Arterial Blood Gases

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ABSTRACT

Arterial blood gases (ABG) results reflect underlying pathology and interpretation of the results are often compounded by ongoing disease processes and clinical interventions. While ABG specimens should be analysed immediately for optimal results the Clinical and Laboratory Standards Institute (CLSI) has recommended a window of 30 minutes at room temperature from blood collection to ABG analysis. A fresh and simple approach to interpreting ABG is provided.

Keywords: anion gap (AG), arterial blood gases (ABG), Base Excess (BE), O_2 Saturation, pCO_2, pH, pO_2, Standard Bicarbonate (SBC)

INTRODUCTION

Assessment of arterial blood gas (ABG) results are the bane of many students and junior doctors as it is poorly learned or poorly taught. We offer a simple approach.

TESTS COMMONLY INCLUDED

pH, pCO_2, pO_2, BE, SBC, O_2 Saturation

pH

The pH is a measurement of the acidity of the blood, indicating the number of hydrogen Ions [H^+] present. Most of the body's hydrogen ions are the result of carbohydrate and protein metabolism. The hydrogen ion concentration is maintained within a tight range of 7.35–7.45 by 3 mechanisms working in concert at different time frames and at different levels. The blood and tissue buffering system is activated within seconds, followed by the respiratory system where CO_2 is moderated within minutes, and the renal system where H^+ is excreted and HCO_3- recovered over several hours or days to reach equilibrium.

Partial Pressure of Carbon Dioxide (pCO_2)

The normal pCO_2 range reflects the amount of CO_2 dissolved in the blood. Carbon dioxide is produced by the internal respiration of tissue cells and excreted from the body by external respiration via the lungs.

Partial Pressure of Oxygen (pO_2)

The partial pressure of oxygen (pO2) reflects the amount of oxygen gas dissolved in the blood.

O_2 Saturation

Oxygen saturation measures the percentage of haemoglobin that is fully combined with oxygen as represented by the oxygen-haemoglobin dissociation (saturation) curve. The plateau of the haemoglobin dissociation curve shows that there is a substantial reserve for oxygen, and haemoglobin saturation remains high at about 75–80% at pO_2 greater than 40mmHg.

Base Excess (BE)

The BE is defined as the amount of acid or base (in mmol) required to titrate 1 L of blood to pH 7.4, at 37°C and pCO_2 of 40mmHg. The calculation of BE is dependent on haemoglobin, pH and pCO_2. The BE does not distinguish a primary metabolic disorder from metabolic compensation for respiratory disorder.

Bicarbonate [HCO_3-]

HCO_3- represents the calculated amount of bicarbonate in the blood.

Standard Bicarbonate

The standard bicarbonate is the calculated bicarbonate concentration corrected to pCO2 of 40mmHg.

SPECIMEN

An ABG specimen should be collected in a heparinised blood gas syringe anerobically and analysed within 30 minutes; otherwise, they should be placed on ice. Prior to an arterial puncture, the Allen Test should be performed to check for the ability of the radial and ulnar arteries to return blood to the hand. By engaging the patient while doing the Allen Test, the phlebotomist engages the patient and thus helps to put him or her at ease further ensuring an optimal outcome. While the use of local anaesthesia in arterial puncture has been recommended[1] it is seldom practised. A comparison of 5 arterial blood sample kits showed that current available products satisfy requirements for speed, ease of sampling, and specimen quality[2]. Glycolysis can cause significant changes in pH, pO_2, and pCO_2 after 20–30 minutes at room temperature[3]. It is important to allay the anxiety of the patient when obtaining the specimen as such a "white coat" effect may skew ABG results. The Clinical Laboratory and Standards Institute (CLSI) recommends that ABG specimens collected in plastic syringes should be analysed within 30 minutes if they are left at room temperature[2]. Guidelines are also available on ABG sampling and analysis[4–6].

PRE-ANALYTICAL SOURCES OF ERROR

Pre-analytical sources of error may be introduced during patient preparation and specimen handling. A patient safety procedure that can never be over-emphasised is positive patient identification — identifying the patient and labelling the specimen at the bedside using 2 primary identifiers. Rapid changes in ABG results may be expected in an agitated patient or following physical exertion. Ideally, the patient should be in a supine and relaxed position, and a lapse of 20–30 minutes be allowed if the fraction of inspired oxygen (FiO_2) has been changed before another patient sample is drawn[4]. Commercial blood gas syringes have been calibrated for the correct amount of blood to anticoagulant and strict anerobiosis. Excess heparin can affect pH and cause specimen dilution while excess blood may cause clotting, all of which will affect the quality of ABG results.

