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INTRAOPERATIVE MONITORING DURING CAROTID CROSS-CLAMPING WITH NEAR INFRARED SPECTROSCOPY: A PRELIMINARY STUDY

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ABSTRACT

Near infrared spectroscopy (NIRS) is a noninvasive and real-time method for monitoring oxy-[HbO_2] and deoxyhemoglobin [Hb] in tissue, and is suitable for intraoperative monitoring. In this study, NIRS monitoring was performed on 10 patients during carotid cross-clamping. The data were analyzed with a theoretical cerebral hemoglobin model developed to identify an ischemic pattern using NIRS parameters. Temporal profiles of changes in [HbO_2] and [Hb] were divided into three phases: initial (immediately after clamping), second (during clamping), and last phase (immediately after clamp release). In the initial phase, [HbO_2] decreased and [Hb] increased in all the cases. In the second phase, recovery patterns of [HbO_2] were classified into three groups: complete (3 patients), incomplete (3 patients), and no recovery (2 patients). In the last phase, the [HbO_2] increased and [Hb] decreased. Relative changes in [HbO_2] and [Hb] measured by NIRS were correlated with changes in blood flow of the internal carotid artery (ICA) measured by a magnetic flowmeter and stump pressure of the internal carotid arteries. The degree of [HbO_2] decrease in the initial phase was significantly correlated with ICA blood flow before clamping (r=0.90, p<0.05). Three of the 4 patients with ICA stump pressure over 50 mmHg showed a complete recovery pattern in the second phase, while all 4 patients with ICA stump pressure under 50 mmHg showed an incomplete recovery or no recovery pattern with NIRS. These results suggest that NIRS is useful in evaluating changes in cerebral blood flow and the extent of hemodynamic reserve during carotid cross-clamping.

Keywords near infrared spectroscopy; hemoglobin; cerebral blood flow and metabolism; carotid endarterectomy; intraoperative monitoring.

1 INTRODUCTION

Near infrared spectroscopy (NIRS) is a method of monitoring oxy-[HbO_2] and deoxyhemoglobin [Hb] in tissue by measuring the emitted laser light, which is attenuated by absorption by these molecules according to the Beer-Lambert relationship of the concentration of chromophore and the attenuated light. Because of the noninvasive and real-time nature of this technology, NIRS is widely used in several clinical situations, such as monitoring neonates in hypoxia,[1] during surgery under cardiac arrest,[2] and during carotid endarterectomy.[3,4] It is also used for brain activation studies[5] or physiological studies[6] in healthy subjects.

However, interpretation of its measured values is not easy and is sometimes misleading. At the moment, NIRS parameters are represented as the relative changes of [HbO_2] and [Hb], and no reliable method has been reported that can correlate the NIRS data with the parameters of cerebral blood flow and metabolism. Therefore interpretation and validation of the NIRS parameters are usually carried out by simultaneously performing other techniques, such as ^{133}Xe clearance,[7] jugular bulb oximetry,[8] transcranial Doppler,[4] or positron emission tomography.[9] To evaluate the ischemic status of the brain by NIRS, the correlation between cerebral hemoglobin and the regional oxygen extraction fraction (rOEF) should be analyzed.

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For carotid endarterectomies, the usefulness of intraoperative monitoring techniques, including internal carotid artery (ICA) stump pressure measurements,[10,11] xenon rCBF studies,[12] transcranial Doppler,[13] electroencephalogram (EEG),[14] and somatosensory-evoked potentials[15] has been reported. The potential of NIRS methodology in endarterectomy was first demonstrated by the work of Ferrari et al. in which a carotid compression test on candidates for carotid endarterectomy showed a correlation between a slowing EEG and a fall in NIRS parameters (hemoglobin content and oxygenation).[16] NIRS could also register changes in cerebral oxygenation during carotid endarterectomy without significant contamination from extracranial tissues and showed a good correlation with the values from transcranial Doppler.[4]

In this study, NIRS monitoring was performed on 10 patients during carotid cross-clamping. The data were analyzed with a theoretical cerebral hemoglobin model developed to identify an ischemic pattern using NIRS parameters. Relative changes in $[HbO_2]$ and [Hb] measured by NIRS were correlated with changes in blood flow of the internal carotid artery measured by a magnetic flowmeter and stump pressure of the internal carotid arteries.

2. MATERIALS AND METHODS

2.1 PATIENTS AND OPERATIONS

NIRS monitoring was performed in 9 patients with carotid endarterectomy and in 1 patient with a subclavian-internal carotid artery bypass. All the patients had internal carotid artery stenosis with transient ischemic attacks (TIAs). A decreased hemodynamic reserve was confirmed by ^{99m}Tc-hexamethlypropyleneamine oxime single-photon emission computed tomography (^{99m}Tc-HMPAO SPECT) or xenon-enhanced computed tomography (Xe-CT). Preoperative CT scans did not show any ischemic lesions in the frontal lobe.

The operations were performed under general anesthesia and maintained mainly with fentanyl and a low dose of isoflurane (0.2%). After exposure of common, internal (ICA) and external carotid arteries, 1 ml of 1.0% xylocaine was injected in the carotid sinus, and 5000 U of heparin was given systematically. After measurements of stump pressure of the ICA, blood flow of the ICA was measured by a magnetic flowmeter (Nihon Kohden, Japan). EEG monitoring was also performed.

