Ch 20: Integrative Physiology II
Fluid & Electrolyte Balance

Objectives

Explain homeostasis (remember homeodynamics) of

1. Water Balance (ECF/ICF volumes)
2. Electrolyte Balance (Na⁺ and K⁺)
3. Acid-Base Balance (pH)
Introduction to Fluid and Electrolyte Balance

- Intake must = Exhaust
  - Water, ‘lytes
  - ECF or ICF
  - \( O_2 \) and CO\(_2\)
- Many systems involved
  - Kidneys most important
- BP Plays a role
- Hydrostatic and osmotic gradients

The kidneys can only conserve fluid. They cannot restore lost volume.

GFR can be adjusted.

If volume drops too low, GFR stops.
Kidneys maintain H$_2$O balance by regulating urine concentration

- Daily H$_2$O intake balanced by H$_2$O excretion (ins and outs)
- Kidneys react to changes in osmolarity, volume, and blood pressure

Fig 20-2

Fig 20-1

Water gain

- Food and drink: 2.2 L/day

Water loss

- Skin: Insensible water loss 0.9 L/day
- Lungs: 0.3 L/day
- Urine: 1.5 L/day
- Feces: 0.1 L/day

Intake

- 2.2 L/day

Metabolic production

- 0.3 L/day

Output

- (0.9 + 1.5 + 0.1)L/day
Urine Concentration

Established by LOH, CD and vasa recta → reabsorption of varying amounts of H₂O and Na⁺

Key player: ADH (= Vasopressin)
Urine concentration, cont’d

- Often expressed in osmolarity mM/L or osmolality mM/kg
  - Blood: 300 mOsm
  - Filtrate in Bowman’s Capsule: 300 mOsm
  - Bottom of LOH: 1200 mOsm
  - Urine: 50-1200 mOsm

- Regulated by ADH (vasopressin)
  - Osmoreceptors in hypothalamus
  - BP and blood volume, too

![Fig. 20-4](https://via.placeholder.com/150)

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Effect of ADH

- Controls Urine concentration via regulation of water reabsorption from the filtrate in the collecting duct
- Osmoreceptors in hypothalamus
- ↑ ADH caused by:
  - ↑ Na\(^+\) and/or osmolality in the ECF
  - H\(_2\)O deprivation
  - ↓ renal blood flow

Fig 20-5
Effect of ADH, cont’d

- ADH Receptors in CD cells
- Luminal CM is generally impermeable to $\text{H}_2\text{O}$
- Aquaporins (remember Ch. 5) on cell membranes of CD are variably active, dependent on ADH
  - “Membrane Recycling” via exocytosis of AQP2
  - Allows osmosis of $\text{H}_2\text{O}$ into vasa recta
Troubles with ADH?

ADH deficiency:

- Diabetes insipidus
  - Central
  - Nephrogenic
- Nocturnal enuresis

ADH Excess:

- AKA Inappropriate ADH secretion
- XS H₂O retention
Concentrated vs. Dilute Urine

**In presence of ADH:**
- Insertion of H$_2$O pores into tubular luminal CM
- At maximal H$_2$O permeability: Net H$_2$O movement stops at equilibrium
- Maximum osmolarity of urine up to 1200 mOsm

**No ADH:**
- DCT & CD impermeable to H$_2$O
- Osmolarity can plunge to ~ 50 mOsm
Countercurrent Exchange

- For temperature exchange:
  - Pampiniform plexus: testicular A. and V are in close proximity
- For solute exchange, a countercurrent multiplier
  - LOH and vasa recta are in close proximity

Fig. 20-9
LOH: Countercurrent Multiplier

leads to

Hyperosmotic IF in medulla

Hyposmotic fluid leaving LOH
Regulation of BP: 
Na⁺ Balance and ECF Volume

- [Na⁺] affects plasma & ECF osmolarity
  - (Normal [Na⁺]_{ECF} ~ 140 Mosm)

