VALVULAR HEART DISESASE

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GENERAL PRINCIPLES OF VALVE MALFUNCTION:

VALVULAR STENOSIS:

Pressure in upstream chamber >> Pressure in downstream chamber *during time of flow* (when valve is normally open).

Hemodynamic Hallmark = "PRESSURE GRADIENT"

VALVULAR REGURGITATION: (Also termed "INSUFFICIENCY" or "INCOMPETENCE")

Retrograde flow of blood "upstream" *during time when valve is normally closed*.

Hemodynamic Hallmark = "VOLUME OVERLOAD"

EXAMPLES OF VALVULAR HEART DISEASE

MITRAL STENOSIS:

Pathophysiology:

Obstruction of blood flow from LA to LV during diastole, causing increased pressure in the left atrium, pulmonary capillaries and, eventually, the right side of the heart. (see fig 1 and fig 1A)

As the valve area (i.e. the cross-sectional area of the valve opening during diastole) becomes smaller, the pressure gradient increases. The relationship between the mitral valve area, the forward cardiac output and the pressure gradient across the valve during diastole is complex, and is defined by this equation:

$$\text{MITRAL VALVE AREA} = \frac{\dfrac{\text{CARDIAC OUTPUT (ml/min)}}{\text{DIASTOLIC FILLING PERIOD (sec/min)}}}{\text{SQ ROOT of PRESSURE GRADIENT (mm Hg)}}$$

Examine the relationship between mitral valve gradient and flow (cardiac output) for various valve areas. (Fig 2) As valve area gets smaller, conditions which increase valve flow (exercise, tachycardia) result in an increase in LA pressure (and hence worsening symptoms i.e. DYSPNEA).

Increased LA pressure -> Pulm HTN -> RV Pressure Overload -> RV failure & Tricuspid Regurgitation

Etiology: Rheumatic Heart Disease

Symptoms:

Dyspnea, Orthopnea, PND
Cough & Hemoptysis
Atrial Fibrillation, LA thrombus and systemic Embolization
RV failure, TR and systemic congestion

Physical Signs:

Diastolic murmur (low-pitched rumble) & Opening Snap

Treatment:

Medical Rx:

Digitalis -- controls heart rate and maintains sinus rhythm

Anticoagulants -- prevent systemic embolization

Diuretics -- reduce pulmonary vascular congestion Surgical Rx

Invasive Rx:

Balloon mitral valvulotomy

Surgical Rx:

Mitral valve commissurotomy
or
Mitral valve replacement

MITRAL REGURGITATION:

Pathophysiology:

LV ejects blood antegrade into the Aorta *and* retrograde into the LA.

COMPENSATORY MECHANISMS:

Frank-Starling: Increased preload --> LV Dilatation --> increased stroke volume
Initially -- Increased LV Ejection Fraction --> increased stroke volume
(Eventually -- LV systolic function deteriorates and CHF ensues)

The severity of MR and the ratio of forward cardiac flow (cardiac output) to backward flow are determined by several, interacting factors:

1) the size of the mitral orifice during regurgitation
2) the systemic vascular resistance opposing forward flow from the ventricle
3) the compliance of the left atrium
4) the systolic pressure gradient between the LV and the LA
5) the duration of regurgitation during systole (not all regurgitation is holo-systolic)

Effect of MR on LA pressure depends on LA Compliance (see Figure 3)

ACUTE MR (non-compliant LA)	CHRONIC MR (compliant LA)
Normal LA size	Dilated LA
Increased LA pressure	LA pressure normal or sl inc
"V" waves on PCW tracing	Absence of V waves
Pulmonary Edema	Low output state

Anatomic structures integral to MV competence:

Posterior LA wall, Ant & Post valve leaflets, chordae tendinae, papillary muscles and their attachment to LV wall.

