

# Oxygen Reserve Index

## A Novel Noninvasive Measure of Oxygen Reserve—A Pilot Study

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### ABSTRACT

**Background:** Pulse oximetry provides no indication of downward trends in  $P_{aO_2}$  until saturation begins to decrease. The Oxygen Reserve Index (ORI) is a novel pulse oximeter–based nondimensional index that ranges from 1 to 0 as  $P_{aO_2}$  decreases from about 200 to 80 mmHg and is measured by optically detecting changes in  $S_{vO_2}$  after  $S_{aO_2}$  saturates to the maximum. The authors tested the hypothesis that the ORI provides a clinically important warning of impending desaturation in pediatric patients during induction of anesthesia.

**Methods:** After preoxygenation, anesthesia induction, and tracheal intubation, the anesthesia circuit was disconnected and oxygen saturation was allowed to decrease to 90% before ventilation recommenced. The ORI and  $S_{pO_2}$  values were recorded from a Masimo Pulse Co-Oximeter Sensor at the beginning of apnea, beginning and end of intubation, beginning and end of the ORI alarm, and 2 min after reoxygenation.

**Results:** Data from 25 healthy children, aged  $7.6 \pm 4.6$  yr, were included in the analysis. During apnea, the ORI slowly and progressively decreased over a mean of  $5.9 \pm 3.1$  min from  $0.73 \pm 0.16$  at the beginning of apnea to  $0.37 \pm 0.11$ .  $S_{pO_2}$  remained 100% throughout this initial period. Concurrently with alarm activation, the ORI began to decrease rapidly, and in median of 31.5 s (interquartile range, 19 to 34.3 s), saturation decreased to 98%.

**Conclusions:** In this pilot study, the ORI detected impending desaturation in median of 31.5 s (interquartile range, 19–34.3 s) before noticeable changes in  $S_{pO_2}$  occurred. This represents a clinically important warning time, which might give clinicians time for corrective actions. (ANESTHESIOLOGY 2016; 124:779-84)

THE relationship between the arterial oxygen saturation ( $S_{aO_2}$ ) and the partial pressure of oxygen ( $P_{aO_2}$ ) is characterized by the oxyhemoglobin dissociation curve that incorporates three distinct ranges: hypoxia, normoxia, and hyperoxia. The relationship follows a well-described sigmoidal curve until  $P_{aO_2}$  reaches about 80 mmHg or greater and the corresponding  $S_{aO_2}$  remains nearly constant at 100%. But at higher oxygen partial pressures,  $S_{aO_2}$  cannot increase further. Thus, pulse oximetry ( $S_{pO_2}$ ) cannot assess oxygen partial pressure once hemoglobin is fully saturated. That the relationship between oxygen partial pressure and hemoglobin saturation plateaus when  $P_{aO_2}$  exceeds about 80 mmHg is generally of little consequence because anesthesiologists are most concerned about low partial pressures of oxygen and rather less so about the large range of partial pressures that fully saturate hemoglobin.

However, there are situations in which it is helpful to know the partial pressure of oxygen in full saturated blood.

### What We Already Know about This Topic

- Oxygen supplementation delays detection of hypoventilation by pulse oximetry in both adults and children because oxyhemoglobin saturation remains 100% over a wide range of oxygen partial pressures exceeding about 80 mmHg
- The Oxygen Reserve Index is a nondimensional index that ranges from 1 (much reserve) to 0 (no reserve) and is measured by optically detecting changes in  $S_{vO_2}$  after  $S_{aO_2}$  saturates to the maximum
- The goal of this pilot study was to test the hypothesis that the Oxygen Reserve Index provides a clinically important warning of impending desaturation in pediatric patients during induction of anesthesia

### What This Article Tells Us That Is New

- During prolonged apnea in healthy anesthetized children, the Oxygen Reserve Index detected impending desaturation in median of 31.5 s (interquartile range, 19 to 34.3 s) before noticeable changes in  $S_{pO_2}$  occurred

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For example, very high partial pressures of oxygen—even for minutes—promote atelectasis<sup>1</sup>; and over days, high partial pressures cause pulmonary injury in critical care patients<sup>2</sup> and retinal injury in premature infants.<sup>3</sup> But there is also a common situation in which having an estimate of oxygen partial pressure of fully saturated blood would be clinically useful: during induction of anesthesia. Most patients are given 80 to 100% oxygen for several minutes before induction of anesthesia. The primary reason is that filling the functional residual capacity of the lungs provides several minutes worth of oxygen “reserve” in case of airway compromise.

