

Heart Disease	Etiology	Mechanism	Pressures	Murmur	Heart Sounds	Clinical Characteristics
<b>Mitral Valve Obstruction (Stenosis)</b>	Rheumatic HD with scarring of valve leaflets, fibrosis, and shortening of chordae tendinae	During diastole valve leaflets open only partially⇒limits blood flow from LA⇒LV	Diastolic P in LA greater than LV	-low pitch, may become louder in end diastole when atrial contraction ↑ flow across valve -DIASTOLIC -heard ONLY at apex	-S1 snap might be heard b/c closure occurs at a higher P -S2 (splitting ↑ and louder P2) -Opening snap: higher the LA P, sooner after A2 the atrial P exceeds ventricular P, and the earlier the mitral leaflets snap open (w/ calcified mitral valve ↓ leaflet mobility and S1 may be ↓ and no opening snap)	-with ↑ HR ↓ diastolic time and ↓ filling⇒↓CO -LA may become dilated and hypertrophied -high LA P reflected into pulmonary bed⇒ chronic passive lung congestion and dyspnea, orthopnea, pnd -↑JVP (due to venous congestion) -with longstanding cases the RV and RA also dilate and hypertrophy
<b>Aortic Valve Obstruction (Stenosis)</b>	-congenital bicuspid or unicuspid valve -calcification of aortic valve -when assoc. with other valve deformities could be rheumatic	During systole delayed and partial opening of aorta⇒limits blood flow and delays aortic closure	-Gradient across aortic valve during systole ⇒ LV P greater than aortic P	-harsh, low, or high pitched -begins after S1, gets louder till peak, then declines and ends before S2 -heard at base to the right of sternum; radiates to neck	-S4 (↓ compliance) -S1 -Ejection Click (at start of systole whole valve may snap open) -S2 may get delayed aortic valve closure – paradoxical split (w/ calcific stenosis valve moves little so no ejection click, and A2↓ or absent)	-pressure overload ⇒LV hypertrophies (Concentric) ⇒ ↓ compliance in diastole -LV failure assoc with dilation (LV compliance problem and/or systolic failure⇒cardiac SOB) -delay in peak of carotid pulse -angina -fainting

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<b>Mitral Valve Regurgitation</b>	Ischemic HD papillary muscle dysfunction, mitral valve prolapse, endocarditis, rheumatic HD, idiopathic hypertrophic subaortic stenosis (hypertrophic cardiomyopathy), marked LV enlargement, mitral annular calcification	-valve leaflets become incompetent during systole, blood flows retrogradely into LA as well as across the aorta (portion of LV stroke volume abnormally ejected into LA⇒volume stress on LV b/c regurgitated V returns to LV in diastole)	<u>Acute MR:</u> -normal or ↓ LA compliance -↑LA P⇒SOB -↑pulmonary venous P ↑ pulmonary venous congestion <u>Chronic MR</u> -↑ LA compliance⇒ LA dilates “normal” LA and PV P, but low CO -compliant LA becomes preferred low P sink for LV ejection vs aorta	-high pitched and blowing -holosystolic begins with S1 and ends after A2 -if clench fists murmur ↑ due to ↑ systemic vascular resistance (more blood will go into lower P LA) -may be heard all over precordium but loudest at apex -radiates to lft axilla and back beneath lft scapula	-diminished S1 -may be a midsystolic click (leaflets prolapse into atrium) -S2 with abnormal split with normal A-P sequence	-added volume load on LV⇒LV dilation and hypertrophy (Eccentric) -MR⇒cardiac SOB dyspnea on exertion <u>Chronic MR</u> -fatigue (low CO) -chronic atrial fibrillation
<b>Aortic Valve Regurgitation</b>	-Rheumatic HD -syphilis -dissecting aortic aneurysm -Marfan’s -ankylosing spondylitis -infective endocarditis	-aortic valve leaflets don’t completely come together during diastole ⇒blood ejected during systole can flow back into LV during diastole	-end diastolic V and SV are larger than normal -no significant P gradient across aortic valve	-Systole: early or midsystolic ejection murmur b/c high volume flow across valve -Diastole: high-pitched decrescendo best heard at Erb’s pt and apex	-S1 -Ejection click -S2 accentuated -S3 often heard during rapid filling in early diastole⇒sign of ventricular failure	-volume overload ⇒Eccentric hypertrophy and dilation of LV -Carotid pulse has forceful early peak and then falls off quickly -widened pulse pressure ex. 180/60 - LV compliance problem and/or systolic failure⇒cardiac SOB

