Vloed en Electric lights

CDP

TABLE 6-1 Pathogenesis of Hypo-osmolar Disorders

Depletion (primary decreases in total body solute + secondary water retention)*:

- Renal solute loss
  - Diuretic use
  - Solute diuresis (glucose, mannitol)
  - Salt-wasting nephropathy
  - Mineralocorticoid deficiency
- Nonrenal solute loss
  - Gastrointestinal (diarrhea, vomiting, pancreatitis, bowel obstruction)
  - Cutaneous (sweating, burns)
  - Blood loss

Dilution (primary increases in total body water ± secondary solute depletion)†:

- Impaired renal free water excretion
  - Increased proximal reabsorption
    - Hypothyroidism
  - Impaired distal dilution
    - Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
    - Glucocorticoid deficiency
  - Combined increased proximal reabsorption and impaired distal dilution
    - Congestive heart failure
    - Cirrhosis
    - Nephrotic syndrome
  - Decreased urinary solute excretion
    - Beer potomania
- Excess water intake
  - Primary polydipsia
  - Dilute infant formula

TABLE 6-2 Criteria for the Diagnosis of SIADH

Essential

1. Decreased effective osmolality of the extracellular fluid (P_{osm} < 275 mOsm/kg H_2O).
2. Inappropriate urinary concentration (U_{osm} > 100 mOsm/kg H_2O with normal kidney function) at some level of hypo-osmolality.
3. Clinical euvolemia, as defined by the absence of signs of hypovolemia (orthostasis, tachycardia, decreased skin turgor, dry mucous membranes) or hypervolemia (subcutaneous edema, ascites).
4. Elevated urinary sodium excretion while on a normal salt and water intake.
5. Normal thyroid, adrenal, and kidney function.

Supplemental

6. Abnormal water load test (inability to excrete at least 80% of a 20-mL/kg water load in 4 hours and/or failure to dilute U_{osm} to < 100 mOsm/kg H_2O).
7. Plasma AVP level inappropriately elevated relative to plasma osmolality.
8. No significant correction of serum [Na^+] with volume expansion but improvement after fluid restriction.

TABLE 6-3 Common Etiologies of SIADH

Tumors
- Pulmonary/mediastinal (bronchogenic carcinoma, mesothelioma, thymoma)
- Nonchest (duodenal carcinoma, pancreatic carcinoma, ureteral/prostate carcinoma, uterine carcinoma, nasopharyngeal carcinoma, leukemia)

Central nervous system disorders
- Mass lesions (tumors, brain abscesses, subdural hematoma)
- Inflammatory diseases (encephalitis, meningitis, systemic lupus, acute intermittent porphyria, multiple sclerosis)
- Degenerative/demyelinative diseases (Guillain-Barré syndrome, spinal cord lesions)
- Miscellaneous (subarachnoid hemorrhage, head trauma, acute psychosis, delirium tremens, pituitary stalk section, transsphenoidal adenomectomy, hydrocephalus)

Drug induced
- Stimulated AVP release (nicotine, phenothiazines, tricyclic antidepressants)
- Direct renal effects and/or potentiation of AVP antidiuretic effects (desmopressin, oxytocin, prostaglandin synthesis inhibitors)
- Mixed or uncertain actions (angiotensin-converting enzyme inhibitors, carbamazepine and oxcarbazepine, chlorpropamide, clofibrate, clozapine, cyclophosphamide, 3,4methylenedioxymethamphetamine ["Ecstasy"], omeprazole, serotonin reuptake inhibitors, vincristine)

Pulmonary diseases
- Infections (tuberculosis, acute bacterial and viral pneumonia, aspergillosis, empyema)
- Mechanical/ventilatory (acute respiratory failure, COPD, positive pressure ventilation)

Other
- AIDS and AIDS-related complex
- Prolonged strenuous exercise (marathon, triathlon, ultramarathon, hot weather hiking)
- Senile atrophy
- Idiopathic

TABLE 7-1 Defects in Thirst

Primary hypodipsia

- Hypothalamic lesions affecting the osmostat
  - Trauma
  - Craniopharyngioma or other primary suprasellar tumor
  - Metastatic tumor
  - Granulomatous disease
  - Vascular lesions
- Essential hypernatremia
- Geriatric hypodipsia

