METABOLIC ALKALOSIS

produced by conditions that increase HCO_3^- or reduce H^+

The diagnosis requires knowledge of the $PaCO_2$, because elevation of the plasma HCO_3^- may be secondary to renal compensation of a chronic respiratory acidosis.

AETIOLOGY:

Usually caused by an increase in HCO_3^- reabsorption secondary to volume, potassium, or Cl^- loss.

An ECF volume reduction can increase the plasma HCO_3^- concentration when combined salt and water losses occur, typically in patients using diuretics. This state forces a contraction of the ECF around a constant plasma HCO_3^-, creating a relative excess in HCO_3^- concentration; this is known as **contraction alkalosis.**

Hypokalaemia can cause H^+ to shift intracellularly, as well as increased H^+ secretion to preserve renal potassium losses.

BOX 122-5 CAUSES OF METABOLIC ALKALOSIS

Volume-Contracted (Saline-Responsive)
Vomiting/gastric suction
Diuretics
Ion-deficient baby formula
Colonic adenomas
Postrespiratory acidosis

Normal Volume/Volume-Expanded (Saline-Resistant)
Hyperaldosteronism (primary, secondary, or exogenous mineralic corticoids, e.g., licorice, tobacco)
Cushing's syndrome
Severe potassium depletion
Adenocarcinoma
Bartter's syndrome
Ectopic adrenocorticotropic hormone

Unclassified
Milk-alkali syndrome
Carbenicillin therapy
Metabolism of organic acid anion (bicarbonate, lactate, citrate)
Massive transfusion with citrate anticoagulant or plasmanate (acetate) if renal impairment
Nonparathyroid hypercalcemia

PHYSIOLOGIC COMPENSATION:

Less predictable; it involves acute compensation with the respiratory centre (hypoventilation) & chronic compensation with the kidneys (excretion of excess HCO_3^-).

Note: a $PaCO_2$ of > 55 mmHg is unlikely to be cause by simple respiratory compensation for metabolic alkalosis & more likely a ventilation disorder.

MANAGEMENT:

Treatment again aimed at identification and reversal of precipitating or causal factors.

SALINE-RESPONSIVE ALKALOSIS.

Usually for people with low urinary Cl^- with administration of NaCl.

In severely volume-depleted patients, consultation for admission and administration of intravenous mineral acids (e.g., arginine monohydrochloride) may be necessary.

In patients with severe oedematous states (where saline is relatively contraindicated), *acetazolamide* increases excretion of $NaHCO_3$.

In severe renal failure, metabolic alkalosis should be treated with *dialysis.*

SALINE-RESISTANT ALKALOSIS.

Usually occurs with mineralocorticoid excess, hypokalaemia and increased secretion of aldosterone.

- Leads to excessive renal excretion of H^+ and a reabsorption of HCO_3^-.

Treatment can be successful with K^+ replacement +/- the addition of *spironolactone* (an aldosterone antagonist).