Chapter 9
Cooximetry

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A patient is in pre-op holding with these vital signs.

HR 102 BP 135/88 RR 22 SaO_2 89%.

Supplemental O_2 6 L/min is administered by face mask with no improvement.

ABG: pH 7.42 PaO_2 206 $PaCO_2$ 35 SaO_2 100%.

The patient is asymptomatic with cyanosis, but an otherwise normal physical exam.

1. Why is the O_2 saturation different between the pulse oximeter and the blood gas?
2. How would cooximetry be helpful?
3. What variants of hemoglobin are detected by the cooximeter?
4. Describe the pathology and treatment of methemoglobinemia (MetHb).
5. Describe the pathology and treatment of carboxyhemoglobinemia (COHb).

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Answers

1. There are three distinct methods of determining the oxygen saturation of blood. The results may be interchangeable in healthy people, but different in dyshemoglobinemias.

   (a) Pulse oximetry utilizes the Beer-Lambert law, which states that light absorbance is proportionate to the concentration (c) of the light attenuating substance. Oxyhemoglobin (O_2Hb) and deoxyhemoglobin (HHb) have differing absorption of light. Oxyhemoglobin (O_2Hb) absorbs more at 940 nm and deoxyhemoglobin (HHb) more at 660 nm, and it is the ratio of absorption of light at 660 nm to 940 nm that determines the saturation, using an algorithm derived from healthy controls. The SaO_2 assumes the presence of only O_2Hb and HHb, thus

$$SaO_2 = \frac{cO_2Hb}{cO_2Hb + \text{cHHb}}$$

   cO_2HB is content of oxy Hb and cHHb is deoxy Hb. It will be inaccurate if abnormal hemoglobin's such as methemoglobin (MetHb) and carboxyhemoglobin (COHb) are present. MetHb is absorbed at both 660 and 940 nm. COHb is absorbed at 940 nm, similar to O_2Hb.

   (b) In the arterial blood gas (ABG) analysis, the pH and partial pressure of oxygen in the blood are measured, and the saturation is calculated from the standard oxygen dissociation curve.

   (c) Cooximetry also utilizes the Beer-Lambert law. Using multiple wavelengths of light, the concentrations of O_2Hb and other Hb species are determined by their different absorption at various wavelengths (Fig. 9.1). This allows the calculation of a fractional SaO_2 or percentage of oxyhemoglobin as a percent of total Hb including abnormal species.

$$\text{Fractional } SaO_2 = \frac{\text{O}_2Hb \times 100}{\text{O}_2Hb + HHb + \text{COHb} + \text{MetHb}}$$

   Cooximetry results for this patient measured 70% O_2Hb, 29% MetHb, and 1% COHb; thus, the fractional SaO_2 would only be 70%. Only 70% of the Hb is available for O_2 transport [1].

2. Cooximetry may be indicated if cyanosis or hypoxia measured by pulse oximetry fails to improve with O_2 administration or if there are discrepancies between O_2 sat and PaO_2 by ABG. It is also indicated for suspected carbon monoxide exposure. The cooximeter measures absorption at multiple wavelengths and can measure the concentration of many different Hb species. Pulse cooximetry applies multiple wave lengths of light to measure dyshemoglobins such as COHb and total hemoglobin concentration. They are not yet as accurate as a lab cooximeter and should be confirmed by the lab.

3. Cooximeters measure absorbance at more than two wavelengths from a minimum of six to as many as 128. Fractions of HHb, O_2Hb, COHb, and MetHb are

