Dibartola, Ch 9: Introduction to Acid-Base Disorders

Key Points

Concept of Acidity
- Bronsted and Lowry definition (most common)
  - Acid: proton donor
  - Base: proton acceptor
- \([H^+]\) nEq/L
  - Other ‘important’ [ions] mEq/L
  - \([H^+]\) 10⁻⁶ that of other ions
  - H⁺ highly reactive
- Body proteins have many dissociable groups that may gain or lose protons as \([H^+]\) changes
  - Results in alterations in charge and configuration
  - May adversely affect protein structure and function

Concept of pH
- pH is defined as the negative base 10 logarithm of \([H^+]\)
- \(pH = -\log_{10} [H^+] = \log_{10} (1/[H^+])\)
- pH and \([H^+]\) vary inversely and exponentially with each other

Law of Mass Action
- States that the velocity of a reaction is proportional to the product of the concentrations of the reactants
- \(HA \rightarrow H^+ + A^-\) \(v_1 = k_1 [HA]\)
- \(H^+ + A^- \leftrightarrow HA\) \(v_2 = k_2 [H^+][A^-]\)
- \(k_1/k_2 = K_a = [H^+][A^-]/[HA]\)
- \(K_a =\) dissociation constant
  - An indication of the strength of the acid
  - Large \(K_a\) means \([H^+]\) and \([A^-]\) are much greater than \([HA]\)
    - The acid is a strong one and is largely dissociated
  - Small \(K_a\) means \([H^+]\) and \([A^-]\) are much smaller than \([HA]\)
    - The acid is a weak one and little of it is dissociated
- Minus log base 10......
- Henderson-Hasselbalch equation form of the dissociation equilibrium equation!
  \(pH = pK_a + \log \text{salt} (A^-)/\text{acid} (HA)\)
Concept of Buffering
- A buffer is a compound that can accept or donate protons (H\(^+\)) and minimize a change in pH
  - A 'buffer solution' consists of a weak acid and its conjugate salt
  - When a strong acid is added to a buffer solution containing a weaker acid and its salt, the dissociated protons from the strong acid are donated to the salt of the weak acid and the change in pH is minimized

The Bicarbonate–Carbonic Acid System: Physical Chemistry
- \(\text{CO}_2\) dissolved in blood + \(\text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-\)
- \(\text{pH} = \text{pK}_a + \log \left(\frac{[\text{HCO}_3^-]}{[\text{CO}_2 \text{ dissolved in blood}]}\right)\)
- In body fluids, pH is a function of the ratio between \(\text{HCO}_3^-\) concentration and \(\text{PCO}_2\)

Body Buffers
- \(\text{HCO}_3^-\) (main buffer in ECF) and non-\(\text{HCO}_3^-\) buffers (proteins, phosphates, primary intracellular buffers)
- Due to 'open' nature of the \(\text{HCO}_3^-/\text{CO}_2\) system, pH can be regulated closely despite the addition of acid due to \(\text{CO}_2\) loss via alveolar ventilation
- Hgb is responsible for 80+% of the non-\(\text{HCO}_3^-\) buffering capacity of whole blood
- Plasma proteins contribute 20% buffering capacity of whole blood
  - Greater contribution from albumin vs globulins
- Intracellular buffers
  - Proteins
  - Phosphates (inorganic and organic)

Terminology
- Acidosis and alkalosis refer to the pathophysiologic processes that cause net accumulation of acid or alkali in the body
- Acidemia and alkalemia refer specifically to the pH of ECF

Primary Acid-Base Disturbances
- Four primary acid-base disturbances
  - Metabolic acidosis, respiratory acidosis, metabolic alkalosis, and respiratory alkalosis
- Each primary acid-base disturbance is accompanied by a secondary/adaptive change in the opposing component of the system
- The adaptive response involves the component opposite the one disturbed
  - Returns the pH of the system toward but not completely to normal
  - Over compensation does not occur
- 'Simple' disorder if the disturbance is limited to the primary disorder and the expected secondary response
- ‘Mixed’ disorder if the disturbance is characterized by the presence of at least two separate primary acid-base abnormalities occurring in the same patient
Should be suspected whenever the secondary response exceeds or falls short of that expected.

Compensatory Response for Acid-Base Disorders

- Respiratory disturbances
  - Acute (<24h): immediate titration of predominantly intracellular non-HCO₃⁻ buffers, resulting in an initial change in plasma HCO₃⁻ concentration.
  - Chronic (>48h): characterized by alterations in net acid excretion and bicarbonate reabsorption via the kidneys.
    - This response begins within hours, but takes 2 to 5 days to achieve maximal effectiveness.
- A patient should be considered to have a mixed disorder only when the blood gas value in question deviates considerably from the calculated expected value.

Sample Collection and Handling

- If sample exposed to air:
  - pCO₂: decreases
  - pO₂: increases
- Run sample within 15-25m if not cooled
- At 4°C, sample stable for 2h

**TABLE 9-5** Expected Renal and Respiratory Compensations to Primary Acid-Base Disorders in Dogs

<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>1.0-mm Hg decrement in PCO₂ for each 1-mEq/L decrement in [HCO₃⁻]⁺</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>[HCO₃⁻]⁺</td>
<td>0.7-mm Hg increment in PCO₂ for each 1-mEq/L increment in [HCO₃⁻]⁺</td>
</tr>
<tr>
<td>Acute respiratory acidosis</td>
<td>[PCO₂]</td>
<td>1.5-mEq/L increment in [HCO₃⁻]⁺ for each 10-mm Hg increment in PCO₂</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>[PCO₂]</td>
<td>3.5-mEq/L increment in [HCO₃⁻]⁺ for each 10-mm Hg increment in PCO₂</td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>[PCO₂]</td>
<td>2.5-mEq/L decrement in [HCO₃⁻]⁺ for each 10-mm Hg decrement in PCO₂</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>[PCO₂]</td>
<td>5.5-mEq/L decrement in [HCO₃⁻]⁺ for each 10-mm Hg decrement in PCO₂</td>
</tr>
</tbody>
</table>

Measurement of Blood Gasses

- Measured: pH, pCO₂
- Calculated: HCO₃⁻, BE
- Base excess (BE) is the amount of strong acid or base required to titrate 1 L of blood to pH 7.40 at 37°C, while PCO₂ is held constant at 40 mmHg.