2.2 NIRS MEASUREMENT

A cerebral oxygenation measuring system, NIRO-500 (Hamamatsu Photonics K.K., Japan), was used to measure the concentration changes in oxy-$[HbO_2]$ and deoxyhemoglobin [Hb]. The NIRO-500 has four laser diodes (LD) for measurements with wavelengths of 775, 825, 850, and 904 nm. The light transmitted through the head is detected by a photomultiplier tube. Two optodes, one for emitting pulsed laser light and the other for detecting the attenuated light, were mounted on the ipsilateral forehead, avoiding close contact with blood vessels and muscles. The distance of these two optodes was set at 5 cm. An optical path length of 30 cm was calculated from the distance of the optodes assuming an optical path length factor of 5.93 for the adult head.[17] Sample time was set at 1 s. The data were collected with a personal computer and 1-min averages were graphically displayed and analyzed. The concentration of the total hemoglobin $[Hb_t]$ was calculated as the sum of $[HbO_2]$ and [Hb].

2.3 THEORETICAL ANALYSIS USING A COMPARTMENT MODEL

2.3.1 Hypothesis

The following hypothetical assumptions are made for the development of the model: (1) A sampled cerebral tissue is assumed to be optically homogeneous with the wavelengths of the light used. (2) No hemoglobin exists in the extravascular space. (3) Intravascular hemoglobin is classified into only two compartments of cerebral arterial and venous blood pool, each at a certain hemoglobin saturation. (4) Total hemoglobin concentrations are equal in the cerebral arterial and venous blood pool. (5) Cerebral arterial oxygen saturation (SaO_2) is equal to the systemic arterial oxygen saturation and remains constant during the measurement. (6) The existence of physically dissolved O_2 is negligible.

2.3.2 Definitions

First, the following values are defined as functions of time in a spectroscopically measured cerebral tissue.

Regional cerebral arterial blood volume: Vol_a (ml)

Regional cerebral venous blood volume: Vol_v (ml)

Regional cerebral tissue volume (including vascular volume): Vol_c (ml)

Second, the following values are defined as functions of time in the volumes defined above:

Regional cerebral arterial oxyhemoglobin concentration: $[HbO_2]_a$ (μmol/100 ml blood)

Regional cerebral venous oxyhemoglobin concentration: $[HbO_2]_v$ (μmol/100 ml blood)

Regional cerebral oxyhemoglobin concentration: $[HbO_2]_c$ (μmol/100 ml brain tissue)

Regional cerebral arterial deoxyhemoglobin concentration: $[Hb]_a$ (μmol/100 ml blood)

Regional cerebral venous deoxyhemoglobin concentration: $[Hb]_v$ (μmol/100 ml blood)

Regional cerebral deoxyhemoglobin concentration: $[Hb]_c$ (μmol/100 ml brain tissue)

Using these functions, the following values are further defined as functions of time:

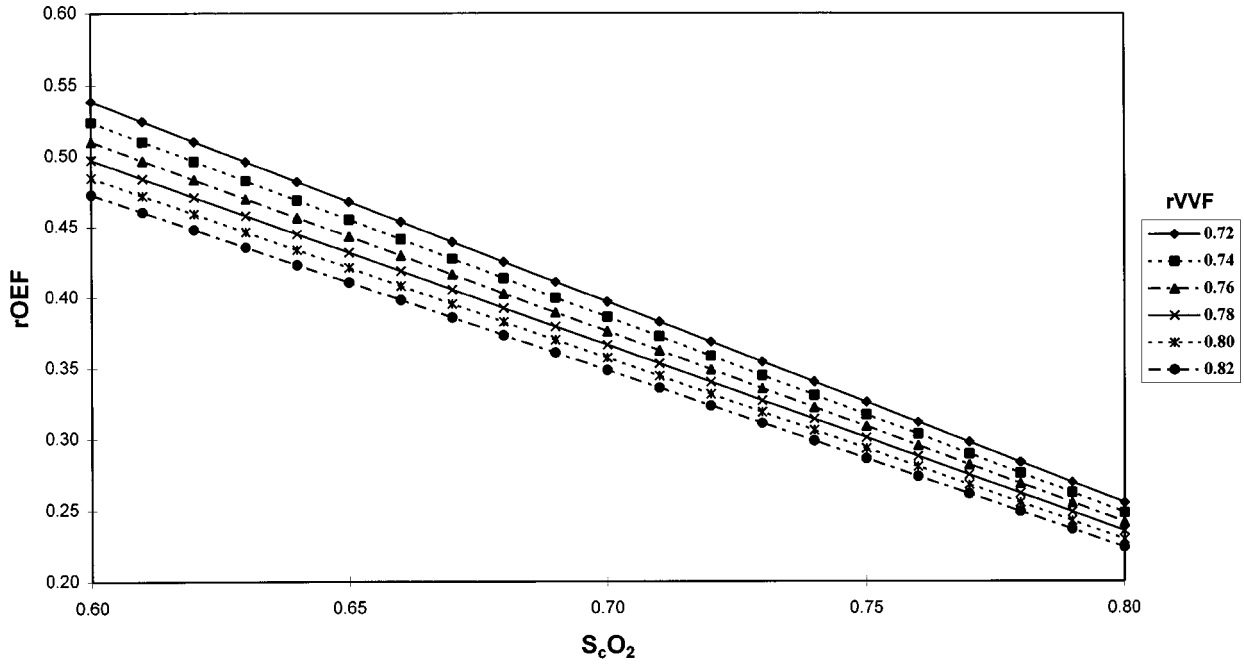

Fig. 1 Correlation between S_cO_2 and *r*OEF under different *r*VVF values (0.72 to 0.82). S_cO_2 and *r*OEF are linearly correlated under different *r*VVF values. S_aO_2=0.98 is adopted as a physiological value.

