Patho Study guide: Final Exam

Ch 1:

Levels of disease Prevention:
Primary: Before disease happens
ex) vaccine
Secondary: preventing reinjury or detecting injury
ex) screening
Tatiary: treating the disease

Atrophy: decreased cell size

Hyperplasia: increased number of cells

Hypertrophy: increased cell size

Dysplasia: disorganized cellular appearence

Neoplasia: cancerous

Metaplasia: different type of cell in the wrong place

Apoptosis: occurs to response in injury that does not directly kill the cell

- Triggers intracellular cascades + activates cellular suicide response

- no inflammation
- triggers
- not always a pathological process

Necrosis: ischeimic or toxic injury

- Cell rupture
- spilling of contents into extracellular fluid
- inflammation

Ch 2: Fluid Volume disturbances consequences/ manifestations, AGB interpretations

- Fluid volume disturbances consequences/manifestations

- increased ADH - dilutes body fluids, retains fluids (water)

- increased aldosterone: expands extracellular fluid volume
  - retains water and salt

ADH: released when increased osmolarity, decreased volume, pain and nausea

Aldosterone: Angiotensin II; activated when there's decreased circulating volume

- Clinical manifestations of excess fluid volume: bounding pulse, neck vein distention, short of breath, chest pain, weight gain

- Clinical manifestations of volume deficit: massive inflammation, pass out, dizzy, decreased urine output, dehydrated, skin turgor, dry mucous membranes, not moving stool easily, weight loss

- isotonic dehydration: loss of fluid and electrolytes

- hypotonic dehydration: loss of more electrolytes than water

- hypertonic dehydration: loss of more fluid than electrolytes

- Hyper/hypokalemia - Heart and Smooth muscle

Hypokalemia

↳ Clinical manifestations: cardiac and smooth muscle, weak, respiratory paralysis, dysrhythmia that can lead to sudden cardiac death

Hyperkalemia

Clinical manifestations: cardiac dysrythmia, muscle weakness, fatigue and nausea.

- Hyper/hyponatremia: too little water per unit of Sodium. Too much water, too much ADH secreted

Hypo

Clinical manifestations: hungry, tired, nauseated, seizures, confusion, coma death, cells from brain absorb more water which is bad

Hyper

Clinical manifestations: cells in brain dry out, confused, lethargic, seizure, coma, death

ABG Interpretation: $PaCO_2$: 35-45 Bicarb: 22-26

Blood PH 7.35-7.45

Bicarb = metabolic CO_2 = respiratory

CO_2 High = acidosis CO_2 low = alkalosis

Bicarb high = alkalosis Bicarb low = acidosis

Ch 4: Pain

Pain Modulation - Gate Control theory, the body can open or close gates, stop transmissions to the brain, disrupting pathways

ex) Coldness of ice will be what your brain is thinking about instead of pain
- ice, pressure, shaking

Gate Control theory?

Acute Pain Characteristics: typically lasts less than 3 months

manifestations of Acute pain: elevated HR, Resp. rate and blood pressure, pallor, sweating, nausea, vomiting, pacing, grimacing, crying, moaning

Chronic Pain Characteristics: may be associated with disease process, lasts longer than expected healing time, > 6 months

ex) significant pain after a bone is supposed to be healed

manifestations of Chronic Pain: generally not associated with signs and symptoms of sympathetic activity, Psychological such as loss of job, irritability, poor self image, depression ← significant factor for individuals with Chronic pain

Ch 6: Epidemic VS. endemic disease

Endemic - continuous transmission in cutined population

Epidemic - rapidly spreading disease to large number of host in a given population in a short time

Ch 7: immunodeficiency characteristics and manifestations

Primary: inborn part of the immune system ex) reoccurent infections

Secondary: illness/medications ex) leukemia, AIDs, viral Hepatitis

opportunistic infections: happen from normal flora or something that doesn't normally cause infection

ex) someone has aids which lets another infection happen

Ch 8: Skin infections/alterations in Skin integrity

Skin lesions
- diagnosed by assessing appearence
- causes of skin lesions - systemic disorders, systemic infections, Allergies

Vitiligo - small areas of hypopigmentation that gradually enlarge overtime

Melasma - areas of darker skin

Skin inflammatory disorders

- Contact dermatitis - exposure to allergin or chemical

- Uticaria (Hives) - Type I hypersensitivity reactions most commonly from ingested shellfish, fruit, or drugs

