Introduction

- An acid is a compound capable of donating a proton and a base is a compound capable of accepting a proton.

- pH is defined as the negative logarithm of the hydrogen ion concentration [H⁺].

General Concepts

- Daily acid production
  - Volatile (CO₂ production – excreted by lungs)
  - Non-volatile (acid production from intermediary metabolism, specifically catabolism of sulfur-containing amino acids in protein)

- There are 2 main homeostatic mechanisms which are responsible for maintaining acid-base balance in response to non-volatile acid production.
  - Internal buffer systems (neutralize acid)
  - Excretion of hydrogen ion (excrete acid)

Metabolic Acidosis

- Metabolic acidosis is defined as a primary decrease in the bicarbonate concentration of the plasma. It is normally accompanied by a decrease in PCO₂ (respiratory adaptation) in order to maintain near normal pH.

- Henderson and Henderson-Hasselbalch. These equations depict the relationship between the three determining variables in acid-base homeostasis.

<table>
<thead>
<tr>
<th>Henderson</th>
<th>Henderson-Hasselbalch</th>
</tr>
</thead>
<tbody>
<tr>
<td>[H⁺] = 24 PCO₂ / [HCO₃⁻]</td>
<td>pH = pK + Log[HCO₃⁻] / [H₂CO₃]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>pH</th>
<th>[H⁺] (neg/L)</th>
</tr>
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<tbody>
<tr>
<td>7.1</td>
<td>80</td>
</tr>
<tr>
<td>7.2</td>
<td>65</td>
</tr>
<tr>
<td>7.3</td>
<td>50</td>
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<tr>
<td>7.4</td>
<td>40</td>
</tr>
<tr>
<td>7.5</td>
<td>30</td>
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<tr>
<td>7.6</td>
<td>25</td>
</tr>
<tr>
<td>7.7</td>
<td>20</td>
</tr>
</tbody>
</table>

- Acid-Base Homeostasis is dependent on the following reactions:
  - [H⁺] + [HCO₃⁻] ↔ H₂CO₃ ↔ H₂O + CO₂ (excreted by lungs)
  - A primary change in either [HCO₃⁻] or PCO₂ will produce an adaptive (compensatory) change in the other in the same direction in an attempt to keep pH constant (Tables 1 and 2)
Table 1. The Four Primary Acid Base Disorders and their Compensatory Responses

<table>
<thead>
<tr>
<th>Acid-Base Disorder</th>
<th>Primary Abnormality</th>
<th>Effect on [H+]</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Acidosis</td>
<td>↓[HCO₃⁻]</td>
<td>increase</td>
<td>↓PCO₂</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑[HCO₃⁻]</td>
<td>decrease</td>
<td>↑PCO₂</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>↑PCO₂</td>
<td>increase</td>
<td>↑[HCO₃⁻]</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>↓PCO₂</td>
<td>decrease</td>
<td>↓[HCO₃⁻]</td>
</tr>
</tbody>
</table>

Renal Regulation of Acid-Base Balance

- **Reabsorption of filtered bicarbonate.** Most or all of bicarbonate filtered at the glomerulus is reabsorbed, primarily in the proximal tubule. Failure of this reabsorption mechanism leads to **proximal renal tubular acidosis** ("type 2 RTA").

- **Excretion of acid.** The kidney excretes acid in 3 fashions:
  - Free hydrogen ion (H⁺) excretion (lowers urine pH) – this is quantitatively the least important mechanism
  - Titratable acid excretion (secreted H⁺ combines with poorly reabsorbable anions such as phosphate)
  - Excretion of ammonium ion (NH₄⁺). The kidney generates ammonia (NH₃) which combines with secreted H⁺ and is excreted as ammonium ion.

- Excretion of acid results in **bicarbonate generation** in the distal tubule.

Failure of H⁺ excretion and/or ammonia production/ammonium excretion result in **distal renal tubular acidosis**. Failure of H⁺ excretion results in "**type 1 RTA**". **Hypoaldosteronism** leads to a form of distal RTA called "**type 4 RTA**" in which there is impaired ammonium excretion.