Regional cerebral blood volume fraction: $rCBV_f=(Vol_a+Vol_v)/Vol_c$

Regional cerebral venous blood volume fraction: $rVVF=Vol_v/(Vol_a+Vol_v)$

Regional cerebral oxygen extraction fraction (based on assumption 6):

$$rOEF=([HbO_2]_a-[HbO_2]_v)/[HbO_2]_a. \quad (1)$$

Regional cerebral arterial oxygen saturation:

$$S_aO_2=[HbO_2]_a/([HbO_2]_a+[Hb]_a) \quad (2)$$

Regional cerebrovascular oxygen saturation:

$$S_cO_2=[HbO_2]_c/([HbO_2]_c+[Hb]_c). \quad (3)$$

2.3.3 Calculations

From assumption (4) the following equation can be described:

$$[HbO_2]_a+[Hb]_a=[HbO_2]_v+[Hb]_v. \quad (4)$$

From the definitions above and assumption (3), the following equations were derived:

Regional cerebral oxyhemoglobin concentration:

$$[HbO_2]_c=rCBV_f\{[HbO_2]_a-rVVF([HbO_2]_a-[HbO_2]_v)\}. \quad (5)$$

Regional cerebral deoxyhemoglobin concentration:

$$[Hb]_c=rCBV_f\{[Hb]_a+rVVF([HbO_2]_a-[HbO_2]_v)\}. \quad (6)$$

Inserting Eqs. (5) and (6) into Eq. (3),

$$S_cO_2=\{[HbO_2]_a-rVVF([HbO_2]_a-[HbO_2]_v)/([HbO_2]_a+[Hb]_a)\}. \quad (7)$$

Inserting Eqs. (1) and (2) into Eq. (7):

$$rVVF\ rOEF=1-(S_cO_2/S_aO_2). \quad (8)$$

Using this equation, the correlation between the *r*OEF and S_cO_2 under different *r*VVF values is graphically displayed in Figure 1.

Inserting Eq. (3) into Eq. (8),

$$rVVF\ rOEF=([Hb]_c-(1/S_aO_2-1)\times[HbO_2]_c)/([HbO_2]_c+[Hb]_c). \quad (9)$$

When the baseline values are represented as $[Hb]_{c0}$, $[HbO_2]_{c0}$, $rVVF_0$, and $rOEF_0$, Eq. (9) is described based on assumption (5) as

$$rVVF_0\ rOEF_0=\{[Hb]_{c0}-(1/S_aO_2-1)\times[HbO_2]_{c0}\}/([HbO_2]_{c0}+[Hb]_{c0}). \quad (10)$$

Now it is necessary to introduce the following terms to represent relative changes in each parameter during a measurement:

Table 1 ICA stump pressure, ICA flow, NIRS parameters of the 10 patients.

Case No.	ICA stump pressure (mmHg)	Pre-clamping ICA flow (ml/min)	Post-clamping ICA flow (ml/min)	Initial decrease in $[HbO_2]$ (μmol)	Final increase in $[HbO_2]$ (μmol)	Recovery pattern of $[HbO_2]$ during clamping	Pattern of $[Hb_t]$ during clamping
1	100	30	160	–1	2	Complete	Increase
2	35	170	NA	–4.5	3.5	No	Decrease
3	37	120	190	–4	NA	NA	NA
4	100	100	100	–3.5	NA	NA	NA
5	70	400	380	–9.5	7	No	Decrease
6	40	180	240	–4	4	Incomplete	Increase
7	38	NA	NA	–5	4	Incomplete	Increase
8	50	80	90	–2	1	Complete	Increase
9	30	20	160	–3	6	Incomplete	Decrease
10	64	100	160	–3.5	3.5	Complete	Increase

Notes: NA=not measured or evaluated. Pre- and post clamping ICA flow values are those of a steady state after the probe application.
Initial decrease represents a difference between the value immediately before clamping and the maximal value within 2 min after clamping.
Final decrease represents a difference between the value immediately before release and the maximal value within 2 min after release.
Complete recovery is when the value recovered almost to the baseline or more during clamping.
No recovery is when the value recovered less than 30% of the initial decrease.
Incomplete recovery is when the value recovered more than 30% of the initial decrease.

$$\Delta(r\mathrm{VVF}\ r\mathrm{OEF})=(r\mathrm{VVF}\ r\mathrm{OEF})-(r\mathrm{VVF}_0\ r\mathrm{OEF}_0)$$

$$\Delta[\mathrm{Hb}]_c=[\mathrm{Hb}]_c-[\mathrm{Hb}]_{c0}$$

$$\Delta[\mathrm{HbO_2}]_c=[\mathrm{HbO_2}]_c-[\mathrm{HbO_2}]_{c0}.$$

From Eqs. (9) and (10) the following equation is derived:

$$\begin{aligned}\Delta(r\mathrm{VVF}\ r\mathrm{OEF})=(1/\mathrm{S_aO_2})([\mathrm{HbO_2}]_{c0}\Delta[\mathrm{Hb}]_c\\ -[\mathrm{Hb}]_{c0}\Delta[\mathrm{HbO_2}]_c)/([\mathrm{HbO_2}]_{c0}\\ +[\mathrm{Hb}]_{c0})([\mathrm{HbO_2}]_{c0}+[\mathrm{Hb}]_{c0}\\ +\Delta[\mathrm{HbO_2}]_c+\Delta[\mathrm{Hb}]_c).\end{aligned}\quad(11)$$

