A simplified approach to the interpretation of arterial blood gas analysis

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The fundamental function of the respiratory system is to provide the correct homeostasis of arterial lung gases, O_2, CO_2, and pH. Arterial blood gas analysis (ABG) represents the "ultimate" test to be used in clinical practice in the evaluation of severity and causes of: 1) lung gas exchange abnormalities; and 2) acid–base (A–B) disturbances. ABG is one of the most useful diagnostic tests, not only in the critical care setting, but also in general clinical practice because of the high incidence of comorbidities (*e.g.* diabetes, heart failure, renal failure) associated with respiratory diseases, particularly in the elderly population.

The aim of this review is to provide the reader with the basic knowledge for ABG interpretation. A brief history of ABG will be presented; then a simple "two-step" approach to the interpretation of the three fundamental variables (*i.e.* arterial oxygen tension (Pa,O_2), arterial carbon dioxide tension (Pa,CO_2) and pH) measured using modern equipment will be discussed. Particular attention will be paid to: 1) the integrated reading of Pa,O_2 and Pa,CO_2 in the evaluation of lung exchange abnormalities; 2) the integrated reading of Pa,CO_2 and pH in the evaluation of A–B disturbances; and 3) the approach to be used in the diagnosis of simple and mixed A–B disorders. Finally, some ABG examples will be presented as teaching cases.

History of ABG

The measurement of ABG has developed during the past century and has had a tremendous impact on clinical practice. In the first years of the 20th century, there was no agreement about the interpretation of the main function of the lung, *i.e.* blood oxygenation. C. Bohr and J.S. Haldane supported the theory that the lung was able to secrete oxygen [1, 2]. The strongest opponent of this theory was one of Bohr's students, A. Krogh, who demonstrated lower Pa,O_2 than alveolar oxygen tension (PA,O_2) values in several animal experiments. A revolution in the understanding of pulmonary gas exchange took place around 1950, sparked by two groups of researchers studying the link between lung ventilation and perfusion. W. Fenn, H. Rahn and A. Otis (the Rochester group) studied the effects of hyperventilation, oxygen breathing and hypoxaemia on alveolar gas composition and developed the well known "O_2–CO_2 diagram" [3]. R. Riley and co-workers concentrated their work on the blood side of the blood–gas barrier, taking into account the O_2 and CO_2 dissociation curves and developing the "four quadrant diagram" [4, 5]. These approaches remained the gold standard in the evaluation of gas-exchange abnormalities for many years until the introduction of the multiple inert gas elimination technique, which permitted a more accurate analysis of ventilation/perfusion (V'/Q') abnormalities [6].

History of ABG equipment

Until the 1950s, the van Slyke technique was used for the measurement of blood O_2 and CO_2 content. Blood gases were extracted with a vacuum and subsequently measured by a manometric method [7]. Although this technique had been available for several years, its large-scale use in clinical practice first occurred during the polio epidemic in Copenhagen, Denmark, in the early 1950s. Physicians had limited experience in interpreting blood gas data and during the first weeks of the epidemic the high CO_2 content observed in patients with

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respiratory muscle paralysis was misinterpreted as metabolic alkalosis. B. Ibsen was the first to propose that the high values observed were caused by to CO_2 retention and, in the following days, P. Astrup, using an electrode to measure blood pH, computed the Pa,CO_2 by the use of the Henderson–Hasselbalch equation and confirmed Ibsen's hypothesis. On the basis of the linear relationship between pH and the logarithm of carbon dioxide tension (PCO_2), Astrup introduced a simpler method of calculating blood PCO_2 by measuring "*in vitro*" changes in pH following blood exposure to different PCO_2 [8]. This method gave life to concepts such as standard bicarbonate (*i.e.* bicarbonate concentration at a normal PCO_2) and base excess (BE), developed in late 1950s by Siggaard-Andersen and co-workers [9, 10], which soon became the favoured method for A–B status interpretation (the Copenhagen School). In the late 1960s the introduction of a small platinum electrode for oxygen tension (PO_2) measurement and of a pH-sensitive glass electrode (surrounded by bicarbonate (HCO_3^-) solution and a thin Teflon membrane) for CO_2 measurement predated the introduction of the modern three-electrode system blood gas analyser that is used nowadays [11–13]. Modern equipment measures three fundamental variables – Pa,O_2, Pa,CO_2 and pH. All other parameters, such as plasma HCO_3^-, are computed by software using standard formulae. In this review, we suggest that the three "measured" variables should be used predominantly when interpreting ABG data.