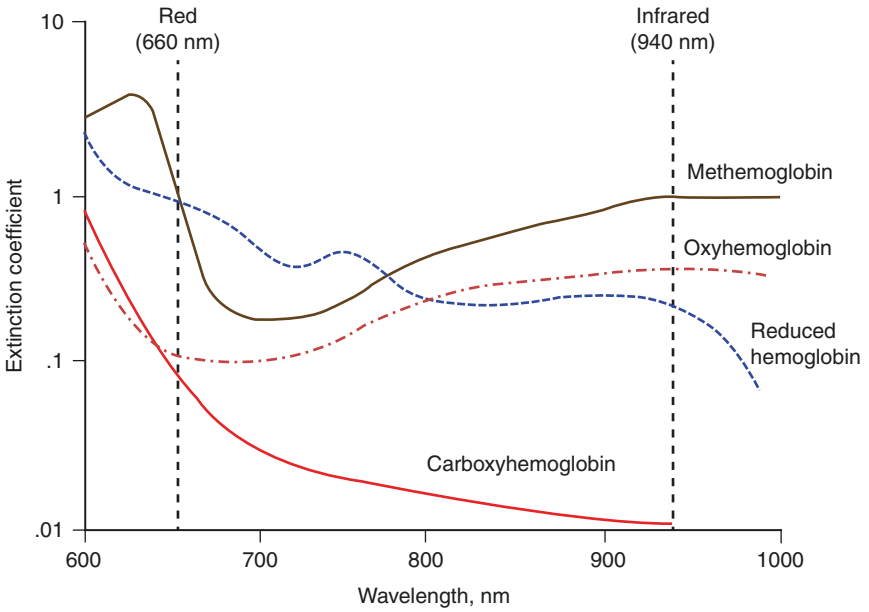

Fig. 9.1 Absorbance spectra of oxyhemoglobin, reduced hemoglobin, carboxyhemoglobin, and methemoglobin. Jubran. Critical Care.2015.19:272 (Open Access under terms of the Creative Commons Attribution License)

routinely measured. Arterial or venous blood may be used. It is important to note the difference of O_2 saturation versus fractional oxyhemoglobin in the presence of increased COHb or MetHb.

4. In methemoglobinemia the normal ferrous (Fe^{++}) in the hemoglobin (Hb) is oxidized to the ferric (Fe^{+++}) state which cannot bind oxygen and also shifts the oxygen dissociation curve to the left. Autoxidation of Hb to MetHb occurs spontaneously with a normal level of <2%. This is balanced by its reduction back to the ferrous state by cytochrome b5 reductase; an alternative is the NADPH generated by G6PD in the RBC, requiring an exogenous electron donor such as methylene blue. Methemoglobinemia may be hereditary, but it is more commonly acquired.

   Substances which may cause methemoglobinemia include:

   With high levels of MetHb, the pulse oximeter reading trends toward 85%; the O_2 dissociation curve shifts to the left. The fractional oxyhemoglobin will be lower than the SaO_2. When acutely acquired, MetHb levels <20% maybe asymptomatic. Symptoms include headache, fatigue, dyspnea, and lethargy. At levels >40%, altered consciousness, seizures, and death may occur. The diagnosis should be considered if the pulse oximetry is lower than the O_2 sat from an ABG. This can be confirmed with cooximetry.

Dapsone	Aniline dyes
Benzocaine	Primaquine
Lidocaine	Chloroquine
Prilocaine	Sulfonamides
Inhaled nitric oxide	Chlorates
Nitrites	Benzene derivatives
Nitroglycerin	Methylene blue

The treatment is to identify and stop the causative agent and administer methylene blue (MB) 1 to 2 mg/kg IV over 5 min. The response is usually rapid, and the MB may be repeated after 1 h if MetHb persists. MB will be ineffective and should be avoided in individuals with G6PD deficiency, more common in those of African or Mediterranean or Southeast Asian descent. If MB is contraindicated, ascorbic acid may be given 300–1000 mg/day orally. Supportive care as indicated may include ventilation, high inspired oxygen, and exchange transfusion [2].

5. Carbon monoxide poisoning is common and causes include faulty home heaters, inadequate home ventilation, auto exhaust, and house fires. Exposure may be chronic or acute. Iatrogenic carbon monoxide poisoning may result from the reaction of halogenated volatile agents, particularly desflurane and isoflurane with desiccated soda lime or baralyme. This has typically occurred on a Monday morning after O_2 was left flowing through the circuit drying out the absorbent canister [3]. Carbon monoxide has 200 times the affinity for Hb as O_2; thus low concentrations can produce significant COHb. COHb is normally 0–2% in nonsmokers and up to 9% in smokers. High levels of COHb reduce oxygen carrying capacity of the blood and will give a falsely high pulse oximetry reading. CO causes inflammatory response and binds to cytochrome c oxidase at the mitochondrial level, impairing cellular respiration. There are high rates of early and late neurocognitive and cognitive deficits, as well as cardiovascular dysfunction and acidosis. Symptoms are nonspecific and range from mild such as headache to severe such as confusion, loss of consciousness, or death.

   Treatment is administration of 100% O_2 at high flow rates. This hastens the release of CO from the Hb. Hyperbaric oxygen has been demonstrated to decrease late neurologic sequelae and may be indicated if COHb > 25% [4].

References

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