

## Spurious oxygen saturation value: A dilemma for anaesthesiologist

Sir,

Hypoxaemia is an important cause of peri-operative mortality and morbidity, making monitoring of arterial oxygen saturation ( $\text{SpO}_2$ ) essential. Pulse oximeter provides information about arterial blood oxygen status and thus used extensively in anaesthesia settings. However, it can be misleading as some individuals may have low  $\text{SpO}_2$  despite a normal partial pressure of oxygen ( $\text{PaO}_2$ ).

We report a case of a 33-year-old, American Society of Anesthesiologists physical status I, female scheduled for diagnostic hystero-laparoscopy under general anaesthesia (GA). During the pre-anaesthetic evaluation, she gave a history of previous hysteroscopy under GA, when she had low  $\text{SpO}_2$  intra-operatively and required post-operative Intensive Care Unit admission for observation. She was discharged next day without further evaluation.

All routine investigations and haemodynamic parameters were unremarkable. She had  $\text{SpO}_2$  of 88% on room air, which increased to 90% with 100% oxygen. It did not improve further after changing probe or probe site. The plethysmograph waveform was normal and of good quality. Cardio-pulmonary pathology was ruled out by chest skiagram, arterial blood gas analysis (ABG) on room air ( $\text{PaO}_2$  96.2 mmHg,  $\text{SaO}_2$  97.4%), pulmonary function test and two-dimensional echocardiography.

On further questioning, she informed about a similar problem in her mother.

We began to suspect possibility of haemoglobinopathy and further laboratory testing was done. Complete haemogram revealed normal haemoglobin (Hb), red blood cell (RBC) count and erythrocyte indices. Peripheral smear showed normocytic normochromic RBCs with no abnormal cells. Variant haemoglobins such as MetHb, HbF, HbD, HbS, HbE and HbC were also found to be within normal limits.

After discussion with the patient, decision was made to proceed with the procedure. The patient

was shifted to the operating room, and standard monitors (electrocardiogram,  $\text{SpO}_2$ , non-invasive blood pressure) were connected. Anaesthesia was induced with injection fentanyl 100  $\mu\text{g}$  and propofol 80 mg. Laryngeal mask airway was inserted, and anaesthesia was maintained with desflurane and oxygen/air mixture. ABG analysis repeated with 100%  $\text{O}_2$  revealed  $\text{PaO}_2$  356 mmHg,  $\text{SaO}_2$  of 98.5%. The 45 min procedure was performed uneventfully, and the patient was shifted to the recovery room. Her  $\text{SpO}_2$  remained between 88% and 90% throughout the procedure and recovery. She was discharged home the next day.

Low  $\text{SpO}_2$  requires immediate attention for cause and correction. Most frequently, we look for cardio-pulmonary pathology which further adds a financial burden to the patient. Various factors such as low perfusion state, motion artifacts, skin pigmentation, nail paint, haemoglobinopathies and faulty probes have been reported as causes for low  $\text{SpO}_2$  on the pulse oximeter.<sup>[1]</sup> Methaemoglobinemia is a common haemoglobinopathy implicated, but other less frequent variant haemoglobins have also been described, which causes a discrepancy between  $\text{SpO}_2$  and  $\text{SaO}_2$ .

Holbrook and Quinn<sup>[2]</sup> reported a patient with such a haemoglobinopathy, where 7% of Hb alpha chains were abnormal. The patient had  $\text{SpO}_2$  of 90% and  $\text{PaO}_2$  of 11.79 kPa (88 mm Hg) on room air. Methaemoglobin level was 0.7%. They identified amino acid alanine substitution for valine at position 62 of alpha chain on mass spectrometry. However, they were unable to confirm their findings.

Pulse oximeter uses two different wavelengths (660 and 940 nm) of light to estimate arterial blood oxygen saturation, based on different absorption spectra of oxy- and deoxy-haemoglobin. In the presence of variant haemoglobin, which has different absorption spectra, accuracy of this measurement may be compromised. Eleven different variants of such Hbs have been identified. Six cases with  $\alpha$ -globin gene missense mutations (Hbs Lansing, Titusville, Bonn, Delaware, M-Iwate and a novel haemoglobin), and five variant Hbs due to  $\beta$ -globin gene missense mutations (Hbs Hammersmith, Cheverly, Okazaki, Regina and Koln). These variant haemoglobins have variable oxygen affinity and carriers may be asymptomatic with low  $\text{SpO}_2$  and normal to discordant  $\text{SaO}_2$ .<sup>[3]</sup>

It is assumed that these variant haemoglobins are benign with less clinical significance, but they may be implicated for haemolysis, polycythaemia and impaired oxygen affinity. Unfortunately, data available for these variant haemoglobins is limited.

In our case, we were unable to detect the cause for low SpO<sub>2</sub> with a normal PaO<sub>2</sub> in an apparently normal patient. Further molecular or genetic studies were required, but it would have added financial, psychological burden and deviated her from the actual treatment she sought. Presence of normal PaO<sub>2</sub> does not necessarily indicate that nothing is wrong, as in cases of methaemoglobinemia and carboxyhaemoglobinemia; these variant haemoglobins provide diagnosis of exclusion in absence of signs and symptoms of hypoxaemia, presence of normal cardiovascular testing and absence of commonly known haemoglobinopathies. We think that not all cases of low SpO<sub>2</sub> with normal PaO<sub>2</sub> should be considered abnormal and these variant haemoglobins should be considered in differential diagnosis while investigating an asymptomatic patient with unexpectedly low SpO<sub>2</sub>.

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#### Conflicts of interest

There are no conflicts of interest.

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