Cushings Disease			
Decreased ACTH	Exogenous glucocorticoids		
	Adrenal Tumor		
Increased ACTH	High dose dexamethasone	Increased ACTH	Ectopic ACTH suppression
	High dose dexamethasone	Decreased ACTH	Cushings Disease(Pituitary Adenoma)
	CRH Stimulation	Increase ACTH/Cortisol	Cushings Disease(Pituitary Adenoma)
	CRH Stimulation	No change ACTH/Cortisol*	Ectopic ACTH secretion.
*Dexamethasone should suppress ACTH and cortisol- if it doesn't then ectopic cause			
**CRH should increase ACTH- if it doesn't then ACTH is being secreted from ectopic cause			

Congenital Adrenal Hyperplasia				
Deficiency	**Build-up**	**BP**	**Sex Hormones**	**Presentation**
17a-hydroxylase	Mineralocorticoids (Aldosterone)	Increased	Decreased	XY: Atypical Genitalia, undescended testes
				XX: no secondary sexual characteristics
21-hydroxylase	Progesterone	Decreased	Increased	XY: salt wasting or precocious puberty
	17-hydroxyproge-sterone			XX: virilization
11B-hydroxylase	11-deoxysterone	Increased (because 11-deoxycorticosterone has weak mineralocorticoid properties)	Increased	XY: severe HTN or precocious puberty
	11-deoxycortisol			XX: virilization
3B-hydroxysteroid dehydrogenase	Pregnenolone	Decreased	Decreased	XY:adrenal insufficiency, ambiguous genitalia

Congenital Adrenal Hyperplasia (cont)

17-hydroxypregnenolone	XX:adrenal insufficiency, ambiguous genitalia
DHEA	

Heavy Menses

- Adenomyosis
- Endometriosis
- Leiomyoma (Fibroids)
- Endometrial Hyperplasia
- Endometrial Cancer

Male Genitalia

DHT	External Male Genitalia+ Prostate
Testos-terone	Internal Male Genitalia- (SEED= Seminal Vesicles, Epididymis, Ejaculatory Duct, Ductus Deferens)

*if female internal genitalia is present in a male then think **Anti Mullerian Hormone deficiency**

Estrogen	Breast development
Aromatase	Converts testosterone--> estrogen

Vaginitis

If there is vaginal inflam-mation	Think **Trichomoniasis** or **Candida vaginitis**

Bacterial vaginosis	fishy odor, clue cells(squ-amous epithelial cells), (+) whiff test	Due to antibiotic use	Treat with metron idazole

Vaginitis (cont)

Trichm-oniasis	frothy, yellow-green discharge, motile tricho-monads,	Due to hx of STIs	Treat with metron idazole
Candida	cottage cheese, pseudo-hyphae	Due to OCP use, antibi-otics, immuno-suppre-ssion, diabetes	Treat with **flucon-azole**

Ovarian Tumor

Struma Ovarii	Germ cell tumor
	Associated with thyrotoxicosis (since it can secrete thyroid hormone)
	Gross: Oily cystic mass
	Microscopic: thyroid follicles filled with colloid and surrounded by ovarian storm

Ducts

Parame son-ephric	form internal female genitalia (fallopian tubes, uterus, upper vagina, cervix)
Mesone phric	In females= degenerate to form Gartner duct
	In males= epididymis, seminal vesicles, ductus deferens, ejaculatory ducts

Genital Ulcers

Painful	**Painless**
Chancroid (H.Ducreyi)	Chlamydia
HSV	Syphilis

Staging/ Gleason Score

High Gleason score	poorly differentiated
Higher Staging	metastasis

Lymph Nodes

Para-Aortic	Ovaries/ Testes
Superficial Inguinal	Scrotum
Deep Inguinal	Glans Penis

Anorexia and Amenorrhea

- Due to hypothalamic suppression of GnRH

Hypertension in Pregnancy

Think Pre-Eclampsia/ Eclampsia	New-onset HTN
	Proteinuria
	End-organ dysfunction
	(+ seizures in eclampsia)

Vaginal Bleeding during Pregnancy

Placenta Accreta (Painful)	Due to absence of endometrial decide basalts
	Or previous uterine scarring (prior C-section)
Molar Pregnancy (Painful)	
	Partial Mole- has fetal tissue

Vaginal Bleeding during Pregnancy (cont)

