FLUID, ELECTROLYTES, ACID-BASE AND SHOCK

Objectives:

1. Discuss the importance of fluids, electrolytes and acid-base elements in ensuring/maintaining proper body function.
2. Describe the movement of fluids, electrolytes and other substances throughout the body by the following: diffusion, osmosis, pressure differential, and other essential mechanisms.
3. Relate common fluid, electrolyte, and acid-base laboratory values and diagnostic tests to normal physiology and pathological disease processes.
4. Discuss common causes of fluid, electrolyte and acid-base imbalances
5. Discuss nursing responsibilities for assessment, identification, treatment and follow-up of fluid, electrolyte and acid-base imbalances
6. Discuss nursing responsibilities for hypovolemic, cardiogenic, neurogenic, anaphylactic and septic shock.

Readings:

2. "Emergency: Hyperkalemia," AJN, Jan 2000, article following content

Please utilize the readings to supplement and further elaborate the topics below, to fulfill the objectives of the course.

FLUIDS, ELECTROLYTES and ACID-BASE STATUS

I. GENERAL CONSIDERATIONS:
   A. The body likes to remain in balance and employs various mechanisms to help maintain homeostasis
   B. Acute rapid changes in fluids and/or electrolytes are more ominous than slow gradual changes
   C. Water, electrolytes and other substances are present in many different areas of the body, yet serum laboratory values only measure conditions in
the intravascular space so cellular and other changes often must be inferred

D. Treatment of imbalances always includes correcting the underlying cause, when possible. In addition, when other treatments are employed, must be careful not to over treat; body is employing its own mechanisms to counteract and serum changes often lag slightly behind treatment so treatment is aimed at approaching, not reaching normal in order to prevent ending up with exact opposite problem that was being treated (e.g., treatment for hyponatremia causing hypernatremia); frequent laboratory and clinical checks are essential

**BODY FLUIDS**

I. **FUNCTION:** vital to life; help maintain body temperature and cell shape; involved in transporting nutrients, gases, and wastes; principle fluid in body is water; skin, lungs and kidneys work together to maintain the proper fluid balance

II. **FLUID COMPARTMENTS:** 2 main; separated by capillary walls & cell membranes:
   A. *intracellular fluid (ICF):* within cells; 40% of body weight and 70% of total body water
   B. *extracellular fluid (ECF):* outside cells; 20% of total body weight and 30% of total body water; comprised of:
      1. *interstitial fluid (ISF):* between cells; ~2/3 ECF
      2. *intravascular fluid (IVF):* plasma; ~1/3 ECF; high protein concentration
      3. *transcellular (TCF):* GI tract, peritoneal, cerebrospinal, pleural, and synovial fluid; small ~1L of ECF

III. **BODY WATER DISTRIBUTION & SPACING:**
   A. Distribution varies with age, sex and body composition. Percentage of body weight that is water is about 80% in a full-term infant, 60% in a typical lean, adult male, and 45-50% of obese and elderly. This puts infants, elderly, and obese individuals at greater risk for fluid related problems.
   B. Fluid spacing is a term used to classify the distribution of water in the body:
      1. **First Spacing:** describes normal distribution of fluid in the body in both the intracellular and extracellular fluid compartments.
      2. **Second Spacing:** describes the excess accumulation of fluid in the interstitial spaces, which we also call edema.
3. **Third Spacing**: occurs when fluid accumulates in areas that normally have no fluid or minimal amount of fluid, such as with ascites, and edema associated with burns. In extreme cases third spacing can cause a relative hypovolemia.

4. **Fluid Status**: 1 liter of water weighs 2.2 pounds. A sudden weight gain or loss is the best indicator of fluid status. Patients that need to have their fluid status monitored should have not only their Intake and Output measured, but also daily bedside weights.

### IV. FLUID, ELECTROLYTE & PARTICLE MOVEMENT

**A. Electrolytes move between ICF & ECF via concentration (toward lower concentration) & electric gradients (toward opposite charge)**

1. **Diffusion**: movement of molecules from an area of higher concentration to one of lower concentration.

