

	What is it? Who gets it?	What causes it?	Clinical manifestations?	Treatment? Assessments?
<b>Mild hypothyroidism</b>	Low levels of thyroid hormone  people who live in areas where there is not seafood	primary = pituitary failure, hypothalamic failure, post thyroidectomy effects, post radiation effects, aging.  secondary = failure to stimulate normal thyroid function, or inability to synthesize thyroid hormone d/t iodine deficiency or use of anti-thyroid meds.	fatigue, sensitivity to cold, dry skin and hair, forgetfulness, depression, weight gain despite reduced appetite, ↑ cholesterol	Synthroid
<b>Myxedema</b>	More severe form of hypothyroidism  Women > 60 years People with Hashimoto's Thyroiditis	Triggered by stress in hypothyroid pts who are undiagnosed or undertreated. Stressors are infection, drugs (phenothiazines, barbituates, opioids, anesthetics), respiratory failure, heart failure, cerebral vascular accident, trauma, prolonged exposure to cold, metabolic disturbances, surgery, seizures.	non-pitting facial and pretibial edema, ↑ cholesterol, CAD, blank expression, dilutional hyponatremia, ↑ CPK, AST, LDH, alopecia, mental dullness, constipation, abdominal distention, voice hoarse and husky, tongue thick, enlarged heart, bradycardia, ↓ cardiac output	Synthroid
<b>Myxedema Coma</b>	Serious complication of myxedema  non compliant pt, stressed pt	Hypothyroidism exacerbated by stress, or chronic noncompliance	hypoventilation, respiratory failure, hypothermia, hypotension, hyponatremia, hypocalcemia, hypoglycemia, coma	Mortality rate is close to 100%, so you want to catch it before it gets severe.

	What is it? Who gets it?	What causes it?	Clinical manifestations?	Treatment? Assessments?
<b>Hyperthyroid / Graves Disease</b>	Elevated levels of thyroid hormone  T3 and T4 ↑ TSH ↓	Cancer, overtreatment of hypothyroid state, Grave's Disease	main = elevated TH, goiter, exophthalmos  also, irritability, anxiety, visual disturbances, tremors, weakness, heat intolerance, chest pain, SOB, cough, edema to extremities, gradual or sudden weight loss despite ↑ appetite.  Critical = temp above 100.4 (no infection) + any change in LOC	Thyroidectomy  s/p thyroidectomy monitor for respiratory obstruction, hemorrhage, hypocalcemia, tetany, injury to recurrent laryngeal nerve, thyroid storm.  nursing care = decrease stress on suture line by supporting head and turning manually, semi-fowler's, pillows and sandbags to support head/neck, keep ampules of calcium gluconate on hand, tracheostomy set and ETT at bedside, ask pt to speak q 30-60 mins and note voice quality. Monitor VS, I/O, rectal temp.
<b>Thyroid Storm</b>	Complication of hyperthyroidism.  Also known as thyrotoxicosis.	A surge of thyroid hormone r/ t infection, thyroid ablation, metabolic catastrophes, surgery, trauma, labor and delivery, myocardial infarction.	high fever without source of infection, irrational and paranoid behavior, severe tachycardia, delirium, dehydration.	Beta-blockers to block tachycardia; propylthiouracil and methimazole to block TH synthesis; cortisone for blocking conversion of T4 to T3; AVOID ASPIRIN, cooling measures, total or subtotal thyroidectomy, monitor ECG, monitor fluids and electrolytes, VS and body temp (use foley with temp sensor), quiet environment, assess LOC and cardiopulmonary status

