Case 1

An internal medicine resident is called to the ER to evaluate a 62-year-old woman transferred from an outside hospital with a 2-day history of profuse vomiting. On exam, NG tube to suction, BP 80/50, pulse 115/minute, decreased skin turgor, diffuse abdominal soreness, bladder not enlarged to percussion. The BMP profile indicated a serum Na 135 mEq/L, K 3.5 mEq/L, Cl 85 mEq/l, and CO₂ (HCO₃⁻) 25 mEq/l, blood sugar 90 mg/dl, BUN 110 mg/dl, creatinine 4.5 mg/dl. ABGs: pH 7.40, pCO₂ 40 mm Hg, calc HCO₃⁻ 25 mEq/L. Over the first 7 hours in the ER, only 20 cc of urine via Foley catheter.
Case 1: Question 1

Which one of the following best applies to this patient?

A. There is no significant acid-base disorder because the blood pH and HCO$_3^-$ are normal.
B. The patient does not have metabolic acidosis because of the normal blood pH.
C. The patient has metabolic alkalosis.
D. The patient has metabolic acidosis and respiratory acidosis.
E. The patient has a mixed metabolic acidosis and metabolic alkalosis.

APPROACH TO ACID-BASE DISTURBANCES

• Is acidemia or alkalemia present?
• Is acid-base disorder metabolic or respiratory?
• Has the “other member” of the HCO$_3^-$/PaCO$_2$ ratio changed quantitatively as one would expect for a single acid-base disturbance?
APPROACH TO ACID-BASE DISTURBANCES

• Metabolic acidosis — check anion gap
• Metabolic alkalosis — chloride-sensitive vs. chloride-resistant
• Respiratory acid-base disorders — acute vs. chronic
• Mixed acid-base disturbance

ACID-BASE DEFINITIONS

<table>
<thead>
<tr>
<th>Acidemia</th>
<th>$\uparrow \left[ H^+ \right] \quad (\downarrow \text{pH})$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alkalemia</td>
<td>$\downarrow \left[ H^+ \right] \quad (\uparrow \text{pH})$</td>
</tr>
<tr>
<td>Acidosis</td>
<td>A pathophysiologic process tending to acidity body fluids</td>
</tr>
<tr>
<td>Alkalosis</td>
<td>A pathophysiologic process tending to alkalinize body fluids</td>
</tr>
</tbody>
</table>
\[ [H^+] \text{ nEq/L} = 24 \frac{PaCO_2 \text{ (mm Hg)}}{[HCO_3^-] \text{ (mEq/L)}} \]

\text{PaCO}_2 \text{ regulated by the physiologic control system maintaining carbon dioxide balance.}

\text{Plasma}[HCO_3^-] \text{ regulated by the physiologic control system maintaining hydrogen ion balance.}

\text{pH} = pK + \log \frac{HCO_3^-}{P_{CO_2}} \text{ (METABOLIC) (KIDNEY) (RESPIRATORY) (LUNGS)}
NORMAL VALUES FOR ACID-BASE PARAMETERS IN ARTERIAL AND VENOUS BLOOD

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>[H⁺] nanoEq/ L</th>
<th>Pco₂ mm Hg</th>
<th>[HCO₃⁻] mEq/ L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial</td>
<td>7.37-7.43</td>
<td>37-43</td>
<td>36-44</td>
<td>22-26</td>
</tr>
<tr>
<td>Venous</td>
<td>7.32-7.38</td>
<td>42-48</td>
<td>42-50</td>
<td>23-27</td>
</tr>
</tbody>
</table>

CONVERSION OF pH TO [H⁺]

<table>
<thead>
<tr>
<th>pH</th>
<th>[H⁺]</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.20</td>
<td>63</td>
</tr>
<tr>
<td>7.40</td>
<td>40</td>
</tr>
<tr>
<td>7.60</td>
<td>25</td>
</tr>
</tbody>
</table>
Is low pH bad?

- Tissue hypoxia causes tissue acidosis which may not be reflected in blood level.
- Tissues are much more acidemic than blood.

