About this Chapter

- Fluid and electrolyte homeostasis
- Water balance
- Sodium balance and ECF volume
- Potassium balance
- Acid-base balance

Fluid and Electrolyte Homeostasis

Why we need to worry about ionic concentrations

Na^+ and water	ECF volume and osmolarity
K^+	Neuron and Muscle function
Ca^{2+}	Exocytosis, muscle contractions, and other functions
H^+ and HCO_3^-	pH balance
Body must maintain mass balance	Excretion routes: kidney and lungs

Fluid and Electrolyte Homeostasis

- The body's integrated responses to changes in blood volume and blood pressure

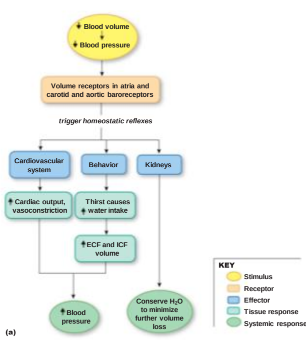


Figure 20-1a

Fluid and Electrolyte Homeostasis

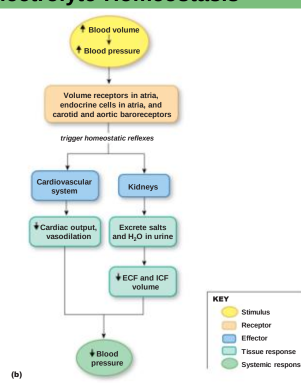


Figure 20-1b

Water Balance (~ 50% of body wieght)

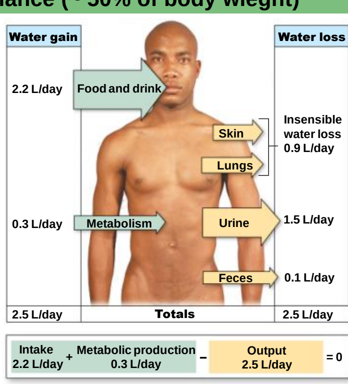


Figure 20-2

Water Balance

- The kidneys conserve volume but cannot replace lost volume

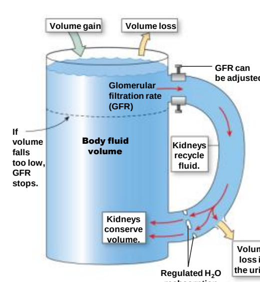
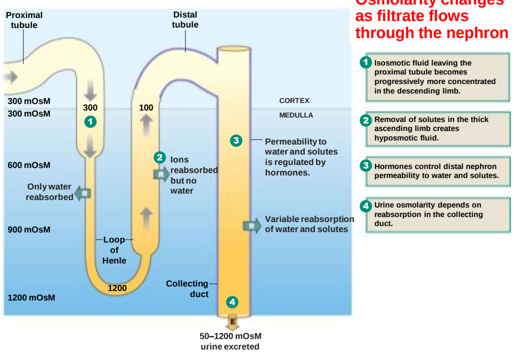


Figure 20-3

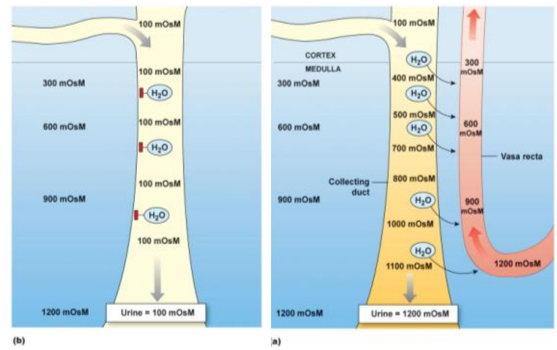
Urine Concentration

Osmolarity changes as filtrate flows through the nephron



Water Reabsorption and Vasopressin (ADH)