- extensive atopic dermatitis - Atopic: Inherited tendency, common problem in infancy, rash with serous exudate
  - in adults it is dry scaly and puritic

- Psoriasis - chronic inflammatory disorder, beleived to be genetic, abnormal t-cell activation, proliferation of keratinocytes.
  - small scaly spots; dry skin

- Scleroderma - cause unknown, increased collagen deposit, inflammation and fibrosis
  - Hard, Shiny, tight, immovable skin

Skin infections: caused by

- bacteria, viruses, fungi, oppurtunistic microbes, cuts, or serous infections

bacterial

- Cellulitis - infection of dermis and subcutaneous tissue, secondary to injury
  - edema, pain, red streaks

- Furnicle - infected hair follicle

- Impetigo - S aureus, small red vesicles which rapidly enlarge, yellow brown, crusty masses

- Acute necrotizing Fasciitis - aerobic and anaerobic bacteria, severe inflammation and tissue necrosis

viral

- Herpes Simplex - cold sores and fever blisters - type 1
type 2 = genital Herpes
  - may be asymptomatic

-Plantar warts- HPV - spread by viral shedding of skin surface

Fungal infections

Tinea Corpis - round lesions with clear center (ring worm)

Scabies - invasion by mite

Pediculosis - lice in hair

Ch 9: Fracture Complications

Fractures initiate inflammatory response

Complications: reffered pain, Bone infection, nerve damage, blood vessel damage, pulmonary embolism ← blood clot in lungs

Compartment syndrome: swollen muscle compresses blood vessels and nerves

Ch 23:

Metabolic Syndrome: precipitated by insulin antagonists from adipose tissue

Three common factors

1. presence of significant abdominal fat
2. changes in glucose metabolism
3. changes in lipoprotein metabolism

- pt may have → CO alterations, Hypertension, Type 2 DM
  - obesity is primary cause
  - weight loss prevents complications

Anorexia nervosa
- extreme weightloss due to self starvation

Strong psychological component
- perfectionist, high acheiving individuals, Family Conflict, Altered body image, fear of fatness

- leads to malnutrition

- Anorexia presentation: Emaciation, Amenorrhea, Hypothermia, Cold intolerance, low BP, Low HR, Dry skin, Brittle nails, Lanugo

Bulimia Nervosa - Binge eating followed by purging, multiple times a day or less.
  - High Calorie

- Self induced Vomiting, laxatives, diuretics

Manifestations - Tooth erosion, oral ulcers, esopnagitis, electrolyte loss, cardiac arrythmias, tetany, severe abdominal pain, compulsive exercise, Normal weight but poor nutrients (Anemias), menstrual irregularities

Ch 28: Industrial Chemical irritants and pulmonary effects

Inhalants: Chronic cough, frequent infections, irritation, inflammation
Carcogenic - increases lung cancer especially w/ cigarette smoking

- iron oxide and Silica cause fibrosis → Chronic lung disease, chronic cough, frequent infections, irritation, and inflammation

- gasses - Sulfur dioxide → chronic inflammation. Carbon monoxide can displace oxygen which would endanger those with cardiovascular disease

-Asbestos - Severe acute inflammation and scarring that could lead to mesothelioma ↑ takes a while to diagnos Abestos disease

Ch 22: Pregnancy

Ectopic pregnancy: Fertilized egg implants outside uterus, most commonly in fallopian tube
- leads to spontaneous abortion or tubal rupture → rupture leads to hemorrhage or peritonitis
- medical emergency - removal of embryo and associated tube

Eclampsia: pregnancy induced hypertension (above 140/90)
- uncontrolled PIH results in preeclampsia and eventually eclampsia
- Higher BP and kidney dysfunction
- Further Complications of eclampsia → HELLP + DIC

Placenta previa - placenta implanted lower in uterus
- may cover cervix
- enlarging uterus and contractions cause tearing of placenta + bleeding
- painless bright red bleeding

Abruptio Placentae
- premature separation of placenta from the uterine wall
  - Bleeding (seen or not seen)
  - Bleeding will be dark and usually abdomen pain

Teratogens:
- Any substance or situation that causes a developmental abnormality
  - greatest impact during first trimester