Once desaturation starts during an airway crisis, it typically progresses rapidly to potentially lethal levels.<sup>4</sup> Desaturation during anesthetic induction is especially rapid in premature babies, infants, and young children,<sup>5–7</sup> as well as in obese children<sup>8</sup> and those suffering from upper respiratory infection.<sup>9</sup> Obtaining  $P_{aO_2}$  values from arterial blood samples are not helpful in such situations because analysis takes far too long, and securing the airway rather than obtaining arterial blood is the appropriate priority. During a crisis, having a reliable estimate of time remaining before hypoxemia becomes critical would help guide management. For example, it would be perfectly reasonable to again attempt intubation knowing that several minutes remained before desaturation, whereas some other ventilation strategy might be more appropriate if only 30 s remained.

Recently, Masimo Corporation (USA) developed a novel continuous and noninvasive measurement called the Oxygen Reserve Index (ORI). The ORI is a nondimensional index that ranges from 1 (much reserve) to 0 (no reserve) and is measured by optically detecting changes in  $SvO_2$  after  $SaO_2$  saturates to the maximum (see appendix). At this time, the device is not cleared in the United States and is limited to investigational use.

In this pilot study, we tested the hypothesis that the ORI provides a clinically important warning of impending arterial desaturation in pediatric patients during induction of anesthesia. Our primary outcome was the time that elapsed between activation of the Oxygen Reserve alarm until saturation reached 98% without ventilation, that is, the warning time the index provided of impending desaturation.

## Materials and Methods

After obtaining approval from the institutional review board (University of Texas Southwestern, Dallas, Texas), we obtained written informed consent from the parents of 33 pediatric patients and assent from 8 patients older than 10 yr. This prospective cohort study was conducted at Children's Medical Center in Dallas, Texas with patients enrolled during a 4-month period extending from January to April, 2014.

Inclusion criteria were pediatric surgical patients with American Society of Anesthesiologists physical status I and II, scheduled for general anesthesia with orotracheal intubation. We excluded patients with substantial cardiorespiratory compromise and those with known or anticipated difficult intubation.

Anesthesia was induced with 8% sevoflurane in 100%  $O_2$  supplemented by intravenous propofol (2 to 3 mg/kg) and fentanyl (1 to 2  $\mu$ g/kg). Muscle relaxants were not used. After patients became apneic, the trachea was intubated, and the endotracheal tube position was confirmed by the end-tidal carbon dioxide response to a single tidal volume breath. The anesthesia circuit was then disconnected to eliminate apneic oxygenation, and saturation was allowed to decrease to 90%. Subsequently, the anesthesia circuit was reconnected, and patients were ventilated with 100% oxygen until  $SpO_2$  returned to 99 to 100%. Thereafter, anesthesia continued per routine.

The ORI and the  $SpO_2$  were measured simultaneously at 1-s interval with a pulse co-oximeter sensor (R1 25L) placed on the patient's toe and connected to a Radical-7 pulse oximeter (Masimo, USA). The monitor averaging time was set at 8 s, per the manufacturer's default.

The Radical-7 pulse monitor, used for this study, was equipped to collect optical raw data needed to compute ORI but was not equipped to display it. Throughout the study, investigators were, thus, aware of oxygen saturation but not the ORI values that were subsequently provided to the investigators by the manufacturer after offline analysis of the collected raw data. The time at which the ORI alarm would have started was also calculated offline using the manufacturer's proprietary algorithm. Alarm activation was based on the fractional rate of change in ORI rather than on a specific oxygen reserve value. The alarm stopped when the ORI reached its minimal value of 0.