Without ADH | With ADH



Water Reabsorption

- **Vasopressin causes insertion of water pores into the apical membrane**

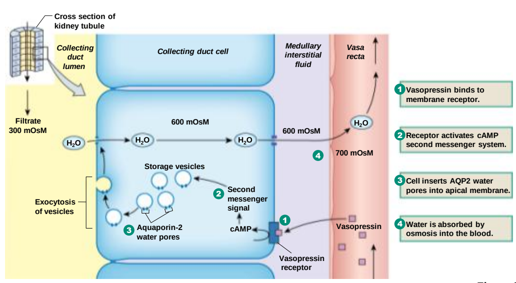


Figure 20-6

Factors Affecting Vasopressin Release

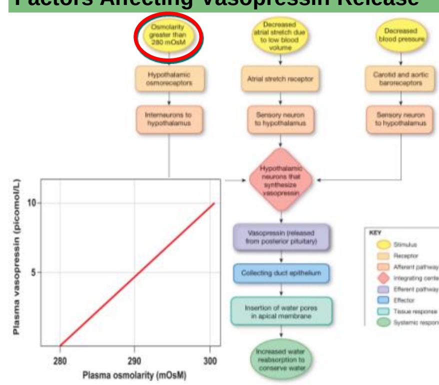
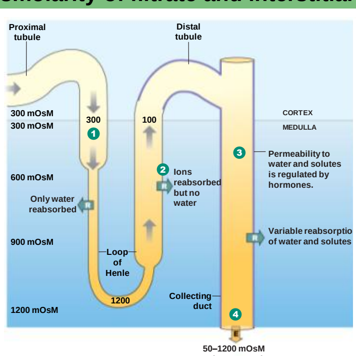


Figure 20-7

Osmolarity of filtrate and interstitial tissue



How do we maintain the concentraion gradients for movement of water out of the tubule?

If water is to move out of tubule (be reabsorbed) osmolarity must be greater in interstitium than in tubule!!!!!

Why doesn't interstitium become dilute if water keeps leaving tubule?

Countercurrent Heat Exchanger

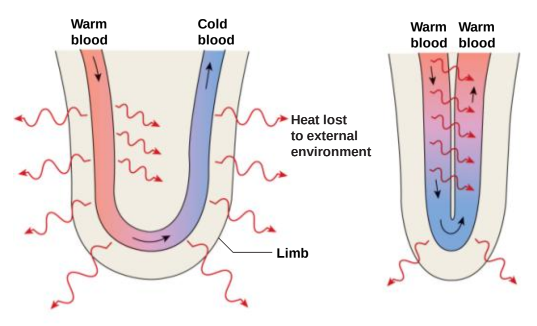
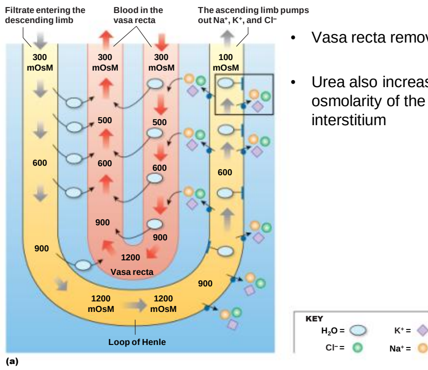


Figure 20-9

Counter Current exchange system (medulla)



- Vasa recta removes water
- Urea also increases the osmolarity of the medullary interstitium

Figure 20-10a

How the Ions are reabsorbed in ascending loop

- **Active reabsorption**
- **Transport driven by Na+/K+ pump**
  - **Na+ moves down gradient from lumen to cell**
  - **K+ and 2C- symported with the Na+**
  - **Cl- and K+ enter interstitium via channels or cotransport**
- **Note: cells not permeable to water here**

