

Med Surg Exam 3

Exam 3:

20% Anemia and Sickle-cell disease

10% Blood transfusion

20% HTN

20% DVT/PVD

20% Fluid and electrolytes

10% Central venous access

Lewis: Chapter 29

And chapter 30

p. (606-625)

Diseases not covering at this time:

Thalassemias, G6PD anemia, acquired hemolytic anemia, hemochromatosis, hemophilia, disseminated intravascular coagulation (DIC), ITP, neutropenia, leukemia, Myelodysplastic syndrome, multiple myeloma, or lymphoma.

- **Know the normal lab values**

*****need to identify and differentiate between normal and abnormal lab values

- WBC= 5,000-10,000
- Hct= men= 42-52% women= 37-47%
- Hgb= men= 14-18 women= 12-16
- Platelets= 150,000- 400,000

Category	SBP (mm Hg)	DBP (mm Hg)
Normal	<120 and	<80
Prehypertension	120-139 or	80-89
Stage 1 Hypertension	140-159 or	90-99
Stage 2 Hypertension	≥160 or	≥100

-
- **Differentiate between decreased production, blood loss, and increased destruction-
etiology of anemia**
 - See table 30-1 p 607
 - Be able to identify a patient scenario with the different types (causes)
- **Differentiate between mild, moderate and severe anemia and recognize**
 - **Clinical manifestations of severe anemia (table 30-1 pg 607)**
 - Mild anemia= Especially Hgb 10 to 12 g/dL [100 to 120 g/L])
 - May have response to the body's response during heavy exercise such as:
 - palpitations, dyspnea, and mild fatigue.

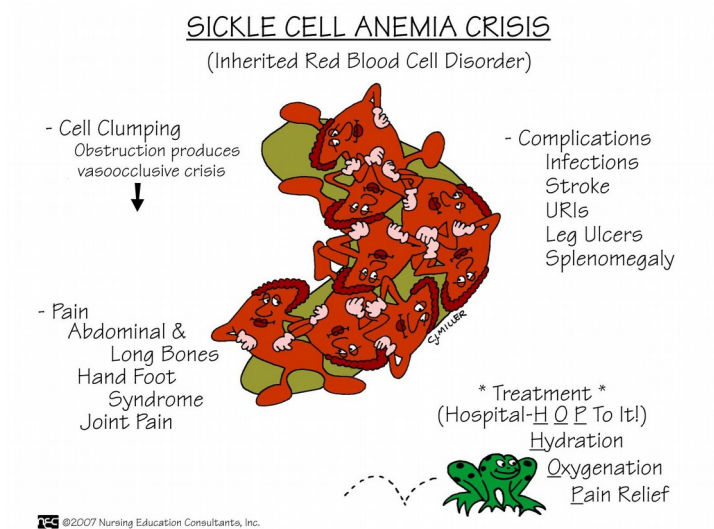
- Moderate Anemia= (Hgb 6 to 10 g/dL [60 to 100 g/L]), the above cardiopulmonary symptoms are increased.
- Symptoms at rest and with activity
- Severe Anemia= (Hgb less than 6 g/dL [60 g/L]), Many body systems become involved when severe
- **Nursing interventions for anemia: related to fatigue/planning daily care activities**
- **Deficient nutrients:**
 - **Iron deficiency anemia**
 - **Pathophys/Etio./Risk factors**
 - Anemia is **not a disease but it is a manifestation of a disease process**
 - First Look at the morphology (Cellular characteristic): Normocytic (normal size) normochromic (normal color)
 - Then, you will look for the cause of the anemia
 - **Decreased production** = From deficient nutrients, Decreased Erythropoietin, Decreased iron availability
 - **Blood loss** = Acute/ Chronic
 - **RBC destruction** = Sickle-cell anemia, Incompatible blood
 - Risk factors = Young and women of reproductive years are at highest risk ,Or poor diet
 - **Clinical manifestations/signs and symptoms**
 - Pallor, jaundice, Glossitis: smooth tongue, Cardio: HR, dyspnea, headache, impaired thought process,
 - GI: anorexia, hepatosplenomegaly, sore mouth, MS: bone pain, Lethargy
 - **Collaborative care**
 - Plan activities at times of most energy -prioritize
 - Monitor CV response
 - Determine endurance
 - Nutritional consult= Determine number of calories to meet requirement, Food diary, Monitor intake (calorie count), Encourage increase of those foods for hemoglobin production, Education about dietary needs
 - **Interventions/Medications**
 - **Dietary implications: Iron therapy-patient education**
 - PO-main medium is Ferrous sulfate-best absorbed in this form
 - 150 -200 mg TID
 - Absorbed in jejunum so extended release is not appropriate
 - Dairy binds to iron and decreases absorption
 - Take iron 1 hour before meals
 - Vitamin C increases absorption
 - Education-black stools -your stools will become black due to excess iron excreted into GI tract
 - 1 hour before meal
 - Constipation, may need laxatives
 - Risk for staining teeth
 - **Nursing diagnoses**
 - Altered Nutrition and Activity Intolerance
 - **Pernicious anemia**

- **Pathophys/Etio./Risk factors**
- Body is not producing intrinsic factor –could be from autoimmune destruction of parietal cells or gastric mucosal atrophy
- **Intrinsic factor is secreted by parietal cells and is necessary for absorption of B12**
- **Other causes of B12 deficiency are those who have had GI surgery and have a loss of gastric mucosal cells**
- **alcoholism-see this a lot, long term use of proton pump inhibitors or strict vegetarians**
- **Clinical manifestations/signs and symptoms**
- These patients will have the same symptoms as the general anemia patients (with tissue hypoxia) but specific to B12 are the sore, red, beefy tongue
- Paresthesias (numbness and tingling of hands and feet) and disturbed thought from mild confusion to dementia
- **Large size normal color=** MCV > 100 MCG > 34
- Exclude folic acid deficiency first
- **Collaborative care**
 - Usually start IM injections of vitamin B12 daily
 - then when Hgb returns to normal monthly injections will be needed for life
 - Cobalamin IM 1000 mg daily x 2 weeks then weekly until hgb normal
 - Neuromuscular complications may not be reversible
- **Interventions/Medications**
 - Dietary implications-
 - If the patient does not have Intrinsic factor than a dietary increase will not help
 - And foods high in vitamin B12
 - Red meats, liver, eggs, enriched grains, milk dairy, and fish
 - Nursing diagnoses
- **Folic acid deficiency**
 - **Pathophys/Etio./Risk factors**
 - Causes: dietary deficiency
 - Malabsorption syndromes
 - Drugs that interfere with folic acid synthesis
 - Such as methotrexate or phenytoin
 - Alcohol Abuse
 - **Clinical manifestations/signs and symptoms**
 - **Be able to differentiate between the (deficient nutrient) anemias based on signs and symptoms/labs**
 - **Collaborative care/Interventions/Medications**
 - Dietary implications-folic acid
 - Green leafy vegetables, Liver, Meat, Fish, Legumes, Whole grains, Orange juice, Peanuts
 - Replacement therapy is the treatment of choice: In malabsorption states or with chronic alcoholism, up to
 - 5 mg per day may be required.

- Duration of treatment depends on cause of deficiency.
 - Nursing Diagnoses
- **Acute blood loss**
 - Pathophys/Etio./Risk factors
 - Acute is sudden and may occur after a
 - Trauma or Surgery/maybe a complication from surgery
 - Clotting disorder =Sudden loss=**Hypovolemic shock**
 - If blood loss is more gradual Body increases plasma volume to compensate
 - Clinical manifestations/signs and symptoms
 - 10% blood loss: may have none
 - 20% tachycardia with exercise or *slight* postural hypotension
 - 30% normal at rest=Postural hypotension and tachycardia with exercise
 - 40% BP, CVP, CO all LOW=Air hunger, rapid thready pulse and cold clammy skin
 - 50% =Shock, lactic acidosis death if not treated
 - The RBC, may seem normal for 2-3 days, but once body compensates by adding plasma then the RBC become less concentrated and therefore lower
 - **Collaborative care/Interventions/Medications: treatment of acute blood loss**
 - IV fluids used in emergencies
 - dextran, hetastarch, albumin, and/or crystalloid electrolyte solutions
 - such as lactated Ringer's
 - Once volume replacement is established, identify source, may need further testing
 - CT scans
 - If trauma-may not be able to prevent
 - If post-op. want to really monitor wounds, drains, and tubes carefully
 - attention can be directed to correcting the RBC loss.
 - The body needs 2 to 5 days to manufacture more RBCs in response to increased erythropoietin.
 - Consequently, blood transfusions (packed RBCs) may be needed if the blood loss is significant.
 - If bleeding from clotting disorder than: replacement of that deficiency is addressed.
 - Anemia after blood loss-decreased iron stores so may need supplemental iron to stimulate erythrocytes.
 - Nursing diagnoses
 - **Chronic blood loss**
 - Patho/Etio/Risk Factors
 - Macrophage phagocytosis of the defective erythrocytes
 - Risk factors →
 - Bleeding ulcer
 - Hemorrhoids
 - Menstrual and postmenstrual blood loss
 - Renal failure → decreased erythropoietin production
 - Clinical manifestations(ATI pg 253)
 - Possibly asymptomatic in mild cases
 - Pallor

- Fatigue
 - Irritability
 - Numbness and tingling of extremities
 - Dyspnea on exertion
 - Sensitivity to cold
 - Pain and hypoxia with sickle-cell crisis
- Collaborative care/ Interventions/Medications
- Rarely blood transfusions
 - Erythropoietin
 - Identifying the source
- Stopping the bleeding
 - Providing supplemental iron as needed
- Nursing diagnoses
- **RBC destruction**
 - **Hemolytic anemia (NOT acquired hemolytic anemia)**
 - Destruction or hemolysis
 - Rate that exceeds production
 - Intrinsic factors→
 - Abnormal hemoglobin
 - Enzyme deficiencies
 - RBC membrane abnormalities
 - Extrinsic to the RBCs→ damage caused by and external force
 - **Patho/Etio/Risk Factors**
 - **Clinical manifestations**
 - Jaundice→ due to increased destruction→ elevated bilirubin levels
 - Enlarged spleen and liver (hepatosplenomegaly) → due to hyperactivity of the spleen removing defective erythrocytes
 - **Collaborative care**
 - treatment→ accumulation of hemoglobin molecules, can obstruct renal tubules and lead to ATN and renal failure
 - Interventions/Medication
 - Nursing diagnoses
- **Sickle cell anemia**
 - **Pathophysiology of /Etiology**
 - Autosomal recessive disorder:
 - 1 out of every 500 african american births
 - Also in mediterraneans, central america
 - Identified in infancy (genetic)
 - Decreased life expectancy
 - **Defective shape of Hgb - HbgS → intrinsic defect, sickling of the RBC**

- Severe, painful, acute exacerbation→ vaso-occlusion occurs
- Severe capillary hypoxia→ leads to tissue necrosis
- Life threatening shock → reduction in O₂ and decreased circulation fluid volume
- **Clinical manifestations→**
 - Repeated sickling→ all body systems become involved (slide 35)
 - Pain **primary symptom**
 - During sickling episodes
 - Ranges moderate to severe
 - Pallor
 - Mucous membranes pale and skin→ gray color
 - Jaundice from hemolysis
 - Prone to cholethiasis
 - Fatigue
 - Chronic disease (due to tissue hypoxia)
 - Kidneys and liver
- Triggers of sickling episodes
 - Deoxygenation of RBC
 - infection→ most common
 - High altitude
 - Stress (physical or emotional)
 - Dehydration
 - Acidosis
 - Low body temperature
- Diagnostic studies→
 - **MCV (80-100) AND MCH (27-34) NORMAL**
 - Increased bilirubin due to accelerated RBC breakdown
 - Hgb and Hct→ LOW



-
- **Interventions/Medication preventative measures**
 - Supportive treatment of
 - **Crisis-**
 - **Chronic, and (how to prevent crisis)/typical daily regimens and preventative care**
- **Nursing diagnoses/management**
 - Need to be hospitalized (sickle cell crisis)
 - O₂ (for hypoxia)
 - Vigilance for respiratory failure
 - Rest
 - and DVT prophylaxis
 - Fluids and electrolytes
 - Transfusion therapy
 - Hematopoietic stem cell transplantation (HSCT) → is the ONLY available cure
 - Nursing management/collaborative care
 - hydraea → chemotherapy medication → it increased RBC volume and production
 - Folic acid → chronic hemolysis → uses up folic acid stores
 - Often undertreated → decreased understanding
 - Pain management → often opioid tolerant, PCA, multimodal approach (TCA, antidepressants, NSAIDs)
 - Acute chest syndrome →
 - Broad spectrum antibiotics
 - Oxygen therapy
 - Fluid therapy

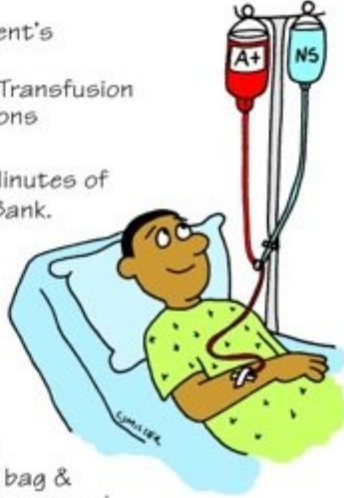
- RBC exchange
 - **What educational topics/triggers would be covered with the patient of SCD to help prevent frequent crisis exacerbations?**
 - **PREVENTION!**
 - Avoid high altitudes
 - Maintain adequate fluid intake
 - Treat infections promptly
 - Patient and caregiver support→ how to avoid crisis, importance of prompt medical attention, pain control and give resources for care and support
 - eyes→ know when to seek medical attention
 - **Collaborative care**
 - Alleviating symptoms and complications
 - Minimizing end-organ damage
 - Promptly treating serious sequelae
- **Define polycythemia: too many RBC, too much Hgb & Hct, too thick, too many platelets**
 - **Cause**
 - Primary versus Secondary-Health history and clinical assessment findings
 - primary→ polycythemia vera
 - Chronic myeloproliferative disorder
 - increased→ RBC, WBC, and platelets→ increased production
 - Insidious onset, 60 years old at diagnosis (sometimes with genetic link→ JAK2 gene)
 - More likely seen in men than women
 - Enhanced blood viscosity/increased blood volume and congestion of organs and tissues with blood
 - Splenomegaly seen in 90% of these patients
 - Hepatomegaly
 - INCREASED risk for clotting
 - **Secondary→**
 - Hypoxia driven→ high altitude, cardiovascular disease, or defective O2 transport
 - Returns to normal once HGB is stabilized
 - Hypoxia independent→ renal cysts or tumors, extrarenal tumors
 - **Treatment of these patients**
 - **Clinical manifestations**
 - HTN
 - Headache, vertigo, dizziness, tinnitus, visual changes

- Paresthesia
 - Pruritis
 - Heart failure, intermittent claudication
 - Thrombophlebitis
 - Complicated by embolism
 - **Most common complication (stroke due to thrombosis)**
 - Hemorrhage from over distension of vessels or rupture
 - Petechiae, ecchymosis, epistaxis, GI bleeding
 - Ruddy complexion
 - Hyperuricemia
- labs→ increased HGB, increased RBC count
- Treatment→ phlebotomy
- Nursing management→ hydration, assess for tissue perfusion complications, reduce blood volume
- Medications used to treat the above
 - (Route, most common side effects, and administration guidelines)
 - Busulfan (Myleran/myelosuppressive)
 - Paxil
 - Low dose heparin
 - Low dose aspirin→ prevents clotting
 - Allopurinol to reduce gout attacks
- Nursing diagnoses related to anemia
 - **(care and interventions related to ND)**
 - **activity intolerance**
 - **fatigue**
 - **altered nutrition: less than body requirements**
 - ineffective self-health management
- Blood products:
 - **Packed RBC's**
 - **Indications**
 - **Special considerations**
 - **Important nursing responsibilities-**
 - **steps of transfusion reaction**
 - How to recognize blood transfusion reactions
 - **Prevention and management of blood transfusion reactions**
 - **Table (30-33)**
 - Platelets
 - Indications
 - Special considerations
 - Nursing responsibilities
 - Fresh frozen plasma
 - Indications
 - Special considerations
 - Nursing responsibilities

BLOOD ADMINISTRATION

- * Determine Client's
 - Allergies
 - Previous Transfusion Reactions
- * Administer Within 30 Minutes of Receiving From Blood Bank.
- * Never Add ANY Meds to Blood Products.

- * Check Crossmatch Record With 2 Nurses:
 - ABO-Group
 - RH Type
 - Client's Name
 - ID Blood Band
 - Hospital #
 - Expiration Date
- * Do NOT Warm Unless Risk of Hypothermic Response THEN Only By Specific Blood Warming Equipment.
- * Infuse Each Unit Over 2-4 Hours BUT No Longer Than 4 Hours.



KEY POINTS

- Verify Client's ID
- Check the Dr's Order.
- Check labels on blood bag & blood bank transfusion record
- Baseline vitals - (Then per policy).
- #18G or #20G gauge needle.
- Normal saline IV solution.
- Blood administration set with filter.
- Severe reactions most likely first 15 min & first 50cc.
- Blood tubing should be changed after 4 hours.

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- Describe the age-related changes that occur in the hematologic system.
- Thrombocytopenia
- Possible causes
- **Common clinical manifestations-(and risks of severely low platelet count)**

How to prioritize care of a patient with thrombocytopenia

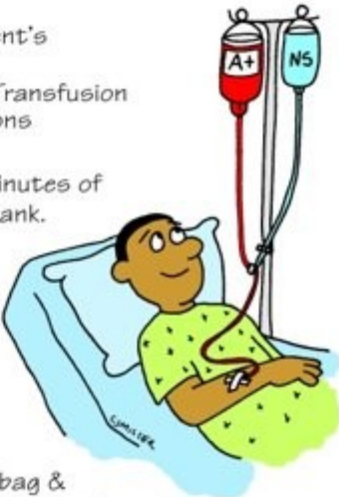
- S&S=
 - Patients are often asymptomatic
 - Mucosal bleeding: epistaxis, bleeding gums
 - Intugumentary:
 - Petechiae – micro hemorrhages
 - Purpura – bruise from numerous petechiae
 - Ecchymoses – larger lesions from hemorrhage
 - Gastrointestinal: splenomegaly, abd distention, guiac positive stools
- Interventions=
 - Control hemorrhage
 - Stop medications (OTC)
 - Aspirin
 - (decreases platelet adhesion)

- Platelet count
- Transfusion Plt. < 10,000 or actively bleeding
- Prevent complications
- Bleeding risk
- Cancer chemotherapy drugs
- Monitor lab values:
 - Platelet count, coagulation studies, Hgb/Hct.
- **What is the collaborative treatment for**
 - **HIT: what labs are you watching for and what do you do?**
 - Platelet count drops 50% from baseline.
 - Major problem- Venous thrombosis
 - DVT and PE often result
 - Arterial thrombosis
 - (risk for skin necrosis, stroke, end organ damage (kidneys))
 - Interventions=
 - STOP Heparin
 - Mark on their chart clearly
 - Should never be given Heparin or LMWH (enoxaparin)
 - Direct thrombin inhibitor
 - Start Coumadin
 - when platelet count reaches 150,000/ μ L
 - Platelet transfusions are not effective=Enhance thrombotic events
 - Severe:
 - Plasmapheresis
 - Clear IgG from the blood
 - Protamine sulfate to interrupt the circulating heparin
 - Thrombolytic agents to treat the thromboembolic events
 - Surgery to remove
 - chemo-induced thrombocytopenia?

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Lewis: Chapter 32

- Define hypertension, pre-hypertension and recognize diagnostic BP parameters

Category	Blood Pressure, mm Hg
Normal	SBP \leq 120 and DBP \leq 80
Prehypertension	SBP 120-139 or DBP 80-89
Stage 1 Hypertension	SBP 140-159 or DBP 90-99
Stage 2 Hypertension	SBP \geq 160 or DBP \geq 100

- Describe the pathophysiologic mechanisms associated with primary HTN.
 - Majority of all cases
 - **Do not know exact cause**
 - Increased sympathetic system response

- **RAAS system* → angiotensin two increased blood pressure in two way → 1. Tells adrenal cortex to hold onto aldosterone (increasing fluids)2. Potent vasoconstrictor affecting arterioles (increasing SVR)**
- **Relate the difference between primary and secondary hypertension.**
 - Primary hypertension→
 - Essential or idiopathic
 - Elevated BP without an identified cause
 - **Accounts for 90-95% of all cases**
 - **Contributing factors**
 - **↑ SNS activity** stress, not sleeping, type A personality
 - **↑ sodium-retaining hormones & vasoconstrictors**
renin overdrive
 - **↑ sodium intake**
 - **> ideal body weight** obesity
 - **Diabetes mellitus**
 - **Excessive alcohol consumption**
 - Secondary hypertension→
 - Elevated BP with a specific cause
 - **Accounts for 5-10% of adult cases**
 - **Contributing factors**
 - **Cirrhosis** changes in the blood flow, causes strain to right side of heart
 - **Coarctation of aorta** change in the way the blood flows
 - **Endocrine disorders** Cushing's, Hyperthyroidism
 - **Medications** norepinephrine, cocaine, birth control
 - **Neurologic disorders**
 - **Renal disease** any time there is renal dysfunction it affects it, kidneys sense they are not getting enough blood causes the increase
 - **Sleep apnea** strain on your heart
- **Discuss risk factors, clinical manifestations diagnostic testing and complications of hypertension.**
 - Risk factors→
 - Age
 - Alcohol
 - Cigarette smoking→ toxic to endothelial lining, causes vasoconstriction and change to cells
 - Diabetes mellitus
 - Elevated serum lipids→ hyperlipidemia
 - Excess dietary sodium
 - Gender→ men at higher risk until age of 64, and then women at higher risk after menopause
 - Family history
 - Obesity
 - Ethnicity → African Americans
 - Sedentary lifestyle→ not exercising
 - Socioeconomic status
 - Stress→ increases sympathetic nervous system

- Clinical manifestations→ Referred to as the “silent killer” because patients are frequently asymptomatic until target organ disease occurs
 - Fatigue
 - Reduced activity tolerance
 - Dizziness
 - Palpitations
 - Angina
 - Dyspnea
- Diagnostic testing→ table 32-11
 - **At least 2 elevated blood pressures within 2 weeks**
 - Use both arms, need to be seated for five minutes with feet on floor, empty bladder
 - **Biggest thing→ correct size of blood pressure cuff**
 - Routine urinalysis→ checking for protein
 - BUN/Creat
 - Serum electrolytes-Serum cholesterol and glucose levels→ baseline, looking at other risk factors
 - Serum cortisol levels → to rule out cushing's
 - ECG→ can look for myocardial ischemia, will also have a tall R wave if they have left ventricular hypertrophy
 - Echocardiogram→ look at ejection factors and left ventricle
 - Chest x-ray→ show if heart is enlarged
- Complications→
 - heart→ usually first organ affected (hypertensive heart disease)
 - coronary artery disease→endothelium stiff and narrow, can have plaques
 - left ventricular hypertrophy
 - heart failure
 - left sided heart failure (pulmonary edema) more common with HTN
 - right sided heart failure (JVD, peripheral edema)
 - brain → cerebrovascular disease
 - atherosclerosis→ vessels that go up to the brain
 - stroke→ 4 times more likely to have a stroke if you have HTN, ischemic or hemorrhagic
 - hypertensive encephalopathy → protective regulation from the brain that does not work, increased intracranial pressure, edema
 - Peripheral vasculature (peripheral vascular disease)
 - Intermittent claudication→ legs are not getting enough oxygen
 - Kidneys (nephrosclerosis)→ narrowing and atrophy of renal tubules
 - End-stage renal disease
 - Lab indications of renal dysfunction: **microalbuminuria**, proteinuria, microscopic hematuria, ↑ serum creatinine and BUN (these are late sign)
 - Nocturia – earliest manifestation of renal dysfunction
 - Eyes (retinal damage)

- blurred vision
 - Retinal hemorrhage
 - Loss of vision
- Discuss strategies for the prevention of hypertension.
- Describe the collaborative care for hypertension, including drug therapy and lifestyle modifications(DASH diet, exercise)
 - Consider strategies for adherence to regimes
 - **Maintain BP goal and decrease risk of CV and target organ damage**
 - **Lifestyle Modifications:**
 - Weight reduction – weight loss of 22 lb (10 kg) may ↓SBP by approximately 5 to 20 mm HG
 - DASH Diet – fruits, vegetables, fat-free or low-fat milk and milk products, whole grains, fish, poultry, beans, seeds, and nuts
 - Dietary sodium restriction - ≤ 2300 mg/day
 - Omega-3 fatty acids
 - Limit caffeine
 - Moderation of alcohol consumption
 - Men: no more than 2 drinks/day
 - Women: no more than 1 drink/day
 - Physical activity
 - Avoidance of tobacco products
 - Management of psychosocial risk factors
- Patient education on new diagnosis, Powerpoint 10 & 15
- Discuss the differences and care of the older adult with hypertension.
 - 60 years and over
 - Start medication therapy for 150/90 and up, but goal is less than 150/90
 - **JNC8 criteria**
 - Isolated systolic hypertension (ISH) – most common form of hypertension in individuals >50 years of age
 - “White coat” hypertension
 - Auscultatory gap – wide gap between the first Korotkoff sound and subsequent beats
Failure to inflate the cuff high enough may result in seriously underestimating SBP
 - More likely to experience medication interactions
 - More likely to experience orthostatic hypotension
- Recognize cultural differences of both incidence and treatment of HTN.
 - Specifically African Americans prevalence and treatment regimens
 - ACE inhibitors may not be as effective and may cause angioedema
 - African Americans increased risk of HTN 2x more likely
 - Mexican Americans are less likely to receive treatment than whites
- Discuss the various age-related changes in the cardiovascular system
- Discuss the goal of pharmacologic management of HTN (JNC 8 guidelines),
 - recognize the main drug classifications,(prototype examples) and the
 - primary nursing implications and precautions associated with each.
 - Slide 3 HTN (know which stage they are in)

Beta-blockers: metoprolol (beta 1 selective, should not affect lungs), atenolol, propranolol (noncardiospecific, can cause bronchospasms in asthma patients), decrease HR, and CO, reduce SVR, also decrease renin secretion→ check heart rate before giving it because **directly** effects the heart rate (if heart rate is low around 50, hold, most times will have parameters when to hold or to give it) **big thing**→ **can cause ED, depression... do not stop abruptly**, patients with diabetes may have different glucose responses

Calcium channel blockers: amlodipine, verapamil, change the way calcium moves across cell membrane, cause vasodilation

Centrally acting Sympatholytic: clonidine, not first line do not like to use a lot, reserved for those having difficulty with therapy (ESRD, hypertensive crisis)→ has a rebound effect, makes patients extremely lethargic, directly affects SNS

11.) Hypertensive crisis: recognition and treatment

BP above 240/120, need to go to ICU or ER cannot be treated on med surg floor

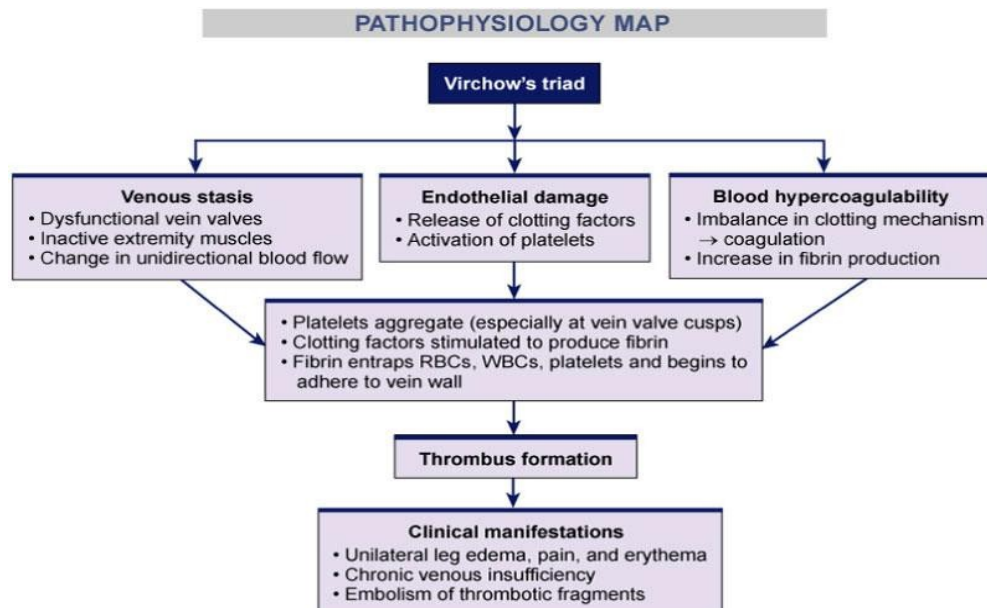
Happens when **NONCOMPLIANT TO PRESCRIBED MEDICATIONS**

- **REFER, REFER, REFER>>>Hospitalization**
- IV drug therapy: titrated to mean arterial pressure (MAP) $MAP = (SBP + 2DBP) / 3$
- Monitor cardiac and renal function
- Neurologic checks (pupils, LOC), cerebral edema
- Determine cause
- Education to avoid future crises

Learning Objectives Vascular Disorders

Chapter 37 (pgs. 816-829)

- Discuss pathophysiology, clinical manifestations and treatment of superficial and deep venous thrombosis.



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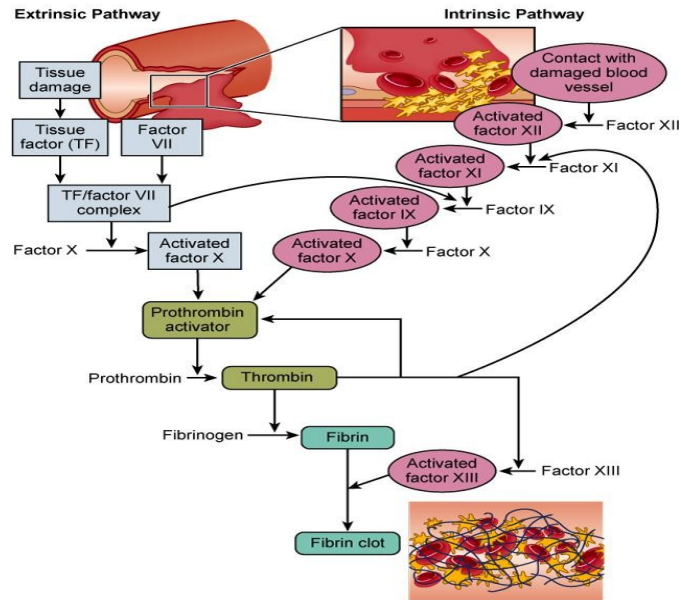
Diagnostic testing related to DVT

- **Identify the risk factors predisposing the development of superficial thrombophlebitis and deep vein thrombosis.**
 - **Identify patients at risk based on the three components: triad of Virchow**
 - Pg. 817
 - Venous stasis, Bed rest, immobility, long trips, surgery, pregnancy, a -fib, obese, spinal cord injury
 - Endothelial damage = by IV or damage caused by trauma burns, sepsis, surgery, diabetes
 - Blood hypercoagulability= abdominal pelvic surgery, hyper IV fluids, fracture of pelvis, history of VTE, central venous catheter, Iv drug use and trauma,
- **Differentiate between clinical manifestations of superficial and deep venous thrombosis**
- **SVT→**
 - Clinical manifestations:
 - Superficial Veins:
 - leg veins and arm veins
 - Untreated→ can extend to deeper veins →VTE
 - Tender to touch
 - reddened, warm
 - inflammation and induration
 - Palpable cord
 - Mild systemic temperature elevation & leukocytosis
 - **Edema rarely occurs**
- **DVT=**
- Clinical manifestations:
 - Deep veins of:
 - arms, legs, pelvis, pulmonary system
 - Tender to pressure
 - Over the vein, venous distention, and EDEMA
 - Change in leg circumference
 - **** Some do not have any obvious changes in the Physical assessment**
 - May break off and → travel to the lungs (pulmonary embolism)→ can result in death

Recognize the main nursing diagnosis associated with venous thrombosis.

- Pain, lack of knowledge
- **Discuss the interventions (both collaborative and independent) for DVT prevention.**
 - **Ambulation is the most affordable and effective**
 - Early mobilization.....leg exercises and out of bed often
 - Ted hose.....fitted correctly
 - SCD's.....unless an active VTE
 - **Must be on the patient to work!!!!!!**
 - If patient does not want them, document you discussed importance
 - **Anticoagulation therapy – PREVENT CLOTS**
 - **Do not want to put on if already bleeding, have a history of bleeding (ulcers)**
 - Complications
 - **Pulmonary embolism (life-threatening)**

- Chronic venous insufficiency – valve destruction
- Discuss specifically the role of the following medications in the treatment of DVT:
 - warfarin, heparin (both unfractionated and low molecular weight (LMW), thrombolytics)
 - Anticoagulants – used routinely for VTE prevention and treatment
 - Prophylaxis – prevent clot formation
 - Treatment – prevent propagation of the clot, development of new thrombi, and embolization
 - thrombolytic drug =CLOT BUSTERS
 - What to check to see if the medication is at a therapeutic level
 - Heparin= Monitor aPTT levels
 - Normal: 25-35 seconds
 - Therapeutic: 46-70 seconds
 - Route: continuous IV, intermittent IV, subcutaneous
 - Antidote: **protamine sulfate**
 - **Warfarin=**
 - Monitor INR levels
 - Normal: 0.75-1.25
 - Therapeutic: 2-3
 - Route: PO
 - Antidote: **vitamin K**
- Describe the nursing management of the patient receiving anticoagulation therapy.
- Recognize the goal and management of the following medications used for thrombosis prevention and/or treatment:
 - Anticoagulants: **heparin sodium, enoxaparin, warfarin**
 - Anticoagulants: used to prevent clot formation for those high at risk, if have a clot already just stops it from getting bigger
 - heparin sodium, enoxaparin,
 - **warfarin→ PO only**, useful for long term, a-fib, DVT, **vitamin K** antagonist
 - **indirect thrombin inhibitor→ heparin**
 - unfractionated heparin (UFH)→ **heparin sodium** subcutaneous, do not massage area, do not aspirate, if given IV then it is the drip (give for people having an active MI as well, works immediately)
 - low-molecular weight heparin (LMWH)→ **LOVENOX (enoxaparin)→** given subcutaneous, can give for people who have a DVT and are treating it, higher dose (prefilled), cannot use in patients with renal failure, less risk of heparin induced thrombocytopenia, treatment of choice
 - **high dose→ have DVT, low dose→ preventive**
 - **PATIENTS ON WARFARIN→** should not take herbal supplements, garlic, ginkgo biloba, NSAIDS
 - Antiplatelets: aspirin
 - Thrombolytics: streptokinase, alteplase
 - Treatment of Heparin overdose: **protamine sulfate**
 - Treatment of warfarin overdose: **vitamin K**



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- Intrinsic → heparin
- Extrinsic → warfarin
- **Know both the normal baseline values and therapeutic levels while on anticoagulants for PT, PTT, INR**
- **Discuss the pathophysiology, clinical manifestations and collaborative care and nursing management of patients with varicose veins, venous insufficiency, and venous leg ulcers.**

Objectives chapter 16 Lewis: (p. 270-286) and (p. 290-294), ATI ch. 43 and 44

- Review homeostasis and the functions and regulatory mechanisms that maintain water and electrolyte balance in the body.
 - **Hypothalamic regulation**
 - Osmoreceptors in the hypothalamus sense body fluid deficit or increase in plasma osmolality
 - This stimulates thirst and ADH (then we increase the amount of water we drink) restored fluid volume
 - Antidiuretic Hormone (also called vasopressin),
 - synthesized in the hypothalamus and stored in the posterior pituitary,
 - Acts in the renal distal and collecting tubules causing water reabsorption.
 - **Cardiac regulation-what does elevation of BNP mean?**
 - B-type Natriuretic Peptide are produced in response to increased atrial pressure
- **Recognize gerontologic considerations regarding fluid and electrolyte imbalances.**
 - **Focus in on power point information**
 - Older adult (less body water) increase risk of dehydration
 - Kidneys have structural changes: decreased GFR and ability to concentrate urine and conserve water
 - Hormonal changes include a **decrease in renin and aldosterone and an increase in ADH (hold onto water and ANP.**
 - Loss of subcutaneous tissue and thinning of the dermis lead to **increased loss of moisture** through the skin and an inability to respond to heat or cold quickly.

- **Skin turgor is not a good indicator** in the elderly because of loss of elasticity
 - Older adult has normal changes with aging that **increase susceptibility to fluid and electrolyte imbalances**
 - Do not automatically assume the fluid and electrolyte imbalances are from natural aging process as they COULD be pathological
 - Attention needs to be paid to long-term care facilities where older adults **may not have access or decreased thirst mechanism**
 - **Identify the primary causes of fluid volume deficit**
 - Which fluid for rapid volume replacement = Normal Saline
- dehydration= is loss of pure water alone (sodium remains normal)**
- Sometimes seen in exercise, alcohol, high elevations
- increased risk in elderly
 - abnormal loss of body fluids (diarrhea, vomiting, hemorrhage)
 - inadequate fluid intake,
 - or plasma to interstitial shift
- treat by find and treat cause and replacing water and electrolytes,
- **Compare/contrast the clinical manifestations presented in the patient with fluid volume deficit/dehydration and fluid volume excess. (table 16-3) and powerpoint**
 - **Volume deficit** = Heart rate is elevated compensate to increase BP
 - Thready pulse (decrease flow)
 - Tachypnea, to compensate for loss of fluid volume
 - Concentrated urine (body is attempting to hold on to water)
 - Kidneys will concentrate the urine to hold on to water, So urine specific gravity will be high, greater than 1.025
 - Hemoglobin and hematocrit: increased due to concentrated plasma
 - Serum OSMolality: greater than 295 (thick/concentrated) 275-295 is normal
 - Serum sodium will be elevated, and BUN/Creat: increased due to hemoconcentration
 - Volume excess=
- Clinical manifestations:
- Headache weakness (can also be due to hyponatremia which usually comes along with fluid deficit)
 - Tachycardia, bounding pulse (increased volume)
 - HTN because of increased CO
 - muscle weakness (due to excess fluid retained depletes energy and increases workload)
 - Weight gain
 - Ascites
 - Dyspnea (at risk for pulmonary edema)
 - Distended neck veins increased CVP

- Edematous skin may feel cool because of fluid accumulation and decreased blood flow, may be taut and hard
 - +1 to +4 pitting
- Recognize the treatment of a patient with a fluid volume excess:
 - Strict I&O monitor
 - Weights increase 1 L fluid =2.2 lb. daily weights, teach patient
 - **Sodium monitor closely-intake=holds onto water, and restrict fluids**
 - Have patient monitor closely
 - Semi-fowlers for skin, and elevate **extremities to decrease edema**
 - Labs, esp. sodium

TREAT with diuretics and fluid restriction, try not to affect the electrolyte balance in ECF

If fluid in the transcellular (pleural) space thoracentesis

Abdominal/peritoneal: paracentesis performed

Complications: ***pulmonary edema Breath sounds big risk of: pulmonary edema: severe fluid overload.

Anxiety, JVD, PVCs, dyspnea at rest, LOC change, restless,

lethargy, crackles productive cough with pink frothy sputum

(HIGH fowlers to increase lung expansion, oxygen/vent or Bipap, nitrates, diuretics if able

- Identify how the patient with fluid volume deficit is treated.
 - Neuro. Status risk for seizures and coma watch closely
 - Weights: MOST accurate measurement of volume status, 2.2 lb. = 1000 mL of fluid retained.**
 - Monitor intake and output closely
 - Daily weights-very consistent, same time/same lines/clothes
 - Increase of 1 kg=2.2 lbs=1 liter
 - Turgor- tenting (not a good assessment in elderly)
 - Monitor intake and output
 - VS with orthostatic measurements
 - Increased risk of falls
 - Encourage call light
 - Change positions slowly
 - Monitor neuro. Status
 - Monitor weight
 - Skin-turgor
 - Oral care
 - Oral and/or IV hydration therapy
 - Rapid-Normal Saline
 - Replace water & electrolytes
 - (Lactated Ringers)
- Treatment:** need IV fluids because we need to Replace volume
- If Rapid needed-give normal saline Isotonic

- If loss of water and electrolytes: give LR
- If volume loss is due to **blood loss: administer blood**
- Shock position: legs above heart to increase blood flow (Trendelenburg)
- prevent shock
- May give colloids (PRBC, plasma, albumin or synthetic plasma expanders) and crystalloids (LR and NS)

Complications: HYPOVOLEMIC SHOCK loss of body fluid,
 MAP low, blood flow decreased perfusion to tissues, O₂, VS q 15, stay w patient,
 colloids (PRBC, plasma) Crystalloids-LR 0.9 NS, may need vasoconstrictors
 (hemodynamic monitoring)
Patients on tube feeding (especially iso-osmolar TF)
should have free water
NG tubes to suction flush with sterile saline

- **Identify the primary nursing diagnosis and explain the nursing care associated with both fluid volume excess and fluid volume deficit.**
 - Identify complications associated with each
 - Recognize treatment of complications

hypovolemia	hypervolemia
Decr. cardiac output Risk for deficient fluid volume Hypovolemic shock	Pulmonary edema ascites impaired skin integrity Activity intolerance Disturbed body image
<i>Hypotonic is: USED TO REPLACE</i> water losses and treat hypernatremia (replaces free water and treats ICF) Nursing consideration: Monitor patient when giving hypotonic fluid: for cerebral edema from cellular swelling (will see change in mentation) Isotonic: expands only the ECF –(ICF not affected inside cell) Ideal for patients with ECF volume deficit Normal saline: Used for fluid and	Initially increases osmolality of ECF and expands volume Draws water out of the cell (cell shrinks) ***Used in hyponatremia (head injury/trauma) Monitor closely @ risk: for increased intravascular fluid volume excess (Monitor: BP, lung sounds, and sodium levels closely can cause intravascular volume excess) D5 1/2NS (432) commonly used D5NS (586)

<p>sodium losses or vascular replacement in hypovolemic shock; immediate fluid replacement</p> <ul style="list-style-type: none"> diarrhea and vomiting, fluid of choice for replacement <p>Compatible with most medications and blood</p> <p>Excessive administration see increased sodium and chloride levels</p> <p>LR (274) contains NA, K, CL, Calcium, and Lactate</p> <p>Good for surgery, burns, losses from lower GI</p> <p><i>**contraindicated in hyperkalemia, liver dysfunction and lactic acidosis b/c Body unable to convert lactate to bicarbonate</i></p> <p>ALBUMIN The 5% (direct expander=increased volume-used in hypovolemia)</p>	<p>3% saline (usually ICU only) with central line access</p> <p>D10W (hypertonic) provides 340 kcal/L parenteral nutrition mixture but anything higher than 10% dextrose WILL need central line</p> <p>Plasma Expanders:</p> <p>made of large molecules increase oncotic pressure and pull fluid into blood vessels.</p> <ul style="list-style-type: none"> (made of proteins) Albumin is used often Packed PRBCs: give primarily RBC and less fluid volume If whole blood watch for circulatory overload (hx. Of heart failure) Will sometimes use diuretics after blood <p>Albumin is available in 5% and 25% solutions.</p> <p>Contrast: 25% albumin is hypertonic, draws fluid from interstitial space and following a paracentesis for ascites.</p> <p>Increases oncotic pressure to keep fluid in ECF (out of interstitial space)</p> <p>Plasma: results in plasma volume expansion equal to volume infused</p>
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- **Most common clinical manifestations and nursing management/interventions of:**

S&S	hypo	hyper
sodium	(<136) Osm (<270) Confusion, tired, cellular swelling, seizures	(>145) Osm (>300) Weakness, decreased level of consciousness, cellular shrinkage, seizures
potassium	(<3.5) Low and slow PVC,	(>5.0) Tall peaked T waves,

	VTACH, U wave, leg cramps, decr. In GI motility, decr. in BS, decr. DTR	Prolonged PR interval, wide QRS, Ventricular fibrillation, abdominal cramps, diarrhea, increase in GI motility,
calcium	(<9) Weak bones, bad teeth, severe cramping, positive chvstek and trousseau, inc. DTR, inc. BS, Tursade D-point (arrhythmia)	(>10.5) Lethargy Bone pain
magnesium	(<1.3) Tremors Inc. DTR, tremors, Tachycardia, confusion, cardiac dysrhythmias	(>2.1) Vasodilation, N/V, hypotension, dec. in DTR, cardiac arrest

Treatment	hypo	hyper
sodium	Treatment-monitor closely neuro status and confusion (seizure precautions) (in mild may see headache severe can lead to brain herniation and death)1. If due to water excess (most cases) Mild treat with water restriction 2. If severe use diuretics and/or 3% central line, pulls fluid out of cell b/c water wants to move (osmosis) where concentration is higher, give slowly monitor close (ICU, labs). If hyponatremia is due to	**Treatment: with fluid: (watch for cerebral swelling water will move into the cell) Monitor labs, neuro., cardiac Seizure precautions Treat underlying cause Primary fluid deficit: Oral hydration for primary fluid deficit: If oral fluids cannot be ingested isotonic IV fluids,(if sodium excess) give hypotonic fluids. Be careful about the rate of infusion.If serum sodium is reduced too fast can

	fluid loss, will need to replace with isotonic-containing sodium,	cause cerebral edema! Usually lab q 6 hours with minimal decrease in sodium each lab draw
potassium	<p>Treatment put on cardiac monitor-biggest issue to watch cardiac alters the resting membrane potential (hyperpolarization) flattened T wave, depressed ST segment, prolonged QRS complex</p> <ul style="list-style-type: none"> • Skeletal muscle weakness and paresthesias may occur • Decreased GI motility • impaired muscle contraction • Decreased DTRs 	<p>***Monitor closely on EKG place on cardiac monitor</p> <ul style="list-style-type: none"> • Look through meds and make sure they are off ALL forms of potassium <p>p. 1083 list of potassium rich foods.examples: <i>Fruits and vegetables (citrus: apricots, avocados, bananas and oranges) nuts/legumes , whole grain foods, meat milk, eggs, coffee). Avoid Salt substitutes, most diabetics know to watch their K before bananas etc. Potassium medications (PO, IV),Stored blood (hemolyzed)</i></p> <p>Meds: to increase elimination of K Diuretics: loop or thiazides Kayexelate binds K in bowel and exchanges for NA excreted in feces excess K excreted (oral or rectal) Add Fluids (not in a renal patient)</p>

		<p>= get dialysis</p> <p>If severe may need to move the K from ECF to ICF (this is only temporary)</p> <p>Loop diuretics increase depletion of potassium from renal system</p> <p>only if renal function is adequate</p> <p>If giving Calcium monitor BP close, if rapid administration, can cause hypotension</p> <p>If severe:</p> <p>Buys you time until you can get them to dialysis</p> <p>use IV REGULAR Insulin</p> <p>Insulin (drives K back into ICF use with dextrose to prevent hypoglycemia)</p> <p>Or sodium bicarb if correcting acidosis</p> <p>Calcium gluconate (<i>reverses membrane excitability</i>)<i>stabilizes cardiac membranes (protects until restore K to lower level)</i></p>
calcium	<p>Treat hypocalcemia with Oral or IV depending on severity</p> <p>If mild oral diet and po supplement may be enough Kale! Dairy and dark green leafy</p> <p>Need Vit d</p>	<p>Treatment: determine cause</p> <p><i>Use loop diuretic (to promote urinary excretion of calcium), Furosemide</i></p> <p>hydrate with NS (isotonic) or oral intake</p>

	<p>IV prep: calcium gluconate or calcium chloride (if severe or unable to take po) Watch for infiltration IVPB with pump over slow rate per hospital protocol –too fast cardiac arrest/hypotension</p>	<p>increase renal excretion of calcium and decrease kidney stone formation If mild can Mobilize enhance bone mineralization If hypercalcemia due to malignancy BIOPHOSPHONATES; pamidronate prevent osteoclast activity Synthetic Calcitonin can lower serum Ca levels –brings it down LOW calcium diet, avoid High calcium: milk, cheese, ice cream, yogurt, lentils, all beans except green. Fish dried fruits, nuts, cocoa and green leafy veg. Stop medications related to hypercalcemia. Increase weight bearing activity.</p>
magnesium	<p>Manage: Oral sup, dietary increase Discontinue mag depleting meds: loop diuretics, osmotic diuretics, phosphorus meds Look at other meds that would deplete Calcium such as diuretics (loop and osmotic) IV mag: NO IM</p>	<p>Not on PPT</p>

	IV give SLOWLY Oral mag-can cause diarrhea Dietary sources Dark green veg, nuts, whole grains, seafood, peanut butter, cocoa,	
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- **What are the nursing implications for the administration of the following electrolyte replacement medications: sodium, potassium, calcium, magnesium?**
 - **See administration guidelines in powerpoint and text**
- **What diagnostic tests are used to identify fluid and electrolyte imbalances?**
 - **Know normal values of Na, K, Calcium, Magnesium-use ATI for reference on numbers**
- **Differentiate between the three types of fluids:**
 - **Isotonic, hypertonic, and hypotonic and discuss why each might be given and any complications that might occur with administration.**
- **How do diuretics work?**
 - **Differentiate the actions of the three major types of diuretics: loop, thiazide and potassium sparing.**
 - **What are the nursing implications related to the administration of these medications?**
- **List the dietary food types that would contain high amounts of sodium, potassium, calcium, magnesium and phosphorus**

Foods high in Sodium:

Processed foods and lunch meats
Canned foods and prepackaged prepared foods
Soy sauce, steak sauce, seasoning salts
Bacon, cheese, olives and pickles, sausage

Foods high in Potassium:

Apricot, avocado, banana, cantaloupe, dates, dried fruits, oranges, kiwi, prunes, raisins
Artichokes, baked beans, squash, broccoli, potatoes, carrots, greens, legumes, spinach
Bran products, chocolate, granola, milk, peanut butter, yogurt, salt substitutes

Foods high in Calcium:

Milk, cheese, ice cream, yogurt, sauces containing milk, lentils, all beans except green
Fish with fine bones (i.e. sardines, kippers, herring, salmon), dried fruits, nuts, cocoa

Green leafy vegetables

Foods high in Magnesium:

Halibut, almonds, cashews, soybeans, spinach, nuts

Whole unrefined grains cereals, oatmeal, baked potato, brown rice

Green leafy vegetables

Lewis: Chapter 16 (p. 294-297) and ATI ch. 27 (p. 165-167)

Vascular Access

- Discuss the types and nursing management of commonly used venous access devices: Peripheral, central (non-tunneled and tunneled), PICC and surgically placed ports.
 - When would a central line be appropriate for treatment? Long term versus short term.

Peripheral	Short term	Check for phlebitis, infiltration, or extravasation
non-tunneled	Usually less than 6 weeks. Called subclavian, percutaneous, acute-care, or short-term catheters. This type of catheter is usually inserted into the internal jugular or subclavian vein, with the catheter tip resting in the superior vena cava just above the right atrium. Nontunneled catheters most often have multiple lumens. Indications for placement of this type of catheter include intravenous therapy, blood sampling, and central venous pressure monitoring. The femoral vein may be used if necessary. This site is not preferred, and the catheter should be removed as soon as possible due to high risk of infection. During puncture risk for pneumothorax.	Monitor for signs of infection.
tunneled	tunneled through subcutaneous tissue and exits through a separate incision on the chest or abdominal wall.	Has a Dacron cuff on the catheter serves to stabilize the catheter and may decrease the incidence of infection by impeding

		bacterial migration along the catheter beyond the cuff.
PICC	<p>PICCs are single- or multiple-lumen, nontunneled, and are up to 60 cm in length with gauges ranging from 24 to 16.</p> <p>They are used with patients who need vascular access for 1 week to 6 months but can be in place for longer periods.</p> <p>Do not use the arm with the PICC for blood pressure readings or blood drawing.</p>	<p>Complications of PICCs include catheter occlusion and phlebitis. If phlebitis occurs, it usually appears within 7 to 10 days following insertion. Change 7 days or earlier if dressing is bloody or becoming undone.</p>
Surgically placed port	<p>Implanted ports are good for long-term therapy and have a <u>low risk of infection</u>. The catheter tip lies in the desired vein and the other end is connected to a port that is surgically implanted in a subcutaneous pocket on the chest wall.</p> <p>The port consists of a metal sheath with a self-sealing silicone septum.</p> <p>To access these devices, a special Huber-point needle with a deflected tip is used to prevent damage to the rubber septum that could make the port useless.</p> <p>Huber-point needles are also available with the tip at a 90-degree angle for longer infusions</p>	<p>Drugs are placed in the port's reservoir either by injection into an already established IV line.</p> <p>Regular flushing is required to avoid the formation of "sludge" (accumulation of clotted blood and drug precipitate)</p> <p>Other surgical placed lines include=</p> <p>Specific types of long-term central catheters are Hickman catheters, which require clamps to make sure the valve is closed, and Groshong catheters, which have a valve that opens as fluid is withdrawn or infused and remains closed when not in use.</p>

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- **Advantages and disadvantages (risks) of central lines**
 - Advantages of CVADs: Immediate access to the central venous system, a reduced need for multiple venipunctures, and decreased risk of extravasation injury.
 - Disadvantages of CVADs: Increased risk of systemic infection and the invasiveness of the procedure. Extravasation can still occur if there is displacement of or damage to the device.

- **Complications associated with placement**

- pneumothorax
- A CVAD can become occluded secondary to clamping or kinking the catheter, the tip coming in contact with the vessel wall, thrombosis, or precipitate buildup in the lumen.
- Clinical manifestations of catheter occlusion include a sluggish infusion or aspiration, or being unable to infuse and/or aspirate.
- Management of catheter occlusion includes the following steps:
 - Instruct patient to change position, raise arm, and cough
 - Assess for and alleviate clamping or kinking
 - Flush with normal saline using a 10-mL syringe; do not force flush
 - Fluoroscopy to determine cause and site
 - Instillation of anticoagulant or thrombolytic agents

An embolism can occur secondary to catheter breakage, dislodgement of thrombus, and/or the entry of air into the circulation.

Clinical manifestations of embolism include chest pain, respiratory distress (dyspnea, tachypnea, hypoxia, cyanosis), hypotension, and tachycardia.

- Management of embolism includes the following steps:
 - Administer oxygen
 - Clamp catheter
 - Place patient on left side with head down (air emboli)
 - Notify physician
- Catheter-related infection can occur secondary to contamination during insertion or use, migration of organisms along the catheter, or immunosuppression.

Clinical manifestations of infection can be local (redness, tenderness, purulent drainage, warmth, edema) or systemic (fever, chills, malaise).

- Management of infection includes the following:

Local

- Culture of drainage from site
- Warm, moist compresses
- Catheter removal if indicated

Systemic

- Blood cultures
 - Antibiotic therapy
 - Antipyretic therapy
 - Catheter removal if indicated
- **A pneumothorax can occur if the pleura is perforated during insertion. Clinical manifestations include decreased or absent breath sounds, respiratory distress (cyanosis, dyspnea, tachypnea), chest pain, or distended unilateral chest. Management includes oxygen administration, semi-Fowler's position, and chest tube insertion.**
- Catheter migration or displacement can also occur. Clinical manifestations would include sluggish infusion or aspiration, edema of chest or neck during infusion, patient complaint of gurgling sound in ear, dysrhythmias, or increased external catheter length. The CVAD will need to be removed and replaced.
- **Troubleshooting clogged ports, cap exchange, and other access issues**
 - Change injection caps at regular intervals according to institution policy or if they are damaged from excessive punctures. Use strict sterile technique. Teach the patient to turn the head to the opposite side of the CVAD insertion site during cap change. If the catheter cannot be clamped, instruct the patient to lie flat in bed and perform the Valsalva maneuver whenever the catheter is open to air to prevent an air embolism.
 - Flushing is one of the most effective ways to maintain lumen patency and to prevent occlusion of the CVAD. It also keeps incompatible drugs or fluids from mixing.
 - Use a normal saline solution in a syringe that has a barrel capacity of 10 mL or more to avoid excess pressure on the catheter. If you feel resistance, do not apply force. This could result in a ruptured catheter or create an embolism if a thrombus is present. Because of the risk of contamination and infection, prefilled syringes or single-dose vials are preferred over multiple-dose vials.
 - The push-pause technique creates turbulence within the catheter lumen, promoting the removal of debris that adheres to the catheter lumen. This technique involves injecting the saline with a rapid alternating push-pause motion, instilling 1 to 2 mL with each push on the syringe plunger.
 - If using a negative-pressure cap or neutral pressure cap, clamp the catheter while maintaining positive pressure (e.g., while instilling last 1 mL of saline) to prevent reflux of blood back into the catheter.
 - If a positive-pressure valve cap is present, it works to prevent the reflux of blood and resultant catheter lumen occlusion. Remove the syringe prior to clamping the catheter in order to allow the positive pressure valve to work correctly. Clamping the catheter during flushing with this cap may actually promote blood reflux.
- **Central line dressing change: steps and what to do if dressing is not in place/alterd.**

- PICC line; Ask patient to turn his head away from insertion site. Raise height of bed and lower head of bed. Don clean gloves and mask. Inspect and palpate site around dressing for swelling or tenderness. With clean gloves, remove dressing by pulling it toward catheter insertion site. When removing transparent dressing, grasp opposite sides, pull outward, and stretch it away from insertion site. Assess site and examine catheter and hub. Remove any catheter-securing device and antimicrobial patch. Measure external portion of catheter. Compare this measurement to previous length to detect migration. Remove gloves. Discard dressing and gloves. Perform hand hygiene. Open dressing kit. Don sterile gloves. Cleanse insertion site. Cleanse skin under central line and line. Allow area to air-dry. Apply skin prep. Apply catheter-securing device or wound closure strips. Apply antimicrobial patch. Apply transparent dressing. Coil external portion of catheter and tape it in place. Leave catheter end cap exposed.
- The CVAD will need to be removed and replaced.
- **Steps in removal of central line**
 - **Put on nonsterile gloves and remove dressing.**
 - **Don sterile gloves and mask; have patient turn head to other side.**
 - **Remove sutures if present.**
 - Slowly and steadily withdraw catheter. If resistance is met, STOP.
 - If resistance is met—can apply warm compresses for 20 minutes and retry. If resistance continues, notify HCP.
 - Have patient perform the Valsalva maneuver as the last 5 to 10 cm of the catheter is withdrawn. “blow out”
 - Pressure should be immediately applied to the site with sterile gauze to prevent air from entering and to control bleeding.
 - Inspect the catheter tip to determine that it is intact.
 - After bleeding has stopped, apply an antiseptic ointment and sterile dressing to the site.

LEARNING OBJECTIVES FROM THIS PAGE DOWN

Lewis Ch: 29, 30(606-615), 30(615-625), 30(647-652), 32, 37(816-829), 16(270-278; 294-297 central line), 16(270-286)

ATI: 39, 41, 40, 35, 36, 42, 27(165-167 vascular access), 43, 44

WBC: 5,000-10,000

Hgb: (M) 14-18 (F) 12-16

Hct: (M) 42-52% (F) 37-47%

Platelets: 150,000-400,000

MCV: 80-95

MCH: 27-31

Etiology of anemia based on MCV/MCH: (Pg 607 table 30-1)

Normal size/color: Normocytic, normochromic MCV 80-100, MCH 27-34

Acute blood loss, hemolysis, chronic kidney disease, cancers, sideroblastic anemia, endocrine disorders, starvation, aplastic anemia, sickle cell anemia, pregnancy

Small size/pale color: microcytic, hypochromic MCV <80, MCH <27

Iron-deficiency anemia, vitamin B6 deficiency, copper deficiency, thalassemia, lead poisoning

Large size/normal color: macrocytic (megablastic), normochromic MCV >100, MCH >34

Cobalamin (B12) deficiency, folic acid deficiency, liver disease (including effects of alcohol abuse)

Mild/Moderate/Severe anemia: Hgb levels (slides) (pg 608 table 30-3)

Mild: 10-12 g/dL (may have response during heavy exercise: palpitations, exertional dyspnea, mild fatigue)

Moderate: 6-10 g/dL (At rest and with activity, may have bounding pulse or dyspnea, fatigue sets in)

Severe: <6g/dL (May body systems become involved when severe. Will see palor, jaundice, blurred vision, glossitis, tachycardia, murmurs, tachypnea, HA, vertigo, anorexia, sore mouth, cold, weight loss)

Iron deficiency anemia: (MCV <80, MCH<27 microcytic/hypochromic... small size/pale color)

Pathophysiology: iron involved in hemoglobin synthesis

Etiology: inadequate dietary intake, malabsorption, gastric bypass, blood loss (GI-peptic ulcer, gastritis, hemorrhoids, GU menses)

Risk factors: Women of reproductive years, poor diet, infants

Manifestations, signs and symptoms:

Pallor – due to decreased blood flow to the skin

Glossitis - Inflammation of the tongue

Cheilitis – inflammation of the lips

Headache – lack of iron to the tissue

Paresthesia – tingling and numbness in fingers/toes dt lack of iron to the tissue. Typically lack of B12. Synapse of the nerves.

Burning tongue – lack of iron to the tissue

If GI blood loss – melena – black is from the iron in the RBCs

Collaborative care:

Treat the cause: malnutrition/malabsorption/blood loss

Absorption: diet foods high in iron, how to maximize absorption (vitamin C, don't take iron with milk)

Replace iron: nutritional therapy, oral iron supplements, transfusion of PRBC (not done until symptomatic)

Interventions/medications:

Oral route: ferrous sulfate 150-200 mg TID

- Don't take with dairy, take 1 hour before meal, vitamin C increases absorption

Parenteral route: indicated for malabsorption, oral iron intolerance, increased need, poor patient compliance.

- Can be given IM or IV. IM may stain skin, use of Z-track

Nursing Diagnosis:

Pernicious anemia: Absence of intrinsic factor (slides)

Morphology: macrocytic normochromic (large size, normal color) MCV >100 MCG >34

Patho: autoimmune destruction of parietal cells or gastric mucosal atrophy

Etiology: gastric surgery, chronic ETOH, long term PPI, strict vegetarians

Risk factors: crohn's, gastric bypass, alcoholic

Clinical manifestations: sore, red, beefy tongue, anorexia, N/V, parasthesias/weakness, impaired thought process

Interventions/medications: dietary cobalamin (B12) does not correct if they are lacking intrinsic factor

Cobalamin IM 1000 mg daily x 2 weeks then weekly until normal Hgb (M-14-18, F 12-16)

Pt will die in 1-3 years if not treated, can be reversed, neuromuscular complications may not be reversed

Foods with B12: eggs, dairy, red meat, fish, liver

Folic Acid deficiency: needed for synthesis of RBC (slides)

Morphology: macrocytic normochromic (large size, normal color) MCV >100 MCG >34

Patho:

Etio: malabsorption, drugs that interfere with folic acid synthesis (methotrexate, phenytoin), alcohol abuse

Risk factors: dietary deficiency or malabsorption syndromes (crohn's, small bowel resection, celiac), alcohol abuse

Clinical manifestations/s&s: sore, red, beefy tongue, anorexia, N/V, parasthesias/weakness

Collaborative care: replacement therapy, dose is 1 mg per day, encourage pt to eat foods with large amounts of folic acid.

Foods: green leafy veggies, liver, meat, fish, legumes, whole grains, OJ, peanuts

Acute blood loss:

Patho: sudden hemorrhage, trauma, complications of surgery, conditions or diseases that disrupt vascular integrity, clotting disorder (sudden loss = hypovolemic shock)

Clinical manifestations/S&S: (slides and table 30-10 pg 615)

10%: (500mL) may have none

20%: (1000mL) tachycardia w/ exercise or slight postural hypotension

30%: (1500mL) normal at rest/ postural hypotension and tachycardia w/ exercise

40%: (2000mL) BP, CVP, CO all LOW. Air hunger, rapid thready pulse, cold clammy skin

50%: (2500mL) shock, lactic acidosis - death if not treated

Pain from internal hemorrhage (tissue distention, organ displacement, nerve compression),

Retroperitoneal bleeding (numbness, pain in the lower extremities)

Collaborative care:

Interventions/medications: replace blood volume, IV fluid to replace / volume expander, identify source (CT scan), stop blood loss (may need iron supplement)

Nursing diagnosis:

RBC destruction: spleen is sight of destruction, jaundice is seen bc bilirubin goes up.

Hemolytic anemia (NOT acquired hemolytic anemia): hepatomegaly, splenomegaly, jaundice = hemolytic anemia

Patho: Rate of destruction exceeds production.

Etio:

Risk factors:

Clinical manifestations:

Collaborative care:

Interventions/medications:

Nursing diagnoses:

Sickle cell anemia:

Patho: Hgb S RBC abnormally shaped. Adhere to wall of endothelium and block the blood flow and then less oxygen to area and over time can destroy organs. (vaso-occlusive crisis)

Etio: autosomal recessive disorder

Risk factors: African American, Mediterranean, Caribbean, Central American

Clinical manifestations (most prevalent): pain, pallor, jaundice from hemolysis, fatigue

Collaborative care: alleviate symptoms & complications, minimize organ damage by prevention, prompt treatment of serious sequelae. (prevention: avoid high altitudes, maintain adequate fluid intake, treat infections promptly, vaccines)

Interventions/medications: Oxygen, anticoagulant, hydration, rest, folic acid (because of chronic hemolysis), pain meds

Supportive treatment of:

Crisis: Oxygen, PCA pump/pain meds, hydration, anticoagulant

Chronic: vaccines (pneumo, flu), opioid tolerance so increase dose or change meds, yearly exams. Medication such as Hydrea (chemo med, increase amount of good RBC and keeps bad ones from sticking to endothelial wall)

Nursing Diagnosis: acute or chronic pain,

What educational topics/triggers would be covered with the patient of SCD to help prevent frequent crisis exacerbations? teach pt to stay hydrated, reduce illness by hand washing and avoiding being around people who are ill, watch altitude, rest. Illness can trigger crisis/ exacerbation

Blood products:

Packed RBC's:

Indications:

Special considerations:

Important nursing responsibilities:

How to recognize blood transfusion reactions:

Prevention and management of blood transfusion reactions:

Table (30-31 and 30-33)

Platelets:

Indications:

Special considerations:

Nursing responsibilities:

Fresh frozen plasma (FFP):

Indications:

Special considerations:

Nursing responsibilities:

Describe the age-related changes that occur in the hematologic system:

Thrombocytopenia:

Possible causes: inherited or acquired

Acquired: immune thrombocytopenia purpura (ITP) – autoimmune disease

Nonimmune: heparin-induced thrombocytopenia (HIT) or thrombotic thrombocytopenic purpura (TTP)

Common clinical manifestations: often asymptomatic

mucosal bleeding (epistaxis, bleeding gums)

intugument: petechiae – mico hemorrhages

purpura – bruise from numerous petechiae

ecchymoses – larger lesions from hemorrhage

gastrointestinal: splenomegaly, abd distention, guaiac positive stools

What is the collaborative treatment for:

HIT: Stop the heparin and mark their chart (should never get heparin again), direct thrombin inhibitor, start coumadin (when platelet count comes back up)

Chemo-induced thrombocytopenia:

Antihypertensive medication classifications

Diuretics: furosemide (Lasix)- more potent, loop diuretic, decreases sodium reabsorption; hydrochlorothiazide - **work in different areas of the kidneys, reduce potassium;** spironolactoneà holds in potassium

Alpha blockers: doxazosin, prazosin- **block the alpha effects, decrease SVR**, often used with patients with BPH- **biggest risk- orthostatic hypotension give at night**

ACE inhibitors: enalapril, captopril, Lisinopril- **blocks conversion of angiotensin 1 to angiotensin 2**, works on lungsà side effect **dry cough, can cause angioedema*** (swelling in lips or tongue can cause airway obstruction)** have to monitor potassium

Angiotensin II Receptor Blockers (ARB): losartan, valsartan, similar to ACE inhibitors, prevent angiotensin 2 from vasoconstriction and holding into aldosterone

Beta-blockers: metoprolol (beta 1 selective, should not affect lungs), atenolol, propranolol (noncardiospecific, can cause bronchospasms in asthma patients), decrease HR, and CO, reduce SVR, also decrease renin secretionà check heart rate before giving it because **directly** effects the heart rate (if heart rate is low around 50, hold, most times will have parameters when to hold or to give it) **big things- can cause ED, depression... do not stop abruptly**, patients with diabetes may have different glucose responses

Calcium channel blockers: amlodipine, verapamil, change the way calcium moves across cell membrane, cause vasodilation

Centrally acting Sympatholytic: clonidine, not first line do not like to use a lot, reserved for those having difficulty with therapy (ESRD, hypertensive crisis)- has a rebound effect, makes patients extremely lethargic, directly affects SNS

Vasodilators: hydralazine, not first line, usually use for HTN crisis given in an IV push- can have side effects of headache, nausea, flushing, tachycardia- **direct acting, goes straight to blood vessels**

Learning objectives HTN

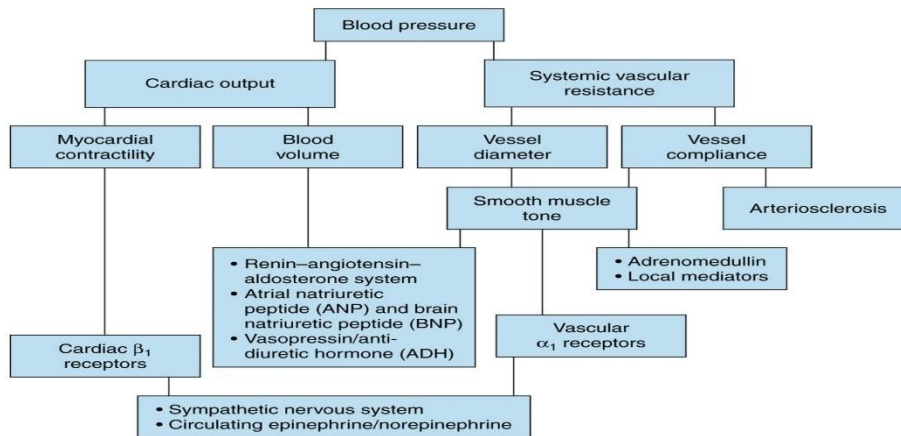
Lewis: Chapter 32

Blood pressure= cardiac output (amount of blood pumped through the left ventricle in the heart in one minute) **x systemic vascular resistance** (force of the vessel walls→ vasoconstriction/vasodilation)

- 1) Define hypertension, pre-hypertension and recognize diagnostic BP parameters

Category	Blood Pressure, mm Hg
Normal	SBP \leq 120 and DBP \leq 80
Prehypertension	SBP 120-139 or DBP 80-89
Stage 1 Hypertension	SBP 140-159 or DBP 90-99
Stage 2 Hypertension	SBP \geq 160 or DBP \geq 100

- a. Need to know for test**
- b. Hypertension→
- c. Pre-hypertension→
- d. Diagnostic BP parameters→
 - i. 60 years and over
 1. Start medication therapy for
 - a. \geq 150 or \geq 90
 - b. Goal is less than 150/90
 - ii. Younger than 60 years old
 1. Less than 140/90 (on medication)
 - iii. Chronic kidney disease or DM
 1. Less than 140/90 (on medication)

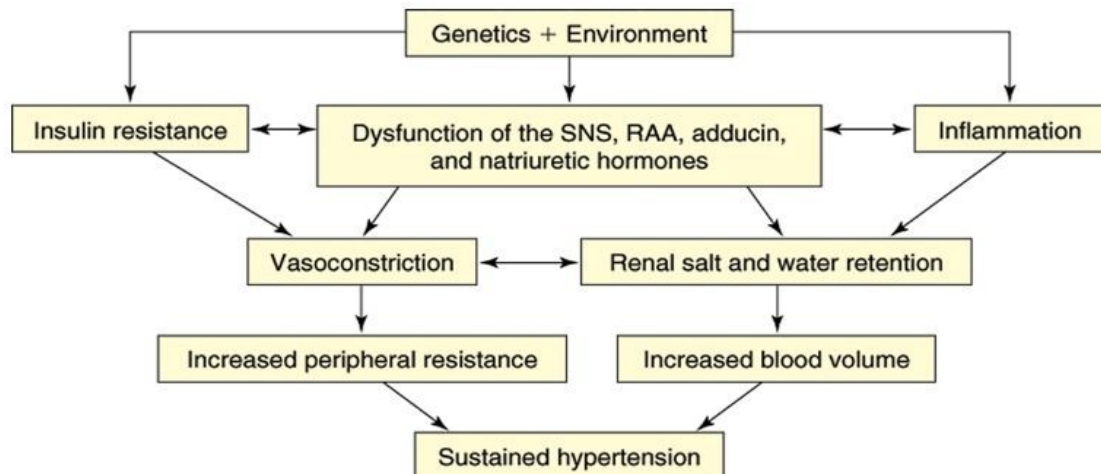


e.
f.

2) Describe the pathophysiologic mechanisms associated with primary HTN.

- Majority of all cases
- Do not know exact cause**
- Increased sympathetic system response
- RAAS system* → angiotensin two increased blood pressure in two way → 1. Tells adrenal cortex to hold onto aldosterone (increasing fluids) 2. Potent vasoconstrictor affecting arterioles (increasing SVR)**

e.



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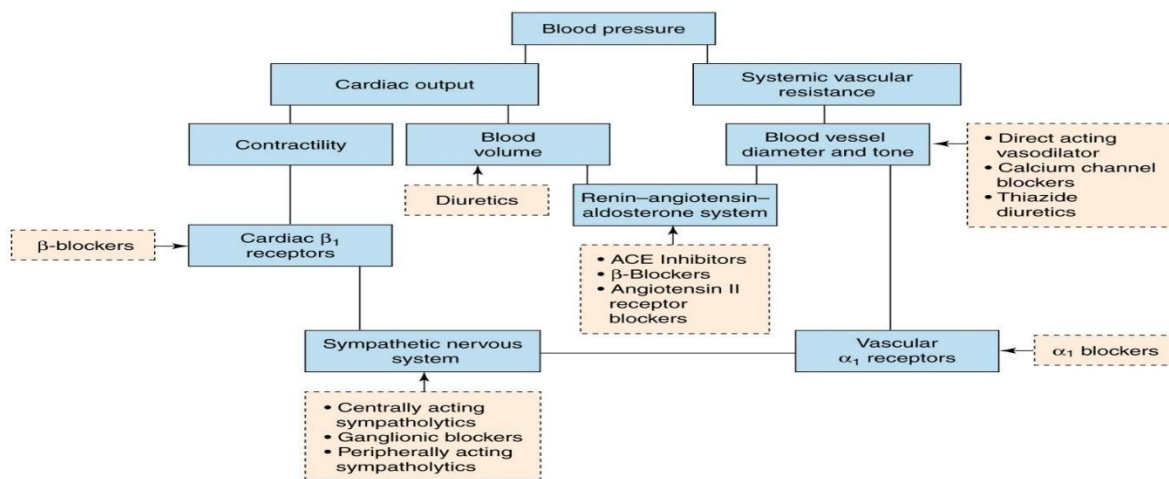
3) Relate the difference between primary and secondary hypertension.

- Primary hypertension→
 - Essential or idiopathic
 - Elevated BP without an identified cause
 - Accounts for 90-95% of all cases**
 - Contributing factors**
 - ↑ SNS activity** stress, not sleeping, type A personality
 - ↑ sodium-retaining hormones & vasoconstrictors**
renin overdrive
 - ↑ sodium intake**

4. **> ideal body weight** obesity
5. **Diabetes mellitus**
6. **Excessive alcohol consumption**
- b. Secondary hypertension→
 - i. Elevated BP with a specific cause
 - ii. **Accounts for 5-10% of adult cases**
 - iii. **Contributing factors**
 1. **Cirrhosis** changes in the blood flow, causes strain to right side of heart
 2. **Coarctation of aorta** change in the way the blood flows
 3. **Endocrine disorders** Cushing's, Hyperthyroidism
 4. **Medications** norepinephrine, cocaine, birth control
 5. **Neurologic disorders**
 6. **Renal disease** any time there is renal dysfunction it effects it, kidneys sense they are not getting enough blood causes the increase
 7. **Sleep apnea** strain on your heart
- 4) Discuss risk factors, clinical manifestations diagnostic testing and complications of hypertension.
 - a. Risk factors→
 - i. Age
 - ii. Alcohol
 - iii. Cigarette smoking→ toxic to endothelial lining, causes vasoconstriction and change to cells
 - iv. Diabetes mellitus
 - v. Elevated serum lipids→ hyperlipidemia
 - vi. Excess dietary sodium
 - vii. Gender→ men at higher risk until age of 64, and then women at higher risk after menopause
 - viii. Family history
 - ix. Obesity
 - x. Ethnicity → African Americans
 - xi. Sedentary lifestyle→ not exercising
 - xii. Socioeconomic status
 - xiii. Stress→ increases sympathetic nervous system
 - b. Clinical manifestations→ Referred to as the "silent killer" because patients are frequently asymptomatic until target organ disease occurs
 - i. Fatigue
 - ii. Reduced activity tolerance
 - iii. Dizziness
 - iv. Palpitations
 - v. Angina
 - vi. Dyspnea
 - c. Diagnostic testing→ table 32-11
 - i. **At least 2 elevated blood pressures within 2 weeks**
 - ii. Use both arms, need to be seated for five minutes with feet on floor, empty bladder
 - iii. **Biggest thing→ correct size of blood pressure cuff**
 - iv. Routine urinalysis→ checking for protein
 - v. BUN/Creat
 - vi. Serum electrolytes-Serum cholesterol and glucose levels→ baseline, looking at other risk factors

- vii. Serum cortisol levels → to rule out Cushing's
- viii. ECG → can look for myocardial ischemia, will also have a tall R wave if they have left ventricular hypertrophy
- ix. Echocardiogram → look at ejection factors and left ventricle
- x. Chest x-ray → show if heart is in large
- d. Complications →
 - i. heart → usually first organ affected (hypertensive heart disease)
 - 1. coronary artery disease → endothelium stiff and narrow, can have plaques
 - 2. left ventricular hypertrophy
 - 3. heart failure
 - a. left sided heart failure (pulmonary edema) more common with HTN
 - b. right sided heart failure (JVD, peripheral edema)
 - ii. brain → cerebrovascular disease
 - 1. atherosclerosis → vessels that go up to the brain
 - 2. stroke → 4 times more likely to have a stroke if you have HTN, ischemic or hemorrhagic
 - 3. hypertensive encephalopathy → protective regulation from the brain that does not work, increased intracranial pressure, edema
 - iii. Peripheral vasculature (peripheral vascular disease)
 - 1. Intermittent claudication → legs are not getting enough oxygen
 - iv. Kidneys (nephrosclerosis) → narrowing and atrophy of renal tubules
 - 1. End-stage renal disease
 - 2. Lab indications of renal dysfunction: **microalbuminuria**, proteinuria, microscopic hematuria, ↑ serum creatinine and BUN (these are late sign)
 - 3. Nocturia – earliest manifestation of renal dysfunction
 - v. Eyes (retinal damage)
 - 1. blurred vision
 - 2. Retinal hemorrhage
 - 3. Loss of vision
- 5) Discuss strategies for the prevention of hypertension.
 - a.
- 6) Describe the collaborative care for hypertension, including drug therapy and lifestyle modifications (DASH diet, exercise)
 - a. Consider strategies for adherence to regimes
 - b. **Maintain BP goal and decrease risk of CV and target organ damage**
 - c. **Lifestyle Modifications:**
 - d. Weight reduction – weight loss of 22 lb (10 kg) may ↓ SBP by approximately 5 to 20 mm HG
 - e. DASH Diet – fruits, vegetables, fat-free or low-fat milk and milk products, whole grains, fish, poultry, beans, seeds, and nuts
 - f. Dietary sodium restriction – ≤2300 mg/day
 - g. Omega-3 fatty acids
 - h. Limit caffeine
 - i. Moderation of alcohol consumption

- i. Men: no more than 2 drinks/day
 - ii. Women: no more than 1 drink/day
- j. Physical activity
- k. Avoidance of tobacco products
- l. Management of psychosocial risk factors
- 7) Discuss the differences and care of the older adult with hypertension.
 - a. 60 years and over
 - i. Start medication therapy for 150/90 and up, but goal is less than 150/90
 - ii. **JNC8 criteria**
 - b. Isolated systolic hypertension (ISH) – most common form of hypertension in individuals >50 years of age
 - c. “White coat” hypertension
 - d. Auscultatory gap – wide gap between the first Korotkoff sound and subsequent beats
Failure to inflate the cuff high enough may result in seriously underestimating SBP
 - e. More likely to experience medication interactions
 - f. More likely to experience orthostatic hypotension
- 8) Recognize cultural differences of both incidence and treatment of HTN.
 - a. African Americans increased risk of HTN 2x more likely
 - b. Mexican Americans are less likely to receive treatment than whites
- 9) Discuss the various age-related changes in the cardiovascular system
 - a.
- 10) Discuss the goal of pharmacologic management of HTN (JNC 8 guidelines), recognize the main drug classifications, (prototype examples) and the primary nursing implications and precautions associated with each.



two main actions of antihypertensive medications→

5. Decrease the volume of circulating blood
6. Reduce SVR

3

Specifically:

Diuretics: furosemide (Lasix)→ more potent, loop diuretic, decreases sodium reabsorption; hydrochlorothiazide → **work in different areas of the kidneys, reduce potassium**; spironolactone→ holds in potassium

Alpha blockers: doxazosin, prazosin→ block the alpha effects, decrease SVR, often used with patients with BPH→ **biggest risk→ orthostatic hypotension** give at night

ACE inhibitors: enalapril, captopril, **Lisinopril**→ blocks conversion of **angiotensin 1 to angiotensin 2**, works on lungs→ side effect **dry cough, can cause angioedema***** (swelling in lips or tongue can cause airway obstruction) have to monitor potassium

Angiotensin II Receptor Blockers (ARB): losartan, valsartan, similar to ACE inhibitors, prevent angiotensin 2 from vasoconstriction and holding into aldosterone

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Vasodilators: hydralazine, not first line, usually use for HTN crisis given in an IV push→ can have side effects of headache, nausea, flushing, tachycardia→**direct acting, goes straight to blood vessels**

11. Hypertensive crisis

BP above 240/120, need to go to ICU or ER cannot be treated on med surge floor

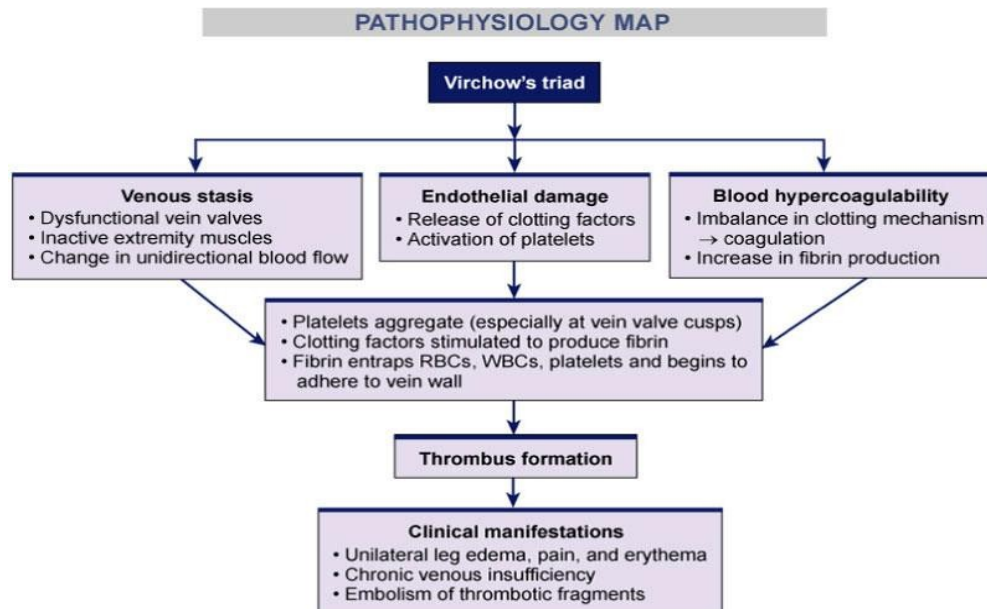
Happens when **NONCOMPLIANT TO PRESCRIBED MEDICATIONS**

- **REFER, REFER, REFER>>>Hospitalization**
- IV drug therapy: titrated to mean arterial pressure (MAP) $MAP = (SBP + 2DBP) / 3$
- Monitor cardiac and renal function
- Neurologic checks (pupils, LOC), cerebral edema
- Determine cause
- Education to avoid future crises

Learning Objectives Vascular Disorders

Chapter 37 (pgs. 816-829)

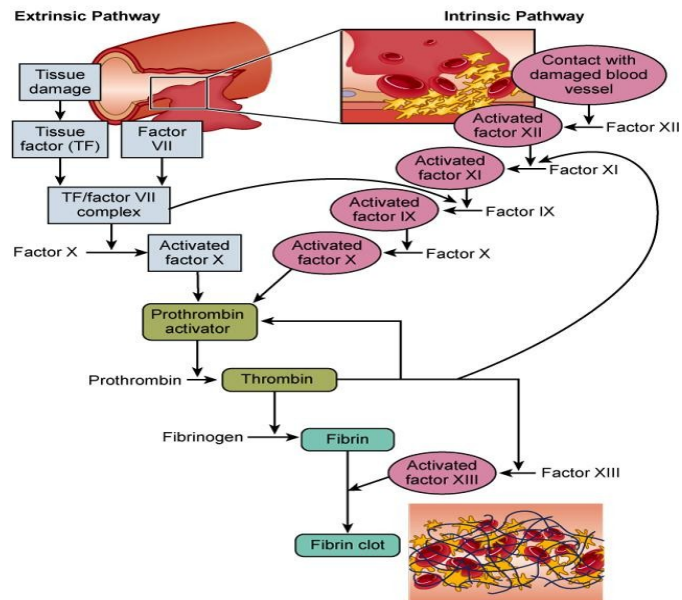
- 2) Discuss pathophysiology, clinical manifestations and treatment of superficial and deep venous thrombosis. **Page 817**



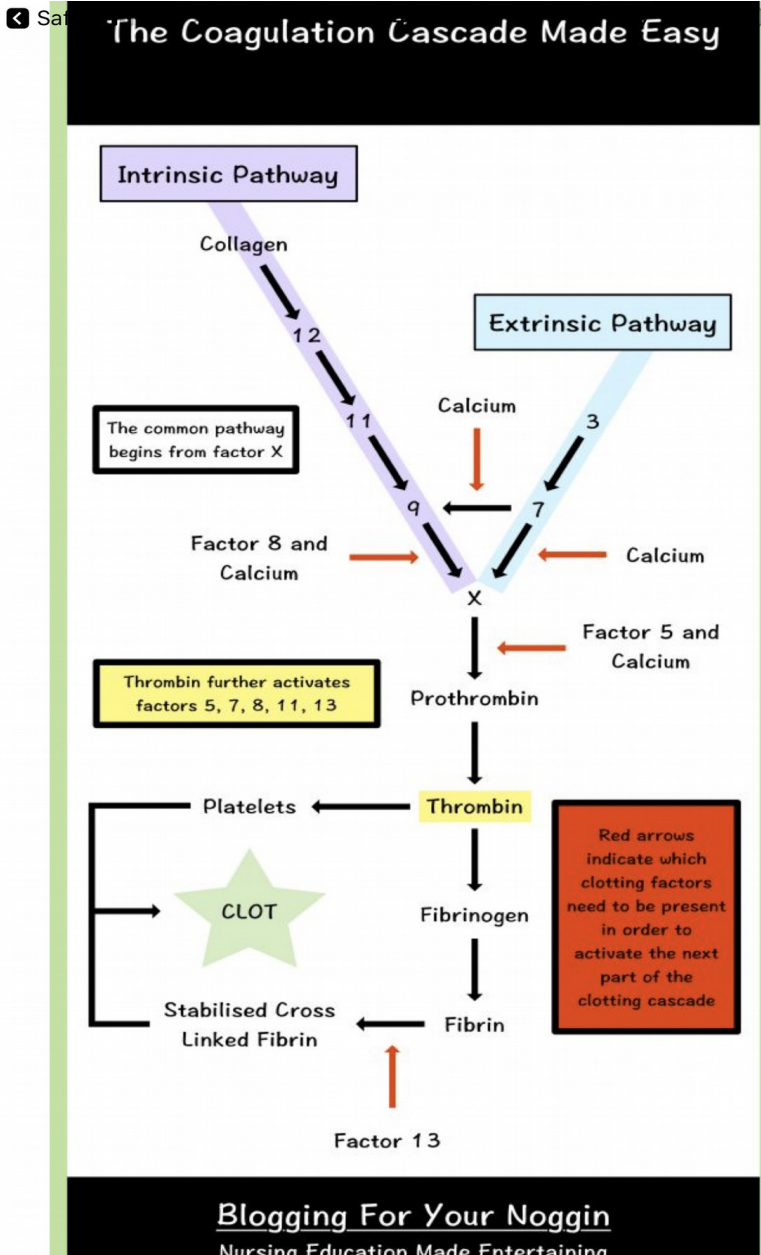
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- a.
 - b. Phlebitis→ inflammation of the walls of the veins, usually in smaller cannulated veins in hands and arms, certain medications can cause it (especially potassium)
 - c. Venous thrombosis→ blood clot, formation of the thrombus in vein, then classify as superficial or deep (deep more worrisome→ bigger veins that lead to lungs→ ileac and femoral)
 - d. Superficial vein thrombosis (SVT)
 - e. Deep vein thrombosis (DVT)
 - f. VTE –
 - i. Represents the spectrum of pathology from DVT to PE
 - ii. from DVT to pulmonary embolism (PE)
- 3) **SVT→**
- a. Clinical manifestations:
 - i. Superficial Veins:
 - ii. leg veins and arm veins
 - iii. Untreated→ can extend to deeper veins →VTE
 - iv. Tender to touch
 - v. reddened, warm
 - vi. inflammation and induration
 - vii. Palpable cord
 - viii. Mild systemic temperature elevation & leukocytosis
 - ix. **Edema rarely occurs**
- 4) **DVT→ more dangerous**
- a. Clinical manifestations:
 - i. Deep veins of:
 - ii. arms, legs, pelvis, pulmonary system
 - iii. Tender to pressure
 - iv. Over the vein, venous distention, and EDEMA
 - v. Change in leg circumference
 - vi. **** Some do not have any obvious changes in the Physical assessment**
 - vii. May break off and → travel to the lungs (pulmonary embolism)→ can result in death

- 5) Identify the risk factors predisposing the development of superficial thrombophlebitis and deep vein thrombosis.
 - a.
- 6) Recognize the main nursing diagnosis associated with venous thrombosis.
 - a. Pain, lack of knowledge
- 7) Discuss the interventions (both collaborative and independent) for DVT prevention.
 - a. **Ambulation is the most affordable and effective**
 - b. Early mobilization.....leg exercises and out of bed often
 - c. Ted hose.....fitted correctly
 - d. SCD's.....unless an active VTE
 - i. **Must be on the patient to work!!!!!!**
 - ii. If patient does not want them, document you discussed importance
 - e. **Anticoagulation therapy – PREVENT CLOTS**
 - i. **Do not want to put on if already bleeding, have a history of bleeding (ulcers)**
 - f. Complications
 - i. **Pulmonary embolism (life-threatening)**
 - ii. Chronic venous insufficiency – valve destruction
- 8) Discuss specifically the role of the following medications in the treatment of DVT: warfarin, heparin (both unfractionated and low molecular weight (LMW), thrombolytic
 - a.
- 9) Describe the nursing management of the patient receiving anticoagulation therapy.
 - a.
- 10) Recognize the goal and management of the following medications used for thrombosis prevention and/or treatment:
 - a. **Anticoagulants:** used to prevent clot formation for those high at risk, if have a clot already just stops it from getting bigger
 - i. heparin sodium, enoxaparin,
 - ii. **warfarin→ PO only**, useful for long term, **vitamin K antagonist**
 - iii. **indirect thrombin inhibitor→ heparin**
 1. unfractionated heparin (UFH)→ **heparin sodium** subcutaneous, do not massage area, do not aspirate, if given IV then it is the drip (give for people having an active MI as well, works immediately)
 2. low-molecular weight heparin (LMWH)→ **LOVENOX (enoxaparin)**→ given subcutaneous, can give for people who have a DVT and are treating it, higher dose (pre-filled), cannot use in patients with renal failure, less risk of heparin induced thrombocytopenia, treatment of choice
 3. **high dose→ have DVT, low dose→ prevenative**
 - iv. **PATIENTS ON WARFARIN→** should not take herbal supplements, garlic, ginkgo biloba, NSAIDS
 - b. Antiplatelets: aspirin
 - c. Thrombolytics: streptokinase, alteplase
 - d. Treatment of Heparin overdose: protamine sulfate
 - e. Treatment of warfarin overdose: vitamin K



- f.
- g. Intrinsic → heparin
- h. Extrinsic → warfarin



- 11) Know both the normal baseline values and therapeutic levels while on anticoagulants for PT, PTT, INR
 - a. INR → warfarin
 - b. PTT → heparin
- 12) Discuss the pathophysiology, clinical manifestations and collaborative care and nursing management of patients with varicose veins, venous insufficiency, and venous leg ulcers.
 - a. Varicose veins →
 - b. Venous insufficiency →
 - c. Venous leg ulcers →

ATI ch 27 Cardiovascular diagnostic and therapeutic procedures

1. A nurse is orienting a newly licensed nurse on the care of a client who is to have a line placed for hemodynamic monitoring. Which of the following statements by the newly licensed nurse indicates effectiveness of the teaching?

- a. A chest x-ray is needed to verify placement after the procedure.

(The nurse should ensure that a chest xray is obtained to confirm proper placement of the lines following placement)

2. A nurse is assessing a client who is undergoing hemodynamic monitoring. The client has a CVP of 7 mm Hg and a PAWP of 17 mm Hg. Which of the following findings should the nurse expect?
 - a. Bilateral crackles in the lungs
 - b. Jugular vein distension
 - c. Hepatomegaly

3. A nurse is teaching a client who is scheduled for an angiography. Which of the following statements should the nurse include in the teaching?

- a. You will need to keep your affected leg straight following the procedure.

(Nurse should instruct the client for the need to remain on bed rest in the supine position with the affected leg straight for a prescribed amount of time. This position decreases the client's risk for bleeding and hematoma formation at the catheter insertion site)

4. A nurse at a provider's office is reviewing the laboratory test results for a group of clients. The nurse should identify that which of the following results indicates the client is at risk for heart disease?
 - a. Cholesterol (total) 245 mg/dL
 - b. LDL 140 mg/dL

(client with cholesterol level >200mg/dL is at an increased risk for heart disease. Client with an LDL [bad cholesterol] > 130mg/dL is at an increased risk for heart disease.)

5. A nurse planning care for a client who has a PICC line in the right arm. Which of the following interventions should the nurse include in the plan of care?
 - a. Use a 10mL syringe to flush the PICC line
 - b. Cleanse ports with alcohol for 15 seconds prior to use
 - c. Maintain a transparent dressing over the insertion site

(Nurse should use a 10mL syringe to flush the PICC line to avoid excess pressure that could cause catheter fracture/rupture. The nurse should clean the insertion ports for 15 sec and allow it to air dry prior to use. This action decreases the risk for bacterial contamination. The nurse should maintain a transparent dressing over the insertion site to decrease the risk for infection and allow for visualization. The nurse should plan to change the dressing at least every 7 days and when wet, loose, or soiled.)

INTERPRETATION OF FINDINGS

27.1 Cardiac enzymes

EXPECTED REFERENCE RANGE	ELEVATED LEVELS FIRST DETECTABLE FOLLOWING MYOCARDIAL INJURY	EXPECTED DURATION OF ELEVATED LEVELS
--------------------------------	---	---

*Creatine kinase MB isoenzyme
more sensitive to myocardium*

0% of total CK (30 to 170 units/L)	3 to 6 hr	2 to 3 days
---------------------------------------	-----------	-------------

Troponin T

Less than 0.1 ng/mL	2 to 3 hr	10 to 14 days
---------------------	-----------	---------------

Troponin I

Less than 0.03 ng/mL	2 to 3 hr	7 to 10 days
-------------------------	-----------	--------------

Myoglobin

Less than 90 mcg/L	2 to 3 hr	24 hr
--------------------	-----------	-------

27.2 Cardiac tests

EXPECTED REFERENCE RANGE	PURPOSE
-----------------------------	---------

Cholesterol (total)

Less than 200 mg/dL	Screening for heart disease
---------------------	-----------------------------

LDL

Less than 130 mg/dL	"Bad" cholesterol Transports cholesterol to the body's cells <u>from the liver</u>
---------------------	--

Triglycerides

MALES: 40 to 160 mg/dL FEMALES: 35 to 135 mg/dL	Evaluates the client's risk for heart disease
--	--

HDL

FEMALES: greater than 55 mg/dL MALES: greater than 45 mg/dL	"Good" cholesterol Protects coronary arteries from heart disease by transporting cholesterol from the body's cells <u>to the liver</u>
--	---

INTRAPROCEDURE

Monitor for manifestations of altered hemodynamics.

27.3 Manifestations of altered hemodynamics

<i>Preload</i>		<i>Afterload</i>	
RIGHT HEART: CVP LEFT HEART: PAWP		RIGHT HEART: pulmonary vascular resistance LEFT HEART: systemic vascular resistance	
ELEVATED	DECREASED	ELEVATED	DECREASED
Crackles in lungs	Poor skin turgor	Cool extremities	Warm extremities
Jugular vein distention	Dry mucous membranes	Weak peripheral pulses	Bounding peripheral pulses
Hepatomegaly			
Peripheral edema			
Taut skin turgor			

ATI ch 35 Peripheral vascular diseases

1. A nurse is caring for a client who has chronic venous insufficiency and a prescription for thigh-high compression stockings. Which of the following actions should the nurse take?

- a. Apply the stockings in the morning upon awaking and before getting out of bed

(applying in the am before getting out of bed reduces venous stasis and assists in the venous return of blood to the heart. Legs are less adenomatous at this time)

2. A nurse is assessing a client who has chronic peripheral arterial disease (PAD). Which of the following findings should the nurse expect?

- a. Pallor on elevation of the limbs, and rubor when the limbs are dependent

3. A nurse is teaching a client who has been a new diagnosis of severe peripheral arterial disease. Which of the following instructions should the nurse include?

- a. Place both legs in dependent position while sleeping

(the nurse should instruct the client to place his legs in a dependent position, such as hanging off the edge of the bed while sleeping. This can alleviate swelling and discomfort of the legs.)

4. A nurse is teaching a client who has a new prescription for clopidogrel. Which of the following instructions should the nurse include in the teaching?

- a. Avoid the consuming grapefruit while taking this medication

- b. Monitor for the presence of black, tarry stools

(clopidogrel is an antiplatelet {arteries - prevent platelet aggregation}; the nurse should instruct the client to avoid consuming grapefruit while taking clopidogrel. Grapefruit interferes with absorption of clopidogrel and can cause severe complications. The nurse should instruct the client to monitor for evidence of GI bleeding, such as abdominal pain, coffee-ground emesis, or black, tarry stools. If this occurs, client should report to HCP.

5. A nurse is caring for a client who has a deep-vein thrombosis (DVT) and has been taking unfractionated heparin for 1 week. Two days ago, the provider also prescribed warfarin. The client asks the nurse about receiving both heparin and warfarin at the same time. Which of the following statements should the nurse give?
 - a. It takes 3 to 4 days before the therapeutic effects of warfarin are achieved, and then the heparin can be discontinued

(Warfarin depresses synthesis of clotting factors but does not have an effect on clotting factors that are present. It takes 3 to 4 days for the clotting factors that are present to decay and for the therapeutic effects of warfarin to occur.)

ATI ch 36 Hypertension

1. A nurse is screening a male client for hypertension. The nurse should identify that which of the following actions by the client increases his risk for hypertension?
 - a. Eating popcorn at the movie theater
 - b. Consuming 36 oz beer daily

(Movie popcorn contains large quantity of sodium and fat - increases risk for hypertension. Consuming more than 24 oz of beer per day for a male client increases the risk for hypertension)

2. A nurse in an urgent care clinic is obtaining a history from a client who has type 2 diabetes mellitus and a recent diagnosis of hypertension. This is the second time in 2 weeks that the client experienced hypoglycemia. Which of the following client data should the nurse report to the provider?
 - a. Takes metoprolol daily after meals

(Metoprolol can mask the effects of hypoglycemia in clients who have DM)

3. A nurse is caring for a client who is admitted to the emergency department with a BP of 266/147 mm Hg. The client reports a headache and double vision. The client states that she ran out of her diltiazem 3 days ago, and is unable to purchase more. Which of the following actions should the nurse take first?
 - a. Obtain an IV access and prepare to administer an IV antihypertensive.

(The greatest risk to the client is injury due to a BP of 266/147, which can be life-threatening and should be lowered as soon as possible. Obtaining IV access will permit administration of an IV hypertensive, which will act more rapidly than an oral med)

4. A nurse is providing teaching for a client who has a new diagnosis of hypertension and a new prescription for spironolactone 25mg/day. Which of the following statements by the client indicates an understanding of the teaching?
 - a. I will report any changes in heart rate to my provider

(The nurse should teach the client to monitor her HR and report changes to her provider)

5. A nurse is providing discharge teaching for a client who has a prescription for furosemide 40mg PO daily. The nurse should instruct the client to take this medication at which of the following times of day?
 - a. Morning

(The client should take furosemide, a diuretic, in the morning so that the peak action and duration of the med occurs during waking hours)

ATI ch 39 Hematologic diagnostic procedures

1. A nurse in a clinic is caring for a client who has suspected anemia. Which of the following laboratory test results should the nurse expect?
 - a. Hgb 10g/dL

(10g/dL is below the expected reference range and is an expected finding of anemia)

2. A nurse is caring for a client who is receiving warfarin for anticoagulation therapy. Which of the following laboratory test results indicates to the nurse that the client needs an increase in the diagnosis?
 - a. INR 1.1

(INR of 1.1 is within the expected reference range for someone who is not taking warfarin. This value is subtherapeutic for anticoagulation therapy. The nurse should expect the client to receive an increased dose of warfarin until the INR is 2 to 3)

3. A nurse is providing teaching for a client who is scheduled for a bone marrow biopsy of the iliac crest. Which of the following statements made by the client indicates an understanding of the teaching?
 - a. I need to stay in bed for about an hour after the test

(Need to be on bed rest for 30 to 60 mins following the test to reduce risk for bleeding)

39.1 Expected reference ranges for blood diagnostic procedures

	EXPECTED REFERENCE RANGE	INTERPRETATION OF FINDINGS
RBC	Females: 4.2 to 5.4 million/uL Males: 4.7 to 6.1 million/uL	Elevated level: erythrocytosis, polycythemia vera, severe dehydration Decreased level: anemia, hemorrhage, kidney disease
WBC	5,000 to 10,000/mm ³	Elevated level: infection, inflammation. Decreased level: immunosuppression, autoimmune disease
MCV	80 to 95 fL	Elevated level: macrocytic (large) RBCs, megaloblastic anemia. Decreased level: microcytic (small) RBCs, iron deficiency anemia.
MCH	27 to 31 pg/cell	Elevated/decreased level: same as above for MCV
TIBC	250 to 460 mcg/dL	Elevated level: iron deficiency anemia, polycythemia vera Decreased level: malnutrition, cirrhosis, pernicious anemia
IRON	Females: 60 to 160 mcg/dL Males: 80 to 180 mcg/dL	Elevated level: hemochromatosis, iron excess, liver disorder, or lead toxicity. Decreased level: iron deficiency anemia, chronic blood loss, inadequate dietary intake of iron.
PLATELETS	150,000 to 400,000 mm ³	Increased level: malignancy, polycythemia vera, rheumatoid arthritis. Decreased level: enlarged spleen, hemorrhage, leukemia
HGB	Females: 12 to 16 g/dL Males: 14 to 18 g/dL Elderly: levels slightly decreased	Elevated level: erythrocytosis, COPD, severe dehydration Decreased level: anemia, hemorrhage, kidney disease
HCT	Females: 37 to 47% Males: 42 to 52% Elderly: levels slightly decreased	Elevated /decreased level: same as above for Hgb
APTT	30 to 40 seconds (1.5 to 2.5 times the control value if receiving heparin therapy)	Increased time: vitamin K deficiency, disseminated intravascular coagulation (DIC), liver disease, heparin administration Decreased time: extensive cancer
PT	11 to 12.5 seconds, 85 to 100%, or 1:1.1 client-control ratio	Increased time: of clotting factors II, V, VII, or X, liver disease, warfarin therapy, disseminated intravascular coagulation Decreased time: vitamin K excess, pulmonary embolus, thrombophlebitis
INR	0.8 to 1.1 (desired goal of 2 to 3 on warfarin therapy)	Measures the mean of PT to provide a universally recognized value. Elevated level: warfarin therapy Decreased level: cancer disorders
D-DIMER	Less than 0.4 mcg/mL	Positive result: disseminated intravascular coagulation, malignancy Negative result: can rule out pulmonary embolus or deep vein thrombosis
FIBRINOGEN LEVELS	200 to 400 mg/dL	Elevated level: acute inflammation, acute infection, heart disease Decreased levels: liver disease, advanced cancer, malnutrition
FIBRIN DEGRADATION PRODUCTS	Less than 10 mcg/mL	Elevated level: disseminated intravascular coagulation, massive trauma resulting in fibrinolysis Decreased level: anticoagulation therapy

ATI ch 40 Blood and blood product transfusions

1. A nurse is preparing to administer packed RBC to a client who has a Hgb of 8g/dL.

Which of the following actions should the nurse plan to take during the first 15 mins of the transfusion?

- a. Assess for acute hemolytic reaction.

(nurse should assess for acute hemolytic reaction during the first 15 min of the transfusion. This form of a reaction can occur following the transfusion of as little as 10mL of blood product)

2. A nurse is caring for a client who is receiving a blood transfusion. Which of the following actions should the nurse expect if an allergic transfusion reaction is suspected?

- a. Stop the transfusion
- b. Maintain an IV infusion with 0.9% sodium chloride
- c. Administer diphenhydramine

(Immediately stop the transfusion if an allergic reaction is suspected. The nurse should administer 0.9% sodium chloride through new IV tubing if an allergic transfusion reaction is suspected. The nurse should administer an antihistamine, such as diphenhydramine, if an allergic transfusion is suspected)

3. A nurse is monitoring a client who began receiving a unit of packed RBCs 10 minutes ago. Which of the following findings should the nurse identify as an indication of a febrile transfusion reaction?
 - a. HR changed from 88/min pretransfusion to 120/min
 - b. Client appears flushed

(Tachycardia and flushed appearance is an indication of febrile transfusion reaction.)

4. A nurse is providing preoperative teaching for a client who requests autologous donation in preparation for scheduled orthopedic surgical procedure. Which of the following statements should the nurse include in the teaching?
 - a. You can donate blood each week if your hemoglobin is stable

(Beginning 6 weeks prior to surgery, the client can donate blood each week for autologous transfusion if his Hgb and Hct remain stable.)

5. A nurse preceptor is observing a newly licensed nurse on the unit who is preparing to administer a blood transfusion to an older adult client. Which of the following actions by the newly licensed nurse indicates an understanding of the procedure?
 - a. Obtains vital signs every 15 min throughout procedure.

(The nurse should check the older adult client's vital signs every 15 mins throughout the transfusion to allow for early detection of fluid overload or other transfusion reaction.)

ATI ch 41 Anemias

1. A nurse is planning care for a client who has Hgb 7.5 g/dL and Hct 21.5%. Which of the following actions should the nurse include in the plan of care?
 - a. Provide assistance with ambulation
 - b. Monitor oxygen saturation
 - c. Obtain stool specimen for occult blood
 - d. Schedule daily rest periods

(Nurse should assist pt with ambulation bc pt who has anemia can experience dizziness.

Monitor oxygen saturation bc of decreased oxygen-carrying capacity of the blood. Stool occult test to identify possible cause of anemia caused from GI bleed. Rest to conserve energy)

2. A nurse is teaching a client who has a new prescription for ferrous sulfate. Which of the following information should the nurse include in the teaching?
 - a. Food high in vitamin C will promote absorption

(Vitamin C enhances absorption of iron by the intestinal tract)

3. A nurse is providing discharge teaching to a client who had a gastrectomy for stomach cancer. Which of the following information should the nurse include in the teaching?
 - a. You will need a monthly injection of vitamin B12 for the rest of your life
 - b. Using the nasal spray form of vitamin B12 on a daily basis can be an option

(B12 monthly bc of lack of intrinsic factor being produced by parietal cells of the stomach.

Cyanocobalamin nasal spray is an option for a client who had gastrectomy)

4. A nurse is completing an integumentary assessment of a client who has anemia. Which of the following findings should the nurse expect?
 - a. Spoon-shaped nails

(deformities of the nails, such as being spoon-shaped, are findings of a client who has anemia)

5. A nurse in a clinic receives a phone call from a client seeking information about a new prescription for erythropoietin. Which of the following information should the nurse review with the client?

- a. The client should have his hemoglobin checked twice a week

(Hgb & Hct monitored twice a week until targeted levels are reached)

41.2 RBC indices			
	NORMAL MCV, MCH, MCHC	DECREASED MCV, MCH, MCHC	INCREASED MCV
CLASSIFICATION	Normocytic, normochromic anemia	Microcytic, hypochromic anemia	Macrocytic anemia
POSSIBLE CAUSES	Acute blood loss Sickle-cell disease	Iron-deficiency anemia Anemia of chronic illness Chronic blood loss	Vitamin B ₁₂ deficiency Folic acid deficiency

ATI ch 42 Coagulation disorders

1. A nurse is caring for a client who has disseminated intravascular coagulation (DIC).

Which of the following laboratory values indicates the client's clotting factors are depleted?

- a. Platelets 100,000/mm³
 - b. Fibrinogen levels 57 mg/dL

(In DIC, platelet levels are decreased, causing clotting factors to become depleted. Clotting times are increased, which raises the risk for fatal hemorrhage. In DIC, fibrinogen levels are decreased, causing clotting factors to become depleted.

Clotting times are increased, which raises the risk for fatal hemorrhage. ↓ platelets
↑ clotting time; Fibrinogen ↓, clotting factors depleted, ↑ clotting times)

2. A nurse is assessing a client and suspects the client is experiencing DIC. Which of the following physical findings should the nurse anticipate?

- a. Epistaxis

(epistaxis is unexpected bleeding of the gums and nose and is a finding indicative of DIC)

3. A nurse is caring for a client who has idiopathic thrombocytopenic purpura (ITP). The nurse should notify the provider and report possible small-vessel clotting when which of the following is assessed?

- a. Cyanotic nail beds

(Cyanotic nail beds indicate microvascular clotting is occurring and should be immediately reported to avoid ischemic loss of the fingers or toes.)

4. A nurse is caring for a client who has DIC. Which of the following should the nurse anticipate administering?

- a. Heparin

(Heparin can be administered to decrease the formation of microclots, which deplete clotting factors. Heparin decreases clotting factors.)

5. A nurse is teaching a newly licensed nurse about heparin-induced thrombocytopenia. Which of the following risk factors for this disorder should the nurse include in the teaching?

- a. Heparin therapy for deep-vein thrombosis

(The client who is receiving heparin therapy for longer than 1 week is at increased risk for development of HIT)

ATI ch 43 Fluid Imbalances

1. A nurse is admitting a client who reports N/V, and weakness. The client has dry oral mucous membranes, temperature 38.5C (101.3F), pulse 92/min, respirations 24/min, skin cool with tenting present, and BP 102/64 mm Hg. Urine is concentrated with a high specific gravity. Which of the following are manifestations of fluid volume deficit for which the nurse should monitor?
 - a. Decreased skin turgor
 - b. Concentrated urine
 - c. Low-grade fever (one of the body's ways of maintaining homeostasis to compensate for the lack of fluid within the body)
 - d. Tachypnea (increased respirations are the body's way to obtain oxygen due to the lack of fluid volume within the body)
2. A nurse is admitting an older client who is experiencing dyspnea, weakness, weight gain of 2lb, and 1+ bilateral edema of the lower extremities. The client has temperature 37.2C (99F), pulse 96/min, respirations 26/min, oxygen saturation 94% on 3L oxygen via nasal cannula, and BP 152/96 mm Hg. Which of the following manifestations of fluid volume excess should the nurse expect?
 - a. Dyspnea (due to excess fluids within the body and lungs, and the client is struggling to breathe to obtain oxygen.)
 - b. Edema (weight gain can be a result of edema)
 - c. Hypertension (BP rises as the heart must work harder due to the excess fluid)
 - d. Weakness (excess fluid retained, which depletes energy and increases the workload for the body)
3. A nurse is assessing a client who is dehydrated for fluid volume deficit. Which of the following findings should the nurse expect in the client?
 - a. Tachycardia (attempt at maintaining blood pressure, manifestation of fluid volume deficit.)
4. A nurse is caring for an older adult client in a long-term care facility. The client has become weak and confused. He ate 40% of his breakfast and lunch. The client's temperature is 38.3C (100.9F), pulse 92/min, respirations 20/min, and BP 108/60 mmHg. He has lost ¾ lb and reports dizziness when assisted to the bathroom. He also has a nonproductive cough with diminished breath sounds in the right lower lobe. Which of the following actions should the nurse take?
 - a. Monitor for orthostatic hypotension (monitor for orthostatic hypotension because he has manifestations of dehydration due to decreased circulatory volume.)

ATI ch 44 Electrolyte Imbalance

1. A nurse is caring for a client who has a serum sodium level 13 mEq/L and serum potassium level 3.4 mEq/L. The nurse should recognize that which of the following treatments can result in these laboratory findings?
 - a. Three tap water enemas (tap water is hypotonic, and GI losses are isotonic. This creates an imbalance and solute dilution.)
2. A nurse is caring for a client who has a serum potassium 5.4 mEq/L. The nurse should assess for which of the following manifestations?
 - a. ECG changes (Potassium levels can affect the heart and result in arrhythmias)
3. A nurse is caring for a client who has a nasogastric tube attached to low intermittent suctioning. The nurse should monitor for which of the following electrolyte imbalances?
 - a. Hyponatremia (nasogastric losses are isotonic and contain sodium)
4. A nurse is assessing a client who has hyperkalemia. The nurse should identify which of the following conditions as being associated with this electrolyte imbalance?
 - a. Diabetic ketoacidosis (hyperkalemia, an increase in serum potassium, is a laboratory finding associated with DKA)
5. A nurse is assessing a client for Chvostek's sign. Which of the following techniques should the nurse use to perform this test?
 - a. Tap lightly on the client's cheek (nurse taps the client's cheek over the facial nerve just below and anterior to the ear to elicit Chvostek's sign. A positive response is indicated when the client exhibits facial twitching on this side of her face.)