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Fluid,Electrolyte & Acid-Base Balance

Foundational Principles of Homeostasis and Clinical Pathophysiology

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Homeostasis & Fluid Balance: The Foundation

Understanding the Body's Internal Equilibrium

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What is Homeostasis?

Homeostasis is the body's ability to maintain a **delicate equilibrium** of water, electrolytes, and hormones despite external changes.

- Supports cellular function and organ integrity.
- Regulates nutrient delivery and waste removal.
- Maintains osmotic balance across membranes.

Cellular Importance

Every cell relies on a specific concentration of ions (electrolytes) and water to:

Nerve Conduction	Muscle Contraction
Enzyme Activity	ATP Production

Adult Body Composition

Total Body Water (TBW) accounts for approximately 60% of weight.

Clinical Implications

Disruptions can lead to life-threatening conditions. Nurses are the first line of defense in:

1. Recognizing early signs of imbalance.
2. Interpreting diagnostic/lab data.
3. Implementing timely interventions.

Body Fluid Compartments

Distribution: Intracellular vs. Extracellular Fluid

% Total Body Water (TBW)

Adults are approximately **60% water** by weight.
For a 70kg adult, this equals ~42 Liters.

ICF (INTRACELLULAR)	ECF (EXTRACELLULAR)
40%	**20%**
~28 Liters	~14 Liters

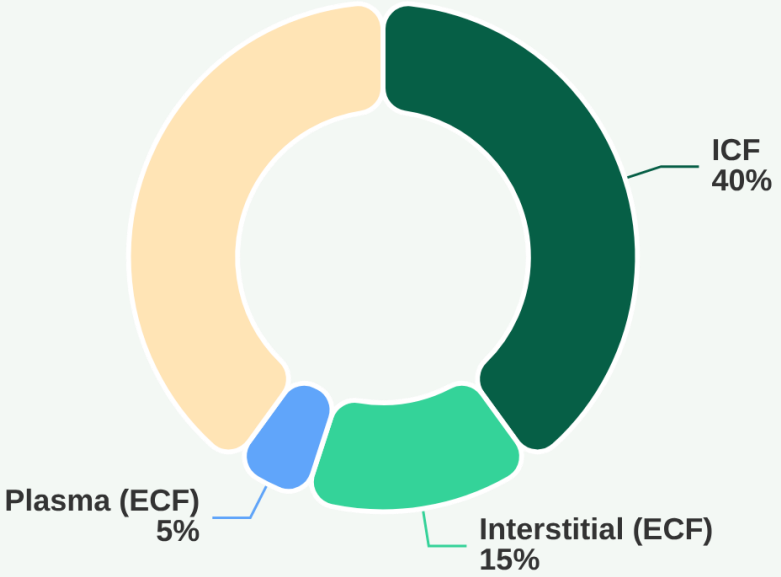
ICF Composition

- **Potassium (K+)** - Primary
- Magnesium (Mg^{2+})
- Phosphate (HPO_4^{2-})

ECF Composition

- **Sodium (Na^+)** - Primary
- Chloride (Cl^-)
- Bicarbonate (HCO_3^-)

Fluid Distribution Ratio



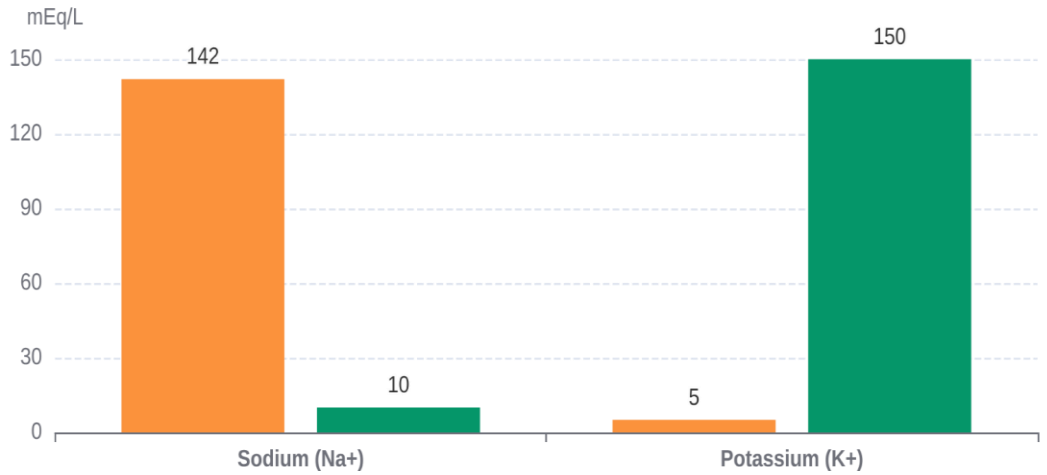
ECF Breakdown:

- Interstitial Fluid: 15% (~10.5L)
- Plasma: 5% (~3.5L)

Electrolyte Composition: ICF vs. ECF

Distinct ionic distribution maintains cellular potential and osmotic balance.

Primary Cation Gradients (mEq/L)



INTRACELLULAR FLUID (ICF)
Dominated by **Potassium (K+)**, Magnesium, and Phosphate.

EXTRACELLULAR FLUID (ECF)
Dominated by **Sodium (Na+)**, Chloride, and Bicarbonate.

The **Na+/K+ ATPase Pump** actively maintains these gradients by moving 3 Na+ out and 2 K+ in.

Na^+
ROLE
Maintains osmotic pressure and fluid volume.

K^+
ROLE
Resting membrane potential & cellular excitability.

Ca^{2+}
ROLE
Neuromuscular transmission & blood coagulation.

Cl^-
ROLE
Most abundant ECF anion; maintains electrical neutrality.

Fluid Movement Mechanisms

Osmosis

Passive movement of **water** across a semipermeable membrane from **low solute** to **high solute** concentration.

KEY FEATURE

Driven by osmotic pressure; Equalizes concentrations.

Diffusion

Passive movement of **solutes** from an area of **higher** concentration to **lower** concentration.

CLINICAL EXAMPLE

Gas exchange (O_2 and CO_2) in the lungs and tissues.

Active Transport

Movement of substances **against** their concentration gradient. Requires energy in the form of **ATP**.

VITAL PROCESS

Na^+/K^+ ATPase pump

(3 Na^+ out / 2 K^+ in).

Mechanism	What Moves?	Direction	Energy Needed?
Osmosis	Water (Solvent)	Low to High Solute	No (Passive)
Diffusion	Solutes (Ions/Gas)	High to Low Solute	No (Passive)
Active Transport	Ions / Glucose	Against Gradient	Yes (ATP)

Starling's Law: Capillary Fluid Exchange

The balance between Hydrostatic and Oncotic pressures

Hydrostatic Pressure (Ph)

The "Pushing Force" generated by the heart. Drives fluid out of capillaries into the interstitial space.
Arterial End: ~35 mmHg Venous End: ~15 mmHg

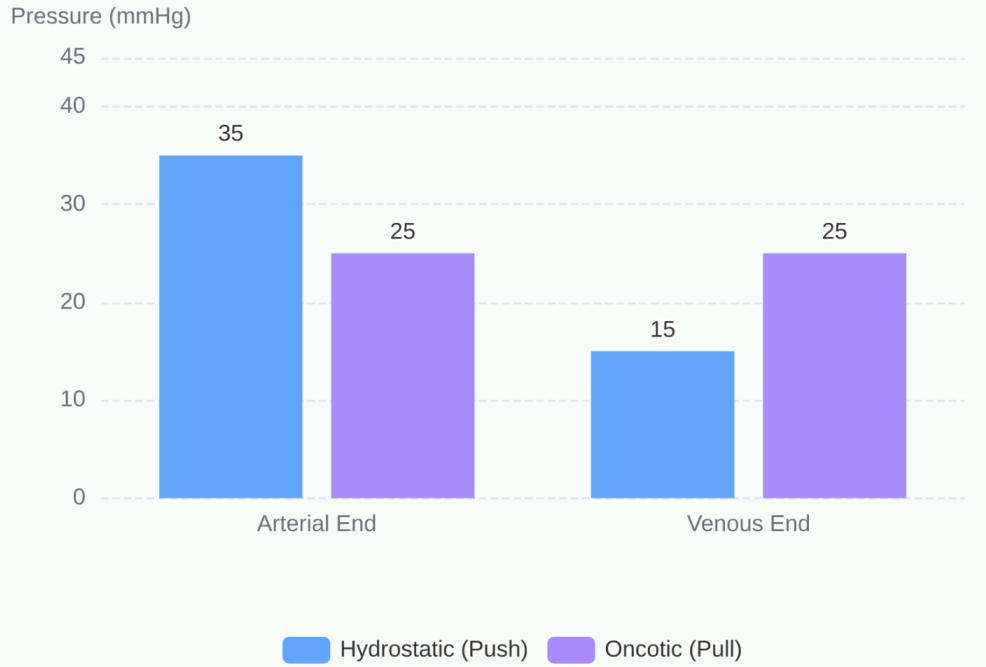
Oncotic Pressure (Po)

