BLOOD GAS ANALYSIS
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### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABE</td>
<td>Actual base excess</td>
</tr>
<tr>
<td>ABG</td>
<td>Arterial blood gas</td>
</tr>
<tr>
<td>AaDO₂</td>
<td>Alveolar to arterial oxygen gradient</td>
</tr>
<tr>
<td>Baro/PB</td>
<td>Barometric pressure</td>
</tr>
<tr>
<td>BB</td>
<td>Buffer base</td>
</tr>
<tr>
<td>BE</td>
<td>Base excess</td>
</tr>
<tr>
<td>BEecf</td>
<td>Base excess in extracellular fluid</td>
</tr>
<tr>
<td>BPD</td>
<td>Bronchopulmonary dysplasia</td>
</tr>
<tr>
<td>CH⁺</td>
<td>Concentration of hydrogen ion</td>
</tr>
<tr>
<td>CO₂</td>
<td>Carbon dioxide</td>
</tr>
<tr>
<td>ECMO</td>
<td>Extra corporeal membrane oxygenation</td>
</tr>
<tr>
<td>FiO₂</td>
<td>Fraction of inspired oxygen</td>
</tr>
<tr>
<td>HCO₃</td>
<td>Bicarbonate</td>
</tr>
<tr>
<td>H₂CO₃</td>
<td>Carbonic acid</td>
</tr>
<tr>
<td>MAP</td>
<td>Mean airway pressure</td>
</tr>
<tr>
<td>O₂CT</td>
<td>Oxygen content of blood</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>Partial pressure of carbon dioxide in arterial blood</td>
</tr>
<tr>
<td>PaO₂</td>
<td>Partial pressure of oxygen in arterial blood</td>
</tr>
<tr>
<td>pAO₂</td>
<td>Partial pressure of oxygen in alveoli</td>
</tr>
<tr>
<td>pH₂O</td>
<td>Water vapour pressure</td>
</tr>
<tr>
<td>PPHN</td>
<td>Persistent pulmonary hypertension in newborn</td>
</tr>
<tr>
<td>RBC</td>
<td>Red blood corpuscles</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
</tr>
<tr>
<td>RQ</td>
<td>Respiratory quotient</td>
</tr>
<tr>
<td>Sat</td>
<td>Saturation</td>
</tr>
<tr>
<td>SBE</td>
<td>Standard base excess</td>
</tr>
<tr>
<td>St HCO$_3$/SBC</td>
<td>Standard bicarbonate</td>
</tr>
<tr>
<td>TCO$_2$</td>
<td>Total carbon dioxide content of blood</td>
</tr>
<tr>
<td>THbA</td>
<td>Total haemoglobin concentration</td>
</tr>
<tr>
<td>UAC</td>
<td>Umbilical artery catheter</td>
</tr>
</tbody>
</table>
The terminology of arterial blood gas (ABG) is complex and confusing. It is made worse by the printouts generated by recent microprocessors. Basically, the machines measure pH, carbon dioxide, and oxygen. All other parameters are derived based on software in the machine which can be obtained manually if one knows how to use Siggaard Andersen Nomograms (given below).
Goals of ABG in newborn is to characterize the type of disorder, quantify the magnitude and assess the nature and extent of compensation.

**Indications for ABG**

(1) Severe respiratory or metabolic disorders

(2) Clinical features of hypoxia or hypercarbia

(3) Shock

(4) Sepsis

(5) Decreased cardiac output

(6) Renal failure

(7) Ideally any baby on oxygen therapy

**Collection of Samples**

Ideal artery for sampling in newborn is radial or umbilical artery. One must perform “Allen Test” to ensure collateral blood supply by ulnar artery before puncturing radial artery. If sample from umbilical artery catheter (UAC) is being taken, one should assure free flow of blood and remove three to four times dead space volume before sample is taken. Indwelling arterial line may only be put if round the clock facilities for ABG estimation are available considering this as a potent source of infection.

Arterialised capillary samples are comparable to arterial blood (Table I). If capillary sample (100-150 micro L) is being taken from prewarmed heel, let the capillary fill from the tissue site from where blood is oozing out (figure I). Avoid squeezing and first drop of blood. Rotate the capillary in palm or use “metal flea” magnet to mix anticoagulant with blood. Seal both ends of capillary till processed. Care should be taken not to include any air bubble in the capillary.
Venous blood is good for HCO$_3^-$ estimation but bad for pH, pCO$_2$ and pO$_2$. While drawing venous sample make sure that no tourniquet is applied, artery is not compressed and sample is drawn against the flow of blood towards heart.

**Table I: Comparison of Blood Gas Analysis at different sites**

<table>
<thead>
<tr>
<th></th>
<th>Arterial</th>
<th>Capillary</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>Same</td>
<td>----------</td>
<td>Lower</td>
</tr>
<tr>
<td>PO$_2$</td>
<td>Higher</td>
<td>$\rightarrow$</td>
<td>Lower</td>
</tr>
<tr>
<td>PCO$_2$</td>
<td>Lower</td>
<td>$\rightarrow$</td>
<td>Higher</td>
</tr>
<tr>
<td>HCO$_3^-$</td>
<td>Same</td>
<td>----------</td>
<td>Same</td>
</tr>
<tr>
<td>Recommendation</td>
<td>Good</td>
<td>Fair</td>
<td>Bad</td>
</tr>
</tbody>
</table>

**Precautions for collection of blood sample**

(1) Heparin is acidic and lowers pH. Use heparin of lower strength (1000 units per ml instead of 5000 units per ml).

(2) Use small volume of heparinised saline just for lubricating syringe and plunger. If volume is more, dissolved oxygen in heparinised saline may increase pO$_2$.

(3) Avoid air bubble and let syringe fill spontaneously.

(4) It is desirable to use a glass syringe as plastic syringes are permeable to air.

(5) Sample may be collected in a heparinised capillary from hub of needle used to puncture artery.

The sample should be processed immediately, preferably within 30 minutes. Blood is a living medium. The cells consume oxygen and produce CO$_2$. Drop in pO$_2$ depends on initial pO$_2$. If the latter is very high, significant drop may be noticed. The changes are as depicted in Table II. Slush of ice (not cubes) should be used for storing samples.
till processing. The sample should be shaken, homogenised before putting in machine.

