

Pericardial Disease	Pathology & Causes	Clinical Presentation	Diagnostics	Management
<p><b>Acute Pericarditis</b></p> <p>1/1000 hospital admissions</p> <p>Men &gt; Women Adults &gt; Children</p> <p>Complications</p> <ul style="list-style-type: none"> <li>- 9% → mild constriction within 30 days → 3 mo spontaneous remission</li> <li>- Recurrence → 25%</li> <li>- Tamponade, constrictive pericarditis</li> </ul>	<ul style="list-style-type: none"> <li>• Idiopathic</li> <li>• <b>Viral</b> – Coxsackie A&amp;B, Echo, Adeno, Mumps virus</li> <li>• Uremic – renal failure</li> <li>• Bacterial – pneumococcus, strep, gram (-) sepsis</li> <li>• Acute MI (Dressler's syndrome)</li> <li>• Cardiac surgery, TB, neoplasm, trauma, radiation, autoimmune disorders, myxedema, drugs, inflammatory diseases</li> </ul>	<ul style="list-style-type: none"> <li>• Chest pain – left precordium radiates to trapezius; begins 10-14 days after viral syndrome; similar to ischemic pain; NOT EFFORT RELATED</li> <li>• Pericardial friction rub</li> <li>• EKG changes</li> <li>- <u>aggravated by</u>: supine, cough, deep inspiration</li> <li>- <u>alleviated by</u>: sitting up, leaning forward</li> <li>- Dyspnea – shallow breathing to avoid pleuritic pain, fever, effusion compressing lung and bronchi</li> </ul>	<ul style="list-style-type: none"> <li>• Friction rub – scratching, grating, high-pitched, 3 components <ul style="list-style-type: none"> <li>○ Presystolic</li> <li>○ Systolic</li> <li>○ Ventricular filling (Cardiac motion)</li> </ul> </li> <li>• Auscultation – Use diaphragm, lower left sternal border, inspiration and full exhalation, sitting up and leaning forward</li> <li>• Chest x-ray</li> <li>• Blood tests – nonspecific markers of inflammation, increase WBC &amp; ESR</li> <li>• Echo – effusion</li> </ul>	<ul style="list-style-type: none"> <li>- NSAIDS</li> <li>- ASA</li> <li>- Glucocorticoids (for resistant situations)</li> </ul>
<p><b>1. Pericardial Effusion</b></p> <p>a. w/o compression</p> <p>b. w/ compression</p> <p><b>2. Cardiac Tamponade</b></p>	<p>1b/2 – caused by ↑ pericardial fluid → ↑ <b>intrapericardial pressure</b></p>	<p>1b/2 – elevation of intracardiac pressures, limitation of ventricular diastolic filling, decrease stroke volume and cardiac output</p> <p><b>2. Jugular venous distention</b></p> <p>Beck's triad – ↓ BP, ↑ systemic venous pressure, ↓ heart sounds</p> <p>Tachycardia, tachypnea</p> <p><b>Pulsus paradoxus</b></p> <p>Friction rub</p> <p>Hypotension in only 1/3 of pts</p>	<p>1a. Echo is most sensitive, accurate, and widely used – size, compression vs. no compression</p> <p>2. right heart cath</p> <ul style="list-style-type: none"> <li>- ↑ RAP with ↓ or absent y descent</li> <li>- RV mid diastolic pressure is ↑ and = to RAP</li> <li>- PCWP = RAP</li> <li>- LV diastolic pressure = RAP</li> <li>- ↓ <b>RAP with inspiration</b></li> </ul>	<p>1. Observation/pericardiocentesis if compression or need for diagnosis</p> <p>2. pericardiocentesis, volume loading prior to procedure for hemodynamic support</p>
<b>Constrictive Pericarditis</b>		<ul style="list-style-type: none"> <li>• Edema, abdominal swelling, ascites and</li> </ul>	<ul style="list-style-type: none"> <li>• ↑ <b>JVP</b> → singular most significant finding</li> </ul>	<ul style="list-style-type: none"> <li>- <b>surgical stripping or</b></li> </ul>

<p>Fibrotic, thickened, adherent pericardium restricts diastolic filling of the heart          Develops after an episode of acute pericarditis</p> <ul style="list-style-type: none"> <li>- Effusion usually develops then is reabsorbed</li> <li>- Chronic stage of fibrosis and thickening of pericardium</li> <li>- Calcium deposits</li> </ul>		<p>hepatic congestion → secondary to ↑ systemic venous congestion</p> <ul style="list-style-type: none"> <li>• Exertional dyspnea, cough, orthopnea → secondary to ↑ R and L heart pressures</li> </ul>	<ul style="list-style-type: none"> <li>• Diastolic pericardial knock             <ul style="list-style-type: none"> <li>○ Early diastole, left sternal border</li> <li>○ Sudden cessation of ventricular filling</li> </ul> </li> <li>• Hepatomegaly</li> <li>• <u>CXR</u> – extensive calcification, pleural effusions</li> <li>• <u>EKG</u> – low voltage; inverted T waves, LA abnormalities</li> <li>• <u>Echo</u> – immobile, dense pericardium dilation of IVC and hepatic veins</li> <li>• <u>CT</u> – pericardial thickening, dilation of IVC and hepatic veins</li> </ul>	<p><b>removal of both layers of the adherent pericardium is the definitive therapy</b></p>
--	--	---	--	--

### Valves

Valvular Disease	Etiology	Pathophysiology	Clinical Presentation	Evaluation
<p><b>Aortic Stenosis</b></p> <ul style="list-style-type: none"> <li>• Onset of angina, mortality in 5 years</li> <li>• Onset of syncope, mortality in 3 years</li> <li>• Onset of CHF, mortality in 18 mo</li> <li>• Congenital bicuspid valve may be asymptomatic for decades</li> </ul>	<ul style="list-style-type: none"> <li>• Rheumatic</li> <li>• Calcific/degenerative</li> <li>• Congenital = <b>bicuspid most common</b></li> <li>• Post-endocarditis</li> </ul>	<ol style="list-style-type: none"> <li>1. Congenital uni- or bicuspid valve functions normally for decades until turbulence induces calcification</li> <li>2. Degenerative calcification occurs similarly, but from years of “wear &amp; tear” – esp. in hypertensives</li> <li>3. High outflow resistance causes concentric LVH</li> <li>4. High LV filling pressure</li> <li>5. LV filling pressure reflected to atrium</li> <li>6. Systolic function preserved until late</li> <li>7. Hypertrophied ventricle becomes less compliant – “stiff”</li> <li>8. Atrial contraction</li> </ol>	<ul style="list-style-type: none"> <li>• Triad of symptoms – occur sequentially             <ul style="list-style-type: none"> <li>○ Chest pain</li> <li>○ Exertional syncope or near syncope</li> <li>○ Dyspneal/orthopnea – CHF</li> </ul> </li> <li>• Delayed &amp; weakened carotid upstroke (“<i>pulsus tardis et parvis</i>”)</li> <li>• <b>Late peaking crescendo/decrescendo murmur at base w/ radiation to carotids</b></li> <li>• Soft or absent S2</li> <li>• Hyperdynamic apical impulse S4 at apex</li> <li>• Murmur may decrease w/ standing</li> <li>• Significant systolic HTN <i>rare</i> (over 180 mm)</li> </ul>	<ul style="list-style-type: none"> <li>• <u>CXR</u>: normal to slightly enlarged LV</li> <li>• <u>2D Echo</u>: structure &amp; mobility of valve leaflets; thickness &amp; function of ventricular walls</li> <li>• <u>Doppler</u>: quantify gradient &amp; calculate valve area (most diagnostic)</li> <li>• Cardiac catheterization</li> <li>• <u>ECG</u>: LVH</li> </ul> <p><b>Therapy</b></p> <ol style="list-style-type: none"> <li>1. Not a “medical” illness – no medical therapy can significantly alter prognosis or symptoms</li> <li>2. Surgery – Most appropriate intervention; can successfully be done even in severely impaired ventricles, w/ improvement</li> </ol>

