Acid Base Part 6

Metabolic Alkalosis

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Major causes of metabolic alkalosis

TABLE 51-6 Causes of Metabolic Alkalosis

I. Exogenous HCO_3^- loads
   A. Acute alkali administration
   B. Milk-alkali syndrome

II. Effective ECFV contraction, normotension, K^+ deficiency, and secondary hyperreninemic hyperaldosteronism

A. Gastrointestinal origin

1. Vomiting
2. Gastric aspiration
3. Congenital chloridorrhea
4. Gastrocystoplasty
5. Villous adenoma

B. Renal origin
   1. Diuretics
   2. Posthypercapnic state
   3. Hypercalcemia/hypoparathyroidism
   4. Recovery from lactic acidosis or ketoacidosis
   5. Nonreabsorbable anions including penicillin, carbenicillin
   6. Mg^{2+} deficiency
   7. K^{+} depletion
   8. Bartter's syndrome (loss of function mutations of transporters and ion channels in TALH)
   9. Gitelman's syndrome (loss of function mutation of Na^{+}-Cl^{-} cotransporter in DCT)

III. ECFV expansion, hypertension, K^+ deficiency, and mineralocorticoid excess
    A. High renin
        1. Renal artery stenosis
        2. Accelerated hypertension
        3. Renin-secreting tumor
        4. Estrogen therapy
    B. Low renin
        1. Primary aldosteronism
            a. Adenoma
            b. Hyperplasia
            c. Carcinoma
        2. Adrenal enzyme defects
            a. 11β-Hydroxylase deficiency
            b. 17α-Hydroxylase deficiency
        3. Cushing's syndrome or disease
        4. Other
            a. Licorice
            b. Carbenoxolone
            c. Chewer's tobacco

IV. Gain-of-function mutation of sodium channel in DCT with ECFV expansion, hypertension, K^+ deficiency, and hyporeninemic-hypoaldosteronism

A. Liddle's syndrome

Maintenance of metabolic alkalosis

- Decreased glomerular filtration rate
  - A: effective circulating volume depletion
  - B : renal failure (usually associated with metabolic Acidosis)
- Increased tubular reabsorption
  - A: effective circulating volume depletion
  - B: chloride depletion (also decreases bicarbonate secretion)
  - C: hypokalemia
  - D: hyperaldosteronism

Urine chloride concentration in the diagnosis of metabolic alkalosis

Less than 25 meq/L	Greater than 40 meq/L
Vomiting or nasogastric suction	Primary mineralocorticoid excess
Diuretics (late)	Diuretics (early)
Factitious diarrhea	Alkali load (bicarbonate or other organic anion
Posthypercapnia	Bartter's or Gitelman's syndrome
Cystic fibrosis	Severe hypokalemia (plasma [K+] < 2.0 meq/L)
Low chloride intake	