**Table II: Changes in ABG every 10 minutes in vitro**

<table>
<thead>
<tr>
<th></th>
<th>37°C</th>
<th>4°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>0.01</td>
<td>0.001</td>
</tr>
<tr>
<td>pCO₂</td>
<td>0.1 mm Hg</td>
<td>0.01 mm Hg</td>
</tr>
<tr>
<td>pO₂</td>
<td>0.1 mm Hg</td>
<td>0.01 mm Hg</td>
</tr>
</tbody>
</table>

* It is obvious that blood sample should be stored at 4°C, if it cannot be processed immediately for minimal error.

**Terminology of ABG**

- **Acidosis**: pH < 7.3
- **Alkalosis**: pH > 7.5
- **Hypercapnia**: pCO₂ > 50 mm Hg
- **Hypocapnia**: pCO₂ < 30 mm Hg
- **Hypoxia**: pO₂ < 50 mm Hg
- **Hyperoxia**: pO₂ > 70 mm Hg

*Acidemia and alkalemia refer to blood while acidosis, alkalosis to tissue pH.

**Normal Neonatal ABG values**

- **PH**: 7.35 – 7.40
- **pCO₂**: 35 – 45 mm Hg
- **pO₂**: 50 – 70 mm Hg
- **HCO₃**: 20 – 24 mEq/L
- **BE**: ± 2
ABG values vary with age of neonate and even with gestational age (Table III, IV).

**Table III: ABG values based on neonatal age**

<table>
<thead>
<tr>
<th></th>
<th>Pre-birth (Scalp)</th>
<th>5 min after birth</th>
<th>1-7 days after birth</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>&gt;7.20</td>
<td>7.20-7.34</td>
<td>7.35-7.40</td>
</tr>
<tr>
<td>pCO₂</td>
<td>&lt;50</td>
<td>35-46</td>
<td>33-35</td>
</tr>
<tr>
<td>pO₂</td>
<td>25-40</td>
<td>49-73</td>
<td>70-75</td>
</tr>
<tr>
<td>Sat%</td>
<td>&gt;50</td>
<td>&gt;80</td>
<td>&gt;90</td>
</tr>
<tr>
<td>HCO₃</td>
<td>&gt;15</td>
<td>16-19</td>
<td>20</td>
</tr>
</tbody>
</table>
Printout generated by blood gas machine
Table IV: Target blood gas values

<table>
<thead>
<tr>
<th></th>
<th>&lt;28 wks</th>
<th>28-40 wks</th>
<th>Term infant with PFC</th>
<th>Infant with BPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂</td>
<td>45-65</td>
<td>50-70</td>
<td>80-120</td>
<td>60-80</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>40-50</td>
<td>40-60</td>
<td>30-35</td>
<td>45-70</td>
</tr>
<tr>
<td>PH</td>
<td>&gt;7.25</td>
<td>&gt;7.25</td>
<td>7.50-7.60</td>
<td>7.35-7.45</td>
</tr>
</tbody>
</table>

Understanding the printout (Appendix – II).

BARO:

It denotes barometric pressure at site where machine is installed. It varies from place to place and it is determined by automated barometer in the machine. **Barometric pressure is required for calculation of alveolar oxygen pressure.**

THb A:

Haemoglobin (Hb) of patient. A few machines measure haemoglobin, others need this information to be fed. If no information is fed, machine may assume any Hb or it may be at mercy of technician. **Haemoglobin is required to calculate oxygen content (O₂ CT) of blood.**

Temp:

Patient temperature has to be fed into machine because the machine measures all values at 37°C. **Temperature affects pH, pCO₂ and pO₂. Hence, it is desirable to have values corrected for patient temperature.**

BE (ABE); BeEcf (SBE); BB

BE refers to actual base excess in variance from (above or below) total buffer base (BB). Normal BB is 48-49 mmol/L. If BB is 40, it means buffer base is reduced by nearly 8
mmol/L, or BE is –8 (also called base deficit). If BB is 60, it means buffer base is increased by nearly 12 mmol/L, or BE is +12.

BB is dependent on haemoglobin, as 25% of BB is constituted by haemoglobin buffer. Fifty percent of BB is contributed by bicarbonate and 25% by other buffers (proteins, phosphate, sulphate).

**HCO\(_3\) (ABC); st HCO\(_3\) (SBC); TCO\(_2\)**

TCO\(_2\) is sum of HCO\(_3\) and amount of CO\(_2\) dissolved in plasma. For each mm Hg pCO\(_2\), 0.03 ml CO\(_2\) is dissolved per 100 ml of plasma. As HCO\(_3\) values change with CO\(_2\) levels, st HCO\(_3\) is used to denote value of HCO\(_3\), independent of CO\(_2\) changes (i.e. at pCO\(_2\) of 40 and temperature of 37°C).

**St. pH:**

It is the pH adjusted for temperature of 37°C and pCO\(_2\) of 40 mm of Hg. This would represent pH value purely due to metabolic status.

**CH+:**

Concentration of hydrogen ion in nmol/L at 37°C and patients temperature.

**O\(_2\) CT:**

It is the sum of oxygen bound to haemoglobin + oxygen dissolved in plasma. For each gm saturated Hb, 1.34 ml O\(_2\) is bound to hemoglobin and for each mm Hg pO\(_2\) 0.003 ml oxygen is dissolved per 100 ml of plasma.

**O\(_2\) sat:**

Proportion/percentage of hemoglobin which is saturated with oxygen.

**Aa DO\(_2\):**

Alveolar to arterial oxygen gradient. Normal value is 5 to 15 mm Hg.
RQ:
Amount of CO$_2$ liberated per minute divided by amount of O$_2$ utilised per minute. Normal values are 200 ml/250 ml = 0.8.

FiO$_2$:
Inspired oxygen fraction concentration. This value has to be fed to machine, it is required for calculation of alveolar oxygen concentration.

DBE/dTHB:
It is called hemoglobin indicator. The normal value of this parameter is 0.32. If this value is more than 0.32 then it indicates, the hemoglobin of the patient should be measured accurately in order to calculate exact base excess.

Details about pH
pH = pK + log (HCO$_3^-$ / H$_2$CO$_3$)  \( \text{(Henderson-Hasselbach equation)} \)

pK = constant, it is the pH value at which H$_2$CO$_3$ is 50% dissociated i.e. concentration of HCO$_3^-$ and carbonic acid in body are equal.