Etiology:

Myxomatous degeneration (MV prolapse)

Coronary artery disease (ischemic papillary muscle dysfunction)

Infectious endocarditis (acute and chronic)

Chronic rheumatic heart disease

Marked LV enlargement from any cause (e.g. dilated cardiomyopathy)

Ruptured papillary muscle &/or chordae tendinae

Hypertrophic Cardiomyopathy with obstruction

Mitral annular Ca++

Congenital cleft MV, etc.

Symptoms:

Acute MR: Pulmonary Congestion

Chronic MR: weakness, fatigue and low output state
± A. Fib., systemic emboli, RV failure (less common than with MS)

Natural Hx => gradual LV dilatation and failure

Physical findings:

Holosystolic apical murmur
± S3 gallop
± laterally displaced apical impulse

Treatment:

Medical Rx:

Digitalis for atrial arrhythmias (a. fib)

Anticoagulation to prevent systemic embolization

Diuretics to reduce pulmonary congestion

Vasodilators to reduce afterload (impedance to LV ejection)

Surgical Rx: Mitral valve replacement or repair

AORTIC STENOSIS:

Pathophysiology:

Obstruction to LV outflow during systole.
Pressure gradient across the aortic valve (pressure higher in LV than aorta during systole), causes chronic LV "Pressure Overload".
Compensatory concentric left ventricular hypertrophy --> reduced LV compliance.
Thus, LV is "stiff" (noncompliant) and LVEDP rises rapidly with increases in LV end-diastolic volume.

Classification:

Valvular -> Rheumatic, Bicuspid, Degenerative Calcific
Subvalvular -> Fixed or Dynamic (ASH)
Supravalvular-> Congenital (rare)

The relationship between the aortic valve area, the forward cardiac output and the pressure gradient across the valve is defined by this equation (similar to the equation for mitral stenosis):

$$\textbf{AORTIC VALVE AREA} = \frac{\frac{\text{CARDIAC OUTPUT ml/min}}{\text{SYSTOLIC EJECTION PERIOD sec/min}}}{\text{SQ ROOT OF SYSTOLIC PRESSURE GRADIENT}}$$

Comparison with Mitral Stenosis:

Aortic valve area smaller than MV area

Systolic Ejection Period << Diastolic Filling Period

Thus Aortic Stenosis has higher pressure gradients and valve flow than Mitral Stenosis (see Fig 4)

SYMPTOM TRIAD:

ANGINA PECTORIS (due to increased MV02)

DYSPNEA ON EXERTION and CHF

SYNCOPE

PHYSICAL FINDINGS:

Pulsus parvus et tardus (carotid pulsation is weak and delayed)
Systolic Ejection Murmur: diamond-shaped, peaks in mid-late systole
2nd heart sound: single or paradoxically split

TREATMENT:

Medical Rx:

Digitalis, diuretics, Na+ restriction, reduced activity

Surgical Rx:

Aortic Valve Replacement

AORTIC INSUFFICIENCY:

Pathophysiology:

Part of the blood that the LV ejects into the Aorta during systole returns to the LV in diastole across an incompetent valve.
This results in "volume overload" of the left ventricle.
Variable pathophysiology depending on acute versus chronic AI (see fig 5).

Compensatory Mechanisms include:

LV Dilatation due to LV "Volume Overload"

"Eccentric" LV Hypertrophy (dilation and hypertrophy)

Two hemodynamic variables influence the extent of AI:

Heart rate:

Inc HR dec regurg by dec time in diastole
Slow HR inc regurg by inc time in diastole

Pressure gradient between Ao and LV:

Increased BP or peripheral resistance (SVR) will increase regurg, while drugs that dec BP or SVR (like vasodilators) will decrease regurg.

Natural History:

Chronic AI -- long asymptomatic period as LV dilatation occurs (significant LV dysfunction may antedate symptoms).

Acute AI -- Acute LV failure and Pulm Edema (without time for LV dilatation). Often refractory to Med Rx and requires emergency aortic valve replacement.

