SPANDIDOS PUBLICATIONS

EXPERIMENTAL AND THERAPEUTIC MEDICINE 19: 353-358, 2020

Comparing the response of pulse oximetry and regional cerebral oxygen saturation to hypoxia in preschool children

YI LU, MEIQIN DI, CHAN LI, MENGMENG CHEN, KAIMING YUAN and WANGNING SHANGGUAN

Department of Anesthesiology and Perioperative Medicine, The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University, Wenzhou, Zhejiang 325027, P.R. China

Received April 12, 2019; Accepted October 15, 2019

DOI: 10.3892/etm.2019.8199

Abstract. Pulse oximetry and measurement of regional cerebral oxygen saturation ($rcSO_2$) are used to monitor peripheral and cerebral oxygenation, respectively. However, the response of $rcSO_2$ and pulse oxygen saturation (SpO_2) to hypoxia in preschool children has not been previously assessed. A total of 36 preschool patients who had undergone a tonsillectomy [age, 4-6 years, American Society of Anesthesiologists grade I or II] were screened and prospectively enrolled in the present study. Hemodynamics, including $rcSO_2$, SpO_2, non-invasive blood pressure, heart rate, electrocardiogram and capnography, were continuously monitored throughout the study. Following pre-oxygenation, pressure-controlled ventilation with 100% oxygen was administered through a mask with a flow rate of 6 l/min, under total intravenous anesthesia, and the end-tidal carbon dioxide partial pressure was maintained between 30 and 40 mmHg. Tracheal intubation was then performed and ventilation was paused until SpO_2 decreased to 90% or $rcSO_2$ decreased by >10% of the baseline level. The duration from pausing of mechanical ventilation to the start of the $rcSO_2$ decline was shorter than that of SpO_2 (80.2±23.6 sec vs. 124.4±20.5 sec; P<0.001). Subsequent to the recovery of ventilation, the duration from the starting point to the increasing point of the baseline of $rcSO_2$ was longer than that of SpO_2 (84.8±24.3 sec vs. 15.2±6.8 sec; P<0.001). From the point where mechanical ventilation was paused to when $rcSO_2$/SpO_2 began to decrease, the $rcSO_2$ and SpO_2 values decreased and a significant correlation of them was observed (Pearson's correlation coefficient=0.317; P=0.027). From the time-point where mechanical ventilation was recovered to the time-point where $rcSO_2$ or SpO_2 began to increase, $rcSO_2$ and SpO_2 values decreased and a significant correlation of them was observed (Spearman's correlation coefficient=0.489; P=0.006). From the baseline to the minimum value, compared with the SpO_2, the $rcSO_2$ declined at a decreased rate (9.7±0.5% vs. 5.3±2.7%; P<0.001). The present clinical trial was registered at http://www.chictr.org.cn on 14th March 2016 (registration no. ChiCTR-OOC-16008095).

Correspondence to: Dr Wangning Shangguan, Department of Anesthesiology and Perioperative Medicine, The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University, 109 West Xueyuan Road, Wenzhou, Zhejiang 325027, P.R. China
E-mail: sgwning@163.com

Key words: hypoxia, pulse oxygen saturation, regional cerebral oxygen saturation, children

Introduction

Consecutive and non-invasive pulse oxygen saturation (SpO_2) may be measured using pulse oximetry, which allows for rapid identification of hypoxic state. Therefore, this technique is a useful clinical alternative to intermittent arterial blood sampling (1). However, measurement of SpO_2 has certain limitations and is difficult to use in the presence of nail polish, anaemia, light interference, skin pigmentation, venous pulsations and low perfusion, as they may cause measurement errors (2). In 1977, Jöbsis (3) introduced, for the first time, the monitoring of regional cerebral oxygen saturation ($rcSO_2$) via near-infrared spectroscopy (NIRS). NIRS takes advantage of the tissue penetration abilities of light of the near-infrared spectrum. In contrast to SpO_2, $rcSO_2$ does not require plethysmography, and pulsatile flow measurement is also not required. NIRS assumes a relative and fixed amount of arterial vs. venous blood to determine the oxygen saturation. Therefore, $rcSO_2$ does not provide an indicator of oxygen delivery and instead provides information regarding the balance between regional oxygen supply and demand (4). Recent studies have suggested that pediatric patients may benefit from $rcSO_2$ monitoring during surgery (5-9). The use of $rcSO_2$ is increasing, but the routine use of $rcSO_2$ as a standard-of-care monitor is still not recommended at present.

Although it has been reported that $rcSO_2$ provides an earlier alert during hypoxia compared with pulse oximetry (10), whether SpO_2 and $rcSO_2$ exhibit similar response curves during acute apnea has, to the best of our knowledge, not yet been reported in preschool children. The purpose of the present study was to determine whether a correlation is present between the changing tendency of SpO_2 and $rcSO_2$ in response to hypoxia in preschool patients. It was hypothesized that SpO_2 may exhibit the same response to hypoxia as $rcSO_2$.