PK = 6.1 for H$_2$CO$_3$.
Normal ratio HCO$_3^-$ / H$_2$CO$_3$ = 20/1 and hence
pH = 6.1 + log 20
= 6.1 + 1.3 = 7.4
pH Normal 7.35-7.45
Ideal 7.4 ± 2 S.D.
Alkalosis > 7.5
Acidosis < 7.3
Severe acidosis < 7.2
If pH is \(<7.25\) stimulation of respiratory centre occurs but if \(<7.0\) depression will occur.

**Relationship of pH and pCO\(_2\)**

pCO\(_2\) elevation of 10 mm Hg decreases pH by 0.08, while pCO\(_2\) decrease of 10 mmHg increase pH by 0.08.

**The effect of buffers on pH**

Buffers stabilize pH. Hemoglobin, bicarbonate and protein are the principal buffers of blood. Extravascular space does not have hemoglobin and hence the buffering capacity is less than that of blood. Because we have no measure of extra and intracellular buffering capacity, it is difficult to predict how much pH will change when the concentration of acid or CO\(_2\) changes. The equation \(\text{CO}_2 + \text{H}_2\text{O} = \text{H}_2\text{CO}_3 = \text{H}^+ + \text{HCO}_3^-\) shows that any addition or subtraction of H\(^+\) or of HCO\(_3^-\) ions causes a change in CO\(_2\) level. By changing ventilation, CO\(_2\) concentration can be altered. The Henderson Hasselbach equation can be used to calculate one variable only if the other two are known; for example, we can calculate \((\text{HCO}_3^-)\) if pH and \((\text{H}_2\text{CO}_3)\) are known. The equation cannot be used to predict what will happen if only one variable changes and if we know nothing about the other two. Although, we can estimate what might happen in response to an acid-load or ventilatory change, we cannot be accurate.

**Oxygenation**

Normal values of arterial oxygen tension in term neonates is 50-70 mm of Hg and in children 70-100 mm of Hg. Spurious hypoxia may be noted in situations with increased cells (polycythemia), delay in processing, venous blood or in a febrile patient. Although universally used, \(\text{paO}_2\) monitoring has recognised shortcoming. Validity of values are optimal when blood gas samples are obtained from indwelling catheters under quiet,
resting conditions. In a crying neonate due to pain of percutaneous puncture values obtained may not reflect steady state conditions. PaO$_2$ values vary considerably throughout the day in sick neonates. Intermittent sampling produces only a limited view of a single point in time. Transcutaneous (TcPO$_2$) monitors are useful for judging trends in oxygenation during management of acute lung disease. These monitors measure skin surface pO$_2$ (not paO$_2$), which under proper conditions is closely correlated with arterial pO$_2$. The TcPO$_2$ sensor combines a miniature blood gas electrode with a servo controlled probe. The sensor is applied to the skin in a way that excludes any effects of environmental air on values measured. The technique depends on heating the skin at the sensor site to 43.5°C to 44°C. This increases the tissues pO$_2$ as oxygen diffuses to the skin surface. With these operating temperatures and proper calibration, skin surface pO$_2$ at the electrode site correlates closely with central arterial pO$_2$.

In certain clinical circumstances, however, correlation is poor and TcPO$_2$ may underestimate paO$_2$. Such conditions include circulatory insufficiency, inadequate electrode temperature, improper calibration and lack of user expertise, patient age greater than 10 weeks (skin thickness factor), and use of vasodilator agents. Maturation and thickening of skin with increasing postnatal age limits the use of transcutaneous monitoring to neonates. All of these artifacts of measurement result in underestimation of arterial pO$_2$.

Under usual circumstances TcPO$_2$ should be in the 40 to 80 mm Hg range. There is a time lag between measured TcPO$_2$ and paO$_2$ values. As a result, oxygen concentration should not be continuously raised and lowered in attempts to “chase” fluctuating TcPO$_2$. 
values. The FiO₂ and management plan selected should be designed to minimize fluctuations of TcPO₂ values as much as possible without constant manipulations of FiO₂.

**Table VI: Causes of hypoxemia**

<table>
<thead>
<tr>
<th>Causes of Hypoxemia</th>
<th>Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoventilation</td>
<td>Loss of respiratory drive, Mechanical interference with lung inflation</td>
</tr>
<tr>
<td>Ventilation/perfusion mismatch</td>
<td>Parenchymal lung disease</td>
</tr>
<tr>
<td>Right-to-left shunt</td>
<td>Congential heart disease, Persistent pulmonary hypertension</td>
</tr>
<tr>
<td>Methemoglobinemia</td>
<td>Abnormal hemoglobin, Nitrate toxicity (following No)</td>
</tr>
</tbody>
</table>

**Oxygen saturation**

Amount of oxygen that is combined with hemoglobin divided by the amount of O₂ that can be combined with Hb i.e. % of saturation of Hb. Pulse oximetry is useful for monitoring trends in oxygenation. This technique is less complex and does not require calibration or the level of user sophistication that TcPO₂ monitors do. The technique measures peripheral hemoglobin O₂ saturation (SaO₂). Movement artifacts may at times, severely limit the applicability of this techniques. Artifacts of saturation measurement may also occur in the presence of high-intensity light, >50% fetal Hb, and some radiant warmers. Pulse oximetry does not measure the paO₂ and, thus is relatively insensitive in detecting hyperoxemia. This is particularly important in the small premature. In acute lung disease, the range of desirable hemoglobin saturation as measured by the pulse oximeter is 90 to 93%. Normal saturation being 95-98%.
Clinical cyanosis becomes evident if saturation is <75%. At paO₂ of 40 mm Hg 75% of hemoglobin A is saturated, while at paO₂ 27 mm Hg 50% of hemoglobin A is saturated (P₅₀) and at paO₂ of 60 mm Hg 90% of hemoglobin A is saturated.
Effects of Temperature and pH on Oxygen Dissociation Curve

Oxygen dissociation curve is sigmoid shaped which plateaus off at $pO_2 > 70$ mm Hg. Thus a patient with very high $pO_2$ may have a saturation 97-99% (Figure II). The position of the oxyhemoglobin dissociation curve is affected by changes in type of hemoglobin, pH, temperature, and concentrations of 2,3-diphosphoglycerate (2,3-DPG) (Table V). Fetal hemoglobin binds $O_2$ more avidly than does adult hemoglobin and tends to shift the curve to the left. As a result, the $paO_2$ at which hemoglobin is 50% saturated ($p_{50}$) is decreased. This shift benefits the fetus since it favours $O_2$ uptake at the low $O_2$ tensions in placenta. During the first months after birth, the oxyhemoglobin dissociation curve begins to shift to the right, and between 4 and 6 months of age it is similar to that of the adult.
Table V: Factors affecting the position of the oxyhemoglobin dissociation curve

