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Reduction of regional cerebral oxygen saturation coincidental with a perioperative focal motor seizure

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Introduction

Continuous, real-time measurement of regional cerebral oxygen saturation (rSo_2) by near-infrared spectroscopy (NIRS) has attracted considerable attention as a simple and noninvasive method of assessing the state of intracerebral oxygenation [1–5]. However, clinical situations in which NIRS can provide quantitatively valid data may be limited because of several assumptions required in constructing algorithms that convert changes in optical density to hemoglobin saturation [5,6]. In addition, a considerable influence of extracranial oxygenation on NIRS signals has been suggested by several investigators [7,8]. Its clinical usefulness, therefore, does not appear to have been fully established. In this report, we describe a patient who developed seizure discharges in electroencephalography (EEG) and a simultaneous reduction of rSo_2 coincidental with focal motor seizure attacks, and we discuss the significance of the data acquired by NIRS.

Case report

A 48-year-old, 62-kg, 153-cm woman with a diagnosis of cancer of the cervix was scheduled to undergo radical hysterectomy. She had a history of hypertension diagnosed 3 years previously and had suffered from three previous left hemiparetic attacks, at 3 years 2 months, 1 year 5 months, and 8 months before admission. The only neurological deficit was a slight sensory disturbance in the left leg. The brain computed tomographic (CT) scan indicated lacunar infarctions at the bilateral basal ganglia. However, the Xe-enhanced CT scan failed to reveal any significant decreases in cerebral blood flow. In addition, cerebral arteriography did not reveal any significant stenotic lesions. She had no apparent history of epileptic attacks. In spite of the previous diagnosis of HT, the blood pressure preoperatively measured in the ward was 100–126/64–88 mmHg without any treatment. She was slightly anemic due to genital bleeding (Hb 10.6 g·dl^{-1}; under medication with ferrous citrate). Her family and life histories were unremarkable.

The patient was premedicated with nitrazepam (6 mg orally) and atropine (0.5 mg intramuscularly) 2 h and 30 min, respectively, before arrival in the operating room. After insertion of an epidural catheter via the L_2/L_3 interspace, anesthesia was induced with thiamylal (200 mg) and the trachea was intubated with the aid of vecuronium (6 mg). Anesthesia was subsequently maintained with O_2-N_2O-isoflurane or O_2-N_2O-epidural mepivacaine. Because of the previous episodes of cerebral ischemia, EEG activity and rSo_2 were monitored in addition to the standard anesthetic safety monitors; direct radial arterial pressure, heart rate, SpO_2 (arterial saturation of hemoglobin with oxygen measured by a pulse oximeter), $ETCO_2$ and temperatures were recorded every 1 min by a computer. The EEG activities of leads F_{p1} to A_1 and F_{p2} to A_2 (according to the International 10–20 System) were recorded continuously with a digital EEG machine (DAE-2000; Nihon Kohden, Tokyo, Japan) and stored on a magneto-optical disk for later analysis. The rSo_2 was monitored with a near-infrared spectrophotometer (INVOS 3100A Cerebral Oximeter, Somanetics, Troy, MI, USA). Since the previous episodes of left hemiparesis

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indicated the patient's vulnerability to right cortical ischemia, the spectrophotometer sensor was attached to the right forehead with adhesive and draped.