The ORI and  $SpO_2$  values were recorded from a Masimo Pulse Co-Oximeter Sensor at the beginning of apnea, beginning and end of intubation, beginning and end of the Oxygen Reserve alarm, and 2 min after reoxygenation. The beginning of induction (mask application) was designated elapsed time 0. The ORI was also recorded when  $SpO_2$  decreased to 98 and 90% or recovered from 90 and 98%. We also recorded the total apnea time defined as the time from the beginning of apnea until  $SpO_2$  reached 90% and ventilation was reinstated. The early warning time was defined from the beginning of the rapid decrease of the oxygen reserve values (indicated by the start of the ORI alarm), until saturation reached 98%.

## Statistical Analysis

The warning time is presented as a median warning time (interquartile range [IQR]) and 95% CI for mean, whereas the rest of the results are presented as mean  $\pm$  SDs. SAS 9.3 software (SAS Institute Inc., USA) was used to conduct the analyses.

## Results

Thirty-three patients were enrolled. Eight resumed spontaneous ventilation during the study period and therefore did not reach the target  $SpO_2$  of 92%. Therefore, these eight were excluded from analysis, leaving 25 patients whose results we report. Demographic and morphometric characteristics

**Table 1.** Patient's Demographics (N = 25)

Age (yr)	7.6 ± 4.6
Gender (male/female)	11/22
Weight (kg)	32 ± 22
Height (cm)	132 ± 31
BMI (kg/m <sup>2</sup> )	20 ± 6

BMI = body mass index.

of the participating patients are presented in table 1. All patients had a SpO<sub>2</sub> of 100% at the beginning of the apneic period.

The induction time (from the beginning of induction with mask application until patients became apneic) was 3.7 ± 1.9 min. Thereafter, the ORI slowly and progressively decreased over a mean of 5.9 ± 3.1 min from 0.73 ± 0.2 at the beginning of apnea to 0.4 ± 0.1 when the ORI began to decrease rapidly. SpO<sub>2</sub> remained 100% throughout this initial period (fig. 1).

Concurrently with alarm activation, the ORI began to decrease rapidly, and in median of 31.5 s (IQR, 19 to 34.3 s), saturation decreased to 98%. Thus, the alarm would have provided a median of 31.5 s (IQR, 19 to 34.3 s; 95% CIs for mean, 23.4 to 60.2 s) warning of impending desaturation had the system been working in real time. The mean (SD), median (IQR), and 95% CI for mean data at all study times are presented in table 2. Among 22 of the 25 patients included in our analysis, the warning time was between 0 and 50 s. The remaining three patients' warning times were at 98, 112, and 221 s (fig. 2).

After reinstatement of ventilation, SpO<sub>2</sub> values continued to decline to a mean saturation of 88 ± 3% before recovering to a SpO<sub>2</sub> of 98% 34 ± 6 s later. The oxygen saturation nadir was 84 ± 2% (table 3).

The ORI was at its minimum value (0) when oxygen saturation was less than 98% and increased rapidly to

0.5 ± 0.3 as saturation increased to 99% and above during reoxygenation.

Mean blood pressures and heart rates at baseline, end of intubation, and at reoxygenation are presented in table 2, along with end-tidal P<sub>CO<sub>2</sub></sub> at the end of intubation and at reoxygenation.

