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Clinical paper

## Regional cerebral saturation monitoring during withdrawal of life support until death<sup>☆</sup>

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### ABSTRACT

**Aims:** The aim of this pilot study was to explore the regional cerebral oxygen saturation (rSO<sub>2</sub>) during the process of dying in Intensive Care Unit (ICU) patients in whom it was decided to withdraw life support.

**Methods:** Regional cerebral saturation was measured from the moment active treatment was stopped until the moment of death, defined as the onset of asystole. Simultaneously, heart rate and arterial blood pressure were recorded using a radial arterial catheter. Baseline rSO<sub>2</sub> values were calculated as mean values over one hour in stable haemodynamic conditions immediately after the decision to withdraw life support.

**Results:** Cerebral saturation was measured in six dying ICU patients. The mean age of patients was 64 year. The median baseline rSO<sub>2</sub> value was 64% (58%–68%). At time of death, median rSO<sub>2</sub> was 33% (7%–40%). The median decrease in rSO<sub>2</sub> from baseline until death was 31% (25%–45%). The median decrease in rSO<sub>2</sub> observed during the last hour before time of death was 20% (12%–31%).

**Conclusion:** A continuous and patient specific decrease in rSO<sub>2</sub> was observed in all patients with a simultaneous decrease in MAP. However, the absolute rSO<sub>2</sub> value at moment death was clinically determined, had a broad range, indicating that there is no clear cut-off rSO<sub>2</sub> value for death probably due to the heterogeneity of the studied population. Taken together, these observations highlight the importance of following trends and comparing rSO<sub>2</sub> values in the cardiac arrest setting.

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### Introduction

Near infrared spectroscopy (NIRS) is a technology used to measure regional cerebral oxygen saturation (rSO<sub>2</sub>). It provides non-invasive, continuous and real-time information. One or two light sources inserted in adhesive sensors and applied to the patient's forehead, emit near-infrared light through the cerebral cortex in a banana-shaped curve after which it is detected by two detectors [1]. According to the Lambert-Beer law, the degree of light absorption can be used to estimate the concentration of a substance [2,3]. In this manner, the difference between oxygenated

and deoxygenated haemoglobin in the capillaries, arterioles and venules of the frontal lobes is calculated which results in the measured rSO<sub>2</sub> values.

Currently, the use of this technique is gaining interest during cardiopulmonary resuscitation (CPR) in both the in-and out-of hospital setting [4–7]. It has been suggested that rSO<sub>2</sub>, both single rSO<sub>2</sub> measurements and changes in rSO<sub>2</sub> values, can be of added value to predict either the return of spontaneous circulation (ROSC) or favorable neurological outcome [4,5,8]. At arrival of the emergency team and start of advanced life support (ALS) after both in-and out-of hospital cardiac arrest (OHCA), the exact time point of collapse is often unknown. This uncertainty often raises the question whether to continue the initiated CPR efforts or whether efforts are futile. Therefore, it would be interesting to identify the changes in rSO<sub>2</sub> leading to cardiac arrest standstill. However, to date, the expected range of rSO<sub>2</sub> values in dying patients is unknown. This makes it difficult to interpret the initial measured rSO<sub>2</sub> values. In this study, rSO<sub>2</sub> was measured in intensive care unit (ICU) patients in whom it was decided to withdraw life support during the last 60 min before

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**Table 1**  
Patient demographics.

Patient number	Age	Sex	Diagnosis
1	76	Male	Pneumonia
2	53	Male	Cerebrovascular accident middle cerebral artery
3	65	Female	Pneumonia
4	55	Male	Liver failure
5	54	Female	Duodenal bleeding
6	83	Male	Respiratory failure

death was clinically determined in order to get a better understanding of the measured rSO<sub>2</sub> values in the cardiac arrest setting. Simultaneously, the change in invasively measured mean arterial pressure (MAP) was monitored and compared with the change in rSO<sub>2</sub>.

**Material and methods**

In this case series, rSO<sub>2</sub> was measured in ICU patients in who was decided to withdraw life support. The study protocol was approved by the local institutional review board (Commissie Medische Ethiek Ziekenhuis Oost-Limburg). Informed consent was obtained from patient’s next of kin.

Regional cerebral saturation measurement was initiated in ICU patients once the decision was made to stop active treatment, however before active treatment was actually terminated. Regional cerebral saturation was measured until the patient died using a portable cerebral oximeter (SenSmart™ Model X-100, Nonin Medical Inc, Plymouth, MN, USA and FORE-SIGHT™ technology, CAS Medical systems, Branford, CT, USA). Death was defined as the onset of asystole. A single sensor was placed on the right side of the forehead of the patient. Together with the rSO<sub>2</sub> measurement, heart rate and arterial blood pressure, measured with an arterial catheter in the radial artery, were monitored. Cerebral saturation values in a healthy population are between 60 and 80% [10]. None of the patients had cerebral pathologies, such as intracranial bleeding, traumatic brain injury, encephalitis or a brain tumor in the region where rSO<sub>2</sub> was measured. Also, none of the patients were brain death at the initiation of the study.