Major Adverse Consequences of Severe Acidemia

CARDIOVASCULAR

- Impairment of cardiac contractility
- Arteriolar dilatation, venoconstriction, and centralization of blood volume
- Increased pulmonary vascular resistance
- Reductions in cardiac output, arterial BP, and hepatic and renal blood flow
- Sensitization to reentrant arrhythmias and reduction in threshold of ventricular fibrillation
- Attenuation of cardiovascular responsiveness to catecholamines
Major Adverse Consequences of Severe Acidemia

**RESPIRATORY**
- Hyperventilation
- Decreases strength of respiratory muscles and promotion of muscle fatigue
- Dyspnea

**CEREBRAL**
- Inhibition of metabolism and cell-volume regulation
- Obtundation and coma

**METABOLIC**
- Increased metabolic demands
- Insulin resistance
- Inhibition of anaerobic glycolysis
- Reduction in ATP synthesis
- Hyperkalemia
- Increased protein degradation
ACID-BASE DEFINITIONS

Metabolic acidosis — An acid-base disturbance initiated by a reduction in plasma \([\text{HCO}_3^-]\)

Metabolic alkalosis — An acid-base disturbance initiated by an increase in plasma \([\text{HCO}_3^-]\)

Respiratory acidosis — An acid-base disturbance initiated by an increase in \(\text{PaCO}_2\)

Respiratory alkalosis — An acid-base disturbance initiated by a reduction in \(\text{PaCO}_2\)

DEFINITION OF TERMS - COMPENSATION

1. Compensatory hyperventilation in metabolic acidosis
2. Compensatory hypoventilation in metabolic alkalosis
3. Secondary fall in \(\text{HCO}_3\) in respiratory alkalosis
4. Secondary rise in \(\text{HCO}_3\) in respiratory acidosis
**ACID-BASE DEFINITIONS**

Simple acid-base disturbance — the presence of one primary abnormality coupled with its anticipated secondary response.

Mixed acid-base disturbance — the simultaneous presence of two or more primary abnormalities.

**ACID-BASE DISORDERS**

<table>
<thead>
<tr>
<th>DISORDER</th>
<th>PRIMARY EVENT</th>
<th>EFFECT ON H⁺</th>
<th>COMPENSATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>↓ HCO₃⁻</td>
<td>↑</td>
<td>↓ pCO₂</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑ HCO₃⁻</td>
<td>↓</td>
<td>↑ pCO₂</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>↑ pCO₂</td>
<td>↑</td>
<td>↑ HCO₃⁻</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>↓ pCO₂</td>
<td>↓</td>
<td>↓ HCO₃⁻</td>
</tr>
</tbody>
</table>
RENAL AND RESPIRATORY COMPENSATION FOR PRIMARY ACID-BASE DISTURBANCES

<table>
<thead>
<tr>
<th>DISORDER</th>
<th>PRIMARY CHANGE</th>
<th>COMPENSATORY RESPONSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>↓ [HCO₃⁻]</td>
<td>1.2 mmHg decrease in PCO₂ for every 1 meq/L fall in [HCO₃⁻]</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑ [HCO₃⁻]</td>
<td>0.7 mmHg elevation in PCO₂ for every 1 meq/L rise in [HCO₃⁻]</td>
</tr>
</tbody>
</table>
# Renal and Respiratory Compensation for Primary Acid-Base Disturbances

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Primary Change</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory acidosis</td>
<td>$\uparrow$ PCO$_2$</td>
<td>1 mEq/L increase in [HCO$_3^-$] for every 10 mmHg rise in PCO$_2$</td>
</tr>
<tr>
<td>Acute</td>
<td></td>
<td>3.5 mEq/L elevation in [HCO$_3^-$] for every 10 mmHg rise in PCO$_2$</td>
</tr>
<tr>
<td>Chronic</td>
<td>$\downarrow$ PCO$_2$</td>
<td>2 mEq/L reduction in [HCO$_3^-$] for every 10 mmHg fall in PCO$_2$</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td></td>
<td>4 mEq/L decrease in [HCO$_3^-$] for every 10 mmHg reduction in PCO$_2$</td>
</tr>
</tbody>
</table>