The operation was uneventful. The estimated blood loss was approximately 1200 g, and the hemoglobin concentration at the conclusion of the operation was 8.4 $g \cdot dl^{-1}$ after transfusion of 1 unit of packed red blood cells (RBC). No significant signs indicating cerebral ischemia were found in pupil size, EEG activity, on rSo_2 values during the operation; the rSo_2 had ranged from 53% to 58% without any apparent trend in its changes. The patient satisfactorily emerged from anesthesia 15 min after discontinuation of administration of the anesthetics; she was orientated, opening her eyes and responding to verbal commands. After further confirmation of the criteria for removal of the endotracheal tube and routine suctioning of the trachea, nasopharynx, and oral cavity, the patient's trachea was extubated. The patient could respond to verbal commands immediately after extubation. A few minutes after extubation, however, the anesthesiologist noticed repetitive bilateral blinking of the eyelids and upward bilateral deviation of the eyes with rapid diagonal nystagmoid movements. These manifestations developed several times, with durations of various lengths (1–5 min) for 15 min. The seizure activities were consistently localized to the bilateral eyelids and eyes, but were not generalized and were without apparent loss of consciousness. On the EEG recording, the rhythmic sharp waves first developed on the right side within 1 min after extubation (Fig. 1, upper panel, B) and subsequently developed on the left (Fig. 1, upper panel, C). The rSo_2 started to decrease immediately following the onset of the seizure discharges, falling to 44%, and the rSo_2 started to increase soon after disappearance of the seizure discharge, finally returning to the preseizure values (Fig. 1, lower panel). During the period when the seizure activity was observed, the patient's hemodynamics were stable except for slight tachycardia (110–120 $beats \cdot min^{-1}$) and hypertension (150/80 mmHg) observed for a few minutes following the extubation, and the SpO_2 was consistently 100%. However, arterial blood analysis made during the seizure revealed slight hypocarbia (pH 7.45, $PaCO_2$ 33.9 mmHg, PaO_2 412.8 mmHg [$FiO_2 = 1.0$, face mask fitted], base excess −0.1, respiratory rate 20–22 min^{-1}) and anemia (Hb 8.1 $g \cdot dl^{-1}$). The blood sugar level was 149 $mg \cdot dl^{-1}$, and any abnormality was not found in serum electrolyte concentrations. No abnormal findings were obtained on neurological examination performed 10 min after disappearance of the seizure attacks. The patient had a postictal amnesia. The patient was intensively observed for about 1 h in the operating room after disappearance of the seizures, receiving 2 units of packed RBC, and then transferred to the ward. No seizure attack was observed in the ward. Her postoperative recovery was uneventful, and she was discharged on the 35th postoperative day.

Discussion

In the present case, EEG monitoring revealed the development of rhythmic sharp waves immediately following extubation, suggesting that extubation triggered the seizures. In addition to slight hypocarbia and anemia, increases in cerebral oxygen consumption due to emergence from general anaesthesia might cause a significant imbalance between oxygen consumption and delivery in some regions susceptible to hypoxia, thereby triggering the seizures. However, the rSo_2, previously suggested as sensitive as analogue EEG to cerebral hypoxia [3], decreased following, but not prior to, appearance of the convulsive EEG waves, and there was no conclusive evidence that cerebral hypoxia triggered the seizures in our patient. The precise mechanisms of the seizures apparently evoked by extubation are therefore currently unknown.

The rhythmic sharp waves first developed on the right side, being consistent with the presumed vulnerability of the right hemisphere to cortical ischemia. The subsequent development of convulsive waves on the left indicates propagation of the seizure activities from the right to the left hemisphere. The observed eyelid blinking, tonic eye deviation, and rapid eye movement suggest that the precentral region (areas 4 and 6; center for eyelid movement) and/or the frontal eye field (area 8; center for conjugate eye movements) were epileptogenic. Therefore, the rSo_2 measured by attaching the spectrophotometer sensor to the right forehead might reflect the changes in oxygen saturation in these regions. Since the majority (about 70%) of blood within the cerebral circulation is venous, the rSo_2 signal is derived principally from the venous blood. The decreases in rSo_2 following development of convulsive waves on the EEG could therefore reflect the imbalance between oxygen consumption and delivery in the seizure foci due to increases in oxygen consumption as a result of excessive neuronal firing.

The cerebral oximeter designed for use in adults measures hemoglobin oxygen saturation in a relatively small area by using reflectance spectroscopy, an ipsilateral source detector system. Although the source-to-detector distance is known to determine the depth and distance of the highest-probability path traveled by the detected photons [4], the path taken by NIR light in reflectance spectroscopy is not yet clearly established [6,9]; therefore, the precise depth of tissue sampled by the cerebral oximeter with a given source-to-detector distance appears to be unknown. It was previously sug-

