Acid-Base Disorders

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General Acid-Base Relationships

Henderson-Hasselbach equation:

\[ \text{pH} = \text{pK} + \log \frac{\text{HCO}_3^-}{\text{pCO}_2} \]

\[ \text{H}^+ = 24 \times \frac{\text{pCO}_2}{\text{HCO}_3^-} \]

\[ \Delta \ 0.1 \text{ pH unit} = \Delta \ 10 \text{ nm/L H}^+ \]
Approach to Acid-Base Disorders

1. Consider the clinical setting!
2. Is the patient acidemic or alkalemic?
3. Is the primary process metabolic or respiratory?
4. If metabolic acidosis, gap or non-gap?
5. Is compensation appropriate?
6. Is more than one disorder present?
### Simple Acid-Base Disorders

<table>
<thead>
<tr>
<th>Condition</th>
<th>pH</th>
<th>Primary Disorder</th>
<th>Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>↓ pH</td>
<td>↓ HCO3</td>
<td>↓ pCO2</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑ pH</td>
<td>↑ HCO3</td>
<td>↑ pCO2</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>↓pH</td>
<td>↑ pCO2</td>
<td>↑ HCO3</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>↑ pH</td>
<td>↓ pCO2</td>
<td>↓ HCO3</td>
</tr>
</tbody>
</table>
### Expected Compensatory Responses

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Compensation</th>
<th>Limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Acidosis</td>
<td>Expected $pCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$</td>
<td>$pCO_2$ cannot go &lt; 10 mmHg</td>
</tr>
<tr>
<td></td>
<td>Expected $pCO_2$ = last 2 digits of pH</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$\Delta \ pCO_2 = 1.2 \times \Delta HCO_3^-$</td>
<td></td>
</tr>
<tr>
<td>Metabolic Alkalosis</td>
<td>$\Delta \ pCO_2 = 0.7 \times \Delta HCO_3^-$</td>
<td>$pCO_2$ cannot go &gt; 55 mmHg</td>
</tr>
<tr>
<td></td>
<td>$HCO_3^- + 15 = pCO_2$ = last two digits of pH</td>
<td></td>
</tr>
<tr>
<td>Respiratory Acidosis</td>
<td>Acute: $\Delta HCO_3^- = 0.2 \times \Delta pCO_2$</td>
<td>$HCO_3^-$ cannot go &gt; 30 mmHg</td>
</tr>
<tr>
<td></td>
<td>Chronic: $\Delta HCO_3^- = 0.4 \times \Delta pCO_2$</td>
<td>$HCO_3^-$ cannot go &gt; 45 mmHg</td>
</tr>
<tr>
<td>Respiratory Alkalosis</td>
<td>Acute: $\Delta HCO_3^- = 0.2 \times pCO_2$</td>
<td>$HCO_3^-$ cannot go &lt; 17-18 mmHg</td>
</tr>
<tr>
<td></td>
<td>Chronic: $\Delta HCO_3^- = 0.5 \times pCO_2$</td>
<td>$HCO_3^-$ cannot go &lt; 12-15 mmHg</td>
</tr>
</tbody>
</table>
Metabolic Acidosis

• **Etiology**: Inability of the kidney to excrete the dietary H\(^+\) load, or increase in the generation of H\(^+\) (due to addition of H\(^+\) or loss of HCO\(_3^-\))
Metabolic Acidosis: Elevated Anion Gap

AG = Na\(^+\) - (Cl\(^-\) + HCO\(_3^-\)) = 12 ± 2

[Note: Diagnostic utility is best when AG > 25]
## Causes of AG Metabolic Acidosis [Classic]

