Real-Time Changes in Brain Tissue Oxygen During Endovascular Treatment of Cerebral Vasospasm

Rune Rasmussen, Søren Bache, Trine Stavngaard, Jane Skjøth-Rasmussen, and Bertil Romner

Abstract The use of endovascular intervention to treat cerebral vasospasm after subarachnoid hemorrhage has increased. Although the effect on angiographic vasospasm can be easily demonstrated, the effect on cerebral blood flow and clinical outcome is still controversial. In this report, we investigate minute-by-minute changes in brain tissue oxygen during balloon angioplasty and intraarterial administration of vasodilators in three patients.

Our results confirm that endovascular intervention is capable of not only resolving angiographic vasospasm, but also of normalizing values of brain tissue oxygen pressure ($PtiO_2$) in target parenchyma. However, during the intervention, dangerously low levels of brain tissue oxygen, leading to cerebral infarction, may occur. Thus, no clinical improvement was seen in two of the patients and a dramatic worsening was observed in the third patient. Because the decrease in brain tissue oxygen was seen after administration of vasopressor agents, this may be a contributing factor.

Keywords Subarachnoid hemorrhage • Cerebral vasospasm • Endovascular intervention • Brain tissue oxygen monitoring

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Introduction

Delayed cerebral ischemia (DCI) is the leading cause of poor outcome in the weeks after subarachnoid hemorrhage (SAH) [3]. The presumed cause of DCI is cerebral vasospasm, although this causality has been challenged lately [1]. No fully effective treatment of DCI exists, but, during the last decade, the use of endovascular intervention has increased. Mechanical dilation (percutaneous transluminal angioplasty (PTA)) and/or intraarterial administration of vasodilating drugs has been used to resolve cerebral vasospasm to prevent or treat DCI. The effect of endovascular intervention is, however, still controversial [6].

To evaluate the effect of endovascular intervention, neuroradiologic imaging has traditionally been performed. However, because the causality between angiographic vasospasm and DCI is complex, other measurements are needed to guide and evaluate treatment. Brain tissue oxygen pressure ($PtiO_2$) offers a continuous measurement of cerebral oxygenation and can be used as a surrogate measure of cerebral blood flow [5]. In this way, measurement of $PtiO_2$ offers a unique way of monitoring the response in target brain parenchyma during manipulation of the feeding arteries. Although some reports describe changes in $PtiO_2$ before and after endovascular intervention [2, 4, 7], none so far has described the minute-by-minute changes during the intervention.

The aim of this study was to investigate changes in $PtiO_2$ during PTA and intraarterial administration of vasodilators. In this report, we describe three cases of responses in $PtiO_2$ during endovascular intervention.

Materials and Methods

During 2012, 12 patients with aneurysmal SAH were monitored with brain tissue oxygen pressure ($PtiO_2$) (Licox, Integra) for clinical reasons at our institution. Three of these

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patients developed symptoms of DCI and underwent endovascular intervention. During the intervention, changes in $PtiO_2$ and the exact time of balloon inflation and intraarterial administration of a vasodilator were prospectively collected. Later, the data were retrospectively analyzed. In all three cases, the $PtiO_2$ probe was placed in the white matter of the left frontal lobe. Indications for endovascular intervention were DCI (as defined by Vergouwen et al. [8]) and no response to triple-H (hypertension, hypervolemia, and hemodilution) treatment.