Types of Metabolic Acidosis

Metabolic acidosis can occur due to overproduction of an endogenous weakly dissociable acid (such as lactic acid). Such acids can be generically represented as H⁺A⁻ (for lactic acid, A⁻ would be the acid anion lactate). In this case the excess H⁺ ions titrate extracellular bicarbonate as before, but now there is accumulation of the excess anion (A⁻) rather than chloride, resulting in a **high anion gap acidosis**.

Metabolic acidosis can also occur due to decreased renal acid excretion leading to retention of acid (H⁺ ions), which titrate extracellular bicarbonate, or it can be caused by loss of bicarbonate from the body. In either instance, the decrease in filtered bicarbonate results in decreased renal bicarbonate and increased renal chloride.
reabsorption (to maintain electroneutrality), resulting in a **hyperchloremic acidosis**.

The plasma chloride rises to the same extent as the plasma bicarbonate falls, and the anion gap remains normal.

- **High Anion Gap Acidosis**

The anion gap is a concept to give a clue as to the cause of metabolic acidosis ([Figure 1](#)). Electroneutrality demands that the number of positive charges equals the number of negative charges (ions) in body fluids. Therefore, in plasma:

\[
\text{Na}^+ + \text{K}^+ + \text{Ca}^{++} + \text{Mg}^{++} = \text{Cl}^- + \text{HCO}_3^- + \text{Alb}^- + \text{OA}^- + \text{PO}_4^{3-} + \text{SO}_4^{2-}
\]

OA = organic anions; Alb = albumin

Simplifying this equation to include only certain monovalent measured anions and calling the other cations unmeasured cations (UC) or unmeasured anions (UA):

\[
\text{Na}^+ + \text{UC} = \text{Cl}^- + \text{HCO}_3^- + \text{UA}
\]

Anion gap = \( \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) = (\text{UA} - \text{UC}) \)

\( \text{UA} - \text{UC} = \text{AG} = 10-12 \text{ mEq/L} \)

Overproduction of an endogenous organic acid (\(\text{H}^+\text{A}^-\)), where \(\text{A}^-\) is the acid anion, leads to high anion gap metabolic acidosis (for example with lactic acidosis \(\text{A}^- = \text{lactate}\)). When lactic acid is produced, the \(\text{H}^+\) is titrated by bicarbonate as follows:

\[
\text{H}^+\text{A}^- + \text{HCO}_3^- \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}_2\text{O} + \text{CO}_2 + \text{A}^-
\]

This reaction consumes 1 mol of bicarbonate for each mole of acid produced. If \(\text{A}^-\) is not excreted or metabolized, it will accumulate in plasma, resulting in an “anion gap”.
The difference between the concentrations of sodium and the sum of chloride and bicarbonate is termed the anion gap. In this figure the anion gap is 12 mEq/L.

Theoretically, with this type of acidosis, the increase in anion gap will equal the decrease in bicarbonate concentration.

- Normal Anion Gap (Hyperchloremic) Acidosis

Metabolic acidosis can also occur from either loss of bicarbonate from the body or failure of the kidney to excrete acid. In either instance, serum bicarbonate level will decrease, and the kidney will reabsorb more chloride than usual along with sodium in order to maintain electroneutrality. This results in hyperchloremic acidosis.

- Causes of Metabolic Acidosis
  - High Anion Gap Acidosis
    - Increased organic acid production (with organic anion shown in parenthesis)
      - Lactic acidosis (lactate)
      - Ketoacidosis (acetoacetate, beta-hydroxybutyrate)
      - Toxin ingestion
        - Salicylate (mostly lactate)
        - Methanol (formate)
        - Ethylene glycol (glyoxylate, oxalate)
    - Failure to excrete inorganic anions (phosphate, sulfate) in renal failure
• Normal Anion Gap (Hyperchloremic) Acidosis
  o Gastrointestinal loss of bicarbonate (diarrhea)
  o Renal loss of bicarbonate
    ▪ Proximal renal tubular acidosis (RTA) (Type 2) (Figure 2)
    ▪ Carbonic anhydrase inhibitors (prevent proximal HCO₃⁻ reabsorption)
  o Failure to excrete acid
    ▪ Distal renal tubular acidosis (RTA) (Type 1, Type 4) (Figure 3)
    ▪ Renal failure
  o Administration of acid
    ▪ Infusion of HCl or its congeners (e.g., ammonium chloride)
  o Administration of large amounts of saline (dilutional acidosis)

