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## Clinical paper

# Usefulness of cerebral rSO<sub>2</sub> monitoring during CPR to predict the probability of return of spontaneous circulation



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## Abstract

**Background:** Cerebral oximetry (rSO<sub>2</sub>) may be useful in assessing the probability of return of spontaneous circulation (ROSC). However, the potential of assessing the trend in the rSO<sub>2</sub> value has not been discussed when determining the probability of ROSC.

**Methods:** This was a retrospective study of out-of-hospital cardiac arrest (OHCA) patients with continuous rSO<sub>2</sub> values recorded during cardiopulmonary arrest. We used logistic regression analysis at each time point to investigate the best subsets of rSO<sub>2</sub>-related variables for ROSC, which included rSO<sub>2</sub> (baseline), the baseline value of rSO<sub>2</sub>; amount of maximum rise, the maximum difference of rSO<sub>2</sub> from rSO<sub>2</sub> (baseline) over *t* minutes;  $\Delta rSO_2(t)$ : (amount of maximum rise)/rSO<sub>2</sub> (baseline) over *t* minutes after hospital arrival.

**Results:** Among the 90 included patients, 35 achieved ROSC. Area under the curve (AUC) analysis revealed that  $\Delta rSO_2$  over a 16-min measurement period was significantly higher than  $\Delta rSO_2$  measured over 4-, 8-, 12-, and 20-min periods. During this 16-min period, the subset showing the best AUC value was interaction of the amount of maximum rise and rSO<sub>2</sub> (baseline) rather than the amount of maximum rise or  $\Delta rSO_2$  alone (AUC = 0.91).

**Conclusions:** The combination of rSO<sub>2</sub> (baseline) with the amount of maximum rise in rSO<sub>2</sub> value over time might be a new index for the prediction of ROSC that could be useful in guiding cardiopulmonary resuscitation. Further studies are needed to validate these findings.

**Keywords:** Amount of rise, Cardiopulmonary, Arrest, Cardiopulmonary resuscitation, Regional cerebral oxygen saturation, Return of spontaneous circulation

## Introduction

It is difficult to determine when cardiopulmonary resuscitation (CPR) should be stopped in patients in cardiopulmonary arrest (CPA). Determining the probability of the return of spontaneous circulation (ROSC) during CPR can help guide the provision of resuscitation efforts. Cerebral oximetry, i.e., the measurement

of regional cerebral oxygen saturation (rSO<sub>2</sub>), has recently been advanced as a promising tool for this purpose. However, the potential for assessing the trend of the rSO<sub>2</sub> value has not been discussed when determining the probability of ROSC. rSO<sub>2</sub> monitoring differs from pulse oximetric saturation (SpO<sub>2</sub>) monitoring in that rSO<sub>2</sub> data can be measured without the necessity of arterial pulsation. Therefore, it is possible to conduct continuous monitoring in CPA patients.

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Many research groups have examined the role of cerebral rSO<sub>2</sub> during cardiac arrest. A higher rSO<sub>2</sub> or increases of rSO<sub>2</sub> have been shown to be strongly associated with ROSC and survival with favorable neurological outcome.<sup>1–4</sup> We have previously reported on the serial changes observed in cerebral rSO<sub>2</sub> values during resuscitation in patients with out-of-hospital cardiac arrest (OHCA). Although chest compression by itself could not increase the cerebral rSO<sub>2</sub> value, ROSC and the initiation of extracorporeal CPR increased the cerebral rSO<sub>2</sub> value.<sup>5,6</sup> In ROSC patients with good neurological outcome, cerebral rSO<sub>2</sub> values promptly increase after ROSC and then gradually decrease thereafter, possibly because cerebral oxygen consumption increases following reperfusion.<sup>7</sup>

Most studies have examined the association between the types of near-infrared spectroscopy (NIRS) measures of rSO<sub>2</sub> (initial, mean, and highest values), resuscitation outcomes (ROSC, survival to discharge, and good neurological outcome), and quality of CPR in CPA patients.<sup>3,8–11</sup> However, we believe that the serial change (in the trend and pattern) of rSO<sub>2</sub> values by continuous measurement seems to be the most important index. Therefore, the purpose of this study was to evaluate the relationship between the rate or amount of rSO<sub>2</sub> increase in patients in CPA undergoing CPR on hospital arrival and the occurrence of ROSC to search for new criteria for the prediction of ROSC.