In this equation, there are only three variables, $\Delta[\mathrm{Hb}]_c$, $\Delta[\mathrm{HbO_2}]_c$, and $\Delta(r\mathrm{VVF}\times r\mathrm{OEF})$. The other terms are constant and positive. This allows us to obtain the following relations,

$$\Delta[\mathrm{Hb}]_c>0\ \text{ and }\ \Delta[\mathrm{HbO_2}]_c<0\rightarrow\Delta(r\mathrm{VVF}\ r\mathrm{OEF})>0$$

$$\Delta[\mathrm{Hb}]_c<0\ \text{ and}$$

$$\Delta[\mathrm{HbO_2}]_c>0\rightarrow\Delta(r\mathrm{VVF}\ r\mathrm{OEF})<0.$$

In these final equations, $\Delta[\mathrm{Hb}]_c$ and $\Delta[\mathrm{HbO_2}]_c$ are measurable by NIRS.

In this relations, if rVVF is constant during the measurement,

$$\Delta[\mathrm{Hb}]_c>0\ \text{ and }\ \Delta[\mathrm{HbO_2}]_c<0\rightarrow\Delta r\mathrm{OEF}>0$$

$$\Delta[\mathrm{Hb}]_c<0\ \text{ and }\ \Delta[\mathrm{HbO_2}]_c>0\rightarrow\Delta r\mathrm{OEF}<0.$$

These relations clearly indicate that reciprocal changes in $\Delta[\mathrm{Hb}]_c$ and $\Delta[\mathrm{HbO_2}]_c$ mean that there is a change in ΔrOEF.

3 RESULTS

No significant bleeding that would influence total hemoglobin values was observed during the carotid cross-clamping. In the early postoperative period, no complications and no aggravation of preexisting clinical symptoms were observed. Arterial oxygen saturation, systemic blood pressure, central venous pressure, and end-tidal CO_2 were maintained almost constant.

In 8 patients, the measurements were successful until the end, and allowed for exact analysis of the data. In the remaining 2 patients, apparent artifacts were recognized during the measurement, probably due to the movement of the optodes. The monitoring data are summarized in Table 1.

In this study, temporary profiles of cerebral hemoglobin signals during carotid endarterectomy

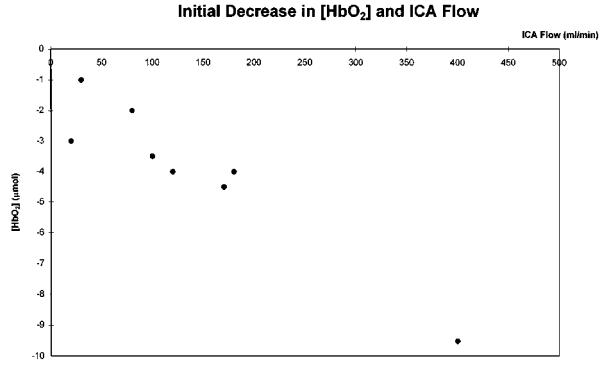

Fig. 2 Correlation between initial decrease in [HbO_2] and ICA blood flow before clamping. A significant correlation was observed (r=0.90, p<0.05).

were classified into three phases: the phase immediately after the clamping (the initial phase), the phase during the clamping (the second phase), and the phase immediately after clamp release (the last phase).

In the initial phase, immediately after carotid clamping, [HbO_2] and [Hb_t] decreased and [Hb] increased very rapidly within 10 s and then stabilized. This change was observed in all the patients. The degree of initial decrease in [HbO_2] was significantly correlated with ICA blood flow before clamping (r=0.90, p<0.05) (Figure 2); however, it did not correlate with the ICA stump pressure (r=0.01, p not significant) (Figure 3).

The second phase begins when the initial changes in the hemoglobin signals have stabilized and lasts until clamp release. According to the degree of recovery of [HbO_2] to the preclamping baseline level, three different patterns were observed in this phase: A complete recovery pattern of [HbO_2], which occurred when the value recovered almost to the baseline or more during clamping, was observed within 5 to 10 min after clamping in 3 patients [Figure 4(a)]. In these patients [HbO_2] values remained near the baseline level thereafter. An incomplete recovery pattern, when the value recovered by more than 30% of the initial decrease, was observed in 3 patients [Figure 4(b)]. No recovery pattern, which occurred when the value recovered by less than 30% of the initial decrease, was observed in the remaining 2 patients [Fig. 4(c)]. In one patient with the no recovery pattern, temporal slowing of the EEG was also observed and a barbiturate was given for cerebral protection against ischemia. The other case with this pattern was a patient with an arteriovenous malformation (AVM) in the ipsilateral frontal lobe. In 2 patients, a rapid decrease in [HbO_2] and their recovery in several minutes was observed [Figure 4(b)].

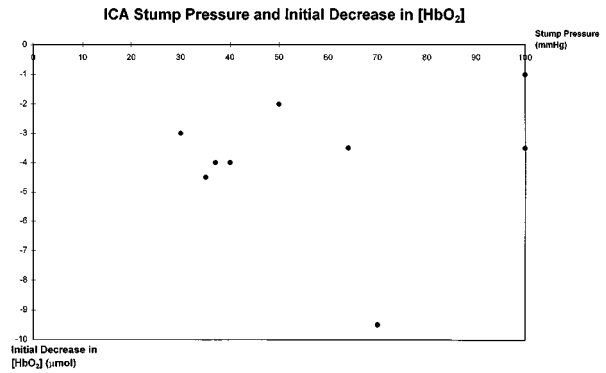

Fig. 3 Correlation between initial decrease in [HbO_2] and ICA stump pressure. No correlation was recognized (r=0.01).

