Oxygenation and Ventilation Monitoring

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Objectives

- Recognize O2 physiology in the neonatal period.
- Recognize carbon dioxide physiology.
- Describe abnormal Hb binding states.
- Interpretation of blood gas.
- Monitoring of CO2 (ventilation) and O2 (oxygenation).
Oxygen Physiology

Oxygen alveolar/arterial (A-a) gradient:

\[(\text{ideal alveolar O2 equation}) - (paO2)\]  
\[= (\text{FiO2} \times 713) - (\text{paCO2}/0.8) - \text{paO2}\]
A-a gradient of O2 approach

- It increases with higher level of inspired O2, normal A-a gradient in room air is 10-15 and 80-100 if FiO2 is 100%.

- In room air, it increases with ventilation/perfusion mismatch.

- If FiO2 is 100%, the gradient increases with shunting.

- If the A-a gradient in 100% FiO2 is above 600 for 8-12 hours, ECMO is indicated.
Effect of altitude on paO2

As altitude increases, barometric pressure decreases and partial P of O2 decreases; thus, need to increase FiO2 to maintain equal paO2.

If a neonate with RDS is requiring 100% O2 in the mountain area where the PB is 687, what percent O2 is needed to result in the same paO2 at the sea level?

\[(p_B - p_{H2O}) \times FiO2_{1} = (p_B - p_{H2O}) \times FiO2_{2}\]

\[p_{H2O} = 47 \text{ in both locals and } P_b \text{ at sea level} = 760;\]

\[(687-47) \times 1.0 = (760-47) \times ?, \quad ? = 89\%\]
Oxyhemoglobin dissociation curve

Curve shift to left (L for Low, low acid, low pCO2, low 2,3-DPG, low temperature) lower P50 (defined as pO2 at which Hb is 50% saturated). Decreased release of oxygen from Hb (poor O2 delivery), occurs with:

- Alkalosis/ high pH
- Lower paCO2
- Increased fetal Hb
- Decreased temperature
- Decreased 2,3-diphosphoglycerate (DPG)
- HbCO (carboxyhemoglobin)
Oxyhemoglobin dissociation curve

Curve shift to right (or RIGHT as in R for Release with/ increased 2,3 DPG, increased Hydrogen and increased Temperature, higher P 50. Increased release of oxygen (improved O2 delivery) occurs with:

- Acidosis/low pH
- Higher pCO2
- Increased adult Hb
- Increased temperature
- Increased 2,3 –DPG
- Alpha thalassemia (associated with decreased fetal Hb)
Oxygen Delivery (ml/kg/min)

- To alveoli = (alveolar minute ventilation) × (FiO2)
  = (tidal volume – dead space) × (frequency) × (FiO2)

- To tissue = (O2 carrying capacity) × (cardiac output) × 10
  = (O2 bound to Hb + dissolved O2) × (cardiac output) × 10
Oxygen Carrying Capacity

O2 content of blood = O2 bound to Hb + dissolved O2

\[(1.34 \text{ ml O2/gHb}) \times \text{Hb} \times \text{O2 sat} + [(0.003 \text{ ml O2/dl torr}) \times \text{paO2}]\]

- A small change in pO2 will give a large increase in O2 content on the steep part of the curve.
- There is profound effect in O2 content with changes in Hb concentration
- There is very little change in O2 content following a change in dissolved O2
- Fetal Hb has a higher affinity for O2 than the adult, so 1.37 ml O2/gHb is often used instead of 1.34
Oxygen Consumption = Fick Principle = VO2

\[ \text{VO2} = \text{CO} \times (\text{CaO}_2 - \text{CVO}_2) \]

\[ = \text{CO} \times (1.34 \text{ ml/g Hb}) \times (\text{Hb concentration}) \times (\text{art. sat} - \text{Ven. Sat}) \]

If O2 delivery to tissues is decreased tissues will attempt to maintain tissue O2 level by:

1. Increasing O2 extraction
2. Recruiting more capillaries

Increased oxygen consumption:

1. Increased caloric intake
2. Decreased body temperature
3. Neonates more than adults (6-8 Vs 3.2 ml/kg/min)
4. Term more than premature infant
5. AGA infant more than SGA
Carbon Dioxide Physiology

CO2 transport evolved to maximize CO2 from the body (with high concentrations) to the atmosphere (low concentration):

Total CO2 = dissolved CO2 (7%) + HCO3 – 70% HbCO2
Bohr Effect

- Changes in paCO2 can shift the oxyhemoglobin dissociation curve leading to increased oxygenation of the blood in the lungs and increased release of O2 from blood to the tissues.

- Alveolar CO2 increases as blood passes through the lungs; thus paCO2 decreases and oxyhemoglobin curve shift to the left, leading to increased amount of O2 bound and increased O2 transport to tissues.

