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Focus on Diagnosis: Co-oximetry

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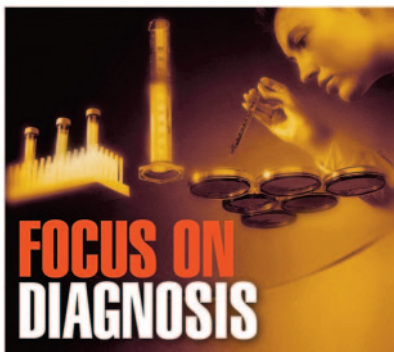
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Co-oximetry

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Introduction

A co-oximeter is a blood gas analyzer that, in addition to the status of gas tensions provided by traditional blood gas measurements, measures concentrations of oxygenated hemoglobin (oxyHb), deoxygenated hemoglobin (deoxyHb or reduced Hb), carboxyhemoglobin (COHb), and methemoglobin (MetHb) as a percentage of the total hemoglobin concentration in the blood sample. Use of co-oximetry is indicated when a history is consistent with toxin exposure, hypoxia fails to improve with the administration of oxygen, there is a discrepancy between the P_{aO_2} on a blood gas determination and the oxygen saturation on pulse oximetry (SpO_2), or the clinician suspects other dyshemoglobinemias such as methemoglobinemia or carboxyhemoglobinemia.

Pulse oximetry measures the oxygen saturation (SaO_2) of hemoglobin in arterial blood or the average amount of oxygen bound to each hemoglobin molecule. Blood gas analyzers calculate oxygen saturation from the measured parameters PO_2 and pH on the basis of standard oxygen-dissociation curves. Unfortunately, pulse oximetry, a noninvasive procedure, does not distinguish among the different types of hemoglobins. For example, in the case of methemoglobinemia, pulse oximetry may read 88%, but desaturation can be demonstrated with co-oximetry, recording 70% oxyHb and 30% MetHb.

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Each of the dyshemoglobins has a unique absorption spectrum, and the concentration can be derived from the Beer-Lambert law by measuring absorption at four specific wavelengths. Normal values are reported as a percentage of normal hemoglobins and include oxyHb, 45% to 70%; deoxyHb, 0% to 5% (arterial) or 15% to 40% (venous); MetHb, 0% to 1.5%; and COHb 0% to 2.5% (non-smoker) or 1.5% to 10% (smoker). The total hemoglobin level is age-dependent in children. An oxygen saturation gap is defined as a difference of more than 5% between the saturation that is calculated from a standard arterial blood gas analyzer and the saturation that is measured by co-oximetry. The presence of an oxygen saturation gap is consistent with the presence of dyshemoglobins.

Accurate collection techniques are necessary for appropriate interpretation of co-oximetry. Co-oximeter gases require a minimum of 0.3 mL of blood (venous or arterial). The blood must be collected in an air-free, heparinized syringe, be mixed well, have the air bubbles removed, and be placed on ice.

Methemoglobinemia

Methemoglobinemia is diagnosed definitively by co-oximetry. Congenital defects in erythrocyte enzyme systems may result in inherited methemoglobinemia. More common causes include drug effects (classically, local anesthetics such as lidocaine or benzocaine), gastroenteritis resulting in nitrite production, exposure to nitric oxide, or ingestion of nitrite-containing well water. These compounds oxidize normal ferrous (Fe^{++}) hemoglobin to the ferric (Fe^{+++}) form (ie, MetHb).

MetHb has no oxygen-carrying capacity and is unable to unload oxygen into the tissues.

Healthy persons should have 0% to 1.5% MetHb. Mild cyanosis occurs at levels below 10%, and symptoms of fatigue, dizziness, and nausea usually develop at concentrations above 20%. Concentrations higher than 45% may result in tissue ischemia, leading to arrhythmias, seizures, acidosis, and coma. Concentrations higher than 70% may be fatal. Patients who have methemoglobinemia may have the classic “chocolate-colored blood.”