- [Na⁺] affects blood pressure & ECF volume
  - [ ] Gradients

**Aldosterone** stimulates Na⁺ reabsorption and K⁺ excretion in last 1/3 of DCT and CD
  - Type of hormone? Where produced? Type of mechanism?
  - ↑ Aldosterone secretion ⇒ ↑ Na⁺ absorption from DCT
  - Secretion of aldosterone by two mechanisms
    - ↑ K⁺ in ECF
    - ↓ BP
  - The signal to release aldosterone is via angiotensin II

- Opposite of Aldosterone?
  - ANP (from the atria) causes loss of Na⁺
Regulation of BP: RAAS Pathways

- RAAS = renin-angiotensin-aldosterone system
- JG cells release renin in response to ↓ BP
  - Renin converts Angiotensinogen to Angiotensin I
  - ANG I converted to ANG II by ACE
RAAS Pathways, cont’d

ANG II causes ↑ BP via
   ↑ ADH Secretion
   Thirst
   Vasoconstriction
   Sympathetic stimulation of heart →
      ↑ HR and CO

ACE inhibitors will ↓ BP
Potassium

- Recall that
  - 2% of $K^+$ is in ECF
  - Major contributor to resting membrane potential

- Hypokalemia
  - MP more negative (weakness)

- Hyperkalemia
  - MP more positive (poor AP and cardiac arrhythmias)
Maintaining the Balance

- Behavioral
  - Thirst
  - Salty foods
  - Avoidance behaviors

- Osmolarity
  - Alsosterone
  - ADH
Acid–Base Balance

- Normal blood pH?
  - $\uparrow$ pH = Alkalosis
  - $\downarrow$ pH = Acidosis

- Enzymes & NS very sensitive to pH changes

- $[H^+]$ is the same in ECF and ICF
  - Kidneys have $K^+/H^+$ antiport
  - Importance of hyperkalemia and hypokalemia

- $\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons H^+ + \text{HCO}_3^-$
  - pH can be altered by respiration

- Renal Compensation
  - $H^+$ excretion, e.g., $\text{NH}_3 + H^+ \rightleftharpoons \text{NH}_4^+$
  - $\text{HPO}_4^{2-} + H^+ \rightleftharpoons \text{HPO}_4^-$
Body deals with pH changes by 3 mechanisms

\[
\begin{align*}
\text{CO}_2 + \text{H}_2\text{O} & \rightleftharpoons \text{H}_2\text{CO}_3 & \rightleftharpoons & \text{H}^+ + \text{HCO}_3^- \\
\text{NH}_3 + \text{H}^+ & \rightleftharpoons \text{NH}_4^+ \\
\text{HPO}_4^{2-} + \text{H}^+ & \rightleftharpoons \text{HPO}_4^- 
\end{align*}
\]

**Buffers** 1\(^{st}\) defense, immediate response

**Ventilation** 2\(^{nd}\) line of defense, can handle \(~75\%\) of most pH disturbances

**Renal regulation** of \(\text{H}^+\) & \(\text{HCO}_3^-\) final defense, slow but very effective

**Fig 20-21**
Acidosis

**Respiratory acidosis** due to alveolar hypoventilation (accumulation of CO$_2$)

Possible causes: Respiratory depression, increased airway resistance (?), impaired gas exchange (emphysema, fibrosis, muscular dystrophy, pneumonia)

**Metabolic acidosis** due to gain of fixed acid or loss of bicarbonate

Possible causes: lactic acidosis, ketoacidosis, diarrhea

Buffer capabilities exceeded once pH change appears in plasma. *Options for compensation?*
Alkalosis

**Respiratory alkalosis** due to alveolar hyperventilation (excessive loss of CO$_2$)

Possible causes: Anxiety, excessive artificial ventilation, aspirin toxicosis, fever, high altitude

**Metabolic alkalosis** due to loss of H$^+$ ions or shift of H$^+$ into the intracellular space. Alkali administration.

Possible causes: Vomiting or nasogastric (NG) suction; hypokalemia; antacid overdose

Buffer capabilities exceeded once pH change appears in plasma. *Options for compensation?*