AFTER CARE

To prevent haematoma at the puncture site apply pressure for up to 10 minutes or even longer for anticoagulated patients. Always check for any bleeding thereafter.

INDICATIONS

ABG results provide information on the acid-base status, oxygenation (pO_2 and haemoglobin saturation) and CO_2 elimination. A utilisation survey of a large tertiary hospital showed that the reasons for requesting ABG tests included change in ventilator setting, respiratory event, part of clinical routine, metabolic episode, pre- and post-intubation, pre- and post-extubation, confirmation of pulse oximetry results and abnormal results, altered mental state, to establish brain death and cardiac event[7].

Most labs have defined "critical/alert values" for pH, pCO_2 and pO_2 which are to be called to the requesting clinician. Active tele-notification has been shown to be more effective than passive electronic systems[8]. These values represent patho-physiologic state at such variance with normal as to be life-threatening if an action is not taken quickly and for which an effective action is possible[9–11]. A report of the College of American Pathologists (CAP) Q-Probes Study in 623 institutions proposed that labs develop policies for handling a second critical value obtained on the same day based on the principle that physicians should not be contacted needlessly with values they know are previously elevated, and suggests that perhaps only the first of a series of values within 24 hours should require notification[12]. (Table 1 lists the reference ranges and alert values reported in the authors' lab.)

As clinicians opt for less invasive assessments of respiratory function such as capnography, pulse oximetry and spirometry, ABG may be indicated only to confirm the patient's status, for example acute asthma and chronic obstructive pulmonary disease (COPD)[13–16].

LIMITATIONS

An ABG should not be ordered for patients presenting with bleeding diathesis, arteriovenous fistula, severe peripheral vascular disease, absence of an arterial pulse; and infection or haematoma

Table 1. ABG parameters, reference range and alert values (reported by the authors' lab).

ABG Parameter	Reference Ranges	Alert Values
pH	7.35–7.45	<7.10 or >7.60
pCO_2 (mmHg)	35.0–45.0	<20.0 or >50
pO_2 (mmHg)	75.0–100.0	<55.0
Base Excess (BE)	-2.0–2.0 mmol/L	
Actual Bicarbonate (HCO_3-)	23–33 mmol/L	
Standard Bicarbonate (SBC)	21.0–27.0 mmol/L	
O_2 Saturation	95.0–100.0 %	

at the site of the specimen collection to prevent iatrogenic complications. Clinicians should interpret AG values derived from point of care testing (POCT) analysers cautiously as there is high probability of error[17].

METHOD

Not much has changed in the principles of the pH, pO_2 and pCO_2 measurements since Severinghaus described their blood gas apparatus in the 1950s[18–20]. What has changed since then are practical innovations such as miniaturisation and automation of the analytical system in the 1970s and addition of new parameters such as electrolytes, glucose, urea, lactate, and haematocrit or haemoglobin in the 1990s. The pH, pO_2 and pCO_2 are directly measured using electrochemistry principles and HCO_3 is calculated from the pH and pCO_2 results using the Henderson-Hasselbalch equation. The O_2 content, O_2 saturation, total CO_2, standard bicarbonate and base excess are calculated parameters. There are numerous instrument designs that seem different but are essentially identical as the differences are not in the sensing or measuring aspects but in the ancillary areas such as calibration, sample introduction and electronic handling of the signal generated by the electrodes.

POCT has been proposed to support immediate treatment decisions, reduction in risk of pre-analytical errors and smaller sample volume required. The counter argument is that POCT reduces risks of errors in only in few steps of the entire testing process but there are serious challenges from a risk management perspective, such as poorly maintained ABG analysers that are carried from 1 room to another, thus becoming reservoirs for nosocomial infections and antibiotic resistant bacteria[21]. Studies have shown that POCT ABG and those performed at the satellite lab[22,23] cost more than central lab testing. Ultimately, the choice of ABG analysers will rest on the definition of "profit" in healthcare terms, namely, cost savings, affordability and a better quality of life[24,25].

