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

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Received April 12, 2019; Accepted October 15, 2019

DOI: 10.3892/etm.2019.8199

Abstract. Pulse oximetry and measurement of regional cerebral oxygen saturation (rcSO₂) are used to monitor peripheral and cerebral oxygenation, respectively. However, the response of rcSO₂ and pulse oxygen saturation (SpO₂) 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 rcSO₂, SpO₂, 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 SpO₂ decreased to 90% or rcSO₂ decreased by >10% of the baseline level. The duration from throwing of mechanical ventilation to the start of the rcSO₂ decline was shorter than that of SpO₂ (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 rcSO₂ was longer than that of SpO₂ (84.8±24.3 sec vs. 15.2±6.8 sec; P<0.001). From the point where mechanical ventilation was paused to when rcSO₂/SpO₂ began to increase, rcSO₂ and SpO₂ 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 SpO₂, the rcSO₂ 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).

Introduction

Consecutive and non-invasive pulse oxygen saturation (SpO₂) 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 SpO₂ 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 (rcSO₂) via near-infrared spectroscopy (NIRS). NIRS takes advantage of the tissue penetration abilities of light of the near-infrared spectrum. In contrast to SpO₂, rcSO₂ 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, rcSO₂ 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 rcSO₂ monitoring during surgery (5-9). The use of rcSO₂ is increasing, but the routine use of rcSO₂ as a standard-of-care monitor is still not recommended at present.

Although it has been reported that rcSO₂ provides an earlier alert during hypoxia compared with pulse oximetry (10), whether SpO₂ and rcSO₂ 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 SpO₂ and rcSO₂ in response to hypoxia in preschool patients. It was hypothesized that SpO₂ may exhibit the same response to hypoxia as rcSO₂.

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Key words: hypoxia, pulse oxygen saturation, regional cerebral oxygen saturation, children
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/m² or >31 kg/m²; 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 anesthesia. Throughout the present study, all patients were continuously monitored, with their rcSO₂ (SenSmart™; Nonin Medical, Inc.) being assessed using a cerebral oximetry probe (reading rcSO₂ every 5 sec), which was placed on the middle of the forehead, and SpO₂ 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 SpO₂ probe. Heart rate (HR), electrocardiogram and end-tidal carbon dioxide partial pressure (P₆CO₂) were also continuously monitored. Induction of anesthesia was performed using propofol 2-3 mg/kg, fentanyl 2-3 μg/kg and cisatracurium 0.1-0.2 mg/kg. Anesthesia 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 P₆CO₂ 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 SpO₂ decreased to 90% or until the rcSO₂ 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, SpO₂ and rcSO₂ were recorded at the designated time-points: T₀ indicates the time-point prior to application of oxygen prior to oxygenation; T₁ indicates baseline, the time-point at which the mechanical ventilation was stopped; T₂ indicates the time-point at which SpO₂ began to drop from the baseline level; t₁ indicates the time-point at which rcSO₂ began to drop from the baseline level; T₃ indicates the time-point of SpO₂ decreasing to 90% or rcSO₂ decreasing by >10% of the baseline level and mechanical ventilation being recovered; T₄ indicates the time-point at which SpO₂ began to rise from the minimum value following ventilation; t₂ indicates the time-point at which rcSO₂ began to rise from the minimum value following ventilation; T₅ indicates the time-point at which SpO₂ returned to the baseline level, t₃ indicates the time-point at which rcSO₂ returned to the baseline level. S₁-T₄ indicates the value of SpO₂ at T₁ (baseline)-the value of SpO₂ at T₄ (the minimum value); R₁-T₄ indicates the value of rcSO₂ at T₁ (baseline)-the value of rcSO₂ at t₄ (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/m², 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 T₀, the SBP, MAP and DBP were decreased at the time-points from T₁ to T₄/t₃, and the HR was decreased at the T₁ time-point (P<0.001). Compared with those at T₁, the MAP and DBP were increased at the T₂ time-point and the HR was increased from the T₂/t₃ to the T₃/t₄ time-point (P<0.001), as presented in Table I.

Changes of rcSO₂ and SpO₂ over time. The values for rcSO₂ and SpO₂ are provided in Table II and the different time-intervals are stated in Table III. Compared with the SpO₂, the rcSO₂ exhibited an earlier decrease in response
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-T_3=13.4±6.2 sec vs. T_4-T_3=18.9±6.5 sec; P<0.001) and the duration of t_4-t_3 was longer than that of T_4-T_3 (84.8±24.3 sec vs. 15.2±6.8 sec; P<0.001). In addition, the duration of t_1-T_3 was longer than that of T_1-T_3 (98.2±24.3 sec vs. 34.1±6.8 sec; P<0.001). From T_1-t_1 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_3-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_1-t_4, R_1-t_4 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
hypoxia. Furthermore, rcSO₂ increased earlier and slower than SpO₂ 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 SpO₂ >85% for cerebral circulation (12). A study performed by Gupta et al (13) reported that by increasing the vascular resistance where the threshold of SpO₂ 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 SpO₂ was set at 90%. It has been reported that a decline of >25% from the baseline level, or the value of rcSO₂ 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 rcSO₂ from the baseline was used as a threshold to ensure patients' safety.

