



# FLUIDS, ELECTROLYTES AND ACID-BASE BALANCING

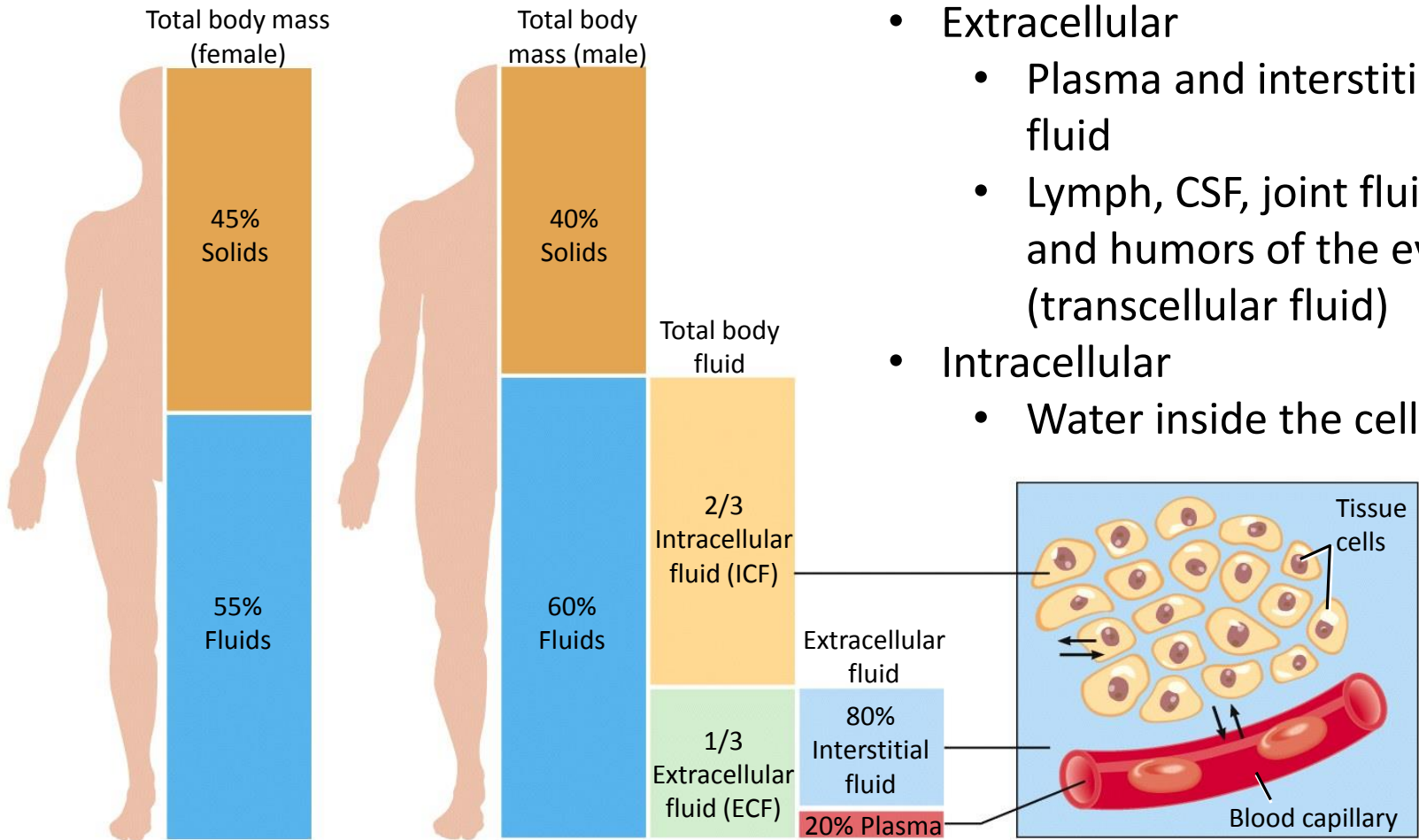
DND Primary Care Paramedicine

Module: 02

Section: 02

- Normal values expressed as a percentage
- Differences occur due to age, sex and fat content
- Water
  - 80% Total body weight (newborns)
  - 65% - 70% TBW (children)
  - 50% - 60% TBW (adults)
  - 45 % TBW (elderly)

# Body Fluid Compartments

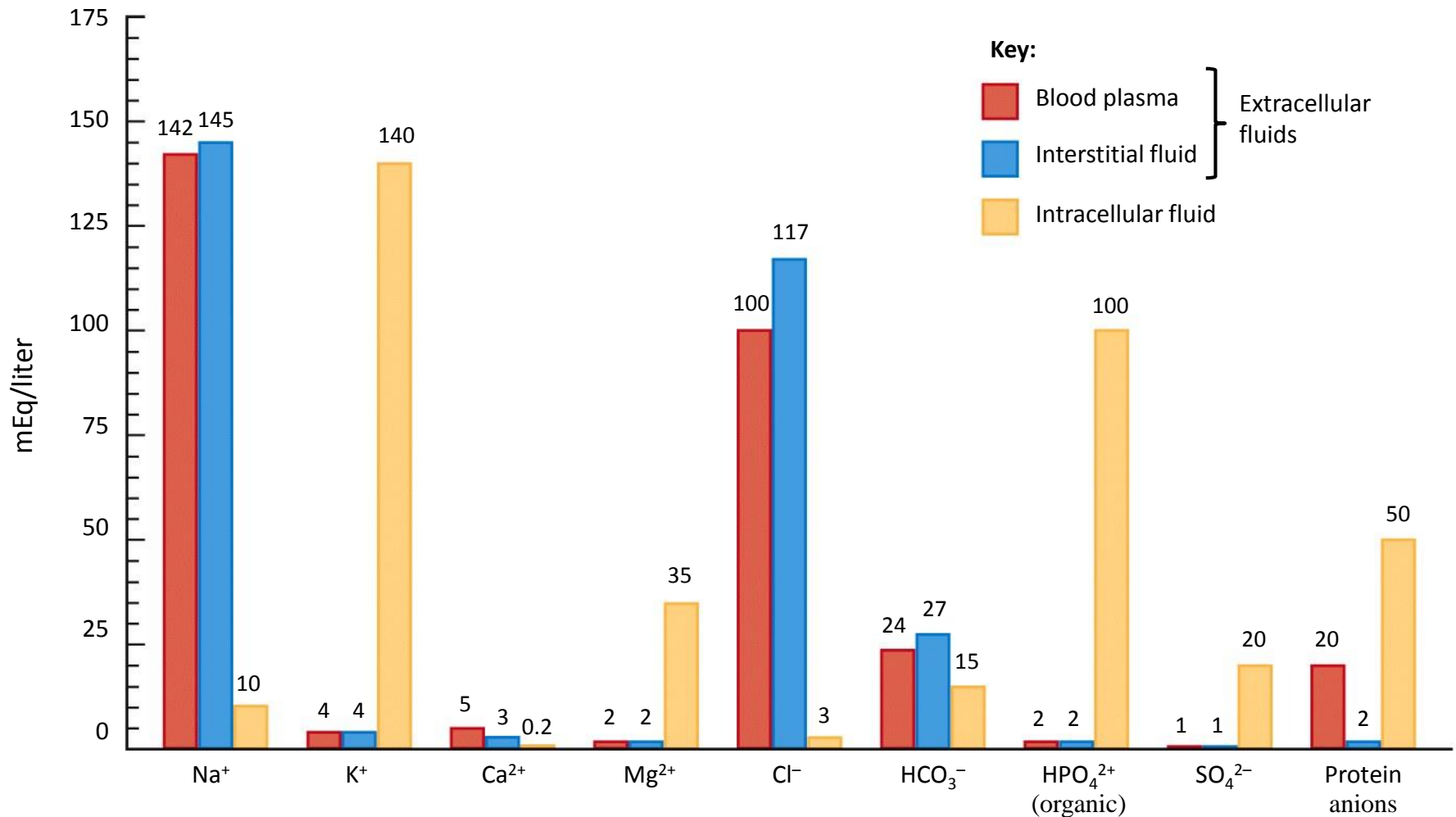


- Extracellular
  - Plasma and interstitial fluid
  - Lymph, CSF, joint fluids and humors of the eye (transcellular fluid)
- Intracellular
  - Water inside the cells

