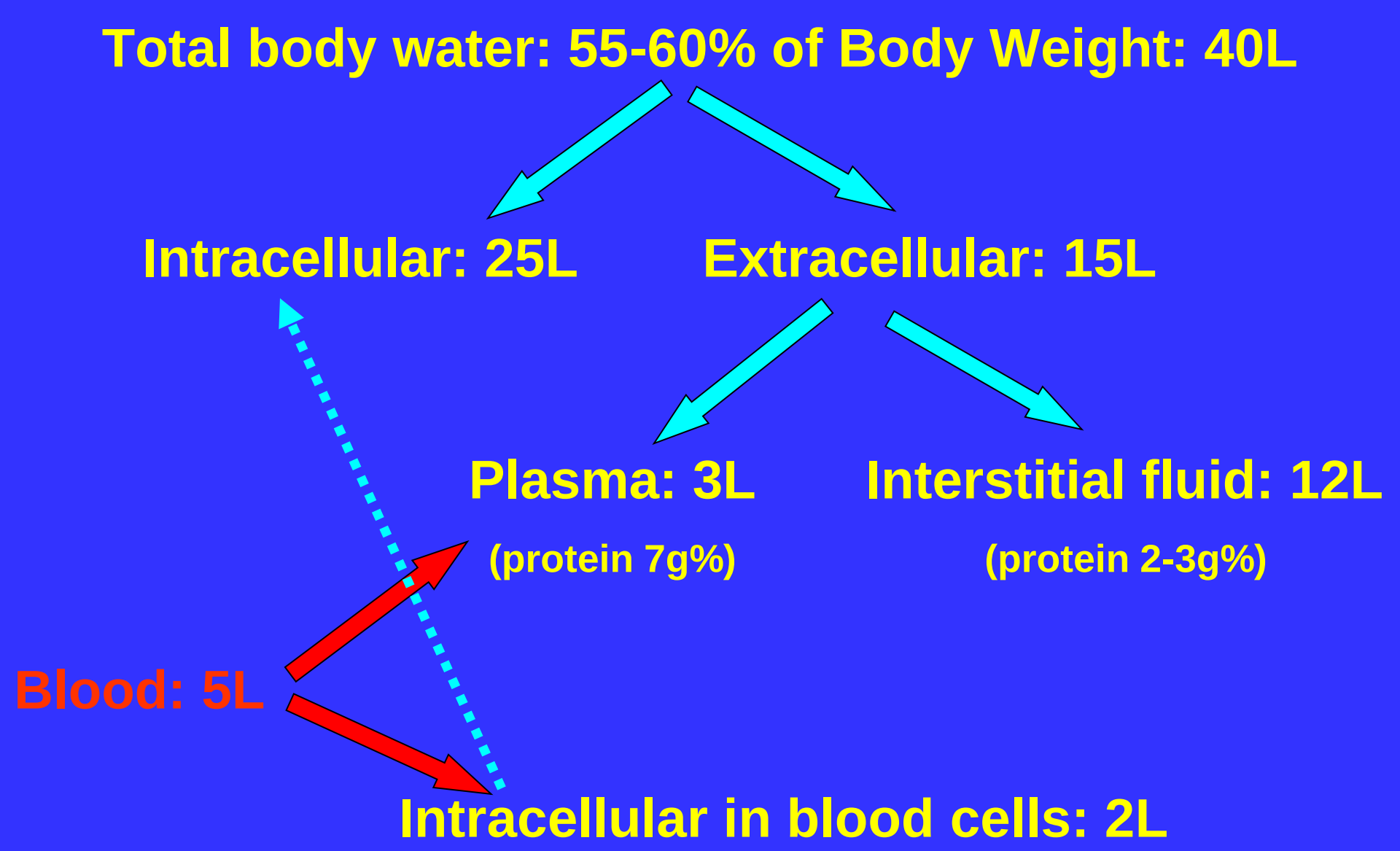