**CAT MUDPILERS**

<table>
<thead>
<tr>
<th>Letter</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>C</td>
<td>cyanide, carbon monoxide, CPK (rhabdo)</td>
</tr>
<tr>
<td>A</td>
<td>alcoholic ketoacidosis</td>
</tr>
<tr>
<td>T</td>
<td>toluene</td>
</tr>
<tr>
<td>M</td>
<td>methanol</td>
</tr>
<tr>
<td>U</td>
<td>uremia</td>
</tr>
<tr>
<td>D</td>
<td>DKA/alcoholic KA</td>
</tr>
<tr>
<td>P</td>
<td>paraldehyde, phenformin</td>
</tr>
<tr>
<td>I</td>
<td>INH, iron</td>
</tr>
<tr>
<td>L</td>
<td>lactic acidosis</td>
</tr>
<tr>
<td>E</td>
<td>ethylene glycol</td>
</tr>
<tr>
<td>R</td>
<td>rhabdo, renal failure</td>
</tr>
<tr>
<td>S</td>
<td>salicylates</td>
</tr>
</tbody>
</table>
Causes of AG Metabolic Acidosis [Updated]

GOLDMARK

G – glycols (ethylene, propylene, diethylene)
O – oxoproline
L – L-lactate
D – D-lactate
M – methanol
A – aspirin
R – renal failure
K – ketoacidosis
Intoxications Causing High AG Acidosis

- Aspirin - [high salicylate level; also primary respiratory alkalosis]
- Methanol - [optic papillitis]
- Ethylene Glycol - [calcium oxalate crystals]
- Paraldehyde
Anion Gap in Hypoalbuminemia

- The true anion gap is underestimated in hypoalbuminemia (= fall in unmeasured anions); AG must be adjusted
- Formulas for adjusted AG:
  - For every 1.0 fall in albumin, ↑ AG by 2.5
  - Consider the patient’s “normal” AG to be (2 x alb) + (0.5 x phosphate)
  - Adjusted AG = Observed AG + (2.5 x [normal alb - adjusted alb])
The Delta/Delta: $\Delta \ AG/ \ \Delta \ HC0_3$

**Rationale:**
For each unit **INCREASE** in AG (above normal),
HC0₃ should **DECREASE** one unit (below normal)

“Normal” values: AG = 12, HC0₃ = 24
## Use of the Delta/Delta: Examples

<table>
<thead>
<tr>
<th>AG</th>
<th>HCO3</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>18 (↑ 6)</td>
<td>18 (↓ 6)</td>
<td>Appropriate; pure AG acidosis</td>
</tr>
<tr>
<td>18 (↑ 6)</td>
<td>22 (↓ 2)</td>
<td>HCO₃ has ↓ less than predicted, so HCO₃ is too high; mixed AG acidosis AND met alk</td>
</tr>
<tr>
<td>18 (↑ 6)</td>
<td>12 (↓ 12)</td>
<td>HCO₃ has ↓ more than predicted, so HCO₃ is too low; mixed AG AND non-AG acidosis</td>
</tr>
</tbody>
</table>
Causes of Low Anion Gap

**Etiology:** Fall in unmeasured anions or rise in unmeasured cations

- Hyperkalemia
- Lithium intoxication
- Hypercalcemia
- Hypermagnesemia
- Bromide (dextromethorphan, ipratropium, pyridostigmine)
- Monoclonal gammopathies [esp. IgG]
Osmolar Gap

Measured serum osmolality >

calculated serum osmolality by > 10 mOsm

Calc Sosm = (2 x Na) + BUN/2.8 + Glu/18
Causes of High Osmolar Gap

- Isotonic hyponatremia
- Hyperlipidemia
- Hyperproteinemia
- Mannitol
- Glycine infusion
- Chronic kidney disease
- Ingestions
  - Ethanol, isopropyl alcohol, ethylene glycol, mannitol
- Contrast Media
## Relationship between AG and Osmolar Gap

<table>
<thead>
<tr>
<th></th>
<th>AG</th>
<th>Osm gap</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethylene glycol</td>
<td>+</td>
<td>+</td>
<td>* Double gap</td>
</tr>
<tr>
<td>Methanol</td>
<td>+</td>
<td>+</td>
<td>* Double gap</td>
</tr>
<tr>
<td>Renal failure</td>
<td>+</td>
<td>+</td>
<td>* Double gap</td>
</tr>
<tr>
<td>Isopropyl alcohol</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Ethanol</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Lipids, proteins</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>
## Causes of Normal AG (Hyperchloremic) Metabolic Acidosis

