

Valvular Heart Disease

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Introduction

- remarkable changes in the evaluation and management of patients with valvular heart disease
- Advances in surgical approaches and interventional cardiology procedures have improve patient's outcomes



Valvular heart disease

Mitral

Aortic

Tricuspid

Pulmonary

Stenosis

Regurgitation



Diagnosis

- Etiology and pathology
- Pathophysiology
- Clinical manifestation
- Physical Findings
- Laboratory Examination
- Differential Diagnosis
- Treatment



Key Concepts

- Echocardiography remains the gold standard for diagnosis and follow up patients with valvular heart disease.
- Stenotic valvular lesions can be monitored clinically until symptoms appear
- Regurgitant valvular lesions require careful echocardiographic monitoring for left ventricular function and may require surgery even if no symptoms are present



- **Medical therapy aims at control of symptoms.**
- **Surgery is the treatment for most symptomatic lesions or for lesions causing left ventricular dysfunction even in the absence of symptoms.**



MITRAL STENOSIS (MS)



ETIOLOGY AND PATHOLOGY

- Rheumatic fever
- Two-thirds are female
- 25% of all patients have pure MS
- 40% have combined MS and mitral regurgitation (MR)
- the incidence of MS is declining
- A major problem in tropical climates and developing countries



- **Rheumatic fever results in four forms of fusion leading to stenosis:**
 - **Commissural**
 - **Cuspal**
 - **Chordal**
 - **Combined**
- **mitral valve cusps fuse at the their edges**
- **fusion of the chordae tendineae results in thickening and shortening of these structures**



- **Calcification of the valve immobilizes the leaflets and narrows the orifice**
- **lead to narrowing of the valve (fish-mouth)**
- **dilated left atrium (LA)**
- **Thrombus frequently arise from LA in patients with atrial fibrillation (AF)**
- **Other causes: congenital, malignant carcinoid, SLE, Amyloid, etc.**



PATHOPHYSIOLOGY

- mitral valve orifice is 4 to 6 cm²
 - mild: 1.5-2cm²
 - moderate: 1-1.5cm²
 - Severe (critical): <1cm²
- transvalvular pressure gradient, pulmonary venous and arterial wedge pressures elevated - exertional dyspnea



- **Tachycardia augments the transvalvular gradient and LA pressure**
- **the CO is abnormal at rest and may fail to rise or may even decline during activity in patients with severe MS**
- **Pulmonary hypertension**



CLINICAL MANIFESTATIONS

- **As MS progresses**
 - **No symptoms**
 - **Dyspnea, cough**
 - **Orthopnea**
 - **Paroxysmal nocturnal dyspnea**
- **Hemoptysis**
 - **rupture of pulmonary-bronchial venous connections**



- **Systemic Embolism**

- more frequent in patients with AF

- **Other Symptoms**

- Hoarseness (Ortner syndrome)
- hepatomegaly, edema, ascites, hydrothorax (right-sided heart failure)



Physical Findings

- **Inspection and Palpation**

- **mitral facies**
- **prominent jugular venous pulse**
- **RV tap-left sternal border**
- **diastolic thrill-apx**
- **Hepatomegaly, ankle edema, ascites, and pleural effusion in patients with MS and RV failure**



● Auscultation

- S1, P2 is accentuated
- The opening snap (OS) follows A2
- low-pitched, rumbling, diastolic murmur at the apex in the left lateral recumbent position
- The Graham Steell murmur of pulmonary regurgitation (PR) results from dilatation of the pulmonary valve ring

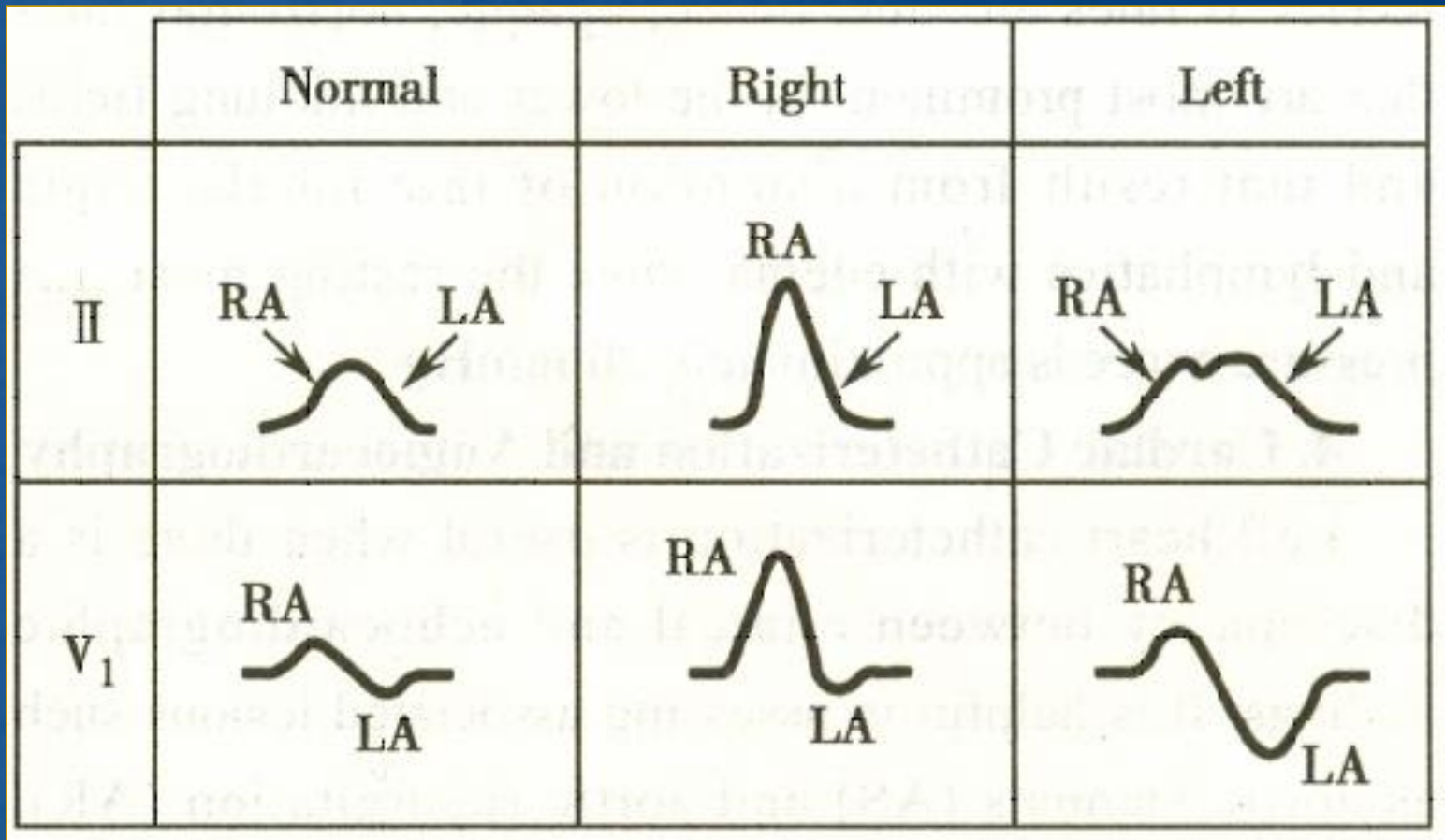


Laboratory Examination

- ECG

- P wave duration in lead II >0.12 s
- P wave axis between $+45$ and -30 degrees
- suggests LA enlargement
- Atrial fibrillation