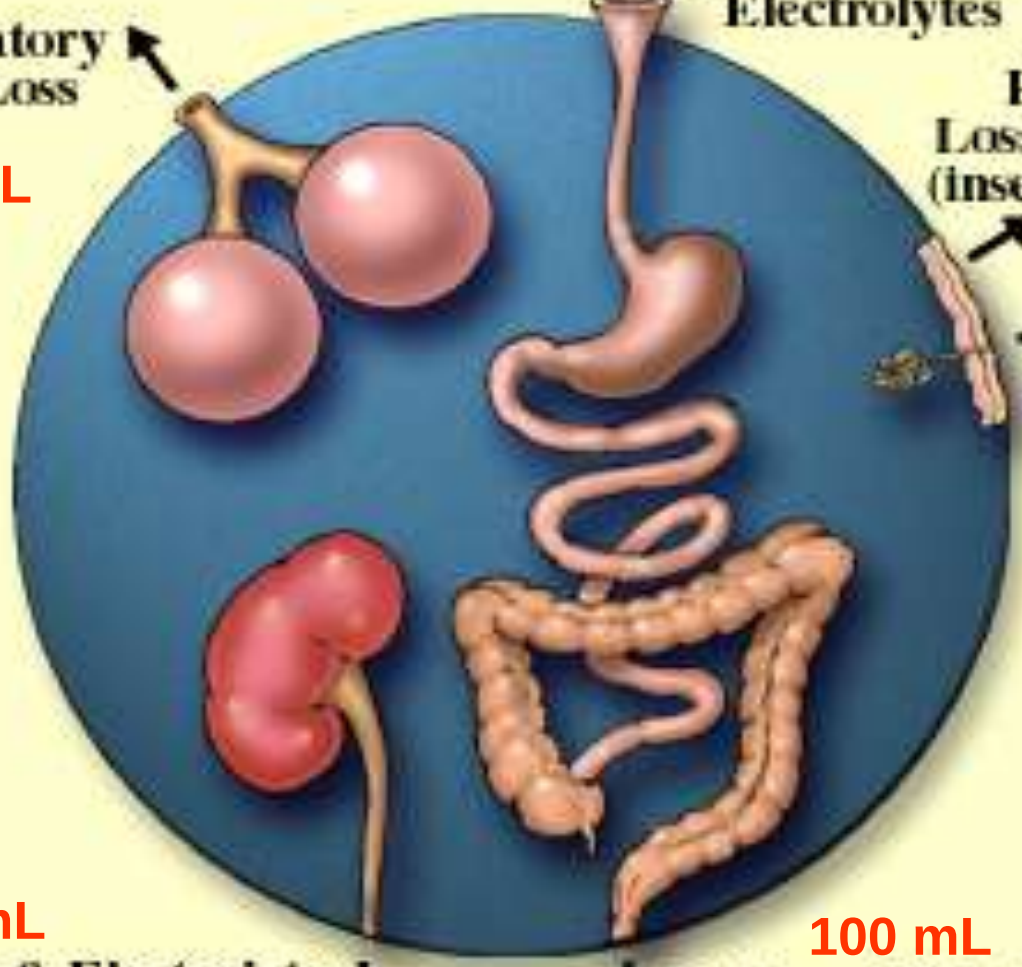
ΔΙΑΤΑΡΑΧΕΣ ΟΜΟΙΟΣΤΑΣΙΑΣ ΝΑΤΡΙΟΥ

