ENDOCRINE SYSTEM

Include the pituitary, thyroid, parathyroid glands, adrenal glands, pancreatic islets, ovaries & testes.

Endocrine glands: secrete hormones directly into the bloodstream
Exocrine glands: secrete onto epithethial surfaces or into GI tract. (eg. Sweat glands)

Negative feedback – mechanism for regulating hormone concentration in the bloodstream. (When the hormone concentration increases, further production is inhibited. When the hormone concentration decreases, the rate of production will increase.)

HORMONES

4 categories:

1. Amines & amino acids (epinephrine, norepinephrine, thyroid hormones)
2. Peptides, polypeptides, proteins & glycoproteins (TRH, FSH, GH)
    a. Interact with receptor sites on the cell surface and/or change membrane permeability.
3. Steroids (corticosteroids)
    a. Penetrate cell membranes & interact with intracellular receptors.
4. Fatty acid derivatives (retinoids)

<u>Assessment</u>
S/S of endocrine imbalances: changes in energy level, tolerance to heat & cold, weight, fat & fluid distribution, secondary sexual characteristic, sexual dysfunction, memory, concentration, sleep patterns, and mood.

<u>Diagnostic</u>
Blood tests – used to determine hormone blood levels.
Radioimmunoassays – radioisotope labeled antigen tests used to measure the levels of hormones or other substances.
Urine tests – used to measure the amount of hormones or other end products of hormones secreted by the kidneys.
Stimulation tests – determine how an endocrine gland responds to the administration of stimulating hormones produced/released by the hypothalamus or pituitary gland.
Suppression tests – used to determine whether negative feedback mechanisms are intact.
Imaging – includes MRI, CT, PET, DEXA
Genetic screening – used to determine the presence of a gene mutation that may predispose an individual to a certain condition.

ENDOCRINE GLANDS AND HORMONES Function and Dysfunction

Abnormalities of function are either ↑secretion or ↓secretion of any of the hormones. All abnormalities are pretty much fatal if left untreated.

Hypothalamus Controls the release of pituitary hormones. Makes ADH & Oxytocin - stored in Pituitary	**Releasing and inhibiting hormones** CRH Corticotropin-releasing hormone TRH Thyrotropin-releasing hormone GHRH Growth hormone–releasing hormone GnRH Gonadotropin–releasing hormone		
Pituitary (hypophysis)	Abnormalities of the anterior and posterior portions of the gland may occur independently	Hypofunction (hypopituitarism) is more common. Can result from disease of the pituitary g itself or disease of the hypothalamus. Results are pretty much the same. **Result**: thyroid, gonads, and adreanal no longer stim'd resulting extreme weight loss, ema atrophy of all endocrine glands & organs, hair loss, impotence, amenorrhea, hypometabol and hypoglycemia. Coma and death if no HRT.	
Anterior Pituitary Hormones stim of secretion is via the pituitary portal circulation	**Function**	**↑secretion** is rare and usually from 2 types of pituitary tumors ("functional" eosinophillic and basophilic tumors), pit tumors can cause visual disturbances & HA	**↓secretion**- radiation tx head & neck area, destruction by trauma, tumor ("non-functio chromophobic tumors make up 90% of pit t vascular lesion, encephalitis, autoimmunity, Postpartum pituitary necrosis-Sheehan's syndrome, occurs w/ hemorrhage, hypovole and hypotension during delivery.
GH Growth hormone (somatotropin)	Stimulates growth of bone and muscle, promotes protein synthesis and fat metabolism, decreases carbohydrate metabolism	**↑GH → acromegaly:** Adults: bone and soft tissue deformities and enlargement of the viscera without an increase in height Children: gigantism 7-8 ft tall **↑GH –stress, exercise, low BG**	**↓ GH** in children: ↓ growth & dwarfism Mild-moderate obesity Reduced cardiac output Fatigue & low BP
ACTH Adrenocorticotropic hormone	Stimulates synthesis and secretion of adrenal cortical hormones	**↑ACTH→ Cushing's syndrome -** hyperstim of adrenoglucocorticoids (cortisol)	**↓ACTH** Fatigue & low BP
TSH Thyroid-stimulating hormone	Stimulates synthesis and secretion of thyroid hormone	**↑TSH** Hyperthyroidism–S&S similar to graves disease	**↓TSH** Mild-moderate obesity Fatigue & low BP Cretinism in children Myexedema in adults
FSH Follicle-stimulating hormone	Female: stimulates growth of ovarian follicle, ovulation Male: stimulates sperm production	Effects unknown	**↓FSH** Failure of sexual maturation Infertility Sexual dysfunction

