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Short communications

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Alteration of regional brain oxygen saturation (rSO_2) in a patient with cerebral damage after aortic arch replacement: carbon dioxide reactivity monitored by near-infrared spectroscopy suggested inverse steal phenomenon

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Introduction

Cerebral damage is a major complication of aortic arch replacement [1]. To monitor the cerebral circulation and cerebral tissue oxygenation during the perioperative period, several invasive and noninvasive monitors have been devised. Although there are some controversies regarding interpretation of the data [2,3], near-infrared spectroscopy is reported to enable physicians to detect intracranial phenomena, such as cerebrovascular alterations or tissue oxygenation status, noninvasively [4–6]. Recently, several reports have noted that cerebrovascular response to carbondioxide could be detected by this spectroscopic method [7–11]. Although hyperventilation induces vasoconstriction in cerebral tissue and decrease of regional brain oxygen saturation (rSO_2), we studied a case in which rSO_2 increased after hyperventilation.

Case report

A 65-year-old man was hospitalized after being diagnosed as having dissecting aortic aneurysm (DeBakey type I). The patient had pre-existing complications of hypertension and atrial fibrillation. No neurological abnormality was observed before the operation. All clinical procedures were performed after obtaining informed consent from the patient or the patient's family and following the approval of the local ethical committee.

Emergency aortic arch replacement under fentanyl anesthesia was completed with temporary retrograde cerebral circulation (total operation time, 14 h; cardiopulmonary bypass, 9 h; retrograde cerebral circulation, 1 h; hypothermia 18°C, 4 h; hemoglobin concentration, 8–9.6 $g \cdot dl^{-1}$ during cardiopulmonary bypass). The brachiocephalic vessels were excised from the aortic wall en bloc and anastomosed with the prosthetic graft. Throughout the operation, the cerebral circulation was monitored at the right forehead by a near-infrared spectroscope (N-1000; Shimazu, Kyoto, Japan) that recorded alterations of oxyhemoglobin, deoxyhemoglobin, and total hemoglobin content in the tissue. An acute reduction in the total hemoglobin and oxyhemoglobin contents (approximately 0.5 optical density for both hemoglobins, which continued for 30 min and then returned to the original value) was recorded just after the suturing of the internal carotid artery graft. Bilateral carotid arterial blood flow was confirmed to be intact after the completion of the cardiopulmonary bypass. After the operation, there was an intermediate dilation of the right pupil, and light reflexes were not obvious on either sides. At this time, the body temperature was 36.8°C and the hemoglobin concentration was 11.3 $g \cdot dl^{-1}$. The systemic circulation was stabilized by the continuous infusion of catecholamines (dopamine 3–5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, dobutamine 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and lidocaine (3.5–5 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Since voluntary breathing could not be detected, respiration was controlled with a respirator.

On the second day after the operation, the pupils dilatated bilaterally, and no involuntary reflex was observed. Electroencephalography with conventional leads recorded an almost flat line for electrical activity in the brain. The rSO_2 was assessed at the right forehead by another type of near-infrared spectroscope (INVOS-3100, Somanetics, Troy, MI, USA), which indicated the

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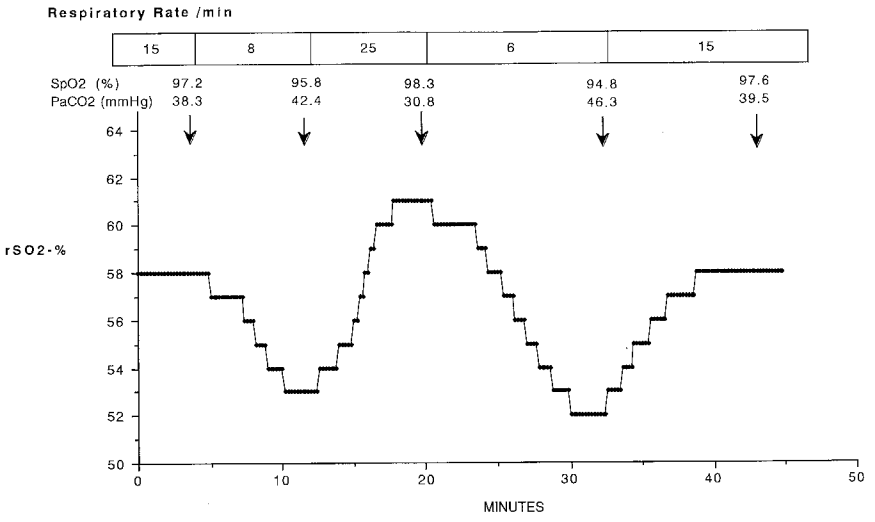

Fig. 1. Response of regional cerebral oxygen saturation value (rSO_2) to change in arterial CO_2 tension. Hyperventilation caused a change in arterial CO_2 tension over the range where the systemic circulatory parameters were maintained stably. Reduction in CO_2 tension induced an increase in rSO_2. *Arrows* indicate the time points when arterial blood samples were collected. The calculated rSO_2 data were averaged every 20 s and displayed stepwise

absolute saturation value for the regional tissue oxygenation [2]. The response to the change in CO_2 tension was also measured over a range where the systemic circulatory parameters were maintained stable (Fig. 1). The inspiratory oxygen percentage was 70% throughout the examination. The original saturation value was 56%–58% and the reduction in CO_2 tension caused by hyperventilation (from 600 ml × 8·min^{-1} to 600 ml × 25·min^{-1}) induced an increase in rSO_2.