## Example of Mixed Metabolic Acidosis and Respiratory Acidosis

If a patient has metabolic acidosis with a HCO$_3^-$ concentration of 15 mEq/L, the finding of a PCO$_2$ of 40 mmHg would establish the presence of superimposed respiratory acidosis. Appropriate compensation for metabolic acidosis consists of hyperventilation. The finding of a normal PCO$_2$ implies inability to hyperventilate appropriately, i.e., a superimposed respiratory acidosis.
METABOLIC ACIDOSIS and RESPIRATORY ACIDOSIS

Cardiac arrest
COPD with sepsis
Renal failure with ARDS
Pulmonary edema

$\text{PaCO}_2 \uparrow$; $[\text{HCO}_3^-] \downarrow$; $\text{pH} \downarrow$; $[\text{H}^+] \uparrow$

Rx: Intubate and ventilate
: $\text{HCO}_3^-$

ANION GAP

$\text{AG} = \text{Na} - (\text{C1} + \text{HCO}_3^-)$
The Ionic Anatomy of Serum

USE OF ANION GAP

1. Differential diagnosis of metabolic acidosis
2. Estimate of concentration of organic anions in metabolic acidosis
3. Elevated anion gap (>16 mEq/L) = metabolic acidosis
4. To check validity of serum electrolytes
5. Special causes of low anion gap (e.g., myeloma, bromide, severe hypernatremia, lithium toxicity)
METABOLIC ACIDOSIS

Primary defect is fall in serum $\text{HCO}_3^-$

Accumulation of metabolic acids caused by:

1. Excess acid production which overwhelms renal capacity for acid excretion (e.g., diabetic ketoacidosis)
2. Renal excretory failure: normal total acid production in face of poor renal function (e.g., chronic renal failure)
3. Loss of alkali: leaves un-neutralized acid behind (e.g., diarrhea)
**METABOLIC ACIDOSIS — INTERPRETATION OF LOW PLASMA HCO$_3^-$**

Rapidly falling plasma HCO$_3^-$ means rapid addition of acid (e.g., diabetic ketoacidosis or lactic acidosis)

Slowly falling plasma HCO$_3^-$ = Either

1. Slow addition of acid over and above ability of kidney to excrete it,... OR
2. Normal addition of acid but kidney is unable to excrete it

**COMPENSATION FOR METABOLIC ACIDOSIS**

**ACUTE RESPONSE:** Hyperventilation BUT fall in pCO$_2$ lags

**STEADY STATE:** Hyperventilation in predictable fashion

\[ \Delta \text{pCO}_2 = 1.3 \times \Delta \text{HCO}_3 \]

OR

\[ \text{pCO}_2 = 1.54 \times \text{HCO}_3 + 8.36 \pm 1.1 \]
CAUSES OF A NORMAL ANION GAP METABOLIC ACIDOSIS

1. Gastrointestinal bicarbonate loss
   a. Diarrhea
   b. Pancreatic fistula
2. Ureteroenterostomy
3. Drugs
   a. Acetazolamide
   b. Sulfamylon
   c. Cholestyramine
   d. Acidifying agents: NH₄Cl, oral CaCl₂, arginine HCl, lycine HC
4. Rapid I.V. hydration
5. Hyperalimentation
6. Post-hypocapnia
7. Renal causes
   a. Chronic renal insufficiency
   b. Renal tubular acidosis
      1) Gradient distal
      2) Bicarbonate wasting proximal
      3) Hypoaldosteronism
NORMAL ANION GAP METABOLIC ACIDOSIS

SERUM POTASSIUM

Elevated
1. NH₄Cl, ArgHCl, HCl, oral CaCl₂
2. Adrenal insufficiency
3. Hyporeninemic hypoaldosteronism
4. Spironolactone
5. Interstitial nephritis

Low
1. Diarrhea, small bowel drainage
2. RTA
3. Sulfur toxicity
4. Ureteral diversions
5. Diamox, sulfamylon

URINE ANION GAP (UAG) IN THE DIFFERENTIAL DIAGNOSIS OF HYPERCHLOREMIC METABOLIC ACIDOSIS

UAG + : RTA
UAG - : Diarrhea
CAUSES OF A HIGH ANION GAP METABOLIC ACIDOSIS

1. Ketoacidosis: b-hydroxybutyrate
2. Lactic acidosis: lactate
3. Renal insufficiency (GFR <20 ml/min): SO₄, HPO₄
4. Ingestions
   A. Salicylate: organic anions, especially lactate
   B. Ethylene glycol: glycolate, oxalate
   C. Methanol or formaldehyde: formate
   D. Paraldehyde: organic anions
   E. Sulfur: SO₄
5. Massive rhabdomyolysis: organic anions

* The substances after the colon represent the major retained anions in the high anion gap acidosis.