LH Luteinizing hormone	F: stims dev of corpus luteum, release of oocyte, production of estrogen & progesterone M: stims secretion of testosterone, dev of interstitial tissue of testes	Effects unknown	**↓ LH** Failure of sexual maturation Infertility Sexual dysfunction
Prolactin	Prepares breast for breast-feeding	**↑ prolactin**-inappropriate milk production	**↓ prolactin** milk production
Posterior pituitary	storage		
ADH Antidiuretic hormone (vasopressin) Stim via ↑osmolality or ↓BP or Hypovolemia	Increases water reabsorption by kidney	**↑ADH** – trauma, stroke, Cancer (lung and panc), meds, stress SIADH	**↓ADH**- stroke, trauma, or idiopathic. most common disorder r/t posterior lobe dysfunc Diabetes Insipidus: ↑↑↑thirst & dilute uri Reduced cardiac output, Fatigue & low BP
Oxytocin stim via pregnancy & delivery	Stimulates contraction (↑ force) of pregnant uterus, milk ejection from breasts after childbirth	Effects unknown	Effects unknown
Adrenal	Regulates Na+ and electrolyte balance Affects PRO, CHO, FAT metabolism Influences dev of sex characteristics Sustains fight or flight response		
Adrenal cortex	adrenocortical secretions make it possible for the body to adapt to stress of all kinds. Without the adrenal cortex, severe stress would peripheral circulatory failure, circulatory shock, and prostration.		
Mineralocorticosteroids, aldosterone Stim'd by RAAS and a little by ACTH, also stim'd hyperkalemia. Aldosterone is the primary hormone for the long-term regulation of sodium balance.	↑Na+ absorption & K+ and H+ loss by kidney	**↑ aldosterone** **SIADH** **Primary Aldosteronism:** occurs in some patients w/ functional tumors of adrenal gland, causes a distinctive pattern of biochemical changes and a corresponding S&S.	**↓ aldosterone** **Diabetes Insipidus**
Glucocorticoids, cortisol Admined often to inhibit inflammatory response to tissue injury and suppress allergic manifestations. Side effects include DM, osteoporosis, peptic ulcer, increased protein breakdown resulting in muscle wasting and poor wound healing, and redistribution of body fat. Lg amts of admined glucocorticoids in the blood inhibit release of ACTH and endogenous glucocorticoids. This can cause adrenal cortex to atrophy. If admin is d/c suddenly, adrenal insufficiency results because of the inability of the atrophied cortex to respond adequately.	Affect metab of PRO, CHO, FAT regulates blood glucose levels, affects growth, anti-inflammatory action, decreases effects of stress	**Cushing's syndrome- ↑adrenocortical** activity: common cause is use of corticosteroid medications. Uncommon cause is excessive corticosteroid production 2° to hyperplasia. But, overproduction of endogenous corticosteroids may be caused by several mechanisms, including tumor in pit gland that produces ACTH and stims adrenal cortex to increase its hormone secretion despite adequate amounts. Primary hyperplasia of adrenal glands in the absence of a pit tumor is less common.	**Addison's disease, or adrenocortical insuffi** adrenal cortex function is inadequate. Autoimmune or idiopathic atrophy of the ac glands is cause of most cases. Other causes: removal of both adrenal glands and infectio (Tuberculosis and histoplasmosis) of the adr glands. Although autoimmune destruction replaced tuberculosis as the principal cause Addison's disease, tuberculosis should be considered in dx workup because of its incre incidence. Inadequate secretion of ACTH frc pituitary gland also results in adrenal insuffi