INTERPRETING ABG RESULTS

A patient's clinical history together with the ABG and electrolyte results provides clues to acid-base disorders. Fig. 1 (overleaf) shows a table that summarises the ABG values expected in various acid-base conditions. Respiratory disorders involve conditions with over- or under- breathing resulting in abnormal pCO_2 and the consequential effect on H^+ with resulting renal [HCO_3] compensatory response. Metabolic conditions include acidosis (acid gain &/or bicarbonate loss) or alkalosis (acid loss &/or bicarbonate gain) resulting in abnormal [HCO_3] and the consequential effect on pH with resulting respiratory (pCO_2) compensation. It is thus evident from Fig. 1 that for respiratory disorders all the arrows follow [H^+] and are in the same direction — Respi-H^+; for metabolic disorders all the arrows follow (pH) and are in the same direction — Meta-pH.

Additional evaluation is required in acidosis — anion gap for metabolic acidosis and oxygen status for respiratory acidosis. A mnemonic for remembering causes of acidosis is provided below. ABG results of mixed acid-base disorders, while confusing, are less crucial than the treatment of the underlying disorder and hence of interest only to the specialist who has a complete understanding of the patient, including underlying pathologies, pharmacotherapy, therapeutic ventilation and

ACID-BASE DISORDER	NO COMPENSATION	EXPLANATION	$[H^+]$ pH	pCO_2	HCO_3
1° Respiratory Acidosis	$[H^+] \uparrow \propto \frac{pCO_2 \uparrow}{[HCO_3^-]\ N}$	CO_2 production > CO_2 elimination	$[H^+] \uparrow\uparrow$	$\uparrow\uparrow$	N
1° Respiratory Alkalosis	$[H^+] \downarrow \propto \frac{pCO_2 \downarrow}{[HCO_3^-]\ N}$	CO_2 elimination > O_2 absorption	$[H^+] \downarrow\downarrow$	$\downarrow\downarrow$	N
1° Metabolic Acidosis	$[H^+]\uparrow = pH\downarrow \propto \frac{pCO_2\ N}{[HCO_3^-] \downarrow}$	Accumulation of acids or loss of alkali	pH $\downarrow\downarrow$	N	$\downarrow\downarrow$
1° Metabolic Alkalosis	$[H^+]\downarrow = pH\uparrow \propto \frac{pCO_2\ N}{[HCO_3^-] \uparrow}$	Loss of acids or gain of alkali	pH $\uparrow\uparrow$	N	$\uparrow\uparrow$

ACID-BASE DISORDER	PARTIAL COMPENSATION	EXPLANATION	$[H^+]$ pH	pCO_2	HCO_3
1° Respiratory Acidosis	$[H^+] \uparrow \propto \frac{pCO_2 \uparrow}{\mathbf{[HCO_3^-]} \Uparrow}$	Kidneys excrete $[H^+]$ ions	$[H^+] \uparrow$	$\uparrow$	$\uparrow$
1° Respiratory Alkalosis	$[H^+] \downarrow \propto \frac{pCO_2 \downarrow}{\mathbf{[HCO_3^-]} \Downarrow}$	Kidneys conserve $[H^+]$ ions	$[H^+] \downarrow$	$\downarrow$	$\downarrow$
1° Metabolic Acidosis	$[H^+] \uparrow = pH \downarrow \propto \frac{\mathbf{pCO_2} \Downarrow}{[HCO_3^-] \downarrow}$	Hyperventilate to eliminate CO_2	pH $\downarrow$	$\downarrow$	$\downarrow$
1° Metabolic Alkalosis	$[H^+] \downarrow = pH \uparrow \propto \frac{\mathbf{pCO_2} \Uparrow}{[HCO_3^-] \uparrow}$	Hypoventilate to conserve CO_2	pH $\uparrow$	$\uparrow$	$\uparrow$

ACID-BASE DISORDER	FULL COMPENSATION	EXPLANATION	$[H^+]$ pH	pCO_2	HCO_3
1° Respiratory Acidosis	$[H^+] \uparrow \propto \frac{pCO_2 \uparrow}{\mathbf{[HCO_3^-]} \Uparrow\Uparrow}$	Kidneys excrete $[H^+]$ ions	N but <7.40	$\uparrow$	$\uparrow\uparrow$
1° Respiratory Alkalosis	$[H^+] \downarrow \propto \frac{pCO_2 \downarrow}{\mathbf{[HCO_3^-]} \Downarrow\Downarrow}$	Kidneys conserve $[H^+]$ ions	N but >7.40	$\downarrow$	$\downarrow\downarrow$
1° Metabolic Acidosis	$[H^+] \uparrow = pH \downarrow \propto \frac{\mathbf{pCO_2} \Downarrow\Downarrow}{[HCO_3^-] \downarrow}$	Hyperventilate to eliminate CO_2	N but <7.40	$\downarrow\downarrow$	$\downarrow\downarrow$
1° Metabolic Alkalosis	$[H^+] \downarrow = pH \uparrow \propto \frac{\mathbf{pCO_2} \Uparrow\Uparrow}{[HCO_3^-] \uparrow}$	Hypoventilate to conserve CO_2	N but >7.40	$\uparrow\uparrow$	$\uparrow\uparrow$