## Methods

### Patients and study protocol

This was a retrospective, single-centre, observational study performed in the Department of Traumatology and Acute Critical Medicine of Osaka University Graduate School. The inclusion criterion was patients aged  $\geq 16$  years old with OHCA who were admitted to our hospital from December 2012 to December 2015.

All patients with or without bystander CPR underwent manual chest compression delivered by emergency life-saving technicians (ELTs). After arriving at the hospital, the patients underwent manual chest compression delivered by the medical staff until load-distributing-band CPR (LDB-CPR) was started. The rSO<sub>2</sub> sensor connected to a TOS-OR device (FUJITA MEDICAL INSTRUMENTS CO., LTD., Tokyo, Japan) was attached to the patients' forehead within 1 min of their arrival in the emergency room. The TOS-OR device measures the oxygen saturation based on the Beer-Lambert law, as do the devices of other companies, by using three different wavelengths of near-infrared LED light that have specific absorbances in oxyhaemoglobin and deoxyhaemoglobin. Tracheal intubation was performed in tandem with chest compression before LDB-CPR if not already completed by the ELTs. Patients underwent LDB-CPR by mechanical chest compression if they were intubated prior to hospital arrival. Rhythm analysis and pulse check were performed every 2 min. If the rhythm was shockable, defibrillation was performed during LDB-CPR. Adrenaline was administered every 4 min. All patients were treated according to the strategy of the Japan Resuscitation Council 2010 guidelines, which are based on the guidelines of the AHA and the International Liaison Committee on Resuscitation (ILCOR).<sup>12</sup> The medical staff could see the rSO<sub>2</sub> values during CPR, but they did not change the treatment according to the rSO<sub>2</sub> data.

### Data collection and method of calculation

We reviewed rSO<sub>2</sub> data, patient characteristics including sex, age, witness status, bystander CPR, initial rhythm on ELT arrival and on

hospital arrival, the number of adrenaline administrations and defibrillation shocks delivered during prehospital and hospital care, ROSC recorded in the medical chart, and each patient's cerebral rSO<sub>2</sub> data from records of the patients admitted to our hospital from December 2012 to December 2015.

We assessed rSO<sub>2</sub> (baseline), rSO<sub>2</sub> (t) max,  $\Delta$ rSO<sub>2</sub> (t), and 'amount of maximum rise' in this study. rSO<sub>2</sub> (baseline) indicates the baseline rSO<sub>2</sub> value, which was calculated as the average of rSO<sub>2</sub> values measured during the initial 1 min of recording. rSO<sub>2</sub> (t) max indicates the maximum rSO<sub>2</sub> value measured over a t-minute period.  $\Delta$ rSO<sub>2</sub> (t) indicates the rate of change in the rSO<sub>2</sub> value and was calculated as follows:

$$\Delta rSO_2 (t) = \{rSO_2 (t) \text{ max} - rSO_2 (\text{baseline})\} / rSO_2 (\text{baseline}) \times 100.$$

$\Delta$ rSO<sub>2</sub> was calculated over periods of 4, 8, 12, 16, and 20 min as  $\Delta$ rSO<sub>2</sub> (4),  $\Delta$ rSO<sub>2</sub> (8),  $\Delta$ rSO<sub>2</sub> (12),  $\Delta$ rSO<sub>2</sub> (16), and  $\Delta$ rSO<sub>2</sub> (20), respectively. The amount of change was calculated as 'amount of maximum rise from rSO<sub>2</sub> (baseline)' as follows: Amount of maximum rise over t minutes = rSO<sub>2</sub> (t) max – rSO<sub>2</sub> (baseline)

This study to monitor cerebral rSO<sub>2</sub> in patients with OHCA required nothing different from the standard approach and management of cardiac arrest conducted in our facility. This study was approved by the Ethics Committee of Osaka University Graduate School of Medicine (No. 12,446). The local institutional review board waived the need for informed consent because all subjects were in CPA.