		<p>provides proportionally more LV filling volume as opposed to passive filling</p> <p>9. As gradient ↑, peak ejection delayed later in systole</p> <p>10. Pulse volume ↓</p> <p>11. Ventricular hypertrophy &amp; high filling pressures lead to subendocardial ischemia</p>	<ul style="list-style-type: none"> <li>• <b>Paradoxical split S2</b></li> <li>• Palpable thrill over base common in severe AS</li> <li>• Rare association w/ colonic angiodysplasia &amp; lower GI bleed</li> </ul>	<p>in symptoms &amp; mortality; critical valve area for intervention is 0.75 cm<sup>3</sup>; surgery should not be delayed until CHF occurs, but must be planned after gradient established</p> <p>3. Balloon valvuloplasty – initial success rate excellent; restenosis rapid &amp; profound, most w/in 6 mo; present indication only for “bridge” to surgery in pts w/ other transient lo-morbid illness</p>
<p><b>Aortic Regurgitation</b></p> <ul style="list-style-type: none"> <li>• Chronic regurgitation tolerated well for yrs until dilation &gt; metabolic accommodation</li> <li>• Symptoms commonly appear <i>after</i> development of irreversible LV dysfunction</li> <li>• Acute AR poorly tolerated, &amp; leads readily to pulmonary edema (PE) &amp; death if not corrected</li> </ul>	<ul style="list-style-type: none"> <li>• Endocarditis</li> <li>• Rheumatic fever</li> <li>• Calcific regeneration</li> <li>• Trauma</li> <li>• Aortic root disease (cystic medial necrosis, Marfan’s syndrome, annulo-ectasia, aortic aneurysm/dissection, syphilitic aortitis, seronegative arthropathies)</li> </ul>	<ol style="list-style-type: none"> <li>1. High pressure leaks ↑ LV filling volume, raises LVEDP, &amp; causes dilation</li> <li>2. High pressure leak also causes LVH</li> <li>3. “<i>cor bovinum</i>” – large, thick-walled heavy hearts</li> <li>4. Well tolerate until myocardial compensation outstrips vascular supply</li> <li>5. Systolic function preserved</li> <li>6. High stroke volume causes most peripheral physical findings of AI</li> <li>7. SVT normal to low</li> <li>8. Wide pulse pressure (&gt; 60 mm Hg) common</li> </ol>	<ul style="list-style-type: none"> <li>• <b>Bounding central pulses</b> (Corrigan’s or water hammer)</li> <li>• <b>Diastolic decrescendo murmur at 2<sup>nd</sup> interspace right, radiating to apex, heard best in end expiration, leaning forward</b></li> <li>• <b>Laterally displaced PMI</b>, w/ hyperdynamia &amp; enlargement</li> <li>• Anterior heave</li> <li>• Wide pulse pressure</li> <li>• <b>Austin Flint murmur</b> (<i>murmur due to aortic regurgitation originating at the mitral valve when blood enters simultaneously from both the aorta &amp; the LA</i>)</li> <li>• Quinke’s pulses (nailbed capillary pulsations)</li> <li>• Duroziez’ sign</li> <li>• <i>Pulsus bisferiens</i> (<i>pulse w/ two strong systolic peaks separated by a midsystolic dip, usually seen in pure aortic regurgitation &amp; aortic regurgitation w/ stenosis</i>)</li> </ul>	<ol style="list-style-type: none"> <li>1. <u>History</u> – functional capacity</li> <li>2. physical exam</li> <li>3. <u>2D Echo</u>: LV chamber dimension, wall thickness, &amp; motion</li> <li>4. <u>Doppler</u>: quantification of regurgitant flow</li> <li>5. Left heart catheterization</li> <li>6. <u>CXR</u>: cardiomegaly</li> <li>7. <u>ECG</u>: LVH</li> </ol> <p><b>Therapy</b></p> <ol style="list-style-type: none"> <li>1. ↓ Afterload</li> <li>2. Inotropes late</li> <li>3. ↓ Preload (if PE is present)</li> <li>4. limit strenuous exercise</li> <li>5. maintain sinus rhythm</li> <li>6. antibiotic prophylaxis</li> <li>7. surgery – indicated at onset of symptoms, or if functional capacity fails; LV end systolic dimension 5 cm or diastolic dimension 7 cm; if root aneurismal, need combined valve/root</li> </ol>

			<ul style="list-style-type: none"> <li>Severity correlates w/ duration of murmur</li> </ul>	replacement
<p><b>Mitral Stenosis</b></p> <ul style="list-style-type: none"> <li>5 yrs to progress from mild to severe disability</li> <li>80% 5 year survival after surgery</li> <li>Surgical risk avgs 6%, higher in elderly</li> <li>Valvuloplasty restenosis rate 80% in 10 yrs</li> </ul>	<ul style="list-style-type: none"> <li><b>Rheumatic fever</b> – seen 2-10 yrs after acute infection (less common in USA due to antibiotics)</li> <li>Degenerative calcific disease – elderly</li> <li>CT disorders (SLE, RA)</li> <li>Congenital</li> </ul>	<ul style="list-style-type: none"> <li>Rheumatic fusion at tips; calcific fusion from cusps</li> <li>Normal orifice 4-6 cm<sup>2</sup>, critical stenosis &lt;1 cm<sup>2</sup></li> <li>Gradient develops, raising arterial pressure</li> <li>Pulmonary HTN – longstanding</li> <li>Atrial dilation → a-fib</li> <li>LV pressure normal</li> <li>LV contractility normal</li> <li>Tachycardia ↓ LV filling volume &amp; CO</li> <li>↑ pulmonary artery pressure can impair RV function</li> </ul>	<ul style="list-style-type: none"> <li>Exertional dyspnea/orthopnea</li> <li>Hemoptysis w/ pulmonary HTN</li> <li>Palpitations</li> <li>Thromboembolism</li> <li>Endocarditis</li> <li><b>Diastolic low pitched apical murmur</b></li> <li><b>Opening snap after S2, before murmur</b></li> <li>S1 loud w/ rheumatic &amp; soft with calcific</li> <li>RV heave</li> <li><b>Normal PMI &amp; function</b></li> </ul>	<ul style="list-style-type: none"> <li><u>CXR</u>: Large RV; pulmonary vascular engorgement. No LV dilation. Large LA</li> <li>Thickened, fused leaflets on echo</li> <li><u>ECG</u>: atrial abnormality, RVH</li> <li><u>Doppler</u>: gradient &amp; valve area</li> <li>Right &amp; left heart cath</li> </ul> <p><b>Therapy</b></p> <ul style="list-style-type: none"> <li>Diuresis for acute pulmonary edema</li> <li>Maintain sinus rhythm: AA</li> <li>Prevent tachycardia: β blockers</li> <li>Digoxin for rate in a-fib</li> <li>Oral anticoagulants</li> <li>Antibiotic prophylaxis</li> <li>Balloon valvuloplasty</li> <li>Open commissurotomy</li> <li>Valvular replacement</li> <li>Surgery (to prevent irreversible pulmonary HTN)</li> </ul>
<p><b>Mitral Regurgitation</b></p> <ul style="list-style-type: none"> <li>Small leaks tolerated for normal life span</li> <li>Marker for significant LV dysfunction indicating need for surgery is LV end diastolic dimension of 7 cm or LV end systolic dimension</li> </ul>	<ul style="list-style-type: none"> <li>Leaflet destruction – infection (SBE), calcific degeneration, CT disorder, trauma</li> <li>Myxomatous degeneration of valve (MVP, Marfan)</li> <li>Papillary muscle dysfunction – ischemia/infarction, myxomatous</li> </ul>	<ul style="list-style-type: none"> <li>Leak unloads LV into low pressure atrium</li> <li>Worsened by ↑ systemic afterload</li> <li>LAP &amp; PWP ↑</li> <li>Atrium dilates</li> <li>LV dilates to accommodate ↑ filling volume</li> <li>Contractility eventually ↓</li> <li>Pulmonary HTN &amp; edema</li> </ul>	<ul style="list-style-type: none"> <li>Symptoms depend on severity of leak and time frame</li> <li>Exertional dyspnea/orthopnea are late findings</li> <li>Palpitations w/ arrhythmia</li> <li>Atrial fib: tolerated better than in MS</li> <li>RHF at end-stage</li> <li>Fatigue, weakness due to ↓ CO</li> <li><b>Laterally displaced PMI w/ enlargement</b></li> <li>S3 common</li> </ul>	<ul style="list-style-type: none"> <li><u>CXR</u>: LV &amp; LA dilation, pulmonary vascular engorgement</li> <li><u>Echo</u>: LV &amp; LA enlargement; wall motion normal until late</li> <li><u>Doppler</u>: identify &amp; quantify regurgitant jet</li> <li><u>ECG</u>: LVH in 1/3, LAA in &gt;50%, RVH in 15%, a-fib</li> <li>EF normal early, reduced late</li> <li>Cardiac cath</li> </ul>