(a) Distribution of body solids and fluids in average lean, adult female and male

(b) Exchange of water among body fluid compartments

- A constant balance between the fluid and the electrolytes must exist for homeostasis
  - Water
  - Electrolytes
    - Salt substances that dissociate into charged components when dissolved in water ( $\text{Na}^+$ ,  $\text{K}^+$ )
  - Non-electrolytes
    - Substances that do not carry an electrical charge in water (glucose, urea)



- Positively Charged Electrolytes (Cation)
  - Intracellular
    - Potassium  $K^+$
    - Calcium  $Ca^{++}$
    - Magnesium  $Mg^{++}$
  - Extracellular
    - Sodium  $Na^+$

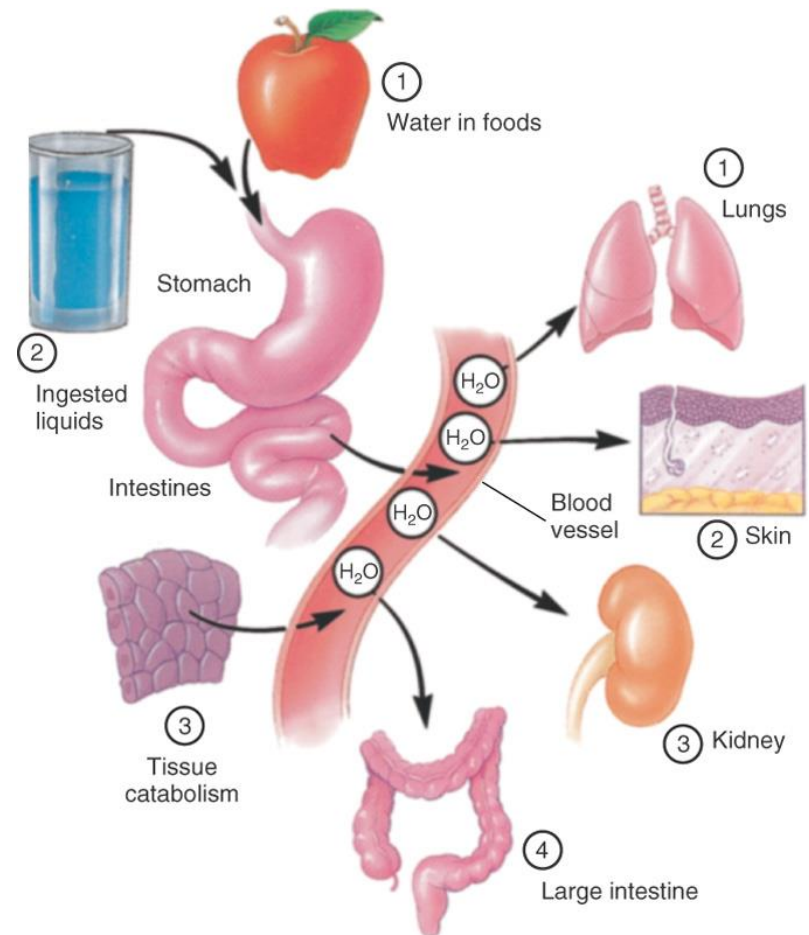
- Negatively Charged Electrolytes (Anion)
  - Intracellular
    - Phosphate  $\text{PO}_4^{3-}$
  - Extracellular
    - Chloride  $\text{Cl}^-$
    - Bicarbonate  $\text{HCO}_3^-$



- Aldosterone is the primary regulator of electrolyte concentrations
- Accomplishes this through reabsorption of  $\text{Na}^+$  and  $\text{K}^+$



- Water enters the body
  - Digestive system
  - Cellular metabolism
- Water exits the body
  - Kidneys
  - Lungs
  - Sweat
  - Feces
- Input = Output



- Net Filtration (Starling's law of the capillaries)
- Net filtration = forces favoring filtration vs. forces opposing filtration
  - Forces favoring filtration
    - Blood hydrostatic pressure (BHP)
    - Interstitial fluid colloid osmotic pressure (IFCOP)
  - Forces opposing filtration
    - Blood colloid osmotic pressure (BCOP)
    - Interstitial fluid hydrostatic pressure (IFHP)
  - Other factors
    - Tonicity
    - Membrane permeability

- The mechanical force of water against cellular membranes
- One of the primary forces in the movement of fluid
- Pressure created by contraction of Left Ventricle
- Filters fluid from blood
  - Allows fluid to pass from vascular space through the capillary wall
- Represents the blood pressure within the capillary network (approximately 25 – 30 mmHg)

- The overall osmotic effect of colloids (plasma proteins) in the vasculature
- Opposes filtration of large solutes
- Maintains levels of intravascular fluids

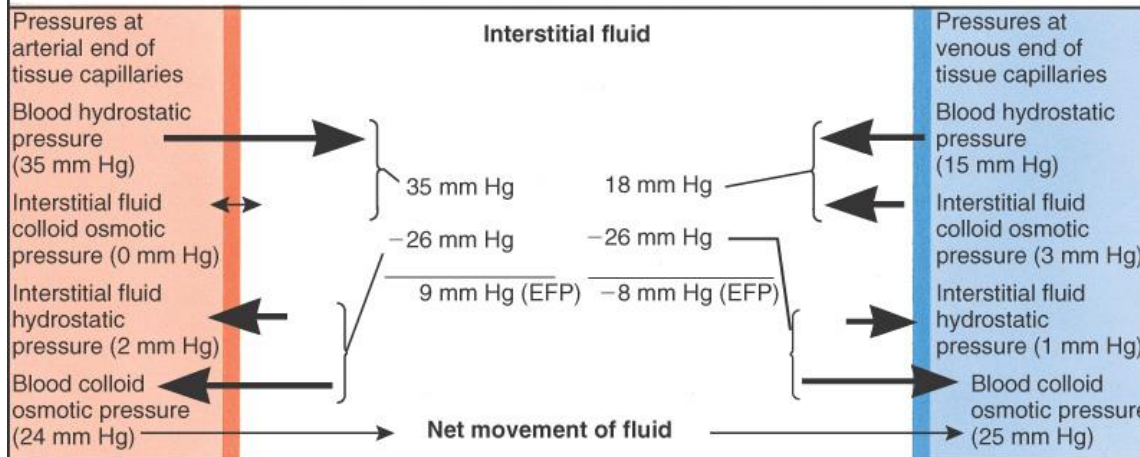
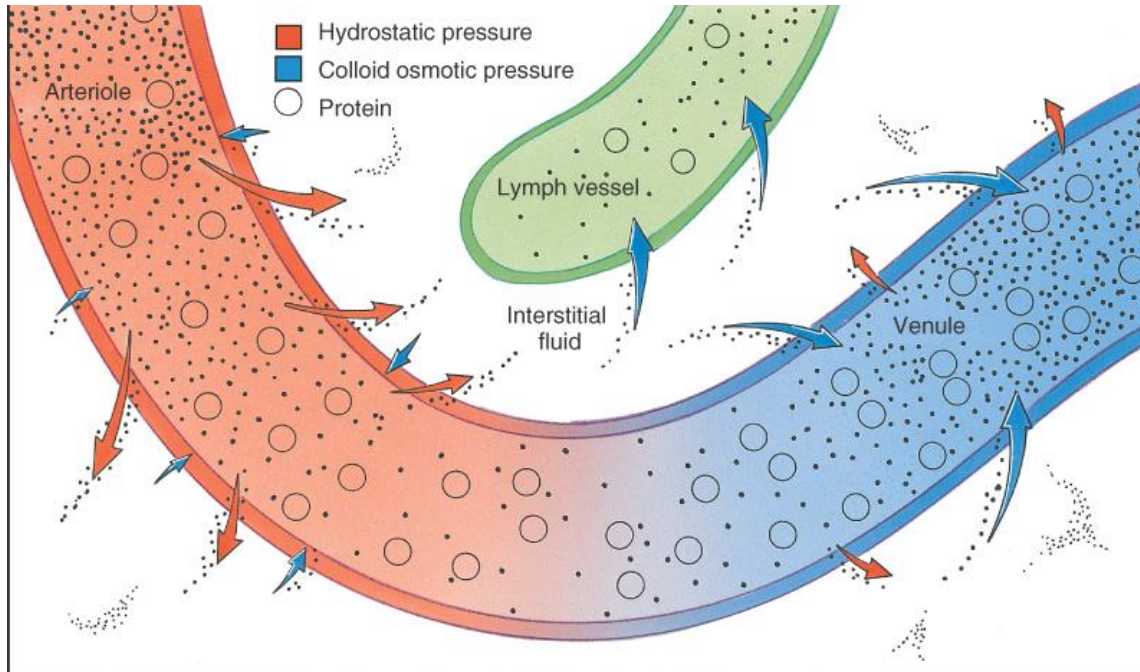
- Generally a negative pressure
- Causes a suction of fluid into the interstitium
- Present due to solute concentration in the interstitium
- Works with capillary hydrostatic pressure in filtration