Etiology:

Diseases of Aortic valve leaflets:

Rheumatic Heart Disease

Bicuspid Aortic Valve

Infectious Endocarditis

Diseases of the Aortic Root:

Cystic Medial Necrosis (Marfan's Syndrome)

Syphilis

Ankylosing Spondylitis and other Connective Tissue Disorders (Reiter's Synd, Rheum Arth, SLE, etc.).

Trauma and Aortic Dissection

Symptoms:

LV failure: dyspnea, orthopnea, etc.

Physical signs:

Hyperdynamic pulses, head bobbing, water hammer pulse, "Quincke's" pulse, Duroziez's murmur

Widened pulse pressure (diastolic blood pressure is less than half of the systolic blood pressure: e.g.: BP = 140/50)

Decrescendo Diastolic Murmur -- Increases with increased SVR (handgrip, squatting)

Austin Flint murmur -- diastolic rumble (differentiate from murmur of mitral stenosis).

TREATMENT

Medical Rx: Vasodilators -- Dec systemic vascular resistance

Digitalis and Diuretics - once heart failure ensues

Surgical Rx: Aortic valve replacement (timing of valve replacement crucial to prevent irreversible LV failure.

FIGURES

Figure 1

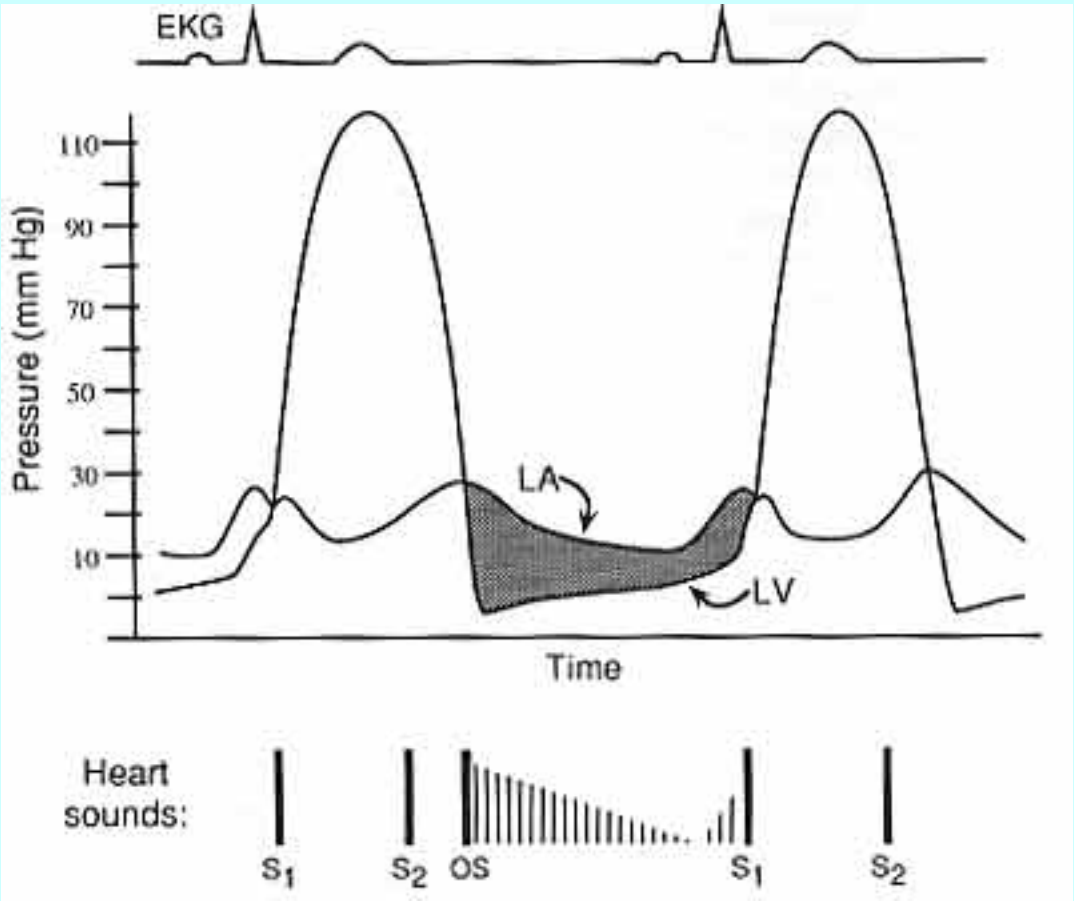
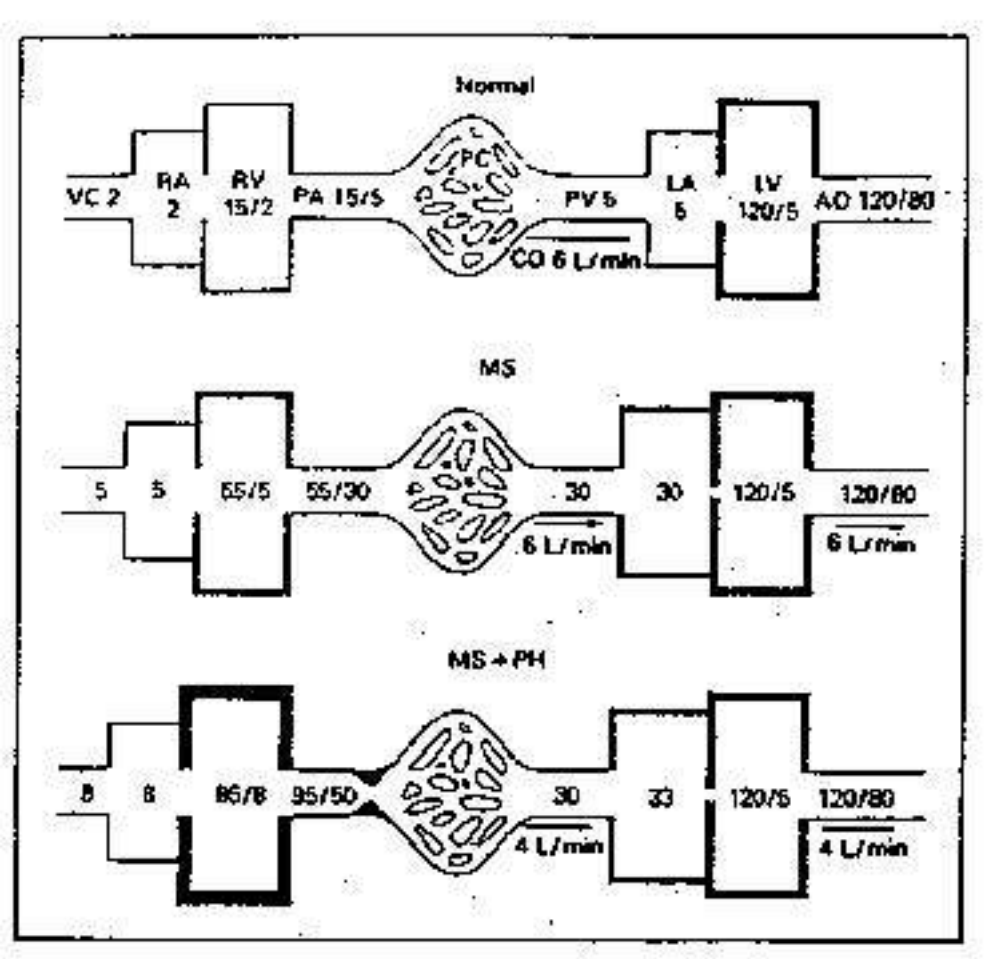