![Diagram of Normal Renal Function and Malfunction](image)

**Figure 2. Mechanism of Proximal RTA.** In normal kidneys, there is complete reabsorption of filtered bicarbonate until the plasma bicarbonate level exceeds normal (i.e., 25 mEq/L). If there is dysfunction of the proximal tubule, there is a defect in bicarbonate reabsorption, leading to a decrease in the plasma bicarbonate level to the point that the kidneys are again able to reabsorb all of the filtered bicarbonate. In the example given in the Figure, this is a plasma HCO₃⁻ of 15 mEq/L. Since filtered bicarbonate is equal to the plasma HCO₃⁻ x GFR, in this case, the kidneys ability to reabsorb bicarbonate would be 15/25 (60%) of normal.
Figure 3. Mechanism of Distal RTA. In normal kidneys, acid (H⁺) is excreted by the distal tubule alpha-intercalated cells, predominantly in the form of ammonium ion (NH₄⁺). When H⁺ is excreted, an equimolar amount of HCO₃⁻ is returned to the blood, thus regenerating the bicarbonate that has been titrated by acid produced from protein metabolism. Type 1 distal RTA can be due to failure of the H⁺-ATP pump on the lumen side of the cell (1), failure of the HCO₃⁻/Cl⁻ exchanger on the blood side of the cell (2), or backleak of H⁺ from the lumen into the cell (3). Aldosterone deficiency decreases the H⁺-ATPase and can also lead to distal RTA (this is called Type 4 RTA).

Treatment of Metabolic Acidosis

- Treat underlying cause(s)
- Bicarbonate (especially with normal anion gap acidosis) – Na or K bicarbonate (or bicarbonate former) depending on etiology and electrolyte values.
Metabolic Alkalosis

Metabolic alkalosis is defined as a primary increase in the bicarbonate concentration, i.e. $[\text{HCO}_3^-]$, of the plasma. In primary metabolic alkalosis, the pH of the blood will be elevated (alkalemia). An increase in $[\text{HCO}_3^-]$ of 1.0 mEq/L should be accompanied by an increase in PCO$_2$ of 0.7 mm Hg (this is termed respiratory adaptation). The rise in PCO$_2$ will keep the blood pH near normal (although mild alkalemia will be present).

Generation of metabolic alkalosis. There are three ways to generate metabolic alkalosis:

- Net loss of hydrogen ions (H$^+$) from extracellular fluid (ECF)
  - Causes of H$^+$ loss
    - GI - loss of HCl from stomach (vomiting, gastric drainage)
      HCl secretion by parietal cells of stomach does not normally cause metabolic alkalosis because HCl is titrated by pancreatic sodium bicarbonate:
      \[
      \text{HCl} + \text{NaHCO}_3 \rightarrow \text{NaCl} + \text{CO}_2 + \text{H}_2\text{O}.
      \]
      However, if HCl is lost from the body because of vomiting or gastric drainage, there is a net gain of bicarbonate. Metabolic alkalosis is then maintained by increased renal bicarbonate reabsorption and decreased bicarbonate secretion due to depletion of ECF and chloride (see Section II below).
    - Renal - loss of H$^+$ into urine (mineralocorticoid excess states)
      Hydrogen ions generated by protein metabolism are normally excreted by the kidneys primarily in the form of ammonium ($\text{NH}_4^+$) ions. In certain conditions, an inappropriate increase in renal H$^+$ excretion leads to metabolic alkalosis. This is characteristic of primary mineralocorticoid excess states. Aldosterone (the naturally occurring mineralocorticoid hormone) increases sodium reabsorption and potassium and H$^+$ secretion in the cortical collecting tubule. Sodium retention expands ECF volume and decreases proximal tubule sodium reabsorption. Metabolic alkalosis then results from the combination of excess mineralocorticoid hormone effect and increased distal delivery of sodium (in the presence of aldosterone, sodium in the lumen will enter the cells of the collecting tubule and potassium and H$^+$ will exit the cells into the tubule lumen). Administration of diuretic drugs that impair ion transport in the loop of Henle or distal convoluted tubule also results in stimulation of aldosterone secretion (secondary to renin release in response to hypovolemia) and increased sodium delivery to the collecting tubule.
      Of note, high levels of aldosterone per se may not result in metabolic alkalosis in the absence of adequate distal sodium delivery. For instance, in congestive heart failure and liver cirrhosis, there is secondary hyperaldosteronism (due to decreased renal perfusion and increased renin secretion); however, distal sodium delivery is decreased and metabolic alkalosis does not normally occur unless diuretics are administered.
  - Shift into cells - severe K$^+$ deficiency
With very severe potassium deficiency, K+ will shift out of cells into the ECF in exchange for H+. Therefore H+ is "lost" into the cells, engendering metabolic alkalosis (Figure 4). The increase in intracellular H+ in renal tubular cells results in increased H+ secretion and bicarbonate reabsorption, thus also maintaining metabolic alkalosis (see Section II below).

**Figure 4. Generation of metabolic alkalosis from potassium deficiency.** With very severe potassium deficiency, K+ will shift out of cells into the ECF in exchange for Na+ and H+. Therefore H+ is "lost" into the cells, engendering metabolic alkalosis.

- **Net addition of HCO₃⁻ to ECF**
  - Causes of HCO₃⁻ gain:
    - Exogenous alkali administration (bicarbonate, lactate, citrate, acetate)
    - This only occurs with either massive administration of alkali (usually iatrogenic) or in the presence of impaired renal function.

- **Loss of fluid containing chloride in excess of bicarbonate ("contraction alkalosis")**
  - Causes of Cl-rich fluid loss:
    - GI - villous adenoma, congenital chloridorrhea.
      - Villous adenomas are tumors that secrete chloride into the stool; K+ depletion also contributes to the generation of alkalosis. Congenital
chloridorrhea is a rare disorder in which there is a failure of gut
reabsorption of chloride secreted by the stomach.

- Renal - diuretics, Bartter's and Gitelman's syndromes.
  - Diuretics are a very common cause of metabolic alkalosis because chloride
    reabsorption by the kidney is impaired and volume contraction results in
    stimulation of the renin-angiotensin-aldosterone axis (secondary
    hyperaldosteronism).
  - Bartter's syndrome is a genetic defect of the loop diuretic-sensitive Na-K-2Cl
    co-transporter in the loop of Henle ("endogenous loop diuretic"). Gitelman's
    syndrome is a genetic defect of the thiazide-sensitive Na-Cl co-transporter in
    the distal tubule ("endogenous thiazide")

During chronic hypercapnia (seen frequently in chronic obstructive lung
disease), there is an appropriate adaptive increase in renal H+ secretion and
thus bicarbonate reabsorption. This is accompanied by loss of chloride in the
urine (sodium is reabsorbed preferentially with bicarbonate rather than
chloride). Rapid restoration of PCO2 to normal with mechanical ventilation is
not accompanied by a similarly rapid change in bicarbonate handling by the
kidney and may result in severe alkalemia. Because of previous chloride
depletion, post-hypercapnic metabolic alkalosis is typically associated with a
low urine chloride concentration and improves with saline administration.

- Skin - cystic fibrosis
  Metabolic alkalosis has been described in children with cystic fibrosis due to loss
  of chloride in excess of bicarbonate in sweat.