The present study demonstrated that after pausing mechanical ventilation (acute apnea), the rcSO₂ decreased earlier and declined slower than SpO₂. A previous study revealed that with SpO₂ maintained in the normal range, a decrease of >20% may be observed in cerebral oxygen saturation (17). Another study indicated that SpO₂ readings were 10-15 sec delayed compared with rcSO₂ readings in neonates (9). Similar results were also reported by Tanidir et al (18). In the present study, the decrease of rcSO₂ occurred ~40 sec earlier than that of SpO₂. Tobias (10) suggested that these changes may be associated with different ‘blood beds’, which are evaluated using monitors. It has been demonstrated that SpO₂ only captures arterial oxygen saturation and measures saturation in the arterial bed, but there is a correlation of rcSO₂ values with mixed venous (70%) and arterial (30%) oxygen saturations (3). In contrast to SpO₂, rcSO₂ 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 rcSO₂ would occur first. During hypoxia, the decline of rcSO₂ reflects a concurrent decrease in arterial oxygen 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 I. Dynamic changes of SBP, MBP, DBP and HR at different time-points.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>T₀</th>
<th>T₁</th>
<th>T₂/₄</th>
<th>T₃</th>
<th>T₄/₅</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>112±12</td>
<td>94±11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>98±13&lt;sup&gt;a&lt;/sup&gt;/97±12&lt;sup&gt;a&lt;/sup&gt;</td>
<td>97±13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>97±12&lt;sup&gt;a&lt;/sup&gt;/97±13&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>80±9</td>
<td>62±8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>68±11&lt;sup&gt;b&lt;/sup&gt;/66±9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>65±9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>65±9&lt;sup&gt;b&lt;/sup&gt;/65±9&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>64±11</td>
<td>46±9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>53±11&lt;sup&gt;b&lt;/sup&gt;/51±9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>49±9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>49±9&lt;sup&gt;b&lt;/sup&gt;/49±9&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>96±16</td>
<td>83±13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>93±12&lt;sup&gt;b&lt;/sup&gt;/91±10&lt;sup&gt;b&lt;/sup&gt;</td>
<td>92±16&lt;sup&gt;b&lt;/sup&gt;</td>
<td>92±16&lt;sup&gt;b&lt;/sup&gt;/92±16&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

All values are expressed as the mean±standard deviation (n=30). Compared with T₀, *P*<0.001; compared with T₁, *P*<0.001. SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure; HR, heart rate. T₀, time-point prior to application of any oxygen for pre-oxygenation; T₁, baseline, the time-point at which mechanical ventilation was stopped; T₂, the time-point at which SpO₂ began to drop from baseline; T₃, the time-point at which SpO₂ decreased to 90% or rcSO₂ decreased by >10% of the baseline and mechanical ventilation was recovered; T₄, the time-point at which SpO₂ began to rise from the minimum value following ventilation; T₅, the time-point at which SpO₂ began to rise from the minimum value following ventilation; T₆, the time-point at which rcSO₂ returned to the baseline level, t₁, the time-point at which rcSO₂ returned to the baseline level.

### Table II. Dynamic changes of SpO₂ and rcSO₂ at different time-points (n=30).

<table>
<thead>
<tr>
<th>Item</th>
<th>T₀</th>
<th>T₁</th>
<th>T₂/₄</th>
<th>T₃</th>
<th>T₄/₅</th>
</tr>
</thead>
<tbody>
<tr>
<td>SpO₂ (%)</td>
<td>99.7±0.5</td>
<td>99.7±0.5</td>
<td>99.7±0.5</td>
<td>90±0.0</td>
<td>84.7±3.2</td>
</tr>
<tr>
<td>rcSO₂ (%)</td>
<td>81.4±3.9</td>
<td>87.0±3.6</td>
<td>87.0±3.6</td>
<td>81.8±4.5</td>
<td>80.4±4.0</td>
</tr>
</tbody>
</table>

