Final Exam Concept Guide

Know the Etiology, Signs/Symptoms, Diagnosis/Diagnostics, Clinical Manifestation, Risks, Treatment and Complications for the following:

- **Gastritis**

  Gastritis – inflammation of the stomach lining
  Acute Gastritis – (just acquired) ingestion of toxins, alcohol, aspirin or other irritating substances
  Chronic- 2 months to become chronic
  Triggers of Gastritis: Alcohol, caffeine, autoimmune disease, viral or bacteria
  Chronic Gastritis: **H Pylori** is always a factor
  H Pylori goes very deep in the lining of the stomach and It causes persistent inflammation
  S/S: N/V – Anorexia- postcranial discomfort
  Post Cranial Discomfort- after eating- goes away and come back 1-2 hrs
  Gastritis- hematemesis- blood in the vomit- coffee brown color
  Treatment: Treat H pylori treat GERD, change lifestyle, PPI

- **Peptic Ulcer Disease**

  Inflammation and ulceration in the stomach (acid and pepsin)
  Gastric: stomach location
  Duodenal: duodenal location
  PUD is a complication of Gastritis
  PUD is caused by **aspirin, H pylori, Nsaids, Stress, Smoking**
  S/S Gastric N/V Anorexia Chest discomfort, asymptotic, Dyspepsia
  Duodenal – normal weight
  Biggest complication of PUD- GI bleeding due to Ulcer perforation- hole in the lining and bleed
  It is life-threatening if it keep bleeding (Anemic, electrolytes imbalance (losing volume)
  Duodenal – Blood in the stool – black and tarry
  Bleeding profusely-frank with cloth
  Hematemesis- Bleeding in vomiting
  Treatment: Cortery of perforation, treatment of H. pylori, PPI, Cessation of smoking

- **Ulcerative Colitis and Crohn's the difference in the complications**

  Complication in UC Malnutrition – dehydration, increased risk factor of colon cancer
  7-10 yrs, rarely in megacolon

  Complication of Chron- Fistulas, perianal fissures, abscesses. The risk of colorectal cancer

- **Bowel Obstruction Manifestations**

  Obstructions in the jejunal area: Vomiting, dehydration, electrolyte depletion
  Obstructions of the distal portion of the small bowl or ileum, dehydration to hypovolemic schock
  Obstructions of the colon: Massive gas distention
  Blockage of the colon by a tumor is the most common cause of colonic obstruction and perforation of the bowel wall adjacent to the tumor.

- **What percentage of the pancreas is dedicated to endocrine functions?**

  Only 5%

- **Pancreatic Cancer**

  Pancreatic Cancer – **2%** of all cancers
  Ranked 4th among death in all malignancies
  Risk Factors; cigarette smoking, obesity
  S/S; head: Jaundice, malabsorption, weight loss tail: Abd pain, nausea'

- **Hepatic Encephalopathy is due to?**

  Hepatic encephalopathy is a decline in brain function due to severe liver disease

Hepatic encephalopathy is usually precipitated by certain well-defined clinical developments, including hypokalemia, hyponatremia, alkalosis, hypoxia, hypercarbia, infection, use of sedatives, GI hemorrhage, protein meal gorging, renal failure, and constipation. In some patients, progressive liver failure leads to chronic encephalopathy without other exacerbating factors.

Hepatic encephalopathy is graded 1 to 4:

- Grade 1: Confusion, subtle behavioral changes, no flap
- Grade 2: Drowsy, clear behavioral changes, flap present
- Grade 3: Stuporous but can follow commands, marked confusion, slurred speech, flap present
- Grade 4: Coma, no flap

- **Gastroesophageal Varices Management**
  - Initial treatment: Fluid resuscitation to stop bleeding
  - Large bore intravenous lines are placed
  - Admin of parenteral vitamin K and plasma, platelet infusion if thrombocytopenia is present
  - Octreotide acetate (synthetic analog) no more vasopressin 3-5 days
  - Metoclopramide and B blockers
  - Esophagogastroduodenoscopy EGD to determine site of bleeding
- **Difference between Diverticulosis and Diverticulitis**

Diverticulosis (diverticular disease) presence of diverticula in the colon.
Diverticula are acquired herniations of the mucosa and submucosa through the muscular coat of the colon

Diverticulosis The presence of one or more diverticula vs diverticulitis inflammation of one or more diverticula

- **Kidney Disease- Assessment-CVA**

Pain associated with intrarenal disorders are assessed by palpating or light percussion over the costovertebral angle (CVA) posteriorly and is recorded as CVA tenderness. Pain is transmitted to the spinal cord between T10 and L1

- **Kidney Cancer signs and symptoms**

Benign renal neoplasm: S/S Hematuria and flank pain
Some may be asymptomatic until large

Renal cell carcinoma: Metastatic disease
Risk factors: smoking, obesity and hypertension
S/S CVA tenderness, hematuria, palpable mass

- **Dialysis- Benefits and Risks**
- Filter blood and rid the waist

Dialysis is the only therapeutic option for those with ESRD unable to obtain transplant
Each treatment of dialysis remove about 2/3 of the total body urea content
Dialysis maintain volume status
Prevent and treat acid-base and electrolyte disturbances
Prevent and treat uremia
Support nutritional needs.
Prevent and treat infection
Orevent and treat anemia
Improve quality of life
Lower mortalty and morbidity rates
Control pains

VS
RISKS
Electrolytes imbalance (potassium and sodium) -need to check hyper or hypo

Low blood pressure (have to check vitals before and after)
Risk for infection

Long-term mobidity
Life threatening
Cardiovascular disease remained the most common cause of death in ESRD patient

- **Acute Kidney Injury- Three phases- Prodromal- Oliguric- Postoliguric**

Prodomal Phase
- Normal or declining urine output
- Serum blood urea nitrogen (BUN) and creatine levels rise

Oliguric phase (10-14 days) range from 1 day to 8 weeks
- Oliguria – Small abnormal amount of urine
- Edema, Hypertension, Hypervolemia
- Distended neck veins, weight gain
- Crackles, possibly heart faily
- WBC and RBC in urine
- Protein in urine
- Hyperkalemka
- Volume overload
- Azotemia and uremia
- Metabolic acidosis