* Maintenance of Metabolic Alkalosis (Figure 5)*

Under normal physiologic conditions, bicarbonate is filtered by the glomerulus (the
filtered load is the product of the GFR and the plasma bicarbonate concentration).
Virtually all of the filtered bicarbonate is then reabsorbed, primarily at proximal nephron
sites. Since normally about 1 mEq/kg of protons are generated by the body each day,
this amount of bicarbonate buffer is titrated in the ECF and needs to be regenerated in
the distal nephron. Both reabsorption (also termed "reclamation") of filtered bicarbonate
and regeneration (also termed "generation") of bicarbonate occur by tubular secretion of
H+. H+ is formed in the tubular cell from the splitting of H2O into H+ and OH-. After the H+
is secreted into the tubular lumen, OH- then combines with CO2 to form HCO3−, which is
reabsorbed into the peritubular capillary. Reabsorption of filtered bicarbonate occurs by
titration of filtered bicarbonate by H+ in the proximal tubular lumen with addition of
bicarbonate formed in the tubular cell into the peritubular capillary. In the distal nephron,
luminal bicarbonate is usually absent; therefore H+ secretion results in urinary H+ loss
(primarily in the form of ammonium ions or NH4+) and thus bicarbonate regeneration.
Figure 5. Mechanism of bicarbonate reabsorption and regeneration by tubular cells. The figure depicts the major cellular and luminal events in bicarbonate reabsorption in the proximal tubule (left panel) and regeneration in collecting tubules (right panel). In the proximal tubule, H+ ions are secreted into the lumen by the Na+-H+ exchanger, whereas HCO3- ions are returned to the systemic circulation primarily via a Na+-3HCO3- co-transporter. In the collecting tubule, a H+-ATPase pump and a Cl--HCO3- exchanger mediate these processes. In the proximal tubule, secreted H+ combines with filtered HCO3- to form CO2 and H2O (this reaction is facilitated by carbonic anhydrase (CA) in the brush border). Bicarbonate is typically mostly reabsorbed by the time the tubular fluid reaches the collecting duct; H+ secretion will then result in regeneration of bicarbonate. Note: the collecting tubule cell shown is a type A intercalated cell; there also are type B intercalated cells, which will be discussed below).

Once metabolic alkalosis has occurred, its maintenance must indicate a failure of the kidneys to excrete the excess bicarbonate. This can occur either because of a decreased filtered load of bicarbonate (due to a decrease in GFR) or an increase in tubular bicarbonate reabsorption (or decrease in bicarbonate secretion). In the absence of renal failure, inability of the kidneys to excrete the excess bicarbonate implies the presence of a factor (or factors) that either increase bicarbonate reabsorption or decrease its secretion, such as ECF or chloride depletion, potassium depletion, and hypercapnia. The mechanism(s) by which these factors maintain metabolic alkalosis are given below.

- ECF depletion
  - Decreases GFR
  - Increases proximal tubular Na+ and HCO3- reabsorption (since there is hypochloremia, decreased filtration of chloride leads to bicarbonate reabsorption with sodium in an attempt to maintain ECF volume)
  - Stimulates renin secretion leading to secondary hyperaldosteronism (increases H+ secretion and HCO3- reabsorption by type A intercalated cells in the collecting tubule) (Figure 6)

- Chloride depletion (recent data suggest that this is the most important mechanism)
  - Decreases distal tubular HCO3- secretion (decreased tubular fluid chloride concentration inhibits chloride-bicarbonate exchange by type B intercalated cells in
the distal tubule) (Figure 7). Note that type B cells have the opposite configuration to type A cells, i.e. the location of the H+-ATPase and chloride-bicarbonate exchangers are reversed).

- Directly stimulates renin production (leading to secondary hyperaldosteronism and increased H+ excretion)

**Figure 6.** Transport mechanisms involved in hydrogen secretion and bicarbonate reabsorption by **type A intercalated cells** in the distal tubule.

**Figure 7.** Transport mechanisms involved in the secretion of bicarbonate into the tubular lumen by **type B intercalated cells** in the distal tubule.