Secondary hypodipsia

- Cerebrovascular disease
- Dementia
- Delirium
- Mental status changes

TABLE 7-2 Classification of Hypernatremia on the Basis of Associated Changes in Extracellular Volume

Pure water deficit (normal extracellular volume)
- Diabetes insipidus
  - Hypothalamic
  - Nephrogenic
- Increased insensible losses

Hypotonic fluid deficit (decreased extracellular volume)
- Renal losses
  - Diuretic administration
  - Osmotic diuresis
  - Postobstructive diuresis
  - Polyuric phase of ATN
- Gastrointestinal losses
  - Vomiting
  - Nasogastric drainage
  - Enterocutaneous fistulae
  - Diarrhea
- Cutaneous losses
  - Burn injuries
  - Excessive perspiration

Hypertonic sodium gain (increased extracellular volume)
- Salt ingestion
- Hypertonic NaCl
- Hypertonic $NaHCO_3$
- Total parenteral nutrition

TABLE 7-3 Hypothalamic Diabetes Insipidus

Pituitary surgery

Head trauma

Neoplasia
- Primary: dysgerminoma, craniopharyngioma, suprasellar pituitary tumors
- Metastatic: carcinoma of the breast, carcinoma of the lung, lymphoma
- Leukemia

Vascular lesions
- Aneurysms
- Cerebrovascular accidents
- Sheehan's syndrome (postpartum pituitary hemorrhage)

Infections
- Encephalitis
- Meningitis
- Tuberculosis
- Syphilis

Granulomatous disease
- Sarcoidosis
- Histiocytosis

Autoimmune

Vasopressin-neurophysin gene mutations

TABLE 7-4 Nephrogenic Diabetes Insipidus

Drug induced
- Lithium
- Demeclocycline
- Methoxyflurane
- Amphotericin B

Electrolyte disorders
- Hypercalcemia
- Hypokalemia

Obstructive uropathy

Congenital
- Vasopressin V_2-receptor mutations
- Aquaporin 2 mutations

TABLE 7-5 Diagnosis of DI

Diagnosis	Urine Osmolality		Plasma Vasopressin Level
	After Dehydration	After Exogenous Vasopressin	
Normal individuals	>700 mmol/kg	<10% increase	>2.0 pg/mL
Hypothalamic DI			
Complete	<300 mmol/kg	>50% increase	<1.0 pg/mL
Partial	>300 mmol/kg	>10% increase	<1.5 pg/mL
Nephrogenic DI			
Complete	<300 mmol/kg	<50% increase	>5.0 pg/mL
Partial	>300 mmol/kg	<10% increase	>2.0 pg/mL