Postoliguric phase (5% recover)
- Fluid volume deficit
- Labs begin to normalize
- Polyuria – sodium waste

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- **Acute Tubular Necrosis and the causes of Acute Tubular Necrosis**

ATN is the result of tubular cell injury
- Ischemia: Exposure to nephrotoxic substances
- ½ of all cases of AKI

Nephrotoxins leading cause of ATN (aminoaglycosides, NSAIDS, amphotericin B, cisplatin, tetracycline.
CI- AKI or Contrast-induced nephropathy

- **Cystitis- signs and symptoms**

(inflammation of the bladder) no fever or flank pain If fever is present – Might be infection (UTI)

Confusion
Cloudy urine
Frequency
Urgency
Dysuria

Intrarenal Kidney Injury- Toxic causes of

Box 28.1

Types of Acute Kidney Injury

Prerenal

- • Absolute decrease in circulating volume
  - • Hemorrhage
  - • Dehydration
  - • Burns
- • Relative decrease in circulating volume

- Distributive shock (neurogenic, anaphylactic, septic)
- Third-spacing and edema
- Decreased cardiac output
    - Cardiogenic shock
    - Dysrhythmias
    - Cardiac tamponade
    - Heart failure
    - Myocardial infarction

- Primary renal hemodynamic abnormalities
    - Occlusion or stenosis of renal artery*
    - Drug-induced impairment of renal autoregulation in susceptible persons‡

Postrenal

- Benign prostatic hyperplasia
- Kinked or obstructed catheters
- Intraabdominal tumors
- Strictures
- Calculi

Intrarenal/Intrinsic

- Tubular (acute tubular necrosis)
    - Ischemic
        - Prolonged prerenal failure
        - Transfusion reactions
        - Rhabdomyolysis
    - Nephrotoxic
        - Prolonged postrenal failure
        - Certain antimicrobials (antibiotics; antifungal and antiviral drugs)
        - Radiographic contrast media
        - Certain cytotoxic chemotherapy agents
        - Recreational drugs (amphetamines, heroin)
        - Environmental agents (heavy metals, carbon tetrachloride, insecticides)
        - Snake and insect venom
- Glomerular
    - Acute glomerulonephritis
- Interstitial
    - Acute allergic interstitial nephritis
    - Acute pyelonephritis
- Vascular
    - Vasculitis
    - Emboli
    - Nephrosclerosis (due to primary hypertension, hypertensive emergencies, and urgency)

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- **Two electrolytes that are affected by the kidney's inability to regulate.**

  Potassium and Sodium
  Hypokalemia and Hyponatremia
- **Renin-Angiotensin-Aldosterone System and the relationship between the autoregulation of the kidneys**

  Renin-Angiotensin-Aldosterone System (RAAS) act to increase the total circulating volume in the kidney attempt to autoregulate perfusion and maintain GFR.

- **What is the Glomerelus?**
- It is a segment of nephron that filters fluid from blood into Bowman capsule, prevents passage of blood cells and proteins.
- **What is Glomerulonephritis?**
- An assortment of immune-mediated conditions that produce inflammation of the glomeruli.

- **Diabetes Type I and Type II**

Type 1: Destruction of the B cells of the pancreas results in absolute insulin deficiency
5-20 yrs
Chromosone 6

Type 2: Most common form of DM – Insulin resistance and B cells dysfunction lead to a resistance lack of insulin
Risk factor: Female sex, obesity, and sedentary lifestyle

Risk factor: non Caucasian, elderly

- **Poly's of Diabetes**
- 3 P : Polyuria, Polyphagia, Polydipsia

- **Cushing's Syndrome characterized by**

Cushing Syndrome	**Adrenocortical Insufficiency**
Truncal obesity Moon face Dorsocervical fat pad Hirsutism Muscle wasting Striae Petechiae Glucose intolerance Hypertension Hypokalemia	Weakness Hypotension Hypoglycemia Hyperpigmentation (Addison disease) Hyperkalemia Weight loss

- **Traumatic Brain Injury**
  - Traumatic brain injury (TBI) refers to injuries of brain tissues sustained as a consequence of trauma
  - Traumatic brain injury == head injury
  - Injuries to the cranials ≠ not always brain injury
  - **TBI is the leading cause of death and disability in the U.S. 50,000 + yr**
  - Causes TBI (falls, sports injuries, firearms, and transportation-related trauma)
  - Fall majority for 0-4 or 65+, motor vehicle TBI – 14-24 yr. Men are more likely to die than women
  - Types of TBI:
    - Severity
    - Location
    - Mechanisms of injury

- Severity of TBI is classified by Glasgow coma scale (CGS) (standardized tool developed for the purpose assessing the LOC in acutely brain-injured patients. To evaluate patient with an altered LO
- Glasgow coma scale: Mild score (13-15), Moderate (9-12), severe (8 – below)
- Primary injury- initial trauma injury on brain cells
  - Focal injuries (coup)
  - Polar injuries (coup contrecoup)
  - Diffuse injury

In addition, as location, Primary Injury can also differentiated by Mechanism of injury

- Concussion
- Contusion
- Intracranial hematoma.

Concussion also known as ***Mild traumatic brain injury*** (MTBI) most common for military and athletes. Alterations or LOC≤30 mn but no evidence of brain damage on CT.
Symptoms: Headache, n/v, dizziness, fatigue, blurred vision, cognitive, emotional disturbances
Contusion CT or MRI reveals area of Brain Tissue damage (necrosis, laceration, bruising)
Intracranial hematoma: Localized collection of blood within the cranium.
Three types; Epidural, subdural, subachnoid.
Secondary Injury : Body's response to initial injury may cause more harm than the initial injury.
Can cause: Ischemia, hypoxic events, vasogenic/neurogenic edema, and other processes that leads to brain swelling and increased ICP.
Treatment: Cardiopulmonary stabilization. Radiologic screening, maintenance of normal body temperature or mild hypothermia, normal PaCO2, normal glucose level and noema intravascular volume recommended. Acutely elevated ICP, mannitol(osmotic diuretic) sedation, hypothermia and mild hyperventilation.

- **Reperfusion Injury- Definition**

Reperfusion injury is the secondary injury that occurs after reestablishing blood flow

- **Increased Cranial Pressure- ICP**
- ICP is the pressure exerted by the contents of the cranium and normal ranges from 0-15 mmhg

The volume of the cranium nerve is made up 3 parts: brain tissue, cerebrospinal, blood
Monroe-kellie hypothesis – the relationship between the 3 components of cranium
Compliance – the ability to accommodate changes in volume w/o sign. Increase in pressure
The most common causes of ICP: stroke, trauma and tumors

- **Most sensitive indicator of altered brain function?**

A change in Level of consciousness LOC is one of the most sensitive indicators of altered brain function

- **Assessment of the brainstem function- How do you assess it?**

Pupil light reflex
Oculovestibular reflex
Corneal reflex

- **CVA- Types and causes**

Transient ischemic attack (TIA) – Episodes of stroke s/s with a duration of less than 24 hr and less than1hr (30% pt with stroke have prior TIA)
Ischemic stroke- Sudden occlusion of cerebral artery secondary to thrombus formation/embolization

- Thrombotic strokes: atherosclerosis and hypercoagulable states
- Embolic strokes: cardiac sources

Hemorrhagic stroke = Hemorrage within the brain parenchyma and after a severe and long-standing
Hypertension. (30 % mortality rate)

- **Test used to diagnosed CVA**

  Non-contrast CT scan /diffusion weighted brain MRI
- **Meningitis what is it?**

  Meningitis is the pyogenic infection that invades the leptomeninges and the subarachnoid space.

  the most comment sequela to microbial invasion of the CNS. (most bacterial but can be viral/fungal)

  Bacteria: Streptoccocus pneumoniae

- **Encephalitis What is it?**

  Encephalitis is the inflammation of the brain caused by viruses, bacteria, fungi and parasites

- **Seizures- how they are classified?**

1. Generalized seizures: Entire brain Surface
   - Absence (petit mal)
   - Atypical absence
   - Myoclonic
   - Atonic (drop attack)
   - Clonic
   - Tonic
   - Generalized tonic-clonic (grand mal)

2. Partial Seizure: Part of the brain surface

   - Simple partial: There is no impairment of consciousness during the seizure.
   - Complex partial: There is impairment of consciousness during the seizure.
   - With secondary generalization: Onset begins as simple partial and then progresses to impairment of consciousness.

- **Status Epilepticus what is it?**

  Status Epilepticus is a continuing series of seizures without a period of recovery between episodes
- **Dementia**

  Progressice deterioration and continuing decline of memory and other cognitive changes
  60 % to 80 % of Alzheimer are dementia- Vascular dementia is the most common cause

  No cure
  Cause is unknown

  Dementia-causing illness: - Alcoholism
  - Alcoholism
  - Intracranial tumor
  - Normal-pressure hydrocephalus
  - Parkinson disease
  - Lewy body disease

- Huntington disease
- Multiple sclerosis MS
- Bocine Spongiform encephalopathy (Mad Cow)

Delerium and depression in elederly different than dementia

Delirium in global mental dysfunction – Acute Confusional state

Primary risk factor: age and family
Others: Head trauma, diabetes, depression, stroke, hypertension diabetes

Signs and Symptoms memory lost, anxiety and agitation, diffulty in judgement, problem solving and communication. Assistance needed for ADL . Difficulty eating and swallowing. Weight loss

- **Parkinson disease**

  Disorder of mobility - 1 million americans affected. 60,000 cases each year
  4 % are younger than 50 Y/O

  idiopatic or acquired

  Common causes: Infection, intoxication, and trauma
  Drug Toxicity: Phenotiazine class: (Chlorpromazine, prochlorperazine, Thioridazine) and Butyrophenone(haloperidol)
  Parkinson disease results from degeneration of the pigmented dopaminergic neurons found in the subtantia nigra and to a lesser extent neurons elsewhere in the brain.

  Lewy bodies are cytoplasmic inclusions are found in the surviving neurons.

- **GERD signs and symptoms**

  Heartburn
  Chest pain
  Regurgitation
  Epigastric pain
  Dry cough
  Hoarseness in the morning
  Esapheogeal strictures with Gerd

- **Multiple Sclerosis- Structures effected by the demyelination?**

  Chronic demyelinating disease of the CNS – sign disability in young adults
  Autoimmune disorder that results in inflammation and scarring of the myelin sheaths covering nerves.
  Age onset (20-50 yr)
  2 -3x more common in women vs men

  In MS, the demyelination of nerves can happen anwhere in the CNS. Structures most frequently affected are the optic nerves, the oculomotor nerves, the corticospinal, cerebellar, and posterior column system.
  Myelin facilitates nerve conduction. So the inflammation with MS slow or interrupt the conduction of nerve impulses.

- **Spinal Cord Injuries- major mechanisms of injury?**

  A problem of the young
  Male 3-4x risk – weekends/summer months
  Motor vehicle crashes- highest number of sci 2) violent gun shot wounds, falls, recreational accident

  The major Mechanisms of injury are hyperflexion, hyperextension and compression

Flexion injury with tearing of the posterior ligaments and dislocation is the most unstable injury and is aften associated with severe neurologic deficits
Hyperextension injury is the most common

S/S complete loss of function below the level of injury , Spinal shock (a few hrs) Flaccid paralysis of all skeletal muscles. Loss of all spinal reflexes, loss of pain, proprioception and other sensations, bowel and bladder dysfunction with paralytic ileus, loss of thermoregulation. Bradycardia, hypotension

- **How to treat Spinal Cord Injuries**

Stabilization of the spinal vertebra (to prevent further trauma to spinal cord)
Sugery – internal fixation or external fixation and bracing
Surgery within 24 hrs if cord compression
Use od high-dose methylprednisolone (1st 8 hrs) (controversial method)
Removing or alleviating the painful stimuli
Use od adrenergic receptor-blocking medication to manage hypertensive crisis
Prevent respiratory and urinary tract infections
Skin pressure sores
Septicemia and fecal impaction

- Hypoventilation build up what?

Hypoventilation causes build up of acid (acidosis) and too little oxygen in the blood.
Hypoventilation occurs when delivery of air to the alveoli is insufficient to meet the need to provide O2 and remove CO2.

- Decrease rate and depth of respiration
- Results in increased PaCO2 ≥45 mm Hg
- Hypoxemia – increased alveolar CO2

- Causes: drugs (morphines, barbiturates) , obesity (Pickwickian syndrome, myasthenia gravis, obstructive sleep apnea, chest wall damage.

- Normal ABG's- Only must know normal values

pH	7.35-7.45
$PaCO_2$	35-45 mm Hg
PaO_2	80-100%
HCO_3	22-26 mEq/L
SaO_2	95-100%

- Acute Respiratory Failure- the goal of treatment

Maintaining ventilatory support by maintaining airway patency and ensuring adequate alveolar is the primary goal of therapy.
Mechanical ventilation
Management of the underlying cause.

- Virchow's Triad of PE

Virchow (1800 Pathologist) 3 Factors that predisposed patient to thrombus formation an increase of PE (Pulmonary Embolism)

- Venous Stasis (sluggish blood flow
- Hypercoagulability
- Damage to the venous wall (intimal injury)

Box 21.4

Factors Predisposing to Pulmonary Embolism of Virchow's Triad

Venous Stasis

- Extended bed rest (delayed venous removal of activated clotting factors)
- Postoperative state
- Immobility (activated clotting factors)
- Vascular disorders (thrombophlebitis of lower extremities and pelvic area)
- Congestive heart failure (venous backflow/stasis)
- Cardiac dysrhythmias (atrial fibrillation)
- Dehydration
- Prolonged air travel
- Obesity

Hypercoagulability

- Oral contraceptives (estrogen therapy), hormone replacement therapy
- Pregnancy, early puerperium
- Polycythemia (chronic high altitude; chronic pulmonary disease with decreased PaO_2 and increased $PaCO_2$)
- Malignant pathologic processes, visceral cancer
- Cigarette smoking
- Inherited resistance to activated protein C
- Deficiency of protein S
- Deficiency of antithrombin III
- Prothrombin gene mutation
- Presence of antiphospholipid antibodies (lupus), anticoagulant and anticardiolipin antibodies

Damage to Vessel Wall (Intimal Injury)

- Blunt trauma
- Penetrating wounds
- Bone fractures with soft tissue injury
- Surgical procedures (hip, pelvic, abdominal, cardiovascular)
- Obstetric manipulations during labor and delivery
- Burns
- Central venous catheter

- Embolisms the different types
- 7 Types of embolisms:
- -Thrombotic
- -Fat
- -Amniotic Fluid
- -Air
- -Tumor
- Foreign material
- Septic
- Parasite

Embolism Type	Cause
Thrombotic	Blood clots develop in venous system, predominantly in thighs and legs
Fat	Globules of fat secondary to fractures of pelvis or long bones
Amniotic fluid	Collections of fluid, hair, or other debris related to complicated labor, especially in olde multiparous women
Air	Venous access through IV catheters
Tumor	Fragments from malignant tissue
Foreign material	Foreign bodies (bullets, sutures, catheter tips, orally prepared medications injected IV)
Septic	Infected tissue or related substances (fungal/bacterial)
Parasitic	Parasites present in lung vasculature

- **Asthma- definition**

Asthma is complex lung disease and associated with the release of inflammatory chemicals from mast cells in the airways.
Asthma is a lung disease characterized by:

- airway obstruction that is reversible (to some patients)
- airway inflammation
- increased airway reactivity to a variety of stimuli

Mast cell release are IgE (immunoglobin E) mediated for both extrinsic and intrinsic asthma.

Extrinsic Asthma: Allergies, family hx of the disease. Positive reaction to environmental triggers.
Intrinsic Asthma: Respiratory Tract infections and psychological factors(harder to treat)

- Exercise-Induced Asthma is due to?

Exercise-induced asthma is due to Running, jogging, bicycling.
Bronchospasm occurs within 3 min after exercise and usually resolve in 60 minute.

- Acute and Chronic Bronchitis

Acute Bronchitis is an acute inflammation of the trachea and bronchi .
80% of the 12 mil cases per yr in UD.

- Influenza Virus A or B
- Parainfluenza Virus
- respiratory syncytial virus
- Coronavirus
- Rhinovirus
- Coxsackie virus
- Adenovirus
- Streptococcus pneumoniae
- Hemophilus influenza
- Mycoplasma
- Moraxella
- Chlamydia pneumoniae

(heat, smoke inhalation, inhalation of irritant chemicals)
Risk factors: smokers, young children, elderly -winter months)
Pathogenesis: The airways become inflamed and narrowed from capillary dilation, swelling from exudation of fluid, infiltration with inflammatory cells.
S/S mild and self-limiting: - low-grade fever

- substernal chest discomfort
- sore throat
- postnasal drip
- fatigue

Associated inflammation of the larynx and trachea produces croup syndrome

Diagnosis: Clinical presentation- onset of the cough.

Treatment: Antibiotic therapy for bacterial infection
Viral infection resolved spontaneously in most normal healthy person
Primary goal is symptom management with antitussive agents – beta-2 agonists and bronchodilatoes
Codeine-containing medication are helpful
Non-pharmacologic: increase fluid intake, avoid smoke, use vaporizer in bedroom

Chronic Bronchitis:

Major causes of chronic bronchitis are:

- Cigarette smoking (90 %)
- Repeated airway infections
- Genetic predisposition
- Inhalation of physical or chemical irritants

Chronic Bronchitis: Type B COPD
Diagnosed: Symptomatically by hypersecretion of bronchial mucus and a chronic or recurrent productive cough of more than 3 months duration and occurring each year for 2 or more successice yes in patient.
Airway obstruction is persistent and irreversible in patients with chronic brochitis and emphysema.
3:1 ratio of annual cases of chronic bronchitis to emphysema.

Pathogenesis: Chronic inflammation and swelling of the bronchial mucosa resulting in scarring, increased fibrosis of the mucous membrane, hyperplasia of bronchial mucous glands and globet cells, hypertrophy of bronchial wall thickness which cause (potentiates) obstruction to airways.

Neutrophil activity cause inflammation.
Interleukin-8 levels are elevated. CD8 T-lymphocyte levels are elevated.
Two bacteria is involved with chronic bronchitis: H. influenza and S.Pneumoniae

The chronic bronchitis patient appear as the blue bloater: Oxygen desaturation (cyanosis) and edmea associated with right-sided heart failure.

S/S of Chronic Bronchitis:

- Overweight (1:2 male to female ratio)
- 30s and 40s
- SOB (shortness of breath) on exertion
- Excessive amounts of sputum (mixture of saliva and mucus) most severe in the am
- Chronic cough
- Excess of body fluids(edema, pypervolemia)
- Hx of smoking
- Complaints of chills
- Malaise
- Muscle aches
- Fatigue
- Loss of libido
- Insomnia

End-stage of Chronic Bronchitis, Patient has

- Right-side heart failure
- Distended neck veins
- Right ventricular heave
- Right ventricular gallop
- Peripheral edema
- Hypoxia- pulmonary hypertension
- Cyanosis

Diagnosis: Chest radiography, pulmonary functions tests, Elevated $PaCO_2$ and decreased PaO_2 (below 65 mmhg)

Severe Chronic Bronchitis: Physical Exam: Scattered crackles, rhonchi, wheezes, jugular vein distension, clubbing, pedal and ankle edema

Treatment: Because Bronchitis and Emphysema has similar therapy

1- Block the progression of the disease
2- Return the patient to optimal respiratory function
3- Return the patient to usual activities of daily living

Inhaled B-2 agonists
Inhaled anticholinergic brichodilatoes
Cough suppressants

Antimicrobial agents for infection
Inhaled or oral cortisosteroids for acute exacerbations
Low-dose of Oxygen therapy for patient with Pao2 less than 55 mmhg
Mechanical ventilation
Home oxygen therapy
Smoking cessation

Emphysema 1

Type A COPD – destructive changes of the alveolar walls and abnormal enlargement of the distal air sacs.
Causes: smoking, air pollution, occupations (Welding, mining, working with asbestos), ∞-1 antitrypsin deficiency (1%).
Develop over a long period
Person over 50
Cigarette smoking in excess of 70 pack -years

Pathogenesis: Pathologic changes leading to alveolar destruction are associated with the released of proteolytic enxymes from inflammatory cells such as neutrophils and macrophages.

How does smoking causes alveolar damages? 2 ways:

- Leads to inflammation in the lung tissue (parenchyma)
- It inactivates ∞-1 antitrypsin which would usually protect the lunc parenchyma.

S/S of patients with emphysema

- Exertional dyspnea
- Thin person over 55 years
- Increased SOB for past 3+4 yrs
- Increase with women who smoke
- Use of accessory muscles to breathe
- Progressive dyspnea
- Pursed-lip breathing
- Minimal Cough
- Barrel chest -Overinflation – increase lung volume
- Digital clubbing
-

At RISK: Pneumothorax, chest pain on the affected side, dyspnea
Late: Major symptom is dyspnea on exertion. Pink puffers

Treatment the same as chronic Bronchitis Cessation of smokins

- Chronic Obstructive Pulmonary Disease

Acute Respiratory Distress Syndrome (ARDS)

ARDS is characterized by damage to the alveolar-capillary membrane
Mortality rates ranges from 30% To 50 %
Decline in the PaO2 (does not respond to O2 therapy)
The common denominator appears to be increased permeability of the pulmonary vasculature

Flooding of the alveoli with proteinaceous fluid

S/S - increase in pulse rate
- Dyspnea
- Low PaO2
- Shallow, rapid breathing

Progression - - - Tachycardia
- Tachypnea
- Hypotension
- Marked restlessness
- Decreased mental status
- Production of frothy secretions
- Phys Assessment: crackles and rhonchi
- Using accessory muscles to breath

Late - Cyanosis

Diagnosis- Hallmark is hypoxemia
- Hypotension
- Decreased UO (urine output)
- Respiratory and metabolic acidosis
- Cardiopulmonary arrest
- Arterial blood gas hypercapnia
- Chest radiography
- Pulmonary function test

Treatment: Addressing the underlying cause (sepsis)

Maintaining fluid and electrolyte balance

Volume ventilator

PEEP (positive end-expiratory pressure)

▪ Tension Pneumothorax- treatment

Tension pneumothorax is medical emergency
Results from the buildup of air under pressure in the pleural space
Severe tachycardia
Hypotension
Tracheal shift to the contralateral disease
Neck vein distension
Hyperresonance
Subcutaneous emphysema

Treatment;
15-25 % : treated symptomatically, monitor closely, resolution within several weeks.

Over 15 – 25% Chest tube placed with water seal and suction
100 % oxygen
Chemical pleurodesis
Thoracotomy

Pneumonia

Pneumonia (breath)
An inflammatory reaction in the alveoli and interstitium of the lung usually caused by an infectious agent

Pneumonia can result from 3 sources

- Aspiration of oropharyngeal secretions of bacterial flora and or gastric contents (20-35 % of all pneumonia)
- Inhalation of contaminants (virus, mycoplasma)
- Contamination from the systemic circulation

Classifications of Pneumonia : Community acquired/hospital acquired
15-20 % with pneumonia requires hospitalization

Pneumonia classified: bacterial
Atypical
Viral

Pneumonia : Gram positive
Gram negative

Gram positive: Staphylococcus and streptococcus
Gram negative: E coli, Hearmophilus influenza, kleb,

Risk: Elderly , diminisged gag reflex, seriously ill., hospiralized, hypoxix immunocompromised

TB- transmission

Transmission: It is a airbone disease

- **inhalation of small droplets containing bacteria**
  TB baccillus
- **Droplets expelled with cough, sneeze, or talking**
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- **Bacteria** Mycobacterium tuberculosis – **acid fast aerobic bacillus**
- Most common sites are the lungs and the lymph nodes
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Sources of Electrolyte Stores

Every fluid compartment contains electrolytes- Electrolytes compositions differs in each compartments.
The concentration of K, Mg, Ca is higher inside cells than fluids outsise of cells.
Total calcim ions content concentration is higher inside cells, intracellular calcium is bound to other molecules.
The concentration of active ionized Ca ions is higher in the extracellular fluids.
The cells and bones are called electrolyte pools as they serve as important reservoir of Ca, K, Mg.

Distribution of electrolytes between the extracellular fluid / the electrolyte polls is influenced by hormones (epinephrine – K ions) (Isulin – K and Ph ions) and PTH (calcium ions)

PG 531 Electrolyte Distribution

- Anemia

Anemia is a deficit of red cell

  - _- Low oxygen-carrying capacity leads to hypoxia.

- Opposite of anemia: polycythemia
  - Excess of red cells
  - Increases blood viscosity and volume
- Relative anemia—normal total red cell mass with disturbances in regulation of plasma volume
- Absolute anemia—actual decrease in numbers of red cells
  - Decreased production
  - Increased destruction

- Hormone that Kidney's secrete
- • The kidney secretes two important endocrine hormones: erythropoietin, a growth factor for red blood cells (RBCs), and active vitamin D, a necessary cofactor for calcium absorption from the intestine.
- • In chronic kidney disease, impaired production of these hormones results in anemia and osteodystrophy.

- Definition of Polycythemia

Red cells are present in excess increasing blood viscosity which turns cause hypertension.
3 Types of polycythemia: - Polycythemia vera- neoplastic transform bones marrow

- Secondary Polycythemia – chrnic hypoxemia (over production of erythropoietin)
- Relative polycythemia

- Thrombocytopenia is caused by
  - Decreased platelet production
    - Folate/b12 deficiency
    - Radiation therapy
    - Chemotherapy
    - Drugs (ETOH, Thiazides, phenytoin)
    - Aplastic anemia
    - Cancer in bone narrow
  - Decreased platelet survival
    - Drugs (Thiazides, digoxin, heparin, furosemide, certain antibiotics
    - Mechanical prosthetic heart valves
    - Viral and bacterial infections
    - Circulating immune complexes
    - Increased destruction in the spleen
    - Disseminated intravascular coagulation
  - Splenic Sequestration (pooling)
    - Splenomegaly
    - Hypothermia
  - Platelet dilution
    - Massive transfusion with blood stored more than 24 hrs

Thronmocytopenia is caused by Generalized bleeding

- Disseminated Intravascular Coagulation (DIC)- What is it? And How to treat it?

Disseminated intravascular coagulation (DIC) is a acquired hemorrhagic syndrome in which both clotting and bleeding occur simultaneously.

Treatment: removal or correction of the underlying cause and support of major organ.

- Replacement of depleted clotting factors with fresh frozen plasma
- Packed red blood cells, platelets
- Cryoprecipitate
- Antifibrinolytics (aminocaproic acid) may be used if life-theating hemorrhage
- Heparin therapy to stop thrombin formation
-

- Chronic Myeloid Leukemia (CML) - diagnosis is by identifying which chromosome abnormality?

Chronic Myeloid Leukemia represents 15 % of all cases od leukemia in US
Onset 40-50 y
Malignat granulocyrs that carry a unique chromosomal abnormality (The Philadelphia chromosome Ph-)
The Philadelphia chromosome is fomed because of the balanced translocation between chromosome 9 and 22.
The overall survival is poor.. 2 years
S/S fatigue, weight loss, diaphoresis, bleeding, abdomral discomfort from enlarged spleen.

- Chronic Myeloid Leukemia (CML)- Treatment prognosis

CML does not respond well to chemotherapy
Temporary remission
Poor survival
2 years survival time if untreated
Anti bcr/abl therapy m(imatinib) to reduce the number of leukemic cells ‘
Patient might develop resistance to drugs

- ONLY CURATIVE TREATMENT is allogenic bone marrow transplantation with a suitable donor
- 25 % mortality with transplant (related donor)with 50 %-60 % disease-free survival
- 50 % mortality with transplant-unrelated
- Once CML which the blast phase – survival is 3-4 months

- GERD

See other study guide

- Initial symptoms of Malignancies
- Hodgkin Disease

30% of malignant lymphoma
Onset 20-40 yrs.. across life span
Males and worst prognosis
Overall 5 yrs survival rate for all stage treated : 85 %

It is a malignant disorder of the lymph nodes
Characterized by Reed-stemberg cells, Eipstein-barr virus
Malignant but grow/spread in predictable way; sets HD apart from other lymphomas
Usually metastasizes along contiguous lymphatic pathways
Usually present in single node or localized node chaint.
There are 2 typed

- Rare lymphocyte Type (5%)
- Classical type cHD (95%)

S/S Early signs are often asymptomatic
Painless lymphadenopathy
Fever
Night sweats
Pruritus

Weight loss
Malaise
-Cerical nodes
-lymp nodes enlargements above diaphragm
Iguinal nodes
-Disease spread from site of origin to other lymph nodes (spleen and bone narrow)
Prognosis and treatment
Ann Arbor staging also used for NHL
Stages of HD
A: Absence of clinical symptoms
B: Symptoms present at time of staging
(Loss of 10 % or more body weight, Unexplained fever, night sweats)
CS: Hx. PA, noninvasive procedures (computed tomography (CT scan)
PS: Invasive procedure such as laparotomy and tissue biopsy

(Non-Hodgkin) B-call, T-call and NK-Cell lymphoma

- **Hypertension- Modifiable and Non-Modifiable Factors**

Most common primary diagnosis in the US>
Heart disease, kidney disease, peripheral vascular disease and stroke.
Responsible for 7.6 mil (worlwide)
Prehypertension – range between normal blood pressure (120/80) to stage 1 hyper

- *Stage that we initiate efforts to prevent or deter progression of disease*

Primary hypertension- essential hypertension – Silent killer - *Sys BP* $\geq 140\,mmhg$ *Dias BP* $<90\,mmHg$
Most common form of hypertension
Rare prior to age 10-
Major risk for cardiovascular disease
Complication: End-organ damage, renal failure, stroke, heart disease, increase myocardial work in HF,
Treatment: Lifestyle modifications, wgt loss, exercise, DASH diet, alcohol moderation. Decrease sodium intake. Drug therapy for heart rare, SVR and stroke

Modifiable: - dietary factors, sedentary lifestyle, obesity/weight gain, metabolic syndrome.
Elevated blood glucose levels/Diabetes, elevated cholesterol, alcohol and smoking

Non-modifiable: Family hx, age, ethinicity/genetics

(maternal smoking, pregnancy induced hypertension, low birth rate)

- **Hypertension- Organs effected**

- Renal (Parenchymal and vascular)
- Cardiovascular
- Tumor
- Endocrine (Hyperthyroidism, cushing disease, congenital adrenal hyperplasia, primary hyperaldosteronism)
- Neurologic (Guillain-Barre Syndrome, Increased intracranial pressure)
- Other (Systemic arteritis. Sleep apnea)
  - Hypertension is the most common primary diagnosis in U.S
  - 32 % of 80 mil adults in U.S has High Blood Pressure
  - Higher in Non-hispanic black adults (44.9 %)
  - Hypertension is the most common Risk factor for cardiovascular worldwide

- Normal Range fore blood pressure is 120 mmHg systolic – 80 mmHg diastolic – 18 and older
- Prehypertension : range between normal BP to Stage 1 Hypertension
- Stage 1 Hypertension : Sys 140 mmHg or dias 90 mmHg

- Primary Hypertension (essential hypertension)
- No clear cause so idiopathic disorder
- Different than secondary hypertension where blood pressure elevation is cause
- Primary hypertension most common 90-95 %

- Systolic blood pressure is the major risk for subsequent cardiovascular disease

- **Hypertension- Management**
  - Lifestyles modifications
  - Drug therapy (heart rate, stroke, Systemic vascular resistance)
- **Coronary Artery Disease**

CHD (Ischemic Heart disease) CA. Insufficient delivery of oxygenated blood to the myocardium (Ischmea) because of the atherosclerotic coronary arteries.
CHD cause 1 in 6 death in UD.
_ (dysrhythmias, sudden cardiac arrest, heart failure)
Ethiology: Atherosclerosis of coronary arteries is the source od nearly all SHD
Risk factors: Family Hx, Abd lipid levels, smoking, hypertension, diabetes, obesity**)**

- **Definition of Atherosclerotic Plaques**

They are plaques that are from injury to the coronary artery endothelium and they are filled with large lipid core, fragiles and prone to ruptures.

- **Myocardial Ischemia- What is it**

It is when blood flow to the heart muscle (myocardium) is obstructed by a partial or complete blockage of a coronary artery by a buildup of plaques (artherosclerosis) If plaques ruptured, you can have a heart attack (myocardial infarction)

- **Acute Coronary Syndrome**

Risk : Smoking, Fam Hx, Obesity, Lifestyle sedentaty, diabetes, poor diets, lipids Up
S/S ; MI Chest pain (more severe than typical angina) *(patient might say elephant sitting on chest)*
Chest pain that may radiate to the arm shoulder , jaw and back
N/V
Diaphoresis (sweating)
SOB (dyspnea)
Etiology: Plaqu rupture with thrombus development
Unstable Angina – Occlusion is partial
With MI- Occlusion is complete
Treatment: Decrease myocardial oxygen demand – increase myocardial oxygen supply
Monitor and manage complications

Diagnosis : EKG and Biomakers

- **Definition of Unstable Angina Pectoris**
- Chest pain associated with intermittent myocardial ischmia (Burning, crushing, squeezing, choking, or referred pain)
  - **Result from inefficient cardiac pumpin (resultant pulmonary congestion and SOB)**

- Condition in which your heart doens;t get enough blood flow and oxygen. May lead to an heart attack. Angina is a type of chest discomfoer caused by a poor blood flow through the blood
  vessels (coronary of the heart muscle (Myocardium)
-
-
-
-
-
-
- **What is reperfusion therapy**

- **Left sided heart failure**

- **Right sided heart failure**

- **Medication management for Systolic Heart Failure**

▪ Effects of Right Sided Heart Failure

▪ MI- Signs and symptoms

▪ Shock

- Shock represents a diverse group of life-threatening circulatory conditions. The common factor among all types of shock is hypoperfusion and impaired cellular oxygen utilization. Inadequate cellular oxygenation may result from decreased cardiac output, maldistribution of blood flow, or reduced blood oxygen content.
- • During the compensatory stage of shock, homeostatic mechanisms are sufficient to maintain adequate tissue perfusion despite a reduction in cardiac output. Manifestations of sympathetic nervous system (SNS) activation are an elevated heart rate, increased myocardial stimulation, bronchodilation, vasoconstriction, cool clammy skin, dilated pupils, and decreased urine output. Blood pressure is maintained even though cardiac output has fallen.
- • During the progressive stage of shock, compensatory mechanisms begin to fail, and hypotension and progressive tissue hypoxia result. Shift of cells to anaerobic metabolism results in lactate production and metabolic acidosis. A lack of cellular adenosine triphosphate (ATP) production leads to cellular swelling, dysfunction, and death. Generation of oxygen free radicals, release of inflammatory cytokines, and activation of the clotting cascade lead to further cellular and organ dysfunction.

Cardiogenic Shock

- Cardiogenic shock is usually a result of severe ventricular dysfunction associated with myocardial infarction. Other causes include cardiomyopathy, ventricular rupture, and congenital heart defects.
- • Diagnostic features of cardiogenic shock include decreased cardiac output as a result of left ventricular dysfunction, along with elevated left ventricular end-diastolic pressure, S_3 heart sounds, and pulmonary edema. Sympathetic activation leads to an increased heart rate, vasoconstriction, and a narrow pulse pressure.
- • Low cardiac output leads to reduced oxygen delivery to tissues. Tissues extract a greater percentage of oxygen from the delivered blood, which leads to reduced Svo_2.
- • Therapy is aimed at improving cardiac output and myocardial oxygen delivery while reducing cardiac workload. Pharmacologic treatment often includes the use of inotropic agents, afterload-reducing agents (e.g., vasodilators), and preload-reducing agents such as nitrates and diuretics. Intraaortic balloon counterpulsation may be used to reduce afterload and improve coronary artery perfusion. Ventricular assist devices (VADs) may be used for longer-term circulatory support, whereas heart transplantation provides definitive treatment.
-

Obstructive Shock

- Obstructive shock results from mechanical obstructions that prevent effective cardiac filling and stroke volume.
- • Pulmonary embolism, cardiac tamponade, and tension pneumothorax are common causes of obstructive shock.
- • Prompt management of the underlying obstruction is necessary to prevent cardiovascular collapse.

Hypovolemic Shock

- Hypovolemic shock results from inadequate circulating blood volume precipitated by hemorrhage, burns, dehydration, or leakage of fluid into interstitial spaces.
- • The classic features of hypovolemic shock are the result of low cardiac output and low intracardiac pressures. Manifestations are due primarily to sympathetic nervous system (SNS) activation: elevated heart rate, vasoconstriction, and increased myocardial contractility.
- • The severity of symptoms of hemorrhagic shock correlates with the amount of blood loss; however, there is significant variation in the clinical presentation of hypovolemic and hemorrhagic shock, and careful assessment is required to diagnose it in the early stages.
- • Therapy for hypovolemic shock is aimed at fluid replacement and control of the source of volume loss. Colloids, isotonic crystalloids, and blood products may be used as replacement fluids.

- **Anaphylactic Shock**

Anaphylactic shock is a result of excessive mast cell degranulation in response to antigen. Mast cell degranulation usually is mediated by IgE antibodies. Release of vasodilatory mediators, such as histamine, into the circulation by mast cells results in severe hypotension. Urticaria, bronchoconstriction, stridor, wheezing, and itching are usually present. Treatment includes maintenance of airway patency and the use of epinephrine, antihistamines, vasopressors, and fluids to restore blood pressure.

- **Septic Shock**
- Septic shock results from a severe systemic inflammatory response to infection. Gram-negative bacteria, gram-positive bacteria, and fungal infections are common causes of septic shock. In gram-negative shock, endotoxins in bacterial cell walls stimulate massive immune system activation. Septic shock from any organism is characterized by release of large numbers of immune mediators (e.g., cytokines) resulting in widespread inflammation. The clotting cascade, complement system, and kinin system are activated as part of the immune response.
- • Widespread inflammation leads to profound peripheral vasodilation with hypotension, maldistribution of blood flow with cellular hypoxia, and increased capillary permeability with edema formation.
- • Initially, septic shock is characterized by abnormally high cardiac output resulting from immune-mediated vasodilation and sympathetic activation of the heart. The patient is usually febrile, pink, and warm. Even though cardiac output is high, cellular hypoxia is present because of maldistribution of blood flow. Reduced cellular oxygen utilization is manifested as a high Svo_2.
-

- **Goal of treating sepsis is**
  Treatment for sepsis is aimed to improve the distribution of blood flow and managing infection with antibiotics. Administration of fluid and drugs to increase cardiac and vascular perfomance to improve blood flow

- **Cancer and its ability to metastasize**

unpredictable- does not follow cellular functions rules
Malignant cells produced specialized enzymes and receptors to enable them to escape their tissue of origin and metastize. The spread generally occurs by way of bloodstream or lyphatics.
Tumor cells lodge in the capillary beds of the organs that drain them
Some cancer are prone to metastize: Breast, lung, bone, colon.

- **CVA**

- **Osteoporosis**

The most common metabolic bone disease (10 mil 50+ in Us)
Happens when rate of bone resorption is greater thatn rate of bone formation and oastoblastic and osteoclastic balance is disrupted

Bone mineral density is greater or equal to 2.5 standard
Risk factor: age, corticosteroid use, Hx of Rheumatoid arthritis, Fam Hx, estrogen level.
Family Hx is a major risk factor
After menopause. The rapid of normal bone loss develop rapidly so estrogen deficiency increase the risk of osteoporosis
Other risk: Small frame, Caucasian or Asian, high dose of thyroid, diet low on calcium and vitamin D, physical inactivity. Smoking or increase alcohol intake/ Patient with chronic renal function who have abnormal parathyroid function. Cushing syndrome,. Hyperthyroidisms. Anorexia nervosa. COPD, Type 1 diabetes. Celiac disease, lupus. Cancer Rheumatoid Arthritis.

S/S: often asymptomatic until bone fracture
Shortened stature
muscle wasting or spasms of back muscles/difficulty bending
Stooped posture
Bone that breaks more easily than expected
C/O of impaired breathing

Complication: Fall risk (spine or hip fractures), disability and increased risk of death

Treatment:
Good nutrition (Protein)
Moderate to regular exercise (walking, riding stationary bicycle)
Calcium (1000-1500 mg daily)
Vitamin D to maintain bone mass (30 mg/mL dihydroxyvitamin D)
Biphosphonates

- **Passive Immunity**
- **Passive immunity involves the transfer of plasma containing preformed antibodies agains a specific antigen from a protected or immunized person to an unprotected or nonummunized person.**
- **Provide immediate but temporary protection**
  Needs: 1) when B-cell immunodefiency 2) highly susceptive person are exposed to disease without active immunization 3) antibody injection

  - **Mom to fetus (IgG crass the placental barrier)**
  - **Newborn receive IgA through Breast milk**
  - **Serotherapy) Immunization of anibodies to unprotected person**

- **Immunoglobulin involved allergic hypersensitivity reactions**
  In the majority of cases the antibody typically responsible is IgE isotype (IgE -Asthma)

- **Clinical indicator of Acute Blood Loss- Orthostatic hypotension**

Acute Blood Loss Anemia – present after trauma or secondary to disease. It rapidly decrease the overall blood volume and impairs O2 delivery
Hemoocrit level is less than 40 % in men and 37 % in women .. Hemoglobin conc is less than 14 in men and 12 in womwn ..

Tachycardia is seen with exercise
Slight drop in BP
(10 % Loss -500 ml) no clinical signs expect vasovagal syncop
(30 % Loss-1500 ml) Flat neck vein with supine postural hypotension
(40% loss-2000 ml) Central venous pressure. Cardiac output, arterial PB below normal
Patient is supine / at rest/ air hunger/ tachycardia/ cold/ clammy skin

(50 % loss -2500 ml) SCHOCK AND DEATH

Treatment_ Blood Volume replacement therapy with crystalloid solutions _ good prognosis if underlying cause is managed

- **Asthma**