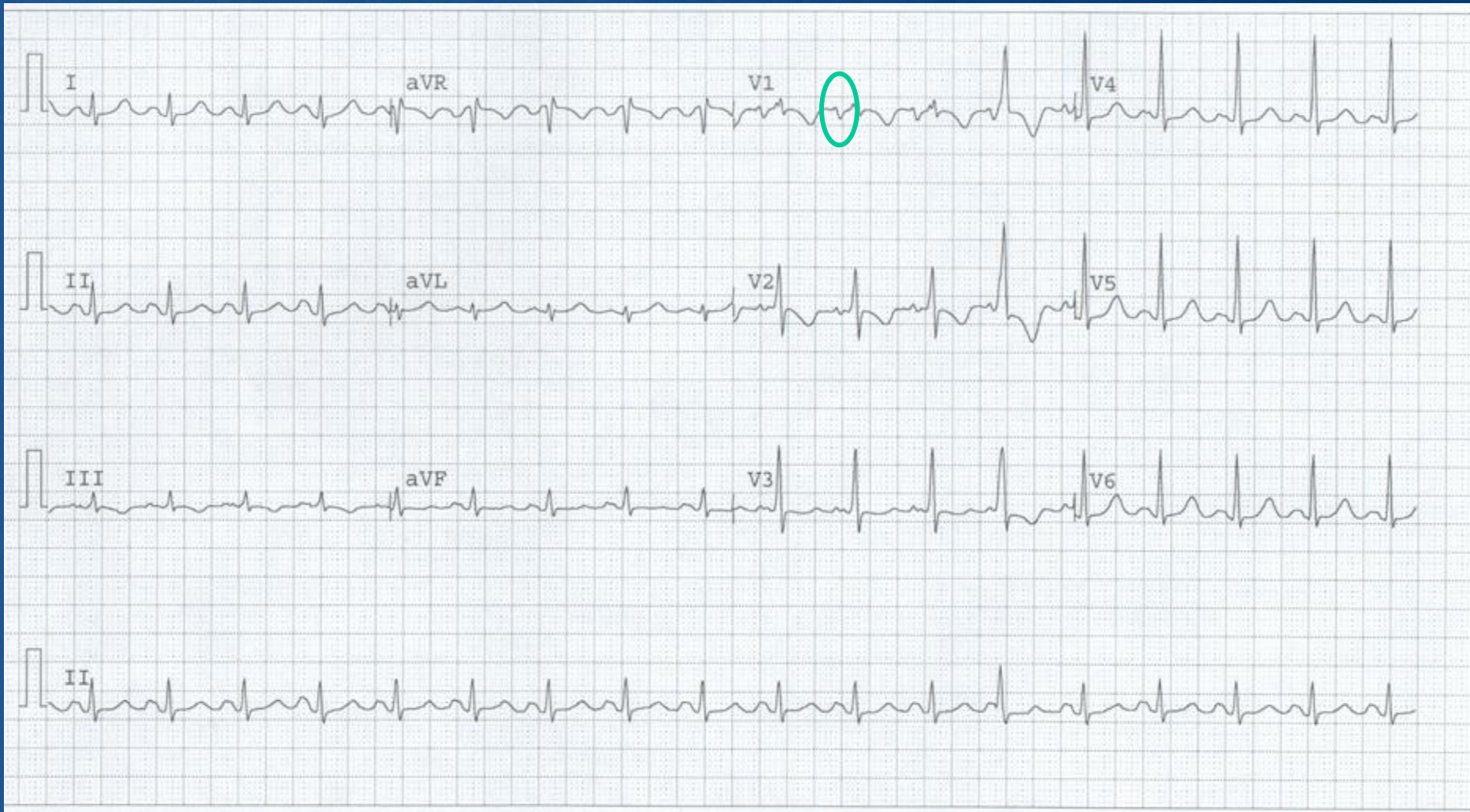


RV overload- tall P wave

LA enlargement-notched, broad P wave



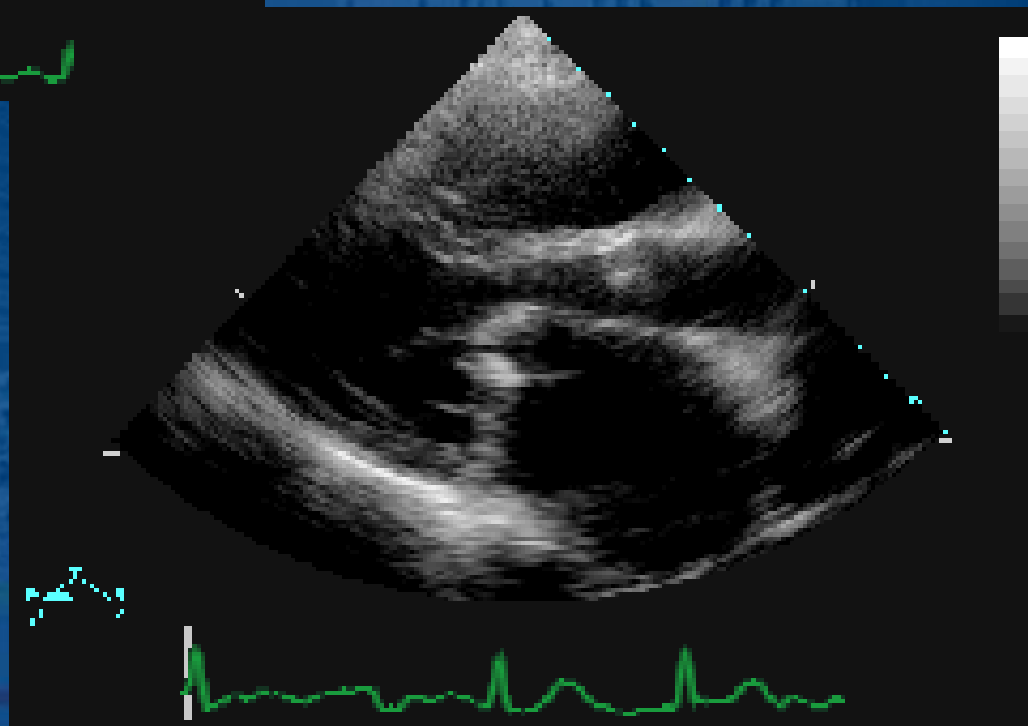
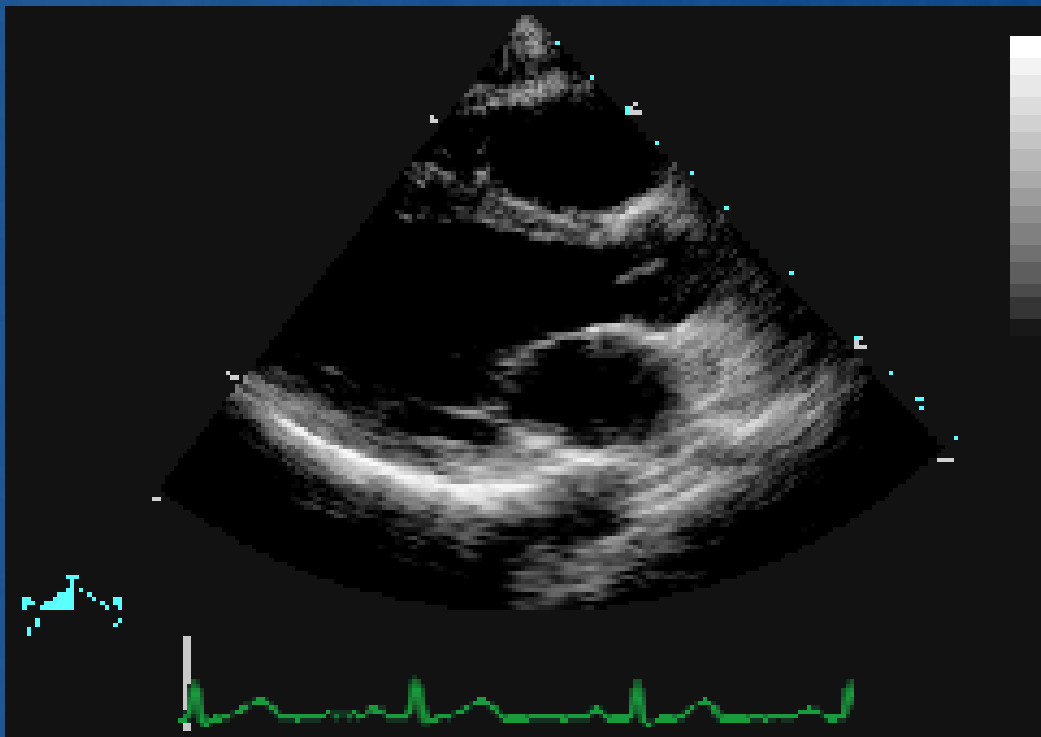
Biphasic P wave in V1

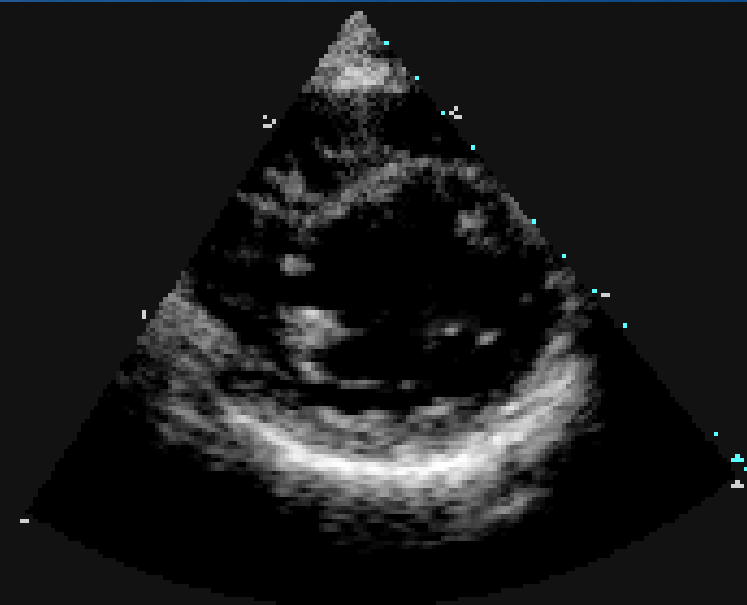


● Echocardiography

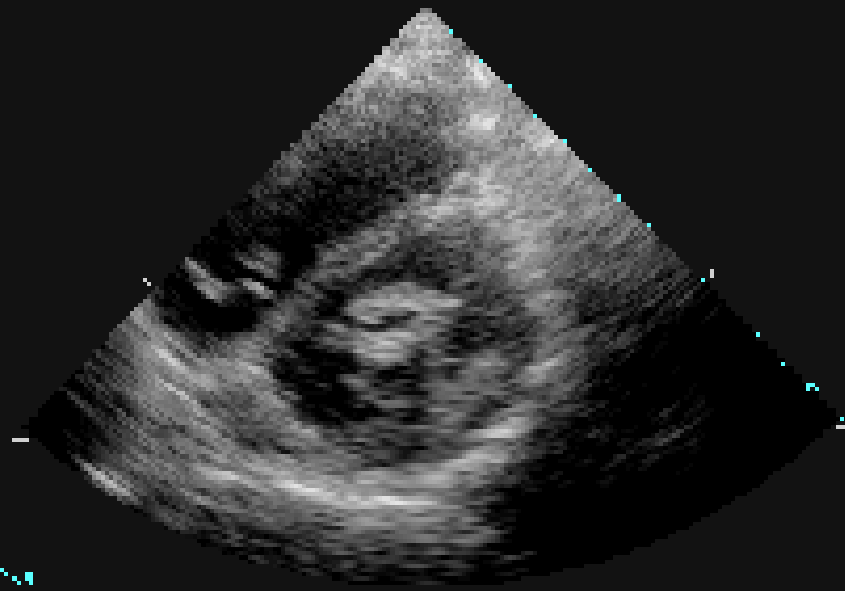
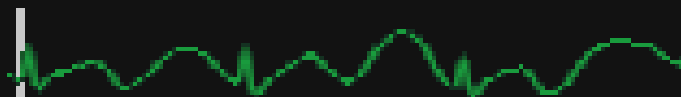
- most sensitive and specific noninvasive method for diagnosing MS
- mitral orifice size, the presence and severity of accompanying MR, the extent of restriction of valve leaflets
- cardiac chamber size, the LV function