The "Pulling Force" created by plasma proteins (Albumin). Pulls fluid back into the capillary lumen.
Constant: ~25 mmHg throughout the capillary

Pathophysiology of Edema

- ↑ Hydrostatic Pressure
- ↓ Plasma Oncotic Pressure
- ↑ Capillary Permeability
- Lymphatic Obstruction

Pressure Gradient Comparison



Net Result:
Arterial: Net Filtration (Out)
Venous: Net Reabsorption (In)

Regulatory Organs

The Three Pillars of Homeostatic Control

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The Kidneys

PRIMARY REGULATORS

- **Water & Na+ Regulation:** Adjusts excretion based on volemic status.
- **Acid-Base Balance:** Reabsorbs HCO_3^- and excretes H^+ ions.
- **Electrolyte Control:** Precise secretion of K^+, Ca^{2+}, and Mg^{2+}.

The Lungs

RAPID RESPONSE SYSTEM

- **CO_2 Elimination:** Controls the concentration of volatile acid in blood.
- **pH Control:** Altering respiratory rate/depth to adjust pH levels quickly.
- **Compensation:** Responds within minutes to metabolic disturbances.

Endocrine System

CHEMICAL ORCHESTRATION

- **Hormonal Signals:** Coordinates responses between distant organs.
- **Volume Control:** ADH and ANP regulate water reabsorption/loss.
- **Pressure Control:** RAAS system manages systemic blood pressure.

Summary: These organs work in concert. A failure in one system (e.g., Kidney Failure) often requires compensation from another (e.g., Respiratory System) to maintain survival.

FLUID VOLUME DEFICIT (HYPOVOLEMIA)

Decreased circulating volume and impaired tissue perfusion.

Etiology

- **GI Losses:** Vomiting, diarrhea, and bleeding.
- **Hemorrhage:** Trauma or surgical complications.
- **Burns:** Significant fluid loss through damaged skin.
- **Third-spacing:** Sepsis, pancreatitis, or bowel obstruction.

Symptoms

VITAL SIGNS	RENAL
Tachycardia Hypotension	Oliguria
SKIN/ORAL	**NEURO**
Dry Mucosa Poor Turgor	Altered Status

Note: Peripheral vasoconstriction often leads to cold, clammy extremities.

Compensation

RAAS Activation

Stimulates aldosterone to promote sodium and water reabsorption in the kidneys.

ADH Release

Increases water reabsorption in collecting ducts to concentrate urine and conserve volume.

Baroreceptors

Triggers SNS response to increase Heart Rate and systemic vascular resistance.

Fluid Volume Excess (Hypervolemia)

Pathophysiology of Fluid Overload and Edema Formation

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Primary Causes

Heart Failure

Reduced cardiac output triggers RAAS, leading to sodium and water retention.

Renal Failure

Decreased GFR impairs the kidneys' ability to excrete excess fluids.

Excessive IV Fluids

Overzealous administration of isotonic saline or dextrose solutions.