With respect to correlation with ICA stump pressure, 3 of the 4 patients with ICA stump pressure over 50 mmHg showed a complete recovery pattern, while all 4 patients with an ICA stump pressure under 50 mmHg showed an incomplete recovery pattern or no recovery pattern with NIRS (Table 2). Also, 5 patients with an increase in [Hb_t] during the clamping showed a stump pressure over 35 mmHg, while, except for the patient with the AVM, 2 patients with a decrease in [Hb_t] showed a stump pressure under 35 mmHg (Table 3).

In the last phase, immediately after clamp release, [HbO_2] and [Hb_t] increased, and [Hb] decreased within 10 s, and then stabilized. These changes were observed in all 8 patients successfully measured until the end. In the majority of the patients, [HbO_2] overshot the preocclusion level, and a possible correlation between the degree of [HbO_2] increase and the ICA blood flow after clamp release was observed (r=0.58) (Figure 5).

4 DISCUSSION

4.1 THEORETICAL MODEL

Several assumptions are necessary in order to establish a cerebral hemoglobin compartment model to correlate the NIRS parameters with the conventional parameters of cerebral blood flow and metabolism. The first assumption (i.e., that the sampled cerebral tissue is optically homogeneous) is the basis for the interpretation of NIRS measurement. The second assumption (i.e., all the hemoglobin is in the intravascular space) seems to agree with reality in the absence of a subcutaneous or intracranial hematoma. The capillaries, in which the hemoglobin saturation may lie between that of arteries or veins, constitute less than 20% of the total cerebral vascular bed and oxygen exchange is almost instantaneous. Therefore, the third assumption (i.e., intravascular hemoglobin is classified into only two compartments) does not distort reality too much. Assumption 4 may be justified, because the difference of hematocrit values among the arteries, capillaries, and veins, although present, is much smaller than the difference of their hemoglobin saturations. Normally cerebral arterial oxygen saturation is equal to systemic arterial oxygen saturation. Arterial oxygen saturation may not be con-