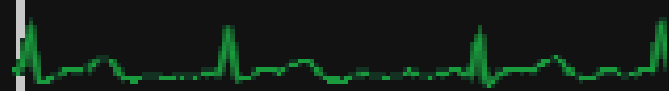




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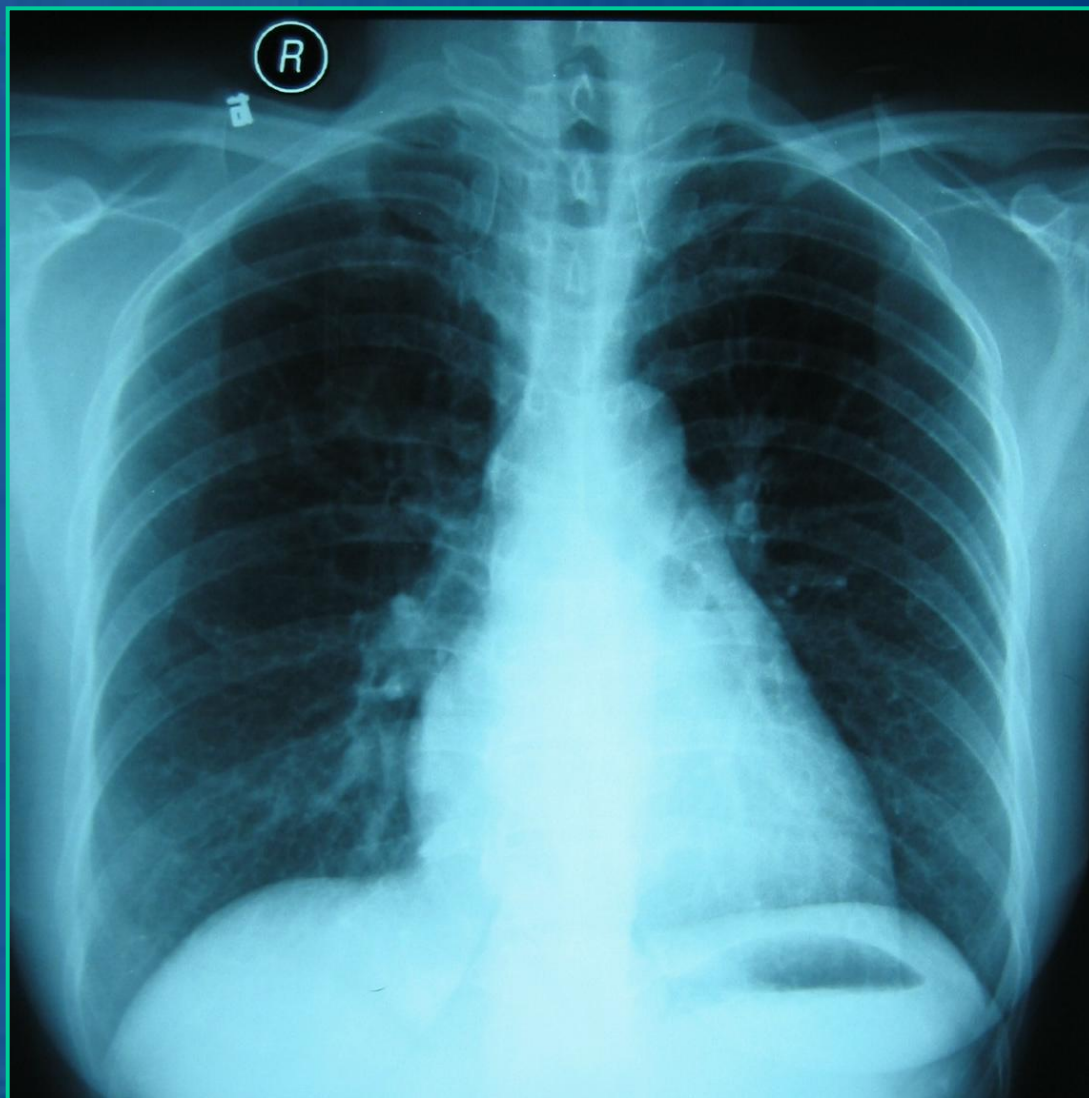
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● X-ray (Roentgenogram)

- straightening of the left border of the cardiac silhouette
- prominence of the main pulmonary arteries
- enlarged LA
- Kerley B lines





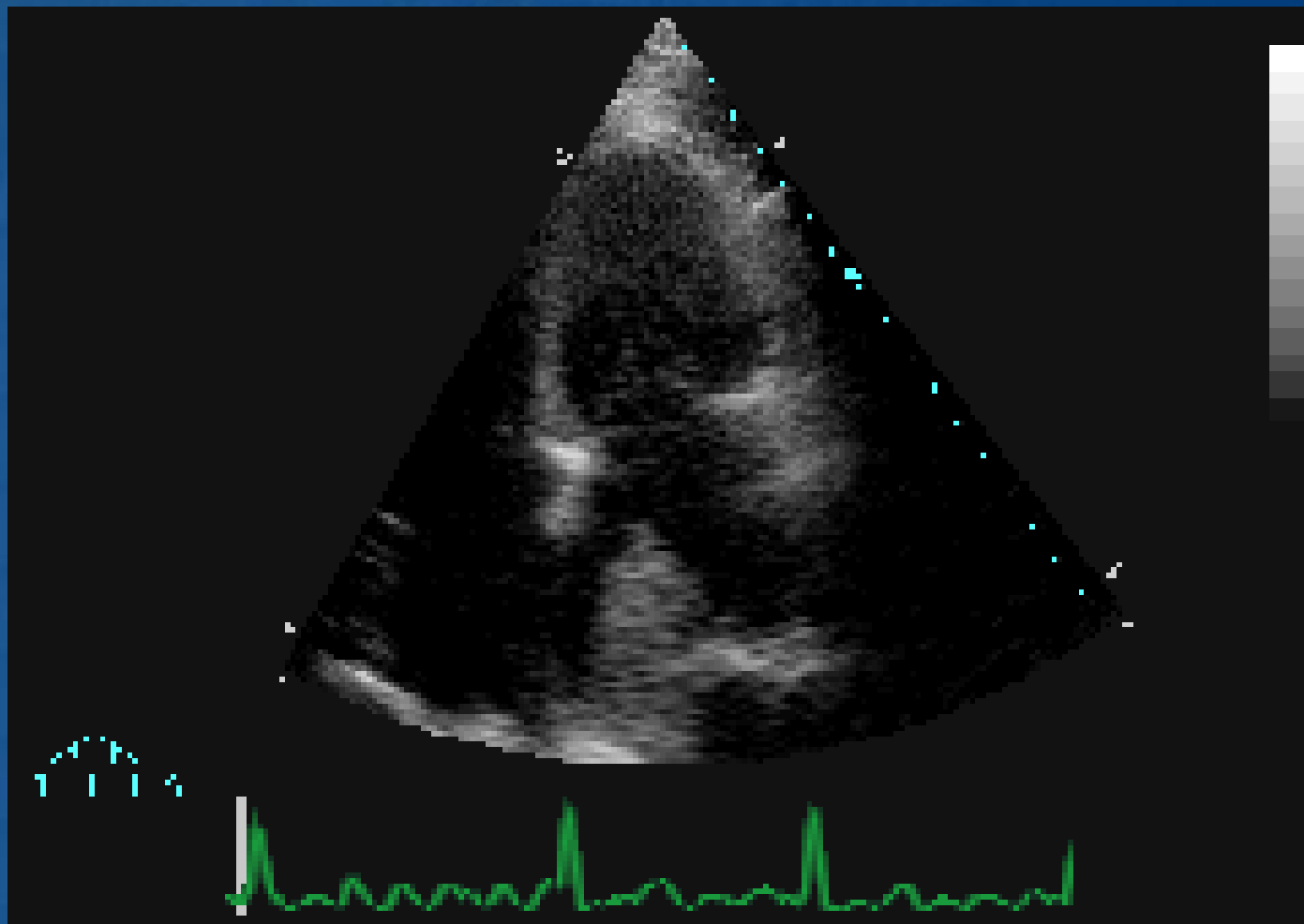
- **Cardiac Catheterization and Angiocardiology**
 - **not usually necessary**
 - **positive noninvasive stress tests for myocardial ischemia, coronary angiography is advisable to detect patients with critical coronary obstructions in males >45 years of age, females > 55 years of age, or younger with risk factors**