- The mechanical force of water within the interstitium against cellular membranes
- Works with plasma osmotic pressure against filtration
- Keeps small quantities of plasma proteins which have leaked into the interstitium within the interstitium

- Therefore effective filtration pressure is:

$$EFP = \underbrace{(BHP + IFCOP)}_{\text{Moves fluid out of the capillaries}} - \underbrace{(IFHP + BCOP)}_{\text{Moves fluid into the capillaries}}$$





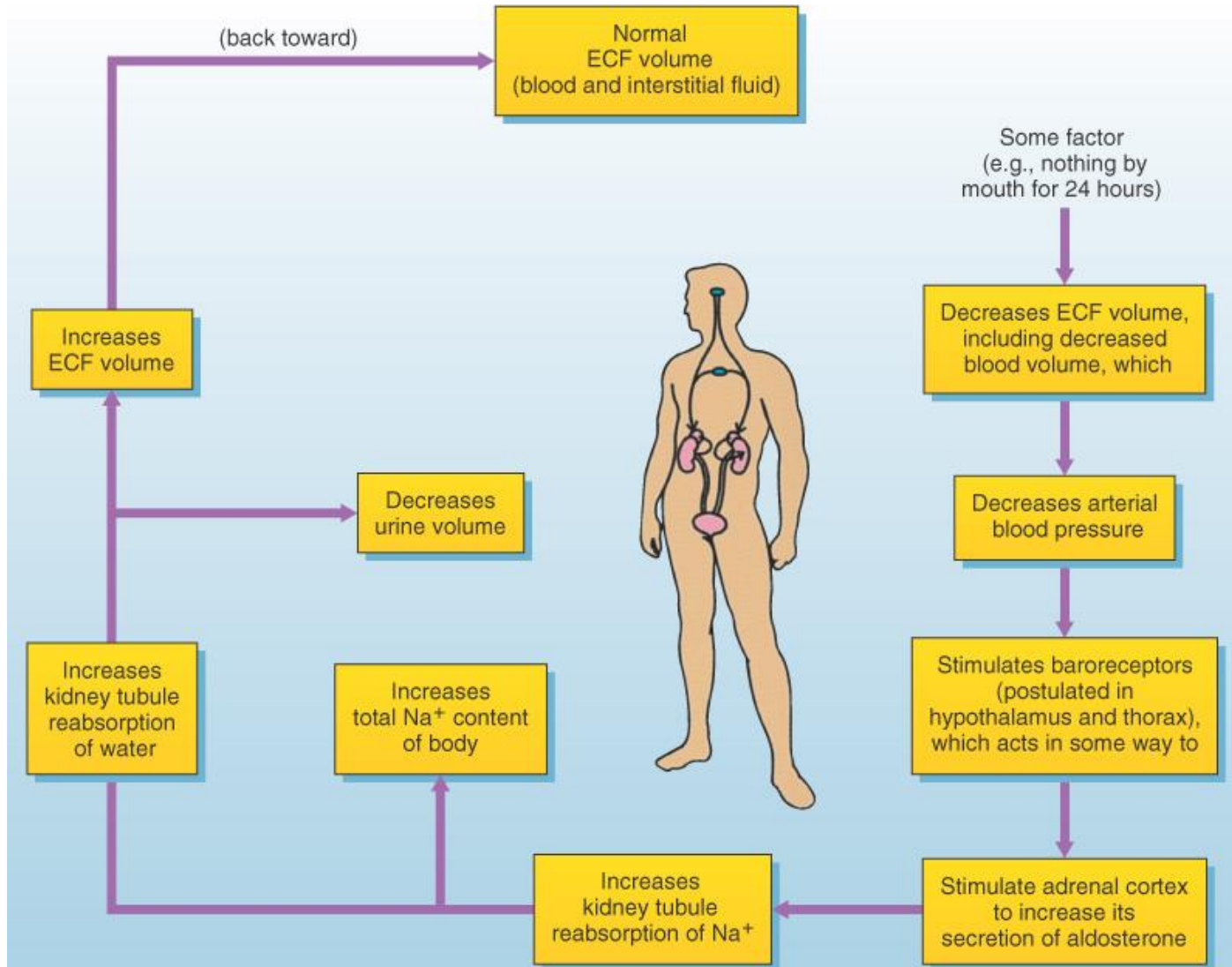
- Isotonic
  - Of **equal** solute concentration...
  - Same pressure
- Hypotonic
  - Of **lesser** solute concentration...
  - Less pressure
- Hypertonic
  - Of **higher** solute concentration...
  - More pressure

...than the fluid being compared

- Only a small portion of plasma proteins cross capillary membranes
- Fluid passes easily, depending on the tonicity of each side of the membrane
- Electrolytes pass readily due their size and function