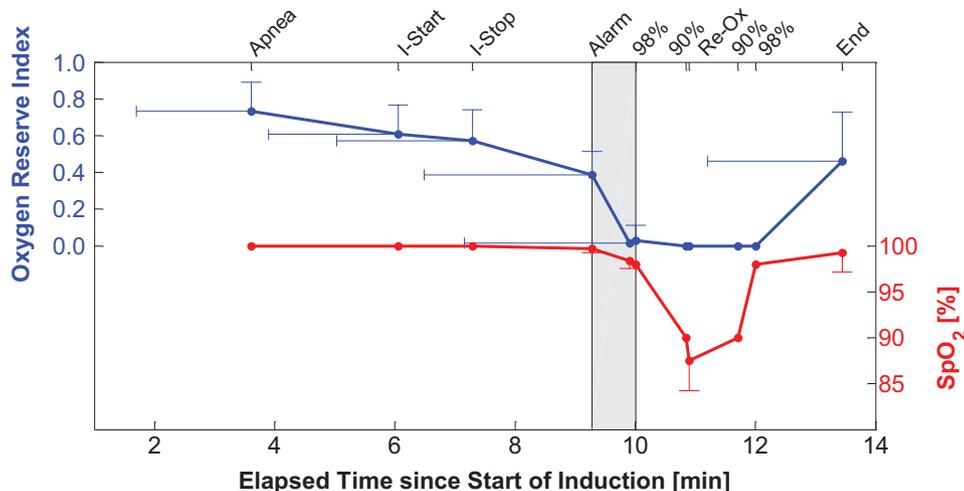
## Discussion

Pulse oximetry provides continuous, noninvasive assessment of arterial oxygen saturation and is a sensitive detector of hypoxemia<sup>10</sup> and major hypoxic events.<sup>11</sup> However, oxygen supplementation delays detection of hypoventilation by pulse oximetry in both adults<sup>12,13</sup> and children<sup>14</sup> because oxyhemoglobin saturation remains 100% over a wide range of oxygen partial pressures exceeding about 80 mmHg.<sup>15</sup>

For the same reason, it is difficult to predict when desaturation will start in apneic preoxygenated patients. For example, our patients maintained 100% oxygen saturation for an average of more than 6 min of apnea, which is consistent with previous clinical<sup>16,17</sup> reports and computational models.<sup>4</sup> However, there was a substantial variability among patients, which means that in any individual, it would have been difficult or impossible to predict just when the rapid desaturation phase would begin.

Our major finding is that monitoring the ORI before and during intubation detected impending desaturation in median of 31.5 s (IQR, 19 to 34.3 s) before noticeable changes in SpO<sub>2</sub> occurred. This represents a clinically important warning time that might give clinicians time for corrective actions. We are unaware of any other method that reliably provides warning that desaturation is imminent.

The ORI is a novel pulse oximeter-based nondimensional index that ranges from 1 to 0 as P<sub>aO<sub>2</sub></sub> decreases from about 200 to 80 mmHg. The ORI is based on the Masimo Rainbow SET technology in which the pulsatile signal is



**Fig. 1.** The Oxygen Reserve Index and SpO<sub>2</sub> values at different times of study. I-Start = beginning of intubation; I-Stop = end of intubation; Alarm = start of the Oxygen Reserve alarm; Re-Ox = reoxygenation; End = end of recording. The points and error bars represent mean (SD) values.

**Table 2.** The Oxygen Reserve Index (ORI) Mean, Median, and CI of Means Times at Various Study Times

	Time (min)			Delta Time (min)	Oxygen Reserve Index
	Mean $\pm$ SD	Median (IQR)	95% CI for Mean		
Start of apnea	3.7 $\pm$ 1.9	3.1 9 (2.7–3.7)	2.9–4.5	N/A	0.73 $\pm$ 0.16
Start of intubation	6.3 $\pm$ 1.9	5.8 (5.1–6.7)	5.5–7.1	2.6 $\pm$ 1	0.60 $\pm$ 0.16
End of intubation	7.5 $\pm$ 2	6.9 (6.3–8.3)	6.7–8.3	1.2 $\pm$ 0.5	0.56 $\pm$ 0.17
ORI alarm start	9.6 $\pm$ 2.4	9.9 (7.9–10.7)	8.6–10.6	2.1 $\pm$ 1.6	0.37 $\pm$ 0.11
ORI alarm stop	10.2 $\pm$ 2.4	10.3 (8.3–11.4)	9.2–11.2	0.6 $\pm$ 0.7	0.01 $\pm$ 0.6
98% SpO <sub>2</sub> (desaturation)	10.3 $\pm$ 2.5	10.3 (8.6–11.7)	9.3–11.3	0.1 $\pm$ 0.2	0.03 $\pm$ 0.8
90% SpO <sub>2</sub> (desaturation)	11.2 $\pm$ 2.4	11.2 (9.6–12.5)	10.2–12.1	0.9 $\pm$ 0.6	0.00 $\pm$ 0.0
Reoxygenation	11.2 $\pm$ 2.4	11.2 (9.6–10.5)	10.2–12.2	0.1 $\pm$ 0.3	0.00 $\pm$ 0.0
90% SpO <sub>2</sub> (reoxygenation)	11.7 $\pm$ 2.2	11.5 (10.3–12.7)	10.5–13.0	0.3 $\pm$ 0.3	0.00 $\pm$ 0.0
98% SpO <sub>2</sub> (reoxygenation)	12 $\pm$ 2.2	11.5 (10.3–12.7)	10.7–13.3	0.3 $\pm$ 0.8	0.00 $\pm$ 0.0
2 min after reoxygenation	13.5 $\pm$ 2.3	13.2 (12.1–14.5)	12.1–14.7	1.4 $\pm$ 1.1	0.46 $\pm$ 0.26