Results

Case 1

Figure 1 shows changes in $PtiO_2$ in the left frontal lobe during endovascular intervention in a 47-year-old woman 5 days after ictus. The clinical symptoms consisted of right-sided hemiparesis and decreased level of consciousness; and bilateral vasospasm was demonstrated on computed tomography (CT) angiography. PTA was performed in the internal carotid artery (ICA), middle cerebral artery (MCA), and anterior cerebral artery (ACA) on both sides, and 2 mg of nimodipin was injected during 20 min into the ICA bilaterally. As can be seen, PTA leads to a rapid response in the level of $PtiO_2$. Initially, a drop in $PtiO_2$ is observed corresponding to the inflation of the balloon in ICA. After 20 min, during which PTA on the MCA and ACA is performed and nimodipin is administrated, values rise above the levels before the intervention. Interestingly, inflation in the right ICA also initially causes a significant drop in $PtiO_2$. At the end of the intervention, $PtiO_2$ values rise to normal levels. Angiography demonstrated resolution of the vasospasm. $PtiO_2$ values in the next 4 days after the intervention was between 20 and 40 mmHg, as compared with below 10 mmHg before intervention; thus, showing a lasting improvement. However, no clinical improvement was observed.

Case 2

A 54-year-old man developed a mild right-sided hemiparesis and confusion 6 days after SAH, and CT angiography revealed vasospasm bilaterally. Figure 2 shows the development of $PtiO_2$ in the left frontal lobe during anesthesia, verapamil infusion, and bilateral PTA. A decrease in MAP after anesthesia made the use of vasopressor agents (ephedrine and phenylephrine) necessary. After initiation of continuous phenylephrine infusion, $PtiO_2$ values decreased to near zero. No immediate effect of intraarterial verapamil was observed; the $PtiO_2$ rose to normal levels and even to

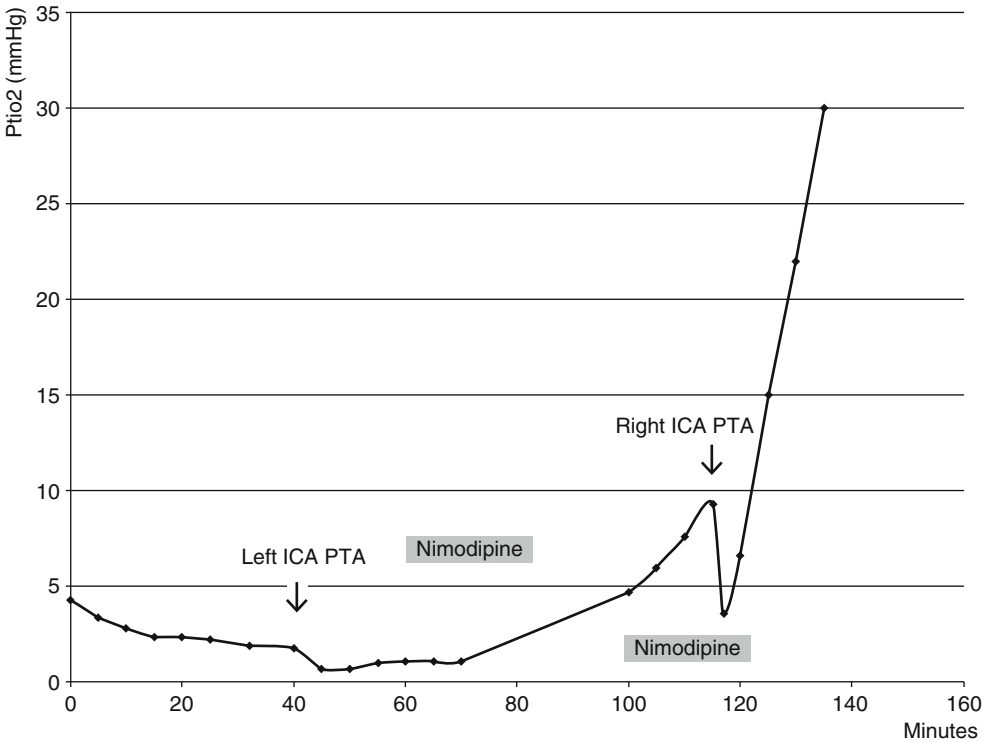

Fig. 1 Development of $PtiO_2$ in the left frontal lobe during endovascular intervention. Times for PTA in the left and right internal carotid artery and period of nimodipin administration are indicated