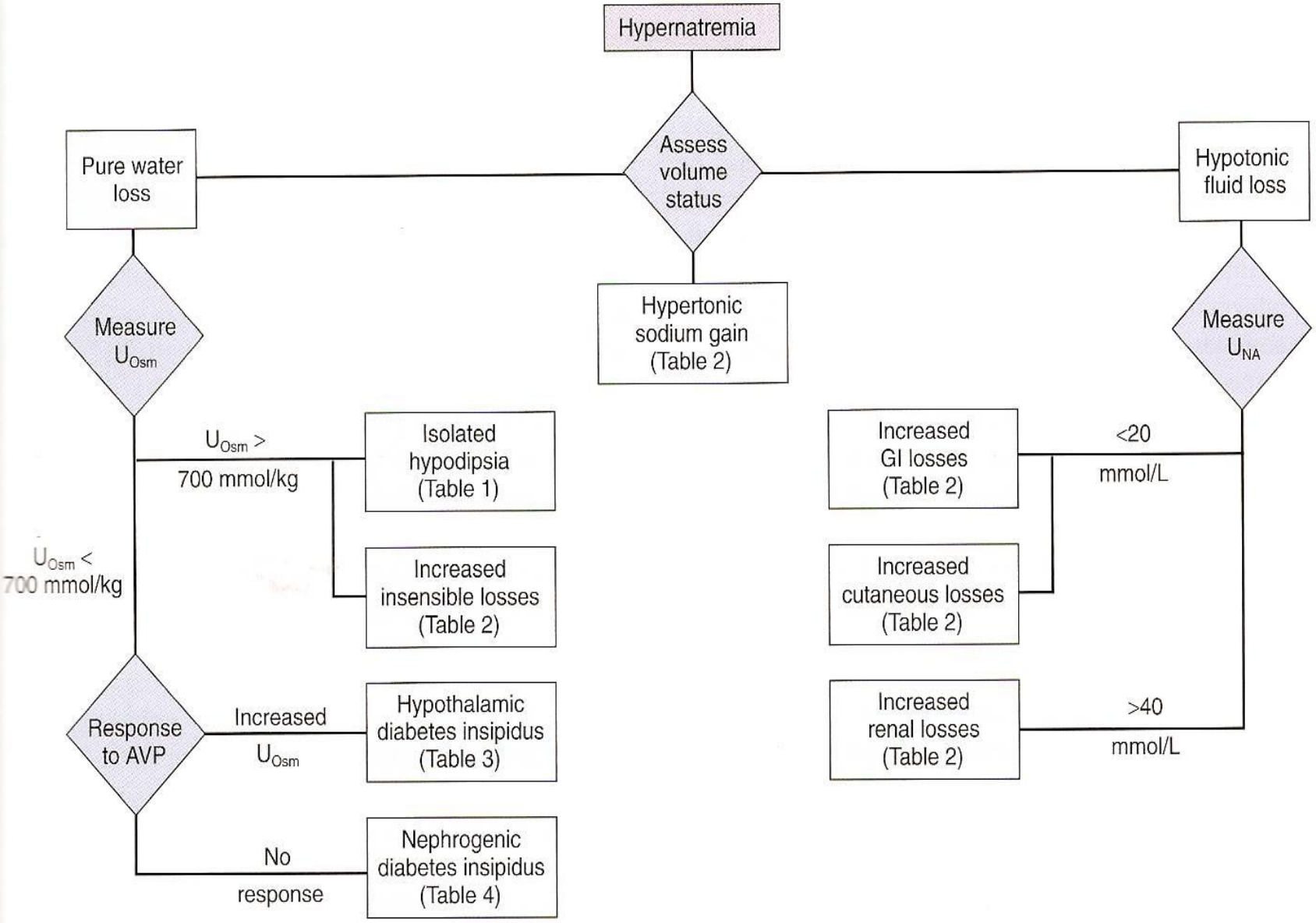


FIGURE 7-1 Algorithm for the evaluation of hypernatremia.

