

- Diabetes insipidus
 - o Lab findings:
 - Urine (think diluted) – decrease in specific gravity (normal 1.010-1.030), decrease in osmolality.
 - Serum chemistry (think concentrated) – increase in osmolality, increased sodium (normal 135-145) and increase in potassium (3.5-5.0)
 - o Pharmacologic treatment and expected findings
 - Expected finding: sunken eyes, tachycardia, hypotension, loss of skin turgor, dry mucous membranes, weak peripheral pulses, decreased cognition, polyuria, polydipsia, nocturia, fatigue and dehydration.
 - Pharm: ADH replacement agents (desmopressin)
 - Nursing considerations: monitor vitals and urinary output, use cautiously in history of CAD because it can cause vasoconstriction, monitor for headache, confusion or other indications of water intoxication.
- SIADH
 - o Manifestations:
 - Early: headache, weakness, anorexia, muscle cramps, weight gain (without edema because water is being retained not sodium)
 - As sodium decreases: personality changes, hostility, sluggish DTRs, N/V/D, oliguria with dark yellow concentrated urine.
 - Renal excretion of sodium leading to water intoxication, cellular edema and dilutional hyponatremia.
 - o Interventions
 - Order urine and blood chemistry
 - Urine will be concentrated
 - Blood will be diluted
 - Nursing: restrict oral fluids, monitor I&O, monitor VS for increased BP, tachycardia and hypothermia, auscultate lung sounds to monitor for pulmonary edema, weigh client daily, report AMS, reduce environmental stimuli and monitor urine and blood chems.
 - Meds: tetracycline derivatives (unlabeled use to correct fluid and electrolyte imbalances by stimulating urine flow, vasopressin antagonist, loop diuretics (furosemide) and hypertonic sodium IV fluid.
 - Complications that can occur: water intoxication, cerebral or pulmonary edema and severe hyponatremia
- Cushing's (caused by an oversecretion of hormones the adrenal cortex produces)
 - o Manifestations:
 - Weakness, fatigue, sleep disturbances, back and joint pain, altered emotional state (irritability, depression), decreased libido
 - Physical: buffalo hump in the neck, heavy trunk and thick extremities, decreased immune function and decreased

inflammatory response, thin, fragile skin, bruising and petechiae, tachycardia, gastric ulcers due to overexcretion of hydrochloric acid and weight gain with increased appetite.

- o Interventions
 - Monitor I&O, daily weight, assess for indication of hypervolemia, maintain a safe environment to minimize risk of pathologic fracture and skin trauma, prevent infection by performing frequent hand hygiene, encourage physical activity within client's limits, change position every 2 hours, provide meticulous skin care, monitor for and protect against skin breakdown and infections, use surgical asepsis when performing dressing changes and any invasive procedures, monitor WBC with differential daily.
 - Meds: tapering off glucocorticoids and managing symptoms, ketoconazole (antifungal that inhibits adrenal corticosteroid synthesis in high doses), mitotane (produces selective destruction of adrenocortical cells) and hydrocortisone (for replacement therapy for clients who have adrenocortical insufficiency)
 - Chemo, radiation, hypophysectomy (removal of pituitary gland) and adrenalectomy
- Addison's
 - o Causes:
 - Autoimmune or idiopathic atrophy of the adrenal glands
 - Others:
 - Surgical removal or infection of adrenal glands
 - TB and histoplasmosis are the most common infections that destroy adrenal gland tissue
 - Inadequate secretion of ACTH from the pituitary gland also results in adrenal insufficiency because of decreased stimulation of the adrenal cortex
 - Therapeutic use of corticosteroids is the most common cause of adrenocortical insufficiency
 - o Manifestations:
 - Weight loss, craving for salt, hyperpigmentation, weakness and fatigue, N/V, abdominal pain, constipation or diarrhea, dizziness with orthostatic hypotension, dehydration, hyponatremia, hyperkalemia, hypoglycemia, hypocalcemia
 - Crisis: hypotension, cyanosis, fever, nausea, vomiting, classic signs of shock, patient may be pallor, complain of headache, abdominal pain, diarrhea and may show signs of confusion and restlessness.
- Pheochromocytoma
 - o Manifestations:
 - Classic triad symptoms: pounding headache, tachycardia and profuse sweating.

