K. J. Lee: Essential Otolaryngology and Head and Neck Surgery (IIIrd Ed)

Chapter 32: Fluids, Electrolytes, and Acid-Base Balance

Part II

Body Fluids

The total fluid content of the human can be estimated on the basis of 60-70% of the body weight. The intracellular water represents 40-50% of the total weight. The circulatory blood volume may be estimated as 7% of the body weight in normal man and 6.5% in women. Thus in a 70 kg man the various estimated volumes are:

Blood volume:	4.9 L
Intracellular volume:	32 L
Total body water:	49 L

The maintenance of a normal circulating blood volume is all-important to maintain the normal function of the heart as a pump and deliver adequate blood and nutrients to all organs but particularly to the lungs and kidneys for their regulatory functions.

Hypovolemia or decreased circulating blood volume may be recognized clinically by:

1. Decreased urinary output: less than 30 mL/hr with an increased specific gravity of the urine.

2. Low central venous pressure: 0-5 cm H_2O

3. Increased pulse rate. Pulse may be soft and thready.

4. Decreasing systolic blood pressure.

5. Thirst and dry skin as it becomes well established.

The ill effects of the hypovolemic state are related to the lack of circulation to all systems but impairment of cardiac function and the development of metabolic acidosis are of greatest concern. Impaired tissue perfusion causes anaerobic metabolism of carbohydrates with formation of lactic acid and increased acidosis. Acute hypovolemic states also may precipitate acute tubular necrosis of the kidney.

The ideal treatment of the hypovolemic state is the replacement of the fluid that has been lost - not only in quantity but in quality. If hemorrhage is responsible then whole blood should be replaced. Other fluid losses should be replaced with solutions of similar electrolyte content. The speed of such replacement depends on the need.

Hypervolemia or increased circulating blood volume may be recognized by:

1. Increased urinary output of a dilute urine.

2. High central venous pressure: azbove 15 cm and rising.

3. Ascites, basal pulmonary rales, and peripheral edema.

4. Distended neck veins.

5. Bounding pulse, increased cardiac rate, and blood pressure.

The ill effects of the hypervolemia are related primarily to the overloading of the heart with precipitation of heart failure and pulmonary edema. The local edema in operative wounds is undesirable because of impairment of healing and increased infection.

Treatment plans are adjusted on the apparent need to correct the overload. Simple withdrawal of intake may be sufficient but the use of diuretics, digitalization, and phlebotomy may be indicated.

Major Electrolytes in Body Fluids

The electrolyte content of the intracellular and extracellular fluids are shown in Table 32-1.

The osmolality of the serum is an expression of the osmotic pressure capability of the ionic constituents of the serum and/or extracellular fluid generated against cell membranes. It is dependent primarily on the sodium ions and the anions chloride and bicarbonate which accompany it with a significantly lesser contribution by glucose and protein. The urea diffuses so freely that it contributes very little to osmotic pressure. The normal osmolality of serum is about 285 mOsm/L. The sodium ions provide about one-half. The body regulatory mechanisms adjust to control the serum osmolality and not the level of serum sodium.

Table 32-1. Major Body Fluid Electrolytes

Substance	Extracellular Fluid (mEq/L)	Intracellular Fluid (mEq/L)
Sodium	140	10
Potassium	4	150
Magnesium	1.7	40
Chloride	105	10
Bicarbonate	28	10
Phosphate and sulfate	3.5	150
Protein anions	15	40

The composition of the intracellular fluids is quite different. The greatest ionic effect is provided by the high potassium and magnesium concentrations. The main anions within the cells are phosphate, sulfate, and protein.

For all practical purposes the body cells maintain perfect osmotic balance between the intracellular and extracellular fluids at all times. Hypoosmolality may be produced by either sodium depletion, potassium depletion, or excess of water, or a combination of the three. Hyperosmolality is produced by sodium or potassium excces or by body water depletion.

The control of the osmolality of the serum is accomplished by several factors. The intake and output of water is under CNS control. A small increase in the concentration stimulates the thirst center as well as production of antidiuretic hormone (ADH). The ADH acts on the distal tubules of the kidney causing water to be reabsorbed thus producing maximally concentrated urine. Fluid loss also occurs in sweating, via the lungs and gastrointestinal tract.