History of A–B disturbance interpretation

The history of A–B abnormality interpretation has been signed by several debates that until now have not been completely resolved. Different theories for the interpretation of A–B disturbances have been generated over the years. Since the 1930s, physicians have known that changes in Pa,CO_2 were associated not only with chemical changes in pH but also with more complex chemical and physiological adaptations. In the late 1950s, after the polio epidemic, Siggaard-Andersen and co-workers [9, 10], from Copenhagen, tried to find a method that permitted a more precise evaluation of A–B alterations. They first performed *in vitro* studies that examined CO_2 titration in human blood; thereafter, they developed the van Slyke equation that permitted the calculation of BE defined as the "amount of strong acid (mmol per L) that must be added to the blood sample to return the sample to pH 7.40

after equilibration while maintaining the partial pressure of carbon dioxide at 40 Torr [5.33 kPa]". Schwartz and Relman [14], from Boston, MA, USA, soon criticised this approach and argued against its applicability "*in vivo*". They studied the effect on pH values of acute and chronic exposure to hypercapnia in dogs and humans [15]. The diagnosis of acute and chronic respiratory acidosis in humans is based on the original data obtained by Schwartz (figure 1). The disagreement between the Copenhagen and the Boston schools, also known as the "Great Transatlantic Acid–Base Debate", led the Danes to formulate variants of the van Slyke equation, corrected for haemoglobin concentration (standard BE (SBE)) and for the concentration of other weak acids such as albumin and phosphate (corrected SBE (cSBE)). The Americans, meanwhile, proposed the six "rules of thumb" based on the expected change of HCO_3^- concentration ($[HCO_3^-]$) for a given change in Pa,CO_2 for respiratory disorders and the opposite for non-respiratory disorders (later on called "metabolic") [16]. In 1983 Stewart [17] proposed a new theory for the understanding and interpretation of A–B disturbances. In the traditional approach, the causes of metabolic disorders were attributed to changes in serum $[HCO_3^-]$; Stewart considered HCO_3^- a "dependent" variable; according to his theory, there are three groups of independent variables in the human plasma that may affect A–B status: CO_2, strong ions (*e.g.* Na^+, Cl^-) and weak acids (*e.g.* albumin, phosphate). An increase or reduction in one or more of these variables can cause A–B abnormalities. A five-equation method, which per-

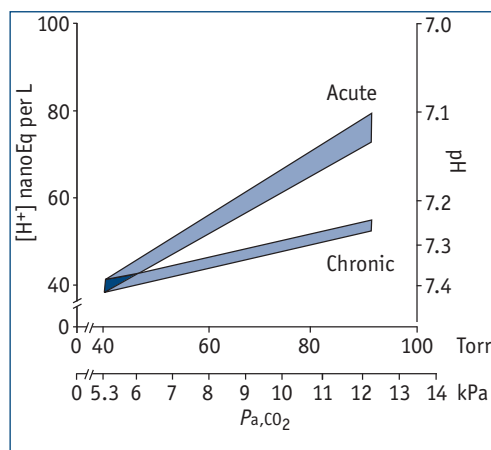

Figure 1

Pa,CO_2–pH relationship in acute and chronic respiratory acidosis. The slopes of the lines indicate the expected change in pH for a given change in Pa,CO_2. Notably, pH does not come back to normal value in the chronic condition.

mits the clinical application of Stewart's theory, was subsequently developed by J. Figge and V. Fencl [18, 19].

An integrated "two-step" approach to ABG interpretation

In this section, a simple and practical two-step approach for ABG interpretation in the clinical setting is proposed (figure 2). The first step aims at the evaluation of lung gas-exchange status while the second step aims at the evaluation of A–B status.

Step 1: Evaluation of lung gas exchange

The causes and the severity of lung gas-exchange abnormalities are evaluated by using an integrated reading of P_{a,O_2} and P_{a,CO_2} values. This integrated view could be obtained by computing the alveolar–arterial P_{O_2} gradient (P_{A-a,O_2}); a simpler alternative is to sum the P_{a,O_2} and P_{a,CO_2} values. As shown in table 1, for an accurate evaluation of the causes of arterial hypoxaemia, P_{A-a,O_2} should be computed according to the following formula:

$$P_{A-a,O_2} = [(P_B - P_{H_2O}) \times F_{I,O_2} - P_{a,CO_2}/R] - P_{a,O_2}$$

where P_B is the barometric pressure (~760 Torr at sea level), P_{H_2O} is the partial pressure of water vapour in the airways (47 Torr at 37°C), F_{I,O_2} is the inspired oxygen fraction and R is the respiratory quotient (the ratio between CO_2 output and O_2 uptake: ~0.90 at rest).

