

ACID BASE BALANCE AND INTERPRETATION OF BLOOD GAS RESULTS

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Introduction

Acid base disturbances are indicators of serious underlying pathology, rather than being the pathology themselves. Arterial blood gas examination is a useful investigation in patients with suspected respiratory or metabolic disease and serial blood gas investigation can monitor the progress or treatment of the underlying disease. The blood gas should be considered in conjunction with the patient's clinical condition. It does have a limitation because we only measure the extracellular fluid and do not know what the intracellular pH and gas tensions are.

Many clinicians find it difficult to interpret the blood gas results. This overview is written to give a basic understanding of the blood gas and a step-wise approach to its interpretation. The section on physics is to give a more complete understanding but you can gloss over it and go straight to the clinical significance.

Some physics

pH is the negative log of the H⁺ ion concentration.

When pH = 7, the H⁺ concentration is 10⁻⁷ or 1/10⁷

This is neutral because the H⁺ and OH⁻ concentration is the same.



When the pH = 1, the H⁺ concentration is 10⁻¹ or 1/10. This is a very strong acid.

- pH 7.00 = neutral
- pH >7 = alkaline
- pH <7 = acid
- pH 7.4 = physiological pH of extracellular fluid. (Range of normal 7.35 - 7.45.)

Because of the log function, a small change in the pH is a significant change in the H⁺ concentration. If the pH drops from 7.4 to 7.0, the acidity is 2^{1/2} times higher.

pH	H ⁺ concentration
7.0	1/10 000 000
7.1	1/12 589 254
7.2	1/15 848 931
7.3	1/19 952 623
7.4	1/25 118 864

Usually pH is measured directly by a special glass electrode that has a H⁺ permeable membrane.

HCO₃⁻ is measured by a bicarbonate electrode or may be calculated.

CO₂ is usually measured directly by a CO₂ electrode.

There are numerous physiological buffers that help prevent sudden swings in the intracellular pH (such as bicarbonate, lactate, phosphate, ammonia, haemoglobin, proteins and others). The bicarbonate system is used to regulate the whole-body pH because it is possible to regulate it at two different sites: HCO₃⁻ is regulated by the kidneys and CO₂ is regulated by the lungs.



The exact pH can be calculated from the Henderson Hasselbach equation

$$pH = pK + \log \frac{[base]}{[acid]}$$

$$= pK + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

pK is a constant for the specific buffer. (For the bicarbonate buffer system at 37°C it is 6.1)

Because HCO₃⁻ is controlled by the kidneys and CO₂ is controlled by the lungs, this equation becomes

$$pH = constant \frac{KIDNEY}{LUNG}$$

Abbreviations used in acid base notation

p	Negative log (lower case "p")
P	Partial pressure (upper case "P")
PA	Alveolar partial pressure (upper case "A")
Pa	Arterial partial pressure. (lower case "a")
Pv	Venous partial pressure

Notes about terminology: acidosis/acidaemia and alkalosis/alkalaemia

The suffix "-aemia" means "in the blood."

The overall acid base status of the blood is correctly referred to as an acidaemia or alkalaemia. This is taken from the pH alone and does not consider if the primary defect is metabolic or

respiratory and if there is compensation or not. The metabolic or respiratory components in the blood or any other body fluid have the suffix “-osis”. If there is (for example) a metabolic acidosis with incomplete respiratory compensation there will be with a low pH and therefore an acidaemia.

Clinical significance

The bicarbonate buffer system is the most important buffer system in the body and is the one measured with the blood gas. The lungs can adjust CO₂ exhalation, and the kidneys can adjust HCO₃⁻ excretion or retention, so the precise ratio of acid to base can be maintained and adjusted.

The respiratory system (CO₂) can make rapid adjustments within minutes.

The metabolic component (renal, bicarbonate) takes hours or even days to adjust.

These two systems work together to maintain a fine balance. They aim to keep the extracellular pH 7.4 as this is the optimal environment for most metabolic activity such as chemical reactions catalysed by enzymes and transport of substances across cell membranes.

Pathological processes such as tissue hypoxia, renal failure, hypoventilation will all disrupt the normal acid base balance. If there is an abnormality in one part of the system, the other part will attempt to compensate and correct the pH.

Acid base disturbances and some examples of how they may occur

Compensatory mechanisms will tend to restore the pH towards normal even though the [HCO₃⁻] and the PCO₂ are not restored until the primary disturbance is corrected. The compensatory mechanisms should not overshoot. For example a metabolic acidosis will drop the pH to <7.4. If there is respiratory compensation the pH will return towards normal but will not overshoot to become >7.4.

Tips about determining which is the primary defect and which is the compensatory effect.

The primary defect (metabolic or respiratory) will go in the same direction as the pH. That is towards an acidosis if the pH is low or towards an alkalosis if the pH is high.

The compensatory effect (respiratory or metabolic) will go in the opposite direction.

The compensation will bring the pH back towards normal but it will never overshoot and will seldom actually reach normal.

For example: if there seems to be a metabolic acidosis and a respiratory alkalosis, the pH tells you which one is primary and which one is compensatory. If the pH is low, the primary defect is metabolic acidosis with respiratory compensation. If the pH is high, the primary defect is respiratory alkalosis with metabolic compensation.

