Emergency assessment of oxygenation

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Hypoxia and hypoxemia describe states of oxygen deficiency: hypoxia is deficiency in oxygenation at tissue or cellular level whilst hypoxemia is a suboptimal normal partial pressure of oxygen. Hypoxia and hypoxemia are interrelated, as untreated hypoxemia will ultimately result in hypoxia.

It should also be noted that although hypoxemia is a common cause of hypoxia, hypoxia can exist without hypoxemia.

Early detection and treatment of hypoxia and hypoxemia is important for patient outcomes, as oxygen deficiency is a known antecedent to adverse events, in particular unplanned intensive care unit admission and cardiac arrest.

Untreated hypoxia results in anaerobic metabolism, cellular acidosis, cell death and organ failure. Oxygenation may be assessed by clinical assessment, pulse oximetry and arterial blood gases. Each of these methods of assessment of oxygenation has strengths and limitations that should be understood by clinicians if assessment and subsequent management of oxygenation is to optimize patient care.

Hypoxia and hypoxemia are states of oxygen deficiency. Hypoxia is deficiency in oxygenation at tissue or cellular level whilst hypoxemia is a suboptimal normal partial pressure of oxygen [1]. Hypoxemia and hypoxia have four major causes.

The major causes of hypoxemia are [1]:

i) poor alveolar ventilation due to decreased tidal volume or respiratory rate

ii) decreased diffusion of oxygen from alveoli to pulmonary capillaries as a result of thickening of the alveolar-capillary membrane

iii) ventilation/perfusion mismatch

iv) shunting which occurs when venous blood travels from the right to the left side of the heart without passing by inflated alveoli, resulting in mixing of venous and arterial blood

The major causes of hypoxia are [2]:

i) hypoxic hypoxia, which occurs when the partial pressure of oxygen in arterial blood (pO_2(a)) is insufficient

ii) anemic hypoxia, which is decreased ability to transport oxygen and which may be the result of low hemoglobin levels (anemia, hypovolemic shock) or reduced functionality of hemoglobin (carbon monoxide poisoning)

iii) ischemic/stagnant hypoxia, which occurs when there is decreased delivery of oxygen to tissue cells as a result of insufficient blood flow and which may be the result of systemic conditions (cardiac failure) or local disruption to circulation (tissue edema or local arterial damage)

iv) histotoxic hypoxia or dysoxia, which occurs when tissues are unable to properly utilize oxygen despite adequate oxygen supply and which occurs as a result of cellular poisons (cyanide), abnormal tissue oxygen requirements, or poor oxygen diffusion across cell membrane (tissue edema)

Hypoxia and hypoxemia are interrelated, as untreated hypoxemia will ultimately result in hypoxia. It should also be noted that although hypoxemia is a common cause of hypoxia, hypoxia can exist without hypoxemia [1].

Supplemental oxygen will only correct hypoxemia due to respiratory dysfunction, e.g. inadequate concentration of inspired oxygen, ventilation/perfusion mismatch and diffusion defects, and it is of limited effectiveness in treating hypoxemia that occurs as a result of shunting [1].

Early detection and treatment of hypoxia and hypoxemia is important for patient outcomes. Suboptimal oxygenation is a well-documented antecedent to adverse events, in particular unplanned intensive care unit admission and cardiac arrest [3, 4, 5, 6]. Anaerobic cellular metabolism is one of the most damaging consequences of hypoxemia and/or hypoxia [7].

Normal cellular metabolism is an aerobic process producing up to 38 molecules of energy (adenosine triphosphate) [2, 7]. In states of oxygen deficiency, anaerobic metabolism occurs as cells attempt to produce energy; however, anaerobic metabolism produces only two energy (adenosine triphosphate) molecules and is only useful in meeting short-term cellular energy requirements [2, 7].

Lactic acid is a detrimental by-product of anaerobic metabolism and results in decreased cellular pH and reduced integrity of cellular and organelle membranes [2]. Untreated hypoxia and cellular acidosis result in cell death and organ failure [2].

Normal partial pressure of arterial oxygen is greater than 80 mmHg and a normal oxygen saturation is greater then 95 % [8]. However, hypoxemia is generally defined as a pO_2(a) of less than 60 mmHg [9] or an oxygen saturation of less than 90 % [10], leaving oxygen saturations in the range of 90-95 % and pO_2(a) ranging from 60 mmHg to 80 mmHg largely undefined [11].

In general, respiratory failure is defined as a pO_2(a) less than 50 mmHg and a pCO_2(a) greater than 50 mmHg [12].

Failure to clearly define hypoxemia results in uncertainty about exact indications for the use of supplemental oxygen [1], and variability of criteria for hypoxemia may also contribute to inconsistencies in the use of supplemental oxygen.

This issue is further complicated by the fact that patients with a normal pO_2(a) or SpO_2 may still have tissue hypoxia, and supplemental oxygen may be warranted irrespective of pO_2(a) or oxygen saturation [1].