<table>
<thead>
<tr>
<th>Shift Curve to the Left and ↓ P50</th>
<th>Shift Curve to the Right and ↑ P50</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Impair Oxygen Delivery)</td>
<td>(Improve Oxygen Delivery)</td>
</tr>
<tr>
<td>- Alkalosis</td>
<td>- Acidosis</td>
</tr>
<tr>
<td>- ↓ Temperature</td>
<td>- ↑ Temperature</td>
</tr>
<tr>
<td>- ↓ 2, 3 – DPG concentrations</td>
<td>- ↑ 2, 3 – DPG concentrations</td>
</tr>
<tr>
<td>- ↑ Fetal hemoglobin</td>
<td>- ↑ Adult hemoglobin</td>
</tr>
</tbody>
</table>

The curve may shift to right due to ↓ pH, ↑ pCO₂, ↑ temperature, ↑ 2-3 DPG and adult haemoglobin indicating less firm affinity of oxygen to haemoglobin while it may shift to left due to ↑ pH, ↓ pCO₂, ↓ temperature, ↓ 2-3 DPG and ↑ HbF thus indicating more firm binding of oxygen to haemoglobin thus resulting in tissue hypoxia (Figure-III).

**O₂ Content (O₂ CT and significance)**

It is concentration of total oxygen in the blood (expressed as vol. %).

1 gm of Hb combines with 1.34 ml of oxygen. Each 100 ml of blood has 0.003 ml of dissolved oxygen for each 1 mm Hg of O₂ tension.

O₂CT = O₂ in saturated Hb + Dissolved O₂ in plasma

Patient with anemia may have normal saturation because of cardiac compensation but decreased oxygen content as less hemoglobin is available for transporting oxygen.

**Oxygen delivery.** Oxygen delivery to the tissues is the product of arterial oxygen content and cardiac output, and it is directly affected by changes in paO₂. Hemoglobin concentration, and cardiac output. A decrease in any one of these components can be offset to some extent by increases in the others.

Oxygen consumption is equal to the cardiac output times the difference between arterial O₂ content and venous O₂ content. Oxygen consumption can be affected by both
O₂ delivery and by the ability of the tissues to extract O₂ from blood. The difference between arterial O₂ content and venous O₂ is a measure of effectiveness of O₂ delivery to tissues. An increase in the gradient between arterial and venous O₂ contents infers that delivery is inadequate and that the tissues are increasing extraction to maintain O₂ tension and increase extraction is limited, since a minimum gradient of oxygen tension must be maintained to facilitate diffusion of oxygen into cells and mitochondria. If mixed venous O₂ tension is too low, O₂ delivery to mitochondria will be compromised and O₂ consumption will decrease as the cell switches from aerobic to anaerobic metabolism. This inefficiency results in ATP depletion and ultimate cell death. In addition, anaerobic metabolism generates two molecules of lactic acid for every molecule of glucose metabolized, resulting in tissue lactic acidosis. Therefore, the appearance of significant amounts of lactic acid in arterial blood (> 3 umol/L) is indicative of inadequate O₂ delivery to the cellular mitochondria.

Calculation of oxygen content difference of arterial blood and venous blood at right atrial level tells how much oxygen is being utilised by tissue. Because of total shut down in cellular enzymatic function in septic shock, no oxygen utilisation occurs. Hence, the oxygen content of venous blood is same as that of arterial.

**Alveolar Gas Equation (AaDO₂)**

Alveolar oxygen can be calculated by following formula

\[
p_{AO₂} = P_{iO₂} - P_{ACO₂} \left( F_{iO₂} + \frac{(1-F_{iO₂})}{R} \right)
\]

Where \( P_{iO₂} \) is partial inspired oxygen pressure and equals \((PB-H₂O) (FiO₂)\). For most clinical purposes, \( R \) is assumed to be 0.8 and a modified equation given below is used
\[ p_{AO_2} = (PB - PH_2O) \times (FiO_2) - \frac{PaCO_2}{0.8} \]

or when FiO\(_2\) is greater than 0.6

\[ PAO_2 = (PB - PH_2O) \times (FiO_2) - PaCO_2 \]

(PB: Barometric pressure; PH\(_2O\): Water vapour pressure).

Alveolar oxygen partial pressure in an individual breathing room air (FiO\(_2\) 0.21) with arterial pCO\(_2\) of 40 mm of Hg is \((760-47) \times (0.21) - 40/0.8\)

\[ = 713 \times 0.21 - 40/0.8 \]

\[ = 149-50 = 99 \text{ mm Hg.} \]

In an infant who is breathing 50% oxygen, (FiO\(_2\) 0.5) and has an arterial pO\(_2\) of 150 and pCO\(_2\) of 36, the calculated Alveolar pO\(_2\) will be:

\[ 0.5 \times (760 \text{ mm Hg} - 47 \text{ mm Hg}) - \frac{36}{0.8} = 311 \text{ mm Hg} \]

AaDO\(_2\) will be 311 – 150 = 161 mm Hg while the arterial to alveolar pO\(_2\) ratio, will be 150/311 = 0.48.

In normal persons breathing room air, the AaDO\(_2\) is less than 10 mmHg. It is about 200 while breathing 100% oxygen. Large gradients (high AaDO\(_2\)) are noted in congenital cyanotic heart disease with shunts exceeding 50%, meconium aspiration syndrome and persistence of fetal circulation. AaDO\(_2\) > 620 for 12 hr on FiO\(_2\) 100% is an indication for ECMO in West, because risk of mortality is > 80%.

An advantage of using arterial pO\(_2\) to alveolar pO\(_2\) ratio (a/ApO\(_2\)) instead of AaDO\(_2\) is that the ratio does not change with varying inspired oxygen concentration. In healthy adult the ratio a/ApO\(_2\) is more than 0.8.
In infants with the severe RDS the a/ApO$_2$ ratio could fall to as low as 0.1 to 0.2. In addition, high arterial pCO$_2$ values indicate reduced ventilation. As baby recovers from RDS the a/ApO$_2$ improve gradually from low (0.1 to 0.3) to normal (0.7 to 0.9). A value of < 0.22 of arterial to alveolar oxygen ratio is indication for administering surfactant.