- K+ depletion
  - Increases tubular HCO3- reabsorption

- Hypercapnia (increased PCO2)
  - Increases tubular HCO3- reabsorption

Note: K+ depletion and hypercapnia will both lead to intracellular acidosis, which increases H+ secretion (K+ depletion leads to shift of K+ out of cells and H+ into cells; CO2 movement into cells results in acidosis because CO2 combines with OH- [formed from splitting of H2O into H+ and OH-]).
Clinical Features of Metabolic Alkalosis

- **History**
  - vomiting
  - gastric drainage
  - diuretics

- **Symptoms**
  - often none
  - sometimes cramps

- **Signs**
  - hypertension (in primary mineralocorticoid excess states)
  - hypoventilation (usually not evident on physical exam)
  - tetany and/or increased deep tendon reflexes (metabolic alkalosis results in increased negative charges on serum albumin, thus increasing binding of calcium to albumin and decreasing the concentration of free or ionized calcium)
  - cardiac arrhythmias (esp. if pH > 7.6)

Laboratory Findings in Metabolic Alkalosis

- Arterial blood gases: increased pH, [HCO₃⁻], and PCO₂

- Electrolytes: elevated [HCO₃⁻], decreased [Cl⁻], usually low [K⁺], slight increase in anion gap (increased negative charges on albumin, increased lactate due to increased intracellular pH)

- Blood urea nitrogen (BUN): frequently increased (due to volume depletion)

- Hematocrit: frequently increased (volume depletion)

- Urine chloride [Cl⁻]: very helpful in differential diagnosis (value < 10 mEq/L indicates volume/chloride depletion)

  **Note:** Urine [Na⁺] may not be low despite volume depletion because urinary loss of bicarbonate obligates urinary cation (Na⁺ and K⁺) excretion.

Differential Diagnosis of Metabolic Alkalosis

- **Chloride-responsive type (urine Cl⁻ low, i.e. < 10 mEq/L):**
  - In these conditions, the kidney is avidly reabsorbing chloride because of persistent volume (and chloride) depletion that developed during the generation of metabolic alkalosis.
    - GI - vomiting, gastric drainage, villous adenoma, chloride diarrhea
    - Renal - diuretics (after drug cessation)
    - Skin - cystic fibrosis

- **Chloride-resistant type (urine Cl⁻ high, i.e., > 20 mEq/L)**
  - Renal - mineralocorticoid excess states -- can be either primary (low renin) of secondary (high renin)
### Treatment of Metabolic Alkalosis

- In general, metabolic alkalosis in the presence of ECF excess (as occurs in mineralocorticoid excess states) is mild and does not require treatment. Alkalosis of sufficient severity to require treatment is generally associated with volume depletion and can be corrected with sodium chloride administration. Volume expansion will decrease $\text{HCO}_3^-$ reabsorption. Administration of potassium is indicated if hypokalemia is present, as entry of $\text{K}^+$ into cells in exchange for $\text{H}^+$ will buffer excess ECF $\text{HCO}_3^-$.  
- Some patients, such as those with severe congestive heart failure, may have metabolic alkalosis due to relative plasma volume depletion (due to loop diuretics) but still have increased ECF volume (edema). In this setting, administration of a carbonic anhydrase inhibitor such as acetazolamide may be beneficial. Carbonic anhydrase inhibitors are proximally acting diuretics which result in decreased bicarbonate reabsorption and bicarbonaturia.  
- In the presence of life-threatening alkalosis, intravenous HCl or ammonium chloride (providing the patient does not have hepatic or renal failure) can be given. Ammonium chloride administration titrates bicarbonate by the following reaction:

\[
\text{NH}_4\text{Cl} + \text{NaHCO}_3 \rightarrow \text{NaCl} + \text{NH}_3 + \text{CO}_2 + \text{H}_2\text{O}
\]

- Finally, prevention of metabolic alkalosis in patients undergoing gastric drainage by drugs that inhibit gastric acid secretion ($\text{H}_2$ blockers, omeprazole) is indicated.