- Severe episodic hypertension accompanied by severe, pounding headache, tachycardia with palpitations, profuse sweating and unexplained abdominal or chest pain.
 - Monitor blood pressure immediately if patient is experiencing an attack.
 - Rare condition caused by a tumor on the adrenal gland. Excess production of catecholamines (epinephrine, norepinephrine).
- Myxedema Coma
 - o Manifestations:
 - Respiratory failure, hypotension, hypothermia, hypoventilation, bradycardia, dysrhythmia, hyponatremia, hypoglycemia and coma
 - o Nursing interventions:
 - Maintain airway patency, initiate aspiration precautions, administer IV fluids as prescribed
- Thyroid storm
 - o Causes:
 - Rare, life threatening condition characterized by severe clinical manifestations of thyrotoxicosis.
 - Uncontrolled hyperthyroidism occurring most often with grave's disease.
 - Also acute events: infection, trauma, emotional stress, DKA and digitalis toxicity.
 - Can also occur following a surgical procedure or a thyroidectomy as a result of manipulation of the gland during surgery.
 - o Manifestations:
 - Hyperthermia/hyperpyrexia (104-106), hypertension, delirium, agitation, vomiting, abdominal pain, tachydysrhythmias, chest pain, dyspnea and palpitations.
 - Labs: Low TSH and high free T4 or T3 concentrations, degree of thyroid hormone excess typically not more profound than that seen in patients with complicated thyrotoxicosis.
 - o Pharmacologic treatment:
 - Beta-blockers
 - Thionamide
 - An iodine solution
 - An iodinated radiocontrast agent
 - glucocorticoids
 - o Nursing interventions:
 - Maintain a patent airways
 - Provide continuous cardiac monitoring for dysrhythmias
 - Administer acetaminophen to decrease temperature
 - ASA are contraindicated because they release thyroxine from protein binding sites and increase free thyroxine levels.
- Diabetes mellitus
 - o Diagnostic labs and values pertinent to each
 - HgbA1c <7, normal 5.6

- Glucose 70-100
- Hypoglycemia
 - o Manifestations:
 - Abnormally low blood glucose level
 - Less than 70, severe is considered less than 40
 - Mild: hunger, nervousness, palpitations, sweating, tachycardia, tremor
 - Moderate: confusion, double vision, drowsiness, emotional changes, headache, impaired coordination's, inability to concentrate, irrational or combative behavior, lightheadedness, numbness of the lips and tongues, slurred speech
 - Severe: difficulty arousing, disoriented behavior, loss of consciousness, seizure
 - o Interventions:
 - Check blood glucose levels
 - Follow guidelines outlined by the provider
 - Instruct client who has hypoglycemia to take 15-20g of readily absorbable carbs (4-6 oz of fruit juice or regular soft drink, glucose tablets or glucose gel per package instructions, 6-10 hard candies or 1 tbsp or honey) and recheck glycose in 15 minutes.
 - Repeat administration of carbs if not within normal limits and recheck in 15
 - If blood glucose is not within normal limits, have a snack containing carbs and proteins.
 - If client is unconscious or unable to swallow, administers glucagon subq or IM
 - In acute care, the nurse should administer 50% dextrose if IV access available.
 - Once consciousness occurs and the client is able to swallow, have the client ingest oral carbs.
 - Simple carbs to treat hypoglycemia: commercially prepared glucose tabs, 6-10 lifesavers or hard candy, 4tsp of sugar, 4 sugar cubes, 1tbsp of honey or syrup, ½ cup of fruit juice or regular soft drink, 8 oz of low fat milk, 6 saltine crackers, 3 graham crackers.
 - Do not attempt to administer oral foods or fluids to a patient experiencing severe hypoglycemia this can lead to aspiration.
- DKA
 - o Manifestations:
 - Hyperglycemia, dehydration or electrolyte loss, metabolic acidosis
 - Fruity breath
 - 3 Ps, blurred vision, weakness and headache, orthostatic hypotension, frank hypotension with a weak, rapid pulse, GI symptoms such as anorexia, N/V and abdominal pain, acetone breath, hyperventilation (Kussmaul respiration)
 - Patient may be alert, lethargic or comatose
 - o Lab findings

