An Approach to Blood Gas Interpretation

Dr Marcin Nejthardt
Dept of Anaesthesia & Perioperative Medicine
University of Cape Town

Step 1 – PO₂

Consider:
Is the blood gas taken from a patient on room air or supplemental oxygen?
Is the blood gas an arterial or a venous sample?
The above two are important to determine if hyper or hypoxaemia is present.

Hypoxaemia is defined as $P_aO_2 < 8$ kPa (60 mmHg).

Hypoxia on the other hand is a low oxygen content in tissue and has a number of causes and can be classified as:
1. Hypoxic (high altitude, low $FIO_2$, $V/Q$ mismatch, diffusion abnormality)
2. Anaemic (low Hb, carbon monoxide poisoning)
3. Stagnant (low cardiac output)
4. Histotoxic (inability of cells to utilize oxygen due to mitochondrial dysfunction eg. cyanide, inflammatory conditions (sepsis), neuro-inflammatory conditions (Alzheimer's, Parkinson's, multiple sclerosis) and ischaemia related reperfusion injury.

What is a normal PO₂?
This will depend on:
1. Sampling site: $P_aO_2$ (arterial) or $P_vO_2$ (venous)
   i. Mixed venous refers to the sample of blood from the Pulmonary Artery.
   ii. Central venous sample refers to blood drawn from the Internal Jugular or Subclavian Veins.
   iii. $PO_2$ will be lowest in a mixed venous sample, followed by the central venous sample and finally from a peripheral vein eg. cubital fossa vein.
2. Altitude: The greater the altitude, the lower the partial pressure. Johannesburg at an altitude of 1 700 m has an atmospheric pressure of 83 kPa (622 mmHg).

<table>
<thead>
<tr>
<th>$P_aO_2$ Normal Value kPa (mmHg)</th>
<th>Room air</th>
<th>40% facemask</th>
<th>100% via ETT</th>
<th>Saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Sea level</td>
<td>$P_aO_2$</td>
<td>13.3(100)</td>
<td>32(240)</td>
<td>97-100</td>
</tr>
<tr>
<td></td>
<td>$P_vO_2$</td>
<td>5.3 (40)</td>
<td>6.7(50)</td>
<td>75- 83</td>
</tr>
<tr>
<td>- Johannesburg (1700m)</td>
<td>$P_aO_2$</td>
<td>10.3(77)</td>
<td>25(187)</td>
<td>95-100</td>
</tr>
<tr>
<td></td>
<td>$P_vO_2$</td>
<td>4.7(35)</td>
<td>6(45)</td>
<td>73-81</td>
</tr>
</tbody>
</table>

What is the Alveolar–arteriolar (A-a) gradient?

$A-a$ gradient = $P_aO_2 - P_vO_2$

$A-a$ gradient = $F_i(P_{atm} - P_{H_2O}) - \frac{P_{ACO_2}}{R} \cdot P_aO_2$

where: $F_i$ = fraction inspired oxygen
$P_{atm}$ = atmospheric pressure
$P_{H_2O}$ = Partial pressure of fully saturated water vapour at 37°C [6.3kPa(47mmHg)]
$R$ = Respiratory quotient. Depends on food source used for metabolic respiration. Typical value of 0.8
$P_aO_2$ = the measure $PO_2$ from an arterial blood gas
Normal A-a gradient is generally less than 2 kPa (15 mmHg) irrespective of altitude but does gradually increases with age.

An increased A-a gradient suggests a V/Q mismatch, diffusion abnormality or right to left shunt.

Note: Under normal physiological conditions (normal extraction of 5 mlO₂/100ml blood) adding supplemental oxygen has very little effect on increasing PvO₂ (mixed venous saturation of 75% equates to Po₂ 5.3 kPa(40 mmHg) as per O₂-Hb dissociation curve).

A problem with tissue extraction may result in mixed venous saturation rising to >90% with a subsequent PaO₂ rising to a higher level and approaching PaO₂ levels.

**Step 2 – pH**

Normal pH: 7.36 - 7.44

If pH < 7.36 = acidaemia

> 7.44 = alkalaemia

**Step 3 – PCO₂**

This is to assess the respiratory contribution to the pH.