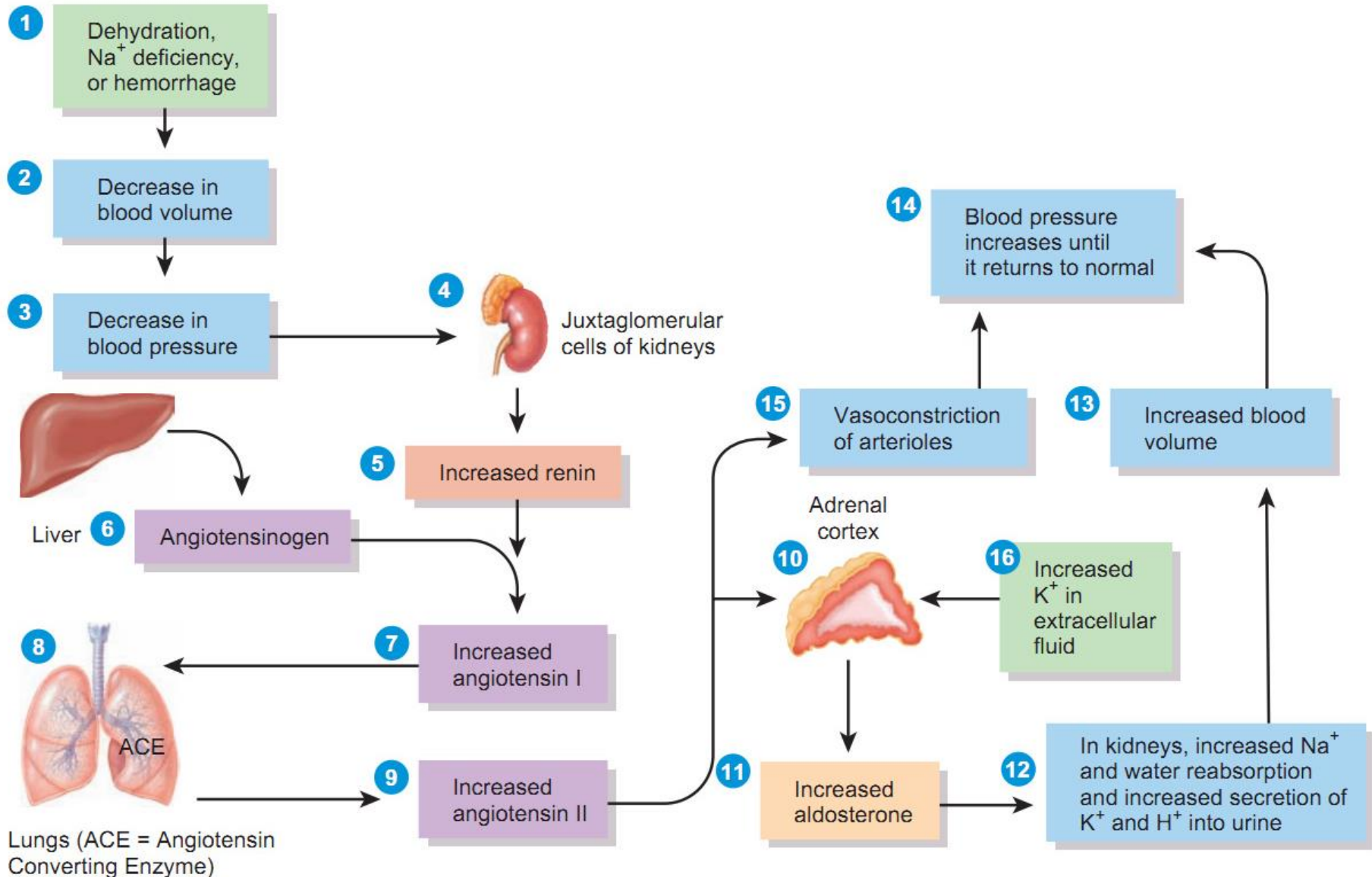
- Osmosis
  - The diffusion of water through a selectively permeable membrane
- Diffusion
  - Movement of atoms, ions or molecules from a region of high concentration to a region of low concentration
- Active Transport
  - Moves substances against (“uphill”) a concentration gradient
- Facilitated Diffusion
  - Moves substances down the gradient by use of a carrier protein

- Water balance regulated by ADH and perception of thirst.
- ADH released due to:
  - Increased plasma osmolality
  - Decreased circulating blood volume
  - Lowered venous and arterial pressure
- Following the release of ADH water is reabsorbed from renal tubules and collecting ducts of the kidneys





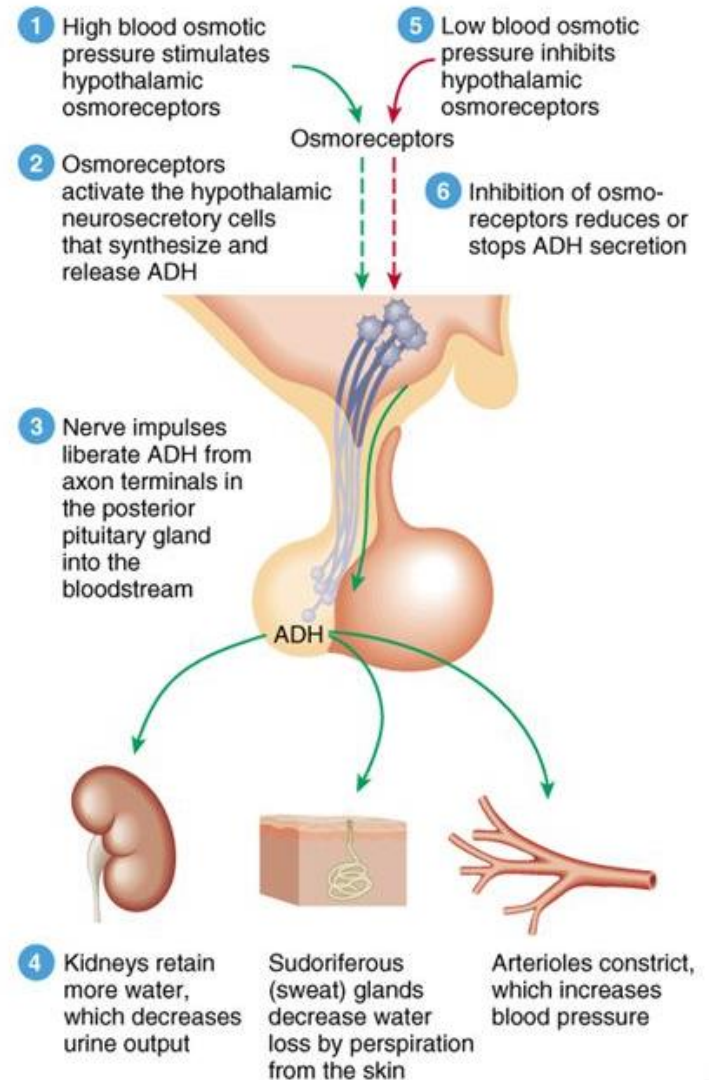
# Renin-Angiotensin-Aldosterone System (RAAS)



