Hypertonic Dehydration

- Second most common type of fluid volume deficit
- When water loss from the ECF is greater than electrolyte loss
- Increase of plasma osmolarity making it hypertonic or hyperosmolar
- Causing cellular dehydration and shrinkage
- The fluid shift causes increase in plasma to normal or greater than normal so….compensatory mechanisms and symptoms of hypovolemic are not present

Hypertonic Dehydration

- Changes in levels of specific electrolytes affecting excitable membrane activity.
- Common causes of hypertonic dehydration are: excessive sweating, hyperventilation, ketoacidosis, prolonged fevers, diarrhea, end-stage renal failure, and diabetes insipidus

Hypertonic Dehydration

- Least common type of fluid volume deficit
- Fluid shifts between spaces causing a decrease in plasma volume
- Excessive loss of NA+ and K+ from the ECF
- Causes decreased osmolarity of blood and interstitial fluid making them hypotonic compared with normal ECF
- Lowers osmotic pressure of ECF
- Water moves from ECF into the cells causing cell swelling
- Dilution of electrolytes causing imbalances in NA+ and K+

Health Promotion

- Mild dehydration is common and can be corrected by matching intake with output
- Advise increase fluid intake with heavy exercise especially in warm climates
- Moderate and severe dehydration is more likely to occur in people who are unable to obtain fluids without help.

Collaborative Management

- History look for risk factors causing dehydration
- Age dehydration occurs more rapidly in older adults
- Height and Weight important for calculating body fluids needs

  1L=2.2 lbs (1kg)

  changes in daily weight are the best indicators of fluid gain or loss

  A weight change of 1 pound is equivalent to fluid volume change of 500ml

Collaborative Management

- Other Changes may indicate loss or gain in fluid.
- Change in fit of rings or shoes
- Palpitations or lightheadedness, orthostatic hypotension
- Any chronic illnesses
- Drugs
- Specific questions about fluid intake and voiding patterns, GI elimination

Physical Assessment Clinical Manifestations

- Hemorrhage-life-threatening -address immediately
- Cardiovascular-increased heart rate, weak, may be difficult to find, easily blocked with light pressure
- Blood pressure-hypotension especially standing (orthostatic bp checks)
- Neck vein and hand vein distention—flat
- Respiratory-increased rate (decreased volume is perceived as hypoxia

Cont. Assessment

- Skin manifestations: Skin color, texture moisture, turgor, and edema
- Oral mucosa-lack moisture may be covered with a thick, sticky, pastelike coating, with cracks and fissures. Tongue surface with deep furrows.
- Neurological change in mental status, (confusion) body temperature (low-grade fever) fever >102 for longer than 6 hours can cause dehydration

Cont. Assessment

- Renal :

Monitor I&O

Urine Output of less than 500 ml/day for any client without renal disease is abnormal

Metabolic weight loss usually accounts for only ½ lb of weight loss /day

Pyschosocial- at first may exhibit a flat affect

As dehydration worsens behavior change to anxious, restless, lethargic, and confused especially in the older adult

Laboratory Assessment

- Lab findings are used along with clinical manifestations
- Isotonic and hypotonic dehydration with plasma volume deficits show hemoconcentration
- Specific urine lab values can help confirm dehydration if the client does not have renal disease----specific gravity >1.030, urine volume is decreased and osmolarity is greatly increased

Nursing Interventions for Deficient Fluid Volume

- Diet Therapy- mild to moderate dehydration is corrected with oral fluid replacement. Replacement fluid varies with the type of dehydration.
- Oral Fluid Rehydration Therapy-Fluid losses from diarrhea are usually 2-3L/day and should be replaced liter for liter especially in the elderly
- Drug Therapy- directed at restoring fluid balance and controlling the cause of the dehydration. Whenever possible replace fluid orally

Continue Nursing Interventions

- If dehydration is severe IV fluid replacement is necessary. Fluid replacement is based on weight loss and clinical manifestations.
- Type of IV fluid depends on the type of dehydration and the client's cardiovascular status. Usually IV infusions of water with whatever solutes (electrolytes) are needed on the basis of labs.
- Generally isotonic dehydration is treated with isotonic fluids

IV Fluid Therapy

- Hypertonic dehydration is treated with hypotonic fluids
- Hypotonic dehydration is treated with hypertonic fluids
- Most importantly monitor pulse rate and quality of urine output

Overhydration

- Fluid overload
- May be either actual excess of total body fluid or relative fluid excess in one or more body fluid spaces.
- Isotonic overhydration
- Hypotonic overhydration
- Hypertonic overhydration
- Most overload problems are related to fluid volume in the vascular space or to dilution of specific electrolytes and blood components.