BODY WATER DISTRIBUTION



PHYSIOLOGICAL ROUTES OF LOSS AND GAIN OF WATER & ELECTROLYTES



Gastrointestinal Gain of H_2O and Electrolytes

2100 mL

Respiratory H_2O Loss

350 mL

Evaporative H_2O Loss from Skin Surface (insensible perspiration)

350 mL

Sensible H_2O & Electrolyte Loss by Sweating

100 mL

1400 mL

Renal H_2O & Electrolyte Loss or Conservation

100 mL

H_2O and Electrolyte Loss in Stool

ELECTROLYTES

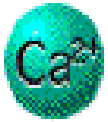
Electrolytes are charged particles **(ions)** that are dissolved in body fluids.

Electrolytes (Dissolved Ions)

Major Positive Ions **(Cations)**

Sodium ion, Na^+

Potassium ion, K^+



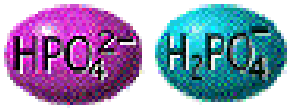
Calcium ion, Ca^{2+}

Magnesium ion, Mg^{2+}

Major Negative Ions **(Anions)**

Chloride ion, Cl^-

Bicarbonate ion, HCO_3^-



Phosphate ions, HPO_4^{2-} & $H_2PO_4^-$

Sulfate ion, SO_4^{2-}


Organic acids


Proteins

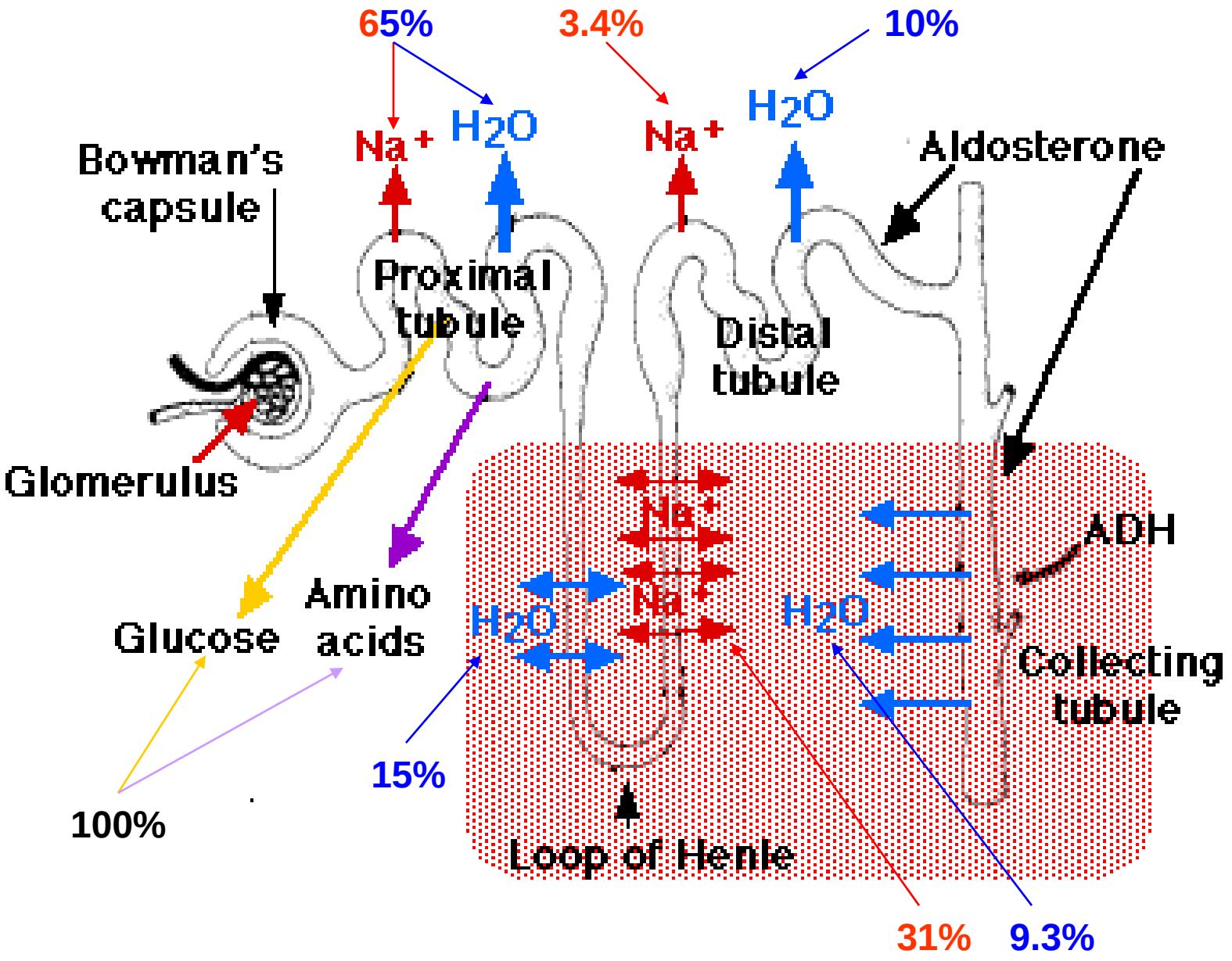
Table 12.1

Electrolyte composition of intracellular and extracellular fluids

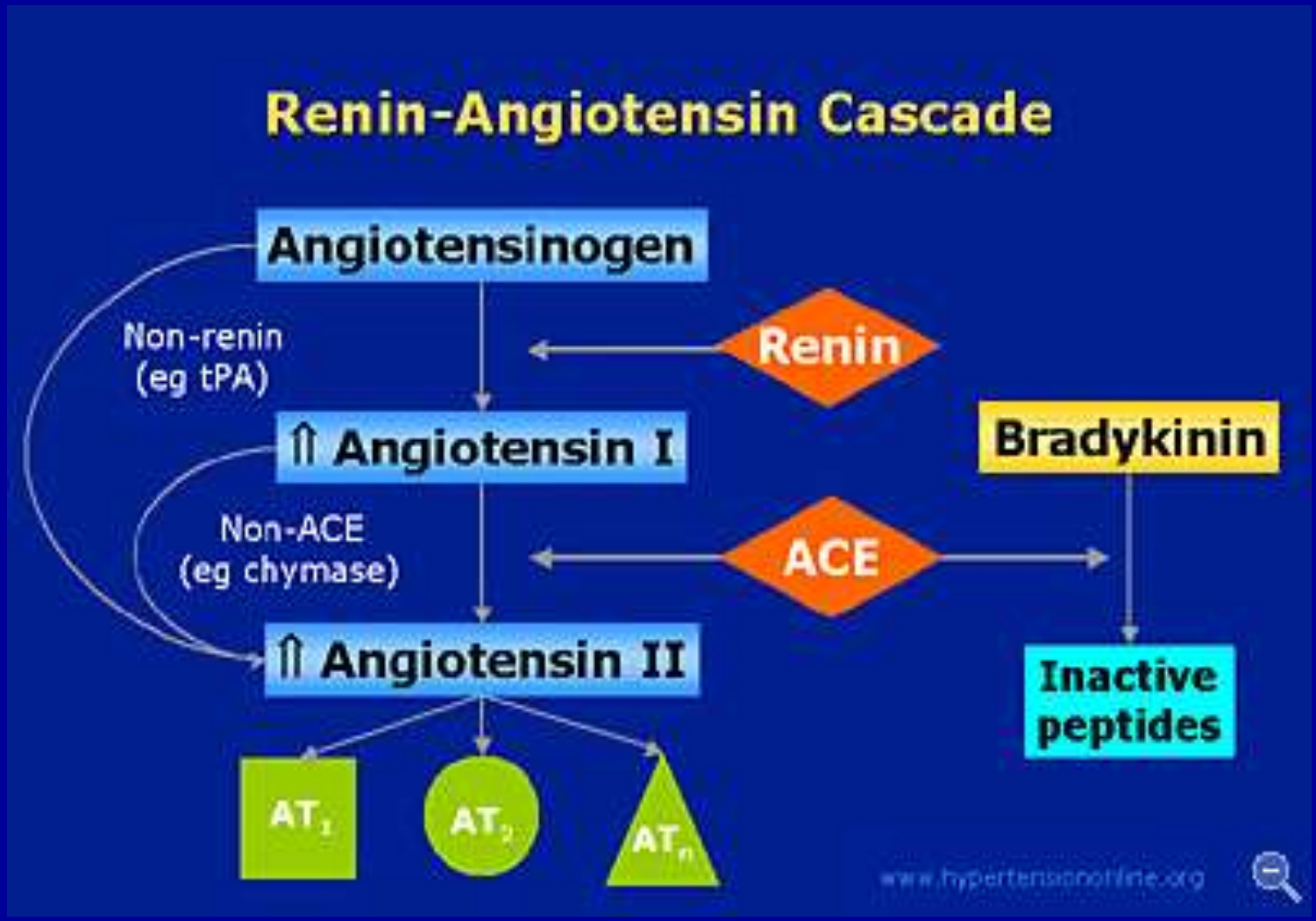
(+) (-)