Differential Diagnosis

- MR
- AR (Austin Flint murmur)
- Atrial septal defect
- Left atrial myxoma





Treatment

- Penicillin prophylaxis to prevent rheumatic fever and infective endocarditis
- restriction of sodium intake and maintenance doses of oral diuretics
- Digoxin, Beta blockers
- Warfarin for systemic and/or pulmonary embolization and AF



- **Recent, mild MS with AF**
- **reversion to sinus rhythm
pharmacologically or by electrical shock**

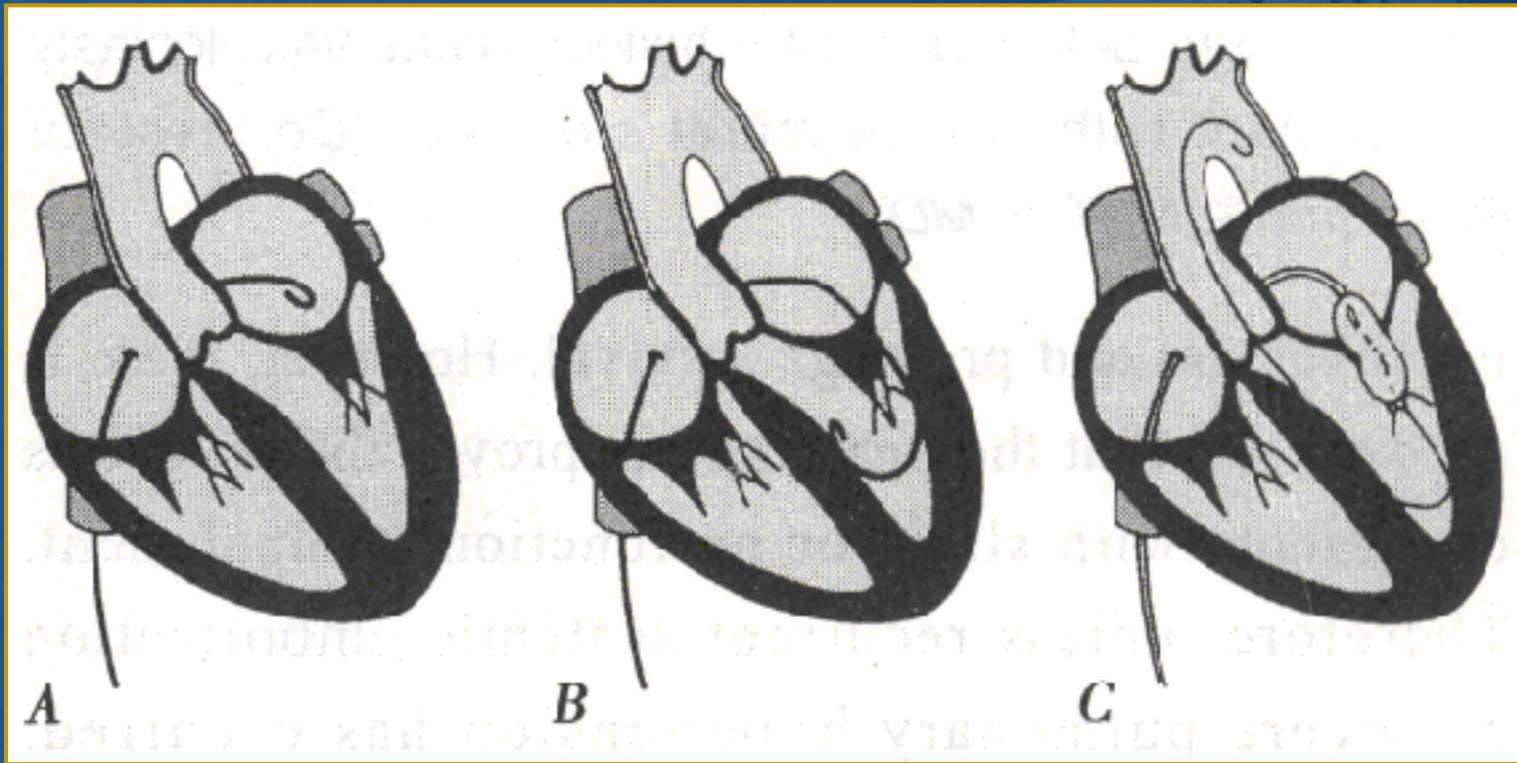


Mitral Valvotomy

- percutaneous balloon mitral valvotomy (PBMV)
- surgical valvotomy
- for symptomatic patients with isolated MS whose orifice is $< 1.0 \text{ cm}^2/\text{m}^2$ body surface area, or $< 1.7 \text{ cm}^2$ in normal-sized adults



PBMV



- **Mitral valve replacement (MVR) is necessary in patients with MS and significant associated MR**



MITRAL REGURGITATION



ETIOLOGY

- abnormal mitral leaflets, chordae tendineae, papillary muscles, and mitral annulus
- mitral valve prolapse (MVP), rheumatic heart disease, infective endocarditis, annular calcification, cardiomyopathy, and ischemic heart disease



PATHOPHYSIOLOGY

- **increased LV and LA volume**
- **elevated LA and PA pressure**
- **reduced forward CO**
- **ejection fraction (EF) rises**
- **with longstanding MR, LV contractility becomes reduced**



SYMPTOMS

- **Fatigue**
- **exertional dyspnea**
- **orthopnea**
- **Acute severe MR--- acute pulmonary edema**



PHYSICAL FINDINGS

- **S1 absent**
- **A holosystolic murmur of grade III/VI intensity is the most characteristic auscultatory finding, most prominent at the apex and radiates to the axilla**



- MVP of posterior mitral leaflet, the regurgitant jet strikes the LA wall, the systolic murmur is transmitted to the base of the heart
- "sea gull" murmur--ruptured chordae



LABORATORY EXAMINATION

- **EKG**
 - **LA, LV, RV enlargement**
 - **AF**
- **ECHO: 2D, color doppler**
 - **most accurate noninvasive technique for diagnosis of MR**



TREATMENT

- **Medical Treatment**
 - **reducing sodium intake**
 - **use of diuretics**
 - **vasodilators and digitalis**
 - **(ACE) inhibitors**
 - **Intravenous nitroprusside or nitroglycerin reduce afterload for patients with acute and/or severe MR**

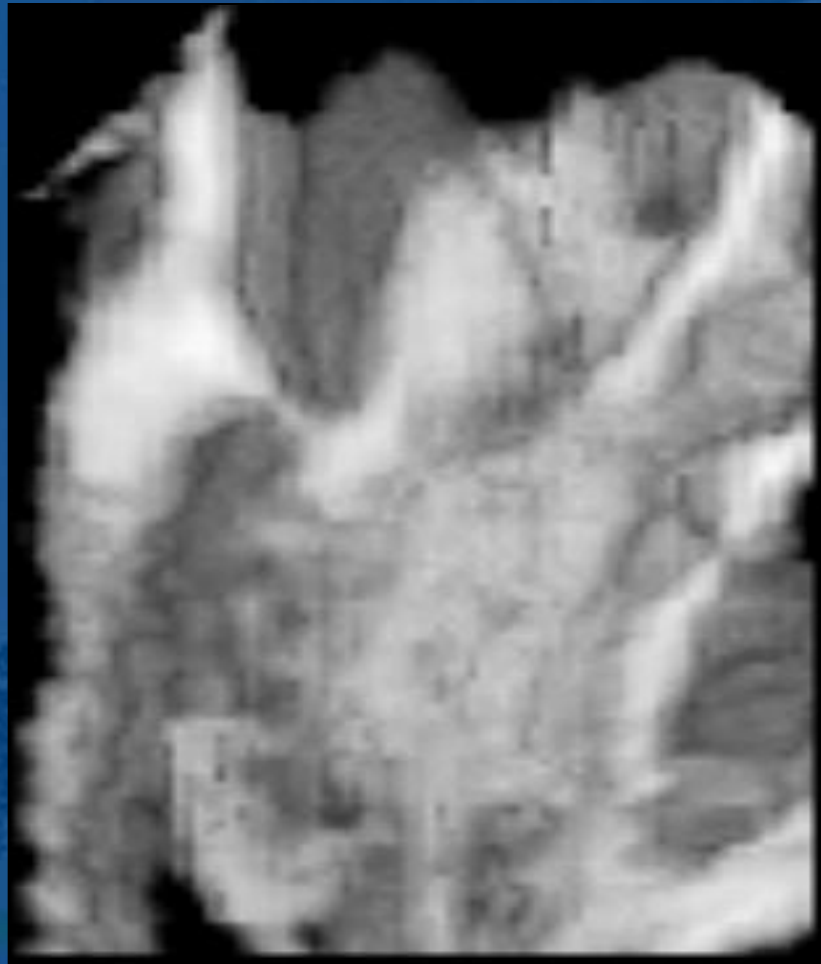


● Surgical Treatment

- asymptomatic or limited only during strenuous exertion, LV functions are normal—no surgery
- severe MR in asymptomatic patients, or LV dysfunction is progressive, with LV EF <60%, and/or end-systolic cavity dimension >45 mm—surgery
- MVR , mitral valvuloplasty/annuloplasty