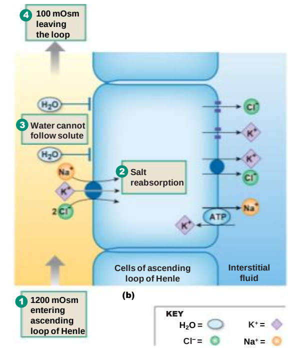


Figure 20-10b

Sodium Balance

- Homeostatic responses to salt ingestion

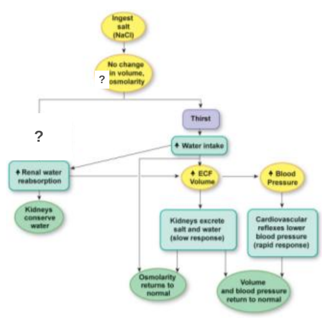


Figure 20-11

Aldosterone Controls Sodium Balance

Na+ reabsorption and K+ secretion

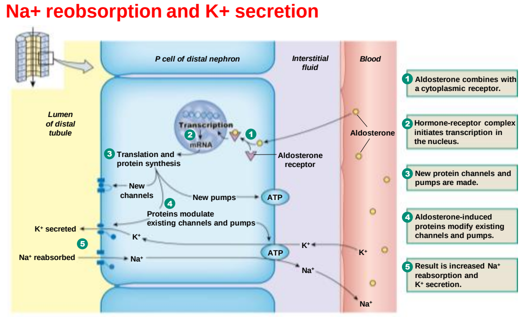
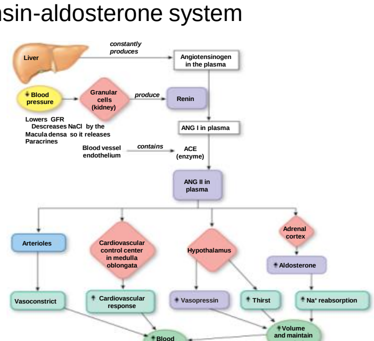


Figure 20-12

Sodium Balance

Renin-angiotensin-aldosterone system



Sodium Balance

- Natriuretic peptides promote Na+ and water excretion
- Does opposite of Aldosterone

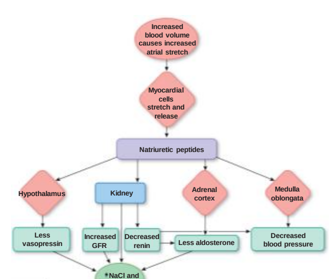
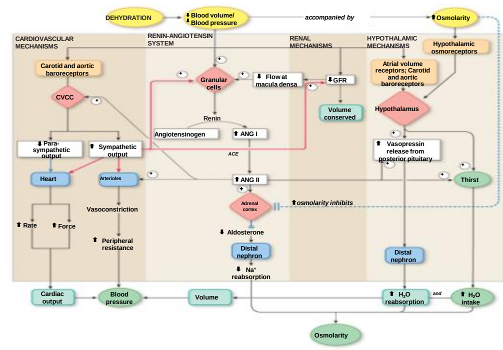


Figure 20-15

Potassium Balance (only 2% of K+ is in ECF)

- **Most K+ is reobsorbed in proximal tubule**
- **K+ is secreted in the Distal tube (= dietary intake)**
- **Regulatory mechanisms keep plasma potassium in narrow range**
  - **Aldosterone plays a critical role**
- **Hypokalemia**
  - **Muscle weakness and failure of respiratory muscles and the heart**
  - **Stop releasing aldosterone**
- **Hyperkalemia**
  - **Can lead to cardiac arrhythmias**
  - **Release aldosterone**
- **Causes include kidney disease, diarrhea, and diuretics**

Volume and Osmolarity (Dehydration)



Acid-Base Balance

- Normal pH of plasma is 7.38–7.42
- H^+ concentration is closely regulated
  - Changes can alter tertiary structure of proteins
- Abnormal pH affects the nervous system
  - Acidosis: neurons become less excitable and CNS depression
  - Alkalosis: hyperexcitable
- pH disturbances
  - Associated with K^+ disturbances

Acid-Base Balance

- Hydrogen ion and pH balance in the body

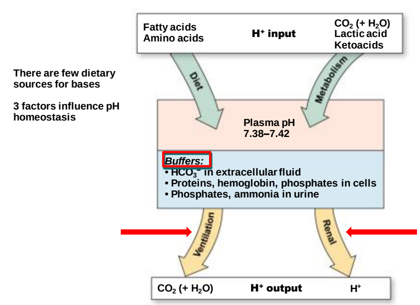


Figure 20-18

pH Homeostasis depends on

- **Buffers**
  - Moderate changes in pH
  - Combines with or releases H^+
  - Cellular proteins, phosphate ions, and hemoglobin
- **Ventilation**
  - Rapid response
  - 75% of disturbances
- **Renal regulation** (Slowest of the three mechanisms)
  1. Directly by excreting or reabsorbing H^+
  2. Indirectly by change in rate at which **HCO_3^-** buffer is reabsorbed or excreted

pH Disturbances

- – don't forget the reflex pathway for respiratory compensation of metabolic acidosis
- But were talking kidneys today

