Kallmann's syndrome	absent GnRH, anosmia, absence of taste
Impotence	failure to sustain an erection; psychogenic in most cases (erections present at night)
Erection	parasympathetic response
Ejaculation	sympathetic response
Leydig cell failure	↑ LH; ↓ testosterone, sperm count; normal FSH
Seminiferous tubule failure	↑ FSH (↓inhibin); ↓ sperm count; normal LH and testosterone

Leydig and seminiferous tubule failure	↑ FSH and LH; ↓ testosterone and sperm count
Y chromosome	determines genetic sex
Testosterone	develops seminal vesicles, epididymis, vas deferens
Dihydrotestosterone (DHT)	develops prostate and male external genitalia
Male pseudohermaphrodite	genetic male; phenotypically female
Testicular feminization	XR; deficient androgen receptors; MCC male pseudohermaphrodite
Klinefelter’s syndrome	XXY; 1 Barr body; female secondary sex characteristics

Herpes genitalis	recurrent painful vesicles; multinucleated squamous cells with intranuclear inclusions
Human papilloma virus	condyloma acuminata; koilocytosis (wrinkled nuclei surrounded by a halo)
Chlamydia trachomatis	metaplastic squamous cells with vacuoles containing elementary bodies
S/S	non-specific urethritis, cervicitis, PID, ophthalmia neonatorum
Neisseria gonorhoeae	urethritis, cervicitis, PID; ophthalmia neonatorum, gram negative diplococcus
Ophthalmia neonatorum first week	N gonorrhoeae
Ophthalmia neonatorum second week	C. trachomatis

Lymphogranuloma venereum	C. trachomatis subtype
S/S	scrotal/vulva lymphedema; granulomatous microabscesses; rectal strictures in females
Chancroid	painful ulcer, adenopathy, Hemophilus ducreyi
Granuloma inguinale	Calymmatobacterium granulomatis; raised ulceration but no lymphadenopathy
Treponema pallidum	spirochete; produces vasculitis of arterioles (plasma cell infiltrate)
Primary syphilis	painless chancre
Secondary syphilis	rash on palms/soles; condyloma lata; generalized adenopathy

Tertiary syphilis	neurosyphilis (e.g., tabes dorsalis), aortic arch aneurysm, gummas
RPR/VDRL	reagin antibodies against cardiolipin; ↓ titer with Rx of syphilis
RPR/VDRL	false positive with anticardiolipin antibodies (common in SLE)
FTA-ABS	confirmatory test for syphilis; not distinguish active from treated disease
FTA-ABS	remains positive after Rx
Trichomonas vaginalis	flagellate protozoan; cervicitis/vaginitis; Rx metronidazole both partners
Gardnerella vaginalis	vaginal pH >5; bacterial vaginosis; clue cells; Rx metronidazole

Candida vaginitis	white, curd-like discharge; DM, antibiotics, pregnancy; Rx fluconazole
Vulvar squamous cancer	MC vulvar cancer; HPV association
Vulvar leukoplakia	biopsy to R/O squamous dysplasia/cancer
Lichen sclerosis vulva	epidermal atrophy; slight risk for squamous cancer
Squamous hyperplasia vulva	leukoplakia; no cancer risk
Paget’s disease	intraepithelial adenocarcinoma (mucin production) of vulva
Malignant melanoma	vulva location; similar to Paget cells but not mucin positive

Gartner’s duct cyst	lateral wall vagina; persistent mesonephric duct
Embryonal rhabdomyosarcoma	bloody, grape-like vaginal mass young girl
Vaginal adenosis	maternal exposure to DES; precursor clear cell adenocarcinoma vagina
Vaginal squamous cancer	usually extension of cervical cancer
Rokitansky-Kiister-Hauser	absence of vagina and uterus
Nabothian cysts	endocervical glands covered by metaplastic squamous epithelium
Pathologic cervicitis	trichomonas, HSV-2, C. trachomatis (follicular cervicitis)

Cervical Pap	superficial squamous (estrogen), intermediate (progesterone), parabasal (no hormone)
Normal	70% superficial, 30% intermediate
Atrophic	100% parabasal cells
Hyperestrinism	100% superficial cells
Pregnancy	100% intermediate cells
Endocervical cells	sign of adequately performed Pap smear
Cervical polyp	bleeding after intercourse; non-neoplastic

Cervical dysplasia	begins in transformation zone; associated with low and high risk HPV
Risk factors cervical dysplasia/cancer	early onset sexual activity; multiple partners; smoking; OC
CIN	cervical intraepithelial dysplasia; mild, moderate, severe (in-situ)
Cervical cancer	↓ incidence (Pap smear); 45-yr-old; COD renal failure from obstruction of ureters
S/S	cervical discharge; bleeding after intercourse
Sequence to menarche	breast budding, growth spurt, pubic hair, axillary hair, menarche
Proliferative phase cycle	estrogen-dependent; ↑estrogen inhibits FSH and stimulates LH

Ovulation	day 14-I6; LH surge; subnuclear vacuoles; ↑body temperature
Secretory phase cycle	progesterone-dependent
Menses	drop in estrogen/progesterone stimulates apoptosis; plasmin prevents clotting
FSH	stimulates follicle and aromatase synthesis in granulosa cells
LH	stimulates androgen synthesis in proliferative phase and progesterone synthesis in secretory phase
Day 21	day of implantation of fertilized egg
Pregnancy	↑plasma volume > RBC mass; ↑GFR; ↑thyroxine/cortisol (increased binding proteins)

hCG	LH analogue produced by syncytiotrophoblast
hCG	stimulates corpus luteum of pregnancy to synthesize progesterone for 8-10 weeks
Estrone	estrogen of postmenopausal woman; aromatization of adrenal androstenedione
Estradiol	estrogen of non-pregnant woman in reproductive life; aromatization of testosterone
Estriol	estrogen of pregnancy
Menopause	↑ FSH (best screen; due to ↓estrogen), ↑LH
S/S	secondary amenorrhea, hot flushes

Hirsutism	↑ hair in normal areas
Virilization	hirsutism + male secondary sex characteristics (clitoromegaly)
Test for hirsutism/virilization	↑ testosterone - ovarian source; ↑DHEA-sulfate - adrenal source
Polycystic ovarian syndrome (POS)	↑ LH; ↓ FSH; ↑ estrogen and androgens
S/S	hirsutism, oligomenorrhea, infertility; enlarged ovaries with subcortical cysts; LH:FSH >2:1
Menorrhagia	excess menstrual flow; MCC iron deficiency in women
Dysmenorrhea	painful menses; 1° PGF2α, 2° endometriosis

DUB	bleeding related to hormone rather than anatomic causes
Anovulatory DUB	menarche and perimenopause; estrogen excess without progesterone
Ovulatory DUB	irregular shedding, inadequate luteal phase
Primary amenorrhea	no menses by 16 years old
Secondary amenorrhea	no menses for 3 months
Amenorrhea-hypothalamic/pituitary dysfunction	↓ FSH/LH; e.g., hypopituitarism
Amenorrhea-ovarian dysfunction	↑FSH/LH; e.g., Turner's syndrome

Amenorrhea-end-organ disease	normal FSH/LH; e.g., imperforate hymen
Asherman syndrome	surgical removal of stratum basalis
Primary amenorrhea-normal secondary sex characteristics	constitutional delay MCC
Primary amenorrhea-lack secondary sex characteristics	Turner’s
Turner’s syndrome	XO; no Barr bodies; XO/XY types have gonadoblastomas; streak gonads (no eggs)
S/S	newborn with lymphedema hands/feet; cystic hygroma in neck (web); short stature; 1° amenorrhea
Secondary amenorrhea	pregnancy MCC; prolactinoma; anorexia nervosa; pituitary adenoma

