Interpreting arterial blood gas analysis

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Introduction

Arterial blood gas (ABG) analysis and its interpretation is important for every specialist. Several comprehensive reviews on this topic have been published [1-3]. This presentation will not provide a detailed derivation of acid-base balance, it will, however, offer a few simple guidelines for the clinical interpretation of ABGs. ABG analysis requires consideration of several separate components:

- pH (acid-base balance)
- PaCO₂ (ventilation)
- PaO₂ (oxygenation)

pH interpretation

pH is defined as the negative logarithm of the hydrogen ion (H⁺) concentration, or more accurately, it is the negative logarithm of the molar concentration of dissolved hydrogen ions (moles of hydrogen ions per litre of solution). The plasma pH reflects acid-base status [4-5]. Maintenance of pH is as important as maintenance of the circulating blood volume. Many essential processes are pH dependent; for example, cell membrane function, metabolic processes, enzyme activity, hormonal effects, activities of mediators, drug effects, etc. Thus, changes in the pH produce powerful regulatory effects at the cellular, organ, and whole body levels. The normal physiological pH range is between 7.35 and 7.45. Within this range, normal bodily function is supported; outside these limits, a series of immediate and delayed compensatory mechanisms are triggered in response to the alteration of pH. These mechanisms act to return the pH to normal. A pH value of 7.40 is considered neutral. Therefore, an acid pH is < 7.40, and an alkaline pH is > 7.40.

The origin of hydrogen ion production needs to be understood in order to be able to interpret the meaning of a high pH (reduced H⁺) or low pH (excess H⁺).

Metabolic source: metabolic acid-base balance

Cellular metabolism generates non-volatile acids (lactic, acetoacetate, etc.) that are buffered by plasma bicarbonate; that is, the acids consume bicarbonate. Thus, metabolic acidosis (excess of non-volatile acids) is characterized by a reduction of plasma bicarbonate.

When hydrogen ions are excreted from the kidney, the corresponding anions are coupled with K⁺ ions, and bicarbonate is regenerated. Thus, the plasma concentration of non-volatile acids (H⁺) or, conversely, bicarbonate, reflects the balance between the production and renal elimination of non-volatile acids. Rather than use the normal value of bicarbonate (24 mmol/l), or the normal range (22-26 mmol/l), a more convenient notation assigns a value of 0 (corresponding to a bicarbonate of 24 mmol/l) to the normal base level. These levels vary between a base excess of 2 mmol/l (corresponding to a bicarbonate of 26 mmol/l) and a base deficit of –2 mmol/l (corresponding to a bicarbonate of 22 mmol/l).
Respiratory source: respiratory acid-base balance

Cellular respiration generates CO$_2$, which is either dissolved in the plasma (5%) or buffered by combining with the haemoglobin (Hb) inside red blood cells (95%). Carbon dioxide is eliminated through the lungs during ventilation. A very small amount of dissolved CO$_2$ (0.1%) combines with water within the plasma to generate carbonic acid, a source of H$^+$ ions. Therefore, CO$_2$ is considered a volatile acid, because it does not consume bicarbonate. Consequently, respiratory acidosis (an excess of volatile acids due to CO$_2$ retention) does not affect plasma bicarbonate.

The arterial plasma concentration of volatile acids (PaCO$_2$) reflects the balance between CO$_2$ production (VCO$_2$) and elimination by alveolar ventilation (VA); this relationship is provided by the equation:

$$\text{PaCO}_2 = k \cdot \frac{\text{VCO}_2}{\text{VA}}$$

The normal PaCO$_2$ value is 40 mmHg (5.3 kPa) (physiological range 35-45 mmHg; 4.7-6.0 kPa).

