with CVS after papaverine infusion. They conclude that routine monitoring of these metabolic parameters after SAH may be helpful in detecting severe CVS. They caution that only a small portion of the brain with problems is sampled and that areas of the brain that are not sampled could be ischemic. Placed in context with other data, these observations tend to support the hypotheses that CVS can be detected early, that it causes harm to the brain that can be measured in a number of ways, and that its reversals can improve the metabolic and imaging measurements. Good data have not been found to show the extent to which CVS is detected and corrected better for patients in the long run.

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1. Fandino J, Kaku Y, Schuknecht B, Valavanis A, Yonekawa Y: Improvement of cerebral oxygenation patterns and metabolic validation of superselective intraarterial infusion of papaverine for the treatment of cerebral vasospasm. *J Neurosurg* 89:93-100, 1998.

In this case report, the authors show that performing TBA in patients with symptomatic CVS after aneurysmal SAH can improve biochemical markers (i.e., glucose level, lactate-to-pyruvate ratio) and $P_{\text{t}}\text{O}_2$ as measured by microdialysis and a Clark-type electrode. On the basis of this observation, they conclude that microdialysis and $P_{\text{t}}\text{O}_2$ monitoring might be useful in monitoring the long-term effect of TBA. That these techniques could be used to detect clinically significant CVS has been shown previously (4), but with documentation of the effect of TBA, this report is a welcome addition to the sparse literature on the subject published to date.

I agree that monitoring clinical signs of CVS is difficult in patients with already low Glasgow Coma Scale scores or in whom focal, unilateral deficits (mostly midline aneurysms) might not develop; therefore, at my institution, my colleagues and I have adopted a policy that a decline in Glasgow Coma Scale score of 2 or more points in the absence of other causes of deterioration (e.g., hydrocephalus, metabolism, pulmonary problems) is an indication for performing angiography, regardless of the transcranial Doppler sonographic findings (2). We have found CVS in approximately 70% of such cases, so the addition of the techniques mentioned here might reduce the need for angiography by some 25%.

The authors do not quote more recent, large series of patients who have undergone TBA showing that the mortality rate with the use of this procedure ranges from 0 to 4% if performed in intubated, chemically paralyzed patients (in lieu of the quoted 0-12%) (1, 3). Thus, the "biochemical success" reported here, even in the absence of immediate clinical improvement but with a good final outcome, leads me to put even more faith in this therapeutic procedure.

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Hoelper et al. provide examples of the types of physiological monitoring technologies that are available and how changes of the blood vessel lumen can alter these measurements. Although interesting from a technological perspective, it seems that the authors have lost sight of the basics of caring for patients after SAH.

The authors instituted multimodal physiological monitoring on Day 6 postbleed and 3 days after the patient had begun to deteriorate after undergoing clipping of his anterior communicating artery aneurysm. At that time, the patient had

deteriorated from hemiparesis to extensor posturing, and his computed tomographic scan then demonstrated diffuse brain swelling. $P_{ti}O_2$ levels at that point were below the threshold for viability, the lactate-to-pyruvate ratio was increased, and the glucose level was decreased with an increase in glutamate. At this point, angioplasty was performed, and a good angiographic and metabolic result was shown (i.e., $P_{ti}O_2$ increased, lactate pyruvate ratio improved, glutamate remained stable), but without any clinical improvement.

Although these measurements are interesting, they were not obtained at the correct point in the patient's clinical course. The ideal use of such measurements is to detect the onset of the compromise of blood flow, not to document that a severe injury has already occurred. Angioplasty performed in a timely manner can be beneficial; when performed after an ischemic injury is already present, however, it is likely to cause additional deterioration because of hemorrhagic transformation of an ischemic injury.

$P_{ti}O_2$ levels below 8 for more than 24 hours should have indicated an irreversible ischemic injury. This type of low

value that increases by 20-fold with improvement in blood delivery is consistent with the insertion of a probe into infarcted brain tissue that became severely hyperemic with reperfusion. An alternative and unfortunately common explanation for the low $P_{ti}O_2$ levels is that the probe never extended beyond the delivery system. The decrease in the lactate-to-pyruvate ratio but the retention of high glutamate levels is consistent with reperfusion hyperemia within an area of infarction.

Thus, although the availability of new systems for monitoring $P_{ti}O_2$ levels and metabolic products should have a role in clinical management, their role is yet to be defined. Will they be able to determine only when tissue has already been irreversibly injured, or will they provide a useful marker of tissue at risk? As with other measures such as CBF, the response to a physiological challenge is often needed to provide valuable predictive insights.

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Sir Victor Horsley (1857–1916) (from, Sachs E: *The History and Development of Neurological Surgery*. New York, Paul B. Hoeber, Inc., 1952).