Examples of cases where supplemental oxygen is indicated despite normal pO_2(a) or normal SpO_2 include

carbon monoxide poisoning [13] or states of increased oxygen demand, such as sepsis [14]. In these instances, the need for supplemental oxygen is based on clinical judgment [13].

Oxygenation may be assessed by clinical assessment, pulse oximetry and arterial blood gases. Pulse oximetry is commonly used to obtain a rapid and continuous assessment of oxygenation. Pulse oximetry measures oxygen saturation, which is the percentage of hemoglobin that is saturated with oxygen [2].

Pulse oximetry does not provide any information about adequacy of ventilation, plasma oxygen transport, hemoglobin levels, cardiac output, oxygen delivery to the tissues or cellular utilization of oxygen [15], and these limitations should be considered when interpreting pulse oximetry findings.

Arterial blood gas analysis quantifies arterial partial pressures of oxygen and carbon dioxide and blood pH [16] and is often regarded as the *"gold standard"* by which to assess oxygenation.

Clinicians should note, however, that arterial blood gas analysis only provides information about oxygenation status at the time of sample collection [16], and that the parameters measured by arterial blood gas analysis provide information about ventilation, oxygenation and acid base balance [16], but do not provide information about the effectiveness of tissue perfusion or cellular use of oxygen [1].

Pulse oximetry and arterial blood gas findings should be interpreted in conjunction with the clinical situation as patients with normal pulse oximetry and/or normal pO_2(a) may still be hypoxic. Although there are limitations to the clinical assessment of oxygenation, untreated hypoxia will ultimately result in physiological signs and symptoms as organs begin to fail [17].

Pallor is associated with the early stages of hypoxia and occurs as a result of peripheral vasoconstriction [18]. Cyanosis is a late sign of severe hypoxia [1, 9], and as not all patients with significant hypoxemia develop cyanosis [9], cyanosis is an unreliable sign of hypoxemia [19].

Inadequate oxygenation causes a stress response resulting in increased respiratory rate, heart rate and blood pressure [1, 18]. The majority of patients who are hypoxemic will exhibit at least one vital sign of abnormality [10].

The respiratory signs and symptoms of hypoxia include dyspnea [1, 9], increased respiratory effort [18, 20], nasal flaring [18, 20], mouth breathing [18] and tachypnea [1, 9, 21]. Peripheral chemoreceptors are stimulated to increase the respiratory rate when pO_2(a) reaches 60-70 mmHg [1], so onset of tachypnea is an indication of physiological change [21].

Tachycardia and mild hypertension are signs of early hypoxia and bradycardia, and hypotension occurs as body systems decompensate as a result of untreated hypoxia, resulting in metabolic acidosis [1]. Because the brain requires a continuous supply of oxygen to function, it is very sensitive to oxygen deficiency, so neurological dysfunction is an early sign of hypoxia [1].

Neurological signs of hypoxia may be subtle (anxiety, agitation or restlessness) [9, 18] but if untreated, hypoxia will cause confusion and loss of consciousness [18]. Each method of assessment of oxygenation has strengths and limitations that should be understood by clinicians if assessment and subsequent management of oxygenation is to optimize patient care.

Two case stories

1. A 28-year-old male presents to the emergency department with increasing shortness of breath, fevers and productive cough. He has a past history of asthma that is usually well-controlled.

On arrival, he is alert but restless, respiratory rate 28 with moderate use of accessory muscles, heart rate 128, blood pressure 125/85, skin pale, cool and dry, oxygen saturation measured by pulse oximetry 92 % in room air and temperature 38.7 °C.

He was treated with high-flow supplemental oxygen: 15 L/minute via a mask, continuous nebulized salbutamol, intravenous steroids and antibiotics and was later admitted to a general medical unit for ongoing care.

This patient required aggressive oxygen management for two reasons. First, he clearly showed clinical signs of hypoxic hypoxia (altered conscious state, pallor, tachycardia, tachypnea, increased respiratory effort and suboptimal oxygen saturation) and second, he was also in a state of increased oxygen demand as a result of febrile illness.

2. A 76-year-old lady presents to the emergency department with increasing dizziness and lethargy. On arrival, she is alert, cooperative, respiratory rate 22 with normal respiratory effort, heart rate 108, blood pressure 135/80, skin pale, warm and dry and oxygen saturation measured by pulse oximetry 97 % in room air. She becomes dyspneic and complains of chest pain on exertion.

This patient was treated with supplemental oxygen at 8 L/minute via a face mask, and a 12-lead ECG showed sinus tachycardia. Her chest pain and dyspnea resolved and her heart rate fell to 94. Laboratory testing showed hemoglobin of 60 g/L (6 g/dL), and she was later transfused with two units of packed cells and admitted for endoscopy.

This patient was profoundly anemic and, showing clinical signs of anemic hypoxia, so warranted administration of supplemental oxygen despite her normal oxygen saturation.

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