Thus, pH can be regarded as a measure of the balance between two sources, bicarbonate (which is the inverse of non-volatile acid production) and ventilation, which determines PaCO$_2$. The acid-base balance or status is expressed as:

$$\text{pH} = (f) \frac{\text{bicarbonate}}{\text{PaCO}_2}$$

(f: function)

Because bicarbonate is produced in the kidney, and PCO$_2$ is regulated in the lungs, the expression can be simplified to:

$$\text{pH} = (f) \frac{\text{kidney}}{\text{lung}}$$

Imbalance: mechanisms of compensation

Because maintenance of pH is so critical, it follows that metabolic disorders that alter pH almost always elicit respiratory compensation, and vice versa, thus maintaining pH within the physiological range. This compensation is derived from the previous function ($\text{pH} = (f) \frac{\text{bicarbonate}}{\text{PaCO}_2}$) by the Henderson-Hasselbalch equation, which measures the pH at equilibrium in an acid-base reaction:

$$\text{pH} = \text{pKa} + \log \left( \frac{\text{HCO}_3^-}{0.03 \times \text{PCO}_2} \right)$$

where pKa is the logarithmic measure of the acid dissociation constant (experimentally derived value of 6.1) and 0.03 is the coefficient of CO$_2$ solubility.

Thus, for example, when there is a reduction in pH, acidosis may occur due to bicarbonate reduction (increase of non-volatile acids; a metabolic acidosis), in which case pH compensation is by a reduction in PaCO$_2$ (increased CO$_2$ elimination by hyperventilation). Alternatively, acidosis may occur due to an increase in PaCO$_2$ (increase of volatile acids; a respiratory acidosis), in which case, the pH compensation occurs with an increase in bicarbonate (through the mechanism of reduced elimination by increased renal resorption).

Although this would appear to be a detailed interpretation of pH and its adjustment, it is not. A few basic premises are required to clarify current practices in pH interpretation.

Basic premises for interpreting pH

The pH defines the body acid-base status

Neutrality is defined as a pH of 7.40. Although the pH may be in the physiological range, it is important to recall that acidosis is defined as a pH lower than 7.40 (but not only below 7.35). Similarly, alkalosis is defined as a pH higher than 7.40 (but not only above 7.45). Some purists may insist on using the terms acidaemia and alkalaemia (but we will not get into semantics).
When correcting for acid-base imbalances the body never overcompensates

In the presence of acidosis (pH < 7.4), the compensatory component (respiratory or metabolic) is initiated before a serious reduction in pH occurs (< 7.35) to maintain the pH within the physiological range (7.35-7.39). In doing so, this mechanism will never cause the pH to rise to an alkaline value (pH > 7.4). Conversely, when alkalosis (pH > 7.4) occurs, compensatory mechanisms are initiated before the pH exceeds 7.45 and tend to maintain the pH between 7.45-7.41, but avoids a fall to an acid pH (< 7.4). This leads to the following premise:

The pH alone defines primary acid-base disorders

When the pH is < 7.40, the primary disorder is acidosis; when the pH is > 7.40, the primary disorder is alkalosis.

Acid-base imbalances are diagnosed by checking the values of PaCO₂, bicarbonate and base excess

For example, consider a case where the pH is 7.36 (within the physiological range, but acidic):

- a high PaCO₂ and positive base excess (BE) (bicarbonate > 26 mmol/l) indicates a respiratory acidosis
- a low PaCO₂ and negative BE (bicarbonate < 22 mmol/l) indicates a metabolic acidosis
- both cases are considered compensated, because the pH is in the physiological range.

Consider a case where the pH is 7.44 (within the physiological range, but alkaline):

- a high PaCO₂ and positive BE (bicarbonate > 26 mmol/l) indicates a metabolic alkalosis
- a low PaCO₂ and negative BE (bicarbonate < 22 mmol/l) indicates a respiratory alkalosis

Again, both cases are compensated, because the pH is in the physiological range.

Consider a case where the pH is 7.31:

- a high PaCO₂ and negative BE (bicarbonate < 24 mmol/l) indicates mixed acidosis

Consider a case where the pH is 7.49

- a low PaCO₂ and positive BE (bicarbonate > 26 mmol/l) indicates mixed alkalosis

Refining the interpretation of acid-base balance

Although it is usually not necessary to apply this analysis in the clinical situation, it can sometimes be useful to calculate the expected pH value based on PaCO₂ and bicarbonate parameters. The pH value will give an indication of the primary disorder, but where both components (respiratory and metabolic) are altered, it is important to establish the magnitude of each to determine which has the predominant effect. Although this may not be done routinely at the bedside (because it is important to treat the primary disorder) it is useful to know how to calculate these values for times when a detailed interpretation of pH is desirable [6-8].