	What is it? Who gets it?	What causes it?	Clinical manifestations?	Treatment? Assessments?
<b>Addison's</b>	<p>Adrenal insufficiency</p> <p>↓ cortisol ↓ aldosterone</p> <p>Pt taking steroids for &gt; 3 weeks with abrupt cessation, pt with Hx of endocrine disorders, TB pt, adrenalectomy pt.</p>	<p>primary = problem with adrenal gland</p> <p>secondary = dysfunction of hypothalamic-pituitary axis (tumor, radiation)</p> <p>Most common cause = chronic treatment with steroid medication (prednisone)</p> <p>Ex: autoimmune, TB, metastatic cancer, corticosteroid meds, HIV, stressful events, bilateral adrenalectomy</p>	<p>Gradual onset.</p> <p>fatigue, weight loss, weakness, ↓ GI enzymes → vomiting, cramps and diarrhea, hypoglycemia, kidneys lose sodium and water and retain potassium → dehydration and cardiac dysrhythmias → decreased cardiac output.</p>	<p>Medication</p>
<b>Addisonian Crisis</b>	<p>Pt with Addisons who is undergoing stress</p>	<p>pregnancy, surgery, infection, dehydration, anorexia, fever, emotional upheaval</p>	<p>back pain radiating to legs, abdominal pain with N/V, depressed mentation, signs of volume depletion, hypotension, decreased LOC, signs of shock, hypokalemia, hyponatremia, elevated BUN, hypoglycemia</p> <p>imaging shows altered size of adrenal gland, calcification of the gland or adrenal hemorrhage.</p>	<p>Rapid IV bolus of 100 mg hydrocortisone Followed by hydrocortisone diluted in NS Aggressive fluid replacement (up to 5 L) and glucose Vasopressors (epi, norepi/Levophed) IV dextrose</p> <p>Nursing management: Monitor VS and O2 sats, respiratory status and lung sounds (possible fluid overload), ECG monitoring (look for tall tented T waves and widening QRS), I/O, daily weights, glucose levels, electrolyte monitoring (look for hyponatremia and hyperkalemia), maintain quiet environment.</p>

	What is it? Who gets it?	What causes it?	Clinical manifestations?	Treatment? Assessments?
<b>Cushing's Syndrome</b>	<p>Excessive adrenocortical activity</p> <p>↑ glucocorticoids aka hypercortisolism</p> <p>Lab Values = hyperglycemia, hypernatremia, hypokalemia</p> <p>slightly ↑ WBC ↓ eosinophils ↓ lymphocytes</p> <p>no diurnal pattern of cortisol (elevated all the time)</p>	<p>Two etiology categories:</p> <p>1) dependent on ACTH...result of a tumor in pituitary or hypothalamus (30% of cases)</p> <p>2) independent of ACTH, may be iatrogenic (too much steroid medication)...most common form of Cushings</p>	<p>persistent hyperglycemia (steroid diabetes), protein tissue wasting (muscle weakness and capillary fragility), pink/purple striae, thinning of skin, ↑ bruising, bone wasting (osteoporosis), loss of height, kyphosis, abnormal fat distribution (moon shape face, buffalo hump, truncal obesity)</p> <p>↑ susceptibility to infection, less resistance to stress, few and subtle signs of infection, poor wound healing.</p> <p>K depletion → dysrhythmias, muscle weakness, renal impairment</p> <p>electrolyte imbalances → water retention, edema, HTN</p> <p>Chronic HTN → left ventricular hypertrophy and CHF</p> <p>Virilism, acne, thinning scalp hair, hirsutism, dull memory, ↓ ability to concentrate, steroid psychosis (alternating euphoria and depression)</p> <p><b>MOST COMMON</b> = central obesity, HTN, emotional lability with depression, acne, amenorrhea, diabetes, easy bruising, moon face, buffalo hump, hypokalemia and metabolic acidosis, generalized weakness, hyperglycemia, hypokalemia, hypernatremia, impaired wound healing, fragile skin, sleep disturbances, osteoporosis, kyphosis, back pain, masculine traits in women (breast atrophy, hirsutism, amenorrhea), impotence in men</p>	<p>Assess and monitor VS, weight changes, serum electrolytes, I/O, blood glucose. Watch for signs of infection, skin breakdown.</p> <p>Fall prevention is very important b/c of osteoporosis and osteopenia.</p> <p>Encourage diet high in protein and calcium, low in calories, fat and sodium.</p> <p>Tx = adrenalectomy → lifelong glucocorticoid and mineralcorticoid.</p> <p>S/P surgery = patient teaching on use of meds, skin care, importance to follow up with health care provider, safety measures r/t weakness, osteopenia, and osteoporosis.</p>