Asherman syndrome	removal of stratum basalis causing scarring; secondary amenorrhea
Endometritis	group B streptococcus; intrauterine device (Actinomyces); chronic - plasma cells
Endometrial polyp	menorrhagia; not a precursor for endometrial cancer
Adenomyosis	functioning endometrial glands and stroma in myometrium; enlarged uterus
Endometriosis	functioning glands and stroma outside uterus; reverse menses; ovary MC site
S/S	dysmenorrhea, painful stooling, bowel obstruction; “powder burn” appearance
Endometrial hyperplasia	unopposed estrogen; simple/complex types; precursor endometrial cancer

Causes	obesity, estrogen Rx, polycystic ovarian syndrome
Endometrial cancer	obesity, nulliparity, estrogen Rx, early menarche/late menopause; OC protective
S/S	bleeding in postmenopausal woman
Leiomyoma uterus	menorrhagia, obstructive delivery; not a precursor for leiomyosarcoma
Leiomyosarcoma	MC sarcoma
Ectopic pregnancy	PID MC risk factor; intraperitoneal hemorrhage; screen with ß-hCG
Follicular cyst	MC ovarian mass in young woman

Risk factors ovarian tumors	nulliparity and genetic factors; OC protective
Serous ovarian tumors	surface-derived; ↑ bilaterality; psammoma bodies in malignant type
Mucinous ovarian tumors	surface-derived; pseudomyxoma peritonei in malignant type
Endometrioid carcinoma	resembles endometrial cancer; association with endometriosis
Cystic teratoma	MC benign germ cell tumor (<1% malignant); hair/teeth; calcifications
Dysgerminoma	MC malignant germ cell tumor; associated with streak gonads of Turners
Yolk sac tumor	MC germ cell tumor young girl; ↑AFP; Schiller-Duval bodies

Meigs syndrome	ovarian fibroma, ascites, right-sided pleural effusion
Granulosa tumor	low grade malignant; hyperestrinism, Call Exner bodies
Thecoma	benign; yellow color; hyperestrinism
Leydig cell and Sertoli cell tumors	hyperandrogenism
Gonadoblastoma	XY phenotype of Turner’s
Krukenberg tumors	metastatic stomach cancer; signet ring cells
Single umbilical artery	↑ incidence congenital defects

Syncytiotrophoblast	lining of villi; produces hCG and human placental lactogen
Human placental lactogen	responsible for mild glucose intolerance in pregnancy
Abruptio placenta	retroplacental clot; painful bleeding; hypertension, cocaine, smoking
Placenta previa	placenta implanted over cervical os; painless bleeding
Placenta accreta	direct implantation into myometrium without intervening decidua; hysterectomy
Twin placenta	monochorionic always identical twins; dichorionic may be identical or fraternal
Siamese twins	monoamniotic monochorionic twin placenta

Enlarged placenta	DM, Rh HDN, syphilis
Complete mole	benign neoplasm of chorionic villi; dilated villi; no embryo; 46 XX (both male)
S/S	preeclampsia in first trimester; ↑ incidence choriocarcinoma
Partial mole	embryo present; 68 XXY; no transformation into choriocarcinoma
Choriocarcinoma	malignancy of trophoblastic tissue (syncytiotrophoblast, cytotrophoblast)
Risk factors	complete mole (MC), spontaneous abortion, normal pregnancy
S/S	↑hCG; lung metastasis; good prognosis

Chorioamnionitis	group B streptococcus (S. agalactiae) infection
Preeclampsia	abnormal placentation causing placental ischemia; ↑ in vasoconstrictors (ATII)
S/S	hypertension, proteinuria, pitting edema; begins in third trimester
Spontaneous abortion	50% have karyotype deformity (trisomy 16)
Amniotic fluid	fetal urine
Polyhydramnios	TE fistula, duodenal atresia, open neural tube defects
Oligohydramnios	infantile polycystic disease

↑ Serum AFP	open neural tube defect
↓ Serum AFP	Down syndrome
Urine estriol	fetal adrenal, placental, maternal liver involved in its production
Down syndrome triad	↑ ß-hCG, ↓ serum AFP, ↓ urine estriol
Fibrocystic change	MC breast mass <50-yrs-old; atypical hyperplasia cancer risk; lumpy, painful breasts
Sclerosing adenosis	component of FCC; involves terminal lobules often has microcalcifications
Fibroadenoma	benign stromal tumor; MC movable mass in women <35-yrs-old

Intraductal papilloma	benign tumor lactiferous duct/sinus; MCC bloody nipple discharge <50-yr-old
Invasive ductal cancer	MCC breast mass in woman >50-yrs-old
Breast cancer risk	unopposed estrogen; family history first-degree relatives
Breast cancer	painless mass upper outer quadrant in woman >50-yrs-old
Mammography	screening test to detect non-palpable masses
Palpable breast mass	order fine needle aspiration (not a mammogram)
Ductal carcinoma in situ	necrotic centers (comedo); microcalcifications common

Paget’s disease of breast	invasive ductal cancer into nipple; Paget’s cells similar to vulvar Paget’s
Medullary carcinoma	bulky tumor with large cells and lymphoid infiltrate; more common in Pt with BRCA 1 mutation
Inflammatory carcinoma	orange peel appearance; lymphatics blocked by tumor (lymphedema)
Lobular cancer	MC cancer of terminal lobule; ↑ bilaterality
Phyllodes tumor	low grade malignant tumor of stroma
ER-PR positive tumors	tumors responding to hormones; candidate for tamoxifen (anti-estrogen)
ERB-B2 oncogene positive breast cancer	aggressive breast cancer

Gynecomastia	estrogen stimulation of male breast
Gynecomastia	normal in newborn, puberty (no surgery), old age; (micronodular) cirrhosis MC pathologic cause
Overactive endocrine syndrome	most often adenomas; use suppression tests (most do not suppress)
Tumors that suppress	prolactinoma (bromocriptine), pituitary Cushings (high dose dexamethasone)
Underactive endocrine syndrome	autoimmune destruction MCC; stimulation tests
Hypopituitarism adults	non-functioning adenoma MCC, Sheehan’s postpartum necrosis (stop lactation)
Hypopituitarism in children	craniopharyngioma (Rathke’s pouch remnant) MCC; visual field defects

S/S ↓ FSH and LH	amenorrhea, ↓ testosterone in male
Growth hormone functions	muscle growth, gluconeogenesis; release of insulin growth factor (IGF)
IGF	synthesized in liver; bone and cartilage growth
S/S ↓ GH/IGF in children	growth retardation; ↓ height and weight
Sleep and arginine infusion	stimulation tests for GH and IGF
S/S ↓ GH/IGF in adults	hypoglycemia
S/S ↓ TSH	secondary hypothyroidism; ↓ T4, ↓ TSH; muscle weakness, dry skin

S/S ↓ ACTH	secondary hypocortisolism; ↓ cortisol, ↓ ACTH; fatigue; hypoglycemia
Metyrapone	stimulation test for ACTH reserve
Metyrapone	blocks adrenal 11-hydroxylase → ↑ ACTH and 11-deoxycortisol (proximal to block)
Metyrapone test ↓ ACTH and 11-deoxycortisol	pituitary/hypothalamic dysfunction
Metyrapone test ↑ ACTH and 11-deoxycortisol	Addison's disease
Diabetes insipidus	loss ADH (central), refractory to ADH (nephrogenic); always diluting urine
Central diabetes insipidus (CDI)	↓ UOsm and ↑ POsm with water deprivation; vasopressin causes ↑ UOsm > 50%

Causes CDI	pituitary stalk transection, hypothalamic lesion (site for ADH synthesis)
Nephrogenic diabetes insipidus (NDI)	↓ UOsm and ↑ POsm with water deprivation; vasopressin causes ↑ UOsm < 50%
Causes NDI	lithium, demeclocycline, nephrocalcinosis, severe hypokalemia
Gigantism	GH secreting pituitary adenoma before epiphyses have fused
Acromegaly	GH secreting pituitary adenoma after epiphyses have fused
S/S acromegaly	cardiomyopathy; large hands, feet, jaw; hyperglycemia
Prolactin	inhibited by dopamine