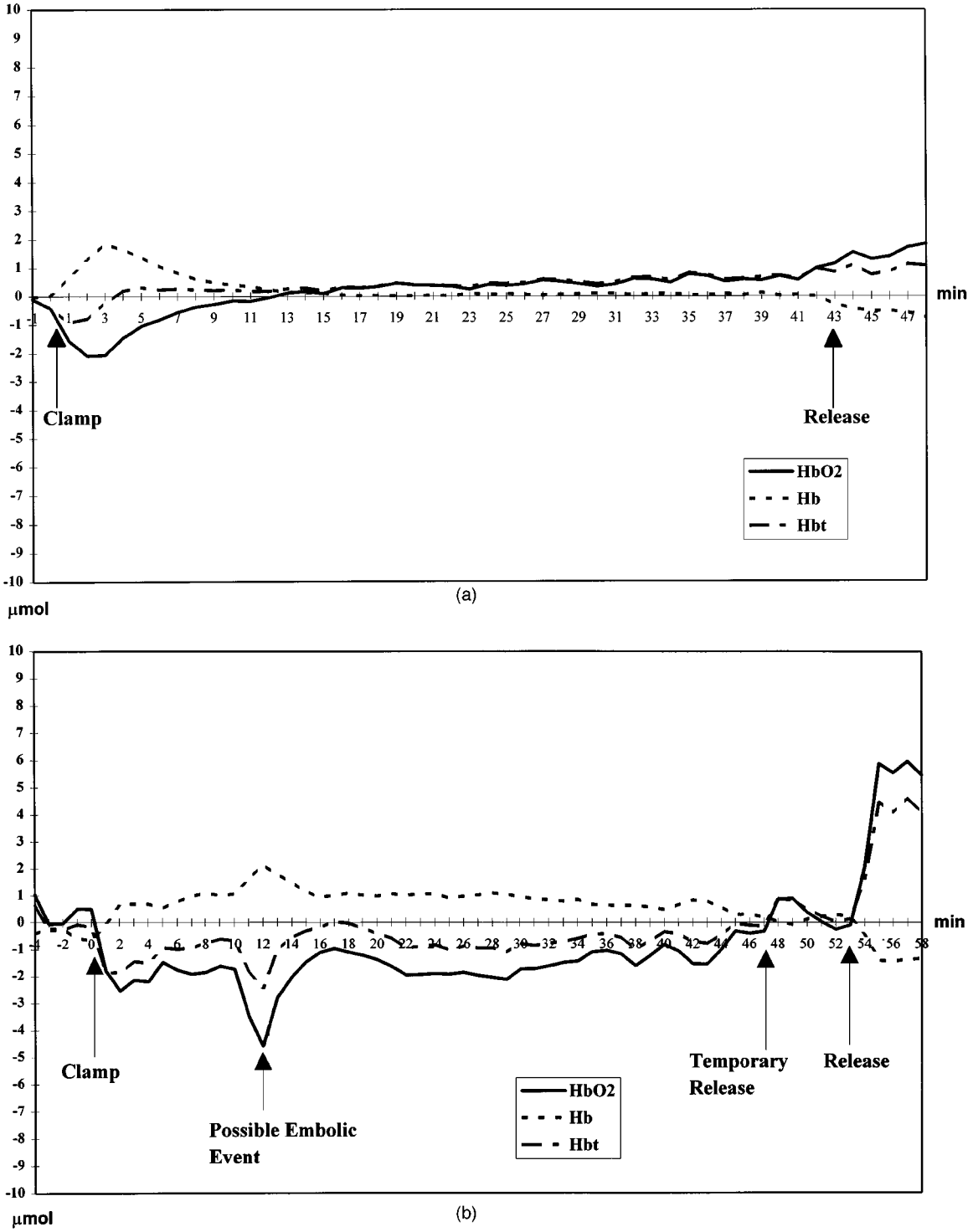

Fig. 4 (a) Case 8: A typical complete recovery pattern. In this case, a reciprocal movement of [HbO_2] and [Hb] was observed, which was followed by a rapid normalization. The systemic blood pressure was stable during the clamping. (b) Case 9: An incomplete recovery pattern. In this case, a significant increase in [HbO_2] was observed after release of clamping, which can be explained by the prominent improvement of blood flow of ICA (20 to 160 ml/min). Also, during the clamping, a transient decrease in [HbO_2] and recovery within several minutes was observed, which could be caused by a transient silent embolic event. (c) Case 2: A typical no recovery pattern. No tendency was observed for [HbO_2] to return to the preclamping baseline. In this case, a slowing of the EEG was recognized, and pentobarbital was given to protect the brain.

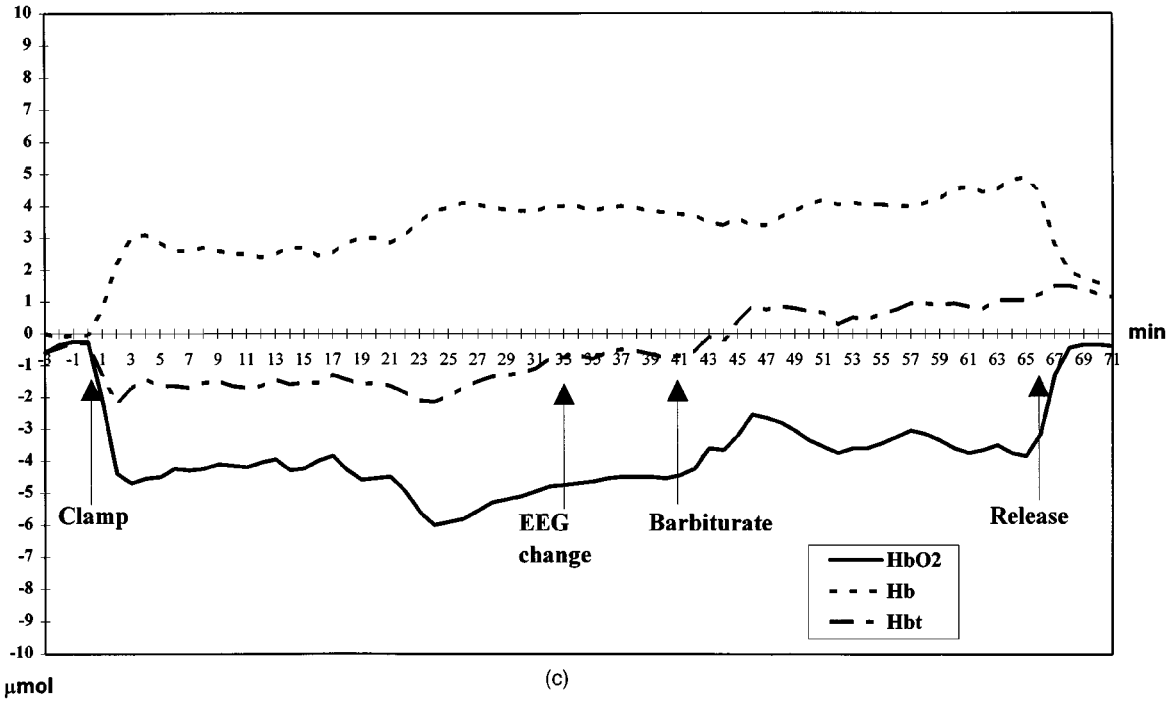

Fig. 4 (*Continued.*)

stant during a measurement, especially in patients with cardiopulmonary disease and subsequent hypoxia. For patients with normal cardiopulmonary function under general anesthesia, SaO_2 is generally stable and therefore the fifth assumption (i.e., cerebral arterial oxygen saturation is equal to the systemic arterial oxygen saturation and remains constant during the measurement) can be applied.

According to our cerebral hemoglobin model, a simultaneous increase in $[Hb]_c$ and a decrease in $[HbO_2]_c$ signifies an increase in the product of rVVF and rOEF. In the same way, a simultaneous decrease in $[Hb]_c$ and an increase in $[HbO_2]_c$ indicates a decrease in the product of rVVF and rOEF. These relations suggest that if rVVF is constant, the changes in rOEF may be directly estimated from the movement of $[Hb]_c$ and $[HbO_2]_c$, and if rCMRO$_2$ is also constant, the changes in rCBF can also be estimated. Thus, reciprocal movements of $[Hb]_c$ and $[HbO_2]_c$ represent an ''ischemic pattern'' or a ''recovery pattern.''

These discussions are based on the assumption that the impact of the change in the rVVF is reasonably small. However, no direct method is known that can evaluate rVVF at the moment, and there are very little hard data regarding the absolute value of this fraction and its consistency. Therefore, we have made a simulation study to evaluate the possible effect of the changes in rVVF on the correlation between S_cO_2 and rOEF. According to this simulation, S_cO_2 and rOEF are linearly correlated at constant rVVF values (Fig. 1). In this simulation, we

Table 2 Correlation between $[HbO_2]$ recovery pattern and ICA stump pressure.

