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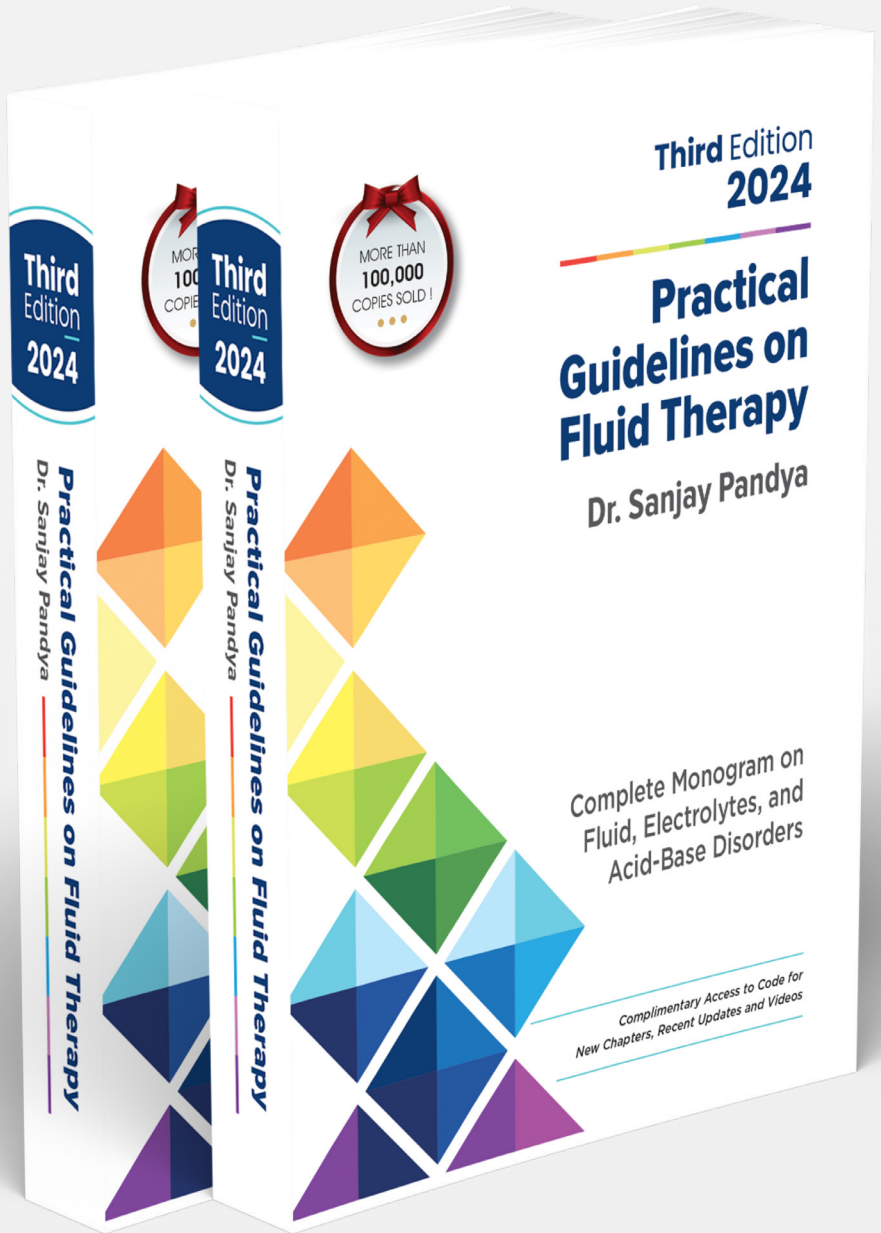
Chapter 32:
Metabolic Alkalosis



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32 Metabolic Alkalosis

Metabolic alkalosis is the most common acid-base disorder, typically developing after hospitalization in critically ill patients, but it is not most frequently present upon admission, unlike metabolic acidosis [1, 2].

DEFINITION

Metabolic alkalosis is a primary acid-base disorder characterized by an increase in serum HCO_3 (>26 mEq/L), a high pH (>7.45), and a compensatory increase in $PaCO_2$ due to alveolar hypoventilation. Hypochloremia and hypokalemia are commonly encountered electrolyte imbalances in metabolic alkalosis.

Respiratory compensation: Hypoventilation, which occurs as a respiratory compensation in metabolic alkalosis, is a relatively slow process compared to the hyperventilation response that occurs in metabolic acidosis. Hypoventilation-induced hypoxia is a limiting factor for compensatory mechanisms in metabolic alkalosis because severe hypoxia (PO_2 <60 mm Hg) is a potent stimulus to increase alveolar ventilation, offsetting this protective respiratory response.

Comparison with chronic respiratory acidosis: Increased HCO_3 and increased $PaCO_2$ are also features of chronic respiratory acidosis, but the differentiating feature is a low pH.

PATHOGENESIS [3–5]

For a proper understanding of the pathogenesis of metabolic alkalosis, it is important to know the two distinct phases involved in sustaining metabolic alkalosis: 1. Generation of metabolic alkalosis and 2. Maintenance of metabolic alkalosis, as summarized in Table 32.1.

Generation of metabolic alkalosis

Mechanisms leading to the primary increase in plasma HCO_3 can involve one or more of the following:

- Gastrointestinal (GI) loss of hydrogen ion: Due to conditions like vomiting,

nasogastric suction, or congenital chloride-losing diarrhea.

- Renal loss of hydrogen ion: It includes the use of diuretics, primary hyperaldosteronism, Bartter syndrome, and Gitelman syndrome.
- Exogenous HCO_3 load: Due to the administration of HCO_3, balanced crystalloids containing buffers, blood products containing citrate as an anticoagulant, and the development of milk-alkali syndrome.

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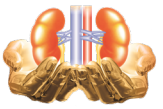
KidneyEducation 

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