Isotonic Overhydration

- Hypervolemia due to excessive fluid in the ECF. Only the ECF expands, fluid does not shift between spaces
- Problems related to Isotonic Overload are circulatory overload and edema. Can lead to Heart Failure and Pulmonary Edema

Hypotonic Overhydration

- Water intoxication the excess fluid is hupotonic to body fluid.
- Osmolarity of the ECF decreases
- Hydrostatic pressure increases
- Fluid moves into the ICF due to decreased osmotic plasma osmotic pressure, and all fluid spaces expand
- Electrolyte imbalances caused by dilution

Hypotonic Overhydration

- Rare
- Causes by an excess sodium intake
- Hyperosmolarity of the ECF draw fluid from the ICF

Interventions

- Restore normal fluid balance
- Drug Therapy-Diuretics if renal failure is not the cause
- Osmotic diuretics (mannitol) first line to prevent severe electrolyte complications, then loop diuretics
- Monitor for weight loss and increased urine output
- Observe for electrolyte imbalances Telemetry
- Diet Therapy- review sodium levels may need fluid and sodium restrictions

Electrolyte Imbalances

- Elderly and individuals with renal failure are at great risk for electrolyte imbalances.
- Those taking drugs that alter fluid and electrolytes are also at risk

Potassium Imbalances

- Hypokalemia K+ below 3.5mEq/L
- 98% of total body K+ is inside the cell minor changes in ECF and cause major changes in cell membrane excitability and in other cellular processes
- Hypokalemia can be life threatening
- Low serum potassium increase the difference in the amount of potassium between the fluid inside the cell in ICF and fluid outside the cell ECF. This causes a reduction in the excitability of cells

Cont. Hypokalemia

- As a result cell membranes of nerve and muscle tissue are less responsive to normal stimuli
- Severity of hypokalemia is related to hoe rapidly the serum potassium level dropped. Gradual decline in K+ the cells adjust and cellular K+ decreases in proportion to the ECF K+. Less of a difference.

Hypokalemia Etiology

- May result from actual total body K+ loss –K+ loss is in excess or intake does not match loss
- May result from movement of K+ from ECF to ICF causing a relative decrease in ECF K+ (caused by metabolic alkalosis and insulin use)
- Hypokalemia is common in acute care and long-term care
- Factors that lead to hypokalemia: age, drugs: diuretics, corticosteroids, beta blockers

Physical Assessment Clinical Manifestations Hypokalemia

- Musculoskeletal--- muscles become weak, hyporeflexia *severe hypokalemia can cause flaccid paralysis
- Respiratory----Skeletal muscle weakness results in shallow breathing
- Cardiovascular--- peripheral pulses are usually weak and thready. Pulse rate can range from slow to rapid
- Neurological---changes in mental status

Cont. Hypokalemia

- GI—decreases smooth muscle contractions within the GI tract leading to decreased peristalsis further causing nausea vomiting constipation
- Psychosocial----behavior changes include lethargy, mental dullness, confusion
- Labs– Serum K+ below 3.5mEq/L
- Other data obtain baseline ECG monitor on Telemetry can cause electrical conduction changes in the heart including ST-segment depression, flat or inverted T-waves, U waves
- Replacement- patient with K+ 3 needs 100-200mEq K+ of 2.0 needs 500 -600mEq

Cont. K+ Replacement

IV replacement for severe hypokalemia

1mEq/10 ml of solution. The maximum rate of infusion is 5-10mEq/hr, never to exceed 20 mEq/hr

Potassium is a severe tissue irritant infiltration can cause necrosis and skin sloughing. Stop IV infusion if infiltration occurs

Hyperkalemia

- Serum Potassium level greater than 5.0mEq/L
- Slight increases of K+ above normal can affect excitable tissue, especially the heart
- High K+ decreases the potassium difference between ICF and ECF. This difference increases the excitability– so excitable tissue respond to less intense stimuli and may discharge spontaneously
- Most seriously can affect the heart
- Hyperkalemia may result from an actual increase in total body potassium or from movement of potassium from the cell into the ECF.
- Rare in people with normal kidney function

Collaborative Management

- History---ask about chronic illness= renal disease, DM. Ask about drug use particularly potassium-sparring diuretics, ACE inhibitors.
- Food intake? Rich in Potassium
- Symptoms--- skipped heartbeats, muscle twitching, weakness in the leg muscles unusual numbness or tingling of the hands or feet

Physical Assessment Clinical Manifestations

- Cardiovascular– Most severe problems and the most common cause of death in patients with a history of hyperkalemia
- Bradycardia,hypotension, ECG changes tall, peaked T waves prolonged PR intervals (Blocks) flat or absent P waves and wide QRS complexes, ectopic beats increase, Complete heart block and asystole
- Neuromuscular—occurs in two stages Twitch of skeletal muscle in the early stages as hyperkalemia worsen twitching moves to weakness and flaccidity

Cont. Clinical Manifestations

- GI--- increased GI motility leading to diarrhea and spastic colonic activity
- Interventions: Aimed at rapidly reducing the serum potassium level

  Eliminate extra K+

  Increase K+ excretion by diuretics (lasix)

  For renal patient cation exchange by using Kayexalate

Dialysis --- rapid removal of serum K+

Hyponatremia

- Sodium level below 136mEq/L
- Problems caused by Hyponatremia involves two mechanisms:
- change in cell excitability = ECF NA+ decreases the difference between ECF and the cellular fluid also decrease, less Na+ is available to move across the excitable membrane depolarization slows
- and movement of water from ECF space into the cells = cells swell and their functions are impaired

Hyponatremia

- Can occur from a loss of total body sodium, movement of sodium from the blood to other fluid spaces, or dilution of sodium from excessive water in plasma.