Calculation of pH variation based on changes in PaCO₂ (expected respiratory pH)

The following table provides a simple rule of thumb to give an immediate approximation of the pH for a given PaCO₂ which is easy to memorise. The formula used in this table states that, for every 20 mmHg (2.7 kPa) above the normal PaCO₂ value of 40 mmHg, the pH is reduced by 0.1 and, for every 10 mmHg (1.3 kPa) below the normal, PaCO₂ value of 40mmHg, the pH is increased by 0.1.

<table>
<thead>
<tr>
<th>PaCO₂ (mmHg)</th>
<th>80</th>
<th>70</th>
<th>60</th>
<th>50</th>
<th>40</th>
<th>30</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.20</td>
<td>7.25</td>
<td>7.30</td>
<td>7.35</td>
<td>7.40</td>
<td>7.50</td>
<td>7.60</td>
</tr>
</tbody>
</table>
The exact calculation of this value can be obtained with the following formulae:

for a PaCO₂ > 40 mmHg:

\[
\text{pH variation} = \frac{(\text{measured PaCO}_2 \text{ (mmHg)} - 40)/2}{100}
\]

and for a PaCO₂ < 40 mmHg:

\[
\text{pH variation} = \frac{(40 - \text{measured PaCO}_2 \text{ (mmHg)})}{100}
\]

From the variation in pH, we can calculate the expected pH, as follows:

\[
\text{expected pH} = \text{normal pH} - \text{variation of the calculated pH}
\]

**Example 1**

PaCO₂ = 70 mmHg (patient on mechanical ventilation, maybe non-invasive ventilation)

When this PaCO₂ is observed at the bedside, minute ventilation must be increased promptly. Nevertheless, because the PaCO₂ is abnormal, it is useful to calculate the pH based on this PaCO₂, and then, compare it with the actual pH. This would give the following interpretation:

\[
\text{change of pH} = \frac{(70-40)/2}{100} = \frac{30}{2}/100 = 0.15
\]

\[
\text{expected pH} = 7.40 - 0.15 = 7.25
\]

Hence, if the actual pH in this example is > 7.25, this would reveal that metabolic compensation is occurring. Alternatively if the actual pH in this example is < 7.25, the interpretation would be that it is due to a superimposed metabolic acidosis. In either case, the initial corrective treatment would be to increase the minute ventilation; thus, the first management response was correct.

**Example 2**

PaCO₂ = 20 mmHg (with the patient being mechanically ventilated)

Out of professional courtesy, we will not ask who set the ventilator for this patient! This is clearly a case of hyperventilation:

\[
\text{change of pH} = 40-20 = 20/100 = 0.2
\]

\[
\text{expected pH} = 7.40 + 0.2 = 7.60
\]

Hence, if the actual pH in this example is < 7.60, the interpretation would be that there is an element of metabolic compensation (since the effect of the PCO₂ alone would establish a pH of 7.60); if the pH was > 7.60, this infers that it is due to a superimposed metabolic alkalosis.

**Diagnoses based on calculations of base excess or deficit**

When there is a difference between the actual pH and the calculated pH (based on the PaCO₂ value), the variation in pH is due to metabolic causes. In the ABG interpretation, this is expressed as a base excess (BE) or base deficit (BD). This calculation is similar in principle to the previous one. The rule of thumb is that the BE is equal to two-thirds of the difference between the actual pH and the expected (calculated) pH, multiplied by 100. If the answer is negative, this indicates that the metabolic component is acidic (in other words, the BE is a actually a base deficit). A similar calculation is performed by a blood gas analyser.

**Example 1**

PaCO₂ = 76 mmHg, pH expected = 7.22, pH measured = 7.04

\[
(7.04-7.22) \times 100 = -18 \times 2/3 = -12 \text{ (BE = -12 mmol/l)}
\]
Diagnosis: this is a combined respiratory acidosis ($\text{PaCO}_2 = 76$) and metabolic acidosis ($\text{BE} = -12$)

Example 2

$\text{PaCO}_2 = 18 \text{ mmHg}$, pH expected = 7.62, pH measured = 7.47

\[(7.47-7.62) \times 100 = -15 \times 2/3 = -10 \text{ (BE} = -10 \text{ mmol/l)}\]

Diagnosis: this is a respiratory alkalosis with a compensatory metabolic acidosis