Materials and methods

Ethical approval and consent to participate. The present study was registered in the research registry (www.chictr.org.cn; registration no. ChiCTR-OOC-16008095; 14 March 2016). The protocol (no. 2016-08; 1 March 2016) was approved by the review board of the Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University (Wenzhou, China). Written informed consent had been obtained by the parents or legally authorized guardians.

Inclusion criteria. A total of 36 pediatric patients [age, 4-6 years; American Society of Anesthesiologists (ASA) grade I or II], scheduled for elective tonsillectomy between May and September 2016 at the Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University were enrolled in the present clinical trial.

Exclusion criteria. Patients were excluded if they exhibited the following: i) No cooperation; ii) body mass index of <13.5 kg/m^2 or >31 kg/m^2; iii) upper airway infection; iv) serious respiratory and/or cardiovascular disease, hepatic or renal insufficiency (the values of alanine aminotransferase, aspartate aminotransferase, blood urea nitrogen, creatinine >1.5 times the upper limit of the normal level); v) asthma or airway hyperresponsiveness, neuromuscular diseases or cachexia; vi) airway abnormalities and a previous history of an abnormal response to anesthesia; vii) an acid-base imbalance or severe electrolyte disorder; viii) participation in another clinical study within 30 days.

Experimental design. After arrival in the operating room, intravenous access was established into the peripheral vein in the forearm for induction of anaesthesia. Throughout the present study, all patients were continuously monitored, with their $rcSO_2$ (SenSmart™; Nonin Medical, Inc.) being assessed using a cerebral oximetry probe (reading $rcSO_2$ every 5 sec), which was placed on the middle of the forehead, and SpO_2 being assessed using an oximetry probe (M1133A; Philips Medical Systems, Inc.), which was placed on the right index finger. Non-invasive systolic blood pressure (SBP), mean arterial pressure (MAP) and diastolic blood pressure (DBP) were measured every 1 min on a different limb to the SpO_2 probe. Heart rate (HR), electrocardiogram and end-tidal carbon dioxide partial pressure ($P_{ET}CO_2$) were also continuously monitored. Induction of anaesthesia was performed using propofol 2-3 mg/kg, fentanyl 2-3 μg/kg and cisatracurium 0.1-0.2 mg/kg. Anaesthesia was maintained with a continuous target-controlled infusion of propofol and remifentanil. Pressure-controlled ventilation of 100% oxygen through a mask, with a flow rate of 6 l/min, was administered, and $P_{ET}CO_2$ was maintained between 30 and 35 mmHg. After a period of 6 min, mechanical ventilation was stopped and the tracheal tube was successfully introduced using a video laryngoscope. The tracheal tube was subsequently disconnected from the circuit and the proximal end was opened until the SpO_2 decreased to 90% or until the $rcSO_2$ decreased by >10% of the baseline level. The tracheal tube was then reconnected to the circuit and ventilation was recovered with a flow rate of 6 l/min of 100% oxygen.

The values of NIBP, HR, SpO_2 and $rcSO_2$ were recorded at the designated time-points: T_0 indicates the time-point prior to application of oxygen prior to oxygenation; T_1 indicates baseline, the time-point at which the mechanical ventilation was stopped; T_2 indicates the time-point at which SpO_2 began to drop from the baseline level; t_2 indicates the time-point at which $rcSO_2$ began to drop from the baseline level; T_3 indicates the time-point of SpO_2 decreasing to 90% or $rcSO_2$ decreasing by >10% of the baseline level and mechanical ventilation being recovered; T_4 indicates the time-point at which SpO_2 began to rise from the minimum value following ventilation; t_4 indicates the time-point at which $rcSO_2$ began to rise from the minimum value following ventilation; T_5 indicates the time-point at which SpO_2 returned to the baseline level, t_5 indicates the time-point at which $rcSO_2$ returned to the baseline level. S_{T1-T4} indicates the value of SpO_2 at T_1 (baseline)-the value of SpO_2 at T_4 (the minimum value); R_{T1-t4} indicates the value of $rcSO_2$ at the T_1 time-point (baseline)-the value of $rcSO_2$ at t_4 (the minimum value; Fig. 1).

Statistical analysis. All data were expressed as the mean ± standard deviation or as n (%), as appropriate. Statistical analysis was performed using SPSS 18.0 (SPSS Inc.). The calculation of the sample size, besides being based on the pilot study, mainly referred to that in previous studies (Koch *et al* (8), where the sample size was n=21, and the authors studied the perioperative use of cerebral and renal near-infrared spectroscopy in neonates; and Eichhorn *et al* (11), where the sample size was n=10, and a clinical trial was used to evaluate the use of near-infrared spectroscopy under apnea-dependent hypoxia in humans).