<ul style="list-style-type: none"> <li>of 5 cm</li> <li>LVEF &lt; 40% indicates severe, possibly inoperable LV dysfunction</li> </ul>	<ul style="list-style-type: none"> <li>degeneration, spontaneous rupture</li> <li>Dilated CM</li> <li>Hypertrophic CM</li> </ul>	<ul style="list-style-type: none"> <li>Acute MR is very poorly tolerated than chronic due to insufficient time for atrial and ventricular adaptation</li> </ul>	<ul style="list-style-type: none"> <li><b>Holosystolic murmur at apex</b></li> <li><b>Murmur radiates to back, left axilla</b>, may radiate to base</li> <li>Pulse upstrokes full</li> <li>S2 normal</li> <li>LV heave</li> </ul>	<p><b>Therapy</b></p> <ul style="list-style-type: none"> <li>Preload ↓ for acute HF</li> <li>Afterload ↓ to improve forward flow</li> <li>Inotrope to maintain contractility</li> <li>AA for ectopy &amp; maintain sinus</li> <li>Antibiotic prophylaxis</li> <li>Surgery – annuloplasty, valvuloplasty, valve replacement, coronary revascularization; timed to prevent irreversible LV dysfunction</li> </ul>
<p><b>Mitral Valve Prolapse (MVP)</b></p> <ul style="list-style-type: none"> <li>Generally benign</li> <li>Can progress to MR</li> <li>Men progress more often and more rapidly than women</li> <li>Small increased risk for SBE, embolus, and arrhythmia</li> </ul>	<ul style="list-style-type: none"> <li><b>Myxomatous degeneration of valve</b></li> <li>Genetically mediated</li> <li>Associated w/ other dyscollagenoses (MARfans, osteogenesis imperfecta)</li> <li><b>Females &gt; males 4:1</b></li> </ul>	<ul style="list-style-type: none"> <li>Leaflets redundant &amp; bulge into atrium during systole</li> <li>Occasionally regurgitant</li> <li>Can continue to degenerate &amp; cause severe MR</li> <li>High circulating catecholamines</li> </ul>	<ul style="list-style-type: none"> <li>Chest pain</li> <li>Palpitations</li> <li>Exertional dyspnea</li> <li>Occasional syncope</li> <li>Usually in 2<sup>nd</sup>/3<sup>rd</sup> decade</li> <li><b>Early to mid systolic click at apex</b></li> <li>Mid to late systolic murmur if MR</li> <li>Click/murmur prolong &amp; ↑ w/ maneuvers which decrease LV filling volume (standing, valsalva, nitrates)</li> </ul>	<ul style="list-style-type: none"> <li>Echo for prolapse</li> <li>Doppler for MR</li> <li>Holter for arrhythmia</li> </ul>
<p><b>Tricuspid Regurgitation</b></p>	<ul style="list-style-type: none"> <li></li> <li></li> <li>Pulmonary HTN</li> <li>Endocarditis</li> <li>Acute pulmonary embolism</li> <li>RV infarction</li> </ul>		<ul style="list-style-type: none"> <li></li> <li></li> <li>Exertional fatigue</li> <li>Anorexia/bloating</li> <li>Peripheral edema</li> <li>Sleep disturbance</li> <li>JVD</li> <li>RV heave</li> <li><b>Epigastric systolic murmur</b></li> <li>Hepatomegaly</li> <li>Liver function test abnormalities</li> </ul>	<p><b>Tx</b></p> <ul style="list-style-type: none"> <li>Diuresis</li> <li>↓ Preload &amp; valve replacement</li> </ul>

<b>Pulmonic Stenosis</b>	<ul style="list-style-type: none"> <li>• Congenital</li> <li>• Endocarditis</li> </ul>	<ul style="list-style-type: none"> <li>• Increase flow gradient</li> <li>• RVH</li> <li>• RV HTN</li> <li>• RV pressure overload</li> <li>• Reduced LV filling</li> <li>• Peripheral venous congestion</li> </ul>	<ul style="list-style-type: none"> <li>• Exertional fatigue</li> <li>• <b>Dyspnea</b></li> <li>• <b>Signs of RV HTN</b></li> <li>• <b>Systolic murmur 2<sup>nd</sup> left interspace</b></li> <li>• Peripheral edema</li> <li>• JVD</li> <li>• RV heave</li> </ul>	<b>Tx</b> <ul style="list-style-type: none"> <li>• Surgery</li> <li>• Judicious preload ↓</li> </ul>
<b>Pulmonary insufficiency</b>	<ul style="list-style-type: none"> <li>• Congenital</li> <li>• Endocarditis</li> <li>• Pulmonary HTN</li> </ul>		<ul style="list-style-type: none"> <li>• Presentation similar to stenosis</li> <li>• <b>Decrescendo diastolic murmur 2<sup>nd</sup> left interspace</b></li> <li>• Signs of RV volume/pressure overload</li> </ul>	<b>Tx</b> <ul style="list-style-type: none"> <li>• preload ↓</li> <li>• Tx of pulmonary HTN</li> </ul>

### Angina

<b>Disease</b>	<b>History</b>	<b>Physical Examination</b>	<b>Evaluation</b>	<b>Therapy</b>
<b>Angina</b> <ul style="list-style-type: none"> <li>- <b>Stable</b> (upon exertion)</li> <li>- <b>Unstable</b> (at rest)</li> <li>- <b>Crescendo</b> (progressive severe symptoms over a short period of time)</li> </ul> <p><u>Ischemic cascade:</u> Decreased oxygen availability</p> <p>Decreased ATP</p> <p>Decreased actin-myosin unbridging; diastolic stiffness</p> <p>Increased end-diastolic filling pressure</p>	<p>“Levine sign” (correlates in males)</p> <p>Pressure or squeezing sensation more correlative w/ pain (substernal)</p> <p>Radiation pattern to left arm, shoulder, neck, or hand</p> <p>Associated w/ diaphoresis and nausea are ominous markers</p> <p>Significant ischemia often results in prolonged periods of fatigue</p>	<p>Evidence of vascular disease (fundoscopic – copper wiring or plaque; large artery bruits; pulse abnormalities)</p> <p>Systolic aortic murmur</p> <p>Cardiomegaly (displaced PMI; anterior heave)</p> <p>Xantholasma</p> <p>Xanthomas</p> <p>Arcus senilis</p> <p>Arrhythmia</p> <p>Enlarged abdominal aorta</p>	<p><u>ECG:</u> ST segment depression; arrhythmia; Q waves indicative of prior injury; normal ECG doesn’t rule out ischemia</p> <p><u>Labs:</u> lipid, troponin or CPK (ER evaluation), glucose, CPK (diagnostic of women)</p> <p><u>Echo:</u> confirm segmental dysfxn in presence of abnormal ECG (LV dysfxn)</p> <p><u>Stress test:</u> screen in males w/ normal ECG (if abnormal ECG, don’t do)</p> <p><u>Stress nuclear imaging:</u> in presence of abnormal ECG; info on ischemia and overall LV fxn</p>	<p><u>Management:</u></p> <ul style="list-style-type: none"> <li>- <u>All patients w/ any risk factor:</u> aspirin and fasting lipid screen</li> <li>- <u>All patients w/ chest pain:</u> ECG (if abnormal, admit to ED)</li> <li>- <u>Patients w/ est. coronary disease:</u> adjust meds and follow if not progressing; if <u>progressing:</u> angiography and adjust meds; if <u>atypical:</u> stress test (if ECG normal) or nuc/echo stress test (if female of ECG abnormal)</li> <li>- <u>w/o est. coronary disease and typical pain in high risk patient:</u> initiate meds and refer to angiography or stress test; if <u>atypical:</u> stress test; if <u>typical and</u></li> </ul>