<table>
<thead>
<tr>
<th>High $K^+$</th>
<th>Low $K^+$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal insufficiency</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>Interstitial nephritis</td>
<td>RTA</td>
</tr>
<tr>
<td>$\text{NH}_4\text{Cl}, \text{Arg HCl}$</td>
<td>Ureteral diversion</td>
</tr>
</tbody>
</table>
Causes of Normal AG (Hyperchloremic) Metabolic Acidosis

HARDUAPS

Hyperalimentation
Acetazolamide, amphotericin
RTA
Diarrhea; overcorrected or early DKA
Ureteral diversion
Pancreatic fistula, posthypocapnia
Spironolactone/saline resuscitation
Use of the Urine Anion Gap (UAG) in Normal AG Acidosis
Batlle DC, et al. NEJM 318:594, 1988

Urine AG = (Na + K) - Cl

Negative UAG = Normal, or GI loss of HCO₃

Positive UAG = Altered distal renal acidification

Caveats: Less accurate in patients with volume depletion (low urinary Na); and in patients with increased excretion of unmeasured anions (e.g. ketoacidosis), where there is increased excretion of Na and K to maintain electroneutrality)
Use of the Urinary AG in Normal Gap Acidosis
Batlle DC, et al.  NEJM 318:594, 1988

<table>
<thead>
<tr>
<th>Plasma K</th>
<th>UAG</th>
<th>U pH</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>-</td>
<td>&lt; 5.5</td>
<td>Normal</td>
</tr>
<tr>
<td>Elevated</td>
<td>+</td>
<td>&lt; 5.5</td>
<td>Aldo deficiency</td>
</tr>
<tr>
<td>Elevated</td>
<td>+</td>
<td>&gt; 5.5</td>
<td>Distal RTA</td>
</tr>
<tr>
<td>Normal-low</td>
<td>+</td>
<td>&gt; 5.5</td>
<td>Classic RTA</td>
</tr>
<tr>
<td>Normal-low</td>
<td>-</td>
<td>&gt; 5.5</td>
<td>GI HCO$_3$ loss</td>
</tr>
</tbody>
</table>
Use of the Urine Osmolal Gap

- When UAG is positive, and it is unclear if increased cation excretion is responsible, urine NH$_4$ concentration can be estimated from urine osmolal gap
- Calc Uosm = (2 x [Na+K]) + urea nitrogen/2.8 + glu/18
- The gap between the calculated and measured Uosm = mostly ammonium
- In patients with metabolic acidosis, urine ammonium should be > 20 mEq/L. Lower value = impaired renal acidification
Calculation of Bicarbonate Deficit

• The bicarbonate “space” differs according to the clinical setting – look it up!
• If metabolic acidosis warrants Rx, bicarb deficit may be hundreds of mEq; need continuous infusion, not random amps
• In metabolic acidosis

Bicarb deficit = $HCO_3^-$ space x $HCO_3^-$ deficit/liter
$HCO_3^-$ space = 0.4 x lean body wt (kg)
$HCO_3^-$ deficit/liter = [desired $HCO_3^-$] - [measured $HCO_3^-$]
Approach to Metabolic Acidosis

- **Osmolar Gap**
  - Normal
  - Increased
    - Uremia
    - Lactate
    - Ketoacids
    - Salicylate
    - Ethylene glycol
    - Methanol

- **Anion Gap**
  - High
  - Normal
    - GI Fluid Loss?
      - Yes
        - Diarrhea
        - Ileostomy
        - Enteric fistula
      - No
        - Urine pH
          - > 5.5
          - < 5.5
            - Serum K
              - Low
              - High
                - Type 4 RTA
        - Proximal RTA (Type 2)
  - Normal
    - No
      - Do UAG
      - Distal RTA (Type 1)
Metabolic Alkalosis

**Etiology:** Requires both generation of metabolic alkalosis (loss of $H^+$ through GI tract or kidneys) and maintenance of alkalosis (impairment in renal $HCO_3^-$ excretion)