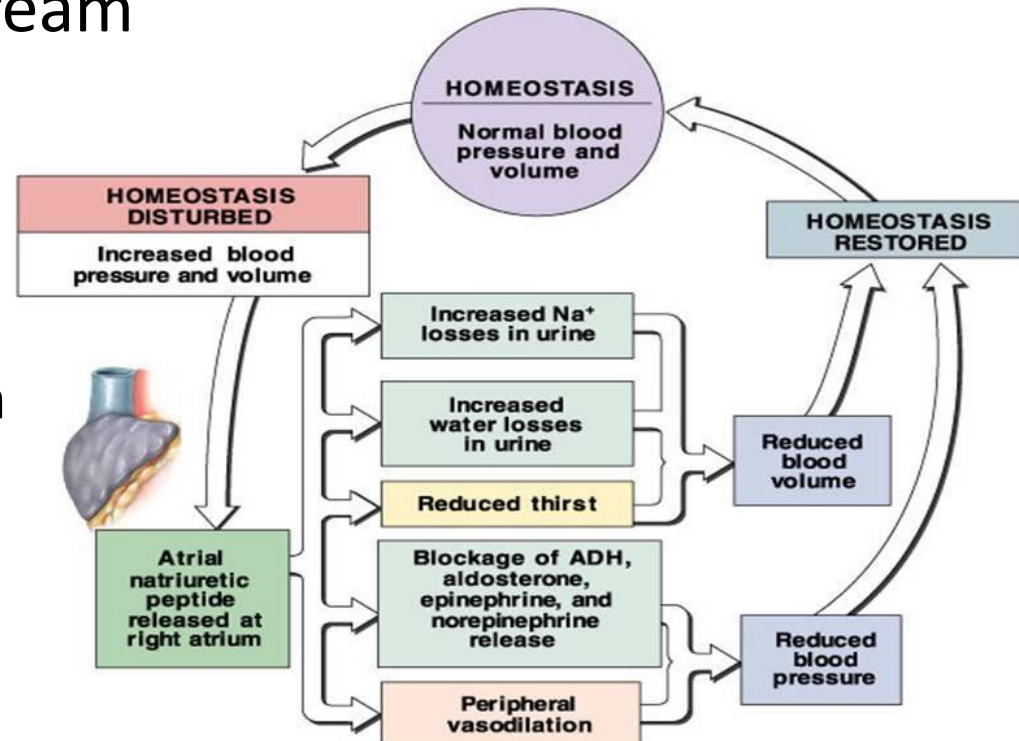


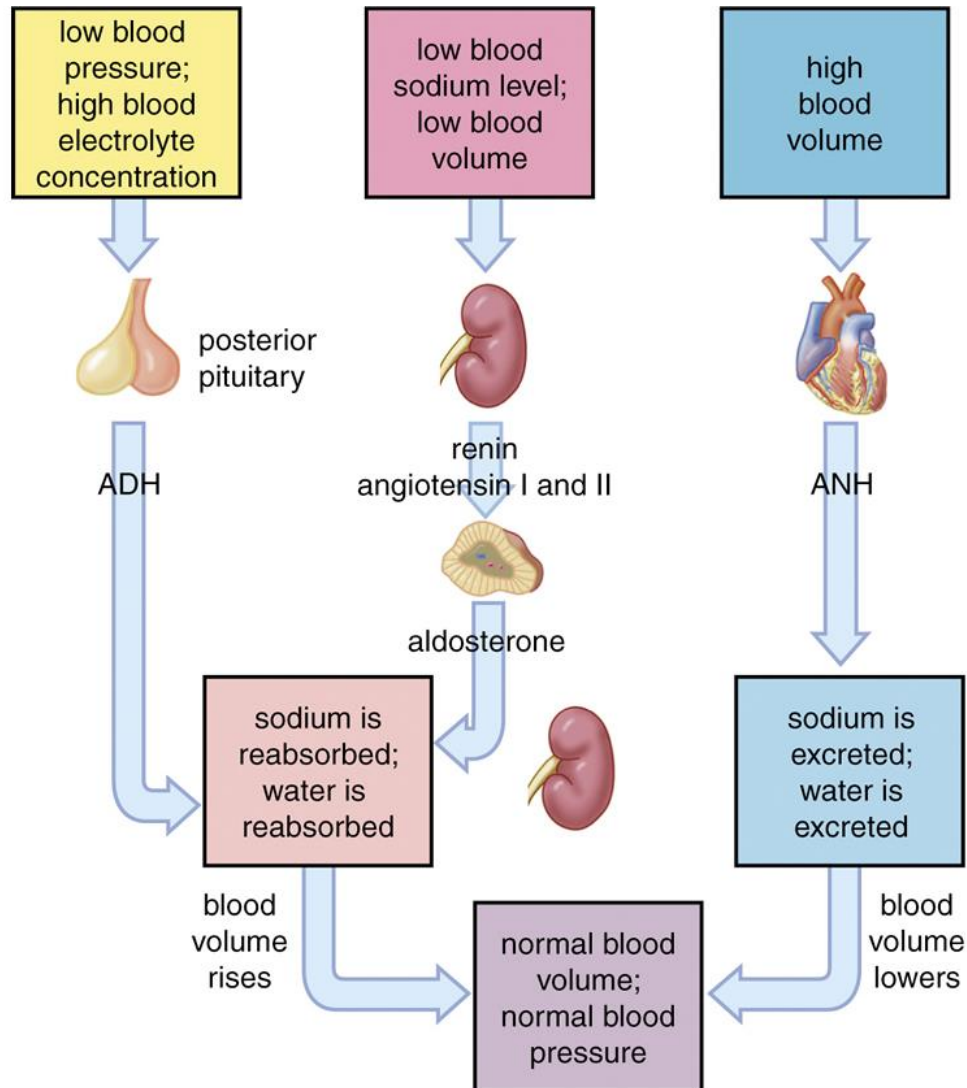
# Antidiuretic Hormone (ADH)

- ADH also referred to as Vasopressin
  - Created in the hypothalamus and stored in the posterior pituitary gland
- Stimulated to be released as a result of Angiotensin II (that was released due to a drop in blood volume)



- Increasing blood volume (RAAS and ADH) and pressure causes right atrium to over stretch
- This stimulates the release of ANH from the cardiac cells into the blood stream
- ANH causes:
  - ↓ Na<sup>+</sup> reabsorption
  - ↑ GFR
  - Peripheral vasodilation
  - Inhibits ADH
  - Inhibits Aldosterone





- Hypovolemia (dehydration)
  - Isotonic
  - Hyponatremic
  - Hypernatremic
- Hypervolemia (overhydration)

- Excessive loss of equal amounts of Na and Water
  - Severe or long term vomiting/diarrhea
  - Systemic infection
  - Intestinal obstruction

- A serum decrease in Na
  - Excess loss of Na
  - Decrease in [Na] relative to water
- Causes
  - Use of salt-wasting diuretics
  - Excessive perspiration
  - Salt losing renal disorders
  - Increased water intake
  - Excessive use of water enemas

- S/S
  - Muscle cramps
  - N/V
  - Postural BP changes
  - Poor skin turgor
  - Fatigue
  - Dyspnea
  - Confusion, hemiparesis, seizures and coma (due to cerebral swelling in severe cases)



- Elevation of serum levels
  - Loss of water in excess of Na
  - Elevated Na levels
- Causes
  - Lack of fluid intake
  - Diabetes Insipidus
  - CHF
  - Renal failure
  - Excessive misuse of diuretics
  - Na intake in absence of water
  - Profuse watery diarrhea

- S/S
  - Similar to hyponatremia
  - Thirst
  - Disorientation
  - Lethargy
  - Seizures

- Increased body water with a decrease in solute concentration
  - Excessive IV fluid administration
  - Impaired cardiac function
  - Impaired renal function
  - Endocrine dysfunction



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- K
  - Nerve, muscle and cardiac function
- Ca
  - Neurotransmission, cell membrane permeability, hormone secretion, growth, ossification of bones and muscle contraction
- Mg
  - Activates many enzymes, nervous system and MSK effects similar to calcium



**TABLE 27.2****Blood Electrolyte Imbalances**

<b>ELECTROLYTE*</b>	<b>DEFICIENCY</b>		<b>EXCESS</b>	
	<b>NAME AND CAUSES</b>	<b>SIGNS AND SYMPTOMS</b>	<b>NAME AND CAUSES</b>	<b>SIGNS AND SYMPTOMS</b>
<b>Sodium (Na<sup>+</sup>)</b> <b>136–148 mEq/liter</b>	<b>Hyponatremia</b> (hī-pō-na-TRĒ-mē-a) may be due to decreased sodium intake; increased sodium loss through vomiting, diarrhea, aldosterone deficiency, or taking certain diuretics; and excessive water intake.	Muscular weakness; dizziness, headache, and hypotension; tachycardia and shock; mental confusion, stupor, and coma.	<b>Hypernatremia</b> may occur with dehydration, water deprivation, or excessive sodium in diet or intravenous fluids; causes hypertonicity of ECF, which pulls water out of body cells into ECF, causing cellular dehydration.	Intense thirst, hypertension, edema, agitation, and convulsions.
<b>Chloride (Cl<sup>-</sup>)</b> <b>95–105 mEq/liter</b>	<b>Hypochloremia</b> (hī-pō-klō-RĒ-mē-a) may be due to excessive vomiting, overhydration, aldosterone deficiency, congestive heart failure, and therapy with certain diuretics such as furosemide (Lasix <sup>®</sup> ).	Muscle spasms, metabolic alkalosis, shallow respirations, hypotension, and tetany.	<b>Hyperchloremia</b> may result from dehydration due to water loss or water deprivation; excessive chloride intake; or severe renal failure, hyperaldosteronism, certain types of acidosis, and some drugs.	Lethargy, weakness, metabolic acidosis, and rapid, deep breathing.
<b>Potassium (K<sup>+</sup>)</b> <b>3.5–5.0 mEq/liter</b>	<b>Hypokalemia</b> (hī-pō-ka-LĒ-mē-a) may result from excessive loss due to vomiting or diarrhea, decreased potassium intake, hyperaldosteronism, kidney disease, and therapy with some diuretics.	Muscle fatigue, flaccid paralysis, mental confusion, increased urine output, shallow respirations, and changes in electrocardiogram, including flattening of T wave.	<b>Hyperkalemia</b> may be due to excessive potassium intake, renal failure, aldosterone deficiency, crushing injuries to body tissues, or transfusion of hemolyzed blood.	Irritability, nausea, vomiting, diarrhea, muscular weakness; can cause death by inducing ventricular fibrillation.

\*Values are normal ranges of blood plasma levels in adults.