<p>Decreased contractility</p> <p>ECG changes</p> <p>Pain</p> <p>Risks:</p> <ul style="list-style-type: none"> <li>- DM</li> <li>- Family Hx of premature atherosclerosis in 1<sup>st</sup> degree relative</li> <li>- Lipid disorder (high LDL or low HDL)</li> <li>- Smoking</li> <li>- Hypertension</li> </ul>	<p>Ischemic pain <u>rarely lasts longer than 30 minutes</u></p> <p>Ischemia presents w/ painless dyspnea (25%) instead of apin</p> <p>Pain w/ activity (stable) or pain w/ activity and rest (unstable) correlates w/ ischemia</p> <p>Claudication separate from chest discomfort</p> <p>Fatigue</p>		<p><u>Stress echo</u>: info on ischemic wall motion abnormalities as well as LV fxn and valve disorders</p> <p><u>Fast CT</u>: eval of calcification of coronary arteries, but not direct relation to degree of obstruction or ischemia</p> <p><u>MRI</u>: info on plaque burden and degree of obstruction (limited to proximal 1/3-1/2 of coronary arteries)</p> <p><u>Coronary Angiography</u>: “gold standard” for diagnosis and risk stratification of coronary disease</p>	<p><u>lower risk</u>: med trial</p> <ul style="list-style-type: none"> <li>- Prominent aortic systolic murmur: echo (screen for AS)</li> </ul> <p><u>Aggressive</u>: hospital CCU; emergent coronary visualization w/ angioplasty; coronary bypass</p>
<b>Disease</b>	<b>History</b>	<b>Physical Examination</b>	<b>Evaluation</b>	<b>Therapy</b>
<p><b>Myocardial Infarction</b></p> <p><b>Q wave infarct</b>: transmural; more myocardial necrosis; acute mortality higher</p> <p><b>Non Q wave infarct</b>: ST segment depression w/ elevated enzymes; less total damage; lower acute mortality</p> <p>Risks:</p> <ul style="list-style-type: none"> <li>- Hypercholesterolemia</li> <li>- Hypertriglyceridemia</li> <li>- Low HDL</li> <li>- Hypertension</li> <li>- Smoking</li> <li>- Hyperglycemia/DM</li> <li>- Obesity</li> </ul>	<p>Prodromal chest pain (40-60%)</p> <p><u>Typical</u>:</p> <ul style="list-style-type: none"> <li>- Substernal squeezing or ache (sharp or heavy)</li> <li>- If longer than 30 minutes, infarction (prolonged severe pain or dyspnea)</li> <li>- Associated diaphoresis, dyspnea, and nausea</li> <li>- Radiation to left arm, hand, neck, or jaw</li> </ul>	<p>Pallor, diaphoresis, dyspnea</p> <p>Normo-hypertensive</p> <p>HR variable (mild tachy common)</p> <p>Low grade fever (hours after infarction)</p> <p><u>Jugular venous pulse</u>:</p> <ul style="list-style-type: none"> <li>- if elevated, right ventricular involvement</li> <li>- if hypotension or rales, consider CHF</li> </ul> <p><u>Heart sounds</u>:</p> <ul style="list-style-type: none"> <li>- S4 at apex; S3 (severe damage w/ impending HF)</li> <li>- new murmur (MR due to papillary muscle injury)</li> <li>- Pericardial friction</li> </ul>	<p><b>Complications:</b></p> <p><u>Arrhythmias</u>:</p> <ul style="list-style-type: none"> <li>- Ventricular premature beat (benign)</li> <li>- VT (lethal: DC cardioversion or AA and cardioversion)</li> <li>- Accelerated idioventricular rhythm (sign of reperfusion and thrombolysis; treat w/ AA; well tolerated)</li> <li>- AFib/Flutter: LV dysfxn</li> </ul> <p><u>Conduction disturbances</u>:</p> <ul style="list-style-type: none"> <li>- <u>1<sup>st</sup> degree block</u>: inferior MI; asymptomatic</li> <li>- <u>2<sup>nd</sup> degree AV block Type 1 (Wenkebach)</u>:</li> </ul>	<p>Intravenous thrombolysis:</p> <ul style="list-style-type: none"> <li>- requires ST elevation in 2 leads</li> <li>- less than 2 hr or less than 6 hr from onset of pain is best</li> <li>- <i>C/</i>: active bleeding, recent CVA, surgery, bleed disorder, trauma</li> </ul> <p>Adjunctive therapy:</p> <ul style="list-style-type: none"> <li>- heparinization</li> <li>- IV nitro</li> <li>- Aspirin</li> <li>- IV B-blocker followed by oral</li> <li>- Oxygen by nasal cannula</li> <li>- ACEI after initial event</li> </ul> <p>Interventional therapy (acute angioplasty):</p>

<ul style="list-style-type: none"> <li>- Sedentary lifestyle</li> <li>- Type A personality</li> <li>- Genetic</li> </ul>	<p><u>Atypical:</u></p> <ul style="list-style-type: none"> <li>- Pain radiating to the right side</li> <li>- Pain in fingers rather than arm</li> <li>- Pain lasting less than 5 min or longer than several hours</li> </ul>	<p>rub (late course 20%)</p> <p><u>Labs:</u></p> <ul style="list-style-type: none"> <li>- <i>Troponin T and I</i> are earliest markers (1-2hr)</li> <li>- <i>CPK</i>: peaks at 12-36hr (MM rises w/ trauma and IM injections; size of infarct related to total CPK over time)</li> <li>- <i>SGOT (AST)</i>: rises 8-12 hours post-MI; elevated w/ liver disease and skeletal muscle injury</li> <li>- <i>LDH</i>: rises 24-26hrs; maintains for 7-10 days (LDH1 predom.); elevated in liver, brain, bone, and blood disorders</li> </ul> <p><u>ECG:</u></p> <ul style="list-style-type: none"> <li>- ST segment elevation</li> <li>- Q wave after 3-6 hr</li> <li>- T wave inverts</li> <li>- Overtime, ST normal, but Q remains</li> </ul>	<p>advanced AV node disease; inferior MI (benign); pacing required if anterior MI</p> <ul style="list-style-type: none"> <li>- <u>2<sup>nd</sup> degree AV block</u></li> <li>- <u>Type 2</u>: may require pacing</li> <li>- <u>Complete Heart Block</u>: requires pacing; inferior MI (transient)</li> </ul> <p><u>LV Failure</u>: correlates w/ degree of damage; asso. w/ pulmonary congestion and low CO; high risk for cardiac mortality</p> <p><u>MR</u>: papillary muscle dysfxn (asympt. or severe)</p> <p><u>LV aneurysm</u>: ant. MI; may cause refractory HF, arrhythmia, or embolus</p> <p><u>RV infarction</u>: inf. wall MI; suspect when BP remains low in spite of therapy; treat w/ high volume infusion</p> <p><u>Thromboembolism</u>: ant. or large MI</p> <p><u>Cardiac rupture</u>: elderly, females, first MI; sudden loss of pulse and electromechanical dissociation; late occurrence</p>	<ul style="list-style-type: none"> <li>- better than thrombolysis if less than 6 hours from onset of pain</li> <li>- Restenosis rate higher in acute MI than elective pt.</li> </ul> <p>Misc. Therapy:</p> <ul style="list-style-type: none"> <li>- AA (acute arrhythmias)</li> <li>- Nonsteroidals (acute pericarditis)</li> <li>- Morphine (pain relief)</li> <li>- Laxatives and sedation while in intensive care</li> </ul>
--	--	---	---	---