Ch 21: Cogenital and Genetic disorders

Autosomal recessive: Both parents must pass on the defective gene to produce affected child
One parent recessive: child is carrier but has no clinical signs of disease

Example: cystic fibrosis, Sickle cell, phenylketonuria

Autosomal Dominant: presence of defect in only one alleles produces clinical expression
- 50% probability of passing on to child of either sex
- no carriers - unaffected do not transfer disorder - may not manifest signs until midlife

Example: Huntington disease

Chromosomal Disorders:
- Abnormal chromosome numbers occurring in any of the 23 pairs

Examples: Down Syndrome, Monosomy X (Turner Syndrome) 45 chromosomes, Polysomy X (Klinefelter syndrome) 47 chromosome

Ch 10: Vascular Disorders

Check on venous insufficiency and peripheral artery disease characteristics

Iron deficiency Anemia: Insufficient iron impairs hemoglobin synthesis
- Frequently sign of underlying problem
- Causes: on going loss of blood
- may need infusion, eat red meat, supplements

DIC: involves both excessive bleeding and clotting
- excessive clotting in circulation → thrombi and infarcts occur
- Clotting factors reduced to dangerous levels
- Widespread, uncontrollable hemorrhage results
- very poor prognosis, with high fatality rate

•complication of many primary problems

PVD: Damage to any veins or arteries outside of the heart
- this includes atherosclerosis
- toes become blue, hair changes
- reduced blood flow

Ch 12: Cardiovascular

Hypertension Etiology: Silent killer
Primary: idiopathic
Secondary: results from renal, endocrine → treatment required to resolve
Malignant or resistant: emergency, SBP, DBP elevated

Bradycardia - Slow HR
Tachycardia - Fast HR
Premature atrial Complex - atria beats early
Atrial flutter and Fibrilation - flutter barely any contraction of the atria
↳ the atria contracts twice before beat

Premature ventricle Complex - ventricle just randomly fire
Ventricular tachycardia -
Ventricular fibrillation
↳ twitching of ventricles
Asystole - No HR

Left Sided Heart Failure: Lungs - left ventricle cannot maintain adequate cardiac output to the body
- manifestations - paroxysmal Nocturnal dyspnea, pulmonary congestion, restlessness, confusion, orthopnea, tachycardia, exertional dyspnea, fatigue, cyanosis

Right Sided Heart Failure: right ventricle cannot handle venous return - pressure backs up into the venous system
- manifestations - fatigue, ↑ peripheral venous pressure, Ascites, enlarged liver and spleen, distended jugular veins, Anorexia + complaints of GI stress, weight gain, dependent edema

Ch 13: Respiratory Disorders

- ~~Chronic bronchitis pathophysiology - what is causing it~~
- COPD complications
- ~~emphysema pathophysiology and manifestations~~
- ~~Obstructive vs restrictive characteristics~~
- ~~open pneumothorax manifestations/mechanics~~

Obstructive characteristics - resisting flow → wall of lumen causing impingement
→ external pressure causing airflow resistance
→ airway lumen obstruction

ex) asthma, emphysema, chronic bronchitis
↳ chronic bronchitis

Restrictive Characteristics → Pulmonary → pneumonia
→ extra pulmonary → pneumothorax, Neuro

Emphysema: proteolytic enzymes from neutrophils and macrophages leading to alveolar damage
- smoking causes alveolar damage
  - inflammation leads to release of proteolytic enzymes
  - inactivates antitrypsin which usually protects lung parenchyma → air trapping → large thin walled cyst in lungs
    - impaired O_2 and CO_2 exchange

manifestations: progressive, exertional dyspnea, use of accessory muscles, pursed lip breathing, cough, digital clubbing, barrel chest, decreased breath sound, prolonged expiration, decreased heart sounds, hyperresonance, chronic morning cough

Chronic Bronchitis — causes: cigarette smoking, repeated airway infections, genetic predisposition, inhalation of physical or chemical irritants
- Chronic inflammation and swelling of the bronchial mucosa and potentiates airway obstruction, hypertrophy of bronchial mucosa

Pneumothorax - results form build up of air under pressure in pleural space
Air enters pleural space during inspiration → ipsilateral lung collapses → mediastinum toward contralateral (opposite side)
- decreased venous return and cardiac output

manifestations - tachycardia, decreased or absent breath sounds on affected side, Sudden chest pain, dyspnea, Hypotension, Neck vein distention, tracheal shift, subcutaneous edema, hyperresonance

