Essential principles: blood gas analysis

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This article outlines a systematic way to interpret core results of arterial blood gases (pH, PaCO₂, PaO₂, HCO₃, BE).

AIMS AND OBJECTIVES

After reading this article, the reader should be able to

- define normal parameters;
- identify what abnormal parameters are likely to indicate;
- recognise when further assessment is indicated;
- · suggest interventions to resolve problems.

INTRODUCTION

With the advent of *Comprehensive Critical Care* (Department of Health, DOH, 2000) and increasing acuity of ward patients, arterial blood gas samples are also often taken. Results are used primarily to guide medical management, but nurses caring for sicker (level 2 and level 3) patients should understand key results. This article provides a systematic method for interpretation.

OVERVIEW

Arterial blood Gas (ABG) analysers measure:

- pH;
- respiratory function;
- metabolic function.

As with any other observation, 'normal' ranges may vary between sources and analysers, so are only a guide-trends or changes in each patient's results are

Author: Philip Woodrow, MA, RGN, DipN, Grad Cert Ed, ENB 100, 298, 870, Practice Development Nurse, Critical Care East Kent Hospitals University NHS Foundation Trust, Intensive Therapy Unit, Kent and Canterbury Hospital, Canterbury, Kent CT1 3 NG, UK Address for correspondence: P Woodrow, Intensive Therapy Unit, Kent and Canterbury Hospital, Canterbury, Kent CT1 3 NG, UK E-mail: philip.woodrow@ekht.nhs.uk more significant than abstract figure. Most analysers also measure some electrolytes and metabolites ('addons'), not discussed here but a table of normal reference ranges is included.

PRELIMINARY INFORMATION

Staff using blood gas analysers will need to enter information. Information required varies between different Trusts and, in some cases, between different clinical areas. Usually, users need an individual log on, and patient identification. Additional information may be required—readers should familiarise themselves with local policies and practices.

A SYSTEMATIC APPROACH

ABG analysers measure:

- pH
- respiratory function
- metabolic function
- electrolytes + metabolites

so analysis should follow a systematic sequence:

- 1. pH
- respiratory function-three core measurements (PaCO₂, PaO₂, SaO₂)
- 3. metabolic function-two core measurements (bicarbonate-HCO₃⁻, and base excess-BE) noting whether each aspect of 1–3 is normal, low or high.
- 4. Is compensation occurring (comparing pH with respiratory and metabolic function)? if so, which way?
- 5. electrolytes + metabolites

pH (acid/base balance) (normal 7.35–7.45 mol/L)

pH is a scale for measuring acidity or alkalinity. The pH scale ranges from 0 to 14. Normally, blood is slightly alkaline, arterial pH being 7.4, with an acceptable range of 7.35–7.45:

- pH < 7.35 = acidosis
- pH > 7.45 = alkalosis

Significant deviations rapidly become life threatening; Marieb and Hoehn (2007, 1057) suggest 'absolute blood pH limits for life' are 7.0–7.8, although occasionally patients may survive if isolated samples exceed this range.

Blood acid/base balance is the sum of respiratory and metabolic acid/base balances, so blood pH only indicates overall balance, not where any problems originate.

Significance: Significant acidosis is life threatening, so any new or worsening acidosis should be urgently reported. Treatments depend on the cause and system function.

Respiratory function

Blood gases show three main respiratory measurements:

- PaCO₂
- PaO₂
- SaO₂

*PaCO*₂ (*carbon dioxide*) [*normal* 4·6–6·1 kPa (*British Thoracic Society*–BTS, 2008)]

Carbon dioxide, usually printed first on ABG results, indicates

- ventilation volume
- respiratory acid/base balance

Air normally does not contain carbon dioxide (0.04%); blood carbon dioxide is a normal waste product of metabolism. Clearance, and so blood levels, depends on air volumes reaching, and exhaled by, lungs (*tidal volume*). Small volumes mean little carbon dioxide clearance, and so high blood carbon dioxide (*hypercapnia*). Excessive (hyper-) ventilation lowers blood carbon dioxide (*hypocapnia*). Air volumes may be measured by minute volume (affected by both tidal volume/depth of breathing and respiratory rate) and tests such as functional residual capacity. Patients breathing shallowly, slowly, or with obstructed airways usually have hypercapnia.