### Angina Treatment

#### Medications:

- *Aspirin* for all cases
- *Nitrates* (first line, at least 8hr free interval per day)
- *B-blockers* (in hypertension, post MI; lung disease and diabetes w/ caution); withhold in patients w/ reduced LV fxn
- *Ca-blockers (Dihydropyridines)* not for post MI; *Verapamil/diltazem* not for LV EF <40%
- Lipid lowering agents
- No longer: antioxidants or estrogen replacement

Conservative:

- High risk: IV heparin, nitro, 2b3a inhibitor; aspirin, B-blocker; NO thrombolysis
- Lower risk: oral therapy w/o heparin; 24 hour inpatient observation w/o ICU; oral nitrates, B-blocker, aspirin; heparin NOT needed and NO thrombolysis

### Heart Failure

Heart Failure	Causes	Symptoms	Therapy
<p>Heart unable to pump adequate output to meet the demands of the tissues</p> <p>Or it does so from abnormally elevated ventricular diastolic volume</p> <p>Associated w/ inflammation and imbalance of hormones released by the body's defense system</p> <p>Overcompensate for poor heart function</p> <p>Progressive – deterioration of cardiac structure, function</p>	<p>Neurohormonal derangements:</p> <ul style="list-style-type: none"> <li>- Norepi</li> <li>- Renin-Angiotension-Aldosterone system</li> <li>- Endothelin</li> <li>- TNF</li> <li>- Natriuretic peptides</li> </ul> <p>Increased Preload</p> <ul style="list-style-type: none"> <li>- Increased circulating hormone</li> <li>- Mitral insufficiency</li> <li>- Aortic insufficiency</li> <li>- Vasoconstrictors</li> <li>- Atrial kick</li> </ul> <p>Increased Afterload</p> <ul style="list-style-type: none"> <li>- Aortic stenosis</li> <li>- Peripheral arteriolar vasoconstriction</li> <li>- Hypertension</li> <li>- Polycythemia</li> <li>- Arteriolar vasoconstrictor drugs</li> </ul> <p>Decreased Contractility</p> <ul style="list-style-type: none"> <li>- Hypoxia (oxygen saturation &lt;50%)</li> <li>- Hypercapnia</li> <li>- Cardiac muscle disease</li> <li>- MI</li> <li>- Decreased HR</li> <li>- Metabolic acidosis</li> </ul>	<p>Dyspnea</p> <p>Fatigue</p> <p>Fluid Retention</p>	<p>Immediate stabilization of acute patients</p> <ul style="list-style-type: none"> <li>- Most have volume overload requiring diuretics</li> <li>- Oxygen</li> <li>- Control dysrhythmias</li> <li>- Reduce preload, afterload</li> <li>- Remove causative factors</li> </ul> <p>Hallmarks of Therapy</p> <ul style="list-style-type: none"> <li>- ACE-inhibitors</li> <li>- B-blockers</li> <li>- Cholesterol-lowering agents</li> <li>- Diuretics (fluid retention; not mono: dioxin – to improve clinical status, use w/ A &amp; B)</li> </ul>

	Physiologic Compensation <ul style="list-style-type: none"> <li>- Increased peripheral vascular disease</li> <li>- Tachycardia</li> <li>- Ventricular remodeling</li> <li>- Renal sodium/water retention</li> </ul>		
--	---	--	--

Type of Heart Failure	Causes	Symptoms
<b>Left Ventricular Systolic Dysfunction</b>  Impairment of left ventricular contraction  Insufficient blood ejected to meet tissue needs	CAD Hypertension Dilated Cardiomyopathy Aortic insufficiency Dysrhythmias Electrolyte disorders Thyroid disease Drug abuse	Weakness, fatigue Changes in exercise tolerance Changes in sensorium (lightheaded, confusion) Dyspnea, DOE, PND Tachypnea Diaphoresis Sacral edema Crackles, ronchi Nocturia Cyanosis (late)
<b>Left Ventricular Diastolic Dysfunction</b>  Impairment of ventricular relaxation Impaired filling	CAD Hypertension Valvular Disease Cardiomyopathy; hypertrophic, infiltrative, restrictive LVH Diabetes, Age	Due to increased filling pressures: <ul style="list-style-type: none"> <li>- Dyspnea, orthopnea</li> <li>- Tachypnea</li> <li>- Pulmonary edema</li> <li>- Exercise tolerance</li> <li>- Signs of R-sided HF</li> </ul>
<b>Right-Sided HF</b>		Peripheral edema Hepatomegaly Ascites JVD Fatigue, weakness Anorexia, N/V
<b>Left-Sided HF</b>		Dyspnea, orthopnea PND Fatigue, weakness
<b>Acute HF</b>		Sudden appearance of dyspnea Acute cardiogenic pulmonary edema Cardiogenic shock Acute decompensation from CHF
<b>Chronic HF</b>		BP stable May have peripheral edema

Type of Heart Failure	Causes	Symptoms
<b>High Output HF</b> Exercise demand Impaired filling	Thyrotoxicosis Sepsis Anemia Cirrhosis AV malformation	
<b>Low Output HF</b> Poor systolic function Low ejection fraction	Dilated cardiomyopathy Myocardial ischemia Valvular heart disease Toxins (EtOH) Dysrhythmias	

<b>Acute Decompensated Chronic HF</b>  Further activation of compensatory mechanisms that:  Enhance sodium and water retention  Increase preload, increase afterload	Most/all current therapies activate detrimental neurohormones  Short term exposure to $\beta$ -agonists and PDEI can greatly increase apoptosis	
--	---	--

### Cardiomyopathies

Cardiomyopathy	Etiology	Clinical Presentation	Evaluation	Therapy
<b>Dilated</b>  Reduced ventricular EF  High EDV/pressure  Reduced CO  High pulmonary capillary wedge pressure  <b>Natural History:</b>	End stage hypertropic disease  Multi-infarct or ischemic disease  Toxins  Infections (myocarditis)  Endocrinopathy (DM,	Cardiomegaly  LV/RV heave  S3  Occasional mitral or tricuspid regurg  JVD  Pulmonary	<u>Echo or nuclear:</u> measure EF  <u>Echo:</u> measure LV wall motion and chamber size  <u>ECG:</u> signs of ischemia or arrhythmia  <u>NYHA1-4:</u> functional capacity	Salt and water retention  Preload and afterload reduction  Inotrope  AA  Transplant or assist device