Prolactinoma	MC pituitary tumor; secondary amenorrhea and galactorrhea; prolactin inhibits GnRH
Rx	surgery or bromocriptine (dopamine analog)
Other causes hyperprolactinemia	primary hypothyroidism, drugs
Inappropriate ADH syndrome	hyponatremia <120 mEq/L; ↑ UOsm (always concentrating urine)
Causes	small cell carcinoma lung, CNS injury, chlorpropamide
Rx	restrict water; demeclocycline in small cell carcinoma
Serum T4	↑ or ↓ in free hormone or thyroid binding globulin (TBG)

↑ Serum T4 and normal TSH	↑ TBG; due to ↑ in estrogen
↑ Serum T4 and ↓ TSH	thyrotoxicosis
↓ Serum T4 and normal TSH	↓ TBG; due to anabolic steroids
↓ Serum T4 and ↑ TSH	primary hypothyroidism
↓ Serum T4 and ↓ TSH	secondary hypothyroidism
TSH	negative feedback with T4 and T3; best screening test
I131 uptake	↑ in Graves; ↓ in thyroiditis, patient taking excess thyroid, hypothyroidism

Cold nodule	non-functioning nodule; no uptake I131
Hot nodule	functioning nodule; ↑ uptake I131
Thyroglossal duct cyst	midline cystic mass
Branchial cleft cyst	cyst in anterolateral neck
Acute/subacute thyroiditis	painful thyroid; early thyrotoxicosis; ↓ I131 uptake
Hashimoto’s thyroiditis	MCC hypothyroidism; HLA Dr3/Dr5; inhibitory IgG TSH receptor antibody
Hashimoto’s thyroiditis	↑ anti microsomal and thyroglobulin antibodies

S/S	muscle weakness, periorbital puffiness, ↓ reflexes, diastolic hypertension, constipation, dry skin
Lab	↓ T4, ↑ TSH
Cretinism	maternal hypothyroidism before fetal thyroid developed, genetic disorder
S/S	mental retardation; short stature and increased weight; coarse skin
Thyrotoxicosis	any cause ↑ thyroid hormone activity; Graves disease, excess hormone, thyroiditis
Hyperthyroidism	↑ synthesis thyroid hormone; Graves disease and toxic nodular goiter
Graves disease	autoantibody against TSH receptor (type II reaction); HLA Dr3

S/S unique to Graves	exophthalmos, pretibial myxedema
S/S thyrotoxicosis	tachycardia/atrial fibrillation, systolic hypertension, diarrhea, brisk reflexes
Lab thyrotoxicosis	↑ T4, ↓ TSH, ↑ glucose, ↑ calcium
I131 uptake	↑ Graves, toxic nodular goiter; ↓ thyroiditis, excess hormone, hypothyroidism
Rx Graves disease	ß-blocker; drug to decrease hormone synthesis (propylthiouracil)
Toxic nodular goiter	hyperthyroidism; develops out of a multinodular goiter; no exophthalmos
Goiter	enlarged thyroid; iodine deficiency MCC; relative thyroid hormone deficiency

S/S	rapid enlargement due to hemorrhage into cyst; Rx thyroxine
Solitary thyroid nodule woman	most often benign (cyst)
Solitary thyroid nodule man or child	often malignant
Papillary carcinoma thyroid	MC thyroid cancer; radiation exposure; psammoma bodies
Follicular carcinoma thyroid	invades blood vessels
Medullary carcinoma thyroid	parafollicular cells; calcitonin; amyloid (calcitonin conversion)
MEN I syndrome	3 P's; pituitary tumor, parathyroid adenoma, pancreatic tumor (ZE or ß-islet cell tumor)

MEN IIa syndrome	2 P's; medullary carcinoma thyroid, pheochromocytoma, parathyroid adenoma
MEN IIb syndrome	1 P; medullary carcinoma thyroid, pheochromocytoma, mucosal neuromas
Alkalotic pH	tetany with normal total calcium, ↓ ionized calcium and ↑ PTH
Hypoalbuminemia	↓ total calcium, normal ionized calcium and PTH
Tetany	↓ ionized calcium level; threshold potential comes closer to resting potential
S/S	thumb adducts into palm, twitching after tapping of facial nerve
PTH	maintains ionized Ca2+; ↑ Ca2+ renal reabsorption; ↓ phosphate/bicarbonate reabsorption in kidneys

Primary HPTH	↑ Ca2+, hypophosphatemia, ↑ PTH
Cause	adenoma MCC, hyperplasia, cancer
S/S	renal stone, peptic ulcers, pancreatitis, hypertension, metastatic calcification
Secondary HPTH	↓ Ca2+, ↑ PTH; hypovitaminosis D from renal failure MCC
Malignancy-induced hypercalcemia	↑ Ca2+, ↓ PTH; all other non-parathyroid causes same results
Causes hypercalcemia	osteolytic lesions, sarcoidosis, ↑ vitamin D, PTH-related peptide, myeloma
Tertiary HPTH	hypercalcemia developing from secondary HPTH

Primary hypoparathyroidism	↓ Ca2+ and ↓ PTH
Causes	previous thyroid surgery, autoimmune, DiGeorge syndrome
S/S	tetany; calcification basal ganglia
Pseudohypoparathyroidism	↓ Ca2+ with normal to ↑ PTH; end-organ resistance to PTH
Other causes ↓ Ca2+	hypomagnesemia (↓ PTH), ↓ vitamin D, DiGeorge
↓ Ca2+ and ↓ PTH	primary hypoparathyroidism
↓ Ca2+ and ↑ PTH	secondary hyperparathyroidism

↑ Ca2+ and ↑ PTH	primary hyperparathyroidism
↑ Ca2+ and ↓ PTH	malignancy induced hypercalcemia; other causes hypercalcemia
Waterhouse-Friderichsen syndrome	meningococcemia with bilateral adrenal hemorrhage due to DIC
Addison’s disease	autoimmune destruction adrenal cortex MCC, adrenogenital syndrome, metastasis
S/S	hypotension (salt loss), hyperpigmentation (ACTH), hypoglycemia
Lab	↓ sodium, ↓ cortisol, ↑ potassium, ↑ ACTH
Adrenogenital syndrome	AR; enzyme deficiency; hypocortisolism; hyperpigmentation from ↑ ACTH

21-Hydroxylase deficiency	↑ 17 KS, ↓ 17 OH, lose salt, hypotension; female pseudohermaphrodite
11-Hydroxylase deficiency	↑ 17 KS, ↑ 17 OH, retain salt, hypertension; female pseudohermaphrodite
17-Hydroxylase deficiency	↓ 17 KS, ↓ 17 OH, retain salt, hypertension; male pseudohermaphrodite
MCC Cushings	long-term corticosteroid therapy
Tests Cushings syndrome	low/high dose dexamethasone suppression; urine free cortisol (best test)
Normal dexamethasone suppression	cortisol analogue; ↓ ACTH and ↑ cortisol
Pituitary Cushings	MCC Cushing's; ACTH secreting pituitary tumor

Lab	low dose dexamethasone not suppress cortisol; high dose suppresses
Adrenal Cushings	adrenal adenoma secreting cortisol; suppressed ACTH
Lab	no suppression with low/high dose dexamethasone
Ectopic Cushings	ACTH secreting small cell carcinoma of lung; high ACTH and cortisol levels
Lab	no suppression with low/high dose dexamethasone
S/S Cushings	purple stria, truncal obesity, hypertension, DM
Primary aldosteronism	benign adenoma in zona glomerulosa