Sodium

The control of sodium loss through the kidney is another powerful mechanism. The variation in the amount of sodium filtered through the glomerulus is influenced greatly by many factors, particularly circulatory factors. The resorption of the sodium in the distal tubule is controlled by the hormone aldosterone from the adrenal glands. The aldosterone causes the cells of the distal tubule to conserve the sodium and excrete potassium. Although the body constantly secretes some aldosterone in a circadian rhythm the known stimuli include:

1. Changes in extracellular fluid volume including acute hemorrhage.

2. Diets low in sodium or high in potassium.

3. Acute heat stress.

Hyponatremia may develop with the followig clinical derangements:

1. Loss from the gastrointestinal tract with vomiting, diarrhea, and fistulae. Excessive water loss also occurs.

2. Starvation states including prolonged surgical illness.

3. Cardiac, hepatic, and renal diseases in which there is overexpanded extracellular fluid volume:

a. Right-sided heart failure

b. Cirrhosis of the liver with portal hypertension

c. Nephrotic syndrome

4. Acute tubular necrosis or other renal states in which excess fluid has been given to encourage renal output.

5. Inappropriate secretion of antidiuretic hormone after major surgical operations.

6. Certain malignant tumors produce antidiuretic hormone resulting in water retention and sodium dilution.

Hypernatremia may occur under the following conditions:

1. Stuporous patient fails to take sufficient water.

2. Inadequate or inappropriate water replacement after operation. Solutions with a high salt content given by tube feeding or intravenously may increase the obligatory urinary loss.

3. Renal disease states in which there is abnormal water loss.

Potassium

Most of the body potassium is intracellular, only about 75 mEq being in the extracellular fluid. The normal range in the plasma is 3.5-4.8 mEq/L. Daily urinary losses of 50 mEq are usual. The required intake averages 50-60 mEq/day. Unfortunately, the plasma concentration does not give an accurate reflection of the intracellular concentration, but is the best available for clinical use.

The greatest clinical effect of hypokalemia is muscle weakness. All muscles are affected, the myocardial most severely and the respiratory muscles minimally. The involvement of the intestinal musculature may be clinically manifested by gastric dilatation of paralytic ileus. Renal damage may occur with prolonged depletion. This is manifested by the inability of the kidney to concentrate urine by the absorption of water - tubular cells having been damaged.

Hyperkalemia is life threatening by its action on the heart. Muscle cells are depolarized and serious impairment of the conductive tissue results. There is a sudden onset of ventricular fibrillation. The heart stops in diastole. The voluntary muscles of the extremities may become painful and weak.

Common Causes of Hypokalemia

1. Abnormal loss of gastrointestinal fluids: vomiting, aspiration, diarrhea, fistula, disease with ulceration, or malabsorption.

2. Loss of fluids in burns or other large granulating wounds.

3. Excessive urinary loss: many renal diseases, hyperaldosteronism, alkalosis, and diuretics.

4. Metabolic disorders such as diabetes mellitus, starvation, and severe trauma.

Causes of Hyperkalemia

1. Renal failure

2. Rapid hemolysis of red blood cells as in hemolytic crisis

3. Dehydration: particularly with acidosis of diabetes

4. Adrenal failure: lack of aldosterone secretion.

Chloride Anion

The chloride in the body is the predominant anion in the extracellular fluid. The plasma chloride level is normally between 100-106 mEq/L. Most of the chloride is in the extracellular fluid. The plasma concentration is obviously affected by the state of hydration and a high concentration may be due to water loss only. The primary role of the chloride anion seems to be related to the acid-base regulation exercised by the kidneys. Depending on the metabolic state and in order to have electrical neutrality the cations excreted in the urine must be balanced by anions, mainly chloride, bicarbonate, and phosphate. Thus in a state of alkalosis with a high bicarbonate, the urinary loss of chloride will be reduced and the bicarbonate loss increased. This is one of the main methods of control of pH of the blood.

The disease state resulting from the derangement of chloride concentration is invariably associated with other related metabolic problems: acute dehydration, hypokalemia, hyponatremia, etc. Symptoms or physiologic changes cannot be attributed to the abnormality of the chloride only.