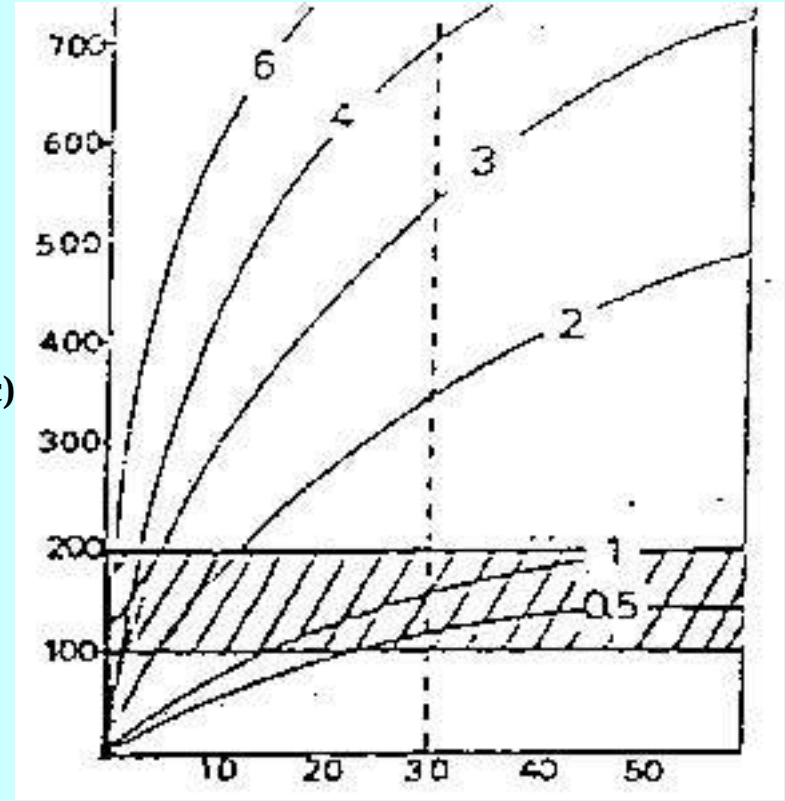
Fig 1: Hemodynamic profile of mitral stenosis. The left atrial (LA) pressure is elevated, and there is a pressure gradient (shaded area) between the LA and left ventricle (LV) during diastole. Abnormal heart sounds are present: there is a diastolic opening snap (OS) that corresponds to opening of the mitral valve, followed by a decrescendo murmur. There is accentuation of the murmur just before S1, due to the increased pressure gradient when the LA contracts. EKG, electrocardiogram.

Figure 1a: Pathophysiology of mitral stenosis (MS)



The top panel demonstrates normal hemodynamics. The middle panel shows a patient with severe mitral stenosis but without reactive pulmonary hypertension (PH). Left atrial pressure is 30mm Hg and there is a 25-mm gradient (30 - 5 mm Hg) across the mitral valve during diastole. The increased left atrial pressure is transmitted to the pulmonary capillaries (PC) and pulmonary artery (PA) and results in an increase in pulmonary arterial pressure to 55/30 mm Hg. Right ventricular systolic pressure must therefore increase to 55 mm Hg as well. There is slight dilatation and hypertrophy of the right ventricle (RV) and left atrium (LA). In the lower panel is depicted an individual with severe reactive pulmonary hypertension. Mitral stenosis is no more severe than in the patient depicted in the middle panel. However, a pulmonary hypertensive reaction has developed. The right ventricle is hypertrophied and dilated. The right ventricle has failed with right atrial and central venous pressures rising to 8 mm Hg and cardiac output falling to 4 liters/minute.
VC = vena cava; RA = right atrium; PV = pulmonary veins; LV = left ventricle; AO = aorta: CO = cardiac output.

Figure 2



Relationship between mitral valve gradient, flow rate and different valve areas.
The crosshatched area indicates the range of normal resting flow values. The vertical line represents the threshold for developing pulmonary edema. The pressure gradient increases as flow rate increases, to a small degree with a normal mitral valve (area = 4 - 6 cm2), to a greater degree with a stenotic valve. With severe stenosis, a substantial gradient is present even at rest.