S/S	hypertension and muscle weakness (hypokalemia), no pitting edema
Lab	hypernatremia, hypokalemia, metabolic alkalosis, ↑ urine K+ and Na+
Pheochromocytoma	benign tumor in adrenal medulla in adults
Associations	von Hippel Lindau, neurofibromatosis, MEN IIa and IIb
S/S	labile hypertension, anxiety, sweating, headache
Lab	↑ 24 hr urine for VMA and metanephrines
Neuroblastoma	malignant tumor adrenal medulla child; widespread metastasis; hypertension

ß-islet cell tumor (insulinoma)	benign tumor; hypoglycemia, ↑ insulin and C-peptide
Patient taking excess insulin	hypoglycemia, ↑ insulin, ↓ C-peptide
Glucagonoma	malignant α-islet cell tumor; hyperglycemia and rash
Zollinger Ellison syndrome	malignant islet cell tumor secreting gastrin; peptic ulcers
Somatostatinoma	malignant δ islet cell tumor; DM, malabsorption, cholelithiasis, achlorhydria
VIPoma	malignant islet cell tumor; diarrhea, hypokalemia, achlorhydria
DM	organ damage correlates with glycemic control

Type 1	young, thin person; no insulin; HLA DR3/4; insulitis; islet cell antibodies; ketoacidosis
Type 2	older person; obese; relative insulin deficiency (↓ insulin receptors, postreceptor problems)
Type 2	family history; fibrotic islet cells with amyloid; hyperosmolar nonketotic coma
↑ Non-enzymatic glycosylation	glucose attaches to amino acids in basement membranes
Non-enzymatic glycosylation	↑ vessel permeability producing hyaline arteriolosclerosis
Osmotic damage	glucose converted into sorbitol by aldose reductase
Osmotic damage	lens (cataracts), Schwann cell (neuropathy), pericytes retinal vessels (microaneurysms)

Pathogenesis hyperglycemia	↑ gluconeogenesis (most important), glycogenolysis
Pathogenesis hyperlipidemia	no insulin to stimulate capillary lipoprotein lipase; ↑ chylomicrons/VLDL
Pathogenesis ketoacidosis	↑ oxidation fatty acids with excess acetyl CoA; liver synthesis ketone bodies
Most commons due to DM	neuropathy, blindness, CRF, hyperglycemia, non-traumatic amputation
Glycosylated HbA1c	measure of long term glycemic control (8-12 weeks)
Gestational DM	↑ placental size, human placental lactogen
Complications	macrosomia (↑ muscle/fat from insulin), RDS, newborn hypoglycemia (↑ insulin)

Hypoglycemia	insulin/oral hypoglycemics MCC, liver disease; carnitine deficiency
Carnitine deficiency	no ß-oxidation of fatty acids; all cells compete for glucose
What two hyperfunctioning endocrine tumors are suppressible?	Prolactinoma - bromocriptine. Cushing syndrome - dexamthasone.
Explain the difference between primary, secondary, and tertiary hypothyroidism.	Primary - issue with the thyroid (e.g. Hashimotos). Secondary - issue with the pituitary secreting TSH. Tertiary - issue with the hypothalamus (e.g. Sarcoidosis).
Name the most common causes of hypopituitarism.	Pituitary diseases: (1) null pituitary adenoma, Sheehan's postpartum necrosis, Craniopharyngioma, pituitary apoplexy, lymphoctyic hypophysitis. Hypothalmic disorders.
What is the MEN I syndrome?	Pituitary adenoma, hyperparathyroidism, pancreatic tumor (either Zollinger or insulinoma).
What bone is the sella turcica located next to?	Clinoid process.

Whats the pathogenesis of Sheehan's postpartum necrosis?	Hypovolemic shock causing an infarction after pregnancy. Loss of prolactin cessating lactation.
What is the most common cause of hypopituitarism in children? Where does the lesion originate? What visual defect might this child have?	Craniopharyngioma: tumor derived from Rathke's pouch remnants. Due to invasion of the optic chiasm the child may develop a bitemporal hemianopsia.
What are the pathological effects of losing GnRH (hypopituitarism) on children, women, and men?	Children - delayed puberty. Women - secondary amenorrhea. Male - impotence.
How are children and adults each effected by the loss of GH?	Children: growth delay, delayed fusion of the epiphyses, pituitary dwarfism. Adults: hypoglycemia (due to decrease gluconeogenesis).
Whats the best stimulation test if you suspect GH insufficiency?	Arginine and sleep stimulation test: no increase in GH or IGF-1. GH and IGF-1 are normally secreted at 5 am.
What two essential amino acids are required for the stimulation of growth hormone?	Arginine and histidine.
Hypopituitarism can cause ACTH deficiency resulting in secondary hypocortisolism. What is the primary effect of lacking cortisol?	Fasting hypoglycemia (decreased gluconeogenesis). Fatigue.

What are the most common causes of central diabetes insipidus?	Lack of ADH: hypothalamic disease, transection of the pituitary stalk (e.g. car accident trauma), posterior pituitary disease (e.g. metastasis).
What is the difference between CDI and NDI? What are the most common causes of nephrogenic diabetes insipidus?	CDI - lack of ADH. NDI - collecting tubules are nonresponsive to ADH. Causes of NDI: drugs (lithium, demeclocycline), hypokalemia, nephrocalcinosis.
What are the clinical findings of CDI and NDI (hint: the same)? What is a differential diagnosis for these symptoms?	Symptoms: polyuria, polydipsia. Other dx: drinking too much water, osmotic diuresis due to diabetes mellitus or use of diuretics.
What is the test for CDI and NDI?	Water deprivation test. Normal: increased plasma Osm (ADH is released) and increased urine Osm. DI: increased POsm and decreased UOsm. To differentiate between CDI and NDI - injection of ADH; in CDI UOsm increases by more than 50%.
Adult presents to your office with a prominent jaw, large lips, and complains all of his hats dont fit him any more. Diagnosis? What organ are you worried about?	Acromegaly: increased GH and IGF-1. IGF-1 stimulates lateral bone, cartilage, and soft tissue growth in adults. GH and IGF-1 are not suppressed by glucose. Patients will also have hyperglycemia. You must worry about cardiomegaly (cause of death).

What are the symptoms of a prolactinoma and how do you treat?	Women - galactorrhea and secondary amenorrhea (prolactin inhibits GnRH release). Men - Impotence. Treat with a dopamine analogues (cabergoline).
What enzyme converts iodides to iodine in the thyroid? What enzyme converts T4 to T3 in the periphery?	Peroxidase. Deiodinase.
What is the carrying protein of thyroxine (T4) and triiodothyronine (T3)? What increases/decreases this protein? What happens to free T4 when this protein increases or decreases?	Thyroid-binding protein. Increased: estrogen (pill, pregnancy, hormone replacement). Decreased: anabolic steroids, nephrotic syndrome. FT4 remains the same, therefore, so does TSH!!!
In a radioactive iodine uptake test, when would the iodide uptake increase and when would it decrease?	Increase (indicates T4 production): Grave's disease, toxic nodular goiter. Decrease: inactivity of gland (taking thyroid hormone) or inflammation of gland (acute, subacute, chronic thyroiditis).
Patient presents with a fever, painful cervical adenopathy and the follow labs: increased serum T4, decreased serum TSH, and decreased I(131) uptake. Diagnosis?	Acute thyroiditis. Bacterial infection (e.g. Staphylococcus aureus). The initial thyrotoxicosis (increased serum T4) is due to gland destruction.
Patient presents with a painful thyroid, but no cervical adenopathy. Biopsy shows granulomatous inflammation with multinucleated giant cells. Diagnosis?	Viral infection of the thyroid leading to subacute granulomatous thyroiditis (e.g. coxsackievirus).