One of the three ways in which blood transports carbon dioxide is dissolving it in water to form carbonic acid. Carbonic acid is normally the main acid of blood, so hypercapnia makes blood more acidic. Therefore,

- hypercapnia (PaCO₂ > 6·1 kPa) = respiratory acidosis
- hypocapnia (PaCO₂ < 4.6) = respiratory alkalosis

Respiratory imbalance may be a disease, or compensating for metabolic imbalance.

Significance: Respiratory acidosis (hypercapnia) can be treated by increasing ventilation:

- tidal volume and/or respiratory rate with invasive ventilation
- increasing IPAP/EPAP (Inspired Positive Airway Pressure/Expired Positive Airway Pressure) difference with non-invasive ventilation
- bronchodilators, positioning and deep breathing with self-ventilating patients

Respiratory alkalosis is usually compensatory, so should not usually be treated.

PaO₂ (oxygen) [normal 12.0–14.6 kPa (BTS, 2008)]

Oxygen is needed for cells to survive.

Some medical staff members remove supplementary oxygen before taking ABGs. If assessing patients for home oxygen this is logical, but in acute illness removing supplementary oxygen is both illogical (part of information sought is whether oxygen therapy is appropriate) and dangerous (hypoxic cells eventually die–*necrose*). Hence, in acute illness, oxygen should not be removed before sampling ABGs.

Significance: Hyperoxia ($PaO_2 > 14.6 \text{ kPa}$)–consider reducing supplementary oxygen with hyperoxia (BTS, 2008). Hypoxia ($PaO_2 < 12.0 \text{ kPa}$)–consider giving/increasing supplementary oxygen.

SaO₂ (saturation of haemoglobin by oxygen) (normal 97–98%)

For practical purposes, peripheral $(SpO_2 = pulse oximetry)$ and arterial (SaO_2) saturation are the same. While PaO₂ measures partial pressure (effectively, oxygen available in plasma for diffusion), saturation measures oxygen carried by haemoglobin. The complex relationship of PaO₂ and SaO₂ is illustrated by the oxyhaemoglobin dissociation curve [described in most physiology textbooks, such as Marieb and Hoehn (2007)].

Significance: BTS (2008) suggests target saturation should be:

- 94–98% for most patients
- 88–92% for people with hypercapnic respiratory failure (generally, COPD) so titrate oxygen therapy to achieve target saturation and PaO₂.

Metabolic

Blood gas analysers measure two metabolic figures:

- HCO₃
- BE

although both may be expressed as standardised (SBC and SBE, or with the suffix–std). Some analysers may give both actual and standardised figures, whereas others give just one pair. 'Standardised' is discussed further below.

HCO_{3}^{-} (bicarbonate) (normal 21–26 mmol/L)

Bicarbonate is the main chemical buffer in blood. Therefore

- HCO₃⁻ < 21 mmol/L = metabolic acidosis
- $HCO_3^- > 26 \text{ mmol/L} = \text{metabolic alkalosis}$

BE (*base excess*) (*normal* \pm 2 *mEq/L*)

BE is a scale of metabolic acid/base balance. Excess of base is therefore positive, whereas lack of base (acidosis) is negative:

- BE < -2 = metabolic acidosis
- BE > +2 = metabolic alkalosis

Analysers calculate BE from HCO_3^- , so both should reflect the same metabolic acid/base status. If one suggests acidosis, and the other alkalosis, the sample was probably poor, making all results from the sample dubious.

Significance (of metabolic imbalance):

- Metabolic alkalosis is usually compensatory (often seen in ventilated patients as a 'metabolic overshoot', where respiratory acidosis is resolved through artificial ventilation, while the compensatory alkalosis continues, causing an overall alkalosis).
- Metabolic acidosis may have various causes: kidney failure, liver failure and hypoperfusion are the most common acute causes. Underlying causes should be treated where possible. Intravenous bicarbonate is not usually infused unless acidosis is severe (pH < 7.1 (Winser, 2001)).
- With dubious results, repeat the ABG.