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<b>Congestive Heart Failure</b>  (inability of heart to deliver sufficient blood to peripheral tissues to meet metabolic demands)	Ischemic HD, hypertension, cardiac valvular disease, cor pulmonale, cardiomyopathy, congenital HD	<u>↑ ventricular wall stress</u> 1. Na retention 2. peripheral vasoconstriction 3. ↓ cardiac contractility <u>↑ neurohormonal activation</u> 1. sympathetic NS 2. renin-angiotensin syst.	-orthopnea (need pillows at night to breathe) -dyspnea on exertion -paroxysmal nocturnal dyspnea (gasping for air at night) -ankle swelling
<b>Left Heart Failure</b>	Ischemic HD, HTN, aortic and mitral valvular disease, myocardial disease	-LV hypertrophies and dilates, often with secondary LA enlargement ⇒ damming back of blood in pulmonary circulation ⇒ pulmonary congestion and edema -↓ systemic output	-pulmonary congestion, edema, hemorrhage ⇒ cough, dyspnea, orthopnea, pnd -↓ renal perfusion: ↑ renin-angiotensin-aldosterone (↑ Na and water retention), ischemic tubular necrosis, pre-renal renal failure -↓ cerebral perfusion: hypoxic encephalopathy
<b>Right Heart Failure</b>	Usually secondary to LV failure or due to cor pulmonale (intrinsic lung disease or of pulmonary vasculature)	-RV hypertrophies and dilates in response to ↑ work load -damming back of blood in portosystemic system ⇒ venous hypertension	-pulmonary congestion is minimal b/c ↓ RV output -venous hypertension: hepatosplenomegaly, nutmeg liver (chronic passive venous congestion) which may ⇒ centrilobar necrosis ⇒ cardiac sclerosis renal congestion ⇒ Na and water retention; peripheral subcutaneous edema, pleural and pericardial effusions, ascites hypoxic encephalopathy

<b>Heart Disease</b>	<b>Etiology</b>	<b>Appearance</b>	<b>Diastolic or Systolic?</b>	<b>Indirect Myocardial dysfunction</b>	<b>Clinical</b>
<p><b>Dilated Cardiomyopathy (Congestive Cardiomyopathy)</b></p> <p>90% of cardiomyopathies</p>	<p>Idiopathic (20% familial with abnormalities of cytoskeletal proteins); late stage myocarditis (infectious, autoimmune); drug toxicity (alcohol or doxorubicin); sarcoidosis, hemochromatosis, peripartum</p>	<p>-progressive hypertrophy and dilation of all 4 chambers -large, flabby, hypocontractile heart -histo: myocyte hypertrophy, interstitial fibrosis</p>	<p>SYSTOLIC dysfunction ⇒ cardiac failure ↓↓↓ ejection fraction</p>	<p>Ischemic HD Valvular HD Hypertensive HD Congenital HD -can all result in dilated pattern</p>	<p>-death from cardiac failure, thromboembolus, or ventricular arrhythmias -tx: transplantation or ventricular assist device</p>
<p><b>Hypertrophic Cardiomyopathy</b> (asymmetric septal hypertrophy, idiopathic hypertrophic subaortic stenosis, hypertrophic obstructive cardiomyopathy)</p> <p>3-4% of cardiomyopathies</p>	<p>Idiopathic (50% familial autosomal dominant) with mutations in sarcomeric proteins (cardiac myosin, tropomyosin, troponin T); amyloidosis; glycogen storage disease; friedrich's ataxia</p>	<p>-massive myocardial hypertrophy in absence of an identifiable extrinsic stress; most pronounced in LV and most pts have disproportionate thickening of ventricular septum -heavy, muscular, hypercontractile heart -histo: massive myocyte hypertrophy, myocyte disarray, interstitial fibrosis, endocardial fibrosis of LV outflow tract</p>	<p>DIASTOLIC dysfunction b/c thick walled ventricle is abnormally stiff and impairs diastolic filling ↓ EDV, ↓ SV -may also be dynamic obstruction to LV outflow</p>	<p>Hypertensive HD Valvular/sub-valvular aortic stenosis -can result in hypertrophic pattern</p>	<p>-variable prognosis: ↑ risk of atrial fibrillation, thromboembolism, cardiac failure, myocardial ischemia (angina)</p>
<p><b>Restrictive Cardiomyopathy (constrictive cardiomyopathy; obliterative cardiomyopathy)</b></p> <p>1-2% of cardiomyopathies</p>	<p>Idiopathic; deposition disease (amyloid, sarcoidosis, neoplasm, hemochromatosis); radiation fibrosis; endocardial diseases</p>	<p>-normal or slightly enlarged ventricles w/o ventricular dilation -contractile function preserved but stiff and inelastic ventricle ⇒ biatrial dilatation -histo: patchy/diffuse interstitial fibrosis</p>	<p>DIASTOLIC stiff ventricle can be filled only with great effort; impaired diastolic relaxation ⇒ Impeded ventricular filling</p>	<p>Pericardial constriction -can result in restrictive pattern</p>	