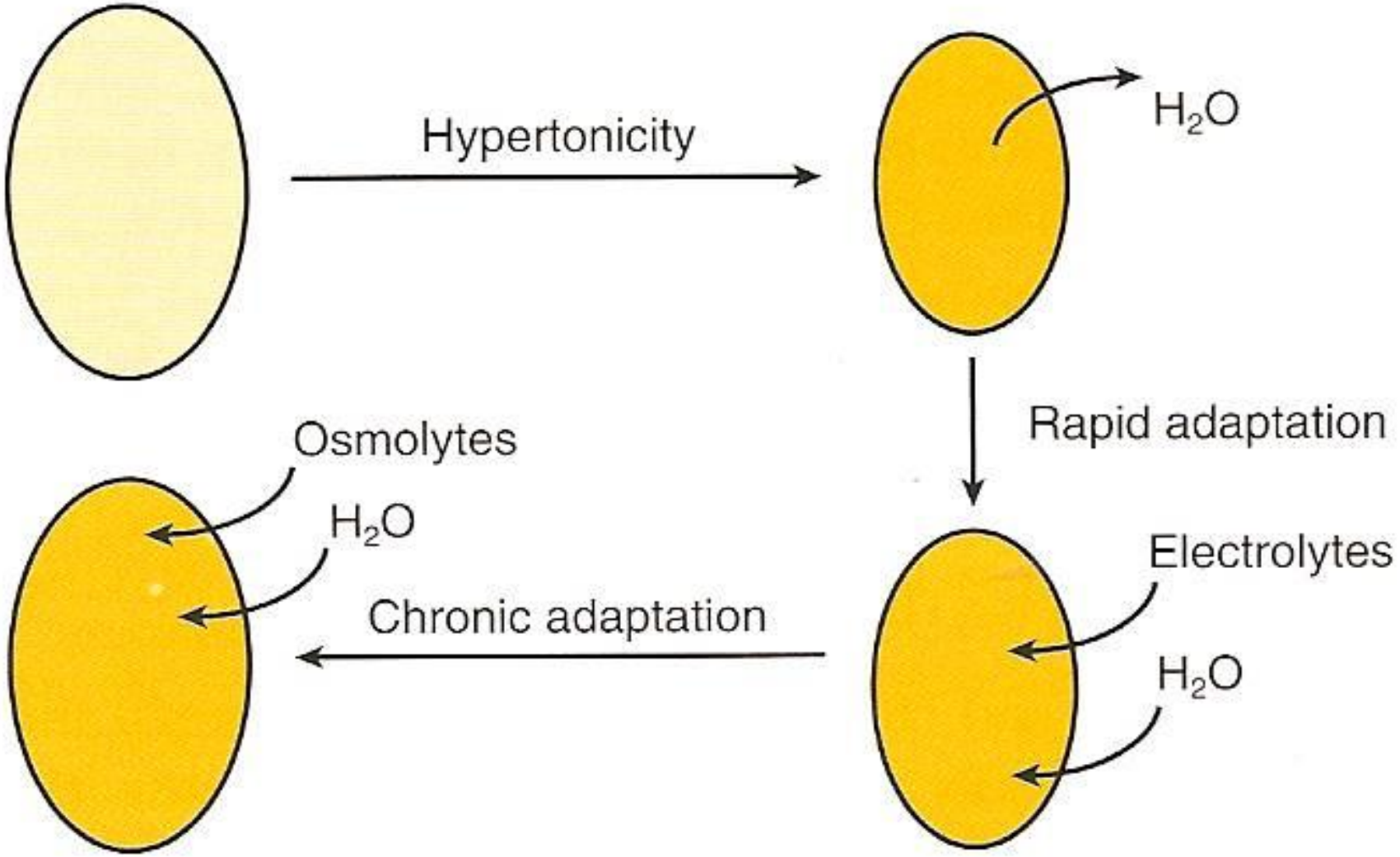
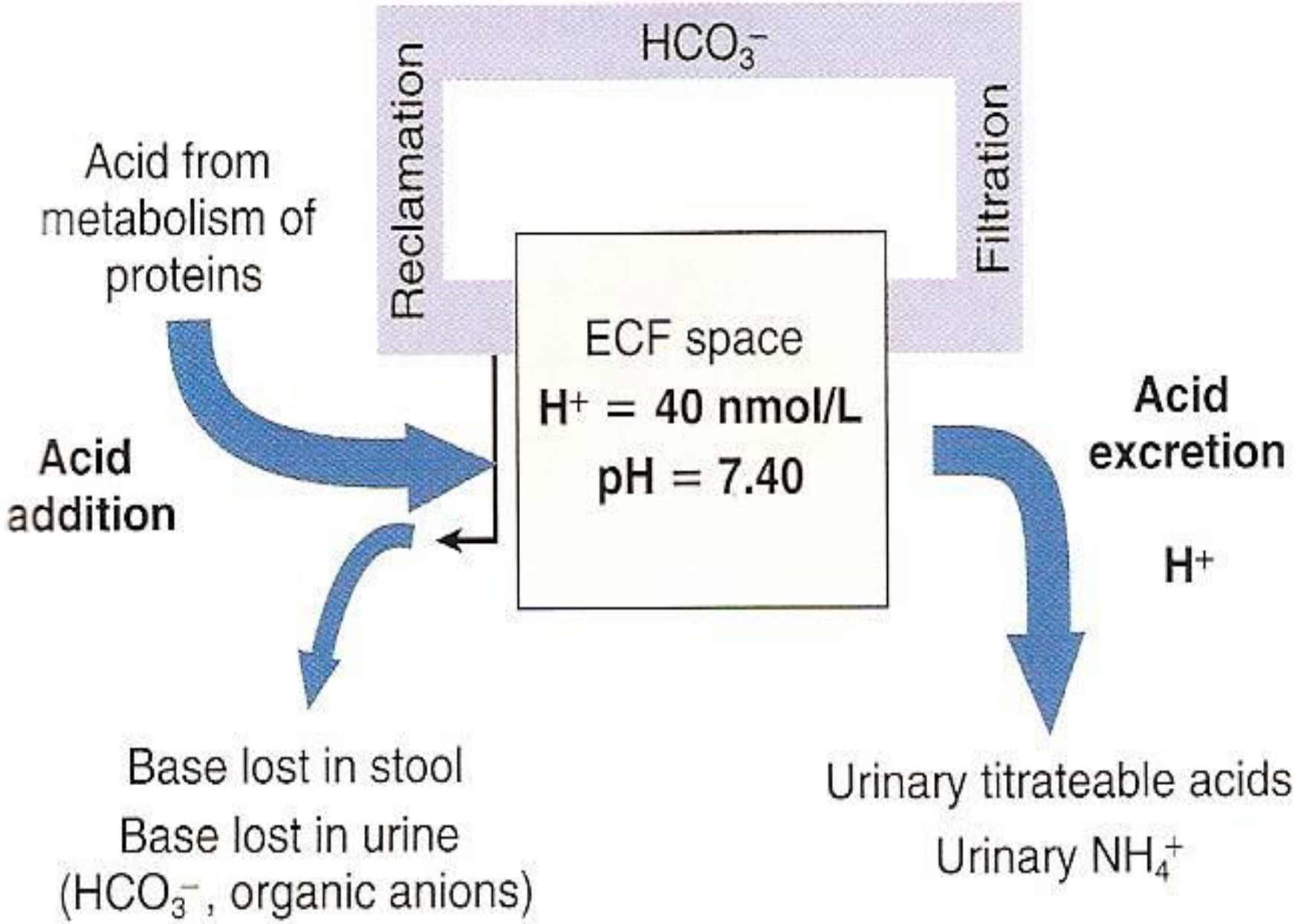