### NIRS rSO<sub>2</sub> monitoring system

A TOS-OR rSO<sub>2</sub> monitor (FUJITA MEDICAL INSTRUMENTS CO., LTD.) was used to measure cerebral rSO<sub>2</sub> values in this study. Three different wavelengths of near-infrared LED light are passed through the skin to a depth of approximately 3 cm, and the wavelengths of the reflected light are sensed by a photodiode. The reflected lights mainly represent the haemoglobin information in the cerebral cortex. The system can measure rSO<sub>2</sub> data every second without the need for arterial pulsation, thus allowing continuous monitoring of the rSO<sub>2</sub> values of CPA patients. Two rSO<sub>2</sub> values, left side and right side, are acquired continuously, and then the average of the two values is calculated. The normal range of cerebral rSO<sub>2</sub> on room air was previously determined from 15 healthy adult volunteers (10 men, 5 women, 43.2  $\pm$  8.9 years) to be 71.2  $\pm$  3.9%.<sup>6</sup>

### Statistical analysis

Continuous data are shown as the median with interquartile range and were compared using the Wilcoxon rank sum test. Categorical variables are expressed as proportions and were compared using the Pearson  $\chi^2$  test. The ability of rSO<sub>2</sub> to discriminate between cases of ROSC and non-ROSC was determined using the area under the receiver operating characteristic curve (AUC-ROC) of a multivariable logistic regression model that included rSO<sub>2</sub>-related variables and a set of covariates chosen *a priori* that were based on clinical relevance including age, sex, total number of electrical defibrillations and administrations of adrenaline, initial rhythm on ELT arrival, bystander CPR, and with or without witnessed arrest. Furthermore, to assess whether the initial rSO<sub>2</sub> value affects the predictive ability of the

multivariable logistic regression model, a cross-product term between baseline  $rSO_2$  and the amount of maximum rise of  $rSO_2$  was included along with the main effect of both variables along with the set of covariates. The model was also adjusted for non-linear associations between ROSC and  $rSO_2$  that were assessed by including restricted cubic splines in the model.

The continuous net reclassification improvement (cNRI) and integrated discrimination index (IDI) values were calculated to assess the incremental predictive ability of the initial value of  $rSO_2$  when added to the model using the amount of maximum rise. The cNRI was estimated by combining proportions of improved reclassification between cases of ROSC and non-ROSC. The IDI was used to quantify the actual change in calculated risk of ROSC for each individual for the patients with ROSC and non-ROSC. A two-sided  $\alpha$  of 0.05 was used to indicate statistical significance. All statistical analyses were performed with R software, version 3.4.3 ([www.r-project.org](http://www.r-project.org)).

## Results

Table 1 shows the characteristics of the 90 patients with OHCA, of whom 35 patients achieved ROSC. There were no significant differences in the variables of age, sex, initial rhythm on ELT arrival, and with or without bystander CPR between the ROSC and non-ROSC groups. However, the existence of a witness and the total amount of epinephrine administered were significantly different between the two groups.

Fig. 1 shows the AUC-ROCs. ROC curve analysis showed that the AUC of  $\Delta rSO_2$  was highest during the 16-min period (i.e.,  $\Delta rSO_2(16)$ ), with the AUC values of  $\Delta rSO_2$  being 0.65, 0.68, 0.71, 0.72, and 0.70 for the 4-, 8-, 12-, 16-, and 20-min periods, respectively. However, when comparing  $\Delta rSO_2(16)$  with the amount of maximum rise from baseline occurring during the 16-min period (i.e.,  $rSO_2(16)_{\max} - rSO_2(\text{baseline})$ ), the ROC-AUC for the amount of maximum rise was higher, at 0.75 (Fig. 1; 16 min proposed). In short, the amount of maximum rise measured during the 16-min period was a better

predictive factor for ROSC than was  $\Delta rSO_2$ . However, the result shown in Fig. 1 might be affected by the baseline  $rSO_2$  value.

Therefore, we plotted the ROC curve using both the baseline  $rSO_2$  and the amount of maximum rise (Fig. 2; (model 3)), which resulted in an AUC-ROC of 0.91. In addition, we compared AUC-ROCs between the 3 different models (Fig. 2). All ROCs were adjusted for the variables of age, sex, total number of electrical defibrillations and administrations of adrenaline, initial rhythm on ELT arrival, bystander CPR, and with or without witness.