MR



心研所



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MITRAL VALVE PROLAPSE (MVP)

- **systolic click-murmur syndrome**
- **Barlow's syndrome**
- **floppy-valve syndrome**
- **billowing mitral leaflet syndrome**



- **excessive or redundant mitral leaflet associated with myxomatous degeneration**
- **The posterior leaflet is usually more affected than the anterior**
- **the mitral valve annulus dilated**
- **rupture or redundant chordae tendineae**
- **cause mitral regurgitation**



CLINICAL FEATURES

- more common in females, 14-30 years of age
- a broad spectrum of severities
 - mild prolapse, only a systolic click and murmur
 - chordal rupture, severe MR
- arrhythmias, syncope, chest pain, infective endocarditis, TIA, sudden death



● Auscultation

- the mid- or late systolic click after the S1 generated by the sudden tensing of elongated chordae or by the prolapsing mitral leaflet when it reaches its maximum excursion.
- may be followed by a high-pitched, late systolic murmur at the apex



LABORATORY EXAMINATION

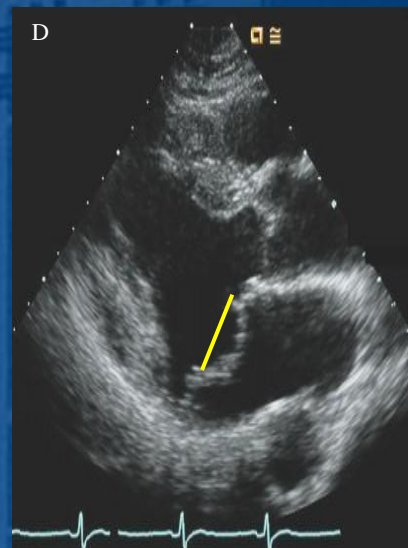
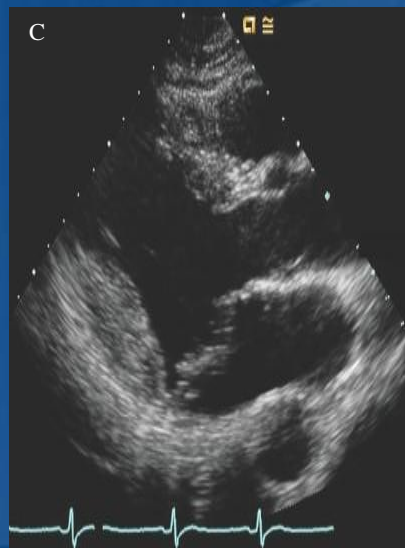
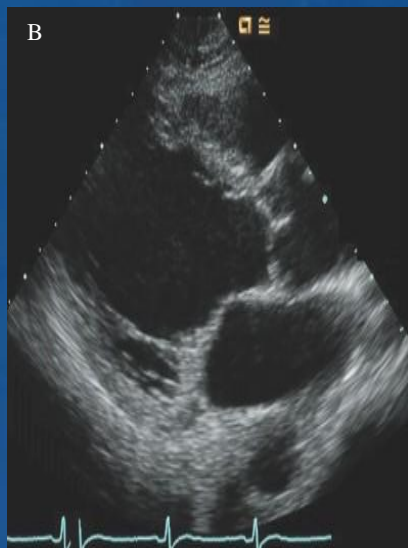
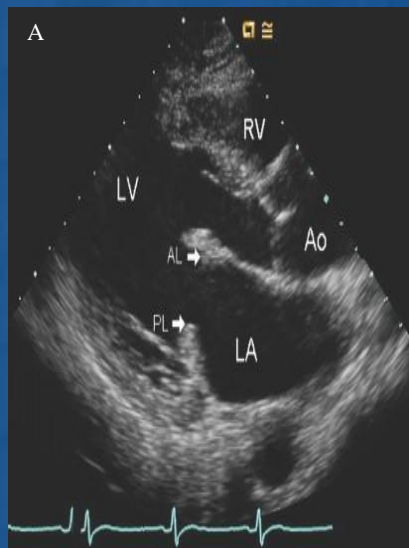
- **EKG**

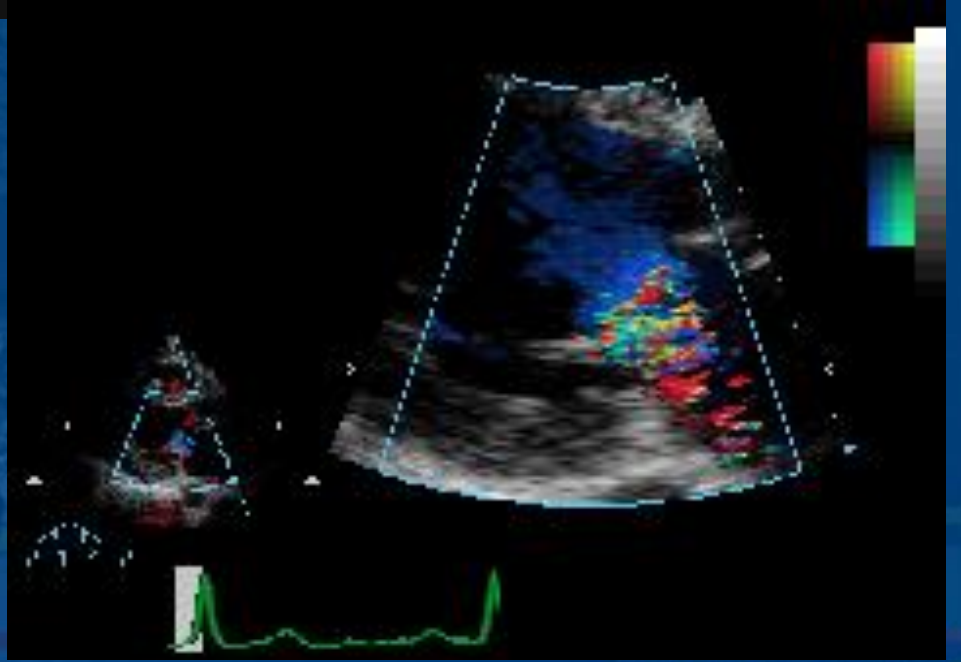
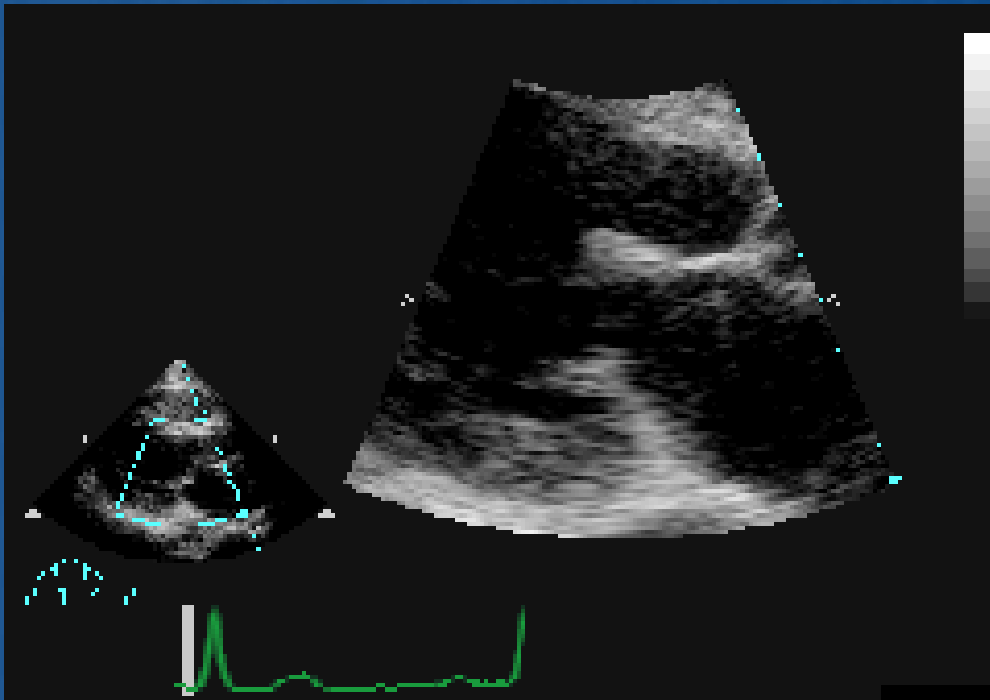
- **biphasic or inverted T waves in leads II, III, and aVF, and premature contractions**

- **ECHO**

- **systolic displacement (in the parasternal view) of the mitral valve leaflets by at least 2 mm into the LA superior to the plane of the mitral annulus**







TREATMENT

- prevention of infective endocarditis
- Beta blockers, antiarrhythmic agents
- Aspirin for TIA patients
- For severe MR, mitral valve repair/replacement



AORTIC STENOSIS



AS

- one-fourth of all patients with chronic VHD
- 80% of adult patients with symptomatic valvular AS are male



ETIOLOGY

- degenerative calcification of the aortic cusps
- congenital or rheumatic inflammation



PATHOPHYSIOLOGY

- The obstruction to LV outflow produces a systolic pressure gradient between the LV and aorta
- A peak systolic pressure gradient >50 mmHg, or an effective aortic orifice < 1.0 cm² or <0.6 cm²/m² body surface area,-- severe obstruction