Mechanisms derived by deductive reasoning

The two previous discussion points can be amalgamated into a simpler formula. This calculates the $\text{PaCO}_2$ that would result from a 'purely' metabolic acidosis.

\[\text{PaCO}_2 = (40 + \text{BE}) \pm 5.\]

Example 1

$\text{PaCO}_2 = 39 \text{ mmHg}$, pH = 7.31, BE = –8

99 % of people would say that this was metabolic acidosis and be done with it. But attention should be directed to determine if the $\text{PaCO}_2$ says anything? It is always useful to understand whether the $\text{PaCO}_2$ has influenced the actual pH. The formula mentioned above will indicate whether $\text{PaCO}_2$ contributes to the acidosis:

\[\text{PaCO}_2 = (40 + \text{BE}) \pm 5 = (40 - 8) \pm 5 = 32 \pm 5 = 27-37 \text{ mmHg}\]

Because the actual $\text{PaCO}_2$ (39 mmHg) is higher than this range, there is a small element of respiratory acidosis because the actual $\text{PaCO}_2$ is above the value expected.

Another example

$\text{PaCO}_2 = 26 \text{ mmol Hg}$, pH = 7.35, BE = –12.6 mmol/l

Subsequent calculation reveals:

\[\text{PaCO}_2 = (40-12.6) \pm 5 = 27.4 \pm 5 = 22.4 – 32.4 \text{ mmHg}\]

This indicates a pure metabolic acidosis, which can be deduced because the $\text{PaCO}_2$ value is within the expected range. Furthermore, the acidosis is fully compensated.

These calculations are also valid for cases of alkalosis.

Example 2

$\text{PaCO}_2 = 57 \text{ mmHg}$, pH = 7.41, BE = +6.3 mmol/l

At first glance, this appears to be compensated respiratory acidosis; however, after performing the calculation, it is can be determined that this is actually a compensated metabolic alkalosis. Subsequent calculation reveals:

\[\text{PaCO}_2 = (40 + 6.3) \pm 5 = 46.3 \pm 5 = 41.3 – 51.3 \text{ mmHg}\]

The expected $\text{PaCO}_2$ is lower than the actual $\text{PaCO}_2$ (57 mmHg); therefore, there is a mild respiratory acidosis.

This reasoning is easily incorporated into daily practice, because the calculation is so very simple, that it can be performed without a calculator.
Compensatory mechanisms

Metabolic alkalosis can be compensated by CO₂ retention (respiratory acidosis), within certain limitations. A compensatory elevated PaCO₂ will not typically rise above 60 mmHg, because higher levels of CO₂ have deleterious physiological effects. Therefore, in a case of metabolic alkalosis, with a measured PaCO₂ > 60 mmHg is encountered, a superimposed respiratory acidosis should be considered.

Hidden ions

The sum of all blood anions (Cl⁻, HCO₃⁻, etc) is equal to 23 mEq/l. They will only be partially balanced by cations (Na⁺, K⁺, etc) that add up to a total of 11 mEq/l. Despite this imbalance in measurable ions, a neutral pH is maintained. Therefore, there must be a quantity of unmeasured ions that equals 12 mEq/L [9, 10]. The magnitude of this unmeasured quantity is referred to as an anion gap. Hence:

\[
\text{anion gap (mEq/L)} = \text{anions (mEq/L)} - \text{cations (mEq/L)} = 23 - 11 = 12 \text{ mEq/L}
\]

By definition, only Na⁺, K⁺, Cl⁻ and HCO₃⁻ are used when calculating the anion gap.

Hence, we get:

\[
\text{anion gap (mEq/L)} = (\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-) \quad \text{(all ions are measured in mEq/L).}
\]

Blood gas analysers generally calculate the anion gap directly, so it is not necessary to calculate this value. An excess of hidden anions (an anion gap > 12) normally indicates excess lactate or ketoacids (as in diabetic ketoacidosis or alcohol abuse); a deficit of hidden anions (an anion gap of < 12) commonly indicates a low concentration of plasma proteins (as in hypoalbuminaemia). The anion gap value is used to explain the buffering capacity of blood when the BE is not the value expected; it facilitates interpretation of the data when lactate, glucose, and albumin are not measured. Scholars of metabolic acid-base disorders must study this area in detail, but this is not necessarily a requirement for the clinician.

