CHAPTER **32**

EVALUATION OF FLUID AND ELECTROLYTE BALANCE IN THE PREOPERATIVE SETTING

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The aim of preoperative preparation is to ascertain the patient's ability to withstand the stresses of surgery and minimize potential risks. Chronic nutritional deficits cannot be rectified in the immediate preoperative setting.[1] However, fluid volume depletion and electrolyte abnormalities can be identified and reversed with intensive intravenous therapy and laboratory testing. Although variation exists according to body cell mass, weight, age, and body fat composition, the volumes of body fluid compartments are surprisingly stable for patients under normal conditions.[1-3] The extracellular fluid compartment however, is dynamic compared with the intracellular compartment.

Water constitutes 22 percent of body weight. The average human oral intake of water is 2000 - 2500 ml per day. This consists of fluid, solids, and by-products of cellular metabolism. Healthy people expel 700 to 1600 ml of water per day as urine, 300 ml as stool, and up to one liter as insensible loss via respiration and perspiration.[4,5] Fluid homeostasis is a delicate balance, ever threatened with disruption by illness or disease. With adequate renal function, however, homeostasis is possible despite mild fever, diarrhea, and vomiting.

Prolonged periods of "nothing per os" (NPO) is at minimum partially responsible for volume depletion in the majority of surgical patients undergoing general anesthesia or monitored anesthesia care. It has been suggested that prolonged liquid fasting is unnecessary and may be harmful in healthy patients prior to ambulatory surgery; ingestion of coffee and pulp-free orange juice (250 ml) before surgery does not increase gastric volume.[6] Goresky and Maltby[7] have offered the following recommendations for elective surgical patients to reduce ECF fluid depletion: unrestricted intake of clear fluids until three hours prior to surgery, allowing oral medications with 30 ml of water up to one hour preoperatively. Less restrictive guidelines may reduce the incidence of patients arriving at the hospital dehydrated or volume depleted the day of surgery. Preoperative emphasis should be placed on correction of volume deficits; however, electrolyte abnormalities should also be addressed particularly when symptomatic.[4]

Certain subgroups of patients require precise determinations of fluid and electrolyte requirements secondary to compensatory deficits. These patients typically have cardiac, renal, or hepatic disease. Diabetic patients with extensive infection or a plantar space abscess may have such compensatory deficiencies. In these patients stringent attention to fluid intake, urinary output records, central venous pressure measurements, and exogenous fluid or solute losses are mandatory. Once fluid and electrolyte requirements are calculated, the rate of IV administration is determined by the patient's cardiovascular status, the scale and urgency of the proposed surgery, renal function and the severity of preexisting abnormalities.

EXTRACELLULAR VOLUME DEPLETION

The most common fluid problem in the preoperative setting is extracellular volume depletion.[5] The diagnosis is ascertained through a thorough history and physical examination. Physical findings of dry mucous membranes, furrowed tongue, sunken eyes, and decreased skin turgor are hallmarks. A fluid deficit of 3-5 liters is necessary for altered skin turgor, and shrunken tongue.[8] Quantifiable parameters include heart rate, body weight, and urine output changes. Volume depletion results in tachycardia and oliguria. Urine osmolarity increases, and urine sodium concentration decreases.[9]

Fluid revitalization of ECF depleted patients is initiated with isotonic 0.9% NaCl infusion and is changed to lactated Ringer's solution when adequate urine output is present. With normal renal function and in the absence of glycosuria, hourly urine output between 35 and 50 ml signifies a normalizing extracellular fluid volume.[10]

ABNORMALITIES OF ELECTROLYTE COMPOSITION

Isolated potassium, magnesium, and calcium abnormalities are rarely seen in the preoperative patient and usually occur in association with volume, sodium, and acid-base disorders. It would be unusual and suspect for a patient to present to the preoperative holding area with significant electrolyte abnormality that went unrecognized after appropriate preadmission testing.

Sodium

The major function of sodium is to maintain ECF volume. Hyponatremia may be caused by actual sodium loss or by an accumulation of water in excess of sodium. A common cause is the administration of D5W to patients with a preexisting sodium chloride deficit.[11] Other causes include severe vomiting, diarrhea, diuretics, or SIADH.[12] The clinical appearance of hyponatremia is similar to isotonic dehydration (dry tongue, mucous membranes, oliguria, tachycardia). Patients may also have impaired sensation of taste, apprehension, and focal weakness such as hemiparesis when serious.[13] Secondary hyponatremia may present as nervous system abnormalities such as increased deep tendon reflexes progressing to convulsions.