LACTIC ACIDOSIS

<table>
<thead>
<tr>
<th>MINOR LACTATE RISE</th>
<th>MAJOR LACTATE RISE</th>
</tr>
</thead>
<tbody>
<tr>
<td>(&lt;5-10 mEq/L)</td>
<td>(&gt;10 mEq/L)</td>
</tr>
</tbody>
</table>

- Alkalosis (Resp>Metab)
- CHO infusion (esp. Fruct.)
- Exercise
- Catecholamines
- Diabetic ketoacidosis
- Alcohol
- Shock
- Severe anemia
- Hypoxia - severe
- Carbon monoxide
- Glycogen storage
- Malignancies
- Metformin
- Idiopathic
TREATMENT OF METABOLIC ACIDOSIS

1. Treat underlying cause (e.g., hypotension, sepsis, diabetic KA)

2. Administration of sodium bicarbonate
   - ALWAYS if very low plasma HCO₃
   - Avoid overzealous administration of HCO₃
     • modest increment in plasma HCO₃, (e.g., 4-6 mEq/L)
     • BEWARE of “overshoot alkalemia”
     • BEWARE of worsening CNS status

A 68-year-old female entered the hospital with acute Salmonella enteritis and profuse diarrhea of 1-week’s duration.

<table>
<thead>
<tr>
<th>Hospital Day</th>
<th>1</th>
<th>2</th>
<th>-</th>
<th>-</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>H⁺ (nEq/L)</td>
<td>57</td>
<td>31</td>
<td></td>
<td></td>
<td>38</td>
</tr>
<tr>
<td>pH</td>
<td>7.24</td>
<td>7.51</td>
<td>7.42</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaCO₂ (mmHg)</td>
<td>12</td>
<td>17</td>
<td>43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCO₃ (mEq/L)</td>
<td>5</td>
<td>13</td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na (mEq/L)</td>
<td>133</td>
<td>137</td>
<td>139</td>
<td></td>
<td></td>
</tr>
<tr>
<td>K (mEq/L)</td>
<td>2.5</td>
<td>4.2</td>
<td>3.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cl (mEq/L)</td>
<td>118</td>
<td>114</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*A (mEq/L)</td>
<td>10</td>
<td>10</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine (mg %)</td>
<td>6.3</td>
<td>--</td>
<td></td>
<td></td>
<td>3.4</td>
</tr>
</tbody>
</table>
Case 2

A 24-year-old Caucasian woman was brought to the ER in coma. On arrival, the serum sodium was 137 mEq/l, potassium 2.1 mEq/L, chloride 110 mEq/l, and CO₂ 14 mEq/l. Blood sugar 90. Arterial blood gases revealed a pH of 6.93, pCO₂ 65, pO₂ 40.

Case 2, continued

The patient was intubated, ventilated, and given potassium replacement. Several hours later, the blood pH was 7.20. A wise CCF resident just happened to note the urinalysis report (heads up ball!!) with a urine pH of 6.10.
Case 2: Question 1

The best explanation for the data given is: (select only one of the 4 possibilities below)

A. Metabolic acidosis due to unexplained lactic acidosis
B. Metabolic alkalosis probably due to vomiting
C. Respiratory acidosis
D. Respiratory acidosis and metabolic acidosis

Case 2: Question 2

The scenario described above suggests: (select only one of the 3 possibilities below)

A. Diabetic ketoacidosis
B. Renal tubular acidosis
C. Severe diarrhea
CAUSES OF METABOLIC ALKALOSIS

Primary accumulation of HCO₃
  – NaHCO₃ administration
  – Milk - alkali syndrome
Primary loss of H⁺
  – Vomiting, GI suction
Contraction alkalosis (diuretics)
Post hypercapneic state
Diuretics
Bizarre diarrhea:
  – Chlordiarrhea - infants
  – Villous adenoma - adults
Primary aldosteronism
Cushing’s syndrome - usually malignancy
Bartter’s /Gitelman’s syndromes
Licorice - glycyrrhizic acid
Carbenoxylone (antiacid)
Severe potassium depletion