**Causes of metabolic alkalosis**
- Loss of hydrogen
- Retention of bicarbonate
- Contraction alkalosis

**Maintenance factors:** ↓ in GFR, ↑ in $HCO_3^-$ reabsorption
Metabolic Alkalosis

**CLEVER PD**

- Contraction
- Licorice
- Endo: Conn’s, Cushing’s, Bartter’s
- Vomiting
- Excess Alkali
- Refeeding alkalosis
- Post-hypercapnia
- Diuretics
## Use of Urine Cl⁻ in Metabolic Alkalosis

<table>
<thead>
<tr>
<th>Chloride –responsive</th>
<th>Chloride-resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td>(U(_{\text{Cl}}) &lt; 15 mEq/L)</td>
<td>U(_{\text{Cl}}) &gt; 20 mEq/L)</td>
</tr>
</tbody>
</table>

- GI loss
- Renal loss
- Low Cl intake
- Exogenous alkali

- With urine K < 15
- Laxative abuse, K depletion
- With urine K > 20
- Hypotensive: Bartter’s
- Hypertensive: hyperaldo, Cushing’s, Liddles, other
Use of Spot Urine Cl and K

**Urine Chloride**
- Very Low (< 10 mEq/L)
  - Vomiting, NG suction
  - Postdiuretic, posthypercapneic
  - Villous adenoma, congenital chloridorrhea, post- alkali

- > 20 mEq/L

**Urine Potassium**
- Low (< 20 mEq/L)
  - Laxative abuse
  - Other profound K depletion

- > 30 mEq/L

- Diuretic phase of diuretic Rx, Bartter’s, Gitelman’s, primary aldo, Cushings, Liddle’s, secondary aldosteronism
Treatment of Metabolic Alkalosis

1. Remove offending culprits.
2. Chloride (saline) responsive alkalosis: Replete volume with NaCl.
3. Chloride non-responsive (saline resistant) alkalosis:
   - Acetazolamide (CA inhibitor)
   - Hydrochloric acid infusion
   - Correct hypokalemia if present
Calculation of Bicarbonate Excess

Bicarb excess = $\text{HCO}_3^-$ space $\times$ $\text{HCO}_3^-$ excess/liter

$\text{HCO}_3^-$ space = 0.5 x lean body wt (kg)

$\text{HCO}_3^-$ excess/liter = [measured $\text{HCO}_3^-$] - [desired $\text{HCO}_3^-$]
Respiratory Disorders

- Result from abnormal hypoventilation (acidosis) or hyper-ventilation (alkalosis)
- Can be due to either CNS, pulmonary, or thoraco-abdominal disorders
Respiratory Acidosis

Causes of Respiratory Acidosis
- Inhibition of medullary respiratory center (e.g. drugs)
- Disorders of respiratory muscles and chest wall
- Upper airway obstruction
- Disorders affecting gas exchange across pulmonary capillaries
- Mechanical ventilation

Treatment of Respiratory Acidosis
- Rx the primary disorder; mechanical ventilation
Respiratory Alkalosis

Causes of Respiratory Alkalosis
- Hypoxemia
- Pulmonary disease
- Stimulation of medullary respiratory center
- Mechanical ventilation

CHAMPS
- CNS Disease
- Anxiety
- Hypoxia
- Mech vent
- Progesterone
- Salicylates/sepsis

Treatment of Respiratory Alkalosis
- Rx the primary disorder; mechanical ventilation; paper bag
Mixed Acid-Base Disorders: Clues

- Degree of compensation for primary disorder is inappropriate
- \( \text{Delta AG/delta HCO}_3^- \) = too high or too low
- Clinical history
## Common Clinical States and Associated Acid-Base Disturbances