2. **Facilitated Diffusion**: addition of specific carrier molecule to aid/accelerate diffusion (e.g., glucose transport into cell facilitated by insulin)

3. **Active transport**: molecules moved from area of low to higher concentration; external energy (ATP) allows movement against concentration gradient (e.g., “Sodium Pump”, where potassium is moved into cell and sodium pumped out)

**B. Water moves according to pressure forces**

1. **Hydrostatic pressure**: pressure of blood fluid against capillary wall; if greater than pressure in interstitial space, fluids and solutes forced out of blood; if less, come back into blood

2. **Osmosis**: water movement from area of higher to lower concentration (“diffusion of water”) through membrane permeable to water, but not to solute; no energy needed; stops when solution concentrations equalize

   a) **Osmotic pressure**: movement of water by osmosis; measured by osmolarity (mOsm/L) and osmolality (mOsm/kg); kidneys are mainly responsible for maintaining concentration of body fluids within normal range of osmolality through changes in antidiuretic hormone (ADH) secretion; normal osmolality is 275-295 mOsm/kg

   b) **Tonicity & IV solutions**: a solution’s solute concentration (e.g., I.V. fluid) compared to another solution (e.g., blood); its “effective osmolality”; IV solutions are categorized into:

      1. **Isotonic**: same effective osmolality as body fluids (e.g., 0.9% NaCl, D5W (prior to infusion), Lactated Ringers); causes intravascular expansion and possibly some interstitial edema but no water shifts into or out of cells.
(2) **Hypotonic**: solute concentration, <270 mOsm/kg (e.g., 0.2 or 0.45% NaCl, D5W (once in the body); causes a shift of water out of vascular space so can result in interstitial and cellular edema

(3) **Hypertonic**: higher solute concentration, >300 mOsm/kg (e.g., 3% NaCl, D5NS, D5 0.45 or D5 0.9% NaCl); causes water to be pulled into vasculature from the interstitium and cells so can result in cellular dehydration and vascular volume overload.

3. **Plasma colloid osmotic pressure (COP)**: osmotic, or pulling force of albumin in the intravascular space; opposes hydrostatic pressure to maintain equilibrium of fluid leaving & returning (if normal BP and plasma protein levels). Hypertension or hypoproteinemia (e.g., malnutrition, malabsorption, impaired albumin synthesis or protein loss), causes hydrostatic pressure to surpass COP and edema results.

V. **FLUID BALANCE MECHANISMS**

A. **Kidneys**: primary regulator of fluid & electrolyte balance through changes in urine volume and electrolyte excretion; renal dysfunction causes multiple fluid & electrolyte disturbances

B. **Thirst**: center in hypothalamus stimulated by hypotension and increased serum osmolality, decreased with hyponatremia; thirst mechanism less effective in elderly, so more prone to dehydration.

C. **Antidiuretic hormone (ADH)**: when low blood volume and increased serum osmolality, hypothalamus signals pituitary gland to secrete ADH which causes the kidneys to retain water, thus increasing blood volume and decreasing serum osmolality. Syndrome of inappropriate ADH (SIADH) results in too much water retention whereas diabetes insipidus (DI) causes diminished ADH secretion and too much water loss.

D. **Renin-Angiotensin-Aldosterone System**: kidney’s juxtaglomerular cells secrete renin when low flow; renin converts angiotensinogen to angiotensin I in the liver; angiotensin I is converted into angiotensin II, a potent vasoconstrictor in the lungs; angiotensin II also stimulates the adrenal glands to produce aldosterone which causes the kidneys to retain sodium and water. High aldosterone is associated with Cushing’s disease and hyperadrenocorticism whereas low aldosterone is associated with Addison’s disease and adrenal insufficiency. ACE inhibitors work by blocking this system.

E. **Atrial Natriuretic Peptide (ANP)**: released when atrial pressure increases; ANP decreases serum renin levels, aldosterone and ADH release, increases glomerular filtration and causes vasodilation; opposes the renin-angiotensin-aldosterone system by decreasing blood pressure and reducing intravascular blood volume. The amount released rises in
response to a number of conditions, including chronic renal failure and heart failure.

F. **GI & Skin:** GI responsible for most of water intake; diarrhea & vomiting can lead to fluid & electrolyte disturbances; insensible losses are water only whereas sweating causes loss of both fluids & electrolytes

VI. **FLUID IMBALANCES:** treatment is aimed at correcting the fluid and sodium (if present) imbalances

A. **Hypovolemia (fluid volume deficit, dehydration):**
   1. **Isotonic:** Normal Na+; causes include blood &/or GI losses; clinical manifestations include dry mucous membranes, poor skin turgor, decreased level of consciousness (LOC), flat neck veins, "shocky" (hypotension, orthostatic initially; tachycardia; weak, thready pulse, cool & clammy skin), sunken fontanelles in infants;
   2. **Hypotonic (hypo-osmolar, hyponatremic):** Low Na+ (Na+ loss exceeds water loss); results in cell swelling/edema as water leaves ECF to correct hypo-osmolar condition; causes include GI fluid loss, diuretic abuse, hypoaldosteronism, adrenal insufficiency; clinical manifestations include s/s of both isotonic fluid deficit plus those of hyponatremia;
   3. **Hypertonic (hyperosmolar, hypernatremic):** High Na+ (water loss exceeds Na+ loss); results in cell shrinkage/dehydration as water is drawn into ECF to correct hyperosmolar condition; causes include profuse sweating, watery diarrhea, poor HOH intake, diabetes insipidus; clinical manifestations include s/s of both isotonic deficit plus those of hypernatremia

