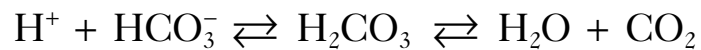


ACID-BASE DISORDERS

The kidneys, lungs, and physiologic buffers normally maintain the serum pH within a narrow spectrum, between 7.36 and 7.44. Such precise physiologic control is required for normal cellular function. Consequently, disorders of kidneys, lungs, and physiologic buffers result in acid-base abnormalities.

Blood pH is determined by the ratio of CO₂ (respiratory influence) to HCO₃⁻ (renal influence).



Acidaemia: pH < 7.36 *Alkaemia*: pH > 7.44

Acidosis is a pathological process that lowers the HCO₃⁻ or raises the CO₂

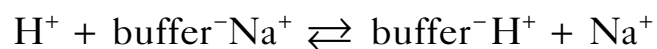
Alkalosis is a pathological process that raises the HCO₃⁻ or lowers the CO₂

Physiologic Buffers.

Defined as a weak acid and its salt, which oppose marked changes in pH.

Buffers mitigate the impact of large changes in available hydrogen ion on plasma pH. These buffers include;

- haemoglobin
- phosphate
- proteins
- bicarbonate



Bicarbonate.

- Open-ended system w/ continuous removal of organic acid made possible by exhalation of CO₂.
- Large quantities in *kidneys & lungs*.

Intracellular Proteins.

- Particularly, *albumin & Hb*.

Bone as Buffer.

- Large reservoir of *bicarbonate & phosphate*.
- In reality, only two types of metabolic acidosis are long-lasting enough to cause bone demineralisation
 - *Renal tubular acidosis*
 - *Uraemic Acidosis*

Pulmonary Compensation.

This involves a relationship between peripheral chemoreceptors (carotid bodies) and central chemoreceptors (medulla); both of which drive minute ventilation.

- A drop in pH results in increasing minute ventilation (lowering CO₂).
- Takes ~ 4-12 hours for respiratory compensation to bring pH back to normal.

Renal Compensation.

The kidneys play little role in acute compensation of acid-base disorders, as they do not immediately respond to changes in pH.

- Requires 6-12 hours of sustained acidosis to result in active excretion of H⁺.
- H⁺ usually excreted in form of ammonia (NH⁺), with retention of HCO₃⁻.
- Conversely, 6+ hours of alkalemia stimulates renal excretion of HCO₃⁻ and retention of H⁺ in form of organic acids.

In metabolic acidosis the kidney attempts to preserve Na⁺ by exchanging it for excreted H⁺ or K⁺.

- The quantity of potassium excreted depends on the level of acidosis & the serum K⁺ level.
- In the presence of large H⁺ load, hydrogen ions move from ECF into intracellular fluid, displacing K⁺ outside the cell (maintaining electroneutrality).

In cases of severe acidosis, there is significant overall depletion of total body K⁺ despite serum hyperkalemia.

In metabolic alkalosis, there is a shift of H⁺ extracellularly (in exchange for K⁺ and Na⁺) with renal excretion of K⁺ to preserve H⁺.

- Kidneys will also excrete H⁺ in cases of severe hypokalemia (eg. prolonged NG suctioning) to preserve K⁺.
- Paradoxical *aciduria* is a clinical clue to the magnitude of hypokalemia.

DIAGNOSTIC STRATEGIES.

Thorough Hx & examination:

- PMHx
- Current medications
- Toxic ingestion
- Vomiting / diarrhoea
- Hydration status
- Urine output
- Level of consciousness
- Respiratory rate

Bloods:

- Serum electrolytes
- pH
- Anion gap & delta gap
- Osmolar gap

THE ANION GAP.

$$AG = Na^+ - (Cl^- + HCO_3^-)$$

- Normal AG = 12 (± 3)
- Estimates 'unmeasured anions'
 - Albumin
 - Phosphate
 - Sulfate
 - Organic anions (citrate).
- If AG is raised; consider an excess in organic acids or other acidic substances.

THE DELTA GAP.

$$\Delta G = (\text{calculated AG} - 12) - (24 - \text{measured HCO}_3^-)$$

- Helps to resolve the possibility of;
 - Mixed acid-base disorder
 - Further differentiation of an elevated AG metabolic acidosis.
- $\Delta G > +6$ = metabolic alkalosis or respiratory acidosis
- $\Delta G < -6$ = a mixed disorder (greater loss of HCO_3^-).

Arterial Blood Gas Analysis

ABG Parameter			ABG result	Calculation and interpretation		
pH	>7.45	Alkalaemia		pH	pCO₂	Interpretation
	7.36-44	Normal				
	<7.35	Acidaemia		↓	↓	Metabolic acidosis
pCO₂	>45	High		↑	↑	Metabolic alkalosis
	35-45	Normal		↑	↓	Respiratory alkalosis
	<35	Low		↓	↑	Respiratory acidosis
HCO₃	>26	High		Corrected standard AG for albumin		
	24+/- 2	Normal		$\frac{\text{Albumin} + 1.5 \times \text{Phosphate}}{4}$		
	<22	Low				
AG	> 16	High		Anion Gap calculation		
	12+/-4	Normal		$\{[\text{Na}^+] - [\text{Cl}^- + \text{HCO}_3^-]\} = 12\pm 4$		
	< 8	Low		Corrected Na⁺ for AG in hyperglycemia		
Glucose	>10	High		$\text{Corrected Na}^+ = \text{Na} + \frac{\text{Glucose} - 5}{3}$		
	< 2	Low				
Gap: Gap	$\frac{\Delta \text{AG}}{\Delta \text{HCO}_3} = \frac{\text{AG} - 12}{24 - \text{HCO}_3}$			Gap: Gap calculation for metabolic acidosis		
				<0.4	Low or Normal AG metabolic acidosis	
				0.4-0.8	Normal + high AG metabolic acidosis	
Lactate	<1.9	Normal		0.8-2.0	Pure high metabolic acidosis	
	>2.0	High		>2.0	Metabolic acidosis with metabolic alkalosis/respiratory acidosis	
pO₂	80-100	Normal		PAO₂ = [713 x FiO₂] - [pCO₂ x 1.25]		
	< 80	Hypoxia		A-a gradient = PAO₂ - PaO₂ = $\frac{\text{Age} + 4}{4}$		
Compensation rules for						
Expected PCO₂	Metabolic acidosis			Metabolic alkalosis		
	$1.5 \times [\text{HCO}_3] + 8 \quad (+/- 2)$			$0.7 \times [\text{HCO}_3] + 20 \quad (+/- 5)$		
Expected HCO₃	Respiratory acidosis			Respiratory alkalosis		
	Acute	Chronic		Acute	Chronic	
	$24 + \frac{\text{pCO}_2 - 40}{10} \times 1$	$24 + \frac{\text{pCO}_2 - 40}{10} \times 4$		$24 - \frac{40 - \text{pCO}_2}{10} \times 2$	$24 - \frac{40 - \text{pCO}_2}{10} \times 5$	