At sea-level, the normal expected P_{A-a,O_2} value is ~15 Torr (~2 kPa) in young subjects or ~20 Torr (~2.7 kPa) in older subjects; higher values reveal the presence of an abnormality in pulmonary gas exchange. To further understand the cause of arterial hypoxaemia (table 1), the effect of supplemental oxygen breathing on P_{A-a,O_2} should be examined. While V'/Q' defects are usually corrected by oxygen breathing, diffusion defects and shunts are only partially corrected or poorly corrected, respectively.

A simple, less accurate alternative to the computation of the P_{A-a,O_2} is to use the rule of "130". If a subject, at sea-level, is breathing room air ($F_{I,O_2}=0.21$) the sum of $P_{a,O_2}+P_{a,CO_2}$ should be ~130 Torr (17.5 kPa). The following examples illustrate how to interpret the $P_{a,O_2}+P_{a,CO_2}$ sum. A patient with $P_{a,O_2}=70$ Torr
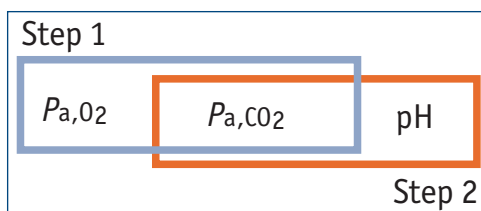

Step 1

P_{a,O_2} P_{a,CO_2} pH

Step 2

Figure 2
A "two-step" approach to ABG interpretation.

and $P_{a,CO_2}=60$ Torr is hypoventilating a "normal" lung ($P_{a,O_2}+P_{a,CO_2}=130$ Torr). A patient with respiratory failure and hypocapnia ($P_{a,O_2}=50$ Torr; $P_{a,CO_2}=20$ Torr) has a greater lung gas exchange defect (130–70=60 Torr) compared with a patient with respiratory failure and hypercapnia (130–($P_{a,O_2}=50$ Torr + $P_{a,CO_2}=50$ Torr)=30 Torr). At sea-level, values of ~150 Torr (~20 kPa) and ~180 (~24 kPa) should be considered normal under $F_{I,O_2}=0.24$ and $F_{I,O_2}=0.28$, respectively.

When evaluating the severity of arterial hypoxaemia, the computation of the $P_{a,O_2}/F_{I,O_2}$ ratio is recommended; lung injury is defined as $P_{a,O_2}/F_{I,O_2}<300$, while adult respiratory distress syndrome (ARDS) is associated with a $P_{a,O_2}/F_{I,O_2}$ ratio <150. The latter condition is usually observed in severely ill patients breathing oxygen at high concentration (true shunt >30%). A limitation of the use of the $P_{a,O_2}/F_{I,O_2}$ ratio in clinical practice is the potential underestimation of the severity of lung injury when $P_{a,O_2}/F_{I,O_2}$ is measured under room conditions [20].

Step 2: Diagnosis of simple and mixed A–B disorders

The second step in ABG interpretation is the evaluation of A–B status; this is best obtained by the integrated reading of P_{a,CO_2} and pH. In fact, if the P_{a,CO_2} is abnormal (e.g. <36 or >44 Torr (<4.8 kPa or >5.9 kPa) and the pH is abnormal (<7.36 or >7.45) an A–B disorder can be diagnosed easily (table 2).

It should be noted that changes in P_{a,CO_2} and pH in non-respiratory disorders are in the same

Table 1 P_{A-a,O_2} in the evaluation of the causes of arterial hypoxaemia

Cause	P_{a,O_2}	P_{A-a,O_2}	P_{A-a,O_2} (when $F_{I,O_2}>0.6$)
Hypoventilation	↓	↔	
V'/Q' mismatch	↓	↑	↔
Diffusion impairment	↓	↑	↑↔
Shunt	↓	↑↑	↑↑↑

Table 2 Pa,CO$_2$ and pH in simple A–B disorders

	Pa,CO$_2$	pH
Respiratory acidosis	↑	↓
Respiratory alkalosis	↓	↑
Non-respiratory (metabolic) acidosis	↓	↓
Non-respiratory (metabolic) alkalosis	↑	↑

where [HCO$_3^-$] is expressed in mmol per L, Pa,CO$_2$ in Torr and [H$^+$] in nmol per L.