B. **Hypervolemia (fluid volume excess, overload, overhydration):**
   1. **Isotonic:** Normal Na+; causes include CHF, renal problems, iatrogenic (IV NS, LR, blood); clinical manifestations include moist mucous membranes, good skin turgor, edema, distended neck veins, possible hypertension, rales, dyspnea, rapid, bounding pulse, warm (hot) skin, bulging fontanelles in infants;
   2. **Hypotonic (hypo-osmolar, hyponatremic):** Low Na+ (Na+ loss exceeds water loss); results in cell swelling/edema as water leaves ECF to correct hypo-osmolar condition; causes include CHF, water intoxication, SIADH, renal failure, hypotonic IVs; clinical manifestations include s/s of both isotonic fluid volume excess plus those of hyponatremia;
   3. **Hypertonic (hyperosmolar, hypernatremic):** High Na+; (Na+ gain exceeds water gain); results in cell shrinkage/dehydration & greater ECF fluid expansion as water is drawn into ECF to correct hyperosmolar condition; causes include hypertonic Na, hyperaldosteronism, ↑ Na intake, Na drugs; clinical manifestations
include s/s of both isotonic fluid volume excess plus those of hypernatremia

**ELECTROLYTES**

**I. GENERAL CONSIDERATIONS:**

A. Disassociate into ions in water
   1. Cations: positively charged; Sodium (Na+), Potassium (K+), Calcium (Ca++), and Magnesium (Mg+)
   2. Anions: negatively charged; Bicarbonate (HCO3-), Chloride (Cl-), Phosphate (PO4-)

B. Composition of Fluid Compartments:
   1. ICF: K+ primary cation; PO4- primary anion
   2. ECF: Na+ primary cation; Cl- primary anion

C. Functions: regulate water distribution & acid-base balance, transmit nerve impulses; important for blood clotting and ATP generation

D. Measured in milligrams/deciliter (mg/dl) or combining power of millequivalents/liter – mEq/L) which is their chemical activity; serum electrolyte levels reflect the ratio of the electrolyte to water, not necessarily loss or gain of the electrolyte

E. Rapid, acute changes are more ominous than slower, gradual changes

**II. SODIUM (Na+) (Normal 135-145 mEq/L)**

A. Function: main ECF cation; maintains ECF concentration & volume (“water follows salt”); primary determinant of ECF osmolality (roughly 2 times serum Na+); transmits nerve & muscle impulses; helps maintain acid-base balance

B. **Hyponatremia:**
   1. water moves from ECF into cells resulting in ICF excess and cellular swelling
   2. causes include loss via Gl fluids (vomiting, diarrhea, fistulas, gastric suctioning), excess sweating with only water replacement, diuretics, particularly with a low-salt diet, aldosterone deficit (adrenal insufficiency [e.g. Addison's], etc.), burns & wound drainage; or excess water gains from hypotonic IVs (D5W, .25 or .45 NaCl), CHF, SIADH, compulsive water drinking (water intoxication), renal failure with excess fluid intake
   3. clinical manifestations may vary; primarily neurological (headache, mental status changes: short attention span, lethargy, confusion leading to stupor, coma, seizures with serum Na+ 110 mEq/L); may also have nausea, abdominal cramps, muscle twitching,
tremors, or weakness plus s/s volume disturbances, either hyper- or hypo-volemia

4. treatment varies with cause & severity: restricted fluid intake if mild hyponatremia with iso- or hyper-volemia; if hypovolemic, isotonic IV fluids such as normal saline to restore volume; hypertonic saline (e.g., 3% or 5%) if significant hyponatremia (caution: must monitor closely for circulatory overload or worsening neurologic status)

C. **Hypernatremia:**

1. water moves from cells into ECF resulting in cell shrinkage and ICF deficit; not as common as hyponatremia because increased thirst and ADH in response to high serum osmolality usually restore balance

2. causes include water loss from fever, heat stroke, huge insensible water loss (e.g., hyperventilation, extensive burn denuding), watery diarrhea, DI; water deprivation (unconscious, debilitated, in infants, young children or retarded individuals, hypertonic (e.g. high protein) tube feedings); or sodium gain from excessive intake (salt tablets, food, medications, IV hypertonic saline or NaHCO3), hyperaldosteronism (Cushing’s), salt water near drowning

3. clinical manifestations include neurologic changes which are most important because of effects of fluid shifts on brain cells (restlessness, agitation, weakness, lethargy, confusion, stupor, seizures, & coma); also low grade fever, flushed skin, intense thirst, muscle twitching; may also have s/s of hypo or hyper-volemia.