**Oxygen Index**

MAP x FiO$_2$ x 100

Postductal pO$_2$

40 – mortality risk > 80-90%

25-40 – Moratlity risk 50-60%

If oxygenation index >40, it is a indication for use of ECMo

**Carbon dioxide transport**

Carbon dioxide transport helps in excreting large amounts of CO$_2$ continuously from high body concentrations to low atmospheric concentrations. Carbon dioxide is carried in several forms: dissolved in plasma as bicarbonate in equilibrium with dissolved CO$_2$, is in the form of plasma or red cell bicarbonate. Carbon dioxide is 20 times more soluble in blood than oxygen and its dissociation curve is nearly linear over physiologic ranges. As a result, large amounts of CO$_2$ can be carried in blood and removed from the body with relatively small changes in partial pressure of carbon dioxide in blood.

Carbon dioxide and oxygen interact in the blood to enhance each other’s loading and unloading capabilities where concentration extremes exist. The bindings of CO$_2$ to hemoglobin in the tissues augments unloading of oxygen from capillary blood-**the Bohr effect**. On the other hand, the binding of oxygen to hemoglobin in the alveolar capillary bed augments CO$_2$ unloading from capillary blood into the alveolar **the Haldane effect**.
**PaCO₂**

Partial pressure of carbon dioxide in arterial blood. Normal value is 35-45 mm Hg (ideal 40 mm Hg).

Normal paCO₂ of venous blood = 45 mmHg

paCO₂ is indicative of alveolar ventilation.

If paCO₂ < 30 = Respiratory alkalosis

paCO₂ > 50 = Respiratory acidosis

High CO₂ is the most important respiratory centre stimulant. If paCO₂ > 65 the respiratory centre becomes insensitive to CO₂. In persistent pulmonary hypertension of newborn (PPHN) values of pCO₂ 40-60 mm of Hg and pO₂ 50-70 mm of Hg are acceptable but not ideal. In acute phase of PPHN once pulmonary artery hypertension has to be reduced by hyperventilation, pCO₂ should be lowered below 30 mm of Hg. In bronchopulmonary dysplasia a high pCO₂ (45-55 mm of Hg) may be acceptable as long as pH is >7.25 and oxygenation normal. In situation of pCO₂ rise, partial tube blockage, decreased minute ventilation or ventilation – perfusion mismatch should be thought.

**CO₂ content**

The partial pressure of CO₂ (the pCO₂) is measured in torr (mm Hg). Torr is then converted into millimoles of H₂CO₃ by assuming that for every torr of pCO₂ at 37°C, there is 0.03 mmol of H₂CO₃. The sum of bicarbonate and carbonic acid is the CO₂ content.

\[
\text{CO}_2 \text{ content} = \text{HCO}_3^- + \text{H}_2\text{CO}_3
\]

(at pCO₂ of 40 mmHg and HCO₃⁻ of 24 mmol/L)

= 24 + 40 X 0.03
= 24 + 1.2
= 25.2 mmol/L
Measuring the CO₂ content in blood

Majority (95%) of CO₂ is inside RBC in form of HCO₃⁻ and carbamino-compound, only 5% is dissolved in plasma.

In clinical practice, CO₂ content is assumed to be largely bicarbonate (which is usually true) and, therefore; it reflects a base excess (if CO₂ content is high) or deficit (if CO₂ content is low). Note that there is a problem with this way of interpretation. Assume one gets a report that the CO₂ content is 12 mEq/l. These 12 mEq/l are the sum of (HCO₃⁻) and (H₂CO₃).

Table VII: CO₂ content can mislead

<table>
<thead>
<tr>
<th></th>
<th>(HCO₃⁻)</th>
<th>pCO₂ x 0.03</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>10</td>
<td>2</td>
<td>6.79</td>
</tr>
<tr>
<td>B</td>
<td>11.8</td>
<td>0.2</td>
<td>7.87</td>
</tr>
</tbody>
</table>

A- Represents a mix respiratory metabolic acidosis

B- is a respiratory alkalosis insufficiently compensated

A CO₂ content, therefore, has to be interpreted cautiously. A simultaneous determination of pCO₂ or pH eliminates the uncertainty.

CO₂ content is a more complete measure of CO₂ present in the plasma in various forms, but it has no additional advantage over HCO₃⁻ and it follows the same changes of HCO₃⁻, which is more accurately measured in acid base disorders.

Actual bicarbonate (HCO₃⁻)

HCO₃⁻ in plasma

(n) 22-24 mEq/L

< 20 Acidosis

> 24 Alkalosis
**CO₂ up and down rule**

Partial pressure of carbon dioxide may change the levels of bicarbonate depending on degree and duration of CO₂ rise.

**Relationship between HCO₃⁻ & PaCO₂**

(1) For acute elevation in PaCO₂ over 40 mmHg, HCO₃⁻ increases by 1 mEq/L for each 10 mmHg paCO₂.

(2) For acute decrease in paCO₂ below 40 mmHg, HCO₃⁻ decreases by 2mEq/L for each 10 mm of Hg decrease in paCO₂.

(3) For chronic elevation in paCO₂ over 40 mm of Hg HCO₃⁻ increases 4 mEq/L for each 10 mm of Hg increase in paCO₂.

**Standard bicarbonate concentration (SBC)**

**(22-26) mEq/L**

It is the concentration of the HCO₃⁻ in the plasma from blood l which is equilibrated to bring the paCO₂ to 40 mm of Hg at 37°C i.e. it overcomes the changes in HCO₃⁻ due to respiratory causes and reflects a nonrespiratory acid-base change.

Under ideal condition SBC=HCO₃⁻ (n) variation = ± 2 mEq/L. If respiratory acidosis is present, HCO₃⁻ > SBC (because this blood will have a pCO₂ > 40 mm of Hg and therefore when equilibrated to 40 mmHg, some of the CO₂ will leave the blood. Hence SBC will be lowered).

If respiratory alkalosis is present HCO₃⁻ < SBC (because during equilibration to 40 mm some CO₂ gets absorbed and therefore SBC increases).

**Remember following**

(1) SBC Low – Metabolic acidosis
High – Metabolic alkalosis

(2) Difference between actual HCO$_3^-$ and SBC indicates

- respiratory acidosis if HCO$_3^-$ > SBC
- Respiratory alkalosis if HCO$_3^-$ < SBC

(3) When HCO$_3^-$ = SBC then respiratory balance is present

- When both are low but equal then compensated metabolic acidosis

(4) When SBC is ↑/↓ then HCO$_3^-$ must also ↑/↓

But ↑/N/↓ HCO$_3^-$ - may be associated with (n) SBC

**Actual base excess (ABE)**

Refers to actual base excess above or below total buffer base (BB). It is in-vitro expression which mainly reflects non respiratory portion of acid-base.