What is the pathogenesis of Hashimoto's thyroiditis?	Autoimmune destruction of parenchyma by cytotoxic T cells. Macroscopically: enlarged gray gland due to lymphocytic infiltrate with prominent germinal follicles. Most common cause of hypothyroidism. Antimicrosomal and antithyroglobulin antibodies are present.
Whats the defining feature of Reidel's thyroiditis?	Fibrous tissue replacement of the gland making it hard.
What are the clinical features of cretinism?	Cretinism is hypothyroidism in infancy or early childhood. Brain requires thyroxine for its maturation. Causes: maternal hyothyroidism, enzyme or iodine deficiency. Clinical features: mental retardation, increased weight and short stature. Unless its pituitary dwarfism (decreased weight and short stature).
What are some clinical findings in adult hypothyroidism?	Proximal muscle myopathy, weight gain (decreased BMR), dry/brittle hair, yellow skin, periorbital puffiness, hoarse voice, myxedema, fatigue, cold intolerance, constipation, diastolic HTN (retention of Na and water), confestive cardiomyopathy, atherosclerotic artery disease (due to hypercholesterolemia), mental slowness, dementia.

What are some clinical findings in adult hyperthyroidism?	Increased BMR, weight loss, fine tremor, heat intolerance, diarrhea, anxiety, lid stare, sinus tachy, atrial fibrillation, systolic HTN, brisk reflexes, osteoporosis.
What are some lab findings in adult hyperthyroidism?	Increased serum T4, decreased serum TSH, increased or decreaesed I(131) uptake, hyperglycemia, hypocholesterolemia, hypercalcemia.
Whats the pathogenesis of Grave's disease and the pathogenesis of ophthalmopathy?	Thyroid stimulating anti-TSH receptor antibody (IgG). Type II hypersensitivity. Exophthalmos - proptosis and muscle weakness of the eye due to adipose and GAG deposited in orbital tissue.
How does Graves' disease in the elderly (apathetic hyperthyroidism) present?	Cardiac abnormalities: atrial fibrillation, congestive heart failure. Muscle weakness, apathy. Thyromegaly.
What is Plummer's disease?	One or more nodules in a multinodular goiter become TSH-independent. This causes hyperthyroidism, but lacks exophthalmos and pretibial myxedema.
What is the mechanism for most of the symptoms observed in hyperthyroidism?	Upregulation of beta adrenergic receptors.

What is the most common cause of a goiter? What are some complications?	Endemic type: due to iodide deficiency. Treat with levothyroxine (reduces gland size). Complications: hemorrhage into cyst (sudden, painful, gland enlargement), primary hypothyroidism, toxic nodular goiter, hoarseness, dyspnea.
Negative feedback in endocrine disease what happens to calcium? PTH (parathyroid hormone)?	↑ calcium, ↓ PTH (parathyroid hormone) ↓ calcium, ↑ PTH (parathyroid hormone)
What is the most common cause of autoimmune disease?	Endoctrine gland hypofunction
Endocrine gland hypofunction what test?	use stimulation tests
Endocrine gland hyperfuncion use which test?	suppression tests
Which exceptions in hyperfunction disorders can be suppressed?	pituitary Cushing syndrome and prolactioma can be suppressed

what is the most common cause of endocrine gland hyperfunction?	benign adenoma
Hypothalamic dysfunction clinical findings?	secondary hypopituitarism, CDI, ↑ prolactin; precocious puberty, visual field defects, mass effects (hydrocephalus)
Precocious puberty: when is it true? pseudo?	true if CNS origin pseudo if peripheral cause
Pineal gland clinical anatomy?	midline above quadrigemianal plate
Melatonin = ?	chemical messenger of darkness
Pineal gland common disorders?	commonly undergoes dystrophic calcification
Pineal gland disorders what is the majority type?	majority are germ cell tumors

Pineal gland tumors clinical findings?	paralysis of upward gaze ("setting sun" sign)
Pituitary infarction invariably produces _____?	Pituitary infarction invariably produces panhypopituitarism
what is the most common cause of hypopituitarism in adults?	nonfunctioning adenoma
MEN(multiple endocrine neoplasia) I syndrome findings?	pituitary adenoma, hyperparathyroidism, pancreatic tumor
Hypopituitarism in children most common cause?	craniopharyngioma
Rathke's pouch develops _____?	Rathke's pouch develops anterior pituitary?
Sheehan's postpartum necrosis = ?	sudden cessation of lactation; pituitary infarction secondary to shock

Pituitary apoplexy most common cause?	hemorrhage into preexisting adenoma
Lymphocytic hypophysitis autoimmune destruction occurs when?	during or after pregnancy
Empty sella syndrome details of primary type?	subarachnoid space extends into sella; ↑ CSF (cerebrospinal fluid) pressure compresses gland
empty sella syndrome what happens with hypertension?	obese with hypertension
Posterior pituitary = ?	storage of ADH and release of oxytocin
what is the most common type of pituitary tumor?	Prolactinoma
Prolactinoma in women clinical and lab findings ?	secondary amenorrhea, galactorrhea

Prolactinoma in men clinical and lab findings ?	impotence due to loss of libido; headache
Rx of Prolactinoma?	dopamine analogues surgery
GH (growth hormone) adenoma functions?	gluconeogenesis; ↑ amino acid uptake in muscle; stimulate IGFF-1 in liver
IGF-1 functions?	stimulates bone, cartilage, soft tissue growth
Gigantism lab findings?	↑ linear/lateral bone growth in children; epiphyses NOT fused
Acromegaly (in adults) lab findings?	↑ lateral bone growth only (epiphyses NOT fused), organomegaly, hyperglycemia
Acromegaly diagnostic tool?	Comparing old versus new photograph is valuable diagnostic tool

Acromegaly what is a common cause of death?	heart failure from cardiomyopathy
Thyroid hormone = ?	iodide attached to tyrosine
TSH (thyroid -stimulating hormone) functions?	mediates trapping, organification and proteolysis
Free T(4) = ?	prohormone; rendered metabolically active by outer ring deiodnase (FT(3))
how does FT(4)/ FT(3) relate to TSH?	a negative feedback in relation to TSH
Total serum T4 bound to?	T4 is bound to TBG + FT4
Estrogen effect on TBG?	↑ TBG which ↑ total serum T4 but NOT FT4

↓TBG effect on T4?	↓ total serum T4 but Not FT4
Alterations in TBG effect on T4?	alter total serum T4; no effect on FT4 and TSH
what is the best screening test for thyroid dysfunction?	Serum TSH
{131}I radioactive uptake evaluates ?	evaluates synthetic activity of thyroid gland
↑ {131}I radioactive uptake effects?	increased synthesis of thyroid hormone; Graves' disease
↓ {131}I radioactive uptake effects?	thyroiditis; patient taking excess thyroid hormone
{131}I radioactive uptake cold nodule? hot nodule?	Cold :↓ {131}I radioactive uptake Hot: ↑ {131}I radioactive uptake

lingual thyroid clinical findings?	Mass at base of the tongue
Thyroglossal duct cyst midline mass?	cystic midline mass is close to byoid bone
Branchial cleft cyst located?	located in the anterolateral neck
Acute thyroiditis clinical findings?	thyrotozicosis; ↓ {131}I radioactive uptake
Subacute granulomatous thyoiditis is the most common cause of ____?	of painful thyroid; virus induced;no adenopathy
Hashimoto's thyroiditis epidemiology?	autoimmune thyroiditis
Hashimoto's thyroiditis is hypersensitive to ?	type IV (mainly) and type II hypersensitivity

what is the most common type cause of hypothyroidism?	Hashimoto's thyroiditis
Reidel's thyroiditis = ?	fibrous tissue replacement of gland and surrounding tissue
Subacute painless lymphocytic thyroidits = ?	develops post partum; progression to hypothyroidism
Pheochromocytoma epidemiology ?	majority benign, unilateral, arise in adrenal medulla
Associations of Pheochromocytoma ?	neurofibromatosis, MEN IIa/IIb; von Hippel-Lindau disease
Pheochromocytoma unique findings?	palpitations, paroxysmal hypertension, anxiety, drenching sweats, headache orthostatic hypotension, chest pain, ileus
Pheochromocytoma Diagnosis test?	plasma free metanephrines is best screen