- **elevated LV end-diastolic pressure**
- **hypertrophied LV wall**
- **diminished compliance of LV wall**
- **LA, PA, and RV pressures rise**
- **myocardial ischemia**



SYMPTOMS

- exertional dyspnea
- angina pectoris
- syncope
- LV failure in the advanced stages of the disease



Auscultation

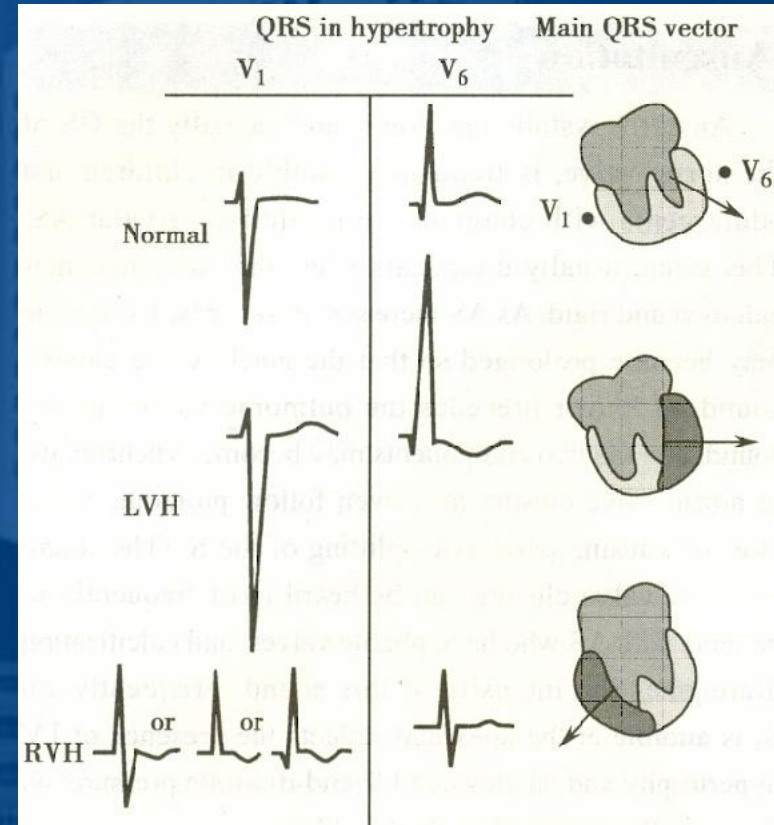
- an early systolic ejection sound
- an ejection (mid) systolic murmur, low-pitched, rough and rasping in character, and loudest at the base of the heart, in the second right intercostal space, transmitted upward along the carotid arteries, grade III/VI



LABORATORY EXAMINATION

- **EKG**

- might be normal
- LV hypertrophy
- ST-segment depression and T-wave inversion



- **Echocardiography**

- **LV hypertrophy**
- **valvular calcification**
- **transaortic valvular gradient**
- **MS, AR**

- **Catheterization**

- **CAD suspected**



● **NATURAL HISTORY**

- **angina pectoris, 3 years;**
- **syncope, 3 years;**
- **dyspnea, 2 years;**
- **congestive heart failure, 1.5 to 2 years**



TREATMENT

- **Medical Treatment**
 - strenuous physical activity should be avoided in patients with severe AS (<0.5 cm²/m²)
 - avoid volume depletion
 - statins may be helpful to slow progression



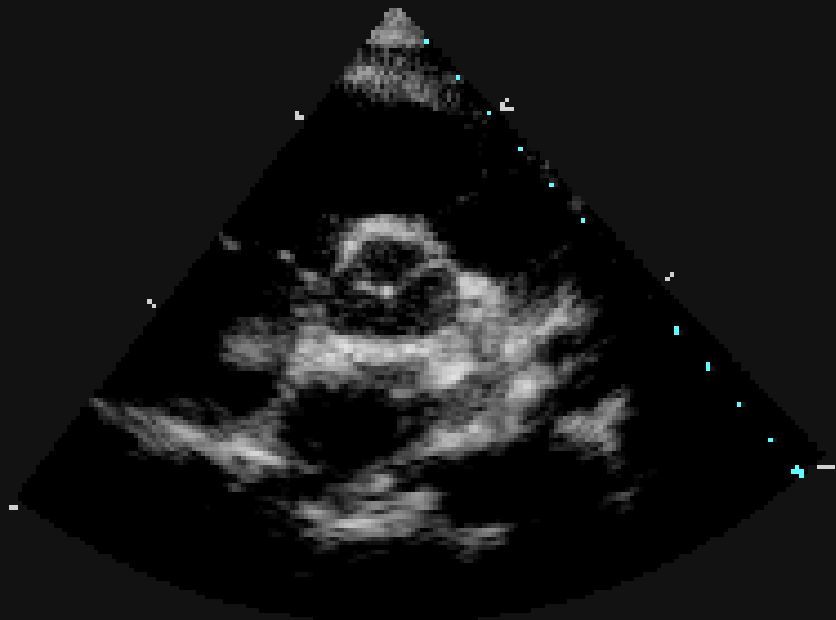
- **Surgical Treatment**

- severe AS (valve area $<1.0 \text{ cm}^2$ or $0.6 \text{ cm}^2/\text{m}^2$ body surface area) who are symptomatic
- LV dysfunction
- expanding poststenotic aortic root, even asymptomatic.

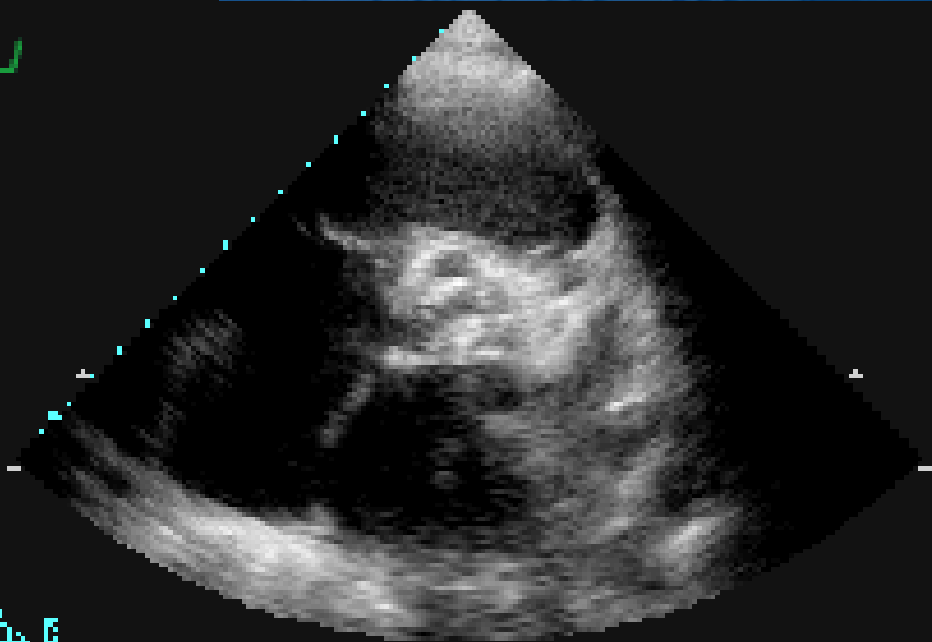
- **Percutaneous Balloon Aortic Valvuloplasty**

- in children and young adults with congenital, noncalcific AS

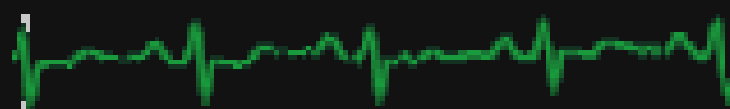




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AORTIC REGURGITATION



ETIOLOGY

- **Primary Valve Disease**
 - three-fourths are males
 - two-thirds is rheumatic in origin
 - degenerative calcification, congenital or rheumatic
 - thickening, deformity, and shortening, prolapse of the aortic valve cusps
- **Primary Aortic Root Disease**
 - aortic root/annulus dilatation
 - Marfan syndrome, hypertension, dissection, syphilis, spondylitis



PATHOPHYSIOLOGY

- **LVEDV↑↑**
- **dilatation and eccentric hypertrophy of the LV**
- **elevation of the LA, PA wedge, PA, and RV pressures**
- **reduced LVEF**
- **myocardial ischemia**



SYMPTOMS

- Acute, severe AR--IE, trauma
 - pulmonary edema, cardiogenic shock may develop rapidly
- Chronic, severe AR--a long latent period
 - palpitation, exertional dyspnea, angina



PHYSICAL FINDINGS

- **Arterial Pulse**

- "water-hammer" (Corrigan's pulse)
- capillary pulsations (Quincke's pulse)
- "pistol-shot" ("pistol-shot")
- to-and-fro murmur (Duroziez's sign)
- arterial pulse pressure is widened



● Palpation

- LV impulse is heaving and displaced laterally and inferiorly
- A diastolic thrill is often palpable along the left sternal border



● Auscultation

- A2 is usually absent
- a high-pitched, blowing, decrescendo diastolic murmur, heard best in the third intercostal space along the left sternal border
- with the patient sitting up, leaning forward, and with the breath held



- **Austin Flint murmur**

- **soft, low-pitched, rumbling middiastolic**
- **diastolic displacement of the anterior leaflet of the mitral valve by the AR stream**
- **Not hemodynamically significant mitral obstruction**