Saline-responsive metabolic alkalosis

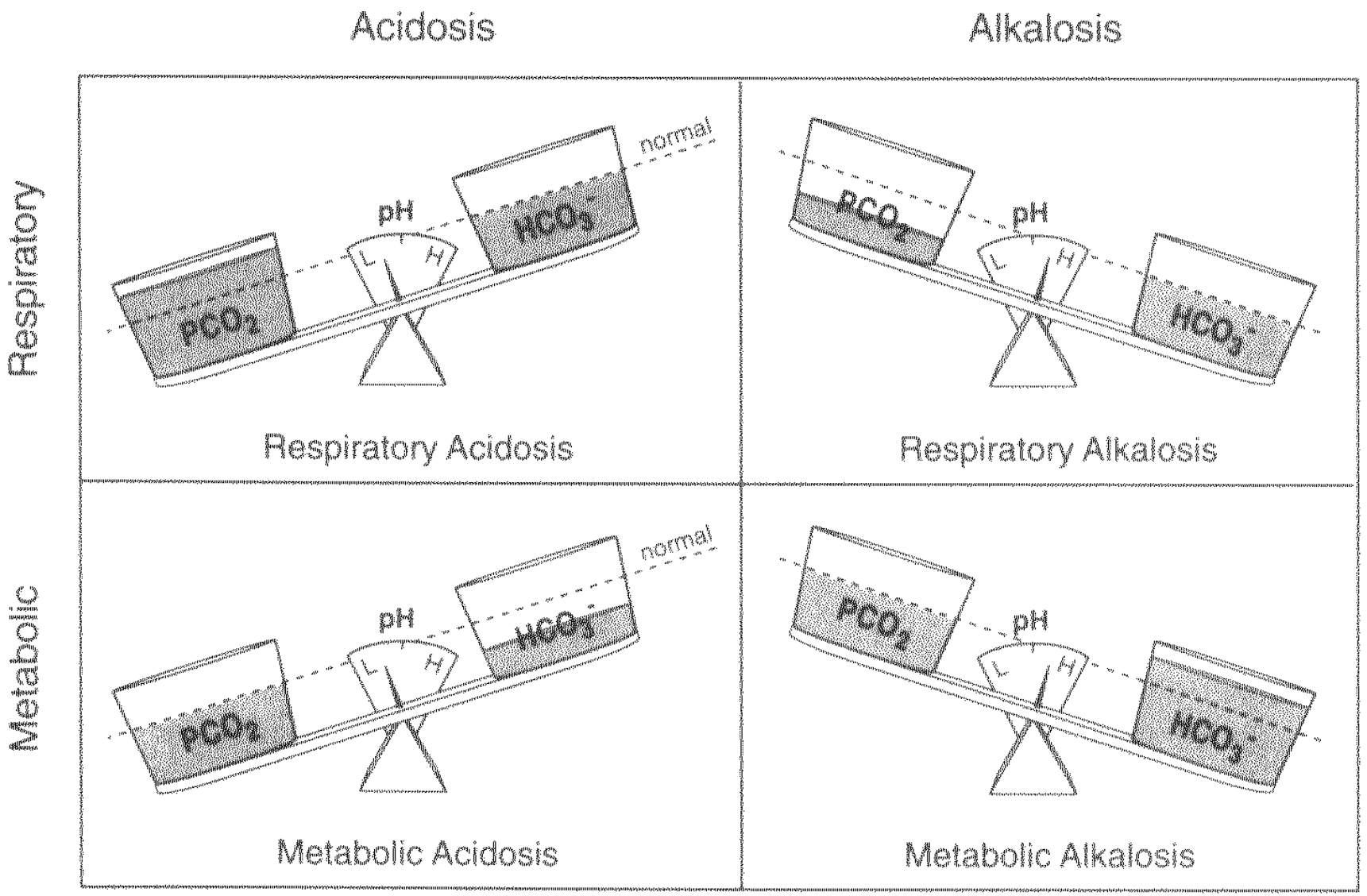
- Vomiting or nasogastric suction
- Diuretics
- Posthypercapnia
- Low chloride intake

Saline-resistant metabolic alkalosis

- Edematous states
- Mineralcorticoid excess
- Severe hypokalemia
- Renal failure

Treatment of metabolic alkalosis(1)

- Correction of volume depletion
- Correction of potassium depletion
- Correction of chloride depletion

