

# Approach to Arterial Blood Gases (ABGs)

1. Adequate oxygenation?

Normal ABG on Room Air = 7.40 / 40 / 90 / 24

Normal  $\text{pCO}_2$  should be  $\text{pCO}_2 \approx 4-5$  ( $\text{pCO}_2$  in room air = 30)

$\text{pCO}_2 < 5$ , then expected

Possible hypoxemia

2. Define the acid-base disturbance

$\text{pH} < 7.40 =$  Acidosis

$\text{pH} > 7.40 =$  Alkalosis

See slide on  
Hypoxemia, Pathogenesis and Clinical Findings

3. Identify the primary ( $\Delta^+$ ) process

4. Compensation mechanisms in lungs and kidneys attempt to keep/retain  $\text{CO}_2$  or  $\text{HCO}_3^-$  to maintain normal pH. Determination of compensation is appropriate (appropriate normal ratio of change in  $\text{pCO}_2$  :  $\text{HCO}_3^-$  is shown in green boxes). If inappropriate, there is a secondary ( $\Delta^-$ ) process accounting

$\Delta^+ \text{ HCO}_3^-$

$\Delta^+ \text{ HCO}_3^-$

$\Delta^- \text{ HCO}_3^-$

5. Calculate anion gap (AG) to determine the presence and type of metabolic acidosis. This is done regardless of the  $\Delta^+$  or  $\Delta^-$  process, as there may also be a hidden metabolic acidosis

Calculate  $\text{AG} = \text{Na}^+ - \text{Cl}^- - \text{HCO}_3^-$  (normal is  $\approx 12$ )

Classically, the compensating anion is  $\text{Cl}^-$ , maintaining a normal AG

If a non-bicarbonate anion is present,  $\Delta^- \text{ HCO}_3^-$  is compensated by  $\Delta^+$  other serum anions to maintain neutral charge

Where the cause is addition of an abnormal acid,  $\text{HCO}_3^-$  is used up as a buffer, and the compensating anion is the abnormal acid's conjugate base – which is not measured in the AG calculation

$\Delta^- \text{ HCO}_3^-$  is compensated by unmeasured anions ( $\Delta^- \text{ the AG} - \text{f}$ )

6. HAGMA: refer to normal blood buffer system, acid gain should match bicarbonate loss. If not, identify if another process is causing an inappropriate loss or gain of  $\text{HCO}_3^-$ .  
 $\text{Gain of acid (AG)} = \text{AG} - 12$   
 $\text{Loss of } \text{HCO}_3^- (\text{AHCO}_3^-) = 24 - \text{HCO}_3^-$

$\text{AG} = 12$

$\text{AG} > 12$

7. Calculate Cation Gap (Net阳离子) or Urine net charge (Net陽離子) to narrow the etiology

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If no metabolic acidosis was identified in steps 2-4:

Normal Anion Gap Metabolic Acidosis (NAGMA)

In this scenario, a normal renal response should be decreasing excess acid

Excess acid is normally excreted as ammonium ( $\text{NH}_4^+$ ) with  $\text{Cl}^-$

The more  $\text{Cl}^-$  in the urine relative to cations, the more acid is being excreted

Calculate urine ( $\text{U}$ ) net charge to determine cause of NAGMA:  $\text{U}_{\text{Cl}^-} - \text{U}_{\text{NH}_4^+} - \text{U}_{\text{K}^+}$

Net  $\text{U}$  net charge

Appropriately large  $\text{Cl}^-$  excretion ( $\Delta^- \text{ HCO}_3^-$  loss is the issue)

AG losses or Type II RBA

Net  $\text{U}$  net charge

Inappropriately small  $\text{Cl}^-$  excretion ( $\Delta^+ \text{ HCO}_3^-$  gain is the issue)

Type II or Type I RBA

$\text{AG} < 12$

High Anion Gap Metabolic Acidosis (HAGMA)

Calculate  $\text{AG} = \text{AG} - 12$  Calculate  $\text{AHCO}_3^- = 24 - \text{HCO}_3^-$

$\Delta \text{HCO}_3^- = \Delta \text{AG}$

Loss of  $\text{HCO}_3^- > \text{Gain of acid}$

Additional process causing further loss of  $\text{HCO}_3^-$

$\text{HAGMA} + \text{NAGMA}$

$\text{HAGMA} + \text{HAGMA netc}$

Toxic alcohol ingestion ( $\Delta^- \text{ serum bicarbonate}$  and  $\text{H}^+$  from metabolites produced, so bicarbonate helps identify etiology)

Calculate anion gap to determine cause of HAGMA,  $= (\text{measured serum cation} - (\text{expected serum cation} + (\text{measured serum anion} - 12.5 \text{mEq}) + \text{U}_{\text{Na}^+} + \text{serum glucose}))$

Urine  $\text{Cl}^- > 10$  (extra cations present)

Toxic alcohol poisoning

Urine  $\text{Cl}^- < 10$  (extra anions present)

Test for other causes

See slide on Normal Anion Gap Metabolic Acidosis: Pathogenesis and Laboratory Findings

See slide on High Anion Gap Metabolic Acidosis: Pathogenesis and Laboratory Findings

# Abmg Study Guide

**Michael A. Paré**

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