ELECTROLYTE COMPOSITION

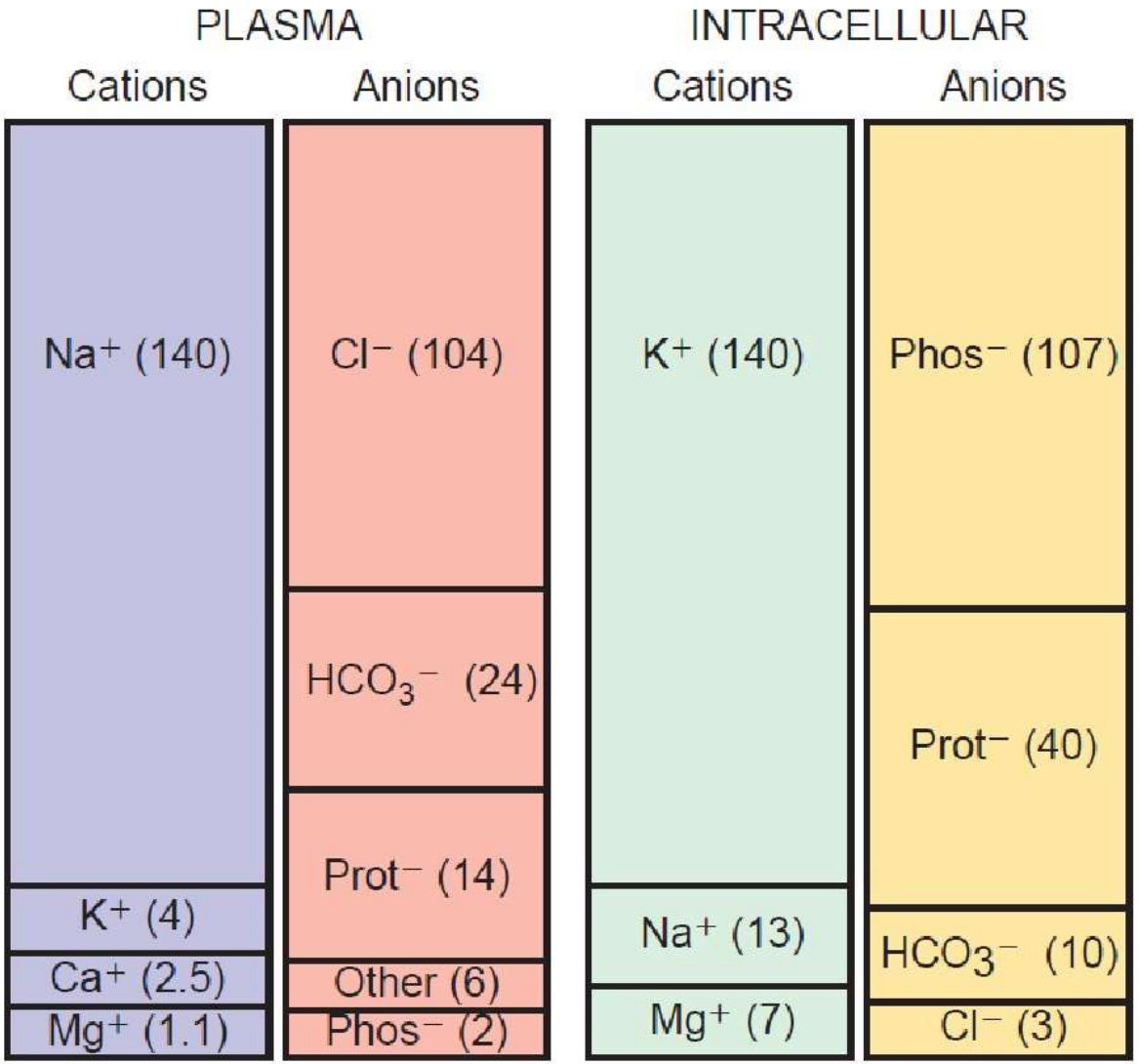

Fig. 68.3 Concentrations of the major cations and anions in the intracellular space and the plasma, expressed in mEq/L.