Pheochromocytoma urine tests?	24- hour collection for metanephrine (best test), VMA
Pheochromocytoma lab findings?	hyperglycemia, neutrophilic leukocytosis
Neuroblastoma malignant tumor = ?	postganglionic sympathetic neurons
Neuroblastoma occurs most often to?	childhood tumor and cause of hypertension
Opsoclonus-myoclonus syndrome = ?	paraneoplastic syndrome; myoclonic jerk; chaotic eye movements
Neuroblastoma: "small tumor" shows _____ under electron microscopy	neurosecretory granules
Neuroblastoma clinical findings?	child with abdominal mass + hypertension

Insulinoma lab findings?	↑ serum insulin ↑ C-peptide
Insulinoma if patient injecting excess insulin: lab findings?	↑ serum insulin ↓ C-peptide
DM (Diabetes Mellitus) is the most common cause of what conditions?	blindness, peripheral neuropathy, chronic renal failure, below -knee amputation
Maturity onset diabetes of the young (MODY) = ?	AD (Autosomal dominant) inheritance; not obese; impaired glucose-induced secretion of insulin
Metabolic syndrome in DM?	insulin resistance exacerbated by obesity
insulin resistance syndrome a associations?	acanthosis nigricans; Alzheimer;s disease
Hyperinsulinemia clinical and lab findings?	↑ VLDL (very low-density lipoprotein), hypertension, CAD (Coronary artery disease) ↓ HDL-CL (high-density lipoprotein cholesterol)

what prevents complications in diabetes?	good glycemic control
NEG (Nonenzymatic glycosylation) = ?	Hb(↷A1c), hyaline, arteriolosclerosis, glomerulopathy
Aldose reductase = ?	converts glucose to sorbitol; osmotic damage
Osmotic damage = ?	cataracts, peripheral, neuropathy, retinopathy
Diabetic microangiopathy = ?	diabetic glomerular disease
what is the most common complication of diabetes?	Insulin-induced hypoglycemia
DKA (Diabetic ketoacidosis) is a complication of which type of DM?	type 1 DM

what is the most important mechanism of hyperglycemia in diabetic ketoacidosis?	Gluconeogenesis
Ketoacids are synthesized from ?	synthesized from acetyl CoA derived from β-oxidation of fatty acids
Hypertriglyceridemia mechanism?	↓ capillary lipoprotein lipase activity; ↓ hydrolysis of chylomicrons and VLDL (very low-density lipoprotein)
DKA (diabetic ketoacidosis) electrolytes lab findings?	↓ serum sodium, bicarbonate (metabolic acidosis); ↑ serum potassium, anion gap
Hyperomolar nonketotic coma is a complication of which type of DM?	type 2 DM
Hb(~A1c) is a marker of ?	long term glycemic control
IGT (Impaired glucose tolerance) = ?	prediabetic state; insulin resistance

GDM (Gestational diabetes) = ?	anti-insulin effect of HPL (human placental lactogen), cortisol and progesterone
Macrosomia (newborn risk) : an increase of insulin causes?	↑ in adipose and muscle
Respiratory distress syndrome (RDS) an increase of insulin causes?	inhibits fetal surfactant
Neonatal hypoglycemia an increase of insulin causes?	drives glucose into hypoglycemic range; give newborn glucose at birth
Type I polyglandular syndrome clinical findings?	Addison's disease, primary hypoparathyroidism, mucocutaneous candidiasis
Type II polyglandular syndrome clinical findings?	Addison's disease, Hashimoto's thyroiditis, type 1 diabetes
What are the two subdivisions of Hypoglycemia?	fed state and fasting state

Fed state hypoglycemia: reactive hypoglycemia most common cause?	excess insulin; adrenergic symptoms
Fasting hypoglycemia causes?	alcohol excess; insulinoma; cirrhosis
Alcohol excess : what happens to glycogen stores? gluconeogenesis?	↓ glycogen stores ↓gluconeogenesis (pyruvate converted to lactate)
Fasting hypoglycemia in children look for signs of ___?	look for inborn errors of metabolism
Neuroglycopenia symptoms?	dizziness, mental status changes, motor disturbances
Diagnosis for fasting hypoglycemia?	prolonged fast; satisfy Whipple's triad
Hypoparathyroidism causes?	autoimmune Hypoparathyroidism is the most common cause

DiGeorge syndrome = ?	failure of descent of 3rd/4th pharyngeal pouches; absent parathyroids and thymus
what is the most common pathologic cause of hypocalcemia in the hospital?	Hypomagnesemia
Hypomagnesemia causes?	diarrhea, aminoglycosides, diuretics, alcohol
Hypomagnesemia lab findings?	↓ serum calcium, PTH ↑ serum phosphorus
what is the most common cause of hypocalcemia; causes hypovitaminosis D?	chronic renal failure
Primary HPTH (hyperparathyroidism) associated with?	MEN I, MEN IIa
most common cause of primary HPTH?	benign adenoma

Primary hyperplasia which glands are involved?	all glands are involved
Primary HPTH what is the most common presentation	renal stones
Primary HPTH gastrointestinal clinical findings?	acute pancreatitis
Primary HPTH bone and joint clinical findings?	osteitis fibrosa cystica; subperiosteal bone resorption; osteoporosis; pseudogout
Primary HPTH: hypertension is caused by?	hypercalcemia
Primary HPTH memory trick for symptoms?	"stones, bones, abdominal groans, and psychic moans"
best screening test for primary HPTH?	Intact serum PTH (iPTH)

Primary HPTH lab findings?	↑ serum calcium/PTH; ↓ serum phosphorus/bicarbonate; chloride/phosphorus ratio > 33; ↑ serum calcitriol
Primary HPTH vs. malignancy ?	↑ PTH in Primary HPTH ↓ PTH in malignancy
what is the most common cause of hypercalcemia in the hospital?	malignancy
Secondary HPTH = ?	compensation for hypocalcemia
What is the danger in Insulin treatment?	danger of developing hypophosphatemia
hypophosphatemia is most commonly caused by ____?	alkalosis
hyperphosphatemia is most commonly caused by _____?	renal failure

Adrenal cortex hormones: glomerulosa →? fasciculata → ? reticularis → ?	glomerulosa → mineralocorticoids fasciculata → glucocorticoids reticularis → sex hormones
Peripheral tissue sites?	skin, testis, prostate, seminal vesicles, epididymis, liver
What produces catecholamines?	adrenal medulla
What are the metabolic end-products of Epinephrine (EPI)/ norepinephrine (NOR)?	metanephrines, vanillylmandelic acid(VMA)
what does abrupt withdrawal of corticosteroids cause?	acute adrenocortical insufficiency
Waterhouse-Friderichsen syndrome = ?	N. meningitidis sepsis → DIC → bilateral adrenal hemorrhage
What is the most common cause of Addison's disease in U.S.?	Autoimmune disease

What is the most common cause of Addison's disease in developing countries?	Miliary TB
What is the most common cause of Addison's disease in children?	adrenogenital syndrome
Addison's disease = ?	diffuse hyperpigmentation; hypotension, weakness
Metyraphone test = ?	↓ cortisol → ↑ ACTH → ↓ 11-deoxycortisol
Addison's disease lab findings?	↓ serum sodium, cortisol, bicarbonate ↑ serum potassium, ACTH
Addison's disease: conditions that develop?	hypoclycemia, eosinophilia, lymphocytosis, neutropenia
↑ 17-KS, testosterone, DHT effects on females? males?	ambiguous genitalia in females, percocious puberty males and females