Liver Cirrhosis

Hypoalbuminemia reduces oncotic pressure, causing fluid leakage.

Edema & Clinical Signs

- **Peripheral Edema:** Pitting swelling in lower extremities.
- **Pulmonary Edema:** *Crackles, Dyspnea, Hypoxia.*
- **Ascites:** Fluid accumulation in the peritoneal cavity.
- **Anasarca:** Severe, generalized whole-body edema.

Key Nursing Alert: Monitor for sudden weight gain, increased blood pressure, and respiratory distress.

Hormonal Regulation of Fluid Balance

ADH, RAAS, and ANP Mechanisms

ADH

THE WATER SAVER

Trigger: Increased plasma osmolarity or low blood volume.

Source: Hypothalamus (Post. Pituitary)

Action: Inserts Aquaporins in collecting ducts. Reabsorbs pure water.

Results in concentrated urine

RAAS

THE VOLUME BOOSTER

Trigger: Decreased renal perfusion (hypovolemia/hypotension).

Mechanism: Renin → Angiotensin II → Aldosterone

Action: Na+ and H2O retention in distal tubules; vasoconstriction.

Increases blood pressure & volume

ANP

THE NATURAL DIURETIC

Trigger: Atrial stretch due to fluid overload (hypervolemia).

Source: Atrial cells of the heart

Action: Excretes Na+ and H2O; inhibits ADH and Aldosterone.

Reduces fluid overload

- ADH: Water Balance
- RAAS: Pressure/Volume
- ANP: Overload Relief

Sodium Imbalance (Na^+)

The Primary Extracellular Cation & Fluid Regulator

NORMAL RANGE: 135 – 145 MEQ/L

↓ Hyponatremia (< 135)

PRIMARY CAUSES

- SIADH (Water retention)
- Heart Failure
- Cirrhosis
- Diuretic Use

Neurological Impact

Water moves INTO cells → Swelling

- ⚠ Cerebral Edema
- ⚠ Confusion/Headache
- ⚠ Seizures/Coma
- ⚠ Lethargy

NURSING MANAGEMENT

Fluid Restriction
Prevents further dilution.

Hypertonic Saline 3%
NaCl (caution: correct slowly).

↑ Hypernatremia (> 145)

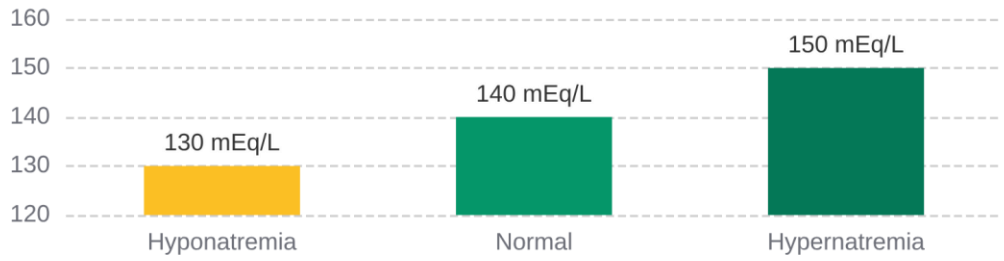
PRIMARY CAUSES

- Dehydration / Low Water
- Diabetes Insipidus
- Excessive Na^+ Intake
- Severe Diarrhea

Osmotic Impact

Water moves OUT of cells → Shrinkage

- Cellular Dehydration
- Intense Thirst
- Irritability
- Flushed Skin



Potassium Imbalance (K)

The Primary Intracellular Cation & Cardiac Regulator

NORMAL SERUM RANGE
3.5 – 5.0 mEq/L

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Low K Hypokalemia

< 3.5 mEq/L

Common Causes

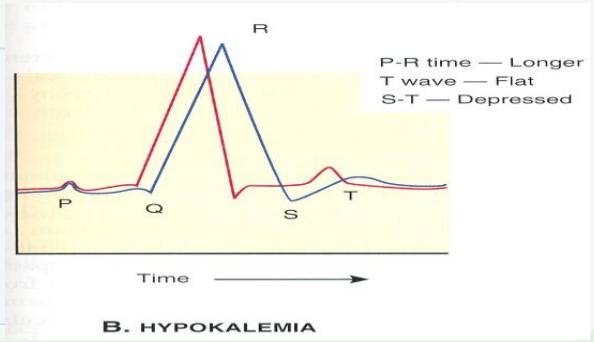
Loop diuretics, GI losses (vomiting/diarrhea), Alkalosis, Insulin therapy.