Delta time: time from one study time measurement to the next.

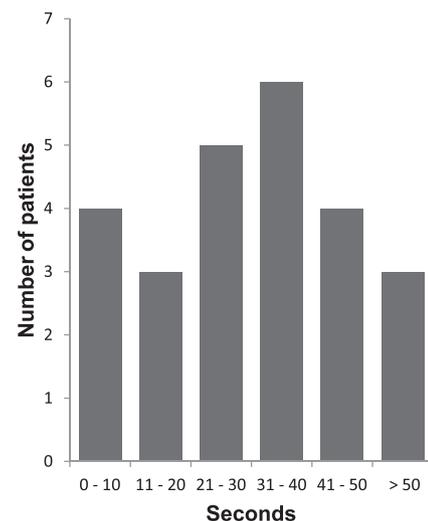
IQR = interquartile range; N/A = not applicable; SpO<sub>2</sub> = oxygen saturation.

extracted, enabling detection of changes in Pao<sub>2</sub> relative to the changes in Svo<sub>2</sub> at the measurement site. The ORI algorithm combines the Fick principle with the absorption properties of both arterial and venous hemoglobin wavelength. Specifically, as Pao<sub>2</sub> increases beyond 100 mmHg, Svo<sub>2</sub> continues to increase, even though Sao<sub>2</sub> has effectively saturated at 100%. This modest increase in Svo<sub>2</sub> above its normal value of approximately 75% will eventually stop as Pao<sub>2</sub> reaches significantly higher values (*i.e.*, more than 200 mmHg). The consequent wavelength change makes it possible to detect changes in Pao<sub>2</sub> up to about 200 mmHg if not even higher.

Potential confounding factors include oxygen consumption, cardiac output, blood pH, Pco<sub>2</sub>, temperature, the amount of perfusion (venous pulsation), and the presence of abnormal hemoglobins. For a more detailed explanation of the relationship between pulse oximetry and ORI, see appendix.

The ORI was validated in an institutional review board–approved study conducted by Masimo per ISO-80601 guidelines (not peer reviewed or published)<sup>18</sup> in 11 adult volunteers who underwent a variety of interventions to change their Pao<sub>2</sub> and SpO<sub>2</sub> levels. A total of 1,885 paired sets of ORI and Pao<sub>2</sub> values were collected. An index of 0.3 provided more than or equal to 80% specificity for a Pao<sub>2</sub> less than 150 mmHg. Although we did not evaluate Pao<sub>2</sub>, our results suggest a consistent and clinically useful relationship between the ORI and blood partial pressure of oxygen.

This being the initial report about the ORI, there remain many questions that will have to be addressed in future studies. For example, we did not evaluate the oxygen index in the setting of abnormally high oxygen consumption states such as fever or hypermetabolic stress. We expect that the relationship between Pao<sub>2</sub> and ORI will remain similar, but that the “warning time” will be reduced because Pao<sub>2</sub> will decrease more rapidly during apnea. More importantly, we did not evaluate the



**Fig. 2.** The early warning time distribution. The early warning time (seconds) was defined from the beginning of the rapid decrease of the oxygen reserve values (indicated by the start of the Oxygen Reserve Index alarm), until saturation reached 98%. Twenty-two of 25 patients had warning times between 0 and 50 s. The remaining three patients' warning times were at 98, 112, and 221 s.

correlation between ORI and Pao<sub>2</sub>, which remains of obvious clinical interest.