4. treatment includes stopping water loss and replacing fluids (often po water or hypotonic IV solutions); fluids should be replaced gradually to avoid shifting water into brain cells; often diuretics along with fluids; restriction of sodium intake

III. **CHLORIDE (Cl-) (Normal 96-106 mEq/L)**

A. Function: main ECF anion (high levels in cerebrospinal fluid; also in bile, gastric & pancreatic juices); helps maintain ECF osmolality, acid-base balance; associated directly with Na+ and inversely with HCO3-

B. **Hypochloremia:**

1. causes include hypochloremic alkalosis (Cl- loss > Na+ loss; e.g., NG sx.), losses thru skin, GI tract, kidneys; changes in Na, K or acid base can alter Cl

2. clinical manifestations include agitation, irritability, coma & seizures; arrhythmias; slow, shallow respirations; muscle cramps & weakness, hypertonicity, hyperactive reflexes, tetany; may also have s/s of hypo-Na &/or –K, metabolic alkalosis
C. **Hyperchloremia**

1. causes include increased intake (e Na), drugs (ammonium chloride, Kayexalate (Cl- exchanged for K+), carbonic anhydrase inhibitors [acetazolamide]); hyperaldosteronism (Cl passively reabsorbed
2. clinical manifestations include decreased LOC, coma; hyperventilation, arrhythmias, lethargy, weakness; major s/s r/t metabolic acidosis; also may be r/t hyper-Na & -volemia
3. treatment includes nonsalty fluids or IVs (LR (if OK liver fcn.) or NaHCO3); possibly diuretics

IV. **POTASSIUM (K+) (Normal 3.5-5.5 mEq/L)**

A. Function: major ICF cation; regulates cell excitability & affects cell’s electrical status, allowing electrical impulses to travel from cell to cell; helps control ICF osmolality

B. **Hypokalemia:**

1. causes include losses from GI tract, kidneys, skin, dialysis; shift into cells and/or decreased intake
2. clinical manifestations include cardiac (e.g., irregular weak pulse, bradycardia, ventricular ectopics, U wave, dig toxicity), decreased GI and neuromuscular function
3. treatment with oral or IV potassium (caution with IV potassium administration)

C. **Hyperkalemia:**

1. causes include excess intake, shift out of cells, and decreased elimination
2. clinical manifestations include cardiac (e.g., irregular pulse, peaked T & widening QRS, ventricular fibrillation/standstill); irritability, anxiety; abdominal cramping & diarrhea, lower extremity weakness & paresthesias
3. treatment includes stopping intake, increasing elimination (diuretics, dialysis, Kayexalate, increased fluid intake); temporary shift into cells (insulin & glucose or NaHCO3); calcium gluconate (reverse membrane effects of hyperkalemia)

V. **CALCIUM (Ca++) (Normal: Total 9-11 mg/dl; Ionized 4.5-5.5 mg/dl)**

A. Function: major cation in teeth and bones (99% & combined with phosphorus); ~ equal concentrations in ICF and ECF; also in cell membranes (helps cells stick together & maintain shape); enzyme
activator within cells (muscles need calcium to contract); helps coagulation; affects cell membrane permeability and firing level; sedative effect on nerve cells; important for muscle contraction; ½ of serum calcium ionized (only part physiologically important) while other half is bound or combined with anions, protein mainly

B. Hypocalcemia:
1. causes include
   a) Decrease in total body calcium from chronic renal failure or alcoholism, loop diuretics; magnesium or vitamin D deficiencies or elevated phosphorus, GI losses (pancreatitis, diarrhea), hypoparathyroidism, decreased albumin
   b) Decrease in ionized calcium from Alkalosis, large amounts citrated blood or hemodilution;
2. clinical manifestations include increased neuromuscular excitability (e.g., paresthesias, hyperactive reflexes, tetany, laryngospasm), diarrhea, anxiety, seizures, decreased cardiac function (prolonged QT & ST, heart block, decreased cardiac output and dig effect)
3. treatment includes correction of high phosphorus & low magnesium levels first, then po or IV calcium replacement (caution with IV); po vitamin D