Chronic obstructive pulmonary disease
Complications: pneumonia, ARDs, depression, heart failure, pulmonary hypertension

Ch 15: Eyes and Ears + 25

~~narrowed angle glaucoma~~
~~Hearing loss causes~~
~~macular degenerations~~
~~meniere syndrome~~

Macular degeneration - loss of central vision, lines are typically wave
Narrowed Angle glaucoma - iris is blocking fluid from leaving, emergency

Hearing loss causes
Conductive - obstruction of the ear canal
Sensorineural - inner ear cochlear damage
ex) Trauma, loud noises, ototoxic medications

Meniere Syndrome - accummulation of endolymph with degeneration of cochlea with tinitis

Ch 15: Endocrine System

Diabetes mellitus- basic problem is inadequate insulin effects in tissues
- results in abnormal carb, protein, and fat metabolism
- some tissues can transport glucose in the absence of insulin

General manifestations of Diabetes
- polypnagia- excess hungry - Fatigue - Hyperglycemia
- Glucosuria- glucose in urine - polyuria - polydipsia- excessive thirst

Hyperthyroidism- Autoimmune, Hyper metabolism, toxic goiter, exophthalmos
Grave disease
Hypothyroidism- replace thyroid hormone, Severe forms- Hashimoto thyroiditis, myxedema, cretinism
Hashimoto's disease
↓ tumors ↓ thyroid not functioning

Thyroid Storm Characteristics—probably a SATA question
1. hyperthermia 3. heart failure
2. tachycardia 4. delerium

Ch 20: Cancer

Benign- doesn't kill but may be life threatening depending on location, non-invasive, non metastatic, more closely resembles original tissue, slower growth, not necrotic
- "oma" indicates benign

Malignant Tumor- can kill host if untreated, invasive or metastasizing nature, does not resemble original tissue, grows rapidly, frequently necrotic, dysfunctional
- "carcinoma" indicates malignant

Characteristics of Malignant Cells
- ignore growth controlling signals
- ignores apoptosis, doesn't look like parent cells, mutates quickly

Tumor Staging and grading - to predict clinical behavior of malignant tumor and guide therapeutic managment
Grading- Histologic, Greater degree of anaplasia = greater degree of malignant potential
Staging- location and spread pattern, tumor size, local growth extent, lymph node, organ involvement, distant metastisis
Staging Continued- results of staging determine treatment protocol + prognosis
T- tumor size
N- nodes involved
M- metastasis

Ch 24: Complications of aging

Osteoporosis risk factors and reductions

-loss of calcium and bone mass
- Hereditary predisposition, decreased estrogen levels, decreased weight bearing activities, decreased intake of calcium, Vitamin C+D, decreased osteoblastic activity which is increased risk with glucocorticoid/ cortisol use

Reductions- increase calcium intake and vitamin D
- walking and other weight bearing exercise
- drugs such as bisphosphates, which inhibit bone resorption
- individualized hormonal therapies, including estrogen receptor modulations

Ch 26: Stress and Associated Problems

Stress Response: A generalized or systemic response to change
- Homeostasis
- Stressors

Ch 27: Addiction vs. physiologic dependence

Addiction- uncontrollable compulsion to use a substance; often with serious consequences

Physiologic dependence- body has adapted to the presence of the drug or chemical so that discontinuing results withdrawal signs

Ch 14: Nervous System Disorders

Stroke risk factors: occlusion of artery by atheroma, sudden obstruction of bolus in artery, intracerebral Hemorrhage- rupture of cerebral artery by hypertension

Brain Injury Mechanism

Primary- direct injuries, lacerations or crushing of the neurons, glial cells, and blood vessels of the brain

Secondary- additional effects of cerebral edema, hemorrhage, hematoma, infection — develops from ruptured blood vessels

Guillan Bar- inflammatory disease of peripheral nervous system, muscle weakness, paralysis, regression of symptoms of weakness

Multiple Sclerosis- chronic demyelinating disease of the CNS, immunologic abnormalities, genetics, often affects cranial nerves

Parkinson's- Dopamine deficiency → tremors, general lack of movement, loss of facial exspression, shuffling gait