When CO$_2$ accumulates as a result of impaired respiration, the following reactions occur

\[
\text{CO}_2 + \text{H}_2\text{O} = \text{H}_2\text{CO}_3 = \text{HCO}_3^- + \text{H}^+
\]

Hb - + H$^+$ = HHb

The decrease in amount of Hb$^+$ buffer is equal to the amount of HCO$_3^-$ released in the reaction. Therefore, total amount of buffer anion content will not change. Therefore, changes in the paCO$_2$ will not change base excess.

Hence, ABE is a indicator of metabolic status. It attempts to quantify the patients total base excess or deficit. Expressed as mmol/L of base above or below the (n) buffer base range. The base excess allows an estimate how much base (if BE is negative) or acid (if BE is positive) is necessary to bring a liter of blood to pH 7.4.
**Standard base excess (SBE) buffer base (BB)**

SBE is same as ABE except that it is an invivo measurement which is dependent on the equilibration of the interstitial or ECF compartment of the body and not only the blood with CO₂.

Unlike the ABE – which is the BE in the whole blood in vitro, where buffering capacity is due to bicarbonate and hemoglobin, the in-vivo buffering capacity is less than in vitro because actually equilibration to the new level CO₂ takes place not only in the blood but also in the interstitial space. Since the extracellular volume of the body contains about three times more extravascular (free of hemoglobin buffer) than intravascular (rich in hemoglobin buffer) volume, some clinicians like to report the standard base excess, a base excess assuming hemoglobin of 5 g/dl. This represents the average buffering capacity of the total extracellular volume. Intracellular buffers play an important role after a disturbance has persisted for some time, particularly in severe acid-base derangements. In such situations, standard base excess does not provide a useful guide to therapy, one has to titrate until the desired result is achieved.

Microprocessors in modern automated analyzers use algorithms that automatically calculate variables such as bicarbonate and standard base excess by deriving the blood’s buffering capacity from known hemoglobin values. If an apparatus displaying BE is not available the Siggaard-Andersen Alignment Nomogram allows us to use either CO₂ content with pCO₂ or pH, or pH and pCO₂ to find bicarbonate. Additionally, it can be used to determine a base excess for well or poorly buffered systems. It is simple to use. Just draw a straight line through any two of the known variables (pH, pCO₂, CO₂ content) and read off not only the actual bicarbonate, but
also BE for any appropriate hemoglobin concentration. For standard BE, use 5g
hemoglobin/dl blood, unless the patient is severely anemic (Appendix-I)

**Significance of base excess**

Total buffer base (BB) in a neonate is 48-49 mmol/L. Half of this is due to HCO$_3^-$,
25% due to haemoglobin buffer and another 25% due to protein, sulfate, phosphate
buffers.

A value of BE of ± 3 is considered normal. Abnormal pH with BE> -5 (based
deficit <5) without any decompensation in a stable neonate does not need treatment.
Abnormal pH with BE< -5 (base deficit >5) with significant imbalance needs
treatment. Treatment of neonatal metabolic acidosis consists of general supportive
care and specific measures directed to treat underlying cause. Treatment of
hypothermia, hypovolemia, (anemia, hypoxia and electrolyte disturbances) will
usually correct metabolic acidosis secondary to asphyxia or poor tissue perfusion.
Antibiotics should be given if sepsis is suspected. Many infants require ventilatory
support. Bicarbonate is considered by some to be unnecessary and even harmful,
leading to changes in cerebral blood flow and paradoxically to increased
cerebrospinal fluid or intracellular acidosis.

**Simple disorder**

In simple acid base disorder pCO$_2$ and HCO$_3^-$ levels change in the same direction.
Simple disorder | pH | pCO₂ | HCO₃⁻
--- | --- | --- | ---
Metabolic acidosis | ↓ | ↓ | ↓
Metabolic alkalosis | ↑ | ↑ | ↑
Respiratory acidosis | ↓ | ↑ | ↑
Respiratory alkalosis | ↑ | ↓ | ↓

**The Mixed Disturbance**

If a patient with respiratory insufficiency develops metabolic acidosis, he loses his ability to compensate and a mixed respiratory-metabolic acidosis supervenes. Correspondingly, a mixed respiratory-metabolic alkalosis is also possible.

**Mixed Disturbances***

<table>
<thead>
<tr>
<th>pH</th>
<th>Bicarbonate</th>
<th>pCO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed Acidosis</td>
<td>↓↓</td>
<td>↓</td>
</tr>
<tr>
<td>Mixed Alkalosis</td>
<td>↑↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

* This table demonstrates that compensation cannot take place when respiratory and metabolic (renal) disturbance conspire. In mixed disturbances, both metabolic (bicarbonate) and respiratory (pCO₂) factors pull in the same direction and pH changes are exaggerated (double arrows).

**Compensation mechanism**

**Compensation for Acid –Base imbalances**

When disturbances in acid-base balance perisist, the body can call into play compensatory efforts through an organ not primarily affected; for example, pulmonary
disturbances resulting in respiratory acidosis or alkalosis will lead to compensation by the
kidney. Conversely, primary disturbances of renal function or metabolism with acid-base
imbalance lead to compensation by the lungs.

The body’s compensatory efforts are governed by complex intracellular and
extracellular stimuli and responses. Assume that a respiratory acidosis triggers a renal
compensatory effort. Compensation will return the abnormal pH toward normal. It does
not re-establish completely normal values or when complete compensation and correction
of a respiratory acidosis succeeds, the drive that sustains the compensatory effort would
cease.

**The direction of compensatory mechanism, bicarbonate, and PCO₂.**

<table>
<thead>
<tr>
<th></th>
<th>PH</th>
<th>Bicarbonate</th>
<th>PCO₂</th>
<th>Compensation*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acidosis</td>
<td>↓</td>
<td>↑↑</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Alkalosis</td>
<td>↑</td>
<td>↓↓</td>
<td>↓</td>
<td>Renal effect on bicarbonate</td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acidosis</td>
<td>↓</td>
<td>↓</td>
<td>↓↓</td>
<td></td>
</tr>
<tr>
<td>Alkalosis</td>
<td>↑</td>
<td>↑</td>
<td>↑↑</td>
<td>Respiratory effect on CO₂.</td>
</tr>
</tbody>
</table>

- Double arrows show direction of compensation. The pH change will be less
  pronounced in the presence of compensatory mechanism than in their absence.