DIAGNOSIS OF METABOLIC ALKALOSIS

Saline dependent (chloride dependent, saline responsive)

1. Vomiting or nasogastric suction
2. Diuretic therapy
3. Chloride-losing diarrhea of infancy (rare)
4. Post hypercapneic state

These are usually associated with a contracted extracellular fluid volume and little chloride in the urine (<10 mEq/L)
**DIAGNOSIS OF METABOLIC ALKALOSIS**

**Saline independent** (chloride independent, saline resistant)

1. Hyperaldosteronism
2. Cushing’s syndrome
3. Bartter’s / Gitelman syndromes
4. Licorice ingestion
5. Severe potassium depletion (e.g., total body potassium deficits of ~1000 mEq)

Potassium replacement, as well as chloride, may be required to relieve the alkalosis; **associated with moderately high urine chloride.**
## TREATMENT OF METABOLIC ALKALOSIS

1. Remove offending agent (diuretic, NG tube)
2. Provide volume (NaCl)
3. Provide chloride
4. Give KCl to correct potassium deficit
5. Acidifying agents (arginine HCl, HCl)
6. Dialysis with low HCO$_3^-$ bath

## CLINICAL CAUSES OF RESPIRATORY ACIDOSIS

<table>
<thead>
<tr>
<th>ACUTE</th>
<th>CHRONIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>General anesthesia</td>
<td>Primary alveolar hypoventilation</td>
</tr>
<tr>
<td>Sedative overdosage</td>
<td>Brain tumor</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>Respiratory nerve damage</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>(e.g., poliomyelitis)</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Primary myopathy involving respiratory muscles</td>
</tr>
<tr>
<td>Severe pneumonia</td>
<td>Restrictive disease of the thorax</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>(e.g., scleroderma)</td>
</tr>
<tr>
<td>Laryngospasm</td>
<td>Prolonged pneumonia</td>
</tr>
<tr>
<td>Aspiration of foreign body</td>
<td>Chronic bronchitis and emphysema</td>
</tr>
<tr>
<td>Mechanical ventilators</td>
<td></td>
</tr>
</tbody>
</table>
CAUSES OF RESPIRATORY ALKALOSIS

Anxiety
CNS disorders
  Cerebrovascular accident
  Tumor
  Infection
Hormones - Drugs
  Salicylates
  Catecholamines
  Progesterone
  Analeptic overdose
  Fever, gram-negative sepsis

Endotoxemia
Hyperthyroidism
Hypoxia
Ventilator-induced
Pregnancy
Liver insufficiency
Pulmonary edema (mild)
Lung disease
  Restrictive disorders (early)
  Pulmonary emboli
  Pneumonia
APPROACH TO ACID-BASE DISTURBANCES

• Is acidemia or alkalemia present?
• Is acid-base disorder metabolic or respiratory?
• Has the “other member” of the $\text{HCO}_3^-/\text{PaCO}_2$ ratio changed quantitatively as one would expect for a single acid-base disturbance?
APPROACH TO ACID-BASE DISTURBANCES

- Metabolic acidosis — check anion gap
- Metabolic alkalosis — chloride-sensitive vs. chloride-resistant
- Respiratory acid-base disorders — acute vs. chronic
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CLUES TO DIAGNOSIS OF ACID-BASE DISORDERS

<table>
<thead>
<tr>
<th>HISTORY AND PE</th>
<th>ACID-BASE DISORDER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholism</td>
<td>respiratory alkalosis</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>respiratory alkalosis</td>
</tr>
<tr>
<td>Jaundice</td>
<td>respiratory alkalosis</td>
</tr>
<tr>
<td>Diuretics</td>
<td>metabolic alkalosis</td>
</tr>
<tr>
<td>Vomiting</td>
<td>metabolic alkalosis</td>
</tr>
<tr>
<td>NG suction</td>
<td>metabolic alkalosis</td>
</tr>
</tbody>
</table>
### CLUES TO DIAGNOSIS OF ACID-BASE DISORDERS