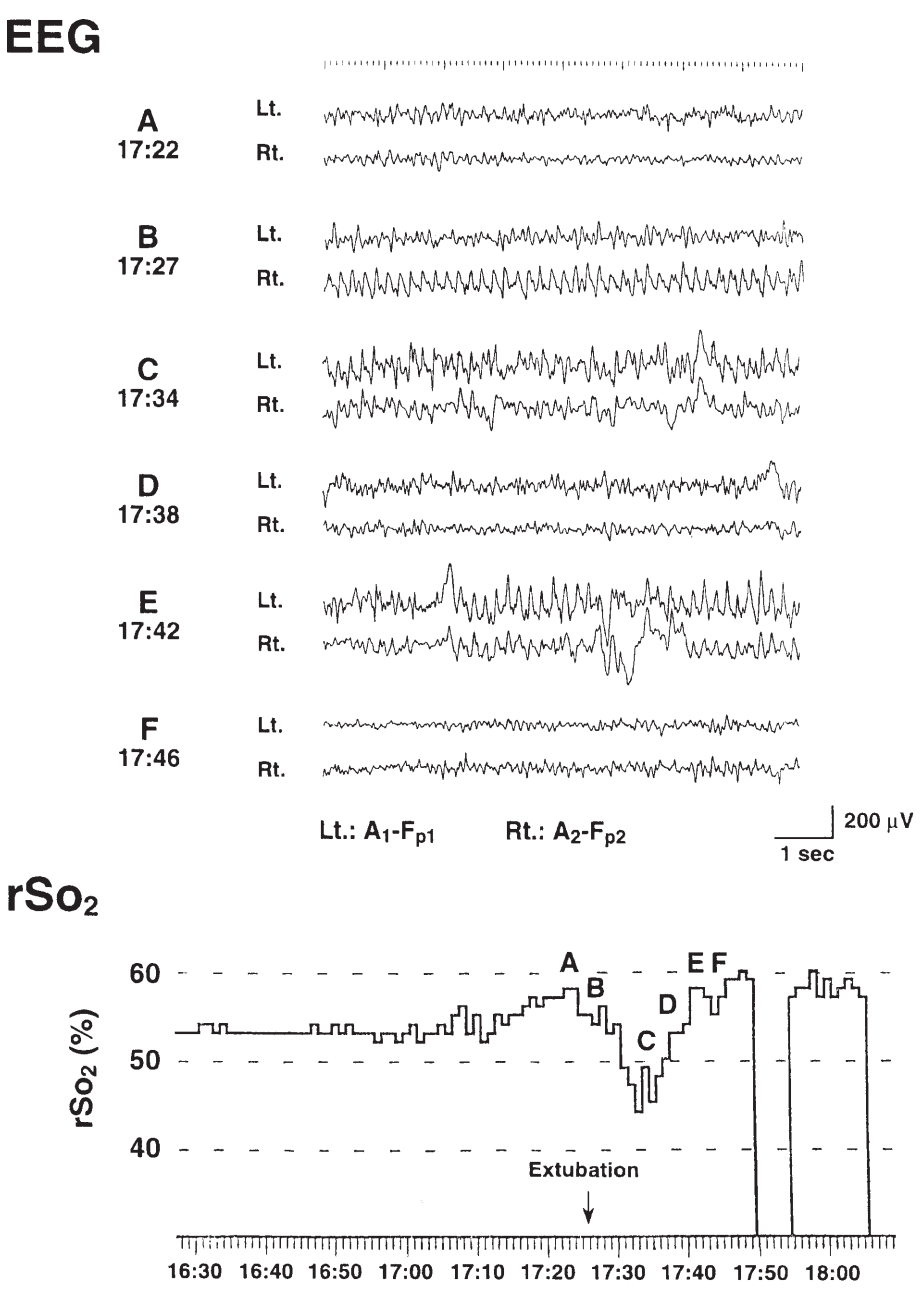

Fig. 1. Simultaneous recording of two-channel (lt., A_1-F_{p1}; rt., A_2-F_{p2}) electroencephalogram (*EEG*) (*upper*) and rSo_2 (*lower*) showing seizure discharges in EEG and concurrent decreases in rSo_2. The trachea was extubated at 17:26. A, B, C, D, E, and F show corresponding times in each recording. A: EEG shows no paroxysm before the extubation. Rhythmic sharp waves first develop on the right appoximately 1 min after extubation (B), propagating to the left side (C); rSo_2 concurrently decreases following the onset of the seizure discharges on EEG. rSo_2 gradually starts to increase after disappearance of the seizure discharges on the EEG (D); however, it again concurrently decreases following redevelopment of rhythmic sharp waves on the EEG (E). The rSo_2 measurement was interrupted for 5 min (from 17:50 to 17:55) while we checked for appropriate (tight) attachment of the spectrophotometrical sensor to the forehead by peeling it once from the forehead. No paroxysmal activities are seen on the EEG after 17:42 (F)