**Anion Gap**

Measurement of anion gap gives a clue to the cause of metabolic acidosis. Anion gap
is the difference between the unmeasured anions and cations. This is calculated as
difference between measured cations and anions.
Serum (Na+ + K+) - (Cl⁻ + HCO₃⁻)

Normal values are 5 to 12 mmol/L. A normal anion gap acidosis suggests a HCO₃⁻ loss or rapid dilution of ECF. Chloride is proportionately increased in such conditions – GIT, Renal loss of HCO₃⁻. Increased anion gap suggest an addition of strong acid in the system as occurs in lactatemia, ketonemia, renal failure, excessive salt therapy (ringer lactate, acetate), ingestion of slicylates, methanol glycol. A decrease in serum K+, Ca++, Mg++ or falsely high Na+ or serum protein can also increase the anion gap. A decrease in anion gap does not help in diagnosis of acid base disorder. This may occur with low serum protein or increase plasma chloride due to bicarbonate loss by intestine or kidneys.

Remember 50% of sick patients with hyperlactatemia may present as no anion gap metabolic acidosis because of hyperchloremia and hypoalbuminemia.

**Approach to ABG and exercises**

**Approach to ABG (Back page)**

Interpretation of ABG should be systematic. Look at pH, pCO₂, HCO₃⁻ / BE and pO₂, (where lungs control CO₂/O₂ while kidneys HCO₃⁻). Try to answer following

1. Is acidosis or alkalosis present?
2. Is the imbalance respiratory (pCO₂) or metabolic (HCO₃⁻) in origin?
3. Is any compensation present?
4. What is paO₂?
5. Identify possible cause of the acid-base imbalance
6. What is the management for the imbalance?

Keep clinical condition, previous ABG and therapeutic interventions in mind while interpreting the ABG report. Compensation by kidneys is slow in neonate with which
while a sick neonate with respiratory disease has limitation for CO₂ excretion and kidneys may be ineffective for HCO₃⁻ conservation.

Let us look at arterial blood gas level for different simple disorders.

<table>
<thead>
<tr>
<th></th>
<th>PH</th>
<th>7.35</th>
<th>7.22</th>
<th>7.49</th>
<th>7.18</th>
<th>7.60</th>
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<tbody>
<tr>
<td>PCO₂</td>
<td>42</td>
<td>55</td>
<td>30</td>
<td>40</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>BE</td>
<td>-2</td>
<td>-4</td>
<td>0</td>
<td>-10</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>23</td>
<td>21</td>
<td>22</td>
<td>16</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>pO₂</td>
<td>60</td>
<td>58</td>
<td>65</td>
<td>55</td>
<td>70</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Interpretation</th>
<th>Metabolic Alkalosis</th>
<th>Normal</th>
<th>Respiratory Acidosis</th>
<th>Respiratory Alkalosis</th>
<th>Metabolic Acidosis</th>
</tr>
</thead>
</table>

32
**ABG exercises:**

For following ABG (A to F) confirm the values of TCO₂, O₂ content, AaDO₂. Are these correctly derived by machine?

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baro Pr.</td>
<td>747</td>
<td>737</td>
<td>747</td>
<td>730</td>
<td>747</td>
<td>747</td>
</tr>
<tr>
<td>Water vap. pr.</td>
<td>47</td>
<td>47</td>
<td>47</td>
<td>47</td>
<td>47</td>
<td>47</td>
</tr>
<tr>
<td>Hb</td>
<td>15</td>
<td>10</td>
<td>12</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>pH</td>
<td>7.418</td>
<td>6.881</td>
<td>7.322</td>
<td>7.516</td>
<td>7.516</td>
<td>7.531</td>
</tr>
<tr>
<td>pCO₂ mmHg</td>
<td>28.8</td>
<td>51.1</td>
<td>36.4</td>
<td>21.4</td>
<td>21.4</td>
<td>29.7</td>
</tr>
<tr>
<td>pO₂ mmHg</td>
<td>43.8</td>
<td>29.5</td>
<td>96.3</td>
<td>112.1</td>
<td>112.1</td>
<td>139.0</td>
</tr>
<tr>
<td>BE (ABE) mmol/L</td>
<td>-3.9</td>
<td>-22.2</td>
<td>-6.7</td>
<td>-2.7</td>
<td>-2.7</td>
<td>+3.5</td>
</tr>
<tr>
<td>Beecf (SBE) mmol/L</td>
<td>-5.4</td>
<td>-21.1</td>
<td>-6.8</td>
<td>-4.9</td>
<td>-4.9</td>
<td>+2.2</td>
</tr>
<tr>
<td>BB mmol/L</td>
<td>44.0</td>
<td>23.4</td>
<td>41.1</td>
<td>45.3</td>
<td>45.3</td>
<td>51.5</td>
</tr>
<tr>
<td>HCO₃ mmol/L</td>
<td>18.1</td>
<td>9.3</td>
<td>18.3</td>
<td>17.4</td>
<td>17.4</td>
<td>25.0</td>
</tr>
<tr>
<td>StHCO₃ mmol/L</td>
<td>20.3</td>
<td>8.2</td>
<td>18.8</td>
<td>21.1</td>
<td>21.1</td>
<td>27.5</td>
</tr>
<tr>
<td>TCO₂ mmol/L</td>
<td>19.0</td>
<td>10.8</td>
<td>19.4</td>
<td>18.0</td>
<td>18.0</td>
<td>25.9</td>
</tr>
<tr>
<td>O₂ ct vol %</td>
<td>15.1</td>
<td>6.8</td>
<td>16.1</td>
<td>13.7</td>
<td>13.6</td>
<td>13.8</td>
</tr>
<tr>
<td>O₂ sat %</td>
<td>81.0</td>
<td>50.0</td>
<td>96.6</td>
<td>98.6</td>
<td>98.6</td>
<td>99.2</td>
</tr>
<tr>
<td>FiO₂</td>
<td>0.50</td>
<td>1.0</td>
<td>0.30</td>
<td>0.30</td>
<td>0.30</td>
<td>0.30</td>
</tr>
<tr>
<td>Aa DO₂</td>
<td>270.6</td>
<td>619.5</td>
<td>70.0</td>
<td>72.1</td>
<td>72.1</td>
<td>35.3</td>
</tr>
</tbody>
</table>
What are possible for simple disorders in newborn?