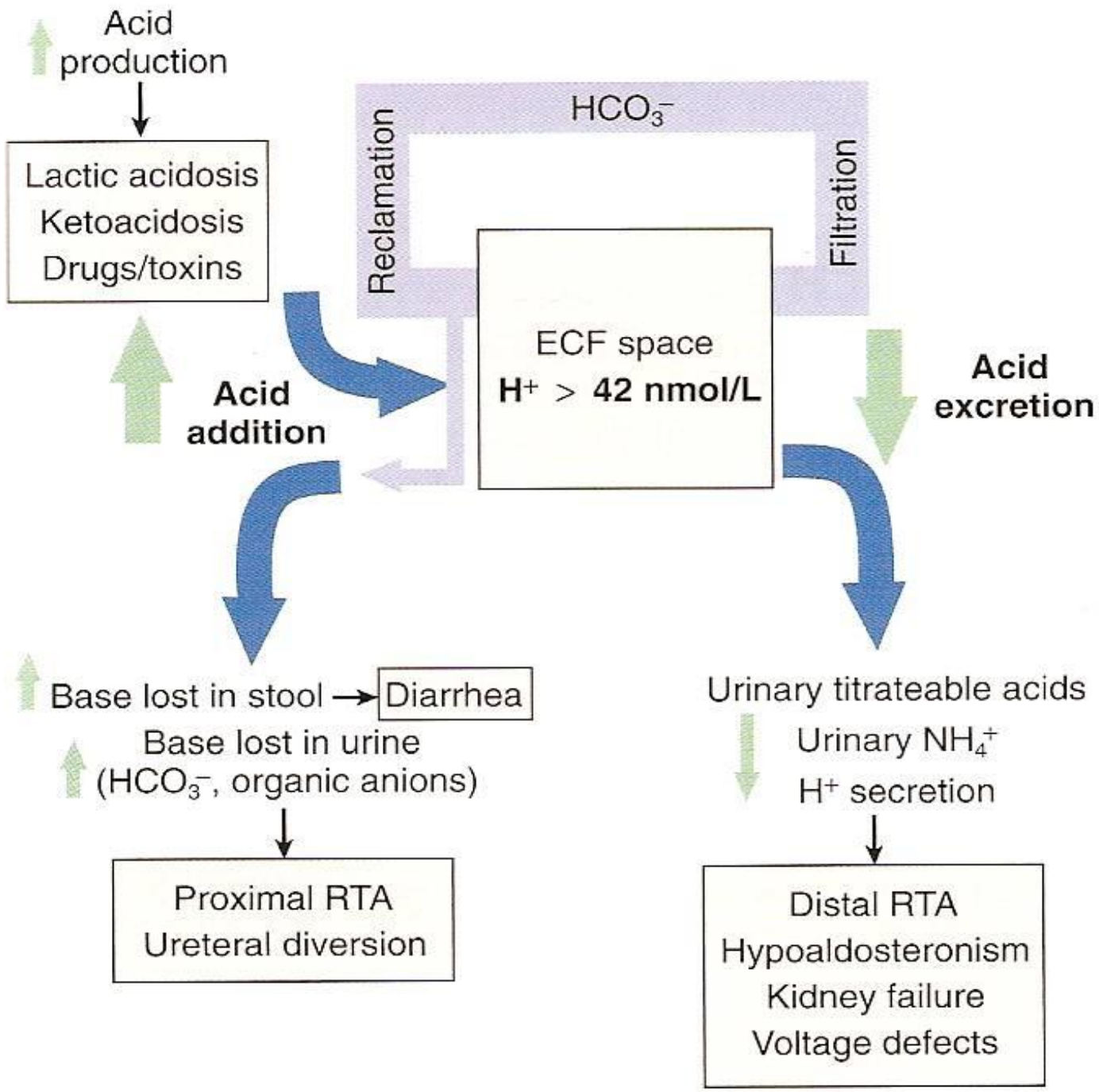