Manifestations

Muscle weakness, fatigue, leg cramps, constipation, decreased deep tendon reflexes.

ECG Changes

Flattened or Inverted **T waves**
Prominent **U waves**
Prolonged QT interval
ST segment depression



B. HYPOKALEMIA

High K Hyperkalemia

> 5.0 mEq/L

Common Causes

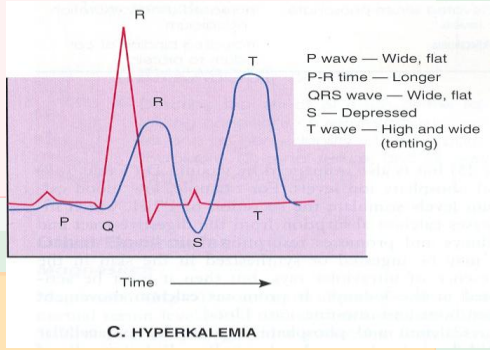
Renal failure (most common), Metabolic Acidosis, Tissue injury/burns, K-sparing diuretics.

Manifestations

Muscle twitching (early), paralysis (late), cardiac arrhythmias, abdominal cramping.

ECG Changes (Medical Emergency)

Tall, **Peaked T waves** (Tent-shaped)
Widened **QRS complex**
Flat or absent P waves
Progression to Ventricular Fibrillation



C. HYPERKALEMIA

Critical Nursing Safety Alert

NEVER administer Potassium via IV push or bolus. It must be diluted and infused slowly (max 10-20 mEq/hr) via infusion pump to avoid immediate cardiac arrest.

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Calcium Imbalance (Ca^{2+})

REGULATING NEUROMUSCULAR EXCITABILITY

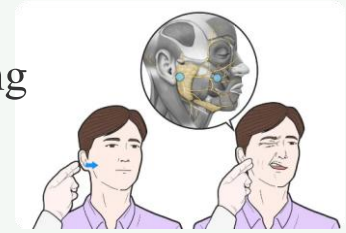
NORMAL RANGE
8.5 – 10.5 mg/dL

Hypocalcemia (< 8.5 mg/dL)

"Increased Neuromuscular Irritability"

Key Clinical Signs

- **Chvostek's Sign:** Facial twitching upon tapping.
- **Trousseau's Sign:** Carpopedal spasm with BP cuff.



COMMON CAUSES

Hypoparathyroidism, Vitamin D deficiency, Acute pancreatitis, Magnesium deficiency.

SYMPTOMS

Tetany, muscle cramps, paresthesias, prolonged QT interval, seizures.

Hypercalcemia (> 10.5 mg/dL)

"Decreased Neuromuscular Irritability"

PRIMARY ETIOLOGY

- **Primary Hyperparathyroidism:** Most common cause.
- **Malignancy:** Bone metastases or paraneoplastic syndromes.

SYSTEMIC MANIFESTATIONS

- Renal Stones
- Confusion
- Lethargy
- Arrhythmias

OTHER CAUSES

Vitamin D intoxication, Thiazide diuretics, Immobilization.

Regulated by PTH, Calcitonin, & Vitamin D

MAGNESIUM & PHOSPHORUS

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Magnesium (Mg^{2+})

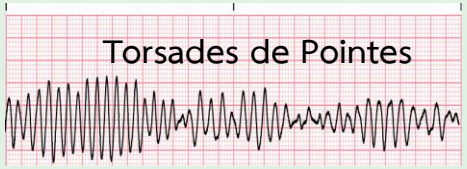
Normal Range: 1.7 – 2.6 mg/dL

Key Physiological Roles

Cofactor for 300+ enzymatic reactions: Energy production (ATP), protein synthesis, and neuromuscular stability.