On the third day after the operation, although the systemic circulation and arterial oxygenation were almost stable, the rSO_2 decreased to 44%–53%, but the response to the change in CO_2 tension was almost identical to that of the previous day. On the fourth day after the operation, there was a deterioration in renal function. However, the arterial oxygen saturation (SpO_2) was 95%–99% and the circulatory parameters were stable. The rSO_2 was 28%–37%, and the response to the change in CO_2 tension was not obvious. All brain death criteria (University of Pittsburgh, 1984) were fulfilled [12]. On the fifth day after the operation, the systemic circulation became unstable. A scheduled CT scan examination was postponed because of the unstable condition, and the patient died 117 h after the operation. Postmortem pathological examination indicated that the entire brain was severely damaged by infarction. However, necrosis of the brain tissue was not evenly distributed but was most prominent in the right temporal and frontal cortex.

Discussion

The cerebrovascular response to change in CO_2 tension is known to be a physiological regulation mechanism of the brain circulation [13]. However, the effect of cerebral damage on the reactivity of the cerebral vessels to CO_2 is still controversial. By a continuous measurement of the partial pressure of brain tissue oxygen, van Santbrink et al. demonstrated that CO_2 reactivity did not show a constant pattern of change over time and was not correlated with clinical outcome [14]. Schwab et al. reported that cerebrovascular reactivity to hyperventilation was only observed during the initial phase after massive stroke [15]. By reviewing the current literature, Bouma and Muizelaar found that in clinical cases, CO_2 reactivity, decrease of cerebral blood flow, and oxygen delivery during hyperventilation diminished in association with the severity of the damage rather than the causative mechanism [16]. In the case described here, we observed that the decrease in CO_2 tension induced an increase in rSO_2. Even though the degree of alteration was small, the alteration in SpO_2 might partly contribute to the alteration in rSO_2. (This alteration in SpO_2 was possibly induced by the replacement of oxygen in the lung with the accumulated CO_2.) However, the 1.4% reduction in SpO_2 should be too small to explain the prompt 5% reduction in rSO_2. The shifting of the blood flow within the brain, known as "inverse steal," was thought to be another possible mechanism underlying this phenomenon. Bouma and Muizelaar reported that the dysregulation of the cerebral vasculature after severe head injury was usually confined to the damaged area, and that the dysfunction of the cerebrovascular response was not a generalized phenomenon [16]. In this case, the destruction of the brain tissue was not evenly distributed. Accordingly, the vasoconstrictive reaction in a relatively conserved area might have induced the shifting of the blood circulation into the nonreactive area, and thereafter improve the rSO_2 in the damaged area. Moreover, hyperventilation might reduce the intracranial pressure by decreasing the blood

content in the reactive area and thereafter increase the arterial blood supply to the monitored area. Ruta et al. demonstrated that the "inverse steal" could not be observed in an experimental stroke model [17]. However, Ashwal et al. reported that the "paradoxical CO_2 response" was observed in a pediatric meningitis case by monitoring cerebral blood flow with stable xenon computed tomography [18]. They proposed the cerebral blood flow shifting mechanism as we considered in this case.

There is a continuing controversy about how to interpret rSO_2 data in clinical settings. The influence of extracranial factors, such as temporal superficial artery blood flow and external jugular vein blood pooling, was a major concern for several years [2]. However, when facial muscle movement is not observed, as in our case, the contamination may be minimal. On the other hand, Pollard and Prough recently reported that the measurement by INVOS 3100 is based on several presumptive parameters, which might not be stable in many situations, and that physicians should be careful when they interpret data from the patients whose cerebral blood flow could be disrupted [19]. Schwarz et al. reported that the values obtained by INVOS 3100 from dead brains were in the same range as those obtained from healthy volunteer's brains, and claimed that it should be problematic to believe in the absolute rSO_2 value in extreme cases [20]. These reports suggested that INVOS 3100 is only useful as a continuous trend monitor. Until further improvement of the instrument and establishment interpretation of the data, simultaneous assessment by other cerebral monitors, such as transcranial Doppler ultrasonography and jugular bulb oximetric catheter, may be indispensable to understand correctly the clinical condition of the neurologically complicated patients.

In this case, we found that an "inverse steal"-like phenomenon could be detected with near-infrared spectroscopy. This kind of interventional assessment of rSO_2 will possibly result in the development of another use for the instrument.

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