	Plasma (mmol/L)	Interstitial fluid (mmol/L)	Intracellular fluid (mmol/L)
Na^+	142	144	10
K^+	4	4	160
Ca^{2+}	2.5	2.5	1.5
Mg^{2+}	1.0	0.5	13
Cl^-	102	114	2
HCO_3^-	26	30	8
PO_4^{2-}	1.0	1.0	57
SO_4^{2-}	0.5	0.5	10
Organic acid	3	4	3
Protein	16	0	55

ΥΠΟΜΝΗΣΕΙΣ ΦΥΣΙΟΛΟΓΙΑΣ



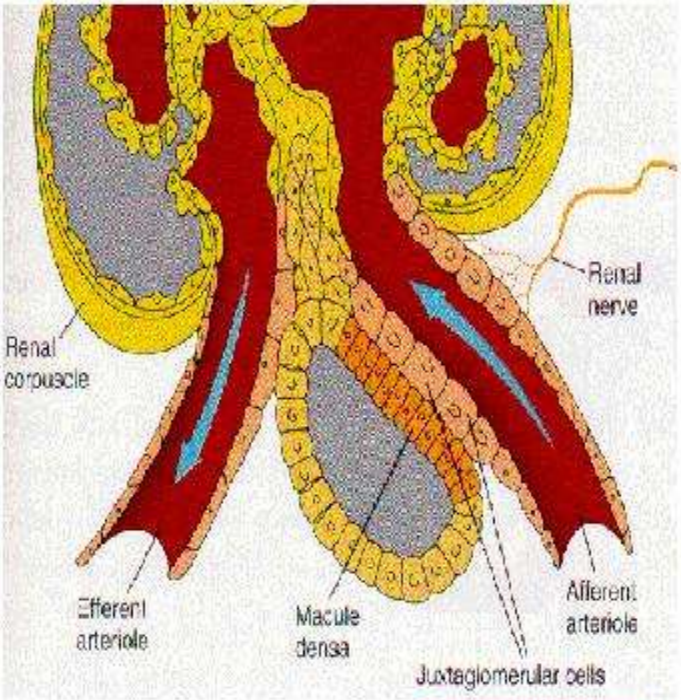
ΣΥΣΤΗΜΑ ΡΕΝΙΝΗΣ-ΑΓΓΕΙΟΤΑΣΙΝΗΣ



ΡΥΘΜΙΣΗ ΕΠΑΝΑΡΡΟΦΗΣΗΣ Na και H2O

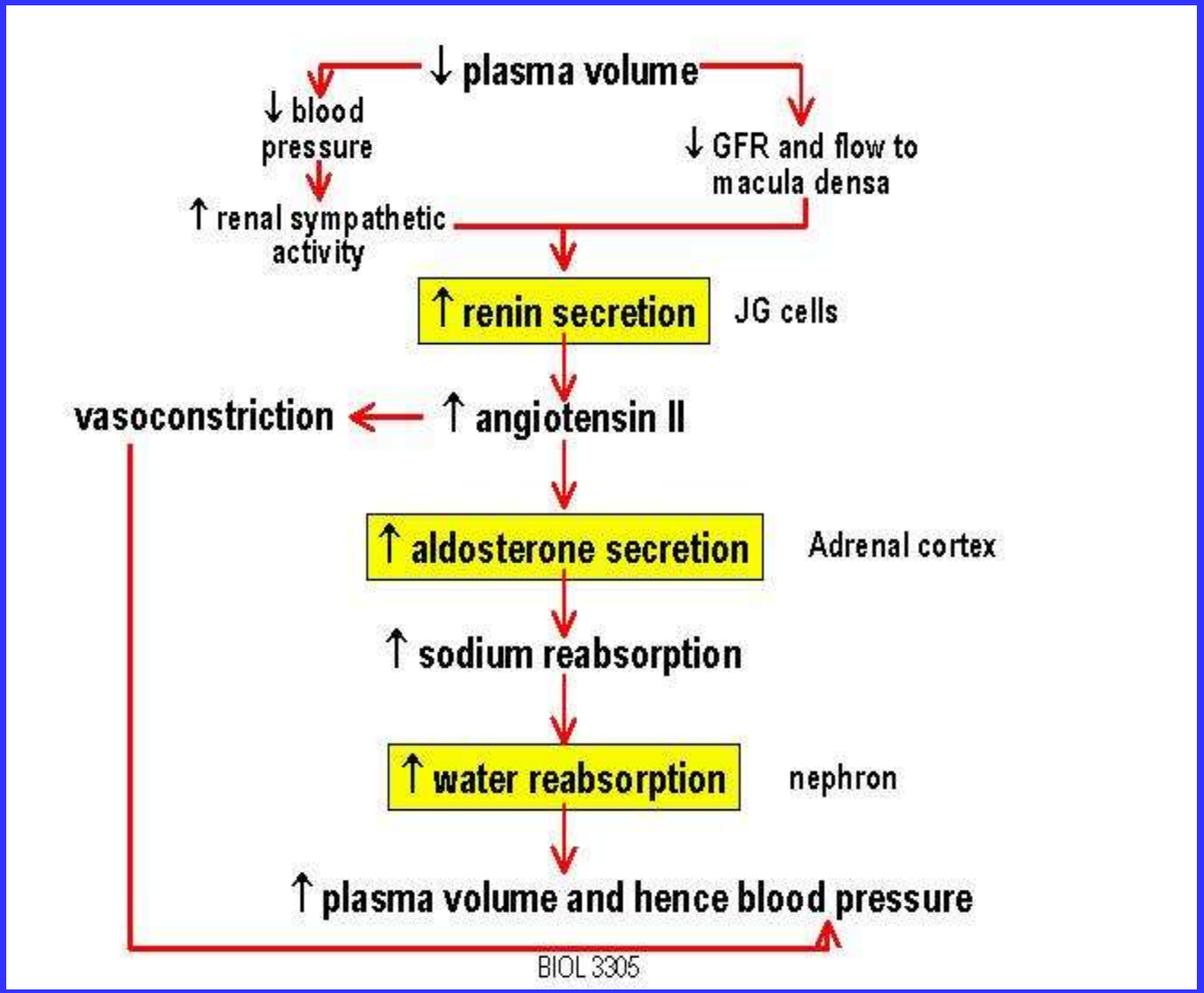
Control of Na^+ reabsorption

- Juxtaglomerular apparatus
  - Macula densa
  - Juxtaglomerular cells
- Stimulated by:
  - ↓ [Na^+]
  - ↓ blood pressure
  - ↓ blood volume
  - sympathetic stimulation
  - circulating catecholamines



See Fig. 14-24

ΡΥΘΜΙΣΗ ΕΠΑΝΑΡΡΟΦΗΣΗΣ Na και H2O



ΕΠΑΝΑΡΡΟΦΗΣΗ Na, H2O ΚΑΙ ΡΕΝΙΝΗ-AII-Aldo

- **Response:**
  - ➪ **renin release**
  - ➪ **angiotensin II**
    - ↑ blood pressure
    - stimulate aldosterone
  - ➪ **aldosterone**
    - promotes Na^+ reabsorption