**TABLE 27.2**
**Blood Electrolyte Imbalances**

ELECTROLYTE*	DEFICIENCY		EXCESS	
	NAME AND CAUSES	SIGNS AND SYMPTOMS	NAME AND CAUSES	SIGNS AND SYMPTOMS
<b>Calcium (Ca<sup>2+</sup>)</b> <b>Total =</b> <b>9.0–10.5 mg/dL;</b> <b>ionized =</b> <b>4.5–5.5 mEq/liter</b>	<b>Hypocalcemia</b> (hī-pō-kal-SĒ-mē-a) may be due to increased calcium loss, reduced calcium intake, elevated phosphate levels, or hypoparathyroidism.	Numbness and tingling of fingers; hyperactive reflexes, muscle cramps, tetany, and convulsions; bone fractures; spasms of laryngeal muscles that can cause death by asphyxiation.	<b>Hypercalcemia</b> may result from hyperparathyroidism, some cancers, excessive intake of vitamin D, and Paget's disease of bone.	Lethargy, weakness, anorexia, nausea, vomiting, polyuria, itching, bone pain, depression, confusion, paresthesia, stupor, and coma.
<b>Phosphate (HPO<sub>4</sub><sup>2-</sup>)</b> <b>1.7–2.6 mEq/liter</b>	<b>Hypophosphatemia</b> (hī-pō-fos'-fa-TĒ-mē-a) may occur through increased urinary losses, decreased intestinal absorption, or increased utilization.	Confusion, seizures, coma, chest and muscle pain, numbness and tingling of fingers, decreased coordination, memory loss, and lethargy.	<b>Hyperphosphatemia</b> occurs when kidneys fail to excrete excess phosphate, as in renal failure; can also result from increased intake of phosphates or destruction of body cells, which releases phosphates into blood.	Anorexia, nausea, vomiting, muscular weakness, hyperactive reflexes, tetany, and tachycardia.
<b>Magnesium (Mg<sup>2+</sup>)</b> <b>1.3–2.1 mEq/liter</b>	<b>Hypomagnesemia</b> (hī'-pō-mag'-ne-SĒ-mē-a) may be due to inadequate intake or excessive loss in urine or feces; also occurs in alcoholism, malnutrition, diabetes mellitus, and diuretic therapy.	Weakness, irritability, tetany, delirium, convulsions, confusion, anorexia, nausea, vomiting, paresthesia, and cardiac arrhythmias.	<b>Hypermagnesemia</b> occurs in renal failure or due to increased intake of Mg <sup>2+</sup> , such as Mg <sup>2+</sup> -containing antacids; also occurs in aldosterone deficiency and hypothyroidism.	Hypotension, muscular weakness or paralysis, nausea, vomiting, and altered mental functioning.

\*Values are normal ranges of blood plasma levels in adults.

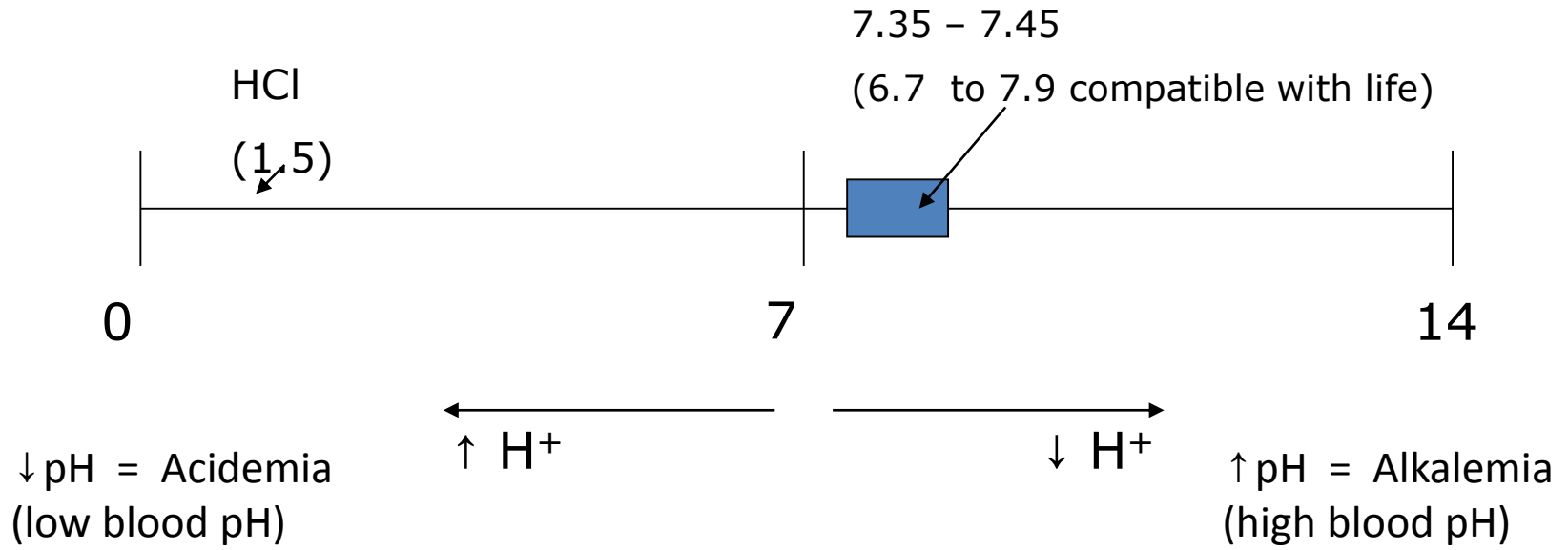


Fluids, Electrolytes and Acid-Base Balancing

# **ACID-BASE BALANCING**

- One of the most important balances in the body homeostatic mechanisms
  - Acids (proton donators)
  - Bases (proton acceptors)
  - Hydrogen ions ( $H^+$ )
  - Hydroxide ions ( $OH^-$ )

- Hydrogen ion concentration
- Measured in moles/L (represented as pH)
- Acidity/alkalinity increases tenfold with every unit change



- Recall, that changes made to a reaction already at equilibrium will result in a shift to either the left or right to return the reaction to equilibrium

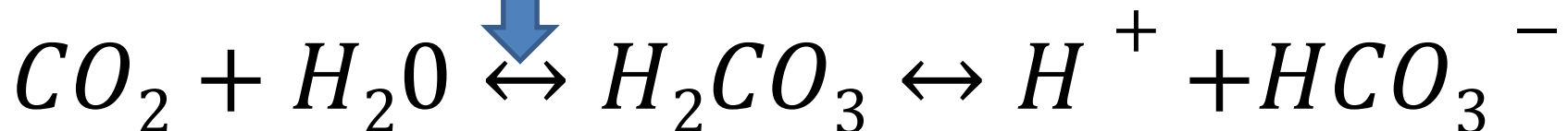


- Increase in concentration of reactants or products will cause shift away from the increase
- Decrease in concentration of reactants or products will cause a shift toward the decrease

- Chemical (rapid)
  - Carbonic acid (bicarbonate buffering)
  - Phosphate buffering
  - Protein buffering
- Physiological (secondary)
  - Respiratory buffering
  - Renal buffering

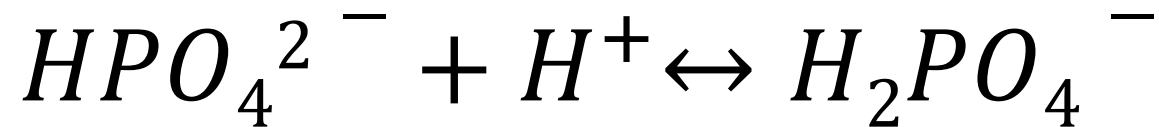
- Normal carbonic acid to bicarbonate ratio is 1 : 20 = normal pH range
  - $\text{HCO}_3^-$ ,  $\text{CO}_2$  and carbonic acid present in blood stream
  - $\text{HCO}_3^-$  results from the transport of  $\text{CO}_2$  in the blood
  - Carbonic anhydrase causes  $\text{CO}_2$  to dissolve in water in the plasma to form carbonic acid ( $\text{H}_2\text{CO}_3$ )
  - $\text{H}_2\text{CO}_3$  breaks down into  $\text{H}^+$  and  $\text{HCO}_3^-$
- Increased  $\text{H}_2\text{CO}_3$  = acidosis
- Increased  $\text{HCO}_3^-$  = alkalosis

Carbonic Anhydrase



- Negative charges allow proteins to serve as buffers for alterations in  $[H^+]$
- Primarily occurs intracellularly
  - Example:
    - In the tissues,  $CO_2$  is high. Once this  $CO_2$  enters the bloodstream, some is converted to Carbonic acid which then dissociates into Bicarb
      - This results in the release of  $H^+$  into the blood
    - Hb then combines with  $H^+$  to form a weak acid
    - At lungs Hb binds with  $O_2$  causing the release of  $H^+$
    - $H^+$  then combines with  $HCO_3^-$  ions to form  $H_2CO_3$  which is converted back to  $CO_2$  and exhaled

- Hydrogen Phosphate / Dihydrogen Phosphate equilibrium helps to buffer the intracellular fluid.