- After traveling to the tissues, CO2 enters the blood from the tissues, leading to increased paCO2 levels and oxyhemoglobin curve shifted to the right. This allows for improved O2 delivery.
Halden Effect

- The reverse of the Bohr effect.
- It is more important in assisting CO2 transport than the Bohr effect in promoting O2 transport.
- The binding of O2 to Hb in alveolar capillaries increases CO2 unloading from the capillary blood into the alveoli.
- In the tissue capillaries, O2 is removed from Hb and thus, increases CO2 binding to hemoglobin.
- CO2 dissociation curve shifts to upwords when O2 decreases.
- CO2 dissociation curve shifts to right when O2 saturation increases.
Henderson-Hasselbalch Equation

Hydrogen concentration = \((24 \times pCO2)/ \text{bicarbonate concentration}\)

- An increase in bicarb concentration leads to an increase in pH.
- An increase in pCO2 leads to decrease in pH.
- Since one can change pCO2 by changing RR, the respiratory system can alter the pH.
- The kidney can alter the bicarbonate concentration and thus, change the pH.
Abnormal Binding Hemoglobin States

- Carboxyhemoglobinemia

- Methemoglobinemia
Carboxyhemoglobinemia

- Excess carbon monoxide from tobacco smoke, fires, motor vehicle exhaust.
- CO binds to Hb much better than O2, very low level of CO will compete with O2 for Hb binding and impair O2 carrying capacity of RBCs.
- CO increases the affinity of Hb for the remaining bound O2 (shift of oxyhemoglobin curve to the left) and decreases O2 delivery to the tissues.
- Since HbCO absorbs light similar to O2 bound to Hb, HbCO will falsely elevate O2 saturation.
- CO can cross the placenta and bind fetal Hb.
- Management: 100% O2
Methemoglobinemia

- **Etiology:** Excess nitrate, nitrite, maternal prilocaine, aniline dyes, hemoglobin M disease.

- **Physiology:** Normally and on daily basis we accumulate metHb and RBCs will reduce it. In methemoglobinemia, the iron of Hb changes from the ferrous (reduced) to ferric (oxidized) state, decreasing O2 carrying capacity.

- Normal paO2 yet decreased O2 sat (less O2 bound to Hb).

- Calculated O2 sat is normal in contrast to measured O2 sat.

- **Diagnosis:** Arterial blood appears brown following exposure to O2.

- **Management:** Methylene blue (alter urine colour to blue/green)
Blood Gas Interpretation

**NORMAL VALUES:**

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<th></th>
<th>Arterial</th>
<th>Venous</th>
<th>Capillary</th>
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<tbody>
<tr>
<td>pH:</td>
<td>7.4 (7.38-7.42)</td>
<td>7.36 (7.31-7.41)</td>
<td>7.35-7.4</td>
</tr>
<tr>
<td>pO2</td>
<td>80-100 mm Hg</td>
<td>35-40 mm Hg</td>
<td>45-60 mm Hg</td>
</tr>
<tr>
<td>pCO2</td>
<td>35-45 mm Hg</td>
<td>41-52 mm Hg</td>
<td>40-45 mm Hg</td>
</tr>
<tr>
<td>Sat</td>
<td>&gt;95% on RA</td>
<td>60-80% on RA</td>
<td>&gt;70%</td>
</tr>
<tr>
<td>HCO3</td>
<td>22-26 mEq/L</td>
<td>22-26 mEq/L</td>
<td>22-26 mEq/L</td>
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<tr>
<td></td>
<td>22-26 mEq/L</td>
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<tr>
<td>BE</td>
<td>-2 to +2</td>
<td>-2 to +2</td>
<td>-2 to +2</td>
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</tbody>
</table>
Blood Gas Interpretation

- Blood gases are helpful in determining the adequacy of respiratory function of the baby (oxygenation and ventilation) as well as the baby's acid/base balance.

- They can be performed from arterial (either a stab or via an arterial line), venous (through an intravenous cannula) or capillary (heel prick) specimens.

- Arterial stabs may be taken from the radial artery (provided there is also a palpable ulnar pulse) or from the brachial artery, although this is in close proximity to the median nerve.

- All three specimens will give a good assessment of acid/base status and pCO2, whereas arterial specimens are required to assess pO2.

- It is always important to note the FiO2 (percentage inspired oxygen) when interpreting blood gases. Each unit should have their own reference ranges.
The pH is a negative logarithm of hydrogen ion concentration \([H^+]\), that is a decrease in pH from 7.0 to 6.0 represents a ten-fold increase in \([H^+]\).