<ul style="list-style-type: none"> <li>- w/o ischemia: 3 yr. mortality – 30%</li> <li>- w/ ischemia: prognosis twice as poor</li> <li>- 60% w/ myocarditis resolve completely</li> </ul>	thyroid) CT disease Muscular dystrophy Post partum	rales/peripheral edema Occasional arrhythmia Fatigue, exertional dyspnea, orthopnea	<u>Labs</u> : electrolytes, thyroid, glucose <u>Stress or cath</u> : ischemia Detailed history Myocardial biopsy	
<b>Restrictive</b> Normal systolic contractility Reduction in diastolic relaxation and filling capacity High resting LV and diastolic filling pressure High pulmonary wedge pressure, especially w/ exercise	Infiltrative disease (sarcoid, amyloid, hemochromatosis) TB DM Fibroelastosis	Similar to dilated cardiomyopathy S4 more common than S3 Resting tachy No LV dilation Valvular dysfunction less common	<u>Diagnosis of exclusion</u> : CHF in presence of normal systolic function <u>Echo</u> : LV size and function <u>NYHA1-4</u> : functional capacity <u>Labs</u> : glucose, ferritin PPD Myocardial biopsy	Judicious preload reduction Direct therapy to underlying disease B-blocker/Ca-blocker (aid in relaxation)

<b>Cardiomyopathy</b>	<b>Etiology</b>	<b>Clinical Presentation</b>	<b>Evaluation</b>	<b>Therapy</b>
<b>Hypertrophic</b> Myositis in disarray May hypertrophy asymmetrically, obstructing outflow Outlet obstruction obstruction pulls anterior mitral leaflet out of position, causing regurg LV filling volumes reduced due to diastolic stiffness	Genetic: Autosomal dominant	Sudden death Palpitations/arrhythmias Chest pain Dizziness/syncope Bifid carotid upstroke Basilar systolic murmur Resting tachy frequent	<u>2D echo</u> : essential Physical exam characteristic Family history revealing <u>ECG</u> : ST changes	Avoid: vigorous physical activity; preload/afterload reducing agents Myomectomy Septal ablation w/ catheters B-blocker/Ca-blocker or disopyramide (aid in slowing HR and relaxation)

High pulmonary capillary wedge pressure				Electronic pacing
Pulmonary hypertension				
Normal LV systolic contractility				

### AV Block

AV Block	Pathology & Causes	Diagnosis	Presentation	Treatment
1 <sup>st</sup> degree	<ul style="list-style-type: none"> <li>• Delay in <b>upper</b> portion of node (cap)</li> <li>• Ischemia</li> <li>• Toxins</li> <li>• Drugs</li> <li>• Conduction disease</li> </ul>	<ul style="list-style-type: none"> <li>• <b>Prolonged PR interval</b> (&gt; 200 ms)</li> <li>• Normal QRS</li> <li>• <b>No effect on HR</b></li> </ul>	<ul style="list-style-type: none"> <li>• Asymptomatic</li> <li>• Often associated with other disease</li> </ul>	<ul style="list-style-type: none"> <li>• Remove offending agents</li> <li>• Treat underlying condition</li> <li>• No specific therapy for block needed</li> </ul>
2 <sup>nd</sup> degree 1. Type I (Wenkebach) 2. Type II (Mobitz II)	<ol style="list-style-type: none"> <li>1. Delay in <b>middle</b> portion of node</li> <li>2. Delay in <b>lower</b> portion of node</li> </ol> <ul style="list-style-type: none"> <li>• Ischemia</li> <li>• Toxins</li> <li>• Drugs</li> <li>• Cardiomyopathy</li> </ul>	<ol style="list-style-type: none"> <li>1. Regular supraventricular rhythm; <b>Progressive prolongation of PR interval until a beat is dropped</b>; Cycle is repetitive, and varies in length; QRS usually narrow (normal); <b>DOES AFFECT HR</b></li> <li>2. Regular supraventricular rhythm; <b>Constant PR interval (fixed ratio of blocked/conducted beats across node)</b>; QRS often widened</li> </ol>	<ul style="list-style-type: none"> <li>• Often asymptomatic</li> <li>• When associated with drugs or toxins, fatigue, or dyspnea common</li> <li>• Often seen in acute MI</li> <li>• May cause syncope or near syncope</li> </ul>	<ul style="list-style-type: none"> <li>• Remove toxins</li> <li>• Reduce or stop drug causing problem</li> <li>• Atropine (blocks vagus → AV node, brings HR/BP up) if acutely symptomatic</li> <li>• Temporary pacing if transient problem</li> <li>• Permanent pacing if MI and symptomatic</li> </ul>
3 <sup>rd</sup> degree	<ul style="list-style-type: none"> <li>• Similar to other blocks, but more</li> </ul>	<ul style="list-style-type: none"> <li>• Regular supraventricular rhythm</li> </ul>	<ul style="list-style-type: none"> <li>• Syncope</li> <li>• CHF</li> </ul>	<ul style="list-style-type: none"> <li>• Immediate intervention usually needed</li> </ul>

	<p>serious</p> <ul style="list-style-type: none"> <li>• <b>Most common with infarction</b></li> <li>• Profound drug toxicity</li> </ul>	<p>with <b>no relation of P waves to QRS</b></p> <ul style="list-style-type: none"> <li>• Ventricular rhythm slower than supraventricular rhythm, called "escape" rhythm</li> <li>• QRS wide if escape rhythm ventricular, and narrow if junctional</li> </ul>	<ul style="list-style-type: none"> <li>• MI</li> <li>• Chest pain</li> <li>• Mental status change (due to poor perfusion)</li> <li>• Weakness/fatigue</li> </ul>	<ul style="list-style-type: none"> <li>• Atropine</li> <li>• Epinephrine</li> <li>• Pacing, temporary or permanent</li> <li>• Remove toxin or drug</li> <li>• Treat acute ischemia</li> </ul>
--	---	--	--	---

### SYNCOPE

sudden spontaneous & brief loss of consciousness → does not require resuscitation (unknown is m/c then vasovagal)

Types	Etiologies	Sx's	Eval	Tx	
Vasovagal/situational	<ul style="list-style-type: none"> <li>◇ Stimulant/psychogenic <ul style="list-style-type: none"> <li>• Associated w/ hyperadrenergic response to stimulus → e.g. pain, fear, physical stress, other intense emotions</li> </ul> </li> <li>◇ Situational (micturition/defecation) <ul style="list-style-type: none"> <li>• Associated w/ hyperadrenergic response to stretch receptor stimulation in bladder and rectum</li> </ul> </li> <li>◇ Vasomotor instability → common in elderly <ul style="list-style-type: none"> <li>• Often associated w/ chronic illness (esp DM)</li> <li>• Often associated w/ other signs of vasomotor dysfxn (e.g. flushing or diaphoresis w/o syncope)</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>◇ Often preceded by a stimulus</li> <li>◇ Brief prodrome of nausea or lightheadedness</li> <li>◇ Usually occurs in standing posture</li> <li>◇ Three phases: <ol style="list-style-type: none"> <li>1. initial rise in HR and BP</li> <li>2. subsequent drop in BP</li> <li>3. rapid recovery in supine posture</li> </ol> </li> </ul>	<ul style="list-style-type: none"> <li>◇ 85% diagnosed w/ H&amp;P</li> <li>◇ No structural or functional abnormalities</li> <li>◇ Specialized testing rarely indicated</li> <li>◇ Tilt table positive in 10-15%</li> </ul>	<ul style="list-style-type: none"> <li>◇ Benign condition w/ excellent prognosis even w/o therapy</li> <li>◇ In high recurrence cases → <input type="checkbox"/>-blocker or anti-anxiety meds</li> <li>◇ Pt education on avoidance &amp; prevention</li> </ul>	
Orthostatic	<ul style="list-style-type: none"> <li>◇ Due to underfilling of the ventricles either by poor venous return or peripheral pooling upon posture change</li> <li>◇ Dehydration</li> <li>◇ Drug effect → vasodilator such as antihypertensive</li> <li>◇ Dysautonomia <ul style="list-style-type: none"> <li>• Inability to increase sympathetic tone in peripheral vessels upon posture change due to primary dysfxn in receptor or discharge</li> <li>• Usually assoc w/ other signs of sympathetic dysfxn → e.g. bowel/bladder/sexual dysfxn</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>◇ Sudden drop in systolic BP upon rising to semi erect or erect posture</li> <li>◇ No prodrome</li> <li>◇ Usually occurs immediately after position change → can occur much later in some patients</li> <li>◇ Usually accompanied by tachycardia</li> </ul>	<ul style="list-style-type: none"> <li>◇ Careful H&amp;P → screen for causes of fluid loss &amp; meds</li> <li>◇ Valsalva maneuver</li> <li>◇ Screening tilt test</li> <li>◇ Tilt table test</li> <li>◇ Serum NE levels w/ position change</li> </ul>	<ul style="list-style-type: none"> <li>◇ Remove drugs responsible</li> <li>◇ Volume expanders (Fluorocortisone)</li> <li>◇ Compression stockings</li> <li>◇ <input type="checkbox"/>-agonist (Midodrine) may help in selected cases</li> </ul>	
C	A	<ul style="list-style-type: none"> <li>◇ Tachy/brady syndrome</li> </ul>	<ul style="list-style-type: none"> <li>◇ Preceded by palpitations</li> </ul>	<ul style="list-style-type: none"> <li>◇ ECG</li> </ul>	<ul style="list-style-type: none"> <li>◇ Pacemaker for pauses</li> </ul>