	What is it? Who gets it?	What causes it?	Clinical manifestations?	Treatment? Assessments?
<b>Type 1 DM</b>	<p>Insulin dependent diabetes</p> <p>&lt; 30 years of age</p>	<p>autoimmune destruction of the islet cells of pancreas.</p> <p>R/F = viral infection</p>	<p>polyuria, polyphagia, polydipsia, weight loss, blood vision, headache, muscle cramps, delayed wound healing, recurrent yeast infections, fatigue</p>	<p>Very rapid insulin (Humalog, Novolog) / onset 15 m / peak 1 hour / duration 3-4 hr</p> <p>Rapid acting insulin (Humlin, Novolin) / onset 30-60 m / peak 1-5 hr / duration 6-8</p> <p>Intermediate insulin (Lente, NPH) / onset 1-3 hr / peak 4-15 hr / duration 8-24 hr</p> <p>Long acting insulin (Ultra Lente) / onset 4-6 hr / peak 8-20 hr / duration 24-48 hr</p> <p>Long acting insulin (Glargine) / onset 1 hr / no peak / duration 24-48 hr</p> <p>Long acting insulin (Lantus) / onset 4-6 hr / no peak / duration 24-48 hr</p> <p><b>Oral hypoglycemics:</b>  Sulfonylureas (stimulates beta cells to secrete insulin)  Biguanides (increases tissue response to insulin);  Alpha glucosidase inhibitors (delays digestion and absorption of complex CHO and simple sugars)  Thiazolidinidiones (increase insulin action at the receptor sites)</p>
<b>Diabetic Ketoacidosis</b>	<p>Acute complication of Type 1 DM</p> <p>Age &lt; 40  BS 250-600  Na = low/normal  pH = low  Bicarb = low  Serum Osm = &lt; 320</p>	<p>Increased production and release of glucose, or decrease of the ability of the cells to use glucose.</p> <p>Can be caused by infection, illness, surgery, stress, noncompliance, insufficient insulin</p>	<p>S/S &lt; 2 days; Polyphagia, ketones in urine, urine acetone, severe metabolic acidosis, glucose 200-800, polyuria, polydipsia, hyperkalemia</p> <p>Lethargy, confusion, Kussmaul's respirations, muscle wasting, weight loss, abd cramping, N/V, fruity odor to breath, tall tented T waves, widened QRS.</p>	<p>Rehydration and dilution of glucose.  Insulin IV or SubQ  Monitor K levels  BS = 250, add dextrose IV  Reduce BS gradually</p> <p>Monitor LOC, BS hourly, respiratory status, ECG, I/O, electrolyte balance</p>

	What is it? Who gets it?	What causes it?	Clinical manifestations?	Treatment? Assessments?
<b>HHNK</b>	<p>Increase in plasma osmolarity &gt; 310</p> <p>BS &gt; 600 mg/dL</p> <p>No Ketoacidosis</p> <p>Age &gt; 40</p>	<p>Occurs in several conditions: type II DM, acute pancreatitis, severe infection (most common), acute MI, TPN.</p> <p>Can be precipitated by ↑ resistance to insulin and an excessive CHO intake.</p>	<p>Occurs gradually over several days to 2 weeks (more than 5 days).</p> <p>-Signs of SEVERE dehydration (polyuria, thirst, tachycardia)</p> <p>-BG really really high! &gt; 600</p> <p>-Neurologic signs (seizures, hemiparesis, aphasia, hyperthermia, visual hallucinations)</p> <p>-Potassium = normal/high</p> <p>-Bicarb = high</p> <p>-Ketones = absent</p> <p>-Fruity breath = absent</p> <p>-pH = normal</p>	<p>Treatment is difficult. Water moves back into brain cells during tx, leading to a risk for cerebral edema. Potassium losses can occur during the diuretic phase and need correction. The prognosis is not as good as for DKA.</p> <p>Little or no insulin is needed to correct the problem. The main focus is on replacing the volume deficit...can be depleted by as much as 12 liters. Replace HALF the fluid shortage in 24 hours starting out with NS. As VS recover, the fluid is changed to 1/2 NS with K to prevent cerebral edema.</p> <p>Assess pt frequently and monitor for signs of fluid overload.</p>

Black, Joyce M., and Jane Hokanson Hawks. *Medical-Surgical Nursing: Clinical Management for Positive Outcomes - Single Volume (Medical Surgical Nursing- 1 Vol (Black/Luckmann))*. St. Louis: Saunders, 2009. Print.