ALS- progressive disease affecting both the upper and lower motor neurons, weakness and wasting of upper extremities followed by speech swallowing + breathing

Pathophysiology and manifestations of brain injuries

-direct injuries, such as lacerations, or crushing of the neurons, glial cells, and blood vessels of the brain Manifestations - seizures, CN impairment, rhinorrhea, otorrhagia, fever, stress ulcers

Pathophysioly and manifestations of Brain infections

-meningitis - microorganisms reach the Brain via blood by extension of near by tissue or by direct abscess through wounds manifestations -

Intracranial Hematomas - collection of blood in the skull usually by a bursted blood vessel

- results from contusions or shearing injuries and may develop several days after injury

Cellular Injury Failure

- unused oxygen and the fuel molecules within the cell leading to damage

Ch 18:

Nephrotic Syndrome - increased glomerular permeability to proteins

-proteinuria leads to hypoalbuminemia and generalized edema
Treatment: conservative symprom management - Lipid lowering agents, Steroids, Antihypertensives

Chronic Kidney Disease Treatment

- Medications, dialysis, or transplant may be needed

Renal Function + urine concentration

Urine concentration - provides specific gravity of urine kidney's ability to balance water and waste

Post infectious glomerulonephritis pathophysiology and manifestations

Pathophysiology - antistreptococcal antibodies, create antigen-antibody complex that lodges into glomerular cavities and causes inflammation response in both kidney's
manifestations - impetigo and throat infections

Acute tubular necrosis stages/progression (Acute Kidney Injury)

1. prodromal phase - injury has occured, Normal or ↓ UO, ↑ BUN and CR
2. Oliguric phase - oliguria / anuria, Hyperkalemia, Azotemia/uremia, metabolic acidosis
3. Postoliguric phase - Fluid Volume deficit, labs begin to normalize

Chronic Kidney Disease Stages

1. Kidney damage with normal or increased GFR - Dx/Rx of underlying condition and comorbities - GFR ≥ 90
2. Mild - estimate rate of progression - GFR 60-89
3. moderate - evaluate and treat complications - GFR 30-59
4. Severe - prepare for renal replacement therapy - GFR 15-29
5. Kidney failure - dialysis or transplantation if uremic - GFR < 15 or dialysis

Polycystic Kidney Disease Characteristics

Autosomal Recessive - evident at birth, kidneys enlarged, systemic hypertension

Autosomal Dominant - most common, symptoms later in life, renal tissue slowly reduced leading to HTN, pain, and dilute urine

Types of Urinary Incontinuence

Stress - due to increased abdominal pressure under stress
Urge - due to involuntary contraction of bladder muscles
overflow - due to blockage of urethra
Neurogenic - impaired function of nervous system

Ch 19:

Characteristics of STI's
STI's and complications - know which ones can be treatable and know the difference
~~Pelvic inflammatory disease - Pathophysiology + complications~~
~~Benign prostatic Hyperplasia~~

Benign Prostatic Hypertrophy

- enlarged gland palpated on digital rectal exam, leads to frequent infections
- continued obstruction causes distended bladder, dilated ureter, hydronephrosis, + renal failure if untreated
- drugs used to slow enlargement and increase urine flow
- surgery options based on obstruction

Pelvic Inflammatory Disease - may be acute or chronic

- infection of uterus, fallopian tubes, or ovaries
- Potential complications - peritonitis, pelvic abscesses, septic shock

Chlamydia - most common - Bacteria

Male - urethritis and epididymus - dysuria, itching, white discharge, painful-swollen scrotum, usually unilateral, fever; inguinal lymphnodes

Females - often asymptomatic, until PID or infertility develops
- newborns affected during birth

Gonorrhea - bacterial

- many strains have become resistant to penicillin and tetracycline
- Males - most common site is urethra, which is inflammed
- Females - Frequently asymptomatic
  - PID and infertility are serious complications

Genital Herpes - Viral

- recurrent outbreaks of blister like vesicles on genitalia
  - proceeded by tingling or itching sensation
  - lesions are extremely painful
- Antiviral drugs are used for treatment and prevention of transmission
- infection is considered lifelong

Trichomoniasis

- localized infection
- Men - usually asymptomic
- Women - may be subclinical
  - flares up when microbial balance in vagina shifts, causes intense itching
- Systemic treatment necessary for both partners