HYPOMAGNESEMIA (<1.7)
- Alcoholism, Diuretics
- Muscle cramps, Tremors
- Cardiac Arrhythmias

HYPERMAGNESEMIA (>2.6)
- Renal Failure
- Muscle Weakness
- Bradycardia / Hypotension



Phosphorus (PO_4^{3-})

Normal Range: 2.5 – 4.5 mg/dL

Key Physiological Roles

Critical component of ATP, DNA/RNA, and cell membranes. Essential for bone mineralization and acid-base balance.

HYPOPHOSPHATEMIA (<2.5)
- Refeeding Syndrome, DKA
- Rhabdomyolysis
- Respiratory Failure

HYPERPHOSPHATEMIA (>4.5)
- Renal Failure, Tumor Lysis
- Soft Tissue Calcification
- Secondary Hypocalcemia

Clinical Monitoring & Nursing Priorities

Essential for high-risk patients (Critical Care, Malnutrition, Chronic Renal Disease)

Routine Lab Monitoring | Dietary Management | Close IV Monitoring

INTRODUCTION TO PH & BUFFER SYSTEMS
Maintaining Acid-Base Homeostasis

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NORMAL PHYSIOLOGICAL RANGE

Acidosis **7.35 — 7.45** Alkalosis

Deviations impair enzymes, disrupt metabolism, and can be life-threatening.

Chemical Buffers
1st Line of Defense. Immediate neutralization of acids or bases.

INSTANT RESPONSE

Respiratory System
Elimination of CO_2 through ventilation adjustments.

MINUTES TO HOURS

Renal System
Regulates H^+ excretion and HCO_3^- reabsorption.

HOURS TO DAYS

Buffer System	Primary Location	Key Components
Bicarbonate	Extracellular Fluid (ECF)	HCO_3^- / H_2CO_3 (Most significant in blood)
Phosphate	Intracellular Fluid (ICF) / Renal	HPO_4^{2-} / $H_2PO_4^-$ (Effective in urine)
Protein	Intracellular Fluid / Plasma	Amino acids, Hemoglobin (Binds H^+)

RESPIRATORY & RENAL REGULATION

Secondary and Tertiary Lines of Defense in Acid-Base Balance

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Respiratory Regulation

RAPID RESPONSE (MINUTES)

Controls pH by adjusting **CO_2** elimination:

- ↑ **In Acidosis:** Hyperventilation (blows off CO_2) to increase pH.
- ↓ **In Alkalosis:** Hypoventilation (retains CO_2) to decrease pH.

Renal Regulation

LONG-TERM RESPONSE (HOURS/DAYS)

Adjusts **HCO_3^-** and **H^+** levels:

- ⊕ **In Acidosis:** Excretes H^+ and reabsorbs/generates new HCO_3^-.
- ⊖ **In Alkalosis:** Reduces H^+ excretion and increases HCO_3^- excretion.

RESPIRATORY CONTROL **$CO_2 + H_2O$** ⇄ INTERMEDIATE **H_2CO_3** ⇄ RENAL CONTROL **$H^+ + HCO_3^-$**

Types of Acid-Base Imbalance

Classification by Primary Disturbance and Systemic Origin

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Respiratory Acidosis

pH ↓
pCO_2 ↑

COMMON CAUSES:

- Hypoventilation (Respiratory failure)
- COPD / Chronic Lung Disease
- Drug Overdose (Opioids, Sedatives)
- Chest wall trauma / Neuromuscular weakness

Renal Compensation: Kidneys retain HCO_3^- and excrete H^+ (takes 24-48 hours).

Respiratory Alkalosis

pH ↑
pCO_2 ↓

COMMON CAUSES:

- Hyperventilation (Anxiety, Panic)
- Acute Hypoxia (High altitude)
- Fever / Sepsis
- Salicylate overdose (early stage)

Renal Compensation: Kidneys increase HCO_3^- excretion.