Complete Mole- NO fetal tissue
Ectopic Pregnancy (Mild Pain)
Abruptio Placentae (Painful)
Placenta Previa (Painless bleeding)

Cervicitis (PID)

Neisseria Gonorrhoeae
Chlamydia Trachomatis
*if antibiotic coverage remains incomplete then can lead to infertility

Combination Therapy

Buserelin and Bicalutamide	Buserelin- continuous stimulation of pituitary gland to down-regulate GnRH--> decrease LH-->decrease testosterone
	Bicalutamide- helps prevent the initial testosterone surge effect from the buserelin administration
Leuprolide and Flutamide	Leuprolide-long acting GnRH antagonist- causes a transient increase in LH-->testosterone levels
	Flutamide-competitive testosterone receptor blocker-counteracts the initial surge of testosterone

Drugs for pregnancy termination

Mifepristone	-partial progesterone agonist--->causes placental separation and uterine contraction
Misoprostol	E1 agonist--> stimulates uterine contractions
Methotrexate	Folic acid antagonist-->destroys proliferating fetal cells

Mullein Agencies vs Androgen Insensitivity

Mullein Genesis	Stage 4: breast; Stage 4: pubic hair
AIS	Stage 4: breast; Stage 2: pubic hair

Phases of Meiosis

Female	Prophase I until ovulation
	Metaphase 2 until fertilization

Postmenopausal bleeding

Think Endometrial Hyperplasia or Cancer

Testicular Torsion

Has absent cremasteric reflex

Physiologic Changes in Pregnancy

Increase CO
Increase Plasma Volume
Decrease SVR
Increase Response Tidal Volume
Decrease FRC
Increase GFR
Chronic hypoventilation (-->respiratory alkalosis)

Congenital Rubella

I heart ruby earrings:
I("eye")--> cataracts
Heart-->PDA
Ruby--> Rubella (enveloped, single-stranded, positive-sense RNA virus)
Earrings--> sensorineural hearing loss

Hypoglycemic Episodes

More common with **Sulfonylureas**:	Inhibits K+ channel to allow for Ca2+ influx

SGLT-2 Inhibitors (-glifozin)

decrease renal absorption of glucose and sodium

Insulin (Endogenous vs Exogenous)

Endogenous	insulin+**C-peptide**	endopeptidases in secretory granules cleave proinsulin into insulin and C-peptide
		Sulfonylureas and Meglitinides are the only ones that stimulate endogenous insulin production
		Insulin producing tumors
Exogenous	insulin only	

Congenital Hypothyroidism

high TSH; low T4	*Initially asymptomatic* because of the presence of material thyroxine

Congenital Hypothyroidism (cont)

Need levothyroxine treatment otherwise can lead to neurocognitive dysfunction

SIADH

Think Euvolemic Hyponatremia

Neonatal Hypoglycemia

Mother had gestational diabetes--> hyperglycemia in mother-->hyperglycemia in baby-> so baby was producing high levels of insulin to counteract high glucose--> pancreatic B cell hyperplasia since it is working extra--> once born then not receiving high levels of glucose from mother but still producing a lot of insulin=neonatal hypoglycemia

MEN Syndromes

MEN 1	Pituitary (Usually a Prolactinoma)
	Pancreas (Usually Zollinger Ellison syndrome)
	Parathyroid
Men 2A	Parathyroid
	Medullary thyroid carcinoma
	Pheochromocytoma
MEN 2B	**Medullary** thyroid carcinoma
	Pheochromocytoma
	Oral Ganglioneuromas

SIADH

Think Euvolemic Hyponatremia

TBG Deficiency

low total T4, normal free T4, normal TSH

Adrenal Gland Secretions

Adrenal Cortex	Zona Glomerulosa	Aldosterone
	Zona Fasiculata	Cortisol
	Zona Reticularis	Sex Hormones
Adrenal Medulla		Catecholamines

Ketone Bodies

Can't be utilize by RBCs since they lack mitochondria

Symptomatic Relief of Thyrotoxicosis

B-blockers

Diabetes

Type 1	Due to autoimmune response against beta cells--> loss of insulin production
	More susceptible to DKA
	Younger patients
Type 2	Due to insulin resistance
	Older patients
	Pancreatic islet amyloid deposition