Normal neonatal pH is defined as 7.30 to 7.40, then

- pH > 7.4 is an alkalosis
- pH < 7.3 is an acidosis

The pH is proportional to HCO\(_3\) (or base excess), therefore:

- an abnormal increase in HCO\(_3\) (or base excess) increases the pH (metabolic alkalosis)
- an abnormal fall in HCO\(_3\) (or base excess) decreases the pH (metabolic acidosis)

The pH is inversely proportional to pCO\(_2\), therefore:

- an abnormal increase in pCO\(_2\) decreases the pH (respiratory acidosis)
- an abnormal decrease in pCO\(_2\) increases the pH (respiratory
Rules Of Interpretation

- Δ in pCO₂ of 10mm Hg should Δ pH by 0.08.

- pH Δ of 0.15 is equal to Δ in HCO₃ of 10mEq/L.

- Normal pCO₂ in the face of respiratory distress is a sign of impending respiratory failure.
Many organic acids are produced during normal metabolism. Sometimes they can accumulate in the blood (e.g. lactic acid).

The hydrogen ion (H+) may be 'mopped up' by buffers including bicarbonate (HCO3).

Bicarbonate is unique because it can be converted to CO2, which can be blown off by the lungs (provided the baby is not in respiratory failure).

The following bi-directional equation demonstrates this:

$$H^+ + HCO_3^- \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2$$
Acid-Base Diagram

Respiratory Acidosis (eg pCO2 $\geq$ 50 mmHg, pH < 7.30)

- The pCO2 is abnormally high and is due to inadequate alveolar ventilation.
- Causes include: depression of the breathing centre in the brain, upper airway obstruction, stiffness of the chest wall or significant ventilation/perfusion imbalance.
- If the respiratory acidosis is chronic, the body will respond by trying to excrete acid and retain bicarbonate in the urine resulting in a compensatory rise in serum bicarbonate (metabolic alkalosis).
- The treatment of a respiratory acidosis is to treat the underlying cause and to consider the need for commencing or increasing mechanical ventilation.
- The latter is achieved by either increasing the tidal volume (increasing PIP or decreasing PEEP) or by increasing the respiratory rate.
Respiratory Alkalosis (eg pCO2 < 35 mmHg, pH > 7.40)

- This occurs when the pCO2 is abnormally low and is usually due to excessive mechanical ventilation or to abnormal control of ventilation (e.g. during hypoxic-ischaemic encephalopathy).

- The baby may also be trying to compensate for a primary (intracellular or extracellular) metabolic acidosis, although the pH will never become alkalotic (as the baby will never over-compensate).

- The treatment of a respiratory alkalosis is to wean the mechanical ventilation by reducing PIP or tidal volume, then respiratory rate.
Metabolic Acidosis (eg HCO$_3$ < 17 mmol/L or B.E. < minus 6.0 mEq/L, pH < 7.30)

- This may occur where there is a rise in free H+ ions that cannot be totally buffered. In this case the anion gap is increased.
- Causes include; lactic acidosis secondary to tissue hypoxia (e.g. hypotension, sepsis and PDA) or the inability to excrete/buffer accumulated organic acids (e.g. protein loading and renal immaturity).
- Another common cause of metabolic acidosis, particularly in the extremely premature infant is excessive loss of HCO$_3$ in the urine or gut. In this case the anion gap is normal.
- Metabolic acidosis is rarely due to an inborn error of metabolism.
The treatment of a metabolic acidosis is to treat the underlying cause, consider volume expansion (e.g. 10 mls/kg of normal saline) if the baby is thought to be hypovolaemic or to administer NaHCO3 if the metabolic acidosis is severe (controversial) or refractory (e.g. bicarbonate wasting).

Bicarbonate should not be given if the pCO2 is elevated as the pH will not change (according to the above formula, a metabolic acidosis is merely being replaced by a respiratory acidosis).
Metabolic Alkalosis (eg HCO₃ > 28 mmol/L or B.E. > plus 4.0 mEq/L, pH > 7.40)

- This occurs where the plasma HCO₃ or base excess is abnormally high.
- Causes include: hypochloraemia (the level of bicarbonate and chloride in plasma are reciprocally related), which may be due to diuretic therapy or upper gastrointestinal obstruction (e.g. pyloric stenosis).
- The baby may also be trying to compensate for a respiratory acidosis, although the pH will never become alkalotic (as the baby will never over-compensate).
- The treatment of a metabolic alkalosis is to treat the underlying cause (e.g. chloride replacement) or the underlying cause of the respiratory acidosis.
Base Excess

- This is one way of looking at the metabolic component.

- It refers to the 'amount of base that would have to be added to one litre of the baby's blood at 40 mmHg pCO2 to return the pH to normal.

- It is a calculated value and will be erroneous if the pCO2 is not normal.

- In these circumstances, the 'metabolic' component of the blood gas should be assessed using the plasma HCO3 level.
Acid-Base Disorders

- Any one of the mentioned four scenarios can occur in isolation, with or without compensation.