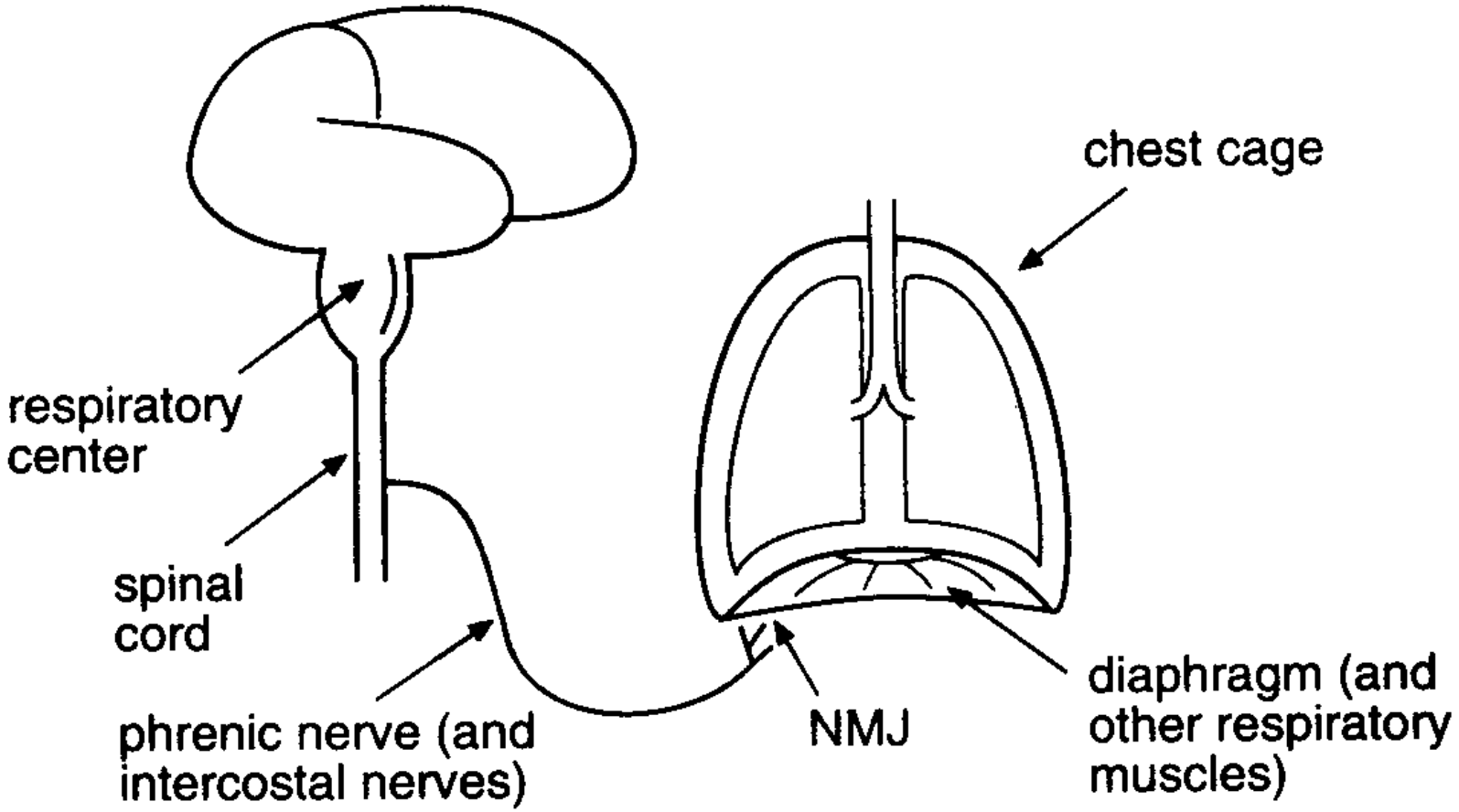


Abelow, Understanding Acid-Base, Williams & Wilkins 1998

RESPIRATORY ACIDOSIS

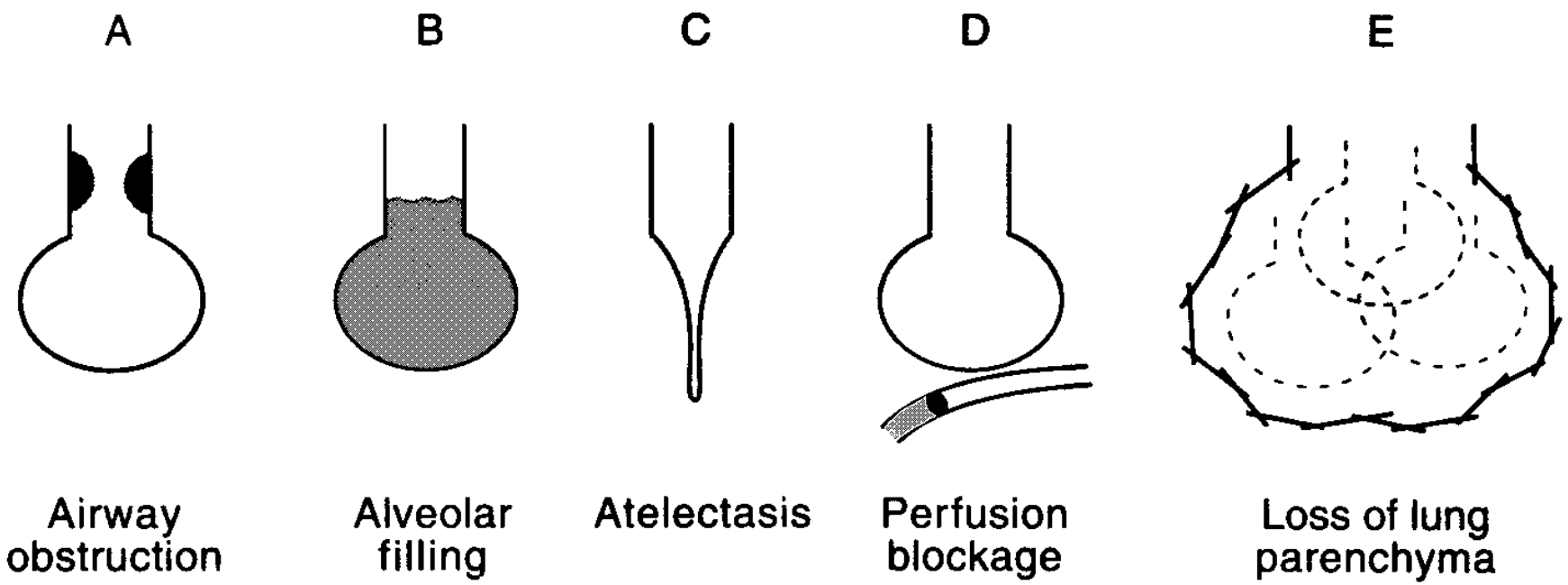
- Alveolar hypoventilation
: acute airway obstruction with underventilation
- Late acute asthma, acute COPD
: CNS - opiate overdose
- stroke
- neuropathy, myopathy

Neuromuscular chain defects -may alter alveolar ventilation



(Abelow B, 1998 "Understanding Acid-Base")

Pulmonary diseases
-may alter alveolar ventilation



(Abelow B, 1998 "Understanding Acid-Base")

RESPIRATORY FAILURE

Type 1: $\downarrow PaO_2$ $\downarrow PaCO_2$

- Alveolar hyperventilation

Type 2: $\downarrow PaO_2$ $\uparrow PaCO_2$

- Alveolar hypoventilation

ACUTE ASTHMA

Early: Alveolar hyperventilation

- ↑ respiratory drive

↓PaO_2 ↓$PaCO_2$

→ give high concentration of O_2 (60%)

Late: Alveolar hypoventilation -↓ respiratorydrive

↓PaO_2 ↑$PaCO_2$

: still relying on hypercapnic drive

→ give high concentration of O_2 (60%)

- may need mechanical ventilation

Permissive Hypercapnea in COPD

RESPIRATORY ALKALOSIS

- Alveolar hyperventilation

: Early acute asthma with over ventilation

: Pulmonary embolus, pneumonia, pulmonary oedema

: Anxiety