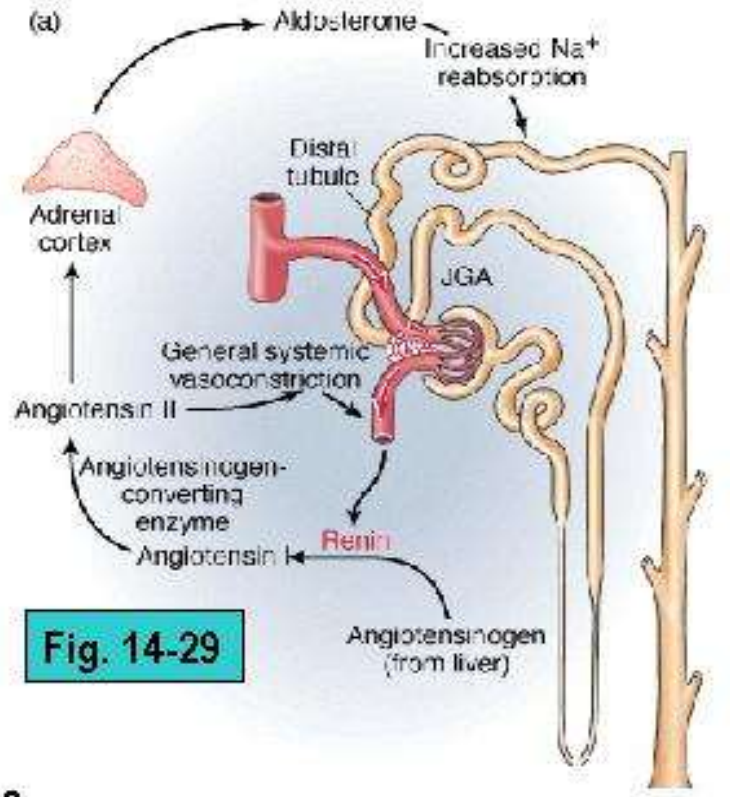
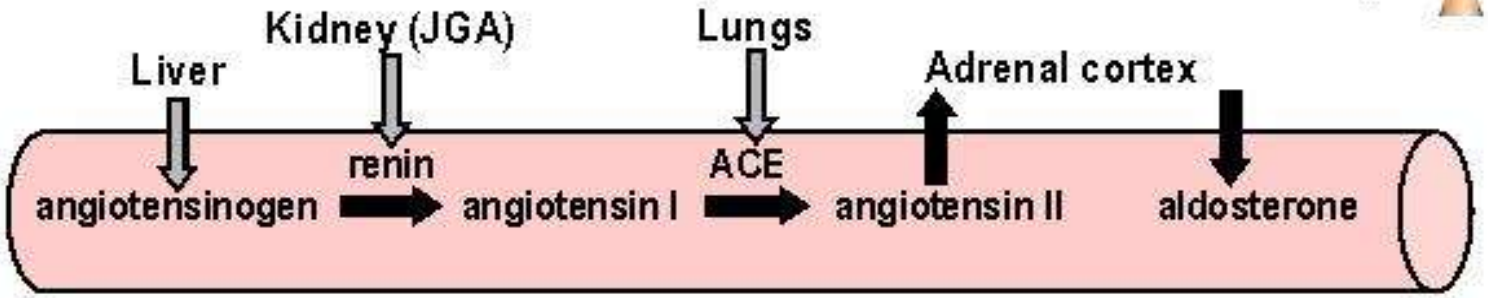


Fig. 14-29



ΡΥΘΜΙΣΗ ΕΠΑΝΑΡΡΟΦΗΣΗΣ H2O ΚΑΙ ADH

Control of water reabsorption

- Collecting duct permeability regulated by ADH
- ADH secretion controlled by:
  - ➪ plasma osmolarity
    - hypothalamic osmoreceptors
  - ➪ blood pressure
    - *via* baroreceptor reflex & atrial stretch receptors (B-type)
  - ➪ alcohol
  - ➪ ecstasy

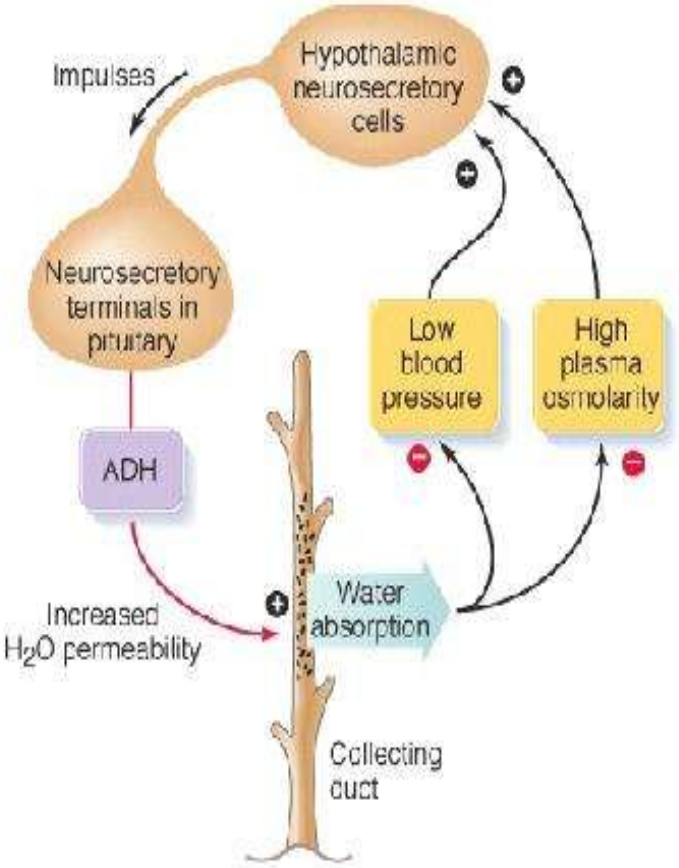


Fig. 14-35

Sodium

- Most abundant Extracellular Cation
- Reflective of relationship between total body sodium and extracellular fluid volume
- Sodium regulated by kidney excretion and reabsorption as well as endocrine regulation