- These are classified as simple acid-base disorders.

- When a combination of simple acid-base disturbances occurs, the baby has a mixed acid-base disorder. When there is a mixed disorder, it is sometimes difficult to know which is the primary and which is the compensatory component.
Acid-Base Disorders

- In such circumstances a helpful principle is that normal physiological processes never over-compensate. The pH can be relatively normal in the following situations:
  - respiratory acidosis with metabolic compensation
  - metabolic acidosis with respiratory compensation
  - metabolic alkalosis with respiratory compensation
  - respiratory alkalosis with metabolic compensation

The fourth is extremely unusual in neonates.
Respiratory Disturbances

- **Acute respiratory acidosis** occurs when CO$_2$ is retained acutely.
- **Chronic respiratory acidosis** occurs when the retained CO$_2$ gets buffered by renal retention of HCO$_3$\textsuperscript{-}. The pH is higher than in acute respiratory acidosis, but it is still <7.4.
- **Acute respiratory alkalosis** occurs when CO$_2$ is blown off acutely.
- **Chronic respiratory alkalosis** occurs when the reduction of CO$_2$ is compensated for by the renal excretion of HCO$_3$\textsuperscript{-}. The pH is lower than in acute respiratory alkalosis, but it is still >7.4.
Metabolic Disturbances

- Acute metabolic acidosis gets compensated by CO₂ reduction within 12-24 hours. The pH is still usually <7.4.

- Metabolic alkalosis: Usual causes are pyloric stenosis, chronic diuretic use, and bicarbonate infusions.

- Otherwise healthy people do not usually retain CO₂ to compensate for metabolic alkalosis.

- Patients who are severely dehydrated or have lung disease will retain CO₂ to compensate for metabolic alkalosis.
Hypoxemia

There are five reasons for hypoxemia:

- FiO$_2$ too low (high altitude)
- Global alveolar hypoventilation
- Right-to-left shunts
- V/Q mismatch
- Incomplete diffusion
Are carbon dioxide detectors useful in neonates?

- Maintenance of neonatal normocarbia may prevent chronic lung disease and periventricular leucomalacia.
- This requires frequent arterial sampling, which has risks.
- Alternative methods for measuring CO₂ are therefore desirable.
- These include:
  - end tidal CO₂, capillary sampling, and transcutaneous measurements.
- CO₂ detectors have also proved effective and rapid indicators of endotracheal intubation. However, this method relies on the presence of exhaled CO₂, which may be reduced in certain situations, such as cardiopulmonary arrest.
- Colorimetric CO₂ detectors are therefore valuable adjuncts for airway management, especially during resuscitation, but Paco₂ is still the best measure of CO₂ in neonatal practice.

Monitoring During MV

- **Frequent ABG:**
  - 20 minutes after initiation of MV
  - After 20 minutes of major alteration
  - Immediately if condition markedly deteriorate
  - Otherwise every 4-8 hours

- **Desired ABG:**
  - pH > 7.25
  - PCO₂ = 40 – 60 mm Hg
  - PO₂ = 50 – 70 mm Hg

- Continuous vital signs monitoring

- Pulse oxymeter
Approach to Blood Gas Changes

- A sudden ↓ in PO$_2$ and ↑ in PCO$_2$ with rapid clinical deterioration:
  - Disconnect from ventilator and bag the baby:
    - If condition improve the problem with ventilator:
      - Concentration of FIO$_2$ going to ventilator
      - Presence of leaks or disconnected tube
      - Mechanical or electrical failure
    - If condition did not improve the problem with infant:
      - Check gas entry bilaterally listen over stomach
      - Check position of heart and trachea
      - Act accordingly
Approach to Blood Gas Changes

- Gradual ↓ in PO₂ and gradual deterioration of PCO₂:
  - Suggest inappropriate ventilatory sitting
    - ↑ PIP
    - ↑ PEEP
    - ↑ I:E ratio or the inspiratory time
- Gradual ↑ in PCO₂ without change in PO₂
  - Insufficient alveolar ventilation (TV)
    - ↑ PIP by 2-5 cm H₂O
    - ↑ ventilator rate by 10
Approach to Blood Gas Changes

- **↓** in PCO2 caused by over ventilation.
  - Dangerous because of respiratory alkalosis:
    - Decreased cardiac output, CBF and tissue O₂ delivery especially if patient is hypotensive
    - Reduce overall alveolar ventilation:
      - **↓** PIP, rate or inspiratory time.

- **↑** in PO2 unaccompanied by changes in PCO₂:
  - Suggest a decrease in intrapulmonary shunting and in degree of atelacasis
    - **↓** O₂ to 40 – 70 before attempting to reduce ventilatory sitting
## Effect of Ventilator Sitting changes on Blood Gases

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<th>Change</th>
<th>PaCO2</th>
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