Hypochloremia occurs frequently in surgical patients with:

1. An excess of water without loss of chloride due to action of antidiuretic hormone after operation or similar stress.

2. Abnormal loss of fluid from the gastrointestinal tract: vomiting, diarrhea, fistulae, etc.

3. Renal losses: diuretics, kidney disease.

4. Chronic respiratory acidosis.

Hyperchloremia is a relatively uncommon clinical problem due to:

1. All forms of excessive water loss.

2. Operative procedure for urinary diversion into the colon. The resorption of the sodium and chloride results in hyperchloremic acidosis.

3. Administration of ammonium chloride to acidify the urine.

Acid-Base Balance

The extracellular fluids are maintained at a pH of 7.4 or in close range thereto. This figure represents the hydrogen ion concentration or the acid ion in the balance. When there is neutrality in water the hydrogen ion concentration is $10^{-6.8}$ the same as the concentration of OH-. The pH designation refers to minus the log of the hydrogen ion concentration; thus pH is 6.8 at neutrality. The plasma pH of 7.4 means that the base is slightly in excess. In plasma the predominant bases are chloride and bicarbonate ions. Phosphate and protein ions are bases of lesser importance. Bicarbonate, phosphate, and proteinate anions are buffer bases since they can modulate the effects of an increase in hydrogen ions by removing them from solution.

The measurement of the respiratory component of the acid-base balance is best accomplished by measuring the pCO_2 of plasma. A rise means that the carbon dioxide is not being cleared adequately by the lungs and a fall indicates excess removal. It may be complicated when metabolic changes also occur but it remains the best evaluation.

The kidney controls the concentration of the acids other than carbonic acid which is eliminated through the respiratory tract. The other acids are, for the most part, metabolic products of all types of food. The kidney eliminates these products by various metabolic and excretory functions. Table 32-2 indicates the fluctuations that occur with various forms of acid-base imbalance.

Respiratory Alkalosis

This occurs as a result of excessive ventilation. The pCO_2 is low. It may be caused by:

1. Overbreathing: either voluntary or involuntary.

2. Involuntary excessive respiratory exchange: exceesive blood ammonia or lesions of the central nervous system may stimulate the respiratory center.

If the compensatory mechanism does not operate quickly enough the pH will rise and symptoms of tetany will develop.

Respiratory Acidosis

This results as a result of subnormal respiration. The blood pH is either low or normal if compensated. It may be caused by:

1. Parenchymal disease of the lungs interfering with gaseous exchange.

2. Impairment of respiratory movements: as with drugs, muscle paralysis, fixation of chest wall by disease, chest wall trauma.

3. Deficient circulation through the lung by cardiovascular failure, embolism.

4. Inadequate respiratory exchange under anesthesia particularly with high oxygen concentration.

The ill effects of acidosis are primarily cardiac arrhythmias, increased susceptibility to shock, and increased vulnerability to drugs.

Table 32-2. Acid-Base Disturbances

		pH	PCO_2	Bicarbonate	Rx
Metabolic acidosis	low	low	low		Bicarbonate, ventilate
Metabolic alkalosis	high	high	high		Tromethamine (THAM)
Respiratory acidosis	low	high	high		Tracheostomy
Respiratory alkalosis	high	low	low		5% CO_2

Metabolic Alkalosis

This commonly occurs with:

1. Loss of chloride in prolonged vomiting, gastrointestinal fistulae, diarrhea.

2. Diuretics, particularly mercurials and chlorothiazide.

3. Excessive potassium loss with large bowel tumors.

4. Alkali overdosage as in ulcer therapy.

The significant loss of potassium which frequently accompanies the chloride loss is the chief concern of this metabolic state. The dehydration also may be of significance.

Metabolic Acidosis

This is one of the most common and serious derangements in the surgical patient. Frequent causes include:

1. Low blood flow with impaired tissue perfusion: shock, cardiac decompensation, hypovolemia.

2. Renal failure.

3. Diabetes mellitus.

The various metabolic processes produce acidosis which under normal conditions may be buffered and eliminated. With poor tissue perfusion anaerobic metabolism of carbohydrate produces an excess of lactic acid adding to the acid load which must be buffered by the bicarbonate buffer system. The lungs cannot remove the other acids produced in normal or abnormal metabolism. Only the normal kidney can effectively offer a complete control or resolution of metabolic acidosis.

The ill effects of the acidosis are the same whether the origin is respiratory or metabolic.