	ICA stump pressure		
$[HbO_2]$ Recovery	>50 mmHg	<50 mmHg	Total
Complete	3	0	3
Incomplete	0	3	3
No recovery	1[a]	1[b]	2
Total	4	4	8

[a] The patient with an AVM in the ipsilateral frontal lobe.

[b] The patient with EEG abnormality during the clamping.

Table 3 Correlation between $[Hb_t]$ during carotid cross-clamping and ICA stump pressure.

	ICA stump pressure		
$[Hb_t]$	>35 mmHg	<35 mmHg	Total
Increase	5	0	5
Decrease	1[a]	2	3
Total	6	2	8

[a] The patient with an AVM in the ipsilateral frontal lobe.

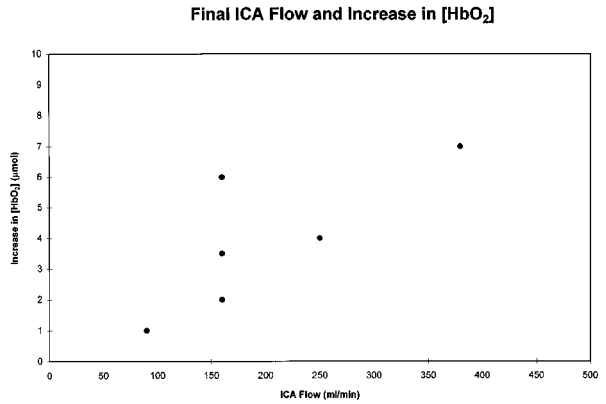

Fig. 5 Correlation between an increase in [HbO_2] and ICA blood flow after clamp release. A tendency for a positive correlation was observed (r=0.58; the number was too small for statistical analysis).

adopted two extreme values of rVVF (0.72 and 0.82).[5] If the shift of rVVF is limited between 0.72 and 0.82, the range of possible rOEF with S_cO_2=0.65 is 0.41 to 0.47; with S_cO_2=0.70, it is 0.35 to 0.40; and with S_cO_2=0.75, 0.29 to 0.33. These results suggest that a significant change in S_cO_2 would be likely to be caused by the change in rOEF and not by the change in rVVF.

A correlation between spectroscopically measured regional S_cO_2 and global cerebrovascular saturation measured by jugular bulb oximetry has also been reported using two extremes of the ratio of regional cerebral venous blood volume to regional total cerebral blood volume (rVVF of our model) (0.72 and 0.82).[8] The authors assumed from these results that distributional changes in blood volume do not significantly alter the relation of spectroscopically measured cerebrovascular oxygen saturation and suggested that NIRS measurements reflect changes in hemoglobin oxygenation mainly in mixed venous blood.

Although the impact of rVVF on this relationship seems small, it is not negligible. Furthermore, the movement of rVVF under pathological conditions has not been studied in detail. Therefore, further studies on the shifts in the relative size of the arterial and venous compartments under normal and pathological conditions are necessary for a more precise estimate of cerebral blood flow and metabolism using NIRS parameters.

4.2 MOVEMENT OF NIRS PARAMETER DURING CAROTID CROSS-CLAMPING

A rapid decrease in [HbO_2] and an increase in [Hb] after carotid cross-clamping have been reported.[4] The present study also confirms this report; the typical reciprocal changes in [HbO_2] and [Hb] were observed in all the cases and successfully measured. According to the theoretical analysis in the previous section, these reciprocal changes in [HbO_2] and [Hb] represent the ischemic status of the brain.

We have classified temporal profiles of [HbO_2] and [Hb] into three phases. In the initial phase, a significant correlation between changes in [HbO_2] and ICA blood flow measured by a magnetic flowmeter was observed (Fig. 2). These results suggest that the initial phase may represent the degree of decrease in cerebral blood flow caused by carotid cross-clamping.

In the second phase, changing patterns of [HbO_2] were individually different and could be classified into the following three groups: complete recovery, incomplete recovery, and no recovery patterns. This is a modification of the classification by Yamane et al.,[3] who proposed classification into no changes, no recovery, and recovery patterns. In the present study, their recovery pattern was further divided into complete and incomplete recovery to correlate with the stump pressure values. Their no change pattern was not observed, probably due to the small number of cases.

Although the number is not enough for statistical analysis, 3 patients with complete recoveries of [HbO_2] during carotid clamping showed a stump pressure that was more than 50 mmHg, while 3 patients with incomplete recoveries of [HbO_2] showed a stump pressure under 50 mmHg (Table 2). Since an ICA stump pressure of more than 50 mmHg is generally regarded as representing sufficient collateral circulation,[11] these correlations suggest that [HbO_2] recovery in the second phase may represent the amount of hemodynamic reserve during carotid cross-clamping. And, as shown in the results, the no recovery pattern, which indicates no hemodynamic reserve, may require special attention; a careful monitoring of EEG, and if necessary, elevation of blood pressure or administration of a barbiturate should be indicated. For the other case with a cerebral AVM, it is speculated that the brain had a smaller hemodynamic reserve due to the AVM.

Rapid reciprocal changes in [HbO_2] and [Hb], which recovered in several minutes, were observed during the second phase in 2 cases. Since no apparent changes were observed in the physiological parameters during these periods, these short-lasting ischemic changes could be caused by transient silent embolic events during carotid cross-clamping.