Primary hyponatremia is treated by salt and volume replacement. The otherwise healthy patient with mild sodium deficiency should undergo repletion with isotonic NaCl. The sodium deficit can be calculated by multiplying the decrease in serum sodium concentration below normal (140 mEq/L) by the estimated normal total body water in liters (body weight in kg x 0.60). This will guide whether isotonic, or hypertonic (3% or 5%) NaCl is most appropriate for symptom relief. Moderate to severe hyponatremia is best treated initially with 3% to 5% NaCl.[14]

Hypernatremia is often iatrogenic, produced by sodium administered in excess of water. Symptomatic hypernatremia may be insidious, however most patients recognize the initial thirst and prevent or correct this disorder themselves with increased oral fluid intake. Other symptoms include irritability, swollen tongue, and red, dry, sticky mucous membranes. The goal in correcting hypernatremia is to gradually lower the serum sodium level. Treatment is initiated by infusion of a hypotonic electrolyte solution such as 0.45% NaCl solution with close monitoring of the serum sodium concentration. If the extracellular osmolality is reduced too rapidly, as it might be when D5W is given, convulsions and coma may occur secondary to cerebral edema as water rapidly crosses the blood-brain barrier.[15]

Potassium

Potassium is the major intracellular cation, with a concentration 30 times greater than plasma. Potassium is acquired through diet and must be ingested daily as the body has no effective storage mechanism.[16] Its key functions are transmission of nerve impulses and influence on both skeletal and cardiac muscle activities. Many conditions lead to potassium deficit, including prolonged diuretic use, glucocorticoids, laxatives, and ingestion of certain varieties of licorice.[17] Other symptoms include anorexia and EKG changes including ST-segment depression, flattened T wave, and the presence of a U wave.[18] Patients with hypokalemia may experience fatigue, muscle weakness, diminished deep tendon reflexes, irritability (early) and flaccid paralysis (late).[19] Clinical signs and symptoms rarely occur before the serum potassium has fallen below 3 mEq/L.

Mild hypokalemia is treated with dietary increases including salt substitutes, which contain potassium. Mild abnormalities do not present significant danger to the surgical candidate. When hypokalemia is severe enough to induce cardiac abnormalities, potassium chloride can be infused intravenously at 20 mEq per hour if renal function is adequate and cardiac monitoring is continuous. IV potassium chloride is associated with pain and phlebitis when administered peripherally. This may be avoided by administering the 20 mEq KCl supplement via central venous catheter in 50 ml 5% dextrose in water.[20]

Hyperkalemia can be a life-threatening emergency and requires prompt treatment.[21-23] The primary cause is renal failure. It seldom occurs in patients who have normal renal function. The most common mechanism is decreased urinary excretion. Hyperkalemia also occurs following sepsis or major tissue destruction secondary to trauma causing K+ liberation from cells (i.e. crush injury). Symptoms include flaccid paralysis, vague muscle weakness, anxiety, nausea and diarrhea. EKG changes include widened QRS complex, prolonged PR interval and ventricular arrhythmias.[17]

In the presence of renal failure, treatment consists of promoting intracellular transfer of potassium as well as increasing potassium excretion. Hypertonic dextrose plus insulin (1 unit/10g dextrose) can shift potassium intracellularly.[24] Neuromuscular toxicity and life-threatening cardiac arrhythmias can be inhibited somewhat by IV calcium gluconate[21] Hemodialysis or peritoneal dialysis can be used to alleviate life-threatening hyperkalemia if other methods are unsuccessful.

Calcium

Calcium is most abundant in the skeletal system, with 99% located in the bones and teeth. Less than 1% is available for rapid exchange in the circulating blood bound to protein. Calcium is instrumental in activating enzymes and stimulating essential chemical reactions. It plays an important role in maintaining normal transmission of nerve impulses. Hypocalcemia is most commonly caused by inadequate secretion of parathyroid hormone, due to hypoparathyroidism.[25] Other causes include loss through diarrhea, wound exudate, pancreatitis, sepsis, inadequate vitamin D, or hyperphosphatemia associated with renal failure. Patients on loop diuretics and digoxin are predisposed to hypocalcemia.[26,27] Signs and symptoms include numbness of the fingers, extremity muscle cramps, positive Trousseau's sign (carpopedal spasm when a tourniquet is applied above systolic pressure to the forearm) and Chvostek's sign (repeated contraction of facial muscles with percussion just below the zygomatic bone). Prolonged QT interval and altered cardiovascular hemodynamics may precipitate congestive heart failure. The most dangerous symptom associated with hypocalcemia is the development of laryngospasm and tetany.

Oral administration of calcium gluconate with calcium supplements is preferred. Intravenous calcium gluconate (10-20 ml of a 10% solution in 5% dextrose in water) over 20 minutes is also effective. IV therapy is viewed as second line treatment.

Hypercalcemia can be very dangerous with a 50 percent mortality rate if untreated.[10] Patients with hypercalcemia have one of the following conditions: malignancy, hyperparathyroidism, or thiazide diuretic use.[29] Signs and symptoms include muscle weakness, lethargy, deep bony pain, pathologic fractures, polyuria or polydipsia.