Some might argue that the diagnosis of severe metabolic acidosis can be made easily in the presence of a value of [HCO$_3^-$] = 5 mEq per L; we can answer that this diagnosis can also easily made with the integrated reading of Pa,CO$_2$=15 Torr (2 kPa) and pH=7.15. In addition, the severity and prognosis of the A–B disorders are better described by the pH value: values <6.9 or >7.8 are not considered compatible with life. However, we should acknowledge, as discussed below, that the [HCO$_3^-$] computation is useful for subsequent calculation of the anion gap (AG$^-$) to be used in the diagnosis of mixed non-respiratory A–B disorders [22].

In order to correctly identify the presence and type (simple *versus* mixed) of A–B disorders, it is useful to compare data measured at ABG with those calculated according to the equations shown in table 3. Although the correlation of Pa,CO$_2$ with pH is not linear, a linear equation could be used to approximate this relationship in the Pa,CO$_2$ range of 25–80 Torr. This limited linear relationship is calculated from existing equations that described the Pa,CO$_2$ *versus* [H$^+$] relationship [22]. In simple acute respiratory disorders, for each 10-Torr (1.3 kPa) variation in Pa,CO$_2$, the expected change in pH value is 0.07 for acidosis and 0.08 for alkalosis, while in simple chronic respiratory disorders it is 0.03 for both acidosis and alkalosis [22]. In simple metabolic disorders, the expected value of Pa,CO$_2$ should correspond to the last two digits of pH. The same information can be more easily obtained by plotting data measured at ABG on a reference nomogram (figure 3).

Pa,CO$_2$–pH values that fall into the acute or chronic, respiratory and non-respiratory (or metabolic) "bands" should be considered as "simple" disorders (Case 1: Pa,CO$_2$ 70 Torr, pH 7.19, acute respiratory acidosis). Pa,CO$_2$–pH values that fall between respiratory and metabolic "bands" should be considered as "mixed" disorders (Case 2: Pa,CO$_2$ 40 Torr, pH 7.20, acute respiratory and metabolic acidosis).

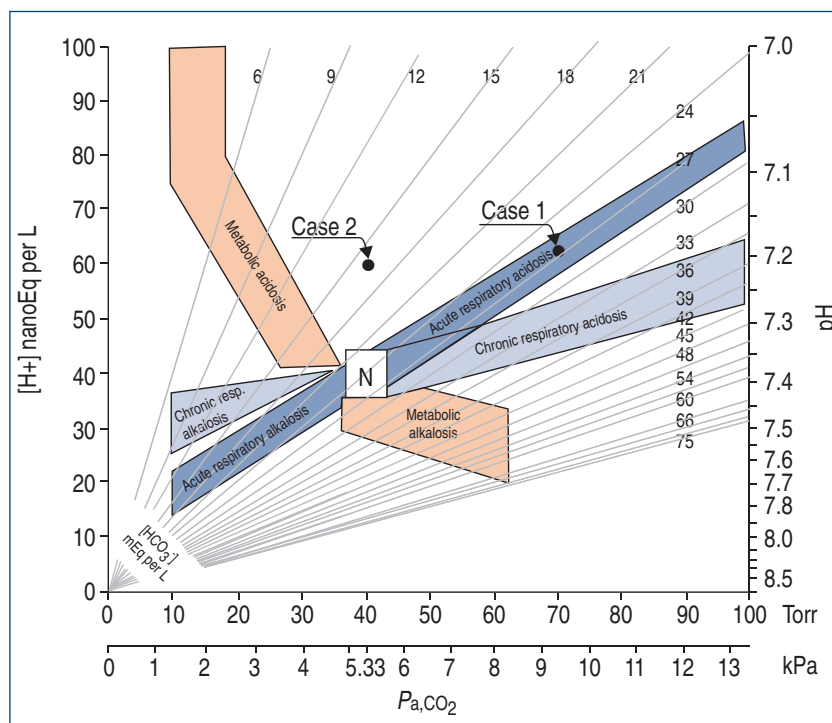The causes of simple A–B disorders are shown in tables 4 and 5.

direction ("concordant"), while they are "discordant" in respiratory disorders.

Traditional teaching has emphasised the role of HCO$_3^-$ in the evaluation of A–B status, often generating confusion between causes and effects. In our opinion, in order to understand the type and the compensation of A–B disorder, [HCO$_3^-$] adds very little to the information already provided by Pa,CO$_2$ and pH. As discussed later on, well-known equations are available to compute the expected acute and chronic changes in pH induced by changes in Pa,CO$_2$ (in respiratory disorders) and *vice versa* (in non-respiratory or "metabolic" disorders) (table 3).