Normal PaCO₂ (arterial)

\( PaCO₂ \) (venous)

<table>
<thead>
<tr>
<th>( PaCO₂ ) (kPa (mmHg))</th>
<th>Room air</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Value</td>
<td></td>
</tr>
<tr>
<td>Sea level</td>
<td></td>
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<tr>
<td>( PaCO₂ )</td>
<td>5.3 (40)</td>
</tr>
<tr>
<td>( PvCO₂ )</td>
<td>6.0 (45)</td>
</tr>
<tr>
<td>Johannesburg (1700 m)</td>
<td></td>
</tr>
<tr>
<td>( PaCO₂ )</td>
<td>4.7 (35)</td>
</tr>
<tr>
<td>( PvCO₂ )</td>
<td>5.3 (40)</td>
</tr>
</tbody>
</table>

Note:

1. Main adaptation to altitude is increased minute ventilation hence lowering PCO₂.
2. Lower PO₂ directly increases PCO₂ (Haldane effect)
3. Increasing FiO₂ has two effects on PCO₂
   a. Decrease in respiratory drive increases PCO₂
   b. Decreases binding to haemoglobin and decreases PCO₂ (Haldane effect)
4. A venous or arterial blood gas are reasonable at assessing a patient's CO₂

If elevated – respiratory acidosis
If decreased – respiratory alkalosis

**Step 4 – Metabolic contribution to pH**

Use standard base excess, anion gap, strong ion difference and \([A_{TOT}]\) to evaluate the metabolic component.

Lactate is a strong ion and thus forms part of the strong ion calculation and interpretation.

Normal values:

| Standard base excess | = -2 to +2 mEq/L |
| Anion gap            | = 8 - 16 mEq/L    |
| Strong ion difference| = 35 - 45 mEq/L   |
| \([A_{TOT}]\)        | = 14 - 17 mEq/L   |
| Lactate              | = 0.5 - 1 mmol/L  |
Step 5 – Is there any compensation?

Interpret the blood gas in the clinical context. Compensation never brings the pH back to normal range. If there is a normal pH in the face of obvious respiratory or metabolic disturbance then a mixed acid-base disturbance is present.

It is important to consider the blood gas in the context of the clinical scenario. Determining the adequacy of compensation can be attempted through a number of empirically developed formulae. One such is Winter’s Formula to assess the expected change in CO$_2$ for a given metabolic acidosis.

**Metabolic Acidosis:** (The One & a Half plus 8 Rule)

The expected P$_{CO_2}$ (in mmHg) is calculated from the following formula:

\[
\text{Expected P}_{CO_2} = 1.5 \times [HCO_3^-] + 8 \text{ (range: +/- 2)}
\]

Other formulae include:

**Acute Respiratory Acidosis:** (The 1 for 10 Rule)

The [HCO$_3^-$] will increase by 1 mmol/l for every 10 mmHg elevation in P$_{CO_2}$ above 40 mmHg. Expected [HCO$_3^-$] = 24 + ((Actual P$_{CO_2}$ - 40) / 10)

**Chronic Respiratory Acidosis:** (The 4 for 10 Rule)

The [HCO$_3^-$] will increase by 4 mmol/l for every 10 mmHg elevation in P$_{CO_2}$ above 40 mmHg. Expected [HCO$_3^-$] = 24 + 4((Actual P$_{CO_2}$ - 40) / 10)

**Acute Respiratory Alkalosis:** (The 2 for 10 Rule)

The [HCO$_3^-$] will decrease by 2 mmol/l for every 10 mmHg decrease in P$_{CO_2}$ below 40 mmHg. Expected [HCO$_3^-$] = 24 - 2((40 - Actual P$_{CO_2}$) / 10)

**Chronic Respiratory Alkalosis:** (The 5 for 10 Rule)

The [HCO$_3^-$] will decrease by 5 mmol/l for every 10 mmHg decrease in P$_{CO_2}$ below 40 mmHg. Expected [HCO$_3^-$] = 24 - 5((40 - Actual P$_{CO_2}$) / 10) (range: +/- 2)

**Metabolic Alkalosis:** (The Point Seven plus Twenty Rule)

The expected P$_{CO_2}$ (in mmHg) is calculated from the following formula:

\[
\text{Expected P}_{CO_2} = 0.7 \times [HCO_3^-] + 20 \text{ (range: +/- 5)}
\]

Compensatory changes in Acid – Base disturbances

A visual representation of the compensatory changes in acid-base disturbances. The numerator indicates the change in [HCO$_3^-$] in mmol/l whilst the denominator indicates the change in P$_{CO_2}$ in mmHg and (kPa). For ease of recall the metabolic compensations have been modified.

These unfortunately are not intuitive and difficult to commit to memory especially at the bedside.
An easier bedside approach may be to use the following approximations (see I Joubert ARC 2017,18)

<table>
<thead>
<tr>
<th>$P_{\text{CO}_2}$</th>
<th>$\text{pH}$</th>
<th>$\text{HCO}_3^-$</th>
</tr>
</thead>
<tbody>
<tr>
<td>12 mmHg</td>
<td>0.1</td>
<td>6 mEq/l</td>
</tr>
<tr>
<td>1.6 kPa</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Understanding the primary disturbance from the clinical scenario would point to the expected compensatory mechanism. Compensation is never complete (returning the pH back to a normal range). *Expected (appropriate) compensation* in clinical practice returns the pH to about halfway to normal.