**Metabolic acidosis**

(i) Underperfusion

(ii) Hypothermia

(iii) Anemia

(iv) Hypoxemia

(v) Sepsis

(vi) Increased protein load > 3g/kg/day while on parenteral nutrition.

(vii) Renal immaturity – loss of bicarbonate

(viii) Late metabolic acidosis – immaturity of kidney to handle high solute load especially sulphur containing aminoacids.

(ix) Metabolic disorder -IEM

(x) Decreased cardiac output

(xi) Acetazolamide (diamox) use

(xii) Use of excessive PEEP ,increase work of breathing

**Metabolic alkalosis**

(i) Iatrogenic – bicarbonate therapy

(ii) Use of diuretics

(iii) Following blood transfusion – citrate in blood gets converted to bicarbonate

(iv) Persistent vomiting – Congential adrenal hyperplasia

(v) Prolonged gastric aspiration

(vi) Urea cycle disorder
Respiratory Acidosis

Due to decreased minute ventilation

(i) Tube block
(ii) Tube dis-lodgement
(iii) Increased dead space – long endotracheal tube, adapters, and small bore tube
(iv) Opening of ductus (PDA)
(v) Pulmonary interstitial edema
(vi) Pulmonary air leak
(vii) Collapse, consolidation
Arterial blood gases decision tree

1. What is the pH? → ACIDOSIS
   < 7.35

2. What is $\text{HCO}_3^-$?
   → METABOLIC ACIDOSIS
   $\text{N or } \uparrow$
   - No help
   → RESPIRATORY ACIDOSIS AS WELL
   → pH ↓↓
   → RESPIRATORY COMPENSATION
   → pH towards normal

3. What is $\text{pCO}_2$?
   → ↑ RESPIRATORY ACIDOSIS
   $\text{N or } \downarrow$
   - No help
   → METABOLIC ACIDOSIS AS WELL
   → pH ↓↓
   → METABOLIC COMPENSATION
   → pH towards normal

4. What is $\text{HCO}_3^-$?
   → METABOLIC ALKALOSIS
   Base Excess
   $\text{N or } \downarrow$
   - No help
   → RESPIRATORY ALKALOSIS AS WELL
   → pH markedly ↑↑
   → RESPIRATORY COMPENSATION
   → pH towards N

5. What is $\text{pCO}_2$?
   → alkALOSIS
   $\uparrow$
   → RESPIRATORY ALKALOSIS
   $\text{N or } \downarrow$
   - No help
   → METABOLIC ALKALOSIS AS WELL
   → pH ↑
   → METABOLIC COMPENSATION

6. What is $\text{pO}_2$?
   → ↑ $\text{FiO}_2$, MAP
   $\downarrow$
   $\text{FEEP, PIP, TI}$

7. $\text{FiO}_2$, MAP
   → $\text{PEEP, PIP, TI}$
Practical tips for sampling for blood gas analysis

- Wait for a steady-state before sampling: Wait 30 minutes after any change in the ventilatory setting.
- The frequency in which the blood gases should be drawn is dependent upon the severity and the progress of the disease.
- The following are recommendations which may be useful.

  Arterial blood gases should be drawn:
  - Within 30 minutes of initiating mechanical ventilation or making a parameter change.
  - Every 2-4 hours during acute phase of illness.
  - Every 4-6 hours on stable infants requiring minimal ventilator manipulations.
  - Every 1-7 days in infants with chronic lung disease.
  - Whenever clinical condition indicates.

Use low strength heparin enough to obtain a final concentration in the sample between 50 and 100 IU/ml.

- Use minute volume of liquid heparin so as to avoid dilution of blood specimen.
- If Ca++ has to be determined on the same sample, use a special “buffered” heparin to avoid the chelation effect of standard heparin on calcium ions.
- Try to use dry heparin (powder or better crystallized or better still, lyophilized). The available commercial kits or sets use mostly crystallized form for capillaries and lyophilized for syringes.
Always carefully mix the blood after sampling by rotating the syringe between your hands and swirling it gently up and down, to assure a good mixing with heparin. Do it again.

Avoid accidental introduction of air bubbles into the syringe. Tighten it carefully immediately after sampling.

Use sampling equipment allowing spontaneous ascension of blood in it, without requirement of pulling on the plunger.

Eliminate immediately and carefully any air bubble present inside the syringe before sending it to the laboratory.

Use glass syringe, if the measurement cannot be done immediately.

Try to reduce delay to a minimum between sampling and measurement. Do not keep sample in ice if measurement can be done within 30 min. after sampling.

If the delay is, or is presumed, to be more than 30 min. , immerse sample in a slush of iced water and keep it so until measurement can be done. Maximum allowable delay in these conditions is two hours, and even then, pO$_2$ value is doubtful.

Be very careful when interpreting the blood gas values measured either between 30 minutes and 2 hours on non iced sample or measured after two hours on an iced sample.

In case of hyperleucocytosis, polycythemia, consider results (mostly pO$_2$) with a critical eye because these may result in spurious hypoxemia

Homogenize blood sample before introduction into the analyzer, particularly if hemoglobin/hematocrit have to be determined simultaneously with pH/pCO$_2$/pO$_2$. 
• Expel the first drops of blood from the syringe before introduction into the analyzer. Inject slowly and carefully when using such a procedure.

• Make a perfect local vasodilation and puncture the appropriate site for capillary sampling with adequate material. Do not try capillary sampling from an hypotensive neonate. Take great care of the risks of local contamination by ambient air. Do not forget to help heparin powder dissolution in the sample in an appropriate way. If a metal flea is required, do not forget to eliminate it before presenting the capillary to the analyser.

• Before sampling from a catheter, aspirate a blood volume four times dead space slowly. Take the sample in desired syringe. Re-infuse the blood withdrawn after the sampling.
Desired blood gas status and the possible change(s) in ventilator settings which will achieve it (using pressure-type ventilator)

<table>
<thead>
<tr>
<th>Desired status</th>
<th>Ventilator settings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rate</td>
</tr>
<tr>
<td>Increase PaCO₂</td>
<td>↓</td>
</tr>
<tr>
<td>Decrease PaCO₂</td>
<td>↑</td>
</tr>
<tr>
<td>Increase PaO₂</td>
<td>↑</td>
</tr>
<tr>
<td>Decrease PaO₂</td>
<td>↓</td>
</tr>
</tbody>
</table>

* Fine tuning, sparingly employed