Modern ABG equipment provides for the computation of HCO$_3^-$ values. We suggest that the reader learns to compute HCO$_3^-$ by using the modified Kassirer–Bleich formula [21], after having transformed pH into [H$^+$] by using appropriate tables:

$$[HCO_3^-] = 24 \times P\text{a,CO}_2 / [H^+]$$

Figure 3
Pa,CO$_2$–pH nomogram for the diagnosis of simple and mixed A–B disorders.

Diagnosis of mixed non-respiratory ("metabolic") A-B disorders

In this section the causes of mixed non-respiratory A–B disorders are discussed. Particular emphasis will be paid to the role of electrolyte studies

Table 3   Expected compensation for simple A–B disorders

| | Respiratory disorders | | Non-respiratory (metabolic) disorders |
	Acute	Chronic	
Acidosis	$\Delta[H^+] = 0.8 \times \Delta P_{a,CO_2}$ or $\uparrow$10 mmHg in P_{a,CO_2} = $\downarrow$0.07 pH	$\Delta[H^+] = 0.3 \times \Delta P_{a,CO_2}$ or $\uparrow$10 mmHg in P_{a,CO_2} = $\downarrow$0.03 pH	P_{a,CO_2} = last 2 digits of pH
Alkalosis	$\Delta[H^+] = 0.8 \times \Delta P_{a,CO_2}$ or $\downarrow$10 mmHg in P_{a,CO_2} = $\uparrow$0.08 pH	$\Delta[H^+] = 0.17 \times \Delta P_{a,CO_2}$ or $\downarrow$10 mmHg in P_{a,CO_2} = $\uparrow$0.03 pH	P_{a,CO_2} = last 2 digits of pH

Table 4   Causes of respiratory disorders

Respiratory acidosis

Nervous system (central nervous system depression, neuromuscolar disorders)
Chest wall abnormalities, pleural diseases
Lung diseases

Respiratory alkalosis

Anxiety, central nervous system disorders
Hormones/drugs (catecholamine, progesterone, hyperthyroidism, salicylate)
Fever, Gram-negative sepsis
Lung diseases, hypoxia
Liver diseases

and in particular the utility of chloride ion (Cl^-) and AG^- measurements. This may be considered as "step 3" in ABG interpretation. Modern ABG equipment usually provides measurements of serum electrolytes. Key points the reader should keep in mind for the correct interpretation of mixed non-respiratory A–B disorders are:

1) HCO_3^- is the dissociated form of carbonic acid, *i.e.* a weak acid. In a solution, all weak acids are in equilibrium with the same $[H^+]$ (isohydric principle). It follows that all the buffer pairs and the derived equilibrium equations can be used to evaluate $[H^+]$.

2) The carbonic acid equilibrium is an open system; alveolar ventilation, besides $[H^+]$, influences this system. In the carbonic acid equilibrium, $[HCO_3^-]$ is a dependent variable influenced by changes, other than $[H^+]$, in alveolar ventilation and P_{a,CO_2}. The role of these variables and their interactions in metabolic disorders are summarised in figure 4. Metabolic disorders originate from changes in strong ions (electrolytes and strong acids) and weak acid concentrations that directly affect $[H^+]$ (figure 4, point 1); as a consequence, all the equilibria of the weak acids in solution are modified (figure 4, point 2). Carbonic acid equilibrium is also influenced by the elimination or retention of undissociated forms of carbonic acid (*i.e.*, CO_2 and thus H_2CO_3) through changes in alveolar ventilation (V_A) (respiratory compensation) (figure 4, point 3). This again affects the pH and moves all the weak acid equilibria (figure 4, point 2). In the figure, the independent variables that can directly affect $[H^+]$ are

Table 5   Causes of metabolic disorders

Metabolic acidosis

Normochloraemic acidosis (or high-AG^- acidosis)
    Ketoacidosis
    Lactic acidosis
    Renal failure
    Toxins
Hyperchloraemic acidosis (or normal- AG^- acidosis)
    Extra-renal loss of Na^+
    Renal tubular acidosis

Metabolic alkalosis

Chloride-responsive type
    Gastric fluid loss
    Volume contraction
    Post-hypercapnic
Chloride-resistant type
    Mineralcorticoid disorders
    Milk-alkali and Bartter syndromes
    Hypoalbumin
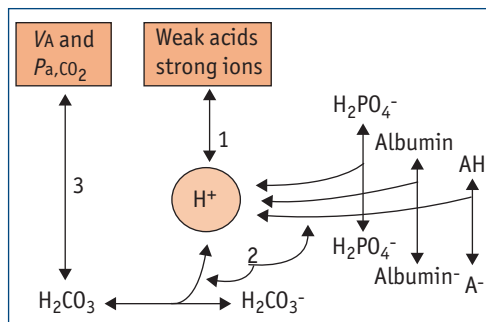
represented in squares. These are V_A and strong ions and weak acid concentrations. Renal excretion or retention of HCO_3^- does not affect the equilibrium.