Antihyperlipidemic drugs

Statins	Primarily lowers cholesterol
Ezetimibe	Lowers cholesterol and LDL
Fibrates	Lowers triglycerides
Niacin	Lowers triglyceride and LDL, Increases HDL
Bile acid resins	Lowers LDL

Glucose Transporters

GLUT-4	Insulin Dependent--> expression increases with insulin concentration
	Muscle cells, Adipocytes
GLUT 1,2,3,5	Insulin-Independent--> does NOT increase with insulin concentration
	Brain, Kidney, Intestine, RBCs, Liver

Bone Formation/ Loss

PTH--> osteoblast-->Bone formation

PTH-->RANK(osteoblasts)+RANK-L/NFkB-L (osteoclasts)-->net bone loss

OPG-bind competitively to RANK-L to prevent bone loss

Insulin Drugs

Long-acting (Glargine, detemir, degludec)	mimic regular insulin
Short-acting (Lispro, aspart, glulisine)	mimics postprandial insulin

Permissive Effect of Cortisol on Ne/Epi

Cortisol indirectly acts Ne/Epi receptors to upregulate them--> this then makes them available for the the catecholamines to bind to them

Turner syndrome (45, XO)

- Widely spaced nipples
- Ovarian dysgenesis (streak ovaries)
- Horseshoe kidney
- Short
- Webbed neck
- Coarctation of aorta
- Aortic Dissection
- Bicuspid Aortic valve
- Cystic Hygroma in neck

Klinefelter (47, XXY)

- Decreased inhibin levels
- Learning disabilities
- Gynecomastia
- Small, firm testes
- Tall

Edwards syndrome

- Hypertonia
- Clenched fists
- Rocker bottom feet

Down syndrome

- Upslanting palpebral fissures
- Epicanthal folds
- Single palmer crease

Drugs and common AE

Levothyroxine	Atrial fibrillation
PTU, methimazole	Agranulocytosis (Nuetropenia)
	PTU- Hepatotoxic (But preferred drug in last two trimesters)
	Methimazole-Teratogenic in FIRST trimester
Sulfonyureas	Hypoglycemia
HIV HAART therapy	Lipodystrophy
B-Blockers	Mask Hypoglycemic episodes
Estrogen	ischemic stoke (therefore don't give in migraines since they already have an increased baseline for an ischemic stroke)
Metformin	Lactic acidosis (CI in renal insufficiency)
Tamoxifen	Gynecomastia
Glucocorticoids	Osteoporosis
GLP-1 agonists (exenatide/liraglutide)	Weight loss
Trazadone	Priapism
Bile acid resins (Cholestyramine, Cholestipol, Colesevelam)	Hypertriglceridemia

Intracellular Pathways

Pathway	Hormones
Bind activated receptors to DNA to modify transcription	Steroid and thyroid hormones
Adenylyl cyclase converts ATP to cyclic amp--> activates protein kinase A	PTH, ACTH, TSH, ADH(V2), glucagon
Bind G protein coupled receptor that activate PLC	GnRH, TRH, Ang II, AHDH(V1)
PLC activates PIP3--> DAG+IP3 to then activate PKC	GnRH, TRH, Ang II, AHDH(V1)
JAK-STAT Pathway	GH

Familial Hyperlipoproteinemia (Type 1)

deficiency of LPL or Apo C-2:	Hypertriglyceridemia
	recurrent acute pancreatitis
	milky appearing retinal vasculature (lipemia retinals)
	yellow populates on extensor surfaces (eruptive xanthomas)

Keep-it-Simple Concepts	
PTH	increase Ca+ (Primary regulator of PTH-if low then PTH increases to increase Ca2+ levels)
	decrease PO4-
	increase Vitamin D (by upregulating 1-a-hydroxylase)
Ca2+	
Calcitonin	counteracts Ca2+
Insulin	decreases glucose (bloodstream--> adipose tissue/skeletal muscle)
	counteracts glucagon
Glucagon	increases glucose (adipose tissue/skeletal muscle-->bloodstream)
Vitamin D	increase Ca+
	increase PO4-
Leydig cells	LH-->secrete testosterone
Sertoli cells	FSH-->spermatogenesis and increase inhibin
	increases glucose by glycogenolysis and gluconeogenesis in the *liver*

Neimann-Pick vs Tay-Sachs	
Neimann-Pick	Hepatomegaly/Splenomegaly
Tay-Sachs	NO hepatomegaly/splenomegaly