The normality of distribution of data was examined using the Shapiro-Wilk test. For the data that did not exhibit a normal distribution, a Wilcoxon signed-rank test and Spearman's rank correlation were used. Data exhibiting a normal distribution were analyzed using a repeated-measures one-way analysis of variance and Pearson's linear correlation. $P<0.05$ was considered to indicate statistical significance.

Results

Patient characteristics. Among the 36 pediatric patients considered for the present study, 6 cases were excluded due to upper airway infection or body mass index >31 kg/m^2, which may have added complexity to the procedure. Finally, a total of 30 patients, including 21 males and 9 females (age, 4.9±0.8 years; body weight, 21.8±5.5 kg) were enrolled in the present study.

Vital signs at different time-points. Compared with the values at T_0, the SBP, MAP and DBP were decreased at the time-points from T_1 to T_5/t_5, and the HR was decreased at the T_1 time-point ($P<0.001$). Compared with those at T_1, the MAP and DBP were increased at the T_2 time-point and the HR was increased from the T_2/t_2 to the T_5/t_5 time-point ($P<0.001$), as presented in Table I.

Changes of $rcSO_2$ and SpO_2 over time. The values for $rcSO_2$ and SpO_2 are provided in Table II and the different time-intervals are stated in Table III. Compared with the SpO_2, the $rcSO_2$ exhibited an earlier decrease in response