Key

$\uparrow$: Increase
$\downarrow$: Decrease
N: Normal
$\Uparrow$: Compensatory Increase
$\Downarrow$: Compensatory Decrease

Fig. 1: ABG values expected in various acid-base conditions.

other clinical information. Indeed a snapshot survey of hospital clinicians showed that only a quarter of the respondents offered correct differential diagnosis[26].

Many articles have been written on the fundamental concepts and interpretation of ABG[27–38].

Clinical History

Respiratory acidosis may occur in situations where there is under-breathing — compromised O_2 and CO_2 movement in the lungs e.g. bronchitis, asthma, pneumonia, COPD and emphysema. Respiratory alkalosis occurs when there is over-breathing — increase in ventilatory rate e.g. hyperventilation during an anxiety attack or mechanical over-ventilation. Acute respiratory failure may not have the benefit of full renal compensation as it takes between 2–3 days for the kidneys to achieve acid-base equilibrium. Serum potassium may be elevated or decreased in renal compensation for respiratory acidosis and alkalosis due to the H^+/K^+ exchange mechanism across cell membranes. Metabolic acidosis may be inferred in situations with acid production (diabetic ketoacidosis, lactic acidosis), acid ingestion (salicylate, methanol) and loss of bicarbonate rich fluids (diarrhea, intestinal fistula). Metabolic alkalosis would be suspected with ingestion of bicarbonate (antacids) and loss of acid (prolonged vomiting, diuretics).

Acid-base Status

The lungs and kidney work in concert to maintain the acid-base balance as represented by the Henderson-Hasselbach equation:

$$pH = pK + \text{Log}\ \frac{[\text{Base}]}{[\text{Acid}]}$$

pH is the negative log of $[H^+]$.

The bicarbonate buffering system is important in restoring acid-base homeostasis. The respiratory (pCO_2) and renal (HCO_3-) systems work in concert to return the plasma pH to near normal within 1–3 minutes and 48–78 hours, respectively. Hyperventilation removes CO_2 efficiently to moderate the adjustment of pH. The kidneys secrete hydrogen ions and also "recover" and "regenerate" bicarbonate ions. Bicarbonate buffering may be represented in the following equation:

$$[H^+] + [HCO_3^-] \Leftrightarrow H_2CO_3$$

By the Law of Mass Action:

$$[H^+] = \frac{K\,[H_2CO_3]}{[HCO_3^-]}$$ Where K is the first dissociation constant of carbonic acid

Dissolved CO_2 is proportional to the partial pressure of CO_2 as explained by Henry's Law. Therefore, in the Law of Mass Action equation, $[H_2CO_3]$ may be substituted with pCO_2. Thus, clinical acid-base disorders may be explained by the relationship:

$$[H^+] \text{ is proportional to } \frac{pCO_2}{[HCO_3^-]}$$

Using what we know about the bicarbonate buffering and compensatory systems, we may proceed to interpret results related to acidosis and alkalosis with reference to the normal ranges quoted by the performing lab as summarised in Table 1 and Fig. 1.

pH

A low pH indicates academia while a high pH indicates alkalemia. Despite the efficient bicarbonate buffering system, primary acid-base disorders will not be fully be compensated, and prior primary acidosis or alkalosis may be discerned by pH <7.4 and pH >7.4, respectively. Thus, a normal, or near normal pH could be present in chronic conditions. It must be noted that complete compensation or over-compensation seldom occurs so a near normal pH in the presence of abnormal levels of pCO_2 and HCO3- suggests a mixed acid-base disturbance.

pCO_2

A high pCO_2 indicates respiratory acidosis which may be primary or secondary to a primary metabolic alkalosis. A low pCO_2 indicates respiratory alkalosis which may be primary or secondary to a primary metabolic acidosis.