LABORATORY EXAMINATION

- EKG

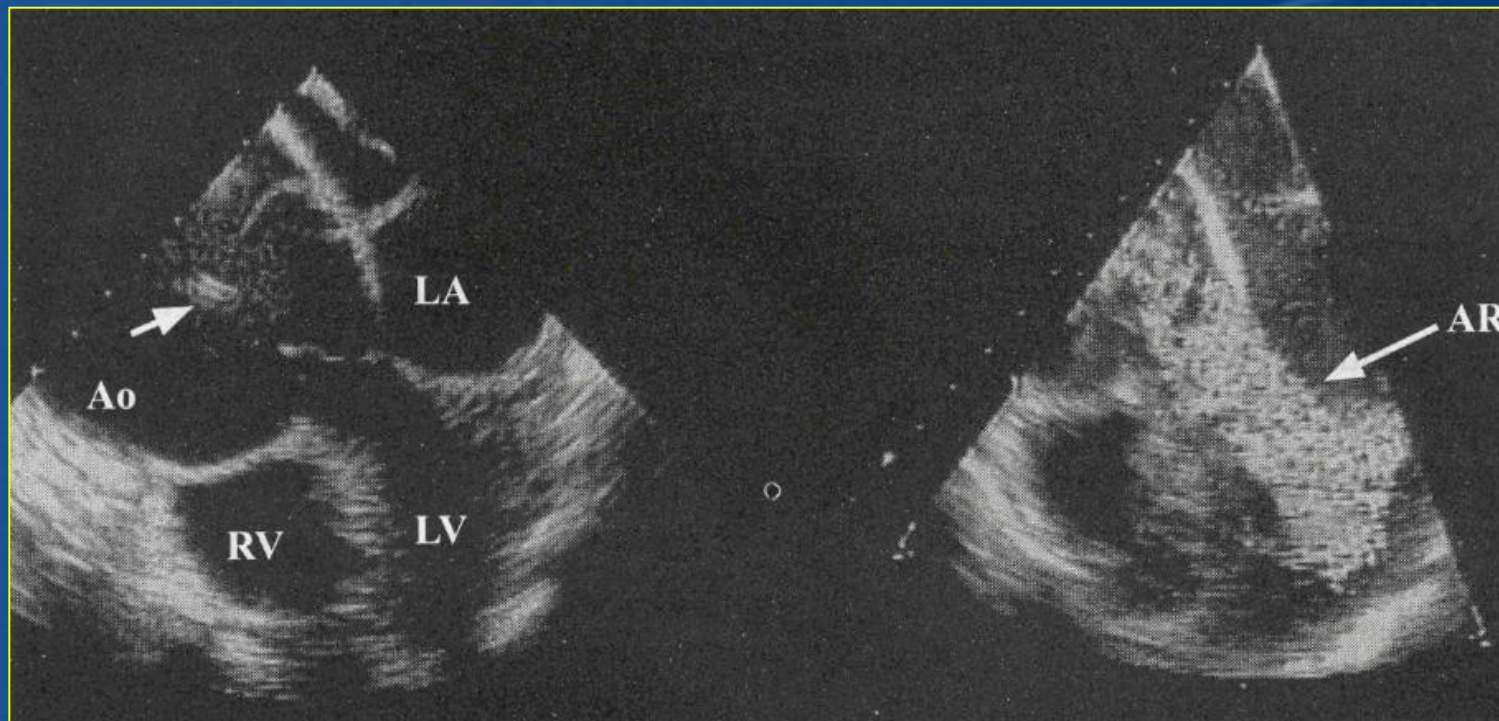
- LV hypertrophy
- ST-segment depression and T-wave inversion leads I, aVL, V5, and V6 ("LV strain")

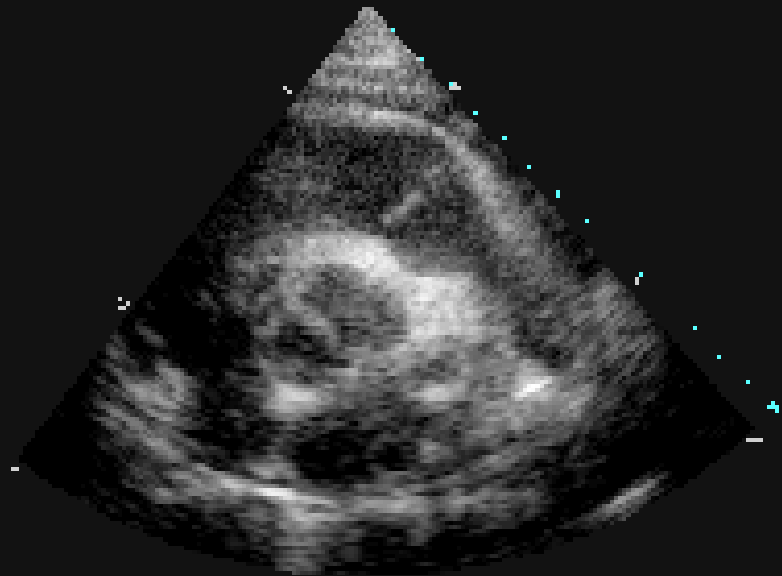


● Echocardiogram

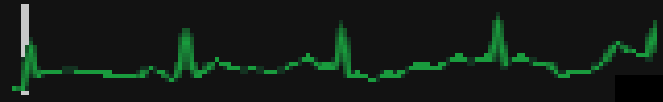
- wall motion are normal or even supernormal
- rapid, high-frequency fluttering of the anterior mitral leaflet
- thickening and failure of coaptation of the leaflets
- dilatation of the aortic annulus
- Color flow Doppler echocardiographic imaging is very sensitive in the detection of AR





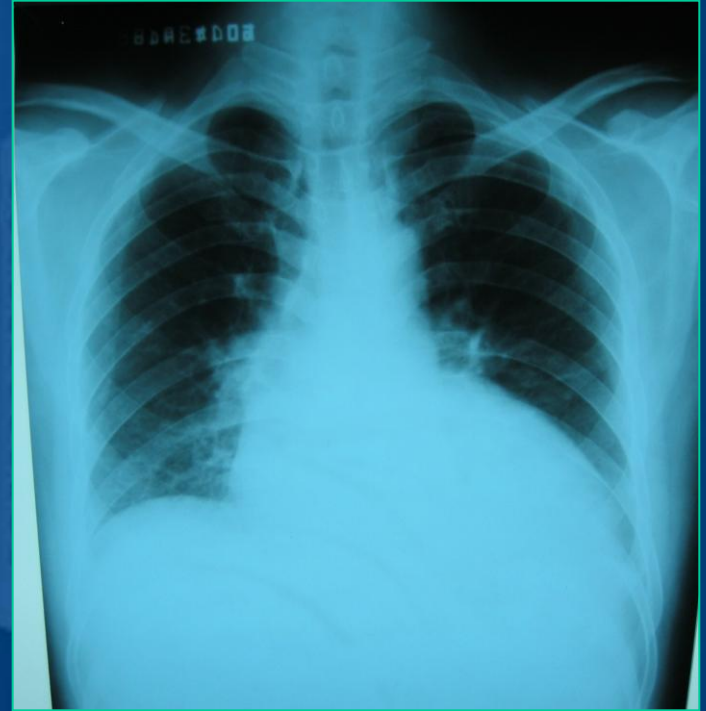


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● X-RAY

- LV enlargement, the apex is displaced downward and to the left
- the ascending aorta and aortic knob may be dilated



TREATMENT

- **Medical Treatment**
 - digitalis, salt restriction, diuretics, and vasodilators, especially ACE inhibitors
 - Cardiac arrhythmias and infections must be treated promptly and vigorously.



● Surgical Treatment

- operation should be carried out in asymptomatic patients, when a left ventricular ejection fraction (LVEF) $<55\%$ or a LV end-systolic volume >55 mL/m².
- AVR, AV repair, narrowing the annulus



TRICUSPID STENOSIS

- **uncommon**
- **generally rheumatic in origin**
- **more common in females than in males**
- **It does not occur as an isolated lesion and is usually associated with MS, TR**



PATHOPHYSIOLOGY

- A diastolic pressure gradient between the RA and RV is elevated
- A mean diastolic pressure gradient of 4 mmHg is sufficient to result in systemic venous congestion
- ascites and edema



SYMPTOMS

- TS can mask the hemodynamic and clinical features of the MS
- Amelioration of MS symptoms should raise the possibility that TS may be developing
- Dyspnea, hepatomegaly, ascites, and edema



PHYSICAL FINDINGS

- **marked hepatic congestion, distended jugular veins, jaundice, splenomegaly**
- **the tricuspid murmur is generally heard best along the left lower sternal margin, augmented during inspiration**