Treatment with 0.45 % NaCl or 0.9% NaCl IV improves the serum calcium levels.[30] This causes saline diuresis, allowing rehydration and promoting urinary excretion. In life-threatening situations hemodialysis or peritoneal dialysis will reduce serum calcium. Ancillary treatments include inorganic phosphates, furosemide, calcitonin, and bisphosphonates.

OVERVIEW OF PARENTERAL FLUIDS

IV fluids are used for fluid and electrolyte management as well as solutions or admixtures for IV drug therapy. It is important for the podiatric physician to be familiar with the selection and administration of various IV fluids. Crystalloid solutions are used most commonly in podiatric medicine. These include: D5-1/2NS, D5LR, LR, and D5W. Colloids such as albumin, and blood products are rarely indicated in podiatric practice.

Crystalloid Solutions

5% Dextrose in Hypotonic Saline (D5NS). 1/4, 1/3, 1/2 Normal Saline

- Replaces excessive fluid loss
- Promotes diuresis
- Prevents alkalosis
- Provides NaCl and calories (170 cal/1000ml)

Lactated Ringers Solution (LR). Na, K, Ca, Cl, Lactates

- (With or without 5% dextrose)
- Restores normal fluid shifts in intraoperative phase of surgical procedures, infections and burns
- Moderates metabolic acidosis (Diabetic ketoacidosis)
- When using extensively, be alert to alterations in blood pH.
- Metabolic alkalosis is a possibility.

Normal Saline .9 NaCL. (NS)

- Used in diabetic acidosis
- Treats alkalosis
- Replaces excessive fluid loss
- Do not use in edema
- Normonatremic patient can become hypernatremic with large quantities of .9 NaCL.

5% Dextrose in water (D5W).

- Prevents and treats ketosis
- Promotes Na diuresis
- Prevents dehydration

Hypotonic Fluid Selection

D51/2 NS	Routine use
D51/3 NS	Contolled hypertensive patients, routine use
D51/4 NS	Uncontrolled Hypertensives
D5W	Diuretic action

Fluid Replacement Calculation

Using D51/2 NS or other fluid:
For the first 10 kg body weight:100 ml/kg/day plus
For the second 10 kg body weight: 50 ml/kg/day plus
For the weight above 20 kg: 20 ml/kg/day

Example:
Patient weighs 98 kg (Approximately 200 lbs.)
10 kg x 100 ml= 1000 ml
10 kg x 50 ml= 500 ml
70 kg x 20 ml= 1400 ml
90 kg 2900 ml D5-1/2 NS per day

A 70 kg patient would require 2500 ml per day by the same calculation. Geriatric patients with CHF or other cardiac patients would require less total fluid. This method of calculation is also applied to the pediatric patient.

Flow Rates

The hourly IV flow rate is calculated as follows: total daily IV fluid requirement/24 hours = Estimated hourly rate (ml/hr).

KVO (keep vein open) or TKO (to keep open) are interchangeable. This flow rate is 50 ml/hr and is merely adequate to prevent retrograde flow into the tubing.

The average rate for healthy nonsurgical podiatric patients with no unusual problems or physical demands 75 to 80 ml/hour. May be used in absence of fever in patients receiving IV antibiotics.

The most commonly used rate is 100 ml/hour. This rate is appropriate following major elective bone surgery.

The flow rate for patients with a fever of 102 degrees or below, or for postoperative management following major hindfoot or ankle trauma is 125 ml/hour.This rate is also used for antibiotic piggyback administration while administering the antibiotic.

The flow rate for a patient with a fever above 102.6 degrees is 150 ml/hour.

IV Antibiotic Administration

There are 3 primary methods of IV antibiotic administration: direct IV injection without dilution; piggyback method in which the antibiotic is diluted in 100 ml of D5W or D5-1/2 NS and is infused at a rate of 125 ml/hour (the most common method of administration); and heprin lock.

A heprin lock or "Hep Lock" is an IV catheter with an injection cap through which medications (antibiotics diluted in 100-250ml of fluid) are run at the required intervals. The use of a heprin lock eliminates the need for constant IV fluids. To maintain patency, the Hep Lock is flushed with sterile saline before and after each antibiotic dose. After the postadministration flush, a special heparin solution is placed in the catheter until the next preadministration flush. The heparin is not sufficient to cause coagulation alterations. It is used only when there is no IV fluid requirement. This method is convenient for patients becase they are no longer tethered to an IV pole, monitor, or tubing.

SUMMARY

The podiatric surgeon should be familiar with symptoms and general treatment parameters for typical volume and electrolyte disorders. Treatments of electrolyte imbalances are most appropriately instituted by primary care physicians. It is impossible to provide "cook book" intravenous therapy rates that can be used for all patients. Fortunately, this fact encourages the majority of specialized health care providers to consult with the appropriate primary care physician or specialist coordinating care who is most familiar with these disorders and their management.

The patient's best interests are maintained via a team approach to surgical candidates with fluid and electrolyte imbalances. Interdisciplinary coordinated care is paramount for a successful outcome and the avoidance of potentially serious complications.

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