Bicarbonate

Bicarbonate is calculated from measured $[H^+]$ and pCO_2 values. A high standard $[HCO_3^-]$ indicates either primary metabolic alkalosis or a compensatory response to a primary respiratory acidosis. Conversely, a low $[HCO_3]$ indicates either primary metabolic acidosis or a compensatory response to a primary respiratory alkalosis.

Base Excess (BE)

Base Excess (BE) refers to the metabolic component of the acid-base disorder as any change in pH due to pCO_2 is not taken into consideration. BE will be positive in metabolic alkalosis and $[H^+]$ must be added to return the blood pH to normal. BE will be negative in metabolic acidosis, and $[H^+]$ must be removed to return blood pH to normal. The limitation of using BE in the interpretation of ABG results is that it does not distinguish a primary metabolic disorder from metabolic compensation for respiratory disorder.

Compensation

pH is maintained at a narrow range of 7.35–7.45 due to the compensatory actions. In primary respiratory acidosis, pCO_2 will be increased and the kidney will compensate by introducing $[HCO_3^-]$.

- In primary metabolic acidosis, the primary disorder is decreased HCO_3^- and the lungs compensate by increased ventilation to "blow off" CO_2, thus reducing pCO_2. In primary respiratory alkalosis, the primary disorder is decreased pCO_2 and the kidneys will compensate by conserving $[H^+]$.

- In primary metabolic alkalosis, the primary disorder is increased $[HCO_3^-]$ in the blood, and the lungs compensate by depressing ventilation. pCO_2 is an effective compensatory trigger as the central and peripheral chemoreceptors initiate increase in ventilation rate at a small change of around 3.8mmHg whereas little effect on ventilation is seen within pO_2 changes at the "physiological" range of 75–105 mmHg[39].

Respiratory compensation occurs within minutes but renal compensation may take up to 2 days for $[HCO_3^-]$ to reach equilibrium. The compensatory mechanisms are efficient and often return the plasma pH to near normal but it is uncommon for either complete compensation or over-compensation to occur.

Compound Acid-base Disorders

A patient may have more than 1 acid-base disorder especially in severely ill patients:

- Metabolic acidosis + respiratory acidosis = BIG acidosis (pH usually <7.15).

- Metabolic alkalosis + respiratory alkalosis = BIG alkalosis (pH usually >7.6)

When 2 acid-base conditions are counter-balancing the pH may be near normal, but the pCO_2 and $[HCO_3^-]$ will be grossly abnormal:

- Metabolic acidosis (low $[HCO_3]$) + Respiratory alkalosis (low pCO_2)

- Metabolic alkalosis (high $[HCO_3]$) + Respiratory acidosis (high pCO_2)

- Metabolic alkalosis + Metabolic acidosis (vomiting and renal failure)

Occasionally, triple acid-base disorders may be encountered e.g. a patient with COPD (respiratory acidosis) on diuretics (metabolic alkalosis) presenting with sepsis (metabolic acidosis).

METABOLIC ACIDOSIS

Assess Anion Gap (AG)

The AG may be used as an adjunct tool to assess acid-base problems. The anion gap is a term that is used to describe the difference between the sum of the two main cations, sodium and potassium and the sum of the two main anions, chloride and bicarbonate: $AG = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$

The AG of a normal person is about 12–20mmol/L. If AG is ≥20mmol/L, there is a 67% likelihood of metabolic acidosis and it AG is ≥30mmol/L, there is a 100% likelihood of metabolic acidosis, for example in instances when there is accumulation of acids such as salicylic, lactic, pyruvic, acetoacetic and sulphuric acids. **SLUM DOG** is useful mnemonic for clues to a high anion gap.

- **S**alicylate poisoning[40];

- **L**actic acidosis (hypoxia, hypoperfusion);

- **U**raemia (renal failure);

- **M**edication (antidiabetics — metformin[41,42] antimicrobials — linezolid[43], anti-seizure — topiramate[44]);

- **D**iabetic or alcoholic ketoacidosis[45];

- **O**rganic acids (metabolic disease in children[46], pyroglutamic acidemia[47]);

- **G**as and heavy metal poisoning (CO, CO_2, cyanide, iron, arsenic).

AG will be decreased in hypoalbuminemia and haemodilution since albumin accounts for most of the AG. **ABC** is useful mnemonic for clues to a low anion gap.