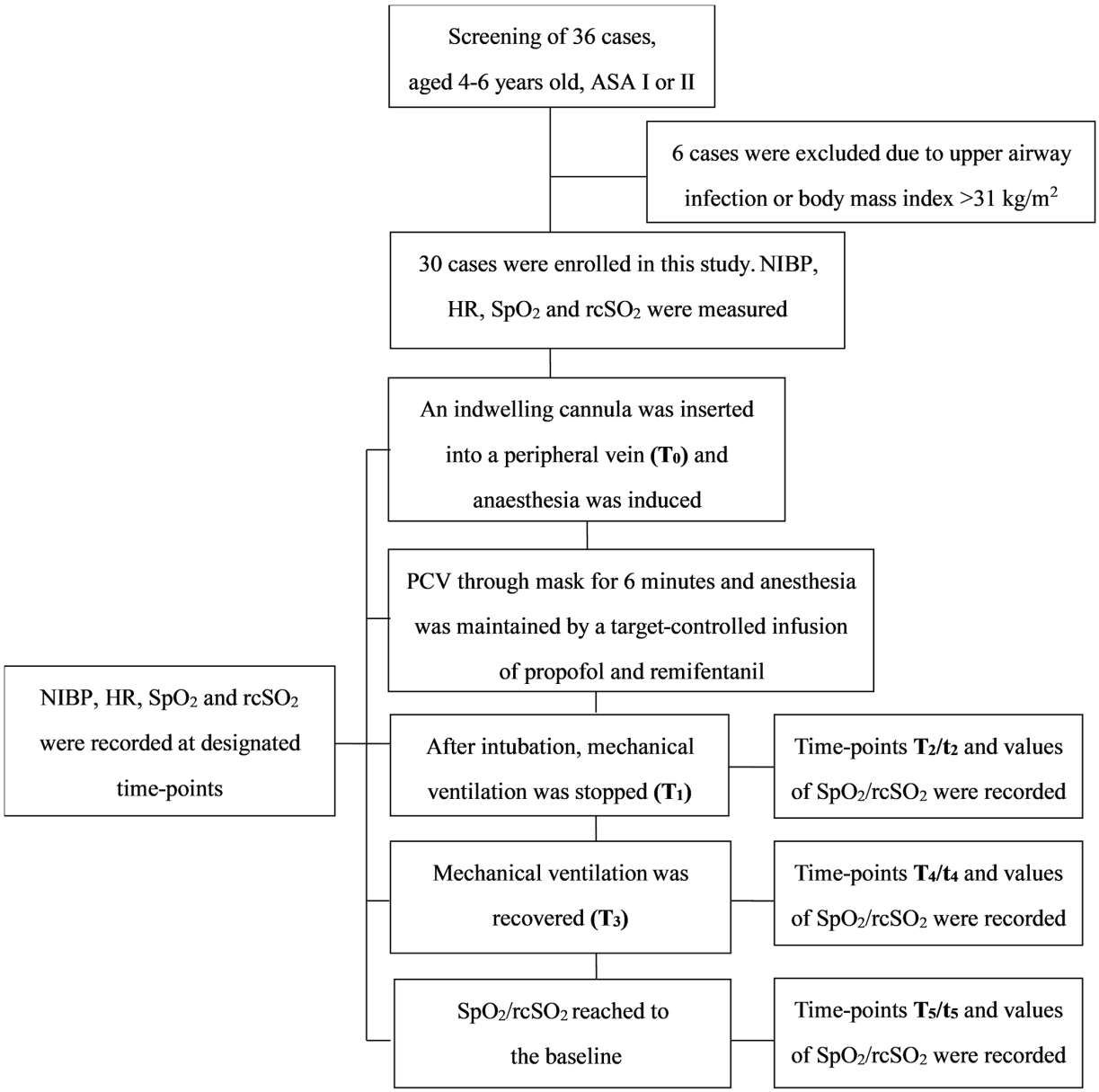

Figure 1. Flow chart of the study. ASA, American Society of Anesthesiologists; NIBP, non-invasive blood pressure; HR, heart rate; SpO_2, pulse oxygen saturation; $rcSO_2$, regional cerebral oxygen saturation; PCV, pressure-controlled ventilation; T_0, time-point prior to application of any oxygen for pre-oxygenation; T_1, baseline, the time-point at which the mechanical ventilation was stopped; T_2, the time-point at which SpO_2 began to drop from baseline; t_2, the time-point at which $rcSO_2$ began to drop from baseline; T_3, the time-point at which SpO_2 decreased to 90% or $rcSO_2$ decreased to by >10% of the baseline and mechanical ventilation was recovered; T_4, the time-point at which SpO_2 began to rise from the minimum value following ventilation; t_4, the time-point at which $rcSO_2$ began to rise from the minimum value following ventilation; T_5, the time-point at which SpO_2 returned to the baseline level, t_5, the time-point at which $rcSO_2$ returned to the baseline level.

to hypoxia (t_2-T_1=80.2±23.6 sec vs. T_2-T_1=124.4±20.5 sec; P<0.001). However, the $rcSO_2$ decreased slower than the SpO_2 (T_3-t_2=104.8±27.3 sec vs. T_3-T_2=60.6±13.7 sec; P<0.001). Furthermore, the decrease of SpO_2 to 90% of the baseline occurred earlier than that of $rcSO_2$ decreasing by >10% of the baseline in all thirty cases. After the recovery of ventilation, $rcSO_2$ was increased earlier than SpO_2 (t_4-T3=13.4±6.2 sec vs. T_4-T3=18.9±6.5 sec; P<0.001) and the duration of t_5-t_4 was longer than that of T_5-T_4 (84.8±24.3 sec vs. 15.2±6.8 sec; P<0.001). In addition, the duration of t_5-T_3 was longer than that of T_5-T_3 (98.2±24.3 sec vs. 34.1±6.8 sec; P<0.001). From T_2/t_2 to T_3, the $rcSO_2$ and SpO_2 values exhibited a decrease and a significant correlation of the two parameters was determined (Pearson's correlation coefficient=0.317; P=0.027). From T_3 to T_4/t_4, the $rcSO_2$ and SpO_2 values decreased significantly and a significant correlation of the two parameters was obtained (Spearman's correlation coefficient=0.489; P=0.006), as shown in Figs. 2 and 3. Compared with S_{T1-T4}, R_{T1-t4} was smaller (9.7±0.5 sec vs. 5.3±2.7%; P<0.001; Fig. 4).

Discussion

The results of the present study demonstrated that $rcSO_2$ and SpO_2 exhibited similar dynamics in their changing curve patterns in response to acute apnea (no ventilation), although $rcSO_2$ decreased earlier and declined slower than SpO_2 during

Table I. Dynamic changes of SBP, MBP, DBP and HR at different time-points.

Parameter	T_0	T_1	T_2/t_2	T_3	T_4/t_4	T_5/t_5
SBP (mmHg)	112±12	94±11[a]	98±13[a]/97±12[a]	97±13[a]	97±12[a]/97±13[a]	98±13[a]/98±12[a]
MAP (mmHg)	80±9	62±8[a]	68±11[ab]/66±9[ab]	65±9[a]	65±9[a]/65±9[a]	66±10[a]/64±11[a]
DBP (mmHg)	64±11	46±9[a]	53±11[ab]/51±9[ab]	49±9[a]	49±9[a]/49±9[a]	50±10[a]/47±12[a]
HR (bpm)	96±16	83±13[a]	93±12[b]/91±10[b]	92±16[b]	92±16[b]/92±16[b]	91±16[b]/98±15[b]

All values are expressed as the mean±standard deviation (n=30). Compared with T_0, [a]P<0.001; compared with T_1, [b]P<0.001. SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure; HR, heart rate. T_0, time-point prior to application of any oxygen for pre-oxygenation; T_1, baseline, the time-point at which mechanical ventilation was stopped; T_2, the time-point at which SpO_2 began to drop from baseline; t_2, the time-point at which $rcSO_2$ began to drop from baseline; T_3, the time-point at which SpO_2 decreased to 90% or $rcSO_2$ decreased by >10% of the baseline and mechanical ventilation was recovered; T_4, the time-point at which SpO_2 began to rise from the minimum value following ventilation; t_4, the time-point at which $rcSO_2$ began to rise from the minimum value following ventilation; T_5, the time-point at which SpO_2 returned to the baseline level, t_5, the time-point at which $rcSO_2$ returned to the baseline level.