We also analysed the relation between  $rSO_2$  (baseline) and the amount of maximum rise from baseline  $rSO_2$  over the 16-min period. Fig. 3 shows the logistic regression analysis for this relation between the amount of maximum rise and the predicted probability of ROSC separately for the baseline  $rSO_2$  values of 30%, 40%, and 50%. These models were also adjusted for the same factors mentioned above. We used initial rhythm on ELT arrival to create these models because we thought that this rhythm, when compared with that on hospital arrival, better reflected the patient's initial status. As Fig. 3 shows, to attain an 87.5% probability of achieving ROSC, the  $rSO_2$  value must rise by 10% at 16 min for patients with a baseline  $rSO_2$  value of 50%, by 20% at 16 min for patients with a baseline  $rSO_2$  value of 40%, and by 35% at 16 min for the patients with a baseline  $rSO_2$  value of 30%. Table 2 shows the values of cNRI and IDI for the initial value of  $rSO_2$  added to the model using the amount of maximum rise (model 3). Adding the baseline  $rSO_2$  model led to a significant improvement in reclassification (cNRI = 0.774,  $p < 0.05$ ; IDI = 0.0839,  $p < 0.05$ ). Supplementing these findings, Fig. 4 shows a scatter plot of the predicted probabilities for ROSC when comparing model (2) using only the amount of maximum rise with model (3) using both the baseline value and the amount of maximum rise.

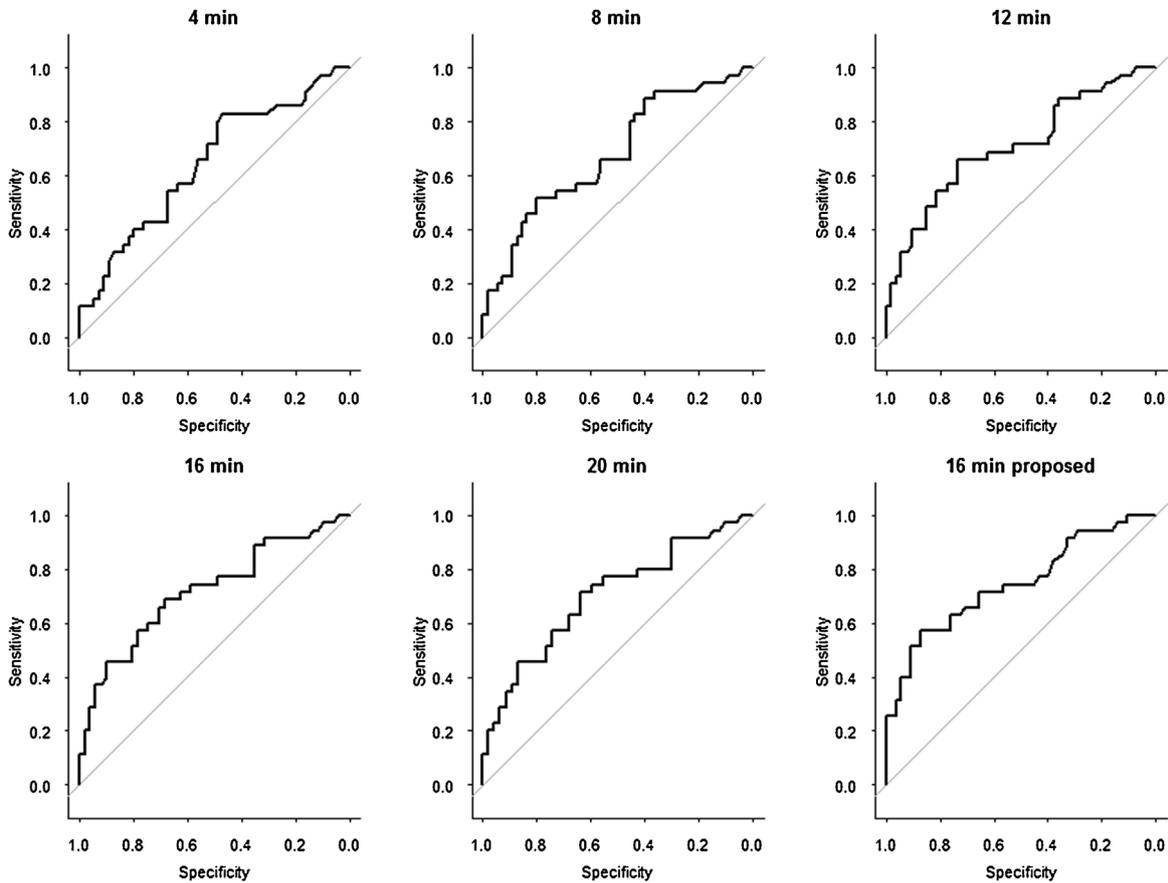
## Discussion

In the present study, we showed the relation between the increasing ratio of cerebral  $rSO_2$  values and ROSC (Fig. 1). ROC curve analysis

