MIXED ACID-BASE DISORDERS

Double and triple primary acid-base disturbances are common...

Clues to the presence of a mixed acid-base disturbance can either be historical (e.g., polydrug ingestion) or clinical, with varied chemistry and arterial blood gas findings that differ from those anticipated.

<u>STEP 1.</u>

Measure the pH.

- pH < 7.36 = acidaemic
- pH > 7.44 = alkaemic

<u>STEP 2.</u>

Is the primary disturbance respiratory vs metabolic ?

pН	pCO2	Interpretation
Ļ	4	Metabolic acidosis
1	Î	Metabolic alkalosis
1	4	Respiratory alkalosis
Ļ	1	Respiratory acidosis

<u>STEP 3.</u>

Calculate the Anion Gap.

• Normal AG = $12 (\pm 3)$

 $AG = Na^{+} - (Cl^{-} + HCO_{3}^{-})$

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<u>STEP 4.</u>

Calculate the Delta Gap.

- $\Delta G = (\text{calculated AG} 12) (24 \text{measured HCO}_3^-)$
- Normal = 0 ± 6
- Positive (>6) = HAGMA + primary metabolic alkalosis
 - eg. DKA w/ severe vomiting or Lactic acidosis w/ chronic diuretic use.
- Negative (< -6) = varied... either
 - mixed HAGMA & NAGMA OR
 - HAGMA w/ chronic respiratory alkalosis & compensating hyperchloraemic acidosis.
 - Often have profound metabolic disease (hypermagnesaemia, hyponatraemia or hypercalcaemia).

<u>STEP 5.</u>

Is the respiratory disturbance acute vs chronic ?

- If acute; for each 10mmHg PaCO₂,
 - pH drops by 0.08
 - HCO3 increases by 1 mmol/L
- If chronic; for each 10mmHg PaCO₂,
 - pH drops by 0.03.
 - HCO3- increases by 4 mmol/L

<u>STEP 6.</u>

Has the respiratory system compensated fully ?

Use Winter's Formula.

BOX 122-6	RELATIONSHIPS IN ACID-BASE DISTURBANCES	
Respiratory Acidosis Acute		
 HCO₃⁻ increases 1 mEq/L (range, 0.25–1.75) for every 10-mm Hg increase in PcO₂. pH drops 0.08 for every 10-mEq/L rise in HCO₃⁻. 		
 Chronic (>5 days of hypercapnia) HCO₃⁻ increases 4 mEq/L for every 10-mm Hg increase in PCO₂ (±4). Limit of compensation: bicarbonate will rarely exceed 38–45 mEq/L. 		
Metabolic A Note: It marries points Paco ₂ = (1) Paco ₂ is eq 20, pH s $\Delta PCO_2 - 1$ For pure A to the fa For pure r HCO ₃ - s ΔHCO_3 Limit of co 10-15 r	cidosis ay take 12–24 hours for maximal respiratory se to develop. $.5 \times \text{HCO}_3$) + 8 ± 2. quivalent to last two digits of pH (e.g., if Pco ₂ is should be 7.20). $[1.3 \times (\Delta \text{HCO}_3^-)]$ AG acidosis, the rise in anion gap should be equal all in [HCO ₃ ⁻] (i.e., ΔG should equal 0). non-AG (hyperchloremic) acidosis, the fall in hould be equal to the rise in [Cl ⁻] (i.e., $= -\Delta \text{Cl}^-$). pompensation: PacO ₂ will not fall below nm Hg.	
Respiratory Alkalosis Acute HCO ₃ ⁻ drops 1 to 3.5 mEq/L for every 10-mm Hg drop in PCO ₂ . Limit of compensation: bicarbonate is rarely below 18 mEq/L.		
Chronic (renal compensation starts within 6 hours and is usually at a steady state by 1½-2 days) HCO ₃ ⁻ drops 2–5 mEq/L for every 10-mm Hg drop in PcO ₂ . Limit of compensation: bicarbonate is rarely below 12–14 mEq/L.		
Metabolic A $Pco_2 = 0.9$ Pco_2 incre HCO_3^- . Limit of co has been	Ikalosis (HCO ₃ ⁻) + 9. ases 0.6 mm Hg for each 1-mEq/L increase in ompensation: PCO ₂ rarely exceeds 55 mm Hg but en reported as high as 75.	

 $PCO_2 = (1.5 \times [HCO_3^-] + 8) \pm 2$