STANDARDISED BICARBONATE AND BASE EXCESS

Analysers measure actual bicarbonate (bicarbonate actually in samples), from which they derive actual base excess. Bicarbonate and base excess are used to assess metabolic acid/base balance. Normally, almost all bicarbonate in blood is produced by the 'metabolic' system (mainly liver and kidneys). However, hypercapnia increases carbonic acid dissociation into bicarbonate. 'Standardised' figures therefore calculate bicarbonate derived from carbon dioxide, and subtract Table 1 Metabolic acid/base balance

Acidosis	Alkalosis
↓ HCO ₃ ⁻ /SBC	↑ HCO ₃ /SBC
↓ BE/SBE	↑ BE/SBE

this from actual measurements to reflect 'metabolic' function. In health, actual and standardised figures are identical, or very similar, but hypercapnia may cause significant differences, so always use standardised measurements (if available). Table 1 summarises metabolic acid/base balance

Compensation

The body attempts to maintain blood pH at 7.4, so respiratory or metabolic imbalance normally triggers an equal but opposite effect in the other system to maintain pH. Hence:

- is pH normal? (overall balance)
- is PaCO₂normal? (respiratory balance)
- are bicarbonate and base excess normal? (metabolic balance)

If all are normal, balance is perfect. If pH is normal, but the two systems are abnormal and opposite, one is compensating for the other. If blood pH is normal (7.35-7.45), one of the following is occurring:

- 1. respiratory and metabolic balance are both normal
- 2. respiratory balance is abnormal, and successfully compensated by an opposite metabolic balance
- metabolic balance is abnormal, and successfully compensated by an opposite respiratory balance
- If pH is abnormal, one of the following is occurring:
- 1. respiratory balance is abnormal, and not successfully compensated by an opposite metabolic balance
- 2. metabolic balance is abnormal, and not successfully compensated by an opposite respiratory balance
- 3. both respiratory and metabolic balances are abnormal, with no compensation occurring

Healthy lungs enable respiratory compensation within minutes (Guyton and Hall, 2006). Metabolic compensation takes considerably longer time-kidneys can increase acid removal 10-fold, but take 7–10 days to achieve this (Worthley, 2003). Hence, with acute metabolic imbalances compensation is normally rapid, whereas compensation for acute respiratory imbalances takes some days. If the other system is

Table 2 ABG differences in COPD

	PaCO ₂	pН	HCO_3^-
Chronic	1	\sim	1
Acute	\uparrow	\downarrow	\sim
Acute on chronic	\uparrow	\downarrow	\uparrow

functioning poorly, compensation is unlikely to succeed. Table 2 shows how this can be used to distinguish acute, chronic and acute-on-chronic obstructive airways disease (COPD).

Usually, imbalances are caused by acidosis; any alkalosis is compensatory. Primary alkalosis is very rare, although occasionally may be seen. Hence:

- if bicarbonate/base excess is opposite to pH, the problem is respiratory in origin
- if bicarbonate/base excess reflects pH, the problem is metabolic in origin

Significance: Identify the problem and any compensatory effects. Patient history usually suggests likely causes. Focus treatment on problems. Do not treat or remove any compensation unless it is problematic.

Electrolytes and metabolites

Not strictly part of blood gas analysis, normal reference ranges are summarised in Table 3.

ADDITIONAL CONSIDERATIONS

Venous samples (normal ScvO₂ 70–75%)

Venous samples are sometimes taken to measure

- ScvO₂ (central venous saturation)
- electrolytes

Venous and arterial electrolytes are usually similar. Differences between arterial and venous saturations reflect tissue uptake of oxygen, low venous saturation indicates excessive uptake, and so potential tissue hypoxia. The Surviving Sepsis Initiative (Dellinger *et al.*, 2004; Surviving Sepsis Campaign, 2007) recommends maintaining ScvO₂ > 70%.

Significance: If $ScvO_2 < 70\%$, attempt to increase oxygen delivery–e.g. increase FiO_2 /blood pressure, transfuse blood/fluids.