<table>
<thead>
<tr>
<th>Clinical State</th>
<th>Acid-Base Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary Embolus</td>
<td>Respiratory Alkalosis</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Metabolic Acidosis</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Metabolic Alkalosis</td>
</tr>
<tr>
<td>Severe Diarrhea</td>
<td>Metabolic Acidosis</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>Respiratory Alkalosis</td>
</tr>
<tr>
<td>Renal Failure</td>
<td>Metabolic Acidosis</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Respiratory Alkalosis/Metabolic Acidosis</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Respiratory Alkalosis</td>
</tr>
<tr>
<td>Diuretic Use</td>
<td>Metabolic Alkalosis</td>
</tr>
<tr>
<td>COPD</td>
<td>Respiratory Acidosis</td>
</tr>
</tbody>
</table>
## Acid-Base Disorders in GI Disease


<table>
<thead>
<tr>
<th>GI Disorder</th>
<th>Acid-Base Disorder</th>
<th>Potassium</th>
<th>ECFV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vomiting, NG suction</td>
<td>Metabolic alkalosis</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Diarrheal states</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholera, infections</td>
<td>Metabolic acidosis</td>
<td>Low</td>
<td>Very low</td>
</tr>
<tr>
<td>Autoimmune</td>
<td>None</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Congenital achloridorrhea</td>
<td>Metabolic acidosis</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Villous adenoma</td>
<td>Variable</td>
<td>Normal-low</td>
<td>Normal-low</td>
</tr>
<tr>
<td>Laxative abuse</td>
<td>None unless severe</td>
<td>Low</td>
<td>Normal-low</td>
</tr>
<tr>
<td>Panc/biliary drainage</td>
<td>Metabolic acidosis</td>
<td>Normal-high</td>
<td>Low</td>
</tr>
<tr>
<td>Ileostomy drainage</td>
<td>Metabolic acidosis, metabolic alkalosis</td>
<td>High Normal</td>
<td>Low Low</td>
</tr>
<tr>
<td>Short bowel</td>
<td>Metabolic acidosis (D-lactic acidosis)</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>
# Acid-Base Disorders with Antibiotic Therapy


<table>
<thead>
<tr>
<th>Drug</th>
<th>Acid-Base Disorder</th>
<th>Mechanism</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin</td>
<td>Anion gap acidosis</td>
<td>Pyroglutamate</td>
<td>Rare</td>
</tr>
<tr>
<td>Linezolid</td>
<td>Anion gap acidosis</td>
<td>Mitochondrial toxicity</td>
<td>Rare</td>
</tr>
<tr>
<td>Most antibiotics</td>
<td>Anion gap acidosis (D-lactic acidosis)</td>
<td>Bacterial overgrowth</td>
<td>Rare</td>
</tr>
<tr>
<td>Tetracyclines,</td>
<td>Non-gap acidosis</td>
<td>Fanconi syndrome</td>
<td>Rare</td>
</tr>
<tr>
<td>aminoglycosides</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>Non-gap acidosis</td>
<td>Blocks eNAC</td>
<td>Frequent</td>
</tr>
<tr>
<td>Ampotericin B</td>
<td>Non-gap acidosis</td>
<td>Proton leak</td>
<td>Frequent</td>
</tr>
<tr>
<td>Aminoglycosides</td>
<td>Metabolic alkalosis</td>
<td>Bartter-like</td>
<td>Rare</td>
</tr>
<tr>
<td>Capreomycin</td>
<td>Metabolic alkalosis</td>
<td>Bartter-like</td>
<td>Rare</td>
</tr>
</tbody>
</table>
Acid-Base Disorders in Liver Disease

<table>
<thead>
<tr>
<th>Acid-Base Disorder</th>
<th>Mechanisms</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anion gap metabolic acidosis</td>
<td>Type B lactic (compensated), Type A lactic (not compensated)</td>
<td>10-20%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30-40%</td>
</tr>
<tr>
<td>Non-gap metabolic acidosis</td>
<td>Diarrhea (lactulose); distal RTA; Wilson’s disease; PBC</td>
<td>Variable</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>Hypoxemia; progesterone</td>
<td>Most common</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Volume contraction from diuretics</td>
<td>Variable</td>
</tr>
</tbody>
</table>
References


4. Fenves AZ, et al. Increased anion gap metabolic acidosis as a result of oxoproline (proglutamic acid): a role for acetaminophen. CJASN 1:441, 2006


Problems 1-7
A 30-yo man with DM presents with a week of polyuria, polydipsia, fever to 102, nausea, and abdominal pain. He is orthostatic on admission.