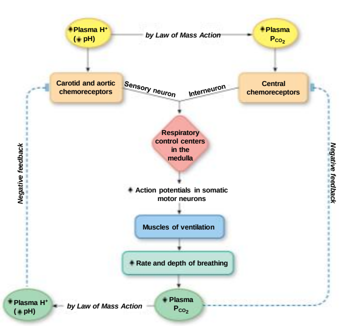


Figure 20-19

pH Disturbances (General)

- What happens during acidosis (low pH)
  - H+ secreted is buffered in urine
  - Bicarbonate ions reabsorbed to buffer H+ in blood

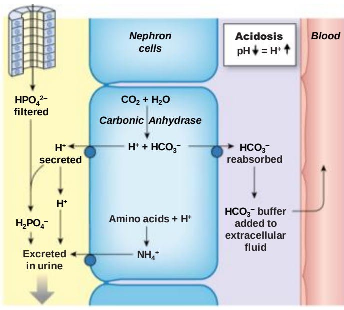


Figure 20-20

Renal Compensation: Transporters

- Apical Na^+-H^+ exchanger (NHE)
- Basolateral Na^+-HCO_3^- symport
- H^+-ATPase
- H^+-K^+-ATPase
- Na^+-NH_4^+ antiport

Renal Compensation Proximal tubule

H^+ secretion and the reabsorption of filtered HCO_3^-

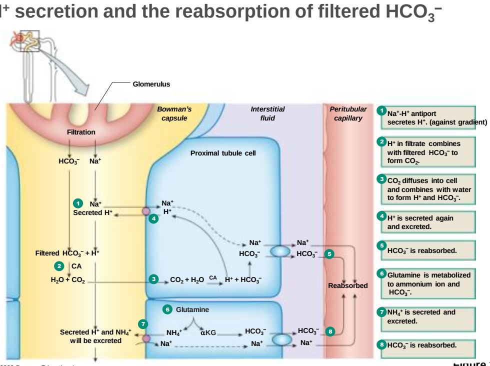
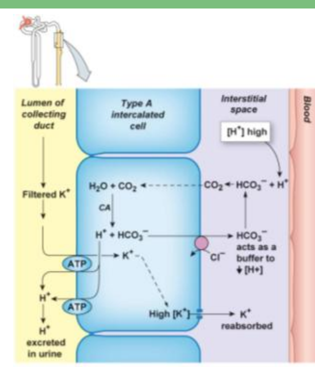


Figure 20-21

Intercalated Cells

- Type A intercalated cells function in acidosis

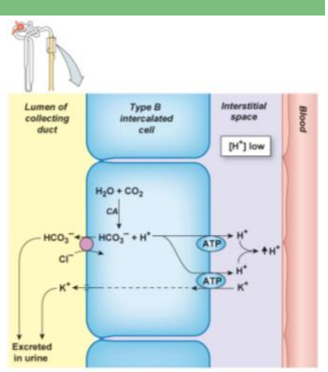


(a) Type A intercalated cell function in acidosis.

Figure 20-22a

Intercalated Cells

- Type B intercalated cells function in alkalosis



(b) Type B intercalated cell function in alkalosis.

Figure 20-22b

Acid-Base Balance

TABLE 20-2 Plasma P_{CO_2}, Ions, and pH in Acid-Base Disturbances

DISTURBANCE	P_{CO_2}	H^+	PH	HCO_3^-
Acidosis				
Respiratory	↑	↑	↓	↑
Metabolic	Normal* or ↓	↑	↓	↓
Alkalosis				
Respiratory	↓	↓	↑	↓
Metabolic	Normal* or ↑	↓	↑	↑

* These values are different from what you would expect from the law of mass action because almost instantaneous respiratory compensation keeps P_{CO_2} from changing significantly.

Table 20-2

Summary

- Fluid and electrolyte homeostasis
- Water balance
  - Vasopressin, aquaporin, osmoreceptors, countercurrent multiplier, and vasa recta
- Sodium balance
  - Aldosterone, principal cells, ANG I and II, renin, angiotensinogen, ACE, and ANP
- Potassium balance
  - Hyperkalemia and hypokalemia

Summary

- Behavioral mechanisms
- Integrated control of volume and osmolarity
- Acid-base balance
  - Buffers, ventilation, and kidney
  - Acidosis and alkalosis
  - Intercalated cells