Sodium (Na+) 135-145 mEq/L

Determined by Blood Chemistry

- **Major functions**
  - Water balance
  - Transmission of nerve impulses (deficits => neurological changes)
- **Na+ absorbed from food & eliminated via urine, sweat & feces**

Hyponatremia (Na+ < 135 mEq/L)

Low Sodium determined by a blood chemistry

- **Causes**
  - Water gain or sodium loss
    - GI loss (diarrhea, vomiting, N/G suctioning)
    - Urine/Renal disease
    - Psychiatric (Excessive water intake)
  - Congestive heart Failure (CHF)
- **Manifestations**
  - Water excess => rapid weight gain
  - Na+ loss => Neuro symptoms ie. irritabilitity

$$\text{Osmolality} = 2(\text{Na meq/L}) + \frac{\text{Glucose mg/dL}}{18} + \frac{\text{BUN mg/dL}}{2.8}$$

Serum Osmolality : 285-295 mosm/kg

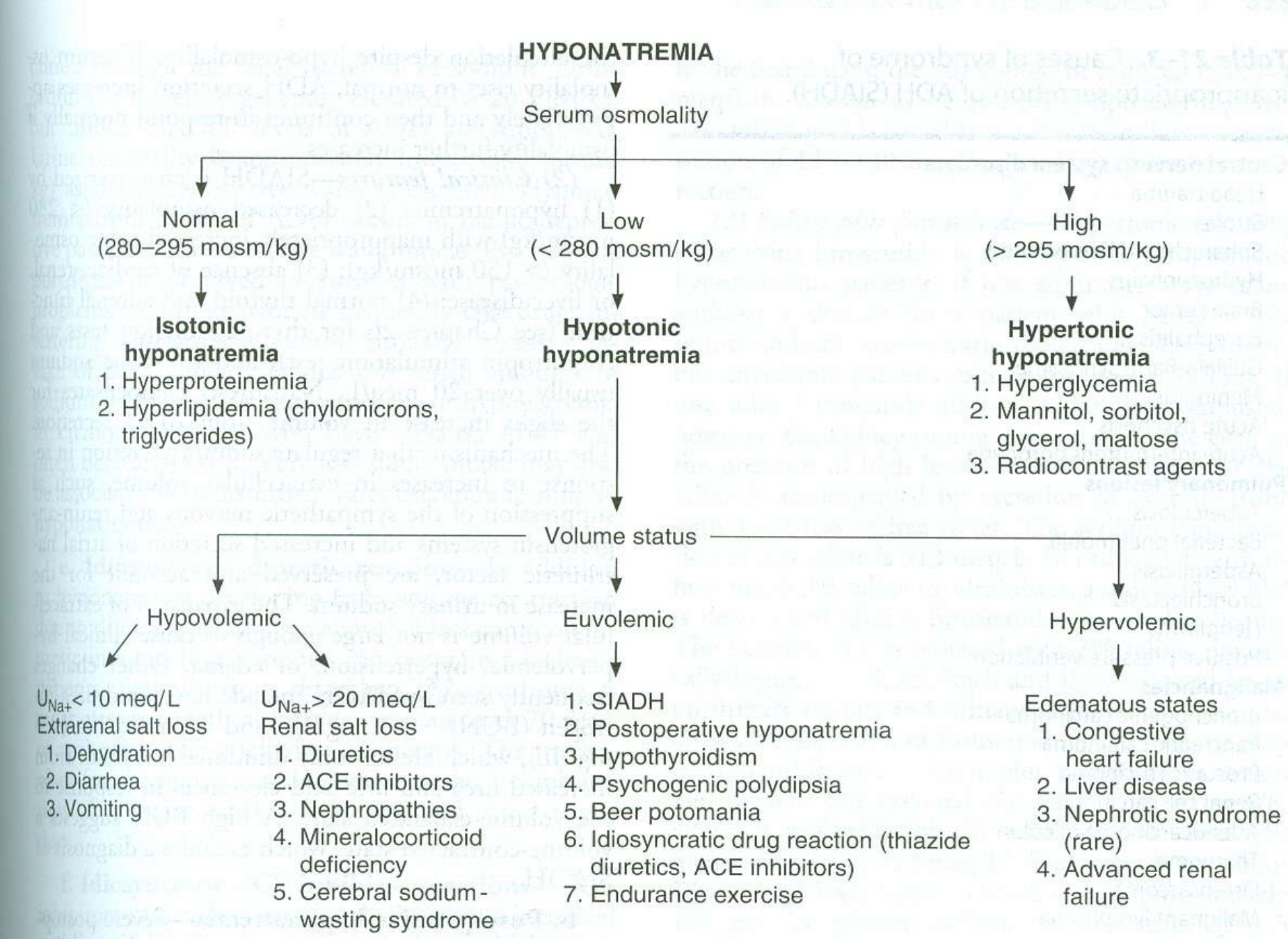


Figure 21–1. Evaluation of hyponatremia using serum osmolality and extracellular fluid volume status.