		Another less common cause of Cushing's syndrome is the ectopic production of ACTH by malignancies; lung carcinoma is most common type. Regardless of cause, normal feedback mechanisms that control the function of the adrenal cortex become ineffective, and the usual diurnal pattern of cortisol is lost. S&S of Cushing's syndrome are primarily a result of oversecretion of glucocorticoids and androgens (sex hormones), although mineralocorticoid secretion also may be affected	Tx use of corticosteroids is most common ca adrenocortical insufficiency. Symptoms of adrenocortical insufficiency may also result the sudden cessation of exogenous adrenoc hormonal therapy, which suppresses the bo normal response to stress and interferes wi normal feedback mechanisms. Treatment w daily administration of corticosteroids for 2 weeks may suppress function of the adrena therefore, adrenal insufficiency should be considered in any patient who has been tre with corticosteroids.
Adrenal androgens, Dehydroepiandrosterone (DHEA) & androstenedione	Have minimal intrinsic androgenic activity; they are converted to testosterone and dihydrotestosterone in the periphery		
Adrenal medulla	regulate metabolic pathways to promote catabolism of stored fuels to meet caloric needs. Major effects of epinephrine release are to to meet a challenge (fight-or-flight response). Secretion of epinephrine causes decreased blood flow to tissues that are not needed in emergency situations, such as the GI tract, and increased blood flow to tissues that are important for effective fight or flight, such as ca and skeletal muscle. Catecholamines also induce the release of free fatty acids, increase the basal metabolic rate, and ↑ BG.		
Catecholamines: **Epinephrine** **Norepinephrine** Stim'd by SNS	Serve as neurotransmitters for the sympathetic nervous system	**↑Epi ↑ Norepi** **Pheochromocytoma** –typically benign tumor of adrenal medulla, also can be in chest,bladder, abd, and brain. Strong genetic link. Peak incidence 40-50 yo. S&S: Hyperglycemia, HTN, severe HA, Palpitations, flushing, diaphoresis, chest and abd pain w/ N & V, heat intolerance, wt loss.	**↓ sx removal of adrenal gland** Can result in CV collapse, hypotension, and
Thyroid - Various hormones and chemicals are responsible for normal thyroid function. Key among them are thyroid hormone, calcitonin, & iodine. Has 5 xs the blood supply of the liver	Controls rate of metabolism & growth	**Hyperthyroid** 2nd most common endocrine disorder, after DM. **Graves' disease** (most common type) from excessive output of thyroid hormones from abnormal stimulation of thyroid by immunoglobulins. Affects women 8Xs more than men, onset bt second and fourth decades. May appear after emotional shock, stress, or infection. Other causes of hyperthyroidism are thyroiditis & excessive ingestion of TH.	**Hypothyroid – Causes:** Autoimmune disease (Hashimoto's thyroiditis {mostly women 30- post-Graves' disease) Atrophy of thyroid gland with aging Therapy for hyperthyroidism Radioactive iodine Thyroidectomy Meds:Lithium, Iodine compound, Antithyroi Radiation to head and neck cancers, lymphc Infiltrative diseases of the thyroid (amyloidc scleroderma, lymphoma) Iodine deficiency and iodine excess

Thyroid Hormone is **triiodothyronine** (T3) **AND thyroxine** (T4) Protein bound amino acids that need iodine for synthesis	Controls cellular metabolic activity ↑the metabolic rate; ↑protein and bone turnover; ↑responsiveness to catecholamines (epi & norepi); necessary for fetal & infant G&D They are important in brain dev and influence every major organ system.	**↑T3 & T4** Increased metabolic rate Opposite of hyposecretion **Thyroid Storm –uncontrollable hypersecretion:** fever, tachycardia, systolic HTN , N V & D, agitation, tremors, anxiety, confusion, seizures, coma. Causes: thyroid surgery, extreme stress and infection.	**↓T3 & T4** (thyrotoxicosis) ↓metab rate-common cause: grave's disea Lethargy/fatigue, myalgia, easily cold, wt g skin & thick, coarse hair, ↓HR, constipatior **myexedema**, forgetfulness, menstrual distu infertility, cardiomegaly and CHF (reduced sensitivity to catacholemines), possible goit when ↓ iodine), poor growth & brain dev ir
Thyroid C cells **Calcitonin (keeps Ca+ in bone)**	Lowers blood Ca++ and P		
Parathyroid glands	Controls Ca+ and P metabolism		
Parathormone PTH-pulls Ca+ from bone Stim'd by Ca++ levels	Regulates serum calcium	**↑ PTH -** Hyper Ca+ & hypo P, fatigue & muscle weakness, bone pain, fractures, anorexia, N & V & epigastric pain, constipation, dysrrythymias, HTN, renal stones	**↓ PTH –** sx removal. Hypo Ca+ and Hyper P facial paresthesias, cramps, positive Trousse Chvostek's sign, tetany including bronchosp dysphagia-dysrrythymias, Hypotension, dep irritability
Pancreatic islet cells (Islets of Langherhorn's)	Influences CHO metabolism, indirectly affects PRO and FAT metabolism		
Insulin	Lowers blood glucose by facilitating glucose transport across cell membranes of muscle, liver, and adipose tissue		**↓ Insulin** DM Type I Type II LADA or Type 1.5 In Blood: Hyperglycemia, Lipidemia, Ketoaci In urine: Glycosuria, Ketonuria, Na+ & K+ lo
Glucagon Stim'd by low BG	Increases blood glucose concentration by stimulation of glycogenolysis and glyconeogenesis		
Somatostatin	Delays intestinal absorption of glucose Suppresses insulin and glucagon release		
Kidney			
1,25-Dihydroxyvitamin D	Stimulates calcium absorption from the intestine		
Renin Stim'd by low BP, or hypovolemia, or SNS	Activates RAAS, returns BP to normal (from low)		
Erythropoietin (EPO) Stim'd by hypoxia	Increases red blood cell production		

Ovaries			
Estrogen	Affects development of female sex organs and secondary sex characteristics		
Progesterone	Influences menstrual cycle; stimulates growth of uterine wall; maintains pregnancy		
Testes			
Androgens, mainly testosterone	Affect development of male sex organs and secondary sex characteristics; aid in sperm production		