What is the first step taken when a newborn has ambiguous genitalia?	determine genetic sex with chromosome analysis
↓ 17-KS, testosterone DHT causes?	delayed menarche and secondary sex characteristics; males develop pseudohermaphroditism
↑ Mineralocorticoids causes what?	sodium retention with hypertension
↓ Mineralocorticoids causes what?	Sodium loss with hypotension
what is the most common cause of adrenogenital syndrome?	Classic 21-OHase deficiency
Classic 21-OHase deficiency = ?	impaired cortisol and mineralocorticoid production (salt loss); ↑ androgens
Nonclassic 21-OHase deficiency = ?	impaired cortisol synthesis only; virilization

11-OHase deficiency = ?	impaired cortisol + mineralocorticoid excess (salt retainer); ↑ androgens
17-OHase deficiency = ?	impaired cortisol and androgens; ↑ mineralocorticoid production
Dignosis of adrenogenital syndrome?	17-OH progesterone screening test; ↑ 21- and 11- OHase deficiency; ↓ 17-OHase deficiency
most common cause of Cushing syndrome?	corticosteroid therapy (iatrogenic)
most common pathologic cause of Cushing syndrome?	pituitary Cushing
Pituitary Cushing, Adrenal Cushing, Ectopic Cushing syndrome: what happens to ACTH? cortisol?	Pituitary Cushing: ↑ACTH, ↑cortisol Adrenal Cushing: ↓ACTH, ↑cortisol Ectopic Cushing: ↑↑ACTH, ↑cortisol
Cushing syndrome clinical findings?	truncal obesity, thin extremities, purple stria hypertension, hirsutism

What does Hypercortisolism cause?	thin extremities, purple stria
What does Hyperinsulinemia cause?	truncal obesity
Cushing syndrome screening tests?	↑ urine free cortisol; no suppression of cortisol with low dose of dexamethasone
Pituitary Cushing syndrome suppresses what?	suppression of cortisol by high-dose dexamethasone
Cushing lab findings?	hyperglycemia; hypokalemia; metabolic alkalosis
Nelson's syndrome = ?	bilateral adrenalectomy causes enlargement of preexisting pituitary adenoma
Primary hyperaldosteronism = ?	hypertension, hypernatremia, hypokalemia, metabolic alkalosis

Secondary hyperaldosteronism = ?	compensation for ↓ cardiac output; activation of renin-angiotensin-aldosterone (RAA) system
Hypothyroidism = ?	patients are hypometabolic
what does the brain need in Hypothyroidism?	brain requires thyroxine for maturation
What is the most common cause for Cretinism?	most often caused by maternal hypothyroidism before fetal thyroid is developed
Cretinism Clinical findings?	severe mental retardation
Hashimoto's thyroiditis clinical findings?	muscle weakness (common complaint), weight gain, dry/brittle hair cold intolerance, constipation, hypertension from sodium retention; delayed reflexes
Hashimoto's thyroiditis : periorbital puffiness, and hoarse voice are signs of ____?	myxedema

Primary Hypothyroidism lab findings?	↓ serum T4/FT4; ↑ serum TSH, cholesterol
Myxedema coma clinical findings?	stupor, hypothermia, hypoventilation; IV levothyroxine, corticosteroids
Thyrotoxicosis describes what condition?	hormone excess from any cause
Hyperthyroidism =?	thyrotoxicosis due to excess synthesis of thyroid hormone
most common cause of hyperthyroidism + thyrotoxicosis	Graves' disease
Graves' disease pathogenesis?	anti-TSH receptor antibody, type II hypersensitivity
Clinical findings unique to Graves' disease?	exophthalmos, pretibial myxedema, thyroid acropachy Transient hyperthyroidism in fetus

Thyroid acropachy = ?	digital swelling and clubbing
Graves' disease in the elderly = ?	cardiac and muscle findings predominate; apathetic appearing
Toxic multinodular goiter = ?	one or more nodules in a multinodular goiter becomes TSH-independent
Thyrotoxicosis constitutional signs?	weight loss with a good appetite; heat intolerance; diarrhea oligomenorrhea, lid stare
Thyrotoxicosis cardiac findings?	sinus tachycardia; systolic hypertension; brisk reflexes
Atrial fibrillation in Thyrotoxicosis?	always order a TSH test to rule out hyperthyroidism
Graves' hyperthyroidism lab findings?	↑ serum T4/FT4, ↑ 131I uptake, ↓ serum TSH

Thyrotoxicosis conditions that develop?	↑ glucosee, calcium, lymphocytes; ↓ cholesterol
Treatment for Graves' disease?	β-blockers, thionamides
Thyroid storm clinical findings?	tachyarrhythmias, hyperpyrexia, coma, shock
ESS (Euthyroid sick syndrome) epidemiology?	serum T3 and T4 abnormalities; normal gland function
ESS (Euthyroid sick syndrome) pathogenesis?	block in outer ring deiodinase conversioni of T4 to t3; T3 converted to inactive reverse T3
ESS what is the most common variant?	↓ serum T3 ↑ reverse T3
Goiter = ?	thyroid enlargement

Nontoxic goiter pathogenesis?	absolute or relative deficiency of thyroid hormone hyperplasia/hypertrophy followed by involution; initially diffuse then nodular
Goiter complications?	Toxic nodular goiter ; one or more nodules become TSH-independent
solitary nodule in a woman vs. man/ child likelihood of being malignant?	woman: majority are benign; 15% malignant man: more likely to be malignant
Solitary nodule with history of radiation exposure: likelihood of being malignant?	more likely to be malignant (40%)
First step in management of solitary thyroid nodule?	fine needle aspiration
what is the most common benign thyroid tumor?	Follicular adenoma
what is the most common cause of papillary carcinoma?	psammoma bodies

Papillary carcinoma microscopic findings?	lymphatic invasion
what is the most common cause of thyroid cancer?	Follicular carcinoma; presenting as a solitary cold nodule
Follicular carcinoma spreads?	hematogenous rather than lymphatic spread
MEN IIa syndrome =?	medullary carcinoma, HPTH, pheochromocytoma
NEM IIb (III) syndrome = ?	medullary carcinoma, mucosal neuromas lips/ tongue, pheochromocytoma
Medullary carcinoma pathogenesis?	derives from C cells; calcitonin is tumor marker calcitonin convered into amyloid
Primary B-cell lymphoma derived from?	Hashimoto's thyroiditis

Anaplastic thyroid cancer= ?	rapidly aggressive; uniformally fatal
Superior and inferior parathyroids derive from?	derive from 4th 3rd pharyngeal pouch, respectively
Parathyroid Gland Disorders (PTH) re-absorption rates? [calcium, phosphorus, bicarbonate?]	↑renal calcium reabsorption; ↓ renal phosphorus, bicarbonate reabsorption
PTH in relation to hypocalcemia/hyperphosphatemia & hypercalcemia/ hypophosphatemia	hypocalcemia/hyperphosphatemia = ↑ PTH hypercalcemia/ hypophosphatemia = ↓ PTH
Sunlight is a major source of _____?	vitamin D
what is the role of the liver in production of Vitamin D?	25-hydroxylase converts cholecalciferol to 25-(OH)D (calcidiol)
what is the role of the kidney in production of Vitamin D?	1α-hydroxylase converts 25-(OH)D to 1,25-(OH){subcase 2}D (calcitriol)