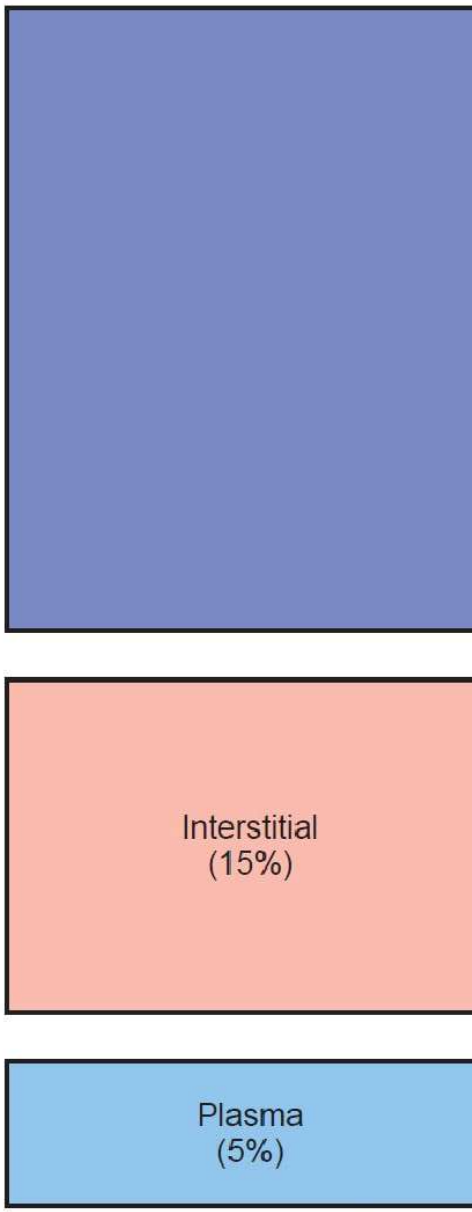

Fig. 68.2 Compartments of total body w
of body weight, in an older child or adu

$$\text{Effective osmolality} = 2 \times [\text{Na}] + [\text{glucose}]/18$$

$$[\text{Na}]_{\text{corrected}} = [\text{Na}]_{\text{measured}} + 1.6 \times ([\text{glucose}] - 100\ \text{mg/dL})$$

$$\text{Osmolality} = 2 \times [\text{Na}] + [\text{glucose}]/18 + [\text{BUN}]/2$$

HYPERNATREMIA is a [Na_+] >145 mEq/L , Sometimes defined as >150 mEq/L.

There are 3 basic mechanisms of hypernatremia (Table 68.1).

Table 68.1 Causes of Hypernatremia

EXCESSIVE SODIUM
Improperly mixed formula
Excess sodium bicarbonate
Ingestion of seawater or sodium chloride
Intentional salt poisoning (child abuse or Munchausen syndrome by proxy)
Intravenous hypertonic saline
Hyperaldosteronism

WATER DEFICIT
Nephrogenic Diabetes Insipidus
Acquired
X-linked (OMIM 304800)
Autosomal recessive (OMIM 222000)
Autosomal dominant (OMIM 125800)

Central Diabetes Insipidus
Acquired
Autosomal recessive (OMIM 125700)
Autosomal dominant (OMIM 125700)
Wolfram syndrome (OMIM 222300/598500)

Increased Insensible Losses
Premature infants
Radiant warmers
Phototherapy
Inadequate intake:
Ineffective breastfeeding
Child neglect or abuse
Adipsia (lack of thirst)

WATER AND SODIUM DEFICITS
Gastrointestinal Losses
Diarrhea
Emesis/nasogastric suction
Osmotic cathartics (lactulose)

Cutaneous Losses
Burns
Excessive sweating

Renal Losses
Osmotic diuretics (mannitol)
Diabetes mellitus
Chronic kidney disease (dysplasia and obstr
Polyuric phase of acute tubular necrosis
Postobstructive diuresis

HYPONATREMIA: serum sodium level <135 mEq/L.

Table 68.2 | Causes of Hyponatremia

PSEUDOHYPONATREMIA
Hyperlipidemia
Hyperproteinemia

HYPEROSMOLALITY
Hyperglycemia
Iatrogenic (mannitol, sucrose, glycine)

HYPOVOLEMIC HYPONATREMIA

EXTRARENAL LOSSES
Gastrointestinal (emesis, diarrhea)
Skin (sweating or burns)
Third space losses (bowel obstruction, peritonitis, sepsis)

RENAL LOSSES
Thiazide or loop diuretics
Osmotic diuresis
Postobstructive diuresis
Polyuric phase of acute tubular necrosis
Juvenile nephronophthisis (OMIM 256100/606966/602088/604387/611498)
Autosomal recessive polycystic kidney disease (OMIM 263200)
Tubulointerstitial nephritis
Obstructive uropathy
Cerebral salt wasting
Proximal (type II) renal tubular acidosis (OMIM 604278)*
Lack of aldosterone effect (high serum potassium):
- Absence of aldosterone (e.g., 21-hydroxylase deficiency [OMIM 201910])
- Pseudohypoaldosteronism type I (OMIM 264350/177735)
- Urinary tract obstruction and/or infection
- Addison disease

EUVOLEMIC HYPONATREMIA
Syndrome of inappropriate antidiuretic hormone secre
Nephrogenic syndrome of inappropriate antidiuresis (C
Desmopressin acetate
Glucocorticoid deficiency
Hypothyroidism
Antidepressant medications
Water intoxication
- Iatrogenic (excess hypotonic intravenous fluids)
- Feeding infants excessive water products
- Swimming lessons
- Tap water enema
- Child abuse
- Psychogenic polydipsia
- Diluted formula
- Beer potomania
- Exercise-induced hyponatremia

HYPERVOLEMIC HYPONATREMIA
Heart failure
Cirrhosis
Nephrotic syndrome
Acute, chronic kidney injury
Capillary leak caused by sepsis
Hypoalbuminemia caused by gastrointestinal disease (
- losing enteropathy)

*Most cases of proximal renal tubular acidosis are not caused b
genetic disorder. Proximal renal tubular acidosis is usually part
syndrome, which has multiple etiologies.