C. Hypercalcemia:
1. causes include:
   a) increased total calcium: increased intake, intestinal absorption (e.g., vitamin A & D overdose), bone release (hyperparathyroidism, malignancies, prolonged immobilization, etc.); decreased excretion (e.g., thiazide diuretics)
   b) increased ionized calcium (iCa++): acidosis;
2. clinical manifestations include decreased neuromuscular excitability, GI motility and neurological irritability; increased cardiac irritability (decreased QT & ST, ventricular arrhythmias, increased dig effect) and renal stones, polyuria
3. treatment includes decreased calcium intake, rapid hydration (po, or IV NS) if client tolerates to increase urinary excretion, then loop diuretics; may also use IV phosphate, drugs to decrease bone resorption (e.g., calcitonin, EDTA), and dialysis if renal failure

VI. MAGNESIUM (Mg+) (Normal 1.5-2.5 mEq/L)
A. Function: ~55% in bone, ~44% in cells, ~1% in ECF (~2/3 ionized (physiologically important) while ~1/3 bound to protein with small amount complexed; contributes to enzyme and metabolic processes, particularly protein synthesis; triggers Na-K pump modifies nerve impulse transmission and skeletal muscle response
B. **Hypomagnesemia:**
1. causes include decreased intake/absorption (e.g., malnutrition, malabsorption); increased GI (vomiting, diarrhea, NG sx.) or renal (DKA, diuretics, some drugs), hyperaldosteronism; at particular risk are alcoholics and critical care patients
2. clinical manifestations include confusion, increased neuromuscular excitability, seizures, cardiac dysfunction (increased QT, ventricular arrhythmias, increased dig effect); may also see s/s hypocalcemia or hypokalemia
3. treatment includes po or IV magnesium (caution with IV); calcium gluconate for hypocalcemic tetany or inadvertent hypermagnesemia

C. **Hypermagnesemia:**
1. causes include primarily renal failure with increased magnesium intake; also acute adrenocortical insufficiency, hypothermia, increased MgSO4 administration (for hypomagnesemia or PIH/eclampsia)
2. clinical manifestations (most from decreased peripheral/central neuromuscular transmission) include lethargy, drowsiness, N/V, decreased neuromuscular excitability (weakness, paralysis, decreased or absent reflexes, somnolence; finally respiratory and cardiac arrest
3. treatment includes stopping intake, increasing fluids with diuretics or dialysis if renal failure; IV calcium (chloride or gluconate) to oppose magnesium’s life threatening effects

VII. PHOSPHORUS (P-) (Normal 2.8-4.5 mg/dl)
A. Function: ~85% in bones & teeth, 14% soft tissue; <1% ECF; almost all phosphorus in body as phosphate (PO$_4^{3-}$), yet labs report elemental phosphorus (used interchangeably); major ICF anion; promotes energy storage and carbohydrate, protein, and fat metabolism; H+ buffer; oxygen transport; WBC and platelet function

B. **Hypophosphatemia:**
1. causes include transient shift into cells (e.g., refeeding syndrome, respiratory alkalosis, extensive burns, hypothermia, androgen treatment); increased urinary loss (e.g., diuretics, hypomagnesemia, hypokalemia, hyperparathyroidism); decreased intestinal absorption or increased intestinal loss; increased utilization: d/t tissue repair (e.g., TPN with decreased phosphorus, malnutrition recovery); also alcoholism, treated DKA, severe burns
2. clinical manifestations (most s/s secondary to decreased ATP & 2,3DPG) include CNS (confusion, coma), cardiac (chest pain, hypotension, reversible cardiomyopathy), platelet
(bleeding/bruising) and leukocyte (infection, inflammation) dysfunction; decreased oxygenation, neuromuscular excitability and GI motility; rhabdomyolysis, osteomalacia, and renal electrolyte wasting.

3. Treatment includes po and IV phosphorus products (caution with IV).

C. Hyperphosphatemia:
1. Causes include primarily renal insufficiency/failure; also increased intake, ECF shift (respiratory acidosis, untreated DKA), cell destruction (chemotherapeutic agents, increased catabolism, rhabdomyolysis) or decreased urinary loss (hypoparathyroidism, hypovolemia).
2. Clinical manifestations are usually few; majority related to hypocalcemia (increased NM excitability, etc.) or metastatic calcifications (calcium phosphate precipitates in soft tissues, joints, & arteries) and include oliguria, corneal haziness, conjunctivitis, irregular HR, dysrhythmias, conduction problems, papular eruptions.
3. Treatment includes decreasing phosphorus intake and absorption, with fluids to flush out and dialysis if needed.