Metabolic Acidosis

pH ↓
HCO_3^- ↓

COMMON CAUSES:

- Diabetic Ketoacidosis (DKA)
- Lactic Acidosis (Shock, Sepsis)
- Severe Diarrhea (Loss of Bicarbonate)
- Chronic Kidney Disease (CKD)

Respiratory Compensation: Kussmaul respirations (Deep, rapid breathing to blow off CO_2).

Metabolic Alkalosis

pH ↑
HCO_3^- ↑

COMMON CAUSES:

- Prolonged Vomiting / NG Suction (Loss of HCl)
- Excessive Diuretic use (Loop/Thiazide)
- Hypokalemia
- Excessive Bicarbonate administration

Respiratory Compensation: Hypoventilation to retain CO_2 (limited by oxygen needs).

● **Acidosis: pH < 7.35**

● **Alkalosis: pH > 7.45**

Normal Range: pH 7.35 – 7.45 | pCO_2 35 – 45 | HCO_3^- 22 – 26

ABG Interpretation

A Step-by-Step Approach to Acid-Base Assessment

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1 Analyze pH

7.35 – 7.45

- ↓ **< 7.35:** Acidosis
- ↑ **> 7.45:** Alkalosis

2 Analyze pCO2

35 – 45 mmHg

RESPIRATORY COMPONENT

- **> 45:** Respiratory Acidosis
- **< 35:** Respiratory Alkalosis

3 Analyze HCO3-

22 – 26 mEq/L

METABOLIC COMPONENT

- **< 22:** Metabolic Acidosis
- **> 26:** Metabolic Alkalosis

4 Compensation

The Body's Response

Determine if the opposite system is moving in the direction to return the pH to normal limits.

Example: In Metabolic Acidosis, the lungs hyperventilate to blow off CO2 (Step 2).

R	Respiratory	PH ↑ PCo2 ↓ Alkalosis
O	Opposite	PH ↓ PCo2 ↑ Acidosis
M	Metabolic	PH ↑ HCo3 ↑ Alkalosis
E	Equal	PH ↓ HCo3 ↓ Acidosis

Nursing Tip: If pH and CO2 move in opposite directions, the problem is **Respiratory**. If they move in the same direction, it's **Metabolic** (ROME).

Compensation Mechanisms

Restoring Physiological pH Balance

CLINICAL ASSESSMENT

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Degrees of Compensation

1 Uncompensated

- **pH:** Abnormal
- **Cause:** Abnormal
- **Opposite:** Normal

2 Partially Compensated

- **pH:** Abnormal
- **Cause:** Abnormal
- **Opposite:** Moving to Correct

3 Fully Compensated

- **pH:** Normal (7.35–7.45)
- **Cause:** Abnormal
- **Opposite:** Abnormal

Respiratory System

FAST RESPONSE (MINUTES TO HOURS)

Adjusts **pCO2** by changing ventilation. In metabolic acidosis, the lungs increase rate (Hyperventilation) to blow off CO2 and raise pH.

Renal System

SLOW RESPONSE (HOURS TO DAYS)

Adjusts **HCO3-** and **H+**. In respiratory acidosis, kidneys excrete H+ and reabsorb HCO3- to neutralize acidity.

Nursing Management

Foundations of Fluid, Electrolyte & Acid-Base Restoration

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Clinical Practice

Comprehensive Assessment

- **Vital Signs:** Monitor for tachycardia, hypotension, or respiratory changes.
- **I/O Tracking:** Precise hourly intake and output measurements.
- **Daily Weights:** The most reliable indicator of acute fluid volume change.

Therapeutic Interventions

- **Fluid Therapy:** Administer IV replacement or implement fluid restrictions (e.g., SIADH).
- **Electrolyte Support:** Oral or IV supplementation based on serum deficits.
- **Underlying Cause:** Manage primary issues (e.g., vomiting, diarrhea, renal failure).

Safety & Monitoring

- **Neuro Status:** Frequent checks to detect cerebral edema or demyelination.
- **ECG Monitoring:** Continuous tracking for arrhythmia risk (K+ imbalances).
- **Lab Frequency:** Serial Na+, K+, and ABG draws as ordered.