3) All metabolic disorders can be attributed to changes in acid concentrations (A^-). These are usually detected as changes in anion gap (AG^-) and in Cl^-. AG^- is the concentration of unmeasurable acids that is computed as the negative charges in excess in solution:

$$AG^- = Na^+ - (HCO_3^- + Cl^-) = \sim 12$$

In normal conditions, AG^- reflects the unmeasured dissociated form of phosphoric acid, albumin (the main non-volatile weak acids) and a small amount of other unmeasured anions (*e.g.* lactate, sulfate). Albumin reduction may cause metabolic alkalosis. As shown in table 6, an increase of other anions is expected to cause high-AG^- metabolic acidosis. An increase or reduction in serum $[Cl^-]$ causes other metabolic disorders, *i.e.* hyperchloraemic acidosis and hypochloraemic alkalosis. Na^+ loss results in a net increase in $[Cl^-]$; conversely Na^+ administration causes a net reduction in $[Cl^-]$. Vasopressin release and thirst stimulus, by affecting renal water excretion/absorption, maintain serum $[Na^+]$ within normal limits. The normal $[Cl^-]$ is ~ 102 mEq per L, but in the presence of water deficit (hypernatraemia) or water excess (hyponatraemia) the observed $[Cl^-]$ has to be corrected

($[Cl^-]_{CORR}$) according to the following formula:

$$[Cl^-]_{CORR} = [Cl^-]_{observed} \times (140/[Na^+]_{observed})$$

4) Renal excretion of Cl^- affects pH. In order to regulate plasma pH, the kidney excretes Cl^- as its ammonium salt (NH_4Cl). Keeping this in mind, it is very useful in the evaluation of the causes of metabolic acidosis (*i.e.* renal or extra-renal) to measure urinary electrolytes and to compute the urinary AG^- (AG_U) [23] by using the following formula:

$$AG_U = Na_U^+ + K_U^+ - Cl_U^- > 0$$

where Cl_U^-, Na_U^+ and K_U^+ are, respectively, the urinary $[Cl^-]$, $[Na^+]$ and $[K^+]$
Negative AG_U values are expected in "extra-renal causes" of acidaemia; positive values indicate "renal causes" of acidaemia. The measurement of urinary $[Cl^-]$ is very useful in the differential diagnosis of the causes of metabolic alkalosis. $[Cl^-] <$ 10 mEq per L suggests a hypovolaemic condition [24] that should respond to saline (NaCl) infusion (chloride-responsive metabolic alkalosis).

Conclusion

In clinical practice, the correct interpretation of ABG provides unique information on the causes and on the severity of lung gas exchange and A–B abnormalities; this represents a fundamental step to be used in the therapeutic approach that should be aimed at the correction of the primary cause of the disease. Figure 5 summarises the interpretative "integrative" approach to be used in the evaluation of the ABG fundamental variables, *i.e.* $Pa_{,O_2}$, $Pa_{,CO_2}$ and pH. As a first step (Step 1), the combined reading of $Pa_{,O_2}$ and $Pa_{,CO_2}$ values, on room air and during supplemental oxygen breathing, should be used to identify the causes and the severity of arterial hypoxaemia (blue squares and blue circles). As a second step (Step 2), the combined reading of $Pa_{,CO_2}$ and pH, with the help of a reference nomogram, is needed for the correct diagnosis of simple and mixed A–B disorders (red squares). The study of serum electrolytes (Step 3), usually measured by modern ABG equipment, will be of great help in the identification of the causes of non-respiratory (or metabolic) disorders (red squares). The causes of metabolic disorders can be further investigated by measuring urinary electrolytes, strong acids and serum albumin (red circles).