Table II. Dynamic changes of SpO_2 and $rcSO_2$ at different time-points (n=30).

Item	T_0	T_1	T_2/t_2	T_3	T_4/t_4	T_5/t_5
SpO_2 (%)	99.7±0.5	99.7±0.5	99.7±0.5	90±0.0	84.7±3.2	99.7±0.5
$rcSO_2$ (%)	81.4±3.9	87.0±3.6	87.0±3.6	81.8±4.5	80.4±4.0	81.4±3.9

All values are expressed as the mean±standard deviation (n=30). Compared with S_{T1-T4}, R_{T1-t4} was smaller (9.7±0.5% vs. 5.3±2.7%, P<0.001). SpO_2, pulse oxygen saturation; $rcSO_2$, regional cerebral oxygen saturation; S_{T1-T4}, the value of SpO_2 at T_1 (baseline)-the value of SpO_2 at T_4 (the minimum value); R_{T1-t4}, the value of $rcSO_2$ at the T_1 time-point (baseline)-the value of $rcSO_2$ at t_4 (the minimum value). T_0, time-point prior to application of any oxygen for pre-oxygenation; T_1, baseline, the time-point at which mechanical ventilation was stopped; T_2, the time-point at which SpO_2 began to drop from baseline; t_2, the time-point at which $rcSO_2$ began to drop from baseline; T_3, the time-point at which SpO_2 decreased to 90% or $rcSO_2$ decreased by >10% of the baseline and mechanical ventilation was recovered; T_4, the time-point at which SpO_2 began to rise from the minimum value following ventilation; t_4, the time-point at which $rcSO_2$ began to rise from the minimum value following ventilation; T_5, the time-point at which SpO_2 returned to the baseline level, t_5, the time-point at which $rcSO_2$ returned to the baseline level.

hypoxia. Furthermore, $rcSO_2$ increased earlier and slower than SpO_2 following the recovery of ventilation.

It has been previously suggested that apneic episodes in infants, which are known to cause an increase in vascular resistance and a reduction of cerebral blood volume, may be avoided with a threshold of SpO_2 >85% for cerebral circulation (12). A study performed by Gupta *et al* (13) reported that by increasing the vascular resistance where the threshold of SpO_2 was 90%, hypoxic load reduced the blood circulation of the middle cerebral artery in normal healthy adults. Therefore, in the present study, the threshold of SpO_2 was set at 90%. It has been reported that a decline of >25% from the baseline level, or the value of $rcSO_2$ of <40%, may influence neurologic dysfunction and exhibit adverse outcomes (14). A reduction to the value of 50% or less or a decrease of 15-20% from the baseline has been used as a critical threshold for interventions (15,16). Therefore, in the present study, a 10% reduction of $rcSO_2$ from the baseline was used as a threshold to ensure patients' safety.

The present study demonstrated that after pausing mechanical ventilation (acute apnea), the $rcSO_2$ decreased earlier and declined slower than SpO_2. A previous study revealed that with SpO_2 maintained in the normal range, a decrease of >20% may be observed in cerebral oxygen saturation (17). Another study indicated that SpO_2 readings were 10-15 sec delayed compared with $rcSO_2$ readings in neonates (9). Similar results were also reported by Tanidir *et al* (18). In the present study, the decrease of $rcSO_2$ occurred ~40 sec earlier than that of SpO_2. Tobias (10) suggested that these changes may be associated with different 'blood beds', which are evaluated using monitors. It has been demonstrated that SpO_2 only captures arterial oxy-hemoglobin saturation and measures saturation in the arterial bed, but there is a correlation of $rcSO_2$ values with mixed venous (70%) and arterial (30%) oxygen saturations (3). In contrast to SpO_2, $rcSO_2$ depends on venous blood. The partial pressure of oxygen would decrease at an approximately equal rate in venous and arterial 'blood beds' during apnea. However, due to the lower venous partial pressure of oxygen, it would reach the bend of the oxy-hemoglobin dissociation curve more rapidly. Therefore, a decrease in the $rcSO_2$ would occur first. During hypoxia, the decline of $rcSO_2$ reflects a concurrent decrease in arterial oxy-haemoglobin saturation and a rise in venous deoxy-hemoglobin saturation (10). In addition, Rasmussen *et al* (19) indicated that cerebral NIRS oximetry responded poorly to changes in tissue oxygenation during hypotension that was induced by decreased preloading. This may be due to the increase in the artery-to-vein ratio that occurs following the decrease in oxygen delivery, which is due to arterial vasodilation and possibly cerebral venous collapse.

Table III. Comparison of the time difference between SpO_2 and $rcSO_2$ during the response to hypoxia (sec).