In the last phase, changes similar those in the initial phase were observed. Reciprocal changes in [HbO_2] and [Hb] were observed, and the degree of [HbO_2] increase had a possible correlation with post clamping ICA blood flow. This last phase represents the reperfusion status after clamp release. Therefore, the lack of an appropriate increase in [HbO_2] in the last phase would be a warning of a possible failure of revascularization and would require an immediate revision of the carotid artery.

These correlations of NIRS values with those of a magnetic flowmeter and ICA stump pressure sug-

gest that NIRS is useful in evaluating changes in cerebral blood flow and the extent of hemodynamic reserves during carotid cross-clamping.

REFERENCES

1. J. S. Wyatt, M. Cope, D. T. Delpy, S. Wray, and E. O. R. Reynolds, ''Quantification of cerebral oxygenation and haemodynamics in sick newborn infants by near-infrared spectrophotometry,'' *Lancet* **8515**, 1063–1066 (1986).
2. J. I. Ausman, P. W. McCormick, M. Stewart, G. Lewis, M. Dujovny, G. Balakrishnan, G. M. Malik, and R. F. Ghaly, ''Cerebral oxygen metabolism during hypothermic circulatory arrest in human,'' *J. Neurosurg*. **79**, 810–815 (1993).
3. K. Yamane, Y. Shima, and M. Okada, ''Validity of spectrophotometric monitoring of O_2 delivery to the cerebrum during carotid cross clamping at carotid endarterectomy,'' *J. Cereb Blood Flow Metab.* **13** (Suppl.) 831 (1993) (Abst.).
4. P. J Kirkpatrick, P. Smielewski, P. C. Whitfield, M. Czosnyka, D. Menon, and J. D. Pickard, ''An observational study of near-infrared spectroscopy during carotid endarterectomy,'' *J. Neurosurg.* **82**, 756–763 (1995).
5. A. Villringer, J. Planck, C. Hock, L. Schleinkofer, and U. Dirnagl, ''Near infrared spectroscopy (NIRS): a new tool to study hemodynamic changes during activation of brain function in human adults,'' *Neuroscience Lett*. **154**, 101–104 (1993).
6. C. E. Elwell, H. Owen-Reece, M. Cope, A. D. Edwards, J. S. Wyatt, E. O. R. Reynolds, and D. T. Delpy, ''Measurement of changes in cerebral haemodynamics during inspiration and expiration using near infrared spectroscopy,'' *Adv. Exp. Med. Biol*. **345**, 619–626 (1994).
7. O. Pryds, G. Greisen, L. L. Skov, and B. Friis-Hansen, ''Carbon dioxide-related changes in cerebral blood volume and cerebral blood flow in mechanically ventilated preterm neonates: comparison of near infrared spectrophotometry and ^{133}Xenon clearance,'' *Pediatr. Res*. **27**, 445–449 (1990).
8. P. W. McCormick, M. Stewart, M. G. Goetting, and G. Balakrishnan, ''Regional cerebrovascular oxygen saturation measured by optical spectroscopy in humans,'' *Stroke* **22**, 596–602 (1991).
9. Y. Hoshi, H. Onoe, Y. Watanabe, J. Andersson, M. Bergström, A. Lilja, B. Långström, and M. Tamura, ''Non-synchronous behavior of neuronal activity, oxidative metabolism and blood supply during mental tasks in man,'' *Neurosci. Lett.* **172**, 129–133 (1994).
10. W. S. Moore and A. D. Hall, ''Carotid artery back pressure: a test of cerebral tolerance to temporary clip occlusion,'' *Arch. Surg.* **99**, 702–710 (1969).
11. R. J. Hays, S. A. Levinson, and E. J. Wylie, ''Intraoperative measurement of carotid back pressure as a guide to operative management for carotid endarterectomy,'' *Surgery* **72**, 953–960 (1972).
12. T. M. Sundt, F. W. Sharbrough, R. E. Anderson, and J. D. Michenfelder, ''Cerebral blood flow measurements and electroencephalograms during carotid endarterectomy,'' *J. Neurosurg.* **41**, 310–320 (1974).
13. H. Benichou, P. Bergeron, M. Ferdani, J. M. Jausseran, M. Reggi, and R. Courbier, ''Pre- and intraoperative transcranial Doppler: prediction and surveillance of tolerance to carotid clamping,'' *Ann. Vasc. Surg.* **5**, 21–25 (1991).
14. C. Perez-Borja and J. S. Meyer, ''Electroencephalographic monitoring during reconstructive surgery of the neck vessels,'' *Electroencephalogr. Clin. Neurophysiol.* **18**, 162–169 (1965).
15. L. A. Jacob, S. D. Brinkman, R. M. Morrel, J. G. Shirley, and S. Ganji, ''Long-latency somatosensory evoked potentials during carotid endarterectomy,'' *Am. Surg.* **49**, 338–344 (1983).
16. M. Ferrari, E. Zanette, G. Sideri, I. Giannini, C. Fieschi, and A. Carpi, ''Effects of carotid compression, as assessed by near infrared spectroscopy, upon cerebral blood volume and haemoglobin oxygen saturation,'' *J. Roy. Soc. Med.* **80**, 83–87 (1987).
17. P. Van der Zee, M. Cope, S. R. Arridge, M. Essenpreis, L. A. Potter, A. D. Edwards, J. S. Wyatt, D. C. MaCormick, D. C. Roth, E. O. R. Reynolds, and D. T. Delpy, ''Experimentally measured optical pathlengths for the adult head, calf and forearm and the head of the newborn infant as a function of interoptode spacing,'' *Adv. Exp. Med. Biol.* **316**, 143–153 (1992).