<table>
<thead>
<tr>
<th>HISTORY AND PE</th>
<th>ACID-BASE DISORDER</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD</td>
<td>respiratory acidosis</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>respiratory acidosis</td>
</tr>
<tr>
<td>Renal failure</td>
<td>metabolic acidosis</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>metabolic acidosis</td>
</tr>
<tr>
<td>Shock</td>
<td>metabolic acidosis</td>
</tr>
<tr>
<td>MI</td>
<td>metabolic acidosis</td>
</tr>
<tr>
<td>Kussmaul respirations</td>
<td>metabolic acidosis</td>
</tr>
<tr>
<td>COPD + diuretics</td>
<td>respiratory acidosis and metabolic alkalosis</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>metabolic acidosis and respiratory acidosis</td>
</tr>
<tr>
<td>Sepsis</td>
<td>respiratory alkalosis and metabolic acidosis</td>
</tr>
<tr>
<td>Salicylate intoxication</td>
<td>respiratory alkalosis and metabolic acidosis</td>
</tr>
</tbody>
</table>
Case 3

A 52-yr-old alcoholic man was brought to the Emergency Department intoxicated and disoriented. He was found in a parking lot lying face down after apparently vomiting. He was febrile 39.6° C with RLL pneumonia on chest x-ray.

The patient appeared volume contracted with BP 100/74 mm Hg. The physical exam demonstrates coarse rhonchi in the RLL, 2/6 systolic ejection murmur in the LSB, abdomen soft and non tender.
Case 3

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>131 mmol/L</td>
</tr>
<tr>
<td>K</td>
<td>2.8 mmo/L</td>
</tr>
<tr>
<td>Cl</td>
<td>80 mmol/L</td>
</tr>
<tr>
<td>HCO&lt;sub&gt;3&lt;/sub&gt;</td>
<td>21 mmol/L</td>
</tr>
<tr>
<td>BUN</td>
<td>60 mg/dL</td>
</tr>
<tr>
<td>Cr</td>
<td>1.6 mg/dL</td>
</tr>
<tr>
<td>Osmolality</td>
<td>310 mOsm/kg</td>
</tr>
<tr>
<td>Glucose</td>
<td>290 mg/dL</td>
</tr>
<tr>
<td>Serum ketones:</td>
<td>weakly positive</td>
</tr>
<tr>
<td>Anion gap</td>
<td>30 mEq/L</td>
</tr>
<tr>
<td>pH</td>
<td>7.53</td>
</tr>
<tr>
<td>PaCO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>23 mm Hg</td>
</tr>
<tr>
<td>PaO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>60 mm Hg</td>
</tr>
<tr>
<td>[H]</td>
<td>30 mmol/L</td>
</tr>
</tbody>
</table>

Case 3, Question:

The acid base disorder in this case is:

A. Metabolic alkalosis
B. Respiratory acidosis, metabolic alkalosis
C. Metabolic acidosis
D. Metabolic acidosis, metabolic alkalosis, respiratory alkalosis
Case 3. Answer is D., metabolic acidosis, metabolic alkalosis, respiratory alkalosis

This patient demonstrates an increased anion gap of 30 mEq/L indicating the presence of a high anion gap metabolic acidosis. The *increase* in the anion gap is about 18 mEq/L (30 – 12), **but the fall** in the plasma HCO$_3^-$ is only 5 mEq/L (26-21). These findings suggest that metabolic alkalosis (vomiting) may have been present before the metabolic acidosis (ECF contraction, hypotension, and likely lactic acidosis).

Case 3, continued

Since the increase in the anion gap is approximately 18 mEq/L, the plasma bicarbonate should have fallen by 18 mEq/L to its measured value of 21 mEq/L (*i.e.*, from 39 – 21). If there was a preexisting metabolic alkalosis with a plasma bicarbonate of about 39 mEq/L, the compensatory rise in PaCO$_2$ should have increased the PaCO$_2$ to about 50 mmHg.

The observed PaCO$_2$ of 23 mmHg is lower than one would have predicted in the setting of this mixed metabolic alkalosis and metabolic acidosis, suggesting a concomitant respiratory alkalosis (pneumonia) (a triple acid-base disorder).