Table 12.7
Causes of hyponatraemia with normal extracellular volume

Abnormal ADH release
Vagal neuropathy (failure of inhibition of ADH release)
Deficiency of adrenocorticotrophic hormone (ACTH) or glucocorticoids (Addison's disease)
Hypothyroidism
Severe potassium depletion

Syndrome of inappropriate antidiuretic hormone
(see Table 18.36)

Major psychiatric illness
'Psychogenic polydipsia'
Nonosmotic ADH release?
Antidepressant therapy

Increased sensitivity to ADH
Chlorpropamide
Tolbutamide

ADH-like substances
Oxytocin
1-Deamino-D-arginine vasopressin (DDAVP)

Unmeasured osmotically active substances stimulating osmotic ADH release
Glucose
Alcohol
Mannitol
Sick-cell syndrome (leakage of intracellular ions)

SIADH
Syndrome of Inappropriate Anti Diuretic Hormone

The usual features are:

- Dilutional hyponatremia due to excessive water retention
- Low plasma osmolality with 'inappropriate' urine osmolality which is higher (>100 mOsm per kg) than plasma osmolality
- Continued urinary sodium excretion > 30 mmol/L
- Absence of hypokalemia (or hypotension)
- Normal renal, adrenal and thyroid function.

Table 18.36
Common causes of the syndrome of inappropriate ADH secretion (SIADH)

Tumours
Small-cell carcinoma of lung
Prostate
Thymus
Pancreas
Lymphomas

Pulmonary lesions
Pneumonia
Tuberculosis
Lung abscess

CNS causes
Meningitis
Tumours
Head injury
Subdural haematoma
Cerebral abscess
SLE vasculitis

Metabolic causes
Alcohol withdrawal
Porphyria

Drugs
Chlorpropamide
Carbamazepine
Cyclophosphamide
Vincristine
Phenothiazines

Table 12.8

Causes of hyponatraemia with decreased extracellular volume

Gut	Kidney
Vomiting	Osmotic diuresis (e.g. hyperglycaemia, severe uraemia)
Diarrhoea	Excessive use of diuretics
Haemorrhage	Adrenocortical insufficiency
	Tubulo-interstitial renal disease
	Unilateral renal artery stenosis
	Recovery phase of acute tubular necrosis

Hypernatremia (Na+ >145 mEq/L)

Sodium Excess

- **Causes p. 332**
  - Water loss or sodium gains
    - Elderly/comatose persons
    - Na intake more than water intake
      - IV's, oral
    - Diabetes insipidus (excessive fluid loss)
- **Manifestations**
  - Thirst, dry tongue
  - Restlessness
  - Weight changes

Table 12.11
Causes of hypernatraemia

ADH deficiency
Diabetes insipidus

Iatrogenic
Administration of hypertonic sodium solutions

Insensitivity to ADH (nephrogenic diabetes insipidus)
Lithium
Tetracyclines
Amphotericin B
Acute tubular necrosis

Osmotic diuresis
Total parenteral nutrition
Hyperosmolar diabetic coma

PLUS
Deficient water intake

TABLE 4
Etiologies of Hypothalamic Diabetes Insipidus

Head trauma
Postoperative (hypophysectomy)
Central nervous system tumors (primary cancer, metastatic and benign)
Infections
Tuberculosis
Syphilis
Mycoses
Toxoplasmosis
Encephalitis
Basilar meningitis
Granulomatous disease
Sarcoidosis
Histiocytosis X
Wegener's granulomatosis
Cerebrovascular disease
Aneurysm
Cavernous sinus thrombosis
Sheehan's syndrome
Cerebrovascular accident
Idiopathic
Sporadic
Familial

TABLE 5
Etiologies of Nephrogenic Diabetes Insipidus

Congenital
Vasopressin V2-receptor defect
Aquaporin-2 water channel defect
Drugs
Lithium
Amphotericin B (Fungizone)
Demeclocycline (Declomycin)
Methoxyflurane (Penthrane)
Foscarnet (Foscavir)
Obstructive uropathy

Chronic tubulointerstitial disease
Analgesic nephropathy
Sickle cell nephropathy
Multiple myeloma
Amyloidosis
Sarcoidosis
Sjögren's syndrome
Systemic lupus erythematosus
Polycystic kidney disease
Medullary cystic disease
Electrolyte disorders
Hypercalcemia
Potassium depletion

Sodium

- **Increased in:**
  - Excessive intake
  - Dehydration
  - Excessive diaphoresis
  - Diabetes Insipidus
  - Primary Aldosteronism

Sodium

- **Decreased in:**
  - Diarrhea
  - Excessive IV fluids without electrolytes
  - Inappropriate secretion of ADH
  - Hyperglycemia
  - Hyperproteinemia
  - Cirrhosis with Ascites
  - Renal Insufficiency and CHF