Calcitriol functions?	↑ calcium/phosphorus reabsorption in bowel; ↑ osteoclast production
Calcitiol feedback =?	hypocalcemia increases synthesis, hypercalcemia decreases synthesis
Total serum calcium = ?	calcium bound + calcium free (ionized)
Hypoalbuminemia = ?	↓ total serum calcium, normal ionized calcium and PTH
Alkalosis in serum calcium= ?	normal total serum calcium; decreased ionized calcium, increased PTH; tetany
Tetany is due to decreased ionized calcium level: what happens to the potentials?	E⇣t (threshold potential)comes close to E⇣m(membrane potential); initiates action potential
Tetany: clinical findings?	thumb adduct into palm; facial twitching after tapping facial nerve

Metyrapone stimulation test	Metyrapone blocks 11OHase which stimulates ACTH and 11-deoxycortisol and decreases cortisol. If ACTH increases and 11-deoxycortisol decreases the problem is adrenal insuficiency. If both increase, the problem is pituitary insuficiency. If 11-deoxycortisol or ACTH dont change look for adrenal or ectopic Cushing's.
High dose dexamethasone suppression test	Dexamethasone is a cortisol analog and should suppress ACTH and cortisol. If it does suppress cortisol its pituitary Cushing's. If it doenst, its adrenal or ectopic Cushing's
Clinical features of hyperthyroidism	Tachychardia, palpitations, atrial fibrillation, systolic hypertension, nervousness, diaphoresis, tremors, diarrhea, weight loss. High free T4 and decreased TSH (if primary) or increased TSH (if secondary)
Graves disease	IgG autoantibodies stimulate TSH receptors. Signs and symptoms of hyperthyroidism plus goiter, exophthalmus and pretibial myxedema (due to glycosamynoglycan deposition)
Effect of oral contraceptives and anbolic steroids on binding proteins	Contraceptives increase binding proteins and increase toal levels of the hormone with normal TSH or ACTH. Anabolics do the opposite. Increased total T4 or total cortisol with normal TSH or ACTH indicates contraceptives. Decreased total T4 or cortisol with normal TSH or ACTH indicates anabolic steroids.

Clinical features of hypothyroidism	Fatigue, cold sensitivity, decreased cardiac output, myxedema, constipation, anovulatory cycles. Low free T4 with high TSH (primary) or low TSH (secondary)
Cretinism	Congenital hypothyroidism. Mental retardation, failure to thrive, stunted bone growth.
Hashimoto thyroiditis	Anti-microsomal antibodies against thyroid gland produces hypothyroidism. Lymphocytic inflamation with germinal centers
Subacute thyroiditis	Preceded by viral illness. Granulomatous inflamation
Reidel thyroiditis	Destruction of the thyroid gland by dense fibrosis. Irregular hard thyroid that is adherent to trachea (dyspnea) and esopahgus (dysphagia)
Thyroid adenoma	Painless solitary cold nodule that may be functional
Papillary thyroid carcinoma	80% of malignant thyroid tumors. Papillary pattern, psammoma bodies, clear "orphan Annie" nuclei. Lymphatic spread to cervical nodes is common

Follicular thyroid carcinoma	15% of malignant thyroid tumors. Hematogenous metastasis to bone or lungs
Medullary thyroid carcinoma	5% of malignant thyroid tumors. Arises from C cells and produce calcitonin. May be associated with MEN II
Anaplastic thyroid carcionoma	Firm enlarging mass that metastasizes to trachea and esophagus and causes dyspnea and dysphagia
Primary hyperparathyroidism	Excess PTH with hypercalcemia caused by parathyroid adenoma (80%), hyperplasia (15%) or paraneoplastic syndromes (lung SCC and renal cell carcinoma). High serum Ca+ and PTH, kidney stones, osteoporosis, short QT interval.
Secondary hyperparathyroidism	Caused by chronic renal failure (no phosphate excretion increases serum phosphate, decreasing Ca+ and increasing PTH), chronic renal failure also causes deficiency of alpha-1-hydroxylase and vitamin D. Vitamin D deficiency and malabsorption

Functions of PTH	Increase reabsorption of Ca+ in distal tubule, decreases phosphate reabsorption in proximal tubule, increases alpha-1-hydroxylase in proximal tubule, binds to PTH receptor on osteoblast releasing IL-1 (osteoclast activating factor) to activate osteoclast resorption
Hypoparathyroidism	Due to surgical removal of glands or DiGeorge syndrome. Hypocalcemia with low PTH, tetany, prolonged QT interval
Prolactinoma	Hyperprolactinemia produces galactorrhea, amenorrhea (tumor mass inhibits GnRH) and infertility
GH producing adenoma	High GH and somatomedin C (IGF-1) produce gigantism (in children, tall stature with long extremities) or acromegaly (in adults, prominent jaw, flat forehead, enlarged hands and feet, diabetes and visceromegaly)
Sheehan syndrome	Ischemic necrosis of pituitary secondary to post partum hypotension results in panhypopituitarism
Diabetes insipidus	Hypotonic polyuria, polydipsia, hypernatremia, dehydration. Central DI is due to lack of ADH. Nephrogenic DI is due lack of renal response to ADH.

SIADH	Excesive ADH. Oliguria, water retention, hyponatremia, cerebral edema. Due to paraneoplastic syndrome (lung SCC) or trauma
Clinical features of Cushing's syndrome	Thin extremeties (protein catabolism), truncal obesity and buffalo hump (hyperglycemia increases insulin with fat deposition), purple stria (low collagen in vessels), hyperlipidemia (hormone-sensitive lipase), hirsutism, hypertension and hypokalemic alkalosis (high aldosterone).
Differentiation of Cushing's syndrome	High ACTH with dexamethasone suppression --> pituitary. High ACTH without dexamethasone suppression --> ectopic (lung SCC). Low ACTH --> adrenal. Low ACTH with low cortisol and adrenal atrophy --> steroid therapy (MCC). High ACTH produces skin pigmentation in pituitary and ectopic.
Conn syndrome	Adrenocortical adenoma causes hypertension, hypernatremia, hypokalemia, metabolic alkalosis, tetany. High aldosterone and low renin
Waterhouse-Friderichsen syndrome	Bilateral hemorrhagic infarction of the adrenals associated with menigococcemia. DIC, hypotension, shock.

Addison disease	Autoimmune destruction of adrenal cortex due to abrupt withdrawal of corticosteroids, miliary TB or menincoccemia. Weakness, hyperpigmentation of skin (high ACTH), hypotension, hypoglycemia, poor response to stress
Pheochromocytoma	Catecholamine-producing benign tumor of the adrenal medulla. Severe headaches, tachycardia, palpitationss, diaphoresis, anxiety, hypertension. Associated with MEN II. Elevated urinary vanillylmandelic acid (VMA) and catecholamines.
MEN I	Tumors of the pituitary (non-functioning), parathyroids (hypercalcemia), and pancreas gastrinoma (zollinger-ellison)
MEN II	Medullary carcinoma of thyroid, pheochromocytoma, parathyroid hyperplasia or adenoma

Pathophysiology of diabetic ketoacidosis	Hyperglycemia (due to increased glycogenolysis and gluconeogenesis). Ketone bodies (low insulin and high cortisol/epinephrine activate hormone-sensitive lipase, B-oxidation and ketogenesis). Osmotic diuresis and volume depletion with loss of potassium. Dilutional hyponatremia due to osmotic effect of hyperglycemia. Low insulin fails to activate lipoprotein lipase leading to hypertriglyceridemia.
Pathophysiology of diabetic vascular disease	Non-enzymatic glycosylation and arteriosclerosis produces ischemic injury and diabetic foot. Accelerated atherosclerosis, abdominal aortic aneurysms and MI.
Pathophysiology of diabetic ocular disease	Cataracts due to conversion of glucose into sorbitol by aldose reductase in the lens. Retinopathy with microaneurysms (non-proliferative) and neovascularization (proliferative)
Pathophysiology of diabetic neuropathy	Destruction of Schwann cells with decreased peripheral sensitivity liads to pressure ulcers on bottom of diabetic foot