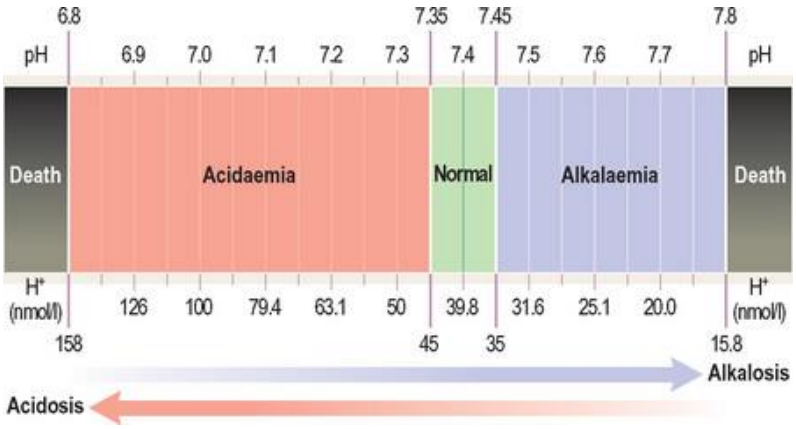

Acid-Base Physiology in a Single Page

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Respiratory Component: When breathing is inadequate (minute ventilation too low) arterial CO_2 builds up. This CO_2 combines with water to make carbonic acid, which in turn lowers arterial pH (raises arterial hydrogen ion concentration, since $[H^+]$ in nEq/L = $10^{(9-pH)}$ or $pH = 9 - \log_{10}[H^+]$) . This is called **respiratory acidosis**. The treatment is to lower the CO_2 tension (arterial pCO_2) by improving the patient's breathing (increasing minute ventilation), for example by employing a ventilator to mechanically breathe for the patient or by giving naloxone to a patient who has received excessive opioid drug (e.g., heroin overdose). Respiratory acidosis can be **acute or chronic**, depending on the degree of compensation (formulas available). To compensate for respiratory acidosis the kidneys increase blood bicarbonate concentration, aka $[HCO3^-]$, via enhanced renal retention of bicarbonate. (Think of bicarbonate as the blood's antacid.) Finally, if an individual breathes with a very large minute volume (hyperventilation) **respiratory alkalosis** is said to occur and the kidneys will eventually dump bicarbonate to compensate so as to keep the pH in the desired range.

Metabolic Component: When blood bicarbonate is diminished, as in a number of pathologic states (e.g., lactic acidosis, diabetic ketoacidosis, renal failure, etc.), the arterial pH drops (arterial hydrogen ion concentration increases). (This effect is a direct consequence of the **Henderson-Hasselbalch equation**, which describes the relationship between the respiratory and metabolic components: **$[H^+]$ in nEq/L = 24 x arterial pCO_2 (mm Hg) / serum bicarbonate**.) This is known as **metabolic acidosis.** To compensate for this, the respiratory control centers in the medulla and pons try to lower the pCO_2 by increasing minute ventilation (seen clinically as **Kussmaul breathing**). The appropriate degree of compensation is given by **Winter's formula**: The **expected arterial pCO_2 in metabolic acidosis (in mmHg) = (1.5 x bicarbonate concentration)** + 8 (range: +/- 2). If the respiratory system is unable to compensate appropriately (lower the pCO_2 sufficiently), the patient is said to have a superimposed respiratory acidosis in addition to a metabolic acidosis.

Acid Base Disorders

Disorder	pH	$[H^+]$	Primary disturbance	Secondary response
Metabolic acidosis	↓	↑	↓ $[HCO_3^-]$	↓ pCO_2
Metabolic alkalosis	↑	↓	↑ $[HCO_3^-]$	↑ pCO_2
Respiratory acidosis	↓	↑	↑ pCO_2	↑ $[HCO_3^-]$
Respiratory alkalosis	↑	↓	↓ pCO_2	↓ $[HCO_3^-]$

Metabolic acidosis comes in two types: normal anion gap and elevated anion gap (elevated over 17), where the anion gap is defined as **anion gap = serum sodium – (serum bicarbonate + serum chloride).** Finally, **metabolic alkalosis** can occur, for example via loss of hydrogen ions from vomiting or from renal retention of bicarbonate. Metabolic alkalosis comes in **two types:** chloride-responsive (urine chloride < 20 mEq/L) versus chloride resistant (urine chloride > 20). **The expected pCO_2 (in mm Hg) in metabolic alkalosis = 0.7 x serum bicarbonate + 20** (range: +/- 5).

File: Acid Base Disorders Summary Rev 3