Duration	SpO_2	$rcSO_2$	P-value
T_2/t_2-T_1	124.4±20.5	80.2±23.6	<0.001
T_3-T_2/t_2	60.6±13.7	104.8±27.3	<0.001
T_4/t_4-T_3	18.9±6.5	13.4±6.2	<0.001
T_5/t_5-T_4/t_4	15.2±6.8	84.8±24.3	<0.001
T_5/t_5-T_3	34.1±6.8	98.2±24.3	<0.001

All values are expressed as the mean±standard deviation (n=30). SpO_2, pulse oxygen saturation; $rcSO_2$, regional cerebral oxygen saturation; T_1, baseline, the time-point at which mechanical ventilation was stopped; T_2, the time-point at which SpO_2 began to drop from baseline; t_2, the time-point at which $rcSO_2$ began to drop from baseline; T_3, the time-point at which SpO_2 decreased to 90% or $rcSO_2$ decreased by >10% of the baseline and mechanical ventilation was recovered; T_4, the time-point at which SpO_2 began to rise from the minimum value following ventilation; t_4, the time-point at which $rcSO_2$ began to rise from the minimum value following ventilation; T_5, the time-point at which SpO_2 returned to the baseline level, t_5, the time-point at which $rcSO_2$ returned to the baseline level.

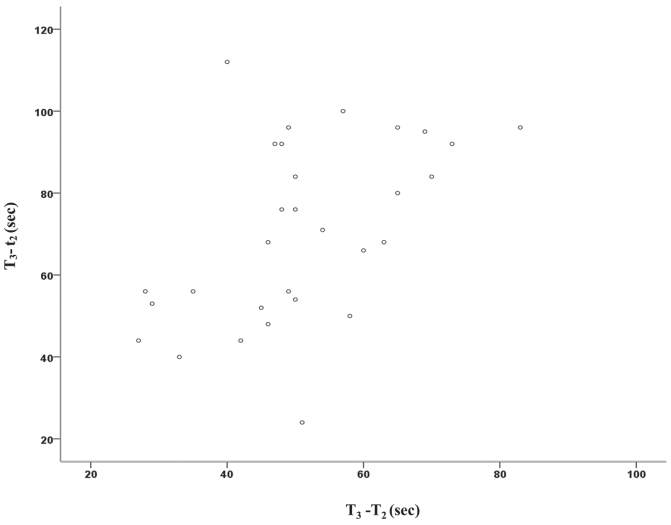

Figure 2. Pearson's correlation scatter plot of T_3- T_2/t_2. From T_2/t_2 to T_3, the $rcSO_2$ and SpO_2 values exhibited a significant correlation (Pearson's correlation coefficient=0.317; P=0.027). T_2, the time-point at which SpO_2 began to drop from the baseline level; t_2, the time-point at which $rcSO_2$ began to drop from the baseline level; T_3, the time-point of SpO_2 decreasing to 90% or $rcSO_2$ decreasing by >10% of the baseline level and mechanical ventilation being recovered.

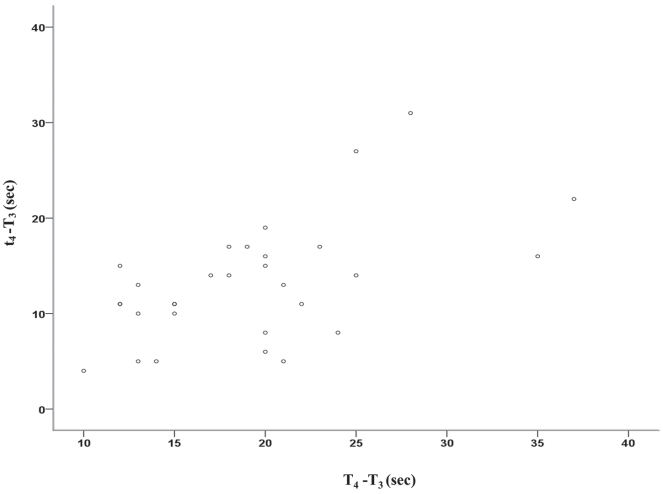

Figure 3. Spearman's rank correlation scatter plot of T_4/t_4-T_3. From T_3 to T_4/t_4, the $rcSO_2$ and SpO_2 values exhibited a significant correlation (Spearman's correlation coefficient=0.489; P=0.006). T_3, the time-point at which SpO_2 decreased to 90% or $rcSO_2$ decreased by >10% of the baseline and mechanical ventilation was recovered; T_4, the time-point at which SpO_2 began to rise from the minimum value following the ventilation; t_4, the time-point at which $rcSO_2$ began to rise from the minimum value following the ventilation.