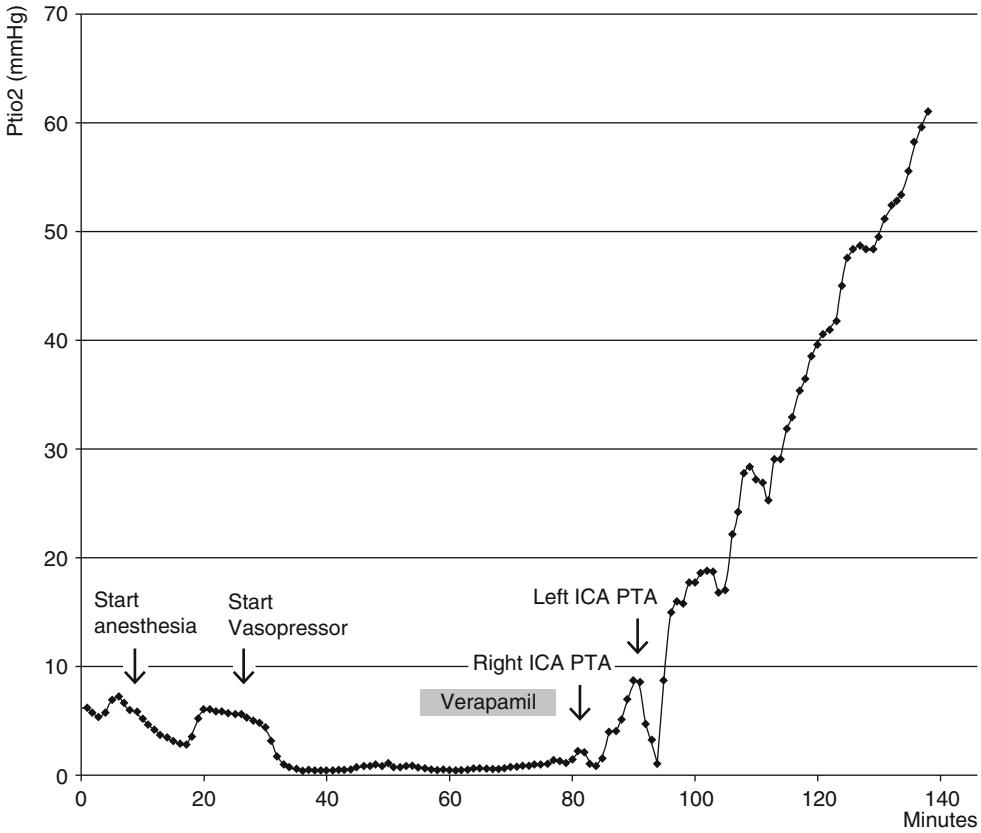

Fig. 2 $PtiO_2$ values in the left frontal lobe during endovascular intervention. Times for anesthesia, vasopressor administration, intraarterial verapamil administration, and PTA are indicated

values suggesting hyperperfusion (>80 mmHg) only after PTA. Angiography after the intervention showed resolution of the vasospasm.

The increased $PtiO_2$ values obtained during intervention were not lasting. Within 24 h after intervention, $PtiO_2$ decreased to the level observed before the intervention and even below. Clinically, a dramatic worsening was observed after the intervention in this case. The mild paresis worsened to paralysis and the Glasgow Coma Scale (GCS) dropped from 14 to 6. CT scan revealed infarction in the left frontal lobe.

Case 3

Figure 3 shows $PtiO_2$ values during administration of vasopressor agents and intraarterial administration of nimopidin (2 mg in the ICA, bilaterally). A significant drop in $PtiO_2$ is observed after administration of vasopressor agents. After nimodipine administration, the $PtiO_2$ level rose to the preintervention level, but not above. No clinical improvement was seen after the intervention.