<table>
<thead>
<tr>
<th>130</th>
<th>94</th>
<th>75</th>
<th>906</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.1</td>
<td>6</td>
<td>2.3</td>
<td></td>
</tr>
</tbody>
</table>

pH 7.14
pCO₂ 18
pO₂ 102
1. Anticipate the disorder
   DKA (with anion gap acidosis)
2. Acidemic or alkalemic?
3. Metabolic or respiratory?
   pH = acidemic; must be metabolic (low HCO₃, low pCO₂)
4. If metabolic acidosis: gap or non-gap?
   AG = 30; + anion gap metabolic acidosis
5. Is compensation appropriate?
   pCO₂ should = last 2 digits of pH [18] or (1.5 x HCO₃) + 8 [17]
6. Mixed disorder?
   AG = 30 (18); HCO₃ = 6 (∫18); thus simple AG met acidosis
Problem 2

A 30-yo man with DM presents with a week of polyuria, polydipsia, fever to 102, and vomiting for four days.

<table>
<thead>
<tr>
<th>135</th>
<th>89</th>
<th>50</th>
<th>1181</th>
<th>pH 7.26</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.1</td>
<td>10</td>
<td>2.3</td>
<td></td>
<td>pCO₂ 23</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>pO₂ 88</td>
</tr>
</tbody>
</table>
1. Anticipate the disorder
   DKA (AG acidosis); met alk from vomiting
2. Acidemic or alkalemic? 3. Metabolic or respiratory?
   pH = acidemic; must be metabolic (low HCO₃, low pCO₂)
4. If metabolic acidosis: gap or non-gap?
   AG = 36; + anion gap metabolic acidosis
5. Is compensation appropriate?
   pCO₂ should = last 2 digits of pH [26] or (1.5 x HCO₃) + 8 [23]
6. Mixed disorder?
   AG = 36 (24); HCO₃ = 10 (↓ 14); HCO₃ is too high; mixed AG metabolic acidosis and metabolic alkalosis
Problem 3

A 30-yo man with DM presents with a week of polyuria, polydipsia, fever to 102, and diarrhea.

<table>
<thead>
<tr>
<th>pH</th>
<th>pCO₂</th>
<th>pO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.26</td>
<td>23</td>
<td>88</td>
</tr>
</tbody>
</table>

138 | 111 | 49 | 650
1. Anticipate the disorder
   DKA (AG acidosis); nongap met acidosis from diarrhea
2. Acidemic or alkalemic? 3. Metabolic or respiratory?
   pH = acidemic; must be metabolic (low HCO₃, low pCO₂)
4. If metabolic acidosis: gap or non-gap?
   AG = 19; + anion gap metabolic acidosis
5. Is compensation appropriate?
   pCO₂ should = last 2 digits of pH [26] or (1.5 x HCO₃) + 8 [23]
6. Mixed disorder?
   AG = 19 (7); HCO₃ = 8 (↓ 16); HCO₃ is too low; mixed AG metabolic acidosis and metabolic acidosis (nongap)
Problem 4

A 30-yo man with DM presents with a week of polyuria, polydipsia, fever, cough, and prurulent sputum.

| 140 | 104 | 75 | 1008 | pH 6.95 |
| 7.0 | 7   | 2.6 |      | pCO₂ 33 |
|      |      |     |      | pO₂ 60 |
1. Anticipate the disorder
   DKA (AG acidosis); resp alk or resp acidosis from hypoxemia/pneumonia
2. Acidemic or alkalemic?
3. Metabolic or respiratory?
   pH = acidemic; must be metabolic (low HCO₃, low pCO₂)
4. If metabolic acidosis: gap or non-gap?
   AG = 29; + anion gap metabolic acidosis
5. Is compensation appropriate?
   \[
   pCO_2 \text{ should } = \text{ last 2 digits of pH [95!!]} \text{ or } (1.5 \times HCO_3^-) + 8 \text{ [18]; } pCO_2 \text{ is too high so he has a superimposed respiratory acidosis}
   \]