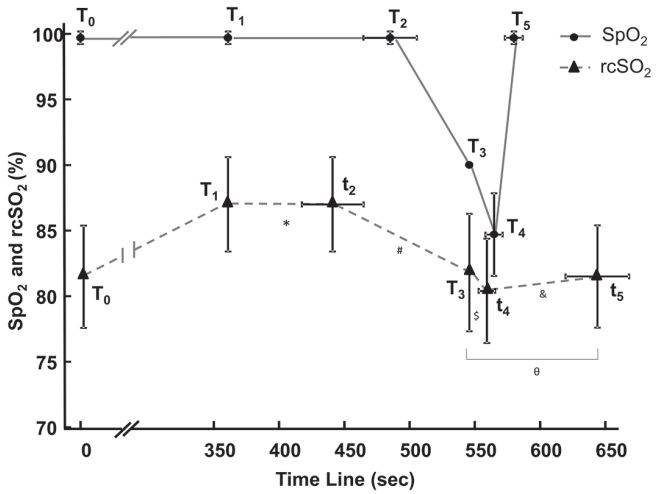

Figure 4. Dynamic changes of SpO_2 and $rcSO_2$ during hypoxia. *P<0.001 vs. T_1/T_2, #P<0.001 vs. T_2/T_3, $P<0.001 vs. T_3/T_4, &P<0.001 vs. T_4/T_5, θP<0.001 vs. T_3/T_5. SpO_2, pulse oxygen saturation; $rcSO_2$, regional cerebral oxygen saturation T_0, time-point prior to application of any oxygen for pre-oxygenation; T_1, baseline, the time-point at which mechanical ventilation was stopped; T_2, the time-point at which SpO_2 began to drop from baseline; t_2, the time-point at which $rcSO_2$ began to drop from baseline; T_3, the time-point at which SpO_2 decreased to 90% or $rcSO_2$ decreased by >10% of the baseline and mechanical ventilation was recovered; T_4, the time-point at which SpO_2 began to rise from the minimum value following ventilation; t_4, the time-point at which $rcSO_2$ began to rise from the minimum value following ventilation; T_5, the time-point at which SpO_2 returned to the baseline level, t_5, the time-point at which $rcSO_2$ returned to the baseline level.

This may cause the arterial part of the NIRS signal to increase, leading to $rcSO_2$ values decreasing more slowly. During the period of paused ventilation, the serum carbon dioxide increased and the blood vessels of the brain became dilated. Venous deoxy-hemoglobin saturation captured by $rcSO_2$ may explain the early change in $rcSO_2$. The effect of perfusion on $rcSO_2$ levels has also been indicated by Schwaberger *et al* (20).

After restarting ventilation, $rcSO_2$ was increased earlier than SpO_2, but its increasing rate was slower, with $rcSO_2$ and SpO_2 exhibiting similar dynamic changing curve patterns. $rcSO_2$ was increased with a mean delay of 13.4 sec, whereas the increase of SpO_2 featured a significant delay of 18.9 sec. These results are similar to those of previous studies (9,11). It is well known that the brain responds to hypoxia through increasing cerebral blood flow. To maintain adequate oxygen supply in organs sensitive to hypoxia, including the brain, blood is being re-distributed (11,21,22). This may explain for

the earlier increase of $rcSO_2$ than that of SpO_2 following the recovery of ventilation, as a result of the oxygenated blood preferentially being distributed to the brain. Delayed vasodilatation in the periphery, in comparison to the cerebral blood, may provide an additional explanation for the time difference observed between the increase of $rcSO_2$ and that of SpO_2 (11).

Of note, the present study has certain limitations. First, the sample size of the present study was relatively small. In addition, the experimental design was relatively simple and the further mechanism exploration was not included. In conclusion, during an episode of hypoxia, $rcSO_2$ and SpO_2 exhibited similar dynamics in their changing curve patterns, and $rcSO_2$ was more sensitive compared with peripheral SpO_2.

Acknowledgements

The authors would like to thank Professor Daqing Ma, expert in Anaesthetics, Pain Medicine and Intensive Care, Department of Surgery and Cancer, Faculty of Medicine, Imperial College London and Chelsea and Westminster Hospital (London, UK) for his critical comments provided throughout the preparation of the manuscript.

Funding

No funding was received.

Availability of data and materials

The datasets used and/or analyzed during the present study are available from the corresponding author on reasonable request.

Authors' contributions

WS and YL contributed to the design of the study and project administration. YL, CL and MD performed the experiments and analyzed the data. MC contributed to data analysis. KY performed the statistical analysis. YL and WS drafted, reviewed and edited the manuscript. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Ethical approval for this study was provided by the Ethical Committee of The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University (Wenzhou, China; no. 2016-08 dated 1 March 2016). Signed informed consent was obtained from the parents and/or guardians. Informed consent was provided by the parents or legally authorized guardians.

Patient consent for publication

Not applicable.

Competing interests

The authors have no competing interests to declare.