Furthermore, all our patients were preoxygenated before apnea was instituted, and ORI values were recorded. Thus, it is probable that at lower Fio<sub>2</sub> (room air), the warning time will be significantly shorter. Future studies will also have to quantify the accuracy or reliability of ORI and the extent to which hemodynamic and other common clinical perturbations might influence the index.

The ORI measurements were obtained from a pulse co-oximeter sensor placed on the patient's toe. Thus, sensor placement in other sites might provide different advanced warning times. Our study group was too small to permit evaluating the relationship between the ORI, age, and

**Table 3.** The Oxygen Reserve Index (ORI) and Oxygen Saturation (SpO<sub>2</sub>) Values, Hemodynamic Data, and End-tidal Carbon Dioxide at Various Study Times

	SpO <sub>2</sub> (%)	Heart Rate (beats/min)	Mean Blood Pressure (mmHg)	End-Tidal CO <sub>2</sub> (mmHg)
Start of intubation	99.9±0.1			
End of intubation	99.9±0.2	117±25	59±12	28±4
ORI alarm start	99.7±0.5			
ORI alarm stop	98.4±0.8			
98% SpO <sub>2</sub> (desaturation)	98.0±0.0			
90% SpO <sub>2</sub> (desaturation)	90.0±0.0			
Reoxygenation	87.3±3.1	109±24	54±7	46±7
90% SpO <sub>2</sub> (reoxygenation)	90.0±0.0			
98% SpO <sub>2</sub> (reoxygenation)	98.0±0.0			
2 min after reoxygenation	99.2±2.1			

weight. But although both likely influence the duration of apnea that can be maintained with full saturation, neither seems likely to directly influence accuracy of the ORI, any more than pulse oximetry is much influenced by morphometric and demographic characteristics.

We evaluated the ORI in one clinical scenario, but believe that warning of impending desaturation is likely to also prove useful during airway maneuvers (suctioning, bronchoscopy, etc.) in critically ill patients. The ORI may also be helpful for assessing the effectiveness of preoxygenation before rapid sequence induction and in avoiding hypoxia in neonates.

In conclusion, most intubations—fortunately—are rapid and smooth. But when they are not, clinicians need to make life-sustaining strategic decisions. In this pilot study, we found that during prolonged apnea in healthy anesthetized children, the ORI detected impending desaturation in median of 31.5 s (IQR, 19 to 34.3 s) before noticeable changes in SpO<sub>2</sub> occurred. Knowing even roughly how much time remains before the rapid desaturation phase begins seems likely to guide proper decisions.

### Acknowledgments

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### Competing Interests

Dr. Szmuk is a member of the Masimo Scientific Advisory Board and has received grant support from Masimo (Irvine, California) for previous and current research projects. The other authors declare no competing interests.

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### Appendix: Relationship between Pulse Oximetry and ORI

The Fick principle<sup>19</sup> relates oxygen consumption (V<sub>O<sub>2</sub></sub>) with cardiac output (CO) and oxygen content of arterial blood (Ca<sub>O<sub>2</sub></sub>) and deoxygenated venous blood (Cv<sub>O<sub>2</sub></sub>).

$$V_{O_2} = CO \times (Ca_{O_2} - Cv_{O_2}).$$

The oxygen content equation for arterial (Ca<sub>O<sub>2</sub></sub>) and venous (Cv<sub>O<sub>2</sub></sub>) blood is given by

$$Ca_{O_2} = (Sa_{O_2} \times tHb \times 1.34) + 0.003 \times (Pa_{O_2})$$

$$Cv_{O_2} = (Sv_{O_2} \times tHb \times 1.34) + 0.003 \times (Pv_{O_2}),$$

where tHb is total hemoglobin.