Table 68.3 Diagnostic Criteria for Syndrome of Inappropriate Antidiuretic Hormone Secretion

- Absence of:
  Renal, adrenal, or thyroid insufficiency
  Heart failure, nephrotic syndrome, or cirrhosis
  Diuretic ingestion
  Dehydration
- Urine osmolality >100 mOsm/kg (usually > plasma)
- Serum osmolality <280 mOsm/kg and serum sodium <135 mE
- Urine sodium >30 mEq/L
- Reversal of "sodium wasting" and correction of hyponatremia with water restriction

Diabetes Insipidus

Table 574.1 Causes of Hypotonic Polyuria

CENTRAL (NEUROGENIC) DIABETES INSIPIDUS

Congenital (congenital malformations, autosomal dominant, arginine vasopressin [AVP] neurophysin gene mutations)
Drug or toxin induced (ethanol, diphenylhydantoin, snake venom)
Granulomatous (histiocytosis, sarcoidosis)
Neoplastic (craniopharyngioma, germinoma, lymphoma, leukemia, meningioma, pituitary tumor; metastases)
Infectious (meningitis, tuberculosis, encephalitis)
Inflammatory, autoimmune (lymphocytic infundibuloneurohypophysitis)
Trauma (neurosurgery, deceleration injury)
Vascular (cerebral hemorrhage or infarction, brain death)
Idiopathic

OSMORECEPTOR DYSFUNCTION

Granulomatous (histiocytosis, sarcoidosis)
Neoplastic (craniopharyngioma, pinealoma, meningioma, metastases)
Vascular (anterior communicating artery aneurysm or ligation, intrahypothalamic hemorrhage)
Other (hydrocephalus, ventricular or suprasellar cyst, trauma, degenerative diseases)
Idiopathic

INCREASED AVP METABOLISM

Pregnancy

NEPHROGENIC DIABETES INSIPIDUS

Congenital (X-linked recessive, AVP V2 rece autosomal recessive or dominant, aquapo gene mutations)
Drug induced (demeclocycline, lithium, cisp
Hypercalcemia
Hypokalemia
Infiltrating lesions (sarcoidosis, amyloidosis)
Vascular (sickle cell anemia)
Mechanical (polycystic kidney disease, bilat
Solute diuresis (glucose, mannitol, sodium,
Idiopathic

PRIMARY POLYDIPSIA

Psychogenic (schizophrenia, obsessive-com
Dipsogenic (downward resetting of thirst th similar lesions, as with central DI)

DI, Diabetes insipidus.

Table 575.1 Differential Diagnosis of Hyponatremia

DISORDER	INTRAVASCULAR VOLUME STATUS	URINE SODIUM
Systemic dehydration	Low	Low
Decreased effective plasma volume	Low	Low
Primary salt loss (nonrenal)	Low	Low
Primary salt loss (renal)	Low	High
SIADH	High	High
Cerebral salt wasting	Low	Very high
Decreased free water clearance	Normal or high	Normal or high
Primary polydipsia	Normal or high	Normal
Runner's hyponatremia	Low	Low
NSIAD	High	High
Pseudohyponatremia	Normal	Normal
Factitious hyponatremia	Normal	Normal

NSIAD, Nephrogenic syndrome of inappropriate antidiuresis; *SIADH*, syndrome of inappropriate antidiuretic hormone secretion.

Table 575.2 Clinical Parameters to Distinguish Among Syndrome of Inappropriate Antidiuretic Hormo Cerebral Salt Wasting, and Central Diabetes Insipidus

CLINICAL PARAMETER	SIADH	CEREBRAL SALT WASTING
Serum sodium	Low	Low
Urine output	Normal or low	High
Urine sodium	High	Very high
Intravascular volume status	Normal or high	Low
Vasopressin level	High	Low

DI, Diabetes insipidus; *SIADH*, syndrome of inappropriate antidiuretic hormone secretion.

Table 575.3 Disorders Associated With Syndrome of Inappropriate Antidiuretic Hormone Secretion

CARCINOMAS	PULMONARY DISORDERS	CENTRAL NERVOUS SYSTEM DISORDERS	OTHER DI
Thymoma	Viral pneumonia	Encephalitis (viral or bacterial)	AIDS
Lymphoma	Bacterial pneumonia	Meningitis (viral, bacterial, tuberculous, fungal)	Prolonged exer
Ewing sarcoma	Pulmonary abscess	Head trauma	Idiopathic (in ol
Oropharyngeal tumor	Tuberculosis	Brain abscess	Nephrogenic
	Aspergillosis	Guillain-Barré syndrome	Acute intermitte
	Positive pressure breathing	Subarachnoid hemorrhage or subdural hematoma	
	Asthma	Cerebellar and cerebral atrophy	
	Pneumothorax	Cavernous sinus thrombosis	
	Cystic fibrosis	Neonatal hypoxia	
		Shy-Drager syndrome	
		Rocky Mountain spotted fever	
		Delirium tremens	
		Cerebrovascular accident (cerebral thrombosis or hemorrhage)	
		Acute psychosis	
		Peripheral neuropathy	
		Multiple sclerosis	