References

1. Sinex JE: Pulse oximetry: Principles and limitations. Am J Emerg Med 17: 59-67, 1999.
2. Kenosi M, Naulaers G, Ryan CA and Dempsey EM: Current research suggests that the future looks brighter for cerebral oxygenation monitoring in preterm infants. Acta Paediatr 104: 225-231, 2015.
3. Jöbsis FF: Noninvasive, infrared monitoring of cerebral and myocardial oxygen sufficiency and circulatory parameters. Science 198: 1264-1267, 1977.
4. Goldman S, Sutter F, Ferdinand F and Trace C: Optimizing intraoperative cerebral oxygen delivery using noninvasive cerebral oximetry decreases the incidence of stroke for cardiac surgical patients. Heart Surg Forum 5: E376-E381, 2004.
5. Steppan J and Hogue CW Jr: Cerebral and tissue oximetry. Best Pract Res Clin Anaesthesiol 28: 429-439, 2014.
6. Abramo TJ, Zhou C, Estrada C, Meredith M, Miller R, Pearson M, Tulipan N and Williams A: Innovative application of cerebral rSO2 monitoring during shunt tap in pediatric ventricular malfunctioning shunts. Pediatr Emerg Care 31: 479-486, 2015.
7. Watanabe T, Ito M, Miyake F, Ogawa R, Tamura M and Namba F: Measurement of brain tissue oxygen saturation in term infants using a new portable near-infrared spectroscopy device. Pediatr Int 59: 167-170, 2017.
8. Koch HW and Hansen TG: Perioperative use of cerebral and renal near-infrared spectroscopy in neonates: A 24-h observational study. Paediatr Anaesth 26: 190-198, 2016.
9. Séguéla PE, Guillet E, Thambo JB and Mauriat P: Ductal closure and near-infrared spectroscopy for regional oxygenation monitoring in ductus-dependent congenital heart disease. Arch Pediatr 22: 857-860, 2015.
10. Tobias JD: Cerebral oximetry monitoring with near infrared spectroscopy detects alterations in oxygenation before pulse oximetry. J Intensive Care Med 23: 384-388, 2008.
11. Eichhorn L, Erdfelder F, Kessler F, Doerner J, Thudium MO, Meyer R and Ellerkmann RK: Evaluation of near-infrared spectroscopy under apnea-dependent hypoxia in humans. J Clin Monit Comput 29: 749-757, 2015.
12. Yamamoto A, Yokoyama N, Yonetani M, Uetani Y, Nakamura H and Nakao H: Evaluation of change of cerebral circulation by SpO2 in preterm infants with apneic episodes using near infrared spectroscopy. Pediatr Int 45: 661-664, 2003.
13. Gupta AK, Menon DK, Czosnyka M, Smielewski P and Jones JG: Thresholds for hypoxic cerebral vasodilation in volunteers. Anesth Analg 85: 817-820, 1997.
14. Edmonds HL Jr, Ganzel BL and Austin EH III: Cerebral oximetry for cardiac and vascular surgery. Semin Cardiothorac Vasc Anesth 8: 147-166, 2004.
15. Samra SK, Dy EA, Welch K, Dorje P, Zelenock GB and Stanley JC: Evaluation of a cerebral oximeter as a monitor of cerebral ischemia during carotid endarterectomy. Anesthesiology 93: 964-970, 2000.
16. Rigamonti A, Scandroglio M, Minicucci F, Magrin S, Carozzo A and Casati A: A clinical evaluation of near-infrared cerebral oximetry in the awake patient to monitor cerebral perfusion during carotid endarterectomy. J Clin Anesth 17: 426-430, 2005.
17. Erdem AF, Kayabasoglu G, Tas Tuna A, Palabiyik O, Tomak Y and Beyaz SG: Effect of controlled hypotension on regional cerebral oxygen saturation during rhinoplasty: A prospective study. J Clin Monit Comput 30: 655-660, 2016.
18. Tanidir IC, Ozturk E, Ozyilmaz I, Saygi M, Kiplapinar N, Haydin S, Guzeltas A and Odemis E: Near infrared spectroscopy monitoring in the pediatric cardiac catheterization laboratory. Artif Organs 38: 838-844, 2014.
19. Rasmussen MB, Eriksen VR, Andresen B, Hyttel-Sørensen S and Greisen G: Quantifying cerebral hypoxia by near-infrared spectroscopy tissue oximetry: The role of arterial-to-venous blood volume ratio. J Biomed Opt 22: 25001, 2017.
20. Schwaberger B, Pichler G, Binder C, Avian A, Pocivalnik M and Urlesberger B: Even mild respiratory distress alters tissue oxygenation significantly in preterm infants during neonatal transition. Physiol Meas 35: 2085-2099, 2014.
21. Joulia F, Lemaitre F, Fontanari P, Mille ML and Barthelemy P: Circulatory effects of apnoea in elite breath-hold divers. Acta Physiol (Oxf) 197: 75-82, 2009.
22. Lindholm P and Lundgren CE: The physiology and pathophysiology of human breath-hold diving. J Appl Physiol (1985) 106: 284-292, 2009.