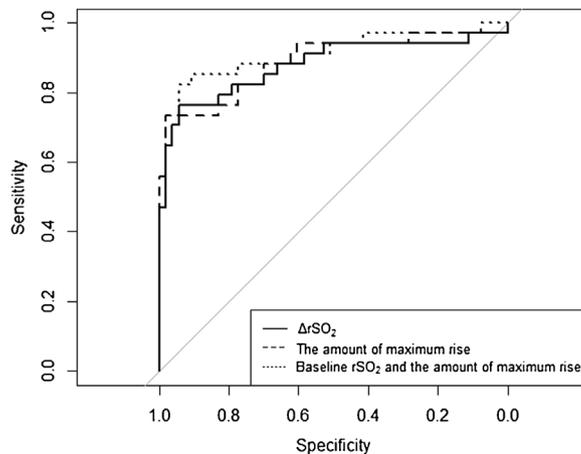
**Table 1 – Characteristics of patients with out-of-hospital cardiac arrest (N = 90).**

Variables	ROSC (N = 35)	Non-ROSC (N = 55)	P value
Age median (IQR)	73 (68-82)	77 (69-85)	0.61
Male n (%)	24 (68.6%)	35 (63.6%)	0.63
Initial rhythm on ELT arrival			0.06
Ventricular fibrillation, n (%)	3 (8.6)	4 (7.3)	
PEA, n (%)	16 (45.7)	13 (23.6%)	
Asystole, n (%)	15 (42.9%)	38 (69.1%)	
Unknown, n (%)	1 (2.9%)	–	
Initial rhythm on admission			0.38
Ventricular fibrillation, n (%)	0	3(5.5%)	
PEA, n (%)	10 (28.6%)	15 (27.3%)	
Asystole, n (%)	24 (68.6%)	37 (67.3%)	
Unknown, n (%)	1 (2.8%)	0	
Witness, n (%)	22 (62.9%)	18 (32.7%)	0.005
Bystander CPR, n (%)	16 (45.7%)	21 (38.2%)	0.48
Total number of electrical defibrillations, median (IQR)	0 (0-0)	0 (0-0)	0.68
Total mg of adrenaline administration, median (IQR)	3 (2-5)	5 (5-7)	<0.01

