Lec 11 continued

Metabolic Alkalosis

- ↑ in bicarb blood level, will lead to ↑ bicarb filtration, ↓ bicarb reabsorption, & ↓ H^+ secretion

Disturbance	*pH*	*HCO_3^-*	*pCO_2*	*HCO_3^-/pCO_2 in plasma*	*Compensation*
metabolic acidosis	⇓	⇓	⇓	⇓	⇑ventilation ⇑renal HCO_3^- production
respiratory acidosis	⇓	⇑	⇑	⇓	⇑renal HCO_3^- production
metabolic alkalosis	⇑	⇑	⇑	⇑	⇓ventilation ⇑renal HCO_3^- excretion
respiratory alkalosis	⇑	⇓	⇓	⇑	⇑renal HCO_3^- excretion

Test	Normal	Decrease Value	Increase Value
pH	7.35-7.45	Acidosis	Alkalosis
PaCO2	35-45	Alkalosis	Acidosis
HCO3	22-26	Acidosis	Alkalosis
PaO2	80-100	Hypoxemia	O2 therapy
SaO2	95-100%	Hypoxemia	----------

Normal values of pH, pCO_2 and HCO_3^-

FOR MEMORIZATION!!

	pH	HCO_3^-	CO_2
Metabolic acidosis	↓	↓	Normal
Metabolic alkalosis	↑	↑	Normal
Metabolic acidosis with respiratory compensation	↓	↓	↓
Metabolic alkalosis with respiratory compensation	↑	↑	↑

Conditions accompanied by Acidosis / Alkalosis

Metabolic Acidosis

- Aspirin poisoning, diabetes, & carbonic anhydrase inhibitors → ↑ H^+
- Diarrhea → ↑ HCO_3^- loss; Renal tubular acidosis → ↓ H^+ secretion & HCO_3^- reabsorption

Respiratory Acidosis

- brain / lung damage (emphysema, pneumonia)

Respiratory Alkalosis

- high altitude, fear, pain → hyperventilation

Metabolic Alkalosis

- ↑ base intake ($NaHCO_3^-$) / vomiting
- mineralocorticoid (Aldosterone) excess → ↑ K^+ & H^+ secretion, ↑ HCO_3^- reabsorption & production
- diuretic overuse → ↑ K^+ & H^+ secretion → ↓ ECF → and aldosterone will be produced to make up for ↓ ECF

↳ except carbonic anhydrase inhibitors

Anion Gap

- used to diagnose Acid/Base abnormality, especially in metabolic disorders

- osmolarity of cations should = osmolarity of anions in body
  ↳ 153 mEq/L ; but some anions are unmeasurable, & are called anion gap ... we calculate gap by ....

Anion gap = Na^+ conc - Cl^- conc - HCO_3^- conc.

10 = 142 - 108 - 24

- Normal gap should be btwn 8-16 mEq/L

hyperchloremic metabolic acidosis

- normal anion gap, high Cl^-, & ↓ HCO_3^-
- associated w/ diarrhea, tubular acidosis, carbonic anhydrase inhibitors, Addisons disease

Normochloric metabolic Acidosis

- high gap, normal Cl^-, low HCO_3^-
- associated w/ diabetes, Aspirin & methanol poisoning, lactic acidosis, starvation

please look at the last few pages of sheet 10 & 11 to answer clinical case questions !!! ♡

Lec 12 male Reproductive System

- the main male reproductive organ is 900 coiled tubes, each about .5 meters long called the testis where

Spermatogenisis takes place

↳ formation of mature sperm from immature spermatogonia under the influence of GnRH by the hypothalamus

- Spermatigonia are present from birth, mature to sperm at puberty (10-13 yrs), & ↓ w/ old age (climacteric period)
- Spermatogonia originate from primordial cells, then migrate to inner lining of seminiferous tubules during 5th gestational week

Sertoli Cells

- Supportive & nourishing cells under the influence of FSH by ant. pituitary
- large w/ overflowing cytoplasm around spermatigonia in the central lumen of semineferous tubule

Leydig Cells

- in the interstitium btwn semineferous tubules that secrete testosterone under influence of LH from ant. pituitary
- numerous in newborns, dissapear, then become active again at puberty – Adulthood

Hormones Stimulating Spermatogenisis

Testosterone

- secreted by genital ridges in first 7 weeks of gestation, then by fetal testes (Leydig cells)
- Supports growth & division of germ cells → spermatogenisis, & Androgenic effect for primary & secondary male features