Hypertonicity
H_2O
Rapid adaptation
Osmolytes
H_2O
Electrolytes
Chronic adaptation
H_2O

Table 2.4.
Principal Causes of Edema[a]

I. Increased capillary hydrostatic pressure
  A. Hypervolemia (increased plasma volume)
    1. Inadequate renal excretion of Na^+
      a. Heart failure
      b. Primary renal Na^+ retention
        i. Renal failure
        ii. Inhibition of prostaglandins synthesis (NSAID)
    2. Massive intake of Na^+
  B. Venous obstruction or stasis
    1. Thrombosis
    2. Tumor compression
    3. Muscle disuse
    4. Tense ascites (liver disease)
II. Decreased plasma oncotic pressure due to hypoalbuminemia
  A. Reduced albumin synthesis
    1. Liver disease
    2. Malnutrition
  B. Urinary protein loss (nephrotic syndrome)
III. Increased capillary permeability
  A. Inflammation
  B. Trauma
  C. Burns
IV. Lymphatic obstruction (lymphedema) e.g. tumor

[a]Modified from Rose BD. Clinical physiology of acid-base and electrolyte disorders. 2nd ed. New York: McGraw-Hill Book Company, 1984:314.


HCO_3^-
Reclamation
Filtration
Acid from metabolism of proteins
ECF space
H^+ = 40 nmol/L
pH = 7.40
Acid addition
Acid excretion
H^+
Base lost in stool
Base lost in urine (HCO_3^-, organic anions)
Urinary titrateable acids
Urinary NH_4^+


Acid production
Lactic acidosis
Ketoacidosis
Drugs/toxins
HCO_3^-
Reclamation
Filtration
ECF space
$H^+ > 42$ nmol/L
Acid addition
Acid excretion
Base lost in stool → Diarrhea
Base lost in urine (HCO_3^-, organic anions)
Proximal RTA
Ureteral diversion
Urinary titrateable acids
Urinary NH_4^+
H^+ secretion
Distal RTA
Hypoaldosteronism
Kidney failure
Voltage defects