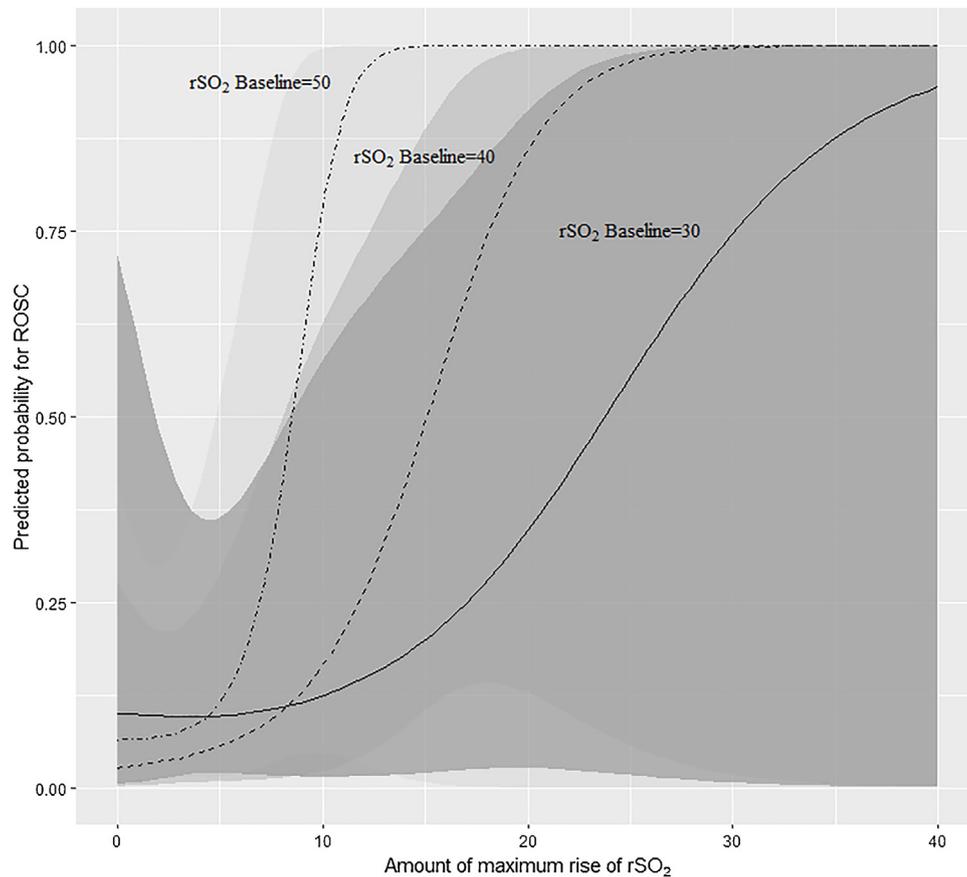
ROSC, return of spontaneous circulation; IQR interquartile range; ELT, emergency life-saving technician; PEA, pulseless electrical activity; CPR, cardiopulmonary resuscitation.



**Fig. 1 – Areas under the receiver operating characteristic curve (AUC-ROC). The rate of change of rSO<sub>2</sub> ( $\Delta$ rSO<sub>2</sub>) was calculated over 4-, 8-, 12-, 16-, and 20-min periods from initiation of rSO<sub>2</sub> measurement. ROC curve analysis showed that the AUC of was the highest during the 16-min period, with the AUC values of  $\Delta$ rSO<sub>2</sub> being 0.65, 0.68, 0.71, 0.72, and 0.70 for the 4-, 8-, 12-, 16-, and 20-min periods, respectively. Furthermore, when the amount of change of  $\Delta$ rSO<sub>2</sub> for the cerebral rSO<sub>2</sub> value from baseline was compared to that measured during the 16-min period, the AUC for the amount of change in rSO<sub>2</sub> was higher, at 0.75 (16 min proposed).**



**Fig. 2 – Comparison of areas under the receiver operation curve (AUC-ROCs) among 3 different models. AUC-ROCs are compared (1) using percent change computed as the rate of change from baseline to the maximum value recorded during the 16-min period ( $\Delta$ rSO<sub>2</sub>), (2) absolute value of the change computed as the maximum rise from baseline during the 16-min period, and (3) using both baseline rSO<sub>2</sub> and the amount of maximum rise from baseline during the 16-min period. AUC-ROCs were 0.88, 0.89, and 0.91 respectively. All ROCs were adjusted for the variables of age, sex, total number of electrical defibrillations and administrations of adrenaline, initial rhythm on emergency life-saving technician arrival, bystander CPR, and with or without witness.**



**Fig. 3 – Logistic regression analysis for the relation between ROSC and rSO<sub>2</sub> value. The associations between amount of maximum rise of rSO<sub>2</sub> and ROSC are shown separately for baseline rSO<sub>2</sub> values of 30%, 40%, and 50% using multivariable non-linear logistic regression. These models were adjusted for age, sex, total number of electrical defibrillations and administrations of adrenaline, initial rhythm on ELT arrival, bystander CPR, and with or without witness. At 50% of baseline rSO<sub>2</sub> value, 87.5% of patients will achieve ROSC if the rSO<sub>2</sub> value rises above 10%. ROSC, return of spontaneous circulation; ELT, emergency life-saving technician.**

showed that the AUC of  $\Delta rSO_2$  was highest (0.72) during the 16-min period, indicating that the probability of ROSC was highest at some point during the 16 min. However, when comparing  $\Delta rSO_2$  (16) with the amount of maximum rise over 16 min, the AUC-ROC for the amount of maximum rise was higher still, at 0.75 (Fig. 1; 16 min