		<ul style="list-style-type: none"> <li>◇ Sudden onset of a-fib or flutter</li> <li>◇ Supraventricular tachycardia</li> <li>◇ Vent tachycardia</li> <li>◇ Sinus pauses (&gt;3 secs)</li> <li>◇ Advanced AV block</li> </ul>	<ul style="list-style-type: none"> <li>◇ More common in pts w/ ischemic or myopathic cardiac conditions</li> <li>◇ May demonstrate profound bradycardia (sick sinus syndrome)</li> <li>◇ Often assoc w/ frequent ectopy</li> </ul>	<ul style="list-style-type: none"> <li>◇ Holter monitor</li> <li>◇ Stress test</li> <li>◇ Event monitor</li> <li>◇ Electrophysiologic study</li> </ul>	<ul style="list-style-type: none"> <li>or block</li> <li>◇ Antiarrhythmic for documented arrhythmias</li> <li>◇ Implantable defibrillator/cardioverter for life threatening cases</li> </ul>
	Valvular	<ul style="list-style-type: none"> <li>◇ Aortic/pulm stenosis</li> <li>◇ Sudden onset severe mitral regurg (as in acute infarction)</li> </ul>		<ul style="list-style-type: none"> <li>◇ ECG</li> <li>◇ Echocardiography</li> <li>◇ Cardiac catheterization</li> </ul>	<ul style="list-style-type: none"> <li>◇ According to valvular lesion</li> <li>◇ Most require surgery</li> </ul>
	Ischemic		<ul style="list-style-type: none"> <li>◇ Chest pain or dyspnea preceding</li> <li>◇ M/C in pts at high risk for coronary disease</li> </ul>	<ul style="list-style-type: none"> <li>◇ ECG</li> <li>◇ Serum cardiac enzymes</li> <li>◇ Monitoring</li> <li>◇ Cardiac catheterization</li> </ul>	<ul style="list-style-type: none"> <li>◇ Address the acute ischemic episode w/ appropriate intervention</li> <li>◇ Consider anticoag for possible thrombus</li> </ul>
Carotid hypersensitivity		<ul style="list-style-type: none"> <li>◇ Abnormally sensitive carotid body reflexes</li> </ul>	<ul style="list-style-type: none"> <li>◇ Syncope while shaving</li> <li>◇ When wearing tight shirt collars</li> <li>◇ When turning head sharply</li> <li>◇ More common in middle aged or older men</li> </ul>	<ul style="list-style-type: none"> <li>◇ Careful carotid massage in supine position</li> </ul>	<ul style="list-style-type: none"> <li>◇ Nonspecific</li> <li>◇ prevention</li> </ul>
CNS dysfxn		<ul style="list-style-type: none"> <li>◇ Cerebrovascular insufficiency → often involves obstruction of the vertebrobasilar arteries</li> </ul>	<ul style="list-style-type: none"> <li>◇ May be associated w/ recurrent small strokes</li> </ul>	<ul style="list-style-type: none"> <li>◇ Careful H&amp;P</li> <li>◇ EEG</li> <li>◇ CT or MRI of train if stroke suspected</li> <li>◇ Cerebral angiogram for suspected vascular event</li> </ul>	<ul style="list-style-type: none"> <li>◇ As appropriate for etiology</li> <li>◇ Anticoag after vaso-occlusive event</li> <li>◇ Anti-anxiety meds for hysteria &amp; hyperventilation</li> <li>◇ Anticonvulsants for seizure disorders</li> </ul>
		<ul style="list-style-type: none"> <li>◇ Acute cerebral infarction or hemorrhage</li> </ul>	<ul style="list-style-type: none"> <li>◇ Sudden onset of neurologic dysfxn along w/ syncope</li> </ul>		
		<ul style="list-style-type: none"> <li>◇ Seizures</li> </ul>	<ul style="list-style-type: none"> <li>◇ Often preceded by a prodrome</li> <li>◇ Usually followed by a period of confusion or lethary</li> <li>◇ May present w/ simple LOC</li> <li>◇ May present w/ syncope and tonic/clonic spasms</li> </ul>		
		<ul style="list-style-type: none"> <li>◇ Psychiatric syncope → hysteria</li> </ul>	<ul style="list-style-type: none"> <li>◇ No objective signs of <input type="checkbox"/> BP/perfusion</li> </ul>		

	◇Psychiatric syncope → hyperventilation	◇Associated w/ anxiety, air hunger, & paresthesias w/o signs of decreased perfusion or BP		
--	---	---	--	--

### Shock

Shock	Etiology	Signs/symptoms	Management
<b>Hypovolemic</b>  1. <u>Minor</u> : <10% blood loss 2. <u>Mild</u> : 20% 3. <u>Mod</u> : 30% 4. <u>Severe</u> : 50%	Hemorrhagic (trauma, iatrogenic)  Non-hemorrhagic (bowel obstruction, GI losses, biliary drainage, pancreatic fistula)	1. equal to giving blood  2. tachy, tilt positive, thirst, cool extremities  3. hypotensive, tachy, confusion, vasoconstriction, decrease urine output  4. profound hypotension, loss of femoral/carotid pulse, no urine output	ABC  IV fluids (crystalloids – lactated ringers; colloids – blood)  Control hemorrhage
<b>Septic</b>  1. <u>Warm</u> (early) 2. <u>Cold</u> (late)	Gram – or + or fungal organisms  Sources of Gram -: - urinary tract - pulmonary - GI - Burns - Soft tissue infections - Indwelling catheters	1. Fever, diaphoretic, flushed, tachy, tachypneic  2. Cold, clammy, lethargic, unresponsive, oliguric, hypotensive; progress to MSOF	Control primary process (antibiotics)  Drainage  IV fluids  Respiratory support  Vasodilators/vasoconstrictors
<b>Cardiogenic</b>	Ischemic heart disease (acute MI, VSD rupture, cardiac aneurysm, papillary muscle rupture)  Valvular heart disease (acute mitral/AI, severe AS)  Arrhythmias (SV, V)  Trauma (tension pneumothorax, pericardial tamponade – Beck’s triad, cardiac contusion)		Recognize etiology  Judicious fluid administration  Swan Ganz catheter  <i>Medical</i> : increase contractility, alter pre/afterload, control arrhythmias  <i>Mechanical support</i> (release pneumothorax, pericardiocentesis, intra-aortic balloon pump, ventilation)
<b>Neurogenic</b>	CNS dysfunction	Hypotension	<u>Supportive</u> : IV fluids; maintain temp.