Principal Causes of Metabolic Acidosis

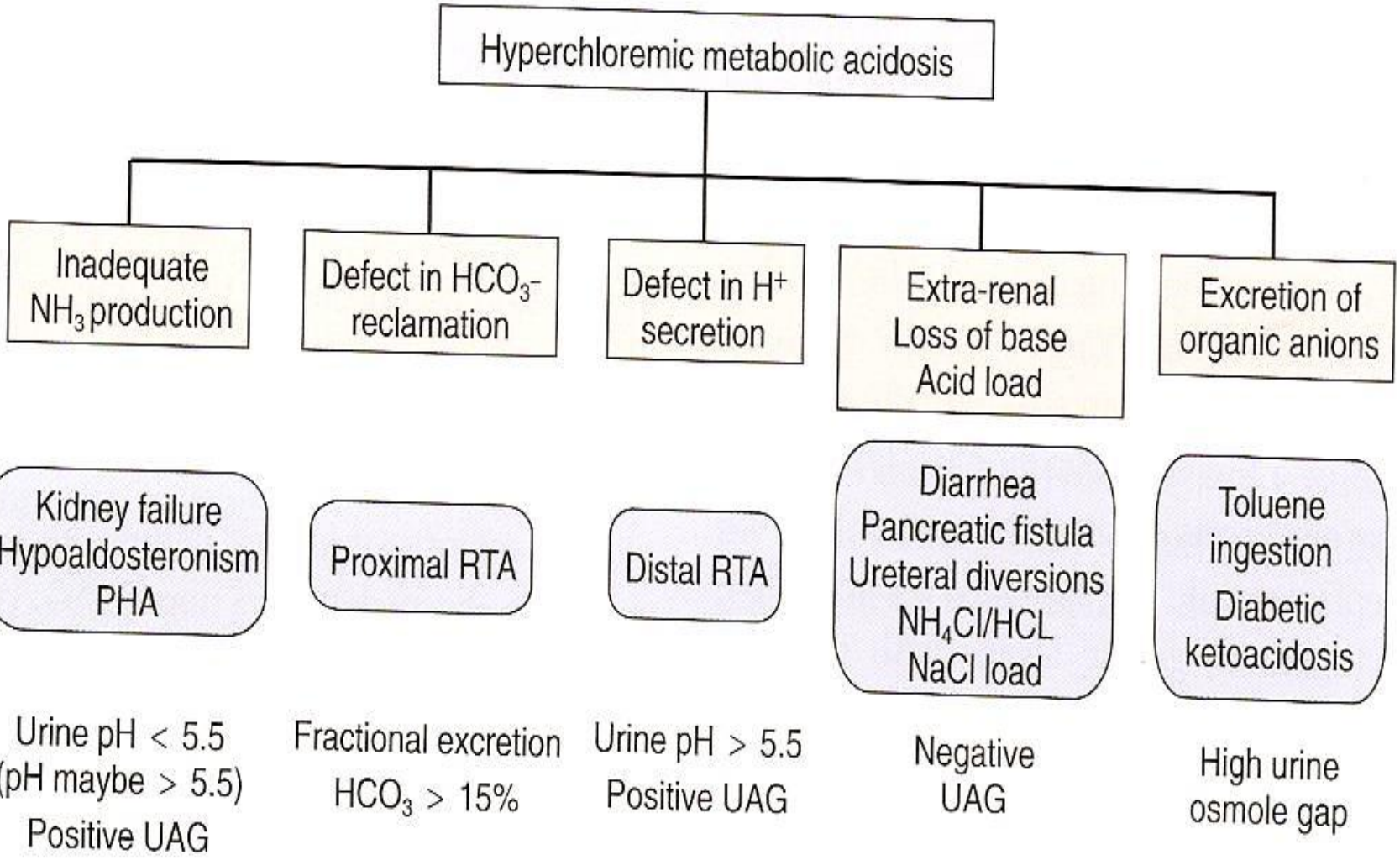
I. Increased Anion Gap
   A. Ketoacidosis
      1. Diabetes mellitus
      2. Starvation
   B. Lactic acidosis
   C. Ingestions
      1. Salicylates
      2. Methyl alcohol
      3. Ethylene glycol
   D. Renal failure
II. Normal Anion Gap
   A. Loss of alkaline intestinal secretions (hypokalemic)
      1. Diarrhea
      2. Fistulae
      3. Biliary or pancreatic drainage tubes
   B. Acetazolamide (hypokalemic)
   C. Renal tubular acidosis (hypokalemic)
      1. Proximal
      2. Distal
   D. Aldosterone deficiency (hyperkalemic)
      1. Primary adrenal insufficiency
      2. Hyporeninemic hypoaldosteronism
      3. Inhibitors of aldosterone effect (K^+ sparing diuretics)
         a. Spironolactone
         b. Triamterene
         c. Amiloride