ACID-BASE STATUS

I. ARTERIAL BLOOD GASES normals (mid-point)
A. pH: 7.35-7.45 (7.4): reflects H+ concentration; pH lower than 6.8 or higher than 7.8 is usually fatal.
B. pO2: 80-100 mm Hg: reflects oxygenation.
C. pCO2: 35-45 mm Hg (40): reflects respiratory side.
D. HCO3−: 22-26 mEq/L (24): reflects metabolic side.

II. MAIN REGULATORS
A. Buffering system: blood chemical buffers (bicarbonate, phosphate & protein) act to decrease effect or neutralize; fastest (within minutes).
B. Respiratory system: changes in carbon dioxide levels can counteract changes in pH rapidly (within minutes), but can only restore normal pH temporarily.
C. Renal system: Kidneys reabsorb or excrete acids or bases in the urine and produce bicarbonate if needed; takes hours to days, but is more permanent.

III. ABG INTERPRETATION
A. Check the pH: does it show acidosis (less than 7.35) or alkalosis (greater than 7.45)?
B. What is the pCO2?: is it low, normal, or high?
1. a PCO2 below 35 normally causes alkalosis
2. a PCO2 above 45 normally causes acidosis
C. What is the bicarb (HCO3-)? Is it low, normal, or high?
1. a HCO3 below 22 normally causes acidosis
2. a HCO3 above 26 normally causes alkalosis
D. Which value (the respiratory component (pCO2) or the metabolic component (HCO3 –)) corresponds with the pH of the ABG?
E. Look for compensation. Sometimes you will see a change in both the PCO2 and the HCO3. If so, the one that corresponds to the same disorder as the pH (if pH is acidotic, then whichever value is also acidotic) is the primary disturbance and the other value which should be the opposite disturbance (in this case alkalotic) indicates the body’s attempt to compensate for that disturbance. If both values are the same disturbance as that reflected by the pH, then that signifies a combined disorder, either a respiratory and metabolic acidosis or respiratory and metabolic alkalosis.

IV. ACID-BASE IMBALANCES
A. Respiratory
1. Acidosis
   a) caused by alveolar hypoventilation (e.g., COPD, sedative or barbiturate OD, severe pneumonia or atelectasis, limited chest expansion)
   b) clinical manifestations include decreased LOC, headache, seizures, hypotension, warm flushed skin, hyperkalemic arrhythmias, hypoventilation with hypoxia
   c) treatment modalities are aimed at increasing ventilation (mechanical and alveolar) and adequate oxygenation
2. Alkalosis
   a) Caused by hyperventilation (e.g., anxiety, CNS disorders, sepsis, mechanical overventilation, hypoxia)
   b) Clinical manifestations include light headedness and confusion, tachycardia, hypokalemic arrhythmias, N/V, and increased NM excitability, including numbness, tingling, seizures/tetany, hyperventilation
   c) Treatment is aimed at eliminating hyperventilation
B. Metabolic
1. Acidosis
   a) Causes include accumulation of acid (diabetic or lactic acidosis, shock) or loss of bicarbonate (severe diarrhea, renal tubular acidosis, renal failure, GI fistulas, starvation)
b) clinical manifestations include decreased LOC, headache, hypotension, warm flushed skin, hyperkalemic arrhythmias, and hyperventilation as a compensating measure
c) treatment is aimed at halting acid accumulation or bicarbonate loss; NaHCO₃ may be administered

2. **Alkalosis**
a) Caused by bicarbonate gain (excess mineralocorticoids, HCO₃⁻ ingestion) or acid loss (prolonged vomiting or NG sx, potassium deficit, diuretics)
b) Clinical manifestations include irritability, dizziness, and confusion, tachycardia, hypokalemic arrhythmias, N/V, and increased NM excitability, including numbness, tingling, seizures/tetany, and compensatory hypoventilation
c) Treatment is aimed at halting acid loss or bicarbonate accumulation

**SHOCK**

I. **General Considerations**
   A. Syndrome of decreased tissue perfusion with impaired cell metabolism leading to oxygenation and nutritional deficits
   B. Often responsible for development of systemic inflammatory response (SIRS) and multiple organ dysfunction syndrome (MODS)
   C. Treatment goals include life sustaining supportive measures (oxygen, IVs, etc.) while correcting underlying cause(s)

II. **Stages**
   A. Initial: may not be clinical manifestations; lactic acid production
   B. Compensatory: neural, hormonal and biochemical; multisystem response to decreasing tissue perfusion; may see some clinical manifestations
   C. Progressive: decreased cell perfusion and increased capillary permeability; full blown clinical symptoms; need aggressive interventions to prevent MODS
   D. Refractory: anaerobic metabolism is exacerbated by decreased perfusion from peripheral vasoconstriction and decreased cardiac output; blood pooling in capillaries causes decreased circulating volume; profound hypotension and hypoxemia; multiple organ failure and death