gested, on the basis of estimated scattering and absorption coefficients of the different tissues, that if the source-to-detector separation distance was less than 3–4 cm, the light would pass predominantly through extracranial tissues, whereas with increasing separation, an increasing proportion of the brain would be seen [9]. The cerebral oximeter used in this patient (Invos 3100) has two detectors, placed 3 and 4 cm from the source; in rSo_2 measurements with this oximeter, in order to reduce the influence of data from extracranial tissues and obtain a pure brain signal, the data derived from the detector nearer the source, which would predominantly reflect absorption events in the extracranial tissues, are subtracted from the data derived from the more distant detector [4]. Although the effectiveness of this system of spatial resolution in detecting changes in intracerebral oxygenation has been suggested [4,10,11], Germon et al. recently suggested that extracranial contamination still confounded the results obtained with the system using source-to-detector distances of 3 and 4 cm [12]. In

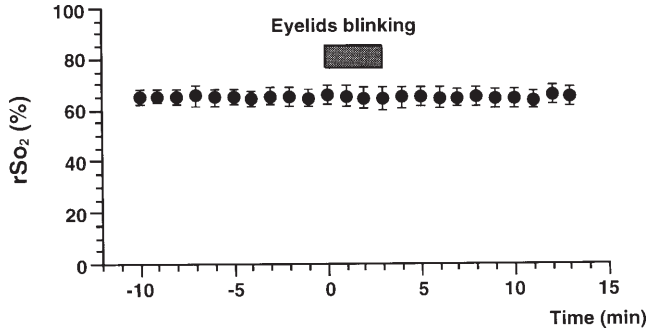

Fig. 2. Effects of intentional bilateral blinking of the eyelids on the rSo_2 values in four healthy volunteers. Time 0 indicates the start of blinking. No significant changes are observed during blinking; one-factor ANOVA with repeated measurements revealed no significant differences in the rSo_2 values throughout the study

addition, it was shown that the considerable extracranial contribution to the NIR signal was still noticeable at 5 to 7 cm separation [13]. Since the focal motor seizure developed at the eyelids very close to the forehead where the spectrophotometer sensor was placed, our concern in interpreting the obtained rSo_2 values was that they might reflect changes in extracranial oxygenation or blood flow at the forehead region, which could be altered by development of the seizure localized at the eyelids.

To address this possibility that eyelid movement (cyclic contraction of the orbicularis oculi muscle) itself might significantly affect the rSo_2 values measured by attaching the sensor to the forehead, we examined the effects of intentional bilateral blinking of the eyelids (or bilateral winks) on the rSo_2 values in four healthy volunteers aged 32 $\pm$ 3 (SEM) yr, weighing 61 $\pm$ 5 kg, and 170 $\pm$ 1 cm in height. After steady-state rSo_2 values had been measured for 10 min, the volunteers were asked to wink bilaterally as fast as possible and as greatly as possible for 3 min; the resultant frequency of the winks was about 3 Hz, and some motions were obviously caused by the winks in the superficial layer of the forehead region. However, no significant changes were observed in rSo_2 values during the winks (Fig. 2). Although the winks made by the volunteers do not completely simulate the rapid eyelid movement observed in our patient, the above results suggest that the changes in extracranial oxygenation or blood flow, or some motions at the forehead region possibly caused by the cyclic contraction of the orbicularis oculi muscle, did not influence the rSo_2 measurements in our patient. The decreases in rSo_2 values observed in our patient might therefore reflect changes in intracerebral oxygenation, based on the assumption that the volumes and proportions of arterial and venous blood remain constant during the seizure on which the algorithm used in the NIRS technology depends [6,9].

In summary, we have reported a patient who developed seizure discharges in EEG and a simultaneous reduction of rSo_2 coincidental with focal motor seizure attacks. Since the seizure developed at the eyelids close to the forehead to which the spectrophotometrical sensor was attached, we questioned the validity of the obtained rSo_2 data because of the possible contribution of changes in extracranial oxygenation or unavoidable motion in the superficial layers of the forehead region to the rSo_2 measurements during the seizure. To address this possibility that the eyelid movement itself might significantly affect the rSo_2 values, we examined the effects of intentional eyelid blinking on rSo_2 values in healthy volunteers. Eyelid blinking did not cause any significant changes in rSo_2 values in our volunteers. The observed decreases in rSo_2 might reflect changes in intracerebral oxygenation due to development of the seizure.

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