Frequent clinical causes

It is essential to interpret blood gases to establish the cause of the pH alteration in order to initiate the correct remedial treatment (for example, in metabolic acidosis, bicarbonate should not always be given). In the following examples, we discuss the most frequent clinical causes of pH disorders [11-13].

Alterations in pH due to primary alterations in PaCO₂ (respiratory causes)

An alteration in PaCO₂ implies an alteration in the factors that affect the balance between CO₂ production (VCO₂) and elimination (VA), according to the familiar alveolar gas equation:

\[
\text{PaCO}_2 \text{ (mmHg)} = k \times \left( \frac{\text{VCO}_2}{\text{VA}} \right).
\]

Being expressed VCO₂ in ml/min and VA in L/min, k value is of 0.863.

Common clinical causes of respiratory acidosis

Respiratory acidosis implies that VA is insufficient to eliminate all the CO₂ that is produced in the body. This can be due to three conditions:

- Alveolar hypoventilation, which can manifest in two forms: hypoventilation with a reduction in the minute ventilatory volume (causes: drugs that depress the respiratory drive, residual effects of muscle relaxants, neurological or neuromuscular illnesses and injuries); and, hypoventilation with a normal or increased minute ventilatory volume (causes: increased dead space due to hypotension, pulmonary hypoperfusion, or pulmonary embolism)
- Overproduction of CO₂ (causes: hyperthermia, use of parenteral or enteral nutrition with high carbohydrate content)
- Both the above: hypoventilation plus overproduction of CO₂
These causes are not exclusive and can occur simultaneously.

**Common clinical causes of respiratory alkalosis**

Respiratory alkalosis, unlike acidosis, implies that the VA exceeds the production of CO$_2$. This can be due to three conditions:

- Alveolar hyperventilation (causes: ventilator settings with a high minute volume, in spontaneous breathing: pain, effect of drugs (theophylline, doxapram), or some traumatic brain injuries). Note: tachypnoea does not always imply hyperventilation: when dead space is increased, there can be hyper, normo, or hypoventilation
  - Reduced CO$_2$ production (cause: hypothermia)
  - Both of the above

**Alterations in pH due to alterations in metabolism (metabolic cause)**

An alteration in pH due to metabolic causes implies an imbalance between the production and renal elimination of non-volatile acids.

**Common clinical causes of metabolic acidosis**

Metabolic acidosis occurs when the existing bicarbonate in the blood is insufficient to buffer the production of non-volatile acids. This can be due to three conditions:

- Elevated H$^+$ production, which can manifest in three forms: lactic acidosis (cause: secondary to circulatory failure; however, up to 20% of this type of patients present with lactic acidaemia (lactate > 2 mmol/l) without alterations in pH or bicarbonate, due to an associated hypochloraemic alkalosis [14]); ketoacidosis (cause: hyperglycaemia secondary to diabetes, surgical stress, SIRS etc., or hyperkalae-mia); or administration of acid solutions (cause: administration of saline solution (0.9 % NaCl) and fluid overload during resuscitation in cardiovascular shock. In this case, an hyperchloraemic acidosis will be the result [15, 16]
  - Reduced plasma HCO$_3^-$ (causes: limited production of bicarbonate in the kidney (renal failure of variable magnitudes) [17, 18]; increased intestinal loss of bicarbonate due to diarrhoea, fistulæ, etc)
  - Any combination of the above.

  The treatment of metabolic acidosis must always be based on the aetiology. Currently, the use of sodium bicarbonate is not recommended for correcting pH. Boyd et al, in a recent review, stated that there is no evidence at present that justifies the use of bicarbonate in lactic acidosis [19].

**Common clinical causes of metabolic alkalosis**

Metabolic alkalosis occurs when the existing bicarbonate in the blood exceeds the buffering capacity required for non-volatile acids. It can be due to three conditions:

- Reduced acids, or a loss of H$^+$; this can manifest in two forms: hypokalaemia: the kidney tries to recover K$^+$ by exchanging it with H$^+$, and thus, H$^+$ is eliminated (causes: furosemide abuse, digestive loss (diarrhoea, etc)); hypochloraemia (cause: high digestive losses (vomiting, gastric aspiration, fistulæ, etc)). Here, an association between metabolic alkalosis and hypokalaemia may develop creating a vicious circle whereby hypokalaemia induces H$^+$ elimination from the kidney, which generates an alkalosis; this alkalosis induces recovery of H$^+$ by the kidney in exchange for K$^+$ which is lost and generates hypokalaemia. Clinically this can be corrected by administration of potassium chloride, which corrects both possible causes of metabolic alkalosis
  - Elevated bicarbonate, HCO$_3^-$ (causes: excess bicarbonate infusion (iatrogenic), hypernatraemia - when the kidney eliminates Na$^+$, it recovers bicarbonate)
  - Both the above. The use of diuretics causes metabolic alkalosis by both mechanisms mentioned above; it increases the loss of potassium (hypokalaemia) and water (which increases plasma Na$^+$).
Other diagnostic parameters to determine the cause of pH changes

To diagnose and treat acid-base disorders confirmed by blood gas analysis, we require additional parameters that will guide us in selecting the most appropriate treatment. For example, the diagnosis of metabolic acidosis ideally requires serum lactate and glucose values. For lactic acidosis, we must evaluate the haemodynamic status of the patient and determine the haemoglobin concentration to determine oxygen transport and delivery. The diagnosis of metabolic alkalosis requires potassium, chlorine, and sodium estimations. These values are often available routinely supplied by blood gas analysers. Furthermore, alterations of potassium and bicarbonate homeostasis must be reviewed in the context of creatinine analysis to reflect renal function; this will give an indication of the capacity of the kidney to compensate for acid-base disturbances.

Interpretation of PaCO₂

PaCO₂ values are related to gas exchange, alveolar ventilation, and dead space. Space constraints do not allow a full discussion; however, PaCO₂ interpretation needs to be made with reference to the patient’s minute volume ventilation.

Interpretation of PaO₂

PaO₂ is influenced by physiological factors associated with ventilation – perfusion mismatches in the lung. PaO₂ evaluation needs to be made with reference to the FiO₂. A blood gas analyser usually provides the PaO₂/FiO₂ ratio, and other indexes of oxygenation that are less commonly used such as the PaO₂/PAO₂ (the gradient of arterial and alveolar O₂), the P50 etc. However, PaO₂ is the most important value for interpreting changes in pH, because it determines oxygen transportation to the tissues. Thus, a low PaO₂ is the most common cause of metabolic-lactic acidosis.

Other factors that determine oxygen delivery to the tissues are shown in the equation:

\[
\text{Delivery of } O_2 = \text{cardiac output } \times \text{arterial } O_2 \text{ content, where arterial } O_2 \text{ content } = (\text{Hb g/dL } \times 1.39 \times \%\text{HbSat}) (\text{PaO}_2 \text{ mmHg } \times 0.0031)
\]

Often a blood gas analyser will calculate the arterial oxygen content directly from the other measured values. Once the oxygen content is known, manipulation of the patient’s cardiac output will enable optimal oxygen delivery to the tissues and improve lactic acidosis states.

Key learning points

- Plasma pH reflects acid-base status. Acid-base imbalances of metabolic origin are compensated by opposite changes in ventilation and vice versa, but interestingly, no overcompensation occurs. This way pH always defines the primary acid-base disorder. When the pH is < 7.40, the primary disorder is acidosis; when the pH is > 7.40, the primary disorder is alkalosis.
- In order to interpret compensatory mechanisms a simple formula for calculating expected PaCO₂ from the Base Excess can be applied. When the actual PaCO₂ differs from the expected value, an associated respiratory acidosis or alkalosis is present.
- The most common cause of a respiratory acidosis/alkalosis in patients on mechanical ventilation is associated with the ventilator settings (low VT or high minute volume). Hyperthermia and hypothermia are also very frequent causes.
- Metabolic acidosis has three main clinical features: lactic acidosis (caused by low oxygen delivery), ketoacidosis (hyperglycaemia) and renal failure (no bicarbonate production). A common cause of alkalosis is due to hypokalaemia secondary to furosemide administration.
- PaCO₂ values are related to alveolar ventilation, hence, its interpretation needs to be made with reference to the patient’s minute volume ventilation.
- PaO₂ is influenced by pathophysiological factors associated with ventilation – perfusion mismatches in the lung. PaO₂ evaluation needs to be made with reference to the FiO₂.
References