III. **TYPES**
   A. Low blood flow
      1. **Hypovolemic**
         a) Causes include anything that decreases circulating blood or fluid volume (massive loss, third spacing, or vasodilation)
2. **Cardiogenic**
   a) Causes include anything that causes myocardial dysfunction resulting in decreased cardiac output (e.g., acute MI and heart failure, tamponade, pulmonary embolus, cardiomyopathy)
   b) clinical manifestations include tachycardia, dysrhythmias, hypotension, tachypnea, rales/rhonchi, anxiety; cool, clammy, pale skin, cyanosis, decreased urine output
   c) specific treatment with vasoactive drugs (to optimize cardiac output)

B. **Maldistribution of blood flow**

1. **Neurogenic**
   a) Causes include spinal cord injury at T5 or above, spinal anesthesia or vasomotor depression (drugs, pain, hypoglycemia) resulting in massive vasodilation
   b) clinical manifestations include bradycardia, hypotension, pale dry skin, temperature abnormalities
   c) specific treatment with meds to reverse bradycardia or causative drug effects if symptomatic

2. **Anaphylactic**
   a) Causes include an acute hypersensitivity/allergy to variety of substances (drugs, chemicals, vaccines, food, insect venom) causing massive vasodilation and capillary permeability with third spacing
   b) clinical manifestations include hypotension, chest pain, swelling of lips, tongue, larynx, epiglottis, SOB, wheezing/stridor, flushed itching skin, urticaria, angioedema, anxiety, confusion, decreased LOC, abdominal pain, N/V, diarrhea
   c) specific treatment with IV epinephrine (po diphenhydramine can buy time til IV epi)

3. **Septic**
   a) Causes include a systemic inflammatory response to infection resulting in hypotension from vasodilation and increased capillary permeability (third spacing); leading cause of death in noncardiac ICUs
   b) clinical manifestations include tachycardia, hypotension, hypoxia, initial hyperventilation progressing to respiratory failure, crackles, decreased urine output, decreased LOC,
initial warm flushed skin progressing to cool and mottled, GI bleeding and ileus

c) specific treatment with anti-infective medications and possibly vasopressors; respiratory support

Marilyn Hickman, Instructor; revised 9/5/05 Jessie Hammond
"Emergency: Hyperkalemia: From AJN, Jan 2000

Emergency: Hyperkalemia

AJN, American Journal of Nursing
January 2000
Volume 100 Number 1
Page 55

Outline
- Abstract
- WHAT ARE THE CLUES?
- FOUR CAUSES OF HYPERKALEMIA
- INTERVENING
- FOLLOWING UP

Abstract
A life-threatening electrolyte imbalance requires immediate reversal-or ventricular fibrillation may ensue.

Joe Galanti, a 25-year-old, 220-lb. professional body builder, arrives at the ED by ambulance complaining of muscle weakness and fatigue that has lasted for three days. He can barely move his lower extremities and has mild difficulty breathing. His vital signs are BP, 100/58 mmHg; P, 84 beats per minute; T, 98.6°F; and R, 20 per minute. His skin is cool and clammy, and pulse oximetry shows 99% \(O_2\) saturation. His exam is unremarkable except for dry mucus membranes and profound muscle weakness with diminished reflexes.

As you insert a peripheral IV, Mr. Galanti tells you he's been training for an upcoming bodybuilding event. He's been taking anabolic steroids and recently started taking KCl 200 mEq/day to 'increase the size and striation of my muscles.' He's ingested only three or four energy fitness drinks-composed of vitamins and minerals, protein, and 360 calories-and two glasses of water daily for five days. Two days ago, he started taking spironolactone (Aldactone) 100 mg BID to further 'exaggerate my muscle definition.' He has lost 25 lbs. in five days.