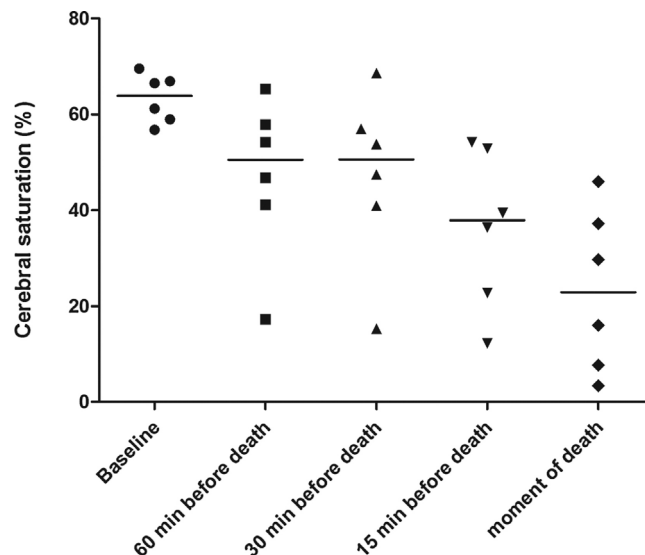
Baseline rSO<sub>2</sub> value was calculated as mean value over one hour in stable haemodynamic conditions immediately after the decision of withdrawal of life support, before active treatment was stopped. Cerebral saturation values at one hour, 30 min and 15 min before death and at the moment of death were calculated as means of periods of 60 s around these specific time points.

*Statistical analysis*

Cerebral saturation values are presented as mean and median with standard deviation and interquartile range respectively. A Pearson correlation test was performed to assess the correlation between rSO<sub>2</sub> and MAP. Differences are considered significant when p-value < 0.05. Statistical analysis was performed using SPSS

**Table 2**  
Baseline cerebral saturation, pulse oximetry, arterial oxygen pressure and arterial carbon dioxide pressure.

Patient number	rSO <sub>2</sub> (%)	SpO <sub>2</sub> (%)	PaO <sub>2</sub> (mmHg)	PaCO <sub>2</sub> (mmHg)	MAP (mmHg)
Patient 1	61	90	136	26,3	81
Patient 2	67	96,3	98,8	40	56
Patient 3	67	91,1	73	64,8	31
Patient 4	57	100	52,9	32,1	69
Patient 5	70	97,8	60,7	29,2	80
Patient 6	59	92,8	67,8	53,5	80



**Fig. 1.** Median cerebral saturation value at different time points.

22.0 (IBM® SPSS® Inc.; Chicago, IL, USA). Figures were made using GraphPad Prism 5.01 (GraphPad Software, La Jolla, CA, USA).

**Results**

In this case series, rSO<sub>2</sub> was measured in six patients who died in the ICU. Four (67%) male patients were included. The mean age of patients was 64 years. Demographic data of the patients are presented in Table 1.

In all patients, rSO<sub>2</sub> could be measured at the right side of the forehead. A baseline value was calculated in stable conditions as the mean rSO<sub>2</sub> over one hour, before active treatment was stopped. The median baseline rSO<sub>2</sub> value was 64% (58%–68%). Median baseline SpO<sub>2</sub> value was 95% (91%–97%), median baseline PaO<sub>2</sub> values were 70 mmHg (62 mmHg–92 mmHg) and median baseline PaCO<sub>2</sub> value was 36 mmHg (30 mmHg–50 mmHg). Individual oximetry data are presented in Table 2. The median rSO<sub>2</sub> measured one hour before death was 50% (35%–60%). At the time of death, median rSO<sub>2</sub> was 33% (7%–40%) (Fig. 1). The median decrease from baseline until death was 31% (25%–45%). The median decrease observed during the last hour before death was 20% (12%–31%). For all patients, the rSO<sub>2</sub> course of the last hour before death is presented for every patient in Fig. 2. Cerebral saturation and MAP were positively correlated, calculated during the last hour before death was clinically determined (r between 0.722–0.968; p < 0.01) (Fig. 3). Fig. 4 represents the rSO<sub>2</sub> and MAP evolution in two patients.

**Discussion**

This is the first observational study in which the evolution of rSO<sub>2</sub> was recorded in the hour before death was clinically determined. A continuous and patient specific decrease in rSO<sub>2</sub> was observed in all patients along with a simultaneous decrease in MAP.

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