Figure 3 -- Pathophysiology of acute and chronic mitral regurgitation

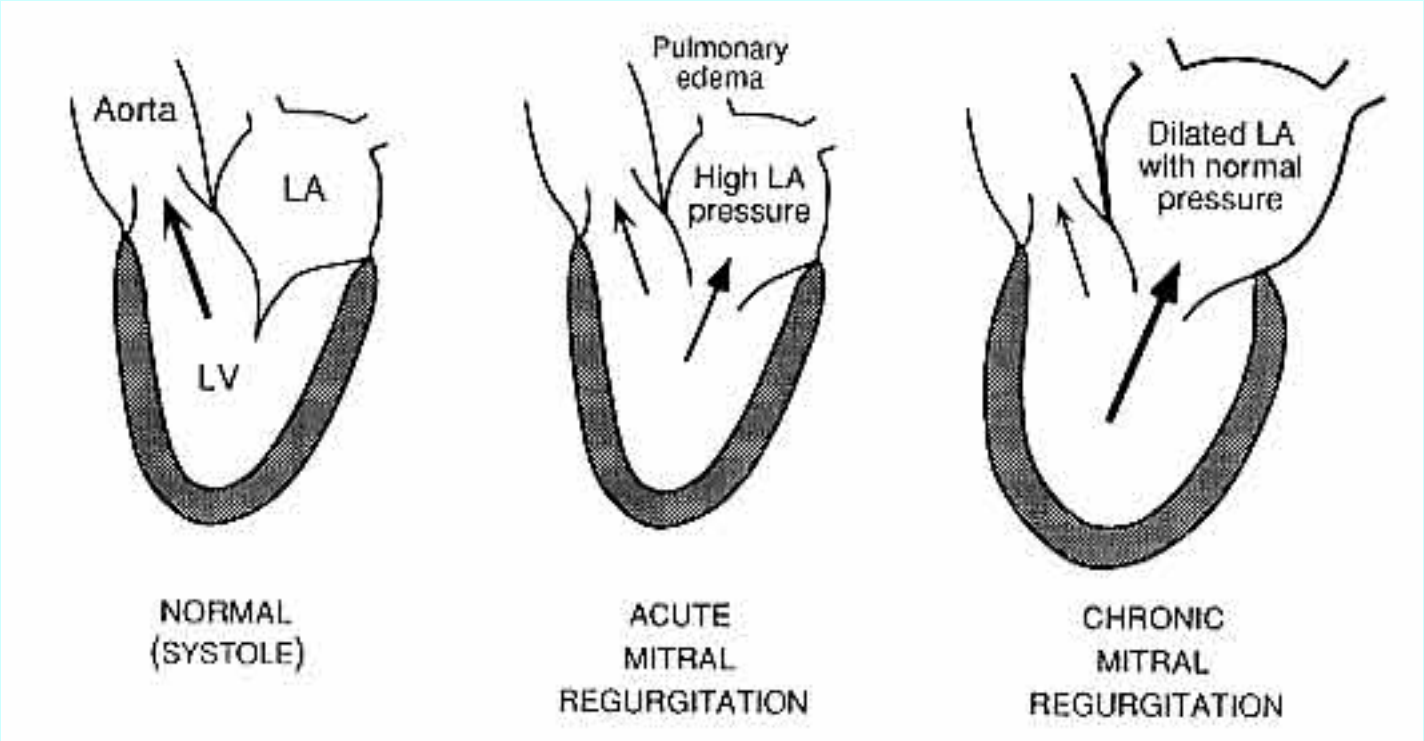


Fig 3: Pathophysiology of mitral regurgitation. In the normal heart, left ventricular (LV) contraction during systole forces blood exclusively through the aortic valve into the aorta; the closed mitral valve prevents regurgitation into the left atrium (LA). In mitral regurgitation (MR), a portion of the LV output is forced retrograde into the LA, so that forward cardiac output into the aorta is reduced. In acute MR, the LA is of normal size and is noncompliant, such that the LA pressure rises markedly and pulmonary edema may result. In chronic MR, the LA has enlarged and is more compliant, such that LA pressure is less elevated and pulmonary congestive symptoms are less common if LV contractile function is intact. There is LV enlargement and eccentric hypertrophy due to the chronic increased volume load.