WHAT ARE THE CLUES?
You suspect Mr. Galanti has an electrolyte imbalance; his symptoms, recent diet, and drug regimen suggest hyperkalemia. You administer normal saline at 150 cc/hr and provide 3 L/min oxygen via nasal cannula. You send blood for CBC, chemistry profile, and ABG analysis; and order a urinalysis and chest X-ray. Because hyperkalemia can cause EKG changes that may lead to ventricular fibrillation, you do a STAT EKG, which shows a widened PR interval, a widened QRS complex, and peaked T waves. You immediately initiate continuous EKG monitoring and ensure that the crash cart is nearby. His blood test results confirm the diagnosis of hyperkalemia, with a potassium level of 9.1 mEq/mL, as well as hyponatremia (124 mEq/L) and hyperchloremia (112 mEq/L)-all effects of diuretic use and dehydration. BUN is 65 mg/dL; creatinine, 8.8 mg/dL; phosphorus 6 mg/dL; and CK-MM, 13,000 µ/L. Urinalysis reveals moderate blood. The ABG values are consistent with metabolic acidosis. Elevated CK-MM, accompanied by elevated serum phosphorus levels and moderate amounts of blood in the urine, suggests rhabdomyolysis, a condition in which marked destruction of muscle tissue releases myoglobin into the circulation, compromising renal function. It may be caused by excessive muscle exercise or sports. Both rhabdomyolysis and dehydration decrease the glomerular filtration rate, raising BUN and creatinine levels and placing the patient at risk for renal failure. Metabolic acidosis further increases potassium levels.

FOUR CAUSES OF HYPERKALEMIA
Potassium, the body's principal intracellular electrolyte, is important for maintaining acid-base and fluid balance. The intracellular and extracellular potassium concentration differential is maintained by a sodium-potassium active transport system. Potassium is critical because it affects the depolarization of electrically excitable tissues, such as heart and skeletal muscle. Potassium homeostasis is regulated by the kidneys, and excess potassium is excreted in the urine. If kidney function is intact, daily intake of 400 mEq—an extremely high dose—increases serum potassium levels less than 1 mEq/mL. However, hyperkalemia (serum potassium level greater than 5.5 mEq/mL) develops when potassium regulation is disturbed. There are four main causes of hyperkalemia:

• **Excessive intake:** oral or IV potassium chloride, nutrition supplementation shakes, salt substitutes, high-dose IV penicillin with potassium salts, blood transfusions, and certain foods, such as bananas, oranges, and tomatoes.
• **Decreased excretion:** acute or chronic renal failure, potassium-sparing diuretics, hypoaldosteronism (Addison's disease), and distal tubular dysfunction.
• **Shift from intracellular to extracellular fluid:** acidemia, insulin deficiency, tissue destruction, drugs.
• **Pseudohyperkalemia:** prolonged tourniquet placement for venipuncture, hemolysis, leukocytosis, thrombocytosis.

Mr. Galanti's hyperkalemia is multifactorial. The combination of high oral doses of
potassium, the use of potassium-sparing diuretics, and poor fluid intake led to potassium retention, hypotension, dehydration, renal insufficiency, and rhabdomyolysis. The severity of symptoms frequently depends on the ratio of intracellular to extracellular potassium. Thus, patient presentations can range from asymptomatic to profoundly symptomatic and may include muscle weakness, cramps, numbness or paresthesias, nausea, diarrhea, abdominal distention, or confusion. EKG changes commonly occur at potassium levels above 6 mEq/dL, requiring emergent treatment.

INTERVENING
Treatment is directed at stabilizing the myocardial conduction system, normalizing extracellular and intracellular potassium, and preventing recurrence. In addition to continuing intense fluid rehydration, you should administer calcium gluconate 10 mL of a 10% solution IV push over two minutes. This temporarily antagonizes the cardiac and neuromuscular effects of hyperkalemia but doesn't decrease potassium levels. The onset is immediate and lasts only one hour; therefore, treatments that normalize serum potassium levels must be given concomitantly. You administer an ampule of D50 (glucose) and 10 units of regular insulin IV. You also give two ampules (100 mEq) sodium bicarbonate by slow IV push, because Mr. Galanti is acidotic. Insulin and sodium bicarbonate shift the extracellular potassium into the cells, which has an immediate effect that lasts several hours. Sodium polystyrene sulfonate (Kayexalate), a cationic exchange resin that binds potassium and increases its excretion through the gastrointestinal tract, is administered. (Dialysis may be required when kidney function is compromised and other measures have failed.)

FOLLOWING UP
Within 30 minutes, Mr. Galanti can move all extremities. He's transferred to the CCU, where he requires continuous EKG monitoring, frequent blood pressure measurements, and serum glucose and potassium testing every one to two hours. Within seven hours, his T waves return to baseline, and potassium is 5.1 mEq/dL. Twelve hours after treatment began, his creatinine is 1.6 mg/dL. During his hospital stay, his nurses explain how potassium supplementation, spironolactone use, and his diet contributed to his illness. They advise him to discontinue anabolic steroids, because longterm use can lead to liver and kidney damage. After a two-day hospital stay, he is discharged with normal laboratory parameters and will have a potassium level drawn in two days by his primary care provider.