- Example:
  - If extra  $H^+$  ions enter the cell,  $HPO_4^{2-}$  can buffer the change and keep the pH within normal range
    - Results in increased  $[H_2PO_4^-]$



- Recovery of bicarbonate and filtered into tubules
- Excretion of  $H^+$  against a gradient to increase urine acidity
- Excretion of ammonium which carries  $H^+$

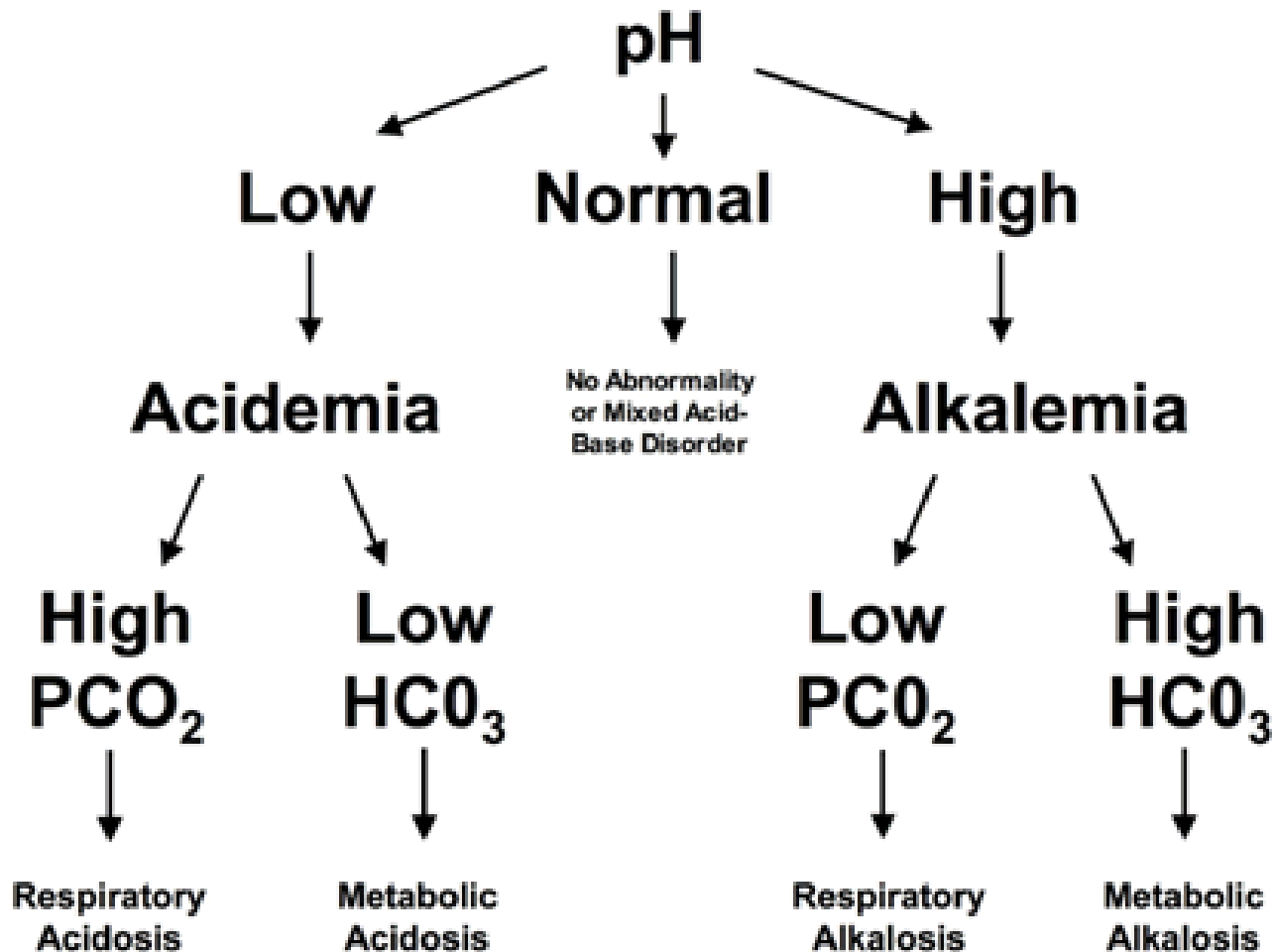
- Metabolic causes
  - $\text{HCO}_3^-$  ion
- Respiratory causes
  - $\text{CO}_2$

## Acidosis

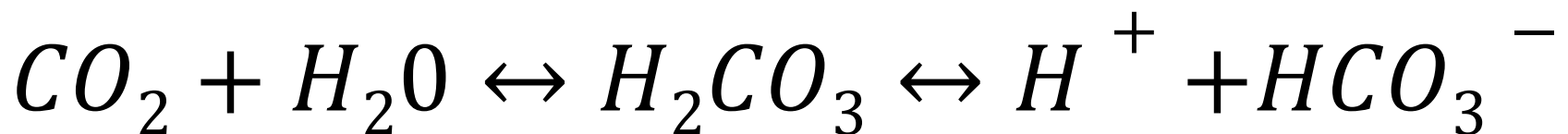
- Metabolic Acidosis
  - $\downarrow \text{HCO}_3^-$
- Respiratory Acidosis
  - $\uparrow \text{CO}_2$

## Alkalosis

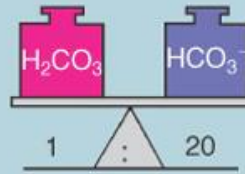
- Metabolic Alkalosis
  - $\uparrow \text{HCO}_3^-$
- Respiratory Alkalosis
  - $\downarrow \text{CO}_2$



- Metabolic Acidosis:
  - Caused by excessive accumulation of acid or deficiency in base
  - Affects the bicarbonate side of equation
  - Excessive acid production = bicarbonate buffer consumption
  - Common Types:
    - Lactic acidosis
    - Diabetic ketoacidosis
    - Renal failure
    - Ingestion of toxins



a) Metabolic balance before onset of acidosis



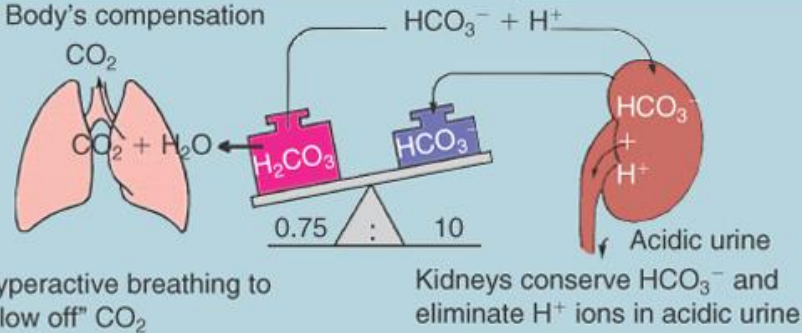
$\text{H}_2\text{CO}_3$ : Carbonic acid  
 $\text{HCO}_3^-$ : Bicarbonate ion  
 ( $\text{Na}^+ \cdot \text{HCO}_3^-$ )  
 ( $\text{K}^+ \cdot \text{HCO}_3^-$ )  
 ( $\text{Mg}^{++} \cdot \text{HCO}_3^-$ )  
 ( $\text{Ca}^{++} \cdot \text{HCO}_3^-$ )