6. Mixed disorder?
   \[
   \text{AG} = 29 \text{ (17); } HCO_3^- = 7 \text{ (17); so metabolic acidosis is pure AG acidosis. Thus, mixed AG metabolic acidosis and respiratory acidosis}
   \]
Problem 5

A 31-yo woman who is 33 weeks pregnant presents with a 2-day history of vomiting.

<table>
<thead>
<tr>
<th>140</th>
<th>104</th>
<th>8</th>
<th>85</th>
<th>pH 7.64</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.0</td>
<td>26</td>
<td>0.6</td>
<td></td>
<td>pCO₂ 25</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>pO₂ 93</td>
</tr>
</tbody>
</table>
1. Anticipate the disorder
   Pregnancy: resp alk  Vomiting: met alk
2. Acidemic or alkalemic?
   pH = alkalemic
3. Metabolic or respiratory?
   If resp, HCO₃ should be low; if metabolic, then pCO₂ should be high; must have both
4. If metabolic acidosis: gap or non-gap?
   N/A; no acidosis; no AG
5. Is compensation appropriate?
   NO (by eyeball, for reasons listed above)

6. Mixed disorder?
   Yes, mixed metabolic and respiratory alkalosis.
   No acidosis component.
A 65-yo man is admitted with congestive heart failure. He responds well to diuretic therapy, with a 4-kg weight loss over the next 2 days.

<table>
<thead>
<tr>
<th>134</th>
<th>91</th>
<th>31</th>
<th>126</th>
<th>pH 7.49</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.4</td>
<td>36</td>
<td>0.8</td>
<td>pCO₂ 48</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>pO₂ 92</td>
<td></td>
</tr>
</tbody>
</table>
1. Anticipate the disorder  
   Hypokalemic, hypochloremic metabolic alkalosis due to volume contraction (diuretics)
2. Acidemic or alkalemic?  3. Metabolic or respiratory?  
   pH = alkalemic; must be metabolic (high HCO₃, high pCO₂)
4. If metabolic acidosis: gap or non-gap?  
   AG = 134 – (91+36) = 7; no anion gap
5. Is compensation appropriate?  
   Δ pCO₂ should = 0.7 x Δ HCO₃ = 0.7 x (36-24) = 8; 40 + 8 = 48  
   HCO₃⁻ + 15 = pCO₂ = last 2 digits of pH → 36 + 15 = 51; pretty close!

6. Mixed disorder?  
   No gap – so he has a pure metabolic alkalosis
The previous patient is discharged on furosemide, but returns 3 days later with crushing chest pain, SOB and diaphoresis. Exam shows BP 88/60, bilateral crackles, S3. EKG shows ischemia; CXR = pulmonary edema.

\[
\begin{array}{c|c|c|c|c}
\text{pH} & \text{pCO}_2 & \text{pO}_2 \\
7.14 & 60 & 52 \\
\end{array}
\]
1. Anticipate the disorder
   Pulm edema -> resp alk or resp acidosis; shock -> metabolic acidosis; furosemide -> metabolic alkalosis
2. Acidemic or alkalemic?
   pH = acidemic
3. Metabolic or respiratory?
   If resp, HCO₃ should be > 24 in compensation; if metabolic, then pCO₂ should < 40; must have both respiratory and metabolic acidoses
4. If metabolic acidosis: gap or non-gap?
   AG = 26; + anion gap metabolic acidosis
5. Is compensation appropriate?
   NO (by eyeball, for reasons listed above)
6. Mixed disorder? Anything else?
   AG = 26 (14); HCO₃ = 20 (↓ 4); so HCO₃ is too high; must have a superimposed metabolic alkalosis.

Thus, triple disorder: respiratory acidosis, anion gap metabolic acidosis, and metabolic alkalosis