LABORATORY EXAMINATION

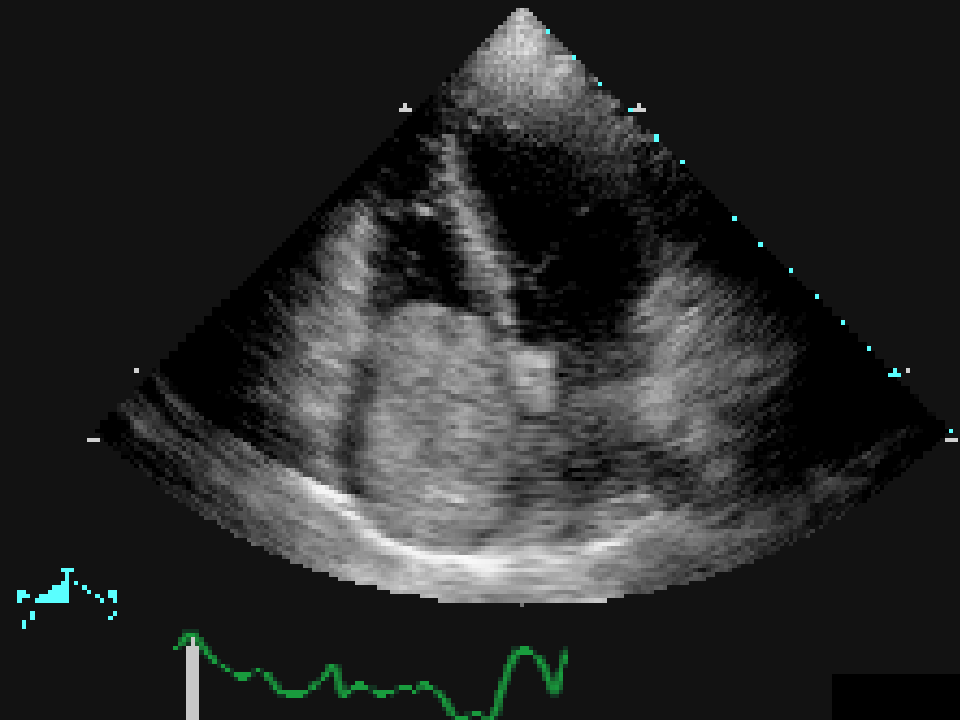
- EKG

- RA enlargement include tall, peaked P waves in lead II, upright P waves in lead V1.

- ECHO

- thickened tricuspid valve; elevated transvalvular gradient





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TREATMENT

- intensive salt restriction and diuretic therapy are required during the preoperative period
- Repair, or replacement



TRICUSPID REGURGITATION (TR)

- Most commonly, TR is functional and secondary to marked dilatation of the tricuspid annulus
- It is commonly seen in the late stages of heart failure with severe pulmonary hypertension, as well as in ischemic heart disease, cardiomyopathy, and cor pulmonale.

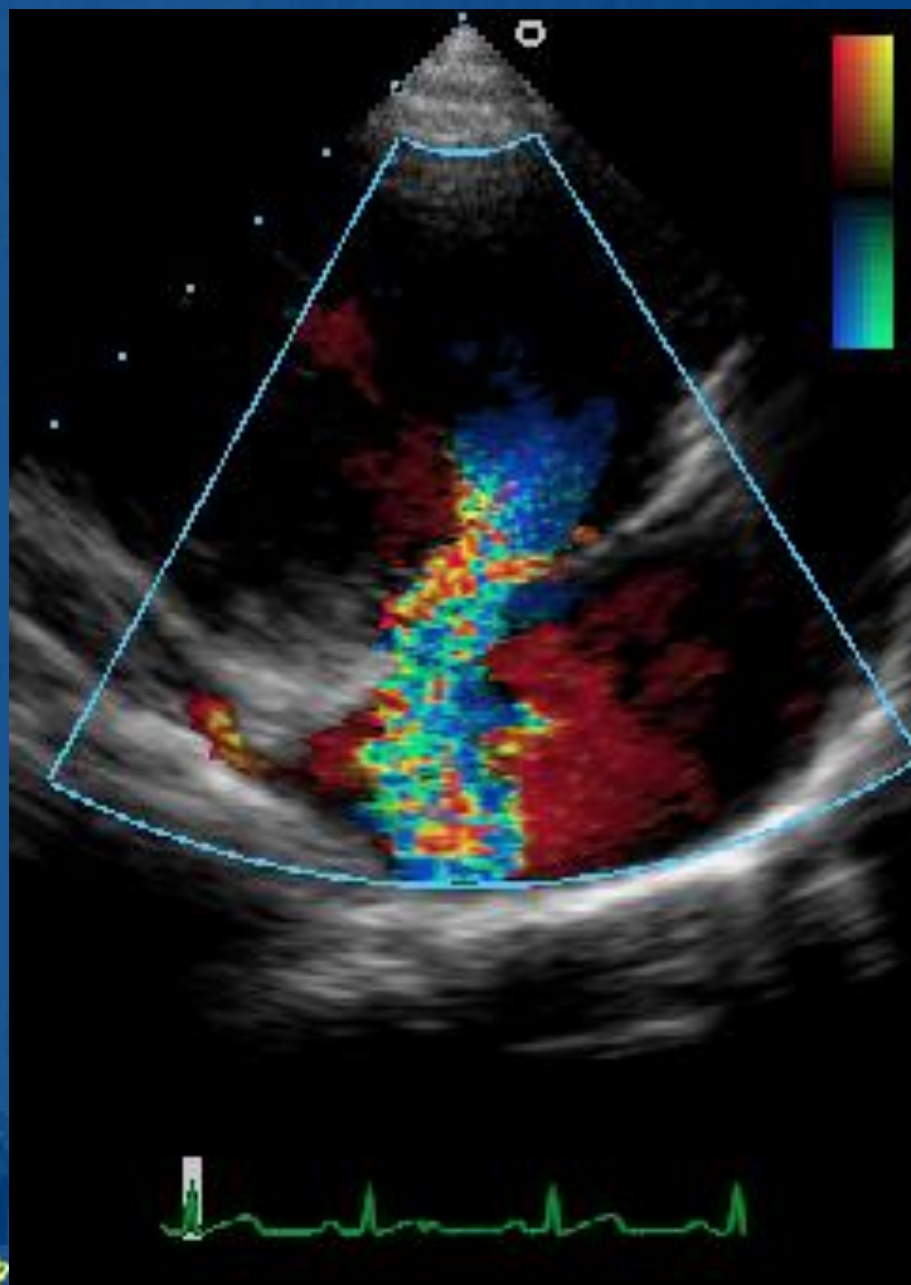


- **Infarction of RV papillary muscles, tricuspid valve prolapse, carcinoid heart disease, endomyocardial fibrosis, infective endocarditis, and trauma**
- **Congenital heart disease: defects of the atrioventricular canal, Ebstein's malformation**



- **Symptoms of right heart failure**
- **A blowing holosystolic murmur along the lower left sternal margin, which may be intensified during inspiration**
- **EKG: RV enlargement, AF**
- **ECHO: RV dilatation, prolapsing of tricuspid leaflets, severity of TR**





TREATMENT

- Isolated TR, in the absence of pulmonary hypertension, may not require operation.
- Treatment of the primary disease, MS
- Tricuspid annuloplasty, tricuspid valve repair/replacement



PULMONIC VALVE DISEASE

- The most common acquired abnormality is regurgitation secondary to dilatation of the pulmonic valve ring as a consequence of severe pulmonary hypertension.
- Graham Steell murmur, a high-pitched, decrescendo, diastolic blowing murmur along the left sternal border
- It is usually of little hemodynamic significance



SUMMARY

- **Etiology**

- **rheumatic**
- **myxomatous degeneration**
- **aging**
- **congenital abnormality**
- **trauma**
- **infective endocarditis**
- **infiltrative diseases**
- **dilation of valve annulus**



PATHOPHYSIOLOGY

- **Preload increases**
 - MR, AR
- **Afterload increases**
 - AS, PS
- **Enlargement of cardiac chamber size**
- **Heart failure**
- **Arrhythmia**



SYMPTOMS

- None
- Nonspecific
- Left heart failure
- Right heart failure
- Arrhythmia



PHYSICAL FINDINGS

- Cardiac size
- S1, S2
- **Murmur**, characteristic
- Heart failure



LABORATORY EXAMINATION

- X-ray
- EKG
- **Echo:** gold standard for diagnosis
- Cath



TREATMENT

- **Medical Treatment**

- **Sodium restriction**
- **diuretics and digitalis**
- **Vasodilators**
- **ACE inhibitor, β -blockers**
- **anticoagulants**
- **Endocarditis prophylaxis**
- **Careful follow-up**



Table 3.6.1 Summary of Useful Medical Treatments in Valvular Heart Disease

Lesion	Symptom Control	Secondary Prevention and Natural History
Mitral stenosis	Diuretics for heart failure; Digoxin, β blockers, and rate-limiting calcium antagonists for rate control in atrial fibrillation	Penicillin prophylaxis against recurrent episodes of rheumatic fever; Anticoagulants to prevent systemic thromboembolism
Mitral regurgitation	Diuretics and vasodilators(usually ACE inhibitors)for heart failure	No proven treatment
Aortic stenosis	Diuretics for heart failure; nitrates and β blockers for angina	No proven treatment but lipid lowering therapy may slow progression of calcific aortic stenosis
Aortic regurgitation	Diuretics and vasodilators(usually ACE inhibitors)for heart failure	Vasodilators(nifedipine or ACE inhibitors)to protect the left ventricular myocardium and delay the need for surgery

Source: NA Boon, P Bloomfield: The medical management of valvular heart disease, Heart 87 : 395, 2002, with permission



- **Surgical treatment:**
 - **Interventional therapy**
 - **Surgical operation**
 - **Valve repair**
 - **Valve replacement**





THANK YOU



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