- Ketoacidosis reflected by low serum bicarb, low pH and low PCO₂ (Kussmauls)
- Ketone bodies in blood and urine
- Increase in creatinine, Hct and BUN
- Dehydration and electrolyte loss
- o Treatment:
 - Rehydration: initial fluid of choice is 0.9% NS, if hypernatremia use 0.45% in order to provide electrolyte free water, once BG reaches 200-300 levels change to D5W to prevent decline in blood glucose levels.
 - Restore electrolytes: major electrolyte of concern is potassium (hyperkalemia), rehydration leads to increased plasma volume and subsequent decreases in serum potassium and increased urinary excretion. Insulin administration enhances the movement of potassium from the extracellular fluid into the cells.
 - Frequent ECGs and lab measurements of potassium
- o Reverse acidosis: reversed with insulin, which inhibits fat breakdown, thereby ending ketone production and acid buildup. Insulin is usually infused IV at a slow, continuous rate
 - Hourly blood glucose must be measured
 - Regular insulin may be added to IV solutions
 - Insulin must be infused continuously until subcutaneous administration of insulin can be resumed.
 - Bicarb infusion to correct severe acidosis is avoided in treatment of DKA because it precipitates further, sudden decrease in serum potassium.
- o Insulin Administration:
- HHS (>600)
 - o Treatment:
 - Rehydration, insulin administration, monitor fluid volume and electrolyte status
- DKA vs HHS:
 - o DKA: patients affected can occur in patients with type 1 and 2 diabetes but more common in type 1, occurs because of the omission of insulin, physiologic stress (infection, surgery, CVA and MI), onset is rapid, usually >250, pH is >7.3, serum and urine ketones present, serum osmolality 300-350, bicarb >15, BUN and creatinine elevated, mortality rate is 1-5%.
 - o HHS: can occur in both types of diabetes but mainly in type 2. Occurs by physiologic stress, occurs slower, usually >600, normal pH level, absent serum and urine ketones, >350 osmolality, normal bicarb, elevated BUN and creatinine and mortality is 10-20%
- Pancreatitis
 - o Manifestations:
 - Severe, constant, knife-like pain (LUQ, mid-epigastric and radiating to back)
 - N/V, weight loss

- Turner sign, cullens sign, generalized jaundice, absent or decreased bowel sounds, warm, moist skin and fruity breath
 - o Lab findings:
 - Serum lipase increases slowly but remains elevated for up to two weeks, WBC increased due to infection or inflammation, serum liver enzymes and bilirubin increased with associated biliary dysfunction.
 - o Pain-relief interventions:
 - Opioid analgesics, histamine receptor antagonists, PPI, pancreatic enzymes, antibiotics
 - NPO, TPN is severe, NG tube if vomiting or ileus, no alcohol, smoking, limit stress, pain management
 - Positioning for comfort: fetal side-lying, HOB elevated, sitting up or leaning forward.
- EGD
 - o Client education pre-procedure:
 - NPO midnight before procedure
 - o Nursing interventions:
 - NPO until gag reflex returns
 - Informed consent
 - Be cognizant of over-sedation, hemorrhage, aspiration, perforation of GI tract
- GI bleeds
 - o Priority assessment:
 - Identify s/sx of shock (hypotension, tachycardia, tachycardia, decreased pulse pressure, cool, clammy skin, decreased LOC, decreased UO and slow cap refill)
 - Monitor urine output
 - Administer isotonic fluids (LR and NS)
 - First line management: endoscopy, performed within 24 hours of bleeding
- AKI
 - o Causes:
 - Sudden cessation of renal function that occurs when blood flow to the kidneys is significantly compromised.
 - o Lab findings:
 - Serum CR gradually increases 1-2 mg/dl every 24-48 hours, 1-6 mg/dl in 1 week or less.
 - BUN can increase 80-100 within a week
 - Urine specific gravity varies in postrenal type, can be elevated up to 1.030 in prerenal type or diluted as low as 1.000 in intrarenal.
 - Sodium decreased (in prerenal azotemia) or increased (intrarenal azotemia), hyperkalemia, hyperphosphatemia, hypocalcemia
 - Hct decreased
 - UA has presence of sediment (RBS, casts)
 - Metabolic acidosis
- CKD