Table 6   Changes in Cl⁻ and AG⁻ in metabolic disorders

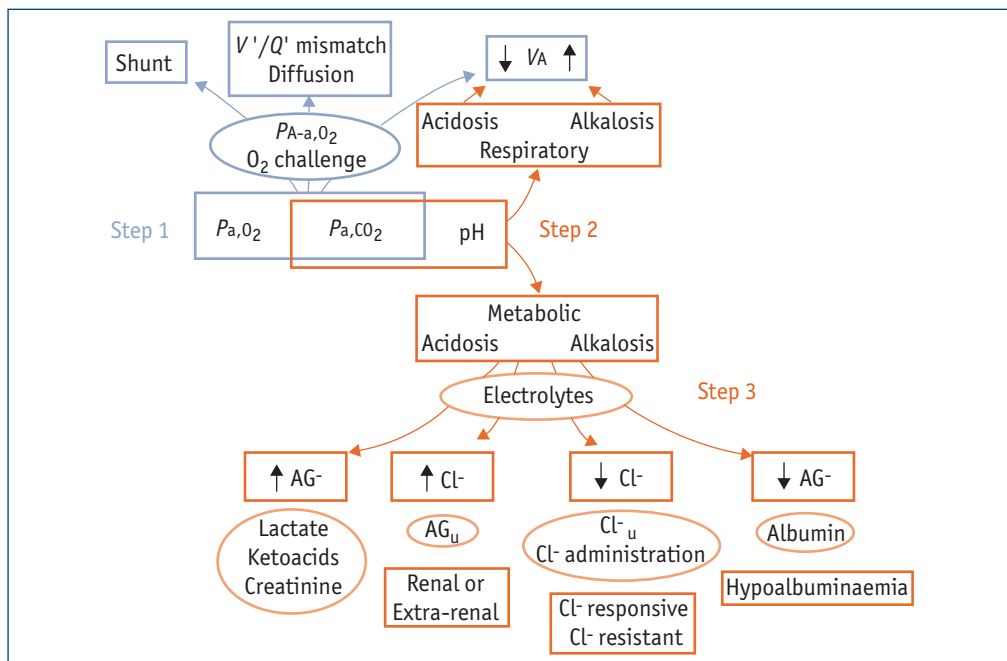|	Cl⁻	AG⁻
Acidosis	↑	↑
	Renal Cl⁻ retention	Lactic acid
	Renal or extra-renal loss of Na⁺	Ketoacids
		Inorganic acids
		Toxins
Alkalosis	↓	↓
	Renal or extra-renal loss of Cl⁻	Albumin reduction
	Na⁺ administration	

Figure 5
A comprehensive approach to ABG interpretation.

Case presentations

In each of the following cases, state which of the answers are true or untrue. There may be more than one true answer per case.

Case 1
Comatose patient, aged 25 years.
ABG measurements (F_{I,O_2} 0.21):

P_{a,O_2}	60 Torr (8 kPa)
P_{a,CO_2}	70 Torr (9.3 kPa)
pH	7.15

a) Pulmonary gas exchange is impaired because of a diffusion defect.
b) Alveolar ventilation is reduced.
c) The patient has a mixed A–B disorder (respiratory acidosis + metabolic acidosis).
d) Electrolyte measurement could help to understand the cause of the respiratory disorder.

Case 2
A 60-year-old diabetic patient with fever (39°C), cough and purulent sputum.
ABG measurements (F_{I,O_2} 0.21):

P_{a,O_2}	50 Torr (6.7 kPa)
P_{a,CO_2}	25 Torr (3.3 kPa)
pH	7.40

Plasma electrolytes:

[Na$^+$]	139 mEq per L
[Cl$^-$]	100 mEq per L
AG$^-$	24 mEq per L

a) A severe impairment in pulmonary gas exchange is present.
b) Diffusion limitation or shunt can cause the gas-exchange defect.
c) The patient has a mixed A–B disorder (respiratory alkalosis + metabolic acidosis).
d) Metabolic acidosis is probably due to renal tubular acidosis with Cl$^-$ retention.

Case 3

A 65-year-old patient with chronic heart failure, under treatment with loop diuretics, develops sudden dyspnoea and hypotension.

ABG measurements (F_{I,O_2} 0.50):

P_{a,O_2}	66 Torr (8.8 kPa)
P_{a,CO_2}	25 Torr (3.3 kPa)
pH	7.50

Serum electrolytes:

$[Na^+]$	135 mEq per L
$[Cl^-]$	90 mEq per L
AG^-	21 mEq per L

a) A mild impairment in pulmonary gas exchange is present.
b) Respiratory alkalosis is present.
c) Low $[Cl^-]$ and high AG^- values suggest the presence of a mixed A–B disorder.
d) Metabolic alkalosis is probably caused by diuretic treatment.