CRITICAL SAFETY LIMITS - NEVER PUSH IV ELECTROLYTES!

Sodium (Na+): Correct gradually (1–2 mEq/L/hour) to prevent brain injury. **Potassium (K+):** Max infusion rate 10–20 mEq/hour. Always use a pump.

CASE STUDY : Severe Dehydration & Diarrhea

Patient Profile

Patient: 65-year-old male

Chief Complaint: Severe watery diarrhea (10-15 times/day) for 3 days

Symptoms: Extreme fatigue, orthostatic dizziness (dizziness upon standing)

Clinical Examination & Labs

Vitals: HR 115 bpm (Tachycardia), BP 90/60 mmHg (Hypotension)

Physical Signs: Dry mucous membranes, poor skin turgor

Initial Labs:

Na^+: 134 mEq/L | K^+: 2.9 mEq/L

ABG: pH 7.28, $PaCO_2$ 30, HCO_3^- 14

Question 1: ABG Interpretation

Acid-Base Status

pH 7.28 (< 7.35)

Indicates

Acidemia

Primary Disorder

HCO_3^- 14 (< 22)

Indicates **Metabolic Acidosis**

(due to loss of base in diarrhea)

Compensation

$PaCO_2$ 30 (< 35)

Indicates **Partial Respiratory Compensation** (pH is not yet normal)

Diagnosis: Partially Compensated Metabolic Acidosis

Question 2: Electrolyte Imbalances

Hyponatremia

Na^+ 134 mEq/L (Normal: 135-145)

Mild hyponatremia due to loss of sodium-rich fluid and volume depletion leading to ADH release.

Hypokalemia

K^+ 2.9 mEq/L (Normal: 3.5-5.0)

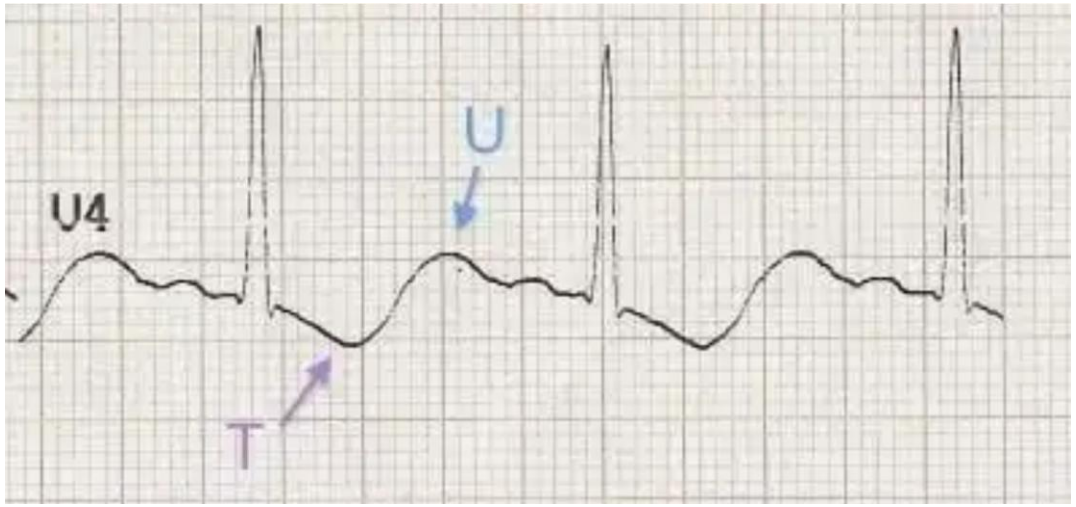
Moderate to severe hypokalemia due to significant potassium loss in intestinal secretions.

Question 3: Pathophysiology & ECG Risks

Mechanism of K^+ Loss

Direct GI Loss: Diarrhea fluid contains high concentrations of K^+ and HCO_3^-.

Secondary Renal Loss: Dehydration activates RAAS, increasing Aldosterone, which promotes renal K^+ excretion.



ECG Risks

Flattened or Inverted T waves

Prominent **U waves**

Risk of lethal arrhythmias (VT/VF)

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