- ↑ testosterone has negative feedback on secretion of FSH, LH, & GnRH by pituitary

Lutenizing Hormone (LH)

- from ant. pituitary to act on leydig for testosterone production

Follicle Stimulating Hormone (FSH)

- GnRH from hypothalamus stimulates FSH secretion from ant. pituitary to act on Sertoli cells (essential for spermatogenisis)

Estrogen

- formed from testosterone when sertoli cells are stimulated by FSH

Growth Hormone (GH)

- from ant. pituitary for metabolic function in testes & early division of spermatagonia

* no GH (dwarfism) = no spermatogenisis
* no FSH = no spermatogenisis

Inhibin → from the name, it inhibits spermatogenisis

- from sertoli cells, & have negative feedback on LH & FSH

Spermatogenesis Steps

- from spermatogonia → mature sperm takes 74 days
- in embryogenesis, Spermatigonia from primordial germ cells migrate from abdomen to line seminiferous tubules of testes, & no division or development will occur until puberty
- At puberty, GnRH ↑, & Spermatagonia proliferate into diploid primary spermatocytes (25 days)
- primary undergo 1st stage of meiosis = haploid Secondary

Spermatocytes (9 days)

- 2ndary undergo 2nd meiosis stage = haploid spermatid (19 days)
- Spermatid differentiate to mature sperm (21 days)

* 1 primary spermatocyte makes 2 sperm w/ X chrom. & 2 sperm w/ Y chrom. (4 sperm total)

- After sperm mature in seminiferous tubules, they take a few days to move to epididymis (non-motile)... & after 18-24 hrs., they are capable of being motile, but still non-motile b/c they are inhibited by epididymal proteins ... After ejaculation, they are motile to fertilize ovum, & stay 1-2 days in female genital tract.
- about 120 million sperms produced each day, small amount stored in epididymis, majority stored in vas deferens w/ a life expectancy of 1 month

Semen Contributions

Sertoli cells / Epididymis epithelium

- contain testosteron/estrogen, enzymes & essential nutrients for sperm maturation

* Vas deferens provides 10% of semenal fluid

Seminal Vesicles (60%)

- pH of 7.2-8 & contains citric acid, nutrients, fibrinogen
- mucoid material w/ fructose → give semen viscosity
- large quantity of prostaglandins → help w/ fertilization by:
  1) ↓ female immune response to sperm

2) makes cervical mucus receptive to sperm movement by reverse peristaltic contractions of uterus & fallopian tubes to move sperm to ovaries

Prostate gland (30%)

- milky Alkaline fluid w/ Ca^{2+}, citrate & phosphate ion, clotting enzyme, profibrinolysin
- Alkaline fluid important for fertilization b/c it neutrilizes acidic vas deferens fluid, & Acidic vaginal secretions (3.5-4 pH) for better sperm motility at (6-6.5 pH)

Bulbourethral Glands

- mucus secretion for facilitation of movement & lubrication

Capacitation of Spermatozoa

- capacitation occurs to fresh ejaculated semen in female genital tract w/in 1-10 hrs. to help w/ fertilization by:

1) washing out factors that inhibit sperm motility, by uterine & fallopian fluid

2) swimming away from cholesterol vesicles ... the acrosome gets thinner ...

3) & more permiable to Ca^{++}

Structure of Mature Sperm

- Acrosomal head w/ hyaluronidase (digest proteoglycans) & proteolytic enzymes (digest proteins) → important for fertilization
- neck & body w/ mitochondria for movement of flagella

- Tail (flagellum)

* mature sperm are motile, & activity is enhanced by Alkaline medium, & inhibited by acidic

Androgens (male sex hormones)

- Steroid hormones causing masculine characteristics, from the testes or Adrenal gland, from Cholesterol or Acetylcoenzyme A

Testosterone

- most abundant, anabolic hormone, secreted from testes to ↑ rate of protein Synthesis in target cells
  - diffuse through cell membrane → converted to DHT by 5-α reductase, & binds w/ cytosolic (androgen) receptors → the dimer/complex is translocated to the nucleus to bind to hormone response element (DNA sequence) for gene expression & protein synthesis