### Table 2. Expected Compensation for Primary Acid-Base Disorders

<table>
<thead>
<tr>
<th>Type</th>
<th>Primary Change</th>
<th>Secondary Adaptation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>Decrease in $\text{HCO}_3^-$ of 1 mEq/L</td>
<td>Decrease in PCO$_2$ of 1.2 mmHg. Winter’s formula: PCO$_2$ = 1.5 x $\text{HCO}_3^-$ + 8 ± 2</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Increase in $\text{HCO}_3^-$ of 1 mEq/L</td>
<td>Increase in PCO$_2$ of 0.7 mmHg</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>Increase in PCO$_2$ of 10 mmHg</td>
<td>Acute: Increase in $\text{HCO}_3^-$ of 1.0 mEq/L; Chronic: Increase in $\text{HCO}_3^-$ of 3.5 mEq/L</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>Decrease in PCO$_2$ of 10 mmHg</td>
<td>Acute: Decrease in $\text{HCO}_3^-$ of 2 mEq/L; Chronic: Decrease in $\text{HCO}_3^-$ of 4-5 mEq/L</td>
</tr>
</tbody>
</table>
ACID-BASE PROBLEMS:

**Patient 1.** A 70-year-old man presents with weakness. Blood chemistries reveal (in mmol/L): sodium 145, potassium 3.0, chloride 120, total CO₂ 16; (in mg/dL): urea nitrogen 30, creatinine 1.2. Arterial blood gases (ABG) reveal: pH 7.35, PCO₂ 30 mmHg, HCO₃⁻ 16 mEq/L.

Q1: What is the acid base disturbance?

A. Metabolic acidosis
B. Metabolic alkalosis
C. Mixed metabolic acidosis/metabolic alkalosis
D. No metabolic disturbance

Q2: How would you further characterize it?

Q3: What is the differential diagnosis and most likely cause?

**Patient 2.** A 30-year-old woman presents with fatigue and weakness. She denies vomiting or diarrhea and takes no medications. Her physical examination is normal except for mild orthostatic hypotension. Blood chemistries reveal (in mmol/L): sodium 138, potassium 2.9, chloride 90, total CO₂ 40. Arterial blood gases (ABG) reveal: pH 7.50; PCO₂ 53 mmHg; HCO₃⁻ 40 mEq/L. The urine pH is 7.5. Urine chemistries reveal (in mmol/L): sodium 30, potassium 30, chloride 5.

Q1: What is the acid base disturbance?

A. Metabolic acidosis
B. Metabolic alkalosis
C. Mixed metabolic acidosis/metabolic alkalosis
D. No metabolic disturbance

Q2: What is the most likely diagnosis?

**Patient 3.** A 60-year-old female patient presents to the emergency room (ER) with fever and confusion. She has known chronic obstructive pulmonary disease (COPD) and is on home oxygen. Serum chemistries reveal (in mmol/L): sodium 136; potassium 3.9; chloride 101; total CO₂ 5. ABG: pH 6.8; PCO₂ 33 mm Hg; HCO₃⁻ 5 mEq/L.

Q1: What is the acid-base disturbance?

A. Metabolic acidosis
B. Respiratory acidosis
C. Respiratory alkalosis
D. Mixed metabolic acidosis/respiratory alkalosis
E. Mixed metabolic acidosis/respiratory acidosis

Q2: Why do you think the patient developed it?
Patient 4. A 30-year-old male patient presents to the emergency room (ER) with confusion. Serum chemistries reveal (in mmol/L): sodium 136; potassium 3.9; chloride 101; total CO₂ 15. ABG: pH 7.5; PCO₂ 20 mm Hg; HCO₃⁻ 15 mEq/L.

Q1: What is the acid-base disturbance?

A. Metabolic acidosis
B. Respiratory acidosis
C. Respiratory alkalosis
D. Mixed metabolic acidosis/respiratory alkalosis
E. Mixed metabolic acidosis/respiratory acidosis

Q2: Why do you think the patient developed it?
REFERENCES:


BOOKS:

