Exercise Testing & Prescription for Special Populations

These lectures will cover the pathophysiology as well as implications for exercise testing and prescription for the following health concerns

- ***Hypertension***
- ***Heart Failure and Valvular Heart Disease***
- ***Peripheral Vascular Disease***
- ***Thromboembolic Disorders***
- ***Obstructive & Restrictive Pulmonary Diseases***
- ***Diabetes***
- ***End Stage Renal Disease***
- ***Pregnancy (Time Permitting)***

Texas A&M Health Science Center College of Medicine

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Hypertension

- **Types of Hypertension**
  - **Primary (essential) hypertension**
    - 95% of cases have no identifiable primary cause but may be related to:
      - Heredity - accounts for up to 50% of the variability in BP
        - A gene has been identified that influences sodium balance
        - CNS abnormalities → ↑ circulating catecholamines → ↑ TPR
      - Stress
      - Obesity / insulin resistance
      - Low levels of nitric oxide (EDRF - a vasodilator)
        - More prevalent in African-Americans
      - Aging → ↓ arterial elasticity (arteriosclerosis) → ↑ TPR
  - **Secondary hypertension**
    - Caused by specific endocrine, metabolic, or renal diseases
      - Renal artery stenosis → ↑ renin → ↑ sodium & fluid retention → ↑ BP
        - Most common cause of secondary hypertension
      - Tumors of the adrenal gland → ↑ circulating catecholamines → ↑ TPR

Hypertension

- **Effects on the physiological response to acute exercise**
  - **Usually, hypertensives have a normal ↑ in BP from baseline levels**
    - May be exaggerated or diminished in a few cases
    - Pre-existing hypertension → absolute ↑ in BP may be classified as a hypertensive response to exercise

- **Effects of exercise training on hypertension**
  - **10 mmHg ↓ in BP for mild to moderate hypertensives**
    - Possible ↓ in plasma NE and ↑ in plasma vasodilator substances
    - Possible ↑ in insulin sensitivity → ↓ BP (mechanism not understood)

- **Implications for exercise testing**
  - **Standard ACSM guidelines apply to hypertensives**
    - SBP > 200 and / or DBP > 110 is a relative contraindication to testing
    - SBP > 250 and / or DBP > 115 is a relative indication to terminate testing
      - Those exhibiting a hypertensive response to exercise may be at high risk for developing hypertension at rest

Hypertension

- **Implications for exercise prescription**
  - **Those with BP > 180/110 should be medicated before exercising**
  - **Normal Exercise Rx parameters may be used**
    - Consider starting at a low intensities and durations
  - **Resistance exercise has not consistently been shown to ↓ BP**
    - Pressor responses to lifting heavy weights may ↑ SBP to dangerous levels
    - “Circuit" types of training with light weights and high reps are permissible

Heart Failure (Acute & Chronic)

- **Pathophysiology**
  - **↓ LV contractile capability → ↓ $\dot{Q}$ …… due to:**
    - Prolonged untreated hypertension
    - Myocardial infarction (acute heart failure)
    - Cardiomyopathy (diseased and malformed heart muscle)
    - Valvular heart disease
      - Aortic stenosis & regurgitation
      - Mitral stenosis & regurgitation
      - Coarctation of aorta (narrowing of aortic outflow tract)
    - Myocardial infection & inflammation (Myocarditis, Restrictive Pericarditis)

Heart Failure (Acute & Chronic)

- **Pathophysiology**
  - **The body tries to compensate for the ↓ Q̇ (compensated HF)**
    - **note: drugs are used to get someone to a "compensated" state**
  - **Consequences of the bodies' compensation in red paretheses**
    - ↑ β sympathetics → ↑ TPR → ↑ venous return & preload (↑ afterload)
    - ↓ Renal perfusion → ↑ blood vol → ↑ venous return & preload (↑ afterload)
    - ↑ In blood vol & preload → ventricular dilatation (max stretch on LV fibers)
    - ↑ tissue O_2 extraction - almost 100% of O_2 is extracted (↑ AVO2-difference)

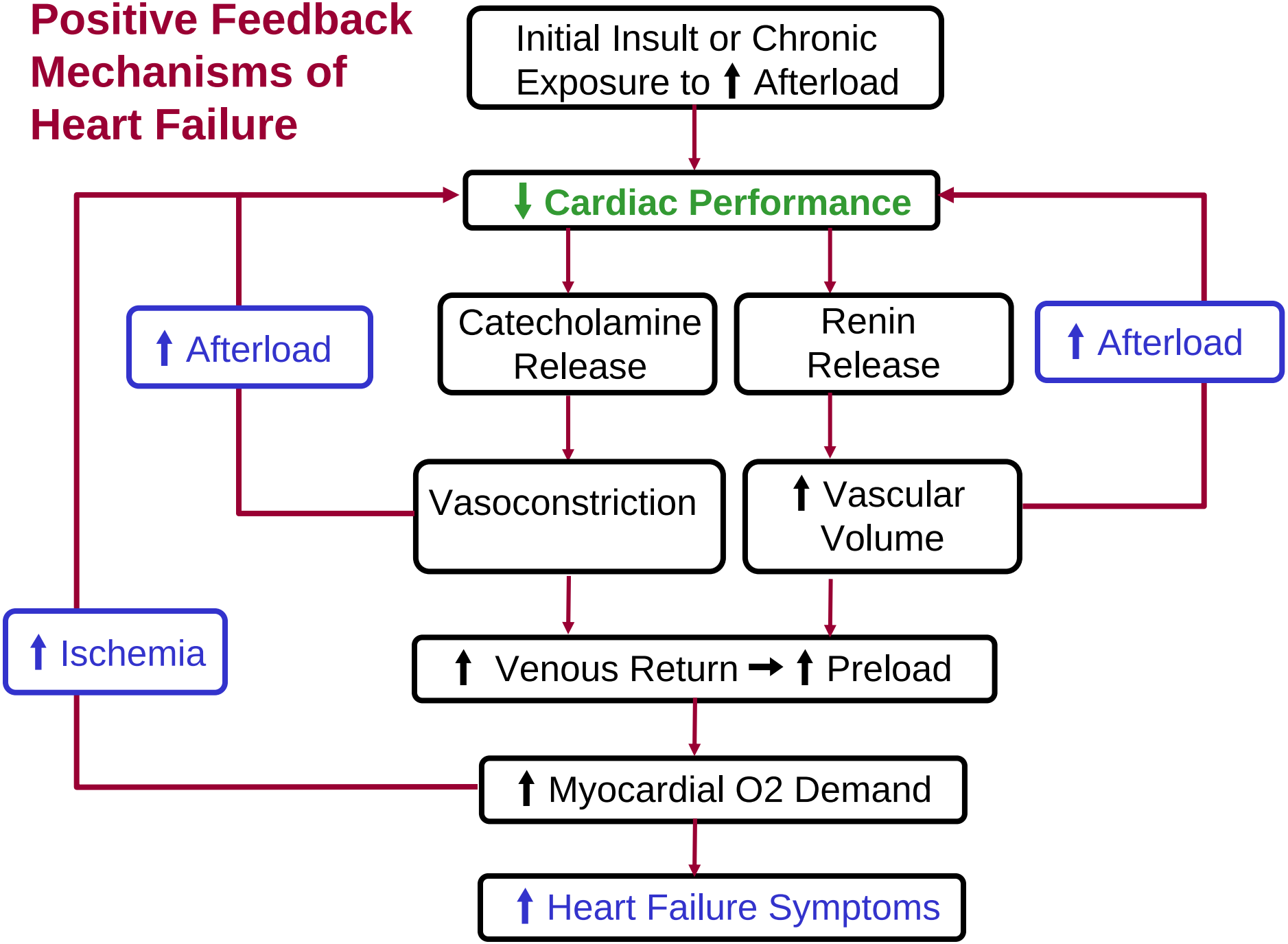
    - **Myocardial hypertrophy** occurs in response to pressure / volume overload
      - Volume overload (↑↑ preload) → eccentric hypertrophy
      - Pressure overload (↑↑ afterload) → concentric hypertrophy
      - Hypertrophy → ↓ ventricular contractility + ↓ ventricular compliance
        - Usually, both systolic function and diastolic function are affected
        - Diastolic dysfunction CHF → inadequate relaxation of LV → ↓ filling → ↓ Q̇
        - Symptoms are similar to those of systolic dysfunction CHF but EF is normal

    - **Dilated Cardiomyopathy** → ↓ contractility + ↑↑↑ compliance
      - Downward portion of Frank Starling Curve
        - More blood in → less blood out

Heart Failure (Acute & Chronic)

- **Pathophysiology**
  - **Summary changes due to heart failure**
    - ↓ $\dot{Q}$ during exercise and, in moderate to severe cases, at rest
    - ↑ Afterload due to ↑ α & β symp. mediated ↑ in TPR and fluid retention
    - ↑ Preload - due to diastolic dysfunction & fluid retention
    - ↑ Pulmonary artery pressure → exertional dyspnea
    - Destructive changes in organs due to ischemia: **Muscles, Kidneys**
- **Effects on the physiological response to acute exercise**
  - **↓ HR + ↓ SV → ↓ $\dot{Q}$**
  - **↓ max HR**
  - **↓ O_2 delivery + muscle lactate buildup → fatigue + hyperventilation**
    - Sometimes occurs before the onset of CHF related dyspnea symptoms
  - **↓ Exercise tolerance due to**
    - ↓ Oxidative phosphorylation → ↑ anaero. glycolysis → ↑ metabolic acidosis
    - ↓ vasodilation of muscle vessels, eventual muscle atrophy
  - **Poor redistribution of blood flow during exercise**

Positive Feedback Mechanisms of Heart Failure



Heart Failure (Acute & Chronic)

- **Signs & symptoms of left ventricular heart failure**
  - **Dyspnea (pulmonary edema)**
    - ↓ Contractility of LV → pressure "backs up" in pulmonary circulation
      - Fluid leaks into alveoli → ↓ gas exchange, cough, dyspnea
    - Mismatch of $\dot{V}_E : \dot{Q}$ → ↑ physiologic dead space in lung → dyspnea
  - **Fatigue & weakness ("heaviness" in arms and legs)**
    - Caused by ↓ perfusion to the muscles
  - **Dizziness, confusion, anxiety, memory loss,**
    - Caused by ↓ perfusion to the brain
- **Signs and symptoms of right ventricular heart failure**
  - **Jugular venous distension**
    - Pressure "backed up" behind RV into the major veins → venous distension
  - **Ascending peripheral edema**
    - ↑ Venous pressure → edema in distal extremities - progresses to thighs
      - Weight gain: HF patients are monitored daily for gain in water weight
  - **Hepatomegaly & ascites (fluid in peritoneal cavity)**
    - ↑ Venous pressure → blood engorged liver + fluid leaks into peritoneum

Heart Failure (Acute & Chronic)

- **Treatment strategies in CHF: main goal: ↓ cardiac workload**
  - **Angiotensin converting enzyme (ACE) inhibitors:**
    - ↓ Angiotensin II → ↓ arterial vasoconstriction → ↓ afterload
    - ↓ Angiotensin II → ↑ venodilation → ↓ preload
    - ↓ Na+ retention → ↓ H2O retention → ↓ blood vol → ↓ preload
      - Only drug shown to both improve symptoms and prolong life
  - **Diuretics + dietary salt restriction**
    - ↓ H2O retention → ↓ blood vol → ↓ preload
  - **Positive inotropic agents: digitalis, sympathomimetics, PD inhibitors**
    - Digitalis → ↑ contractility → ↑ $\dot{Q}$ → ↓ CVP → ↓ preload
    - Sympathomimetics → ↑ contractility → ↑ $\dot{Q}$ → ↓ CVP → ↓ preload
    - PD inhibitors → ↑ cyclic AMP in myocardium and vascular smooth muscle
      - ↑ Contractility + ↑ arterial & venous dilation → ↓ preload & afterload
  - **Arterial and venous dilators**
    - Nitrates → ↑ venodilation → ↓ preload (CA dilation → ↑ O2 supply)
    - Hydralazine (peripheral vasodilator) → ↑ arterial dilation → ↓ afterload
  - **Antiarrhythmics - CHF is the most arrhythmogenic CVD (V-tach)**

Heart Failure (Acute & Chronic)

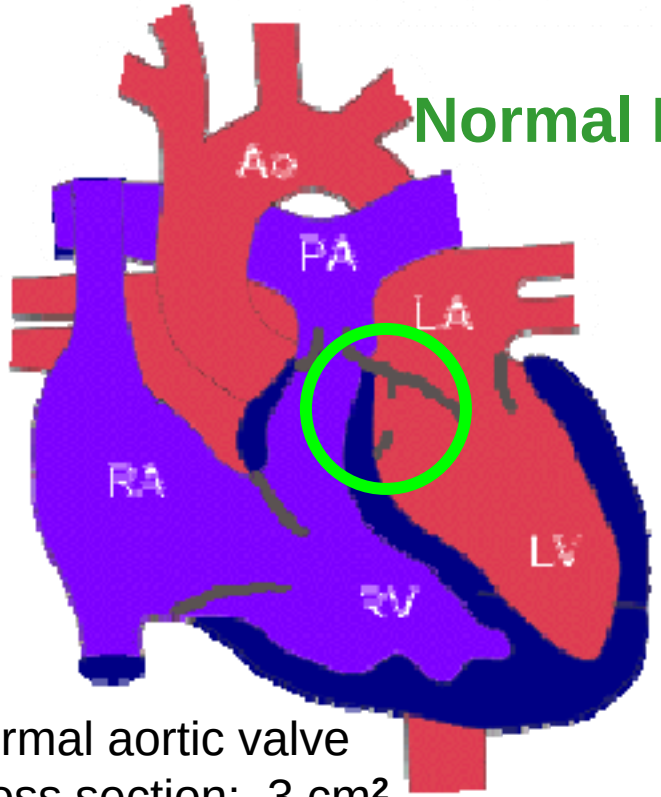
- **Implications for exercise testing:**
  - **Unstable or decompensated CHF is a contraindication to testing**
  - **Maine goals of exercise testing in CHF patients:**
    - Identify the severity of CHF (precisely quantify functional capacity - $\dot{V}O_{2max}$)
    - Test the efficacy of various interventions
    - Evaluate the possibility of other disease (CAD, PVD, VHD, arrhythmias, etc.)
  - **Make sure testing protocol, equipment, and staff are appropriate**
    - Begin protocol at < 3 METS with small / moderate stage workload increments
    - Be prepared for hypotension, arrhythmias, and chronotropic incompetence
    - Use respired gas measurements if possible
      - Breathing patterns can be assessed for efficiency
  - **Observe conservative test endpoints**
    - Fatigue, weakness, pallor → ↓ $\dot{Q}$
    - CNS symptoms (dizziness, unsteady gait) → ↓ cerebral perfusion
    - ST-segment changes (especially if accompanied by symptoms)
    - PVC's & ventricular ectopy (especially in aortic stenosis patients)
    - Atrial flutter or fibrillation accompanied by a fast ventricular response

Heart Failure (Acute & Chronic)

- **Implications for exercise Rx:**
  - **Observe conservative contraindications to training:**
    - Decompensated CHF, LV outflow tract obstruction, unstable arrhythmias
  - **Maine goals of exercise training in CHF patients:**
    - ↓ Symptoms + ↑ functional capacity ($\dot{V}O_{2peak}$) & T-vent → ↑ quality of life
  - **Make sure exercise Rx and patient monitoring is appropriate**
    - Prolong warm-up and cool-down sessions
    - Use RPE and dyspnea scales instead of THR or absolute workload targets
    - Begin at a low workload (Borg 11-16) - always below point of symptoms onset
      - Workload should be less than that which produces:
        - LV wall motion abnormalities or a drop in ejection fraction
        - A pulmonary capillary wedge pressure (filling pressure) > 20 mmHg
        - Anaerobic threshold (ventilatory threshold)
    - Progress by advancing the duration of the bout
    - Avoid isometric exercise but strength training exercise (low weight, hi reps) OK
      - Discourage Valsalva maneuver → ↑ thoracic pressure → ↑ afterload
    - CHF patients may deteriorate rapidly → frequent re-assessment

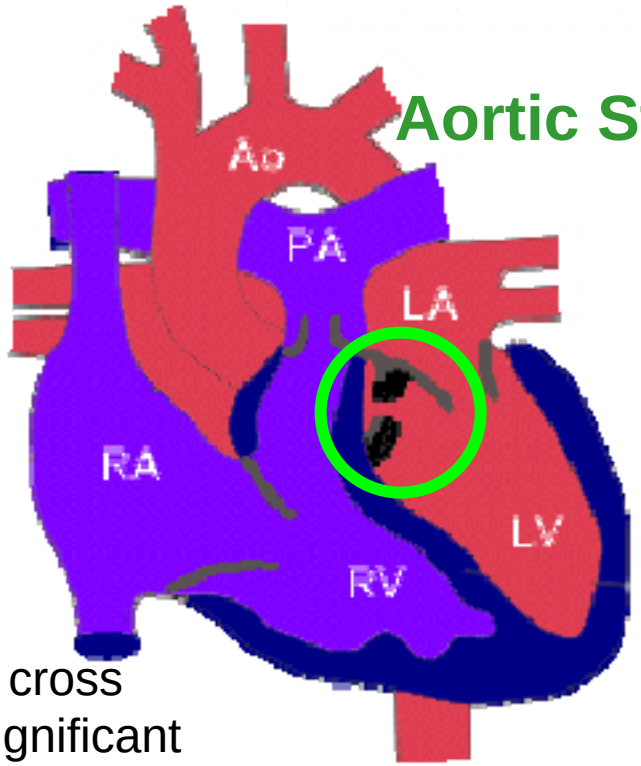
Valvular Heart Disease: Aortic Stenosis

- **Pathophysiology**
  - Narrowing of the aortic outflow tract at, above, or below the valve due to valve fibrosis & calcification, congenital abnormality, or damage from rheumatic fever
  - Idiopathic hypertrophic subaortic stenosis (form of cardiomyopathy)
    - asymmetric hypertrophy of the ventricular septum → ↓ outflow



Normal Heart

normal aortic valve cross section: 3 cm^2



Aortic Stenosis

aortic valve cross section in significant aortic stenosis: 1 cm^2

Valvular Heart Disease: Aortic Stenosis

- **Pathophysiology (continued)**
  - **↑ pressure required to eject blood into aorta → ↑ LV muscle mass**
    - The thickened LV eventually dilates, stiffens, and begins to fail
  - **In some cases the normal tricuspid valve is abnormally bicuspid**
  - **Usually occurs in children (congenital) or after age 70**
  - **Pressure gradient across the valve is critical diagnostic factor**
    - Mild AS: peak LVSP is 10 - 40 mmHg higher than aortic pressure
    - Severe AS: peak LVSP is > 60 mmHg higher than aortic pressure
- **Symptoms**
  - **Fatigue & syncope**
  - **Dyspnea (with exertion and at night)**
  - **Anginal type chest pains**
  - **Sudden death**

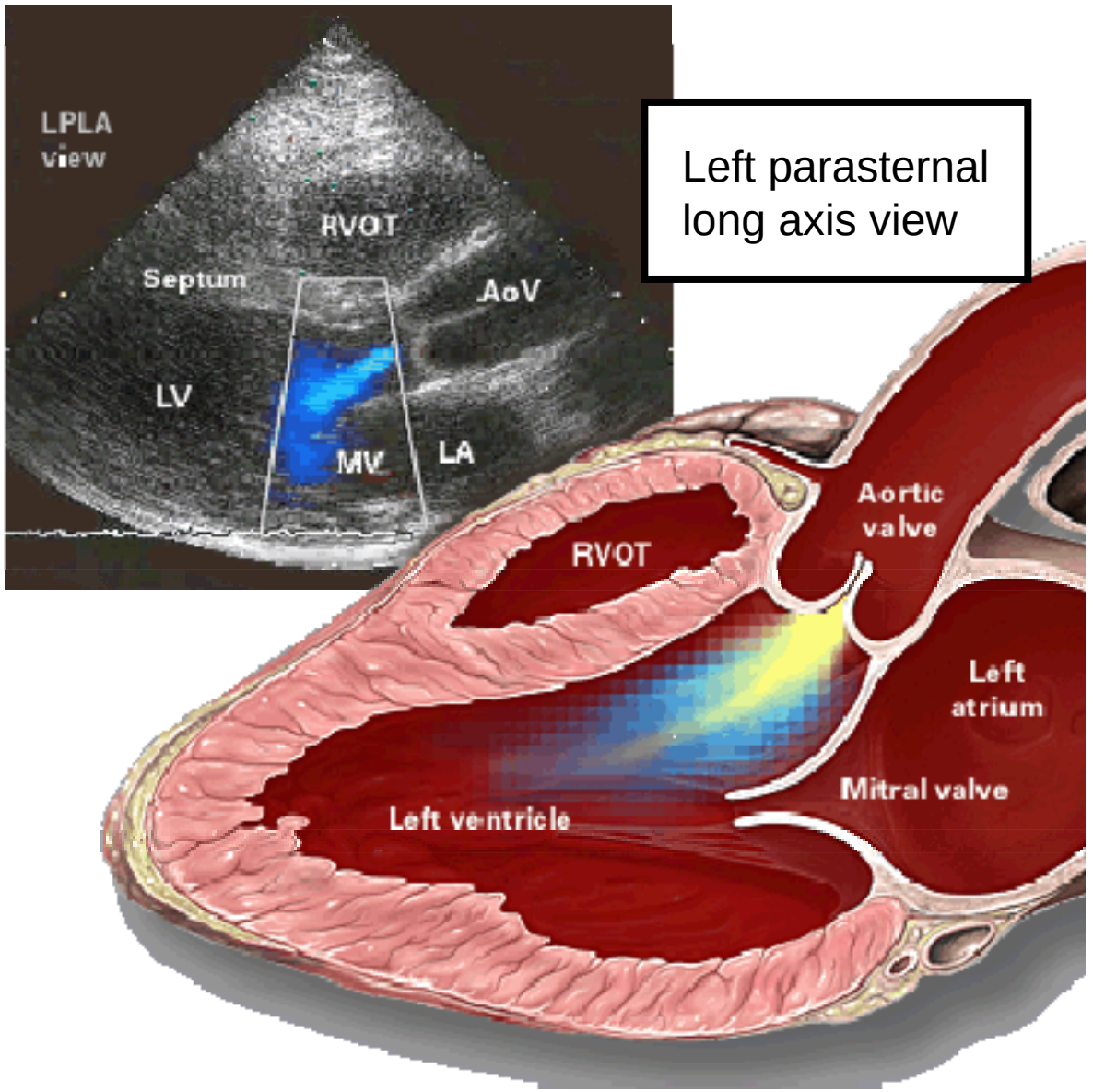
Valvular Heart Disease: Aortic Stenosis

- **Treatment**
  - **Balloon valvuloplasty - "stretching" the aortic valve opening**
    - May improve condition for a period of time, but procedure is not curative
  - **Valve replacement surgery - artificial valve or autograft valve**
    - Ross Operation - autografted valve can grow with the child
      - Patient's pulmonary valve grafted into aortic valve
      - Donor valve replaces transplanted pulmonary valve
- **Implications for exercise testing**
  - **Severe aortic stenosis is an absolute contraindication to testing**
    - Aortic stenosis is associated with ventricular arrhythmias & sudden death
  - **Sometimes GXT 's are done to quantify functional capacity**
    - ST-segment depression is often seen
    - Important to use low stage increments so FC can be accurately identified
    - Important to closely monitor pressure for falloffs

Valvular Heart Disease: Aortic Stenosis

- **Implications for exercise training**
  - **Clinically mild AS can be prescribed exercise - normal parameters**
    - Asymptomatic, negative GXT should be secured before exercise begins
    - Begin at low intensities and durations
  - **Patients with gradients > 40 mmHg should not do intense exercise**
    - Exercises with high cardiac demands should be avoided
      - No competitive activities
  - **Surgery is usually recommended when gradient exceeds 60 mmHg**

Valvular Heart Disease: Aortic Regurgitation (Aortic Insufficiency)



Valvular Heart Disease:

Aortic Regurgitation (Insufficiency)

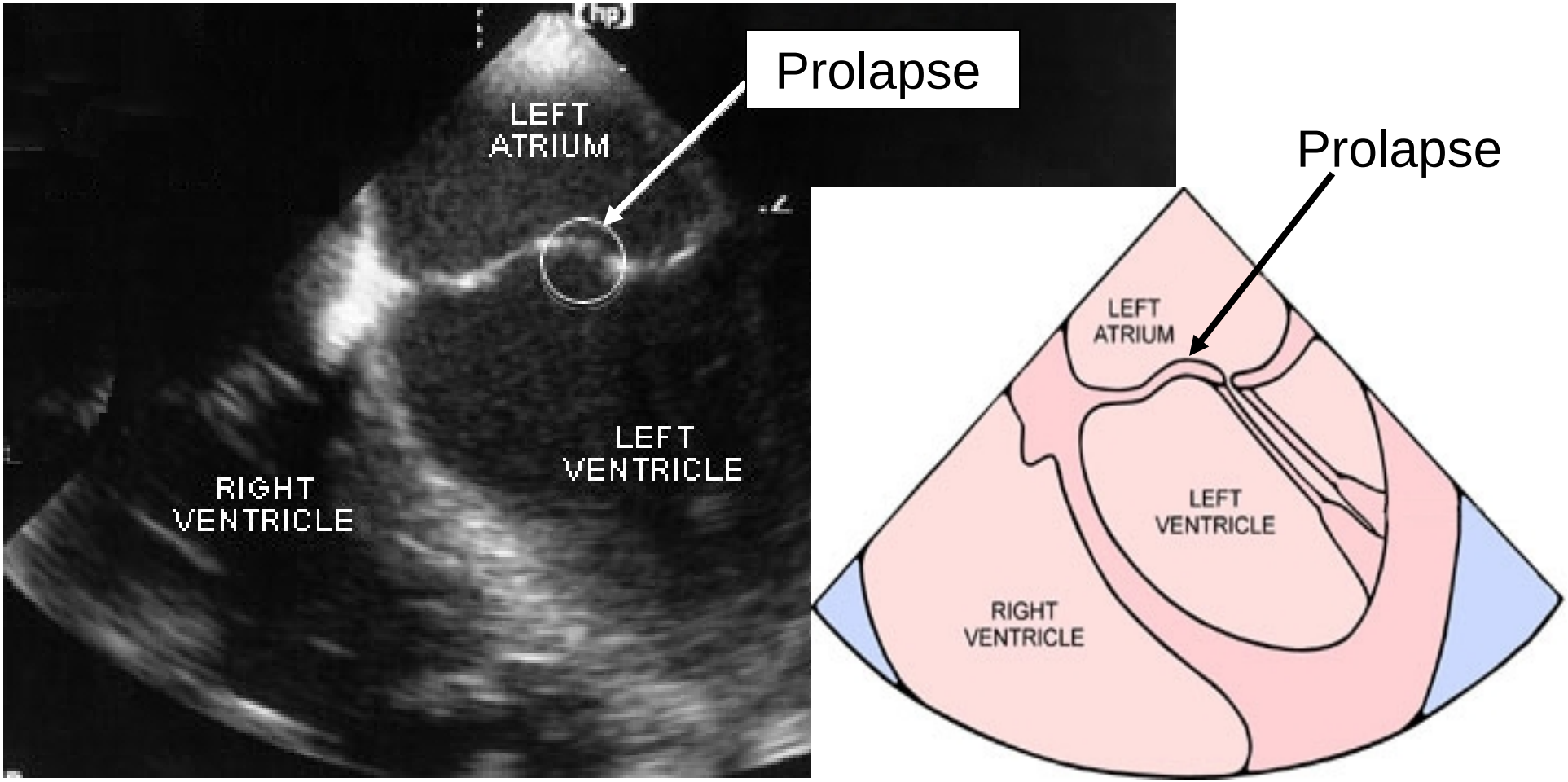
- **Pathophysiology**
  - **Retrograde flow from the aorta back into the LV**
  - **Usually caused by:**
    - Rheumatic fever or bacterial endocarditis
    - Congenital valve defect (valve is bicuspid instead of tricuspid)
    - Marfan's syndrome: composition defects in connective tissue → ↓ stiffness
  - **Heart must pump normal EDV + regurgitant volume**
    - Pressure & volume overload → eventual LV failure
    - Acute AR → ↑ left atrial pressure → pulmonary edema (EMERGENCY)
- **Signs & Symptoms** - **note that most are reflective of heart failure**
  - **Fatigue, syncope, dyspnea (with exertion and at night)**
  - **Sensation of forceful heartbeat**
  - **↑ SV → ↑ SBP , regurgitation during diastole → ↓ DBP**
  - **Chest pain**
  - **Arrhythmias**

Valvular Heart Disease:
Aortic Regurgitation (Insufficiency)

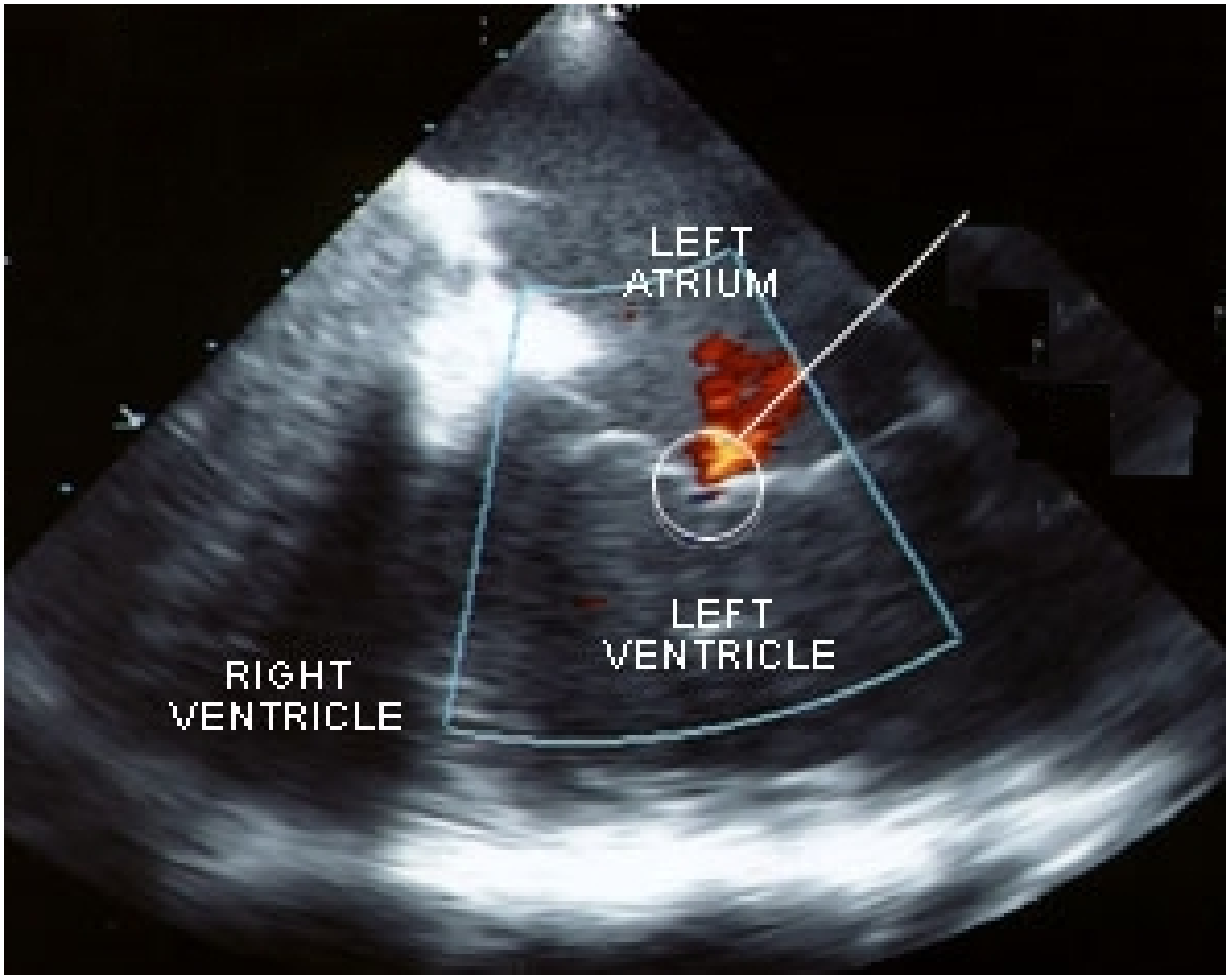
- **Treatment**
  - **Mild / asymptomatic cases**
    - Appropriate antibiotic prophylaxis
    - Vasodilator drugs to reduce afterload
  - **Acute or severe chronic cases**
    - Valve replacement surgery
      - Should be done before irreversible damage is done to LV
- **Implications for exercise testing & RX**
  - **Same precautions & guidelines as in heart failure**
  - **Strenuous or competitive exercise should be avoided**

Valvular Heart Disease: Mitral Valve Prolapse

- **Pathophysiology**
  - **MV leaflet "prolapses" (bulges) back into left atrium during systole**



Valvular Heart Disease: Mitral Valve Regurgitation



Valvular Heart Disease:
Mitral Valve Prolapse - Regurgitation

- **Pathophysiology (continued)**
  - **5% - 10% of US population has some degree of MV prolapse**
  - **Most common in women ages 40 to 50**
  - **Severe MV prolapse will lead to MV regurgitation**
    - Retrograde blood flow back into the left atrium
- **MVP Symptoms - most patients are asymptomatic**
  - **Chest palpitations**
  - **Arrhythmias**
  - **Fatigue & anxiety**
  - **Sharp chest pains (possible related to strain on papillary muscle)**
  - **Resting & Orthostatic hypotension**
  - **Dyspnea (with exertion and at night while in prone position)**
  - **SIGNIFICANT REGURGITATION ➞ HEART FAILURE SYMPTOMS:**

Valvular Heart Disease:
Mitral Valve Prolapse - Regurgitation

- **Treatment**
  - For asymptomatic MV prolapse: antibiotic prophylaxis
  - β-blockers or Ca^{++} channel blockers may relieve chest palpitations
  - Severe MV prolapse / regurgitation: valve repair or replacement
    - Should be done before irreparable damage is done to LV
- **Implications for exercise testing and Rx**
  - Exam should be performed to rule out other valve problems
  - Normal parameters for exercise testing - symptoms limited test
  - Normal exercise Rx parameters in most asymptomatic patients
  - Patients may be sensitive to exercise induced hypovolemia
  - Patients that should avoid strenuous/competitive/contact sports:
    - Moderate to severe regurgitation
    - History of arrhythmogenic syncope or exercise induced tachycardias
    - Family history of sudden death or embolism associated with MVP

Valvular Heart Disease: Mitral Valve Stenosis

- **Pathophysiology**
  - **Narrowing of the mitral valve - usually due to rheumatic fever**
    - Thickening & calcification of the valve leaflets
    - Normal valve area 4 - 6 cm^2 - pressure gradient occurs when area < 2 cm^2
  - **Women have MV stenosis 4 X more than men:**
    - First symptoms may occur during pregnancy
  - **↑ LA pressure transmitted back to lungs → ↑ pulmonary edema**
    - LA becomes dilated → conduction fibers are stretched → A-fib may occur
    - Essentially, patients have left sided heart failure without LV dysfunction
- **Signs & Symptoms**
  - **Exertional dyspnea is most common symptom in mild MS**

  CONDITION PROGRESSES (VALVE AREA OF 1 CM^2 OR LESS)
  - **Marked fatigue & dyspnea due to pulmonary congestion**
  - **Paroxysmal nocturnal dyspnea**
  - **Cough or hoarseness**
  - **Stagnation of LA blood flow → ↑ risk of thrombus formation**

Valvular Heart Disease: Mitral Valve Stenosis

- **Treatment**
  - **For asymptomatic MV stenosis: antibiotic prophylaxis**
  - **β-blockers used to ↑ diastolic filling time**
  - **Mild pulmonary congestion can be treated with diuretics**
  - **A-fib patients require antiarrhythmics and anticoagulants**
  - **Sever MS requires valve replacement or balloon valvuloplasty**
- **Implications for exercise testing and Rx**
  - **SV may fall during exercise due to inadequate ventricular filling**
    - Exercise → ↑ HR → ↓ diastolic filling time → ↓ SV → ↓ $\dot{Q}$ → ↓ SBP
    - ↓ $\dot{Q}$ → ↓ muscle perfusion → ↑ lactate → ↓ functional capacity
    - Pulmonary congestion → ↑ work of breathing → dyspnea is limiting factor
  - **Normal parameters for exercise testing - symptoms limited test**
    - Use precautions similar to those for heart failure patients
  - **For exercise Rx, use same precautions as in heart failure**
  - **Strenuous / competitive / contact sports should be avoided**

Peripheral Arterial Disease (PAD)

- **Peripheral Arterial Disease - atherosclerotic obstruction of peripheral arteries (Usually in lower extremeties)**
  - **Signs & Symptoms**
    - Claudication (usually earliest & most common symptom):
      - Cramping in the hips, thighs, & especially the calves
      - Cramping caused by skeletal muscle ischemia
    - Numbness, weakness, or heaviness of lower extremity muscles
    - Severe cases ➞ burning - aching pain at rest in feet & toes
    - Pale color & palpable coldness of lower extremities
    - Diminished or absent peripheral pulses (tibial & dorsalis pedis pulses)
  - **Risk Factors**
    - Hypertension, Diabetes, Age, CAD, Smoking (similar to CAD risk markers)
  - **Diagnosis**
    - Ratio of ankle SBP to brachial SBP (AB index or ABI) is less than .9
    - Doppler assessments of flow & pressure
    - Angiography
    - Treadmill testing to assess functional capacity (time to claudication pain)

Peripheral Arterial Disease (PAD)

- **Treatment**
  - **Exercise → ↑ vascularization and blood flow + ↑ pain tolerance**
    - May double time to claudication or exertion level before it appears
  - **Cilostazol - helps to relieve PAD symptoms**
    - PDE III inhibition → ↑ C-amp → ↓ Ca++ in smooth muscle → vasodilation
    - ↓ platelet aggregation
  - **Pentoxifylline (Trental)**
    - ↓ blood viscosity → ↑ blood flow
  - **Dipyridamole (Persantine)**
    - Inhibition of platelet adhesion → ↑ blood flow
  - **Warafin Sodium (Coumadin)**
    - Inhibition of vitamin K dependent coagulation factors → ↑ blood flow
  - **Aspirin & other platelet inhibitors → ↓ platelet aggregation**
  -
  - **Angioplasty (stents in large arteries)**
  - **Bypass surgery**

Peripheral Arterial Disease (PAD)

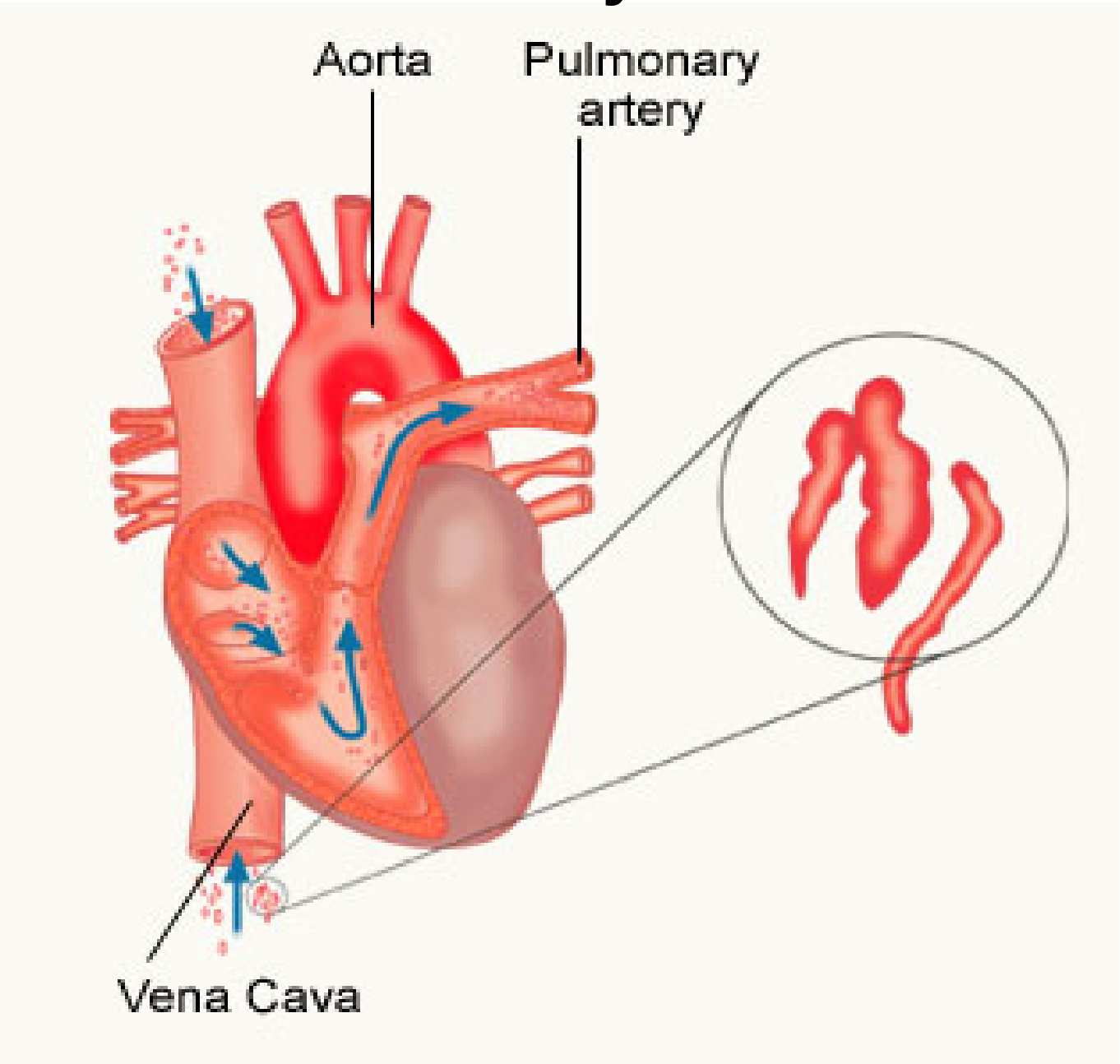
- **Implications for exercise testing and Rx**
  - **Exercise Testing**
    - ABI's are assessed pre & post exercise
    - Gradual stage workload increments are used to precisely assess FC
    - Pain scale of 1-4 is used to assess claudication pain throughout GXT
    - Record elapsed time from volitional termination to symptoms disappearance
    - Closely monitor patients for signs of CAD
      - **50% - 80% of PAD patients have CAD**
  - **Exercise Rx**
    - Mode: Treadmill, walking, stair climbing
    - Frequency: minimum 3 days per week
    - Intensity: 3 on a 4 point pain scale (≅ 40% Karvonen to start)
    - Duration: 20 minutes - exercise to point of pain tolerance, rest, repeat
      - Usually, 5 minutes of exercise will produce severe claudication
      - Allow full recovery between bouts
      - Increase duration before intensity
    - Progression: 40 - 60 minutes of intermittent exercise within 6 months

Thromboembolic Disorders

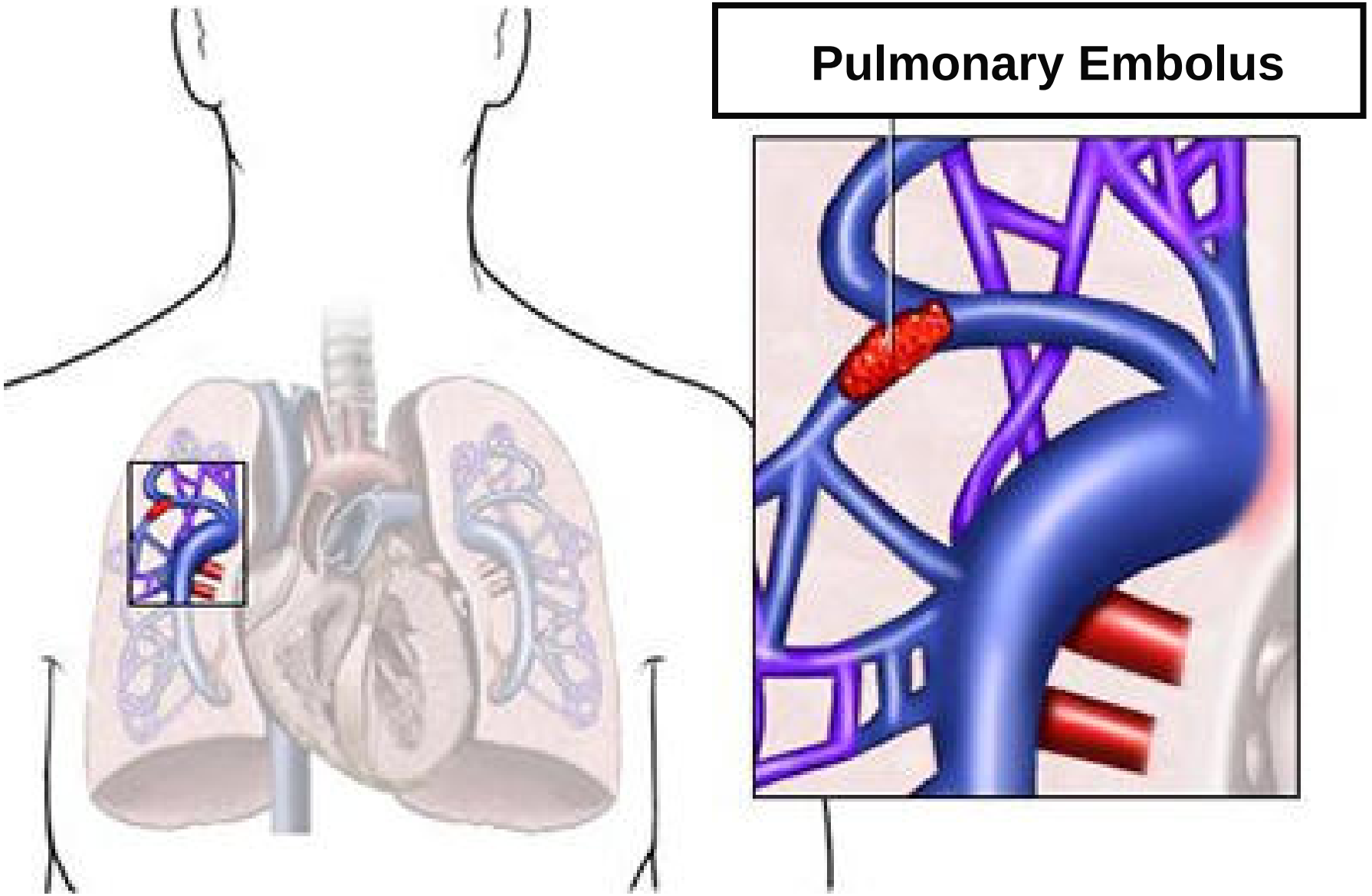
Pulmonary Embolism (PE)

- **Symptoms**
  - Chest pain, back pain, shoulder pain, upper abdominal pain
  - Shortness of breath or wheezing
  - Painful and or fast respiration (respiratory distress)
  - Syncope
  - Fever & sweating
  - Chest wall tenderness
  - Sudden death
  - Coughing up blood
  - Right ventricular failure
    - Caused by high pulmonary pressures

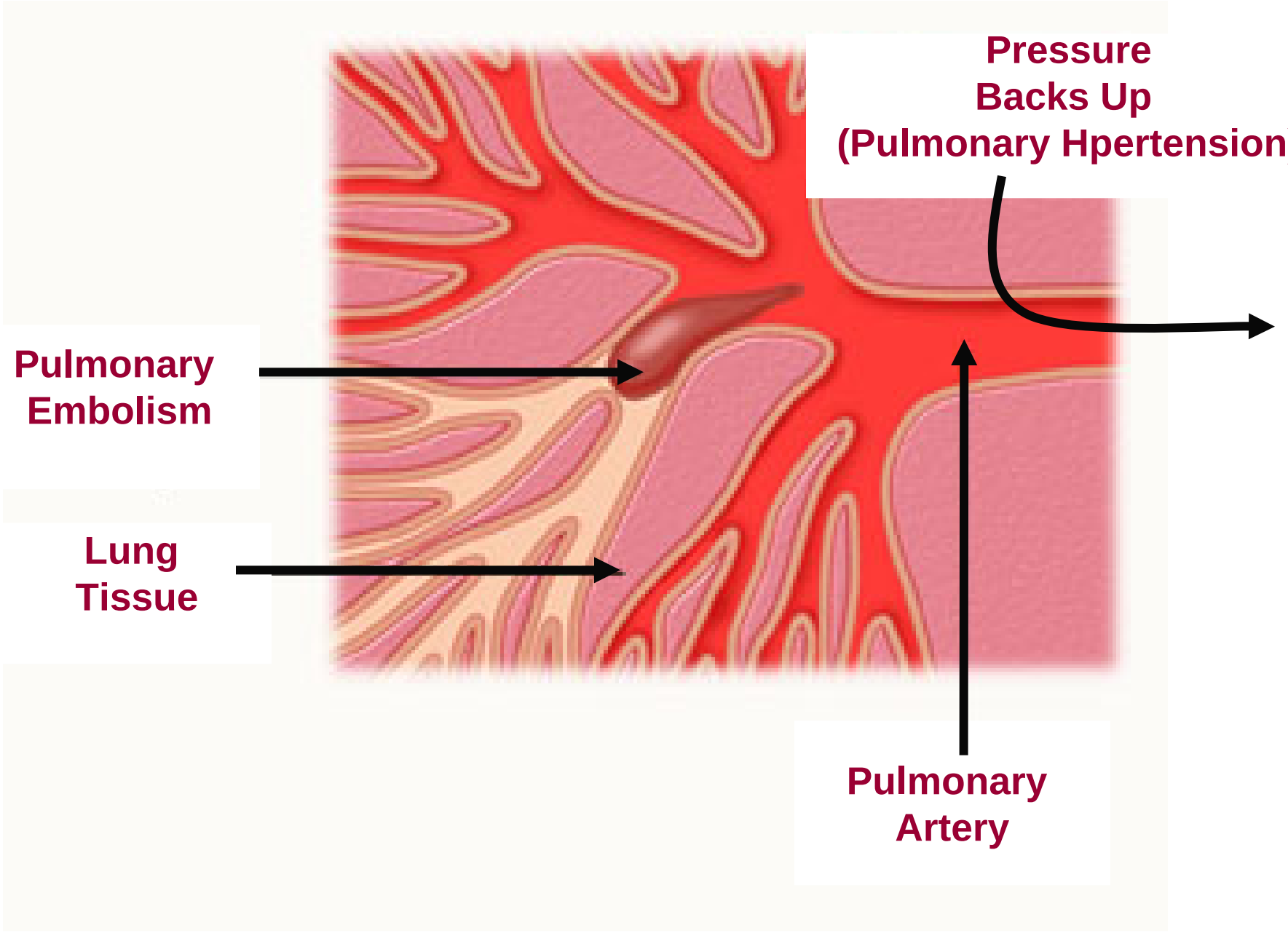
Pulmonary Emboli



Pulmonary Embolism



Pulmonary Embolism



Pulmonary Embolism

Causes and Risk Factors

- Surgery lasting > 30 min (most common cause of DVT)*
  - Especially surgeries of the hip, lower abdomen, and leg
- Prolonged immobility *
- A long-haul flight in an aircraft *
- Prior PE
- A fracture of a lower limb or the pelvis
- High dose estrogens meds (birth control & HRT)
- Hypercoaguable states
- Any debilitating disease (especially cancer)
- Pregnancy and childbirth
- Smoking
- Age
- Obesity
- Phlebitis

* Commonly Arise from Deep Vein Thrombosis (DVT)

Pulmonary Embolism

Pulmonary Embolism and Cancer

Why is cancer a risk for thromboembolic events

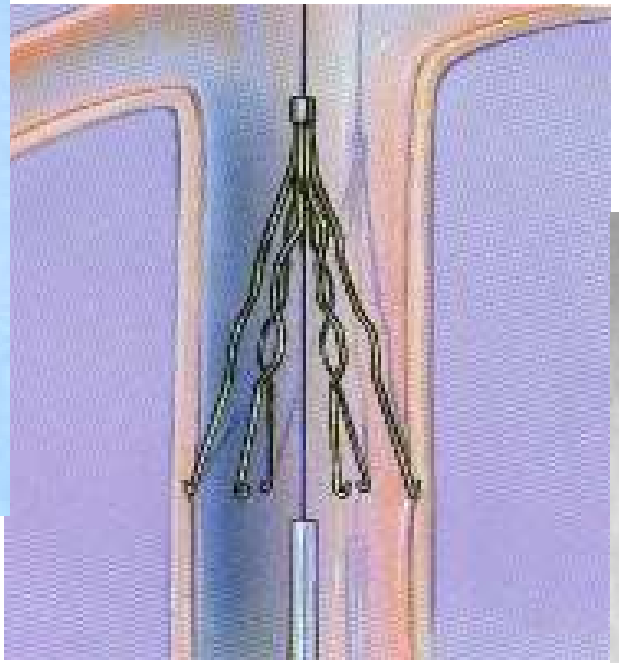
- **Chemotherapy destroys cancer cells. These destroyed cancer cells release pro-coagulant substances**
- **Surgical intervention for cancer can damage vessel walls triggering coagulation**
- **Cancer therapies may reduce the body's ability to produce adequate amounts of natural anticoagulants**

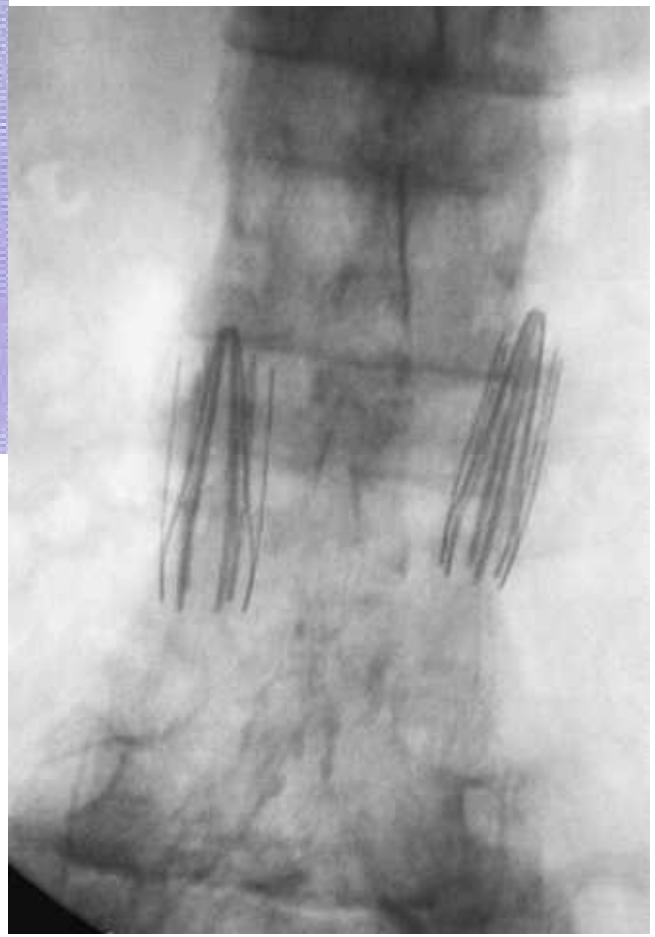
- **Note: 90% of cancer patients usually experience and increase in the blood's clotting activity**

Pulmonary Embolism

- **Treatment**
  - Hospitalization & leg elevation
  - Pain medication
  - Compression stockings
  - Thrombolytic drugs
    - (Urokinase, Streptokinase, TPA)
  - Surgical removal of clot (only if drugs don't work)
  - Supplemental oxygen
- **Prevention**
  - Mobility / Exercise !!!
  - Compression stockings
  - Keep feet raised higher than hips during prolonged immobility
  - Anticoagulant drugs
    - Warfarin (Coumadin), Heparin
  - Vena caval filter insertion
    - Usually in inferior vena cava for DVT

IVC Filter

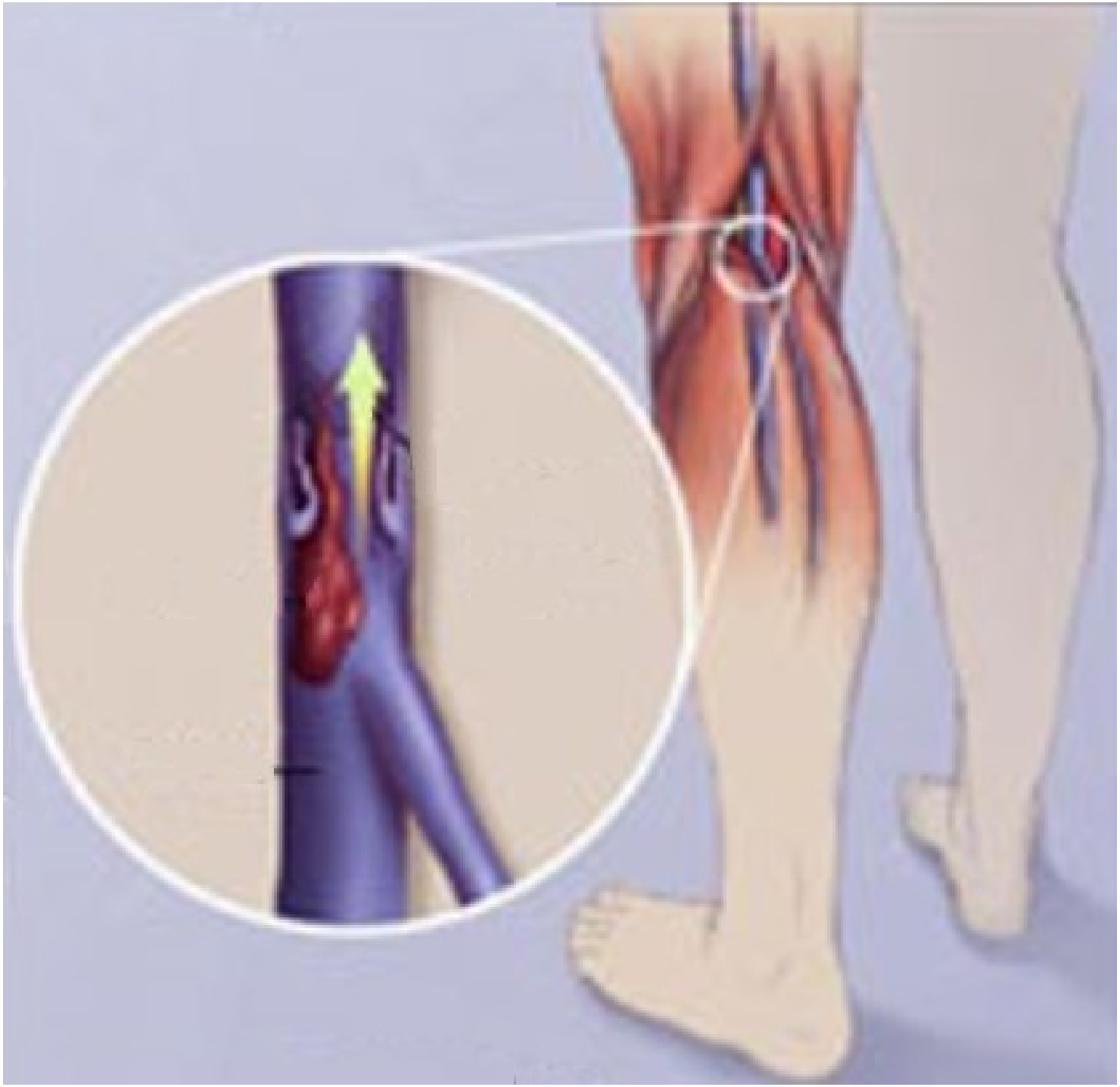




Used when person is not a candidate for anti-thrombolytic drug therapy or that therapy fails

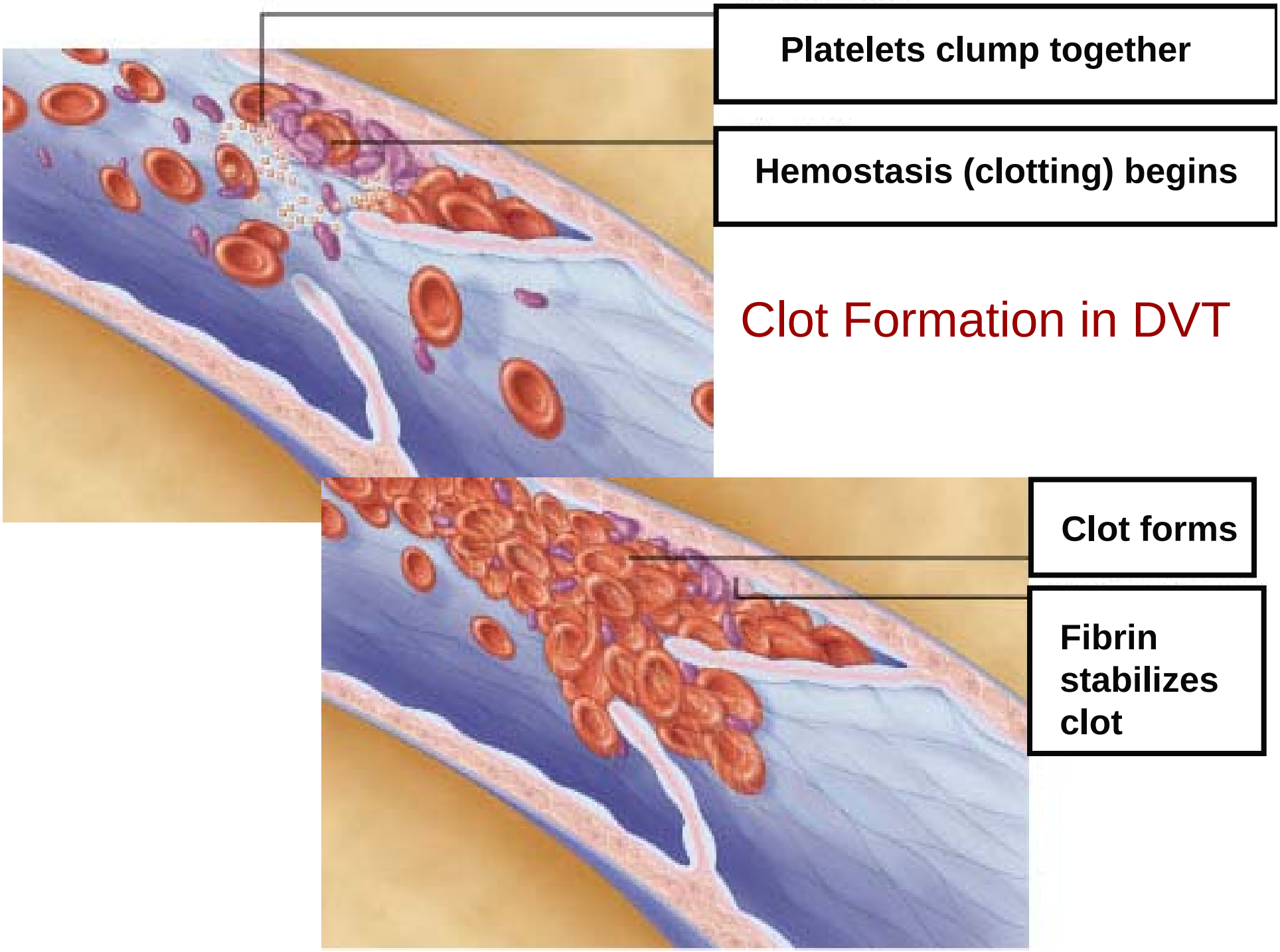
Deep Vein Thrombosis (DVT)

- DVT
  - Formation of large clot usually in calves or thighs
    - Clot may form in axillosubclavian area, abdomen, or pelvis
    - More common in calves
    - Danger of DVT is **pulmonary embolism**
    - Post thrombotic syndrome may also occur:
      - Calf pain, swelling, rashes, skin ulcerations
  - Causes / Risks of DVT
    - Similar to PE
  - Symptoms of DVT (only ½ of DVT patients have symptoms)
    - Acute PE symptoms
    - Pain / tenderness in the calf or leg
    - Swelling & redness of leg – leg becomes warm
    - Surface veins become more visible
  - Treatment / Prevention of DVT
    - Similar to Pulmonary Embolism



Deep Vein Thrombosis (DVT)

Clot Formation in DVT



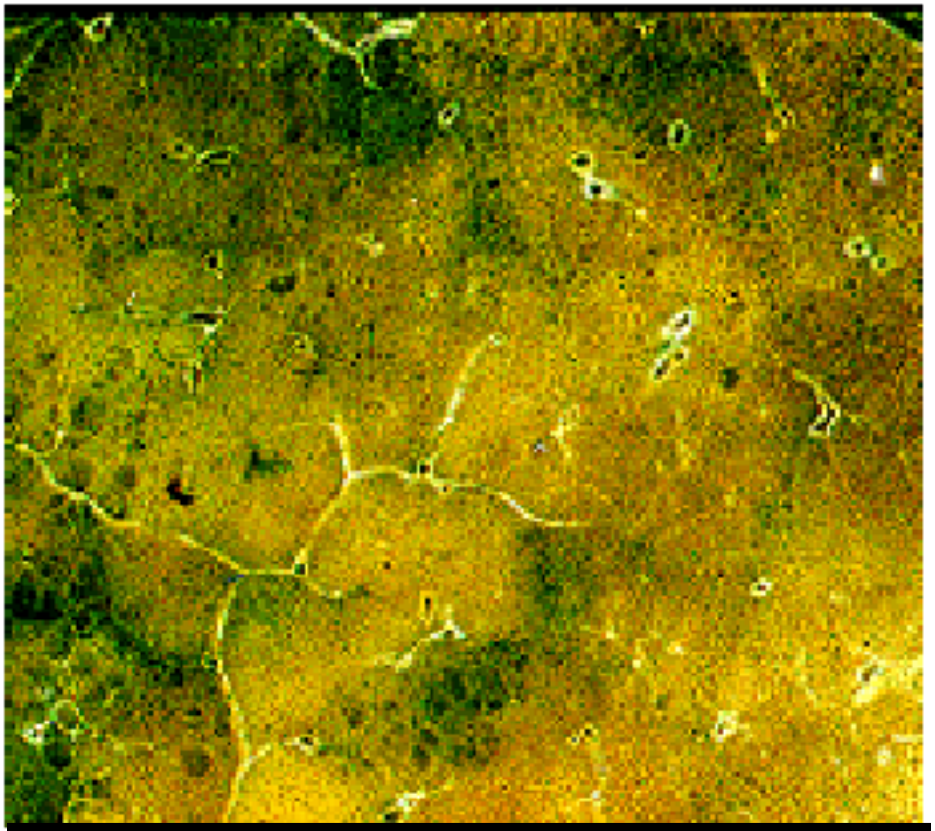
Deep Vein Thrombosis (DVT)

Chronic Obstructive Pulmonary Disease (COPD)

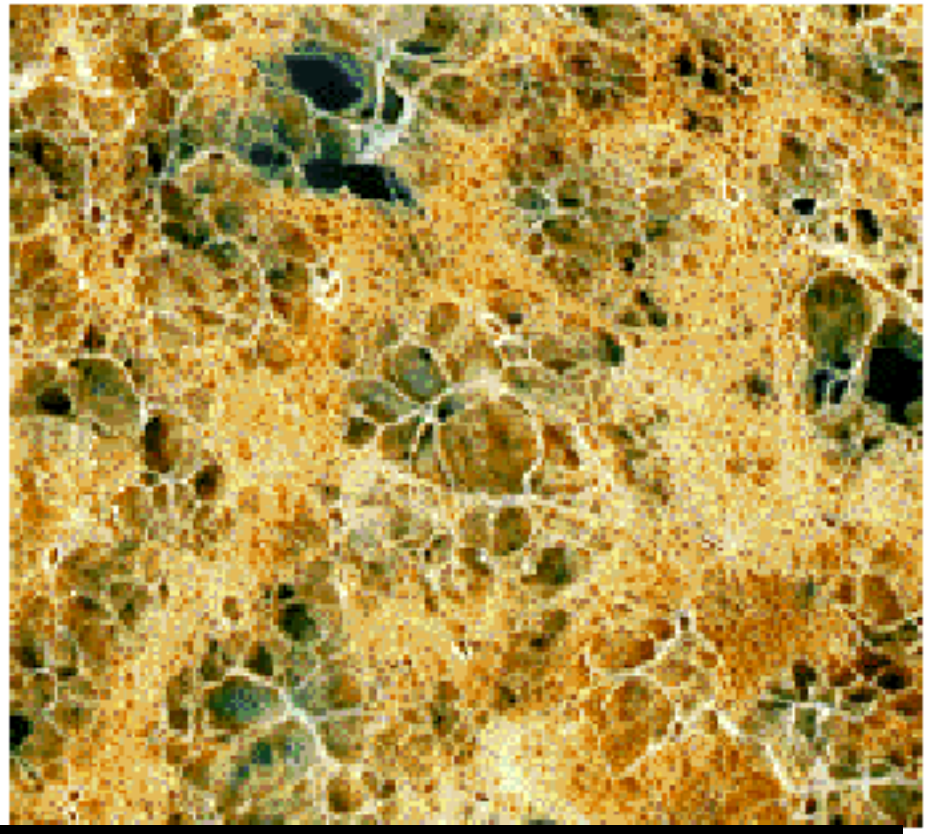
- **3 Main Conditions Comprise the Majority of COPDs**
  - **Asthma - reversible bronchospasm + airway inflammation**
    - may be related to: genetics, allergens, cold air, exercise, smoke, and smog
    - **SYMPTOMS:** wheezing, shortness of breath, coughing
  - **(Chronic) Bronchitis - inflammation & obstruction of small airways**
    - smoking is most common cause **(90% of COPD patients are smokers)**
    - other causes: respiratory infections, Industrial pollutants (smog)
    - characterized by chronic production of sputum & thickened bronchial walls
    - **INITIAL SYMPTOMS:** shortness of breath and productive coughing
      - usually not diagnosed until person has symptoms 3 months / year
  - **Emphysema - destruction and distension of alveoli**
    - again, smoking is most common cause
    - alveoli destruction results in the loss of elastic recoil
    - patients have usually lost 50% - 70% of tissue before symptoms appear
    - often difficult to distinguish from bronchitis - both may occur simultaneously
    - **INITIAL SYMPTOMS:** shortness of breath (exertional dyspnea)

Chronic Obstructive Pulmonary Disease (COPD)

Emphysema



Normal Lung Tissue



Lung Tissue with Emphysema

* Same magnification

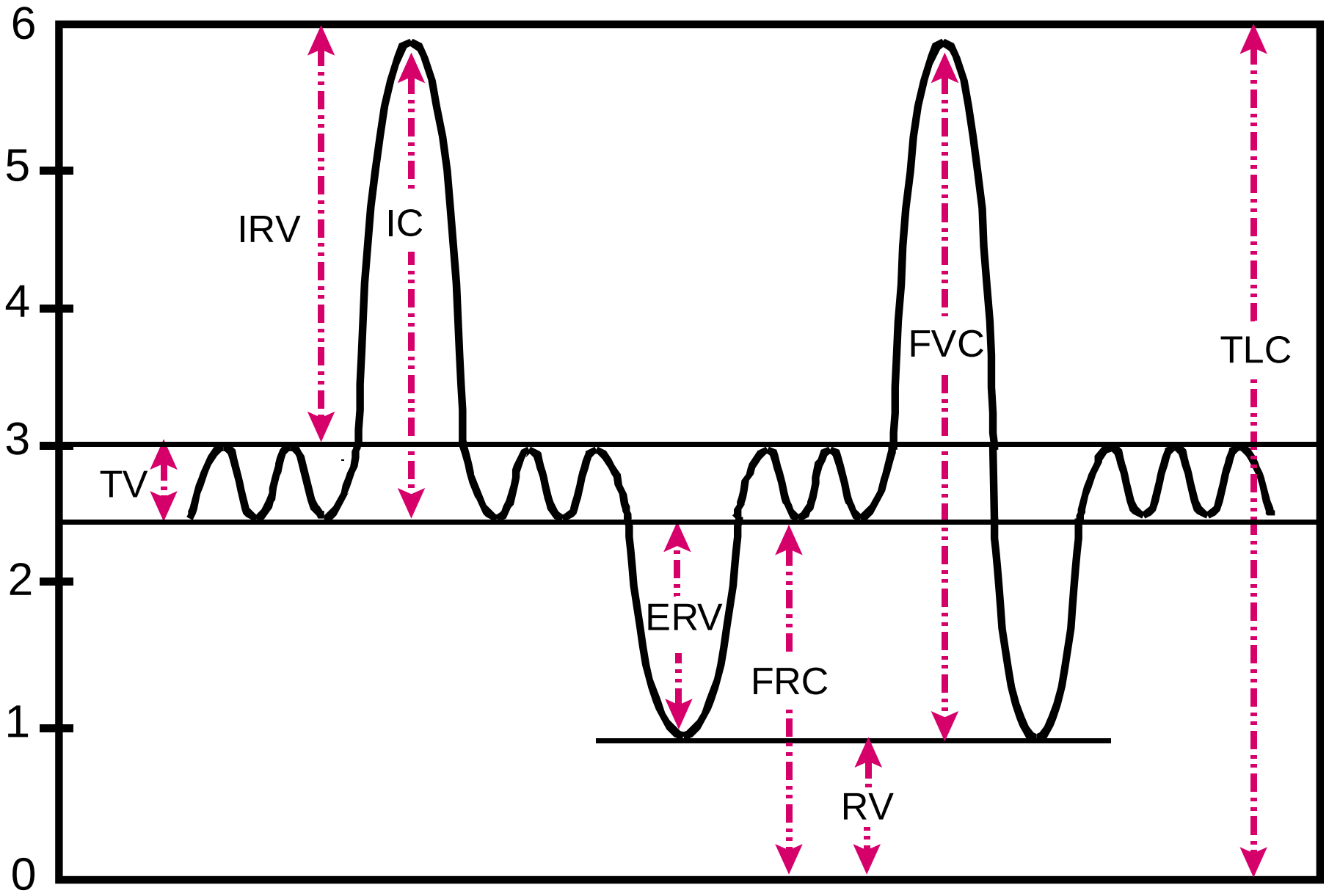
Chronic Obstructive Pulmonary Disease (COPD)

- **COPD epidemiology**
  - **13 million people have COPD (40 X lung cancer) - 4th leading COD**
  - **10 year mortality rate after diagnosis of chronic bronchitis: 50%**
  - **10 year mortality rate with FEV_1 < 20% predicted: 95% (any COPD)**
  - **Asthma is most common chronic disease in those < 17 years old**
    - responsible for 23% lost school days and 2 million ER visits per year
    - prevalence of asthma is increasing (75% from 1980 to 1994)
- **COPD pathophysiology notes**
  - **85% of COPD's are caused by smoking**
  - **COPD → ↑↑ chance of secondary infections: pneumonia & flu**
  - **Initially, COPD may be difficult to distinguish from CHF**
    - earliest sign of emphysema is exertional dyspnea
  - **Many patients have symptoms of both emphysema & bronchitis**
    - most COPD cases are individual combinations of bronchitis & emphysema
  - **Onset of chronic bronchitis is insidious**
    - person never fully recovers from a cold or a bout of influenza
    - has relapsing respiratory infections that become increasingly worse

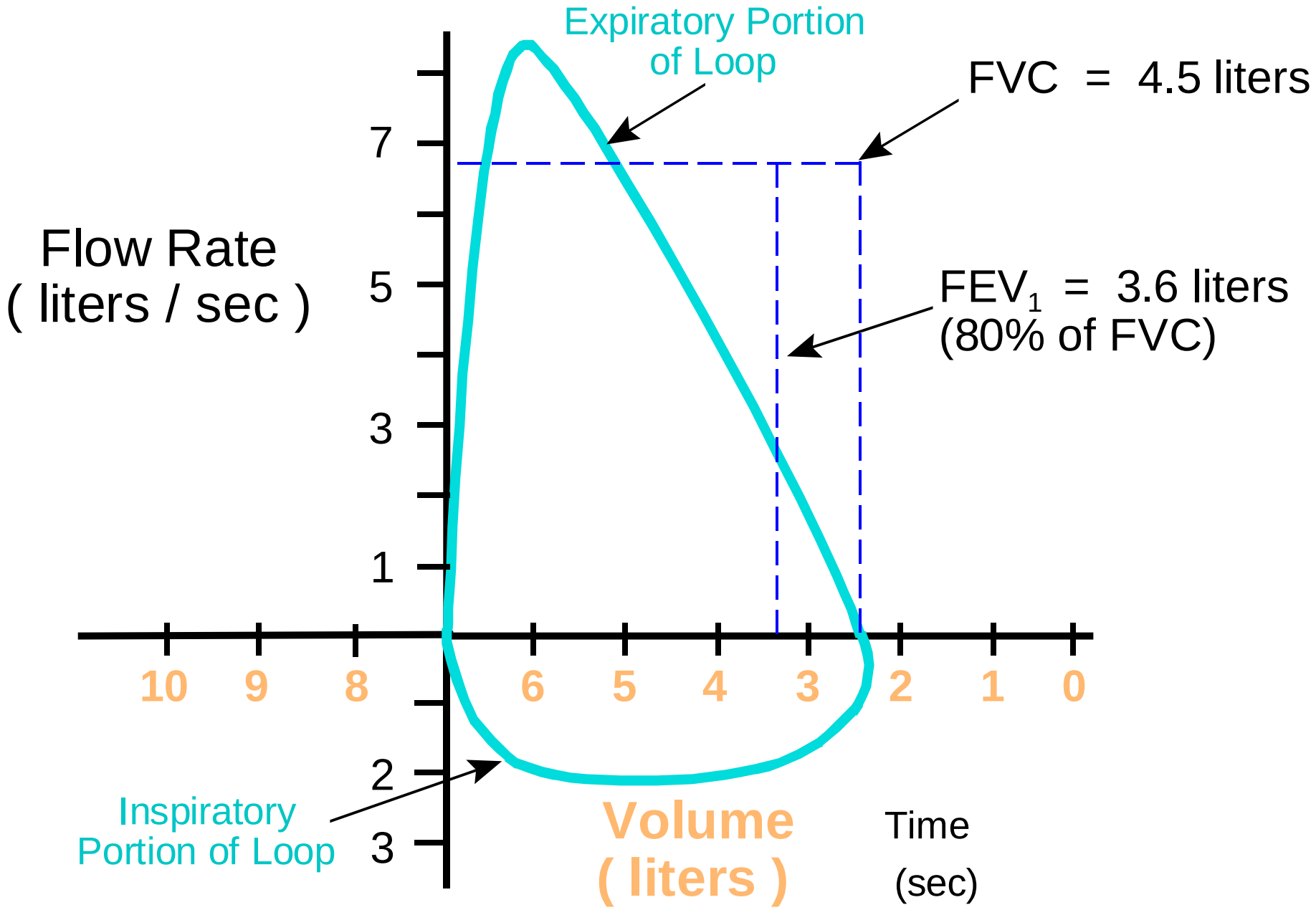
Chronic Obstructive Pulmonary Disease (COPD)

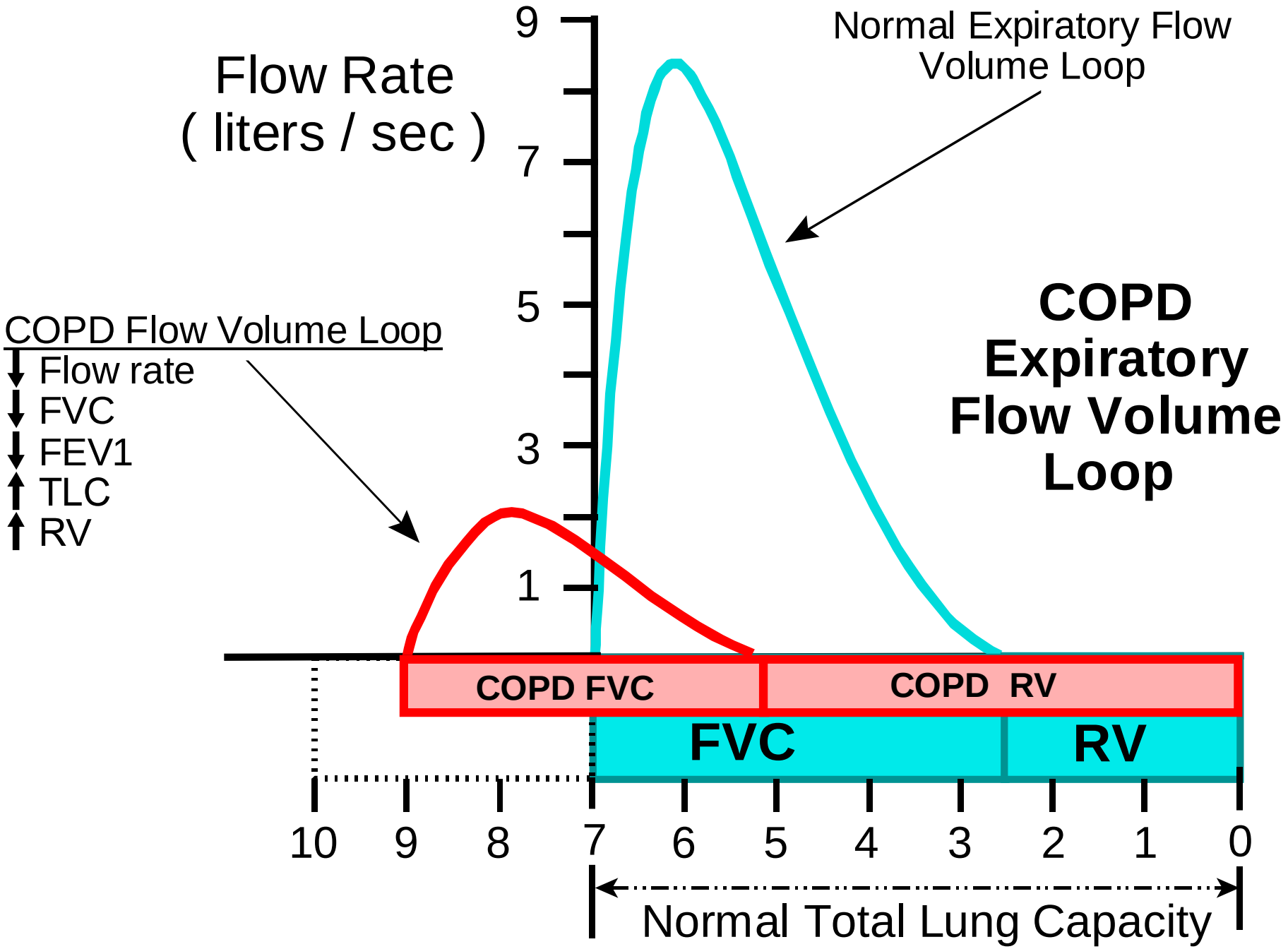
- **COPD pathophysiology notes (cont.)**
  - **In COPD, as the disease progresses:**
    - ↓ ventilation → ↓ $\dot{V}a / \dot{Q}$ → ↓ O_2 (hypoxia) + ↑ CO_2 (hypercapnea)
      - hypercapnea → headache
    - ↓ amount of lung tissue ventilated → body will not perfuse these areas
      - hypoxic vasoconstriction **(HV)**
    - ↓ lung vascular tissue (emphysema) + HV → ↑ PA pressure
      - ↑ PA pressure (pulmonary hypertension) → RV failure
        - RV failure called Cor Pulmonale
    - ↑ work of breathing (up to 17 fold)
    - In emphysema: lung hyperinflation → "barrel chest" (↑ lung capacity)
    - person breathes through pursed lips to optimize airflow
    - person may have a bluish discoloration (cyanosis) due to hypoxia
- **Diagnosis of COPDs**
  - **FVC and FEV_1 < 85% of predicted (severe: < 50% predicted)**
  - **FVC : FEV_1 ratio < .75** (others: chest x-ray, blood analysis)

Dynamic Lung Volumes



Normal Flow Volume Loop




Flow Rate
(liters / sec)
9
7
5
3
1
Normal Expiratory Flow
Volume Loop
COPD Flow Volume Loop
Flow rate
FVC
FEV1
TLC
RV
COPD
Expiratory
Flow Volume
Loop
COPD FVC
COPD RV
FVC
RV
10 9 8 7 6 5 4 3 2 1 0
Normal Total Lung Capacity

Chronic Obstructive Pulmonary Disease (COPD)

Pulmonary Insufficiency Scale and Grades

Grade	Dyspnea Cause	FEV1	MAX VO_2	BLOOD GASSES
1	Fast walking	> 60% pred.	> 25 ml/kg/min	norm. $PaCO_2$ & SaO_2
2	Regular walking	< 60% pred.	< 25 ml/kg/min	SAO_2 > 90% rest & exer.
3	Slow walking	< 40% pred.	< 15 ml/kg/min	SAO_2 < 90% during exer.
4	Walking limited to < 1 block	< 40% pred.	< 7 ml/kg/min	↑ PCO_2 is a problem SAO_2 < 90% rest & exer.

Chronic Obstructive Pulmonary Disease (COPD)

- **Treatment of COPD's**
  - **β_2 agonist bronchodilators (mainstay of COPD treatment)**
    - activation of β_2 receptors → bronchodilation
  - **Anti-cholinergic drugs**
    - ↓ acetylcholine → ↓ c-GMP in bronchial smooth muscle → ↓ constriction
  - **Theophylline**
    - ↑ c-AMP in bronchial smooth muscle → dilation
  - **Corticosteroids (AZMACORT PREDNISONE)**
    - ↓ prostaglandins → ↓ bronchoconstriction
    - side effects:
      - long term use reduces the effectiveness of the immune system
      - osteoporosis
  - **Combination of corticosteroids & β_2 agonist (ADVAIR)**
  - **Oxygen replacement therapy (for respiratory failure)**
    - needed when PO_2 drops below 60 mmHg (about 90% Hb saturation)
  - **Antibiotics for secondary lung infections & pneumonia**
  - **Lung transplantation and lung resection surgery**

Chronic Obstructive Pulmonary Disease (COPD)

- **Implications for exercise testing and Rx**
  - **Exercise Testing**
    - consider monitoring O_2 saturation with ear oximeter
    - gradual stage workload increments are used to precisely assess FC
    - for some patients, a discontinuous test may be used
    - for more impaired people, a 6 or 12 minute walking test may be used
      - measure the distance covered in 6 or 12 minutes - test is reproducible
    - severely impaired with O_2 → treadmill at 1-3 mph until symptoms appear

Chronic Obstructive Pulmonary Disease (COPD)

- **Implications for exercise testing**
  - **Effects of Exercise Training for Pulmonary Patients**
    - little or no improvement in spirometry measures
    - ↑ FC (exercise tolerance) with little or no increase in $VO_{2\ max}$
      - exercise is limited by the respiratory system
      - a minimum HR training intensity may not be attainable in some cases
      - respiratory muscles require more O_2 → ↓ O_2 available for exercising muscles → workloads that can be tolerated are low
        - for a given O_2 consumption COPD patients will have ↑ HR
        - for a given HR, COPD patients will be exercising at a ↓ workload
        - training adaptations may not occur in spite of attaining a THR
    - desensitization to dyspnea pain and sensation → ↓ anxiety

Chronic Obstructive Pulmonary Disease (COPD)

- **Implications for exercise training and Rx**
  - **Exercise Rx**
    - For Grade I patients, normal prescription parameters may be used
      - exercise capacity not limited by pulmonary insufficiency
    - For Grade II & III patients, intensity should not exceed a workload that requires more than approximately 30 breaths per minute or 60% - 80% of their ventilatory capacity
      - exercise duration may be limited to 5-10 minutes → multi-session days
    - For the more severe patients:
      - short bouts of low intensity exercise, progressing where possible
      - goal: ↑ duration of bouts → better day-to-day functioning
      - supplemental O2 may be needed

Diabetes: Type I and Type II

- **Diabetes Type I - Insulin Dependent - Juvenile Onset**
  - **Pathophysiology / Complications from Type I Diabetes**
    - marked reduction in insulin secreting β cells in the pancreas → ↓ insulin
    - ↑ plasma glucose → glucose in urine → ↑ urination → dehydration + ↑ thirst
    - ↑ fat metabolism → ↑ ketone production → KETOACIDOSIS
      - symptoms: fruit breath, thirst, ↑ need to urinate, hot dry skin
      - ↑↑ KETOACIDOSIS: leads to lethargy, unresponsiveness, & coma
    - diabetics must balance all parameters related to blood glucose levels
      - food & alcohol intake
      - exogenous insulin
      - normal "average" daily physical activity + exercise
    - ↓↓ food, ↑↑ exogenous insulin, ↑↑ physical activity → HYPOGLYCEMIA
      - symptoms: weakness, confusion, pale, dizziness, sweating, shakiness
      - ↑↑ HYPOGLYCEMIA (insulin shock) → unconsciousness and coma
        - diabetics should always carry a source of simple carbohydrate
        - exercise should be prescribed with caution
    - In a diabetic emergency, if in doubt, give sugar - DO NOT INJECT INSULIN

Diabetes: Type I and Type II

- **Diabetes Type I - insulin dependent – usually juvenile onset**
  - **Complications from Type I Diabetes**
    - **PAD** - poor perfusion in the extremities is possible
      - ↑ atherosclerosis + destructive changes in arterioles → ↓ blood flow
      - ↑ susceptibility to infections + ↓ wound healing → ↑ risk of amputation
      - prophylactic foot care is essential
    - **RETINOPATHY** - microaneurysms in retinal capillaries may hemorrhage
      - eventually occurs in 25% of patients with diabetes
      - bleeding → scar formation → retinal detachment → blindness
        - leading cause of blindness in US: ≅ 5000 new cases per year
        - bi-annual eye examinations are prudent
    - **NEUROPATHY** - nerve damage from ↓ blood supply + ↑ sugar toxicity
      - occurs in 50% of patients who have been diabetic for 25 years
      - causes tingling, pain, and loss of sensatory discrimination

Diabetes: Type I and Type II

- **Complications from Type I Diabetes (cont.)**
  - **NEPHROPATHY** - destruction of kidney glomeruli
    - symptoms appear only after 30% of glomeruli have been destroyed
    - will develop in 25% - 40% of diabetic patients
    - most common cause of kidney failure in US
    - initial symptom is albumin in the urine (albuminuria)
    - as disease progresses → ↑ BUN, ↑ creatinine (filtration begins to fail)
      - eventually, kidney failure → dialysis
  - **HYPERTRIGLYCERIDEMIA + HYPERLIPIDEMIA** → ↑ CAD risk (2.5 - 5 X)
    - lipoprotein lipase → plasma triglycerides stored in adipocytes
      - ↓ insulin → ↓ LPL activity → ↑ plasma triglycerides
    - overproduction of VLDL & TG from liver → ↑ plasma lipids + ↓ HDL-C

Diabetes: Type I and Type II

- **Type II Diabetes**
  - accounts for approximately 90% of all diabetes cases
    - 16 million diabetics in US - 14.5 million are type II
    - almost half of all diabetic individuals remain undiagnosed
  - prevalence of Type II diabetes is in epidemic proportions due to:
    - ↑ longevity
    - ↑ obesity
    - ↑ physical inactivity
- **Pathophysiology**
  - ↑ insulin resistance due to receptor dysfunction is the initial abnormality
    - this is termed impaired glucose tolerance (IGT)
    - hepatic glucose production increases, exacerbating the IGT state
  - IGT → ↑ insulin secretion (hyperinsulinemia) → ↑ glucose uptake
    - compensation occurs
  - ↑↑ IGT + overworked β cells fail to secrete enough insulin → hyperglycemia

Diabetes: Type I and Type II

- **Risk Factors for Type II Diabetes**
  - family history (very strong - as yet, specific genes have not been identified)
    - Type I diabetes also has a strong heredity component
  - obesity (80% of type II diabetics are obese at diagnosis)
  - age: most type II diabetics are over 50 years of age
- **Treatment for Type II Diabetes**
  - early diagnosis of IGT → diet modification & exercise for 3 months
  - first line drug of choice: Sulfonylurea drugs which stimulate insulin secretion
  - Glucophage: inhibits hepatic glucose production
  - Acarbose: delays the breakdown of complex carbohydrates to glucose
  - Troglatazone: ↓ insulin resistance in skeletal muscle
  - combinations of the above
  - insulin therapy in some cases

Diabetes: Type I and Type II

- **Implications for exercise testing and Rx**
  - **Exercise Testing**
    - make sure a source of simple carbohydrate is available
    - consider lower limb inspection to make sure no infections are present
    - exercise hemodynamic & metabolic responses may be different
      - diabetes can impair ventricular function
      - max HR and $\dot{V}O_{2\ max}$ may be 15% - 20% below normal
    - hypoglycemic responses may be masked in those taking β-blockers
  - **Benefits of Moderate Exercise for the Type I or Type II Diabetic**
    - ↑ insulin sensitivity → exogenous insulin requirement may ↓ by 20% - 60%
      - reduced need for oral hypoglycemic agents in type II diabetics
    - ↓ cardiovascular risk factors
    - ↓ stress → ↓ counterregulatory hormones → improved glucose control
      - stress, shock, or very intense exercise produces:
        - ↑ glucagon → ↑ production of hepatic glucose → ↑ blood glucose
        - ↑ epinephrine → ↑ hepatic lipase activity → ↑ plasma triglycerides
        - both of the above reactions to stress may exacerbate diabetic state

Diabetes: Type I and Type II

- **Considerations for Prescribing Exercise**
  - make sure an ID is worn that identifies the person as diabetic
  - make sure lower extremities are examined before beginning program
    - consider a doppler examination of lower extremities to rule our PVD
  - do not exercise if:
    - blood glucose levels are 1) > 200 mg/dl or 2) < 100 mg/dl
      - 1) exercise → ↑ glucagon → ↑ hepatic glucose → ↑ hyperglycemia
      - 2) exercise → ↓ glucose → ↑ hypoglycemia
      - **EXERCISE ACTS LIKE INSULIN**
    - there has been recent retinal hemorrhage or other retinopathy problems
    - illness or infection is present
  - Once again, remember that diabetics must balance:
    - food intake
    - daily normal energy expenditure
    - exogenous insulin dosage
    - exercise **(EXERCISE ACTS LIKE INSULIN)**
  - **Excess insulin + exercise can quickly lead to hypoglycemia**
    - **have a source of simple carbohydrate available during exercise**

Diabetes: Type I and Type II

- **Considerations for Prescribing Exercise (cont.)**
  - at the onset, consider monitoring blood glucose levels pre & post exercise
  - **example: anticipated exercise exceeds 1 hour in the morning**
    - **↓ insulin 25% before breakfast (regular insulin)**
      - **35% - 55% ↓ if sustained activity is vigorous**
  - avoid insulin injection in exercising muscle
    - ↑ insulin absorption and rate of action → ↑ chance of hypoglycemia
  - avoid exercising during peak levels of insulin activity
    - 2 - 4 hours after injection (depending on the type of insulin used)
  - best time to exercise is 1 - 2 hours after eating a small meal or snack
    - post prandial glucose levels peak during this time
    - ideal pre-exercise glucose value is 120 – 180 mg / dL
    - optimum fuel availability without danger of hypoglycemia
    - snack containing 30 - 50 calories of carbohydrate should be consumed every 30 - 45 minutes during bouts of prolonged exercise

Diabetes: Type I and Type II

- **Exercise Rx**
  - normal parameters for exercise Rx may be used keeping in mind:
    - many diabetics have been sedentary
      - consider beginning at low intensities and durations

Renal Disease and Renal Failure

- **Chronic Renal Failure - irreversible loss of large numbers of nephrons which decrease glomerular filtration capacity**
- **End Stage Renal Disease - progression of chronic renal failure to the point where the kidneys are functioning at < 10% of capacity**
  - **↓ the ability to take certain drugs (cannot be metabolized & filtered)**
  - **Epidemiology**
    - **mortality from ESRD has increased 52% in the last 16 years.**
    - affects 4 in 10,000 people
    - about 100,000 people in the US are on some sort of dialysis
    - chronic renal failure may exist from 10 - 20 years before ESRD onset
  - **Pathophysiology**
    - structural damage of parenchymal tissue → loss of glomerular filtration
      - damage is usually caused by
        - diabetes
        - hypertension
        - chronic infection & inflammation (glomerulonephritis)
        - auto-immune disorders

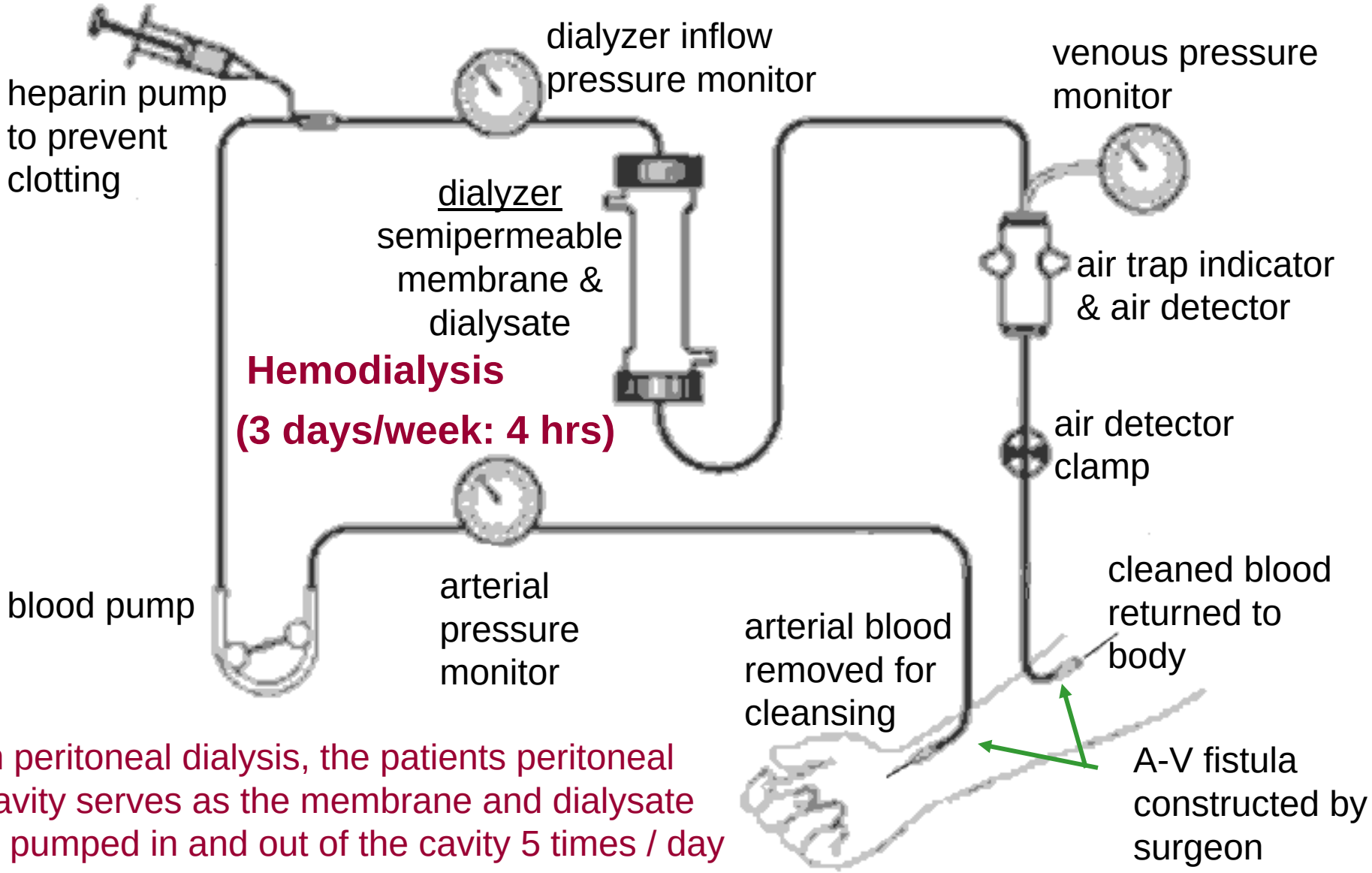
Renal Disease and Renal Failure

- **Pathophysiology (cont.) and Symptoms**
  - nephron # ↓ to 30% of normal → ↓ GFR → fluid & electrolyte retention
    - generalized edema and abdominal bloating,
    - ↑ blood pressure
    - fluid on the lungs → dyspnea
    - electrolyte imbalance → leg and muscle cramps, severe hyperkalemia
  - ↓ glomerular filtration → ↑urea (uremia), N_2 and creatitine from protein metabolism
    - ↑ BUN (common test) - normal range from 7–20 mg/dL
    - ↑ creatinine [↓ creatinine clearnace] (common test) normal range .8–1.4 mg/dL
    - ↑ buildup of H^+ → ↓ pH (acidosis: pH of < 6.4….normal pH = 7.3)
  - loss of kidney secreted erythropoietin → ↓ RBC production → anemia
  - electrolyte imbalances → arrhythmias
  - fluid overload → pathogenic changes in the heart muscle
  - abnormal platelet function → ↑ bruising and bleeding
  - renal osteodystrophy (abnormal Ca^{++} metabolism → ↓ bone mineralization)
    - ↓ bone density, ↑ fiber deposition in bones → pain + bone weakness
  - accumulation of toxic waste products → fatigue & weakness
    - $\dot{V}O_{2\ max}$ is usually between 15 - 20 ml / kg / min

Renal Disease and Renal Failure

Treatment: Hemodialysis (90%) or Peritoneal Dialysis (10%)

Usually done 4 hrs 3/wk – recent study → nocturnal HD for 8 hr 3/wk → 80% ↑ in survival



In peritoneal dialysis, the patients peritoneal cavity serves as the membrane and dialysate is pumped in and out of the cavity 5 times / day

Renal Disease and Renal Failure

- **Implications for exercise testing and Rx**
  - **Exercise Testing**
    - GXT may not be efficacious in determining cardiac status because:
      - inability to ↑ HR (max HR in dialysis patients is about 75% of normal)
      - ECG findings may be abnormal, precluding interpretation of ischemia
        - electrolyte disturbances → arrhythmias
        - digitalis (many ESRD patients are in heart failure)
        - LVH
      - hypertensive response to exercise (18%↑ SBP by > 22 mmHg / MET)
    - testing should be done on non-dialysis days
    - start protocol at 1.5 METs, increasing it .5 METs each stage
  - **Benefits of Exercise for the ESRD patient**
    - ↑ $\dot{V}O_{2\ max}$ by 20% - 40% → ↑ functional capacity for everyday living tasks
      - ↓ anxiety & depression
    - ↓ CHD risk factors (remember CHD is highly prevalent in ESRD patients)
      - ↑ HDL-C and improve insulin sensitivity
    - ↑ Hematocrit & Hemoglobin

Renal Disease and Renal Failure

- **Considerations for Prescribing Exercise**
  - remember: some patients may not be good candidates for exercise
  - because of ↑ CHD risk, exercise in a monitored setting if possible
    - problem: Medicare & other insurance may not pay
  - consider the amount of time dedicated to the disease
    - dialysis for 3 hours, 3 times / week, now exercise 1 hour, 3 times / week
  - exercise during dialysis is both practical and feasible
- **Exercise Rx**
  - since most ESRD patients have low functional capacities:
    - begin at very low intensity using the RPE scale as an indicator
      - HR response to exercise may be abnormal in ESRD patients
    - multiple bouts of exercise with rest intervals may be necessary
    - Consider beginning as follows:
      - 5 minutes, 2 times /day, 3 days per week (possibly during dialysis)
      - increase bout duration 2 minutes each day up to 15 minutes / bout
      - consider increasing frequency to 4 - 6 days per week

Pregnancy

- **Benefits of Exercise for Pregnant Women**
  - ↓ excessive swelling, backache, leg cramps, constipation, and bloating
  - Lower overall weight gain
  - Slightly lower birth weights (still well within normal limits)
  - Psychological benefits (↓ depression & anxiety, ↑ sense of well being)
  - Larger placenta (better nutritional base for the baby)
  - Possible reduction of time in labor
  - Possible easier delivery and speedier recovery
  - Possible reduced chance of Caesarean birth
  - Possible reduced likelihood of resultant varicose veins

Pregnancy

- **Contraindications to Exercise (reasons to discontinue)**
  - Significant structural heart disease, ventricular dysfunction, or arrhythmia
  - Uncontrolled hypertension, thyroid disease, diabetes, or other systemic maladies
    - asthma, severe headaches, dizziness or syncope, visual disturbances
  - Diagnosis of incompetent cervix
  - Spontaneous abortions or premature labor in previous pregnancies
  - Bleeding or diagnosis of placenta previa (placenta in lower segment of uterus)
  - Breech presentation in third trimester (fetus oriented "feet first"), multiple fetuses
  - Ruptured membranes or premature labor in the current pregnancy
    - any type of contraction, abdominal pain, vaginal discharges
  - Pre-eclampsia / eclampsia (hypertension, edema & proteinuria / convulsions)
  - Elevation of HR or BP that persists after exercise, chest palpitations or pain
  - Sudden swelling of ankles, hands, or face
  - Smoking, excessive alcohol intake, or inadequate weight gain
  - Excessively low body fat percentage (bad nutrition, history of eating disorder)
  - Anemia, iron deficiency, or excessive fatigue after exercise
  - Swelling, redness, or pain in the calf of one leg (phlebitis)
  - Taking medications that can alter cardiac output or blood flow distribution

Pregnancy

- **Considerations for Prescribing Exercise for Pregnant Women**
  - choose exercises that minimize risk of falling & abdominal trauma
    - consider extra pounds and center of gravity shift
    - no bench stepping, kick boxing, roller blading, etc
    - walking, cycling, low impact aerobics, water aerobics are best
      - water aerobics → body support and heat dissipation
  - gestational hormones (relaxin) ↑ connective tissue laxity:
    - ↑ chance of muscle / ligament strain and overuse injury
    - again, avoid heavy weight bearing or bounding exercises
  - start slowly - especially if the mother has never exercised before
  - avoid exercise at altitude or hot, humid environments (hypoxia and hyperthermia)
  - do regular Kegel exercises regularly (tightening the pelvic floor muscles)
    - helps prevent incontinence

Pregnancy

- **Considerations for Prescribing Exercise for Pregnant Women (cont.)**
  - General rule: THRs < 140 - 150 bpm, durations < 35 minutes, 3 - 5 days / week
  - 12 or 13 or the 20 point Borg RPE scale is also a good intensity guideline
    - **RATIONALLE**:
    - RHR is already ↑ by 5 - 12 bpm from the pregnancy (↓ TPR → NC in RBP)
    - fetal bradycardias have been reported after exercise suggesting hypoxia
    - less chance of reduced uterine blood flow and elevated fetal temperatures
      - mother's body temperature should never exceed 100 degrees F
    - ↑ exercise intensity → ↑ maternal glucose utilization → ↓ fetal hypoglycemia
  - Avoid exercises where you lay flat of your back as this ↓ blood flow to uterus
    - especially important after the first trimester
  - Do not ↑ the volume of exercise prior to the 15th week or after the 25th week
  - Weight lifting is recommended with the following modifications:
    - ↑ rest periods between sets
    - do not strain to maximally do a given number of reps - no one rep max's
      - no valsalva maneuvers
    - avoid exercises requiring balance (lunges, squats, etc.) or abdominal work

Rheumatoid Arthritis & Osteoarthritis

- **Benefits of Exercise for the Arthritis Patient**
  - **↑ functional capacity and $\dot{V}O_{2max}$ (diminish the effects of inactivity)**
  - **Reduction in the speed of disease progression**
  - **Improved psychosocial profile**
  - **Possible reduction in joint swelling, stiffness, and pain**
  - **Possible reduction in bone loss**
  - **Assistance with weight management → ↓ stress on joints**
  - **Movement → body produces proteoglycans (joint lubricant)**

- **Exercise Testing & Training for the Arthritis Patient**
  - **Exercise Testing**
    - a normal symptoms limited maximal test may be administered if tolerable
      - cycle ergometry may be mode of choice (non-weight bearing)
      - start protocol at low workloads and make small stage advances
        - better to assess accurate functional capacity

Rheumatoid Arthritis & Osteoarthritis

- **Considerations for Prescribing Exercise for the Arthritis Patient**
  - Do not do any kind of weight bearing exercise in the presence of acute flare-ups
    - red, hot, swollen, and painful joints
    - still should do non weight bearing flexibility & warm-up exercises
  - Initially, range of motion and flexibility exercises should be the prime component
  - Choose exercises that reduce the load on hip & knee joints (low impact activities)
    - stationary cycle, rowing machine, water aerobics, water running
    - consider cross training to gradually implement some weight bearing activity that is representive of daily activities (walking, stair climbing, lifting)
    - implement a strength training program for major muscle groups as tolerated
      - ↑ strength of skeletal muscles → ↓ stress on joint
    - consider using interval training with substantial rest periods or exercising 3 times per day
    - although some discomfort may be expected during and post exercise, avoid activities that provoke severe joint pain that lasts > 2 hours
  - Normal age-associated parameters may be used to compute a THR, but remember that most arthritic patients are sedentary → start at low intensities and durations