- o Dietary restrictions with examples of each:
 - Drink at least 2L of water daily, stop smoking, limit alcohol, control protein intake based on clients stage of CKD, restrict na, k, ph and mg, provide a diet high in carbs and moderate in fat
- o Lab findings:
 - Hematuria, proteinuria, decrease in specific gravity, gradual increase over months to years of serum creatinine, gradual increase in BUN with elevated serum CR over months to years, decreased na, increased k, ph and mg, decreased hgb and hct
- o Nursing interventions:
 - Report and monitor irregular findings, assess and monitor vascular access or peritoneal dialysis insertion site, obtain a detailed med and herb history, control protein intake, restrict dietary na, k, ph and mg, provide diet high in carbs and moderate fat, restrict intake of fluids, adhere to meticulous cleaning of areas on skin not intact and access sites to control infections, balance clients activities and rest, prepare the client for hemodialysis, peritoneal dialysis and hemofiltration, provide skin care, provide emotional support, encourage the client to ask questions, administer meds.
- Peritoneal dialysis
 - o What is it, how does the system work:
 - rids body of excess fluid and electrolytes, achieves acid-base balance, eliminates waste products and restores internal homeostasis by osmosis, diffusion and ultrafiltration
 - Involves instillation of hypertonic dialysate solution into the peritoneal cavity and subsequent dwell times, drain the dialysate solution that includes the waste products, the peritoneum serves as the filtration membrane.
 - Client should have an intact peritoneal membrane without adhesions from infections
 - Treatment of choice for older adults, treats clients requiring dialysis who are unable to tolerate anticoagulants, have trouble with vascular access, have chronic infections or are unstable, have chronic diseases such as DM, HF or HTN.
- ESRD
 - o Manifestations:
 - N/V, loss of appetite, fatigue and weakness, sleep problems, changes in urination, decreased mental sharpness, swelling of feet and ankles, persistent itching, chest pain if fluid fills up enough to affect lining of heart, shortness of breath and high blood pressure that is hard to control
- Kidney biopsy
 - o Nursing interventions:
 - Clients receive sedation and ongoing monitoring
 - Pre-procedure: obtain informed consent, obtain urine specimen, review coagulation studies, NPO for 4-6 hours

- Post-procedure: monitor vitals following sedation, assess dressing and urinary output, review hgb and hct values and administer prn pain medications
- Kidney transplants
 - o Contraindications:
 - Organ rejection, ischemia, renal artery stenosis, thrombosis and infection
 - o Organ rejection manifestations:
 - Hyperacute: occurs within 48 hours after surgery
 - Acute: occurs 1 week to 2 years after surgery
 - Chronic: occurs gradually over months to years
- Dialysis types:
 - o Hemodialysis: shunts blood from the body through a dialyzer and back into circulation, requires vascular access
 - o Peritoneal dialysis: instillation of hypertonic dialysate solution into the peritoneal cavity and subsequent dwell times, drain the dialysate solution that includes the waste products; the peritoneum serves as the filtration membrane
 - o CRRT: provides a means to remove uremic toxins and fluids while acid-base status and electrolytes are adjusted slowly and continuously in a hemodynamically unstable patient
 - CVVH
 - SCUF
 - CVVHD
 - CVVHDF