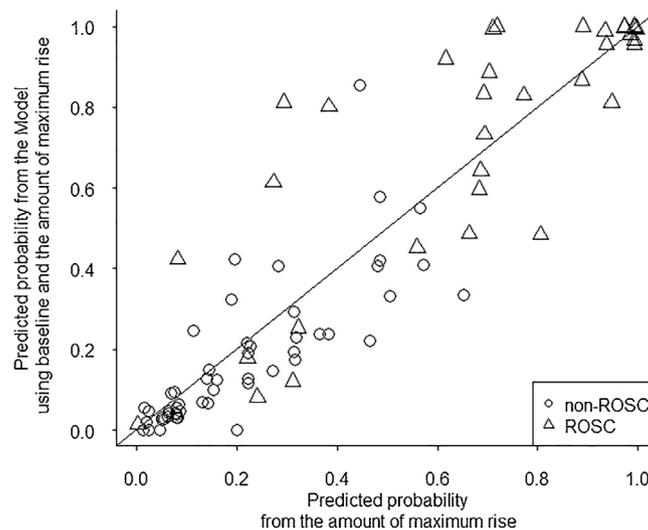
proposed). In short, the amount of maximum rise over 16 min was a better predictive factor for ROSC than was  $\Delta rSO_2$  alone. Furthermore, to discuss these results in terms of increasing percentage, we assessed the interaction between the amount of maximum rise from baseline rSO<sub>2</sub> (i.e., rSO<sub>2</sub> (baseline)) and rSO<sub>2</sub> (baseline) alone with a multivariable logistic regression model (Fig. 3). The model revealed that the probability of ROSC was low, and a rhythm check every 2 min could be meaningless if a constant increase in rSO<sub>2</sub> from baseline was not achieved. CPR to elevate the cerebral rSO<sub>2</sub> as soon as possible would result in ROSC, although it is also true that the rSO<sub>2</sub> value increases when the patient achieves ROSC.

Recently, some reports have identified the usefulness of rSO<sub>2</sub> monitoring to detect ROSC. Higher rSO<sub>2</sub> values or increases in rSO<sub>2</sub> have been shown to be strongly associated with ROSC<sup>2</sup>. These findings show that rSO<sub>2</sub> monitoring during CPR could be used to identify ROSC, and CPR using rSO<sub>2</sub> monitoring as an index of ROSC enables the performance of more continuous chest compression. In contrast, other reports have discussed rSO<sub>2</sub> values in terms of an absolute value or an initial single time point during CPR at hospital arrival and at the prehospital scene. The probabilities of ROSC and a favourable neurological outcome based on an initial rSO<sub>2</sub> value measured at a single time point have been reported as an absolute

**Table 2 – Continuous net reclassification improvement index (cNRI) and integrated discrimination index (IDI) for initial rSO<sub>2</sub> value added to the model using the amount of maximum rise.**

	Index	P value
(1) Proportion of increase for ROSC	0.559	–
(2) Proportion of increase for non-ROSC	0.208	–
(3) Proportion of decrease for ROSC	0.441	–
(4) Proportion of decrease for non-ROSC	0.792	–
(1-3) cNRI for ROSC	0.118	0.490
(4-2) cNRI for non-ROSC	0.585	<0.001
(1-3+4-2) cNRI	0.774	0.012
IDI	0.084	0.016

ROSC, return of spontaneous circulation.



**Fig. 4 – Predicted probability from the model using the amount of maximum rise and the model adding baseline rSO<sub>2</sub>. The scatter plot shows the predicted probabilities for assessing the ability to predict ROSC using multivariable non-linear logistic regression models. A black circle indicates the pair of predicted probabilities when a subject is in the non-ROSC group. A red circle indicates the pair of predicted probabilities when a subject is in the ROSC group. ROSC, return of spontaneous circulation. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).**

value.<sup>8,13–15</sup> However, we believe that it is important to discuss the relative value and the serial change in rSO<sub>2</sub> values during CPR because each cerebral oximeter is different in their mechanical characteristics, and the values of rSO<sub>2</sub> are always changing depending on the patient's situation at the time cerebral rSO<sub>2</sub> is measured. We can recognize that patients achieve ROSC when the rSO<sub>2</sub> value increases to a certain extent on the basis of the baseline rSO<sub>2</sub> value, as Fig. 3 shows.