Substituting the oxygen content equation for the arterial (Ca<sub>O<sub>2</sub></sub>)<sup>20</sup> and venous (Cv<sub>O<sub>2</sub></sub>) blood, we are left with the following equation (where Sv<sub>O<sub>2</sub></sub> is the oxygen saturation in the venous blood and Pv<sub>O<sub>2</sub></sub> is the partial pressure of oxygen in the venous blood):

$$V_{O_2} = CO \times \left[ \left( (Sa_{O_2} \times tHb \times 1.34) + 0.003 \times Pa_{O_2} \right) - \left[ \left( Sv_{O_2} \times tHb \times 1.34 \right) + 0.003 \times Pv_{O_2} \right] \right].$$

This equation can be modified *via* oxygen saturation equations to the following format:

$$V_{O_2} = CO \times \left( 1.34 \times tHb \times (Sa_{O_2} - Sv_{O_2}) + 0.003 \times (Pa_{O_2} - Pv_{O_2}) \right). \tag{1}$$

The oxygen dissociation curve provides a relationship between Sa<sub>O<sub>2</sub></sub> and Pa<sub>O<sub>2</sub></sub> as given by the equation below.

$$Sa_{O_2} = f(Pa_{O_2}) \text{ and } Sv_{O_2} = f(Pv_{O_2}). \tag{2}$$

Substituting equation 2 in equation 1, we get:

$$V_{O_2} = CO \times \left[ 1.34 \times tHb \times (f(Pa_{O_2}) - f(Pv_{O_2})) + 0.003 \times (Pa_{O_2} - f^{-1}(Sv_{O_2})) \right].$$

Hence, for a constant oxygen consumption and cardiac output,  $SvO_2$  is directly proportional to  $PaO_2$ , as  $f$  (defined in eq. 2) is an increasing function. This results in the following relationship:

$$SvO_2 \propto PaO_2 \text{ for constant } V_{O_2}, CO. \quad (3)$$

Pulse oximeters work by measuring the absorption of pulsatile blood at the measuring site (finger). Pulsatile changes are observed at the arteries, capillaries, and in the venules, although to a lesser degree in the venules. A pulse oximeter absorption measurement at wavelength  $\lambda$ , denoted by  $A(\lambda)$ , is thus affected by both arterial and venous blood absorption changes.

$$A(\lambda) = A_a(\lambda) + \alpha A_v(\lambda), \quad (4)$$

where  $\alpha \ll 1$  and  $\alpha$  is dependent on perfusion at the measurement site.

In the absence of dyshemoglobins:

$$A_a(\lambda) = SaO_2 \times A_a^{O_2Hb}(\lambda) + (100 - SaO_2) \times A_a^{HHb}(\lambda) \quad (5)$$

$$A_v(\lambda) = SvO_2 \times A_v^{O_2Hb}(\lambda) + (100 - SvO_2) \times A_v^{HHb}(\lambda) \quad (6)$$

where  $A_a^{O_2Hb}$  is the absorption of oxyhemoglobin in arterial blood,  $A_v^{O_2Hb}$  is the absorption of oxyhemoglobin in venous blood,  $A_a^{HHb}$  is the absorption of deoxyhemoglobin (reduced) in arterial blood, and  $A_v^{HHb}$  is the absorption of deoxyhemoglobin (reduced) in venous blood.

Substituting equations 5 and 6 into equation 4:

$$A(\lambda) = (SaO_2 \times A_a^{O_2Hb}(\lambda) + (100 - SaO_2) \times A_a^{HHb}(\lambda)) + \alpha (SvO_2 \times A_v^{O_2Hb}(\lambda) + (100 - SvO_2) \times A_v^{HHb}(\lambda)). \quad (7)$$

Combining equation 3 and equation 7, we observe that  $A(\lambda)$  changes as a function of  $PaO_2$ .

The units of measurement for the different components in the equations are as follows:

- $SaO_2, SvO_2$  = percent saturation expressed as decimal fraction (e.g., 0.98).
- $tHb$  is measured in g/dl.
- $PaO_2$  and  $PvO_2$  are expressed in mmHg.
- Cardiac output is expressed in ml/min.
- $V_{O_2}$  is expressed in ml/min.