	Trauma (most common)	Normal/slow pulse	Vasoconstrictors (neosynephrine)
	Iatrogenic (regional anesthetics)	Warm extremities	Surgical intervention
		Little or no agitation	
		Loss of motor function	

### Arrhythmias

Arrhythmia	Etiology	Physical Findings	Evaluation	Treatment
<b>Atrial Fibrillation</b>  Irreg. irregular rhythm w/ no discernable P waves	Ischemia  Acute inflammation (pericarditis, pneumonia/pleuritis)  Chronic inflammation (connective tissue disorder, chronic pericardial effusion)  Valvular disease  Chronic/acute hypertension  Cardiotoxins (chemotherapeutics, alcohol, stimulants)  Thyroid dysfunction  Cardiomyopathy  "Lone" (no discernable etiology - younger ages)	Irregular rhythm  Vary intensity of 1 <sup>st</sup> heart sound  Pulse deficit compared to ausculted rate	<u>Echo</u> : valve disorder, cardiomyopathy, pericardial effusion  <u>Stress test</u> : ischemia  <u>Labs</u> : thyroid, glucose, inflammation  <u>Cardiac cath</u> : if ischemia suspected  <u>If fibrillation not present at time of examination</u> : - 24 hr Holter monitoring - 30 day event recorder - continuous loop recorder - implanted continuous loop recorder	<u>Rate Control</u> (reduce AV node conduction to slow ventricular response – if underlying etiology can't be reversed): - Digoxin, B-blocker, Verapamil, Diltiazem  <u>Conversion to Sinus</u> (if hemodynamically unstable – less significant w/ enlarged LV, LA, RA, or valve disease): - <i>Electrical</i> : Bipolar countershock (effective at lower energy; sedation; reversion freq. within 24hr) - <i>Maintenance of Sinus</i> : no AA for low risk; higher risk require AA - <i>Medical options</i> : Digoxin (low risk); Verapamil/diltiazem (low-mod risk); Amiodarone (best for all); Sotalol (NOT for HF)  <u>Prevention of Thrombosis</u> (most important – espec. cerebral): - High risk (rheumatic/valvular disease; dilated LA or LV w/ cardiomyopathy; prior embolism) - <i>Warfarin</i> (INR of 2) for 3-4 weeks - <i>Heparin</i> (acute embolic event) - Not indicated for bleeding disorder, hemorrhage, compliance problems, CNS tumor (use <i>aspirin</i> instead)

				<u>Invasive Therapy:</u> <ul style="list-style-type: none"> <li>- <i>Maze procedure</i> (incise atrium and create electrical channel from sinus to AV node); may require anticoagulation</li> <li>- <i>Catheter AV node ablation w/ pacemaker insertion</i> (disallows AV synchrony); requires anticoagulation</li> </ul>
<b>Premature Atrial Contractions (PAC)</b>  Early occurring QRS identical to sinus preceded by P wave	Normal variant  Stimulants (Caffeine, EtOH, Cocaine, Catecholamines)  Atrial enlargement  Hypertension  Valvular disease (MR, TR, AS)  Ischemia  Irritants (pneumonia, surgery, mediastinitis)		Asymptomatic/minimally symptomatic – no investigation	No therapy unless symptomatic  B-blocker (rarely AA)
<b>Paroxysmal Atrial Tachy (PAT)</b>  Reg rate 150-250  P waves before QRS  Narrow QRS  Sudden onset and cessation	Hyperthyroidism  Toxins (EtOH; sympathetic amines)  Hypertension  Rarely ischemia  Digoxin toxicity		ECG  TSH, Digoxin levels  Echo  Stress Test	Immediate intervention to ablate: <ul style="list-style-type: none"> <li>- DC cardioversion (if hypotensive or CHF)</li> <li>- Adenosine</li> <li>- Diltiazem or Verapamil</li> <li>- Metoprolol or Atenolol</li> </ul> Long term: <ul style="list-style-type: none"> <li>- Metoprolol or Atenolol</li> <li>- Verapamil or Diltiazem</li> <li>- AA if above unsuccessful</li> </ul>
<b>Atrial Flutter</b>  Reg atrial waves 250-350	Irritants  Toxins		Drug screen  Echo	Acute intervention for tachy disease: <ul style="list-style-type: none"> <li>- B-blocker, Ca Blocker, digoxin</li> <li>- Vagal maneuver</li> <li>- Adenosine (occasionally)</li> </ul>

QRS generally regular  Atrial waves often biphasic	Stimulants  Atrial Enlargement  Valvular Disease		Stress Test	- DC cardioversion if extremis  Long term: - B-blocker, digoxin, Ca blocker - Consider: AA, Anticoagulation, Ablation/pacemaker
<b>Multifocal Atrial Rhythm</b>  Irreg. w/ P waves before QRS, but at least 3 different morphologies	Decompensated pulmonary disease (most common)  Valvular disease			Anticoagulation (prevent embolus)  Poorly responsive to meds that slow AV conduction  25-35% response to AA
<b>Ventricular Tachy</b>  Wide QRS  AV dissociation  Reg. rate >100  Concordance across precordium	Structural heart disease  Nonischemic Cardiomyopathies  Metabolic disorders  Drug toxicity  Prolong QT syndrome	Hypotension, syncope  Asymptomatic		W/o structural heart disease: - Benign (except: Long QT syndrome) - B-blockers, verapamil, AA  W/ structural heart disease: - Hemodynamically stable - Pharm therapy - Pacing Catheters - Programmed Stimulation - Antitachy Pacing - AICD - Surgery
<b>Torsade de Pointes</b>  VT w/ polymorphic QRS	QT prolongation  Electrolytes, diets  AA  CVA, arrhythmias  Congenital			Remove precipitating factors  Correct metabolic derangements  Remove drugs  Overdrive pacing  B-blockers  AICD
<b>Accelerated Idioventricular</b>  Slow VT  Rate 60-120	Acute MI/reperfusion  Following cardiac operations  Cardiomyopathy			

	Rheumatic fever Digitalis intoxication Idiopathic			
<b>Ventricular Fibrillation</b>  Majority of Sudden Death	Ischemia/Hypoxia AA WPW and A Fib Electrical accidents	After MI: good prognosis  Unrelated to MI: recurrence common		Treat underlying conditions  AA (termination, prevent recurrence, prevent life-threatening arrhythmia; monitor therapeutic levels)  Electrical: <ul style="list-style-type: none"> <li>- <u>Pacemakers</u> (used for termination; reserved for refractory cases; EPS guidance; prevention)</li> <li>- <u>Cardioversion and Defibrillation</u> (reliable, synchronous)</li> <li>- <u>Implanted Cardioverter/Defibrillator</u> (antitachy pacing ability; lower energy; increased survival; indications – depressed LV fxn, prior MI, VT on EPS)</li> <li>- <u>Ablative</u> (procedure of choice – concealed tracts, AV nodal reentrant SVT, atrial flutter, poorly controlled ventricular responses, recurrent episodes; creation of AV block)</li> <li>- <u>Surgical</u> (aneurysmectomy)</li> </ul>
<b>Sinus Tachy</b>  HR>100  Gradual onset/offset	Response to stress			
<b>AV Nodal Reentrant Tachy (AVNRT)</b>  Narrow QRS  Rate 120-250  P waves present		Palpitations  Syncope  Heart failure		Vagal maneuvers  AV nodal blockers  Pacing  DC cardioversion  AA (prevention)

<b>Wolff-Parkinson-White</b>  Short PR  Slurred upstroke of QRS (wide)  Atrial arrhythmias common  AV bypass tracts; antegrade conduction				B-blocker, verapamil (caution)  DC cardioversion  Ablation
---	--	--	--	--