- **A**lbumin (haemorrhage, nephrotic syndrome, intestinal obstruction, liver cirrhosis, multiple myeloma);

- **B**icarbonate;

- **C**hloride.

The AG will not change if reduced [HCO_3-] is balanced by increased [Cl-], for example in chronic diarrhoea or when [HCO_3-] is lost through the intestinal fistula. **GRIM** is a useful mnemonic for providing clues to a normal anion gap.

- **G**astrointestinal (chronic diarrhoea, uretero-colonic and pancreatic fistula);

- **R**enal tubular disease (proximal and distal tubular acidosis);

- **I**atrogenic (calcium chloride, total parenteral nutrition);

- **M**ineralocorticoid deficiency (Addison's disease).

Following a survey of 220 ABG analyses of older patients in an Irish hospital, the authors recommended that the AG should be calculated on every sample and every negative and positive AG should be fully investigated[48]. Since AG >30mmol/L and AG <3mmol/L are rare, the quality system of the laboratory should be investigated in these instances[49,50].

RESPIRATORY ACIDOSIS

Assess Oxygen Status

Oxygenation is linked to the acid-base status as [H^+] and pCO_2 in addition to temperature and 2,3-diphosphoglycerate (DPG) levels affects the affinity of oxygen to haemoglobin. Respiratory failure may be classified as Type I hypoxaemic respiratory failure and Type II hypercapnic respiratory failure, respectively[51]. Acute respiratory failure is a common in the intensive care unit (ICU) and 1 study found it to be present in 49% of patients admitted to ICU and another 8% developing it during their ICU stay[52]. Clinical manifestations include altered mental state (agitation or drowsiness), breathing difficulties, diaphoresis, tachycardia and hypertension. Respiratory failure is the consequence of lung failure to fully oxygenate the blood giving rise to hypoxia (pO_2 <60 mmHg) and/or respiratory muscle pump failure resulting in hypoventilation and hypercapnia (pCO_2 >45mmHg).

Type I (hypoxaemic) respiratory failure is characterised by increased pH, decreased pO_2 and decreased pCO_2. Low pCO_2 in venous blood will result in a reduction in the hypercapnic ventilatory drive to maintain a constant pCO_2 but consequently pO_2 is also decreased as a result of alveolar hypoventilation. Type I respiratory failure may be explained by 1 of 5 pathological mechanisms of hypoxemia and the mnemonic, AVAIL is useful as a memory aid for its explanation:

- **A**lveolar hypoventilation, e.g. opiate overdose;

- **V**entilation-perfusion mismatch, e.g. COPD, pulmonary embolus, cystic fibrosis;

- **A**natomical right-left shunt, e.g. intrapulmonary shunt, intracardiac shunt, persistent left superior vena cava with "unroofed" coronary sinus[53];

- **I**mpaired diffusion, e.g. diffuse parenchymal lung disease;

- **L**ow partial pressure of inspired oxygen, e.g. high altitude.

Type II (hypercapnic) respiratory failure is an imbalance between neural respiratory drive, the load on respiratory muscles and capacity of the respiratory muscles. Its diagnosis often requires a neurological examination. The underlying pathologies that cause decreased pH, increased pO_2 and decreased pCO_2 seen in Type II respiratory failure may be represented by the mnemonic MANNERS:

- **M**usculoskeletal problems (e.g. muscular dystrophy, severe kyphoscoliosis);

- **A**irway obstruction (e.g. epiglotitis);

- **N**europathy (e.g. motor neuron disease, Guillain-Barre syndrome);
- **N**euromuscular junction problems (e.g. myasthenia gravis);
- **E**xtrapulmonary issues (e.g. severe ascites with diaphragmatic splinting);
- **R**espiratory centre suppression (e.g. brainstem stroke, spinal anesthesia — propofol and midazolam[54]).
- **S**pinal cord lesion.

CONCLUSION

A good ABG specimen is the first step in ensuring medically useful data. A simple approach for interpreting ABGs has been suggested using our understanding of the relationship between $[H^+]$ with pCO_2 in respiratory and pH and $[HCO_3^-]$ in metabolic disorders as summarised in Fig. 1. Mnemonics have also been suggested as aids to interpret ABG. However, ABG results are only meaningful when interpreted in relation to the patient's medical history, underlying pathologies, pharmacotherapy, therapeutic ventilation and other clinical information.

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