TABLE 8-3 Lactic Acidosis

Type A
- Generalized seizure
- Extreme exercise
- Shock
- Cardiac arrest
- Low cardiac output
- Severe anemia
- Severe hypoxemia
- Carbon monoxide poisoning

Type B
- Sepsis
- Thiamine deficiency
- Uncontrolled diabetes mellitus
- Malignancy
- Hypoglycemia
- Drugs/toxins
  - Ethanol
  - Metformin
  - Zidovudine
  - Didanosine
  - Stavudine
  - Lamivudine
  - Zalcitabine
  - Salicylate
  - Propofol
  - Niacin
  - Isoniazid
  - Nitroprusside
  - Cyanide
  - Catecholamines
  - Cocaine
  - Acetaminophen
  - Streptozotocin
  - Pheochromocytoma
  - Sorbitol/fructose
  - Malaria
- Inborn errors of metabolism

Other
- Hepatic failure
- Respiratory or metabolic alkalosis
- Propylene glycol
- D-Lactic acidosis


Hyperchloremic metabolic acidosis
Inadequate NH_3 production
Defect in HCO_3^- reclamation
Defect in H^+ secretion
Extra-renal Loss of base Acid load
Excretion of organic anions
Kidney failure Hypoaldosteronism PHA
Proximal RTA
Distal RTA
Diarrhea Pancreatic fistula Ureteral diversions NH_4Cl/HCL NaCl load
Toluene ingestion Diabetic ketoacidosis
Urine pH < 5.5 (pH maybe > 5.5) Positive UAG
Fractional excretion HCO_3 > 15%
Urine pH > 5.5 Positive UAG
Negative UAG
High urine osmole gap

Principal Causes of Metabolic Alkalosis

I. Loss of acid (urine Cl^- <15 mEq/L)
   A. Extrarenal
      1. Vomiting
      2. Gastric drainage
   B. Renal
      1. Diuretics[a]
         a. Thiazides
         b. Furosemide
         c. Ethacrynic acid
      2. Posthypercapneic alkalosis

II. Gain of Alkali (urine Cl^- >20mEq/L)
   A. Rapid administration of $NaHCO_3$
   B. Primary mineralocorticoid excess
      1. Endogenous
         a. Primary aldosteronism
         b. Cushing's disease
      2. Exogenous
         a. High dose corticosteroid administration
         b. Disorders simulating mineralocorticoid excess[b]
            i. Licorice
            ii. Chewing tobacco
      3. Primary aldosteronism

Principle Causes of Respiratory Acidosis

I. Inhibition of respiratory center
   A. Drug overdose: opiates, etc.
   B. Oxygen therapy in chronic hypercapnia
II. Musculoskeletal disorders
   A. Muscle weakness, e.g., myasthenia gravis
   B. Extreme obesity
   C. Chest cage abnormalities, e.g., kyphoscoliosis
III. Airway obstruction
   A. Aspiration of foreign body
   B. Asthma
   C. Chronic obstructive lung disease
IV. Abnormal pulmonary capillary gas exchange
   A. Pulmonary edema
   B. Severe lung disease
V. Inadequate mechanical ventilation

Principal Causes of Respiratory Alkalosis

I. Hypoxemia
   A. Congestive heart failure
   B. Pneumonia
   C. Pulmonary emboli
   D. High altitude
II. Pulmonary disease
III. Stimulation of respiratory center
   A. Salicylate intoxication
   B. Sepsis
   C. Psychogenic hyperventilation
   D. Brain lesions
IV. "Excessive" mechanical ventilation