b) Metabolic acidosis

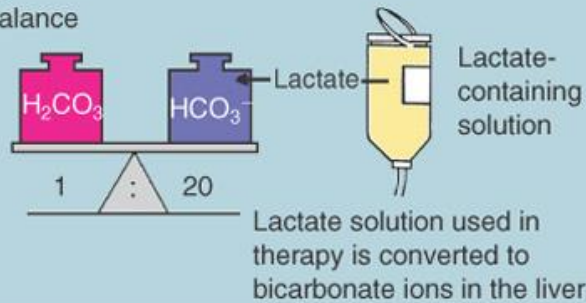
$\text{HCO}_3^-$  decreases because of excess presence of ketones, chloride, or organic acid ions



c) Body's compensation

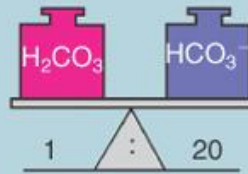


d) Therapy required to restore metabolic balance



- Metabolic Alkalosis:
  - Rare
  - Results from a loss of  $H^+$ 
    - Primarily through the GI related to excessive antacid ingestion
    - Over administration of IV  $NaHCO_3$
    - Over administration of diuretics

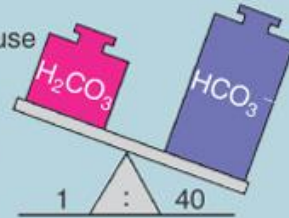
a) Metabolic balance before onset of alkalosis



$H_2CO_3$ : Carbonic acid  
 $HCO_3^-$ : Bicarbonate ion  
 ( $Na^+ \cdot HCO_3^-$ )  
 ( $K^+ \cdot HCO_3^-$ )  
 ( $Mg^{++} \cdot HCO_3^-$ )  
 ( $Ca^{++} \cdot HCO_3^-$ )

b) Metabolic alkalosis

$HCO_3^-$  increases because of loss of chloride ions or excess ingestion of sodium bicarbonate



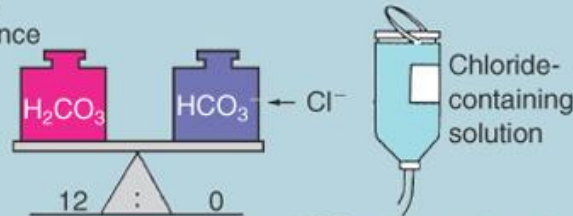
c) Body's compensation



Breathing suppressed to hold  $CO_2$

Kidneys conserve  $H^+$  ions and eliminate  $HCO_3^-$  in alkaline urine

d) Therapy required to restore metabolic balance

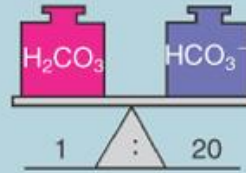


$HCO_3^-$  ions replaced by  $Cl^-$  ions



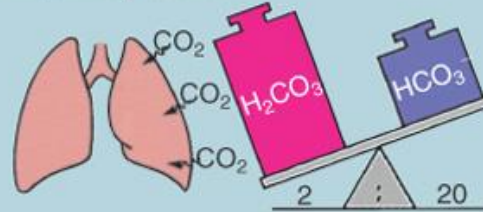
- Respiratory Acidosis:
  - Results from retained  $\text{CO}_2$  and increased  $\text{PCO}_2$ 
    - Depressed respiratory centre
      - Drug abuse, injury or disease
      - Anesthetics, sedatives, narcotics
    - Obstructive airways disease
      - Emphysema
      - Chronic Bronchitis
      - Asthma
      - Severe pneumonia
    - Blockages
      - Inhaled foreign object
      - Vomit
    - Bronchoconstriction (acute asthma)

a) Metabolic balance before onset of acidosis



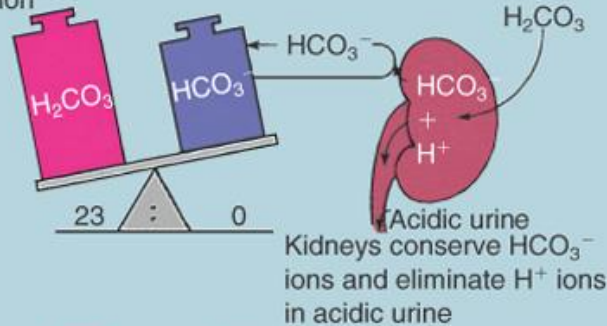
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 ( $Na^+ \cdot HCO_3^-$ )  
 ( $K^+ \cdot HCO_3^-$ )  
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 ( $Ca^{++} \cdot HCO_3^-$ )

b) Respiratory acidosis

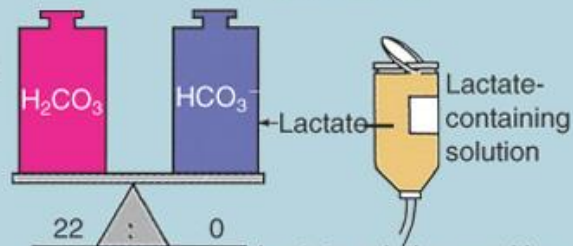


Breathing is suppressed, holding  $CO_2$  in body

c) Body's compensation



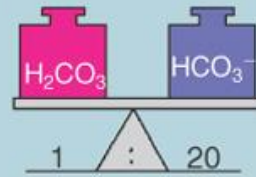
d) Therapy required to restore metabolic balance



Lactate solution used in therapy is converted to bicarbonate ions in the liver

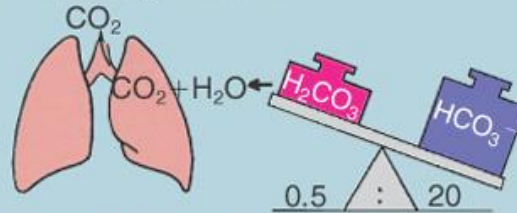
- Respiratory Alkalosis:
  - Results from decreased  $\text{PCO}_2$  through hyperventilation
    - Sepsis
    - Peritonitis
    - Shock
    - CO poisoning
    - Head injury
    - DKA

a) Metabolic balance before onset of alkalosis



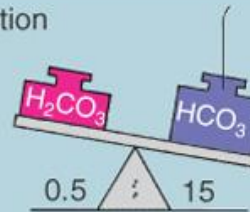
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( $K^+ \cdot HCO_3^-$ )  
( $Mg^{++} \cdot HCO_3^-$ )  
( $Ca^{++} \cdot HCO_3^-$ )

b) Respiratory alkalosis



Hyperactive breathing  
"blows off"  $CO_2$

c) Body's compensation



Alkaline urine  
Kidneys conserve  $H^+$  ions  
and eliminate  $HCO_3^-$   
in alkaline urine

d) Therapy required to  
restore metabolic balance



Chloride-containing solution  
 $HCO_3^-$  ions are replaced  
by  $Cl^-$  ions

- Determines
  - Blood oxygenation
  - Acid-base balance
- Arterial blood used to identify respiratory function
- pH indicates acidosis/alkalosis
  - $\text{PCO}_2$  indicates presence/absence of respiratory component
  - $\text{HCO}_3$  indicates presence/absence of metabolic component

Disorder	pH	Primary Disturbance	Compensatory Response
Metabolic Acidosis	↓	↓ [HCO <sub>3</sub> <sup>-</sup> ]	↓ pCO <sub>2</sub>
Metabolic Alkalosis	↑	↑ [HCO <sub>3</sub> <sup>-</sup> ]	↑ pCO <sub>2</sub>
Respiratory Acidosis	↓	↑ pCO <sub>2</sub>	↑ [HCO <sub>3</sub> <sup>-</sup> ]
Respiratory Alkalosis	↑	↓ pCO <sub>2</sub>	↓ [HCO <sub>3</sub> <sup>-</sup> ]

- Complete compensation (pH within normal limits)
- Partial compensation (pH near normal limits)
- Uncompensated (pH above or below normal limits)