References

1. Haldane JS, Lorrain Smith J. The absorption of oxygen by the lungs. *J Physiol (London)* 1897; 22: 231–258.
2. Douglas CG, Haldane JS, Henderson Y, Schneider EC. Physiological observation made on Pike's Peak, Colorado, with special reference to adaptation to low barometric pressures. *Philos Trans R Soc Lond B Biol Sci* 1913; 203: 185–381.
3. Fenn WO, Rahn H, Otis AB. A theoretical study of the composition of the alveolar air at altitude. *Am J Physiol* 1946;146: 637-653.
4. Riley RL, Cournand A. "Ideal" alveolar air and the analysis of ventilation–perfusion relationships in the lungs. *J Appl Physiol* 1949; 1: 825–847.
5. Riley RL, Cournand A. Analysis of factors affecting partial pressures of oxygen and carbon dioxide in gas and blood of lungs: theory. *J Appl Physiol* 1951; 4: 77–101.
6. Evans JW, Wagner PD. Limits on VA/Q distribution from analysis of experimental gas elimination. *J Appl Physiol* 1977; 42: 889–898.
7. Van Slyke DD, Neill JM. The determination of gases in blood and other solutions by vacuum extraction and manometric measurement. *J Biol Chem* 1924; 61: 523–573.
8. Astrup P. A simple electrometric technique for the determination of carbon dioxide tension in blood and plasma, total content of carbon dioxide in plasma and bicarbonate content in 'separated' plasma at fixed carbon dioxide tension. *Scand J Clin Lab Invest* 1956; 8: 33–43.
9. Siggaard-Andersen O, Fogh-Andersen N. Base excess or buffer base (strong ion difference) as a measure of a non-respiratory acid-base disturbance. *Acta Anaesthesiol Scand* 1995; 39: Suppl. 107, 123–128.
10. Siggaard-Andersen O. The acid–base status of blood. 4th Edn. Copenhagen, Munksgaard, 1974.
11. Clark LC, Wolf R, Granger D, Taylor Z. Continuous recording of blood oxygen tension by polarography. *J Appl Physiol* 1953; 6: 189–193.
12. Severinghaus JW, Bradley AF. Electrodes for blood pO_2 and pCO_2 determination. *J Appl Physiol* 1958; 13: 515–520.
13. Stow R, Baer RF, Randall B. Rapid measurement of tension of carbon dioxide in blood. *Arch Phys Med Rehabil* 1957; 38: 646–650.
14. Schwartz WB, Relman AS. A critique of the parameters used in the evaluation of acid–base disorders. "Whole-blood buffer base" and "standard bicarbonate" compared with blood pH and plasma bicarbonate concentration. *N Engl J Med* 1963; 268: 1382–1388.
15. Brackett NC Jr, Cohen JJ, Schwartz WB. Carbon dioxide titration curve of normal man. Effect of increasing degrees of acute hypercapnia on acid–base equilibrium. *N Engl J Med* 1965; 272: 6–12.
16. Story DA. Bench-to-bedside review: a brief history of clinical acid–base. *Crit Care* 2004; 8: 253–258.
17. Stewart PA. Modern quantitative acid–base chemistry. *Can J Physiol Pharmacol* 1983; 61: 1444–1461.
18. Figge J, Rossing TH, Fencl V. The role of serum proteins in acid–base equilibria. *J Lab Clin Med* 1991; 117: 453–467.
19. Fencl V, Jabor A, Kazda A, Figge J. Diagnosis of metabolic acid–base disturbances in critically ill patients. *Am J Respir Crit Care Med* 2000; 162: 2246–2251.
20. Allardet-Servent J, Forel JM, Roch A, *et al.* FIO$_2$ and acute respiratory distress syndrome definition during lung protective ventilation. *Crit Care Med* 2009; 37: 202–207.
21. Kassirer JP, Bleich HL. Rapid estimation of plasma carbon dioxide tension from pH and total carbon dioxide content. *N Engl J Med* 1965; 272: 1067–1068.
22. Narins RG, Emmett M. Simple and mixed acid–base disorders: a practical approach. *Medicine* 1980; 59: 161–187.
23. Batlle DC, Hizon M, Cohen E, Gutterman C, Gupta R. The use of the urinary anion gap in the diagnosis of hyperchloremic metabolic acidosis. *N Engl J Med* 1988; 318: 594–599.
24. Mersin SS, Ramelli GP, Laux-End R, Bianchetti MG. Urinary chloride excretion distinguishes between renal and extrarenal metabolic alkalosis. *Eur J Pediatr.* 1995;154(12):979-82.

Suggested answers

1. a) False. b) True. c) False. d) False.
2. a) True. b) True. c) True. d) False.
3. a) False. b) True. c) True. d) True.