Discussion

The above cases show that decreases in $PtiO_2$ values can be dramatically improved by mechanic/pharmacological dilation of the main cerebral arteries. However, no clinical improvement was seen in any of the cases. Several reasons can account for this. In Figs. 1 and 3, very low levels of $PtiO_2$ are seen during the intervention. In Fig. 1, the $PtiO_2$ level dropped to values below 2 mmHg for several minutes as the feeding artery was manipulated during PTA. No vasopressors were used in this case. In Fig. 3, a dramatic drop in $PtiO_2$ to near 0 mmHg was seen after initiation of vasopressor agents. The curves suggest that both manipulation of the arteries and the use of vasopressors in patients with cerebral vasospasm can cause ischemia, potentially leading to poorer outcome than without any intervention.

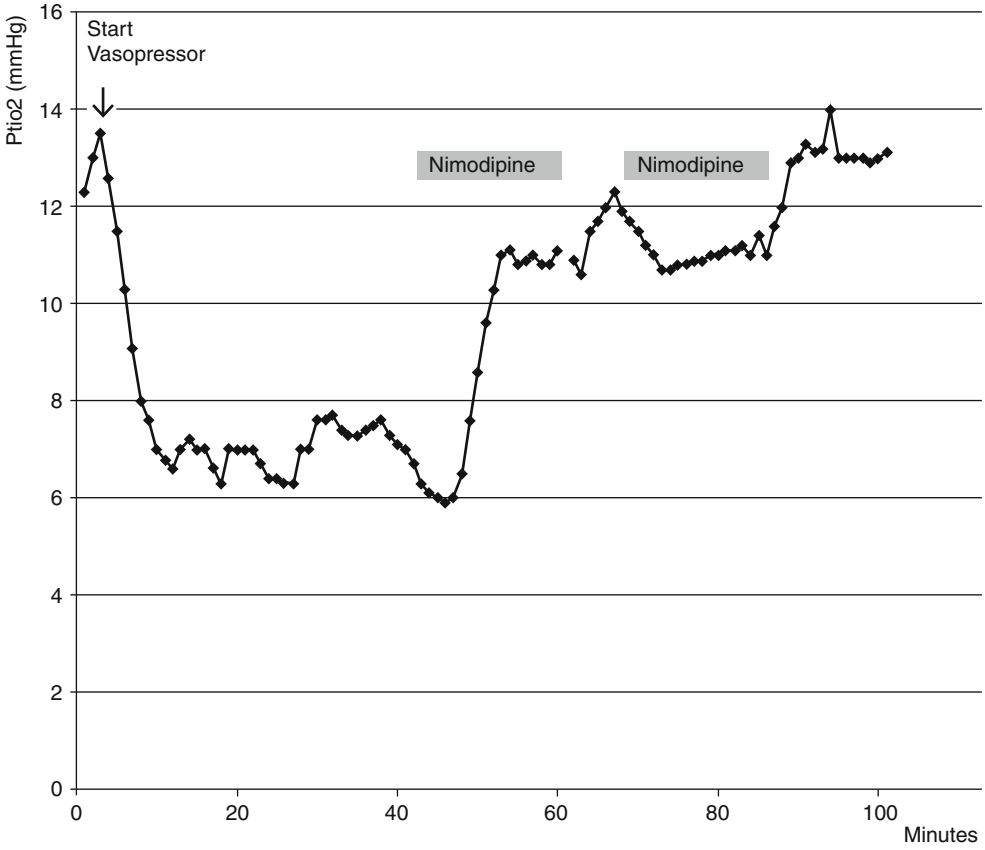

Fig. 3 $PtiO_2$ values in the left frontal lobe during endovascular intervention. Times for vasopressor administration and intraarterial nimodipin administration are indicated

Conclusion

Endovascular intervention is capable of not only resolving angiographic vasospasm, but also normalizing values of $PtiO_2$ in the target parenchyma. However, dangerously low levels of $PtiO_2$ leading to cerebral infarction can be seen during interventions. One reason for this may be the use of vasopressor agents during the anesthesia, but manipulation within the arteries may also be a cause. Further studies are needed for clarification.

Conflict of Interest Statement We declare that we have no conflict of interest.

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