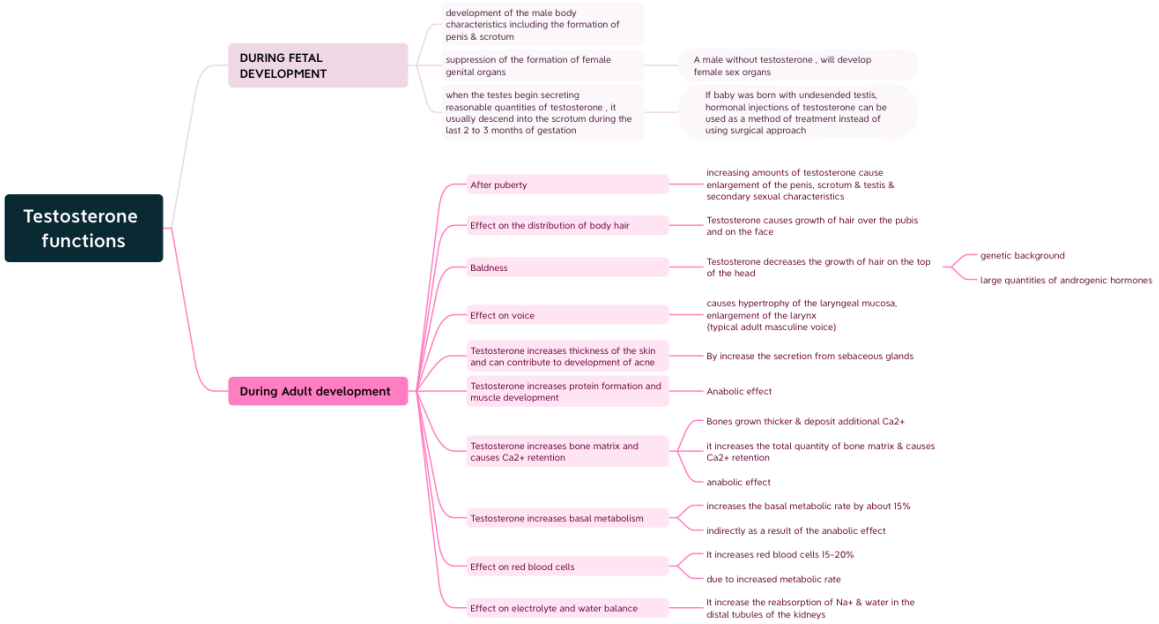
* Small amount from adrenal gland in both sexes

- Secreted in mid trimester (fetus) due to ↑ HCG
- Secreted in neonates (10 weeks after birth) for development of male reproductive organs (no Spermatogenesis)
- After puberty → spermatogenesis & enhance male characteristics

Dihydrotestosterone (DHT)

- more active/potent & less abundant

Androstenedione



just read through this, its pretty self explanitory & make sense

Fertility depends on ...

Sperm count

- 3-5 ml of semen during sex, & each ml has 120 million Sperm

Sperm shape

- if count is normal, but still infertile, there could be abnormal Sperm shape

Motility

- if count & shape are normal, sperm may be non-motile

Abnormalities

Prostate gland

- prostate tissue overgrowth → Benign prostatic fibroadenoma → older age (not from testosterone)
- prostate cancer from testosterone stimulation

Hypogonadism

* pay attention to what stage of life has testes

- if testes are non-functional in fetal life = no male

characteristics → female organs are formed

- if testes lost before puberty (eunuchism) → boy has developed infantile sex organs & characteristics
- if man castrated after puberty → regression of sex organ size & voice, 2ndary characteristics remain

Adiposogenital / Fröhlich Syndrom (hypothalmic eunuchism)

- genitic (hypogonadism) innability to secrete GnRH → obesity w/ eunuchism

Cryptorchidism

- testes dont descend into scrotum in fetal life

Testicular Tumor / hypergonadism

- rare tumor in leydig cells → testosterone overproduction
  - in children = rapid muscular/bone growth & sexual organ development
  - Adults = hard to see testosterone effects

Stages of Sex Act in Males

Penile erection → parasympathetic

Lubrication → parasympathetic

- mucus secretion by urethral & bulbourethral glands

Emission → sympathetic

- contraction of vas deferens, ampulla, prostate, & seminal vesicles, for sperm & fluid to mix in internal urethra w/ mucus of bulbourethra = semen

Ejaculation → sympathetic

- full internal urethra sends impulses to pedundal nerve → Sacral plexus → rhythmic contractions of internal genital organs → ↑ pressure → ejaculation