INTRODUCTION

A. In the context of physiology and medicine, the term "electrolytes" refers to the small inorganic ions prevalent in body fluids and important in normal function: mainly sodium (Na^+), chloride (Cl^-), potassium (K^+), bicarbonate (HCO_3^-), hydrogen ion (H^+), and sometimes calcium (Ca^{2+}) and phosphate (PO_3)

B. The volume of the extracellular fluid (ECF) depends on the sodium content of the body because Na^+ and its salts (e.g. NaCl, $NaHCO_3$) are the major osmotic solutes in the ECF (extracellular fluid)

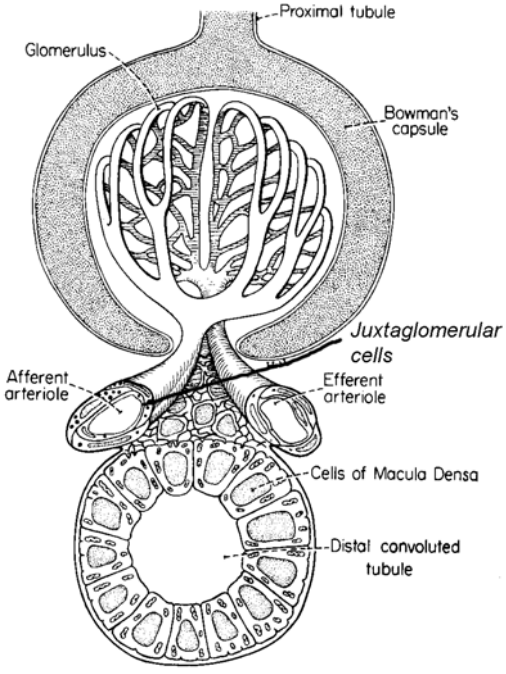
C. Renal excretion of Na^+, K^+, and Cl^- are controlled by

1. renal sympathetics: increase Na reabsorption
2. Aldosterone: increase Na reabsorption, increase K secretion/excretion
3. Atrial Natriuretic Peptide (ANP): decrease Na reabsorption

RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM

A. Renin

1. Synthesized by and released from the juxtaglomerular cells (modified smooth muscle cells) of the renal afferent arteriole



2. Release controlled by

   a. renal arterial/arteriolar hydrostatic pressure: P ⇓ ⇒ renin ⇑; mechanism: decreased stretch of afferent arteriole granular cells

   b. renal sodium at the distal tubule macula densa: Na^+ ⇓ ⇒ renin ⇑

   c. renal sympathetic activation ⇒ renin ⇑, due to:

      a. fall in "central venous pressure", as mediated by the low-pressure (atrial & venous) baroreceptors; very sensitive

      b. decrease in systemic arterial pressure; mediated by carotid and aortic baroreceptors; less sensitive

3. Action: conversion of Angiotensinogen to Angiotensin I

RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM (continued)

B. Angiotensin

1. Originates from an inactive precursor -- Angiotensinogen -- produced in the liver and circulating in the blood

2. In the presence of Renin, Angiotensinogen is converted to Angiotensin I (A-I)

3. Angiotensin-I is essentially biologically inactive

4. Angiotensin-I is converted to Angiotensin II (A-II) in the presence of Angiotensin Converting Enzyme (ACE), an enzyme which is present in the capillary endothelium, especially in the pulmonary circulation

5. actions of Angiotensin-II

   a. causes release of Aldosterone from the adrenal cortex

   b. at high concentrations, can cause general vasoconstriction, leading to increased systemic arterial blood pressure (can cause to systemic hypertension)

   c. increases proximal tubule Na^+ and Na^+-linked HCO_3 reabsorption

   d. contributes to thirst sensation

C. Aldosterone

1. Synthesized by and released from the adrenal cortex

2. Release stimulated by
   a. Angiotensin II
   b. plasma potassium: $K^+ \Uparrow \Rightarrow$ aldosterone $\Uparrow$ (local adrenal effect)

3. Steroid structure, so termed a "mineralocorticoid"

4. Action: increase active transport of Na-K-ATPase pump, particularly at the distal tubule and collecting duct (where it binds to a corticoid receptor), leading to increased Na reabsorption and K excretion by the kidney (also capable of stimulating Na-K-ATPase transport in some other tissues)