Current guidelines recommend continuous chest compression and a rhythm check with an electrocardiographic monitor every 2 min.<sup>16</sup> However, the relation between continuous chest compression and a rhythm check every 2 min during resuscitation is definitely contradictory. Berg et al. reported that the integrated coronary perfusion pressure was lower in the group receiving chest compression and rescue breathing during each minute of CPR than in the group receiving chest compression alone.<sup>17</sup> Meex et al. reported that a decrease in the cerebral rSO<sub>2</sub> value was observed during every-2-min rhythm assessments.<sup>18</sup> Although even the brief interruption of CPR has the possibility of affecting the probability of ROSC and neurological outcome, it remains unclear whether chest compression must be continued without cessation, i.e., the meaning of brief interruption has not been clearly defined. From our wide reading, we could find no definite evidence that a rhythm check must be performed every 2 min. ILCOR 2015 recommended the treatment for the “Timing of CPR cycles” to be pausing chest compression every 2 min to assess the cardiac rhythm (weak recommendation, low-quality evidence) because no studies directly address the question of optimal CPR intervals and their effect on the identified critical outcomes of survival with favourable neurologic or functional outcome at discharge or survival only at discharge or the important outcomes of ROSC, coronary perfusion pressure, and cardiac output.<sup>16</sup>

We performed mechanical chest compression in this study. Ogawa et al. reported that mechanical chest compression resulted in a statistically significant increase in cerebral rSO<sub>2</sub> values during

resuscitation.<sup>19</sup> Parnia et al. reported that significantly higher mean rSO<sub>2</sub> levels were observed during CPR in patients who were resuscitated using an automated mechanical chest compression device compared with those undergoing manual chest compression.<sup>3</sup> Our CPR protocol with mechanical chest compression may affect the probability of ROSC and elevation of the rSO<sub>2</sub> value. However, if mechanical chest compression and rSO<sub>2</sub> monitoring are combined, continuous chest compression can be performed for as long as possible without the need to change chest compressors or perform unnecessary rhythm checks.

As a consequence of our study, it might be possible to devise a CPR protocol that pauses continuous chest compression at a different interval, e.g., a protocol that precludes the every-2-min rhythm check, until the rSO<sub>2</sub> value rises to a certain extent on the basis of the baseline rSO<sub>2</sub> value. In prehospital settings, it is often difficult for ELTs to adhere to the standard guideline protocol of every-2-min rhythm checks and high-quality chest compression when patients are transported up and down stairs and through narrow routes. To handle such situations, a simpler CPR protocol could be devised that omits the unnecessary rhythm checks and allows for longer periods of continuous chest compression based on the index of change in the rSO<sub>2</sub> value.

This study has several limitations. First, it is a single-centre, retrospective study with a small sample size. Second, it was not possible to evaluate the cerebral rSO<sub>2</sub> value in all CPA patients. In total, 155 CPA patients were admitted during the study period. However, 14 trauma patients were excluded because they underwent thoracotomy in the emergency room, and adequate rSO<sub>2</sub> data could not be obtained from the other 51 patients. Third, we used the AutoPulse<sup>®</sup>R mechanical chest compression device in our study. The association between differences in mechanical chest compression devices and ROSC or rSO<sub>2</sub> values is not clear. However, the differences in these devices may affect the probability of ROSC. Furthermore, we did not consider the duration of CPR in the prehospital and in-hospital settings in our analyses because we

could not collect accurate data on these factors, which also might also affect the probability of ROSC. Fourth, our data might include contamination from subjects who already achieved ROSC during chest compressions because the ECG rhythm was only checked every 2 min. Finally, patients with low probability of ROSC might also have been included because all patients in CPA are transferred to hospital in Japan. The number of patients with a shockable rhythm in this study was small, and as a result, this might have affected the percentage of patients with ROSC.

## Conclusion

The combination of rSO<sub>2</sub> (baseline) with the amount of maximum rise from the baseline rSO<sub>2</sub> value during CPR might be a new index for the prediction of ROSC in OHCA that could be useful in guiding cardiopulmonary resuscitation. Further studies are needed to validate these findings.

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## Conflict of interest

All authors report that they have no conflicts of interest to declare in relation to this report.

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None.

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