Treatment is indicated for patients who have more than 30% MetHb and includes removal of the offending agent and administration of methylene blue, a cofactor of NADP-MetHb reductase. Methylene blue increases the capacity of this enzyme to reduce ferric iron concentrations and usually is administered intravenously in doses of 1 to 2 mg/kg. Clinicians can expect MetHb concentrations to fall in 1 to 2 hours. If this decrease does not occur, the dose may need to be repeated. However, caution is advised at total doses greater than 15 mg/kg because this amount of methylene blue paradoxically can cause methemoglobinemia. In refractory cases, exchange transfusion is recommended.

Carbon Monoxide (CO) Poisoning

CO is a colorless, odorless, nonirritating gas produced by incomplete combustion of organic matter. CO poisoning is the most common cause of death by poisoning in the United States. Sources of CO poisoning include house fires, car exhaust, indoor heaters, and stoves. Endogenous production occurs during the degradation of heme and contributes to

the baseline COHb concentration found in healthy people. In patients afflicted with hemolytic anemias, COHb can reach concentrations of 8%. Values also may be elevated in severe sepsis.

CO binds Hb with an affinity 200 times that of oxygen and causes a leftward shift in the oxygen-hemoglobin dissociation curve, resulting in decreased oxygen delivery and tissue hypoxia. CO also binds fetal Hb with greater affinity, making infants more vulnerable to its effects. CO often is called the “great mimicker” because it produces flulike or gastroenteritis-type symptoms; diagnosis requires a high degree of suspicion. The cherry-red color of the skin described in textbooks rarely is seen.

Pulse oximetry is not adequate to diagnose CO poisoning because COHb and oxyHb both absorb at 660 nm with the same absorption coefficient, thus falsely elevating the SpO₂ value. In addition, when a patient who already has received supplemental oxygen presents to an emergency department, his or her COHb concentration by co-oximetry may have normalized.

Common symptoms of acute CO intoxication include headache, dizziness, weakness, nausea, confusion, shortness of breath, and vision changes. Severe poisoning may result in dysrhythmias, hypotension, rhabdomyolysis, myocardial ischemia, cardiac or respiratory arrest, noncardiogenic pulmonary edema, seizures, and coma. Late clinical effects (not seen for 2 to 40 d) involve delayed neurologic sequelae, which may include any conceivable psychiatric or neurologic symptom.

Effects of chronic exposure to CO include headache, nausea, cerebellar dysfunction, mood disorders, low birthweight, reduced exercise toler-

ance, polycythemia, and cardiomegaly. Chronic exposure often occurs in smokers, who may have concentrations of COHb up to 8% or even 10% immediately after a cigarette.

Treatment of acute poisoning includes oxygen administration (hyperbaric oxygen may be considered in specific cases) and the usual support of airway, breathing, and circulation. Administering higher FIO₂ decreases the half-life of the COHb. Room air results in a half-life of 320 minutes, 100% oxygen causes a half-life of 80 minutes, and administration of hyperbaric oxygen reduces the half-life to 25 minutes.

Sulfhemoglobinemia

Sulfhemoglobinemia is a rare condition that may result from exposure to sulfonamides, acetanilide, phenazopyridine, nitrates, trinitrotoluene, and phenacetin. Sulfhemoglobin is unique because its presence is not detected by co-oximetry. Unlike the other dyshemoglobins, sulfhemoglobin shifts the oxygen dissociation curve to the right, resulting in oxygen being more readily available to tissues. Sulfhemoglobinemia should be suspected when a patient who has cyanosis has a normal PaO₂ and elevated methemoglobin concentration by co-oximetry but does not respond to methylene blue therapy. Treatment involves administering oxygen and discontinuing the offending agent.

Suggested Reading

- Kao LW, Nanagas KA. Toxicity associated with carbon monoxide. *Clin Lab Med.* 2006;26:99–125
- Konig MW, Dolinski SY. Pulmonary and critical care pearls. *Chest.* 2003;123:613–616

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