## References

1. Edmark L, Kostova-Aherdan K, Enlund M, Hedenstierna G: Optimal oxygen concentration during induction of general anesthesia. *ANESTHESIOLOGY* 2003; 98:28–33
2. Altemeier WA, Sinclair SE: Hyperoxia in the intensive care unit: Why more is not always better. *Curr Opin Crit Care* 2007; 13:73–8
3. Bizzarro MJ, Li FY, Katz K, Shabanova V, Ehrenkranz RA, Bhandari V: Temporal quantification of oxygen saturation ranges: An effort to reduce hyperoxia in the neonatal intensive care unit. *J Perinatol* 2014; 34:33–8
4. Hardman JG, Wills JS: The development of hypoxaemia during apnoea in children: A computational modelling investigation. *Br J Anaesth* 2006; 97:564–70
5. Laycock GJ, McNicol LR: Hypoxaemia during recovery from anaesthesia—An audit of children after general anaesthesia for routine elective surgery. *Anaesthesia* 1988; 43:985–7
6. Dupeyrat A, Dubreuil M, Ecoffey C: Preoxygenation in children. *Anesth Analg* 1994; 79:1027
7. Cote CJ, Rolf N, Liu LM, Goudsouzian NG, Ryan JF, Zaslavsky A, Gore R, Todres TD, Vassallo S, Polaner D, Alifimoff JK: A single-blind study of combined pulse oximetry and capnography in children. *ANESTHESIOLOGY* 1991; 74:980–7
8. Tait AR, Voepel-Lewis T, Burke C, Kostorzewa A, Lewis I: Incidence and risk factors for perioperative adverse respiratory events in children who are obese. *ANESTHESIOLOGY* 2008; 108:375–80
9. Kinouchi K, Tanigami H, Tashiro C, Nishimura M, Fukumitsu K, Takauchi Y: Duration of apnea in anesthetized infants and children required for desaturation of hemoglobin to 95%. The influence of upper respiratory infection. *ANESTHESIOLOGY* 1992; 77:1105–7
10. Severinghaus JW, Kelleher JF: Recent developments in pulse oximetry. *ANESTHESIOLOGY* 1992; 76:1018–38
11. Coté CJ, Goldstein EA, Coté MA, Hoaglin DC, Ryan JF: A single-blind study of pulse oximetry in children. *ANESTHESIOLOGY* 1988; 68:184–8
12. Fu ES, Downs JB, Schweiger JW, Miguel RV, Smith RA: Supplemental oxygen impairs detection of hypoventilation by pulse oximetry. *Chest* 2004; 126:1552–8
13. Ayas N, Bergstrom LR, Schwab TR, Narr BJ: Unrecognized severe postoperative hypercapnia: A case of apneic oxygenation. *Mayo Clin Proc* 1998; 73:51–4
14. Keidan I, Gravenstein D, Berkenstadt H, Ziv A, Shavit I, Sidi A: Supplemental oxygen compromises the use of pulse oximetry for detection of apnea and hypoventilation during sedation in simulated pediatric patients. *Pediatrics* 2008; 122:293–8
15. Severinghaus JW: Simple, accurate equations for human blood  $O_2$  dissociation computations. *J Appl Physiol Respir Environ Exerc Physiol* 1979; 46:599–602
16. Patel R, Lenczyk M, Hannallah RS, McGill WA: Age and the onset of desaturation in apnoeic children. *Can J Anaesth* 1994; 41:771–4
17. Cook TM, Wolf AR, Henderson AJ: Changes in blood-gas tensions during apnoeic oxygenation in paediatric patients. *Br J Anaesth* 1998; 81:338–42
18. Oxygen Reserve Index (ORI™). Available at: [http://www.masimo.co.uk/pdf/ori/LAB8543A\\_Whitepaper\\_ORI\\_British.pdf](http://www.masimo.co.uk/pdf/ori/LAB8543A_Whitepaper_ORI_British.pdf). Accessed October 8, 2014
19. Fritts HW, Cournand A: The application of the Fick Principle to the measurement of pulmonary blood flow. *Proc Natl Acad Sci USA* 1958; 44:1079–87
20. Martin L: All You Really Need to Know to Interpret Arterial Blood Gases. Lippincott Williams & Wilkins, 1999, pp 68–75