All values are expressed as the mean±standard deviation (n=30). Compared with S₃₁₁₃₈₆, R₃₁₄₄ₐ was smaller (9.7±0.5% vs. 5.3±2.7%, *P*<0.001). SpO₂, pulse oxygen saturation; rcSO₂, regional cerebral oxygen saturation; S₃₁₄₄₉, the value of SpO₂ at T₁ (baseline)-the value of SpO₂ at T₃ (the minimum value); R₃₁₄₄₉, the value of rcSO₂ at the T₁ time-point (baseline)-the value of rcSO₂ at T₃ (the minimum value). T₀, time-point prior to application of any oxygen for pre-oxygenation; T₁, baseline, the time-point at which mechanical ventilation was stopped; T₂, the time-point at which SpO₂ began to drop from baseline; T₃, the time-point at which SpO₂ began to drop from baseline; T₄, the time-point at which SpO₂ decreased to 90% or rcSO₂ decreased by >10% of the baseline and mechanical ventilation was recovered; T₅, the time-point at which SpO₂ began to rise from the minimum value following ventilation; T₆, the time-point at which rcSO₂ began to rise from the minimum value following ventilation; T₇, the time-point at which SpO₂ returned to the baseline level, t₁, the time-point at which rcSO₂ returned to the baseline level.
This may cause the arterial part of the NIRS signal to increase, leading to rcSO₂ 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 rcSO₂ may explain the early change in rcSO₂. The effect of perfusion on rcSO₂ levels has also been indicated by Schwaberger et al (20).

After restarting ventilation, rcSO₂ was increased earlier than SpO₂, but its increasing rate was slower, with rcSO₂ and SpO₂ exhibiting similar dynamic changing curve patterns.

rcSO₂ was increased with a mean delay of 13.4 sec, whereas the increase of SpO₂ 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

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**Table III. Comparison of the time difference between SpO₂ and rcSO₂ during the response to hypoxia (sec).**

<table>
<thead>
<tr>
<th>Duration</th>
<th>SpO₂</th>
<th>rcSO₂</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>T₀/T₁</td>
<td>124.4±20.5</td>
<td>80.2±23.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T₁/T₂</td>
<td>60.6±13.7</td>
<td>104.8±27.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T₂/T₃</td>
<td>18.9±6.5</td>
<td>13.4±6.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T₃/T₄</td>
<td>15.2±6.8</td>
<td>84.8±24.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>T₄/T₅</td>
<td>34.1±6.8</td>
<td>98.2±24.3</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

All values are expressed as the mean±standard deviation (n=30). SpO₂ pulse oxygen saturation; rcSO₂ regional cerebral oxygen saturation; T₀, baseline, the time-point at which mechanical ventilation was stopped; T₁, the time-point at which SpO₂ began to drop from baseline; T₂, the time-point at which rcSO₂ began to drop from baseline; T₃, the time-point at which SpO₂ decreased to 90% or rcSO₂ decreased by >10% of the baseline and mechanical ventilation was recovered; T₄, the time-point at which SpO₂ returned to the baseline level; T₅, the time-point at which rcSO₂ returned to the baseline level.

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Figure 2. Pearson’s correlation scatter plot of T₀-T₄/T₅. From T₀ to T₄/T₅, the rcSO₂ and SpO₂ values exhibited a significant correlation (Pearson’s correlation coefficient=0.317; P=0.027). T₀, the time-point at which SpO₂ began to drop from the baseline level; T₄, the time-point at which rcSO₂ began to drop from the baseline level; T₅, the time-point at which rcSO₂ decreasing to 90% or rcSO₂ decreasing by >10% of the baseline level and mechanical ventilation being recovered.

Figure 3. Spearman’s rank correlation scatter plot of T₀/T₅-T₄. From T₀ to T₅/T₄, the rcSO₂ and SpO₂ values exhibited a significant correlation (Spearman’s correlation coefficient=0.489; P=0.006). T₀, the time-point at which SpO₂ decreased to 90% or rcSO₂ decreased by >10% of the baseline and mechanical ventilation was recovered; T₄, the time-point at which SpO₂ began to rise from the minimum value following the ventilation; T₅, the time-point at which rcSO₂ began to rise from the minimum value following the ventilation.

Figure 4. Dynamic changes of SpO₂ and rcSO₂ during hypoxia. *P<0.001 vs. T₀/T₅, *P<0.001 vs. T₀/T₄, *P<0.001 vs. T₄/T₅, *P<0.001 vs. T₄/T₄, SpO₂ pulse oxygen saturation; rcSO₂ regional cerebral oxygen saturation; T₀, time-point prior to application of any oxygen for pre-oxygenation; T₅, baseline, the time-point at which mechanical ventilation was stopped; T₄, the time-point at which SpO₂ began to drop from baseline; T₅, the time-point at which rcSO₂ began to drop from baseline; T₄, the time-point at which SpO₂ decreased to 90% or rcSO₂ decreased by >10% of the baseline and mechanical ventilation was recovered; T₅, the time-point at which SpO₂ began to rise from the minimum value following ventilation; T₅, the time-point at which SpO₂ returned to the baseline level, T₅, the time-point at which rcSO₂ returned to the baseline level.
the earlier increase of rcsO₂ than that of SpO₂ following the recovery of ventilation, as a result of the oxygenated blood preferentially being distributed to the brain. Delayed vasodilation in the periphery, in comparison to the cerebral blood, may provide an additional explanation for the time difference observed between the increase of rcsO₂ and that of SpO₂ (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, rcsO₂ and SpO₂ exhibited similar dynamics in their changing curve patterns, and rcsO₂ was more sensitive compared with peripheral SpO₂.

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