Further reading

1. Update in Anaesthesia No 13
2. Alan Grogono has produced a very good tutorial on acid base. It is at <http://www.acid-base.com/>

Acid base disturbances		
Respiratory acidosis	PaCO ₂ increased	This occurs when there is inadequate ventilation and CO ₂ production is greater than CO ₂ elimination. It may occur with airway obstruction, respiratory depression due to drugs or head injury, lung diseases, etc
Respiratory alkalosis	PaCO ₂ decreased	This occurs with hyperventilation. The hyperventilation may be in response to hypoxaemia and hypoxic respiratory drive. The lungs are more efficient at eliminating CO ₂ than at absorbing O ₂ so patients with diseased lungs frequently have hypoxaemia with a normal or low CO ₂ . Mechanical ventilation with a large minute volume also leads to respiratory alkalosis.
Metabolic acidosis	HCO ₃ ⁻ decreased (base deficit)	Multiple aetiologies <ul style="list-style-type: none"> ● Loss of bicarbonate due to GIT losses or chronic renal disease (Normal anion gap) ● Addition of inorganic acids such as diabetic ketoacidosis, lactic acidosis associated with tissue hypoxia, salicylate, ethylene glycol and other toxins, decreased acid excretion in renal failure (increased anion gap)
Metabolic alkalosis	HCO ₃ ⁻ increased (base excess)	Occurs with loss of gastric acid (e.g. pyloric stenosis) and diuretic therapy. Metabolic alkalosis is commonly associated with low serum chloride.
Mixed and respiratory acidosis	PaCO ₂ increased metabolic and HCO ₃ ⁻ decreased	This is very dangerous and may occur in severe diseases such as septic shock, multiple organ dysfunction, cardiac arrest

<i>Blood gas normal values</i> <i>* You should remember the numbers in bold</i>			
Item	Normal range*	Units	Notes
pH	7.35 - 7.4 - 7.45		(no units)
PCO ₂	4.8 - 5.3 - 5.9	kPa	
	36 - 40 - 44	mmHg	
PO ₂	11.9 - 13.2	kPa	at sea level, FiO ₂ = 21%
	90 - 100	mmHg	lower at high altitude, higher if supplemental oxygen
HCO ₃ (actual bicarbonate)	22 - 24 - 26	mmol/l	normal values vary if the PCO ₂ is abnormal
standard bicarbonate	22 - 24 - 26	mmol/l	the [HCO ₃ ⁻] after the sample has been equilibrated with CO ₂ at 40mmHg (5,3kPa)
base excess	-2, 0, +2	mmol/l	a negative number is a base deficit

<i>What do the different numbers mean?</i>	
pH	The total acidity or alkalinity of the sample. This indicates if the patient has an acidaemia or an alkalaemia.
PCO ₂	The respiratory component
PO ₂	Indicates the oxygenation status of the patient and must not be confused with the acid base status. In general it is an indicator of the severity of lung disease, but cannot really be interpreted without knowing the FiO ₂ . The PO ₂ could be up to 650mmHg (85kPa) if the lungs are normal and the FiO ₂ is 100%. The predicted PaO ₂ for normal lungs can be calculated from the alveolar gas equation (which I am not going to discuss) A rough approximation of the predicted PaO ₂ is percentage inspired O ₂ x 6mmHg. (eg a patient breathing 40% oxygen should have a PaO ₂ of 6 x 40 = 240mmHg. If it is less than that, it means there is a shunt. Blood is not passing a ventilated alveolus before getting to the aorta. The worse the lung disease, the lower the PaO ₂ will be at any given FiO ₂ .
HCO ₃ (Actual bicarbonate)	The renal component.
Standard Bicarbonate	Another measure of the renal (metabolic) component. More useful than the actual bicarbonate as it has been corrected for an abnormal PCO ₂
Base Excess	The amount of strong acid (or base if there is a base deficit) needed to titrate 1litre of blood back to pH 7,4 at PCO ₂ = 5,3kPa and Temperature = 37°C Another measure of the renal (metabolic) component. It gives the same information as the standard bicarbonate except that the normal value is 0 instead of 24.

<i>Stepwise interpretation of the blood gas</i>		
1	Is the overall picture normal, acidaemia, alkalaemia?	pH < 7.35 = acidaemia [...go to step 2] pH > 7.45 = alkalaemia [... go to step 5]
2	If there is an acidaemia: is the primary defect metabolic or respiratory or mixed?	CO ₂ high = respiratory acidosis [...3] Bicarbonate low or BE negative = metabolic acidosis. [...4] Both of the above = mixed metabolic and respiratory acidosis.
3	If there is respiratory acidosis: is there metabolic compensation?	The CO ₂ is high (resp acidosis) but the metabolic component is going in the opposite direction (BE or SB high, towards metabolic alkalosis) then there is metabolic compensation.
4	If there is metabolic acidosis: is there respiratory compensation?	BE is negative (metabolic acidosis) but the respiratory component is going in the opposite direction (CO ₂ low, towards resp alkalosis), then there is respiratory compensation.
5	If there is an alkalaemia, is the primary defect respiratory or metabolic?	The primary defect will go in the same direction as the pH (towards alkalosis): respiratory alkalosis will have low CO ₂ metabolic alkalosis will have high SB and positive BE.
6	If metabolic or respiratory alkalosis, is there any compensation by the other one?	Same principles as above
7	Look at the oxygenation	Is the PO ₂ consistent with the FiO ₂ ? If it is lower than expected, it either indicates lung disease, right to left shunt, or venous sample. (A venous sample usually has PO ₂ < 40mmHg, saturation < 75%). The lung is much more efficient at eliminating CO ₂ than absorbing oxygen so lung disease will show in the low PO ₂ but the PCO ₂ is often normal or even low. If the CO ₂ is very high, the O ₂ will also be low.
8	Summarise the interpretation	eg. There is a metabolic acidosis (because the pH is low and BE is negative) with respiratory compensation (because the PCO ₂ is low).
9	Try to establish the cause	