Figure 4 -- Hemodynamics of aortic stenosis: Relationship between valve flow rate and mean systolic pressure gradient for various aortic valve areas. The cross-hatched area indicates the range of normal resting flow rates. The vertical dashed line indicates a pressure gradient of 50-mm Hg. Low pressure gradients are noted with normal valve areas even at high flow rates. Significant reduction in valve area results in substantial pressure gradients with normal or even depressed values of resting flow.

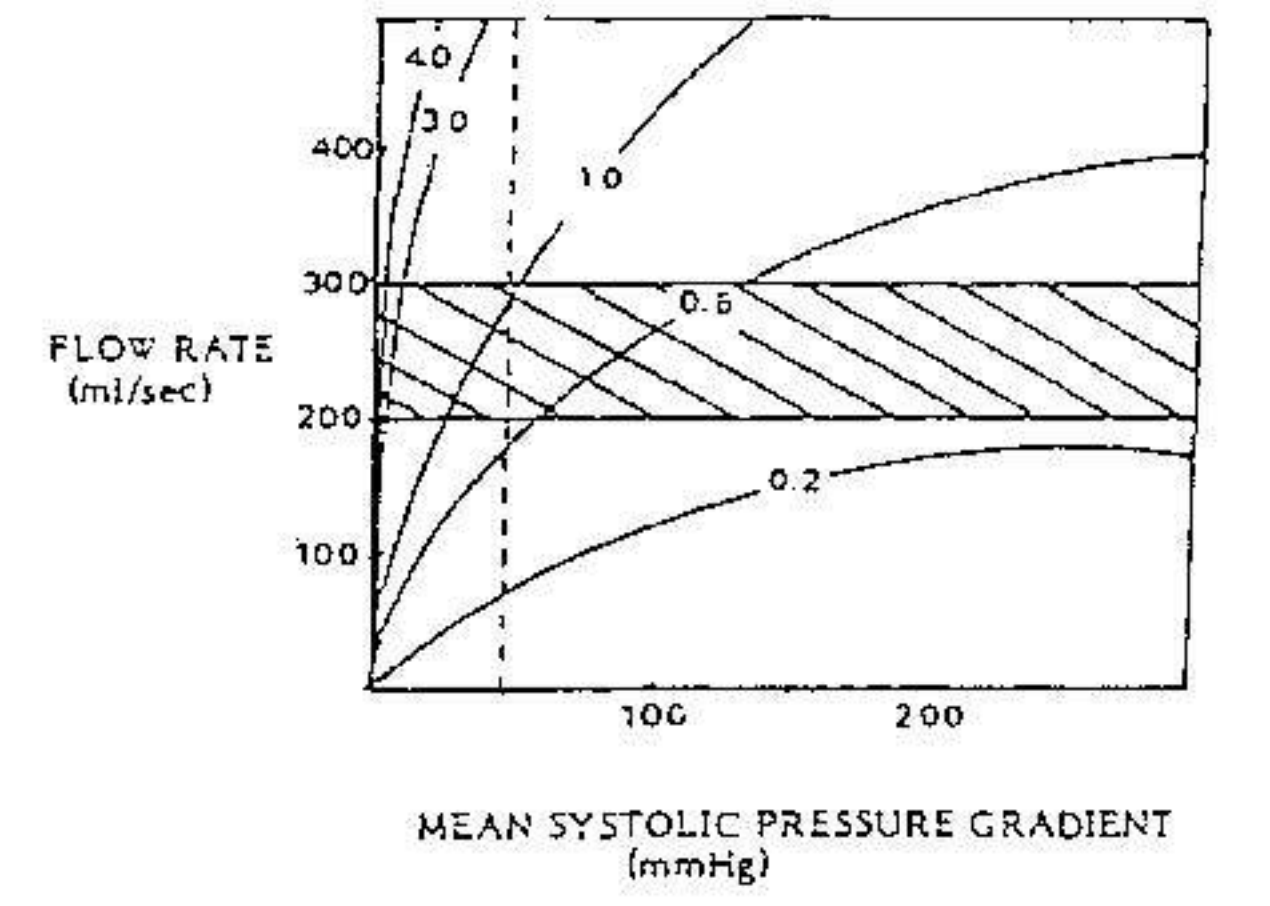
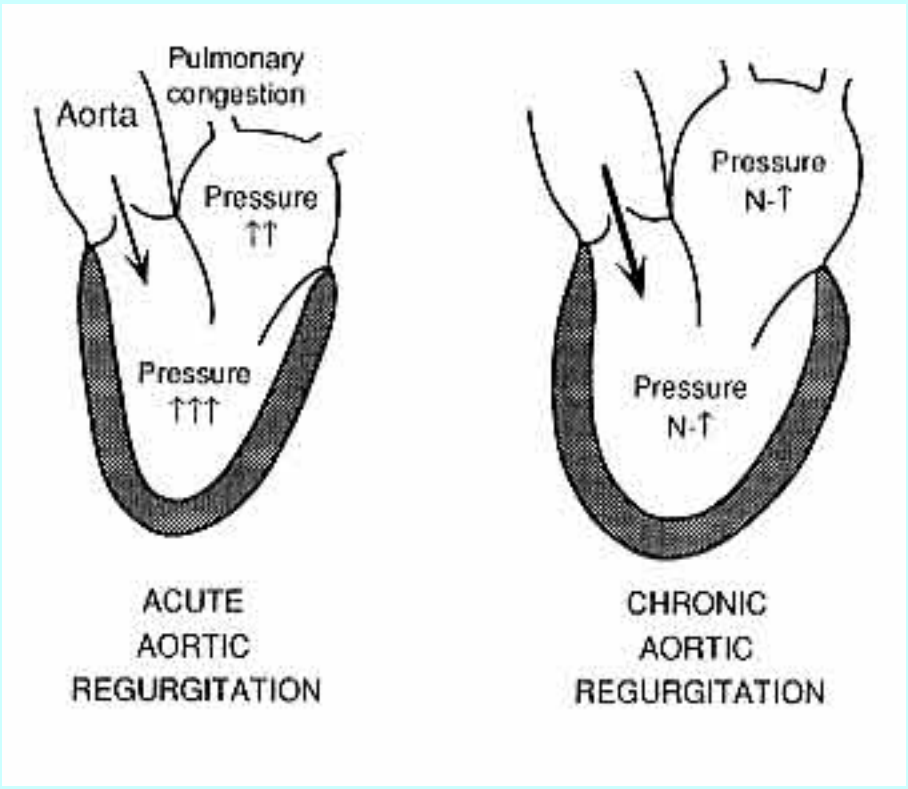


Figure 5.
Pathophysiology of acute and chronic aortic regurgitation (AR).
Abnormal regurgitation of blood from the aorta into the left ventricle (LV) is shown in each schematic drawing (large arrows). In acute AR, the LV is of normal size and relatively low compliance, such that its diastolic pressure rises markedly; this is reflected back to the left atrium (LA) and pulmonary vasculature, resulting in pulmonary congestion or edema. In chronic AR, adaptive LV and LA enlargement have occurred, such that a greater volume of regurgitation can be accommodated with less of an increase in diastolic LV pressure, so that pulmonary congestion is less likely. N, normal.


Pulmonary congestion
Aorta
Pressure ↑↑
Pressure ↑↑↑
ACUTE AORTIC REGURGITATION
Pressure N-↑
Pressure N-↑
CHRONIC AORTIC REGURGITATION