Table 3 Normal ranges for electrolytes and metabolites

Sodium = $135-145$ mmol/L	
Potassium = 3.5-4.5 mmol/L	
Calcium = $1.0-1.5$ mmol/L (n.b. ABG analysers measure ionised calciu	m;
laboratory U + Es measures whole blood calcium–normally $2 \cdot 0 - 2 \cdot 5$)
Chloride = $98-106 \text{ mmol/L}$	
Glucose = $4 \cdot 4 - 6 \cdot 1 \text{ mmol/L(van den Berghe et al., 2001)}$	
Lactate < 2.0 mmol/L	

Errors

Analysers usually have good quality control systems, alerting users to machine error–e.g. printing question marks or other symbols beside dubious results. However, errors when obtaining samples or during transportation may affect results, without analysers detecting problems. Interpreting results should include considering possible errors, especially if results conflict with other clinical information.

Significance: If in doubt, take another ABG, or send samples elsewhere (e.g. laboratory).

CONCLUSIONS

Interpreting ABGs is a skill that, like all skills, improves with practice. Hence, use a systemic approach to core interpretation:

- 1. pH
- 2. respiratory
- 3. metabolic
- 4. compensation
- 5. electrolytes + metabolites

take every opportunity to analyse samples and whenever possible discuss interpretation with more experienced colleagues.

KEY LEARNING POINTS

- ABGs can be a useful component of treatment planning for level 2/3 patients
- Analyse samples systematically starting with pH and compare this with respiratory and metabolic function then electrolytes + metabolites
- · take every opportunity to practice interpretation
- if in doubt about significance of any results, ask

RECOMMENDED TEXTS FOR FURTHER READING

Hennessey and Japp (2007) and Foxall and Kelsey (2008) are two recent books focussing on ABGs, although many texts on laboratory investigations (e.g. Higgins, 2007) usually contain useful chapters. Coggan (2008) provides a useful two-part introductory article.

ARTERIAL BLOOD GAS-EXAMPLES

pН	7.465
pCO ₂	6-21 kPa
pO ₂	7-98 kPa
SO ₂	94.7 %
HCO3	32-8 mmol/l
BE	9.0 mmol/L

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pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7-454 6-18 kPa 20-30 kPa 99-5% 31-7 mmol/L 7-8 mmol/L	pH pC pC SC HC BE
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7-083 16-22 kPa 7-44 kPa 80-7% 25-3 mmol/L 1-3 mmol/L	pł pC pC SC HC BE
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7-586 4-82 kPa 9-24 kPa 98-1 % 33-5 mmol/L 11-8 mmol/L	pl pC pC SC HC BE
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7·247 6·71 kPa 9·92 kPa 96·4% 21·4 mmol/L –5·9 mmol/L	RE Briti Cog
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7-435 5-44 kPa 6-76 kPa 88-6 % 26-4 mmol/L 2-4 mmol/L	Delli G F S Dep
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7-334 4-28 kPa 11-10 kPa 98-1% 16-6 mmol/L –9-2 mmol/L	Foxa Guy Hen
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7-358 7-82 kPa 12-88 kPa 99-9 % 32-1 mmol/L 6-8 mmol/L	Higg I Mar Surv C
pH pCO ₂ pO ₂ SO ₂ HCO ₃ BE	7.426 4.84 kPa 6.02 kPa 82.2% 23.6 mmol/L –0.6 mmol/L	van i F Win <i>I</i> Wor F
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pH	6.916
pCO ₂	5-49kPa
pO ₂	14-44 kPa
SO ₂	95.5%
HCO₃	8-2 mmol/L
BE	-24.5 mmol/
рН	7.333
pCO ₂	12-35 kPa
pO ₂	11-54 kPa
SO ₂	97-8 %
HCO3	48-1 mmol/L
BE	22-2 mmol/L
рН	6.992
pCO ₂	4-35 kPa
pO ₂	11.3 kPa
SO ₂	91.5%
HCO3	8.3 mmol/L
BE	–21.5 mmol/

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