Assessment and Clinical Manifestations

- Manifestations of hyponatremia are caused by its effects on excitable cellular activity
- Cerebral---The most obvious signs may be either depressed or excessive activity. Behavioral changes result from cerebral edema and increased intracranial pressure
- Neuromuscular—general muscle weakness, occurs bilaterally, worse in arms and legs
- GI—increased motility, nausea, diarrhea and abdominal cramping, peristalsis may be palpated or seen

Cont.

- Cardiovascular---has little direct effect on cardiac muscle contractility Cardiac output is changed

Interventions

- Drug and Diet Therapy---restore normal sodium level. Drug therapy varies due to whether or not fluid imbalances occur with hyponatremia.
- In chronic hyponatremia NA+ is replaced slowly
- With hypovolemia IV saline infusions to restore both fluid and NA+
- Severe hyponatremia may be treated with small volume infusions of hypertonic (2%-3%) saline

Cont.

- When hyponatremia occurs with fluid excess osmotic diuretics that promote the excretion of water are used

Hypernatremia

- Serum Na+ >145mEq/L
- High serum NA+ can be caused by or cause changes in fluid volumes.
- Mild hypernatremia ---excitable tissues are excited more easily (irritability), osmolarity of ECF increases as Na+ increases causing water to move from the cells to ECF to dilute the ECF worsening cellular dehydration and cells may no longer respond to stimuli

Assessment

- CNS—Altered cerebral function (attention span, recall of recent events and cognitive function. Manic moods or seizures. If hypernatremia occurs with fluid overload lethargy, drowsiness, stupor, and coma.
- Neuromuscular—Mild rises cause muscle twitching, as hypernatremia increases nerves less able to respond to stimuli
- Cardiovascular---increased Na+ slows the movement of calcium into the heart cells, which decreases contractility

Interventions

- Drug Therapy—hypernatremia caused by fluid loss, drug therapy is used to restore fluid balance. Hypotonic infusions usually 0.225 or 0.45 sodium chloride

  Hypernatremia caused by fluid and sodium losses require IV infusion of isotonic sodium chloride

  Hypernatremia caused from poor renal excretion of sodium require diuretics (lasix, bumex)

Diet therapy---may need dietary sodium restriction

Hypocalcemia

- Ca++ level below 9.0 mg/dl
- Ca++ stored in bone with only a small amount in ECF, so any change in calcium level has a major effect
- Ca++ is an excitable membrane stabilizer, regulating depolarization of action potential
- Calcium decreases sodium movement across excitable membranes, decreasing the rate of depolarization—low Ca++ increases NA+ movement and at inappropriate times

Hypocalcemia

- Actual calcium loss occurs when Ca++absorption from GI tract slows or when Ca++ is lost from the body

Assessment

- Dietary History – intake of calcium
- Frequent painful muscle spasms
- Endocrine problems (thyroid surgery)
- Neuromuscular---limb symptoms Paresthia, tingling and numbness ,if hypocalcemia worsens actual muscle twitching or cramps
- Trousseau and Chvostek sign's
- Cardiovascular---heart rate slower or slightly faster than normal, weak, thready pulse prolonged ST and QT (decreased depolarization)
- GI increased peristalsis painful abdominal cramps

Interventions

calcium replacement (see Chart 16-12)

Vitamin D

Diet---high calcium

Excitable membranes of the nervous system and skeletal system are over-stimulated so decrease stimulation

Use seizures precautions, keep emergency drugs nearby

Prevent injury---brittle bones, fracture easily

Hypercalcemia

- Serum calcium > 10.5 mg/dl
- Hypercalcemia means that either the amount of serum Ca++ is so great that the normal ca++ controlling mechanism can not keep pace or that one or more mechanism is not working properly
- Hypercalcemia causes excitable tissues to be less senitive to normal stimuli. Tissues affected are cardiac muscles, nerves, and intestinal smooth muscles.
- Calcium is needed by many enzymes involved in clotting
- Causes of hypercalcemia , increased absorption of CA++

Assessment

- Cardiovascular---Mild Hypercalcemia—at first cause increase heart rate and blood pressure
- Severe or prolonged hypercalcemia causes depressed electrical conduction, slowing heart rate, check ECG for shorten QT. Increase blood clot formation whenever flow is constricted or blocked
- Neuromuscular---severe muscle weakness and decreased deep tendon reflexes, altered level of consciousness ranging from confusion to lethargy
- GI—Decreased peristalsis
- Renal—increased urine output leading to dehydration---calculi

Interventions

- Drug therapy---Fluid volume replacement IV 0.9ns sodium increases the excretion of calcium
- Diuretic that enhance excretion of CA++ (lasix),

Phosphorus, calcitonin inhibit ca++ resorption from bone.

Dialysis or blood filtration

Cardiac Monitoring watch for changes in T wave and QT