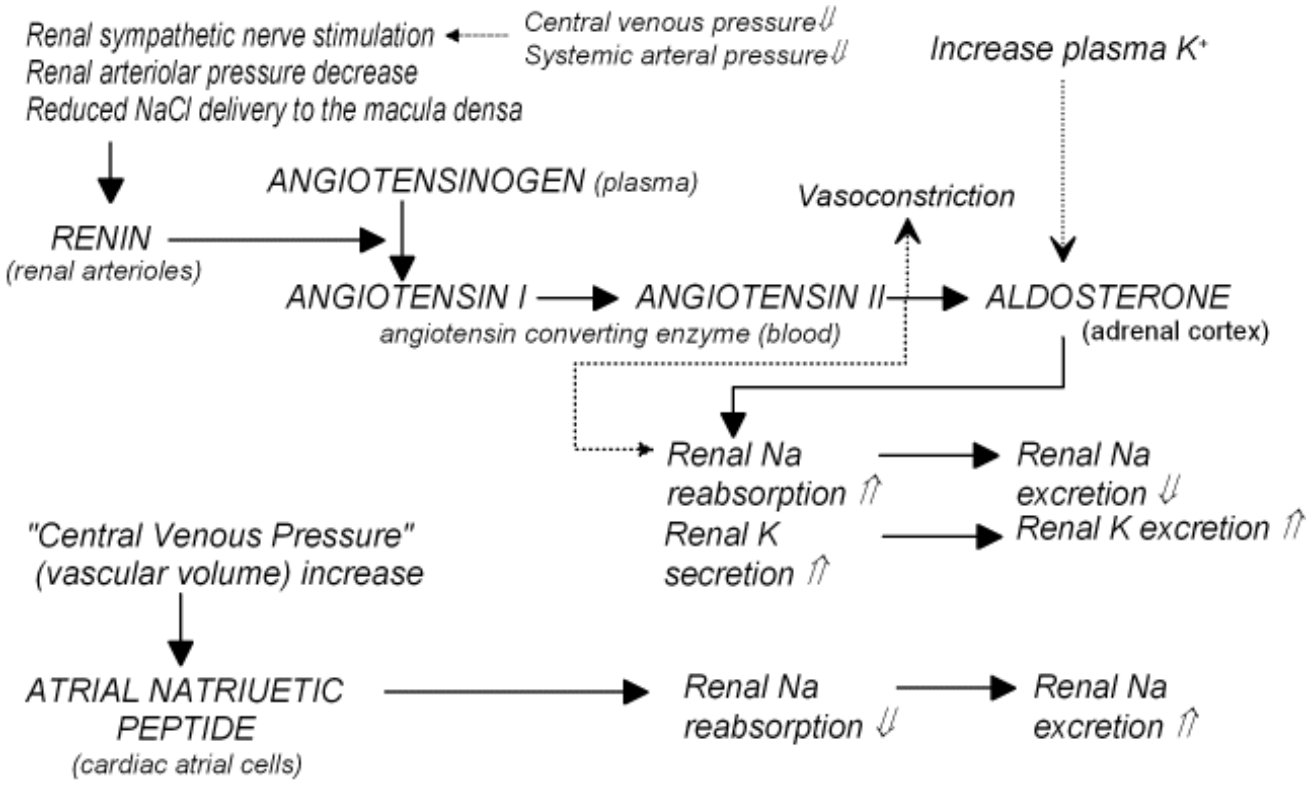
ATRIAL NATRIURETIC PEPTIDE (ANP)

A. Synthesis: synthesized by and released from atrial myocardial cells

B. Actions: increase renal Na excretion by
   1. inhibits Na active transport in the kidney
   2. inhibits renin and aldosterone secretion

C. Control: released by atrial stretch, when atrial (or "central venous pressure") increases

SUMMARY OF CONTROL



SODIUM AND EXTRACELLULAR VOLUME REGULATION

A. Relation of sodium to extracellular volume

1. Na^+ and associated anions (Cl^- and HCO_3^-) account for about 90% of the osmotic activity of extracellular fluid

2. changes in Na^+ and associated ion content of the body cause changes in extracellular volume by the osmoreceptor-ADH and the Renin-Angiotensin-Aldosterone/ANP mechanisms

B. Sodium and ECF volume homeostasis

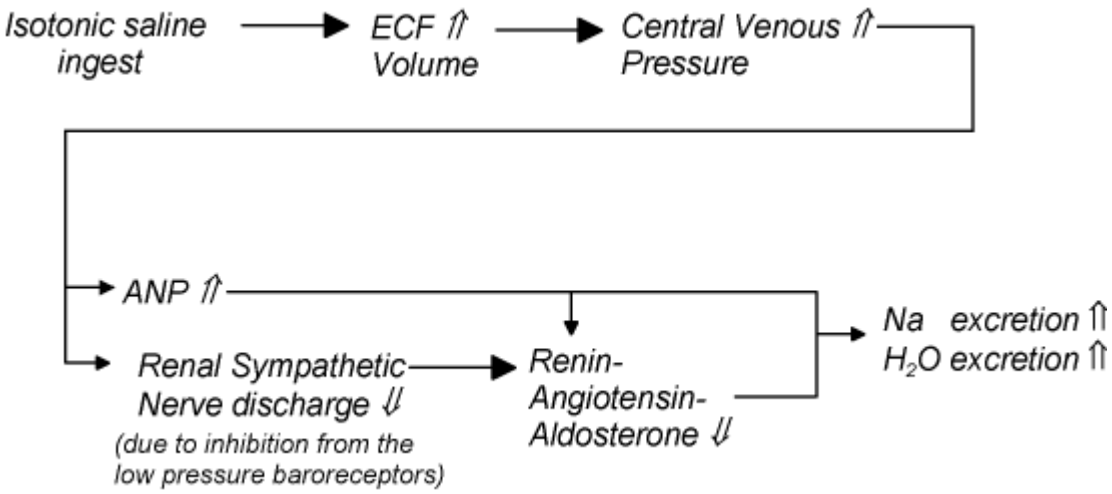
1. ADH acts to maintain body osmolality at normal levels

2. Renin-Angiotensin-Aldosterone system acts to maintain body extracellular fluid (ECF) volume and sodium & potassium concentrations within normal limits

3. Atrial Natriuretic Peptide system aids in maintaining body fluid volume by its action on sodium excretion

4. Speed of action:
   a. ADH: fast, minutes
   b. Aldosterone: slow, up to a day or so

C. Example: drink isotonic saline



Note: If Na and H_2O excretion are not balanced so that plasma osmolality drifts from its normal value, the ADH-osmoreceptor mechanism will be invoked to restore normal osmotic concentration